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Kent Stuber DC, MSc

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The Journal of the Canadian Chiropractic Association is delighted to announce that Dr. Kent Stuber has joined the JCCA as an Assistant Editor.

Dr. Stuber graduated from the University of Calgary with a BSc in Cellular, Molecular & Microbial Biology in 1998, and received his DC from CMCC in 2002, graduating Magna Cum Laude and with Clinic Honours.

In 2008 he received his MSc (with Distinction) in Health and Social Care Research from the University of Sheffield School of Health and Related Research (ScHARR), Sheffield, UK. His dissertation topic was "Chiropractic care for low back pain during pregnancy."

Dr. Stuber is an Editorial Board member for JCCA and a peer reviewer for several journals and scientific conferences including JMPT. He has published in numerous peer-reviewed journals including JMPT, Journal of Chiropractic Humanities, Journal of the Canadian Chiropractic Association, Journal of Chiropractic Medicine, Clinical Chiropractic, Chiropractic & Osteopathy, and Infection and Immunity. His work has been presented at the World Federation of Chiropractic's International Conference on Chiropractic Research and the Association of Chiropractic Colleges Research Agenda Conference.

Dr. Stuber was the recipient of the CCA's Young Investigator Award in 2008. He was also the co-recipient of a significant CIHR peer-reviewed award to support the "Workshop to Advance the Canadian Chiropractic Research Agenda." Recently he was a member of the Program Development Committee for the 2011 workshop entitled "Advancing the Canadian Chiropractic Research Agenda" held in Toronto in September of 2011.

In addition to maintaining clinical practice in Calgary, Alberta, he is an Adjunct Professor at the Canadian Memorial Chiropractic College in the Division of Graduate Education and Research. Previously he worked at the Canadian Chiropractic Examining Board from 2006 until 2010 in different roles including Chiropractic Resource Officer, and Chiropractic Research Officer.

Dr. Stuber's current research interests include pregnancy-related musculoskeletal conditions, the validity and reliability of orthopaedic testing, sports injuries, vertigo, spinal stenosis, and the utilization of nutraceuticals in clinical practice. His preferred research methods include qualitative methods, surveys, and systematic reviews. He is currently co-supervising several student research projects at CMCC.

Dr. Mana Rezai DC, PhD Candidate

Dr. Mana Rezai
University of Toronto

The CCA is delighted to announce that Dr. Mana Rezai has been named a recipient of the 2011 CCA Young Investigator Award.

This award recognizes young researchers working in the field of chiropractic and is given for a paper submitted for this competition that has not yet been published, or for a *recently* published paper. The investigator has not had his/her degree longer than two years before submitting the work.

Dr. Rezai has made very significant and extraordinary contributions to the chiropractic community. Among her many achievements, she recently published in the scientific literature the research paper set out below. As the "co-author", Dr. Rezai has clearly distinguished herself as a young researcher with exceptional skills who can work effectively in a collaborative manner with world class investigators.

Kristman VL, Côté P, Hogg-Johnson S, Cassidy JD, Van Eerd D, Vidmar M, Rezai M, Wennberg RA. The burden of work disability associated with mild traumatic brain injury in Ontario compensated workers: a prospective cohort study. The Open Occupational Health & Safety Journal 2010; 2: 1–8.

Dr. Rezai is an Epidemiology PhD candidate at the Dalla Lana School of Public Health, University of Toronto. Her doctoral dissertation is an examination of the association between work-ability and return-to-work following a traffic injury. Her research goals are to better understand the course and determinants of recovery from traffic injuries in order to implement effective health care interventions, develop return-to-work programs and policies, and promote healthy lifestyle changes. Dr. Rezai's doctoral work takes place at the Toronto Western Research Institute, under the supervision of Dr. Pierre Côté. Her MHSc work included a project at the Institute for Work and Health examining the association between quality of life and neck pain as well as two international projects in Cambodia examining rates of injury, access to care and education for persons with disabilities. In December 2010, Dr. Rezai returned to Cambodia to evaluate access to education barriers for persons with disabilities. This research was funded by CIHR and the International Centre for Disability and Rehabilitation at the University of Toronto.

Congratulations to Dr. Rezai!

Dr. Maja Stupar DC, PhD Candidate

Dr. Maja Stupar
University of Toronto

The CCA is delighted to announce that Dr. Maja Stupar has been named a recipient of the 2011 CCA Young Investigator Award.

This award recognizes young researchers working in the field of chiropractic and is given for a paper submitted for this competition that has not yet been published, or for a *recently* published paper. The investigator has not had his/her degree longer than two years before submitting the work.

Dr. Stupar has made exemplary contributions to the chiropractic community. She recently published the research paper set out below which has had very high impact in the scientific literature. As the "lead author" in this paper, Dr. Stupar has established herself as a very capable young researcher and clearly in a position to collaborate effectively with world class researchers.

Stupar M, Côté P, French MR, Hawker GA. The association between low back pain and osteoarthritis of the hip and knee: a population-based cohort study. J Manip Physiol Ther. 2010 Jun;33(5):349–54.

Dr. Maja Stupar is a doctoral candidate in Clinical Epidemiology at the Department of Health Policy, Management and Evaluation, Faculty of Medicine, University of Toronto. Her thesis addresses measurement of outcomes in musculoskeletal injuries, specifically evaluating psychometric properties of an outcome measure developed for assessing whiplash-related disability and her supervisor is Dr. Pierre Côté.

Dr. Stupar is a CIHR Vanier Canada Scholar and a recipient of the Michael Smith Foreign Study Supplement. Her work at the Karolinska Institute in Sweden assessed an outcome measure in the Swedish adult population with back and neck pain. A collaboration with Dr. Gillian Hawker, a rheumatologist and Chief of Medicine at Women's College Hospital, resulted in a publication in the JMPT in 2010. This paper "The Association Between Low Back Pain and Osteoarthritis of the Hip and Knee: A Population-Based Cohort Study" was awarded the 2nd place World Federation of Chiropractic (WFC) research prize. Dr. Stupar has presented her work at the Primary Care Musculoskeletal Research Congress in Rotterdam, Netherlands and the International Society of Quality of Life Research conference in London, UK.

Congratulations to Dr. Stupar!

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Treatment of post-traumatic myositis ossificans of the anterior thigh with extracorporeal shock wave therapy

David Allen Torrance, BSc, DC*
Christopher deGraauw, DC, FRCCSS(C)†

Objective: *This case study demonstrates the effectiveness of a novel approach to the treatment of post-traumatic myositis ossificans with extracorporeal shockwave therapy in an elite athlete.*

Clinical Features: *A 20 year-old male semi-professional rugby player presented with progressive pain and loss of range of motion after sustaining a severe, right quadriceps contusion nine weeks earlier. The differential diagnosis of myositis ossificans was suspected and confirmed on radiographic examination.*

Intervention and Outcome: *A two week treatment protocol was undertaken consisting of three sessions of extracorporeal shockwave therapy and an unsupervised exercise program consisting of active and passive range of motion, gradual strengthening and balance exercises. The patient experienced appreciable improvements in pain and range of motion in two weeks and was able to participate in sport specific activity four weeks after presentation.*

Summary: *This case illustrates the successful conservative management of post-traumatic myositis ossificans of the anterior thigh with extracorporeal shockwave therapy and a primarily unsupervised graded exercise program within a condensed treatment time frame of 2 weeks.*
(JCCA 2011; 55(4):240–246)

KEY WORDS: myositis ossificans, high-energy shock waves, muscles, injuries

Objectif : *cette étude de cas démontre l'efficacité d'une nouvelle approche relative au traitement de la myosite ossifiante post-traumatique avec thérapie par onde de choc extracorporelle sur un athlète d'élite.*

Caractéristiques cliniques : *un joueur de rugby semi-professionnel de 20 ans éprouvait des douleurs et une perte d'amplitude des mouvements après avoir subi une grave contusion au quadriceps droit neuf semaines auparavant. Le diagnostic différentiel de myosite ossifiante soupçonné fut confirmé suite à une radiographie.*

Intervention et résultat : *un protocole de traitement de deux semaines fut entrepris. Celui-ci comprenait trois séances de thérapie par onde de choc extracorporelle et un programme d'exercice non supervisé comprenant l'amplitude des mouvements actifs et passifs, le renforcement graduel et des exercices d'équilibre. Le patient a senti une grande amélioration au niveau de la douleur et de l'amplitude des mouvements en l'espace de deux semaines, et fut en mesure de participer à une activité sportive quatre semaines après avoir ressenti des douleurs.*

Sommaire : *ce cas démontre le succès de la gestion conservatrice de la myosite ossifiante post-traumatique de la cuisse antérieure avec thérapie par onde de choc extracorporelle et un programme d'exercice non supervisé dans un délai de 2 semaines.*
(JCCA 2011; 55(4):240–246)

MOTS CLÉS : myosite ossifiante, ondes de choc de haute énergie, muscles, blessures

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Introduction

Posttraumatic myositis ossificans (MO) occurs as a complication in approximately 20% of large haematomas associated with muscle contusions and strains. It is responsible for considerable morbidity, with symptoms of prolonged pain, diminished flexibility, local tenderness and stiffness lasting an average of 1.1 years.[1–3] The majority of contusions and strains are typically responsive to conservative treatment within a few weeks of injury, however, MO becomes clinically suspected when a strain or contusion is unresponsive to conservative care and patients begin to demonstrate increasing pain and progressive loss of range of motion (ROM).[1,4] MO is a non-neoplastic proliferation of bone and cartilage tissue at the site of a previous injury, most commonly after blunt trauma or repeated micro-injuries.[1,5] It is also known as post-traumatic heterotopic ossification, non-hereditary heterotopic ossification and myositis ossificans circumscripta.[3,6] Radiographic imaging is used for a definitive diagnosis with evidence of bone formation being detected 3 to 4 weeks after the initial trauma.[7] It is most commonly found in athletes in contact sports but ectopic bone formation is also a frequent complication in approximately 19% of total hip arthroplasties.[1,3,8–10] MO is typically found in muscles but can also occur in tendons, joint capsules, ligaments and fascia.[5,6,11,12]

The goal of therapy for MO is the restoration of strength and ROM. Therapy has been largely based on the RICE principle of rest, ice, compression and elevation and non-painful, passive stretching and strengthening routine.[3,13] Due to the relative rarity of the condition and variability of the diagnosis in location and severity, evidence for effectiveness of manual therapy has been sparse. A Medline literature search from January 1991 to January 2011 for conservative therapies for myositis ossificans revealed mostly case reports[2,3,14–16] and review articles.[4,5,9,12–14] However, a case-series was published in 2010 demonstrating the effectiveness of extracorporeal shock wave therapy (ESWT) for the treatment of MO in elite or sub-elite athletes in significantly less time than traditional therapeutic approaches reported.[1] After three sessions of ESWT spaced over six weeks and an intense program of supervised exercises and stretching, the athletes all experienced significant short-term improvements with 87% of the patients returning to competitive sport activities after 13 weeks of therapy.[2,3] This was the first study to evaluate the use of ESWT for the treatment of MO.

The following is a case of a young rugby player that sustained a severe contusion to the right quadriceps. Two months after the initial trauma, he presented to the clinic with progressing pain and disability and a significant loss of ROM of the knee. He was diagnosed with posttraumatic myositis ossificans of the right vastus lateralis and a trial of ESWT was initiated.

Patient History

A 20 year-old male semi-professional rugby player presented to a chiropractic clinic with a severe, right quadriceps contusion that he sustained nine weeks before in Scotland where he was attending school. The injury occurred during a game of touch rugby where there was a significant collision with another player who fell into his anterior thigh while running. The injury was quite debilitating and he was forced to bed-rest for one week. The rehabilitation began in Scotland with pool exercises, hot/cold baths and ultrasound. Soft tissue therapy was attempted but not well tolerated. His thigh seemed to get worse with more pain, loss of strength and reduced range of motion (ROM). He saw an orthopaedic surgeon in Edinburgh three weeks after the injury who ordered an MRI (Figure 1) which revealed a significant haematoma in the vastus lateralis. The quadriceps tendon and structures of the knee were intact.

Upon presentation to our clinic nine weeks later, he walked with a significant limp and could not bend his knee more than 45 degrees. He reported difficulty walking, going up or down stairs and difficulty sleeping due to pain. Past medical history revealed that he was otherwise healthy. On physical examination, the contusion was located in the proximal one-third of the vastus lateralis with notable swelling and tenderness along the length of vastus lateralis. Palpation revealed a mass approximately 12 cm × 8 cm that was firm and very tender. Knee extension strength was graded 2/5 on the right (he could not extend against gravity while supine) and 5/5 with knee flexion. Sensation of the lower limb was intact. He could perform a double leg squat comfortably to 45° of right knee flexion. Passive range of motion of the knee revealed 70° of flexion with the hip flexed at 90°. He was diagnosed with a large, severe chronic contusion of the right vastus lateralis. A differential diagnosis of MO was considered due

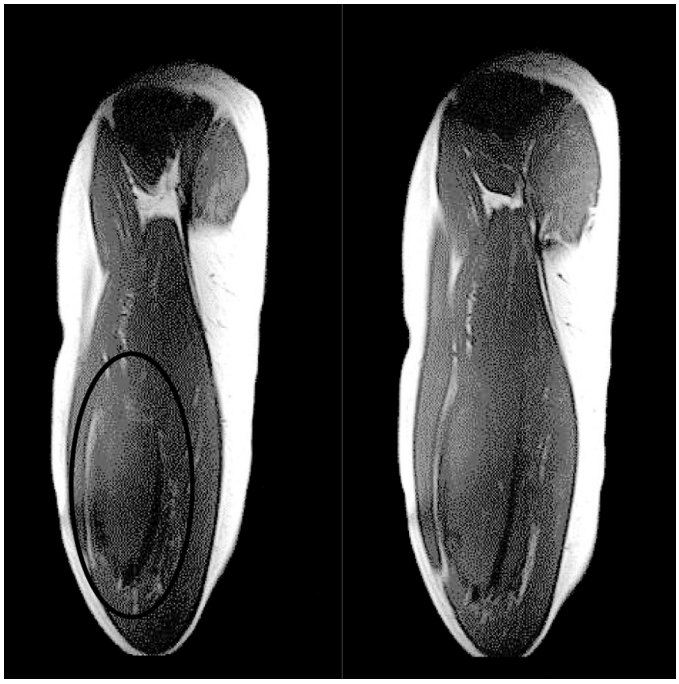

Figure 1 *The lateral view of the MR images of the right thigh three weeks post-injury demonstrating the lesion in the vastus lateralis.*

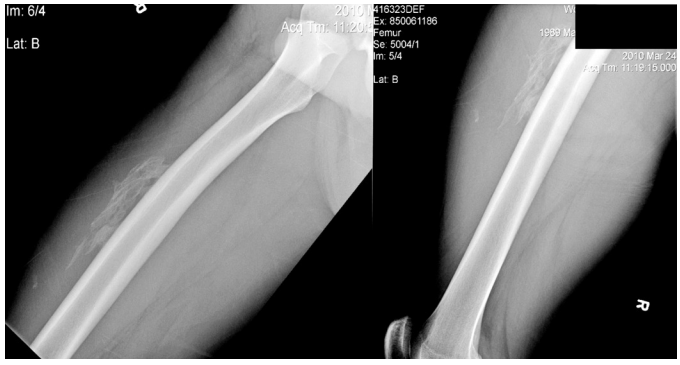

Figure 2 *Radiographs taken nine weeks post-trauma demonstrating the appearance of fine, lacy, heterotopic ossification in the right thigh consistent with the diagnosis of MO. Frog-leg (left) and lateral (right) thigh series.*

to the progressive loss of function. He was advised to ice at home and to begin a progression of stretching (active hamstring contraction and gentle passive manoeuvres) and strengthening exercises (isometric contraction, supine straight-leg raises and one-foot balance) on his own. He was instructed that all components of the treatment plan were to be gentle and pain-free. The patient had radiographs taken the following day which verified the diagnosis of MO (Figure 2).

Based on an article by Buselli et al., a trial of shockwave therapy was initiated with the patient five days after the initial assessment (10 weeks post-injury). Before the first ESWT treatment, the patient was fully informed and consent was obtained. On examination, the right knee had regained some strength and the patient could lift his heel off the table 10 cm while supine. Passive flexion was 90° and he reported to continue to have pain at night. There was more delineation of the contours of the mass with exquisite tenderness at the proximal end. The first EWST treatment (Masterpuls MP100, Storz Medical) consisted of 100 impulses at 1.5 bar (1 bar = 0.1 MPa = 0.1 N/mm^2) delivered to each of five locations over the mass: two at the proximal end, one in the middle and two at the distal end. Home instructions remained the same.

Two days later, the patient reported that he had experienced about 12 hours of soreness after the treatment but presently felt great and had no night pain. Objectively, his ROM increased to 115°. He had a second shockwave treatment for 100 impulses at 3.0 bar over every square centimeter of the mass (2500 impulses). He was recommended to have another treatment one week later and was told to continue with his home care.

When the patient returned the following week (11 weeks post-injury), he reported that he was able to walk for over an hour with no pain and had no night pain. The ROM of his knee was not restricted and was equal to the contralateral side (125°). He had a final treatment of 2500 impulses over 25 points and was advised that he could start shallow, body weight squats and lunges to 45° of knee flexion. He was advised to gradually increase his ROM with the lunges and squats before adding resistance.

The patient returned to Scotland the next day so the remaining follow-ups were through written correspondence. The patient remained diligent with the gradual stretching and strengthening. Two weeks after the last ESWT session (13 weeks post-injury), he reported that he was running an average of four km/day and had just played his first 60-minute touch rugby session with no pain. The following week he was running over 5 km in 25 minutes

with speed intervals of up to 85% capacity and playing touch-rugby three times a week. He reported full ROM with only some muscle fatigue that seemed to be similar in both legs, possibly due to deconditioning. At 17 weeks post-injury, he reported he was playing touch rugby at full pace, his timed sprints were back to pre-injury levels, and his squat and deadlifts were back to full strength and ROM. He reported being unrestricted in training with the rest of the team and felt that he had made a full recovery.

Discussion

Post-traumatic myositis ossificans is the proliferation of bone and cartilage within a muscle after the formation of an intramuscular haematoma. It is more common in the sporting community as a complication of muscle contusions and strains by either a major trauma or repeated injury.[1,5,11] Muscle strains and contusions account for over 90% of all injuries sustained in sports and, of these, approximately 9% to 20% are complicated by MO.[3,5,17] The case presented was typical in that it is usually found adjacent to the diaphysis of tubular bones, most commonly in the anterior thigh and is also commonly found in the anterior third of the arm involving the brachialis.[3,7]

Diagnosis can be obtained from the clinical history, physical examination, radiographic imaging, and less commonly by diagnostic ultrasound or magnetic resonance imaging (MRI).[3,18] The symptoms of pain, tenderness, erythema, soft-tissue swelling and periarticular stiffness overlap between muscle strains/contusions and MO. However, MO becomes clinically suspected after the injury is unresponsive to 10–14 days of rest.[12] As in the case presented, MO causes intensified pain with progressive loss of ROM and a painful, palpable mass in the weeks following trauma.[1,12] Sporting activities as well as activities of daily living are impaired by the reduced ROM and soft-tissue contractures.[1,4,12] Radiographic signs of ectopic bone usually develop approximately 3–5 weeks after the injury.[5,13,18] The radiographic signs of MO during the early stages demonstrate a fine, lacy radiopacity that later appears cloudy within a well-defined mass (Figure 2).[6,17] MRIs will demonstrate well-defined margins and a lack of soft-tissue invasion which helps to differentiate it from sarcomatous lesions (Figure1).[6,17,19,20]

The pathogenesis and primary etiological factors of bone formation remains unclear.[1,12] After a muscle is crushed or torn, the tissues at the center of the traumatized area usually liquefy or form sheets of non-specific cells. It has been hypothesized that the ectopic bone and cartilage are from the differentiation of osteoblasts from these rapidly proliferating mesenchymal cells under anoxic conditions.[7,14,21] Inflammation caused by the damage, with heat, swelling and tenderness, recruits an influx of inflammatory cells and macrophages into the necrotic tissue that release osteogenic bone mediators which encourage the formation of heterotopic bone.[1,3,7,21]

During the first 2 to 4 weeks, osteoid formation is minimal and a biopsy can lead to an erroneous diagnosis of neoplasia if the history of trauma is not disclosed.[7,12] The tissue in the periphery is the most prompt to repair, organize and mineralize into mature tissue with the lesion becoming progressively less differentiated towards the center. Differentiation of the tissues continues through the second and third months, leading to the characteristic zoning of MO.[6,7] The zoning helps to distinguish MO from osteosarcomas with the latter being the least differentiated at the periphery and most differentiated at the center.[6,7,11] MO tends to shrink as it matures over a six month period.[12] Surgery is reserved until approximately 12–14 months post-injury, when the lesion becomes stable and the periosteum has formed. Surgery is contraindicated for immature lesions due to the high recurrence rates.[5] Muscle function typically does not recover with or without surgery, however, muscle contours typically return to normal.

The literature for the effectiveness of typical therapeutic approaches to MO, such as stretching, strengthening and proprioceptive retraining, is sparse.[3] Acute care of a severely injured muscle during the initial two weeks aims to reduce local inflammation which is hypothesized to reduce the chance of developing MO. This typically includes immobilization of the muscles in slight tension with compression, along with ice and elevation in order to limit the intramuscular bleeding.[1,5,22] During the first 15 days after trauma, the lesions are vulnerable to further trauma and, therefore, excessive activity, forceful stretching and massage should be avoided to limit bleeding.[5,11] Non-steroidal anti-inflammatories are also incorporated and have been shown to reduce the onset of MO after hip arthroplasty, but have not been studied in post-traumatic contusions.[12] If MO develops despite the best efforts of prevention, the graded restoration of flexibility and strength with minimal pain becomes the goal of rehabilitation.[3]

Extracorporeal shock-wave therapy (ESWT) is a sequence of high intensity sonic pulses with a short duration (10 ms).[23,24] ESWT was originally used for fragmentation of urinary stones and other types of body calculi and has since been extended in its use and shown to be effective for calcification of tendons and pseudo arthroses.[3,5,25,26] ESWT, when applied to a tissue, produces a mechanical action through a cavitation effect that can induce biological action through microdisruption of avascular tissues. This leads to suppressive effects on local nociceptors as well as stimulation and reactivation of tissue repair through neovascular angiogenesis, release of local growth factors and anti-inflammatory mediators, such as nitrous oxide, and recruitment of local stem cells for the repair process.[23,26–30] It is thought that the analgesic ability of ESWT may be due to a form of hyperstimulation analgesia[8,31,32] which occurs when a moderate to intense sensory input is applied to the site of greatest discomfort in a chronic pain patient. The relief can last for days, weeks, months or sometimes permanently.[31–33]

Consideration for the use of ESWT for the treatment of MO for this particular patient came from a case-series by Buselli, et al., which demonstrated successful treatment of 21 of 24 MO athletes treated with EWST.[1] A significant portion of the patients in that study were similar to the patient presented here. Twelve of the 24 patients with MO in Buselli's study were the result of contusions to the quadriceps, were relatively young athletes (average age of 25 years), and were also treated within twelve weeks of the initial trauma.

In Buselli's study, ESWT was administered every other week for a total of three treatments. The application of ESWT was in accordance of the International Society of Medical Shock Waves Therapy in which 100 impulses were applied at medium power (1.3 to 2.3 bar), within the patient's tolerance, to every square centimeter of the ossification.[24] In the case presented here, due to the novelty of the treatment, the first session of ESWT was limited to 500 impulses at a low-medium intensity (1.5 bar) to gauge the patient's level of tolerance. Of note, even at the reduced intensity, the patient reported a notable analgesic effect 24 hours post-treatment and an improvement in ROM of the knee. Physical examination also confirmed a reduction in swelling and tenderness of the vastus lateralis muscle. It is possible that the gentle stretching and strengthening exercises also contributed to the improvement of his symptoms. However, the ESWT was suspected to have contributed to the majority of the analgesia due to the dramatic reduction in pain and improvement of ROM within 48 hours of the first ESWT application. The patient had a more intense ESWT treatment (2500 hits, 3.0 bar) two days after the first and the last one seven days later. After the last treatment, there was full restoration of knee flexion and significant reduction in swelling and tenderness over the lesion and no pain at rest.

The patients in Buselli's study also underwent a much more intense regimen of physical therapy (6 times per week for six weeks, 80 minutes per session) consisting of stretching, proprioceptive exercises, manual and mechanical active and passive mobilizations. After six weeks of treatment, over 90% of those patients were able to return to competitive sporting activities after an average of 11 weeks (24 max., 8 min.) and complete return to activities after an average of 14 weeks (28 max., 10 min.). In the case presented here, the two week timeline and predominantly unsupervised, home-based rehabilitation program contrasted with the daily physiotherapy sessions that was proposed in Buselli's study. Our therapy consisted of unsupervised active and passive stretching and a graded exercise program of basic movements, from non-weight bearing to weight bearing, with the instructions that it was to all be completed pain free. The patient was permitted to return to sport-specific training after he was able to demonstrate equal ROM of the injured and uninjured knees, as well as pain-free use of the injured muscle during functional testing.[5] In this case, the patient was able to return to sport-training thirteen weeks post-injury (two weeks after the final ESWT application).

No serious side-effects from the use of ESWT on MO have been reported. However, the procedure can be painful and cause pain over the treatment area afterwards for 24 to 48 hours. Other known side-effects include minor skin bruising, reddening and short-term swelling.[1,25,34] In this case, the first treatment was quite sensitive even at the lower pressure settings. Subsequent treatments were only moderately painful. The patient reported twelve hours of soreness after the first treatment with only mild, short-term discomfort after the last two applications.

The MO patients in Buselli's study did not demonstrate a reduction in the size of the ossification despite the improvement in ROM and strength after treatment with ESWT.[1] This may be because the ossification in MO

tends to be well organized which is consistent with the finding that ESWT does not cause fractures in cortical bone.[35] No follow-up radiographs were available for the patient presented in this study, however, a firm mass was still palpable in his lateral thigh at the last visit. Therefore, a reduction in the size of the ossification area may not be necessary for patients to regain their functional ROM, strength and to experience a reduction in pain.

It should be noted that the evidence for the effectiveness of ESWT on MO is limited to descriptive case reports and has not been validated in controlled trials. A controlled trial can help to delineate the relative contribution of ESWT on pain relief and disability versus the traditional exercise protocols commonly used. Of note, all of the patients in these case reports were male, elite or sub-elite athletes and between 18 and 54 years of age (mean age of 25 years) which restricts the generalizability to other patient populations prone to MO, such as those receiving hip arthroplasty. More research is also needed to establish timelines for the onset of treatment, the number of treatments that are necessary, and to confirm the safety of the modality.

Conclusion

MO is a chronic, debilitating condition and is not an uncommon diagnosis in contact sports due to the high incidence of traumatic contusions. Non-invasive treatment options for MO have remained consistent for over 20 years with stiffness and tenderness lasting 1.1 years on average[1–3]; however, a recent study has demonstrated that ESWT may be a promising treatment modality for the management of pain and loss of ROM that result from MO.[1] Although the results have not yet been confirmed in a controlled trial, the appreciable improvement in pain scores and ROM in elite and sub-elite athletes after only 3 treatments of ESWT in a relatively short period of time is encouraging. In the case presented here, a two week program of ESWT and an unsupervised, progressive strengthening program were effective modalities in the treatment of a post-traumatic MO in the quadriceps of a 20 year old, male, semi-professional rugby player. Despite relatively high disability for 10 weeks before treatment with ESWT, he regained much of the function of his right quadriceps and was back to sport specific training four weeks after initiating treatment. The current literature on ESWT indicates that it can have a substantial analgesic effect on the lesion although the mechanism remains elusive.[36,37] ESWT is an attractive modality for the treatment of MO given that it is non-invasive, affordable and with few side effects.

References

1 Buselli P, Coco V, Notarnicola A, et al. Shock waves in the treatment of post-traumatic myositis ossificans. Ultrasound in Medicine & Biology. 2010; 36(3):397–409.

2 Giombini A, Di Cesare A, Sardella F, Ciatti R. Myositis ossificans as a complication of a muscle tendon junction strain of long head of biceps. A case report. J Sports Med Phys Fitness. 2003; 43(1):75–77.

3 Miller AE, Davis BA, Beckley OA. Bilateral and recurrent myositis ossificans in an athlete: a case report and review of treatment options. Archives of Physical Medicine and Rehabilitation. 2006; 87(2):286–90.

4 Booth DW, Westers BM. The management of athletes with myositis ossificans traumatica. Can J Sport Sciences. 1989; 14(1):10–16.

5 Järvinen T a H, Järvinen TLN, Kääriäinen M, et al. Muscle injuries: optimising recovery. Best practice & research. Clinical Rheumatology. 2007; 21(2):317–31.

6 Yochum TR, Rowe LJ. Essentials of skeletal radiology. 2nd ed. Baltimore: Williams and Wilkins; 1996.

7 Ackerman LV. Extra-osseous localized non-neoplastic bone and cartilage formation (so-called myositis ossificans): clinical and pathological confusion with malignant neoplasms. J Bone Joint Surg(AM). 1958; 40-A(2):279–98.

8 Melzack R. Trigger points and acupuncture points for pain: Correlations and implications. Pain. 1977; 3(1):3–23.

9 Vanden Bossche L, Vanderstraeten G. Heterotopic ossification: a review. J Rehabil Med. 2005; 37(3):129–136.

10 Neal B, Gray H, MacMahon S, Dunn L. Incidence of heterotopic bone formation after major hip surgery. ANZ J Surg. 2002; 72(11):808–21.

11 Järvinen T a H, Järvinen TLN, Kääriäinen M, Kalimo H, Järvinen M. Muscle injuries: biology and treatment. Am J Sports Med. 2005; 33(5):745–64.

12 Pignolo RJ, Foley KL. Nonhereditary heterotopic ossification. Implications for Injury, Arthropathy, and Aging. Clinical Reviews in Bone and Mineral Metabolism. 2005; 3(3):261–266.

13 Beiner JM, Jokl P. Muscle contusion injury and myositis ossificans traumatica. Clinical Orthopaedics and Related Research. 2002; (403 Suppl):S110–9.

14 Micheli A, Trapani S, Brizzi I, et al. Myositis ossificans circumscripta: a paediatric case and review of the literature. Eur J Pediatrics. 2009; 168(5):523–529.

15 Sodl JF, Bassora R, Huffman GR, Keenan MAE. Traumatic myositis ossificans as a result of college

fraternity hazing. Clinical Orthopaedics and Related Research. 2008; 466(1):225–30.
16 Sokunbi G, Fowler JR, Ilyas AM, Moyer RA. A case report of myositis ossificans traumatica in the adductor magnus. Clinical J Sport Med. 2010; 20(6):495–6.
17 Danchik JJ, Yochum TR, Aspegren DD. Myositis ossificans traumatica. J Manip Physiol Thera. 1993; 16(9):605–614.
18 Wang SY, Lomasney LM, Demos TC, Hopkinson WJ. Radiologic case study. Traumatic myositis ossificans. Orthopedics. 1999; 22(10):1000, 991–5.
19 Shirkhoda A, Armin AR, Bis KG, et al. MR imaging of myositis ossificans: variable patterns at different stages. J Magnetic Resonance Imaging: JMRI. 1995; 5(3):287–292.
20 Tyler P, Saifuddin A. The imaging of myositis ossificans. Seminars In Musculoskeletal Radiology. 2010; 14(2):201–216.
21 Aro HT, Viljanto J, Aho HJ, Michelsson JE. Macrophages in trauma-induced myositis ossificans. APMIS. 1991; 99(5):482–6.
22 Bleakley C, McDonough S, MacAuley D. The use of ice in the treatment of acute soft-tissue injury: a systematic review of randomized controlled trials. Am J Sports Med. 32(1):251–61.
23 Thiel M. Application of shock waves in medicine. Clin Orth Rel Res. 2001; (387):18–21.
24 Thiele R. New Guidelines for ESWT. ISMST Newsletter. 2009; 5:19.
25 Wang C, Wang F, Yang KD, Weng L, Ko J. Long-term results of extracorporeal shockwave treatment for plantar fasciitis. Am J Sports Med. 2006; 34(4):592–596.
26 Wang L, Qin L, Lu HB, et al. Extracorporeal shock wave therapy in treatment of delayed bone-tendon healing. Am J Sports Med. 2008; 36(2):340–347.
27 Tu J, Matula TJ, Bailey MR, Crum LA. Evaluation of a shock wave induced cavitation activity both in vitro and in vivo. Physics in Medicine and Biology. 2007; 52(19):5933–5944.
28 Wang C-J, Wang F-S, Yang KD, et al. Shock wave therapy induces neovascularization at the tendon-bone junction. A study in rabbits. J Orth Res. 2003; 21(6):984–989.
29 Speed C a. Extracorporeal shock-wave therapy in the management of chronic soft-tissue conditions. J Bone Joint Surg(Br). 2004; 86(2):165–71.
30 Barakat C, McCluskey M. Shock wave treatment speeds wound healing. Equus. 2010; (388):8.
31 Melzack R. Prolonged relief of pain by brief, intense transcutaneous somatic stimulation. Pain. 1975; 1(4):357–73.
32 Rompe JD, Hope C, Küllmer K, Heine J, Bürger R. Analgesic effect of extracorporeal shock-wave therapy on chronic tennis elbow. J Bone Joint Surg(Br). 1996; 78(2):233–237.
33 Rompe JD, Riedel C, Betz U, Fink C. Chronic lateral epicondylitis of the elbow: A prospective study of low-energy shockwave therapy and low-energy shockwave therapy plus manual therapy of the cervical spine. Arch Phys Med Rehab. 2001; 82(5):578.
34 Leeuwen MT van, Zwerver J, Akker-Scheek I van den. Extracorporeal shockwave therapy for patellar tendinopathy: a review of the literature. Br J Sports Med. 2009; 43(3):163–8.
35 Pauwels FET, McClure SR, Amin V, Van Sickle D, Evans RB. Effects of extracorporeal shock wave therapy and radial pressure wave therapy on elasticity and microstructure of equine cortical bone. Am J Vet Res. 2004; 65(2):207–212.
36 Wang C-J, Wang F-S, Huang C-C, et al. Treatment for osteonecrosis of the femoral head: comparison of extracorporeal shock waves with core decompression and bone-grafting. J Bone Joint Surg(Am). 2005; 87(11):2380–7.
37 Wess OJ. A neural model for chronic pain and pain relief by extracorporeal shock wave treatment. Urological Research. 2008; 36(6):327–34.

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Avulsion fractures of the pelvis – a qualitative systematic review of the literature

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Objective: *To assess a causal relationship between physical activity or boney surgical intervention and the occurrence of avulsion fracture in the pelvis. Secondarily to assess the average age at which avulsion fracture occurs in cases associated with physical activity or boney surgery.*

Method: *A literature search was performed on a variety of databases using text words and MeSH terms. Results were limited to English language. Cases involving trauma or pathological disease were excluded. Causation Criteria scores were calculated for each paper to establish a link between the suspected mechanism of injury and avulsion fracture.*

Results: *48 papers were retrieved encompassing 66 cases of avulsion fracture. 88% of cases were associated with physical activity while 12% were associated with a history of surgery. Average age in the physical activity cases was 16.8(range 13–43) and 56.4(range 31–74) in the surgery related cases. Causation Criteria scores were definite in 76% of activity related cases and probable in 60% of boney surgery related cases.*

Conclusions: *Avulsion fractures of the pelvis represent a highly prevalent pathology among the adolescent athletic population. A population of skeletally mature*

Objectif : *évaluer la relation de cause à effet entre l'activité physique ou l'intervention chirurgicale des os, et l'occurrence d'une fracture d'avulsion du bassin. Ensuite, évaluer l'âge moyen des victimes d'une fracture d'avulsion dans les cas associés à l'activité physique ou à l'intervention chirurgicale des os.*

Méthode : *une recherche littéraire fut effectuée dans diverses banques de données à l'aide de mots du texte et de termes du MeSH. Les résultats obtenus étaient en anglais. Les cas impliquant une maladie traumatique ou pathologique furent exclus. Les pointages des critères de causalité furent établis pour chaque document afin de créer un lien entre le mécanisme soupçonné de blessure et de fracture d'avulsion.*

Résultats : *48 documents furent trouvés, et ceux-ci comprenaient 66 cas de fracture d'avulsion. 88 % des cas étaient liés à l'activité physique, tandis que 12 % étaient liés à des antécédents de chirurgie. Dans les cas liés à l'activité physique, la moyenne d'âge était de 16,8 ans (plage allant de 13 à 43) et de 56,4 ans (plage allant de 31 à 74) dans les cas liés aux interventions chirurgicales. Les pointages des critères de causalité furent définis dans 76 % des cas liés à l'activité physique, et jugés probables dans 60 % des cas d'intervention chirurgicale aux os.*

Conclusions : *des fractures d'avulsion du bassin représentent une pathologie très prévalente chez les athlètes adolescents. Il existe un groupe de patients dont*

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patients with history of boney surgical intervention are also at risk.
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KEY WORDS: avulsion, fracture, pelvis, athlete

le squelette est mature qui ont subi une intervention chirurgicale aux os.
(JCCA 2011; 55(4):247–255)

MOTS CLÉS : avulsion, fracture, bassin, athlète

Introduction

Avulsion fractures represent a unique type of bone pathology which occurs when a fragment of bone is pulled away from the main boney mass as a result of a sudden tensile force applied through a powerful muscle contraction. The current literature reports the prevalence of avulsion fracture as being the highest among adolescents, which leads one to consider the stage of development of the bone to be crucial.[1–12] Accordingly, the weakest morphological structure in the bone is the physis. Once the growing cartilage ossifies, the connection between the apophysis and the body of the bone strengthens. As a result, the musculotendinous unit becomes the weakest structure in transferring the force between muscle and bone.

The body of literature surrounding avulsion fractures is largely comprised of case reports and case series. This form of literature provides researchers with direction for further research. However, it also provides a body of literature for situations where other forms of research may be unethical or too costly. Case reports and case series also provide an opportunity for authors to provide a great deal of information regarding the given case. This added information may be crucial in establishing a causal association.

The literature reports that the majority of pelvic avulsion fractures occur during the eccentric phase of a sporting activity due to the higher forces generated during eccentric muscular contractions.[2,6,10] Reports of pre existing pain in the hip or diagnosed osteitis have been shown in the literature; however, the reporting of these phenomena is inconsistent and may often be missed by practitioners.[13–20] Reports of avulsion fracture do exist in older populations despite skeletal maturity; however, these typically involve pathological processes.[21] Surgical interventions involving boney resection provide another possible explanation for a weakened state to exist at a pelvic apophysis despite skeletal maturity. Removal or alteration of a bone may leave it in a state of weakness making it susceptible to avulsion fracture. To date the strength of this causal association has not be studied.

The purpose of this review is to assess the strength of a causal association between activity or surgical intervention and the occurrence of avulsion fracture. The secondary aim of the paper is to assess the average age at which avulsion fracture occurs within cases reporting activity or history of surgery at onset of symptoms. To the knowledge of the authors, there has not been a review of the literature of this nature.

Methods

Search Strategy

A literature search was performed in EBSCO in the databases MEDLINE, CINAHL, Alt HealthWatch, AMED, SPORTDiscus, Rehabilitation & Sports Medicine Source, ICL and MANTIS. Text words used were Avulsion Fractur*, Pelvic Bones, Anterior Superior Iliac Spine (ASIS), Anterior Inferior Iliac Spine (AIIS), Ischial Tuberosity (IT), Pubic Crest (PC), Pubic Symphysis (PS) and Iliac Crest (IC). The MeSH Term Pelvic Bone was used in MEDLINE. The search consisted of literature published from the start date of each database up to December 2010. Limiters included papers published in the English language and only case reports or case series were accepted for analysis. Reference lists were hand searched for additional relevant articles. Data from literature reviews on similar topics were not used for analysis, however were utilized for comparison with our results.

Inclusion and Exclusion Criterion

Papers accepted for analysis must have shown objective evidence of osseous involvement in each case of reported avulsion fracture, and there must not have been a history of direct trauma to the area of injury or evidence of pathological disease such as metastasis. Only studies involving injury to an apophysis of the pelvis were included. Each paper retrieved by the search was assessed by the lead author for inclusion.

Table 1 *Causation scoring strategy for avulsion fracture*

Activity Factor	Value		
	Yes	No	Unknown
Did the adverse event appear within 24 hours of activity?*	+2	–1	0
Are alternative causes available in the case that could have caused the adverse reaction?	–1	+2	0
Was the adverse reaction proportional to the force of the suspected mechanism?	+1	0	0
Was the adverse reaction comparably related to the area of injury?	+1	0	0
Did the patient have a similar reaction to any previous exposure?	+1	0	0
Was the adverse event confirmed by objective evidence?	+1	0	0
Is the description of the mechanism of avulsion fracture first hand?	+1	0	0
Total Score of each column			
Final Score			
Scoring§: ≤ 0.68 = doubtful; 0.69 – 3.45 = possible; 3.46 – 6.18 = probable; ≥ 6.19 = definite.			

* 24 hours chosen as patient excitement and adrenaline levels during physical activity may mask pain at initial time of injury; for cases with history of surgery a time line of 6 weeks was used as this is the natural history for osseous healing.

§ The original causation criterion included a question on dose response which was deemed inappropriate for the current study; scores were adjusted accordingly.

Causation Criteria of the Studies

In 1982, Naranjo et al. state that the lack of a method for establishing causality generates large inter and intra rater variability during assessment.[22] They also state that the estimation of the probability that an adverse event occurs due to a proposed mechanism is based on clinical judgement.[22] Therefore, Naranjo et al. devised an instrument to evaluate the degree of association and level of certainty that a drug treatment in question caused an observed adverse event.[22] McGregor et al. adapted the instrument to address adverse events related to spinal manipulation.[23] The instrument was revised for use in case literature reporting on vertebrobasilar compromise associated with cervical spine manipulation. Reliability of the adapted tool between practitioners was found to be high (r = 0.84).[23] Scoring for Naranjo et al.'s work ranged from –4 to 13 with sub scoring ranges as follows: ≤0 being doubtful of a cause and effect relationship; 1–4 as possible; 5–8 as probable; ≥9 as definite.[22] McGregor et al. used the same boundaries as Naranjo et al., and therefore with a maximum score of 11 on their adapted version of the instrument had sub scoring ranges as follows: ≤0 being doubtful; 0.84–4.22 being possible; 4.23–7.55 being probable; ≥7.56 being definite.[23] Adaptations to the instrument formulated by McGregor et al. were made to ensure suitability when dealing with case literature on avulsion fracture. Following these changes the maximum total score for the instrument utilized in the current study was 9. In keeping with the original boundaries set out by Naranjo et al., and maintained by McGregor et al., the resulting sub scoring ranges for our adapted version of the instrument are as follows: ≤0.68 being doubtful; 0.69–3.45 being possible; 3.46–6.18 being probable; ≥6.19 being definite.[22,23] These sub scoring ranges represent accurate boundaries for division of groups, however it is to be expected that individual scores for each case will result in a whole number which will fall into one of four sub scoring ranges.[23] Each paper was independently assessed by two of the authors according to the Causation Criteria, and consensus was then met for a final score for each paper.

Data Collection

All cases presented in the accepted papers were reviewed for relevant data. Data was collected on age, sex, causative

mechanism, signs and symptoms, imaging, displacement, treatment rendered and complications. Results of data collection are available upon request as Appendix 1.

Analysis

Causation Criteria scores were analyzed between the activity related papers and the surgical papers to assess for a significant difference between groups. Descriptive statistics, including mean scores and standard deviations, will also be reported.

Results

The authors were able to retrieve 48 case reports and case series on avulsion fractures that met the inclusion criterion. This accounted for 66 cases reported in the literature, 8 (12%) which were associated with previous surgical procedures and 58 (88%) which were associated with physical activity. A summary of the cases of avulsion fracture by site can be found in Table 3. The average age at onset of avulsion fracture in the activity causally related cases was 16.8 (range 13–43) and in the surgical causally related cases it was 56.4 (range 31–74). Conservative treatment was utilized in 68% (45/66) of cases and surgical intervention was used in 32% (21/66) of cases. All cases of avulsion fracture with a proposed history of surgery as a causative agent were treated by conservative means. Cases related to physical activity had a gender distribution of 84% males, while the cases related to a surgical causal agent had a 100% female gender distribution. Detailed results for data collection are available upon request as Appendix 2.

Causation Criteria

For the papers reporting on avulsion fracture occurring as a result of physical activity, 76% (37/49, range 7–3) scored a definite rating on the causal relationship between avulsion fracture and a history of physical activity. They had a mean score of 6.63 with a standard deviation of 1.07 (95% CI 6.2 – 6.9). In the surgical group 60% (3/5, range 6 to –1) scored in the probable range for a causal relationship between avulsion fracture and a history of surgical intervention. They had a mean score of 4.4 with a standard deviation of 3.05 (95% CI .613 – 8.19). Evaluation of the criterion-based scored data indicated a non-normal distribution. Wilcoxon rank-sum was undertaken in order to determine if there was a statistically significant differ-

Table 2 *Summary of causation scores for all accepted papers*

Paper	Causation Criteria Score
Aksoy et al., 1998[24]	7
Atalar et al., 2007[25]	7
Bahk et al., 2000[26]	7
Bolgla et al., 2001[27]	7
Byrne et al., 2008[28]	7
Davis et al., 1998[13]	7
Deehan et al., 1992[29]	7
DePalma et al., 1965[14]	7
Doral et al., 2005[30]	7
Dosani et al., 2004[31]	7
Draper et al., 1992[32]	7
Gidwani et al., 2004[3]	7
Gomez, 1996[33]	7
Kaneyama et al., 2006[34]	7
Karakas et al., 2009[35]	7
Kusma et al., 2004[36]	7
Khoury et al., 1985[37]	7
Lambert et al., 1993[38]	7
Mader, 1990[39]	7
Nanka et al., 2003[40]	7
Oldenburg et al., 2009[41]	7
Pointinger et al., 2003[15]	7
Rajasekhar et al., 2001[42]	7
Resnick et al., 1996[16]	7
Rosenberg et al., 1996[43]	7
Rossi et al., 2001[9]	7
Salvi et al., 2006[44]	7
Schlonsky et al., 1972[45]	7
Servant et al., 1998[46]	7
Steerman et al., 2008[47]	7
Swischuk, 2004[48]	7
Thanikachalam et al., 1995[49]	7
Vajnar, 2008[50]	7
Valdes et al., 2000[51]	7
Vogt et al., 2007[52]	7
Watanabe et al., 1995[18]	7
Yazzie, 2001[53]	7
Yildiz et al., 2005[20]	7
Kuhn, et al., 1986[54]	6
Stellon et al., 1985[55]	6
Zijderveld et al., 2004[56]	6
Samartzis et al., 2006[57]	5
Winkler et al., 1987[19]	5
Spinner et al., 1998[17]	4
Tompkins et al., 2010[58]	4
Miller et al., 1987[59]	3
Miller, 1982[60]	3
Smith et al., 1998[61]	–1

Table 3 *Number of cases by site of avulsion fracture and associated causative mechanism*

Site/ Mechanism	Running	Kicking	Extreme ROM	Jumping	Surgical Hx£	Other	Total	%
ASIS	6	2	1	0	7	3*	19	28.8
AIIS	4	8	0	1	0	0	13	19.7
PC	0	0	1	0	0	0	1	1.5
AC	0	0	1	0	0	0	1	1.5
IC	7	1	0	0	0	2¤	10	15.2
IT	10	2	4	1	1	4§	22	33.3
Total	27	13	7	2	8	9	66	
%	40.9	19.7	10.6	3.0	12.1	13.6		100.0

* 1 breakdancing, 1 raising from seated position, undefined athletic training.
¤ 1 wrestling, 1 batting.
§ 1 weighted squatting, 1 raising from seated position, 1 getting out of vehicle, 1 skating.
£ All cases occurred following harvesting of IC for grafting except for IT case that followed total hip replacement.
ASIS – Anterior Superior Iliac Spine, AIIS – Anterior Inferior Iliac Spine, PC – Pubic Crest, AC – Acetabulum, IC – Iliac Crest, IT – Ischial Tuberosity.

ence between groups (physical activity versus surgery). A statistically significant difference was found with $z = 4.19$ and $p < 0.00$ with 12% of the activity-related papers scoring five or less on the Causation Criteria, and 40% of the surgical cases scoring five or less on those same criteria. A summary of the Causation Criterion Scores for all papers is summarized in Table 2 (detailed results are available upon request as Appendix 2).

Discussion

To date the largest analysis of avulsion fracture of the pelvis is a retrospective analysis of competitive athletes by Rossi and Dragoni, where they analyzed 203 cases that presented to an Italian Sports clinic.[9] The authors reported an average age of 13.8 with 68.5% of cases occurring in males.[9] The percentage of males reported with avulsion fracture is significantly lower than what was found by the authors, however this could be a reflection of the larger sample size. Rossi and Dragoni further reported that the IT, ASIS and AIIS were the three most commonly reported sites of avulsion fracture in the pelvis, which is in accordance with the results of the activity related cases in this study.[9]

Typically avulsion fractures are described as having a specific presentation and more importantly a specific patient history involving a forceful muscular contraction during sport activities.[2,6,7,9,10] Patients are frequently skeletally immature and experience pain and a popping sensation at the site of injury.[2,6,7,9,10] The most commonly reported sites of avulsion fracture in the pelvis are the ASIS, AIIS and IT.[7,9,41] The results of this review are in agreement with previous work; however, several important factors have been revealed. Although avulsion fractures most commonly occur in a skeletally immature population, specifically in the cases involving physical activity, a population of skeletally mature post surgical patients was identified as being at risk. Within the cases involving physical activity as a causative agent, there were cases in the literature of avulsion fractures occurring in skeletally mature patients. These cases involved extreme ranges of motion and likely an associated protective forceful muscular contraction.

The strength of causative association evaluated in this study showed that the mean score for 76% the activity related cases placed them in the definite category while in the surgical groups 60% scored in the probable group.

It appears from these results that there is a very strong causal association between physical activity and avulsion fracture. Although there does appear to be a causal association between the surgical group and avulsion fracture it is significantly different from the causal link with activity ($p = 0.00$). This difference may be due to the significantly fewer number of cases available in the literature at this time. Another explanation may be that there was much less thorough reporting in the surgical cases than the activity cases.

The role of apophysitis in the occurrence of avulsion fracture has been proposed in the literature.[13–20] It is thought that perhaps apophysitis may act as a precursor. Saunder et al. discuss that avulsion fractures may occur in cases where no acute episode is present and instead results from chronic repetitive traction on a developing apophysis.[10] In the current study 6 cases were presented with a history of preexisting boney pain in the area of avulsion fracture representing preexisting osteitis.

Of the cases involving physical activity the most commonly reported mechanisms were kicking (19.7%) and running (40.9%) (Table 2). This is in accordance with previous work identifying soccer and running sports as the two most prevalent activities associated with avulsion fractures.[5,9,43] Rossi and Dragoni reported soccer and gymnastics as the two activities most commonly related to the onset of avulsion fractures of the pelvis.[9] Reporting of the mechanism of injury was poor across the majority of cases analyzed making it impossible to confirm eccentric loading as the prime mechanism of injury. Cases were not reported first hand and there may have also been difficulty recounting the details of injury by the patient as many of these injuries occur in a very short period of time and in a very dynamic manner. Also proper historical data on pre-exercise warm up activities and pre-season strengthening programs may be of value in understanding possible preventative mechanisms.

All but one of the cases proposing history of surgery as a causative mechanism for avulsion fracture involved bone harvesting from the iliac crest for the purpose of bone grafting elsewhere in the body. Authors have suggested that removal of a segment of bone close to the ASIS may predispose skeletally mature patients to avulsion fractures in this region.[56] The current study revealed 7 cases of avulsion fracture in the area of the ASIS as a result of bone harvesting from the iliac crest. In response to the occurrence of avulsion fractures following bone harvest from the iliac crest, authors have recommended using a site 3–4 cm posterior to the ASIS.[62] In these 7 cases the details of the mechanism of injury such as hip or knee positioning, were poorly described leaving clinicians with no indications for possible preventative measures. Reporting of preexisting co-morbidities such as osteoporosis and osteomalacia are also essential details needed in these cases.

Three cases of sciatic nerve irritation and one case of meralgia paresthetica were presented in the literature. Patients may present to their primary care provider with chief complaints revolving around these two pathologies. Thorough history and physical examination are essential to rule out the various causes of these pathologies.

The diagnosis of avulsion fracture in nearly all cases was made using radiographic imaging, although use of CT, MRI and bone scan was also reported in the literature. Patient history was an integral part of diagnosis and typically revealed a history of physical activity with forceful muscular contraction followed by a popping sensation and pain in the affected area resulting in difficulty with ambulation. The clinical presentation of an avulsion fracture is often identical to that of a simple muscle strain and therefore must always be considered when dealing with at risk populations. In the cases with a history of surgery present, often minimal trauma such as tripping or rising from a seated position was associated with the onset of symptoms.

Treatment for avulsion fractures included surgical or conservative interventions. The determinant for treatment method depended on the site of avulsion and the amount of displacement of the avulsed segment. Some authors recommend that fragments displaced greater than 2 cm be treated surgically.[14,34,46] While 53% of IT avulsion fracture cases in the Rossi and Dragoni paper were treated conservatively[9], a study by Barnes et al. reported that 68% of AF of the IT do not reunite which may lead clinicians to consider surgery for AF affecting this area.[63] Another argument for surgical intervention does exist for the athletic population arguing improved return to full function, however no difference was noticed in the current study regarding recovery times for surgical versus conservative intervention.[45]

The typical conservative treatment included a period of bed rest for 3 days followed by a progressive ambulation

program typically involving a period of crutch use until the patient was able to walk without pain. A continual increase of pain free activity was prescribed with return to full sport occurring around the 6 week mark. A similar approach is also utilized for post operative therapy. Although this was the trend, no set guidelines or protocols have been reported on in regards to conservative or surgical treatment. Metzmaker and Pappas[2] propose a five stage conservative treatment timeline which correlates patient perceived pain, palpation findings, range of motion, muscle strength and osseous separation to the amount of activity the patient should be engaging in. The level of activity ranges from none to normal pre-injury levels.

All cases involving a history of surgery as a causative mechanism were managed conservatively. This is in accordance with work by Zijderveld et al. where they propose that treatment of iliac crest fractures is most often conservative.[56]

Limitations

Several limitations exist within the study. Only English language papers were included for this search which may have excluded potential cases of avulsion fracture. One must also be cautioned when concluding causality based on low level evidence such as case reports and case series. Due to ethical and logistical concerns, the study of avulsion fractures is limited to retrospective analysis. Therefore the use of a tool such as the Causation Criteria helps establish causality when clinical judgment is typically employed. Often case reports and case series are the only forms of literature that provide enough detail for such an instrument to be utilized. While the sample size for the activity related cases was relatively strong, the surgical cases are highly under reported in the literature, and therefore only allowed for a small sample size in the current study. Lastly, the reliability of the Causation Criteria following the alterations is unknown.

Future Research

The muscles that are typically involved in pelvic avulsion fractures act very differently from a functional point of view on the low back, hip and knee. As such, an understanding of the functional implications of avulsion fractures at each site on the pelvis may be very important for the patient. This information would be of particular importance in the treatment and rehabilitation of these patients both from a surgical and non surgical perspective, and therefore is an important direction for future research.

Conclusion

Patients suffering from avulsion fractures of the pelvis typically present as adolescents engaging in physical activity that requires sudden and forceful muscular contraction that results in a popping sensation with local pain, tenderness and difficulty with ambulation. A special population of skeletally mature patients at risk of avulsion fracture has been identified as those with a history of bone harvest from the iliac crest. A diligent history and physical is required along with radiographic imaging for an accurate diagnosis of avulsion fractures, typically followed by a course of conservative therapy. Surgical consultation may be warranted in cases displaying a displacement greater than 2 cm or involvement with the IT.

References

1 Lynch SA, Renstrom PA. Groin injuries in sport: treatment strategies. Sports Med. 1999; 28(2):137–144.
2 Metzmaker JN, Pappas AM. Avulsion fractures of the pelvis. Am J Sports Med. 1985; 13(5):349–358.
3 Gidwani S, Bircher MD. Avulsion injuries of the hamstring origin - a series of 12 patients and management algorithm. Ann R Coll Surg Engl. 2007; 89(4):394–399.
4 Meyer NJ, Schwab JP, Orton D. Traumatic unilateral avulsion of the anterior superior and inferior iliac spines with anterior dislocation of the hip: a case report. J Orthop Trauma. 2001; 15(2):137–140.
5 Morelli V, Smith V. Groin injuries in athletes. Am Fam Physician. 2001; 64(8):1405–1414.
6 Orava S, Ala-Ketola L. Avulsion fractures in athletes. Br J Sports Med. 1977; 11(2):65–71.
7 Fernbach SK, Wilkinson RH. Avulsion injuries of the pelvis and proximal femur. AJR Am J Roentgenol. 1981; 137(3):581–584.
8 Pisacano RM, Miller TT. Comparing sonography with MR imaging of apophyseal injuries of the pelvis in four boys. AJR Am J Roentgenol. 2003; 181(1):223–230.
9 Rossi F, Dragoni S. Acute avulsion fractures of the pelvis in adolescent competitive athletes: prevalence, location and sports distribution of 203 cases collected. Skeletal Radiol. 2001; 30(3):127–131.
10 Sundar M, Carty H. Avulsion fractures of the pelvis in children: a report of 32 fractures and their outcome. Skeletal Radiol. 1994; 23(2):85–90.
11 Vandervliet EJ, Vanhoenacker FM, Snoeckx A, Gielen JL, Van DP, Parizel PM. Sports-related acute and chronic avulsion injuries in children and adolescents with special

emphasis on tennis. Br J Sports Med. 2007; 41(11):827–831.
12 White KK, Williams SK, Mubarak SJ. Definition of two types of anterior superior iliac spine avulsion fractures. J Pediatr Orthop. 2002; 22(5):578–582.
13 Davis JR, Charalambides C, Bircher MD. Avulsion fracture of the ischium. Injury. 1998; 29(8):632–635.
14 DePalma AF, Silberstein CE. Avulsion fracture of the ischial tuberosity in siblings. A report of 2 cases. Clin Orthop Relat Res. 1965; 38:120–122.
15 Pointinger H, Munk P, Poeschl GP. Avulsion fracture of the anterior superior iliac spine following apophysitis. Br J Sports Med. 2003; 37(4):361–362.
16 Resnick JM, Carrasco CH, Edeiken J, Yasko AW, Ro JY, Ayala AG. Avulsion fracture of the anterior inferior iliac spine with abundant reactive ossification in the soft tissue. Skeletal Radiol. 1996; 25(6):580–584.
17 Spinner RJ, Atkinson JL, Wenger DE, Stuart MJ. Tardy sciatic nerve palsy following apophyseal avulsion fracture of the ischial tuberosity. Case report. J Neurosurg. 1998; 89(5):819–821.
18 Watanabe H, Shinozaki T, Arita S, Chigira M. Irregularity of the apophysis of the ischial tuberosity evaluated by magnetic resonance imaging. Can Assoc Radiol J. 1995; 46(5):380–385.
19 Winkler AR, Barnes JC, Ogden JA. Break dance hip: chronic avulsion of the anterior superior iliac spine. Pediatr Radiol. 1987; 17(6):501–502.
20 Yildiz C, Yildiz Y, Ozdemir MT, Green D, Aydin T. Sequential avulsion of the anterior inferior iliac spine in an adolescent long jumper. Br J Sports Med. 2005; 39(7):e31.
21 Bui-Mansfield LT, Chew FS, Lenchik L, Kline MJ, Boles CA. Nontraumatic avulsions of the pelvis. AJR Am J Roentgenol. 2002; 178(2):423–427.
22 Naranjo CA, Busto U, Sellers EM, Sandor P, Ruiz I, Roberts EA et al. A method for estimating the probability of adverse drug reactions. Clin Pharmacol Ther. 1981; 30(2):239–245.
23 Haldeman S. Principles and Practice of Chiropractic. 3rd ed. New york: McGraw Hill; 2005.
24 Aksoy B, Ozturk K, Ensenyel CZ, Kara AN. Avulsion of the iliac crest apophysis. Int J Sports Med. 1998; 19(1):76–78.
25 Atalar H, Kayaoglu E, Yavuz OY, Selek H, Uras I. Avulsion fracture of the anterior inferior iliac spine. Ulus Travma Acil Cerrahi Derg. 2007; 13(4):322–325.
26 Bahk WJ, Brien EW, Luck JV, Jr., Mirra JM. Avulsion of the ischial tuberosity simulating neoplasm--a report of 2 cases. Acta Orthop Scand. 2000; 71(2):211–214.
27 Bolgla LA, Jones DL, Keskula DR, Duncan JB. Hip Pain in a High School Football Player: A Case Report. J Athl Train. 2001; 36(1):81–84.
28 Byrne A, O'Hare J, Rice P. Bilateral pelvic avulsion fractures. Emerg Med J. 2008; 25(12):853.
29 Deehan DJ, Beattie TF, Knight D, Jongschaap H. Avulsion fracture of the straight and reflected heads of rectus femoris. Arch Emerg Med. 1992; 9(3):310–313.
30 Doral MN, Aydog ST, Tetik O, Atay OA, Turhan E, Demirel HA. Multiple osteochondroses and avulsion fracture of anterior superior iliac spine in a soccer player. Br J Sports Med. 2005; 39(3):e16.
31 Dosani A, Giannoudis PV, Waseem M, Hinsche A, Smith RM. Unusual presentation of sciatica in a 14-year-old girl. Injury. 2004; 35(10):1071–1072.
32 Draper DO, Dustman AJ. Avulsion fracture of the anterior superior iliac spine in a collegiate distance runner. Arch Phys Med Rehabil. 1992; 73(9):881–882.
33 Gomez JE. Bilateral anterior inferior iliac spine avulsion fractures. Med Sci Sports Exerc. 1996; 28(2):161–164.
34 Kaneyama S, Yoshida K, Matsushima S, Wakami T, Tsunoda M, Doita M. A surgical approach for an avulsion fracture of the ischial tuberosity: a case report. J Orthop Trauma. 2006; 20(5):363–365.
35 Karakas HM, Alicioglu B, Erdem G. Bilateral anterior inferior iliac spine avulsion in an adolescent soccer player: a typical imitator of malignant bone lesions. South Med J. 2009; 102(7):758–760.
36 Kusma M, Jung J, Dienst M, Goedde S, Kohn D, Seil R. Arthroscopic treatment of an avulsion fracture of the ligamentum teres of the hip in an 18-year-old horse rider. Arthroscopy. 2004; 20 Suppl 2:64–66.
37 Khoury MB, Kirks DR, Martinez S, Apple J. Bilateral avulsion fractures of the anterior superior iliac spines in sprinters. Skeletal Radiol. 1985; 13(1):65–67.
38 Lambert MJ, Fligner DJ. Avulsion of the iliac crest apophysis: a rare fracture in adolescent athletes. Ann Emerg Med. 1993; 22(7):1218–1220.
39 Mader TJ. Avulsion of the rectus femoris tendon: an unusual type of pelvic fracture. Pediatr Emerg Care. 1991; 7(2):126.
40 Nanka O, Havranek P, Pesl T, Dutka J. Avulsion fracture of the pelvis: separation of the secondary ossification center in the superior margin of the acetabulum. Clin Anat. 2003; 16(5):458–460.
41 Oldenburg FP, Smith MV, Thompson GH. Simultaneous ipsilateral avulsion of the anterior superior and anterior inferior iliac spines in an adolescent. J Pediatr Orthop. 2009; 29(1):29–30.
42 Rajasekhar C, Kumar KS, Bhamra MS. Avulsion fractures of the anterior inferior iliac spine: the case for surgical intervention. Int Orthop. 2001; 24(6):364–365.
43 Rosenberg N, Noiman M, Edelson G. Avulsion fractures of the anterior superior iliac spine in adolescents. J Orthop Trauma. 1996; 10(6):440–443.
44 Salvi AE, Metelli GP, Corona M, Donini MT. Spontaneous healing of an avulsed ischial tuberosity in a young football player. A case report. Acta Orthop Belg. 2006; 72(2):223–225.

45 Schlonsky J, Olix ML. Functional disability following avulsion fracture of the ischial epiphysis. Report of two cases. J Bone Joint Surg Am. 1972; 54(3):641–644.
46 Servant CT, Jones CB. Displaced avulsion of the ischial apophysis: a hamstring injury requiring internal fixation. Br J Sports Med. 1998; 32(3):255–257.
47 Steerman JG, Reeder MT, Udermann BE, Pettitt RW, Murray SR. Avulsion fracture of the iliac crest apophysis in a collegiate wrestler. Clin J Sport Med. 2008; 18(1):102–103.
48 Swischuk LE. Wrestling: pain in the thigh. Avulsion fracture of the anterior inferior iliac spine. Pediatr Emerg Care. 2004; 20(4):259–260.
49 Thanikachalam M, Petros JG, O'Donnell S. Avulsion fracture of the anterior superior iliac spine presenting as acute-onset meralgia paresthetica. Ann Emerg Med. 1995; 26(4):515–517.
50 Vajnar J. A 13-year-old boy with a common athletic injury. Avulsion fracture of the pelvis. JAAPA. 2008; 21(2):75–76.
51 Valdes M, Molins J, Acebes O. Avulsion fracture of the iliac crest in a football player. Scand J Med Sci Sports. 2000; 10(3):178–180.
52 Vogt S, Ansah P, Imhoff AB. Complete osseous avulsion of the adductor longus muscle: acute repair with three fiberwire suture anchors. Arch Orthop Trauma Surg. 2007; 127(8):613–615.
53 Yazzie K. Clip & file. Dx-ray... anterior inferior iliac spine avulsion fracture. Physician Assistant. 2001; 25(22):49–50.
54 Kuhn DA, Moreland MS. Complications following iliac crest bone grafting. Clin Orthop Relat Res. 1986; (209):224–226.
55 Stellon A, Davies A, Williams R. Avulsion of the anterior superior iliac spine complicating bone biopsy. Postgrad Med J. 1985; 61(717):625–626.
56 Zijderveld SA, ten Bruggenkate CM, van Den Bergh JP, Schulten EA. Fractures of the iliac crest after split-thickness bone grafting for preprosthetic surgery: report of 3 cases and review of the literature. J Oral Maxillofac Surg. 2004; 62(7):781–786.
57 Samartzis D, Shen FH. What's your call? Postoperative iliac-crest avulsion fracture. CMAJ. 2006; 175(5):475–476.
58 Tompkins M, Ehrlich M. Bracing treatment for chronic avulsion of the iliac crest apophysis. Clin J Sport Med. 2010; 20(2):122–124.
59 Miller A, Stedman GH, Beisaw NE, Gross PT. Sciatica caused by an avulsion fracture of the ischial tuberosity. A case report. J Bone Joint Surg Am. 1987; 69(1):143–145.
60 Miller M. Avulsion fractures of the anterior superior iliac spine in high school track. Athletic Training Spring. 1982; 17(1):57–59.
61 Smith PN, Gie GA. Avulsion fracture of the ischium following complex total hip arthroplasty: an unusual cause of hip pain. J Arthroplasty. 1998; 13(5):603–606.
62 Hu R, Hearn T, Yang J. Bone graft harvest site as a determinant of iliac crest strength. Clin Orthop Relat Res. 1995; (310):252–256.
63 Barnes ST, Hinds RB. Pseudotumor of the ischium. A late manifestation of avulsion of the ischial epiphysis. J Bone Joint Surg Am. 1972; 54(3):645–647.

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The effects of aerobic physical activity on adiposity in school-aged children and youth: a systematic review of randomized controlled trials

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Context: *The role of aerobic physical activity as a standalone treatment in decreasing adiposity in school-aged children and youth has not been well established.*

Objective: *To systematically search and assess the quality of the literature on the efficacy of aerobic physical activity to decrease adiposity in school-aged children and youth.*

Methods: *An electronic search strategy was conducted in EBSCO databases, including MEDLINE and CINAHL. Retrieved articles that met the eligibility criteria were rated for methodological quality by using the Downs and Black checklist.*

Results: *10 articles met the inclusion criteria in the form of RCTs. Results indicate that five articles had positive results in decreasing adiposity compared to controls and five articles had no change in adiposity compared to controls.*

Conclusion: *There is a paucity of evidence to support aerobic physical activity as a successful standalone treatment for decreasing adiposity. Despite the heterogeneity of the methods there is some evidence to support that school-aged children and youth benefit from*

Contexte : *le rôle de l'activité physique aérobique en tant que traitement autonome afin de réduire l'adiposité chez les enfants d'âge scolaire et les adolescents n'a pas été clairement établi.*

Objectif : *rechercher systématiquement et évaluer la qualité de la documentation sur l'efficacité de l'activité physique aérobique afin de réduire l'adiposité chez les enfants d'âge scolaire et les adolescents.*

Méthodes : *une recherche électronique fut menée dans les banques de données EBSCO, notamment MEDLINE et CINAHL. Les articles consultés qui répondaient aux critères d'admissibilité furent évalués en fonction de leur qualité méthodologique à l'aide de la liste de vérification Downs and Black.*

Résultats : *10 articles répondaient aux critères d'inclusion sous la forme d'essais cliniques aléatoires. Les résultats indiquent que cinq articles démontraient qu'il était possible de réduire l'adiposité par rapport aux contrôles, et que cinq articles démontraient qu'il n'y avait aucun changement par rapport aux contrôles.*

Conclusion : *il existe suffisamment de preuves solides démontrant que l'activité physique aérobique constitue un traitement autonome efficace pour réduire l'adiposité. Malgré l'hétérogénéité des méthodes, il existe des preuves démontrant que les enfants d'âge scolaire et les adolescents bénéficient de l'activité physique*

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aerobic physical activity to decrease adiposity and to limit weight gain.
(JCCA 2011; 55(4):256–268)

KEY WORDS: systematic review, physical activity, exercise, adiposity, body mass index, body composition, children, youth

aérobique pour réduire l'adiposité et limiter la prise de poids.
(JCCA 2011; 55(4):256–268)

MOTS CLÉS : évaluation systématique, activité physique, exercice, adiposité, indice de masse corporelle, composition du corps, enfants, adolescents

Introduction

Physical inactivity is one of the leading causes of major chronic illness tracking from childhood into adulthood[1]. Overweight and obese children are four times more likely to become overweight adults, thus leading to major chronic illnesses such as type-two diabetes, heart disease, and cancer[1]. Physical inactivity is leading to a global epidemic of childhood obesity[1]. In 2010, The World Health Organization (WHO) stated that physical inactivity is the fourth leading risk factor for global mortality.[1] There is a widespread concern in Canada that the prevalence of overweight and obese children and youth are reaching epidemic proportions.[2] Obesity is on the rise in Canada with approximately 26% of Canadian children and youth between the ages of two and 17 years of age over the 90th percentile for body weight.[3]

Canada first set physical activity guidelines for children and youth in 2002 to promote healthy active living in the Canadian population.[4,5] Health Canada, now the Public Health Agency of Canada (PHAC), in conjunction with the Canadian Society for Exercise Physiology (CSEP) developed basic guidelines to give physical activity guidance to Canadian children (5–11) and youth (12–17).[4,5,6,7] The recommended target in 2002 was for children and youth to complete a minimum of 30 minutes of physical activity per day and progressively increase to 90 minutes per day of moderate to vigorous intensity activity.[1,4,5]

As of early 2011, the PHAC in conjunction with the CSEP changed their physical activity guidelines for children and youth.[4,5,6,7] For health benefits, children (5–11) and youth (12–17) should accumulate at least 60 minutes of moderate to vigorous physical activity daily.[4,5,6,7] Thus, there has been a decrease in the recommended amount for physical activity of Canadian children and youth. A common intervention for overweight and obese children is an aerobic physical activity program to decrease adiposity.

Reversing the trend of increasing adiposity in children has been proven to be an extremely difficult task. It has been widely acknowledged that increasing physical activity to increase total energy expenditure and reducing caloric intake may form the theoretical basis for the management of obesity.[4,5,6,7] Thus, increasing aerobic physical activity may be the foundation for decreasing childhood obesity and a first step to behavioural change. Increasing physical activity has been supported in recent years by the WHO, PHAC, and CSEP.[1,4,5,6,7] The objective of this systematic review was to determine the quality of current evidence forming the relationship between aerobic physical activity and adiposity changes in school-aged children and youth. We reviewed randomized controlled trials (RCTs) that assessed the efficacy of physical activity programs for decreasing adiposity in school-aged children and youth with sedentary controls.

Physical Activity Guidelines for Children and Youth

There are currently two separate guidelines published to give physical activity guidance to Canadian children and youth.[4,5,6,7] These new guidelines recommend children and youth should accumulate at least 60 minutes of moderate to vigorous intensity physical activity daily.[4,5,6,7] Moderate physical activity is defined as 3–6.9 metabolic equivalents (METS) for example brisk walking and bicycle riding.[4,5,8] Vigorous physical activity is defined as >7 METS for example running, tennis, and jumping jacks.[4,5,8] Children and youth should engage in vigorous physical activity at least three days per week and resistance training three days per week for health benefits. CSEP also developed the Canadian Sedentary Behaviour Guidelines. Children and youth should minimize the time spent being sedentary each day by limiting recreational screen time to no more than two hours per day which is now consistent with the guidelines published by the Can-

adian Pediatric Society.[4,5,6,7] Screen time refers to the amount of time spent sedentary in front of electronic devices including televisions, computers, and video games.

In 2011 the Canadian physical activity guidelines changed significantly to reflect global harmonization in the guidelines.[1,4,5,6,7] The guidelines now define physical activity participation in an absolute amount and no longer incorporate progression. Previously the physical activity guidelines stated that children and youth should increase the time spent being physically active by 30 minutes per day and progress to a minimum of 90 minutes per day.[4,5,6] Now the guidelines recommend a minimum of 60 minutes per day everyday.[4,5,6,7] Further, the guidelines used to recommend that children and youth should decrease screen time to less than 90 minutes per day.[4,5] Today, it is recommended that screen time should be decreased to less than two hours per day.[4,5,6,7]

Canada's physical activity guidelines formerly pertained to a limited age span of children between six to nine years of age and youth between 10–14 years of age, allowing for specific promotional and educational materials to be directed at different age groups.[4–10] As of 2011 the guidelines have included five year olds up to 17 year olds to comprise a larger target.

The PHAC and CSEP have created physical activity guidelines for the Canadian population to increase healthy active living.[4,5,6,7] A sedentary lifestyle is one of the leading causes of obesity. According to a systematic review performed by Janssen and LeBlanc the relationship between physical activity and obesity in school-aged children and youth has been extensively studied via observational studies.[11] Low-level physical activity studies have reported that there is a weak to modest relationship between physical activity and obesity.[11] Interestingly, studies that have looked at moderate to vigorous physical activity have reported a stronger correlation than low-level physical activity.[11] These studies have reported that there is a dose-response relationship between physical activity and obesity.[11–25] Thus, with an increase in physical activity in school-aged children and youth there will be a decrease in adiposity leading to a decrease in obesity.

We hypothesize that aerobic physical activity, as a standalone intervention will decrease measures of total adiposity in response to an increase in aerobic training in school-aged children and youth.

Methods

Search Strategy

Figure 1 depicts the flow of trials through the review. Studies were identified by searching electronic databases and scanning reference lists of articles identified. The electronic literature search was conducted by one independent reviewer in the EBSCO databases in MEDLINE and CINAHL up to and including December 2010. The MeSH terms used were ["Exercise", "Leisure Activities", "Physical Endurance" OR], ["Body Mass Index", "Body Fat Distribution", "Overweight", "Skinfold Thickness", "Adipose Tissue", "Obesity", and "Body Composition" OR]. Text words were used ["Physical activity", "fitness", "exercise", "energy expenditure" OR] and ["obese", "obesity", "overweight", "body composition", body mass index". All MeSH terms and text words were combined with aerobic* and the following limiters: English language; from 2000–2010; age related: All children and adolescents 0–18 years; and Publication Type: Randomized Controlled Trials. The reference lists from all retrieved papers were reviewed for further relevant articles that may not have been included in the initial electronic search.

Screening of citations

The authors independently examined the electronic search results. The titles and abstracts of the 38 articles found in the search strategy were examined to determine which full-text manuscripts met the eligibility criteria to be included in the review. The eligibility criteria used is indicated in Table 1. The eligibility criteria were applied to all of the obtained full text manuscripts and consisted of studies that were RCTs conducted on children between 0–18 years of age. Interventions included aerobic physical activity with no co-interventions of caloric restriction allowed; these could be compared to a sedentary control group, an active controlled group, or a sedentary control with lifestyle education only. Studies must have had an outcome measure for determining adiposity. Only articles published in a peer-reviewed journal in the English language within the past 10 years (2000–2010) were considered. These criteria were applied to all of the obtained manuscripts. The eligibility assessment was performed independently by two reviewers and if disagreement between reviewers was identified it was resolved by consensus.

Figure 1 *Literature Search Flow*

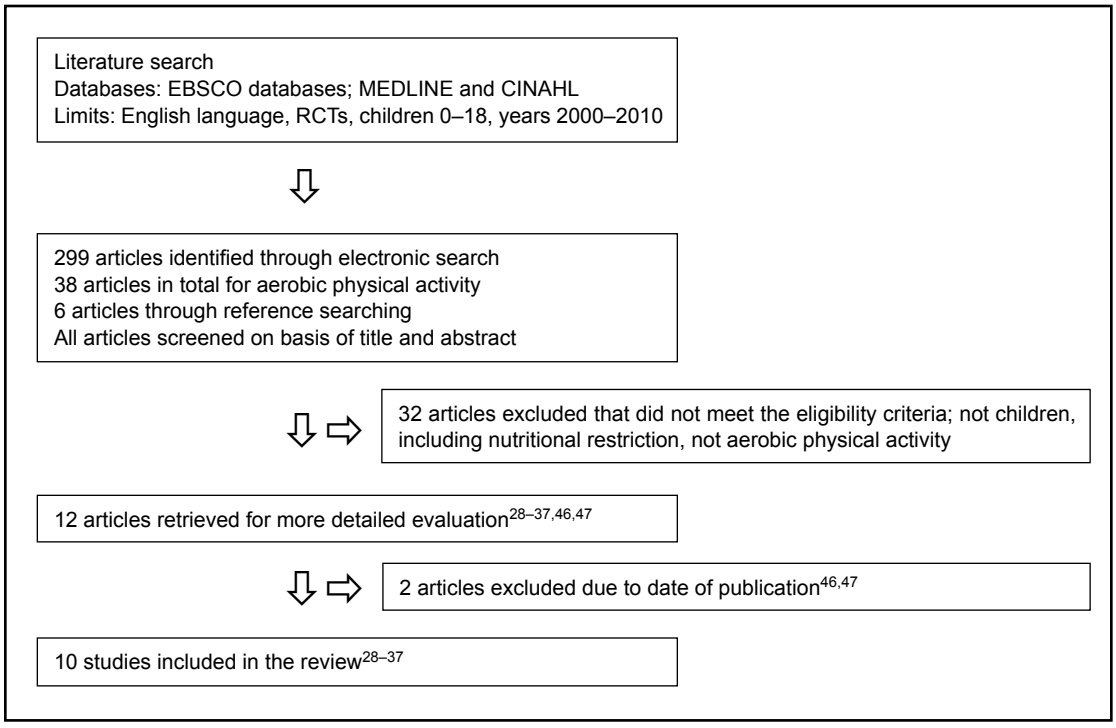

Table 1 *Review of eligibility criteria*

Inclusion Criteria	**Exclusion Criteria**
RCTs	Cross-sectional studies, case-control studies, case studies, prospective or retrospective cohort studies, reviews, or personal narratives
Published in peer-reviewed journal	Published in a non-peer reviewed journal
Published in the English language	Not published in the English language
All children between 0-18 years of age	Adult subjects
Published between January 2000 and December 2010	Published before January 2000
Studies must have used some outcome measure for determining adiposity (body composition, percent body fat, weight, body mass index (BMI), skinfold thickness, trunk and visceral fat composition, and adiponectin levels)	Studies without an outcome measure
Intervention group provided with aerobic physical activity	Intervention group did not perform any aerobic physical activity or included nutrition in the intervention
Control group consisting of sedentariness, maintaining current levels of physical activity, or sedentariness with lifestyle counseling	No control group

**For the purpose of this review fitness and exercise are considered proxy measures for physical activity*

Data collection process

The authors independently reviewed the published studies meeting the inclusion criteria and conducted a critical appraisal of the studies. The following items were extracted: objectives, interventions, outcome measures used, results, adverse events, and drop-outs. The specific design of the study was extracted to ensure it was a RCT. Details of the population used including all baseline characteristics and samples sizes for the intervention groups and the control groups were recorded. Specific details of the intervention groups and control groups were extracted including the frequency, intensity, and duration of the intervention, the number of sessions per week, and the format for each intervention (group training sessions, supervised sessions, or individual sessions). (See Table 2 and 3). In addition, the method of randomization, blinding procedures, statistical analysis, sample sizes, and power were extracted and recorded. To ascertain the validity of eligible RCTs, the two reviewers worked independently. With adequate reliability the two reviewers determined the adequacy of the randomization process, the concealment of randomized allocation to groups, blinding of the participants in the study, blinding of the authors and data collectors.

Quality of the Studies

The methodological quality of all studies that met the eligibility criteria were assessed independently by the authors using the Downs and Black checklist.[26] The checklist is considered a reliable and valid tool to report on the methodological quality of randomized and non-randomized studies.[26] (See Table 4).

Due to the extreme heterogeneity in the study results we hypothesized that the effect size may differ between studies according to the methodological quality of the studies analyzed. The Downs and Black assessment of power is provided in Table 5. There are 27 questions with a total score out of 32.[26] Scores above 20 were considered of high methodological quality; 11–20 moderate quality; and below 11 were considered of poor methodological quality. All scoring can be found in Table 4. No attempts were made at meta-analysis as there was too much heterogeneity between the studies to allow for a suitable synthesis.

Results

A total of 10 RCTs were identified for inclusion in the review.[28–37] The search of EBSCO databases in MEDLINE and CINAHL using all MeSH terms and search terms combined with limiters produced a total of 299 articles. To specify only aerobic physical activity aerobic* was added as a text word and resulted in 399 articles. Adding all the MeSH terms with all search terms, including aerobic*, and limiters resulted in 38 articles. Of these, 32 studies were discarded because after reviewing the titles and abstracts it appeared that these papers did not meet the inclusion criteria of the review. These studies were primarily discarded due to inclusion of a nutritional intervention or exclusion of aerobic physical activity in the intervention group. The full-text of the remaining six citations were examined in more detail. It appeared that all six studies met the inclusion criteria and were included in this review. An additional six studies met the criteria for inclusion and were identified by checking the references of relevant papers. Two of the additional studies were excluded because they did not meet the inclusion criteria. No unpublished preliminary studies were included in this review. (See Figure 1) Appendix 1 provides a summary of the results for each of the 10 studies.

Methodological quality

Table 4 depicts the quality score of each of the included studies. The Downs and Black checklist was used to adequately evaluate the methodological quality of all 10 of the reviewed studies and allowed for greater objectivity in the results.[26] All 10 of the RCTs reviewed contained several significant methodological limitations. Many of the intervention group sizes were too small and not fully representative of the entire population. The lack of power was a significant issue for most studies and few studies addressed this deficiency.[28,30,31–36] Aguilar at al.[37] and McMurray et al.[29] had the largest intervention groups and thus ultimately had significant power ratings.

The majority of the studies (n = 7) included in this review were of moderate methodological quality.[28,31–36] Aguilar et al.,[37] McMurray et al.,[29] and Farpour-Lambert et al.[30] were all considered to be of high methodological quality.[29,30,37] The highest score on the Downs and Black scoring system was 23/32 achieved by the Aguilar et al. study.[37]

All 10 of the studies failed in reporting the important adverse events that may have been a consequence of the intervention.[28–37] Adverse events such as musculoskeletal

Table 2 *Study Characteristics*[28-37]

Reference	N	Power	Age	Sex	Subjects	Total Score	Intervention outcome
Aguilar et al.[37]	1044	5/5	9–10	Both	Average weight children and adolescents	23/32	Decreased BMI
McMurray et al.[29]	1140	5/5	11–14	Both	Average weight children and adolescents	22/32	No change in BMI or body composition
Farpour-Lambert et al.[30]	44	0/5	6.5–10	Both	Pre-pubertal obese children	21/32	Decreased BMI, body composition, abdominal fat, & triglycerides
Meyer et al.[32]	67	0/5	11–16	Both	Obese adolescents	19/32	Decreased BMI, waist/hip ratio, triglycerides
Kelly et al.[33]	20	0/5	Mean 10.9	Both	Over-weight children and adolescents	18/32	No significant difference in body weight, BMI, percent body fat, triglycerides
Gutin et al.[34]	80	0/5	13–16	Both	Obese adolescents	17/32	Decreased total body composition
Heyman et al.[28]	16	0/5	Mean 16.1	Female	Type 1 Diabetes	16/32	Unchanged body fatness Increased body fatness on controls
Hagstromer et al.[35]	31	0/5	13–15	Both	Obese adolescents	16/32	No change in BMI & total body weight
Kelly et al.[31]	19	0/5	Mean 10.8	Both	Over-weight	15/32	No change in total body weight, adipocytes, or adipokines
Tan et al.[36]	60	0/5	9–10	Both	Obese children	14/32	No difference between BMI, skinfolds & waist girth

**Results listed in order of methodological quality based on the Downs and Black checklist.[22]*
**All study designs are RCTs.*

Table 3 *Characteristics of Exercise Intervention*[28-37]

Reference	Frequency d/wk	Duration mins/d	Length Wks/mos	Intensity
^Aguilar et al.[37]	3 d/wk	90 mins/d	28 wks	Unknown
McMurray et al.[29]	3 d/wk	30 mins/d	8 wks	Unknown
^ Farpour-Lambert et al.[30]	3 d/wk	60 min/d	3 mos	55–60 max HR
^ Meyer et al.[32]	3 d/wk	60–90 mins/d	6 mos	unknown
Kelly et al.[33]	4 d/wk	30–50 mins/d	8 wks	50–80% $V0_2$max
^ Gutin et al.[34]	5 d/wk	Unknown	8 mos	Grp 2: 55–60% $V0_2$max Grp 3: 75–80% $V0_2$max
Heyman et al.[28]	2 d/wk	90 mins/d	6 mos	80–90% max HR
Hagstromer et al.[35]	1 d/wk	60 mins/d	13 wks	unknown
Kelly et al.[31]	4 d/wk	30–50 mins/d	8 wks	50–80% $V0_2$max
Tan et al.[36]	5 d/wk	50 mins/d	8 wks	Lactate Threshold

** Results listed in order of methodological quality based on the Downs and Black checklist.*[24]
^ Denotes significant change in outcome for intervention group.

injuries may occur in physical activity programs. Also, the majority (n = 8) of the studies failed in reporting the characteristics of the subjects that were lost to follow-up.[28–30,33–37] Four of the studies reviewed achieved a score of zero for external validity highlighting that the authors failed to describe if the subjects were representative of the entire population.[31–33,35] Four of the studies reviewed achieved a score of two for external validity highlighting that some of the reviewed studies did report if the subjects, staff, and facilities were representative of the entire population.[28,30,34,37]

The majority of the studies (n = 9) failed in reporting the blinding procedures and if there was compliance with the intervention.[28,29,31–37] Shortcomings in selection bias were also evident in the majority of the studies that were reviewed. Eight of the studies failed to report if the randomization process was concealed from both parents and health care staff until recruitment was complete.[28,29,31–36]

Only two of the studies reviewed had significant power to detect a clinically important effect of physical activity on adiposity according to the Downs and Black checklist.[29,37] A small intervention group will not have significant power to see a decrease in adiposity with a physical activity intervention. Two of the studies revealed a power rating of 5/5 on the Downs and Black checklist, although this is due to large intervention groups.[29,37] Conversely, eight of the other studies reviewed had a rating of 0/5 on the Downs and Black checklist due to small intervention groups.[28,30–36] It may be possible to rate question 27 on the Downs and Black checklist as 1/1 for significant power and 0/1 insufficient power. This review did not change the Downs and Black checklist but there have been recent papers that have changed question 27.[29,38] Thus, it is important to bring to the readers' attention the potential flaw of question 27 on the Downs and Black checklist.

Participants

The sample sizes in the 10 studies ranged from 16 to 1140.[28–37] Five of the studies included sample sizes that ranged from 44 to 90.[30,31] All the studies had participants that ranged in age from 6.5 to 18.5 years old.[28–37] Six of the studies included subjects that were considered to be over the 90th percentile in weight.[30–34,36] Three of the studies used normal healthy weight children.[29,35,37] Only one study looked at subjects that were previously diagnosed with diabetes and were considered to be overweight or obese.[28]

Primary Outcome

It is important to note that of all the RCTs examined,

Table 4 *Methodological quality scoring method*

No.	Item Description	Hagstromer et al.[35]	Kelly et al.[31]	Meyer el al.[32]	Gutin et al.[34]	Heyman et al.[28]	McMurray et al.[29]	Kelly et al.[33]	Tan et al.[36]	Aguilar et al.[37]	Farpour-Lambert et al.[30]
1	Hypothesis/aim/objective described?	1	1	1	1	1	1	1	1	1	1
2	Main outcomes to be measured described?1	1	1	1	1	1	1	1	1	1	1
3	Characteristcs of patients described?	1	0	1	1	1	1	1	0	0	1
4	Interventions of interests clearly described?	1	1	1	1	1	1	1	1	0	1
5	Distributions of confounders described?	2	2	2	1	2	0	2	0	2	2
6	Main findings clearly described?	1	1	1	1	1	1	1	1	1	1
7	Estimates of random variability in data?	1	1	1	1	1	1	1	1	1	1
8	Important adverse events reported?	0	0	0	0	0	0	0	0	0	0
9	Described patients lost to follow-up?	0	1	1	0	0	0	0	0	0	0
10	Actual probability values reported except where P values <.001?	0	0	1	1	0	1	1	0	1	1
11	Subjects asked to participate representative of entire population?	0	0	0	1	1	0	1	0	1	1
12	Subjects prepared to participate representative of population?	0	0	0	0	1	1	0	1	1	1
13	"Staff, places, facilities representative of treatment majority of patients receive?"	0	0	0	1	0	0	0	0	0	0
14	Attempt made to blind subjects?	1	1	1	1	1	1	1	1	1	1
15	Attempt made to blind those measuring the outcomes to intervention?	0	0	0	0	0	0	0	0	0	1
16	"Any of the results based on ""data dredging,"" was this made clear?"	1	1	1	1	1	1	1	1	1	1
17	"Analysis adjust for different lengths of follow-up of patients, or is this time period between the intervention and outcome the same for cases and controls?"	1	0	1	0	0	1	1	1	1	1
18	Statistical tests appropriate	1	1	1	1	0	1	1	1	0	1
19	Compliance with treatments reliable?	0	0	0	0	0	0	0	0	0	1
20	Outcomes measures valid/reliable?	1	1	1	1	1	1	1	1	1	1
21	Patients in intervention groups or cases and controls recruited from the same population?	1	0	1	1	1	1	1	1	1	0
22	Subjects in different intervention groups or cases and controls recruited over same time period?	0	0	0	0	0	0	0	1	1	1
23	Subjects randomized to groups?	1	1	1	1	1	1	1	1	1	1
24	Randomized assignments concealed until recruitment was complete?	0	0	0	0	0	0	0	0	1	1
25	Adjustment for confounding in analyses?	1	1	1	1	1	1	1	1	1	1
26	Losses to follow-up accounted for?	0	1	1	0	0	0	0	0	0	0
27	Sufficient power to detect clinically important effect where P value for differences due to chance is <5%	0	0	0	0	0	5	0	0	5	0
	Total Score out of 32	16/32	15/32	19/32	17/32	16/32	21/32	18/32	15/32	23/32	22/32

obesity was not always the primary health outcome measured. In many of the experimental studies the primary aim of the study was to improve other health measures and not specifically measures of adiposity.[28–33] All 10 of the studies looked at different methods of measuring obesity including weight, body mass index, skinfolds, percentage of body fat, waist circumference, trunk and visceral fat composition.[28–37] The most common control intervention used within the studies was a sedentary control group (n = 5). Four of the studies maintained current levels of physical activity as a control intervention and one study used a sedentary control group with lifestyle counseling. The time of the exercise session varied between all 10 studies in this review. The length of each intervention ranged from eight weeks to eight months in duration. The results show that interventions of longer duration can have beneficial effects on adiposity in children and youth.[30,32,34,37] The frequency of exercise prescription also ranged significantly from one day per week to five days per week of aerobic exercise. The duration of each session varied between studies from 30 minutes per day to 90 minutes per day. (See Table 3).

Five of the exercise intervention studies showed no change in body composition, percentage of fat, or body mass index in response to a training program.[29,31,33,35,36] Four of the exercise intervention studies showed a decrease in body mass index, hip to waist ratio, or body fat composition in response to the training program.[30,32,34,37] Heyman et al. showed an increase in body fat composition in response to an aerobic training program of six months duration however less weight gain compared to controls.[28] Only Hagstromer et al. employed more than aerobic physical activity by incorporating strength training into the intervention.[35] However, Hagstromer et al. did not show a change in body mass index after 13 weeks of exercise compared to the control group.[35]

According to Aguilar et al., the study with the highest methodological quality, aerobic physical activity three

Table 5 *Downs and Black assessment of power*[27]

Downs and Black	Size of smallest intervention grp	Power	Score
A	<58	.70	0
B	59–72	.80	1
C	73–82	.85	2
D	83–96	.90	3
E	97–118	.95	4
F	>119	.99	5

days per week for 90 minutes for 28 weeks in duration can significantly decrease body mass index.[37] The intensity of exercise in the intervention group in Aguilar et al. study is unknown.[37]

Discussion

Overall, the evidence is not sufficiently strong to determine the efficacy of aerobic physical activity as a standalone treatment for decreasing adiposity of school-aged children and youth. Only five RCTs showed positive results of aerobic physical activity as a standalone treatment for decreasing adiposity.[28,30,32,34,37] There is a paucity of evidence to suggest that aerobic physical activity should be used as a standalone treatment for obesity.[28,30,32,34,37] Thus, chiropractors and other healthcare practitioners should potentially use a combined approach of diet and exercise to decrease adiposity in school-aged children and youth.

There is considerable evidence on the health benefits of physical activity in children and youth.[8,11,40] Leading a physically active lifestyle is an extremely important part of healthy living for children and youth.[8] Strong et al. performed a systematic review of the literature to evaluate the evidence linking physical activity to health and behavioural outcomes in school-aged children and youth.[40] The results showed that physical activity has beneficial effects on adiposity, musculoskeletal health and fitness, cardiovascular health, blood pressure, plasma lipids, lipoprotein levels, and on several components of mental health.[40] Thus, prescribing physical activity as part of a healthy lifestyle is an important component of clinical practice.

Due to variation in the age, sex, exercise frequency, exercise intensity, and exercise duration, limited conclusions can be made on recommendations of aerobic physical activity frequency, duration, and intensity. Also, due to the lack of adequate intervention sizes and power in the majority of the studies (n = 8) it is difficult to draw any definite conclusions regarding whether or not aerobic physical activity effects adiposity in school aged children and youth.

This review shows that there is a paucity of evidence to support that aerobic physical activity has beneficial effects on adiposity of those with normal body mass and those with a body mass in the 90th percentile of weight.[30,32,34,37] However, fifty percent of the studies did not find a decrease in adiposity. Thus, for the practicing chiropractor there is limited evidence to support the sole use of aerobic physical activity to treat obesity based on the results of five positives studies and five studies with no change and all with varying quality. This review shares similar results to a meta-analysis on the effects of school based physical activity programs on BMI in children.[41] The results of the meta-analysis performed by Harris et al. showed that school-based physical activity programs may not be sufficient to decrease BMI in school-aged children.[41] The author further concluded that a combined approach of diet and exercise may be the appropriate treatment measure for increased adiposity in young children and youth.[41]

A recent study analyzing sedentariness, screen time, physical activity, and nutrition found that all may be significant contributors to decreasing body composition in children and youth and should be incorporated into any intervention.[40,45] The role of proper nutrition and caloric restriction combined with a physical activity program may play a role in decreasing adiposity in school-aged children and youth. Knopfli et al.[42] performed a study on the effects of an eight-week multidisciplinary inpatient program on body weight, body composition, aerobic fitness, and quality of life in obese children and youth. The results showed that a multidisciplinary inpatient treatment program including moderate calorie restriction to 1200–1400k/cal per day, daily physical activity of 60 to 90 minutes in duration two times per day, and behaviour modification can significantly decrease adiposity in school-aged children and youth.[42]

There are currently no intervention studies that have reviewed the health effects of the previous or current Canadian physical activity guidelines.[4,5,6,7] Based on Janssen and LeBlanc's systematic review on the health benefits

associated with physical activity, there is strong evidence to suggest as little as two to three hours per week of moderate to vigorous physical activity are associated with health benefits.[11] Thus, it seems appropriate to set a minimal target of 30 minutes per day of physical activity that reflect the low levels of physical activity in inactive and sedentary children and youth.[11] However, a recommendation of at least 60 minutes per day is more appropriate for overall health benefits.[1,11] Implementing an average of 60 minutes per day instead of 60 minutes everyday has been recommended because physical activity on a daily basis has not been validated within the current literature.[11] It is known that physical activity should be at least moderate intensity but more consideration on the impact of low intensity activities on health benefits for children and youth is needed.[11] Aerobic activities should make up the majority of the physical activities performed by children and youth because of its effects on body mass index, cardiovascular health, blood lipid profiles, and metabolic syndrome.[11]

This review has some possible limitations including the limitations in the original literature itself; there may be inherent bias in the original studies. Another possible limitation in this review is the language bias as we only permitted articles published in the English language. The studies reviewed predominately involved young children who may not have appreciated the importance of not breaching protocol from their assigned groups, thus corrupting the study to some content. We did not search EMBASE, MANTIS, or Cochrane libraries. However, we did conduct a thorough search strategy using two electronic databases with hand reference searching of obtained articles, thus measures were taken to evaluate the current state of literature. It could be argued that a weakness in this review was the inclusion criteria. This systematic review only included RCTs according to the eligibility criteria employed to review the highest quality of evidence. Only reviewing RCTs can exclude other clinical studies that may change the results of this review.

The primary limitation amongst the selected studies was the lack of homogeneity of the study designs and thus definitive conclusions in regard to frequency, intensity and duration of aerobic physical activity to decrease adiposity is difficult to make. There are no studies in the current literature that utilize the current or previous guidelines created by CSEP for their intervention strategies. In particular, future research on physical activity interventions should consider utilizing the Canadian physical activity guidelines for children and youth outlined by CSEP to determine exact exercise prescription.

Future research in this area should take place to determine the rates at which chiropractors recommend or prescribe physical activity to decrease adiposity in children and youth. Future systematic reviews should be conducted to assess the value of only caloric restriction on childhood obesity. Moreover, the combination of both physical activity and caloric restriction should be addressed in a systematic review of the literature. The current body of literature on physical activity and obesity in children is lacking evidence for a combined approach of increased physical activity and decreased caloric intake for weight loss or overall changes in body composition and adiposity. It may be hypothesized that a combined approach of caloric restriction and physical activity may essentially be an effective method for decreasing adiposity in school-aged children and youth.

Chiropractors and other healthcare practitioners need to educate themselves, parents, and children on the benefits of regular physical activity and sport participation. A combined approach of physical activity and caloric restriction may be the most effective method to combat childhood obesity. Chiropractors and other healthcare practitioners should promote a healthy lifestyle by emphasizing proper nutrition and following the physical activity guidelines. Parents can strongly influence physical activity behaviours in children through role-modeling and direct involvement in physical activities.[43,44] Parental influence on physical activity may also track into adulthood leading to more physically active adults and less health complications.[43,44] There is a growing need for chiropractors to play a more influential role in the community when it comes to physical activity and sport participation in young school-aged children and youth.

Conclusion

The childhood obesity epidemic is rapidly growing and affects all socioeconomic levels and ethnicities.[44] Excessive weight in children and youth is linked to cardiovascular disease, orthopedic problems, and psychosocial constraints that track into adulthood.[44] The increase in sedentary behaviour and decrease in aerobic fitness may cause an increase risk of obesity in school-aged children

and youth.[45] A focus on early physical activity intervention is thus urgently needed. There is a paucity of evidence to support aerobic physical activity as a successful standalone treatment for decreasing adiposity. Despite the heterogeneity of the methods there is some evidence to support that school-aged children and youth benefit from aerobic physical activity to decrease adiposity and to limit weight gain. Results of this systematic review make it difficult for chiropractors and other healthcare providers to justify the recommendation of aerobic physical activity as a standalone treatment for decreasing adiposity. Further, an effective approach to weight loss may be a combination of physical activity and caloric restriction. Further research is necessary before any definitive statements can be made with regards to the recommendation of only aerobic physical activity and its effects on adiposity in young children and youth.

References

1 World Health Organization. Global Strategy on Diet, Physical Activity, and Health. The World Health Organization; 2010.

2 Tremblay MS, Willms JD. Is the Canadian childhood obesity epidemic related to physical inactivity? International J Obesity. 2003, 27: 1100–1105.

3 Lau DCW, Douketis JD, Morrison KM, Hramiak IM, Sharma AM, Ur E. Canadian clinical practice guidelines on the management and prevention of obesity in adults and children [summary]. Can Med Assoc J. 2006; 176(8): 1–13.

4 Health Canada, Canadian Society for Exercise Physiology: Canada's Physical Activity Guide for Youth**.** Ottawa: Minister of Public Works and Government Services Canada; 2002.

5 Health Canada, Canadian Society for Exercise Physiology: Canada's Physical Activity Guideline for Children**.** Ottawa: Minister of Public Works and Government Services Canada; 2002.

6 Tremblay MS, Warburton DER, Janssen I, Paterson DH, Latimer AE, Rhodes RE, Kho ME, Hicks A, LeBlanc AG, Zehr L, Murumets K, Duggan M. New physical activity guidelines. Appl Physiol Nutr Metab. 2011; 36:36–46.

7 Tremblay MS, LeBlanc AG, Janssen !, Kho ME, Hicks A, Murumets K, Colley RC, Duggan M. Canadian sedentary behaviour guidelines for children and youth. Appl Physiol Nutr Metab. 2011; 36:59–64.

8 Janssen I. Physical activity guidelines for children and youth. Appl Physiol Nutr Metab. 2007; 32:109–121.

9 Timmons B, Naylor P, Pfeiffer K. Physical activity for preschool children – how much and how? Can J Public Health. 2007; 98:122–134.

10 Hearst W, Sharratt M. Canada's physical activity guides: background, development, and process. Appl Physiol Nutr Metab. 2007; 32:9–13.

11 Janssen I, Leblanc A. Systematic review of the health benefits of physical activity and fitness in school-aged children and youth. International J Behav Nutr Phys Activity. 2010; 7(40):1–16.

12 Eisenmann JC, Bartee RT, Wang MQ. Physical activity, TV viewing, and weight in U.S. youth: 1999 Youth Risk Behavior Survey**.** Obes Res. 2002; 10(5):379–385.

13 Dencker M, Thorsson O, Karlsson MK, Linden C, Eiberg S, Wollmer P, Andersen LB. Daily physical activity related to body fat in children aged 8–11 years**.** J Pediatr. 2006; 149(1):38–42.

14 Laxmaiah A, Nagalla B, Vijayaraghavan K, Nair M. Factors affecting prevalence of overweight among 12- to 17-year-old urban adolescents in Hyderabad, India**.** Obesity (Silver Spring). 2007, 15(6):1384–1390.

15 Hernandez B, Gortmaker SL, Colditz GA, Peterson KE, Laird NM, Parra-Cabrera S. Association of obesity with physical activity, television programs and other forms of video viewing among children in Mexico city**.** Int J Obes Relat Metab Disord. 1999, 23(8):845–854.

16 Janssen I, Katzmarzyk PT, Boyce WF, King MA, Pickett W. Overweight and obesity in Canadian adolescents and their associations with dietary habits and physical activity patterns**.** J Adolesc Health. 2004, 35(5):360–367.

17 Veugelers PJ, Fitzgerald AL. Prevalence of and risk factors for childhood overweight and obesity. CMAJ. 2005; 173(6):607–613.

18 Smith BJ, Phongsavan P, Havea D, Halavatau V, Chey T. Body mass index, physical activity and dietary behaviours among adolescents in the Kingdom of Tonga**.** Public Health Nutr. 2007; 10(2):137–144.

19 Guerra S, Teixeira-Pinto A, Ribeiro JC, Ascensao A, Magalhaes J, Andersen LB, Duarte JA, Mota J. Relationship between physical activity and obesity in children and adolescents**.** J Sports Med Phys Fitness. 2006; 46(1):79–83.

20 Eisenmann JC, Laurson KR, Wickel EE, Gentile D, Walsh D. Utility of pedometer step recommendations for predicting overweight in children**.** Int J Obes (Lond). 2007; 31(7):1179–1182.

21 McMurray RG, Harrell JS, Deng S, Bradley CB, Cox LM, Bangdiwala SI. The influence of physical activity, socioeconomic status, and ethnicity on the weight status of adolescents**.** Obes Res. 2000; 8(2):130–139.

22 O'Loughlin J, Paradis G, Renaud L, Meshefedjian G, Gray-Donald K. Prevalence and correlates of overweight among elementary school children in multiethnic, low income, inner-city neighbourhoods in Montreal, Canada**.** Ann Epidemiol. 1998; 8(7):422–432.

23 Fiore H, Travis S, Whalen A, Auinger P, Ryan S. Potentially protective factors associated with healthful

body mass index in adolescents with obese and nonobese parents: a secondary data analysis of the third national health and nutrition examination survey, 1988–1994**.** J Am Diet Assoc. 2006; 106(1):55–64–64. quiz 76–59.
24 Hanley AJ, Harris SB, Gittelsohn J, Wolever TM, Saksvig B, Zinman B. Overweight among children and adolescents in a Native Canadian community: prevalence and associated factors**.** Am J Clin Nutr. 2000; 71(3):693–700.
25 Kuriyan R, Bhat S, Thomas T, Vaz M, Kurpad AV. Television viewing and sleep are associated with overweight among urban and semi-urban South Indian children**.** Nutr J. 2007; 6:25.
26 Downs SH, Black N. The feasibility of creating a checklist for the assessment of the methodological quality both of randomised and non-randomised studies of health care interventions**.** J Epidemiol Community Health. 1998; 52:377–384.
27 Malcomson KS, Dunwoody I, Lowe-Strong AS. Psychosocial interventions in people with multiple sclerosis: A review. J Neurol. 2007; 254:1–13.
28 Heyman E, Toutain C, Delamarche P, Berthon P, Briard D, Youssef H, Dekerdanet M, Gratas-Delamarche A. Exercise training and cardiovascular risk factors in type 1 diabetic adolescent girls**.** Pediatr Exerc Sci. 2007; 19(4):408–419.
29 McMurray RG, Harrell JS, Bangiwala SI, Bradley CB, Deng S, Levine A. A school-based intervention can reduce body fat and blood pressure in young adolescents. J Adoles Health. 2002; 31:125–132.
30 Farpour-Lambert NJ, Aggoun Y, Marchand LM, Martin XE, Herrmann FR, Beghetti M. Physical activity reduces systemic blood pressure and improves early markers of atherosclerosis in pre-pubertal obese children. J Am Coll Cardiol. 2009; 54:2396–406.
31 Kelly AS, Steinberger J, Olson TP, Dengel DR. In the absence of weight loss, exercise training does not improve adipokines or oxidative stress in overweight children**.** Metabolism. 2007; 56(7):1005–1009.
32 Meyer AA, Kundt G, Lenschow U, Schuff-Werner P, Kienast W. Improvement of early vascular changes and cardiovascular risk factors in obese children after a six-month exercise program**.** J Am Coll Cardiol. 2006; 48(9):1865–1870.
33 Kelly AS, Weitzteon RJ, Kaiser DR, Steinberger J, Bank AJ, Dengel DR. Inflammation, insulin, and endothelial function in overweight children and adolescents: The role of exercise. J Pediatr. 2004; 145:731–6.
34 Gutin B, Barbeau P, Owens S, Lemmon CR, Bauman M, Allison J, Kang HS, Litaker MS. Effects of exercise intensity on cardiovascular fitness, total body composition, and visceral adiposity of obese adolescents**.** Am J Clin Nutr. 2002; 75(5):818–826.
35 Hagstromer M, Elmberg K, Marid S, Sjostrom M. Participation in organized weekly physical exercise in obese adolescents reduced daily physical activity. Acta Pediatrica. 2008; 98:352–354.
36 Tan S, Yang C, Wang J. Physical training of 9-to-10-year-old children with obesity to lactate threshold intensity. Pediatr Exerc Sci. 2010; 22:477–485.
37 Aguilar FS, Martinez-Vizcaino V, Lopez MS, Martinez MS, Gutierrez RF, Martinez SS, Lopez-Garcia E, Rodriguez-Artalejo F. Impact of after school physical activity program on obesity in children. J Pediatr. 2010; 157:36–42.
38 Kaminskyj A, Frazier M, Johnstone K, Gleberzon BJ. Chiropractic care for patients with asthma: A systematic review of the literature. J Can Chiropr Assoc. 2010; 54(1): 24–32.
39 Takito MY, D'Aquino Benicio MH, Lopes Nevri LdC. Physical activity by pregnant women and outcomes for newborns: A systematic review. Rev Saude Publica. 2009; 43(6): 1–10.
40 Strong WB, Malina RM, Blimkie CJ, Daniels SR, Dishman RK, Gutin B et al. Evidence based physical activity for school-aged youth. J. Pediatr. 2005; 146:732–737.
41 Harris KC, Kuramoto LK, Schulzer MS, Retallack JE. Effect of school-based physical activity interventions on body mass index in children: a meta-analysis. CMAJ. 2009; 180(7):719–26.
42 Knopfli BH, Radtke T, Lehmann M et al. Effects of a multidisciplinary inpatient intervention on body composition, aerobic fitness, and quality of life in severely obese girls and boys. J Adoles Health. 2008; 42:119–127.
43 O'Connor TM, Jago R, Baranowski T. Engaging parents to increase youth physical activity: A systematic review. Am J Prev Med. 2009; 37(2):141–157.
44 Norton DE, Froelicher ES, Waters CM, Carrieri-Kohlman V. Parental influence in models of primary prevention of cardiovascular disease in children. Eur J Cardiovasc Nurs. 2003; 2:311–322.
45 Kriemier S, Zahner L, Schindler C et al. Effect of school based physical activity programme (KISS) on fitness and adiposity in primary school children: cluster randomized controlled trial. BMJ. 2010; 340:785–794.
46 Owens S, Gutin B, Allison J, Riggs S, Ferguson M, Litaker M, Thompson W. Effect of physical training on total and visceral fat in obese children**.** Med Sci Sports Exerc. 1999; 31(1):143–148.
47 Ferguson MA, Gutin B, Le NA, Karp W, Litaker M, Humphries M, Okuyama T, Riggs S, Owens S. Effects of exercise training and its cessation on components of the insulin resistance syndrome in obese children**.** Int J Obes Relat Metab Disord. 1999; 23(8):889–895.

Appendix 1 – *Results Table*

Author(s)	Score	Population	Sample Size	Intervention	Outcome
Hagstromer et al., 2008	16/32	10–18 year old adolescents	31 subjects; Exercise grp n = 16 Control grp n = 15	Exercise; 1hr/wk for 13wks Session 1 brisk walking, 2–5 spinning, 6–9 strength training 50–70% 1RM, 10–13 swimming	No change in BMI, total body weight, or BMI SD-score
Kelly et al., 2007	15/32	Overweight children (BMI >85th percentile) Exercise grp 10.0 ± 0.67 Control grp 11.0 ± 0.71	20 children randomly assigned to exercise or control grp	4d/wk for 8wks of stationary cycling wks 1–3 50–60% V02 max for 30 mins wk 4–7 60–70% V02 max for 40 mins wk 8 70–80% V02 max for 50 mins Control grp – maintain normal physical activity	No change in total body weight, adipocytes, or adipokines
Meyer et al., 2006	19/32	11–16 year old obese children Exercise grp 13.7 ± 2.1 Control grp 14.1 ± 2.4	67 obese children randomly assigned to control (34; 17 boys and 17 girls) and exercise grp (33; 17 boys, 16 girls)	3 d/wk for 6 months (Mon: swimming, aqua aerobic class for 60 mins; Wed: Sports for 90 mins; Fri: Walking for 60 mins).	decreased BMI, waist/hip ratio, insulin, insulin resistance, triglycerides, low-density lipoproteins, fibrinogen, and C reactive protein
Gutin et al., 2002	17/32	Obese 13–16 year old youth	80 obese youth were randomly assigned to 1 of 3 grps group 1; lifestyle ed group 2; moderate physical exercise + lifestyle ed group 3; vigorous physical training + lifestyle ed	5 d/wk for 8 months; target HR >170 bpm; mod PA 55–60% V02max, vigorous PA 75–80% V02max	Moderate and vigorous PA decreases total body composition. Vigorous PA is more effective than moderate PA for cardiovascular health. PA enhances body composition in obese children but intensity is unknown
Heyman et al., 2007	16/32	Diabetic girls 13–18.5	16 diabetic girls were randomly assigned to 6 months training program (n = 9) and 6 months non training program (n = 7)	3hrs/wk for 6 months at 80–90% max HR	Body fatness remained the same in the PA grp and increased in the control grp
*McMurray et al., 2002	21/32	11–14 year old children and youth	1140 children and youth were randomly assigned to into 4 grps 1. exercise only 2. education only 3. exercise and education combined 4. control grp	30 mins aerobic exercise 3 d/wk for 8 wks	No change in BMI or body composition but decrease in skinfold thickness after 8 wks
Kelly et al., 2004	16/32	Overweight and obese children (10.9 ± 0.4)	20 overweight and obese children were randomly assigned to exercise grp or control grp	Exercise grp – 4 d/wk for 8 wks of stationary cycling for 30mins 50–60% V02max and increasing over 8 wks to 50 mins 70–80% Control – maintain current levels of PA	No significant difference btwn grps over 8wks in body weight, BMI, percent body fat, LDL, or triglycerides
Tan et al., 2010	15/32	Obese children 9–10 years of age	60 obese children; 26 girls and 34 boys were randomly assigned to 8wk training program at lactate threshold and a control grp	Exercise grp – 5 d/wk for 8wks 50 mins per session for a total of 40 sessions maintaining HR at lactate threshold	No difference between BMI, skinfolds, and waist girth
*Aguilar et al., 2010	23/32	1044 4th–5th grade children from 20 different schools	1044 10 schools were randomly assigned to a 28 week training program and 10 schools to a control group	Exercise group – 3 d/wk for 28 wks 90minutes each session	Significant decrease in BMI in exercise group
*Farpour-Lambert et al., 2009	22/32	Obese children 8.9 ± 1.5 years old	44 pre-pubertal obese children were randomly assigned to an exercise grp (n = 22) or a control grp (n = 22)	3 d/wk 60 mins/d for 3 months at 55–65% V02 max	Exercise grp had a significant decrease in BMI, body composition, abdominal fat, and triglycerides

*Highest methodological quality.

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Chronic Achilles tendinopathy: a case study of treatment incorporating active and passive tissue warm-up, Graston Technique®, ART®, eccentric exercise, and cryotherapy

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Tracy L. Bougie, BPHE, BSc, CSCS, DC**

Objective: *To describe the subjective pain and functional improvements of a patient with chronic Achilles tendinopathy following a treatment plan incorporating active and passive tissue warm-up, followed respectively by soft tissue mobilization utilizing both Graston Technique® and Active Release Techniques®, eccentric exercise, and static stretching in combination with cryotherapy.*

Background: *The primary characterization of chronic Achilles tendinopathy is gradual onset of pain and dysfunction focused in one or both Achilles tendons arising secondary to a history of repetitive use or excessive overload.*

Intervention and Outcome: *Conservative treatment is commonly the initial strategy for patient management. Tissue heating, soft tissue mobilization, eccentric training, and static stretching with cryotherapy were implemented to reduce pain and improve function.*

Summary: *A specific protocol of heat, soft tissue mobilization, eccentric exercise, stretching, and cryotherapy appeared to facilitate a rapid and complete recovery from chronic Achilles tendinopathy.*
(JCCA 2011; 55(4):269–279)

KEY WORDS: Achilles, tendon pathology, tendinopathy, soft tissue therapy, thermotherapy, cryotherapy, eccentric exercise, Active Release Techniques, Graston Technique

Objectif : *décrire la douleur subjective et les améliorations fonctionnelles d'un patient souffrant de tendinopathie achilléenne chronique qui suit un traitement comprenant le réchauffement des tissus actifs et passifs, suivi respectivement de la mobilisation des parties molles à l'aide de la technique Graston® et des techniques Active Release®, de l'exercice excentrique, et de l'étirement statique jumelé à la cryothérapie.*

Information de base : *la tendinopathie achilléenne chronique se caractérise principalement par une douleur et une dysfonction qui évoluent graduellement dans l'un des tendons d'Achille, ou les deux, suite à un usage répétitif ou une surcharge excessive.*

Intervention et résultat : *le traitement conservateur constitue la stratégie initiale de gestion des patients. Le réchauffement des tissus, la mobilisation des parties molles, l'exercice excentrique et l'étirement statique avec cryothérapie furent utilisés pour atténuer la douleur et améliorer les fonctions.*

Sommaire : *un protocole spécifique de chaleur, la mobilisation des parties molles, l'exercice excentrique, l'étirement et la cryothérapie semblent faciliter la récupération rapide et complète d'une tendinopathie achilléenne chronique.*
(JCCA 2011; 55(4):269–279)

MOTS CLÉS : Achille, pathologie des tendons, tendinopathie, thérapie des parties molles, thermothérapie, cryothérapie, exercice excentrique, techniques Active Release, technique Graston

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Introduction

Chronic painful injuries of the Achilles tendon are relatively common in athletes, especially among runners.[1–7] The annual incidence of Achilles disorders in top-level runners has been reported to be between 7% and 9%.[2] In such cases, overuse is generally considered to be the inducing factor, however, the exact pathogenesis has not been demonstrated.[1–4] Postulated alternative theories include poor vascularity, diminished flexibility, heredity, age, gender, as well as endocrine and/or metabolic factors.[3,6] Realistically, the pathogenesis is likely a combination of multiple intrinsic and extrinsic factors.[2] Alfredson (2005) theorizes that physical activity may be involved with provocation of symptoms as opposed to acting as the primary cause of the pathology.[4]

The lack of a conclusive pathogenesis for chronic achilles tendon disorders has resulted in considerable debate regarding the diagnostic terminology used in the literature.[1–3,5,7,8] The terms "tendinitis" and "tendonitis" have been used, despite the absence of scientific evidence indicating inflammation.[1–4] Under diagnostic ultrasound (DxUS) and magnetic resonance imaging (MRI), tendons exhibit a localized area of structural degeneration which has provoked the use of the term "degenerative tendinosis".[5] Recent investigations have indicated that the morphology of tendinosis involves changes in collagen fiber structure and arrangement, an increased amount of interfibrillar glycosaminoglycans (GAGs), and local vasculo-neural growth (neo-vascularisation) within the tendon structure.[4,5,9] Alfredson (2003,2005), an authority on achilles pain disorders, states that it is now common opinion among investigators and clinicians that for chronic pain symptoms arising from a tender area of mid-substance tendon tissue, the term "tendinopathy" should be applied. Alternatively, when chronic pain symptoms are combined with diagnostic imaging showing changes in tendon fiber structure, arrangement, and/or evidence of local neo-vascularisation (via colour doppler ultrasound), the term utilized should be "tendinosis".[4,5,8] These terms could simply be interpreted as representing an academic continuum of severity and/or chronicity, with the former indicating a less progressed, and therefore less severe stage. This is an important distinction when developing a plan of management, as approaching treatment with the sole purpose of reducing inflammation is unlikely to resolve the condition as seen with the use of anti-inflammatory agents for chronic tendinopathies.[10] Terminology aside, clinically determining the diagnosis of chronic Achilles tendinopathy via comprehensive patient history and physical exam is typically not difficult.

This report will describe the clinical presentation and treatment of a case of chronic Achilles tendinopathy which resulted from repetitive athletic activity (running). This case is of particular interest due to the rapid and successful patient response to a treatment plan incorporating an active and passive tissue warm-up, followed respectively by soft tissue mobilization utilizing both Graston Technique® (GT®) and Active Release Techniques® (ART®), eccentric training, and static stretching in combination with cryotherapy. The report will provide an overview of symptomatology, rationale supporting the management strategy, and expected outcomes associated with the diagnosis of chronic Achilles tendinopathy.

Case Presentation

A 40-year-old physically active male presented with intermittent bilateral Achilles pain of approximately 3.5 years duration which initially was felt in the right Achilles following a 7-minute dash in street shoes. Later, the patient began to notice an achy stiffness first thing in the morning, when starting to walk after prolonged sitting, and during the beginning of a regular fitness jog (10 km). The symptoms started out relatively mild and intermittent depending on activity level. Approximately six months later, the patient began training for a marathon and greatly increased the volume of running. During this period, the symptoms in the right Achilles gradually worsened, and the left Achilles began to exhibit similar symptoms. Approximately four months later, the stiffness and discomfort progressed to the point where the patient was limping while walking and was unable to continue training for the marathon. The patient then felt it was necessary to seek care. Orthotics were obtained from a chiropractor, a topical anti-inflammatory from a primary care physician, and physical therapy which included rest, therapeutic ultrasound, general massage, basic calf stretching, and needle acupuncture. The patient attended 10–15 physiotherapy sessions, 10 massage treatments and stated that the sessions provided only temporary relief, as the symptoms would re-occur with even light physical activity. Six months after the initiation of treatment the patient no longer pursued the goal of running a marathon, had

Table 1 *Office Therapy Protocol*

Patient: _____

Plan of Management:

_____ / Week for _____ Weeks.

In Office Protocol:

1. 5 minutes heat + cycle warm-up
2. GT® and ART®
3. Specific Exercise
    a. Slow eccentric calf lowering. 3 Sets of 10 Reps. Within pain tolerance.
4. Flexibility + Ice application (10 minutes)
    a. Gastrocnemius and soleus stretch 3 reps with 30 second hold each

greatly reduced his physical activity, and discontinued the physiotherapy sessions due to no evidence of long term improvements.

After almost a year of reduced activity and self-care, during which time the symptoms lessened but did not resolve, the patient then attended the author's clinic. The patient characterized the pain (present in both achilles tendons but slightly worse on the left) as a bothersome "stiff, achy painful" sensation, with an intensity rating of 6–7 out of 10 as reported on a numeric pain rating scale (zero indicating "no pain" and 10 equalling the "worst pain ever"). Pain was described to be worse in the morning, and after prolonged inactivity, but too much activity also aggravated the pain. Based on self-report, the patient was able to run short distances of not greater than 5 km, but both Achilles were always very painful afterward. The patient had no previous history of significant foot or ankle injuries, related surgeries or traumas, medication or supplement use, and was a non-smoker and non-drinker. As an information technology manager, the daily job demands involved sedentary activities. Systems review and illness history was non-contributory.

Gait and postural observation were unremarkable. Physical examination revealed bilateral Achilles tendon pain induced by palpation which was slightly worse on the left. Upon visual inspection, the left tendon was mildly red, showed no evidence of ecchymosis and also exhibited a visual and palpable enlargement of the mid-substance tendon, just proximal to its insertion at the calcaneus. Mild pain in both tendons was provoked with resisted plantar flexion, passive dorsiflexion, and with bodyweight heel raises. Bilateral palpation of the posterior calf muscle complex subjectively revealed tightness and tenderness in the following muscles; soleus, flexor hallucis longus, flexor digitorum longus, and tibialis posterior. Orthopedic lower limb joint provocative testing indicated normal ligament structure and joint function. Chiropractic evaluation of lumbar, sacroiliac, knee, ankle mortis, subtalar, and tarsal joint motion were within normal limits. Neurological and vascular functions were likewise determined as normal.

Following the examination, the patient was diagnosed with chronic bilateral Achilles tendinopathy. The plan of management included two in-office treatments per week for three weeks, followed by one session every seven to ten days for an additional three sessions. Therefore, the patient received a total of nine sessions over an eight week period. The treatment plan (see Table 1) began with active and passive tissue heating accomplished by five minutes of heat pack application in combination with stationary cycling (see Figure 1). This was followed respectively by GT® (see Figure 2) and ART® (see

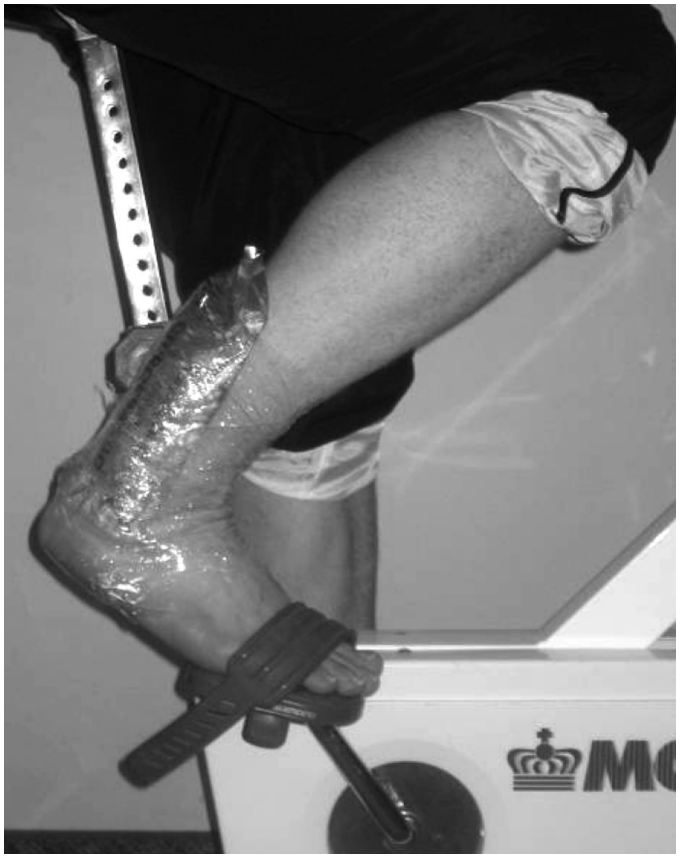

Figure 1 *Active and passive tissue warm-up utilizing a heat pack in combination with stationary cycling.*

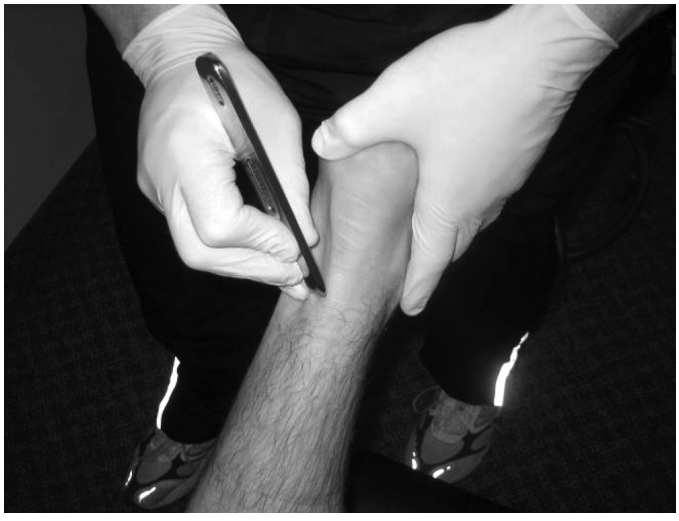

Figure 2 *Graston Technique® performed on the Achilles tendon.*

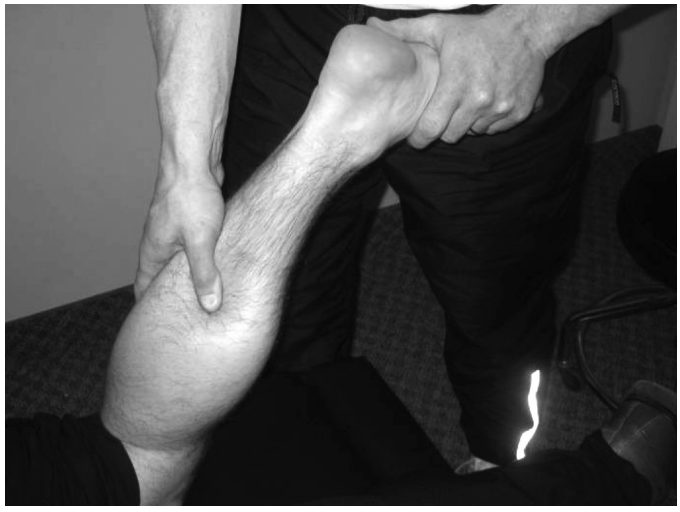

Figure 3 *Active Release Techniques® performed on the gastrocnemius muscle.*

Figure 3) applied to the affected muscles of the posterior leg (gastrocnemius, soleus, plantaris, flexor digitorum, tibialis posterior, and flexor hallucis longus). Slow eccentric calf lowering exercises (see Figure 4) were performed after the soft tissue mobilizations utilizing the sets and repetitions prescription consistent with previously published protocols.[17–22] Finally, static gastrocnemius and soleus stretching was utilized in conjunction with ice pack application (see Figure 5). The patient was also required to follow a specific protocol of home therapy which included ice application, calf stretching, and eccentric heel lowering exercises (See Table 2). The patient was instructed to maintain his current level of physical activity, but not to increase it. During re-evaluation on the sixth visit, the pain level was reduced to 3–4 out of 10 (a 50% improvement), and Achilles discomfort was experienced less often in the mornings, as well as during and after running. Upon conclusion of the plan of management, the patient reported minimal discomfort in the Achilles tendons when squeezing or rubbing them, little to no discomfort with running, no pain with activities of daily living, and a pain level of 0–1 out of 10. Resisted plantar flexion and passive ankle dorsiflexion was non-painful. The patient considered the condition to be almost completely resolved and was thus discharged with instructions to continue with the home therapy protocol for

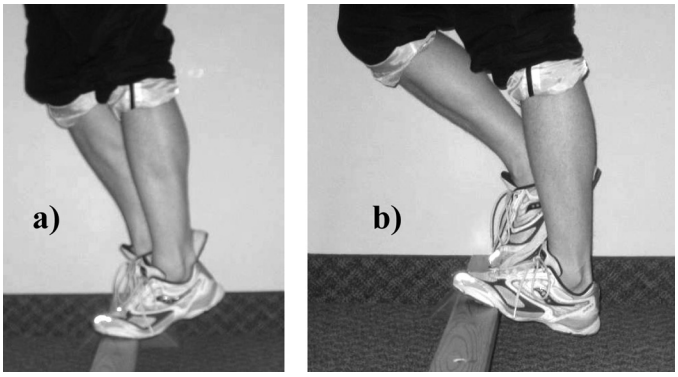

Figure 4 *Eccentric calf strengthening exercise. a) Start position, b) End Position.*

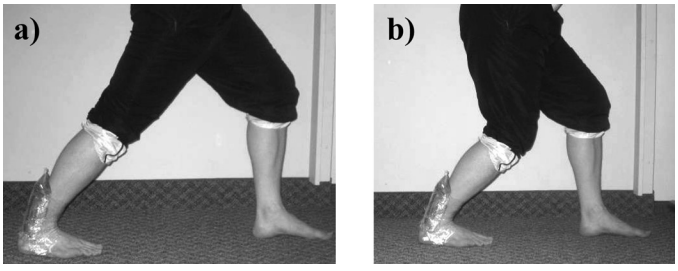

Figure 5 *Static stretching with ice pack application. a) Gastrocnemius stretch, b) Soleus stretch.*

an additional three weeks and to incorporate the eccentric calf lowering exercises into a regular training routine.

During follow up seven months later, the patient reported equal or perhaps slight improvement over the discharged status. Orthopaedic and manual muscle testing were non-provocative and the patient reported a pain level of 0–1 out of 10. It was also noted that the weekly training routine was altered to include cross-training activities (cycling, swimming, circuit training, weights, yoga), and therefore the load on the Achilles was perhaps less than when training for a marathon, but significantly more loading than when the patient initially presented for therapy. The change in exercise pattern to include alternative forms of exercise may have also contributed to the overall improvement in symptoms. In summary, the patient reported to be at, or near, pre-injured physical status with the only residual symptom being a non-painful thickening of the Achilles tendons which was more pronounced on the left compared to the right.

Discussion

As illustrated by the above case presentation, patients with chronic Achilles tendinopathy typically present with a gradual onset of pain and dysfunction focused in one or both Achilles tendons.[11] In athletic patients (competitive or recreational), symptoms generally arise secondary to a history of repetitive use or excessive overload.[1–6,11] Factors included in the clinical presentation may include sudden increases in training volume or intensity, or both, a change of terrain (for example, hill running), an increase in interval training, or a solitary intense run.[7] However, as mentioned previously, overuse is not always associated with the diagnosis.[1–4] The pain associated with Achilles tendinopathy can range from mild to severe, depending on the phase of injury at initial presentation.[11]

Physical examination may reveal pain with resisted plantar flexion and passive dorsiflexion, and the patient will typically find it difficult to stand on tiptoe and/or to perform repeated single leg heel raises.[11] Tenderness may be localized or diffuse, possibly extending several centimeters along the tendon, starting just proximal (2 to 6 centimeters) to the insertion of the Achilles onto the calcaneus.[1–3, 11] The area of tenderness may be accompanied by tendon swelling, fibrous or nodular thickening, and possibly crepitus.[3,11] If the diagnosis of Achilles tendinopathy is still in debate after a careful and thorough history and physical examination, medical imaging including diagnostic ultrasound or magnetic resonance imaging may be warranted.[11]

Most sources of information on the treatment of chronic Achilles tendinopathy suggest a conservative treatment protocol as the initial strategy for patient management, however, evidence is sparse in regards to which conservative treatment method and/or modality is most effective.[1–5,11–13] Conservative treatment options include; eccentric strength training, therapeutic modalities, soft tissue mobilization, rest, corticosteroids, cryotherapy, heat, non-steroidal anti-inflammatory drugs (NSAIDs), deep friction massage, stretching, acupuncture, and podiatry.[1,9,12–32] Among these treatments, calf muscle eccentric strength training, either alone or in combination with one or more of the other therapies, has the most supportive evidence.[12,14–22] It is cost effective and low risk which makes it an ideal first-line therapy.[12,14–22] In patients that have failed first-line conservative therapy, alternative treatments including extracorporeal shockwave therapy

Table 2 *Patient Home Therapy Protocol*

Patient: _____

In Office Management:

_____ / Week for _____ Weeks.

Home Care:

Day of Treatment:

1. Apply ice after treatment at least twice using the "10 minutes on, 10 minutes off, 10 minutes on" protocol. If possible use real ice applied through a wet tea towel. Apply ice no more frequent than once per hour.

2. Perform gentle stretching after the second ice application.
    a. Straight leg calf stretch 2 x 30 sec
    b. Bent knee calf stretch 2 x 30 sec

1st Day after Treatment:

1. Ice at least three times throughout the day using the "10-10-10" protocol.

2. After each ice application perform the above mentioned stretching routine.

2nd Day after Treatment: (and up to next office visit)

1. Once during the day perform the following…
    a. Perform the above mentioned stretching routine 1 x 30 sec
    b. Complete 3 sets of 15 reps of slow eccentric heel lowering exercises within tolerance
    c. Repeat stretch routine for 2 x 30 sec

2. Apply Ice at least two additional times throughout the day using the "10-10-10" protocol.

(ESWT), ultrasound-guided sclerosing agent injections and platelet-rich plasma (PRP) injections show promise in early studies, however, significant additional research is needed.[9,32,33] Most commonly, the conservative therapy approach consists of some combination of the available therapies.[1] Considering that the pathology of chronic Achilles tendinopathy is due to tissue overload and degeneration rather than inflammation, the rationale behind the plan of management developed for the presented patient was to reduce pain and to improve function. The aim was to re-initiate a state of healing and promote new tissue synthesis along lines of function-induced stress, while avoiding re-aggravation. The presented plan of management sought to achieve these outcomes via a specific treatment protocol incorporating an active and passive tissue warm-up, followed respectively by soft tissue mobilization (GT®, ART®), eccentric strength training, and static calf stretching in combination with cryotherapy. A brief review of the evidence in support of each of these specific interventions is presented.

Thermotherapy

Thermotherapy or heat application in a clinical setting has traditionally been used for the management of pain, joint stiffness and/or soft tissue stiffness associated with chronic conditions.[34] Heat therapy application has also been thought to relieve muscle spasms, increase local blood flow, and assist in the resolution of inflammatory infiltrates, edema, and chemical exudates.[35] Evidence on whether heat application can increase the extensibility of collagen fibers and therefore improve the elasticity of muscle, fascia, and tendon tissue remains inconclusive.[36–38] Some evidence does support the use of heat therapy to create vasodilation, increased blood flow, warming of superficial tissues, and an increase in cellular metabolism.[34–37]

Graston Technique®

GT®, otherwise known as "Graston Technique Instrument-Assisted Soft Tissue Mobilization", is a patented form of augmented soft tissue mobilization (ASTM).[28,39,40] The therapy involves the utilization of custom designed stainless steel instruments to augment a clinician's ability to perform soft tissue mobilization (see Figure 2).[39] Essentially, the technique is an instrument assisted form of deep transverse friction massage (DTFM) as proposed by Cyriax in 1975.[25,28] DTFM is theorized to reduce abnormal post injury fibrous adhesions, to make scar tissue more mobile in sub-acute and chronic injury, and to facilitate healing in chronically degenerated soft tissues by inducing controlled micro-trauma and facilitating the normal alignment of soft tissue fibers.[3,25–29] This therapy creates a state of touch induced analgesia, inflammation, hyperemia, and increased fibroblast recruitment and activation, which contribute to the repair and regeneration of damaged collagen.[3,25–29] Experimental animal investigations and human case reports support the above theories.[26–29,39] Although there have been no controlled human studies, GT® and ASTM therapies have been reported to produce positive clinical benefits in the treatment of chronic ankle pain, sub-acute lumbar compartment syndrome, and trigger thumb.[28,29,39]

Active Release Techniques®

ART® is perhaps the most popular of the soft tissue therapy/mobilization techniques utilized by chiropractors and other manual therapists.[40] It is proposed that repetitive or constant micro-trauma in the form of pressure, tension, and/or friction can lead to chronically tight and weak muscles, which eventually progresses to tissue injury, degeneration, and/or inflammation.[38,41,42] If this situation is sustained, tissue adhesions and fibrosis can lead to chronically dysfunctional and painful tissue injuries.[38] This theory is in line with the repetitive overuse pathogenesis theory of chronic Achilles tendinopathy.[3] During ART® therapy, the clinician applies deep digital tension (utilizing either the thumb or fingers) to the affected site as the tissue is moved both actively and passively from a shortened position to a lengthened position (see Figure 3).[38,41] The goal is to improve tissue function by reducing tissue stiffness, fibrosis, and/or adhesion.[38,41] Numerous recent case reports suggest that ART®, in combination with active rehabilitation, is effective in the treatment of conditions including chronic lateral epicondylosis, external coxa sultans and for increasing the strength in a post-operative shoulder.[40,43,44] Future research involving randomized control trials will more conclusively determine the efficacy of ART® in the treatment of musculoskeletal injuries.

Eccentric Exercise

Rehabilitative exercise has long been included in the conservative treatment approach to chronic Achilles tendinopathy.[1–5,11,12,14–16] Although clinical research appears to support the efficacy of eccentric calf loading for the treatment of chronic Achilles tendinopathy, evidence regarding the mechanism of action is still unclear.[17–22] Theories include a direct mechanical effect on tendon pathology in terms of tissue shape and alignment, an improvement in strength which relates to improved function, an improvement in muscle-tendon stiffness, a reduction in tendon volume, or a reduction or reversal of local tendon neovascularisation.[17–19,45,46] Shalabi et al. (2004) found that eccentric training of the gastrocnemius-soleus complex in chronic Achilles tendinopathy resulted in decreased tendon volume and decreased intratendinous signal as evaluated by MRI.[17] In another study, Öhberg and Alfredson (2004) found that painful Achilles tendinosis is associated with a local neovascularisation within the tendon structure, as demonstrated by colour doppler US.[19] Furthermore, after 12-weeks of eccentric calf exercise, 34/36 subjects had decreased pain and improved function and 32/36 subjects had US evidence showing resolution

of the neovascularisation. Öhberg and Alfredson concluded that positive clinical results after eccentric calf muscle training in patients with chronic Achilles tendinopathy seem to be associated with a more normal tendon structure and no remaining evidence of local tendon neovascularisation.[19] A recent systematic review by Kingma et al. (2007), examined the efficacy of eccentric overload training on outcome measures of pain and physical functioning in patients with chronic Achilles tendinopathy.[47] The study included three randomized control trials and six controlled trials. All were prospective in nature and had a mean duration of symptoms between 3.6 and 22 months. Six of the studies used the protocol described by Alfredson et al., while the other three used eccentric exercises with other co-interventions such as stretching, and cryotherapy.[47,48] The duration of the eccentric training was 6 or 12 weeks and was compared to concentric training (three studies), surgery (one study), with a night splint or combined with a night splint (one study) and four studies did not include a control group. The results revealed a mean reduction in pain of 60% with eccentric overload training compared to 33% among controls.[47] Although the effects of eccentric training are promising for reducing pain in chronic Achilles tendinopathy, the full magnitude of its effect cannot be determined due to the lack of satisfactory methodological quality of the studies. The results of the abovementioned findings offer strong support for the inclusion of eccentric calf muscle training in the management of chronic Achilles tendinopathy.

Cryotherapy and Stretching

Ice application (cryotherapy) and stretching are generally considered to be staple components of most injury management plans.[49] In fact, ice is the most often applied therapeutic modality, despite the fact that there is minimal understanding regarding the actual physiological effects on soft tissue.[49] Generally speaking, the effects associated with the application of cryotherapy include a reduction in cell metabolism and blood flow, decreased nerve conduction velocity, and decreased muscle spindle activity.[34] Together, these physiological responses lead to the therapeutic effects of decreased secondary cell hypoxic injury, decreased pain, and decreased muscle spasm.[34] Although evidence is not conclusive, the ideal method of ice application may be to apply real ice (as opposed to gel packs or chemical packs) through a wet towel interface to the injured area utilizing a protocol of 10 minutes on, 10 minutes off, 10 minutes on.[49] Static gastrocnemius and soleus muscle stretching was included in the plan of management to improve ankle range of motion, isometric force production, stretch tolerance, and to promote a focused Achilles stress during the theorized tissue remodelling phase induced by soft tissue mobilization. Improvements in joint range of motion, isometric force production, and stretch tolerance following regular static stretching are supported by recent evidence.[50–52]

Prognosis

The natural history of Achilles tendinopathy is largely unknown.[3] However, it is known that 24% to 45% of patients presenting with a chronic Achilles pain problem will fail to respond to conservative therapies and will choose operative management.[3,53] No studies have reported the prognosis of patients with chronic Achilles tendinopathy treated conservatively with soft tissue mobilization either alone or in combination with a regime of eccentric calf strengthening. Considering that the best prognosis reported in the available literature is a return to full activity within 12 weeks, regardless of the therapeutic intervention, the response to treatment of the presented patient is notable.[24] The patient obtained a near complete resolution of symptoms and a complete return to physical activity after a total of nine therapy sessions over an eight week period. Upon follow-up, seven months later, the patient had maintained the positive therapeutic result.

Summary

Chronic Achilles tendinopathy is a common injury, especially among distance runners.[1–7] The diagnosis is relatively straightforward, and can be made from a thorough history and physical examination. The onset of pain and dysfunction are typically gradual and progressive, associated with overuse or high levels of physical activity.[1–4,11] Early recognition and initiation of conservative therapies typically allow patients to return to their previous levels of activity anywhere between 12 weeks and 1 year, however, recovery can be long and frustrating as re-aggravation is possible.[24]

Although a conservative management approach is recommended, there is a considerable lack of evidence supporting a specific conservative management strategy. General treatment guidelines supported by the literature

include methods to relieve pain, avoid re-aggravation, and treat injured tissues from the perspective of affecting the conditions pathology of angiofibroblastic degeneration and functional impairment.[1–5,11–13] Current literature suggests that the conservative management of chronic Achilles tendinopathy should include an eccentric calf muscle strength training protocol.[12,14–22] Whether or not the inclusion of some other conservative therapy intervention in combination with eccentric training improves the prognosis is not known. As seen in this case report, the implementation of a specific treatment protocol incorporating both an active and passive tissue warm-up, followed respectively by GT® and ART® soft tissue mobilization, eccentric calf muscle strength training, and static calf stretching in combination with cryotherapy appears to have resolved a case of chronic Achilles tendinopathy quite rapidly compared to the prognosis of 12 weeks reported by the literature.[24] It is hypothesized that a specific treatment plan involving in-office and home based focused soft tissue therapy and tissue rehabilitation which relates directly to the current understanding of the pathology of chronic Achilles tendinopathy is the possible reason as to why this patient responded so positively, when previous passive treatment efforts failed. Consequently the conservative treatment protocol used in this case report may be beneficial for patients experiencing symptoms arising from chronic Achilles tendinopathy; however significant additional study is clearly necessary.

References

1 Alfredson H, Lorentzon R. Chronic achilles tendinosis: Recommendations for treatment and prevention. Sports Med. 2000; 29(2):135–46.

2 Paavola M, Kannus P, Jarvinen T, Khan K, Jozsa L, Jarvinen M. Current concepts review: Achilles tendinopathy. J Bone Joint Surg. 2002; 84A(11):2062–2076.

3 Kader D, Saxena A, Movin T, Maffulli N. Achilles tendinopathy: Some aspects of basic science and clinical management. Br J Sports Med. 2002; 36(4):239–49.

4 Alfredson H. The chronic painful achilles and patellar tendon: Research on basic biology and treatment. Scand J Med Sci Sports. 2005; 15(4):252–9.

5 Alfredson H. Chronic mid-portion achilles tendinopathy: An update on research and treatment. Clin Sports Med. 2003; 22:727–741.

6 Huang TF, Perry SM, Soslowsky LJ. The effect of overuse activity on achilles tendon in an animal model: A biomechanical study. Ann Biomed Eng. 2004; 32(3):336–41.

7 Schepsis AA, Jones H, Haas AL. Achilles tendon disorders in athletes. Am J Sports Med. 2002; 30(2):287–305.

8 Magnussen RA, Dunn WR, Thomson AB. Nonoperative treatment of midportion achilles tendinopathy: A systematic review. Clin J Sport Med. 2009; 19(1):54–64.

9 Alfredson H, Ohberg L. Sclerosing injections to areas of neo-vascularisation reduce pain in chronic achilles tendinopathy: A double-blind randomised controlled trial. Knee Surg Sports Traumatol Arthrosc. 2005; 13(4):338–44.

10 Rees JD, Maffulli N, Cook J. Management of tendinopathy. Am J Sports Med. 2009; 37(9):1855–67.

11 Sorosky B, Press J, Plastaras C, Rittenberg J. The practical management of achilles tendinopathy. Clin J Sport Med. 2004; 14(1):40–4.

12 Murray IR, Murray SA, MacKenzie K, Coleman S. How evidence based is the management of two common sports injuries in a sports injury clinic? Br J Sports Med. 2005; 39(12):912–916.

13 McLauchlan GJ, Handoll HH. Interventions for treating acute and chronic achilles tendinitis. Cochrane Database Syst Rev. 2001; 2:1–11.

14 Kongsgaard M, Aagaard P, Kjaer M, Magnusson SP. Structural achilles tendon properties in athletes subjected to different exercise modes and in achilles tendon rupture patients. J Appl Physiol. 2005; 99(5):1965–71.

15 Ng GY, Ng CO, See EK. Comparison of therapeutic ultrasound and exercises for augmenting tendon healing in rats. Ultrasound Med Biol. 2004; 30(11):1539–43.

16 Beneka AG, Malliou PC, Benekas G. Water and land based rehabilitation for achilles tendinopathy in an elite female runner. Br J Sports Med. 2003; 37(6):535–7.

17 Shalabi A, Kristoffersen-Wilberg M, Svensson L, Aspelin P, Movin T. Eccentric training of the gastrocnemius-soleus complex in chronic achilles tendinopathy results in decreased tendon volume and intratendinous signal as evaluated by MRI. Am J Sports Med. 2004; 32(5):1286–96.

18 Roos EM, Engstrom M, Lagerquist A, Soderberg B. Clinical improvement after 6 weeks of eccentric exercise in patients with mid-portion achilles tendinopathy – a randomized trial with 1-year follow-up. Scand J Med Sci Sports. 2004; 14(5):286–95.

19 Ohberg L, Alfredson H. Effects on neovascularisation behind the good results with eccentric training in chronic mid-portion achilles tendinosis? Knee Surg Sports Traumatol Arthrosc. 2004; 12(5):465–70.

20 Fahlstrom M, Jonsson P, Lorentzon R, Alfredson H. Chronic achilles tendon pain treated with eccentric calf-muscle training. Knee Surg Sports Traumatol Arthrosc. 2003; 11(5):327–33.

21 Mafi N, Lorentzon R, Alfredson H. Superior short-term results with eccentric calf muscle training compared to concentric training in a randomized prospective multicenter study on patients with chronic achilles tendinosis. Knee Surg Sports Traumatol Arthrosc. 2001; 9(1):42–7.

22 Silbernagel KG, Thomee R, Thomee P, Karlsson J. Eccentric overload training for patients with chronic achilles tendon pain--a randomised controlled study with reliability testing of the evaluation methods. Scand J Med Sci Sports. 2001; 11(4):197–206.

23 Speed CA. Fortnightly review: Corticosteroid injections in tendon lesions. BMJ. 2001; 323(7309):382–6.

24 Paoloni JA, Appleyard RC, Nelson J, Murrell GA. Topical glyceryl trinitrate treatment of chronic noninsertional achilles tendinopathy. A randomized, double-blind, placebo-controlled trial. J Bone Joint Surg Am. 2004; 86-A(5):916–22.

25 Brosseau L, Casimiro L, Milne S, Robinson V, Shea B, Tugwell P, Wells G. Deep transverse friction massage for treating tendinitis. Cochrane Database Syst Rev. 2002; 4:1–8.

26 Gehlsen GM, Ganion LR, Helfst R. Fibroblast responses to variation in soft tissue mobilization pressure. Med Sci Sports Exerc. 1999; 31(4):531–5.

27 Davidson CJ, Ganion LR, Gehlsen GM, Verhoestra B, Roepke JE, Sevier TL. Rat tendon morphologic and functional changes resulting from soft tissue mobilization. Med Sci Sports Exerc. 1997; 29(3):313–9.

28 Hammer WI, Pfefer MT. Treatment of a case of subacute lumbar compartment syndrome using the graston technique. J Manipulative Physiol Ther. 2005 Mar; 28(3):199–204.

29 Melham TJ, Sevier TL, Malnofski MJ, Wilson JK, Helfst RH. Chronic ankle pain and fibrosis successfully treated with a new non-invasive augmented soft tissue mobilization technique (ASTM): A case report. Med Sci Sports Exerc. 1997:801–804.

30 Yeung CK, Guo X, Ng YF. Pulsed ultrasound treatment accelerates the repair of achilles tendon rupture in rats. J Orthop Res. 2006; 24(2):193–201.

31 Demir H, Menku P, Kirnap M, Calis M, Ikizceli I. Comparison of the effects of laser, ultrasound, and combined laser + ultrasound treatments in experimental tendon healing. Lasers Surg Med. 2004; 35(1):84–9.

32 Rompe JD, Nafe B, Furia JP, Maffulli N. Eccentric loading, shock-wave treatment, or a wait-and-see policy for tendinopathy of the main body of tendo achillis: A randomized controlled trial. Am J Sports Med. 2007; 35(3):374–83.

33 Alsousou J, Thompson M, Hulley P, Noble A, Willett K. The biology of platelet-rich plasma and its application in trauma and orthopaedic surgery: A review of the literature. J Bone Joint Surg Br. 2009; 91(8):987–96.

34 Bélanger, A. Evidence-based guide to therapeutic physical agents. Philadelphia: Lippincott Williams & Wilkins; 2002: 263–98, 299–321.

35 Lehmann, JF. Therapeutic heat and cold. 4th ed. Baltimore: Williams & Wilkins; 1990:417, 590–591, 627–627, 633–637.

36 Kubo K, Kanehisa H, Fukunaga T. Effects of cold and hot water immersion on the mechanical properties of human muscle and tendon in vivo. Clin Biomech (Bristol, Avon). 2005; 20(3):291–300.

37 Kumamoto T, Ito T, Kubota K, Yamamoto I, Abe K, Fujiwara T. The influence of thermotherapy on muscle elasticity: Measurement of pennation angle with the use of ultrasound images. J Phys Ther Sci. 2006; 18:193–199.

38 Leahy, PM. Active Release Techniques®, soft-tissue management system for the lower extremity. 2000.

39 Howitt S, Wong J, Zabukovec S. The conservative treatment of trigger thumb using graston techniques and active release techniques. J Can Chiropr Assoc. 2006; 50(4):249–54.

40 Howitt SD. Lateral epicondylosis: A case study of conservative care utilizing ART and rehabilitation. J Can Chiropr Assoc. 2006; 50(3):182–9.

41 Schiottz-Christensen B, Mooney V, Azad S, Selstad D, Gulick J, Bracker M. The role of active release manual therapy for upper extremity overuse syndromes – a preliminary report. J Occup Rehabil. 1999; 9(3):201–211.

42 Drover JM, Forand DR, Herzog W. Influence of active release technique on quadriceps inhibition and strength: A pilot study. J Manipulative Physiol Ther. 2004; 27(6):408–13.

43 Spina AA. External coxa saltans (snapping hip) treated with active release techniques: A case report. J Can Chiropr Assoc. 2007; 51(1):23–9.

44 Buchberger DJ. Use of active release techniques in the postoperative shoulder: A case report. J Sports Chiropr Rehabil. 1999; 13:60–65.

45 Lorenz D. Eccentric exercise interventions for tendinopathies. J Strength Cond Res. 2010; 32(2): 90–98.

46 Khan KM, Scott A. Mechanotherapy: How physical therapists' prescription of exercise promotes tissue repair. Br J Sports Med. 2009; 43(4):247–52.

47 Kingma JJ, de Knikker R, Wittink HM, Takken T. Eccentric overload training in patients with chronic achilles tendinopathy: A systematic review. Br J Sports Med. 2007; 41(6):e3.

48 Alfredson H, Pietila T, Jonsson P, Lorentzon R. Heavy-load eccentric calf muscle training for the treatment of chronic achilles tendinosis. Am J Sports Med. 1998; 26(3):360–6.

49 MacAuley, D, Best, TM. Evidence-based sports medicine. 2nd ed. Malden, Mass.: Blackwell Pub.; 2007. 615 p.

50 Zakas A. The effect of stretching duration on the lower-extremity flexibility of adolescent soccer players. J Bodywork Mov Ther. 2005;9:220–225.
51 Shrier I. Does stretching improve performance? A systematic and critical review of the literature. Clin J Sport Med. 2004; 14(5):267–73.
52 LaRoche DP, Connolly DA. Effects of stretching on passive muscle tension and response to eccentric exercise. Am J Sports Med. 2006; 34(6):1000–7.
53 Paavola M, Kannus P, Paakkala T, Pasanen M, Jarvinen M. Long-term prognosis of patients with achilles tendinopathy. an observational 8-year follow-up study. Am J Sports Med. 2000; 28(5):634–42.

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Utilization of Vascular Restriction Training in post-surgical knee rehabilitation: a case report and introduction to an under-reported training technique

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Introduction: *The objective of this paper is to introduce a new and reportedly safe training technique, utilizing a vascular restriction stimulus during low intensity rehabilitative exercise and provide a case example within a post-surgical rehabilitation scenario. A brief review of the most commonly reported mechanisms of action behind the purported success of the training stimulus is included.*

Methods: *19-year-old athlete presented for an accelerated post-operative knee rehabilitation program. She received a commonly utilized rehabilitation program that was supplemented with vascular restriction stimulus.*

Results: *The patient maintained muscle cross-sectional area and had improved function at a 12-week follow-up.*

Conclusion: *Low intensity exercise supplemented with vascular restriction may prove to be an efficient and effective means of maintaining post-surgical muscle size and subjective knee function.*
(JCCA 2011; 55(4):280–287)

KEY WORDS: knee, rehabilitation, post surgical, vascular restriction

Introduction: *ce document a pour objet de présenter une nouvelle technique d'entraînement supposément sécuritaire, qui a recours à un stimulus de restriction vasculaire durant les exercices de réhabilitation à faible intensité, et un exemple de cas faisant partie d'un scénario de réhabilitation post-opératoire. Le document comprend une brève évaluation des mécanismes d'action communément jugés comme étant à l'origine du succès du stimulus d'entraînement.*

Méthodes : *une athlète de 19 ans s'est présentée afin de suivre un programme de réhabilitation post-opératoire accéléré pour son genou. On lui a prescrit un programme de réhabilitation communément utilisé, avec comme ajout un stimulus de restriction vasculaire.*

Résultats : *la patiente a maintenu la section transversale du muscle et la fonction du genou s'était améliorée après 12 semaines.*

Conclusion : *l'exercice à faible intensité, auquel s'ajoute une restriction vasculaire, pourrait s'avérer une méthode efficace et efficiente pour maintenir la taille du muscle suite à une opération, ainsi que la fonction du genou.*
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MOTS CLÉS : genou, réhabilitation, post-opératoire, restriction vasculaire

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Introduction

Thigh muscle weakness and dysfunction secondary to an acute knee injury or surgical reconstruction procedure has been well established and has been linked to poor functional outcomes.[1–3]

From a muscular standpoint, post-injury/surgical care initially focuses on proper quadriceps activation and prevention of muscle atrophy. Common training paradigm states that in order to promote the most optimal muscle hypertrophy and strength gains, one has to work at a moderate to high intensity level – in the range of at least 60–70% of 1 repetition maximum (1RM).[4] Early application of this principle in the rehabilitation setting is often hindered due to the compromised nature of the affected limb. However, a growing amount of evidence gathered over the past decade suggests that low intensity resistance exercise (20–50% of 1RM) under the stimulus of blood flow restriction can result in greater strength and muscle cross sectional area (CSA) gains when compared to exercise at the same intensity with normal flow,[5–8] and comparable to the gains seen with traditional high intensity resistance exercise.[5]

Training with vascular restriction (VRT), aka "KAATSU Training,"[9] is accomplished using an instrument such as a blood pressure (BP) cuff placed around the base of an extremity and pumped up to a desired level to produce restricted vascular flow while exercise is performed. Restrictive pressures as low as 100 mmHg have been shown to produce significant strength gains when combined with low intensity resistance exercise.[5,7]

This educational case report will briefly highlight a case of post-arthroscopy knee rehabilitation with the implementation of VRT. More importantly, the objective of this paper is to introduce the reader to an under-reported training technique and discuss the commonly proposed mechanism associated with it by reviewing the relevant literature. It is not the purpose of this article to educate the reader regarding all domains relevant to training with vascular restriction or to provide a structured protocol for its use within a rehabilitative setting.

Case

A 19-year-old female provincial level soccer player presented 3 days following a hamstring tendon autograft ACL arthroscopic reconstructive surgery. Twenty-three weeks prior to surgery, the athlete sustained an on-field injury, which yielded the unhappy triad of a right ACL rupture, a grade 3 medial collateral ligament sprain and a large bucket-handle tear of the medial meniscus. Prior to surgery the athlete completed a Knee Injury and Osteoarthritis Outcome Score (KOOS), a Lower Extremity Functional Scale (LEFS), and thigh muscle girth measurements were taken using a standard tape measure at 3.9 in (10 cm) proximal to the superior portion of the patella and at the thickest portion of the leg. Muscle girth measurements were compared to the unaffected side and were deemed equal on both sides.

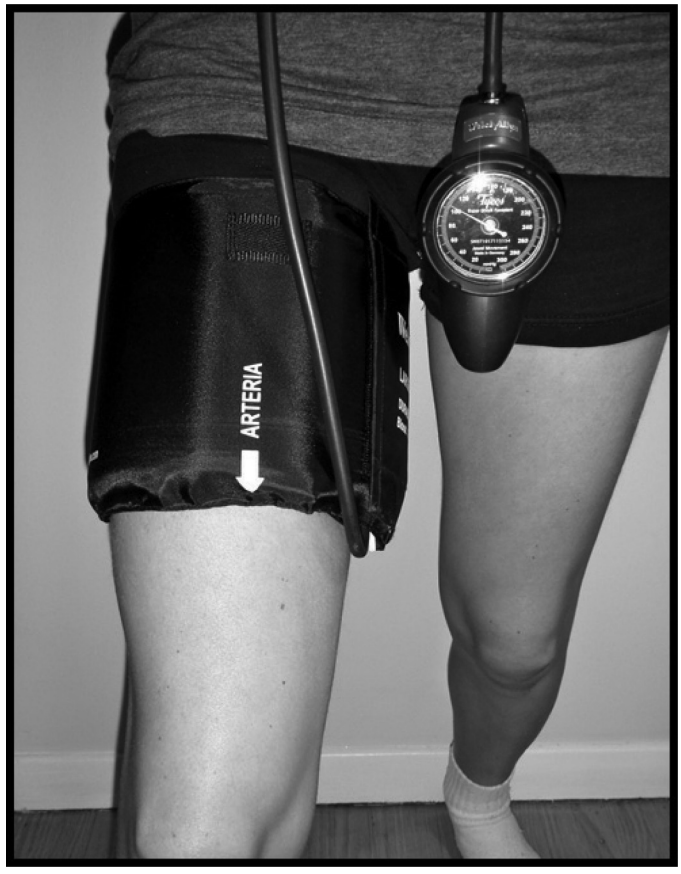

Figure 1 *Demonstration of vascular restriction stimulus application. Blood pressure cuff was applied at the base of the thigh in the affected lower limb.*

Upon post-surgical presentation, the patient began an accelerated post-surgical ACL rehabilitation program as proposed by Wilk et al. (1999).[10] The protocol included joint and soft tissue mobilizations, cryotherapy, electro-

Table 1 *Application of the vascular restriction stimulus*

Period	Vascular Restrictive Protocol	Exercise
Days 1–3	• Application of BP cuff to the proximal thigh • Inflation of BP cuff to 100 mm Hg for 5 min • 5 repetitions (3 min rest) • 2x/day	Rest
Days 4–7	• Application of BP cuff to the proximal thigh • Inflation of BP cuff to 100 mmHg • 20 reps/fatigue, 2 sets (30 sec rest b/w sets) • Deflate cuff @ completion of 1st exercise • Rest 5 mins • Repeat with 2nd exercise	1) Knee extensions from 90–40° (hip @ 90°) 2) Heel Slides
Week 2	• Same as above	1) Knee extensions from 90–40° (with extra-light Theraband™) 2) Heel slides (with extra-light Theraband™)
Week 3	• Same as above	1) Front step-ups (height = 1′ 6″) 2) Lateral step-ups (height = 1′ 6″)
Week 4–12	• Same as above	1) Lateral lunges 2) Vertical squat

***Immediately deflate BP cuff upon any of the following:** Pain, burning, tingling, loss of sensation, bruising or anything else that feels different then exercise-induced fatigue.

modalities, electrical muscle stimulation, proprioceptive exercises, as well as stretches and active range of motion exercises. VRT was also included in the regimen. This was implemented via the use of a standard 6-inch (15.2 cm) wide blood pressure cuff. The cuff was inflated to maintain 100 mmHg at the base of the affected lower limb. Although the amount of vascular restriction was maintained at 100 mmHg throughout the rehabilitation program, the exercises used under the stimulus changed periodically with phases, as outlined in Table 1.

During the first phase, days one to three (days four to seven post-surgery), the vascular restriction stimulus was applied without coupled exercises and modeled after a previous study attempting to use vascular restriction to prevent post-surgical quadriceps atrophy.[11] The subsequent phases incorporated a coupling of vascular restriction with low intensity resistance exercise that were already part of the original program as proposed by Wilk et al. (1999). Readers are encouraged to refer to the above reference for the full and detailed rehabilitation protocol. Rehabilitation exercises were performed four to five times per week.

The vascular restrictive stimulus was well tolerated. Rapid onset of fatigue and some associated transient discoloration of the restricted lower limb were the only reported symptoms. The patient reported no pain, paresthesia or other symptoms.

Re-assessment three months after the surgery yielded LEFS scores that were similar to the pre-operative values. Some improvements in KOOS scores were made when

Table 2 *Outcome measures and results*

Outcome Measure	Prior to Surgery		12 Weeks Post-Surgery	
LEFS	66/80		65/80	
KOOS • **Pain:** • **Symptoms:** • **ADLs:** • **Sports & recreation function:** • **Knee-related quality of life:**	92 82 97 68 43		100 85 99 78 56	
Muscle Girth (tape measure) • **Thigh** • **Leg**	Unaffected: 14.2in (36cm) 12.6in (32cm)	Affected: 14.2 in (36cm) 12.6in (32cm)	Unaffected 14.2in (36cm) 12.6in (32cm)	**Affected:** **14.4in (36.5cm)** **12.8in (32.5cm)**
	***Girth maintained when measured every 3 weeks**			

comparing the pain, symptoms, activities of daily living, sport and recreational function, and knee-related quality of life domains of the pre-operative and post-operative assessment. Muscle girth was measured periodically (every 3 weeks) over the 3-month post-surgical period. When compared to the unaffected lower limb, the girth measurements of the thigh and leg remained identical and at no point did they decrease suggesting atrophy. These remained a stable 14.2 in (36 cm) and 12.6 in (32 cm) measured at the thigh and calf respectively. At 3 months following surgery, the subject exhibited a 0.2 in (0.5 cm) greater thigh and leg circumference on the affected limb. Please refer to Table 2 for result details.

The patient and parent gave informed consent and agreed to allow the use of their medical information without disclosing personal identifiers for this case report.

Discussion

The study design is the inherent limitation of this paper as well as the lack of strength measures and more valid measurements of girth. However, it was not the intention of the authors to provide the readers an in-depth look at training with vascular restriction or to outline a specific utilization protocol. Such information does not exist. It was our intention to simply educate health-care professionals on an innovative training stimulus and to highlight how this may be utilized within a clinical setting to help prevent post-surgical atrophy and secondarily to maintain and improve function.

Advanced measurements of muscle cross-sectional area and strength were unavailable in this case. Alternatively, common clinical measurements (i.e. tape measure and a subjective functional scale) were utilized. Although the validity of clinical muscle girth measurements is questionable, our results in this case suggest that post-surgical atrophy was prevented. There was evidence of some improvement in subjective functional impairment as demonstrated in all of the KOOS subscales. It should be noted that the previously determined minimal detectable change was not reached for any of the domains (pain: 13.85, symptoms: 9.97, activities of daily living: 11.92, sports and recreational function: 22.96, knee-related quality of life: 15.45).[12] This could easily be attributed to the high baseline scores prior to surgery. Interestingly,

when the scores at the 3 month follow up in this case are compared to a previous study including a comparable subject pool, the scores for each domain are similar and in some cases supersede the average KOOS scores of subjects assessed at 6 months post-reconstructive surgery.[13] It is possible that similar results could have been achieved without the additional implementation of VRT within the rehabilitation regimen. Unfortunately the design of this study does not allow for any conclusions to be made regarding the efficacy of VRT in this scenario.

The lack of more significant subjective functional improvements after 3 months of the proposed rehabilitation protocol could be attributed to the fact that the patient suffered a significant injury involving more then the one repaired anatomical structure. Secondly, with the follow-up being only 12 weeks after the surgery, it is clear that the rehabilitation process is not complete. In this case however, the clinical decision to terminate VRT at 12 weeks was made based on the finding that the girth measurements in the affected extremity were already surpassing those of the control limb.

Evidence supporting favourable muscular adaptations secondary to vascular restriction exercise is well established.[5–7,14,15] Application of vascular restriction within the rehabilitative setting has also showed promise. The first report of implementation of vascular restriction post-operatively, was by Takarada et al. (2000).[11] Here the authors utilized repeated bouts of transient vascular restriction with no resistance exercise in post-surgical ACL reconstructed limbs. This type of protocol showed promise as the associated disuse atrophy was markedly diminished (MRI CSA) in the vascular restriction group compared to a control at a 14-day follow-up. Kubota et al. (2008)[16] later reproduced the above protocol in a design that immobilized healthy volunteer lower limbs for two weeks. In this study, the vascular restriction group was compared to a group of subjects performing isometric exercises (knee extensor/flexor and plantar flexors) 2 times per day for two weeks, and a control group (no intervention). The results showed that only the vascular restriction group demonstrated a relative maintenance of strength, as well as thigh and leg circumference.

The first (and only) randomized control trial utilizing vascular restriction in conjunction with post-surgical rehabilitation was by Ohta and collegues (2003).[17] To test the efficacy of low-load resistance training with moderate vascular restriction following ACL reconstruction, the authors randomized a group of post-surgical patients (n = 22) to a 16-week VRT rehabilitation regimen. Another group of patients acted as controls, performing the exact same regimen without the vascular restriction stimulus. Although several study limitations were present, many significant differences were found in favour of VRT. Specifically, the VRT group demonstrated significantly superior knee flexor/extensor muscle strength and knee extensor muscle CSA over the control, as measured by isokinetic dynamometry and axial magnetic resonance imaging slices respectively. The vascular restriction stimulus was maintained for up to 15 minutes at 180 mmHg, as the subjects were performing their assigned exercises. Many subjects reported a deep ache associated with the VRT as they reached the end of the 15-minute application. No serious side effects to VRT were reported in this study. In the present case, the estimated average that the blood pressure cuff remained inflated was roughly 5 minutes per set.

The gains associated with VRT are substantial, yet utilization is low. This is most likely due to the lack of knowledge about the safety surrounding this training technique, as well as knowledge outlining concrete mechanisms of action. VRT has been practiced for decades in Japan. Although side effects have been reported, a study looking at utilization and safety amongst its users has suggested that even though training with vascular restriction is widely used amongst all age groups in Japan, the incidence of serious complications seems rare.[18] In this retrospective study, the most common side effects included transient bruising and paresthesia that occurred in roughly 13 and 1.3 percent of the cases respectively. Other more serious side effects were also reported but were extremely rare. It is worthwhile to mention that out of the ~12.6 thousand individuals who were reportedly receiving VRT, roughly 29% of them were seniors (60+).

The authors of the present study were unable to find any scientific literature studying VRT that reported any serious side effects. The decision to use VRT in this case was strictly based on the similarity of the present case with previous reports of utilization in a rehabilitation setting and patient consent. The patient reported no significant side effects related to the treatment and was at no point hesitant about complying with the planned protocol even though she was advised she could withdraw at any time.

The following two subsections of this discussion relate to the two most commonly reported mechanisms of action pertaining to the effects seen with VRT.

Acute Hormonal Responses

Acute responses of anabolic hormones such as testosterone, growth hormone(GH) and insulin-like growth factor 1 (IGF) have long been guiding exercise practices. In a recent review, Kraemer & Ratamess concluded that the acute responses of the above hormones are critical for strength gains and muscular growth.[19] These conclusions are based on the observable increases of these hormones in individuals post-exercise. It is suggested that exercise utilizing high intensity loads, low repetitions, short rest intervals and large muscle groups will result in higher acute hormonal responses.[4] Training dogma has long taken this advice and incorporated it. An example of this is the common recommendation of performing high intensity training and mixing large and small muscle group exercises with the intention that small muscles will benefit from the large increases of systemic anabolic hormones stimulated by intense exercise of large muscle groups.[4] Although low intensity exercise does not fit within the specifications of the above guidelines, when combined with the stimulus of vascular restrictions, significant acute hormonal changes are noted.

Several studies have shown significant increases up to ~290-fold in GH following low intensity training with vascular restriction.[20–22] Acute increases in human GH have been considered as one of the primary mechanisms responsible for the observed muscular adaptations associated with VRT. This hypothesis however is based solely on correlation and does not speak well to causation. Recent investigations into the effects of the ostensibly anabolic hormones concludes that physiological increases of such hormones associated with high intensity exercise of large muscle groups does not equate to any differences in strength, hypertrophy and myofibrillar synthesis in general.[23,24] Also, recent investigations into the effects of GH specifically are suggesting that the hormone plays significant role in tendon collagen synthesis as opposed to muscle protein synthesis.[25] The importance of GH should not be understated, however, with respect to the synthesis of contractile proteins in muscle tissue, evidence suggest other factors are responsible for this. Therefore the theory that the demonstrated gains related to VRT are secondary to associated increases in GH is not well supported by recent empirical evidence. This is not however the only proposed mechanism.

Acute Neuromuscular Adaptations

A more likely proposed explanation for the favourable gains in strength and CSA observed with VRT at low loads is rooted in the acute neuromuscular adaptations that occur while it is being performed.

The "size" principle of motor unit activation (MUA) was first proposed by Henneman in 1965.[26] This principle explains motor unit and therefore muscle fibre recruitment patterns. It states that low-load resistance exercise recruits small motor units and their associated slow twitch, low threshold, oxidative muscle fibres. As the exercise intensity increases (load), there is an increase of motor unit recruitment and larger, higher threshold motor units are recruited. These larger motor units are associated with larger, fatigable (glycolytic) Type II muscle fibres. Since type II fibres respond to exercise with more substantial hypertrophy, the goal of modern resistance exercise progressions is to recruit these fibres in order to maximize gains.[27] Due to the size principle, recruitment of type II motor units primarily occurs when lifting substantial loads. This could also be accomplished by maintaining submaximal fatiguing exercise which eventually exhausts smaller oxidative motor-units leading to the recruitment of larger (non-oxidative) ones in order to maintain work.[28]

Training with a vascular restriction stimulus has demonstrated a unique ability to accelerate muscular fatigue at light loads. The transient hypoxia associated with blood flow restriction at light loads demonstrates hastening fatigue through increases in blood lactate concentrations, MUA, and deceases in motor unit firing rate; all of which suggest fatigue.[29]

Moritani et al. (1992),[29] were the first to show all three changes occurring during very low intensity exercise (20% maximum voluntary contraction) with blood flow restriction (200 mmHg). These changes were not seen in the control group (no vascular restriction). Studies utilizing electromyography (EMG) to study MUA via root mean square and integrated EMG (iEMG) analysis have demonstrated on a number of occasions that vascular restriction leads to increases in recruitment.[5,7,29,30] For example, in a study comparing iEMG of the biceps brachii during vascular restriction (40% 1 RM) and a heavy resistance exercise group (80% 1 RM), Takarada et al. (2000)[5] showed almost equal iEMG output values between the groups. These findings indicate a similar amount of MUA and therefore similar effort at drastic-

ally different exercise intensities. This highlights the most likely mechanism for the purported gains in strength and CSA seen with VRT.

Conclusions

This case report outlines a supplementation of the typical accelerated post-surgical knee rehabilitation program with low intensity exercises utilizing vascular restriction. The results suggest maintenance of muscle girth and subjective function 3 months after the surgery for this particular patient. It cannot be stated unequivocally that these results were not due to the other, more traditional elements of the post-surgical program. Low intensity exercise supplemented with vascular restriction may prove to be an efficient and effective means of maintaining post-surgical muscle size and subjective knee function. Future studies should aim to further elucidate the efficacy behind such an approach to rehabilitation.

References

1 Palmieri-Smith RM, Kreinbrink J, Ashton-Miller J, Wojtys EM. Quadriceps inhibition induced by an experimental knee joint effusion affects knee joint mechanics during a single-legged drop landing. Am J Sports Med. 2007; 35(8):1269–75.

2 Daniel DM, Stone ML, Dobson BE. Fate of the ACL-injured patient: a prospective outcome study. Am J Sports Med. 1994; 22(5):632–644.

3 Ageberg E, Thomeé R, Neeter C, Silbernagel KG, Roos EM. Muscle strength and functional performance in patients with anterior cruciate ligament injury treated with training and surgical reconstruction or training only: a two to five-year followup. Arthritis Rheum. 2008; 59(12): 1773–9.

4 Ratamess NA, Alvar BA, Evetoch TK, Housh TJ, Kibler B, Kraemer WJ, Triplett T. American college of sports medicine position stand: Progression models in resistance training for healthy adults. Med Sci Sports Exerc. 2009; 41(3):687–708.

5 Takarada Y, Takazawa H, Sato Y, Takebayashi S, Tanaka Y, Ishii N. Effects of resistance exercise combined with moderate vascular occlusion on muscular function in humans. J Appl Physiol. 2000; 88:2097–2106.

6 Takarada Y, Sato Y, Ishii N. Effects of resistance exercise combined with vascular occlusion on muscle function in athletes. Eur J Appl Physio. 2002; 86(4):308–314.

7 Moore DR, Burgomaster KA, Schofield LM, Gibala, MJ, Sale DG, Phillips SM. Neuromuscular adaptations in human muscle following low intensity resistance training with vascular occlusion. Eur J Appl Physio. 2004; 92(4–5): 399–406.

8 Sumide T, Sakuraba K, Sawaki K, Ohmura H, Tamura Y. Effect of resistance exercise training combined with relatively low vascular occlusion. J Science Med Sport. 2009; 12(1):107–12.

9 Sato Y. The history and future of KAATSU training. International J KAATSU Training Research. 2005; 1(1): 1–5.

10 Wilk KE, Arrigo C, Andrews JR, Clancy Jr WG. Rehabilitation after anterior cruciate ligament reconstruction in the female athlete. J Athletic Training. 1999; 34(2):77.

11 Takarada Y, Takazawa H, Ishii N. Applications of vascular occlusion diminish disuse atrophy of knee extensor muscles. Med Sci Sports Exerc. 2000; 32(12):2035–9.

12 Logerstedt DS, Snyder-Mackler L, Ritter RC, Axe MJ, Godges JJ. Knee stability and movement coordination impairments: knee ligament sprain. J Orthop Sports Phys Ther. 2010; 40(4):A1–A37.

13 Roos EM, Roos HP, Lohmander LS, Ekdahl C, Beynnon BD. Knee Injury and Osteoarthritis Outcome Score (KOOS) – development of a self-administered outcome measure. J Orthop Sports Phys Ther. 1998; 28(2):88–96.

14 Shinohara M, Kouzaki M, Yoshihisa T, Fukunaga T. Efficacy of tourniquet ischemia for strength training with low resistance. Eur J Appl Physio. 1998;77(1–2):189–91.

15 Teramoto M, Golding L. Low-intensity exercise, vascular occlusion, and muscular adaptations. Research in Sports Medicine: An International Journal. 2006; 14(4):259–271.

16 Kubota A, Sakuraba K, Sawaki K, Sumide T, Tamura Y. Prevention of disuse muscular weakness by restriction of blood flow. Med Sci Sports Exerc. 2008; 40(3):529–34.

17 Ohta H, Kurosawa H, Ikeda H, Iwase Y, Satou N, Nakamura S. Low-load resistance muscular training with moderate restriction of blood flow after anterior cruciate ligament reconstruction. Acta Orthop Scand. 2003; 74(1): 62–8.

18 Nakajima T, Kurano M, Iida H, Takano H, Oonuma H, Morita T, Meguro K, Sato Y, Nagata T. Use and safety of KAATSU training: results of a national survey. International J KAATSU Training Research. 2006; 2:5–13.

19 Kraemer WJ, Ratamess N. Hormonal responses and adaptations to resistance exercise and training. Sports Med. 2005; 35(4):339–61.

20 Takarada Y, Nakamura Y, Aruga S, Onda T, Miyazaki S, Ishii N. Rapid increase in plasma growth hormone after low-intensity resistance exercise with vascular occlusion. J Appl Physiol. 2000; 88:61–65.

21 Pierce JR, Clark BC, Ploutz-Snyder LL, Kanaley J. Growth hormone and muscle function responses to skeletal muscle ischemia. J Appl Physiol. 2006; 101(6):1588–95.

22 Reeves GV, Kraemer RR, Hollander DB, Clavier J, Thomas C, Francois M, Castracane VD. Comparison of hormone responses following light resistance exercise

with partial vascular occlusion and moderately difficult resistance exercise without occlusion. J Appl Physiol. 2006; 101(6):1616–22.
23 West DWD, Kujbida GW, Moore DR, Atherton P, Burd N, Padzi JP, De Lisio M, Tang JE, Parise G, Rennie MJ, Baker SK, Phillips SM. Resistance exercise-induced increases in putative anabolic hormones do not enhance muscle protein synthesis or intracellular signalling in young men. J Physiol. 2009; 587:5239–47.
24 West DWD, Burd N a, Tang JE, Moore DR, Staples AW, Holwerda AM, Baker SK, Phillips SM. Elevations in ostensibly anabolic hormones with resistance exercise enhance neither training-induced muscle hypertrophy nor strength of the elbow flexors. J Appl Physiol. 2010; 108(1): 60–7.
25 Doessing S, Heinemeier KM, Holm L, Mackey AL, Schjerling P, Rennie M, Smith K, Reitelseder S, Kappelgaard AM, Rasmussen MH, Flyvbjerg A, Kjaer M. Growth hormone stimulates the collagen synthesis in human tendon and skeletal muscle without affecting myofibrillar protein synthesis. J Physio. 2010; 588:341–51.
26 Henneman E, Somjen G, Carpenter DO. Functional significance of cell size in spinal motoneurons. J Neurophysiol. 1965; 28(3):560.
27 McCall G, Brynes W, Dickinson P, Patany P, Fleck S. Muscle fiber hypertrophy, hyperplasia, and capillary density in college men after resistance training. J Appl Physiol. 1996; 2004–2012.
28 Sale DG. Influence of exercise and training on motor unit activation. Exerc Sport Sci Rev. 1987; 15(1):S135–45.
29 Moritani T, Sherman WM, Shibata M, Matsumoto T, Shinohara M. Oxygen availability and motor unit activity in humans. Eur J Appl Physio. 1992; 64(6):552–6.
30 Yasuda T, Brechue W, Fujita T, Sato Y. Muscle activation during low-intensity muscle contractions with varying levels of external limb compression. J Sports Science Med. 2008; 7:467–474.

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Heel pain due to psoriatic arthritis in a 50 year old recreational male athlete: case report

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Heel pain is a common presentation in a sports injury practice, with a list of common differentials including achilles tendinopathy and retrocalcaneal bursitis. However, seronegative arthritis can also cause enthesopathies that produce heel pain and should be considered in a differential diagnosis list. In this case, a 50 year old recreationally active male presented with non-traumatic insidious heel pain and without history of any skin conditions or any other symptoms of seronegative spondyloarthritis. Clinical suspicion led to laboratory testing and radiographs / bone scan which yielded the diagnosis of psoriatic arthritis.
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KEY WORDS: psoriatic arthritis, seronegative spondyloarthropathy, athlete, enthesopathy, heel pain, achilles

La douleur au talon est courante dans la pratique du sport, et comporte des variantes telles que la tendinopathie achilléenne et la bursite rétrocalcanéenne. Cependant, l'arthrite séronégative peut également causer des enthésopathies produisant une douleur au talon, et doit apparaître sur la liste des diagnostics différentiels. Dans ce cas, un homme de 50 ans actif de façon récréative ressentait une douleur insidieuse non traumatique au talon, et ses antécédents ne démontraient aucune maladie de la peau ou autres symptômes de spondylarthrite séronégative. Les soupçons cliniques ont mené à des tests en laboratoire et des radiographies / une scintigraphie osseuse qui ont permis de diagnostiquer le rhumatisme psoriasique.
(JCCA 2011; 55(4):288–293)

MOTS CLÉS : rhumatisme psoriasique, spondylarthrite séronégative, athlète, enthésopathie, douleur au talon, Achille

Introduction

Heel pain is a common presentation in a sports injury practice. Foot and ankle injuries make up approximately 25% of all sports related injuries, while 16% of all sports related injuries involve the foot.[1] The vast majority of these injuries can be attributed to a mechanical diagnosis but it is important to maintain an index of suspicion for systemic inflammatory diseases such as psoriasis.

In the United States, psoriasis affects approximately 2% of the population.[2] Psoriasis is a chronic autoimmune skin disease and 10% to 40% of individuals with psoriasis develop chronic inflammatory arthritis.[3] The overall age and sex adjusted population incidence of psoriatic arthritis in the United States was calculated at 6.59/100,000.[4] Psoriatic arthritis is a seronegative spondyloarthropathy which is a group of inflammatory arthritides characterized by enthesitis, arthritis of the peripheral joints and/or the axial skeleton, while being negative for rheumatoid factor.[5] Psoriatic arthritis usually develops at or after the diagnosis of psoriasis, but in 15% to 20% of cases, the arthritis precedes the onset of skin lesions by as much as 2 years.[5] Heel pain is a common sign of psoriatic arthritis

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with ten percent of psoriatic arthritis sufferers reporting heel pain as a significant symptom.[6] However, in an athletic setting, the clinician may intuitively lean towards a mechanical diagnosis first in an active 50-year-old male with no previous diagnosis of psoriasis.

Case Report

A 50-year-old male presented to a sports chiropractic clinic with insidious bilateral foot and heel pain that began after a one-week ski trip eight months prior. He self described his injury to be achilles tendonitis and sought soft tissue care. He reported that his heels did not bother him during the trip, despite walking in ski boots. The pain was focused at the posterior calcaneus and distal attachment of the achilles tendon, with some radiation into the medial arch of the foot and first digit. At rest, the pain was rated a 1 or 2 out of 10 on the numeric pain scale, but with palpation it intensified to 5 or 6 out of 10 (with zero being no pain at all and 10 being the worse imaginable pain). Both feet were affected, but the right foot was generally reported to feel worse. The patient reported that NSAIDs, avoiding aggravating physical activity or footwear have helped with the pain. There was no history of trauma to the feet. The patient had to discontinue playing hockey, due to the pronounced bump on the posterior aspects of both of his heels which contributed to a progressive intense discomfort while skating. The heel pain was also aggravated by golf, which was attributed to his golf shoes and walking the course inclines. He reported no problems during his usual weight training, wind surfing or cycling. He scored a 70 on the 20 question Lower Extremity Functional Score (LEFS). The LEFS max score is 80 and this outcome measure is utilized to monitor function, progress and effectiveness of an intervention.[7] The patient denied a personal or family history of arthritides, low back pain, eye problems or gastrointestinal complaints. He was a non-smoker, an occasional (social) drinker and sporadically took a multi-vitamin. There were no recent changes in his diet and the patient did not report indulging in rich cheeses and wine on further questioning. He did not take any medication and had never been diagnosed with a skin disease nor sought the opinion from a medical professional for any dermatological reason. Observation revealed a significant enlargement over the posterior calcaneus of the right foot (see figure 1), which was very tender to diagnostic palpation, an 8/10 at presentation. His

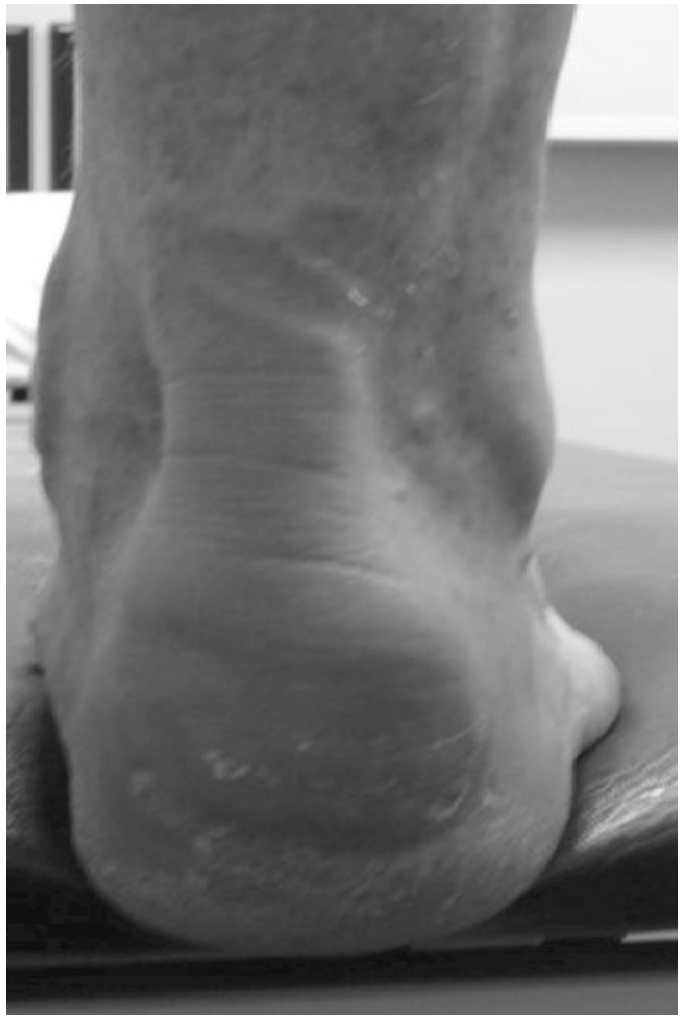

Figure 1 *Patient's heel*

right first hallux appeared red and the medial foot was warm to the touch. The skin over the heels and the soles of the feet were scaly and the nail of the right first hallux was thickened. The right foot appeared generally swollen and the circumference of the right midfoot was 1.5 cm larger when compared to the left. During the physical exam, active, passive and resisted ranges of motion of the ankle and foot were full and pain free. The only physical test that aggravated the heel pain was a one legged squat in which the patient described an increase in pressure in the achilles and ankle joint. The patient's leg was noted as unsteady and he demonstrated an obvious joint coupling dysfunction with a valgus knee, internal rotation of the

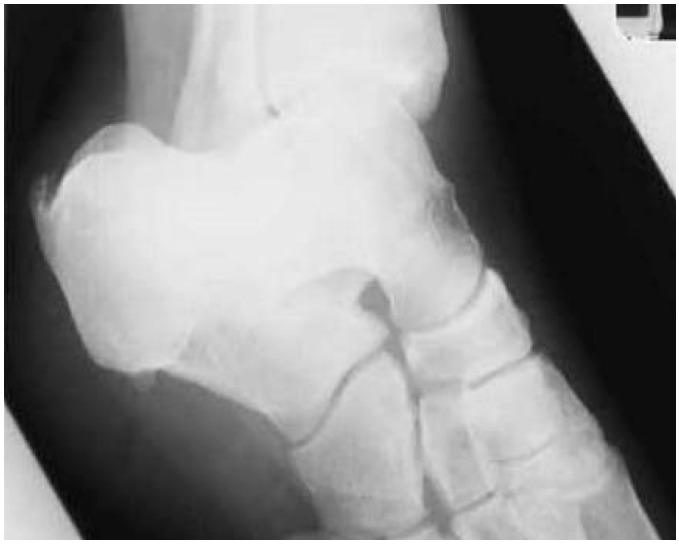

Figure 2 *Radiograph of the patient's heel*

tibia and over pronation of the foot during the one legged squat.

The patient had radiographs and a diagnostic ultrasound of the right foot 5 months previous that were arranged by his family medical doctor (see figure 2). The radiographs revealed a calcaneal plantar spur and a calcaneal Achilles spur and the patient was told he had arthritis. The ultrasound showed calcific deposits at the insertion of the Achilles tendon. In light of the history, physical and imaging results, a working diagnosis of an acute retrocalcaneal bursitis was given to explain the irritated heel, however, the patient was referred to a sport medicine specialist to assist in the possible differential diagnosis of Reiter's syndrome or Psoriatic arthritis. Additional testing included a bone scan and blood tests. The bone scan revealed features that are consistent with an enthesopathy of the Achilles' tendons, as can be typically seen with seronegative arthropathies. Other non-specific asymmetric arthritic features compatible with an underlying seronegative arthropathy were also seen by the radiology specialist, most prominently involving the right 1st metatarsophalangeal joint and, to a lesser degree, the right ankle and adjacent tarsus, patellofemoral joints and right hip.

Blood work in this case revealed that ESR, rheumatoid factor and C-reactive protein were all within normal limits. The patient was also negative for HLA-B27 and antinuclear antibody. There was no evidence of a past or present syphilis infection. Due to the presence of skin lesions on the heel and sole of the foot, as well as thickened toe nails, a diagnosis of psoriatic arthritis was made and the patient was referred to a rheumatologist for treatment which resulted in a trial of methotrexate and nonsteroidal anti-inflammatory drugs (Diclofenac). At a one year follow up / check up, prior to resuming his recreational hockey the patient reported to be consistent with a stretching routine and post exercise icing which were allowing him to be active without pain, including skating. His LEFS improved by 10 points to a score of 80 (a change of 9 is the minimal significant change).[7] He reported to rarely notice any pain, if at all. However, he did report to purchasing larger shoes and skates that allowed for an accommodation of his calcaneal bump.

Discussion

The differential diagnoses for heel pain in a healthy active 50-year-old male typically include achilles tendinopathy and retro calcaneal bursitis. Retrocalcaneal bursitis is an inflammation of the retrocalcaneal bursa and can also cause pain at the posterior heel.[8] Achilles tendonopathy is a degeneration and failed healing of the tendon that can cause pain at the insertion on the calcaneus, as well as within the mid substance of the tendon. While these diagnoses are typical, practitioners might consider the other systemic differential diagnoses that are possible. To help in the diagnosis of a systemic inflammatory problem, the practitioner may also identify constitutional symptoms, morning stiffness, elevated acute-phase reactants and worsening of symptoms despite activity modification.[9] Additionally, if the mechanism of injury is absent or not proportional to the injury, this may also suggest an underlying disease.[9] In this case, even though the chief complaint was reproduced with palpation of the enthesis of the Achilles tendon and aggravated by a squat, the presence of skin and nail lesions and a mild dactylitis of the hallux led the clinicians to suspect a seronegative spondyloarthropathy.

Individuals with psoriatic arthritis test negative for rheumatoid factor but they may have an association with HLA-B27, especially those with involvement of the axial skeleton.[10,11] This perhaps atypical case had a negative test for HLA-B27 and lacked any axial complaints yet the bone scan demonstrated multiple asymmetrical joint involvements.

There are many different diagnostic criteria available for psoriatic arthritis. The CASPAR criteria have specificity of 98.7% and sensitivity of 91.4%.[12] To meet the CASPAR (Classification for Psoriatic Arthritis) criteria, a patient must have inflammatory articular disease (joint, spine or entheseal) with ≥3 points from the following 5 categories:

1. Evidence of current psoriasis, a personal history of psoriasis or a family history of psoriasis.
2. Typical psoriatic nail dystrophy including onycholysis, pitting and hyperkeratosis observed on physical examination
3. A negative test result for the presence of rheumatoid factor
4. Either current dactylitis, defined as swelling of an entire digit or a history of dactylitis recorded by a rheumatologist
5. Radiographic evidence of juxtaarticular new bone formation, appearing as ill-defined ossification near joint margins (but excluding osteophyte formation) on plain radiographs of the hand or foot.

Current psoriasis is assigned a score of 2, and all other features are assigned a score of 1.

Uncomplicated psoriasis arises in the second or third decade, with the arthritis typically settling in two decades later.[3] In 15 to 20% of cases, the arthritis precedes the onset of skin lesions by as much as 2 years.[5] Since this patient had never been diagnosed with psoriasis, it is difficult to know if the skin condition appeared before the arthritis. The patient's age in this case coincides with the range for this disease, as the mean age of psoriatic arthritis onset ranges from 30 to 55 years.[5] Psoriatic arthritis affects men and women equally.[13] All forms of psoriasis are associated with arthritis, but classic psoriasis vulgaris is seen most frequently. Typical psoriatic lesions are erythematous plaques that produce scaling with scratching. Many patients with psoriatic arthritis have mild to moderate skin disease, but there has been no consistent correlation in the literature between the degree of psoriasis and the extent of joint involvement. The psoriasis may be subtle and careful examination of the entire skin surface may be required to visualize the skin lesions. The scalp, ears, periumbilical and perianal region should be examined carefully.[5]

The etiology of psoriatic arthritis is unknown. Genetic, immunologic and environmental factors all play a role in psoriatic arthritis.[3] Individuals with a first degree relative with psoriatic arthritis are 50-fold more likely to develop the disease.[13] A father with psoriasis is twice as likely to pass on the disease than a mother with psoriasis.[13] Of note to the sports injury practitioner, psoriatic lesions have been known to arise at areas of trauma, which is also known as the Koebner phenomenon.[13] It has also been suggested that environmental factors play a role and that they are additive to the genetic background.[3] Immunology is also implicated which is shown in the aggressive nature of psoriatic arthritis in HIV patients.[3]

Most patients present with psoriatic arthritis that is considered oligoarthritis or monoarthritis.[5] A typical patient will present with stiff, swollen and tender DIP joints, in an asymmetric fashion. The joints most often affected are the DIP joints, other small joints of the hands and feet, the sacroiliac joint and the spine. Knees, hips and shoulders are occasionally involved.[10,14,14] In some patients dactylitis and enthesitis may be the only clinically apparent manifestations of psoriatic arthritis for months or years.[16] The MCP joints and wrists are usually spared, which helps to differentiate this condition from rheumatoid arthritis.[5] Without treatment, the destruction of joints will continue, which will be seen clinically with joint deformities and radiographically with the development of juxta-articular erosions, joint space narrowing and in some cases, bony ankylosis.[5]

The enthesis is the attachment site of tendons, ligament and joint capsule into bone. Their function is to reduce stress concentrations during the transmission of force.[17] Enthesitis is inflammation at the site of the insertion of tendons, ligaments or capsules and is a feature of up to 40% of patients with psoriatic arthritis. However, it is also found in other spondyloarthropathies. This enthesitis can occur anywhere in the body, but it most commonly occurs at the achilles tendon, the calcaneal insertion of the plantar fascia and at the insertion of the hamstrings on the ischial tuberosity.[18] Physical examination will reveal soft tissue swelling, usually accompanied by tenderness to palpation and sometimes erythema and warmth over the area. Entheseal inflammation may lead to destruction of the adjacent bone and joints.[5] Entheses are prone to experience high mechanical stresses that make them vulnerable to microdamage. It has been suggested that

biomechanical stress and microdamage at the enthesis may be the trigger that causes enthesitis,[14] much like the Koebner phenomenon in skin lesions. Theoretically, this is important to note as athletes with psoriasis would be at a higher risk of being affected by enthesitis and possibly psoriatic arthritis.

A review of the literature revealed a similar case report of a 39-year-old kicker in the National Football League who developed mild pain in the medial right knee during the preseason.[10] His injury was thought to be mechanical and he was able to finish the season with the help of NSAIDs. A large knee effusion developed while he was resting during the off-season. A corticosteroid injection and a knee arthroscopy, only provided temporary relief from the swelling. It was then noted that the player had several small patches of psoriasis on his elbows. The diagnosis of psoriatic arthritis was made and he was treated with pharmacotherapy.[10] The case is similar to this presentation, as it is an example of a common sports injury presentation that leads to a diagnosis of psoriatic arthritis and points to how easily the correct diagnosis may be missed.

Dactilytis is the swelling of a single digit of the hand or the foot and it is also known as a "sausage digit". It is found in spondyloarthropathies and is common in psoriatic arthritis. It is found in 1/3 to1/2 of patients at some point during the disease. It is more commonly found in the toes than in the fingers.[5] It is caused by a combination of tenosynovitis and enthesitis of the tendons and ligaments as well as synovitis involving the entire digit.[18] On rare occasion, the patient may present without joint involvement, but with considerable enthesitis, as evidenced by multiple sites of aching and stiffness, which may be confused with fibromyalgia or overuse tendonitis.[18]

Nail involvement may be the only indication of psoriasis. Psoriatic nail changes include ridging, pitting, onycholysis and hyperkeratosis. Nail changes are most likely associated with psoriatic arthritis involvement of the DIP joint of that digit.[5]

Other spondyloarthropathies exhibit extra-articular manifestations, such as ocular inflammation (conjunctivitis, iritis, scleritis and episcleritis), oral ulcerations and urethritis. These also occur with psoriatic arthritis, but less frequently.[3]

There are no diagnostic laboratory findings for psoriatic arthritis, however, considering the systemic inflammatory nature of this disease C-reactive protein and the erythrocyte sedimentation rate may be elevated, but to a lesser degree than other inflammatory arthritides.[5] Elevation of these reactants may correlate with disease activity, more commonly in patients with multiple joints affected by psoriatic arthritis.[3] Psoriatic arthritis patients are generally rheumatoid factor negative, yet 10% may test positive. A positive rheumatoid factor test does not exclude psoriatic arthritis,[5] but psoriasis is an exclusion factor for seronegative rheumatoid arthritis.[3]

Radiographic signs include joints space narrowing and marginal bone erosions. The entheses are subject to similar erosions and bone proliferation, especially in the calcaneus, hand and foot.[15] Periarticular osteopenia is usually absent in psoriatic arthritis, which in fact is another feature that helps distinguish psoriatic arthritis from rheumatoid arthritis.[5] Non-marginal syndesmophytes can be found in the spine, along with paravertebral ossification. Together, these two types of ossification can fuse to adjacent vertebral bodies causing bony ankylosis.[15]

When comparing individuals with psoriasis to healthy control groups, individuals with psoriatic arthritis have a reduced quality of life and functional capacity. [18] Early diagnosis of psoriatic arthritis may lead to better management and therefore increased quality of life and ability to engage in physical activity.

Medical treatment for psoriatic arthritis includes several different types of medications. NSAIDs have commonly been used to control mild symptoms of synovitis.[19] Intra-articular injections of glucocorticoids have also been used. However, systemic glucocorticoids need to be used with caution as they are associated with the occasional risk of post steroid psoriasis flare.[20] Methotrexate is one of the most commonly used systemic medications for psoriatic arthritis.[20–23] Tumor Necrosis Factor (TNF) blockers, such as Infliximab and Etanercept, have shown to be an important mediator of inflammation in the skin and synovitis of psoriatic arthritis patients.[24,25] Sulphasalzine has also shown some benefit for patients with peripheral joint activity, but does not seem to have a significant effect on axial disease.[19, 26–28]

Considering arthritis can appear before the psoriatic skin lesions, diagnosing psoriatic arthritis can be challenging. The varying presentation patterns and possibility of overlap with other rheumatic syndromes also add to the difficulty of diagnosis. A thorough physical examina-

tion with a careful assessment of nails and skin may help the clinician. Future research may investigate if psoriatic arthritis is more prevalent in athletes with psoriasis when compared to sedentary individuals with psoriasis.

References

1 Garrick J, Requa R. The epidemiology of foot and ankle injuries in sports. Clinics in Sports Medicine. 1988; 7:29–36.
2 Pardasani A, Feldman S, Clark A. Treatment of psoriasis: an algorithm-based approach for primary care physicians. Amercian Family Physician. 2000; 61(3):725–733.
3 Bennett R. Psoriatic Arthritis. In: Koopman W, Moreland L, eds. Arthritis and Allied Conditions. Vol 1. 15 ed. Philadelphia: Lippincott Williams & Wilkins; 2005.
4 Shbeeb M, Uramoto K, Gibson L, O'Fallon W, Gabriel S. The epidemiology of psoriatic arthritis in Olmsted County, Minnesota, USA, 1982–1991. J Rheumatology. 2000; 27(5):1247–1250.
5 Lam G, Bingham III C. Psoriatic Arthritis. In: Imboden J, Hellman D, Stone J, eds. Current Rheumotology Diagnosis & Treatment. 2 ed. New York: Lange Medical Books/ McGraw-Hill; 2007.
6 Dailey J. Differential diagnosis and treatment of heel pain. Clinics in Podiatry Medicine and Surgery. 1991; 8(1):153–166.
7 Binkley JM, Stratford PW, Lott SA, Riddle DL. The Lower Extremity Functional Scale (LEFS). Physical Therapy. 1999; 79(4):371–383.
8 Heckman DS, Gluck GS, Parekh SG. Tendon Disorders of the Foot and Ankle, Part 2. Am J Sports Med. 2009; 37:13.
9 Jennings F, Lambert E, Fredericson M. Rheumatic diseases presenting as sports-related injuries. Sports Med. 2008; 38(1):14.
10 Brophy RH, MacKenzie R, Gamradt SC, Barnes RP, Rodeo SA, Warren RF. The diagnosis and management of psoriatic arthritis in a professional football player presenting with knee effusion: a case report. Clin J Sport Med. 2008; 2008(18):3.
11 Turkiewicz A, Moreland L. Current concepts on pathogenesis-oriented therapeutic options. Arthritis & Rheumatism. 2007; 56(4):15.
12 Taylor Q, Gladman D, Helliwell P, Marchesoni A, Mease P, Mielants H. Classification criteria for psoriatic arthritis. Arthritis & Rheumatism. 2006; 54(8):2665.
13 Rahman P, Gladman D, Schentag C, Petronis A. Excessive paternal transmission in psoriatic arthritis. Arthritis & Rheumatism. 1999; 42:4.
14 McGonagle D, Tan AL, Benjamin M. The biomechanical link between skin and joint disease in psoriasis and psoriatic arthrits:what every dermatologist needs to know. Ann Rheum Dis. 2008; 67.
15 Yochum T, Rowe L. Essentials of Skeletal Radiology. Vol 2. 2 ed. Baltimore: Williams & Wilkins; 1996.
16 Olivieri I, Scarano E, Padula A, Giasi V, Priolo F. Dactylitis, a term for a different digit disease. Scand J Rheumatology. 2006; 35:8.
17 Doschak MR, Zernicke RF. Structure, function and adaption of bone-tendon and bone-ligament complexes. J Musculosk Neuronal Interaction. 2005; 5(1):6.
18 Myers WA, Gotlieb AB, Mease P. Psoriasis and psoriatic arthritis: clinical features and disease mechanisms. Clinics in Derm. 2006; 24:9.
19 Liu Y, Cortinovis D, Stone MA. Recent advances in the treatment of the spondyloarthropathies. Current Opinion in Rheumatology. 2004; 16:357–365.
20 Mease P. Psoriatic arthritis assessment and treatment update. Current Opinion in Rheumatology. 2009; 21:348–355.
21 Cutolo M, Seriolo B, Pizzorni C, Craviotto C, Sulli A. Methotrexate in psoriatic arthritis. Clinical and Experimental Rheumatology. 2002; 20 (Suppl 28):S76-S80.
22 Jones G, Crotty M, Brooks P. Interventions for treating psoriatic arthritis. Cochrane Database of Systematic Reviews. 2009.
23 Jones G, Crotty M, Brooks P, Group TPAM-AS. Psoriatic Arthritis: A quantitative overview of therapeutic options. Br J Rheumatology. 1997; 36:95–99.
24 Antoni C, Manger B. Infliximab for psoriasis and psoriatic arthritis. Clinical and Experimental Rheumatology. 2002; 20 (Suppl. 28):S122–S125.
25 Mease P. Psoriatic arthritis: The role of TNF inhibition and the effect of its inhibition with etanercept. Clinical and Experimental Rheumatology. 2002; 20(Supplement 28):S116–S121.
26 Clegg D. Comparison of sulfasalazine and placebo in the treatment of psoriactic arthritis. Arthritis & Rheumatism. 1996; 39(12):2013–2020.
27 Dougados M, Linden Svd, Leirisalo-Repo M, et al. Sulfasalazine in the treatment of spondyloarthropathy. Arthritis & Rheumatism. 1995; 38(5).
28 Coumbe B, Goupille P, Kuntz JL, Tebib J, Liotes F, Bregeon C. Sulphasalazine in Psoriatic Arthritis: a randomized, multicentre, placebo-controlled study. Br J Rheumatology. 1996; 35:664–668.

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Intraosseous ganglion cyst of the humeral head in a competitive flat water paddler: case report

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Objective: *To present the diagnostic and clinical features of an intraosseous ganglion cyst of the humeral head of a female flat water canoe athlete.*

Clinical Features: *An 18-year old female flat water canoeist complaining of right shoulder pain following a strenuous paddling training camp.*

Intervention and outcome: *A trial of passive care was conducted, including soft tissue therapy, spinal manipulative therapy, acupuncture, and rehabilitation. The patient seemed to be responding with treatment, but pain would always resume with paddling. A diagnostic ultrasound displayed mild thickening and effusion in the subacromial/subdeltoid bursae. Continued passive care was not able to resolve the symptoms and she underwent an MRI which revealed an intraosseus ganglion cyst subjacent to the lesser tuberosity and floor of the intertubercular groove. A subsequent MRA was ordered to assess the labrum, which was intact, but the cyst had progressed in size. She was referred to an orthopedic surgeon who performed surgery.*

Conclusion: *An IOG cyst within the humeral head is a rare, potentially painful condition that can mimic other pathologies including impingement and labral tear. It is important to be aware of the clinical features to obtain*

Objectif : *soumettre un diagnostic et les caractéristiques cliniques d'un kyste ganglionnaire intraosseux de la tête humérale d'une athlète pratiquant le canoë en eau plate.*

Caractéristiques cliniques : *une canoéiste en eau plate de 18 ans se plaint de douleurs à l'épaule droite suite à un camp d'entraînement très exigeant.*

Intervention et résultat : *un essai de soins passifs fut mené, notamment la thérapie des parties molles, la manipulation rachidienne, l'acupuncture et la réhabilitation. La patiente semble avoir bien réagi au traitement, mais la douleur revient lorsqu'elle recommence à ramer. Un ultrason diagnostic démontra un épaississement léger et une effusion dans les bourses sous-acromiales/des courts rotateurs de l'épaule. Les soins passifs continus n'ont pu éliminer les symptômes, et elle subit un examen à résonnance magnétique qui révéla un kyste ganglionnaire intraosseux sous-jacent à la tubérosité moindre et au plancher de la cannelure intertuberculaire. Une angiographie à résonnance magnétique subséquente fut exigée pour évaluer le bourrelet marginal, qui était intact, mais le kyste avait grossi. Un chirurgien orthopédique dut l'opérer.*

Conclusion : *un kyste ganglionnaire intraosseux dans la tête humérale est un état pathologique douloureux plutôt rare, qui ressemble à d'autres états pathologiques, notamment la déchirure du bourrelet marginal et le coincement. Il est important de connaître les caractéristiques cliniques afin de diagnostiquer*

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a prompt diagnosis and appropriate treatment of this condition.
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KEY WORDS: intraosseous cyst, ganglion cyst, paddler, flat water, humeral head, canoe

rapidement cet état pathologique et de prescrire un traitement adéquat.
(JCCA 2011; 55(4):294–301)

MOTS CLÉS : kyste intraosseux, kyste ganglionnaire, canoéiste, eau plate, tête humérale, canoë

Introduction

Canoeing and kayaking are two sports that have their origins in North America.[1] Native Americans and Canadians and Inuit developed and used the canoe and kayak for trading, transportation and fishing. The canoe and kayak played a huge role in the history and transformation of Canada into a country when the Voyageurs adapted their use and procured new trade opportunities in the New World.[2] In a manner similar to the use of horses and horseracing, the mechanization of society diminished the role of the canoe and kayak but their traditions live on through competition.

In competition, canoeing and kayaking are categorized into "whitewater" and "flat water"; whitewater referring to paddling on a moving body of water and flat water a non-moving body of water. Men's flat water canoeing and kayaking debuted in the 1936 Berlin Olympics while women's flat water kayaking was added in 1948.[2]

The American Canoe Association estimates that there are approximately 24 million Americans involved in canoeing and kayaking.[2] Another study, done in 2004, showed similar results, indicating that participation had increased from 16.7 million in 1994–95 to 22.6 million in 1999.[3] Of the 22.6 million participants, the study indicated 28% were women.[3]

Injury data for these two sports is difficult to ascertain due to a lack of published studies. In a survey done in 2000 on whitewater canoe and kayak injuries, the estimated incidence of any type of injury was 4.5 injuries per 1000 days paddled with an incidence of significant injuries (an injury for which medical evaluation/care was sought) at 1.9 injuries per 1000 days paddled.[3] They also found a median average for days paddled was 50 days per year with a range of 5 to 300 days.[3] A similar survey for flat-water paddlers could not be found and makes any extrapolation to this type of paddling difficult. Precipitating factors in paddling injuries include intense, high-volume on-water training, less-than-optimal paddling stroke mechanics and maximum strength training during dry-land workouts.[4]

Intraosseous ganglion (IOG) cysts of the humerus is a rare and often painful condition.[5–9] There is only one other case in the literature describing an IOG cyst of the humerus, while most papers describe cystic lesions of the carpal and tarsal bones.[6,10,11] Symptomatic IOG cysts present with intermittent pain, occasional swelling and it is aggravated via movement of the affected area.[6,12] IOG cysts are often misdiagnosed or there is a delay in diagnosis due to the variable presentation.[6–8,11–13] Early diagnosis is important to avoid mismanagement of patients.

Another case of a ganglion cyst affecting the shoulder in a canoeist was reported in a 15 year old female.[14] The athlete complained of pain, reduced abduction and external rotation of her left shoulder with intermittent parasthesia over the left scapula. An EMG and MRI revealed an entrapment neuropraxia of the left suprascapular nerve due to a ganglion cyst. The authors felt the repetitive nature of the paddling stroke caused an otherwise asymptomatic, pre-existing ganglion cyst to become symptomatic.

The purpose of this case report is to: present a rare case of intraosseus ganglion cyst (IOG) of the humerus diagnosed in a flat water canoe athlete; outline the difficulties in its diagnosis due to an IOGs ability to mimic other pathologies; outline the sports' biomechanics predisposing the athlete to other, more likely, pathologies (impingement syndromes, rotator cuff tendinopathy …); the imaging needed to diagnose an IOG; and the subsequent surgical management of the patient.

Case Presentation

An 18 year old female national level flat water canoe athlete was assessed for a right anterior shoulder and upper back complaint that started following an intensive paddling training camp three weeks prior. She paddles on the left side with her right being the high hand on the paddle. The pain was progressing and not improving. She re-

ported a previous, similar right shoulder complaint two years prior that had resolved within three weeks with chiropractic care. She also complained of some numbness and tingling into the posterolateral arm that she had not had previously.

On examination, her cervical spine range of motion and neurologic exam were within normal limits. During orthopedic evaluation of the shoulder, both empty and full can were found to be positive, while crank test elicited pain and clicking. Passive joint play of the glenohumeral joint demonstrated mild to moderate laxity. Joint play of the cervical and thoracic spine revealed locally tender restrictions. An upper limb tension test of the radial nerve recreated the numbness and tingling in the arm, doorbell test was locally tender and Hoffman's test was negative.

The patient was tentatively diagnosed with a rotator cuff tendinopathy and radial nerve entrapment (posterior interosseous nerve syndrome), with differential diagnoses of labral tear, supraspinatus tear, and cervical spine radiculopathy. Treatment of this patient included soft tissue therapy (myofascial release) of the affected right shoulder musculature and radial nerve, spinal manipulative therapy of the cervical and thoracic spine, rehabilitative exercises and a progressive return to paddling. Her MD had prescribed Celebrex but she was unsure if it was helping. She was also referred for a diagnostic ultrasound of the right shoulder.

The diagnostic ultrasound performed within 1 week of referral (4 weeks post-injury) suggested that all rotator cuff musculature was intact and no effusion was visualized in any of the tendons. However, mild thickening and effusion was noted in the subacromial/subdeltoid bursae.

Her shoulder responded moderately well to treatment but would re-aggravate following a paddling session longer than 10 or 15 minutes. Upon further evaluation by the primary author, she had tenderness of the long thoracic, dorsal scapular, and suprascapular nerves with palpation and tension of the nerve with palpation increased the pain. ***(Author's Note: In the primary author's experience, these peripheral nerve findings are consistent with other nerve entrapments that involve predominantly the C5 nerve root.)*** She also had pain in the shoulder with shoulder flexion and abduction. Treatment of the entrapment sites of these nerves using myofascial and vibration-assisted myofascial release and acupuncture was added to her plan of management as well as acupuncture to the arm and shoulder including Li4, 11, 12, 15, TW14, Lu5, GB20 and 21. Nerve flossing exercises of the affected nerves were added to her treatment program as well. With the addition of these therapies, the posterolateral arm tingling resolved and her pain free shoulder range of motion increased.

Paddling continued to aggravate her condition and following a particularly painful training session several SLAP lesion tests were now positive including: O'Brien's, Biceps Load 2 and anterior slide. She was recommended to get an MRA, ideally to assess the labrum, or an MRI and reduce the paddling as much as possible.

An MRI performed 5 months post-injury revealed an 8 mm × 6 mm intraosseus ganglion cyst subjacent to the lesser tuberosity and floor of the intertubercular groove (see Figure 1). She was told by the medical doctor that the cyst was not the reason for her pain. Due to its close proximity to the biceps tendon in the intertubercular groove, it was suggested that the cyst may be causing a tendinopathy and she received soft tissue treatment and rehabilitation. All other structures in the shoulder were intact and no other effusion was noted in the shoulder or bursal complex upon MRI evaluation.

The patient's shoulder complaint was not improving and she was referred to an orthopedic clinic at the university she was attending. The patient was prescribed physical therapy that mainly consisted of exercises which re-aggravated her condition. She was subsequently referred for an MRA. An MRA performed 8 months post-injury showed that the labrum was intact but the cyst had progressed to a size of 12 mm (see Figure 2). It also showed a partial thickness tear of the bursal surface of the supraspinatus tendon, and fraying of the subscapularis tendon. She was referred to an orthopedic surgeon who performed surgery 9 months post injury.

The orthopaedic surgeon assessed the shoulder arthroscopically and reported an intact rotator cuff with slight fraying of the subscapularis that was felt to be minimal and required no intervention. A small incision was made in the anterior shoulder, the cyst site was identified due to a small hole noticed in the bone deep to bicep tendon. The area was drilled and a curette was used to remove any material but little was extracted. The site was then injected with a small amount of demineralized bone matrix. The surgeon's prognosis in terms of recovery time was

Figure 1 *MRI revealing intraosseous ganglion cyst. The white arrows indicates the IOG cyst.*

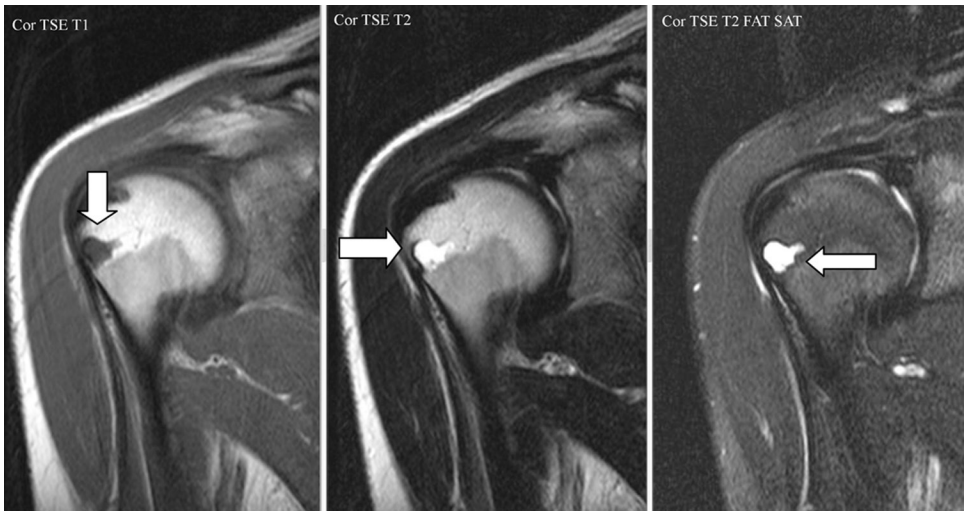
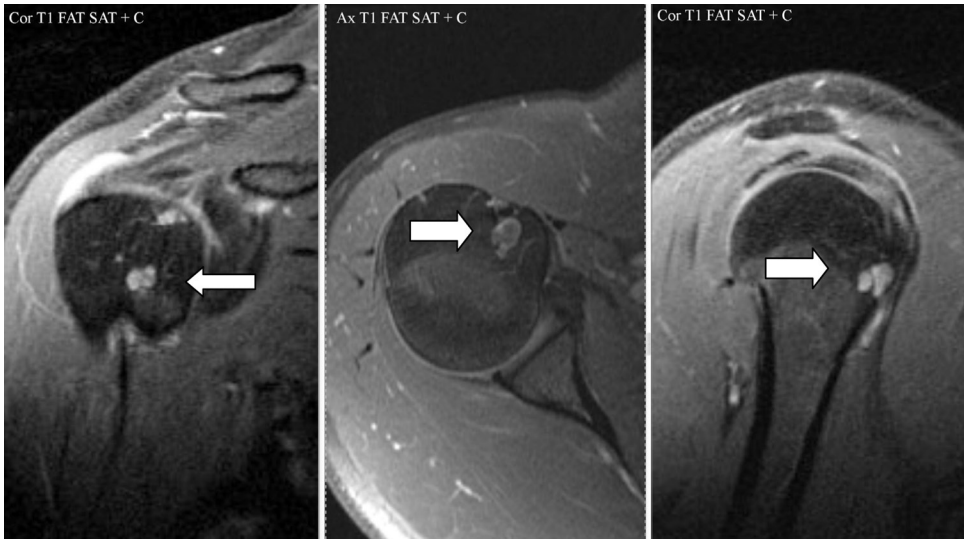


Figure 2 *MRA revealing the intraosseous ganglion cyst. The white arrows indicates the IOG cyst.*



unknown – he had not seen a similar case previously. The patient has returned twice for follow-up with the surgeon who feels she is making good progress.

Physical therapy was initiated approximately 2 weeks after surgery that included range of motion exercises, postural/scapular setting, and kinesiotaping to aid proper positioning. The program was progressed to light strengthening and lower body cardiovascular exercise. At 14 months post injury (5 months post surgery), the patient reported full range of motion and that she was progressing through her weight training program.

She also reported a return to paddling one time per week for 1 hour sessions with no return of the original pain.

Discussion

This is the second case known to the authors to describe IOG cysts of the humeral head and the first in a flat water paddler. The discussion will outline the reasons why the shoulder is an oft injured area, including the biomechanics of the canoe stroke (see Tables 1), as well as IOG cysts in bones. Because IOG cysts in the humeral head are very rare, the IOG cyst discussion will primarily be based on information in bones more commonly involved.

Shoulder injuries in paddlers are very common.[4,15,16] Berglund and McKenzie[16] suggest that the most common types of shoulder injuries in paddlers are impingement syndrome, bicipital tendonitis, and glenohumeral dislocations/subluxations although there were no statistics or references related to the prevalence of these conditions. They report that impingement syndrome is a constant source of lost training or missed competition, however, degeneration and subsequent tearing of the rotator cuff is very rare in the paddling athlete.

A condition known as "paddler's shoulder" is a soft tissue injury of the shoulder that includes shoulder impingement syndrome, bicipital tendonitis and subacromial bursitis.[4] Predisposing factors for paddler's shoulder include maintaining a high (upper) pivot arm in the canoe paddling stroke as well as hypertrophy of the musculotendinous structures of the rotator cuff that fills the subacromial space.[4] Interestingly, a comparison of canoe athletes between the 1976 and 2000 Olympics showed that both male and female athletes were 5 kg heavier although having similar skinfold measurements, and had increased shoulder breadth and chest girth indicating hypertrophic changes in these and other muscle groups.[17] These changes were attributed to "advances" in off and on water training programs. Although suggestive of a predisposing factor,[4] no injury data for comparison was available.

Our patient was initially diagnosed with a rotator cuff tendinopathy secondary to shoulder impingement and this differential remained high on the list even after the MRI showed an intraosseus ganglion cyst. It was not until the surgery and the lack of tendon damage being found that rotator cuff tendinopathy could be seemingly ruled out.

Ganglion cysts most commonly exist in soft tissue, but there are rare occurrences where they have been diagnosed within the bone termed as an intraosseous ganglion cyst.[5] IOG cysts present as solitary well-defined, sharply marginated lytic lesions that are commonly found within the subchondral region and epiphyseal areas of long and short tubular bones.[6,7,8] They come in close proximity to the joint but rarely perforate the joint cavity or articular surface.[6,7,8] These cysts form near joints that do not undergo significant degenerative changes.[7] They most often occur in the lower end of the tibia, medial malleolus, femoral head, and carpals.[6,7,9] However, they rarely affect the neck of the scapula, head of the humerus, ulnar head and acetabulum.[6,7,9]

The exact pathogenesis of ganglion cysts is still unclear but a few theories exist. One theory suggests that ganglia are due to intramedullary metaplasia of mesenchymal cells into synovial type cells as an idiopathic process in response to an unknown stimulus.[7,8,12] The second theory describes penetration of an extra-osseous synovial cyst which begins to erode into the bone and eventually becomes isolated within the bone by new bone formation filling the intracortical gap.[6,7,8,11,13] The last theory proposes a microvascular disorder that leads to the development of cysts after focal ischemic bone necrosis.[8]

The clinical presentation of IOG cysts is quite variable and nonspecific, which can impact obtaining prompt diagnosis.[7,13] In most cases there is intermittent mild localized pain, aggravated with activity of the affected area.[6] There may or may not be associated swelling and soft tissue masses depending on the location of the cyst.[6,13] Pain may be present for many months due to a delay in diagnosis[11] which can vary from two months to three years.[6,7,8,10,11,18] In the case presented, the patient was told by the MD that the pain was not due to the IOG cyst, which delayed adequate treatment. Interestingly during the surgery, the surgeon noted that there was a small hole in the bone leading into the cyst suggesting that there was communication between the biceps tendon and the ganglia. If the cyst was enlarging during and after activity, and its contents were pushing through the communicating channel, the biceps tendon may have been forced anteriorly potentially increasing the risk of impingement.

Diagnosis of IOG cysts is achieved via radiographic or special imaging. Upon x-ray analysis, IOG cysts appear as a single or multiple confluent radiolucent, eccentric defects measuring 1–2 millimeters surrounded by a narrow zone of sclerosis.[6,7,12,18] The lesion, however, usually does not extend deep into the bone.[6,10] CT scans display details of spatial orientation of the lesions, confirms the cysts liquid aspect and is helpful in detecting any cortical defect

Table 1 *Canoe biomechanics*[1,4,19]

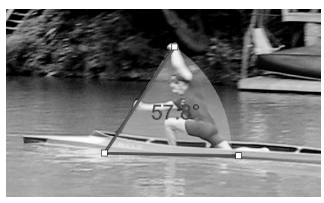
Canoe stroke position	Anterior view	Lateral view
Set up The canoeing stroke involves the athlete kneeling on one knee, with the contralateral hip and knee flexed to 90 degrees. The bottom arm is held at approximately 100 degrees of shoulder flexion, with the hand positioned just above the blade while the top arm is at 170 degrees of shoulder flexion, with the hand at the top of the paddle (A-frame position). The stroke side hip and torso are rotated forward to allow the bottom arm to reach as far forward as possible.	 **Note the Neer impingment position.**	 
Catch The torso flexes forward to allow the blade to enter the water. The top and bottom arms drive downward, with the blade entering the water at approximately 60 degrees.		 
Draw or Pull The pulling motion is initiated by the stroke side hip rotating back away from the bottom hand. The elbows stay extended throughout the pull phase. The body then rotates posteriorly, keeping both elbows extended. Both shoulders will move into extension, with the top shoulder adducting and crossing the midline of the body.		

Table 1 *(Concluded)*

Exit As the blade nears the hip, the bottom arm will flex at the elbow to allow the blade to exit the water.		
Recovery The top arm quickly flexes upward as the body rotates towards the front of the boat to place the blade back in the water. The forward rotation is initiated by the stroke side hip rotating forward as the blade exits the water.	Return to Set up position	Return to Set up position

or articular communication.[10] MRI scans exhibit a well-defined fluid collection with low intensity of T1-weigthed images and very high intensity on T2-weighted images.[11] In our present case, the initial MRI clearly shows the IOG on the T2 weighted images (see figure 1) and the MRA (see figure 2).

Differential diagnoses of IOG cysts include: aneurysmal bone cyst, osteoblastoma, giant cell tumour, fibrous dysplasia, chondromyxoid fibroma, osteiod osteoma, rheumatoid arthritis, simple bone cyst, enchondroma, chondroblastoma, and subchondaral bone cyst.[6,12] Many of these differentials can be ruled out based on patient age, symptomatolgy, and location. IOG cysts are commonly confused with subchondral cysts, which communicate with the joint, while IOG cysts rarely do so and are not associated with a degenerative a process.[6,7,8]

Conservative management is always considered the first step to treatment. There are a limited number of reports outlining the conservative treatment of IOG. Conservative care typically consists of NSAIDs and splinting of the affected area.[18] In our present case, the patient received physical therapy, chiropractic care (16 visits), acupuncture and appropriate activity modification. This regimen was moderately successful in reducing the pain when the patient was not paddling but the pain quickly returned following a session on the water. It was due to this unusual recurrence pattern, atypical for a normal soft tissue/overuse injury, that further investigation was initiated.

If conservative therapy fails, surgery is an option. There are several indications to guide operative intervention. These include: i) failure of conservative modalities to provide adequate relief of symptoms, ii) suspicious x-ray changes, iii) the cyst progressively entering and replacing the cancellous substance of the bone eroding the cortex. Even if the lesion is not painful, it weakens the bone putting it at risk for fracture.[12,18] The surgical procedure with the lowest recurrence rate is known as curettage[6,18] which involves excision of the cyst followed by bone grafting to prevent recurrence and the risk of collapsing fracture.[12,18]

Conclusion

The biomechanics of the canoe stroke and the hypertrophy of the musculotendinous contents of the subacromial space seemingly predispose the paddling athlete to shoulder overuse/impingement injuries.[4] Combine these factors with an acute episode of extreme training,[4] as in

our case, and this can lead to a shoulder injury. Whether the extreme training caused the intraosseus ganglion cyst to become symptomatic is debatable but has been suggested previously[14] although the mechanism is unclear.

IOG cyst of the humeral head is a rare entity that may result in a great deal of pain. It is important for clinicians to be aware that cysts not only exist in soft tissues but also within the bone. Patients may present with enduring pain lasting anywhere between two months to three years and delays in diagnosis can lead to inappropriate management and delays in proper treatment.

References

1 Shephard RJ. Science and medicine of canoeing and kayaking. Sports Medicine. 1987; 4: 19–33

2 Kenal K, Trela P. Canoeing and Kayaking. In: Drinkwater, BL, editor. Women in Sport. Oxford UK: Blackwell Science LTD; 2008. p.600

3 Schoen RG, Stano MJ. Year 2000 Whitewater Injury Survey. Wilderness Environ Med. 2002; 13(2):119–24.

4 Pelham TW, Holt LE, Stalker RE. The etiology of paddler's shoulder. Australian J Science Med Sport. 1995; 27(2):43–7.

5 Fealy MJ, Lineaweaver W. Intraossrous ganglion cyst of the scaphoid. Ann Plastic Surgery. 1995; 34(2): 215–217.

6 Kambolis C, Bullough PG, Jaffe HL. Ganglionic cystic defects of bone. J Bone Joint Surg. 1973; 55: 496–505.

7 Pope TL, Fechner RE, Keats TE. Intra-osseous ganglion. Report of four cases and review of the literature. Skeletal Radiology. 1989; 18: 185–187.

8 Schrank C, Meirer R, Stabler A, Nerlich A et al. Morphology and topography of intraosseous ganglion cysts in the carpus: an anatomic, histopathologic, and Magnetic Resonance Imaging correlation study. J Hand Surg. 2003; 28A:52–61.

9 Tuzner T, Subasi M, Alper M et al. Penetrating type intraosseous ganglion cyst of the lunate bone. West Indian Med J. 2005; 56(6):384–386.

10 Mnif H, Koubaa M, Zrig M et al. Ganglion cyst of the carpal navicular. a case report and review of the literature. Orthopaedics & Traumatology: Surgery and Research. 2010; 96:190–193.

11 Nishimura T, Tsujii, Kusuzaki, Hoki Y et al. Intra-osseous ganglion of the proximal humerus: case report. J Ortho Surg. 2007; 15(1):102–105.

12 Uriburu, Levy VD. Scaphoid and lunate bones: report of 15 cases in 13 patients. J Hand Surg. 1999; 24A:508–515.

13 Tan EW, Dharamsi FM, McCarthy EF et al. Intramuscualr synovial cyst of the shoulder: case report. J Shoulder Elbow Surg. 2010; 19:e20–e24.

14 Knossalla F, Nicolas V, Tegenthoff M. Suprascapular nerve entrapment in a canoeist. Arch Neurol. 2006; 63(5):781.

15 Kameyama O, Shibano K, Kawakita H et al. Medical check of competitive canoeists. J Ortho Science. 1999; 4(4):243–9.

16 Berglund B, McKenzie D. Injuries in canoeing and kayaking. In: Renstron, editor. P.A.F.H. Clinical Practice of Sports Injury, Prevention, and Care. Encyclopaedia of Sports Medicine. Oxford; Blackwell Scientific Publications; 1994. P.633–640

17 Ackland TR, Ong KB, Kerr DA, et al. Morphological characteristics of Olympic sprint canoe and kayak paddlers. J Science Med Sport. 2003; 6(3):285–94.

18 Kural C, Sungur I, Cetinus E. Bilateral lunate intraosseous ganglia: case report. Orthopedics. 2010; 33(7):514.

19 Canoe Kayak Canada [Internet]. Ottawa; c2009–2011 [updated 2002 Sep; cited 2011 Apr 11]. CKC Canoe Technical Page. Available from: http://www.canoekayak.ca/files/49/72/Canoe_TECHNICAL_TEMPLATEwebsite_version.pdf

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Acute compartment syndrome of the foot in a soccer player: a case report

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Objective: *To present the diagnostic and clinical features including management of acute compartment syndrome (ACS) of the foot and to create a sense of emergency amongst clinicians of this rare and dangerous condition.*

Clinical Features: *A 28-year old male soccer player on acetylsalicylic acid (ASA) and verapamil presented with severe swelling, paresthesia, and pain in the left ankle after an acute grade three-inversion ankle sprain. A diagnosis of foot compartment syndrome was made.*

Intervention and Outcome: *A fasciotomy was not performed and subsequent neurological sequelae occurred. We hypothesize that the edema caused by the ankle sprain was excessive due to the use of ASA and verapamil, resulting in increased compartmental pressure and neurological signs in the foot.*

Summary: *Although rare, it is extremely important to be aware of the clinical features of ACS of the foot to obtain an appropriate diagnosis and manage this medical emergency promptly.*
(JCCA 2011; 55(4):302–312)

KEY WORDS: acute compartment syndrome, inversion ankle sprains, soccer, aspirin, verapamil

Objectif : *présenter le diagnostic et les caractéristiques cliniques, notamment la gestion du syndrome des loges aigu du pied, et sensibiliser les cliniciens à cet état pathologique rare et dangereux.*

Caractéristiques cliniques : *un joueur de soccer de 28 ans consommant de l'acide acétylsalicylique et du vérapamil souffrait d'une grave enflure, de paresthésie et de douleurs dans la cheville gauche suite à une entorse d'inversion de la cheville de catégorie trois. On lui a diagnostiqué un syndrome des loges du pied.*

Intervention et résultat : *la fasciotomie n'a pas été exécutée, et des séquelles neurologiques s'ensuivirent. Nous supposons que l'œdème causé par l'entorse à la cheville était grave en raison de l'utilisation de l'acide acétylsalicylique et du vérapamil, qui a eu pour effet d'accroître la pression et les signes neurologiques dans le pied.*

Sommaire : *bien que rare, il est très important de connaître les caractéristiques cliniques du syndrome des loges aigu afin d'émettre un diagnostic précis et de gérer cette urgence médicale rapidement.*
(JCCA 2011; 55(4):302–312)

MOTS CLÉS : syndrome des loges aigu, entorses d'inversion de la cheville, soccer, aspirine, vérapamil

Introduction

Inversion ankle sprains causing disruption to the anterior talofibular ligament are one of the most common injuries seen in the athletic population.[1] Approximately 25% of all musculoskeletal injuries leading to medical treatment are acute ankle sprains and ruptures of the lateral ligaments of the ankle, most commonly caused by an inversion mechanism of injury.[2] Further, the most common injury occur-

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ring in the sport of soccer is injury to the ankle region resulting in ankle sprains.[3] Ankle sprains are common in soccer players due to frequent contact with other players, repetitive jumping, running, and frequent abrupt changes in direction of motion also known as cutting movements. Cutting movements cause an excessive amount of angular and rotational strain on the ankle joint.[4] Initially, a common treatment plan for an acute inversion ankle sprain is to protect, rest, ice, compress, and elevate[5] (PRICE). This simple protocol is well documented in current literature to decrease swelling thereby decreasing time to heal and decreasing the time it takes for an athlete to return to play. However, this may not be the proper protocol when there is an increase in intracompartmental pressure (ICP) leading to acute compartment syndrome (ACS).[6,7,8] ACS of the foot is extremely rare after an inversion ankle sprain and may be complicated with the use of platelet inhibitors and calcium channel blockers.

This article presents a case of ACS of the foot following a grade three inversion ankle sprain in a 28-year-old male soccer player on a daily regimen of ASA (162 mg) and Verapamil (320 mg) pharmacotherapy. The patient consented to release all information in regard to his case for publication.

Case report

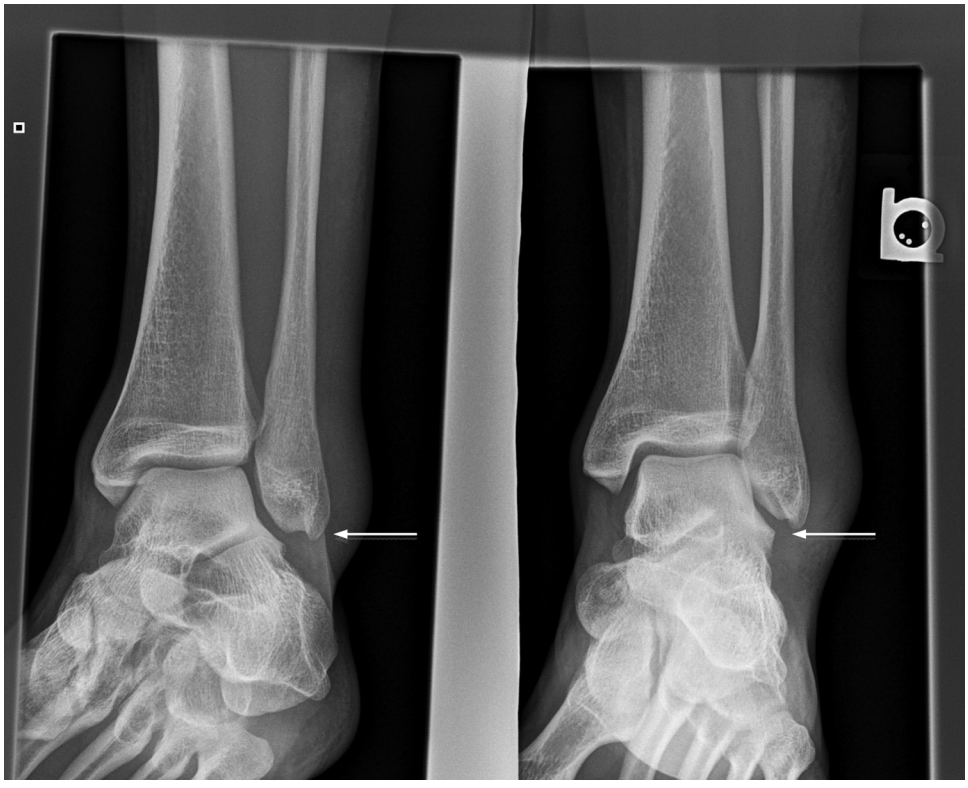
A 28-year old male, recreational soccer player, presented to a chiropractor with a primary complaint of left lateral ankle pain and swelling surrounding and up to 6 cm proximal to the lateral malleolus following a high-energy deceleration injury into ankle inversion and plantar flexion. The patient attributed this injury to attempting to block a kick of the opposing player while playing soccer. Presentation to the chiropractor was fifteen minutes after initial trauma. The patient reported immediate sharp pain and swelling of the lateral aspect of the ankle. Initially, the pain intensity was 6/10. Medical history revealed this patient previously suffered two cryptogenic (of undetermined origin) ischemic strokes at age 17 and was prescribed 162 mg of ASA for platelet inhibition and 360 mg of verapamil to increase vasodilation. Hospitalization occurred for approximately one week after each stroke and pharmacotherapy was prescribed to decrease the chance of recurrent stroke. The patient was also on 40 mg of omeprazole used as a proton pump inhibitor to decrease hydrochloric acid formation in the stomach. No nutriceuticals were reported. The patient reported no other history of significant injury, trauma, or surgeries, and systems review was unremarkable.

On immediate physical examination, observation revealed that the distal 1/3 of the anterolateral aspect of the left leg and left foot was extremely swollen. There was pain on palpation of the distal 1/3 of the left extensor digitorum longus, extensor hallicus longus, the left lateral malleolus, and 6 cm above the lateral malleolus. Range of motion revealed limitations in dorsiflexion and plantarflexion of 10 degrees total in the left ankle. There was an inability to perform inversion and eversion due to extreme pain and swelling. Neurological examination revealed paresthesia on the dorsum of the foot, the 2nd, 3rd, 4th digits, and one inch inferior to the medial malleolus. Motor examination revealed a significant decrease in strength in all ranges of motion. Deep tendon reflexes were within normal limits (L4 and S1).

The chiropractor proceeded with radiographical evaluation of the left ankle and left tibia and fibula. Radiological examination revealed a small flake fracture of the distal aspect of the left fibula and severe soft tissue swelling (see Figure 1 and 2). The chiropractor provided the patient with crutches and a strict protocol of rest, ice, compression, and elevation for a grade three-inversion ankle sprain. Approximately nine hours after release from the chiropractor the patient experienced extreme swelling, hypersensitivity, and shooting pain in the dorsum of the left foot. The patient proceeded to the emergency department due to extreme constant sharp, electric like pain out of proportion of injury and rapidly deteriorating neurological signs.

Upon examination in the emergency room, vital signs revealed a temperature of 36.1, pulse rate 118 bpm, respiratory rate 20 bpm, and blood pressure was 134/86. The left foot was severely swollen, tender, and extremely painful and weak on dorsiflexion and plantarflexion of the toes and ankle. The character of the pain was reported as sharp and electric like. Pain intensity was reported as 10/10. Observation revealed a large one-inch blister forming on the lateral aspect of the left ankle (see Figure 3). Severe bruising and swelling of the left leg and foot was evident (see Figure 4 and 5). Further, there was severe pitting edema on the dorsum of the left foot and 1/3 of the distal aspect of the leg. The toes were slightly pale and capillary refill was approximately 2 seconds on left and

Figure 1 *AP and medial oblique radiograph of the left ankle. Arrows indicate an avulsion fracture of the distal fibula (fragmentation).*



<2 seconds on right. Sensory examination revealed continued paresthesia in the same distribution. The dorsalis pedis and posterior tibial pulses were not palpable due to extreme swelling. (See Table 1 and 2).

The patient was admitted to the hospital for observation and consultation by an orthopedic surgeon due to rapidly progressing pain and swelling in left foot and ankle and a differential diagnosis of ACS of the foot. The orthopedic surgeon suspected the patient might have sustained a subtalar dislocation with spontaneous reduction although no dislocation was visualized on radiographic imaging of left ankle and foot.

Due to the past history of cryptogenic ischemic strokes the patient was sent for complete doppler examination of left leg. The visualized portions of the common femoral, superficial femoral, popliteal, and calf veins were free of echogenic thrombus, with normal compression, augmentation, and phasicity (the cyclic rise and fall of venous flow with normal respiration). There was no evidence of deep vein thrombosis in the left leg. Further, doppler ultrasound showed intact dorsalis pedis and posterior tibial pulses of the left foot.

A diagnostic ultrasound of the left ankle revealed a large hematoma on the lateral malleolus measuring $3.7 \times 3.3 \times .$ 69 cm, the left anterior talofibular ligament (ATFL) was not intact, and bony fragments were found surrounding the distal aspect of the fibula (see Figure 6, 7 and 8).

Continuous inpatient monitoring occurred for 6 days with repeated clinical assessment every 2 hours. The ICP was not measured. The patient was placed in an open plaster cast with the foot raised to the level of the heart for 6 consecutive days. No change in medication was provided. Morphine 5–7 mg was prescribed intravenously every 15 minutes PRN (as needed), 10 mg oxycodone was administered orally every 6 hours PRN for pain control, and

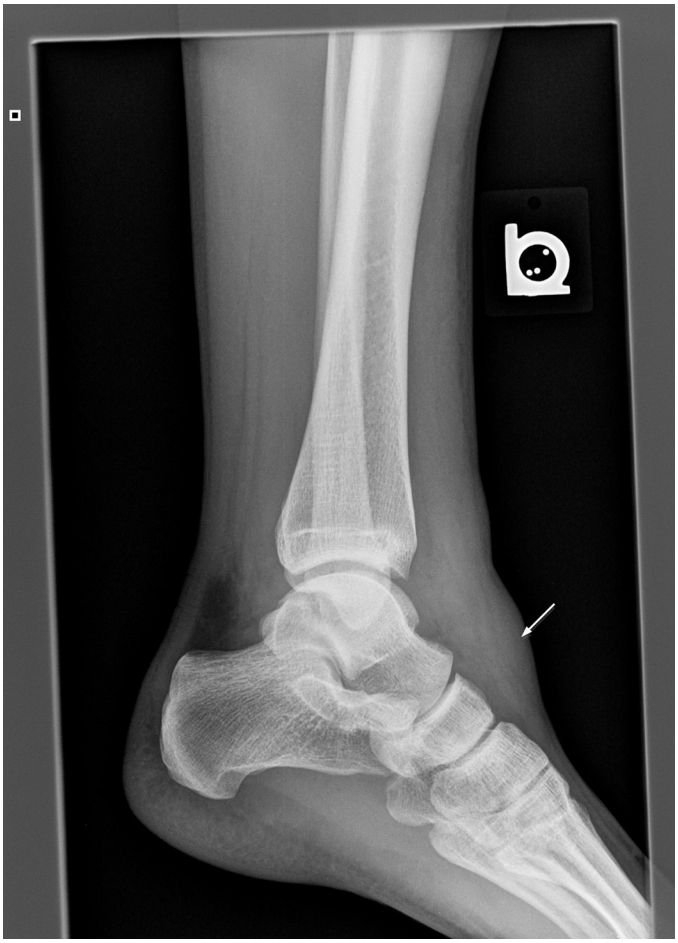


Figure 2 *Lateral radiograph of the left ankle. The arrow indicates severe soft tissue swelling of the left foot and ankle.*

Table 1 *Blood Chemistry Results*

Procedure	Value	Reference Range	Units
Urea	**8.3 H**	[2.0–8.0]	MMOL/L
Creatinine	**114 H**	[44–110]	UMOL/L
Anion Gap	**20 H**	[6–15]	MMOL/L
Potassium	**3.1 L**	[3.3–4.5]	MMOL/L
Carbon dioxide	**20 L**	[22–29]	MMOL/L
Glucose random	5.8	[3.8–6.1]	MMOL/L
Sodium	144	[133–145]	MMOL/L
Chloride	104	[95–107]	MMOL/L

H – *Value higher than reference range*
L *– Value lower than reference range*

25 mg dimenhydrinate intravenously every 4 hours PRN for nausea relief.

The patient's left leg was immobilized and placed in a below-knee airform walker and released from the hospital after a 6-day duration. A fasciotomy was not performed. This patient was reviewed in the Outpatient Fracture Clinic 14 days after release from the hospital where another radiological ankle series was performed and read as unremarkable.

The patient received three weeks of extensive chiropractic treatment after discharge from the hospital, once the airform walker was removed and ambulation was near pre-injury state. Active Release Technique® was provided to the anterior tibialis, extensor hallicus longus, extensor digitorum longus, extensor retinaculum, flexor hallucis longus, tibialis posterior, flexor digitorum longus, and achilles tendon. No mobilizations or manipulations of the ankle were performed. Range of motion exercises, rehabilitation, and proprioception exercises including single leg heel raises and theraband® concentric and eccentric exercises were prescribed. Rehabilitation exercises were completed at home for three months duration.

Upon one-year follow-up, weakness in dorsiflexion of the great toe on the left could be elicited. Motor examination of the extensor hallicus longus was 4/5. Weakness of the extensor hallux longus may have been due to severe swelling of the foot leading to hypoxia and focal tissue necrosis of the deep fibular nerve or axonotmesis directly from the mechanism of injury. All other motor testing was within normal limits. The patient could actively dorsiflex the foot to 15 degrees and plantarflex to 45 degrees, normal ranges are 20 degrees and 50 degrees respectively. Sensory deficits of the left foot included paresthesia of the dorsum of the left foot, 2nd, 3rd, and 4th digits, and one inch inferior to the medial malleolus. Axonotmesis, a peripheral nerve injury occurred to the superficial fibular nerve causing sensory disruption in the dorsum of the foot and digits and to the tibial nerve causing sensory disruption one inch inferior to the medial malleolus. Posterior tibial and dorsalis pedis pulses were palpable. No contractures of the toes or deformities of the affected leg or foot were noted. The patient was not engaged in any intervention

Table 2 *Hematology Results*

Procedure	Value	Reference Range	Units
Leukocyte count	**15.0 H**	[4.0–11.0]	10E9/L
Red blood cell distribution	**0.147 H**	[0.115–0.140]	
Neutrophils	**10.90 H**	[2.0–7.5]	10E9/L
Hemoglobin	**129 L**	[130–180]	g/L
Hematocrit	**0.379 L**	0.390–0.540]	L/L
Red Blood Cell Count	4.52	[4.50–6.50]	10E12/L
Mean corpuscular volume	84	[78–98]	fL
Volume distribution of erythrocytes	339	[320–360]	g/L
Platelets	385	[150–400]	10E9/L
Lymphocytes	3.00	[1.0–4.0]	10E9/L
Monocytes	0.90	[0.1–1.2]	10E9/L
Eosinophils	0.10	[0–0.4]	10E9/L
Basophils	0.05	[0–0.2]	10E9/L
International Normalized Ratio Blood Test	1.15	[0.84–1.16]	
Partial Thromboplastin Time	30.3	[26.0–38.0]	seconds

H – *Value higher than reference range*
L *– Value lower than reference range*

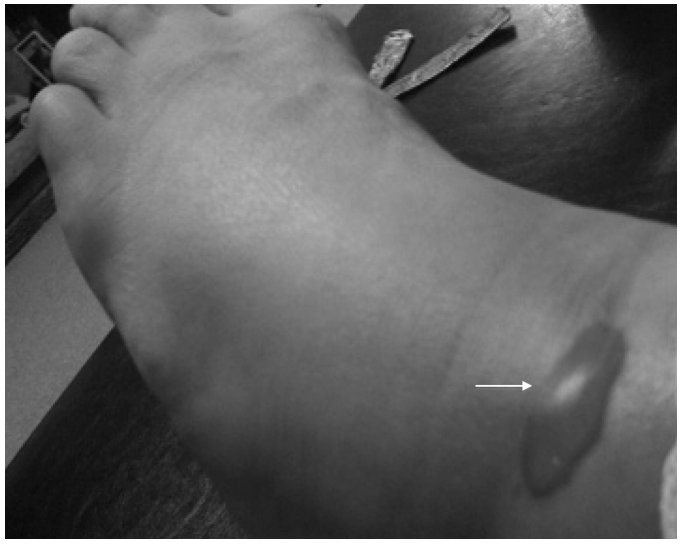


Figure 3 *Photograph of subject's left ankle 3 days post injury. The arrow indicates a large blister on lateral aspect of ankle.*

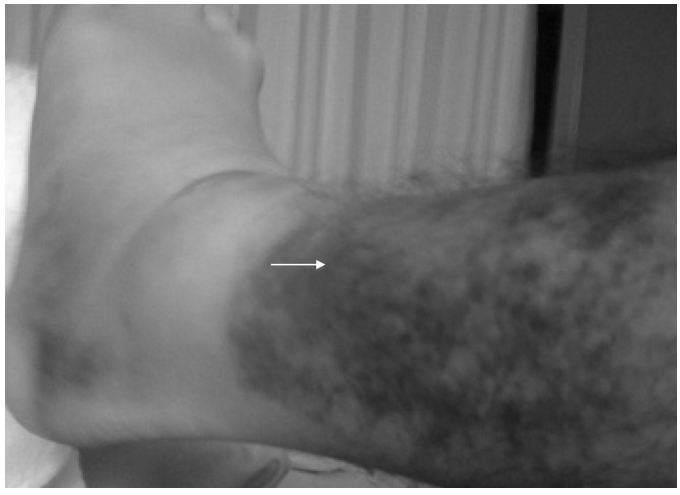


Figure 4 *Photograph of subject's left ankle 5 days post injury. The arrow indicates severe bruising on the lateral aspect of left leg.*

or rehabilitation at one-year post injury. This patient successfully returned to recreational sport participation with a left ankle brace for stability at one-year post injury.

Discussion

Ankles are one of the most traumatized sites in the body with respect to sports injuries and account for 10–30% of all sports injuries.[9] Seventy-six (76%) of all injuries that occur in soccer are related to the ankle joint with sprains accounting for over 80% with 77% involving the lateral aspect of the ankle joint and 73% involving rupture to the anterior talofibular ligament specifically.[9,10,11]

We present a case of a patient who was given a presumptive diagnosis of severe grade three-inversion ankle sprain after a thorough history of the mechanism of in-

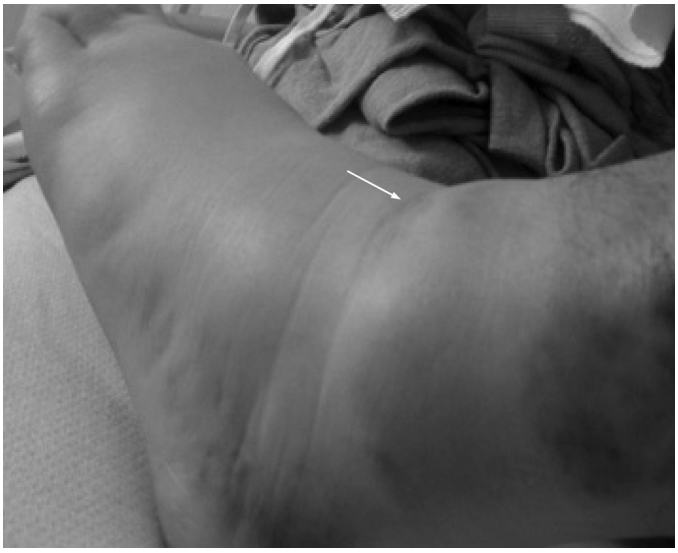


Figure 5 *Photograph of subject's left ankle 7 days post injury. The arrow indicates severe swelling surrounding ankle and dorsum of left foot.*

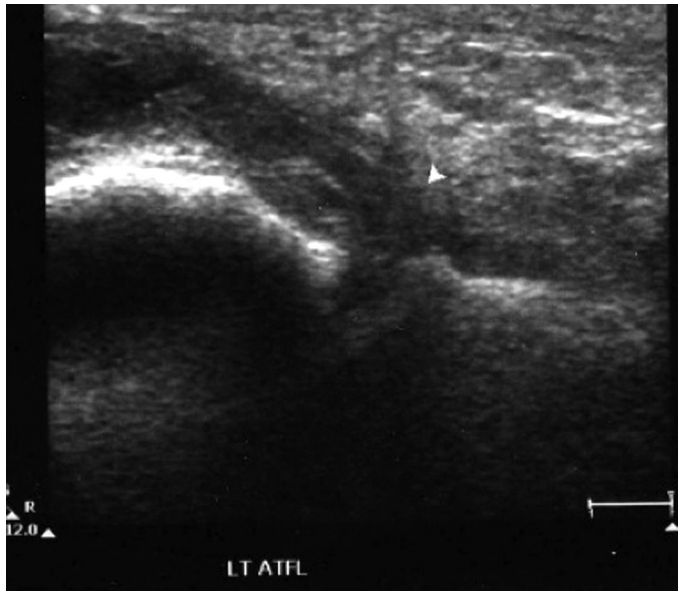


Figure 6 *Diagnostic ultrasound of lateral aspect of left ankle. The arrowhead indicates the left ATFL is not intact.*

jury and complete physical examination of the ankle and foot. Plain film radiographs confirmed the diagnosis. The patient may have sustained a subtalar dislocation with spontaneous relocation; however, there were no findings present clinically or radiographically of such an event.[12,13] Deteriorating neurological signs over a nine-hour period lead the surgeon to suspect ACS of the foot.

In this case ACS was a complication of a severe grade three-inversion ankle sprain. ACS typically occurs after a traumatic event such as a fracture, soft tissue injury, or vascular trauma.[6,8,14,15,16,17] It can be a devastating medical emergency that occurs from an increase in ICP within a closed osteofascial space. The circulation and viability of the tissues within the compartment are compromised due to an initial traumatic or haemorrhagic injury.[8] Hypoperfusion occurs to the tissues in response to the increased ICP leading to tissue ischemia and necrosis of muscles and nerves in the compartment.[7,8,14,18] Specifically, ACS occurs when the perfusion pressure of one of the osteofascial compartments falls below the tissue pressure.[7,8,14,18] Increased volume within a tissue compartment is usually caused by bleeding due to injury but can be caused by a coagulopathy, a disorder of blood coagulation.[7,8,19] This patient presented with severe pain and allodynia in the foot due to hypoperfusion of the tissue causing tissue ischemia. Further, the paresthesia that followed was due to an increase in volume within the foot compartment leading to an increased ICP causing necrosis of nerves and soft tissues within the compartment.

The ischemia caused by ACS causes perifascicular and intrafascicular edema.[8] The end result of ACS without surgical intervention includes severe neurological deficits, ischemic contracture, gangrene, and tissue necrosis leading to amputation.[6,7] Therefore, early diagnosis is extremely important and requires a high index of suspicion during evaluation of the patient.[7,14,15] The initial symptomatology of this case may have contributed to confusion resulting in an accurate but non-comprehensive diagnosis of acute grade three-inversion ankle sprain. The initial symptomatology and a low index of suspicion also may have resulted in the delayed diagnosis of ACS of the foot. This case emphasizes the importance of a high index of suspicion when assessing an ankle sprain with pain out of proportion from the initial injury with concomitant paresthesia in the foot.

ACS occurring in the four compartments of the lower leg has been well reported within the current literature.[8,17,18,20] There are currently nine specific compart-

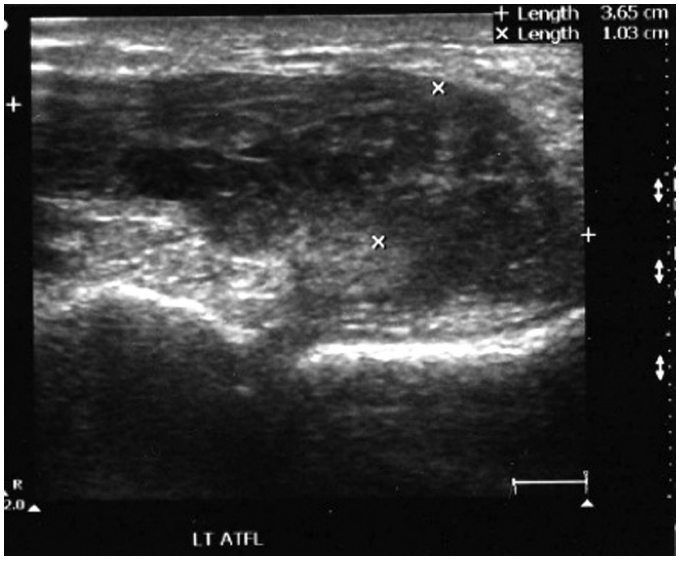


Figure 7 *Diagnostic ultrasound of lateral aspect of left ankle. The 2 x's show a large hematoma over the left lateral malleolus and a complete avulsion of AFTL.*

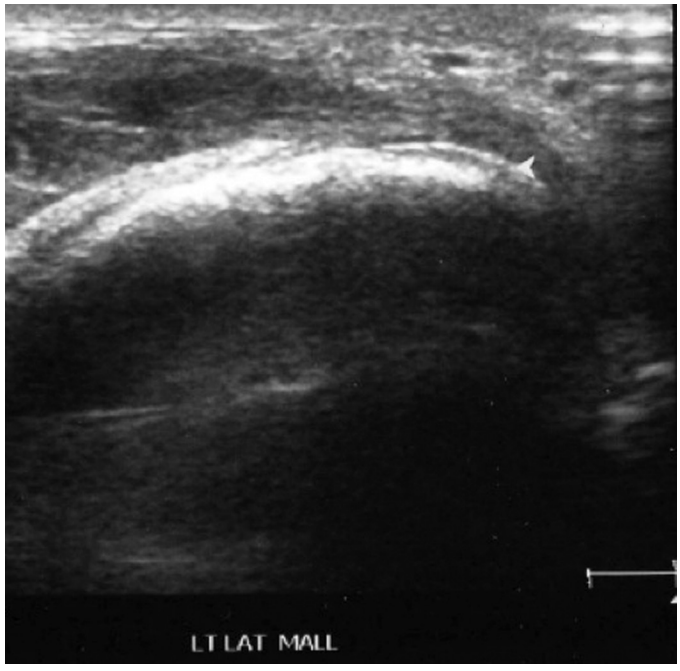


Figure 8 *Diagnostic ultrasound of lateral aspect of left ankle. The arrowhead shows fragmentation of the left lateral malleollus.*

ments within the foot that have been described: medial, lateral, superficial, adductor, four interossei, and calcaneal[7,16,17,21,22] (see Table 3 and Figure 9).

The signs and symptoms of ACS of the foot are subtle and hard to distinguish from the primary foot injury. Particular attention to the mechanism of injury, the amount of energy involved with the injury, the presence of trauma, and coagulopathies are important.[7,16]

The clinical diagnosis of ACS is made based on a constellation of physical signs and symptoms; increased pain out of proportion of original injury, pallor, paresthesia, paralysis, and pressure[7,15,16] (see Table 4). ACS may present as overt pain that is particularly accentuated by active and passive range of motion of the ankle joint, forefoot, or toes, sensory deficits in the specific compartment and a significant amount of swelling.[14,18] Pulselessness is uncommon and only seen in the late stages of ACS.[7] Pulselessness is usually caused by a vascular injury because the compartment pressure does not reach systolic blood pressure allowing blood flow.[8,17] If pulses are present palpation of the dorsal artery of the foot or the posterior tibial artery may be falsely reassuring as pulses can persist with a compartment pressure of >80 mmHg.[15] The examination of pulses is unreliable in the diagnosis of ACS since ICP does not reach systolic pressure.[17] Necrosis of the soft tissues of the foot may appear with compartment pressures ranging from 30–60 mmHg.[15]

Severe and spontaneous pain has been identified as the earliest and most sensitive clinical sign that manifests with ACS of the foot.[23,24,25] No evidence based recommendations can be made as to how to manage ACS, however, it has been suggested that serial examination of motor and sensation of the foot should be done at least every hour, since muscle necrosis can occur within 4 hours.[26]

Most often the clinical diagnosis of ACS is confirmed by measurement of the ICP. In many cases, the direct measurement of ICP is not warranted if the classic signs and symptoms of ACS are clinically evident and the patient is conscious.[4,6] ICP monitoring may not show any differences in outcome or time to surgery when compared to serial monitoring.[27] In our patient the ICP was not measured due to convincing signs and symptoms of ACS at approximately nine hours post-injury.

The use of platelet inhibitors (ASA) and calcium channel blockers (verapamil) are extremely common today.[28]

Table 3 *Compartments of the foot*[7,16,17,22,23]

Compartments	Muscles	Vessels	Nerves
Medial	1. Flexor hallucis brevis 2. Abductor hallucis	n/a	n/a
Lateral	1. Abductor digiti quinti 2. Flexor digiti minimi	n/a	n/a
Superficial	1. Flexor digitorum brevis 2. Lumbricals (4) 3. Flexor digitorum longus tendons	n/a	1. +/– Medial plantar nerve
Interosseus (×4)	1. Interossei	n/a	n/a
Adductor	1. Adductor	n/a	n/a
Calcaneal	1. Quadratus plantae	1. Posterior tibial artery 2. Posterior tibial vein 3. Lateral plantar artery 4. Lateral plantar vein	1. Posterior tibial nerve 2. Lateral plantar nerve 3. +/– Medial plantar nerve

Figure 9 *The compartments of the foot*[33] *(Reprinted with permission from Mubarak SJ and Hargens AR: Compartment Syndromes and Volkman's Contracture, p. 45. Philadelphia, W. B. Saunders, 1981 and Bonutti PM and Bell GR Compartment syndrome of the foot. A case report. JBJS. 1986; 68:1449–1451)*

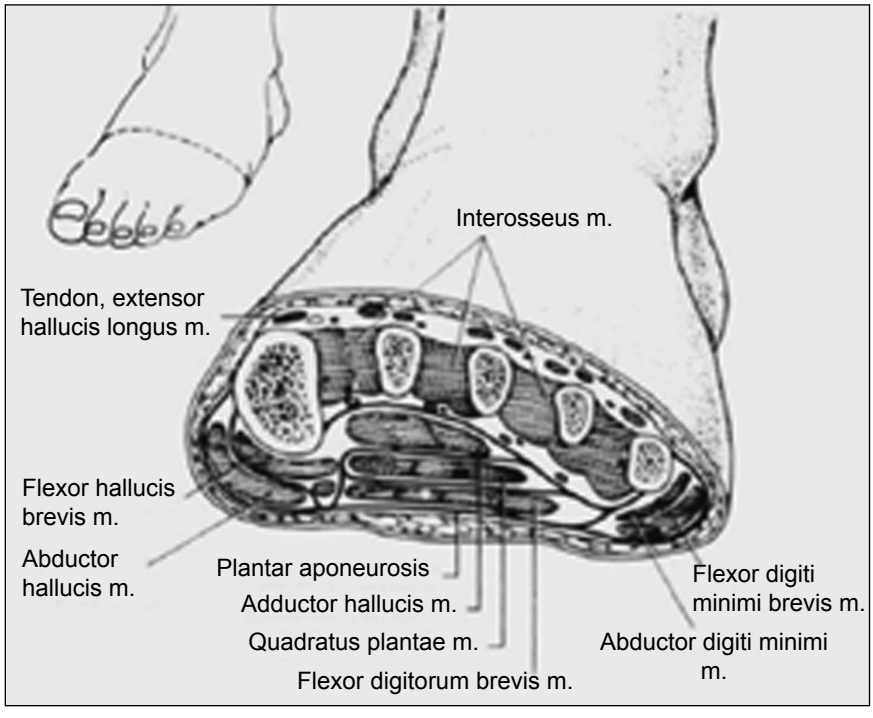


Table 4 *Signs and Symptoms of ACS*[7,8,15,16]

ACS of the foot
Severe pain that is out of proportion to the apparent injury
Pain provoked by passive range of motion of the foot
Severe swelling in the specific compartment
Progression of pain and swelling over a short period of time
Paraesthesia
Pallor of the surrounding cutaneous skin
Pulselessness (only occurs in the later stages)
Paralysis of the affected foot (only occurs in the later stages)

However, the frequency of these medications causing severe peripheral edema causing ACS seems particularly rare. ASA is used as a platelet inhibitor and verapamil is traditionally used as a vasodilator in the treatment of hypertension, angina pectoris, and cardiac arrhythmias and is an L-type calcium channel blocker of the Phenylalkylamine class. Verapamil may cause a decrease in arteriolar resistance with no change in venous circulation. This difference causes a change in the peripheral resistance causing an increase in hydrostatic pressure in the precapillary circulation.[29] When this occurs the vessels permit a fluid shift into the interstitial compartment.[29] Further, ASA causes anti-platelet aggregation leading to a decreased clotting time. These two distinct components lead to an increase in the peripheral perfusion to the injured area, which may cause the likelihood of ACS to increase exponentially. We hypothesize that the grade three-inversion ankle sprain combined with these medications may have increased the amount of swelling into the foot resulting in severe pain and paresthesia and ultimately leading to an increased ICP.

Upon review of the literature there are only three other case reports that are similar.[30–32] Beall et al. described a case of an 18 year-old male football player with ACS of the anterolateral aspect of the leg associated with the intake of platelet inhibitors used for an inherited platelet defect.[30] Compartment pressure measurements of the anterolateral compartment of the leg was taken with a slit catheter and revealed a pressure of 40 mmHg. Decompression of the anterolateral compartment of the leg was performed by a fasciotomy. No neurological sequelae followed due to the immediate decompression.[30]

Secondly, Rancan et al. described a case of a 57-year old male with ACS of the anterior aspect of the leg caused by intramuscular hemorrhage of the anterior tibialis muscle following a low energy, non-contact injury.[31] The patient was on a daily regimen of platelet inhibitors (75 mg ASA daily). The clinical diagnosis of ACS was confirmed in this case with a handheld ICP monitoring system. The ICP was measured as 95 mmHg and immediate surgical decompression of the anterior and lateral compartments were performed. A six-week follow-up was performed and no neurological deficits were found and there was full function of the knee and ankle joints.[31]

A third case report by Dhawan et al. described a case of 35-year old male who sustained a severe inversion ankle sprain while playing basketball.[32] The patient was initially prescribed a treatment at home of rest, ice, elevation, and compression. Examination of the foot 26 hours after injury revealed a swollen foot and ankle with severe blistering. Posterior tibial and dorsalis pedis pulses were not palpable; capillary refill was brisk in all toes. No fracture was diagnosed on plain film radiographs. The patient's dorsal compartment pressures were measured at 120 mmHg, confirming a diagnosis of ACS of the foot and a disruption of the anterior tibial artery. A seventy-seven month follow-up was performed and no neurological deficits were found.[32]

For patients who sustain a high-energy deceleration injury resulting in pain, severe swelling, and paresthesia ACS should be suspected. ICP measurement may be considered if the patient is unconscious and the signs and symptoms of ACS are not evident.[6,7] Patients with a measured ICP of 30 mmHg are recommended to undergo an immediate fasciotomy to relieve the pressure in the tissue compartment.[6] Normal ICP in the foot is approximately 4.7–6 mmHg and in the lower leg 0–8 mmHg.[6,8] In patients presenting with ACS, pain and paresthesia first appears at an ICP between 15–30 mmHg. Therefore, many surgeons will perform fasciotomies of the affected compartment at 30 mmHg.[6,8] Irreversible changes occur to soft tissues and neurological structures with an ICP of 30 mmHg for durations of 4 hours or longer.[6,8,27] The highest compartment pressures are often found 12–36

hours post injury.[27] In this patient a fasciotomy was considered but not performed resulting in sensory deficit at a one-year follow-up. A fasciotomy was not performed due to a recent outbreak of methicillin-resistant staphylococcus aureus in the hospital that may have resulted in a serious life threatening infection. Decompression surgery in this case would have occurred if the patient's neurological signs and symptoms deteriorated further from initial presentation.

Summary

ACS is often not suspected in common ankle injuries. We report a case of a 28-year old recreational soccer player who was eventually diagnosed with ACS of the foot. Although ACS is usually diagnosed with an ICP measurement, this case represents a diagnosis of ACS of the foot without ICP measurement. This case highlights the necessity for all primary health care practitioners responsible for diagnosing and treating ankle sprains to be aware of the potential complication of ACS of the lower limb or foot. A delayed diagnosis and inappropriate management may lead to devastating neurological sequelae and even amputation of the affected limb, especially in patients presenting with early signs and symptoms and those on antiplatelet therapy or calcium channel blockers[6]. ACS may have been exacerbated by the use of ASA and Verapamil pharmacotherapy in this case.

References

1 Garrick JG. The frequency of injury, mechanism of injury, and epidemiology of ankle sprains. Am J Sports Med. 1977; 5:241–242.
2 Pihlajamaki H, Hietaniemi K, Paavola M, Visuri T, Mattila VM. Surgical versus functional treatment for acute ruptures of the lateral ligament complex of the ankle in young men: A randomized controlled trial. J Bone Joint Surg Am. 2010; 92:2367–74.
3 Schmikli SL, de Vries WR, Inklaar H, Backx FJG. Injury prevention target groups in soccer: Injury characteristics and incidence rates in male junior and senior players. J Sci Med Sport. 2010, doi:10.1016/j.jsams.2010.10.688
4 Waterman BR, Owens BD, Davey S, Zacchilli MA, Belmont PJ. The epidemiology of ankle sprains in the United States. J Bone Joint Surg Am. 2010; 92:2279–84.
5 Lynch SA, Renstrom PA. Treatment of acute lateral ankle ligament rupture in the athlete. Conservative versus surgical treatment. Sports Med. 1999; 27:61–71.
6 Kostler W, Strohm PC, Sudkamp NP. Acute compartment syndrome of the limb. Injury, Int. J Care Injured. 2004; 35:1221–1227.
7 Malik AA, Khan WSA, Chaudhry A, Ihsan M, Cullen NP. Acute compartment syndrome – a life and limb threatening surgical emergency. J Preop Practice. 2009; 19(5):137–142.
8 Tiwari, A, Haq AI, Myint F, Hamilton G. Review: Acute compartment syndrome. Br J Surg. 2002; 89:397–412.
9 Tik-Pui Fong D, Hong Y, Chan L, Shu-Hang Yung P, Chan K. A systematic review on ankle injury and ankle sprain in sports. Sports Med. 2007; 37(1):73–94.
10 Garrick JG, Requa RK. The epidemiology of foot and ankle injuries in sports. Clin Sports Med. 1988; 7(1): 29–36.
11 Gerber JP, Williams GN, Scoville CR, Arciero RA, Taylor DC. Persistent disability associated with ankle sprains: a prospective examination of an athletic population. Foot Ankle Int. 1998; 19(10):653–60.
12 DeLee JC, Curtis R. Subtalar dislocation of the foot. J Bone Joint Surg Am. 1982; 64:433–437.
13 Zimmer TJ, Johnson KA. Subtalar dislocations. Clin Orthop. 1989; 238:190–4.
14 Kosir R, Moore FA, Selby JH, Cocanour CS, Kozar RA, Gonzalez EA, Todd SR. Acute lower extremity compartment syndrome (ALECS) screening protocol in critically ill trauma patients. J Trauma. 2007; 63:268–275.
15 Elliott KGB, Johnstone AJ. Diagnosing acute compartment syndrome. J Bone Joint Surg [Br]. 2003; 85:625–632.
16 Fulkerson E, Razi A, Tejwani N. Review: Acute compartment syndrome of the foot. Foot & Ankle International. 2003; 24(2):180–187.
17 Frink M, Hildebrand F, Krettek C, Brand J, Hankemeier S. Compartment syndrome of the lower leg and foot. Clin Orthop Relat Res. 2010; 468:940–950.
18 Shadgan B, Menon M, O'Brien P, Reid D. Diagnostic techniques in acute compartment syndrome of the leg. J Orthop Trauma. 2008; 22:581–587.
19 Roberge RJ, McLane M. Compartment syndrome after simple venuuncture in an anticoagulated patient. J Emerg Med. 1999; 17:647–649.
20 Kashuk JL, Moore EE, Pinski S, Johnson JL, Moore JB, Morgan S, Cothren CC, Smith, W. Lower extremity compartment syndrome in the acute care surgery paradigm: safety lessons learned. Patient Safety in Surgery. 2009; 3(11):1–6.
21 Perry MD, Manoli A. Foot compartment syndrome. Ortho Clin North America. 2001; 32(1):103–111.
22 Richter J, Schulze W, Klaas A, Clasbrummel B, Muhr G. Compartment syndrome of the foot: an experimental approach to pressure measurement and release. Arch Orthop Trauma Surg. 2008; 128:199–204.
23 Myerson M. Diagnosis and treatment of compartment syndrome of the foot. Orthopedics. 1990;13:711–717.

24 Myerson MS. Management of compartment syndromes of the foot. Clin Orthop Relat Res. 1991; 239–248.
25 Ulmer T. The clinical diagnosis of compartment syndrome of the lower leg: are clinical findings predictive of the disorder? J Orthop Trauma. 2002; 16:572–577.
26 Blaisdell FW. The Pathophysiology of skeletal muscle ischemia and the reperfusion syndrome: a review. Cardiovascular Surgery. 2002; 10(6):620–630.
27 Al Dadah OQ, Darrah C, Cooper A, Donell ST, Patel AD. Continuous compartment pressure monitoring vs. clinical monitoring in tibial diaphyseal fractures. Injury. 2008; 39:1204–1209.
28 Kirshner HS, Biller J, Callaban AS. Long-Term Therapy to Prevent Stroke. J Am Board Fam Pract. 2005; 18:528–40.
29 Sica DA. Calcium channel blocker-related peripheral edema: Can it be resolved? J Clin Hypertens. 2002; 5:291–297.
30 Beall S, Garner J, Oxley D. Anterolateral compartment syndrome related to drug-induced bleeding: A case report. Am J Sports Med. 2003; 11:454–455.
31 Rancan M, Esser MP, Kossmann T, Cameron P, Fitzgerald M. Acute compartment syndrome following low energy non-contact injury. J Surg. 2004; 11:1023–1025.
32 Dhawan A, Doukas WC. Acute compartment syndrome of the foot following an inversion injury of the ankle with disruption of the anterior tibial artery. J Bone Joint Surg Am. 2003; 85:528–532.
33 Bonutti PM, Bell GR. Compartment syndrome of the foot. A care report. J Bone Joint Surg Am. 1986; 68:1449–1451.

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Growth Restart / Recovery Lines involving the vertebral body: a rare, incidental finding and diagnostic challenge in two patients

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Michelle Wessely, BSc, DC, DACBR, FCC (UK), DipMEd§

Objective: *To present the phenomenon of growth restart lines and create awareness of the possible differential diagnoses.*

Clinical Features: *Two case reports outlining the presentation of growth restart lines found in the vertebrae of trampolinists. Emphasis in each case is placed on correlating the patient history with radiographic findings.*

Intervention and Outcome: *In both cases a conservative chiropractic treatment plan was initiated once the differential diagnoses could be ruled out.*

Conclusion: *Although the range of etiologies of growth restart lines is extensive, these case reports illustrate the importance of a comprehensive case history when presented with the radiographic finding of growth restart lines.*
(JCCA 2011; 55(4):313–317)

KEY WORDS: growth, restart line, vertebrae

Objectif : *présenter le phénomène des lignes de reprise de la croissance et faire connaître les diagnostics différentiels possibles.*

Caractéristiques cliniques : *deux rapports de cas mettant en relief la présentation des lignes de reprise de la croissance dans les vertèbres de trampolinistes. Dans les deux cas, nous mettons l'accent sur la corrélation entre les antécédents du patient et les résultats des radiographies.*

Intervention et résultat : *dans les deux cas, un traitement chiropratique conservateur fut mis en œuvre lorsque la possibilité de diagnostics différentiels fut éliminée.*

Conclusion : *bien que l'éventail des étiologies des lignes de reprise de croissance était vaste, ces cas démontrent l'importance des antécédents détaillés lorsque les résultats des radiographies portant sur les lignes de reprise de croissance sont connus.*
(JCCA 2011; 55(4):313–317)

MOTS CLÉS : croissance, ligne de reprise, vertèbres

Introduction

Growth restart lines are generally encountered in the ends of the bones of the lower limb that grow most rapidly, especially about the knee.[1] Although less common, other regions of the skeleton may be affected, particularly the spine. Growth restart lines (also known as Harris lines or growth arrest lines) are typically formed in the long bones when the trabeculae become dense and horizontally oriented to the long axis of the bone, resulting in the radiographical feature of lines. These restart lines may be due to a variety of disorders affecting skeletal development such as severe childhood infection, leukemia, malnutrition, immobilization, and medication use.[2–7] Interestingly, their identification has been used by paleontologists and anthropologists as a means of determining the health of ancient populations.[8,9] It is important to recognize and identify growth restart lines in order to determine their origin and to be able to differentiate growth restart lines

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from the reinforcement lines encountered in patients with reduced bone quantity.[10] This report presents two cases to illustrate the importance of a comprehensive case history along with imaging findings to aid with the determination of the etiology of growth restart lines.

Case One

History

A sixteen-year-old male elite trampolinist presented to a chiropractic clinic with neck pain which began immediately after falling from a trampoline during a practice where the athlete landed on the right side of his neck and right shoulder. At the time, he saw his family physician and was referred for cervical and thoracic spine radiographs, which were interpreted as unremarkable. The physician subsequently prescribed anti-inflammatory medications and advised him to rest for two weeks. With continued pain and restriction of neck movement one week after the fall, the athlete presented to a chiropractor.

The patient indicated that the pain was constant and the intensity was rated verbally as six out of ten, where zero equals no pain at all and a ten rating is the most pain the individual has ever experienced in his lifetime. The patient localized the pain to the cervicothoracic junction and reported that moving his head to the right and upwards aggravated the pain, whereas stretching provided some temporary relief. The patient had no previous history of neck pain but reported a history of recurrent low back pain. The patient denied having any previous childhood infections, malnutrition, immobilization, or prescription medication use. Further investigation into his past sporting activities revealed a history of competitive gymnastics that involved three hours of practice each day for six days per week since he was six years old. The athlete ceased competitive gymnastics at the age of nine to train for the trampoline competitively. At the time of his injury, his time commitment to trampoline training was approximately four hours per day, five days per week.

Physical Examination

Upon examination of the head and neck regions, no obvious deformities, effusion or muscle atrophy were noted. His cervical spine active ranges of motion (ROM) were all within normal limits except for right rotation, which was 45 degrees, extension was 30 degrees and right lateral flexion was 15 degrees. Passive ROM was five degrees greater in each direction. Resisted cervical spine flexion, extension, bilateral lateral flexion and rotation were all graded as 4/5 as the patient was unable to achieve full muscle strength due to reported pain during this procedure. A neurological sensory examination of the upper limbs for light touch, vibration, pain and crude touch was unremarkable. Deep tendon reflexes (DTR) at C5, C6, and C7 were graded 2+ bilaterally (normal). Motor testing for the upper limb myotomes of C5 to T1 were graded 5/5 in all directions, where a 5/5 is indicative of the client being able to full resist the practitioner's applied force. Orthopedic testing was not reliable due to the prominent muscular spasm in the cervico-thoracic spinal region. Aberrant motions with reported tenderness were identified through motion and static palpation in the right C7-T1 and T3-4 facet joints. The patient's right posterior scalene, upper trapezius, levator scapulae and rhomboid major/minor were mildly hypertonic and tender upon palpation.

Diagnostic Imaging

Cervical and thoracic spine radiographs requisitioned by the patient's medical physician were interpreted by the radiologist as unremarkable for fractures and dislocations. However these radiographs did reveal multiple linear growth recovery lines most prominent in the thoracic region, and less so in the cervical region (please see Figure 1).

Diagnosis

The patient was diagnosed by his chiropractor with acute mechanical cervicothoracic facet joint dysfunction with associated myofascial pain.

Plan of Management

The initial plan of management consisted of interferential current therapy (on a continuous bipolar setting at 120 Hz for pain relief), Active Release Techniques® to the affected musculature, and spinal manipulative therapy of the cervical and thoracic spine. The patient was seen three times per week for two weeks, followed by two times per week for one week. The patient was seen twice more over the next two weeks and at the end of this treatment plan the patient's cervical spine active, passive and resisted ROM were all within normal limits and reported as pain free.

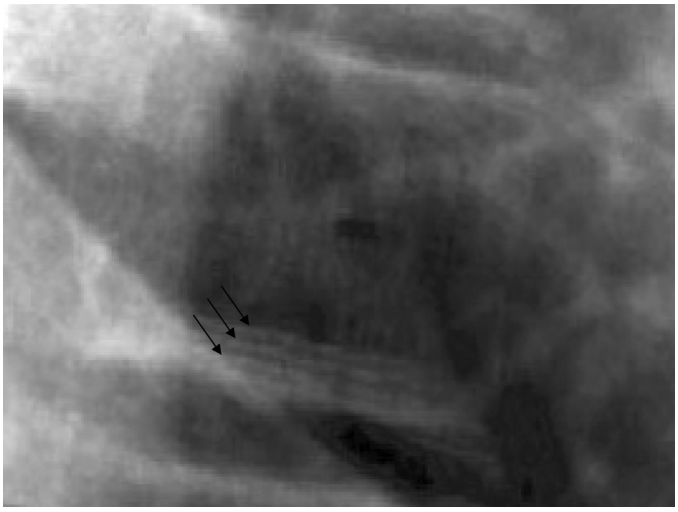

Figure 1 *Lateral thoracic spine view demonstrating growth restart lines (indicated by arrows).*

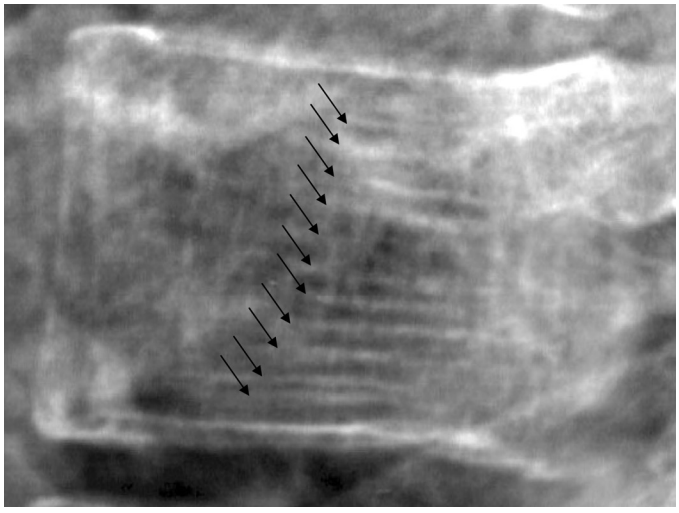

Figure 2 *Lateral lumbar spot view demonstrating growth restart lines (arrows).*

Case Two

History

A 27-year-old flight attendant presented to the chiropractor with a three-month history of low back pain that began when his flying hours were increased. He initially consulted the airline's occupational health physician who diagnosed him with a "lower back sprain". Due to the continuous pain the patient subsequently consulted his family physician who examined him and prescribed a seven-day course of non-steroidal anti-inflammatory medication.

The chiropractic history examination revealed the patient had a history of recurrent lower back pain as a young gymnast and that injury previously responded to conservative care. The current pain was localized to the thoracolumbar region without radiation to the buttocks or leg and was not associated with any sensory or neurological deficits. The patient reported that the pain was a consistent achy sensation and was uncertain of any aggravating or relieving factors. The patient reported that his annual airline medical examination included a full blood count, which had been reported as negative the previous month.

Physical Examination

The physical examination revealed that the patient's lumbar ranges of motion were within normal limits although he reported pain at the end-range of passive extension. All orthopedic and neurological testing was unremarkable. Motion and static palpation revealed segmental restrictions at the thoracolumbar junction. Muscle palpation revealed a mildly hypertonic quadratus lumborum bilaterally. Subsequently, the chiropractor determined that radiographs were required prior to commencing spinal manipulation due to patient's prior history of lower back pain and to rule out a spondylolysis owing to his high level of involvement in gymnastics.

Diagnostic Imaging

Radiographs of the thoracolumbar spine region demonstrated the presence of multiple radio-opaque horizontal lines along the vertebral bodies of both the lower thoracic and most of the lumbar spinal segments (please see Figure 2). The remaining alignment, bone, articulations and soft tissue structures were within normal limits.

Differential Diagnosis

The medical radiologist discussed the imaging findings with the patient and offered the diagnosis of leukemia as the explanation for the radio-opaque lines in the noted vertebral bodies. The patient returned to his family physician for further blood tests. To follow up the chiropractor consulted a chiropractic radiologist (MW) for additional imaging interpretation. Following analysis of the multiple radio-opaque linear lines, a more in-depth health his-

tory was performed and the patient reported that during his adolescence a relative had systematically physically abused him and that during these periods he suffered from mild bouts of anorexia nervosa. In addition, between the ages of 13 and 16 years of age he was in the national training program for trampolining, which consisted of two to three hours of daily training. This additional clinical information and the fact that repeated blood tests were unremarkable assisted in determining the most likely explanation that the presence of the linear radio-opaque lines was benign in nature and not due to leukemia as otherwise suggested.

Management

The patient received chiropractic treatment, which included spinal manipulation directed towards the thoracolumbar segmental dysfunction, along with soft tissue therapy for quadrates lumborum myofascial pain syndrome. At the patient's request he was referred to a counselor regarding his past physical abuse. The response to chiropractic treatment was rapid, with cessation of all pain after two chiropractic treatments.

Table 1 *Differential diagnoses for growth restart lines.*

Osteopetrosis
Hyper/Hypoparathyroidism
Sclerosing Spondylosis
Radiation Exposure
Cushing's Syndrome
Rickets (Healing)
Avascular Necrosis
Osteoporosis
Congenital Syphilis
Paget's Disease
Leukemia
Nutritional Deficiency
Scurvy
Fractures (Healing)
Childhood Stress
Infection

Discussion

Historically, conventional radiography has been an integral part of chiropractic practice to rule out specific sources of pain, such as infection, malignancy, fracture and dislocation. Abnormal findings apparent on radiographs are not overly common, with reported rates between one in 100 to one in 2500.[11–13] However, if such pathologies are missed the consequences could be severe for both the patient and the practitioner. The cases presented illustrate not only the importance of identifying growth restart lines on radiographs but also linking these findings with the clinical history.

Numerous authors have described the morphologic characteristics of growth restart lines and a general consensus has not been agreed upon.[2–5,13,14] The lines are more appropriately referred to as growth restart lines or recovery lines rather than the previously preferred term of growth arrest lines. This change in bony appearance on conventional radiography is due to the osteoblasts forming a thin transverse bone layer below the zone of provisional calcification during the time when growth has resumed after a period of growth cessation. When growth is re-established, cartilaginous proliferation and increased osteoblastic activity contribute to the thickening and metaphyseal migration of the transverse line.[14,15]

In the spine the restart lines typically parallel the vertebral endplates and occasionally form a bone within a bone (endobone) appearance when a remnant of the anterior vertebral surface persists. The appearance is similar to those seen in some patients with osteopetrosis or heavy metal poisoning.[16] As a clinician it is imperative to distinguish whether these lines may have developed as a result of repetitive trauma or from other pathologies such as infection,[2] malnutrition,[2,21] immobilization, [4,8] hypoparathyroidism,[16] alcohol consumption during growth,[9] juvenile chronic arthritis,[18] psychosocial short stature,[19] endemic skeletal fluorosis,[20] bisphosponate medication,[21] heavy metal poisoning[13] or leukemia.[5,23] (Please see Table 1 for a detailed list of differential diagnoses)

The cases presented above demonstrate that it can be difficult to solely attribute the presence of growth restart/recovery lines to one common factor but the authors postulate that repetitive axial compression activities such as in trampoline and gymnastics may contribute to these radiological findings. These cases also emphasize the im-

portance of conducting a comprehensive history and performing the appropriate investigative tests to render this diagnosis by exclusion.

Conclusion

The cases presented in this report illustrate an incidental radiological finding of growth restart/recovery lines in two trampoline athletes. It is the authors' opinion that health care practitioners should be cognizant of restart/recovery lines and should take every precaution not to misdiagnose or misinterpret these radiological findings as one of the more serious disorders with a similar radiographic appearance. Lastly, it is clear from the limited research in this area that further studies are warranted, in particular investigations into the hypothetical contribution of sporting activities that involve repetitive axial loading movements and their role in the possible cause of these radiological findings.

References

1 Siffert RS, Katz JF. Growth recovery zones. J Pediatr Orthoped. 1983; 3:196–201.
2 Park EA. The imprinting of nutritional disturbances on growing bone. J Pediatr. 1964; 33:815–862.
3 Silverman FN. Variants due to diseases of bone. In: Caffey's Pediatric X-ray Diagnosis: An Integrated Imaging Approach 9th Edition. Philadelphia: Mosby, 1994; 1521–1527.
4 Bar-On E, Beckwith JB, Odom LF, Eilert RE. The effect of chemotherapy on human growth plate. J Pediatr Orthop. 1993; 13(2):220–224.
5 Schwartz AM, Leonidas JC. Methotrexate osteopathy. Skeletal Radio. 1984; 11:13–16.
6 Peters W, Irving J, Letts M. Long-term effect of neonatal bone and joint infection on the adjacent growth plate. J Paediatr Orthopaed. 1992; 12(6):806–810.
7 Rosen RA, Desmukh SM. Growth arrest recovery lines in hypoparathyroidism. Radiology. 1985: 155(1):61–62.
8 Yao L, Seeger LL. Epiphyseal growth arrest lines: MR findings. Clin Imag. 1997; 21:237–240.
9 Gonzalez-Reimers E, Perez Ramirez A, Santolaria-Fernandex F, Rodriguez-Rodriguez E, Martinez-Riera A, del Carmen Duran-Castellon M, Alemain-Valls MR, Gaspar MR. Association of Harris lines and shorter stature with ethanol consumption during growth. Alcohol. 2007; 41:511–515.
10 Kursunoglu S, Pate D, Resnick D, Haghighi P, Tyson R, Pitt M. Bone reinforcement lines in chronic adult osteopenia: A hypothesis. Radiology, 1986; 158:409–415.
11 Brekkan A. Radiographic examination of the lumbosacral spine: An "age-stratified" study. Clinical Radiology. 1983; 34: 321–324.
12 Nachemson AL. The lumbar spine: An orthopaedic challenge. Spine. 1976; 1:59–71.
13 Resnick D. Diagnosis of Bone and Joint Disorders 4th Edition. W.B. Saunders Company, 2002; 2397–2401.
14 Frager DH, Subbarao K. The bone within a bone. JAMA. 1983; 249:77–79.
15 Nowak O, Piontek J. The frequency of appearance of transverse (Harris) lines in the tibia in relationship to age at death. Ann of Hum Bio. 2002; 29(3):314–325.
16 Rosen RA, Deshmukh SM. Growth arrest recovery lines in hypoparathyroidism. Radiology. 1985; 155:61–62.
17 Mays S. The relationship between Harris lines and other aspects of skeletal development in adults and juveniles. J Archaeol Sci. 1985; 22:511–520.
18 Fiszman P, Ansell BM, Renton P. Radiological assessment of knees in juvenile chronic arthritis (juvenile rheumatoid arthritis). Scand J Rheumatol. 1981; 10:145–152.
19 Khadilkar VV, Frazer FL, Skuse DH, Stanhope R. Metaphyseal growth arrest lines in psychosocial short stature. Arch Dis Child. 1998; 79:260–262.
20 Wang Y, Yin Y, Gilula LA, Wilson AJ. Endemic fluorosis of the skeleton: Radiographic features in 127 patients. AJR Am J Roentgenol. 1994; 162:93–98.
21 Hong IK, Suh JS, Lee YA, Kim DY. Scintigraphic findings of growth arrest lines after bisphosphonate administration in a steroid-induced osteoporosis patient. Clin Nuc Med. 2010; 35(9):740–742.
22 Hummert JR, Van Gerven DP. Observations on the formation and persistence of radiopaque transverse lines. Am J Phys Antropol. 1985; 66:297–306.
23 Meister B, Gassner I, Streif W, Dengg K, Fink FM. Methotrexate osteopathy in infants with tumors of the central nervous system. Med Pediatr Oncol. 1994; 23(6):493–496.

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Weight cycling in adolescent Taekwondo athletes

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Background: *Weight reduction cycles are used by weight classed athletes in Taekwondo to make a weight category. Tension, dizziness, headaches, and confusion have been associated with rapid weight loss (RWL). There is a lack of research in weight cycling and its benefits among Taekwondo athletes.*

Purpose: *To investigate the rate of weight cycling in Junior Taekwondo athletes and its effect on performance.*

Methods: *Athletes were weighed prior to competition, then again before their first match. Body mass difference in relation to winning was compared.*

Results: *A significant increase from weigh-in to pre-match measurements was consistently found in both genders with no significant difference between them. Winners had a mean body mass gain (1.02 kg) which was non-significantly less than the non-winners (1.09 kg).*

Conclusions: *RWL practices do not define which athlete will perform better. Negative effects of weight cycling coupled with RWL has unclear performance benefits which indicates a need for further research.*
(JCCA 2011; 55(4):318–324)

KEY WORDS: competitive behaviour, martial arts, Taekwondo, weight loss

Information de base : *les cycles de perte de poids sont utilisés par les athlètes pratiquant le taekwondo afin de se conformer à une catégorie de poids. La tension, les étourdissements, les maux de tête et la confusion ont été associés à la perte de poids rapide. Il existe peu de recherches concernant le cycle de poids et ses avantages chez les athlètes pratiquant le taekwondo.*

Objet : *enquêter sur le cycle de poids des athlètes d'âge junior pratiquant le taekwondo et ses conséquences sur leur rendement.*

Méthodes : *les athlètes étaient pesés avant une compétition, puis encore une fois avant leur premier combat. La différence de poids fut comparée aux victoires remportées.*

Résultats : *une importante hausse de poids entre la pesée et le début d'un combat fut constatée chez les deux sexes, sans différence significative entre ceux-ci. Les gagants avait une masse corporelle moyenne supérieure de 1,02 kg, alors que cette hausse était de 1,09 kg chez les perdants.*

Conclusions : *la perte de poids rapide ne peut déterminer quels athlètes performeront le mieux. Les conséquences négatives du cycle de poids, jumelées à la perte de poids rapide doivent être analysées plus amplement avant de déterminer si les athlètes sont avantagés ou non.*
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MOTS CLÉS : comportement compétitif, arts martiaux, taekwondo, perte de poids

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The research conducted involved human subjects. All procedures conformed to the ethical standards of the IRB located at the Canadian Memorial Chiropractic College. Prior to the commencement of the study ethical approval was granted.
Competing interests – None.

Introduction

Taekwondo (TKD) is a full contact free-sparring weight classed sport. This form of martial arts originated in Korea and became an Olympic demonstration sport in the summer of 1988 which led to its eventual inclusion into the summer 2000 Olympic Games. It has since gained in popularity and is now practiced worldwide.

Taekwondo competitions are divided by weight classes, as is done in wrestling, judo, and other combat sports. This classification system aims to equalize differences in strength, leverage and agility amongst competitors.[1] Athletes often aim to compete in the weight class below their natural body mass, as it is believed that this will provide a competitive advantage, sparring against smaller and theoretically weaker opponents. In order to achieve this feat, weight classed athletes often use rapid weight loss practices prior to weigh-in which occurs 16–20 hours before competition. After weigh-in occurs, athletes tend to abandon body mass loss practices to replenish nutrients, hydrate and thus, increase weight before the first match. In this study, Taekwondo athletes were weighed at the usual pre-match time 16–20 hours prior to initiation of the competition, then again immediately prior to sparring in order to indirectly measure the rate of weight cycling occurring in adolescent Taekwondo athletes.

The RWL that occurs prior to competition leads to *weight cycling* a term used to describe RWL following self-induced food limitation and/or dehydration. Both gradual (seasonal) and rapid (weekly) weight reduction cycles are used by TKD athletes, and have been investigated for potential effects on nutrition and performance.[2] Some initial data on pre-competition weight cycling in TKD showed that 53% of the respondents fasted prior to competition.[3] A follow up study[4] comparing the group in the 2005 study (Group A) to the Canadian National team (Group B) found that the National Taekwondo team athletes were more likely to have more years of practice ($p = 0.03$) and were less likely to fast prior to competition ($p = 0.03$) as compared to the Group A athletes. Not surprisingly, more than half of the competitors in Group A and eighteen percent of Group B athletes dieted prior to competition in order to make their weight class. This may suggest that the elite athletes (Group B) have been preparing for the competition more systematically.

Current literature reveals controversy regarding weight cycling, RWL and performance. Artioli et al.[1] reported that rapid reduction of approximately 5% body mass achieved by typical judo athletes, when followed by a 4 hour recovery period, did not impair simulated judo performance or arm power. In contrast, Filare et al. reported that left hand grip values and 30-second jump test output were decreased after seven days of food restriction in judo athletes.[5] All mean micronutrient intakes were below recommended values, while triglyceride levels and free fatty acids were increased.[5] Koral and Dosseville found that RWL procedures did not have an effect on short intense efforts (squat jump, countermovement jump, repetitions of Judo movements), but adversely affected prolonged physical performance.[6] There is a need to find out what this translates to for TKD athletes who often compete in many matches over a day of tournament. Varied performance outcomes associated with weight cycling has been a common finding in weight cycling research. Koral and Dosseville suggest that alterations in performance may depend on the type of body mass lost, type of sport undertaken and on the type of dietary restriction the competitor is committed to.[6]

The focus of this study is on adolescent athletes participating in TKD competition. Apart from a lack of knowledge as to how this may affect the health of adolescent athletes, it must not be ignored that the possibility of adolescent athletes developing eating disorders as a consequence of being overly concerned with weight control in weight class sports cannot be ruled out, as most athletes begin weight cycling at impressionable, young ages.[1] What is particularly striking are the methods used to induce RWL which range from nutritional restrictions to extreme physical demands. Specifically, dieting, fasting, fluid restriction, increased physical activity, passive (sauna) and active (sweat suit) dehydration, and even pathogenic methods such as diuretics, laxatives, and self-induced vomiting are practiced to achieve RWL.[5] These drastic methods for RWL can have severe short term and long term impacts on health, particularly associated with water restriction. Water balance is essential for metabolic processes and temperature regulation to properly cool down the body via sweating. It is no surprise that 60 percent of the subjects reported that during weight loss they felt fatigued, 30 percent perceived that their performance was decreased, 30 percent felt increased tension, 23 percent felt their vigour was reduced, 20 percent felt increased anger and 17 percent felt dizzy.[6] Collegiate

wrestlers undergoing RWL suffered similar symptoms to that of Taekwondo athletes as reported by Alderman et al.,[7] including headaches, dizziness, nausea, and fever sensations. Furthermore, Koral and Dosseville[6] reported an increased perceived effort during physical activity for Taekwondo athletes, even if their overall results and performance were maintained.

Athletes using weight control techniques may believe an advantage will be gained over the opponent competing at his/her natural body mass. There are many warnings against weight cycling and RWL in the literature, and while some aspects of performance have been shown to decline, many studies have not revealed the effects of weight cycling and RWL on performance to be negative. Therefore, it is important to illuminate the potential long term health hazards and exposure of young TKD athletes to dangerous short term effects. It is also suggested that experienced weight cyclers adapt to weight loss procedures and become less affected by negative effects on performance. This may mask declines in performance that could present in less experienced weight cyclers. Considering the lack of consistently negative performance consequences, it can be postulated that athletes may have a misplaced sense of improved strength and performance capabilities when weight cycling for competition. These views may be reinforced if a weight cycling athlete wins a competition, thus increasing the likelihood of using these strategies in the future.

To date, there is a lack of research in the areas of weight cycling and its perceived benefits among Taekwondo athletes. The purpose of this study is to investigate rate of weight cycling utilization in Junior Taekwondo Athletes (14–17 years old) and its effects on performance (winning a medal).

Methods

The junior athletes (14–17 year-old) were weighed one day prior to the competition (weigh-in) as required for qualification to compete in their specific weight category. Weight scale CAS model DL-100N, serial # 080441216 and CAS Model DL-200N, serial # 080441232 were used to weigh female and male athletes respectively. The scales were calibrated before weighing. Subsequently, athletes were weighed again, utilizing the same weight scales used for initial weigh in, just before their first match (16–20 hours later). This initial body mass and pre-match body mass were recorded on a separate sheets so that the recorder was blinded to the athlete's initial weight.

Statistical Analysis

Descriptive statistics (proportions, means and standard deviations) were used to summarize the sample of athletes. A two factor repeated measures analysis of variance with time (weigh in versus pre fight weight) and gender was used to analyze the weight measurements. A comparison of winners (medalist) versus non-winners in terms of body mass-gain after official weigh-in was conducted using a two sample t test. Statistical packaged used was "Statistica". Statistical evaluation was carried out by Dave Soave, Research Methodologist.

Results

A total of 108 athletes (72 males, 36 females) participated in the study. Mean male body mass weigh-in was 62.7 kg, and pre-match body mass was 63.7 kg indicating a 1 kg body mass gain during the recovery period. Mean female weigh-in was 55.5 kg while mean pre-match body mass was 56.7 kg, indicating a 1.2 kg body mass gain during the recovery period. Body mass gain was significant for both sexes, but not significant across sexes (males did not gain significantly more than females and vice versa) (Table 1).

Table 2 depicts mean body mass differences between weigh-in and pre-match per weight category. The 'light-heavy' weight class had the highest change in body mass amongst males at 1.9 kg, while the bantam and light-middle weight classes had the highest change amongst females, both with a mean body mass gain of 1.6 kg. In contrast, females and males in the 'heavy' weight class both lost body mass during recovery period.

Winners had a body mass difference of 1.02 kg after recovery period while non-winners had a slightly larger body mass difference of 1.09 kg. Both body mass changes were significant, however the difference of body mass change between winners and non-winners was not significant (0.11) ($P = 0.81$) (Table 3).

Discussion

The main objective of this study was to determine if pre-competition weight cycling had an effect on overall performance (winning a medal) in Junior TKD athletes. Though there are several studies investigating perform-

Table 1 *Mean weigh-in and pre-match body mass (kg) by sex*

	Males	Females	Combined
N	n = 72 (66.7%)	n = 36 (33.3%)	n = 108
Weigh-In (kg [SD])	62.7 [13.3]	55.5 [6.5]	60.3 [11.9]
Pre-match (kg [SD])	63.7 [13.0]	56.7 [6.4]	61.4 [11.7]
Age (Year [SD])	16.3 [0.6]	16.1 [0.9]	16.2 [0.7]

*SD = Standard Deviation – Statistics derived from "Statistica"

Table 2 *Mean body mass changes from weigh-in to pre-match per weight category*

Weight Category	Males N	Body mass Difference from weigh-in to pre-match Mean(SD) kg	Females N	Body mass Difference from weigh-in to pre-match Mean(SD) kg
FIN	4	0.43(1.3)	–	–
FLY	2	1.1(1.8)	1	1.0(–)
BANTAM	3	1.5(1.2)	1	1.6(–)
FEATHER	14	1.6(0.7)	3	1.3(0.2)
LIGHT	11	1.3(1.0)	8	1.4(0.5)
WELTER	10	0.9(0.7)	7	1.4(1.1)
LIGHT MIDDLE	10	0.5(2.2)	5	1.6(0.6)
MIDDLE	7	0.7(0.7)	5	1.1(0.8)
LIGHT HEAVY	5	1.9(1.0)	5	0.7(0.9)
HEAVY	6	–0.2(0.7)	1	–0.2(–)

*SD = Standard Deviation – Statistics derived from "Statistica"

ance and weight cycling in TKD and other weight classed combat sports,[1,6,8] current literature reveals controversy in results regarding weight cycling, RWL and performance, indicating a need to tease out this relationship. Our findings suggest that pre-competition weight cycling has no significant effect on overall competitive performance in both male and female weight classes following a 16–20 hour recovery period. Body mass gained during the recovery period between the initial "weigh-in" and secondary "pre-match" measurements is an indirect method of measuring body mass lost during weight cycling. It is assumed that the more body mass lost during weight cycling, the more body mass will be gained during the recovery period.[9] All athletes in this study were allowed about 16–20 hour recovery period between the "weigh-in" and "pre-competition" measurements whereby athletes re-fed and rehydrated. In a similar study by Artioli et al.[1] investigating weight cycling amongst judo athletes, subjects who lost body mass proceeded to regain 51 ± 13% of their body mass during a 4 hour recovery period between weight-in and performance evaluation. In the present study, as seen in Table 1, both males and females gained less than 0.5% of their body mass back between weight-in and pre-match which is considerably less than the body mass gain values found by Artioli et al.[10] This can be attributed to the fact that the average age of the participants in the present

Table 3 *Body mass changes in Taekwondo athletes: Winners vs. Non-Winners*

	Weigh-in	**Pre-match**	**Difference**
Winners (kg [SD])	60.13 [11.94]	61.23 [11.77]	1.02 [1.03]
Non-Winners (kg [SD])	61.54 [12.21]	62.55 [11.68]	1.09 [1.11]

*SD = Standard Deviation – Statistics derived from "Statistica"

study is 4 years younger and presumably less experienced at RWL and recovery then the participants used in the Artioli et al.[10]study, which had the average age 20 ± 4 years and were all males. These differences in results could also be due to the sport specific variability between judo and TKD.

All weight categories, with the exception of the 'Heavy' weight class (for both males and females) gained body mass during the recovery period. The weight classes that gained the most body mass differed between male and female groups. The "Light Heavy" group in males and "Bantam" and "Light Middle" groups in females are the groups that gained the most body mass during recovery. This discrepancy between sexes may be due to the differing weight loss methods undertaken by female and male athletes in those groups to re-hydrate and feed during the recovery period. It may also be due to low number of participants in these weight categories. Interestingly, both male and female "Heavy Weight" groups lost the same amount of body mass during the recovery period at 0.2 kg. The athletes in the 'Heavy' weight class do not need to weight cycle, as there are no upper limits for their weight category.

Table 3 depicts the difference in "weigh-in" measurements between *Winners* and *Non-winners* as less than 1% (exactly 0.27%). Because body mass changes due to RWL between winners and non-winners was not significant it may be inferred that RWL cannot solely be a determining factor in winning a TKD match. Instead, it may be postulated that *Winners* are simply more skilled at their craft when compared to *Non-winners*, and RWL is not a factor affecting performance. Body mass gained during the recovery period in non-winners was slightly and non-significantly greater than that gained in winners. Non-winners on average gained 0.7kg or 0.8% more body mass than winners. Weight gained during recovery may have affected performance but this is currently not supported by other research. A study by Koral and Dosseville[6] found that mass loss procedures (gradual weight loss over 3–4 weeks) employed by elite judoists prior to competition had no affect on short duration performance. In this study a 1–2 hour recovery period was allowed after weigh-in and prior to performance evaluation. As mentioned previously, Artioliet al.[1] allowed a 4 hour recovery period for a weight-loss group. In both studies the weight-loss group performance, following recovery, had no effect on overall performance when compared to a control non-weight losing group.

RWL is a technique that is not practiced by TKD athletes alone. Artioli et al.[10] noted that 86% of the judo athletes participating in the study subjected themselves to RWL techniques. A very similar percentage was found in a study by Fleming and Costarelli[8] whereby 87% of the TKD athletes in their study were subjecting themselves to RWL practices.

Rapid weight loss is a potentially dangerous technique to partake in that may have negative effects on athletes. It has been noted in various studies[6,9] that RWL practices can cause increased tension, dizziness, fatigue, increased confusion and loss of vigour. Artioli et al.[1] also mentions that three young athletes in 1997 died due to hyperthermia and dehydration in the USA as a result of RWL practices. Since RWL seems to have no foreseeable benefits on overall performance the incentive for TKD athletes to subject themselves to this potentially harmful practice is not justified. Monitoring dietary habits of athletes in weight classed sports is recommended since most weight class athletes compete 5–10% under their natural body mass and do this up to 10 times a year.[1] It is prudent to assume that larger body mass losses and more frequent dieting could potentially result in negative physiological and performance consequences. Twenty percent of the athletes reported that they had never received advice on healthy eating and appropriate weight loss practices.[8] It

is clear that athletes in weight classed sports are not receiving adequate education regarding the potential health risks and negative performance outcomes associated with weight cycling.

A limitation of this study was the failure to record the methods of weight cycling employed by participating athletes, and who provided the information regarding how to weight cycle. The methods of RWL employed could have shed some light into the physiological and psychological states of the competitors. This could have elicited why there were differences in pre-match weight gain between different weight classes, and determined the severity of the RWL practices being conducted in TKD today. Also, different methods of RWL may have manifested in different associated symptoms, and could have affected performance in various ways.

The methods of recovery employed by athletes during the 16–20 hour recovery period are unknown and act as another limitation in this study. What is clear is that most groups (except the 'Heavy' weight class) in the male and female categories gained body mass during recovery. Previous studies[1,6] have shown that implementing a recovery period after RWL methods negates negative effects on overall performance. Additionally, sample sizes in this study were relatively small, thus limiting conclusions from the descriptive statistics. Further research with larger sample sizes should be conducted in the future.

One subject that remains to be explored is the effects of weight cycling on first time competitors. The current study has not explored at what age the athletes began to practice RWL. Artioli et al.[1] and Koral and Dosseville[6] studied experienced martial art athletes with an average age over 20 years old, who have been implementing RWL techniques for many years. Even though the average age of the participants in the present study were younger (on average 16 years of age), these athletes may have been using RWL techniques for some time. Artioli et al.[10] found that on average, male and female judo competitors begin weight cutting for competition at 12.6 ± 6 years of age. As mentioned in the study conducted by Artioli et al.,[1] seasoned competitors have time for their bodies to adapt to the weight cycling practice. This can help athletes overcome some of the negative side effects of RWL. It would also be interesting to study the effects of RWL on athletes that were not allowed a recovery period following weigh-in. Currently this does not follow real world scenarios since in TKD tournaments, recovery periods of various durations are allowed. However, such a study can potentially demonstrate if RWL itself results in negative effects on performance. The other area to be explored is the effect of RWL on endurance sports versus anaerobic sports.

Conclusion

RWL is a common practice that many athletes undergo prior to competitions. RWL seems not to have a significant effect on overall performance in young TKD athletes. This may be due to the fact that RWL is followed by a recovery period which may mitigate negative effects associated with RWL.

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References

1 Artioli GG, Iglesias RT, Franchini E, Gualano B, Kashiwagura DB, Solis MY, Benatti FB, Fuchs M, Lancha AH. Rapid weight loss followed by recovery times does not affect judo-related performance. J Sports Sci. 2010; 1:21–32.

2 Fogelholm GM, Koskinen R, Laakso J, Rankinen T, Ruokonen I. Gradual and rapid weight loss effects on nutrition and performance in male athletes. Med Sci Sports Exerc.1993; 371–377.

3 Kazemi M, Shearer H, Choung YS. Pre-Competition habits and injuries in Taekwondo athletes. BMC Musculoskeletal Disorders. 2005; 6:26.

4 Kazemi M, Shearer H. Differences in pre-competition habits between national team and other competitive Taekwondo athletes. Proceeding of 1st International Symposium for Taekwondo Studies, Beijing, China 2007.

5 Filare E, Maso F, Degoutte F, Jouanel P, Lac G. Food Restriction, Performance, Psychological State and Lipid Values in Judo Athletes. Int J Sports Med. 2001; 22:454–459.

6 Koral J, Dosseville F. Combination of gradual and rapid weight loss: Effects on physical performance and psychological state of elite judo athletes. J Sports Sci. 2009; 2:115–120.

7 Alderman BL, Landers DM, Carlson J, Scott JR. Factors related to rapid weight loss practices among international-style wrestlers. Med Sci Sports Exerc. 2004; 249–252.

8 Fleming S, Costarelli V. Eating behaviours and general practices used by Taekwondo players in order to make

weight before competition. Nutrition & Food Science. 2009; 1:16–23.
9 van Dijik F**,** Garthe I, Wisnes A. Rapid weight loss practices among elite Taekwondo players. In: Proceedings of 1st International Symposium for Taekwondo Studies, Beijing, China 2007.
10 Artioli GG, Gualano B, Franchini E, Scagliusi FB, Takesian M, Fuchs M, Lancha AH. Prevalence, magnitude, and methods of rapid weight loss among judo competitors. Med Sci Sports Exerc. 2010; 3:436–442.

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Lateral epicondylosis and calcific tendonitis in a golfer: a case report and literature review

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Objective: *To detail the progress of a young female amateur golfer who developed chronic left arm pain while playing golf 8 months prior to her first treatment visit.*

Clinical Features: *Findings included pain slightly distal to the lateral epicondyle of the elbow, decreased grip strength, and positive orthopedic testing. Diagnostic ultrasound showed thickening of the common extensor tendon origin indicating lateral epicondylosis. Radiographs revealed an oval shaped calcified density in the soft tissue adjacent to the lateral humeral epicondyle, indicating calcific tendonitis of the common extensor tendon origin.*

Intervention and Outcome: *Conventional care was aimed at decreasing the repetitive load on the common extensor tendon, specifically the extensor carpi radialis brevis. Soft tissue techniques, exercises and stretches, and an elbow brace helped to reduce repetitive strain. Outcome measures included subjective pain ratings, and follow up imaging 10 weeks after treatment began.*

Conclusion: *A young female amateur golfer with chronic arm pain diagnosed as lateral epicondylosis and calcific tendonitis was relieved of her pain after 7 treatments over 10 weeks of soft tissue and physical therapy focusing specifically on optimal healing and decreasing the repetitive load on the extensor carpi radialis brevis.*
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KEY WORDS: epicondylosis, calcific tendonitis, golf

Objectif : *exprimer en détail la progression d'une jeune golfeuse de calibre amateur qui a développé une douleur chronique au bras gauche en jouant au golf 8 mois avant son premier traitement.*

Caractéristiques cliniques : *douleur légèrement distale à l'épicondyle latérale du coude, prise plus faible et test orthopédique positif. L'ultrason diagnostic a démontré l'épaississement du tendon extenseur commun, ce qui indique une épicondylose latérale. Les radiographies ont révélé une densité calcifiée de forme ovale dans les parties molles adjacentes à l'épicondyle humérale latérale, ce qui indique une tendinite du tendon extenseur commun.*

Intervention et résultat : *les soins conventionnels visaient à diminuer la charge répétitive sur le tendon extenseur commun, particulièrement le muscle court extenseur radial du carpe. Les techniques des parties molles, les exercices et les étirements, ainsi qu'un support de coude ont contribué à réduire la tension répétitive. Les niveaux de douleur subjectifs ont été établis, et une imagerie a eu lieu 10 semaines après le début du traitement.*

Conclusion : *une jeune golfeuse de calibre amateur souffrant d'une douleur chronique au bras a reçu un diagnostic d'épicondylose latérale et de tendinite calcifiante et fut guérie de sa douleur après 7 traitements répartis sur une période de 10 semaines de thérapie physique et des parties molles axée sur la guérison optimale et la réduction de la charge répétitive sur le muscle court extenseur radial du carpe.*
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MOTS CLÉS : epicondylose, tendinite calcifiante, golf

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Introduction

The sport of golf has grown in popularity over recent years, especially in North America. In any sport, with an increase in popularity comes an increase in injury incidences. Golfers commonly report injuries to the lower back, lower extremity, and upper extremity. The most common upper limb area to be injured in amateur golfers is the elbow.[1] The term "golfer's elbow" has traditionally been used as a common expression to describe medial epicondylosis. A much more widespread elbow problem in golfers is in fact lateral epicondylosis (LE). Among amateur golfers, lateral elbow pain was found to be 5 times more common than medial elbow pain.[2] In golfers, LE results from repetitive forearm extension and excessive gripping of the club during the swing. Additional identified risk factors that can contribute to elbow injuries in golfers include poor swing mechanics, poor conditioning, inadequate warm-up, inappropriate equipment, overuse, age, and preexisting pathological conditions.[1,2]

The diagnosis of LE was first recorded in the literature by Runge in 1873.[3] It is the most common cause of lateral elbow pain affecting approximately 1–3% of adults each year, and is more commonly seen in the dominant arm.[4,6,7] Men and women are affected with an equal prevalence, typically in their 4th to 5th decade of life.[5–7] Though special imaging is rarely ordered for cases of LE, the most common radiographic finding is calcification along the lateral epicondyle within the common extensor tendon.[8]

Calcified tendinopathy or calcific tendonitis (CT) is a chronic condition where deposits of calcium phosphate crystals accumulate in the midsubstance of the tendon fibers.[9–11] Epidemiological studies report that the prevalence of CT is highly variable (3–22% in the general population) depending on the location within the body.[10,11] CT has been shown to worsen tendinopathy conditions causing increased rupture rates, slower recovery times, and high frequencies of complication after surgery.[9,14] Women are affected slightly more often than men, with patients typically between 30 and 60 years of age.[12,15]

This case report will describe an amateur female golfer who experienced lateral arm pain while playing golf and during her activities of daily living. Imaging revealed an oval shaped calcific density in the soft tissue adjacent to the lateral epicondyle of the humerus. The patient underwent a simple non-invasive conservative treatment plan using soft tissue techniques, an elbow brace, as well as stretching and strengthening exercises.

Case Report

In this case, a 27-year old female amateur golfer developed left arm pain while playing. The pain first occurred for a short duration 8 months prior to the patient's initial visit. She also recalls starting a new job one month later, where typing on a laptop re-aggravated her symptoms. Hitting golf balls at the driving range was described to be extremely painful at that time. The patient reported trouble picking up heavy objects with her left hand. The symptoms then subsided for a period of 6 months, until an acute exacerbation 1 week prior to her initial visit. At that time the patient found it difficult to move her elbow and experienced shooting pains down her forearm. Rest, ice, an elbow brace, and medication (ibuprofen taken 4 times a day) were self administered by the patient but failed to improve her condition. She rated her pain as 4/10 on a Visual Analog Scale (VAS). The patient reported no previous history of trauma or injury and a systems review was unremarkable.

On physical examination, postural observation revealed mild anterior head carriage, and slight anterior rolling of the left shoulder. No swelling or discoloration was noted. Palpation reproduced pain and tenderness over the left lateral epicondyle, wrist extensor muscles, and radiocapitellar joint. The left extensor carpi radialis brevis was particularly tight, as was the sternocleidomastoid, levator scapulae, and scalenes. The chief complaint was reproduced with resisted wrist extension and passive wrist supination. Resisted wrist supination and finger extension were both pain free. Orthopedic testing found Cozen's test and Varus stress test to be positive for left elbow pain (Table 2). A cervical spine screen was negative and an upper limb neurological exam was within normal limits. Grip strength was found to be weaker on the left when compared to the right.

The patient was diagnosed with left lateral epicondylosis. Due to the acute nature of the patient's pain and the extreme difficulty moving her elbow, she was referred for ultrasound and radiographic imaging to rule out more sinister differentials (osteochondritis dissecans of the capitellum or radiocapitellar arthritis). Diagnostic ultrasound showed thickening of the origin of the common extensor tendon from the lateral humeral epicondyle,

indicating LE. Radiographs revealed an oval shaped calcified density in the soft tissue adjacent to the lateral humeral epicondyle, indicating CT of the common extensor tendon origin (Figure 1).

Treatment

The patient was treated with 7 sessions over 10 weeks. Passive treatment consisted of NSAIDs (Naprosyn taken as required) and ice to help decrease inflammation. A fitted tennis elbow brace was prescribed to help reduce the repetitive load on the common extensor origin. Soft tissue techniques in the form of forearm interosseous release and light massage of the extensor muscles, specifically to the extensor carpi radialis brevis (ECRB) muscle, were performed during each session for 3 to 5 minutes. The wrist extensors and flexors were stretched. Isometric bicep curls and triceps extensions were prescribed. Postural strengthening exercises included scapular setting, chin retraction, and wall angels. Wall-clocks were given as a shoulder girdle proprioceptive exercise. Additionally, active care consisted of the patient being given plasticine crunches to increase grip strength. See Table 1 for a complete list of exercise progression.

After 10 weeks the patient had fully recovered. She was able to return to playing golf pain free. Follow up imaging studies showed that the common extensor tendon thickening and calcification had resolved. This patient was not considered a surgical candidate, as a trial course of conservative care was successful in allowing her to return to sport pain free.

Discussion

Calcific tendonitis

The pathogenesis of CT is still not fully understood. Suggestions as to the origin of calcification include vascular or mechanical trauma, hypoxia, extracellular matrix vesicle organelles, metaplasia of tendon cells, and metabolic endocrine disorders.[12,13] In CT, calcium phosphate (hydroxyapatite) deposits within the tendon cause inflammation and painful symptoms. These deposits may trigger an inflammatory reaction through the up regulation of inflammatory mediators. The potential for crystals to elicit an inflammatory reaction is reported to depend on their morphology. Surface area, size, density, and calcium phosphate ratio of the crystal all display positive correlations with clinical symptoms.[16] Asymptomatic patients tend to have calcific deposits that appear granular with sharply defined and well-circumscribed borders. Alternatively, acutely painful patients have enlarged deposits

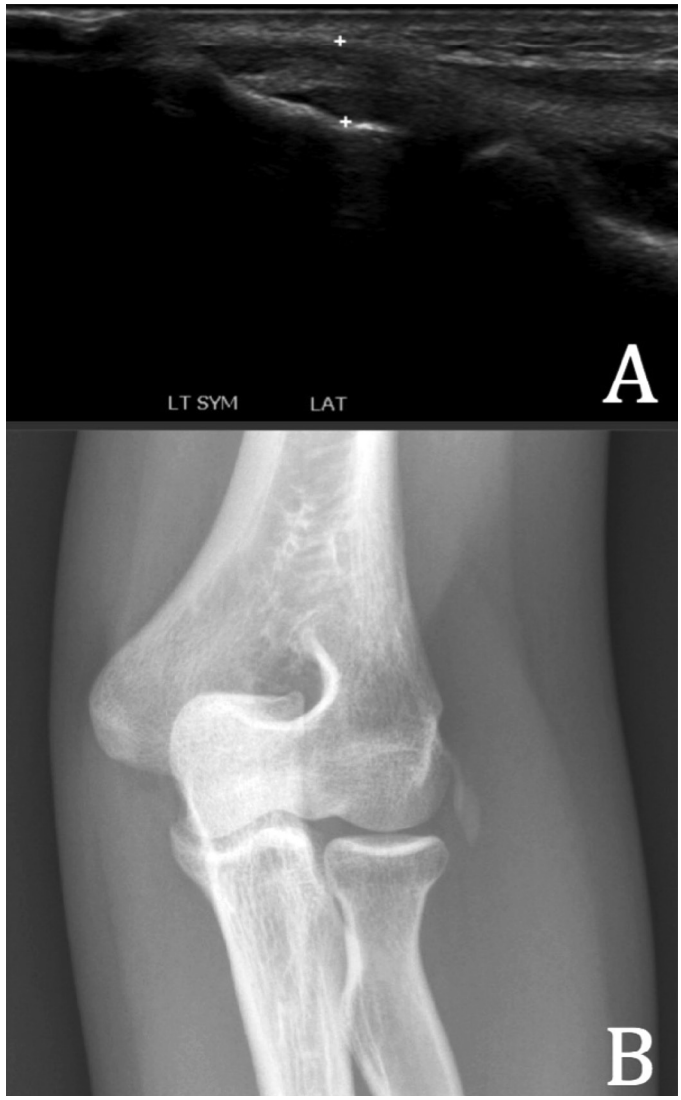

Figure 1 *Imaging results for the patients left elbow. A) Diagnostic ultrasound showing thickening of the origin of the common extensor tendon, indicating lateral epicondylosis. B) Anterior to posterior radiograph showing an oval shaped calcified density in the soft tissue adjacent to the lateral humeral epicondyle, indicating calcific tendinosis.*

Table 1 *Exercises and stretches prescribed to improve strength, flexibility and grip endurance of the forearm musculature.*

Exercise or Stretch	Repetitions	Sets	Time for hold and/ or contraction (Sec)
Wrist extensor stretch	3	1	30
Wrist flexor stretch	3	1	30
Scapular setting	10	3	5
Chin retraction	10	3	
Wall angels	5	3	
Wall clocks	5	1	30
Plasticine crunches	10	3	
Isometric bicep curls	10	3	5
Isometric tricep extension	10	3	5

with liquefied, poorly circumscribed borders.[17] The condition itself has 4 well-defined phases. The first, formative phase, initiates from one of the causes listed above. During this phase the tendon undergoes fibrocartilaginous transformation. Then the calcific phase occurs, in which the calcific deposit enlarges producing pain and mechanical symptoms. Next, the resorptive phase involves macrophages and multinuclear giant cells absorbing the deposit and is often very painful. Finally, the reparative phase consists of fibroblasts restoring the collagen pattern back to normal tendon tissue.[12,15]

As mentioned in the introduction, there is strong evidence that suggests CT worsens the clinical manifestations of tendinopathy. Tendon biology can also be influenced by age, gender, previous injury, and hormonal variations (especially thyroxine). Furthermore, endocrine and connective tissue conditions may be implicated in the development of CT. With this in mind, the presence of an endocrine co-morbidity could alter the natural history of CT and lead to a chronic symptomatic state.[10] For example, it is known that hypothyroidism causes a build up of glycosaminoglycans in the extracellular matrix of tendons, which could predispose the tendons to develop calcific deposits. Tendinitis is often found as a presenting complaint in patients with hypothyroidism, and symptom resolution is frequently obtained with treatment of the underlying deficiency.[10]

Lateral Epicondylosis

The lateral epicondyle has a pyramidal morphology, with each of the three sides (anterior, posterior, and inferior) serving as a specific attachment site. The anterior surface contains attachments for the ECRB and extensor digitorum communis muscles, which are in fact part of the common extensor tendon origin. Posteriorly the anconeus muscle originates, while the apex and inferior surface are attachments for the lateral collateral ligament.[4] Important anatomical consideration must also be given to the radial nerve (Figure 2). The posterior interosseous nerve (PIN) branch is commonly entrapped in the supinator muscle at the radial tunnel, causing symptomotology similar to LE. Thus PIN entrapment is an important differential that should be considered in longstanding cases of LE.[4]

Epicondylitis is a term that implies the presence of inflammation, however, it is only the very early stages of LE that demonstrate this histological trait. Investigations have shown the disease process to be a degenerative tendinopathy, thus tendinosis or lateral epicondylosis is cited as the preferred terminology.[3,6] However, biochemical analysis has shown increased levels of the neurotransmitter glutamate and substance P receptors within the common extensor origin of patients with LE. These findings suggest a mechanism for neurogenic pain generation and provide a potential explanation for pain relief from steroid injection into a condition that is largely absent of inflammation.[4]

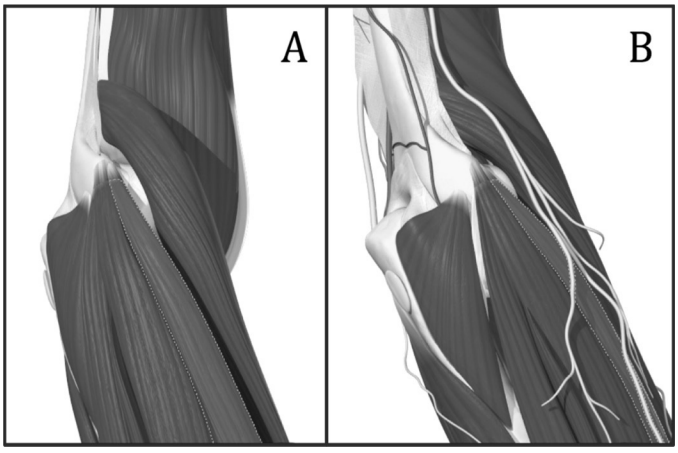

Figure 2 *The most common mechanism for lateral epicondylosis is caused by repetitive eccentric contraction of the extensor carpiradialisbrevis (ECRB) muscle. This causes microtears within the common extensor tendon origin. A) ECRB muscle at proximal lateral epicondyle attachment. B) ECRB muscle with overlying posterior interosseous nerve.*
(Image courtesy and copyright Primal Pictures www.anatomy.tv)

Biomechanical assessment shows that LE is primarily caused by excessive ECRB eccentric contraction causing a microtraumatic tear in the common extensor tendon origin (Figure 2).[3] Extensor carpi radialis longus attaches to the lateral supracondylar ridge of the humerus, not the lateral epicondyle, and thus is not involved in LE. Other hypothesized causes of LE include direct trauma, hypovascularity of the region, and anatomical predisposition.[3,6,7] Identified risk factors associated with LE include a history of heavy tool use, and significant forearm strain while performing repetitive tasks.[4]

During the clinical encounter, patients with LE will present with pain localized to the lateral aspect of the elbow that radiates down the forearm with certain movements or carrying items in their hand.[3,6] Symptoms are usually exacerbated by resisted wrist extension or passive wrist flexion. There will often be more pain in the morning or after the elbow has been held in a flexed position for a prolonged period of time.[4,7]

The physical exam should start with screens of the cervical spine and shoulder to rule out any radicular or referred pain from these structures.[3] As was reported with the amateur golfer, palpation will typically discover a point of maximal tenderness just distal and anterior to the lateral epicondyle over the origin of the ECRB muscle.[4] Several orthopedic tests are regularly positive with this condition; these include Cozen's test, Mill's test, Thomson test, Bowden test, and the Chair test (Table 2).[6,7] Grip strength should be tested and compared with the opposite hand to determine if there is a significant difference.[3,6,7] Two important differentials to rule out during the physical exam are PIN entrapment and posterolateral rotatory instability. Approximately 5% of LE patients simultaneously have PIN entrapment. This condition is indicated by pain on resisted supination with the elbow flexed (entrapment in the supinator muscle) or pain with resisted long finger extension (Maudsley's test) when the elbow is extended (entrapment in the ECRB muscle).[3,6] Abnormal sensation to the web of the thumb also suggests injury to the PIN.[2] Posterolateral rotatory instability is caused by injury and laxity in the lateral collateral ligament of the elbow. It is important to evaluate the integrity of this structure with a Varus stress test. This condition should be considered if patients have undergone treatment for LE and continue to have persistent symptoms.[6]

The differential diagnosis list for LE also includes synovial plica, cervical radiculopathy, osteochondritis dissecans of the capitellum, and radiocapitellar arthrosis,[3,4,6] several of which require special imaging in order to rule out. In fact, imaging studies are often more helpful in ruling out a differential cause of elbow pain than in making a diagnosis of LE.[3] Radiological examination of LE patients is usually normal.[5] Occasionally intra-articular pathology or calcifications within the surrounding soft tissue structures are observed.[4] As was demonstrated in the case report, calcifications have been associated with persistent LE and are reported to be present in approximately 7–25% of patients.[3,4,6,8] Ultrasound examination of LE patients can identify thickening of the common extensor tendon, intrasubstance tendon tears, focal hypoechoic regions within the tendon, adjacent bony irregularity, and calcifications.[4,6,7,12] Magnetic resonance imaging is generally not indicated for LE patients. Its main usefulness is in the exclusion of osteochondral defects, identifying non-displaced physeal fractures, quantifying the extent of tendon tearing, and preoperative planning.[3,4,6]

The success rate for conservative treatment of LE patients is around 90%.[2,3] It can include several interven-

Table 2 *Orthopedic tests regularly used to aid in the diagnosis of lateral epicondylosis.*[6,7]

Cozen's test	Patient resists the examiner while in a position of 90 degrees of elbow flexion and full wrist extension.
Mill's test	Examiner passively takes the patient into maximal elbow extension and wrist flexion.
Thomson test	Patient resists the examiner while in a position of 60 degrees of shoulder flexion, maximal elbow extension, forearm pronation, and 30 degrees of wrist extension.
Bowden test	Patient squeezes a blood pressure cuff and attempts to maintain a specific degree of pressure.
Chair test	Patient attempts to lift a moderately lightweight chair by gripping it from the chair back with both elbows extended and forearms pronated.

tions used either alone or in combination. Ice should be applied several times throughout the day during the acute phase of LE. Once the subacute phase begins, heat can be applied to help promote increased blood flow to the area and reduce muscle spasm.[2] Ultrasound therapy can also be useful to influence blood flow and tissue extensibility while decreasing pain.[2] Medications suggested for pain control include acetaminophen as well as oral or topical NSAIDs.[4] Acupuncture was also shown to display a trend of shortening pain symptoms in LE patients.[6,18] Low-level laser therapy at 904 nm wavelength has demonstrated positive results for tendon pathology through stimulation of fibroblast activity.[6] Additionally, counterforce wrist extension braces have been shown to be helpful. The brace is believed to decrease the repetitive load on the elbow by preventing the forearm muscles from fully contracting, leading to decreased tension within the common extensor origin.[2,4,6] It is important to note that braces placed just distal to the lateral epicondyle reduce loads greater than braces placed directly over the lateral epicondyle.[3,18] Shock wave therapy has produced contradicting evidence in terms of its efficacy for treating LE. Some investigators have found patients improved, while others have reported a lack of effectiveness.[3,19] Osteopathic and chiropractic manipulation of the elbow has been effective in certain cases of LE. The goal of manipulation is to release adhesions within joint articulations, promote realignment, and normalize function.[2,18] Injections of various substances have also been used to decrease pain and promote healing in numerous tendonopathies including LE. See Table 3 for a complete list of substances used for injection and proposed mechanisms of action.[3,4,6] Regarding physical therapy, exercises are generally aimed at strengthening the wrist extensors and flexors, but supinator, pronator, and grip strength exercises should also be included in an effort to prevent re-injury. Stretching exercises and eccentric training for the forearm extensor and flexor muscles can also aid in recovery.[3,4] Theoretically, eccentric training induces hypertrophy in the musculotendinous junction, increasing tensile strength to the area and reducing the amount of strain on the tendon. Eccentric contractions may also stimulate tendon cells to produce collagen and decrease neovascularization that contributes to pain.[4] There has been some research done on injury prevention programs for golfers. These tend to focus more on developing muscular endurance and motor control of the lower back and shoulder areas.[20]

There are several poor prognostic factors to successful conservative treatment of LE patients. These include dominant arm involvement, high baseline pain levels of long duration, poor coping mechanisms, and manual labor occupation.[4] A wide variety of surgical treatment options exist for LE patients. Both open and arthroscopic techniques have high success rates of 84–97%. However, only 4–11% of LE patients who seek medical care will require surgery.[3,4] Operative management is indicated for patients with: 1) Persistent pain. 2) Functional disability. 3) After 6 to 12 months of failed conservative therapy.[3,4,6] Complications that may arise from elbow surgery include peripheral nerve injury, heterotropic ossification of soft

Table 3 *Substances commonly used for injection of tendonopathies and the proposed mechanisms by which they mediate their actions.*[3,4,6]

Anesthetic and Corticosteroids	Act directly at the site of injection to reduce local pain and inflammation.
Botulinum Toxin A	Causes a paralytic effect in muscles by irreversibly inhibiting acetylcholine release at the neuromuscular junction.
Platelet Rich Plasma (PRP)	Releases massive quantities of advantageous cytokines, such as growth factors, known to be important to tendon healing.
Whole Blood	Pain relief is hypothesized to be secondary to growth factors acting as humoral mediators and inducing a healing cascade.
Prolotherapy	The practice of injecting an irritating substance into a tendon or ligament to promote the growth of new tissue.

tissues, and posterolateral rotatory instability from overly aggressive debridement.[4]

Summary

Injuries in amateur golfers have been widely reported.[1,2] Some of the more common injuries to be discussed in the literature include the lower back, wrist, elbow, and stress fractures.[1,21,22] The cause of injury to LE patients is believed to be multi-factorial and include repetitive microtrauma to the ECRB of the common extensor tendon as well as significant forearm strain during repetitive tasks, such as swinging a golf club. Conservative treatment is aimed at physical therapy featuring stretching and strengthening exercises along with several other possible co-interventions. In this case, a young female amateur golfer with elbow pain diagnosed as LE and CT was relieved of her pain after 7 treatments over 10 weeks.

References

1 Bayes MC, Wadsworth LT. Upper extremity injuries in golf. Phys Sportsmed. 2009; 37(1):92–6.
2 Stockard AR. Elbow injuries in golf. J Am Osteopath Assoc. 2001; 101(9):509–16.
3 Van Hofwegen C, Baker CL 3rd, Baker CL Jr. Epicondylitis in the athlete's elbow. Clin Sports Med. 2010; 29(4):577–97.
4 Calfee RP, Patel A, DaSilva MF, Akelman E. Management of lateral epicondylitis: current concepts. J Am AcadOrthop Surg. 2008; 16(1):19–29.
5 Matthews P, Leyshon R. Acute calcification in tennis and golfer's elbow. Rheumatol Rehabil. 1980; 19(3):151–3.
6 Scher DL, Wolf JM, Owens BD. Lateral epicondylitis. Orthopedics. 2009; 32(4):276–282.
7 Levin D, Nazarian LN, Miller TT, O'Kane PL, Feld RI, Parker L, McShane JM. Lateral epicondylitis of the elbow: US findings. Radiology. 2005; 237(1):230–4.
8 Pomerance J. Radiographic analysis of lateral epicondylitis. J Shoulder Elbow Surg. 2002; 11(2):156–7.
9 Lui PP, Chan LS, Lee YW, Fu SC, Chan KM. Sustained expression of proteoglycans and collagen type III/type I ratio in a calcified tendinopathy model. Rheumatology (Oxford). 2010; 49(2):231–9.
10 Harvie P, Pollard TC, Carr AJ. Calcific tendinitis: natural history and association with endocrine disorders. J Shoulder Elbow Surg. 2007; 16(2):169–73.
11 Holt PD, Keats TE. Calcific tendinitis: a review of the usual and unusual. Skeletal Radiol. 1993; 22(1):1–9.
12 Siegal DS, Wu JS, Newman JS, Del Cura JL, Hochman MG. Calcific tendinitis: a pictorial review. Can Assoc Radiol J. 2009; 60(5):263–72.
13 Hayes CW, Rosenthal DI, Plata MJ, Hudson TM. Calcific tendinitis in unusual sites associated with cortical bone erosion. Am J Roentgenol. 1987; 149(5):967–70.
14 Gohr CM, Fahey M, Rosenthal AK. Calcific tendonitis: A model. Connect Tissue Res. 2007; 48(6):286–91.
15 Mouzopoulos G, Stamatakos M, Mouzopoulos D, Tzurbakis M. Extracorporeal shock wave treatment for shoulder calcific tendonitis: a systematic review. Skeletal Radiol. 2007; 36(9):803–11.
16 Chiou HJ, Hung SC, Lin SY, Wei YS, Li MJ. Correlations among mineral components, progressive calcification

process and clinical symptoms of calcific tendonitis. Rheumatology (Oxford). 2010; 49(3):548–55.
17 Wainner RS, Hasz M. Management of acute calcific tendinitis of the shoulder. J Orthop Sports Phys Ther. 1998; 27(3):231–7.
18 Valen PA, Foxworth J. Evidence supporting the use of physical modalities in the treatment of upper extremity musculoskeletal conditions.Curr Opin Rheumatol. 2010; 22(2):194–204.
19 Sems A, Dimeff R, Iannotti JP. Extracorporeal shock wave therapy in the treatment of chronic tendinopathies. J Am Acad Orthop Surg. 2006; 14(4):195–204.
20 Lehman GJ. Resistance training for performance and injury prevention in golf. J Can Chiropr Assoc. 2006; 50(1):27–42.
21 McHardy AJ, Pollard HP. Golf and upper limb injuries: a summary and review of the literature.Chiropr Osteo. 2005; 25:13:7.
22 Lee AD. Golf-related stress fractures: a structured review of the literature. J Can Chiropr Assoc. 2009; 53(4):290–9.