Treatment of Myositis Ossificans with acetic acid phonophoresis: a case series

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Objective: To create awareness of myositis ossificans (MO) as a potential complication of muscle contusion by presenting its clinical presentation and diagnostic features. An effective method of treatment is offered for those patients who develop traumatic MO.

Management: Patients in this case series developed traumatic MO, confirmed on diagnostic ultrasound. Patients participated in a treatment regimen consisting of phonophoresis of acetic acid with ultrasound.

Outcome: In all cases, a trial of phonophoresis therapy significantly decreased patient signs, symptoms and the size of the calcification on diagnostic ultrasound in most at a 4-week post diagnosis mark.

Discussion: Due to the potential damage to the muscle and its function, that surgical excision carries; safe effective methods of conservative treatment for MO are

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Introduction
Myositis ossificans (MO) is a non-neoplastic proliferation of cartilage and bone in an area of muscle that has been exposed to trauma. Following a blunt trauma, 9-20% of athletes develop this condition and it is then termed myositis ossificans traumatica or myositis ossificans circumscripta. MO is diagnosed via findings of calcification in muscle identified on imaging. MO is self-limiting, with resolution reported after 1-2 years, and therefore can be managed conservatively in most cases. Literature looking at conservative treatment of MO is sparse, consisting of one non-controlled clinical trial, case studies and expert opinion. Surgical removal is considered in cases of persistent pain, limitations in range of motion (ROM), a decrease in function due to compression of neurological or vascular tissues. An attempt of 4-6 months of conservative care is tried initially prior to the consideration of surgery.

The purpose of this study is to offer manual therapists a method of conservative treatment for patients presenting with MO. This is achieved by describing three cases of athletes with traumatic MO confirmed on x-ray and diagnostic ultrasound and successfully treated with acetic acid phonophoresis.

Case 1
A 27-year-old male soccer player presented to a chiropractor with right groin pain approximately one month after kicking a soccer ball. The patient had not been treated in the past month and kept re-aggravating his groin while trying to continue to play soccer. This current re-aggravation was described as achy groin pain was rated as a 7 on a 10-point numerical scale. No ecchymosis was present and active and passive ROM of the hip displayed a decrease by 50% in adduction, abduction, and internal rotation. Diagnostic ultrasound confirmed MO in the proximal adductor longus muscle due to a large calcific mass measuring 7.7 x 1.8 x 0.29 cm (Figure 1). Along with the ultrasound findings, radiographic examination showed a linear calcification over 15 cm adjacent to the proximal femur. The patient was treated 3x/week for 4 weeks with therapeutic ultrasound and a 2% acetic acid solution as the medium. After the 4-week treatment plan, the patient rated his pain as a 2 on a 10-point numerical scale and had

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Figure 1.
Right Adductor, Transverse Proximal View. A large calcific mass measuring 7.7 x 1.8 x 0.29cm in the proximal adductor longus muscle is present.
regained full ROM of his hip. Post treatment diagnostic ultrasound demonstrated a decrease in the size of the calcification to 6.8 x 1.4 cm (Figure 2).

Case 2
A 45-year-old male horseback rider presented to a chiropractor approximately one month after being kicked in the right lateral quadriceps muscle by a horse. The pain was described as sharp and was rated as an 8 on a 10-point numerical scale. This patient presented with a mild limp and atrophy of his right quadriceps. Active and passive ROM of the right knee displayed 75% reduction in knee flexion and pain with adduction and abduction. Diagnostic ultrasound confirmed MO in the right vastus lateralis due to a finding of calcification in the muscle measuring 13.6 x 3.7 x 1.2 cm (Figure 3). In agreement with the findings on ultrasound, the same measurements were found on radiographic examination. The patient was treated with the same protocol as the patient in case 1 with acetic acid phonophoresis. After the plan of management, a follow-up ultrasound showed that the calcification had not decreased with an updated measurement of 14 x 3 x 0.61 cm (Figure 4). His pain score went from an 8 to a zero on a 10-point numerical scale. On the lower extremity functional scale (LEFS) the patients scored increased from 37 on an 80-point scale to 68 demonstrating significant improvement in function corresponding with full gains in ROM.

Case 3
A 24-year-old male soccer player presented to a chiropractor one year after injuring his right hip while kicking...
a soccer ball. The injury has never been treated and gets re-aggravated with cutting, forceful kicking, and sprinting. Active ROM of the right hip created pain with full flexion and resisted ROM displayed pain with hip flexion and adduction. Diagnostic ultrasound confirmed MO in the right iliacus muscle due to a finding of calcification in the muscle measuring 2.7 cm (Figure 5). In agreement with the findings on ultrasound, measurements of the calcification on radiographic examination measured 1.8 x 1.1 cm. The patient was treated with the same protocol as the patient in cases 1 and 2. After the plan of management, a follow-up ultrasound showed that the calcification had decreased to 2.3 cm (Figure 6). The patient also has pain free ROM.

Discussion

The incidence of myositis ossificans ranges from 0% to 9% for mild contusions to 17% to 72% for moderate-to-severe contusions suggesting that the more severe the contusion, the greater likelihood of progressing to MO. Rates increase with severity and with recurrent injuries to the same location. Myositis ossificans traumatica is a benign proliferation of cartilage and bone in skeletal muscle that has been exposed to trauma. A direct blow to a limb causes compression of the deep muscles to the adjacent bone. Within 24 hours, bleeding occurs between and within muscle. The calcification can be from 2 up to 12 cm and can be debilitating in an athlete. MO of a muscle can develop after a muscle contusion, tear, or wound and a bony fracture although there have been a few cases presented in the literature of MO that have not been associated with any trauma and must be differentiated from a malignant mass.

The exact pathogenesis of MO formation is unclear but has been thought of as rapidly proliferating mesenchymal cells that differentiate into osteoblasts in the presence of localized tissue anoxia, producing abnormal bone and cartilage growth. Also, trauma to muscle leads to prostaglandin synthesis, which attracts inflammatory cells to the site of injury, fostering the formation of bone. Four theories of pathogenesis have been suggested in the literature, 1) displacement of bony fragments into adjacent soft
tissues and hematoma with subsequent proliferation, 2) detachment of periosteal fragments into surrounding tissues with proliferation of osteoblasts, 3) migration of subperiosteal osteoprogenitor cells into adjacent soft tissues through periosteal tears due to trauma, and 4) metaplasia of extraosseous cells exposed to growth factors derived from the breakdown of bone fragments displaced within soft tissue during trauma. The fourth theory is the most commonly reported along with a hematoma as an important precursor.9

A patient with MO presents with local pain, warmth, ecchymosis, and swelling at the site of ossification for one week without a decrease in symptoms. Inability to bear weight or limited function of the adjacent joint may be evident depending on severity. MO should be suspected in patients who have had major direct trauma to muscle and have not responded to conservative treatment after 5 days or have worsening symptoms after 2 weeks.1 Associated stiffness and decreased ROM is evident by 1-2 weeks in any joint related to the involved muscle and can significantly impact athletic performance. Within 1-2 months, a solid palpable mass appears with muscle atrophy.2 MO usually affects young active males between 10-30 years of age, involved in contact sports.2,4,8 It is most common in the quadriceps, gluteal muscles and biceps brachii, although it has been reported in the psoas, hand, neck, shoulder, hip, calf, deltoid ligament, and pterygoid muscles.4,5,8-14 The diagnosis of MO is made based on imaging and histological examination.

Classification of MO based on its radiographic appearance is 1) flat bone formation adjacent to the shaft of bone with damage to the periosteum (periosteal), 2) bone formation that remains attached to the shaft of bone with damage to the periosteum (stalk), 3) intramuscular bone formation without disruption of the periosteal sleeve (intramuscular or disseminated).1

The goal of examination is to rule out other causes of a palpable mass. Differential diagnoses for MO include osteosarcoma, malignant fibrous histiocytoma, osteochondroma, foreign-body granuloma, giant cell tumor of soft tissue (osteoclastoma), atypical fibroxanthoma, pseudosarcomatous fibromatosis (nodular fasciitis), and deep vein thrombosis.2,15,16 Criteria to help differentiate these cases from a malignant osteosarcoma are 1) MO is located close to the diaphysis of the bone, whereas osteosarcoma is located near its metaphysis and 2) in MO, pain and swelling increase during the early phase, then subside gradually, whereas in osteosarcoma, both pain and swelling increase slowly, but continuously.8 In 1958, Ackerman described three zones in the lesion, 1) central zone (fibroblasts); 2) middle zone (osteoblasts that deposit trabeculae of woven bone); 3) outer zone (well-formed mineralized trabeculae).2 The three zones are identified via imaging and differentiate MO from malignant tumors giving a peripheral calcified appearance and a radiolucent centre.10 Occasionally, there is difficulty differentiating calcification and ossification, and a biopsy can be performed displaying the same 3 zones described above and to differentiate a benign lesion from a malignant one.5,17

Radiographs will show floccular calcified density in the soft tissues 2 to 6 weeks from the disease onset; within 6 to 8 weeks the calcification becomes well circumscribed and ossifications begin to adhere to the periosteum. A peripheral calcified appearance and a radiolucent centre is diagnostic of MO on radiographs.8 It is also important to note that the cortex and periosteum remain intact.8 Ultrasound can pick up MO as early as one week and before an x-ray based on changes in the soft tissue.18 Similar to its histological appearance, ultrasound shows a homogenous hypoechoic soft tissue mass with a circumscribed border and central reflective core. The mass appears oval on longitudinal images and round on transverse images. The peripheral calcifications as seen on radiographs is also seen on ultrasound along with its ultrasound-specific finding of lamellar calcification. Earlier phases show neovascularization, a center of decreased echogenicity with no clear zonal demarcation and no calcification while later phases show ossification with a more reflective rim and a distal acoustic shadowing. Although in the muscle belly, no damage to muscle fibers is seen. Ultrasound can also be used for guided surgical excision.19,20

MRI has been useful in the acute stages of the disease but radiographs are more beneficial in later stages.2 On T2, MO is seen with a high signal intensity core and a low signal intensity rim representing the zonal pattern of the condition. The specificity of MRI increases when injecting MO with gadolinium but radiographs remain the diagnostic tool of choice due to the inability of MRI to detect soft-tissue calcification.8,20

Computed tomography (CT) has been shown to be the imaging modality of choice in diagnosis and surgical
treatment in terms of identifying the exact location of the lesion with an axial image and, it is the best imaging modality to show the zonal pattern of MO.9,20

Triple-phase bone scans are helpful in the early detection of MO and are typically positive a few weeks before bone mineralization is detected on radiographs. They are also helpful in determining the maturity of the lesion once a diagnosis has been made.1

In 2010, Beck et al. wrote a case report in which he utilizes and recommends the use of hybrid imaging with single photon emission tomography (SPECT) and CT helped to confirm the diagnosis of MO in the paraspinal cervical muscles. It provides anatomical imaging with CT and the ability to determine the activity of the lesion via SPECT.10

Further confirmatory studies include blood work. Serum alkaline phosphatase and erythrocyte sedimentation rate levels are elevated in patients with MO.1

All of the cases presented in this series were athletic men, from 24-45 years of age, presenting with pain or decreased ROM. In our cases we chose to image patients with radiographs and diagnostic ultrasound, which showed calcifications from 2.7-14 cm in size. Based on the history of trauma and the radiographic and ultrasound findings all patients were diagnosed with MO. Cases 1 and 3 similarly report re-aggravation of a pre-existing muscle injury. This suggests trauma to an injured area may predispose an individual to developing MO. No imaging was performed at the time of initial injury so the presence of calcification prior to re-aggravation is unknown. The patient in case 2 presented to the chiropractor 4 weeks after being kicked by a horse. This coincides with the timeline of progression from a muscle contusion to MO. A palpable mass along with muscle atrophy was evident in this patient at presentation, which typically presents around one month post trauma.

For treatment of MO, the literature suggests immobilization, ice, elevation, rehabilitation, ultrasound, cold laser, iontophoresis, non-steroidal anti-inflammatory drugs, therapeutic injections, and surgical excision. Rehabilitation of patients with MO should include restoration of ROM, strength and proprioception.2 Heat, continuous ultrasound, massage, stretching, and exercise should be avoided initially to prevent further bleeding in the area that may promote the progression to MO.5 Protective padding should be worn to prevent further injury to the area.6 NSAIDs have been shown to be beneficial if used for at least 2-6 weeks and the literature suggests that if implemented immediately after injury, it would further decrease the risk of progression to MO.6 When swelling has subsided and the patient is pain free, light active and passive ROM exercises are recommended while those with lower limb contusions are able to bear weight. Progressions for functional rehabilitative exercises are made within patient tolerance. When patients have regained near full ROM and strength they can progress to sport specific activities. Throughout conservative care, patients must be monitored for a palpable mass or other signs of MO. If the mass is not painful, rehabilitation may be continued while continuous monitoring for pain and growth is performed.6

Elbow mobilizations have shown to be effective at restoring normal ROM and sports specific function in a basketball player with MO of his brachialis muscle. The patient presented with a severe flexion contracture and a palpable mass in his brachialis confirmed as MO on radiographs. Joint mobilizations allowed for the gains in ROM with out overstressing the brachialis muscle.21

A recent non-controlled study by Buselli et al. looked at the effect of extracorporeal shockwave therapy (ESWT) for the treatment of MO. Twenty-four athletes with radiographic and MRI confirmed MO were given 100 shocks per cm2 of ossification, every 2 weeks for 3 sessions at an intensity of 0.15 mJ/mm2. Concurrently, subjects were participating in a physiotherapy program consisting of ROM, progressive muscle strengthening, and proprioceptive exercises. Post intervention, only partial reduction of the ossification was observed on radiographs but all patients showed signs of functional improvement immediately after treatment. At 2 months post intervention, normal ROM and strength was observed. At 3 months post intervention, 87.5% of patients resumed regular sports activities. The theory this method of treatment is that ESWT creates mechanical stimulation to the tissues, inducing bleeding and ultimately, tissue repair.2 Torrance and deGraauw also found success with a treatment plan consisting of icing, progressive stretching and strengthening, and ESWT on a 20 year-old male semi-professional rugby player with MO in his right vastus lateralis. The patient was able to return to full sport participation after three ESWT treatments.22

In the presented cases, a 2% acetic acid solution was
administered via phonophoresis into the MO (8 minutes of pulsed ultrasound at 1.5 W/cm²), 3 times per week for 4 weeks. This method was chosen based on a study by Wieder in 1992 on a case of a 16-year-old male with MO in his quadriceps from a diving accident. The patient participated in a 3-week physical therapy program consisting of a 2% acetic acid iontophoresis treatment followed by pulsed ultrasound and mild ROM movements. Results showed 98.9% decrease in the size of the patient’s MO based on radiographic measurement. The patient also regained full knee ROM and was able to return to play without pain. As a follow-up to Weider’s case, Gard and Ebaugh (2010) performed acetic acid iontophoresis (2% solution) as a primary method of treatment in a case of a 19-year-old college, club hockey player with MO in his brachialis muscle, diagnosed through clinical examination conducted by an orthopedic surgeon. He was referred for physiotherapy where he received 9 treatments over 29 days consisting of acetic acid iontophoresis and active ROM exercises. After his treatment plan the patient went from a significant decrease in elbow extension and inability to play hockey to full ROM and back to full sport participation.

Iontophoresis is the application of, physiologically active ions through the skin using continuous direct current. The theory of iontophoresis was first described by Le Duc in 1908 as an electrical charge that will repel a similarly charged ion. Psaki and Carroll (1955) and Kahn (1977) described the use of acetic acid iontophoresis for treatment of calcium deposits as effective in reducing the size of calcium deposits through the absorption of calcium due to the negative polarity of the acetate ion found in acetic acid. Before completely ossifying, MO usually consists of precipitates of calcium carbonate that are non soluble in normal blood pH levels. It has been proposed that the acetate radical replaces the carbonate radical in the insoluble calcium carbonate deposit, forming a more soluble calcium acetate. Other clinical implications for acetic acid iontophoresis mentioned in the literature are, calcific joint deposits, frozen shoulder, and heel spur formation. There currently are no studies on acetic acid phonophoresis but theory is similar to that of iontophoresis except the application of the ions is directed through the skin via sound waves instead of an electrical current.

Although most cases of MO are managed conservatively, 3 cases in the literature went on to have surgery. One of the cases requiring surgery was due to an intra-articular MO in the knee associated with significant ROM loss and its unique attachment to the anterior cruciate ligament and the 2 other cases being MO in the masseter muscle affecting temporomandibular joint function. Surgery may be necessary with significant decreases in ROM, muscle atrophy, unrelenting pain, and deterioration of function after 6-12 months of unsuccessful conservative care. It is important to note that there have been reports of exacerbation of symptoms, prolonged disability, and recurrence of MO after surgery. To prevent recurrence, MO cannot be operated on until the lesion has reached complete maturity. A case series by Lipscomb et al presented 4 football players all necessitating surgical removal of a MO. All athletes reported satisfactory recovery and were able to return to play. After surgery, NSAIDs are recommended for up to 6 weeks followed by a similar rehabilitation protocol as mentioned above for those managed conservatively.

In our cases, all of the patients experienced residual calcification, and one with no change in the size of the calcification at all. This residual calcification is found in most other cases presented in the literature, similar to this report, with no further pain or limitations in strength and ROM.

As previously stated, the natural history of MO is 1-2 years. Conservative care may shorten this healing time. A shortened healing time is crucial in an elite athlete and therefore, surgical consultation may be considered sooner in these individuals.

All of the cases presented in this report had significant improvements on range and function within 4 weeks of conservative treatment. Based on these results, acetic acid phonophoresis can significantly improve pain and ROM caused by calcification in a muscle with or without reduction in the size of the lesion. Because the lesion did not fully absorb, we cannot be certain that the decrease in size of the MO on radiograph was not due to natural history. No long-term follow-up has been reported so we do not know the long-term effects of phonophoresis but we know that it may create benefits in the short-term and should therefore be considered as a treatment option for MO by manual therapists.

Research on conservative care is necessary for chiropractors and other manual therapists to appropriately manage patients with MO. Proving to be effective in this
References: