

Conservative management of posterior interosseous neuropathy in an elite baseball pitcher's return to play: a case report and review of the literature

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This report documents retrospectively a case of Posterior Interosseous Neuropathy (PIN) occurring in an elite baseball pitcher experiencing a deep ache in the radial aspect of the forearm and altered sensation in the dorsum of the hand on the throwing arm during his pitching motion. The initial clinical goal was to control for inflammation to the nerve and muscle with active rest, microcurrent therapy, low-level laser therapy, and cessation of throwing. Minimizing mechanosensitivity at the common extensor region of the right elbow and PIN, was achieved by employing the use of myofascial release and augmented soft tissue mobilization techniques. Neurodynamic mobilization technique was also administered to improve neural function. Implementation of a sport specific protocol for the purposes of maintaining throwing mechanics and overall conditioning was utilized. Successful resolution of symptomatology and return to pre-injury status was achieved in 5 weeks. A review of literature and an evidence-based discussion for the differential diagnoses, clinical examination, diagnosis, management and rehabilitation of PIN is presented.
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KEY WORDS: radial nerve, posterior interosseous nerve, radial neuropathy, baseball, arm injury.

Ce rapport documente rétrospectivement un cas de neuropathie interosseuse postérieure (NIP) s'étant produit chez un lanceur élite au baseball qui avait une forte douleur dans la face radiale de l'avant-bras et une sensation modifiée de la face dorsale de la main du bras qui lance lors du mouvement du lancer. Le premier objectif clinique était de contrôler l'inflammation du nerf et du muscle avec un repos actif, un traitement par micro-courant, une thérapie au laser à faible niveau et la cessation des lancers. On a réussi à minimiser la mécano-sensibilité à la région commune de l'extenseur du coude droit et à la NIP par un soulagement des douleurs myofasciales et des techniques d'augmentation de la mobilisation des tissus mous. La technique de mobilisation neurodynamique a également été administrée pour améliorer la fonction neurale. On a mis en place un protocole propre aux sports aux fins de maintien des mécaniques de lancer et de conditionnement général. La résolution réussie de la symptomatologie et le retour à l'état qui prévalait avant la blessure ont été réalisés en cinq semaines. Une revue de la littérature et une discussion fondée sur des preuves pour les diagnostics différentiels, l'examen clinique, le diagnostic, le traitement et la réadaptation pour la NIP sont présentées.
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MOTS CLÉS : nerf radial, nerf interosseux postérieur, neuropathie radiale, baseball, blessure au bras

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Introduction

Posterior Interosseous Neuropathy (PIN) is a peripheral nerve injury commonly characterized by a sensation of a deep ache in the posterior forearm which can be accompanied by weakness of the forearm extensors and brachioradialis, and/or sensory alterations, or a combination of both. In the upper extremity PIN occurs with less frequency than median and ulnar mononeuropathies.¹ Among athletes, the incidence of PIN is unknown. Mechanical soft tissue injuries can present in a self-limiting nature and with similar symptomatology (pain and weakness) as a peripheral neuropathy which can make differentiating each condition a challenge.²

This report highlights a case of PIN in an elite baseball pitcher associated with throwing. Many case reports of peripheral neuropathies unresponsive to conservative care typically involve surgical management.^{3,4} Other neuropathies of a repetitive nature provide recommendations of rest, nonsteroidal anti-inflammatory medications, physiological modalities, and rehabilitation regimes in the early stages of management.

Case Report

A 21 year-old male, right-handed pitcher, drafted to play professional baseball presented with an insidious onset of pain, fatigue, and altered sensation of the right posterior forearm of 1-week duration. This pain was experienced while pitching, more specifically during follow-through, both at maximal and submaximal efforts. The onset of pain and fatigue commenced with the initiation of pitching and progressively worsened with throwing to a maximum of 35 pitches which resulted in the discontinuation to pitch. The pain was rated as a 6/10 on the visual analogue pain scale and was described as a deep ache in the radial, posterior compartment of the forearm. The pain subsided upon cessation of throwing. The patient reported an altered sensation over the dorsum of the thumb and index finger, and acknowledged that the wrist and finger extension felt "weaker and more fatigueable" since the onset of the pain. The patient reported no previous injury of this nature to the throwing arm. The patient denied any history of trauma, surgery, or previous injury to the right upper extremity and/or neck.

Upon visual inspection, no swelling, redness, ecchymosis, deformity, spontaneous muscle activity (i.e. fibrillations), or muscle wasting of the forearm when compared

to the non-throwing arm was observed. Active and passive ranges of motion of elbow extension elicited difficulty attaining a bony end-feel at end range of extension due to tightness in the antecubital fossa. Elbow flexion for all range types was within normal limits and a muscular end feel obtained. All active and passive ranges of motion of the wrist and hand were also within normal limits, while supination was limited at the end range actively due to the patient verbalizing discomfort in the proximal radial forearm. Passive forearm pronation combined with wrist flexion elicited patient discomfort in the proximal radial forearm. Resisted wrist extension with the elbow extended reproduced the patient's chief complaint.

Direct palpation of the supinator was markedly tender with a "snapping band" effect over the Arcade of Froshe. Proximal to the elbow joint, on the lateral aspect of the distal humerus, direct palpation of the brachioradialis and brachialis elicited tenderness. Palpation over the radial nerve branches that transverse the elbow joint (see figure 1) proved to be non-pain provoking. Joint play of the radiocapitellar and ulnotrochlear joints proved to be unremarkable and non-pain provoking. Direct palpation over the common extensor origin was unprovocative.

Orthopaedic testing revealed Cozen's Test (resisted wrist extension from a flexed elbow and pronated and extended wrist position) reproduced the patient's chief complaint. Repeat muscle testing, using a 15-pound dumbbell resulted in progressive fatigueability of the wrist extensors. A Modified Cozen's Test (modification with the elbow fully extended) also reproduced the chief complaint producing greater fatigueability and discomfort in the radial forearm. Pain and fatigueability subsided within minutes of stopping the test. Mill's Maneuver (passive extension of the elbow from a flexed, pronated forearm and flexed wrist/finger position) elicited tightness in the forearm and proximal to the elbow joint. Active cervical ranges of motion and Maximal Cervical Compression test (cervical spine lateral flexion, extension, and vertical compression) were unprovocative. Shoulder examination was unremarkable for any capsular pathology, rotator cuff pathology, acromioclavicular joint pathology, or glenohumeral labrum pathology. Elbow examination was unremarkable for Valgus-Overload Syndrome, Postero-lateral instability, and ligamentous insufficiencies. Thoracic outlet syndrome testing was deemed to be unremarkable for reproduction of symptomatology.

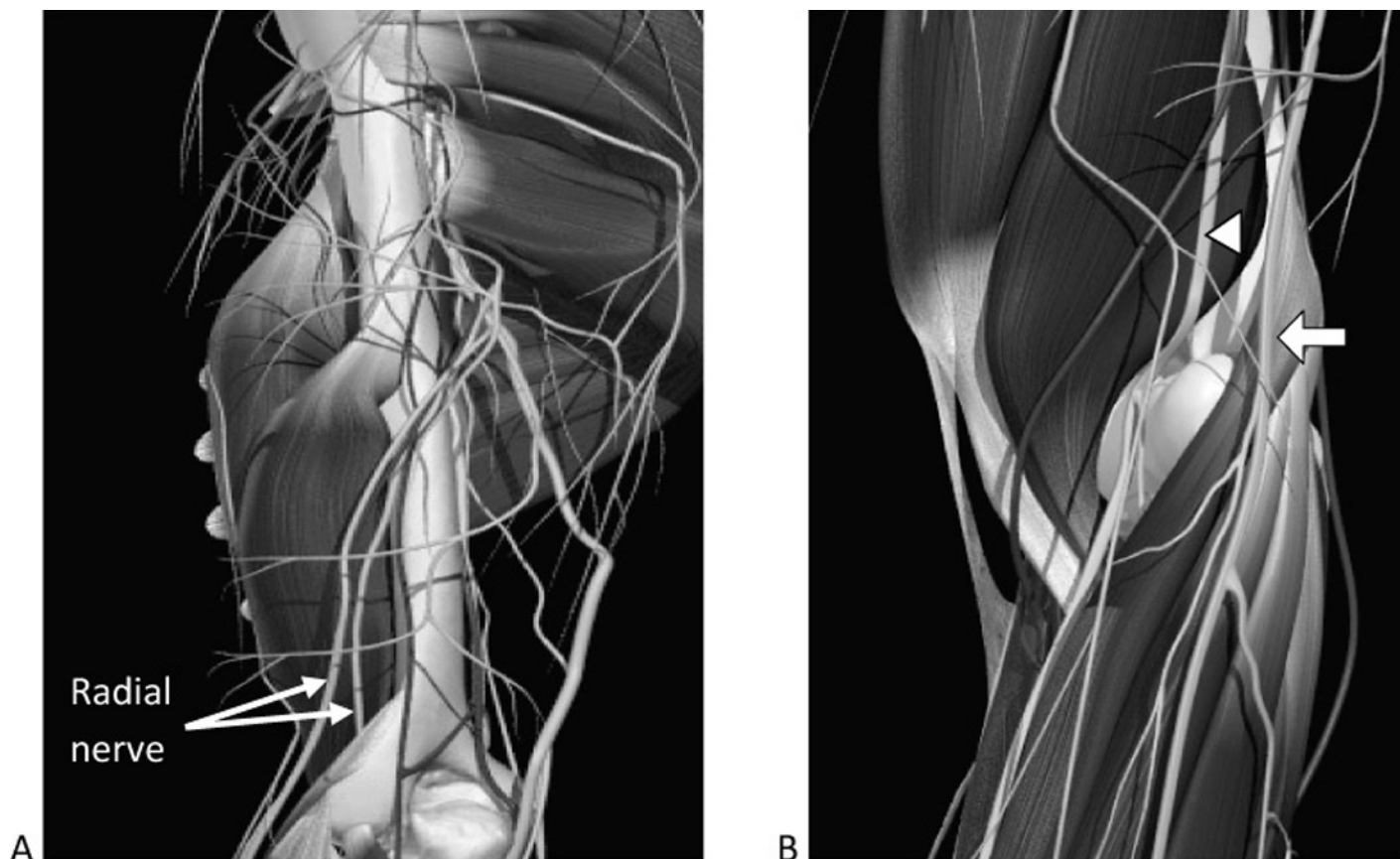


Figure 1 *Graphic depiction of the Radial Nerve at the distal humeral and elbow joint. (A) Radial Nerve branches coursing along the posterior compartment of the upper arm superficial to the brachioradialis. (B) Radial Nerve trunk and its branches (posterior interosseous (arrow head) and superficial radial (small arrow) nerves) coursing distally through the elbow and forearm region. (3D anatomy images copyright Primal Pictures Ltd.)*

The Upper Limb Tension test for the radial nerve trunk was used to assess the radial nerve as described in previous studies^{5,6} (see figure 2). The protocol involves shoulder girdle depression with the elbow flexed to 90 degrees; this is followed by forearm pronation, elbow extension, wrist and finger flexion, shoulder abduction and cervical spine lateral flexion to the opposite side (see figure 1). Reproduction of the patient's chief complaint and altered sensation over the dorsum of the radial 3 digits which included the thumb was achieved. Pin sensation (sharp and dull) was symmetrical and intact over the dorsum of the thumb and index finger, the antero-lateral border of the forearm, and the postero-lateral border of the forearm. Po-

sition and vibratory senses were present, deep tendon reflexes were 2+ and symmetric in the upper extremity.

The clinical impression was Posterior Interosseous Neuropathy, as a first degree injury or neuropraxia, with an associated myofasciopathy of the supinator, brachioradialis, and brachialis which was consistent with the history and clinical findings.

The patient was treated six times over two weeks with soft tissue therapy (Active Release Techniques), augmented soft tissue mobilization technique (Graston Techniques) as presented in figure 3, and low level laser therapy to the affected muscles. Neural gliding was employed (figure 4) for the radial nerve. Mobilizations with

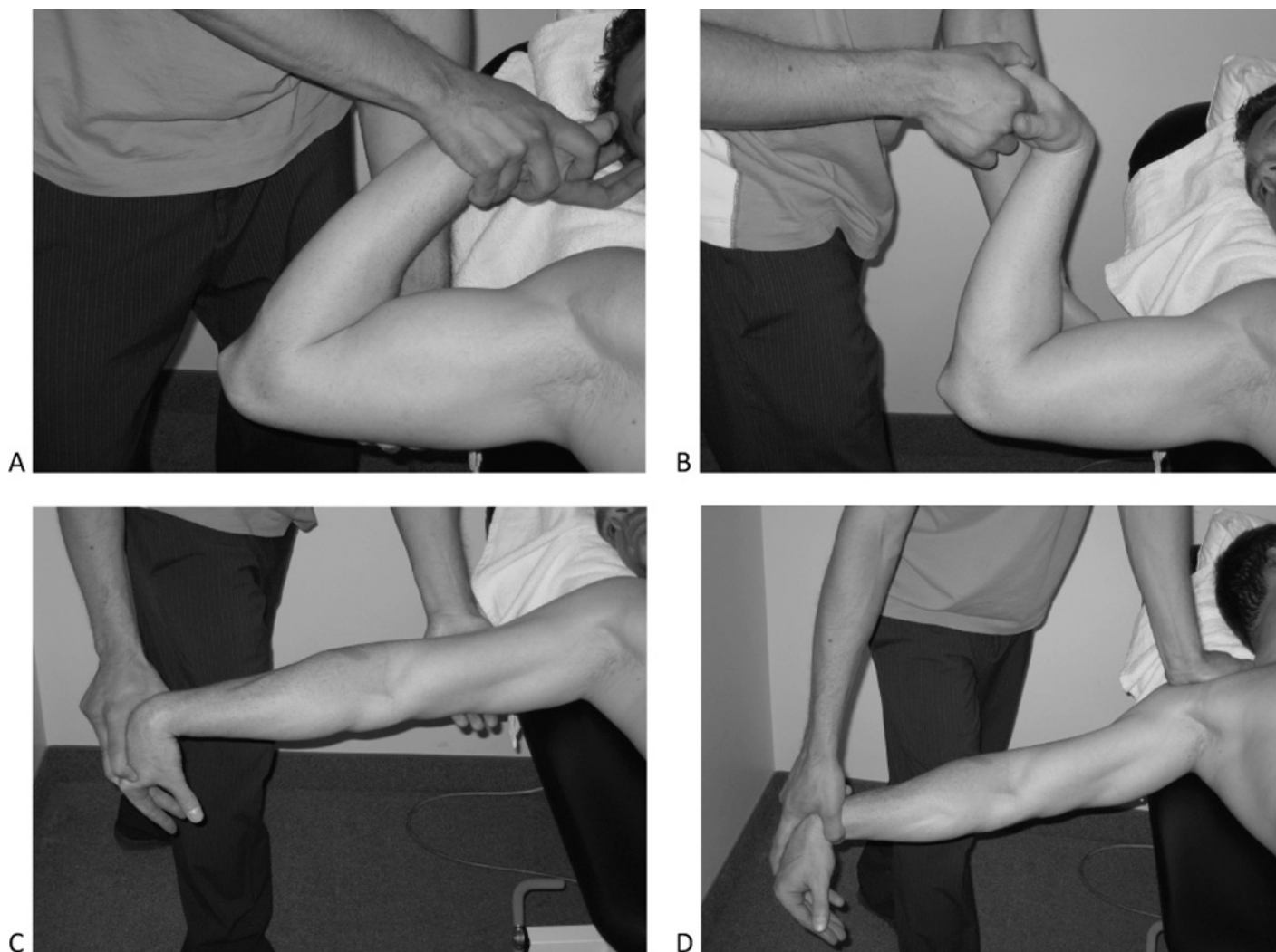


Figure 2 *Upper Limb Tension Test: Radial Nerve Protocol. (A) Starting Position: shoulder is in 90 degrees of abduction, elbow in full flexion, the forearm is fully pronated, the wrist and fingers placed in full extension and ipsilateral lateral flexion of the cervical spine. (B) Initiation of motion is first conducted by placing the fingers and wrist in to full flexion while maintaining the elbow, forearm, shoulder, and cervical spine positions. (C) Elbow extension and forearm pronation is started while still maintaining the shoulder in 90 degrees of abduction. (D) The shoulder is depressed and the cervical spine is laterally flexed to the contra-lateral side.*

movement were administered to the radiocapitellar and ulnotrochlear joints. Rehabilitative exercises for the right elbow and forearm were performed simultaneously with microcurrent therapy applied to the radial tunnel and distal course of the radial nerve. A sport specific rehabilitation program was performed to maintain conditioning of the lower extremity, core, and throwing shoulder. In-

structions were to avoid pitching during the treatment period, except for light throwing to focus on appropriate motor control and technique.

Having a clear prognosis and clearance to participate in a baseball try-out camp is dependent upon having a healthy arm to pitch.

Upon the third visit the patient was referred to his



Figure 3 Myofascial Release of the Elbow employing (A) Active Release Technique and (B) Graston Technique.

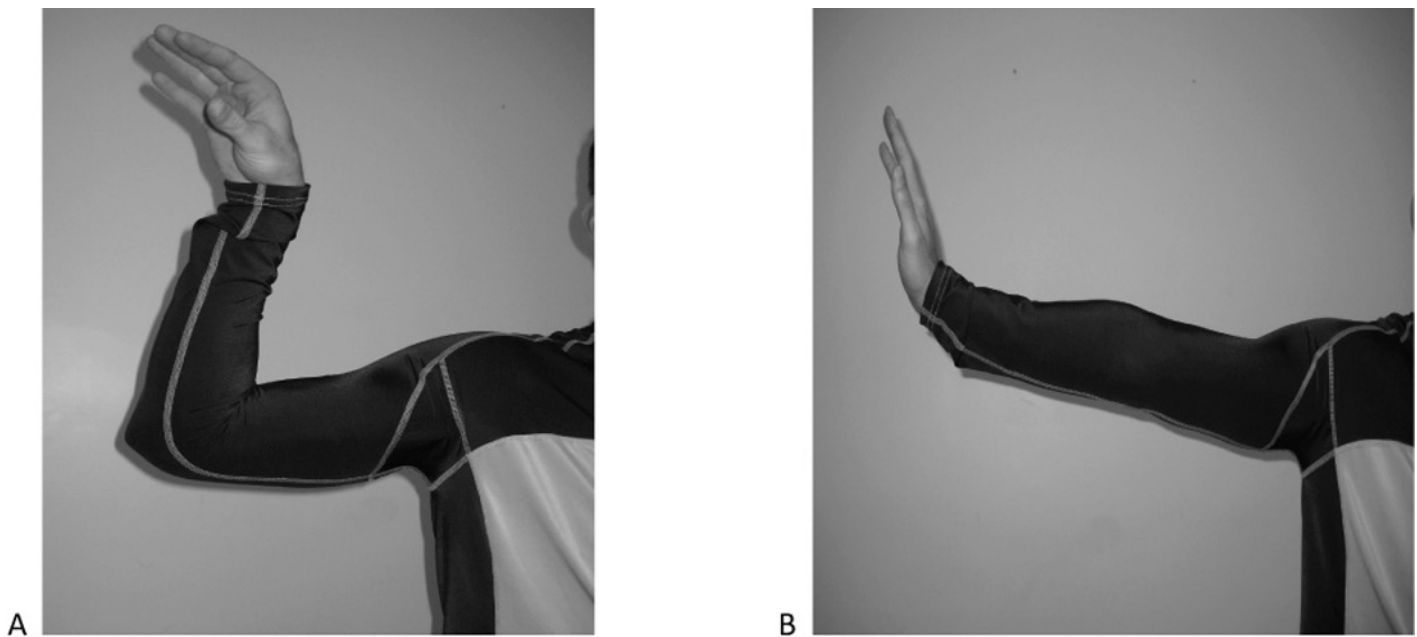


Figure 4 Neuromobilization Technique for the radial nerve. (A) Start with the wrist and fingers in flexion, elbow in flexion, shoulder at 90 degrees of abduction. This is intended to permit maximal radial nerve excursion at the wrist and less at the elbow joint. (B) Commence by placing the wrist and fingers into extension and the elbow into extension. This is intended to permit maximal excursion of the radial nerve at the elbow joint and less at the wrist joint.

medical doctor for a nerve conduction study to confirm the severity and ascertain prognosis of the injury. The nerve conduction analysis measuring the amplitudes of the motor action potentials was graded as within normal limits. It should be noted that patient's right radial nerve conduction velocity value was graded as within lower end-range of the normal limits measuring 53 m/s but this result was still above what is typically viewed upon as pathological (a decrease of equal or greater than 20%). This nerve conduction velocity study revealed a slight decrease in values from the norm for both the sensory and motor conduction velocities of the radial nerve at the elbow of the throwing arm when compared to the normative population ranges (64 m/s).⁷ The remaining nerve conduction velocity study was normal and no advanced diagnostic imaging was taken.

The patient attended a scout camp in the third week, for 10 days, with minimal reproduction of symptoms in the posterior forearm and had his pitches measured over 85 mile per hour on average. Upon return from the camp, the patient was treated four additional times over a 14 day period with the same treatment plan as described above. At the end of the last treatment (38 days post first visit), the patient experienced a pain free throwing motion. The patient no longer reported any stiffness in the elbow, wrist extensor strength was powerful and absent of fatigueability during dumbbell testing, and his pitches were regularly measured at speeds over 90 mph which was a velocity previously achieved prior to injury.

Discussion

Peripheral neuropathies have been described in case reports and reviews among baseball pitchers involving the suprascapular nerve,² axillary nerve,² ulnar nerve,² musculocutaneous nerve,⁸ and the lateral femoral cutaneous nerve.⁹ In baseball pitchers, nerve injury has been cited in the literature accounting for approximately 2% of all diagnosed shoulder injuries.² There is a paucity of literature describing the occurrence of radial neuropathy in baseball and athletes alike. As a result, the incidence of radial neuropathy in athletes is unknown. The majority of the literature assessing the management of peripheral neuropathies consists predominantly of surgical nerve decompression.^{8,9} This case report is a first to document a variant radial neuropathy in a baseball pitcher with conservative management.

The prevalence of nerve injuries are found to be rare among throwing athletes and can be a challenge to diagnose as a result of overlapping presentations for muscle pain and weakness. Consequently, delayed diagnosis can compromise prognosis with progressive neurological sequelae and deterioration of pitching performance. Even with resolution, determining the plan of management in the absence of definitive identification of tissue damage is limited.² Understanding how pitching exposes the nerve to insult will assist with tissue identification and direct treatment initiatives.

The proposed mechanism for injury in this case is the mechanics during the late stages of the throwing motion, the follow-through. The follow-through is defined from maximal shoulder internal rotation to a balanced fielding position after the ball has been released.^{10,11} This is the phase of throwing that exhibits high levels of eccentric loading at the forearm along and along with repeated supination-pronation as a potential effect that places stress to the soft tissues of the throwing arm.¹²

The muscles involved with the deceleration phase are the rotator cuff muscles, triceps brachia, brachialis, supinator, and forearm extensor muscles. These muscles undergo eccentric loading that can theoretically compress or apply excessive tensile loading along the course of the radial nerve. This theory has not been substantiated in the literature, however observing the similarity of the position of the follow-through and the upper limb tension test for the radial nerve (see figure 5) suggests a plausible mechanism for assault to the radial nerve. This requires further investigation to demonstrate a relationship between radial nerve injury and the follow-through in pitching.

The pathophysiology of nerve injuries can be instigated by mechanical events involving repetitive movements as suggested with throwing or through direct blunt trauma.^{13,14} The effect, either tensile or compressive, on the nerve stimulates the *nervi nervorum*, causing disruption of the microvasculature, and the deformation of the connective tissue. Irritation potentiates a transient inflammatory response (i.e. macrophagic activity) that can induce chemosensitive and immunosensitive reactions with prolonged exposure producing a noxious response to be perceived as pain.¹³ Furthermore, the product of swelling is a consequence of compromised microcirculation at the endoneurial level and may lead to hypoxia, endoneurial edema, and restricted axoplasmic flow.¹⁴ Endoneurial edema



Figure 5 Comparison of the Follow-through (A) and the Upper Limb Tension Test for the Radial Nerve (B). The extension that is observed in the follow-through is similar to the extension necessary to test the radial nerve as presented in the Upper Limb Tension Test.

accumulation is compounded by the lack of lymph tissue present at this level of the nerve to facilitate flow of fluids.¹⁵ Continued mechanical exposure, in this case throwing, potentiates increased nerve mechanosensitivity, propagating inflammatory responses inflicting chemosensitivity to the nerve and the surrounding musculature.^{14,15} The manifestation can result in dysesthesia, hyperesthesia, hypoesthesia, paresis, and/or paresthesia. Clinical presentations include dysfunction, fatigueability, and disability. Muscular tightness also occurs as a result of excessive fibrosis, neural budding, and reflexive muscle tone to protect the injured nerve from further damage. Management involves eliminating any mechanical stimuli that are prevalent to reduce irritation and promote proper healing.

Differential diagnostic considerations of pathology afflicting the radial forearm were ruled out on the basis of the history, physical examination, and nerve conduction

study. An exertional posterior forearm compartment syndrome was ruled out in the absence of pressure, disproportionate pain, and paresthesia. Cervical discogenic and/or radiculopathy were ruled out with a normal cervical and neurological screen. A possible referral from the rotator cuff was ruled out on the basis of normal palpatory findings, resisted muscle testing and specific orthopaedic testing. A suspicion of a stress reaction or fracture to the radial head and neck was not considered as local pain was not identified. Suspicion of a local space occupying lesion (i.e. ganglion cyst, lipoma, intraneurial neuroma) should always be suspected with neuropathies but a normal nerve conduction study and relatively quick resolution of symptomatology assisted with ruling this out. Consideration of a possible entrapment or compression of the nerve should be suspected when examining this patient, however only diagnostic imaging (magnetic resonance imaging or diag-

nostic ultrasound) or surgical exploration is definitive.^{16,17} The clinical examination does suggest the consideration of a concomitant grade I muscle strain of the supinator, brachioradialis, and extensor carpi radialis longus (ECRL) as a result of the patient reporting pain upon palpation and reduced ranges of motion.

A neuropathy is typically defined as a nerve injury that exhibits neuro-pathophysiological adaptations, lacks a definitive pathophysiological cause (entrapment/compression, neuroma, ganglion cyst, lipoma, etc.) as confirmed on advanced diagnostic imaging (MRI, ultrasound), aggravated with movement and/or unequivocal electrophysiological testing.¹⁵ In addition, the presentation of neurologically related symptomatology can be categorized into Sunderland's or Seddon's Classification of a nerve injury (Table 1). Nerve "Entrapment or Compression" can be definitively identified during surgery when the nerve is dissected and observed to demonstrate the constricted region that is associated with the neurological presentation.^{16,17} However, there are protocols manual therapists perform that attempt to identify and localize the area of suspected "entrapment" or irritation (i.e. Upper Limb Tension Tests).^{5,6}

The current understanding for conservative management of peripheral neuropathy emphasizes reducing mechanosensitivity to the peripheral nervous system and restoring neurodynamic function.³¹ A fundamental understanding of the pathophysiology and the magnitude of involvement between nonneural tissue (muscle, fascia) and neural tissue are necessary.^{13,15}

Treatment to restore non-neural tissue function can consist of a variety of soft tissue techniques consisting of myofascial release and augmented soft tissue mobilization. Myofascial release (Active Release Techniques ©) has been successfully employed for various soft injuries: lateral epicondylitis,¹⁸ dorsal interosseous strain,¹⁹ hamstring tightness,²⁰ and trigger thumb.²¹ Augmented soft tissue mobilization (Graston Technique ©) has been used in the treatment of thoracic spine dysfunction,²² trigger thumb,¹⁸ costochondritis,²³ and subacute lumbar compartment syndrome.²⁴ These two techniques are theorized to breakdown adhesive and fibrotic deposits at the level of the tissues, restore physiological range of motion, and provide biofeedback through manual application or instrument assisted applications thereby reducing noxious stimulation. The theoretical mechanism of action suggests

that stimulation at the site of the injury increases the concentration levels of satellite cells (mesenchymal cells with highly differentiating capabilities) that differentiate into myoblasts to facilitate the production of healthy connective tissue.²⁵ The implementation of myofascial therapy has demonstrated resolution of symptomatology within 4 weeks.¹⁸

Physical modalities can be employed to control the inflammatory response and promote healing of the nerve through the utilization of microcurrent therapy and low-level laser therapy directed over the nerve trunk between the BR and Brachialis and radial tunnel. Microcurrent is an electrotherapeutic modality inducing a subsensory electric charge to the skin surface producing no sensation during treatment. Microcurrent has been shown to significantly reduce pain thresholds over a 5 day period.²⁶

Low level laser therapy (LLLT) involves applying wavelengths of energy, between 630 and 1064 nm, at varying doses to penetrate specific depths.²⁷ The conditions known to be treated with LLLT include tendinopathy,²⁸ arthropathies,²⁹ and myofasciopathies.³⁰ Laser has been shown to increase collagen synthesis and reduce pain thresholds in as little as 10 and 21 days respectively since the start of treatment.²⁷⁻³⁰

Neuromobilization was implemented using the nerve gliding technique designed to facilitate the enhancement and restoration of nerve mobility through the numerous nerve beds (see figure 3). Nerve gliding is strategic joint movements designed to elongate the nerve bed at one joint while simultaneously reducing the length of another nerve bed at an adjacent joint. Nerve gliding exhibits greater excursion of the nerve through the nerve bed than nerve tensioning.³¹ Nerve gliding is theorized to apply less tension to the affected nerve because of the segmental partitioning of the nerve beds to minimize the ectopic axonal discharge (i.e. fibrillations, dysesthetic pain) and to permit extravasation of intraneural edema and reduce symptoms.³¹ Caution is warranted with excessive nerve tensioning as this can impede microcirculation and exacerbate neural symptomatology.³²

A common rehabilitative strategy prescribed to overhead athletes is the "Ballistic Six" exercises as suggested by Pezzullo and colleagues.³³ These exercises were used as rehabilitative and preventative strategies. The concept emphasized that throwing should involved both eccentric and concentric movements that mimicked the various

Table 1 *Classification of Nerve Injuries*

Seddon's	Sunderland's	Dysfunction	Pathology	Mechanism	Prognosis
Neuropraxia	Type 1	Focal conduction block – motor, proprioception most common; Sensory, sympathetic loss	Myelin injury of large fibers Axons intact No Wallerian degeneration	Electrolyte imbalance; Disruption of myelin; Ischemia due to compression or traction	Transient to hours or days (anoxic or ionic imbalances); Mechanical compression or stretch take weeks to months
Axontomesis	Type 2	Loss nerve conduction at and below injury site	Disrupt axon with Wallerian degeneration; connective tissue intact	Compression	Recovery of function proportionate to distance from end of nerve
	Type 3	Loss of nerve conduction at and below injury site	Axon continuity and endoneurial architecture lost. Peri/epineurial structures intact	Compression	Scarring due to endoneurial deformation, edema, and hemorrhaging; poor prognosis; potential surgical intervention
	Type 4	Loss nerve conduction at and below injury site	Near complete deformation of nerve architecture, except for perineurium	Compression	Intraneural scarring and axonal misdirection; Poor prognosis requiring surgical intervention
Neurotomesis	Type 5	Loss of nerve conduction at and below injury site	Complete deformation of nerve architecture	Traction, laceration, compression	Recovery necessitates surgical resection, with incomplete acquisition of function.

Adopted from Zachazewski, Magee, and McQuillen. Chapter 20 Peripheral Nerve Injuries, page 658.³⁶

phases of the pitching motion. The sport specific movement patterning and conditioning is also suggested to prevent injury and enhance ball velocity. This upper extremity plyometric regime has been demonstrated to increase concentric internal rotation and eccentric external rotation and pitching velocity (~2 mph) in 8 weeks. This

regime was performed for 3 sets of 10–20 repetitions, 2 days per week. The exercises included: latex tubing external rotation, latex tubing 90/90 external rotation, overhead soccer throw using a 6-lb medicine ball, 90/90 external rotation side-throw using a 2-lb medicine ball, deceleration baseball throw using a 2-lb medicine ball,

and baseball throw using a 2-pound medicine ball. This protocol has been implemented in a similar manner with collegiate players.³⁴ Modification of this program was implemented to avoid the throwing based exercises to minimize irritation to the throwing arm until full range of motion and strength testing was pain free. This program was implemented to maintain sport specific movement patterns and conditioning of the upper extremity.

The limitations of this case report are those inherent with the type of study design.³⁵ This study lacked objectivity by not using a dynamometer to assess muscular strength. The subjective nature by which the muscle strength was assessed was not incorrect to clinically identify muscle weakness when compared to the unaffected forearm; notwithstanding it does not accurately validate and reliably assess fatigueability pre and post conservative management. The absence of diagnostic imaging to visualize the nerve injury site did not permit definitive correlation of symptomatology with pathophysiological changes to the nerve. This limitation results in the use of the categorization of the injury as a neuropathy to generalize the clinical entity even though the presentation is consistent with a neuropraxia/grade I nerve injury. Furthermore, because of multi-modal approach it is difficult to ascertain the contributions of each therapy mode would have on the early recovery. Finally, the natural history of the injury cannot be ruled out as an explanation for the resolution of symptomatology in a short time frame. Finally, this is the first report of conservative management of a variant radial neuropathy and important to understand that generalizing the results of this report are individualized to this particular patient.

This case report demonstrated the successful application of soft tissue therapy, electrophysiological interventions, neuromobilization techniques, and rehabilitative regimes. The goal was to restore an elite pitcher to pre-injury status afflicted with a neuropraxia of the PIN due to a repetitive use mechanism associated with baseball pitching. To provide assistance to clinical decision-making practices with neuropathy presentations the results of this case are for hypothesis generation in advanced research methodology trials involving the non-surgical management of neuropathies.

Conclusion

Peripheral nerve injuries in pitchers are rare. Diagnosing

peripheral nerve injuries that present with only muscle weakness and pain can be challenging as mechanical muscle injuries can present concomitantly. This case of Posterior interosseous Neuropathy was a diagnosis of exclusion, upon ruling out various pathologies that afflicting the forearm. Uncertainty of the true pathophysiology (i.e. compression or entrapment causing nerve injury) was evident yet imaging was unnecessary as the resolution of symptomatology was swift with conservative management. The application of a multimodal conservative approach to managing a peripheral neuropathy can be a potential option for treatment but further research investigations should be implemented as this may be a viable alternative to surgery.

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