12-1-2021

# Isolated Anterior Interosseous Neuropathy Affecting Only the Flexor Digitorum Profundus to the Index Finger After Shoulder Arthroscopy: A Case Report and Review of the Literature

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# Isolated Anterior Interosseous Neuropathy Affecting Only the Flexor Digitorum Profundus to the Index Finger After Shoulder Arthroscopy

## *A Case Report and Review of the Literature*

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**Abstract:** Anterior interosseous nerve neuropathy is an uncommon neuropathy with multiple potential etiologies. We present a rare case of anterior interosseous nerve neuropathy affecting only the flexor digitorum profundus to the index finger and occurring after shoulder arthroscopy. This unique presentation used a combination of both electrodiagnostic testing and neuromuscular ultrasound to obtain an accurate diagnosis and highlights the importance of these complementary tests in the evaluation of nerve disorders. To our knowledge, anterior interosseous nerve neuropathy after shoulder arthroscopy affecting only the flexor digitorum profundus to the index finger has not been previously described in the literature.

**Key Words:** Anterior Interosseous Nerve, Ultrasound, EMG, Case Report

(*Am J Phys Med Rehabil* 2021;100:e188–e190)

The anterior interosseous nerve (AIN) branches from the posterior aspect of the median nerve 1.5–8 cm distal to the intercondylar line and innervates the flexor pollicis longus (FPL), flexor digitorum profundus (FDP), and the pronator quadratus (PQ).[1] Anterior interosseous nerve neuropathy is an uncommon phenomenon. Multiple etiologies exist including trauma, entrapment, iatrogenic injury, isolated neuritis, and neuralgic amyotrophy.[2,3] The most common etiology is a subject of debate. An AIN neuropathy leads to clinical weakness of the FDP to the index and middle finger, the FPL, and the PQ. The most common presentation in AIN neuropathy is weakness or absence of FPL function and pain in the volar forearm. The AIN is frequently affected in neuralgic amyotrophy (also known as Parsonage-Turner syndrome), involved in 35.3% of cases in one retrospective study.[4]

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Financial disclosure statements have been obtained, and no conflicts of interest have been reported by the authors or by any individuals in control of the content of this article.

Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's Web site (www.ajpmr.com).



ISSN: 0894-9115
DOI: 10.1097/PHM.0000000000001829

Postoperative AIN neuropathy after shoulder arthroscopy is rare but has been described in the literature. The etiology of AIN neuropathy after shoulder arthroscopy remains elusive. Therefore, guidelines for the diagnosis and management of this condition are not currently standardized. The following case illustrates an atypical presentation of AIN neuropathy after shoulder arthroscopy. This study conforms to all case report guidelines and reports the required information accordingly (see Supplemental Checklist, Supplemental Digital Content 1, http://links.lww.com/PHM/B335).

## CASE REPORT

A 45-yr-old man presented for electrodiagnostic evaluation for right finger weakness. Onset of his weakness was 9 mos prior, after a right arthroscopic posterior labral repair, superior labrum anterior posterior tear repair, subscapularis repair, and glenohumeral debridement. Postoperatively, he received an interscalene ropivacaine 0.2% block performed by regional anesthesia for pain relief. The block was removed on postoperative day 3. At the 2-wk postoperative mark, the patient was experiencing right-sided elbow and forearm pain along with stiffness. He was unable to actively flex the distal interphalangeal joint of his right index finger. The patient had full passive range of motion at the affected joint. The arm pain and stiffness improved as he weaned out of his shoulder sling and began therapy. At his 6-wk follow-up, he was still unable to actively flex the distal interphalangeal joint of the index finger. It was recommended that he be evaluated by hand surgery. At his initial visit with the hand surgeon, the patient was also complaining of new paresthesias along the anatomic lateral/volar distal forearm. However, these paresthesias resolved by the time of electrodiagnostic testing. An electromyography (EMG) was pursued 9 mos after his shoulder arthroscopy. Examination performed at the time of the EMG revealed 0/5 strength in the right FDP to the index finger and 5/5 strength in the right FDP to the middle, ring, and small fingers, as well as the FPL. Sensory examination of the right upper limb was normal.

Electrodiagnostic testing was performed using a Cadwell Sierra Summit (Kennewick, Washington). Testing was performed by a fellow physician under the supervision of a physiatrist board certified in electrodiagnostic medicine and neuromuscular ultrasound (US, American Board of Electrodiagnostic Medicine) with 8 yrs of experience with musculoskeletal US. Nerve conduction studies (NCSs) included the right median antidromic sensory recording from the index finger (14 cm), right radial antidromic sensory recording from the thumb (10 cm), and right ulnar antidromic

sensory recording from the small finger (14 cm). The right ulnar motor NCS to the abductor digiti minimi (8 cm) and right median motor NCS to the abductor pollicis brevis (8 cm) and FPL (8 cm) were within normal limits. Ultrasound examination was then performed using a Sonosite X-Porte (Bothell, Washington) US machine with a 15-MHz linear transducer. The right median nerve was scanned from the level of the wrist and proximally to the elbow. Maximum cross-sectional area of the right median nerve at the wrist was 13 $mm^2$, which is mildly increased (laboratory normal ≤11 $mm^2$). This increase in median nerve cross-sectional area may represent a mild median neuropathy at the wrist; however, NCSs were within normal limits. No other regions of median nerve focal enlargement or loss of fascicular architecture were appreciated proximal to the wrist. Ultrasound was used for accurate needle EMG localization of the FDP, FPL, and PQ muscles. Prescan was performed to identify the FDP muscle belly in the transverse plane at the midforearm with the patient's supinated hand lying flat on the examination table. Individual slips of the FDP were identified by performing passive and active flexion-extension at the distal interphalangeal joint while blocking the PIP joint. Ultrasound revealed hyperechoic changes in the FDP slip to the index finger, consistent with chronic denervation atrophy (Fig. 1). Needle was introduced into the muscle belly using an out-of-plane approach. Needle EMG revealed increased insertional activity with positive sharp wave and fibrillation potentials in the FDP slip to the index finger. There were no motor unit action potentials appreciated in this muscle. The remaining AIN-innervated muscles tested (FDP to the middle finger, FPL, and PQ) were within normal limits. Electromyography was consistent with a right AIN mononeuropathy affecting solely the branch to the FDP slip of the index finger. The lesion appeared complete with no evidence of nerve continuity on needle EMG.

## DISCUSSION

Anterior interosseous nerve neuropathy after shoulder arthroscopy is not a well-established complication but is becoming increasingly recognized. To our knowledge, there are currently 16 reported cases of AIN neuropathy after shoulder arthroscopy in the literature, which includes the previously mentioned case report.[5–9] The potential etiologies for this condition, diagnostic workup, and management strategies are reviewed hereinafter.

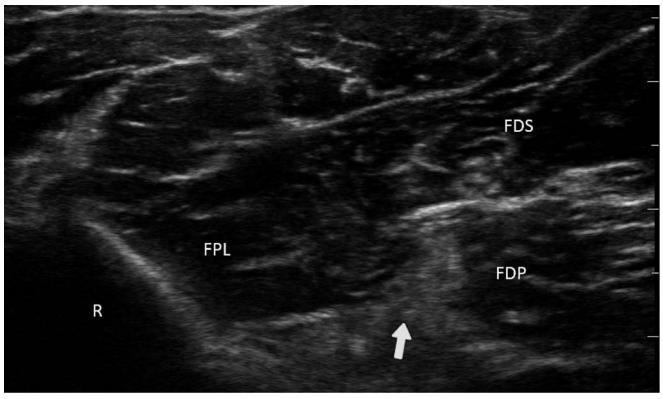


**FIGURE 1.** Needle EMG of the FDP to the index finger in short axis. Arrowhead, EMG needle tip within the hyperechoic flexor digitorum profundus index finger; FDS, flexor digitorum superficialis; R, radius.

Interscalene regional blocks were performed for analgesia in 11 of the 16 cases. Peripheral nerve blockade can lead to mechanical, vascular, or chemical nerve injury.[10] Mechanical injury is dependent on the needle type, size, site, and pressure achieved.[11] With regard to vascular injury, damage to the vasa nervorum can cause local ischemia. Local anesthetics have also been shown to reduce nerve blood flow.[10,12] The neurotoxic effects of anesthetics appear concentration and duration dependent. The site of injection, intrafascicular versus extrafascicular, determines the extent of nerve damage.[10,11] However, in a prospective study including 660 patients receiving an interscalene block for surgical purposes, only two patients developed motor weakness at follow-up, one of which was diagnosed with an AIN neuropathy.[13]

Lateral decubitus positioning was implemented in at least 9 of the 16 cases. The lateral decubitus position has been associated with transient paresthesias and neurapraxic injury, with incidence ranging from 10% to 30%.[14] The widely used semiupright beach chair position implemented in the current case has decreased this risk.[15] Traction was implemented in an estimated 10 of the 16 cases. Anterior interosseous nerve neuropathy after shoulder arthroscopy may represent a traction-type injury. Applying our current knowledge of AIN neuropathy after supracondylar fractures, the AIN is prone to traction injury. The proximal median nerve proper is free moving when compared with the AIN in cadaveric models. This is partially due to an anchoring effect from its interosseous portion.[16,17] A study on sensory evoked potentials during shoulder arthroscopy supports this theory. Results revealed that joint distension and traction caused abnormal sensory evoked potentials in the upper limb. Abnormal sensory evoked potentials were most pronounced in the musculocutaneous nerve, with variable involvement of the median, radial, and ulnar nerves.[18]

To our knowledge, no cases of AIN neuropathy with isolated involvement of the FDP to the index finger after shoulder arthroscopy have been described in the literature. However, "incomplete" spontaneous AIN neuropathies have been recognized. In 1 retrospective study, 7 of 14 patients had incomplete AIN neuropathy with isolated loss of function of the FPL. Two of the 14 patients presented with isolated involvement of the FDP to the index finger.[19] The lack of neurogenic findings in the FDP to the middle finger may be related to an anatomical anomaly. A study evaluating 50 cadaveric specimens contradicted the standard textbook innervation of the FDP. The most common innervation pattern (50%) in this study revealed that all four slips (second–fifth) of the FDP were innervated by the median nerve, with dual innervation from the ulnar nerve occurring in the third–fifth slips.[20] The possibility of dual innervation of the FDP to the middle finger may explain the lack of denervation in our current case.

In a prospective study implementing magnetic resonance neurography in patients with spontaneous AIN neuropathy, an organized somatotopic internal fascicular pattern was identified within the median nerve at the level of the upper arm. Based on anatomical localization, the fascicles affected were the motor fascicles to become the AIN distally.[21] A smaller case series implementing high-resolution US identified three patients with AIN neuropathy in the setting of neuralgic

amyotrophy. Each case presented with fascicular enlargement of the median nerve above the elbow.[22] Both magnetic resonance neurography and high-resolution US have identified proximal median nerve enlargement, hourglass constrictions or torsions, and fascicular intertwinement in AIN neuropathy.[21–23] Based on the currently available knowledge, spontaneous AIN neuropathy is likely immune-mediated inflammatory in origin, similar to that described in neuralgic amyotrophy.[21] The current case may be autoimmune-inflammatory, involving only the fascicles of the AIN to the index finger. Under this theory, shoulder arthroscopy could have served as the inciting immunologic event.

A diagnostic EMG was reported in 12 of the 16 cases, with variable impressions. There are several indications for the application of EMG in AIN neuropathy. Electromyography assists in localizing the injury and helps exclude diagnoses, such as multifocal motor neuropathy with conduction block or neuralgic amyotrophy. The lack of a more thorough evaluation of the brachial plexus was a limitation in our current case. Our patient's transient sensory symptoms may have been related to involvement of the lateral antebrachial cutaneous nerve in the setting of neuralgic amyotrophy, and a lateral antebrachial cutaneous sensory NCS should have been completed. Other peripheral nerves commonly affected in neuralgic amyotrophy (i.e., suprascapular or long thoracic nerves) should also be evaluated. However, clinically, these did not seem to be involved at the time of his EMG. Electromyography can also rule out a coexistent peripheral entrapment neuropathy, anatomical anomaly (i.e., Martin-Gruber anastomosis), or cervical radiculopathy. As discussed earlier, there is potential anatomical variability in the pattern of innervation to the FDP between the median and ulnar nerve, which can lead to discrepancies in examination findings and presumptive EMG findings.[20] Electromyography categorizes the severity of the injury and assists with prognostication. Serial EMG studies with careful clinical examination assist in determining whether a lesion is due to neuropraxia versus axonotmesis, which is important for management.[24] In our case, EMG revealed no motor unit action potentials to the FDP to the index finger at 9 mos, supporting a poor clinical outcome.

Ultrasound was implemented for needle localization of the FPL, PQ, and FDP. Compared with anatomical landmark technique, US has been shown to improve the diagnostic accuracy of EMG.[25] Given the atrophic changes of the FDP to the index noted on US, blind needle localization would have been very difficult to perform. Ultrasound also rules out an underlying flexor tendon rupture or tendon adhesions. This is of particular importance with incomplete lesions of the AIN. With respect to management, the AIN was surgically explored in 11 of the 16 reported cases. The indications for operative treatment for spontaneous AIN neuropathy remain controversial. No studies currently exist, which compare conservative versus surgical management. Current experts recommend serial electrodiagnostic testing and observation. If no clinical improvement occurs after 3–6 mos, surgical neurolysis or nerve transfer should be considered.[26] Electromyography, US, and magnetic resonance neurography help with both the diagnosis and decision-making process. The presence of AIN torsions or fascicular constrictions seems to support an unfavorable outcome in the literature. The presence of which may serve as a future indication for surgical intervention.[22] Tendon transfer can also be considered to improve hand function if the previously mentioned treatment approaches are ineffective.

## CONCLUSIONS

Anterior interosseous nerve neuropathy after shoulder arthroscopy is becoming an increasingly recognized complication, albeit still rare. This case highlights the added value of neuromuscular US to electrodiagnostic testing in reaching an accurate diagnosis.

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