



Case Report

Unique case of anterior interosseous nerve injury following distal volar radial plating: A case report

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ABSTRACT

Trauma itself, surgery, nerve entrapments, amyotrophic neuropathy, or neuritis may result in anterior interosseous nerve (AIN) palsy. A 76-year-old woman who was operated for distal end radius fracture with plating was referred back referred back to the clinic by her general practitioner with complaints of inability to flex the interphalangeal joint of the thumb and index finger approximately 3 years after the index surgery. The proximal interphalangeal joint flexion (flexor digitorum superficialis) of the index finger was intact and the pinch power was reduced. She had normal function of her other forearm and hand muscles. Ultrasonography and nerve conduction velocity (NCV) studies were done to investigate further to rule out either tendon flexor pollicis longus (FPL) or nerve injury (AIN). NCV studies reported subacute denervation within the FPL. The patient recovered to near normal without any surgical intervention in 18 months. Literature reported various treatment options in the form of conservative as well as surgical depending on the cause and evidence of recovery.

Keywords: Anterior interosseous nerve injury, Fracture, Kiloh-Nevin syndrome neuritis, Paralysis, Palsy

INTRODUCTION

Tinel (1918) was a pioneer who described anterior interosseous nerve (AIN) palsy as “dissociated paralysis of the median nerve.”^[1] Later on, Feinberg and Radecki reported the same in patients with neuralgic amyotrophy and a few years later, Kiloh and Nevin described it as isolated neuritis.^[2,3] AIN palsy associated with supracondylar fracture was described by Lipscomb and Burleson and as an entrapment syndrome by Fearn and Goodfellow.^[4,5] However, literature reported that such palsy accounts for <1% of all compression syndromes in the upper extremity.^[6] Rask observed pain as the earliest symptom of such entrapment neuropathy.^[7] However, brachial neuritis can be differentiated from compression neuropathy, as motor symptoms are usually preceded by arm or forearm pain/viral illness in neuritis.^[8] Moreover, such pain is not a predictive sign for differentiating an inflammatory from a mechanical cause. The patients frequently had inability to form an “O” with the thumb and index finger due to paralysis of flexor pollicis longus (FPL) and flexor digitorum profundus (FDP). Sometimes, pronator quadratus (PQ) is also paralyzed, but goes unnoticed by the patient and it is difficult to elicit with clinical examination.

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The involvement of AIN following fractures of the distal radius is quite rare. Most traumatic causes of AIN palsy have been reported due to supracondylar humerus, proximal humerus, or shaft or proximal ulna fractures.^[9-11] We want to convey a message to trauma surgeons that delayed AIN palsy can occur post-surgery following distal end radius fractures. Early surgical exploration of the nerve is indicated in entrapment neuropathy, however, in an isolated neuritis, conservative treatment is usually preferred. Following surgery, any suspected iatrogenic injury may require reexploration and repair of the nerve. The temporal relationship of the onset of palsy to the surgery is important in defining a possible injury from the approach.^[12-14]

CASE REPORT

A 76-year-old lady sustained a closed bilateral distal radial fracture following a fall in her garden [Figure 1]. This lady had been previously diagnosed with deep venous thrombosis and was treated with warfarin. A thorough examination was documented by an orthopedics resident before her admission, with no motor or sensory deficit reported. Warfarin was stopped 5 days before surgery and started after 3 days after surgery.

She underwent operative fixation of her left side fracture using a volar approach through the bed of flexor carpi radialis under general anesthesia. The distal radius was exposed and reduced under vision. The fracture revealed a long thin radial styloid fragment and a larger distal radial fragment. A stable fixation was accomplished (using a standard sized, Acu-Loc® Volar Distal Radius Plate, Acumed®, Hillsboro, Oregon, USA) [Figure 1]. She made a good recovery postoperatively and her distal neurovascular status was intact before discharge. She was discharged with a below-elbow back slab (as per protocol, we give backslab to all our patients operated for distal radius fractures for a week which is taken off by physiotherapist) and discharged to hand physiotherapist for rehabilitation. The patient was regularly followed up with physiotherapists till 1 year and then discharged to general practitioner (GP) without any complaints.

She was referred to the fracture clinic by her GP 3 years post her index surgery with complaints of inability to flex the interphalangeal joint of her left thumb and index finger [Figure 2]. She gave a history of gradual onset of weakness in pinch that started 2 years after the surgery which was initially managed by the GP with pain killers and pregabalin. Pain was insidious in onset and progressed over a period of 8–12 months, limiting her pinch severely and was referred to us for further management.

The flexor digitorum superficialis muscle to the index finger was intact (MRC grade 5) and the tripod pinch power was reduced as compared to the other side on manual muscle



Figure 1: Anteroposterior and lateral radiographs pre-operative and following plate fixation.

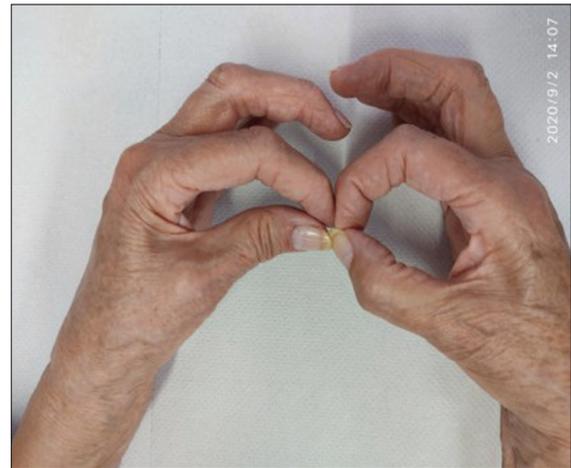


Figure 2: The left side loss of flexor pollicis longus function and weakness of flexor digitorum profundus in the index finger.

testing. The patient had normal function of her forearm and hand muscles as well as no tenderness at watershed line. The symptoms were gradual in onset and progressed in the sense that she had been finding it difficult to perform some of her tasks (unbuttoning, holding cup of tea, etc.) as well as with her usual daily living activities. She did not report any history of trauma or jerk activities or any history of fever or illness preceding these symptoms.

A presumptive diagnosis of FPL rupture or AIN palsy was made, and ultrasound was requested to look for any tendon rupture or compression neuropathy. Ultrasonography

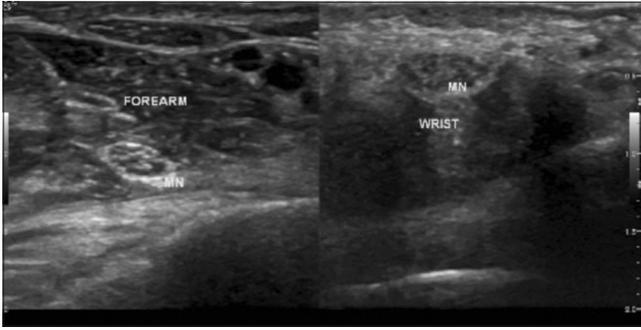


Figure 3: Ultrasound image of forearm and wrist.

reported no obvious neural compression or disruption of AIN nerve [Figure 3].

To further investigate the pathology, neurophysiology studies were done which reported subacute denervation within the left FPL although there was evidence of signs of some reinnervation albeit with a reduced motor function within the left FPL. Similarly, there was a reduced motor function within the anterior interosseous component of the FDP but again with evidence of physical continuity and some degree of function within the FDP albeit that this is significantly reduced. These features were compatible with a subacute axonal lesion affecting the AIN.

The patient was treated conservatively (activity modification, soft-tissue massage, and exercise) and reassurance. The patient was not keen for another surgery and wanted to explore non-operative management whatever time it may take. We agreed to wait for 1 year if no recovery then surgical treatment options (nerve grafting/tendon transfer) were discussed. During 6–8 months of conservative treatment under physiotherapist, her symptoms improved. The patient got near normal recovery around 18 months after she presented to us, and was happy with the final outcome.

DISCUSSION

AIN palsy can occur due to atraumatic or traumatic causes. The lesion of AIN palsy can be “complete” or “incomplete.” A patient with a complete palsy would present with no motor function to the FPL, FDP to the index and middle fingers, and PQ, whereas only the FPL or the FDP of the index finger is paralyzed in incomplete palsy.^[14]

Some of pathological conditions might mimic AIN palsy like, flexor tendon rupture or adhesion, stenosing tenosynovitis, Parsonage-Turner syndrome, and a brachial neuritis.^[2,15,16] After trauma, it is quite difficult to distinguish an isolated palsy of the FPL muscle from other local pathological conditions.^[16] Mostly, electrophysiological studies are used to diagnose AIN palsy (electromyography may show denervation). These can be used to diagnose as well as ascertain the severity of neuropathy. Moreover, magnetic resonance imaging can be used to confirm

the diagnosis of neuropathy as well as any local pathology around the nerve.^[15,17,18]

The treatment of AIN palsy is mostly related to the etiology and pathology. Literature review reported both conservative and operative treatment options for AIN palsy. Conservative treatment in the form of avoidance or modification of activity, immobilization, and anti-inflammatory medication is a viable option to be explored before any operative intervention. However, the time to improvement to appear varies from several weeks to months.^[15,19] Nevertheless, some researchers have recommended to follow conservatively up to 1 year as a spontaneous partial to complete recovery may take 9 and 24 months. Seki *et al.* recommended operative intervention only after 1 year of conservative management.^[20]

We searched the literature and found that in patients with complete paralysis of either muscle-tendon unit and who have shown no improvement (clinical and/or EMG/NCV) after 1–2 years of clinical observation (depending on the case), exploration and neurolysis of the AIN are recommended as it offers a rapid and often a complete recovery. Spinner advocated exploration if no signs of clinical and electromyographic improvement occur within 6–8 weeks,^[6] Nigst and Dick recommended it to exactly 8 weeks,^[12] and Ulrich *et al.* prolonged it to 12 weeks.^[19] Tendon transfer option is reserved, if motor function does not recover after 12 months.^[21] The flexion of interphalangeal joint of the thumb is restored using brachioradialis muscle. The transfer of the tendon of FDP of the ring or middle finger to that of the index finger at the wrist can provide satisfactory flexion of the distal phalanx of the index finger.^[22] Schantz and Riegels-Nielsen recommend delay in the use of tendon transfer until 1 year after the onset of palsy.^[23]

The patients with paresis should be observed probably for a longer time. However, regardless of severity, a complete spontaneous recovery may occur in all patients, it is concluded that atraumatic AIN lesions are most likely to show a circumscribed form of brachial neuritis and that surgical decompression should be deferred for at least 12 up to 24 months or even indefinitely if progressive recovery is evident. In short, conservative treatment is recommended for neuroamyotrophy or neuritis, whereas decompression is advised for compression syndromes. The most important pitfall is that there are no clinical signs and symptoms to differentiate between these lesions. Another noteworthy point to remember is to keep the patients under physiotherapy to keep the joint and muscles functional until AIN recovers. Moreover, a very late recovery of the atraumatic AIN palsy is possible, so it is advised to continue with non-operative treatment modalities for longer time before any aggressive surgical intervention is planned.

CONCLUSION

Trauma surgeons should be aware of the anatomy and course of AIN in the forearm while fixing fractures. Special care should be taken while examining patients with such nerve injuries as these are easily missed by registrars or junior doctors. Surgical intervention is only reserved for cases where conservative treatment fails or no signs of recovery.

RECOMMENENDATIONS

AIN palsy after trauma or surgical intervention may represent an injury to the nerve and should prompt further investigation and if warranted surgical exploration. Non-operative observation can be followed when there is a clinical diagnosis of a neuritis and a partial injury with evidence of progressive recovery within 3 months at clinical review. Most nerve injuries can be readily identified using a thorough clinical examination.

AUTHORS' CONTRIBUTIONS

GAS involved in collection, draft, and writing; NA concept and revising; JH gathering figures and revising; CK final revision; AA involved in drafting and revising. All authors have critically reviewed and approved the final draft and are responsible for the manuscript's content and similarity index.

Declaration of patient consent

The authors' certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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