CASE REPORT – OPEN ACCESS

International Journal of Surgery Case Reports 21 (2016) 44–47

Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.casereports.com

Anterior interosseous nerve syndrome diagnosis and intraoperative findings: A case report

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ARTICLE INFO

Article history:
Received 11 November 2015
Received in revised form 13 February 2016
Accepted 14 February 2016
Available online 20 February 2016

Keywords:
Anterior
Case report
Interosseous
Nerve
Neuropathy
Palsy

ABSTRACT

INTRODUCTION: Anterior Interosseous Nerve (AIN) is a motor branch from the Median nerve and runs deep in the forearm along with the anterior interosseous artery. It innervates three muscles in the forearm; an isolated palsy of these muscles is known as AIN Syndrome. There are several documented causes of AIN syndrome but its pathophysiology remains unclear.
PRESENTATION OF CASE: A 48-year old male that presented with right elbow pain and inability to flex his right interphalangeal joint of the thumb and the distal interphalangeal joint of the index finger. MR images denoted mild atrophy of the radial half of the flexor digitorum profundus and the pronator quadratus. Although there were no compressing lesions identifiable on MRI, Electrodiagnostic studies suggested compression neuropathy affecting the AIN. During surgical decompression of the median nerve in the proximal forearm, the operative findings were several tendinous fasciae and a tight fibrous arch of the flexor digitorum superficialis compressing the median nerve at the level of the AIN branch.
DISCUSSION: Different treatment schemes with reasonable outcome have been reported. Both nonsurgical and surgical intervention have been described in most of these schemes but differed in the timing of intervention with variable outcome.
CONCLUSION: Clinical suspicion should arise in the presence of isolated paralysis of the AIN-supplied muscles. MRI and electrodiagnostic studies will confirm the diagnosis and identify the etiology. The optimal treatment of AIN syndrome has not been established. We recommend surgical intervention in confirmed AIN syndrome from compression neuropathy, refractive to conservative therapy.
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1. Introduction

Anterior Interosseous nerve (AIN) syndrome is a rare syndrome that comprises less than 1% of all upper extremity nerve palsies, arising due to compression or inflammation of the AIN of the forearm [1]. The AIN is purely a motor branch of the median nerve that arises from its dorsomedial aspect, just inferior to the elbow. It is about 5–8 cm distal to the lateral epicondyle and 4 cm distal to the medial epicondyle. It passes distally, along the anterior interosseous membrane with the anterior interosseous artery. The AIN innervates three muscles in the forearm: flexor pollicis longus (FPL), pronator quadratus (PQ), and the radial half of flexor digitorum profundus (FDP) [2]. In the hand, the median nerve innervates 5 muscles: The 2 lateral lumbricals, opponens pollicis, abductor pollicis brevis, and flexor pollicis brevis [3].

2. Presentation of case

A 48-year old otherwise healthy male, presented to our orthopedic outpatient clinic complaining of a 1-week history pain in his right elbow associated with weakness in his right index finger and thumb. Physical examination showed weakness of the FLP and FDP to the index finger with a positive Pinch Grip test (Froment's sign). Examination of the interphalangeal (IP) joint of the thumb and distal interphalangeal (DIP) joints of the 2nd and 3rd digits all revealed powers (3/5), where the patient could overcome gravity, but not resistance. Full neurological and musculoskeletal examinations were otherwise normal. With the impression of AIN syndrome, the patient was initially treated conservatively with rest, analgesia with anti-inflammatory medications and physiotherapy for forearm flexor muscle stretching exercises and activity modification. Upon follow up 3-months later, the patient's symptoms progressed. Physical examination showed paralysis at the IP joint of the thumb and DIP joints at the 2nd and 3rd digits. Magnetic resonance imaging (MRI) scans of the forearm revealed abnormal high signal intensity of the both the PQ and the radial FDP uscles on proton density fat-saturated images (Fig. 1) and mild atrophy represented by some fatty streaks on T1 weighted images (T1WI)(Fig. 2).

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http://dx.doi.org/10.1016/j.ijscr.2016.02.021
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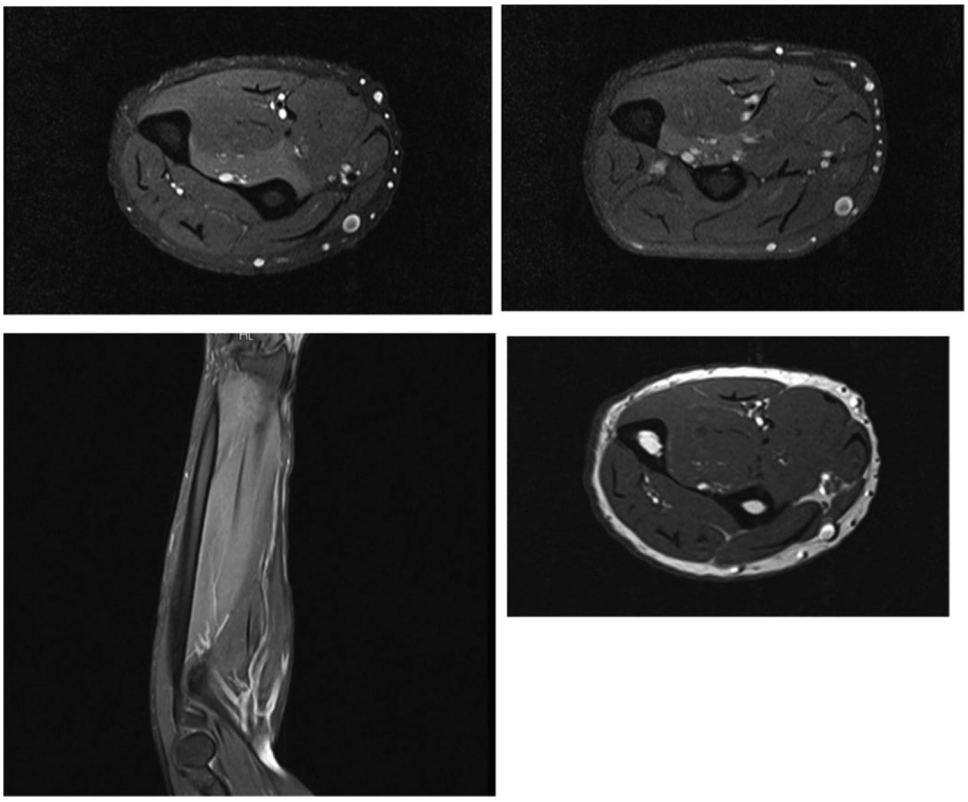

Fig. 1. Axial and sagittal fat-saturated proton density images demonstrating diffuse increased SI of the proximal portion of the flexor digitorum profundus muscle and axial T1WI demonstrates decreased muscle bulk and a few streaks of high SI within the flexor digitorum profundus muscle. Both images denote mild atrophy.

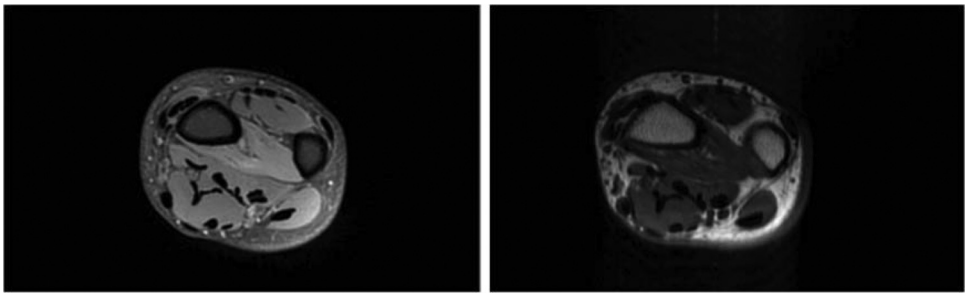

Fig. 2. Non-contrast MR images of the distal forearm: axial T1WI demonstrates few streaks of high SI seen within the pronator quadratus muscle and axial fat-saturated proton density image demonstrating diffuse increased SI of the pronator quadratus muscle. Both images denote mild atrophy.

Although there were no compressing lesions identifiable on MRI, electrodiagnostic studies by needle examination suggested compression neuropathy affecting the AIN as the cause of paralysis of the FPL, the radial FDP, and PQ muscles. After 3 months of failed conservative management, the patient underwent complete surgical decompression of the median nerve throughout its course in the proximal forearm through a lazy S-shaped incision over the volar forearm. With careful dissection from proximal to distal, the median nerve was exposed releasing the lacertus fibrosus, the deep head of the pronator teres muscle, the proximal fascial margin of the flexor digitorum superficialis arch and other possible fibrous bands or edges compressing the nerve. Several tendinous fasciae and a tight fibrous arch of the flexor digitorum superficialis were also found to compress the median nerve at the level of the AIN branch (Fig. 3), which were surgically released. Postoperatively, the patient was followed up for 6 months with slight improvements in his symptoms. His pain was well controlled with oral analgesia. His physical examination revealed power 2 at the IP joint of the thumb and DIP joints at the 2nd and 3rd digits, where the patient was able to flex with gravity eliminated

3. Discussion

Anterior interosseous nerve syndrome is a pure motor neuropathy. Nevertheless, associated dull pain in the forearm had been reported [4]. Patients with AIN syndrome are typically unable to form an “O” by using the index finger and thumb due to paralysis of FPL and the radial FDP (impaired flexion of the interphalangeal joint of the thumb and the distal interphalangeal joint of the index finger). The patients will lose the ability to button their shirts or turn on their car keys to start it, for example. On physical examination, the Pinch Grip test is positive where patients will not be able to demonstrate the “OK” sign, instead clamping the sheet between an extended thumb and index finger [5]. The absence of sensory deficits in AIN syndrome differentiates it from carpal tunnel syndrome and other nerve palsies (e.g., Pronator Syndrome,

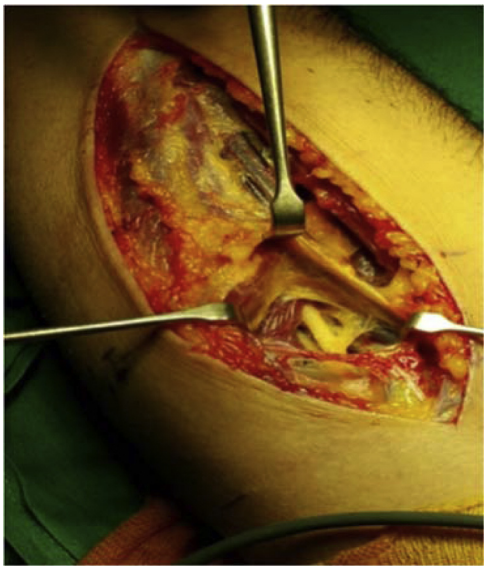

Fig. 3. Intraoperative photograph showing compression of the median nerve by tendinous fascia off the deep head of pronator teres and the fibrous arch of the flexor digitorum superficialis proximal to it.

Parsonage-Turner Syndrome), which usually lead to decreased sensation, tingling, or numbness in the upper limb. The FPL, the radial part of the FDP and PQ are affected in complete AIN syndrome. However, incomplete AIN syndrome occurs when isolated FPL or FDP of the index finger is either paretic or paralyzed [6]. An isolated weakness of the thumb may indicate isolated involvement of the particular fascicle that innervates the FPL [7].

There are several documented causes of AIN syndrome, the pathophysiology of which remains unclear. Etiological factors can be categorized into either traumatic or spontaneous [8,9]. Traumatic causes include: penetrating injuries, forearm fractures, venipuncture, cast fixation, and a complication of open reduction and fixation of fractures [10]. The commonest of the spontaneous causative factors are compression neuropathy and brachial plexus neuritis (neuralgic amyotrophy) [9,11,12].

Causes of AIN compression include the heads of the pronator teres muscle, the proximal edge of the flexor digitorum superficialis arch, enlarged bicipital tendon bursa, lacertus fibrosus, a thrombosed radial artery branch in the mid-forearm, a thrombosed ulnar artery, tendinous bands and osseous spurs [4,6]. Furthermore, the AIN is prone to entrapment by anatomical variants such as Gantzer's muscle; an anomalous head of the FPL [13].

Electrodiagnostic examination is a useful adjunct in the investigation of AIN syndrome of spontaneous etiology. It helps in differentiating idiopathic AIN syndrome as part of a neuralgic amyotrophy from compression neuropathy [14,15]. MRI is also useful in diagnosing AIN syndrome [16]. On fluid-sensitive sequences such as short tau inversion recovery (STIR), proton density or T2-weighted fat saturated images, an increased signal intensity within some or all muscle groups innervated by the AIN could be seen. The most reliable finding is increased signal intensity within the PQ [16].

While most cases of AIN syndrome improve spontaneously without surgical intervention, its optimal treatment remains controversial. Conservative management with rest, analgesia (Steroids, NSAIDs), physiotherapy and splinting has been generally advocated [17,18]. Meanwhile, early surgical exploration and neurolysis have demonstrated promising results [19]. Other options include observation for spontaneous recovery, in which surgical intervention is indicated after 3 months of failed recovery [20]. Although electrodiagnostic studies showed compression neuropathy in our patient, we decided to treat conservatively in light that most cases do resolve or improve spontaneously. Nevertheless, in our case, the patients' symptoms progressed during the 3-months of conservative management and have not improved spontaneously. Therefore, the decision for surgical decompression was made. Although surgical decompression did not achieve full recovery, improvement of power has been achieved. Further investigations are warranted to delineate the optimal treatment of AIN syndrome.

4. Conclusion

Although AIN syndrome is rare, clinical suspicion should arise in the presence of weak FPL and the radial FDP muscles. The use of MRI in conjunction with electrodiagnostic studies will help in diagnosing AIN syndrome as well as aiding in specifying the possible etiology. The optimal treatment of AIN has not been established. Until further research is made, we recommend surgical intervention be considered in patients who fail to show clinical improvements during the first 3 months or those with confirmed compression neuropathy. During surgical decompression of the median nerve at the known compression sites, meticulous dissection is advised to identify and release any other compressing fibrous bands or edges.

Conflict of interest

The authors declare no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors did not receive any outside funding, grants or additional benefits for the work involved with this report.

Ethical approval

Our institutional review board does not require ethical approval for case reports. Only informed consent of the patient is necessary and was obtained.

Consent

The patient gave his consent to publication of this article.

Author contribution

Abdulla Aljawder: Collection of data, patient's informed consent, writing and revising the article.

Mohammed Khalid Faqi: Writing and revising the article.

Abeer Mohamed: collection of data and providing the radiology critique and paper write up.

Fahad Alkhalifa: Aritcle supervisor, critically revised the article and was the operating surgeon.

Guarantor

Abdulla Aljawder.

Acknowledgements

We would like to thank Maj. Gen. Prof. Khalid Bin Ali Al-Khalifa for his continuous support and encouragement towards research at the Royal Medical Services, Bahrain Defense Force hospital.

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