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Case Report

Post Traumatic Anterior Interosseous Nerve Syndrome in a Child, Wait and See Approach - A Case Report

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Abstract

Introduction: Peripheral entrapment neuropathies commonly present as pain and sensory-motor changes in the area innervated by that particular nerve. There are multiple causes of entrapment neuropathies and trauma is one of them. Involvement of the anterior interosseous nerve (AIN) is the most commonly seen entity following supracondylar humerus fractures in children. In the forearm, the motor division of the median nerve is known as AIN. Together with the anterior interosseous artery, the AIN travels deep in the forearm and supplies flexor pollicis longus (FPL) and flexor digitorum profundus (FDP) to the index finger and pronator quadratus (PQ) muscles. The clinical manifestations of AIN compression are seen as a weakness in the thumb and the index finger. This is typically presented as the inability to flex the interphalangeal joints of the thumb and the distal interphalangeal joint of the index finger.

Presentation of Case: A nine-year-old male child from a poor family presented in our pain clinic with right forearm pain along with weakness in the right thumb and index finger, because of which the patient was not able to hold the pen and write in the classroom. There was restricted joint movement at the elbow. The patient gave a history of right-sided supracondylar fracture humerus (Gartland type III with posterior displacement), four and half months back. The fracture was treated with C-arm guided closed reduction and pinning with Kirschner wires under general anesthesia. There was no history of vascular compromise or any other associated nerve involvement. Diagnosis of AIN injury was missed when the child had visited the orthopedic hospital for the initial injury, as the child was extremely anxious, noncooperative, and frightened. Also, there was lot of edema around the elbow joint due to the fracture.

On the basis of detailed history taking and physical examination, the clinical diagnosis of AIN syndrome was made by us. The diagnosis was confirmed after performing different clinical tests including a positive pinch grip test on the affected side. The electrophysiological studies showed typical electromyographic findings. No further investigations were possible because of the poor economic condition of the family. The child was treated with wait and see policy along with physiotherapy, stretching exercises, and rehabilitation. The patient was regularly coming for follow-ups for the next three months. All the complaints of the patient were resolved spontaneously and complete restoration of AIN function was observed within six months of the initial trauma.

Discussion: Understanding the anatomy of the anterior interosseous nerve as well as the etiology of the anterior interosseous syndrome. Primary diagnosis, analysis of sensory-motor involvement and the multimodal treatment approach in children with AIN syndrome. An interdisciplinary and multimodal treatment plan will lead us to an exact diagnosis and this is how the health care providers can help the patient most effectively.

Keywords: *Anterior Interosseous Nerve Palsy; Supracondylar Fracture Humerus; Clinical Suspicion; Wait and See*

Introduction

Supracondylar humeral fracture is the most common elbow fracture in children [1]. Most of the studies have stated that the occurrence of nerve injury in cases of supracondylar fracture humerus is variable from 6% to 16% [2-5] up to a maximum of 42% [6]. In these fractures, all the nerves around the elbow joint have the chance of getting injured but the sole involvement of the AIN remains the commonest finding [3,7]. The brachial artery and the median nerve travel across the elbow joint and the AIN has the highest probability of getting injured if the distal fragment of the fractured bone is displaced posterolaterally [9]. Multiple studies have shown that the function of AIN gets restored on its own in most cases within 6 to 10 weeks [2,4,8].

Anatomy of AIN and its clinical correlation

The AIN is purely a motor nerve. In their studies, Sunderland [10], Spinner and Mangin have discussed topography and different anatomical variations of AIN. The actual formation of AIN occurs at the level of the brachial plexus and the fibers contributing to AIN get separated at this level. In the proximal forearm, AIN originates from the median nerve at a distance of 5 - 8 cm distal to the lateral epicondyle of the humerus. Then AIN traverses with the anterior interosseous artery and lies within the pronator teres muscle, or below the muscle on the anterior aspect of the interosseous membrane to the wrist. The AIN provides motor supply to three muscles the pronator quadratus (PQ), the flexor digitorum profundus (FDP) to the index finger and the flexor pollicis longus (FPL). The damage to the AIN can lead to various clinical manifestations of motor weakness, and the interphalangeal joints of the thumb, as well as the distal interphalangeal joint of the index, can't be flexed. Dorsal to the PQ muscle, the AIN terminates as a thin branch which in turn provides sensory innervations to carpal joints [10]. But the innervation to FDP by median and ulnar nerves varies considerably [10-12]. The knowledge of topography and the anatomical variations of AIN and its application in our day-to-day practice is very important in a case of entrapment neuropathy.

Depending on the site and percentage of axonal damage, the AIN injury can present with various signs and symptoms. There is always confusion in cases of exclusive FPL muscle palsy after trauma and closed tendon rupture can be mistaken as a diagnosis.

Anterior interosseous nerve syndrome was first described as a complication of supracondylar fractures by Lipscomb and Burleson [2] and later by Spinner and Schreiber [8] who suggested that the mechanism that is producing the paralysis is traction on the nerve caused by the relatively fixed position of the AIN in the proximal forearm and the posterior displacement of the distal fragment of the fractured humerus.

Another explanation for the origin of this palsy is an injury of the median nerve proximal to the anterior interosseous nerve. The fibers are located in the posterior portion of the median nerve and might be damaged directly at the fracture site. This is called a pseudoanterior interosseous nerve syndrome [13]. Because there is no sensory loss, this hypothesis seems debatable. A cross-sectional map of the median nerve shows that the anterior interosseous nerve fibers are located immediately adjacent to the median sensory fibers in the supracondylar area [14] so selective damage to the motor fibers by traction with sparing of the sensory fibers is not likely. If there is motor loss accompanied by an inability to flex the terminal joints of the thumb and index finger, together with sensory loss, the nerve injury must be located proximal to the anterior interosseous nerve branch.

The third hypothesis of transient ischemia is suggested as a possible cause of anterior interosseous nerve syndrome. Geutjens [15] reported three cases of this palsy following supracondylar fractures of the humerus in children in whom the onset of anterior interosseous nerve paralysis was delayed and occurred only after surgery. During manipulation of the fracture, the children suffered a transient episode of ischemia, so the added insult to the nerve may have precipitated the palsy.

But in all cases, full spontaneous recovery from the paralysis is expected within 4 to 17 weeks. A second surgery to treat the anterior interosseous nerve palsy is not necessary. The anatomical location of the anterior interosseous nerve is distal to the supracondylar fracture. Complete division of this nerve is therefore is not likely and has never been described.

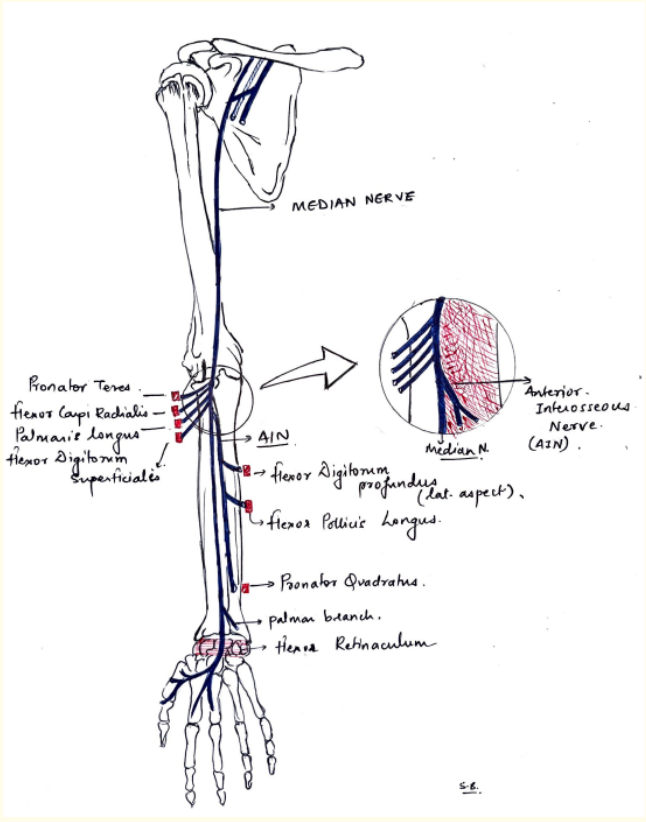

Diagram A: *Shows the anatomy of AIN and its branches.*

Case Presentation

A conscious, oriented, nine-year-old anxious male child from a poor family, presented in our pain clinic with right cubital fossa and forearm pain which was deep, dull aching, poorly localized, non-radiating, not associated with tingling or numbness, and with a VAS score of 7/10 since last 3 months. Along with the pain, there was weakness in the right thumb and index finger. The patient was unable to button the shirt, unable to write in the school which was noted by his teacher, was unable to make a fist and was also unable to turn on the bicycle key. The flexion at the right interphalangeal joints of the thumb was not possible along with difficulty in flexing the distal interphalangeal joint of the right index finger.

The patient gave a history fall from his bicycle and right-sided supracondylar fracture humerus (Gartland type III with posterior displacement), four and half months back. The fracture was treated with C-arm guided closed reduction and pinning (Kirschner wires) under general anesthesia four and half months back. There was no history of vascular compromise or any other associated nerve

involvement at the time of initial trauma. The diagnosis of AIN injury was missed when the child had visited the orthopedic hospital for the initial injury, as the child was extremely anxious, noncooperative, and frightened. Also, there was a lot of edema around the elbow joint due to the fracture.

No history of major illnesses, or drug allergies in the past. The patient had received rest, splinting, analgesics, anti-inflammatory medication and physiotherapy as a conservative treatment for the presenting symptoms at an orthopedic hospital.

When the patient was referred to our Pain clinic, after four and half months old trauma the primary diagnosis of AIN syndrome was made based on history and clinical examination. The patient complained of pain in the cubital fossa and forearm. Clinically there were no sensory symptoms such as tingling, numbness, loss of sensation, etc. There was restricted joint movement at the elbow. The fracture showed complete union. There were no visible skin changes or visible veins. The scars of pinning were seen at the right elbow joint. Mild to moderate atrophy and muscle wasting was noted in the right upper limb as compared to the normal left side. There was mild tenderness at the right cubital fossa and forearm. No trigger points were noted. There was no local rise in temperature, no allodynia or hyperalgesia. The physical examination also showed the weakness of the FPL and FDP to the index finger. The motor power was assessed in the interphalangeal (IP) joints of the thumb and distal interphalangeal (DIP) joint of the index on the right side, which was reduced to 3/5. The patient was able to overcome gravity without resistance. A complete evaluation of musculoskeletal and nervous systems revealed no abnormality. The patient was unable to form "O" with the fingertips of the right thumb and index, showing a positive Froment's sign. Clamping the sheet of paper between an extended thumb and index finger was also not possible. Considering the possibility of AIN syndrome, and the non-willingness of the patient and his parents for further investigations and interventions, conservative treatment with rest, analgesics, and physiotherapy was continued. The patient and his parents were not ready for any intervention although perineural injection of the anterior interosseous nerve with hydro dissection could have been an alternate treatment option.

The patient came for follow-up after 3-weeks and clinical improvement was noted. The motor power evaluation showed improvement up to 4/5 in the interphalangeal joints of the right thumb and distal interphalangeal joint of the right index finger and it was possible to overcome gravity with resistance. Due to the financial concerns of the family further investigations were not done. The child was treated with Wait and see policy along with physiotherapy, stretching exercises, and rehabilitation. The patient was regularly coming for follow-ups for the next three months. Total recovery of the AIN function was seen within six months of the initial trauma.

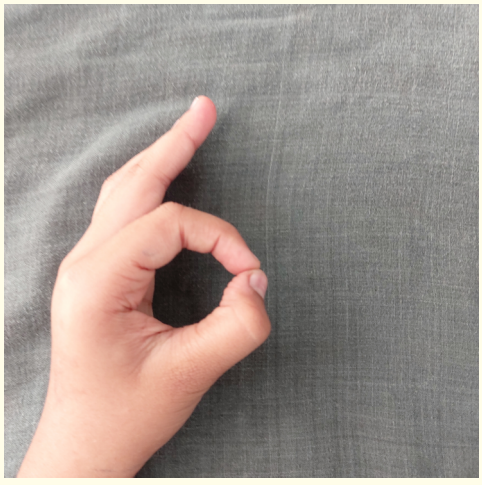

Diagram B: *Showing Pinch grip test of AIN function. There is no injury to the left upper extremity. The muscles innervated by AIN (FDP, FPL) showed adequate flexion.*

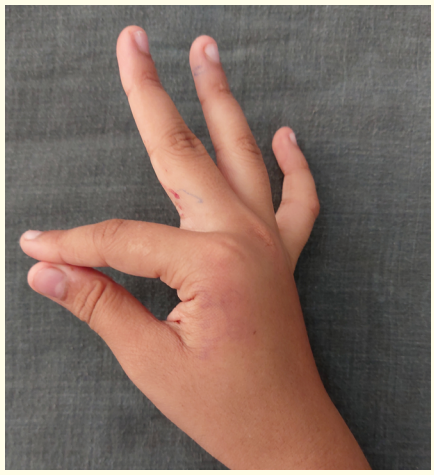

Diagram C: *Showing Pinch grip test of AIN function. The injured right extremity shows the abnormal activity of FDP and FPL, where flexion is not possible.*

Discussion

The supracondylar humerus fractures along with AIN injury is the most frequent picture [8]. A study was carried out on 138 patients with displaced supracondylar humerus fractures of non-articular type by McGraw., *et al* [16]. This study revealed a 2.9% incidence (4/138 patients) of pure AIN involvement. Another study was done by Cramer., *et al*. [7] which showed a 5.9% incidence (6/101 patients) of exclusive AIN injuries along with the supracondylar humerus fractures. One more study was done by Lyons., *et al*. [3] on 210 patients and they demonstrated the incidence of 6.2% (13/210) of pure AIN injuries in supracondylar humerus fractures of type III. Considering the high incidence of AIN injury with the supracondylar fracture humerus, a big index of suspicion is necessary whenever you come across such patients.

The AIN syndrome is considered to be pure motor neuropathy. But many studies have demonstrated dull aching forearm pain in association with AIN syndrome [17]. Characteristically AIN syndrome patients find difficulty in forming an "O" (circle) with the tip of the index finger and thumb because of weakness in the muscles supplied by AIN. The patients are unable to write, button their shirts or turn on their keys. The physical examination shows a positive Pinch Grip test and patients find difficulty in making "O" using the thumb and index, which indicates weakness in the muscles FDP and FPL [18]. To compensate for this weakness, patients use extra pinch strength whenever they are asked to grasp a piece of paper between extended thumb and index finger, demonstrating the deformity (Froment's test). The motor power of PQ should be tested by asking the patient to pronate the forearm actively against resistance when the elbow is in flexion to eliminate the action of pronater teres (PT) muscle. In cases of AIN syndrome, the clinical examination below the elbow shows no sensory loss or changes [12]. This helps the clinician to isolate it from the Median nerve or any other nerve involvement where sensory loss and paraesthesia in the upper extremity is a common findings. Considering the age, anxiety, and associated swelling in pediatric patients, it is always a challenge to make the exact diagnosis of AIN syndrome amongst different etiologies [19]. A practical solution to examine the sensory system without the patient's cooperation is to put the diseased part under water or inside a wet towel for some time and look for the wrinkles in various areas [20]. In spite of putting underwater, the areas with sensory loss do not show any wrinkles. In all doubtful cases, this test is of tremendous help. When the AIN is affected completely, all the three muscles- the PQ, the radial side FDP and FPL are involved. But if the AIN is affected partially then either FPL or FDP of the index shows weakness [21]. When only thumb weakness is seen, it indicates the fault of a specific fascicle supplying the FPL [22].

Though we couldn't perform further investigations because of no financial support from the family, the electrodiagnostic studies along with USG and MRI are the gold standard triad of investigations.

Electrodiagnostic testing

Electrophysiological evaluation remains one of the important investigations for AIN syndrome of spontaneous origin. The nerve conduction evaluation of the sensory part of the median nerve has to be normal since AIN has only a motor component. Electromyography (EMG) of the muscles innervated by AIN should be performed. The results of EMG testing should be compared with the same muscles of the normal extremity. This comparison differentiates between amyotrophy of neurological etiology and neuropathy due to external pressure. A baseline EMG seems to be useful for further comparison. Repeat the EMG to monitor the recovery and for necessary timely intervention if needed.

USG

Since the AIN is very thin and is located deep in the forearm, the nerve may not be visible easily [23]. USG evaluation is difficult for deeper structures with increasing tissue thickness. The anterior interosseous artery which always accompanies the nerve should be visualized first using the colour doppler and this is taken as a guide [24]. The AIN may get entrapped between the PT and the proximal margin of the flexor digitorum superficialis muscle, just similar to the median nerve [25]. On USG evaluation, the cross-sectional area of AIN is increased due to the swelling of the nerve when compared to the normal side. Under the ultrasound examination, there can be atrophy of the muscles supplied by AIN, loss of normal fibrillar echo structure seen as hyperechoic muscles, and reduced muscle contraction in the affected area. Injecting a small volume of local anesthetic solution around the affected nerve provides pain relief and also confirms the source of pain. The use of ultrasound evaluation allows us to rule out any structural pathology and dynamic abnormalities.

MRI is useful in diagnosing AIN syndrome [26]. On MRI, the T2 weighted images may show changes in the affected nerve which is seen as enlargement showing increased signal. The T2 weighted images also show a hyperintensity signal in the muscles supplied by the AIN. The hyperintensity signal seen in the PQ is the most reliable guide [26].

Differential diagnosis

Diagnosis of AIN syndrome can be difficult. Considering the exclusive motor component of AIN, the diagnosis is frequently confused with ligamentous injury or tendon rupture. Tenosynovitis, tendon rupture, ligamentous injury, brachial neuritis, etc. can be the variable diagnosis. A similar picture is seen in cases of brachial neuritis [27,28].

Treatment

Even though anterior interosseous nerve syndrome is very rare as compared to all the peripheral neuropathies of the upper limb, in children it is the most common occurrence with supracondylar fracture humerus with posterior displacement. And whenever there is weakness in the muscles innervated by AIN, the clinician must think of AIN syndrome as a diagnosis. After the exact diagnosis, proper management should be done in consideration of the underlying cause. A proper clinical history with thorough clinical examination and rational use of electrodiagnostic studies, USG coupled with MRI will definitely assist in the diagnosis and help to specify the possible etiology and management. Optimum treatment for AIN syndrome has not been established yet.

When the final opinion of AIN syndrome is made, the patient is advised to continue rest, avoid harmful activities, and have analgesics and anti-inflammatory drugs for quite a long period before going for any surgery [29]. Mostly no surgery is needed and a spontaneous improvement is seen [30,31]. We followed a similar treatment option for our patient with a wait-and-see policy, and complete recovery occurred without surgical intervention. According to the pattern of pain and symptoms, physiotherapy can be of help. Surgical intervention

may be necessary when the conservative treatment fails with no signs of clinical and electromyographical improvement. If the motor function is not recovered, tendon transfer can restore the function satisfactorily.

An interdisciplinary and multimodal approach to management is the key to complete recovery. This approach includes primary care providers who encounter the disease first, the orthopedic surgeons who follow the course of treatment, the Pain physicians who can help to find out the exact pain generator as well as help in the management by doing USG-guided diagnostic as well as therapeutic perineural injections. Electrophysiologists, pharmacists, and physiotherapists also play a major role. Participation of the nursing staff in patient care can bring value to the whole outcome.

Conclusion

Anterior interosseous nerve injury is the most common type of nerve injury associated with supracondylar humeral fracture. Underdiagnosis of this complication results from the inadequate neurological examination at the time of injury. The traction on the anterior interosseous nerve usually causes neurapraxia and delayed recovery of nerve function occurs in only those patients who are not aware of the disability. An orthopedic surgeon should be aware of the possibility of AIN syndrome, and special attention should be given while examining patients with such injuries. A correct and early diagnosis contributes to optimal treatment. If weakness or paralysis is seen only in the muscles supplied by the AIN, the possibility of AIN syndrome should first come to mind. The diagnosis and pathophysiology can be confirmed by EMG, ultrasound examination, and MRI.

In most patients with isolated anterior interosseous nerve injuries along with a supracondylar fracture humerus, surgical intervention is not necessary unless signs of recovery are not evident at the three-month follow-up and, wait and see approach can be followed. Observing and keeping an eye on the improvement of nerve function is requisite, once the definitive treatment of the fracture is done.

But emergency surgery may be required if there is any neurovascular compromise, another nerve involvement, and any additional fractures of the forearm in the same extremity.

Conflict of Interest

No conflicts of interest are stated by the authors.

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The authors didn't get any external finance or aids, and for this case report, they have worked without any added interest.

Ethical Approval

For the case reports, no special permission is required from the examination committee of our institute. A proper written informed consent was taken from the child and his parents.

Consent

To publish this article, written consent was given by the child and his parents.

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Volume 5 Issue 8 August 2022