

ANTERIOR INTEROSSEOUS NERVE SYNDROME

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SYNOPSIS

The anterior interosseous nerve syndrome (AINS) is an interesting neuropathy first noted by Parsonage and Turner in 1948(1) and later described by Kiloh and Nevin in 1952(2). It presents classically as an inability to flex the interphalangeal joint of the thumb and distal interphalangeal joint of the index finger. This article presents an illustrative case and reviews the literature in an attempt to enhance awareness of this syndrome which is crucial to its diagnosis.

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CASE REPORT

A forty four year old Malay man presented to the Orthopaedic Outpatient Clinic in February 1987 with an acute onset of pain over the medial volar aspect of his left elbow. This symptom was initiated by forced hyperextension of the wrist and fingers while the fingers were flexed. The patient also had a significant history of frequent hunting with a shotgun in which the left hand held the double barrel of the gun with the proximal interphalangeal joints flexed and the fingers firmly clasp the barrels.

The patient was initially treated conservatively for strain of the common flexor originating with a period of rest, nonsteroidal anti-inflammatory drugs, physiotherapy and local injection of steroids. This did not result in any improvement and when referred to the Hand and Microsurgery Clinic, he was noted to be unable to flex the interphalangeal joint of the left thumb and the distal interphalangeal joint of his index finger (Figure 1). Physical examination also revealed marked wasting of the medial forearm flexor muscles and weakness of pronator quadratus on stressing. A diagnosis of the AINS was made and electromyographical studies were performed.

It was not possible to test the flexor pollicis longus muscle (FPL) as it was wasted. However the main trunk of the median nerve was found to be normal. Surgical exploration was carried out in July 1987.

OPERATIVE FINDINGS

The median nerve was exposed in front of the elbow and traced distally. The tendinous arc of the flexor digitorum superficialis (FDS) was identified and found to be compressing both the median and the AIN (Figure 2). A vascular pedicle was found anterior to the AIN. Both these nerves were noted to be oedematous proximal to the compression band but only the AIN was atrophied distal to it.

The fibrous arc of the FDS, the lacertus fibrosus and vascular pedicle were released and divided (Figure 3). Post-operatively at two weeks follow up the patient had recovered function in the FDP to the index finger but the power in the FPL remained at grade 0. Five months post-operatively FDP II function had recovered to a power of grade 5 but FPL function had only grade I power (MRC grading).

DISCUSSION

The AIN is a motor branch arising from the postero-lateral aspect of the median nerve usually 5 to 8 cm distal to the lateral epicondyle(3). It then passes under both heads of pronator teres. After passing beneath the fibrous arch of the FDS origin it courses distally down the forearm on the anterior aspect of the interosseous membrane, giving off branches to the FPL and FDP II muscles. It then ends in the pronator quadratus muscle supplying it. It does not supply sensory innervation to any skin over the forearm.

Anteriorly it is crossed by collateral vessels of the interosseous artery. A communication between the ulnar and median nerves, in which the motor component of the ulnar nerve is carried by the median nerve (Martin-Gruber Anastomosis) is present in 15% of limbs and half of these communications may involve the AIN directly (4). In these cases some of the intrinsic muscles of the hand namely first dorsal interosseous, adductor pollicis and abductor digiti quinti may also be paralysed(5).

Diagnosis is made on the basis of a detailed history and careful physical examination. The patient may complain of initial pain in the forearm as was the case with our patient, or loss of pinch grip. The thumb and index finger assumes a characteristic pinch attitude in which there is extension of the interphalangeal joints of the thumb and distal interphalangeal joint of the index and sometimes the middle finger.

Weakness of pronator quadratus can be demonstrated by having the examiner hold the patient's forearm in a supinated position by clasping the hand, then asking the patient to pronate it against resistance. The two sides are then compared with each other. There is no sensory loss in this syndrome as the AIN does not carry sensory innervation to the skin. There may only be a partial paralysis of either FDP or FPL alone. Hill et al(6) has reported 33 cases of the incomplete syndrome. O'Brien and Upton(7) reported on a case in which they carried out neurophysiological investigations with a unipolar concentric needle electrode which was used to record the electromyographic interference pattern of the muscles and also for the motor conduction studies. They demonstrated a significant reduction in the interference patterns during maximal volitional activity in the affected pronator quadratus, FPL and part of the FDP.

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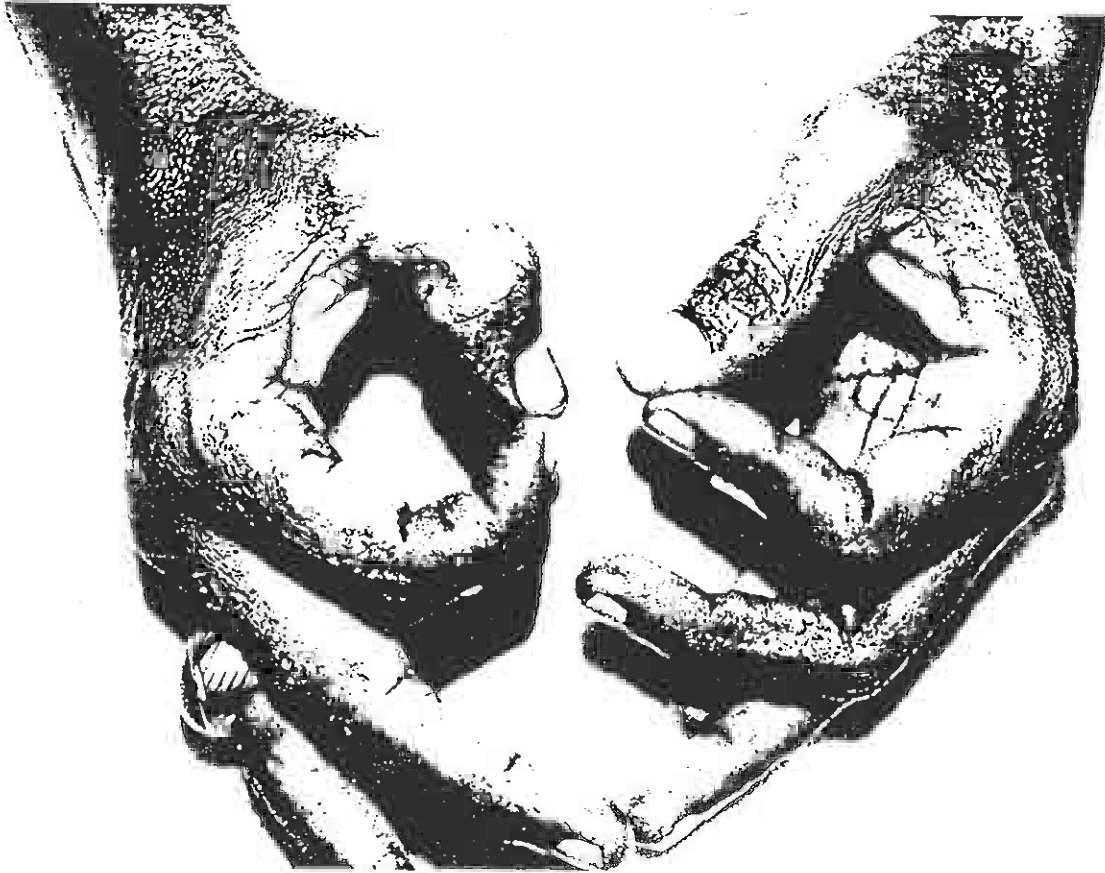


Figure I The typical square (pulp to pulp) pinch in the affected left hand.

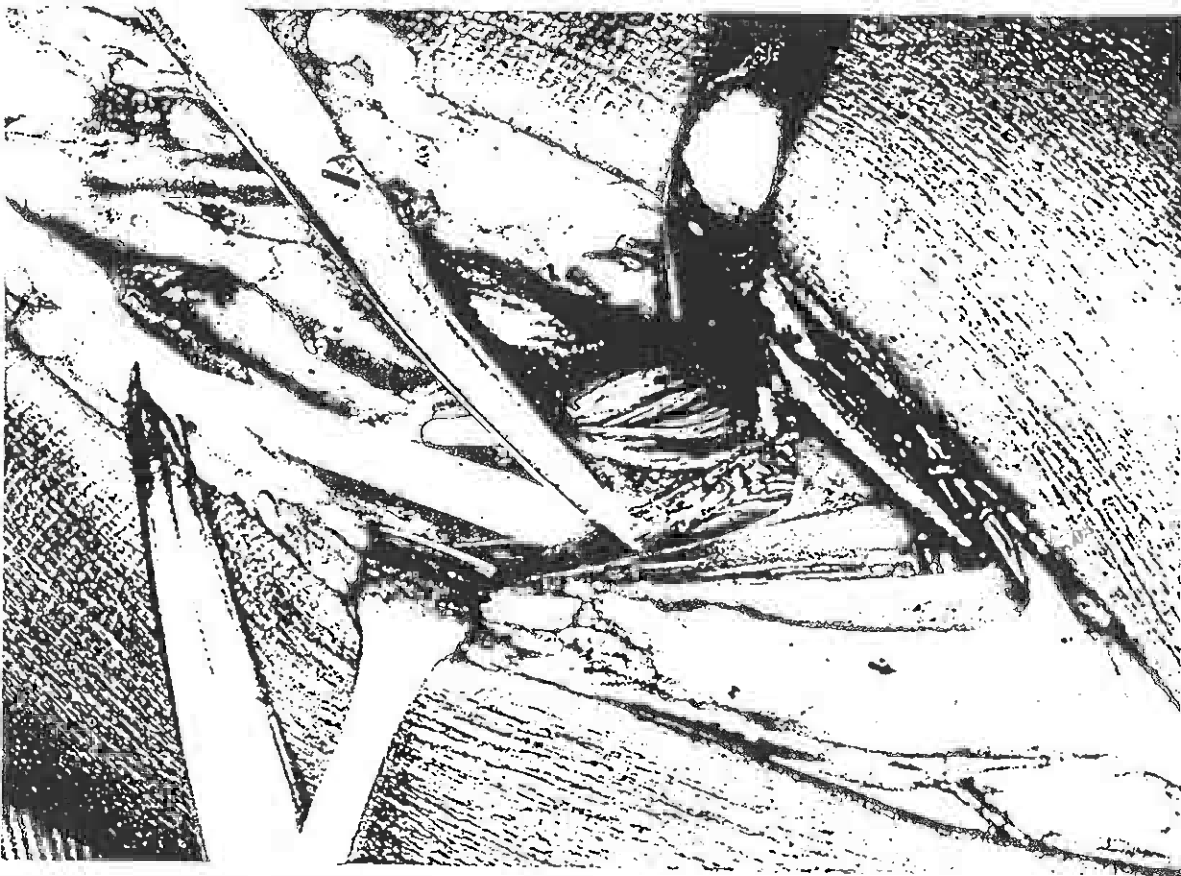


Figure II The fibrous arch of FDS origin compressing both median and AIN nerves (scissors slipping beneath the arch).



Figure III After division of the fibrous arch — oedema of the proximal AIN and atrophy of the distal AIN (forceps pointing to the AIN).

The most common aetiology of this syndrome is due to compression by fibrous bands at the deep head of pronator teres muscle or at the tendinous origin of FDS. Other reported cases include:-

- (1) Local trauma e.g. penetrating injuries or forearm fractures especially supracondylar fractures of the humerus(8,9)
- (2) Neuralgic amyotrophy
- (3) Thrombosis of the anterior interosseous artery(10)
- (4) Direct pressure on the volar aspect of the forearm
- (5) Metastatic bronchogenic carcinoma(11)
- (6) Idiopathic causes

The differential diagnosis of this condition includes rupture of the FPL tendon in rheumatoid arthritis, lesions of the infra clavicular portion of the brachial plexus, congenital absence of the profundus and the Pronator Teres Syndrome. The latter produces paralysis of all forearm muscles supplied by median nerves as well as sensory deficit in the radial three and half fingers. It can also present as pain in the proximal forearm on the volar surface. If the arch of the FDS com-

presses the median nerve, it is aggravated by resisted flexion of the flexor superficialis muscle of the middle finger. It may also be due to compression by the ligament of Struthers or by the lacertus fibrosus, in which case, the symptoms will be aggravated by flexion of the elbow against resistance between 120° and 135° of flexion.

Most authorities would recommend an initial trial of conservative treatment with a period of rest in a sling with perhaps galvanic stimulation of the muscles; followed by surgical exploration at the end of eight to twelve weeks if there was no improvement in symptoms(12,13,14).

We would advocate immediate surgical exploration and decompression when the diagnosis has been made, as we feel that any delay in release will result in unnecessary prolonged period of disability and progressive compression resulting in chronic damage to the nerves leading to unpredictable recovery following release.

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