Review Article

Compressive neuropathy in the upper limb

Mukund R. Thatte, Khushnuma A. Mansukhani[1]

Departments of Plastic Surgery, [1]Electro Physiology, Bombay Hospital Institute of Medical Sciences, India

Address for correspondence: Dr. Mukund R. Thatte, Department of Plastic Surgery, 402, Vimal Smruti, 770 Dr. Ghanti Road, Dadar, Mumbai – 400 014, India. E-mail: mthatte@gmail.com

ABSTRACT

Entrampment neuropathy or compression neuropathy is a fairly common problem in the upper limb. Carpal tunnel syndrome is the commonest, followed by Cubital tunnel compression or Ulnar Neuropathy at Elbow. There are rarer entities like supinator syndrome and pronator syndrome affecting the Radial and Median nerves respectively. This article seeks to review comprehensively the pathophysiology, Anatomy and treatment of these conditions in a way that is intended for the practicing Hand Surgeon as well as postgraduates in training. It is generally a rewarding exercise to treat these conditions because they generally do well after corrective surgery. Diagnostic guidelines, treatment protocols and surgical technique has been discussed.

KEY WORDS

Entrapment neuropathy, carpal tunnel, minimal access

INTRODUCTION

Compressive neuropathy is one of the most fascinating yet most complex aspects of Hand Surgery. It is also quite often the most rewarding surgery in terms of clinical outcomes with some exceptions. Compresssive or entrapment neuropathy results from compression on a nerve at some point over its course in the upper limb. It can result in altered function and if left untreated leads to considerable morbidity—some of which can be difficult to reverse, if left too late. It is therefore worthwhile to be able to diagnose and treat these conditions early.

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Common conditions

The nerves discussed in this article are

- Median nerve
- Ulnar nerve
- Radial nerve

The common conditions in our practice are

- Carpal tunnel syndrome (CTS)
- Ulnar neuropathy at the elbow (UNE)

The rare conditions are

- Anterior interosseous nerve syndrome
- Pronator syndrome
- Radial tunnel syndrome
- Posterior interosseous nerve syndrome
- Ulnar neuropathy in Guyon's canal

PATHOPHYSIOLOGY, HISTOLOGY AND BIOCHEMISTRY OF NERVE COMPRESSION

A peripheral nerve consists of myelinated and

unmyelinated axons originating in the dorsal root ganglion (in case of sensory fibres) and the grey matter of the anterior horn (in case of motor fibres) to form a mixed peripheral nerve. Some autonomic fibres too are carried by the nerve. The role of the connective tissue is very important in this discussion.

Compression of a nerve in a given area can lead to a cascade of physiological changes resulting in pathological situations and then anatomical changes in the later stages. Eventually there is severe compromise of nerve function if left untreated. Mackinnon in her seminal article[1] on pathophysiology has discussed it very well.

The axons themselves are neuroectodermal in origin, whereas the connective tissue is mesodermal in origin. When surgeons operate on a nerve or repair it all terms used by them refer to the respective connective tissue layer in the nerve. Each axon is covered by endoneurium, a group of axons is surrounded by perineurium which is the most critical layer in neurophysiology as it represents the 'Blood–Nerve Barrier'. Between the fascicles there is the internal epineurium, and the whole nerve is covered by the epineurium, the connective tissue around the nerve is mesoneurium and often carries the segmental blood supply of the nerve. Nerves have both axial (e.g. the median artery) and segmental vasculature (e.g. the Superior Ulnar colateral artery) all along its course and compression results in altered pressure in the vessels on and within the nerve creating an internal compartment syndrome and/or a breakdown of the blood–nerve barrier with consequent leakages.

Blood–Nerve barrier

The inner layers of the perineurium and the endothelial cells of the endoneurial microvessels create the blood–nerve barrier. These cells have tight junctions that are impermeable to many substances. Thus, the blood–nerve barrier provides a privileged environment within the endoneurial space. There are no lymphatic vessels within the endoneurial or perineurial spaces.

Breakdown in the blood–nerve barrier will result in the accumulation of proteins and the ingress of lymphocytes, fibroblasts and macrophages as a reaction to previously shielded antigens contained within the perineurial space. This will initiate inflammation and eventually there is scar formation. If the barrier site at the inner layers of the perineurium remains relatively intact, this will then result in increased endoneurial fluid pressure and a mini compartment syndrome within the fascicle.[2]

Acute nerve compression

Rydevik *et al*[3] studied the effects of graded compression on intraneurial blood flow and showed that external pressures of 20 mmHg reduced epineurial venule blood flow, pressures of 30 mmHg inhibited both antegrade and retrograde axonal transport, and with pressures of 80 mmHg, all intraneurial blood flow ceased. These changes were transitory and therefore reversible in the short term. Prolonged acute pressure could result in lasting damage. Tourniquet palsy is a good clinical example of acute compression leading to deficit. It too is often reversible in 3–6 weeks but not always.

Chronic nerve compression

Models of chronic nerve compression have been attempted by using silastic cuffs placed around the rat sciatic nerve and the median nerve in primates.[4-9] The results of these studies were similar to that noted above in that a dose–response relationship between the duration of compression and neural injury was noted. The initial changes were a breakdown in the blood–nerve barrier, followed by subperineurial oedema and fibrosis; localized, then diffuse, demyelination occurred, and finally Wallerian degeneration. These changes were most impressive in the periphery of the nerve directly under the area of compression. Perhaps explaining why the middle finger fibres of the median nerve are the first to be symptomatic.

The histopathology of chronic nerve compression follows a continuum that parallels patient sensory complaints, which progress from intermittent paresthesia to constant numbness. Motor complaints progress from aching to weakness to atrophy. Sunderland[10] quoted 2 rare reports where necropsy material became available. A brief description of their findings is as follows: Marie and Foix (1913)[11] described a 'neuromatous' enlargement just above the retinaculum with an abrupt reduction in size in the tunnel, distal to which the nerve regained its normal dimensions. The nerve bundles beneath the retinaculum were thinned with an increase in the endoneurium which had destroyed the 'myelin sheaths'. The swelling showed a considerable increase in both the epineurial and intrafunicular connective tissue though the great bulk of the swelling was due to the former. Thomas and Fullerton (1963)[12] reported a bilateral case, where the right nerve

(marked symptoms) had a similar picture to that reported by Marie and Foix (1913) but the left (milder symptoms) appeared normal. Histological examination of sections of both nerves from beneath the retinaculum revealed an increase in perineurial and endoneurial connective tissue and a marked reduction in the calibre of the nerve fibres.

Sud *et al*[13] have discussed the biochemistry of nerve compression and the resultant effects on the nerve and the synovium around it. They mention that Serum and tissue levels of the free oxygen radical malondialdehyde bis diethyl acetate (MDA) are higher in people undergoing continued oxidative stress. The cellular injury created by this reactive oxygen species initiates the metabolism of arachidonic acid into cyclooxygenase products such as PGE2, a potent vasodilator known to increase the sensitivity of nerve endings to chemical and mechanical stimuli contributing to pain stimuli in CTS patients. Cellular damage created by neural and synovial ischemia contributes to the production of cytokines. High levels of IL-6 cause fibroblast proliferation and synovial thickening.

Ischemia-induced reperfusion injury thus has a role to play in the symptomatology of CTS. These findings and chain of events, interestingly, point to swelling of the flexor tenosynovium with indirect compression of the nerve. The histology of the synovium in the author's experience is always non-specific inflammation and the above-mentioned chain of biochemical changes explains this phenomenon.

The final common pathway is increased interstitial fluid pressure. The experiments conducted in laboratories on animals to simulate CTS showed changes in the nerve, and these were extrapolated to humans.[14-16] Several pathways have been proposed:

- Flexor tenosynovitis,
- Vascular sclerosis,
- Fibrous hypertrophy
- Oedema of the synovium

Initial reduction in epineural blood flow is followed by reduction in endoneural blood flow and oedema.[17] This process, at least partially, may be irreversible, resulting in abnormal impulse generation, conduction delay or complete nerve block.[18] Thus there is an entire spectrum to the pathophysiology of nerve compression and depending on the severity and duration of the compression various levels of damage can be seen.

Finally, it is a common clinical experience, that even chronic entrapments with longstanding muscle weakness and sensory disturbances sometimes show a very rapid reversibility of some or all of the symptoms after surgical decompression of the nerve. These findings suggest the existence of a local metabolic block in the compressed segment of the nerve. The rapid reversibility indicates that such a disturbance may be based upon temporary microvascular disorder in the compressed part of the nerve in addition to local myelin changes

Multiple and double crush syndrome

The double crush hypothesis was introduced by Upton and McComas[1,19] who stated that a proximal level of nerve compression could cause more distal sites to be susceptible to compression. They noted a high incidence of carpal and cubital tunnel syndrome with associated cervical root injuries. They concluded that the summation of compression along the nerve would result in alterations of axoplasmic flow and subsequent pathology and symptomatology. The possibility of a distal site of compression making the more proximal nerve susceptible to secondary compression has been suggested: A reverse double crush. Similarly, systemic diseases such as diabetes can be considered to lower the threshold for the occurrence of a nerve compression. Thus, anything that would hypothetically alter axoplasmic transport would render that nerve more susceptible to developing compression neuropathy and act as a 'crush'.[20]

This concept of double or multiple crush is important clinically in patients who demonstrate multiple levels of nerve compression, as failure to diagnose and treat these multiple levels of injury will result in failure to relieve patients' symptoms. Systemic conditions such as obesity, diabetes, thyroid disease, alcoholism, rheumatoid arthritis and other neuropathies will similarly render a given individual more susceptible to the development of CTS and other compressions.

Electrodiagnosis in entrapment neuropathies

'Entrapment' implies chronic and often slowly increasing compression of a nerve as it passes through a fibroosseous space – the most common example being CTS. An outline of the abnormalities detected in electroneuromyographic evaluation of the nerve entrapment syndromes mentioned earlier is provided in this section. Chronic compression of a nerve usually results in a combination

of focal demyelination (below the site of entrapment) and axon degeneration, depending on the chronicity and severity of the lesion. These changes are responsible for the abnormalities detected on electrophysiological evaluation.

Electroneuromyography consists of a series of tests done sequentially to aid the diagnosis of the neuromuscular dysfunction. It helps to accurately localise the site of the lesion, give an objective diagnosis, assess its severity, determine its predominant pathophysiology and extent, provide a basis for comparison and recognise minimal defects. Most importantly it is the only test to assess the function of the nerve. The tests done are nerve conduction studies for sensory and motor nerves and needle electromyography.

Sensory nerve conduction studies are the earliest to show abnormality of slowing (focal demyelination) in the nerve across the site of the entrapment. Motor conduction abnormalities generally present later with slowing across the site followed by loss of axons (both sensory and motor) if the entrapment is unrelieved. Needle electromyography is used to detect axon loss which is chronic unless there is a super added acute external pressure on an existing entrapped nerve.

Carpal tunnel syndrome

Routine tests include sensory motor conductions across the wrist in median and ulnar nerves. Early abnormality is slowing of the median sensory nerve action potential (SNAP) with normal conduction in the Ulnar SNAP defined as Mild CTS. When the terminal motor latency of the median nerve to the Abductor Pollicis Brevis muscle is prolonged it is termed as moderate CTS. Severe CTS is indicated by absent SNAP or low amplitude motor response, and extreme CTS is indicated when both sensory and motor conduction is absent.

When routine tests are negative for CTS but symptoms are very suggestive, sensitive internal comparative tests are done which compare the SNAP from the median nerve to that recorded from the Radial and Ulnar nerves to demonstrate differential slowing across the wrist only in the median nerve.

Cubital tunnel syndrome

This term is reserved for compression of the Ulnar nerve in the cubital tunnel distal to the medial epicondyle in the forearm. Nerve conduction study would show variable loss in the amplitude of the Ulnar SNAP, depending on the severity of the entrapment. Motor conduction would localise the site of the nerve entrapment and quantify the axon loss if any. Needle Electromyography would detect the chronicity of the lesion and localise it specially if there is chronic long-standing entrapment with severe axon loss.

True neurogenic thoraxic outlet syndrome

Electrodiagnosis would localise the site of the lesion to the lower trunk of the brachial plexus. On the affected side the findings would be low amplitude Ulnar SNAP, absent Medial cutaneous nerve of forearm SNAP, low amplitude motor-evoked responses median > ulnar nerves and chronic denervation of the muscles innervated by the lower trunk (T1 > C8)

Supinator syndrome/radial tunnel syndrome/ posterior interosseous nerve entrapment

Chronic entrapment of the posterior interosseous nerve shows normal radial sensory conduction with chronic denervation in the muscles supplied by that nerve.

Carpal tunnel syndrome

It is the most common entrapment neuropathy in our clinical practice in India. Although national data is missing over here, there is data from USA to show that there too it is the most common compressive neuropathy.[21] Incidence in the United States has been estimated at 1–3 cases per 1000 subjects per year, with a prevalence of 50 cases per 1000 subjects per year.[21]

Anatomy

The carpal tunnel situated in the proximal palm, it contains the median nerve and nine flexor tendons from the forearm into the palm. The tendons are covered by a variable amount of tenosynovium, the role of this in the pathology is alluded to the above.

The tunnel is bounded by the carpal bones dorsally and the transverse carpal ligament, a fibrous unyielding structure, volarly. The median nerve lies superficially in the carpal tunnel, immediately beneath the transverse carpal ligament. The palmar cutaneous nerve branches 5 cm proximal to the wrist crease and passes into the palm above the ligament. The median nerve divides into sensory and motor branches after passing through the carpal tunnel, the motor branch lying volarly and radially in the nerve.[10]

Etiology

There are multiple causes discussed without consensus about them. Where no specific cause exists it is primary CTS. These are generally women in the age group of 30–50 years.

Secondary causes are

- Hypothyroidism
- Rheumatoid arthritis
- Malunited fracture distal end of radius
- Pregnancy
- Repetitive strain—highly disputed in many studies
- Workers using vibratory tools

Symptoms

Graham *et al*[22] have published the six most important symptoms to diagnose CTS:

1. Numbness and tingling in the median nerve distribution
2. Nocturnal numbness
3. Weakness and/or atrophy of the thenar musculature
4. Tinel sign
5. Phalen's test
6. Loss of 2-point discrimination

Treatment

Initial treatment can be symptomatic, comprising of rest, avoidance of vibratory tools, or repetitive activity. Changing work profile and drug therapy which can be a combination of NSAIDs and oral as well as local steroids. In the author's experience a small subset of people, about 10% will settle with these measures. A substantial majority though require surgical release. The American Academy of Orthopaedic Surgeons' (AAOS) guidelines on the subject are quoted below.[21]

AAOS Clinical Practice guidelines for the treatment of CTS[21]:

Recommendations:

1. Nonsurgical treatment is an option for early CTS. Surgery is an option when there is evidence of median nerve denervation.
2. A second nonsurgical treatment or surgery is recommended when initial nonsurgical treatment fails after 2–7 weeks.
3. There is no evidence to support specific treatment recommendations for CTS associated with diabetes, cervical radiculopathy, hypothyroidism, polyneuropathy, pregnancy, rheumatoid arthritis or CTS in the workplace.
   - 4a. Local steroid injection or splinting is recommended prior to treatment with surgery.
   - 4b. Oral steroids and ultrasound are also options for treatment.
   - 4c. Carpal tunnel release is recommended for treatment of CTS based on level I evidence.
   - 4d. Heat therapy does not have evidence to support its use in CTS.
   - 4e. Other nonsurgical treatment modalities are not recommended for treatment of CTS.
5. Surgical treatment with complete division of the flexor retinaculum is recommended, regardless of the technique used.
6. Skin nerve preservation, epineurotomy, flexor retinacular lengthening, internal neurolysis, tenosynovectomy, and ulnar bursa preservation are not recommended in the performance of carpal tunnel surgery.
7. Use of preoperative antibiotics is an option that may be decided upon by the surgeon.
8. Wrist immobilization is not recommended postoperatively after routine carpal tunnel release. No recommendation is made regarding use of postoperative rehabilitation.
9. It is suggested that physicians use one or more patient response tools to assess results after carpal tunnel treatment in performing research.

Surgical technique

Once the decision is made based on clinical findings and electrophysiology the treatment consists of Carpal Tunnel Release. There are three main techniques to perform this procedure.

- Conventional Wide Exposure (Extended open Carpal Tunnel release or ECTR) [Figure 1]
- Minimal access open technique (MAOT) [Figure 2]
- Endoscopic release

There is no difference in outcome from either[21] as far as decompression of the nerve is concerned or therefore the relief from symptoms but there is considerable difference in the postoperative morbidity and recovery process. The 2nd and 3rd method have considerably lower morbidity as compared to the older conventional wide exposure which crosses the wrist crease. Between the minimal access and endoscopic release there is not much to choose in terms of the small incision; however, the endoscopic technique has reported higher complication rate vis-a-vis accidental

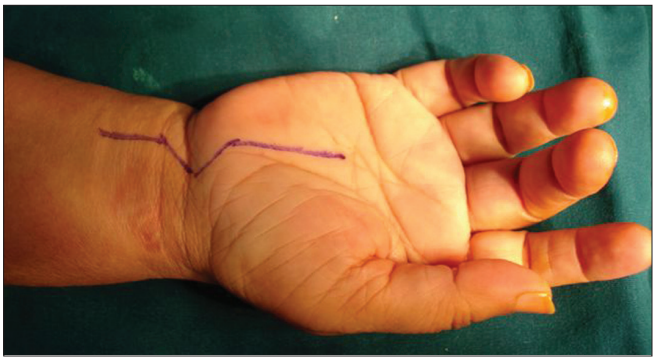

Figure 1: Incision marking ECTR

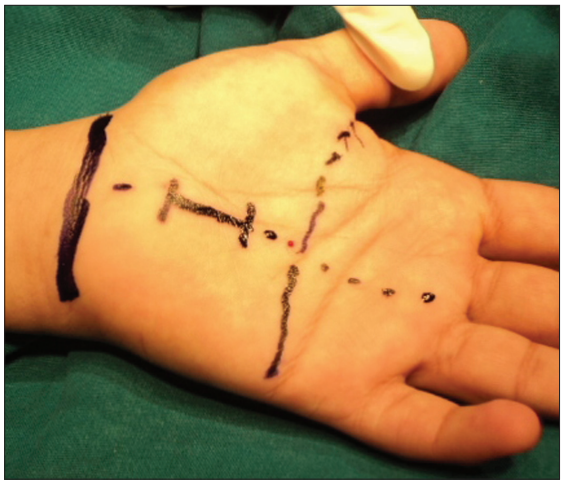

Figure 2: Incision MAOT

nerve damage. The minimal access open technique is therefore the author's preferred choice. [Figures of all three with details of MAOT].

Technique of MAOT (author's personal preference)

The incision is taken in the radial aspect of the 4th ray starting from 1 cm distal to the wrist crease for a further 2 cm. It is important not to cross the wrist crease to avoid scar morbidity due to continuous wrist movement. The layers are

- Skin
- Subcutaneous fat
- Palmar aponeurosis
- Roof of the carpal tunnel

The palmar aponeurosis incision [Figures 3 and 4] is extended into the distal forearm under vision using a right-angled retractor to hold the skin and subcutaneous tissue away. The retractor is then repositioned so that the volar carpal ligament and the distal deep fascia of the forearm in continuity with it is visible with a properly positioned light. The incision of the volar carpal ligament in the palm is started with a no. 15 blade till the edge of the forearm [Figures 5 and 6]. After this a blunt tipped but sharp straight scissor is advanced with one blade under the roof of the tunnel and the other above it. Under vision the complete volar carpal ligament and the deep fascia of the distal forearm in continuity with it is divided with one quick movement [Figure 7]. The scissor is withdrawn and the division inspected to rule out and remaining strand, if found this is cut sharply. The distal part of the palmar aponeurosis is then cut under vision till the superficial palmar arch is seen [Figure 8]. This is the end of the release. In the end the median nerve and its motor branch is inspected for continuity and the other contents noted. Unless the synovium is abnormally thick (such patients complain of an inability to make a fist preoperatively) it is left alone. If the patient has mentioned an inability to make a fist then a limited synovectomy is done until tendons are clearly seen. For the proximal synovium a separate transverse incision may be needed on the wrist [Figure 9], if there is considerable hypertrophy. Sometimes incidental findings like seeing a large lumbrical belly [Figure 10] originating in the FDP within the carpal tunnel is noted but is left alone. Postoperatively a bulky padded dressing with a cock up slab [Figures 11 and 12] is given for 48 h to avoid pain and oedema. This is removed at 48 h and replaced with a strap on splint, which is removable. This is, used by the patient as per his or her comfort levels. Typically patients give up the splint in 2 weeks. In bilateral cases the author gives a gap of 2–6 weeks between the 2 sides based on patients' ability to use the first operated side. Currently we use vicryl Rapide® sutures and these dissole on their own between day 7 and day 10, depending on frequency of washing, which is encouraged from day 3.

Rare compressions of the median nerve

An excellent review article on this subject by Dang *et al*[23] has listed the various areas. Possible areas for median nerve compression proximal to the carpal tunnel:

- The ligament of Struthers
- The bicipital bursa
- Anomalous arteries, and anomalous muscles (such as Gantzer's muscle, an accessory FPL muscle)
- Pronator syndrome
- Anterior interosseous nerve syndrome

The last two remain the two most frequently referenced compression neuropathies of the median nerve in the forearm.

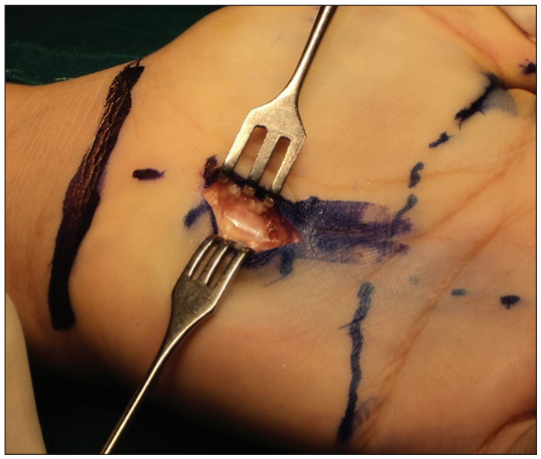

Figure 3: Sup palm apon

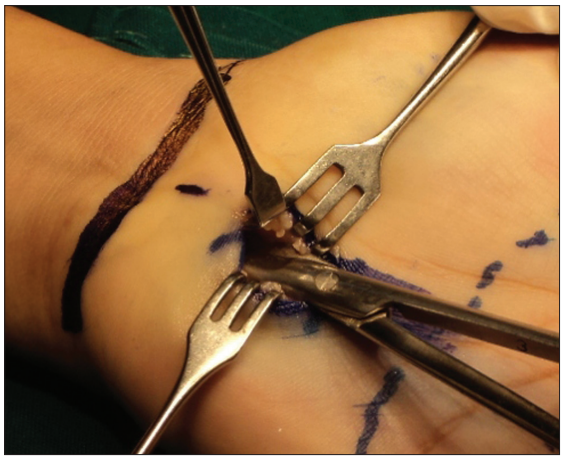

Figure 4: Palmar aponeurosis complete in palm and forearm

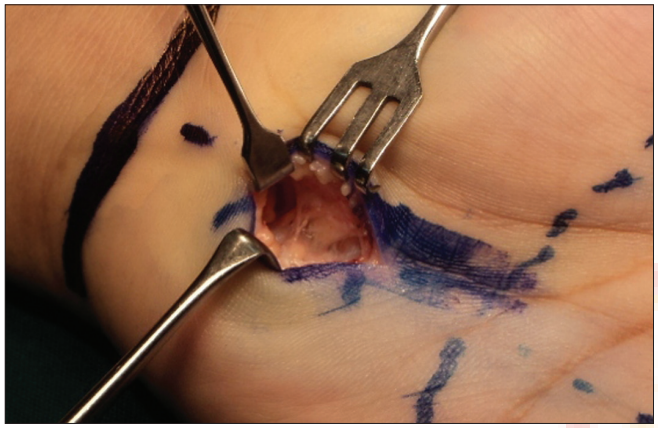

Figure 5: Roof of CT with distal hold fast fibres seen

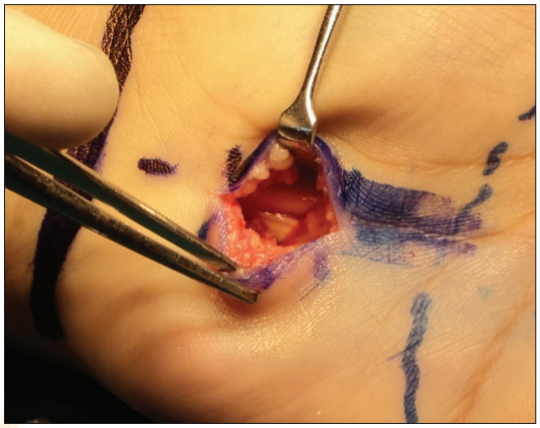

Figure 6: Median exposed after dividing the roof

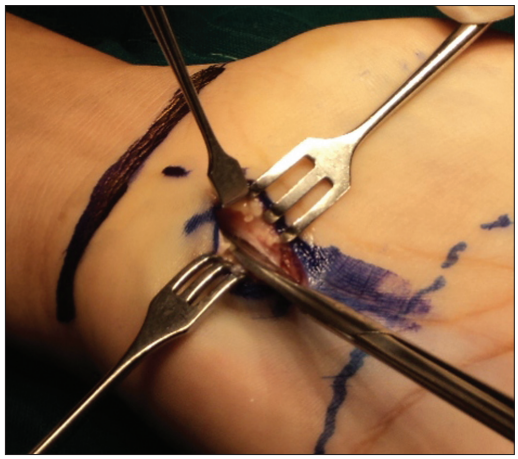

Figure 7: Completion in distal forearm under vision

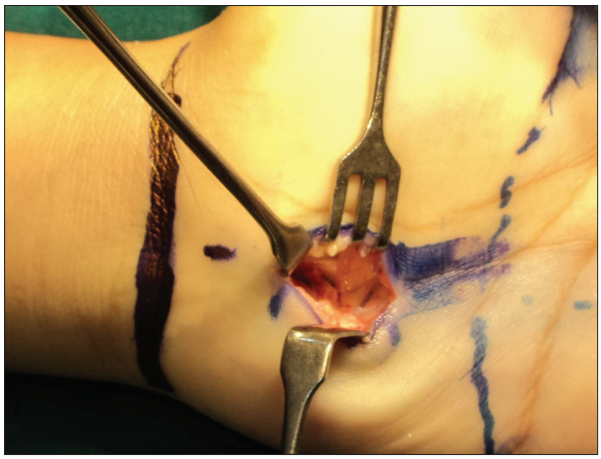

Figure 8: Note palmar arch distally

Pronator syndrome

Pronator syndrome (PS) commonly refers to compression of the median nerve as it passes between the two heads of the pronator teres muscle or under the proximal edge of the proximal FDS arch. Symptoms usually have an insidious onset and typically are not diagnosed as part of an overall clinical syndrome for months to years.[23]

Presenting symptoms:

- Aching pain in the proximal, volar forearm
- Paresthesias radiating into the median innervated fingers

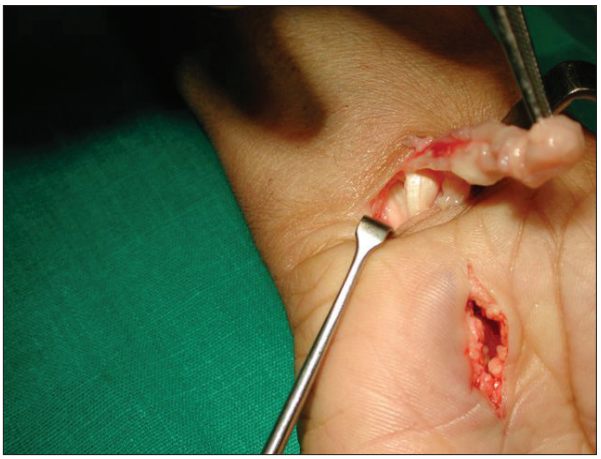

Figure 9: CT Syn. Cont

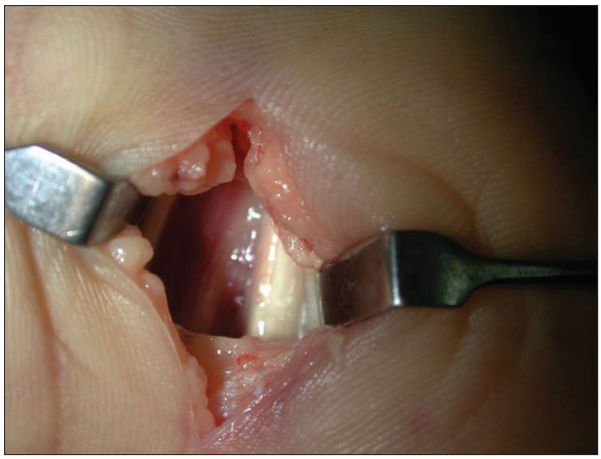

Figure 10: Carpal tunnel lumbrical

Figure 11: Bulky dressing using fluffed gauze

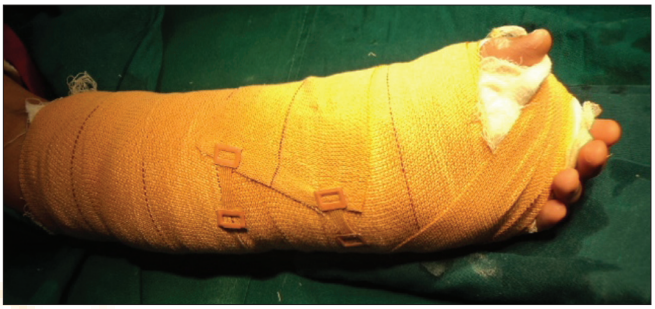

Figure 12: Slab with crepe bandage

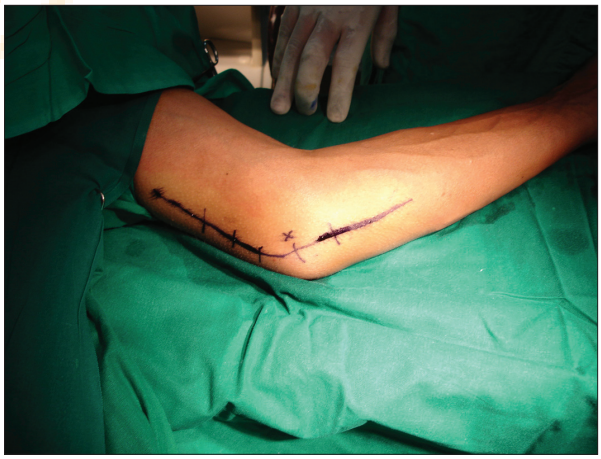

Figure 13: Incision for the release of cubital tunnel syndrome

- Worsened by repetitive pronosupination movements.

Discriminating clinically between PS and CTS:

- Loss of sensation over palmar cutaneous branch territory
- No Tinel's on the wrist
- No nocturnal disturbance
- Pain on resisted pronation from a neutral position, especially as the elbow is extended
- If resisted contraction of the FDS to the middle finger reproduces symptoms, median nerve compression at the level of the fibrous arch between the heads of the FDS might be suspected.[24]

If symptoms are elicited by resisted flexion of the forearm in full supination, compression at the more proximal level of the lacertus fibrosus might be considered.[25]

Electrodiagnostic studies can be useful in the diagnosis of PS in that they may exclude other sites of nerve compression or help identify a double-crush with a more proximal lesion in the neck.

Management

Surgery is usually not necessary to treat PS. Conservative therapy should be tried first. This includes

- Rest
- Avoid precipitating activity
- Steroids orally

Surgery is indicated after adequately long trial (several months) is given to conservative therapy. If a neoplasm

is suspected to compress the nerve or electrophysiology shows gross affection, again surgery is indicated. Surgery is similar to that done for anterior interosseous nerve (AIN) and is discussed in the next section.

Anterior interosseous nerve syndrome

Duchenne de Boulogne reported an isolated palsy of the FPL in 1872.[26] The AIN innervates the deep muscles of the forearm (FPL, FDP to the index and middle fingers, and pronator quadratus), a patient with a complete AIN palsy would present with

- Absent motor function to all three of these muscles.
- Pain may be present in the forearm along the course of the nerve.
- Inability to make an "OK" sign when asked by the examiner to flex his thumb interphalangeal joint and index finger distal interphalangeal joint [Figure 13].

In patients with mild AIN compression, subtle weakness of these muscles may be the only clinical finding. Such weakness of the FPL and index finger FDP may be uncovered by asking the patient to pinch a sheet of paper between his thumb and index finger using only the fingertips and then trying to pull the paper away. A patient with AIN syndrome may be unable to hold on to the sheet of paper with just his fingertips and may compensate by using a more adaptive grip in which the interphalangeal joint of the thumb and distal interphalangeal joint of the index finger remain extended

Differential diagnosis

- Brachial neuritis
- Viral neuritis (Parsonage–Turner syndrome)
- Rupture of the FPL tendon

1 and 2 above will resolve spontaneously with conservative management and 3 is usually a result of rheumatoid arthritis if there is no history of trauma.

Electrodiagnostic studies are an important element in the workup of AIN syndrome because they confirm the diagnosis and objectively assess the severity of the neuropathy. Electromyography is often helpful in ruling out more proximal lesions as a cause of the symptoms.

Management

Treatment is initially conservative with the rest, splinting, and observation. It is suggested that observation under this regime should be for a period of several months before surgery is planned for failure of conservative treatment. AIN syndrome seems to improve without any surgical intervention,[27,28] It must be noted here that there is a paucity of high-quality literature supporting the appropriate duration of conservative treatment.[23]

Technique

Common for PS and AIN syndrome. The incision spans the elbow crease and is on the anterior aspect of the forearm in a lazy S shape. After the deep fascia is reached the author likes to see the lacertus fibrosus and incise it. This reveals the median nerve with the brachial artery. Keeping the vascular bundle safe the median is then traced up in the upper arm to see the ligament of Struthers if it is present and compressing the nerve. After that all the possible sites of compression are checked out including the two heads of PT, the arcade at FDS origin, the AIN in its entire course etc. Please note that the AIN comes out radially from the median nerve while most other branches come out Ulnarward.[23] If abnormal muscles like the Muscle of Ganzer are present they too are released. The wound is closed with a drain after meticulous haemostasis and a padded dressing with slab in elbow flexion at 135° is given for 2 weeks after which mobilisation is allowed. Results are usually very good, especially if a cause of compression is found.

Cubital tunnel syndrome/ulnar neuropathy at elbow

Entrapment of the ulnar nerve is the second most common compression neuropathy in the upper extremity after CTS.[29] Although the ulnar nerve may be compressed at multiple points along its course, the most common location is at the elbow.

The ulnar nerve arises from the medial cord which derives from the lower trunk of the brachial plexus. It represents C8T1 roots. It courses between the medial head of the triceps brachii and the brachialis muscles. The nerve is posteromedial to the brachial artery and just posterior to the intermuscular septum. The arcade of Struthers is a band of fascia that connects the medial head of the triceps with the intermuscular septum of the arm. It crosses the ulnar nerve approximately 8 cm proximal to the medial epicondyle. The ulnar nerve then becomes more superficial and enters the ulnar sulcus approximately 3.5 cm proximal to the medial epicondyle. The nerve courses posterior to the medial epicondyle and medial to the olecranon. The nerve then enters the cubital tunnel. The

roof of the cubital tunnel is defined by the Osbourne's ligament[29] which is a thickened transverse band between the humeral and ulnar head of the flexor carpi ulnaris (FCU). The floor of the cubital tunnel consists of the medial collateral ligament of the elbow, the elbow joint capsule and the olecranon. After passing through the cubital tunnel, the ulnar nerve courses deep into the forearm between the ulnar and humeral heads of the FCU. Potential ulnar nerve entrapment can occur at five sites around the elbow:

The arcade of Struthers (different from Struthers ligament in case of median nerve)
- The medial intermuscular septum
- The medial epicondyle
- The cubital tunnel, and
- The deep flexor pronator aponeurosis.

The most common site of entrapment is the cubital tunnel.
Symptoms:
- Numbness and paresthesia in the Ring and Little Fingers
- Difficulty in gripping—intrinsic weakness (CTS patients say the same but the cause is in the opposition failure and sensory proprioceptive failure)
- Pain, tenderness over elbow region medially
- Particularly over the ulnar nerve
- Wasting of ulnar innervated intrinsics

Categories of nerve dysfunction (McGowan[30] and Dellon[31])
- Mild nerve dysfunction implies intermittent paresthesias and subjective weakness.
- Moderate dysfunction presents with intermittent paresthesias and measurable weakness.
- Severe dysfunction is characterized by persistent paresthesias and measurable weakness.

Provocative tests[29,32]:
- Tinel's test along the course of the ulnar nerve
- Elbow flexion test.
- Pressure provocation test (where direct pressure is applied to the cubital tunnel for 60 s) and a
- Combined elbow flexion–pressure test.

A positive Tinel's test is only 70% sensitive, whereas the elbow flexion test is 75% sensitive after 60 s. However, after 60 s, the pressure test is 89% sensitive, and the combined elbow flexion–pressure test is 98% sensitive. These examination findings can be used in combination to best diagnose cubital tunnel syndrome.

Predisposing causes:
- Childhood supracondylar fracture (tardy ulnar nerve palsy)
- Chronic valgus stress
- Elbow fractures that are treated without ulnar nerve transposition (olecranon fractures, distal humerus fractures, medial epicondylar fractures).

Always examine if nerve does/does not subluxate over the medial epicondyle.

Treatment

Mild cubital tunnel syndrome can often be treated conservatively. There is a tendency toward spontaneous recovery among patients with mild and/or intermittent symptoms if provocative causes can be avoided and adequate rest and bracing used for the elbow.

Surgical treatment

Surgical treatment of cubital tunnel syndrome is indicated with motor weakness or when conservative measures have failed. There are multiple techniques currently recommended for treatment of cubital tunnel syndrome, and there is ongoing controversy as to which is the optimal surgical treatment of this nerve entrapment. The most common surgical treatments include
- In situ decompression
- Subcutaneous transposition
- Intramuscular transposition
- Submuscular transposition
- Medial epicondylectomy.

Surgical technique

The technique preferred by the author is being described here. The incision is taken in a curved fashion on either side of the medial epicondyle, going superiorly for about 8–10 cm and inferiorly for about 8 cm [Figure 13]. The ulnar nerve is visualised entering the cubital tunnel from behind the groove on the medial epicondyle. It is traced up till the purported site of the Struthers arcade and all five sites described earlier are checked out and decompressed if needed [Figures 14–16]. Care is taken to preserve the posterior branch of the medial antebrachial cutaneous nerve during the approach to the nerve [Figure 17] and the motor branches during the exploration between the two heads of the FCU. If the nerve is seen subluxating on elbow flexion then a subcutaneous transposition is done

using a flap of fascia to hold it in place. Care must be taken to avoid creating a new tight tunnel. If the nerve does not subluxate under vision on elbow flexion then it is left *in situ*.

Meta-analysis

In a scholarly review Calliandro *et al*[33] in a Cochrane Systematic Review have concluded:

'The available evidence is not sufficient to identify the best treatment for idiopathic ulnar neuropathy at the elbow on the basis of clinical, neurophysiological and imaging characteristics. We do not know when to treat a patient conservatively or surgically. However, the results of our meta-analysis suggest that simple decompression and decompression with transposition are equally effective in idiopathic ulnar neuropathy at the elbow, including when the nerve impairment is severe. In mild cases, evidence from one small randomised controlled trial of conservative treatment showed that information on movements or positions to avoid may reduce subjective discomfort.'

Radial tunnel syndrome and posterior interosseous nerve compression and superficial radial nerve compression

Although comparatively rare, clinical cases of compression of radial nerve are occasionally encountered. The radial nerve arises from the posterior cord with contributions from all three trunks. Consequently its root value is C5–T1.

Anatomy

The nerve begins posterior to the axillary artery and travels through the triangular space and then continues along the spiral groove of the humerus. The branches to the triceps are given off before this transition. All branches (sensory or motor) beyond the spiral groove pertain to the hand and forearm (Anconeus is the exception). The nerve travels from the posterior compartment of the arm into the anterior compartment as it penetrates the lateral

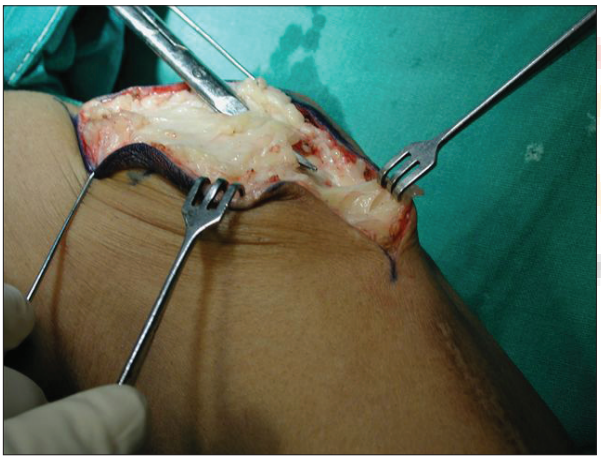

Figure 14: Cubital tunnel opened

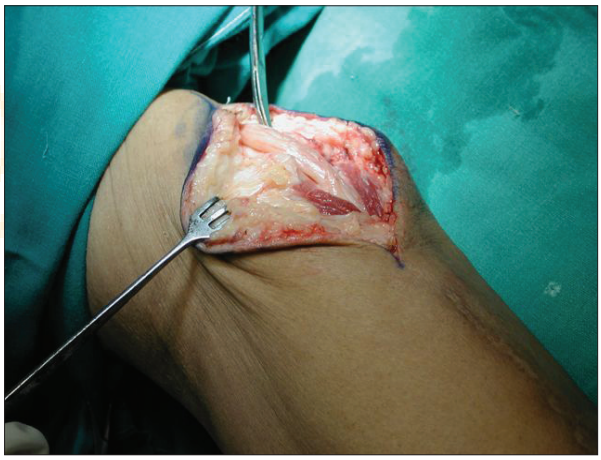

Figure 15: Cubital tunnel opened

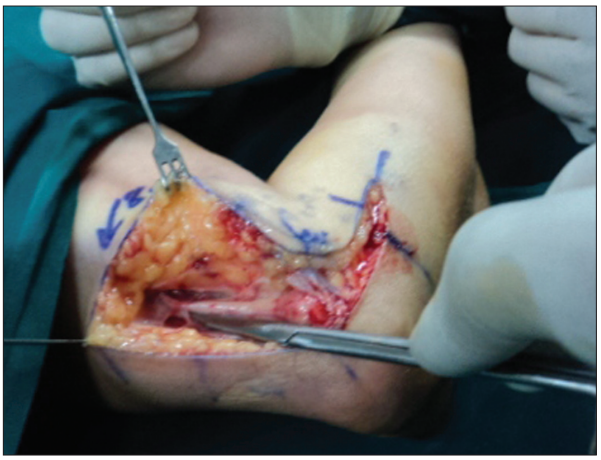

Figure 16: Arcade of Struthers and neuroma at the entrance of cubital tunnel

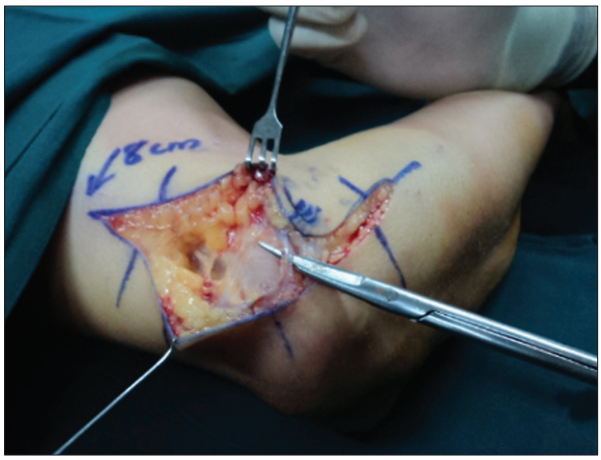

Figure 17: Subcutaneous nerve

intermuscular septum approximately 10–12 cm proximal to the elbow. The radial nerve continues to travel distally and ultimately bifurcates into deep (PIN) and superficial (SRN) branches approximately 6.0–10.5 cm distal to the lateral intermuscular septum and 3–4 cm proximal to the leading edge of the supinator.[35,36] The PIN is a motor nerve that courses deep beneath the supinator muscle; the SRN is a sensory nerve that travels anteriorly on the undersurface of the brachioradialis and, in the distal one-third of the forearm, travels subcutaneously to provide sensation to the dorsoradial hand.[34,35]

Posterior interosseous nerve compression

The PIN travels through the radial tunnel. Distally innervating the ECRB, supinator, ECU, EDC, EDM, APL, EPL, EPB, and EIP. It does not innervate the extensor carpi radialis longus (ECRL). The radial tunnel[34] is a potential space 3–4 finger breadths long, lying along the anterior aspect of the proximal radius through which the PIN travels. The floor of the radial tunnel is created by the capsule of the radiocapitellar joint, which continues as the deep head of the supinator muscle. Anatomically, there are five potential sites of compression of the PIN in the area of the radial tunnel[34]:

- Fibrous bands of tissue anterior to the radiocapitellar joint between the brachialis and brachioradialis
- The recurrent radial vessels that fan out across the PIN at the level of the radial neck as the so-called leash of Henry
- The leading (medial proximal) edge of the extensor carpi radialis brevis (ECRB)
- The proximal edge of the superficial portion of the supinator, commonly referred to as the arcade of Fröhse
- The distal edge of the supinator muscle.

Causes:

- Neoplasm
- Elbow pathology—such as synovitis associated with rheumatoid arthritis
- Abnormal anatomical compression in the above-mentioned five sites

Symptoms and signs:

- Inability to extend fingers and thumb
- ECRL function intact—the wrist extends and radially deviates

Interestingly, RTS and PIN syndrome describe compression of the same nerve, and therefore can be approached with identical surgical interventions. Although in each syndrome the same nerve is affected, the clinical presentations are divergent. Whereas patients with PIN syndrome have a loss of motor function, patients with RTS typically, present with mobile wad and lateral forearm pain without motor involvement. The difference in clinical presentation may well be attributed to a difference in the degree of compression.

Radial tunnel syndrome

The same nerve is being compressed in the same location in RTS. However, it is typically only pain, discomfort and inability to perform routine tasks that is the presenting complaint. There is no motor weakness. Unlike a case of lateral epicondylitis the pain is not on the lateral epicondyle of the humerus but slightly distal to it. It is described as being in the area of the mobile wad and radial tunnel.[34]

Symptoms and signs:

- Pain distal to lateral epicondyle, tenderness over the radial tunnel along the path of PIN[37]
- Pain worsened by extending the elbow, pronating the forearm and flexing the wrist
- Pain with resisted active supination or wrist extension
- Pain with active supination against resistance
- Pain with wrist extension against resistance
- Pain with resisted middle finger extension at the metacarpophalangeal joint[38,39]
- No neurological deficit
- Pain disappears after instilling local anaesthetic at the site of entry of PIN

Electrodiagnostic studies in the diagnosis of RTS have been described, although they are almost always unrevealing in the absence of a PIN syndrome. Forearm rotation can produce differential latencies in nerve conduction studies under laboratory conditions, but the vast majority of patients with RTS have normal electrodiagnostic testing.

Differential diagnosis:

- Lateral epicondylosis
- Osteoarthritis of the radial capitellar joint
- Impingement of the articular branch of the radial nerve
- Synovitis of the radiocapitellar joint
- Muscle tear of the extensor carpi radialis brevis.[40]

Management

Initially conservative management is recommended. It

consists of

- Rest and splinting
- Avoid provocative postures
- Injection of local steroids with or without local anaesthetic

Surgical decompression

Various methods have been described to expose the PIN at the elbow. Techniques differ about which interval to approach. It is either the gap between ECRL and BR[41] or ECRL and EDC,[42] essentially both approaches take a semi-curved/longitudinal incision over the radia aspect of elbow and find the appropriate muscle interval to identify the radial nerve. The author prefers the former as he feels the dissection is easier but that is matter of experience with individual technique [Figures 18–21]. The radial nerve is seen under the BR and can then be followed. After this essentially we need to look for all sites of compression and decompress them. If a neoplasm is seen compressing the nerve it should be removed. Some believe that the lengthening of the extensors should be added as a precaution, both to reduce pressure and also to treat an element of lateral epicondylosis if it exists simultaneously. Padded dressing is given and a slab to hold the elbow in 45° flexion and mid prone position for 2 weeks. Gentle mobilisation is started thereafter.

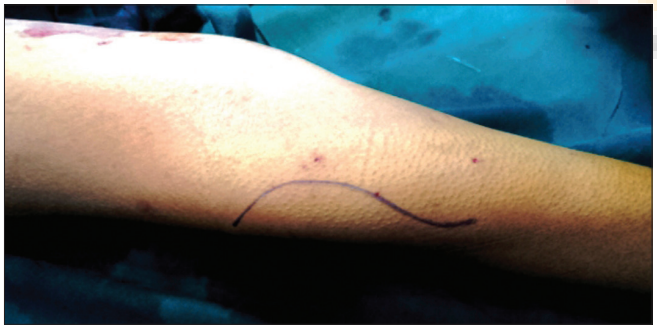

Figure 18: Incision RN

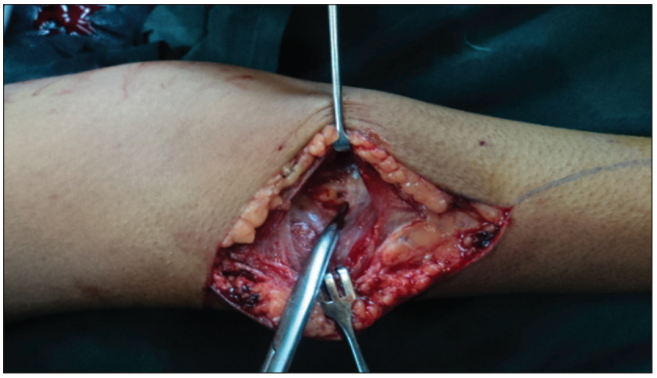

Figure 20: RN identification

DISCUSSION

RTS is a much rare entity than CTS or cubital tunnel syndromes. The quoted incidence of RTS in the series reported by Weitbrecht and Navickine[43] were 957 CTS, 143 cubital tunnel syndrome, and 12 cases of supinator syndrome (RTS), a rate of 1%. However, a high index of suspicion may uncover some cases which are being conserved for too long with no relief.

Compression of the superficial radial nerve

Also called Wartenburg syndrome after the person who described it in 1932.[44] Wartenberg published a series of five adult patients describing an isolated neuropathy of the SRN. Due to its clinical similarity to isolated neuropathy of the lateral femoral cutaneous nerve in the lower extremity (also known as meralgia paresthetica), Wartenberg coined the term 'cheiralgia paresthetica'. It must be added that others have described it before,[45,46] but Wartenberg's name is widely known.

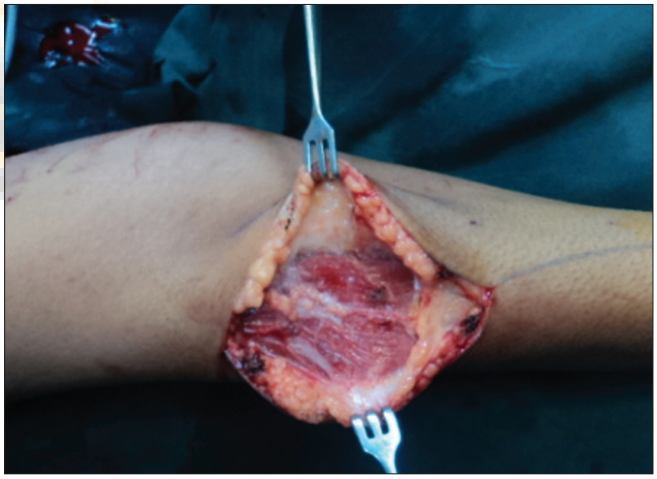

Figure 19: BR and ECRL plane

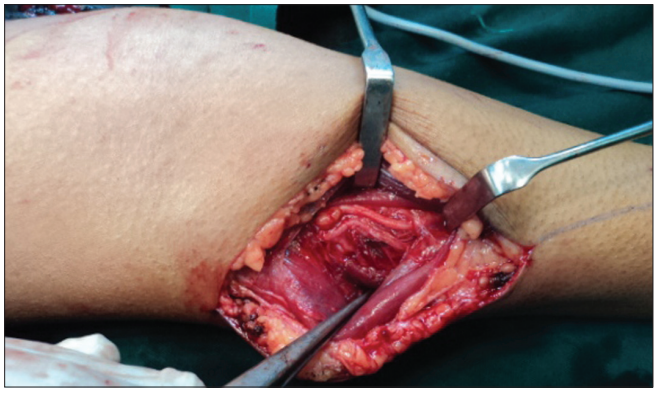

Figure 21: Released RN

Approximately 9 cm proximal to the radial styloid, the SRN becomes a subcutaneous structure by traveling between the brachioradialis and ECRL tendons[34]; it supplies sensation to the dorsum of the thumb, index, and middle fingers proximal to the proximal interphalangeal joints.

The SRN can be compressed at any point along its course in the forearm, but it is believed to be at greatest risk at the posterior border of the brachioradialis as the nerve transitions from a deep to a subcutaneous structure. Trauma is also a common etiology for SRN compression, which can occur from direct pressure on the nerve (i.e., by a wristband[45,46] or handcuffs[47-49]) or from a stretch injury to the nerve (i.e., during a closed reduction of a forearm fracture[50]).

Symptoms

Patients with SRN compression typically report pain or dysesthesias on the dorsal radial forearm radiating to the thumb and index finger, although the distribution of symptoms may vary owing to differences in anatomy.[34]

Management

Conservative treatment is preferred. It consists of

- Removing causative items, such as bangle, watch, bracelet
- Rest
- Splinting
- Non-steroidal anti-inflammatory drugs
- Occasional trial of local steroid.

Surgical decompression is offered only to patients who failed conservative therapy or whose symptoms are longstanding and have no distal progression of a Tinel's sign,[34] it had a 74% success rate in reported series.[51] Surgical decompression may also be indicated in post-traumatic situations to remove scar tissue and achieve decompression.

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