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Upper Extremity Nerve Compression Syndromes – More Than Just a Carpal Tunnel Syndrome: A Review Article

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Abstract

BACKGROUND: Besides carpal tunnel syndrome and cubital tunnel syndrome, other nerve entrapment sites in the upper extremity are less recognized. Only half of the upper extremity compression neuropathy syndromes are actually carpal tunnel and cubital tunnel compressions. This suggests that the rest of the entrapment syndromes are potentially not treated adequately. They are often misdiagnosed or the level of compression is not being distinguished.

AIM: To raise awareness of compression syndromes (other than the carpal tunnel) in the arms and to describe the clinical triad to diagnose them.

METHODS: This is a narrative review of the clinical features of the compression syndromes of the median, ulnar, and radial nerves in the upper extremities. The diagnostic triad for each syndrome is analyzed. The review focuses especially on the lacertus syndrome and describes the surgical release.

CONCLUSION: A correct diagnosis is essential for targeted treatment of upper extremity nerve compression syndromes. It should also include an assessment of individual muscle strength, which is a critical part of the clinical neurological tests.

Introduction

Carpal tunnel syndrome (CTS) is a widely known, diagnosed, and treated compression syndrome. Unfortunately, it is misdiagnosed in 10-20% of cases because most median nerve compression symptoms are erroneously recognized as compression of the median nerve at the level of the wrist joint, while other compression sites are overlooked. In those cases, the compression was higher in the forearm and elbow area [1], [2], [3], [4]. Diagnostic difficulties exist in distinguishing the level of compression of the ulnar nerve, whether at the elbow (Cubital tunnel syndrome) or at the wrist (Guyon's canal) [5]. Besides carpal tunnel syndrome and cubital tunnel syndrome, other nerve entrapment sites in the upper extremities are less recognized by the majority of surgeons treating musculoskeletal pathologies. About 60% of the upper extremity compression neuropathy syndromes are actually carpal tunnel and cubital tunnel compressions [6]. This suggests that the rest of the entrapment syndromes are potentially not treated adequately. They are either misdiagnosed as cervical radiculopathy or carpal tunnel/cubital tunnel syndrome.

Neuropathy is a broad term referring to the clinical presentation of sensory abnormalities (pain, paresthesia, numbness) or motor weakness in the expected distribution of a particular nerve and can be caused by metabolic abnormalities, such as diabetes or by structural abnormalities. Entrapment syndrome is neuropathy due to a structural abnormality, such as compression, displacement, or traction of the nerve, or by an intrinsic abnormality of the nerve, such as a nerve

cell tumor [5].

Without a proper diagnosis, adequate treatment cannot be provided. In practice, many years and even decades pass until the correct diagnosis is reached in a large number of compression neuropathy patients. Neurolytic surgery (nerve decompressions, dynervology) for upper extremity nerve entrapments has been undertaken for years, but its success depends on the precise location of the entrapment, and in some cases, it is challenging. The term “dynervology" refers to eliminating the pain caused by nerve entrapment.

This manuscript aims to raise awareness of compression syndromes (other than carpal tunnel) in the arms and how to diagnose them, with special emphasis on the lacertus syndrome.

Three major nerves course through the forearm to innervate the distal extremity: the median, ulnar, and radial nerves. Each can be affected in compression syndromes. Understanding the muscular anatomy, innervation, and sensory distribution of the upper extremities is critical for diagnosing and treating compressive neuropathies. The surgeons treating upper extremities need to be familiar with the following nerve entrapments: Radial nerve entrapments: Lateral intermuscular syndrome and Posterior Interosseous Nerve-Radial tunnel syndrome; Median nerve entrapments: Lacertus syndrome and Carpal tunnel syndrome; and Ulnar nerve entrapments: Cubital tunnel syndrome and Guyon’s canal.

Clinical Diagnosis

The triad of neuropathic symptoms, pain, weakness, and numbness/tingling suggests an involvement of the peripheral nerves. Consequently, the physical examination must be able to identify or rule out an impaired function of these. Focal neuropathies cause rather specific neurological patterns: If muscles are innervated distally to a nerve lesion, they are likely to be weak; the sensibility in supplied cutaneous territories will be altered; there will be abnormal soreness where the nerve trunk is affected. The classical neurological examination is based on these three principles. Still, there seems to be an unwillingness to perform a detailed neurological examination in upper extremity patients [7].

The clinical examination techniques have a pivotal role, emphasizing the clinical triad of muscle, sensory, and pain testing in diagnosing nerve compression syndromes [8].

Compression neuropathies are commonly caused by mechanical pressure and are found in areas where the nerve passes a joint or a distinct compressive anatomical structure [9]. The effect of the compression is dose-dependent, meaning that a lesser compression will only result in decreased endoneurial circulation, neural edema, and a Seddon’s grade IV weakness [10], whereas a marked nerve compression will result in axonal degeneration, demyelination, and evident muscular atrophy [9]. The latter scenario is easier to diagnose but difficult to treat. In contrast, the former presents a clinical challenge as it will not be readily revealed in nerve conduction or magnetic resonance imaging studies [6].

In the last decade, several clinical options to diagnose compression neuropathies in the upper extremity have been published. A thorough physical examination of the upper limb should comprise inspection and palpation in the search for atrophies and focal tenderness; evaluation of joint status in terms of pain, swelling, and mobility; and sensory function, motor function, and grip strength. To complete the examination, testing of the strength of individual muscles has to be done in order to diagnose impaired nerve function [6].

A nerve branch that is severed close to its entry point into a muscle will result in paralysis of that specific muscle. If the injury occurs further up the nerve, more muscles will be affected by paralysis. By understanding the branching of the nerve, we can identify the exact location of the injury based on the muscles that are paralyzed [6]. A classic example known to all clinicians is "drop hand," which serves as a reliable indicator of a radial nerve injury at the level of the upper arm.

The validity and reliability of the technique for manual testing of the strength of individual muscles have been well documented in peer-reviewed studies [11], [12], [13]. Dr. Elizabet Hagert elaborated on and popularized it [6], [8].

To diagnose nerve compressions effectively, clinical evaluations must assess both motor and sensory function while identifying pain at the compression site. The clinical triad for a structured assessment includes: Manual muscle testing, from proximal to distal, to determine the level of compression; Sensory provocative testing, such as sensory-collapse test (SCT), to confirm compression levels; and Pain testing: Evaluating for pain over the site of compression to further verify the diagnosis [8], [14].

Electrodiagnostic studies

Electrodiagnostic studies are commonly used to diagnose suspected nerve pathologies in the upper extremities, but their sensitivity and specificity are limited, ranging between 30 and 65% [15], [16]. Electrophysiological evaluation is limited in the detection of mixed nerve injuries and is unable to provide comprehensive muscle function assessments

or identify early-stage compressions. Clinical examination techniques should be integrated with or prioritized over sole reliance on electrodiagnostic [8].

The accuracy in correctly diagnosing radial tunnel and pronator syndromes using electrophysiological techniques is disappointing [15], [17], [18]. Of the pronator syndromes analyzed electrophysiologically, 50% had been diagnosed as CTS and subsequently surgically treated with open carpal tunnel release without improvement [6]. Eighteen percent had negative findings, 4% had diabetic polyneuropathy, and 18% had "anterior interosseous" syndrome. Of all EMG studies performed on patients with pronator and radial tunnel syndromes, only one correctly diagnosed the level of entrapment [6].

Nerve conduction studies can be valuable in identifying focal neuropathies and assessing potential polyneuropathies. However, they should not be relied upon as the sole diagnostic method for determining the presence or severity of nerve damage. Instead, these studies should be considered as an adjunct to a comprehensive clinical evaluation for a more accurate diagnosis.

Manual muscle strength testing

The use of manual muscle testing in clinical practice has proven to be an essential and reliable method for diagnosing the entrapment level of focal neuropathies in patients with compression neuropathy syndromes and chronic pain conditions in the upper extremities.

For accurate results, manual muscle testing requires a thorough understanding of the upper extremity's topographical and functional anatomy. Clinicians must be familiar with the specific muscles to test and must correctly interpret any observed muscle weaknesses. Accurate interpretation of these findings is crucial before drawing any conclusions about the patient's condition.

When a nerve is completely transected or crushed, it is crucial to acknowledge that every muscle it innervates distal to the injury will inevitably become paralyzed. However, with nerve entrapment, we must be careful not to expect weakness and uniform findings across all associated muscles. Research by Rydevik and Lundborg reveals that the pressure impacting a nerve diminishes as it penetrates deeper [19]. As a result, the most superficial fascicles endure the most harm, while those deeper within may remain unaffected. This distinction clarifies why not all muscles in the innervation zone exhibit weakness during nerve entrapment scenarios [6]. For instance, in a study investigating median nerve entrapment at the elbow, known as pronator syndrome, the flexor carpi radialis (FCR) was weak in 24 out of 25 cases. In comparison, the flexor pollicis longus (FPL) and flexor digitorum profundus II (FDP-II) displayed weakness in 23 out of 25 cases. Remarkably, the flexor digitorum superficialis (FDS) and abductor pollicis brevis (APB) showed no weakness in 12 out of 25 cases. This variability underscores the complexity of nerve entrapment and emphasizes the need for careful evaluation [6] [20].

The muscle strength has been classified into six grades, as proposed by Seddon [10], on a scale from 0 (no muscle power) to 5 (full strength against maximum resistance). Grades 0 through 3 refer to either a complete loss of muscle strength or a significant reduction of active range of motion. Although grades 4 and 5 may appear normal, a paresis grade 4 may, in fact, entail a 15%–30% reduction of muscle strength as compared to the healthy, contralateral side [6]. A systematic, individual muscle strength testing is needed to reveal the presence of this grade 4 paresis, as it may be a significant sign of impaired nerve function, as the one in nerve entrapments. To be even more challenging to diagnose, the EMG studies will not reveal this low-grade compression neuropathy [21], [22], [23]. Therefore, this grade 4 paresis is difficult to identify and needs to be searched for by using the manual muscle testing technique.

Out of the 60 muscles in the upper limb, about 45 are accessible to manual testing of individual strength [6]. As suggested by Carl Göran and Elisabet Hagert, that number is both unrealistic and quite unnecessary and the following eight muscles give a reasonable representation of the upper extremity nerve tree as a whole: pectoral, deltoid, biceps, triceps, infraspinatus, flexor carpi radialis (FCR), extensor carpi ulnaris (ECU), and abductor digiti minimi (ADM) [6]. They suggest that weakness of the FCR with a normal deltoid and biceps is a very reliable sign of a median nerve affliction at the elbow level (lacertus syndrome). A selective weakness of the ECU is a reliable sign of radial tunnel syndrome. If both FCR and ECU appear weak, it is a reliable sign of concomitant lacertus and radial tunnel syndromes. A selective weakness of the ADM is a reliable sign of an ulnar nerve entrapment, either at the elbow or at the wrist. If both the FCR and ADM are weak, it is indicative of a concomitant pronator and cubital tunnel syndrome.

Upper extremity muscle testing should be done as a standardized algorithm to identify the level of nerve compression [8], [11]. General aspects of muscle testing include starting proximally and working our way distally in muscle testing; performing bilateral comparisons of strength, being consistent in testing, and applying adequate force. Figure 1 depicts the method for testing the muscle strength of eight upper extremity muscles as suggested by E. Hagert and C-G Hagert [8] and Jepsen [24].

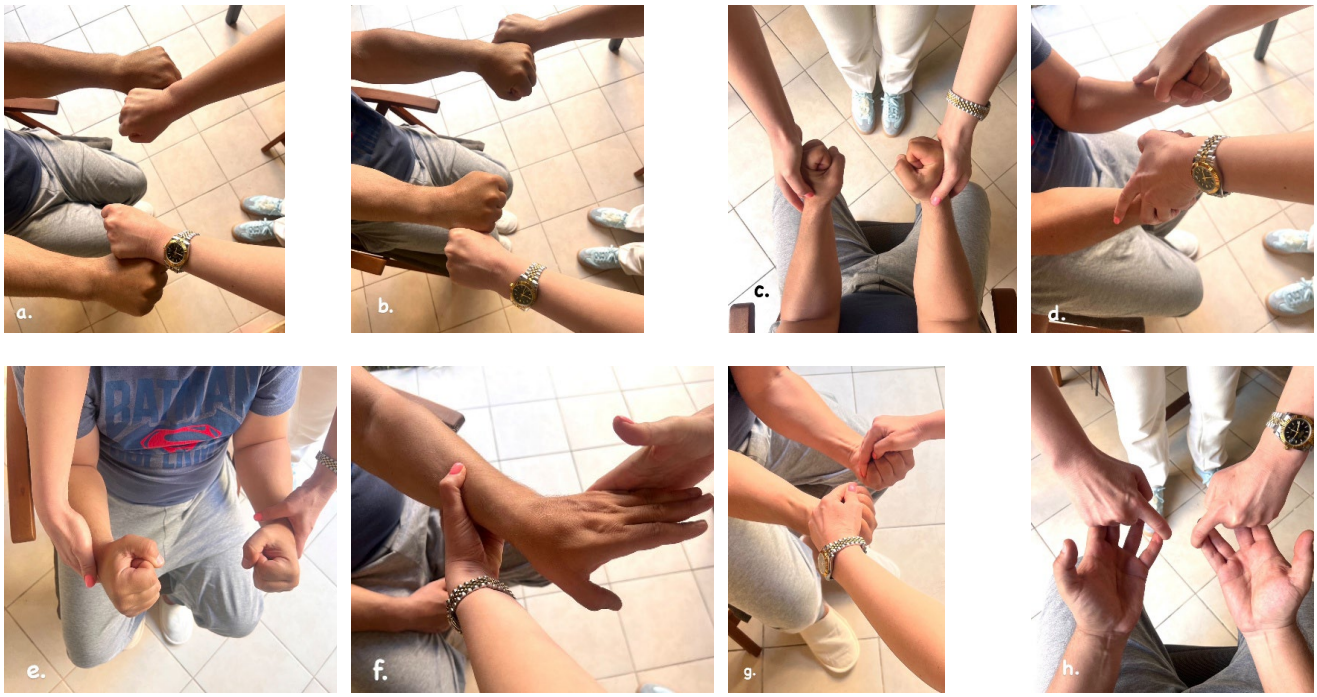

Figure 1: Upper extremity muscle strength testing. a. Shoulder adduction (pectoralis), b. Shoulder abduction (posterior deltoid), c. External shoulder rotation (infraspinatus), d. Elbow flexion (biceps), e. Elbow extension (triceps), f. Wrist ulnar deviation (ECU), g. Wrist flexion (FCR), h. Little finger abduction (ADM)

Sensory Provocative Testing

The sensory-collapse test (SCT), first described as a scratch-collapse test, is a useful confirmatory tool used alongside other diagnostic methods, not as a stand-alone tool for nerve compression diagnosis [8], [25]. The superficial fascicles of the nerve at any compression point will usually generate motor or sensory deficits associated with a specific nerve compression syndrome. The axonal organization of the nerve at that point will, therefore, explain the patterns of weakness found in muscle testing [26].

The clinical triad in most common nerve compressions

I. Median nerve compressions

Table 1, published by Dr. Elisabeth Hagert, provides a clear guideline on the clinical triad for diagnosing and distinguishing the different sites of median nerve entrapments: Lacertus syndrome, Superficialis Syndrome, Anterior Interosseus Syndrome, and Carpal Tunnel Syndrome [8].

Table 1: The clinical triad for median nerve compression syndromes [8]

Median Nerve Triads	Motor Weakness	The sensory-collapse test	Pain (compression site)
Lacertus Syndrome	FCR FDL FDP to index	Over the lacertus fibrosis	At the ptoximal ede of the lacertus fibrosis
Superficialis Syndrome	FDS to middle ring fingers	5-7cm distal of the elbow crease, over the superficialis arcade	At the superficialis arcade
Anterior Interosseous Sy	FPL FDP to index PQ	Mid-forearm level, 2-3 cm distal of the superficialis arcade	Volar mid-forearm
Carpal Tunnel Syndrome	APB	At the level of the carpal tunnel	Wrist flexion crease (+Tinel's test)

Abbreviations: FCR flexor carpi radialis, FPL flexor pollicis longus, FDP flexor digitorum profundus, PQ pronator quadratus, APB absuctor pollicis brevis

Lacertus syndrome

Lacertus syndrome is a compression of the median nerve in the proximal forearm at the level of the lacertus fibrosus.

The clinical triad for the diagnosis of lacertus syndrome is: Weakness in the flexor carpi radialis (FCR), flexor pollicis longus (FPL), and flexor digitorum profundus in the index (FDP2); Sensory testing is directed to the area from the ulnovolar antecubital fossa to the LF; and Pain will be present at the level of the LF [8].

Carpal tunnel syndrome (CTS)

CTS is the most commonly described nerve compression. The clinical trial would be [8]: Thenar atrophy may be seen in advanced CTS, and in earlier stages of nerve compression, a subtle weakness in the abductor pollicis brevis (APB) is most commonly found.; A SCT would be positive over the proximal edge of the carpal tunnel; and CTS is only sometimes associated with pain on compression. The Tinel's test, percussion of the median nerve at the carpal tunnel will result in paresthesias distally in the area innervated by the nerve.

II. Ulnar nerve compressions

Ulnar-sided numbness in the hand can be a result of compression anywhere from the C8 level, through the thoracic outlet to the hand. In addition to the clinical triad, a careful assessment of the distribution of numbness – ulnar hand, ulnar forearm, ulnar upper arm, shoulder – is essential to stage the level of nerve affliction in these patients [27]. Table 2 presents the clinical triad to diagnose and distinguish different levels of compression over the ulnar nerve [8].

Table 2: The clinical triad for diagnosing ulnar nerve compression syndromes [8]

Ulnar nerve triads	Motor Weakness	The sensory-collapse test	Pain (compression site)
C8-radiculopathy	FCU FDP little finger Intrinsic hand muscles	Level of the C8, back of the neck	No specific pain point on compression; generalized pain on neck movement; radicular pain
Thoracic outlet syndrome (TOS)	FDP to index FPL Intrinsic hand muscles	Scalene interval, side of the neck	Pain at the lower scalene interval on the side of the neck
Cubital tunnel syndrome	FCU FDP small finger Intrinsic hand muscles	Staged SCT from proximal to distal of the cubital tunnel to confirm level of compression	Pain test based on SCT finding. Common: distal of the cubital tunnel
Ulnar tunnel (Guyon's canal)	Ulnar intrinsic hand muscles, especially ADM FDI	Ulnovolar wrist crease and/or hook of hamate	Proximal of Guyon's canal and/ or hook of hamate

Abbreviations: C8 cervical level 8, FCU flexor carpi ulnaris, FPL flexor pollicis longus, FDP flexor digitorum profundus, ADM abductor digiti minimi, FDI first dorsal interosseous

Cubital tunnel syndrome

The ulnar nerve can be compressed at various locations around the elbow. The most frequent site is at Osborne's ligament [28], and other potential areas of compression include the arcade of Struthers [29], the medial intermuscular septum located proximal to the cubital tunnel, and the flexor carpi ulnaris (FCU) arcade.

Muscle testing [8]: Weakness is consistently found in the flexor digitorum profundus for the little finger (FDPV). At times, it is also possible to isolate weakness in the FCU. SCT: The most common site of entrapment is in the distal cubital tunnel, at the level of Osborn's ligament and the FCU arcade. SCT should be performed at all potential sites of compression [28]. Pain: Pain site is dependent on the compression site; most commonly, pain will be noted at the FCU arcade and Osborn's ligament.

Guyon's canal – ulnar tunnel syndrome

The symptoms associated with compression in Guyon's canal vary based on the specific location [30]. Compression in zone I (Guyon's canal proper) may cause both motor and sensory deficits, while zone II (deep branch of the ulnar nerve) affects motor function only, and zone III (ulnar of zone II) leads to sensory symptoms exclusively.

Muscle testing of the ADM and first dorsal interosseous IOD1 [8]. Both muscles will be weak in zone I whereas only IOD1 in zone III compression. SCT: Staged SCT based on muscle testing, will be positive in either zone I, II or III. Pain: Also staged and should be correlated to motor and SCT findings for zones I, II, III [8].

III. Radial nerve compressions

Compression of the radial nerve can occur at multiple points, including two locations in the upper arm (The triangular Interval Syndrome and the Lateral Intermuscular Syndrome), two in the proximal forearm (Posterior Interosseous Nerve- Radial tunnel syndrome and Brachioradialis – "High" Wartenberg Syndrome), and one in the wrist ("Low" Wartenberg Syndrome) [8], [31]. Table 3 presents the clinical triad for the five different radial nerve compression syndromes [8].

Table 3: The clinical triad for the radial nerve compression syndromes [8]

Radial nerve triads	Motor Weakness	The sensory-collapse test	Pain (compression site)
Triangular Interval Syndrome (TIS)	Triceps ECRB EIP EPL	Posterior upper arm, just distal of the axilla	Just distal of the axilla and proximal of the radial (spiral) groove
Lateral Intermuscular Syndrome (LIS)	ECRB EIP EPL	Lateral aspect of the distal upper arm	9–10 cm proximal of the lateral epicondyle
Posterior Interosseous Nerve (Radial Tunnel) Syndrome	ECU (EDM)	Posterolateral aspect of proximal the forearm	Three fingerbreadths distal of the lateral epicondyle, over the arcade of Frohse
Brachioradialis Syndrome (BRS) -"High" Wartenberg	ECRB	Radiovolar aspect of the proximal forearm, just distal of the radiovolar elbow crease	Volar proximal edge of the brachioradialis
"Low" Wartenberg Syndrome	No motor weakness	9–10 cm proximal of the radial styloid, along the radial border of the forearm	Painful positive Tinel's test where the nerve exists between ECRL-BR

Abbreviations: ECRB extensor carpi radialis brevis, EIP extensor indicis proprii, EPL extensor pollicis longus, ECU extensor carpi ulnaris, EDM extensor digit minimi, BR brachioradialis

Lateral intermuscular syndrome (LIS)

After passing through the spiral groove of the humerus, the radial nerve pierces the lateral intermuscular septum approximately 9–10 cm proximal to the lateral epicondyle to exit between the brachialis and brachioradialis (BR).

Muscle testing [8]: Weakness in wrist extension, index finger (extensor indicis proprii, EIP) and thumb (extensor pollicis longus, EPL). SCT: Tested in the lateral aspect of the distal upper arm. Pain: At the level of the nerve piercing the lateral intermuscular septum, 9–10 cm proximal of the lateral epicondyle.

Radial tunnel syndrome (RTS)

The posterior interosseous nerve (PIN) is prone to compression under the proximal supinator edge (the arcade of Frohse). RTS is frequently misdiagnosed as lateral epicondylitis and commonly presents as dull, aching forearm pain in the mobile extensor wad [32].

Muscle testing: The superficial fascicles in the PIN are to the extensor carpi ulnaris (ECU), which is tested by having the patient extend the arm fully and ulnarly deviate the hand maximally while the examiner tries to forcefully radially deviate the wrist. SCT: The SCT is directed to the posterolateral aspect of the forearm, over the arcade of Frohse. Pain: Deep pressure is applied approximately three fingerbreadths distally and obliquely from the lateral epicondyle, over the arcade of Frohse [8].

Lacertus syndrome

The site of the proximal median nerve compression has been widely debated. The initially described pronator syndrome in 1951 has been recently challenged, and lacertus fibrosus has been identified as responsible for the compression of the median nerve [3], [4], [33]. The lacertus syndrome is underdiagnosed and found to be responsible for the unsatisfactory outcomes after carpal tunnel release [4].

The lacertus fibrosus (LF) is an aponeurosis originating from the medial border of the distal biceps brachialis tendon, directed medially and distally, and in direct contact with the median nerve in almost half of individuals, crossing over the common flexor muscle mass and blending with its fascia [34] (Figure 2). This entrapment caused by the LF is commonly known as lacertus syndrome (LS). The entrapment has a dynamic nature and its symptomatology often goes unnoticed [4], [35].

Some patients may have simultaneous CTS and LS, which is an example of “double-crush syndrome”. If someone is still having symptoms after carpal tunnel release, the hand should be examined for lacertus syndrome. A decrease in the power of FPL, FDP2, and FCR as well as tenderness at the medial edge of the lacertus fibrosus over the median nerve will make the diagnosis (Figure 3). Nerve conduction studies are not helpful [36].

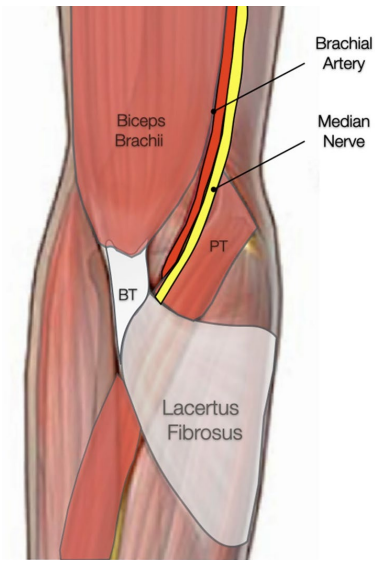

Figure 2: Anatomy of the lacertus fibrosis [34]

A minimally invasive surgical treatment under WALANT, using only local anesthesia with lidocaine–epinephrine and no tourniquet, results in direct perioperative return of strength in median innervated muscles in almost all subjects [3], [37].

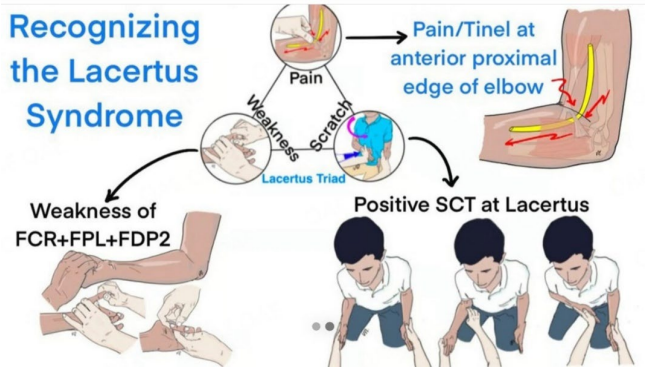

Figure 3: The clinical triad of lacertus syndrome [2]

The surgical technique proposed by Elisabeth Hagert and used in most reports of lacertus release is the following [3]: A 2–3-cm transverse incision is placed in the flexion crease of the cubital fossa, from 1 cm medial of the biceps tendon to 2 cm lateral of the medial epicondyle. Careful dissection is made subcutaneously to the pronator teres fascia, taking great care to identify and protect branches of the medial antebrachial cutaneous nerve. The pronator teres fascia is incised and followed laterally, allowing exposure of the lacertus fibrosus, which is subsequently divided. By retracting the pronator teres muscle medially, the median nerve is readily exposed. Any focal adherences to the underlying brachialis muscle may then be released. At this point, the strength of the FPL and FDP II is again tested intraoperatively before the skin is closed, as the return of muscle strength is usually immediate after the proper release of the nerve. After cauterization, the wound is closed with interrupted sutures, a small soft

dressing is applied, and immediate mobilization is encouraged. A load of 1 kg is allowed until suture removal 2 weeks postoperatively. Full load is permitted 4 weeks postoperatively. Patients with no manual labor return to work within 1–2 days postoperatively.

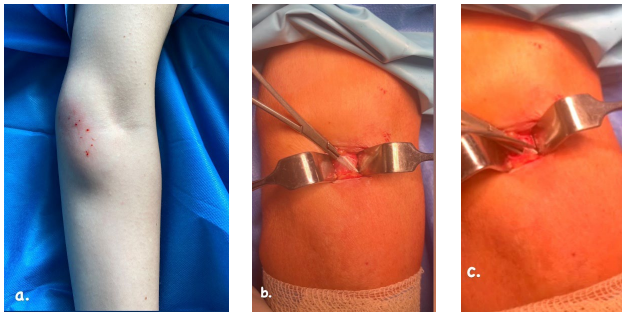

Figure 4: Lacertus tunnel syndrome surgical release through a transverse incision (left arm). a. Local tumescent anesthesia is applied with skin pallor as a vasoconstrictive effect from the adrenaline; b. Lacertus fibrosus identified and isolated (a hemostat underneath); c. Division of the lacertus fibrosus with scissors

Conclusion

Assessing individual muscle strength is a critical part of clinical neurological tests. Surgeons who treat upper extremity patients should routinely integrate into their physical examination a screening approach consisting of manual muscle testing of representative arm muscles. This approach is sensitive and permits the identification of patients who should be physically examined further to confirm the location of an upper extremity nerve entrapment. A manual assessment of the strength of individual arm muscles should be learned. It is feasible in any clinical setting as it does not require any equipment or funds.

A correct diagnosis is essential for the targeted treatment of upper extremity nerve compression syndromes. The success of the dynervology surgery for upper extremity nerve entrapments depends on the precise location of the entrapment. Including a systematic neurologic examination in the diagnostic physical approach may eventually constitute a step toward improved treatment.

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