

Review

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Painful nerve compression beyond the carpal tunnel: recognizing the lacertus syndrome

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Abstract

The most common nerve compression in the upper extremity is that of carpal tunnel syndrome. Although generally recognized and treated, as much as a 20% failure rate is reported. Recent publications are indicating that one of the sources of persistent median nerve symptoms may be missed proximal median nerve entrapments, of which the lacertus fibrosus represents a principal cause of compression, and rarely other sites such as the flexor superficialis arch or pronator teres. Compression by the lacertus fibrosus is called lacertus syndrome, and as this is a clinically diagnosed entity, only rarely confirmed using electrodiagnostic or imaging studies, it is frequently overlooked. Clinicians regularly treating patients with carpal tunnel syndrome or patients with signs of median nerve neuropathy should be aware of the lacertus fibrosus as a possible compression site. In this review, we will define lacertus syndrome, describe its clinical manifestations and diagnosis, and demonstrate surgical techniques used to treat it.

Keywords: Carpal tunnel syndrome, dynamic nerve compression, lacertus syndrome, median nerve, nerve decompression, nerve surgery



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INTRODUCTION

Carpal tunnel syndrome (CTS) is one of the most common diagnoses in hand surgery. While there is an 80%-90% success rate in carpal tunnel release surgery, there is thus an estimated 10%-20% failure rate^[1]. A failure rate of up to 20% begs the question: (1) Was the surgery performed correctly? and (2) Was the initial diagnosis correct? This review paper's objective is to educate readers about lacertus syndrome (LS), or proximal median nerve compression caused by compression of the lacertus fibrosus (LF) in the elbow, as an alternative diagnosis to CTS, especially when patients have residual symptoms post-surgical carpal tunnel release. This can help increase cognitive awareness among surgeons and minimize the unconscious misdiagnosis of CTS^[1].

Some patients may have simultaneous CTS and LS, which is an example of "double-crush syndrome" (DCS). DCS is defined by compression at two or more locations across a peripheral nerve that can coexist and mutually increase the intensity of symptoms. It was first described in 1973 by Upton and McComas^[2], who theorized that "compression at one site (at times symptomatic) predisposed a peripheral nerve to increased susceptibility to impairment at another anatomic location". This "double crush" subsequently results in the disruption of axonal transport along the nerve, increasing the fragility of distal axons to compression syndromes and symptomatology^[3].

Additionally, dissatisfaction after treatment at one site may be the result of persistent pathology at another site along the same peripheral nerve. For example, a patient presenting with a "classic" CTS may visit the physician after an open or endoscopic carpal tunnel release (CTR) with persistent symptoms of pain and numbness in the median nerve distribution. This clinical picture can point the examiner towards a more proximal nerve involvement, and its diagnosis should be highly considered using a complete and thorough clinical examination as delineated below^[1].

DEFINITION OF LACERTUS SYNDROME

Lacertus syndrome (LS) is one of the described proximal median nerve entrapments (PMNE)^[4], related to pressure of the LF, or bicipital aponeurosis^[5] on the median nerve at the elbow. The LF originates from the distal biceps brachialis tendon at its medial border and is directed medially and distally across the pronator teres just above the median nerve^[3,6] [Figure 1].

A recent cadaveric study investigating the perineural pressure of the median nerve at the level of the LF was conducted, where repetitive elbow motion was performed with a pressure sensor inserted into the flexor-pronator mass^[7]. Following isolated sectioning of the LF, over 80% pressure reduction was observed in the median nerve. The LS differs from prior descriptions of the classic pronator syndrome in that it is a dynamic nerve compression, usually a Sunderland Zero compression (as described by Peters *et al.*)^[8], and that treatment by simple sectioning of the LF results in immediate perioperative return of strength in median nerve innervated muscles, regardless of whether the median nerve is located immediately under the LF or, as rarely reported, within the pronator muscle mass^[9].

SYMPTOMS & DIAGNOSIS OF LACERTUS SYNDROME

Symptoms

In LS, pain is one of the symptoms found in approximately 35%-40% of patients^[9] and, if present, will primarily be found at the level of the median nerve under the LF or along its course in the forearm.

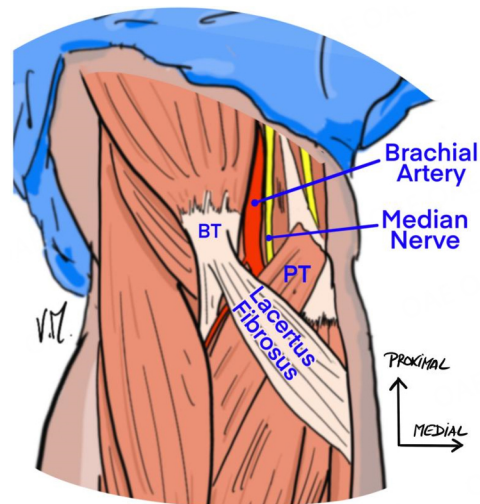


Figure 1. Illustration of the anatomy of the lacertus fibrosus in relation to the biceps tendon (BT), median nerve, brachial artery, and pronator teres (PT) in a right elbow.

The three classical symptoms in patients with isolated LS are: (1) loss of hand strength; (2) loss of hand endurance and hand fatigue; and (3) forearm pain^[9]. These symptoms can also be described as: loss of tip pinch and key strength, a general feeling of clumsiness and weakness of the hand, non-specific pain in the forearm in addition to occasional extension to the upper arm, and a transitory sensory deficit in the thenar eminence along the palmar cutaneous branch of the median nerve (PCBMn) and, at times, in the digits innervated by the median nerve^[4,10-12] [Table 1].

In patients with DCS of the median nerve at both the carpal tunnel and lacertus, in addition to the three symptoms listed above, the most common symptom is numbness of the hand along the median nerve territory, including the PCBMn.

Diagnosis

Clinical examination

The diagnosis of LS is established through a comprehensive history taking and clinical examination of the patient. Finding a complete clinical triad of muscle weakness, sensory provocative test, and pain at the level of nerve compression is essential to establish the diagnosis. The clinical triad for lacertus syndrome includes (1) weakness in the flexor pollicis longus (FPL), flexor digitorum profundus II (FDP II) and flexor carpi radialis (FCR) muscles upon manual muscle tests; (2) a positive sensory (or scratch) collapse test (SCT) at the LF; and (3) local tenderness over the median nerve at the LF^[4,11,13-15] [Figure 2].

SCT can be difficult to perform in cases with rotator cuff or shoulder pathologies. In these instances, using SCT by testing the lower extremities to look for collapse on the lower limbs in response to cutaneous stimulation at the elbow is necessary^[16]. SCT is not reliable or possible to do in the event of a neurological or psychiatric illness.

It is important to distinguish the diagnosis of LS from CTS as they may present with similar clinical manifestations. In line with the above described clinical triad for LS, a similar clinical triad can be used for isolated carpal tunnel syndrome, with: (1) weakness in the abductor pollicis brevis (APB) on muscle testing (not in FPL, FDP II, FCR)^[5], positive SCT over the median nerve at the carpal tunnel, positive Tinel's test and/or pain on compression of the median nerve at the carpal tunnel (not at the lacertus or pronator

Table 1. Symptoms present in isolated carpal tunnel syndrome, isolated lacertus syndrome, and double-crush syndrome of the median nerve

	Sensory Examination: Numbness	Motor Examination: Weakness	Pain
Carpal Tunnel	Lateral hand, digits I- radial IV (median nerve)	Thumb abduction (APB) Thumb opposition (OP)	Pain in wrist and hand
Lacertus	Thenar eminence (PCBMn)	Wrist flexion (FCR) Thumb IP flexion (FPL) Index DIP flexion (FDP2)	Pain in medial elbow and forearm
Double-crush	Thenar eminence and digits	Thumb, index flexion Thumb abduction	Pain from medial elbow to hand

PCBMn: palmar cutaneous branch of the median nerve; APB: abductor pollicis brevis; OP: opponens pollicis; FCR: flexor carpi radialis; FPL: flexor pollicis longus; FDP2: flexor digitorum profundus index

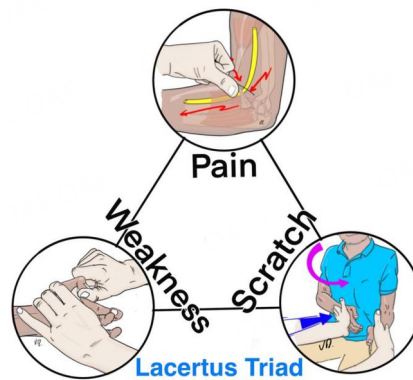


Figure 2. The clinical triad of lacertus syndrome includes weakness in FPL, FDP II, FCR; positive scratch collapse test; and pain over the median nerve at the level of the lacertus fibrosus.

region) [Table 1]. Other provocative maneuvers (Phalen's test, Durkan's test) may, of course, be used to additionally support the diagnosis of CTS.

DCS with associated LS and CTS will be found to have the following clinical triad: (1) weakness is observed in all four muscles: FCR, FPL, FDP2, and AP; (2) positive SCT at the carpal tunnel and lacertus level; (3) pain and/or positive Tinel's test over the median nerve at both the carpal tunnel and lacertus level.

Careful examination of strength helps distinguish LS from other possible sites of PMNE:

Isolated anterior interosseous nerve compression: Weakness of FPL and FDP-II but with complete strength of FCR.

Superficialis arcade: weakness of flexor digitorum superficialis (FDS) III-IV.

In line with the above description of a clinical triad for nerve compressions, SCT and pain on provocation will be found at these respective sites^[17].

Imaging

As the diagnosis of lacertus syndrome is essentially acquired upon a comprehensive patient history and clinical examination, the use of imaging modalities is not a required step to confirm the diagnosis and may be inconclusive. However, recent studies have discussed the use of ultrasound (US), magnetic resonance

imaging (MRI), and MRI neurography in the diagnosis of LS.

Ultrasound

Ultrasound is a reliable, fast, and inexpensive imaging modality that can be used in the diagnosis of CTS^[18,19], and a recent publication has shown that ultrasound findings correlate with nerve conduction studies in its diagnosis^[20]. Presently, however, there are currently no systematic methods or protocols in place for the use of ultrasound in diagnosing dynamic proximal median nerve entrapments, such as the LS; in case of static entrapment, ultrasound can help to identify a loss of nerve caliber or a change in texture under the PT or flexor superficialis arcade. It is also helpful to rule out cyst or solid tumor as a cause of compressive syndrome^[21] [Figure 3]. Considering this, future research ought to analyze and compare the normal and pathological appearance of the median nerve with respect to its structural characteristics and nerve size, from the distal upper arm to the mid-forearm^[18]. The fact that ultrasound allows for dynamic imaging means that it has the potential to be an important imaging modality to aid in the diagnosis of dynamic nerve compressions, such as the LS.

MRI

MRI, on the contrary, is a static imaging tool that is sensitive yet nonspecific for detecting peripheral nerve lesions^[4,22,23]. In recent studies, MRI has been proposed to be a potential assist in diagnosing proximal entrapments. However, the results have been shown to be normal in the event of a proximal median nerve entrapment (PMNE) unless there is clear axonal degeneration and muscle wasting of the forearm^[23].

MRI neurography

There have been a few recent studies looking into newer and advanced technology-based imaging modalities that primarily focus on the benefit of MRI neurography (MRN) in pre-operative diagnosis of upper extremity neuropathies.

A recent study showed that MRN has the potential to visualize high-resolution characteristics in peripheral nerves of the hand and wrist, where the precise lesion location and confirmation of the diagnosis of neuropathy can be depicted^[24]. It is also a good tool for better identification of physiological and internal structural changes^[25]. Nonetheless, additional research is still required to establish optimal imaging methods and sequences of MRI.

Electromyography studies

As mentioned above, LS is a dynamic nerve compression, frequently classified as a Sunderland “zero”. As a result, there will be no axonal injury but rather only changes in perineural blood flow and axonal transport^[26], making the specificity in electromyography (EMG) studies very low^[8].

Furthermore, compression at one level can cause symptoms distal to it due to changes in intraneural circulation^[27] and neuroinflammatory reactions^[28], while the pressure levels of compression are too low to cause axonal injury, thus resulting in no detectable changes in EMG^[29]. Therefore, it is not recommended to use EMG for the diagnosis of PMNE/LS, as the results are often normal or inconclusive, with the specificity shown to be as low as 30% and ranging to 70%^[30].

In CTS, EMG studies are routinely performed as a complement to the clinical examination, yet their validity is still largely unproven. Additionally, EMG studies have been shown to be poor predictors of symptom severity or functional impairment^[31], and patients with clinical evidence of CTS and negative EMG studies have been shown to have identical clinical improvement after surgery as patients with positive EMG

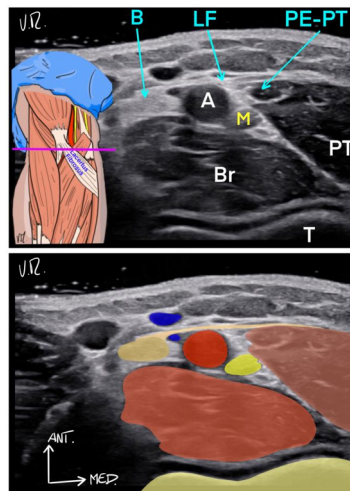


Figure 3. Transverse Ultrasonographic view of the anterior aspect of the Elbow. A: Humeral Artery; B: Biceps tendon; Br: Brachialis; LF: Lacertus Fibrosus; M: Median Nerve; PT: Pronator Teres; PE-PT: Proximal Edge of Pronator Teres; T: humeral Trochlea.

analysis^[32].

Overall, limitations in EMG studies include technical and operator-dependent errors, the inability to distinguish mixed pattern types of nerve injuries with only a portion of muscle function evaluated, and an inability to detect early nerve compression syndromes^[33]. These shortcomings have even led some authors to advise against the use of EMG in upper extremity nerve entrapments^[34], instead advocating the use of clinical examination techniques^[35].

Treatment

Conservative

Patients who have early signs of LS may benefit from a local cortisone injection at the proximal edge of the LF. Additionally, a program consisting of gliding exercises of the nerve in addition to evaluation of work ergonomics are important parts of a conservative treatment strategy in such patients^[1].

Surgical

In patients with apparent or evident weakness in the FCR-FPL-FDPII (with or without sensory symptoms), and prior failed conservative management, a surgical release of the LF is indicated. This is generally done on an outpatient basis or in-office, using wide-awake anesthesia, as it allows the surgeon to test the strength of muscles intraoperatively after the proximal median nerve release^[1].

To plan a surgical incision, the “tripod grip” is used to best locate the LF from superficial landmarks. By placing the thumb on the medial epicondyle and simultaneously the middle finger on the biceps tendon, the index finger should naturally fall between the two fingers in a tripod position, which will directly lead to the location of the LF [Figure 4A and B]. Correct centering requires that the side of the patient’s arm and the examiner’s hand are ipsilateral, meaning that the right hand of the surgeon is used to identify the LF in the right elbow of the patient (and vice versa).

The surgical technique that has been used in most published reports of lacertus release is the following^[5,9,12,36]: After the surgical incision landmark is marked, the tumescent anesthesia, 20-30 mL 1% lidocaine (10 mg/mL) is mixed with epinephrine (5 µg/mL) and buffered with 2-3 mL of sodium bicarbonate (50 mg/

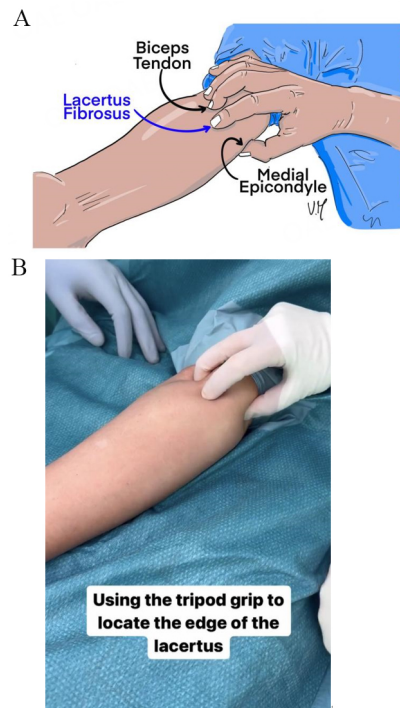


Figure 4. (A) Illustration of the tripod grip to localize the proximal edge of the lacertus fibrosus (LF) when planning surgical release. The ipsilateral hand of the surgeon should be used when doing the tripod grip to correctly center the index finger over the LF; (B) Intraoperative image of the tripod grip.

mL). Using a 27G needle, it is subcutaneously injected from the medial elbow crease and obliquely over the area of the LF, which is about 4 cm distal and central to the elbow crease. A 2-3 cm transverse skin incision is placed transversely in the volar medial elbow crease or slightly distal, to provide proper postoperative aesthetics. To recognize and secure the medial antebrachial cutaneous nerve, the dissection is carried subcutaneously with care before reaching the pronator teres (PT) fascia. The PT fascia is then incised to allow identification of the LF laterally and centrally in the wound. At this point, the proximal edge of the LF should be lifted to ensure that underlying neurovascular bundle is not injured while dividing the LF completely. Be aware of possible perforator vessels through the LF that may cause postoperative hematoma if injured. Underneath the split lacertus is where the median nerve can be identified, and on rare occasions within the muscle belly of the PT. After appropriate hemostasis, the wound is closed with intradermal 4-0 monocryl sutures, covered with surgical strips, and a small soft dressing applied. Prompt mobilization is then encouraged^[1].

Patients who do not undergo manual labor can return to work within 1 to 2 days postoperatively but are instructed to avoid heavy lifting of more than 1-2 kgs. Gradual manual work with heavier lifting is allowed after 4 weeks^[1].

Ultrasound-guided lacertus release

As an alternative to the in-situ approach described above, surgeons trained in ultrasonosurgery may prefer to perform an ultrasound-guided lacertus release (USLR)^[37]. The advantages of USLR are the following: (1) the surgeons are guided by what they see on the screen; (2) the anesthesia can be performed more precisely; (3) anatomic variations may be detected; and (4) the procedure can be performed in-office by the surgeon alone (no assistant required). The disadvantages, on the other hand, include a high threshold to

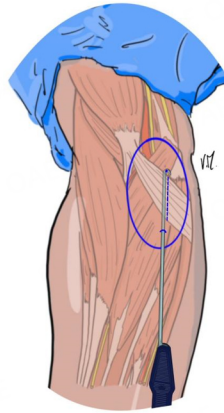


Figure 5. Illustration of the retrograde technique for ultrasound-guided lacertus release.

master the technique, and no possibility for intraoperative hemostasis leading to a higher risk of postoperative hematoma.

USLR can be performed using a retro- or antegrade technique. The retrograde technique is as follows: Following the anesthetic approach as described above, a 1-2 cm incision is made along the distal edge of the lacertus. A specific retrograde hook is inserted with US guidance. The hook is positioned over the proximal edge of the lacertus, as visualized using US, and pulled in a retrograde manner to divide the LF [Figure 5]. For the antegrade technique: Surgery is performed as with the open technique but with a smaller skin incision, allowing positioning of the US-guided knife on the proximal edge of the lacertus, which is then cut by pushing the knife distally under US guidance.

RETURN OF POWER

One advantage of undergoing a lacertus release in wide-awake anesthesia is the possibility of testing the power in the FPL, FDP II, and FCR instantly after the release is completed. If the release is adequate, the power will markedly improve (often normalized) immediately. This rapid return of power is explained by the nature of the Sunderland zero and the dynamic ischemic compression of the median nerve without complete and permanent axonal damage, allowing for immediate return of strength, as shown in a recent publication^[36]. The absence of immediate improvement may be a sign of more severe nerve compression, longstanding neuropathy, or incomplete release. If the latter is suspected, additional intraoperative release may be required (i.e., of the leading edge of the superficial flexor arcade) until recovery is noted. In addition to confirming adequate release of the nerve, the intraoperative testing of power enables the surgeons to cooperate with their patients and appreciate the success of the surgery.

CONCLUSION

The awareness of lacertus syndrome as a separate diagnostic entity or in conjunction with CTS is increasingly recognized. Clinical signs are sufficient to diagnose a patient with LS and present as a triad of the following: (1) FPL, FDP II, and FCR weakness upon manual muscle testing; (2) a positive SCT at the LF; and (3) local tenderness over the median nerve at the LF^[4,11,13-15]. Complementary imaging tools such as US, MRI, and MRN may aid in diagnoses or be used to rule out other causes of compression, while EMG has little to no diagnostic benefit. Initial conservative treatment includes nerve gliding exercises, upper arm strengthening, and ergonomic considerations. Surgical treatment is generally done as an outpatient case using local anesthesia with wide-awake patients to allow for testing of muscle strength perioperatively.

DECLARATIONS

Author's contributions

Writing the first draft of the manuscript: Al-Hashimi Y

Mentoring and guidance throughout the writing process: Hagert E

Critical review and editing of the final manuscript draft: Hagert E, Ferembach B

Original drawings: Martinel V

Read and approved the final version of the manuscript: Hagert E, Ferembach B, Al-Hashimi Y, Martinel V

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All authors declare that there are no conflicts of interest.

Ethical approval and consent to participate

Not applicable. All study participants provided informed consent.

Consent for publication

Patient written informed consent was obtained, as per institutional requirements, for the [video](#) included in the manuscript. The content of the [video](#) is entirely anonymized.

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