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REVIEW

Fascial Entrapment Neuropathy

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Entrapment neuropathies are debilitating clinical conditions, creating significant morbidity in the upper and lower extremities in terms of pain, sensory abnormalities, and motor weakness, becoming a challenge to diagnose and treat. Because entrapments can have multiple origins, a misinterpretation of anatomy during examination can lead to incorrect diagnosis and treatment. This review addresses understanding of the anatomy of fascia that can play an important role in this syndrome. There is a specific microenvironment around the nerve composed of connective tissues that include deep fascia, intermuscular septa, epineurium, and perineurium. The microenvironmental modifications can be translated into change in mobility with consequence decreasing of the independency of the nerve from the surrounding structures lading to entrapments and "internal stretch lesion." The entrapments reported in this article reinforce the importance of fascia tissue in generating common symptoms that pose more difficult diagnostic challenges and may often be confused with more common clinical conditions. Clin. Anat. 32:883–890, 2019.

Key words: nerve; nerve entrapment; fascia; Parsonage-Turner

INTRODUCTION

Entrapment neuropathies are the most prevalent type of peripheral neuropathy (Schmid et al., 2018) and can have profound physical, psychological, and economic impacts on patients (Toussaint et al., 2010). The defining criteria of an entrapment, according to Kashuk (1977), include altered transmission due to mechanical irritation related to anatomical neighbor impingement. Nerve entrapment is broadly defined as compression or entrapment of a nerve as it passes through an anatomical structure such as a fibro-osseous tunnel or fascial opening (Flanigan and DiGiovanni, 2011) or below a thickened overlying retinaculum (Toussaint et al., 2010) that represents a fascial reinforcement (Stecco et al., 2010). Other than piriform and cubital tunnel syndromes, which represent the only entrapment neuropathies attributed to muscles and are caused by anconeus and epitrochlearis (Park et al., 2018), virtually all other types are attributed to compression by fascial bands, aponeuroses, ligaments, or other rigid structures (Campbell, 1997).

Entrapment neuropathies are more frequent and overall better understood in the upper extremity (Papanicolaou et al., 2001) than the lower extremity; thus, nerve entrapments in the lower extremities are believed to be underdiagnosed and underreported (Flanigan and DiGiovanni, 2011).

Surrounding fascial structures have been increasingly highlighted recently as possible causes of nerve entrapment. The aim of this commentary review is to assess the role of fascial structures (retinaculum, collagen bundles and fascial reinforcement, aponeurosis, etc.) in entrapment neuropathy.

MATERIALS AND METHODS

This article is not intended as a comprehensive review but rather a commentary review of published

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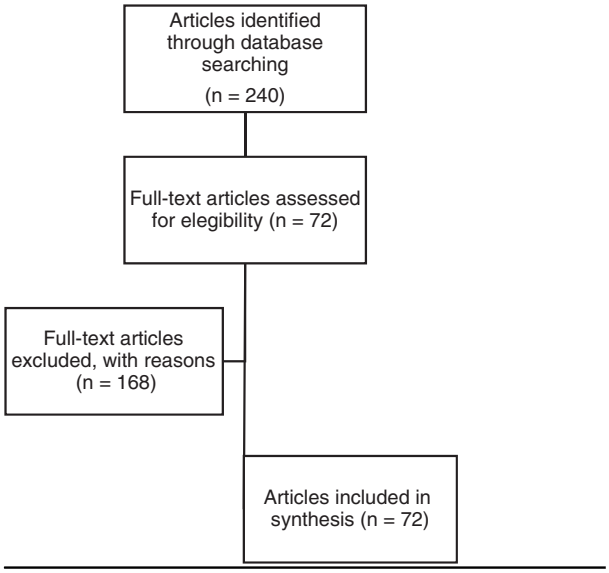
TABLE 1.



articles that include the terms "entrapment," "fascia," or "connective tissue" in the title. The PubMed database was searched for clinical studies with the following key terms: "nerve entrapments," "entrapment neuropathies," "fascia," and "connective tissue." We combined those terms between the Boolean operators "AND"/"OR." Research was limited to case reports, clinical trials, controlled clinical trials, reviews, comparative studies, multicenter studies, and randomized controlled trials in humans. The search was extended through the reference lists of the recruited texts. Relevant secondary references were also captured. English studies in which fasciae were the main causes of nerve entrapment were considered. All the others tissue entrapments were excluded from the review.

RESULTS

Using "entrapment neuropathies" as key term on PubMed recovered 21,790 articles. The addition of other search keys words or other selection criteria, as described in the research strategy, reduced this number. The PubMed search for "entrapment neuropathies and fascia" yielded 240 articles including other articles describing previous research cited in the reference lists. Finally, 72 articles dating from 1947 to 2019 were obtained (Table 1). These articles were then classed anatomically as upper or lower extremity.

Lower Extremity Entrapment Neuropathies

Ilioinguinal, iliohypogastric, and genitofemoral nerve entrapment. Entrapment of the ilioinguinal, iliohypogastric, and genitofemoral nerves can be spontaneous (e.g., resulting from variations in the musculoaponeurotic connections of the nerve) or iatrogenic (Toussaint et al., 2010), particularly following hernia repair surgery or secondary to scar formation along fascial planes of the oblique musculature (Stulz and Pfeiffer, 1982; Gaines, 1978).

Lateral femoral cutaneous nerve entrapment or meralgia paresthetica. Meralgia paresthetica is a pain disorder resulting from neuropathy of the lateral femoral cutaneous nerve (LFCN). Hanna (2017) defined a new fascial canal that completely ensheathed the nerve in the thigh in all specimens that he dissected. He described a fascial plane separating the nerve from skin and superficial fascia. It is superficial to the nerve and proximally attached to the inguinal ligament. He also identified a second fascial plane, deep to the nerve, which separates it from surrounding muscles (sartorius and tensor fasciae latae). This plane was continuous proximally with the posterior lamina of the iliac fascia and thickened toward the inguinal ligament. Hanna (2017) demonstrated that the two fascial planes along the nerve thicken to become continuous with the deep fascia of the thigh, forming a circumferential canal around the nerve. This canal extends distally until the LFCN branches and pierces its walls toward the subcutaneous tissue.

Omichi et al. (2015) also reported a case of meralgia paresthetica caused by entrapment of the LFCN at the fascia lata level.

Obturator nerve entrapment. Bradshaw et al. (1997) reported 32 cases of a previously undescribed condition in athletes, "obturator neuropathy," in which a thickened fascia overlying adductor brevis muscle resulted in nerve entrapment. The authors suggested surgical intervention through fascia lata incision, dividing it in line with the nerve, to decrease the compression of the entire thigh, but he also underlined that conservative measures should be considered.

Sciatic neuropathy. In clinical practice, extraspinal causes of sciatic nerve entrapment are sometimes overlooked. Hernando et al. highlighted the role of the fascia, describing how a compressive or bridge-type fibrous band extending from the posterior border of the greater trochanter and along the plane of the gluteus maximus toward the sciatic notch can limit anteroposterior movement of the sciatic nerve (Hernando et al., 2015).

Venna et al. (1991) described progressive sciatic palsy in a 12-year-old boy secondary to a constricting myofascial band in the lower thigh. Sayson et al. (1994) found a fascial constricting band around the sciatic nerve at the inferior border of piriformis muscle, generating hip and buttock pain in a 38-year-old woman.

Common peroneal nerve entrapment. Garland and Moorhouse (1952) suggested that a cause of significant compression of the common peroneal nerve is a fibrous tunnel of variable length made by a fascial layer lying superficial to the nerve but deep to peroneus longus muscle. It should be noted that the

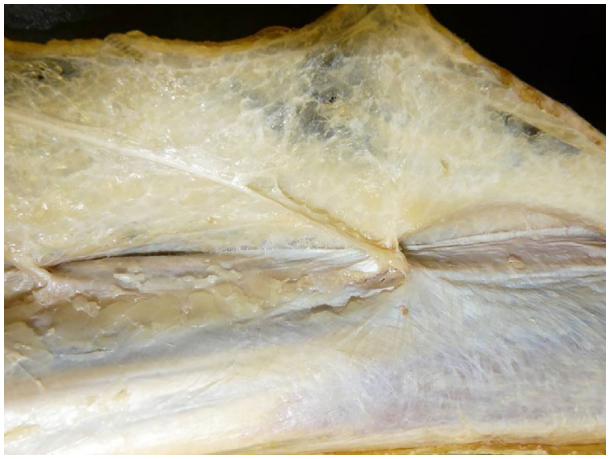


Fig. 1. Superficial peroneal nerve that pinches the anterolateral part of crural fascia (deep fascia) merging inside the superficial fascia. [Color figure can be viewed at wileyonlinelibrary.com]

narrowest region of this tunnel is where the superficial and deep fascial layers merge to form a tight band. This band is the posterior crural intermuscular septum and is the fascial layer between the lateral and posterior leg compartments (Garland and Moorhouse, 1952).

Fishman et al. (2002) also found that the entrance to the fibular tunnel was tight and restrictive in cadaveric specimens.

Superficial peroneal nerve entrapment. Entrapment of the superficial peroneal nerve is relatively rare, implicated in 3.5% of leg pain cases (Styf and Morberg, 1997). It results from mechanical compression at or near the point where the nerve leaves the lateral compartment and pierces the fascia to travel within the subcutaneous tissue (Yang et al., 2006). Kalenak (1996) demonstrated variability in the distal–proximal location at which the nerve pierces the lateral compartment fascia. Adkison et al. (1991) also found variabilities in the point at which the nerve emerges between compartments. In 14%, the nerve passes into and subsequently exits from the anterior compartment fascia; in another 12%, it bifurcates proximally, one limb exiting the lateral fascia and the other exiting the anterior fascia.

Typically, surgical intervention is described as release of the superficial peroneal nerve from the surrounding fascia at the point where it exits the crural fascia (Fig. 1).

Saphenous nerve entrapment. Saphenous nerve entrapment is a rare condition not even recognized by many authors, but Elazab (2017) emphasized that it should be considered in cases of medial knee or leg pain. The entrapment occurs at the distal narrow aperture of the adductor canal or at one of its cutaneous branches when it pierces the fascia between vastus and adductor musculature.

Superior cluneal nerve entrapment. Several authors (Kuniya et al., 2013; Matsumoto et al., 2018) have reported entrapment of the superior cluneal nerve in an osteofibrous tunnel in the space surrounded by the iliac crest and the thoracolumbar fascia as a cause of low back pain.

Ermis et al. (2011) identified an alterative reason for cluneal nerve entrapment: a peri-iliac thickened hypoechogenic nonspecific band. They found a correlation between the duration of complaints and band thickness: the longer the duration, the thicker the band (r = .361, P = .01).

Sural nerve entrapment. The superficial anatomical location of the sural nerve predisposes it to injury and entrapment, although these conditions are uncommonly reported and could be undiagnosed (Fabre et al., 2000). The sural nerve is most commonly entrapped by compression and fixation due to fascial thickening (Pecina et al., 1991). Paraskevas et al. (2014) defined a potential entrapment site for the sural nerve at the fascial opening (the emergence point of the nerve) where it passes through a fibrous arch. The same author found an anastomotic peroneal branch of the sural nerve traveling within a fibrous fascial tunnel that caused nerve flattening. Fabre et al. (2000) hypothesized that the "superficial sural aponeurosis" can be thickened and doubled, forming a fibrous tunnel that can compress the nerve and cause an undiagnosed entrapment.

Tarsal tunnel syndrome. The condition is rare but leads to a range of symptoms affecting the plantar margins of the foot (Ahmad et al., 2012). It is related to compression of the tibial nerve or its plantar divisions at the level of the tarsal tunnel as the nerves course deep to underlying fascia. The superficial margin of the tunnel is marked by the flexor retinaculum (a fascial thickening) and the distal end point is narrow and merges with both the superficial and deep fasciae of abductor hallucis muscle (Harries et al., 2000; Kohno et al., 2000). Costales et al. (2018) affirmed that during surgery, it is common to encounter local nerve swelling, a narrow tarsal tunnel, fibrous adhesions around the nerve, and constricting bands.

Common digital plantar nerve entrapment. Stecco et al. (2015) hypothesized that alterations in foot support and altered biomechanics acting on the interosseous muscles can increase the stiffness of the dorsal fascia, particularly at the points where those muscles are inserted. Chronic rigidity of this fascia can result from repetitive microtrauma and can increase the stiffness of the intermetatarsal space and lead to entrapment of the common digital plantar nerve.

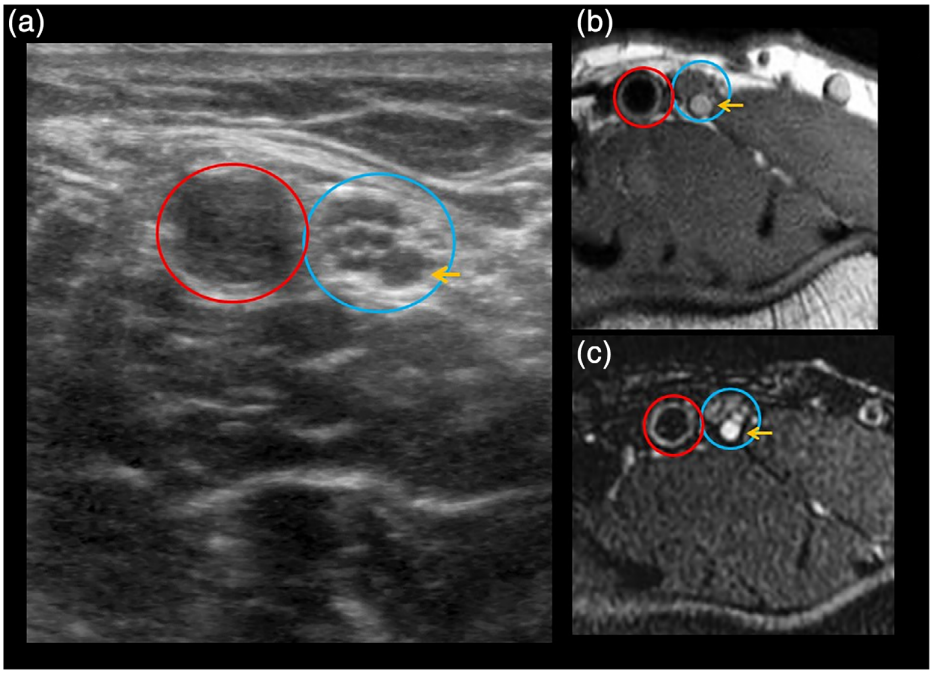


Fig. 2. Parsonage-Turner syndrome: Grayscale ultrasound (a) and axial proton density (b) and T2-weighted Dixon fat suppression (c) magnetic resonance images demonstrate focal enlargement and decreased echogenicity/signal hyperintensity of the posteriorly positioned fascicular bundle (yellow arrow) of the median nerve (blue oval) (corresponding to the anterior interosseous nerve) immediately above the level of the medial epicondyle. Note that adjacent brachial artery (red oval). [Color figure can be viewed at wileyonlinelibrary.com]

Upper Extremity Entrapment Neuropathies

Thoracic outlet syndrome. Millesi and Schmidhammer (2006) showed that passive motion of the brachial plexus in relation to movement of the upper extremity is provided by gliding tissue. Pathological changes in the gliding tissue could contribute to the development of thoracic outlet syndrome. These authors also stated that recurrent fibrosis develops in rare cases. Chavhan et al. (2017) affirmed that the neurovascular bundle can also be compressed by soft-tissue abnormalities such as a fibrous band.

Intrinsic constriction of suprascapular nerve. - In pathological and post-trauma conditions, the fascia can be retracted or thickened, and the suprascapular nerve can become entrapped along its course in the supraspinatus fossa, between the suprascapular notch and in the spinoglenoid notch (Duparc et al., 2010). Also, Jelev and Surchev (2007) suggested that certain fibrous and muscular structures could be an anatomical basis for supraclavicular nerve entrapment syndrome. These structures could include a thickened stiff fascia generating chronic compression as a tight band. Kara et al. (2010) suggested a possible overlap between chronic compression and Parsonage-Turner syndrome (idiopathic brachial plexus neuropathy) (Fig. 2). The greatest mechanical deformation occurs both in the superficial region and between compressed and uncompressed areas of the nerve (Gelberman et al., 1993). It is also hypothesized that multiple entrapments along the same nerve impede proper epineurial gliding, generating interfascicular nerve friction and/or ischemia. Torsion of the nerve results between the entrapped sites, as a rope that is turned in opposite directions at its ends develops a twist in the middle.

Radial nerve entrapment. There are several potential sites of compression along the path of the radial nerve where many connective tissue adhesions can develop (Clavert et al., 2009). These include the intermuscular septum between the triceps and brachialis muscles, where the nerve leaves the spiral groove (radial nerve), and at the ligament of Frohse (posterior interosseous or deep radial nerve). Congenital constriction bands in the upper arm have been implicated in combined median, ulnar, and radial neuropathies (Weeks, 1982). Ouvrier and Shield (1999) said that the posterior interosseous branch of the radial nerve can be entrapped and compressed at the elbow level or from a fibrous bundle.

Wartenberg's syndrome/cheiralgia paresthetica (superficial radial nerve entrapment). Patel et al. (2014) asserted that owing to its anatomical location, the superficial branch of the radial nerve is vulnerable to compression from trauma, masses, and constriction from the brachial fascia. Also, Kim et al. (2014) attested that the most critical region for generating entrapment of the superficial radial nerve is where it pierces the brachioradialis fascia horizontally.

Median nerve entrapment. There are many potential entrapment sites of the median nerve in the upper extremity, which include the biceps brachii aponeurosis (lacertus fibrosis) (Hobson-Webb and Juel, 2017), and Struthers' ligament (a band of connective tissue) (Bilge et al., 1990), which is present in 1–13% of the general population (Dang and Rodner, 2009; Siqueira and Martins, 2005). Costales et al. (2018) noticed during surgery that abnormal fibrous bands, adhesions, or neuromas could be found at sites of medial nerve entrapment.

Ettema et al. (2006) hypothesized that idiopathic carpal tunnel syndrome is the result of shear injury to the subsynovial connective tissue, causing fibrosis that ultimately leads to nerve compression and ischemia. Jones et al. (2012) found during revision surgery of carpal tunnel syndrome that incomplete release of either the distal transverse carpal ligament or proximal antebrachial fascia is the most common intraoperative finding (Level of Evidence: Therapeutic, IV). Piyawinijwong et al. (2011) reported two anatomical cases of median nerve entrapment, one potential cause of carpal tunnel syndrome. The first case was thickening of the brachial fascia, which resembles the Struthers' ligament. The second was thickening of the bicipital aponeurosis. Numerous case reports in the literature also describe a compressive cause of anterior interosseous syndrome, but care should be taken in attributing anterior interosseous neuropathy to compression, particularly in cases with an abrupt onset.

Ulnar nerve entrapment. The ulnar nerve passes through a fibrous tunnel (cubital tunnel) within the medial intermuscular septum into the posterior compartment of the upper arm in more complicated patterns than those described in anatomy textbooks. These unreported patterns could be related to idiopathic ulnar nerve entrapment at the mid-arm (Won et al., 2011). Also, Choi et al. (2018) documented scant attention to the deep fascial plane, along which the ulnar nerve travels in the forearm.

The medial collateral ligament of the elbow defines the floor of the cubital tunnel, whereas the roof consists of the thickened fascia known as the Osborne (arcuate) ligament. This ligament lies between the medial and ulnar heads of flexor carpi ulnaris (Hobson-Webb and Juel, 2017).

Costales et al. (2018) said that nerve also becomes entrapped by repetitive trauma and scar formation in the cubital tunnel.

Many authors have identified and released fascial bands compressing the ulnar nerve during endoscopic cubital tunnel release (Amadio and Beckenbaugh, 1986; Hoffmann and Siemionow, 2006).

Cadaveric dissections by Siemionow et al. (2007) revealed fascial "bands" or segmental fascial thickenings within flexor carpi ulnaris of the proximal forearm, capable of compressing the ulnar nerve.

Liu et al. (2015) proved through short-segment nerve conduction studies that the point where the ulnar nerve is most vulnerable to compression is 2 cm proximal the medial epicondyle.

Limitations

The aim of this commentary review is to present the role of fasciae in the etiology of nerve entrapments, without any intention to be systematic. The authors decided to include only entrapments caused by fascia, aponeuroses, and connective tissue bundles, excluding bones, muscles, and other anatomical structures.

DISCUSSION

Our research has underlined a finding common to many authors: fascial tissue is relevant to the etiology of entrapment neuropathy. With different prevalences and definitions, most of the articles emphasize the importance of general connective tissue (other than ligaments) in peripheral nerve entrapments. Acute or chronic compression-related injury can occur in almost any peripheral nerve, but considerations such as anatomical course, superficial location, or adjacent fibrous or osseous structures can predispose certain nerves to intrinsic or extrinsic compression-related injury (Arnold and Elsheikh, 2013). Each sensory nerve has to cross the deep fascia in order to reach the skin, so entrapment at the fascial opening site is possible. Even more clinically relevant are the tunnels in the intermuscular septa where sensory and motor fibers can be entrapped.

Dahlin et al. (1989) induced higher pressures (130–150 mmHg) experimentally in the epineurium, resulting in acute conduction block and changes in nerve fiber morphology, causing both epineural and perineural thickening and rapid loss of nerve function (Braidwood, 1975). It has been suggested that entrapment neuropathies result from compression and secondary intraneural microenvironmental changes such as ischemia, edema, and scarring (Lundborg and Dahlin, 1996), but a few have thought that this microenvironment also includes fascial tissue (epineurium, perineurium, and endoneurium).

Peripheral nerve mobility is characterized by sliding along the longitudinal nerve axes. It involves stretching and shortening of the fibers and sliding of the nerve bundles along the nerve beds and within the epineurium and perineurium (Millesi et al., 1990). Longitudinal sliding reduces the local stretching that would otherwise occur during limb movement. Excursion of peripheral nerve trunks thus appears to involve a complex array of intra- and extra-neural gliding surfaces (Abe et al., 2005). The longitudinal

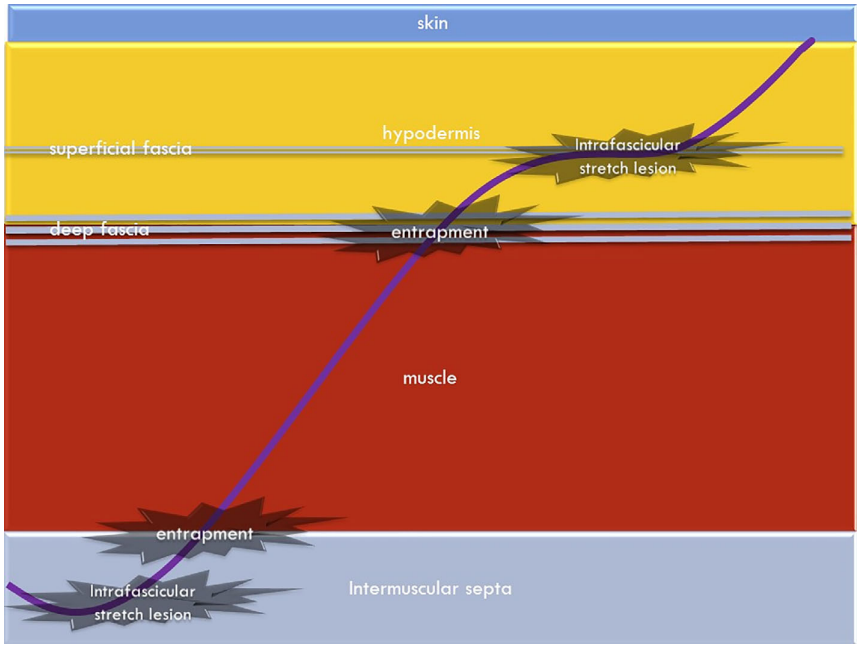


Fig. 3. Locations of nerve entrapments and decrease of intra-fascicular sliding. [Color figure can be viewed at wileyonlinelibrary.com]

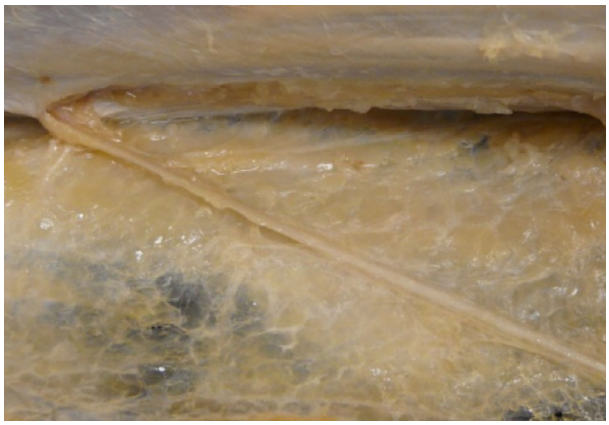


Fig. 4. Sensory nerve inside a split of superficial fascia. [Color figure can be viewed at wileyonlinelibrary.com]

movement of the nerve is closely associated with the presence of loose connective tissue and its components (glycosaminoglycans, hyaluronan, adipose cells).

The management of peripheral nerve entrapment syndromes depends on multiple factors including the chronicity and severity of symptoms, the underlying mechanism, and associated predisposing factors. Understanding the underlying mechanism of injury and the associated natural history is fundamental to designing an appropriate treatment strategy (Arnold and Elsheikh, 2013). It has to be considered that entrapment can also occur inside the hypodermis when nerves are located within a superficial fascia split (Fig. 4).

Sensory nerves must cross intermuscular septa and deep and superficial fasciae, exposing them, according to anatomical topography, to possible entrapments (Fig. 3). Mechanical compression arises where a nerve pierces the fascia and an increased fascial thickness can exacerbate it. We hypothesize that, in idiopathic nerve entrapment, repetitive microtraumas, and/or overuse can transform the extracellular matrix from sol to gel in multiple regions within the deep fasciae (Stecco et al., 2013). Larger nerve fibers are often surrounded by different layers of loose connective tissue that protect the nerve from the traction to which the fascia is subjected (Macchi et al., 2007). An increase in extracellular matrix viscosity would reduce fascial adaptability and gliding, resulting in abnormal stress on the nerve when it passes through intramuscular septa or deep fascia. The same consequences can ensue when sensory nerves travel within superficial fasciae (Fig. 4). An increased extracellular matrix viscosity will affect the telescopic structure of the epineurium (Lundborg and Dahlin, 1996; Lundborg and Rydevik, 1973), which in turn will impair intra-fascicular gliding. Loss of intra-fascicular gliding has been shown to create an internal stretch lesion (Abe et al., 2005; Lundborg and Dahlin, 1996) that could alter both afferent and efferent conduction. In the light of this study, fascial origins for nerve entrapments have to be considered during differential diagnosis.

CONCLUSION

Entrapment mononeuropathies represent a common reason for visiting primary care and outpatient neurology practices (Hobson-Webb and Juel, 2017). Accurate diagnosis is paramount because these presentations can be very similar to radiculopathy or systemic neuropathy. Effective management depends very much on proper identification of the involved nerve and the anatomical location of compression (Flanigan and DiGiovanni, 2011). Entrapment neuropathies are not frequent in children, but clinicians treating pediatric age patients must always keep them in mind (Costales et al., 2018).

Numerous experimental and clinical studies (Allison et al., 2002; Bove et al., 2003; Dilley et al., 2005; Van Der Heide et al., 2006) have demonstrated that nerve mechanosensitivity can be heightened in the absence of nerve injury and therefore of neuropathic pain. In such cases, the underlying pain is classed as nociceptive (Marchettini et al., 2006), which could be initiated by activation of nociceptors within the connective tissue surrounding peripheral nerves (nerve nervorum) (Schmid et al., 2013). All the nerves in their course have direct contact with fasciae and cross them. Until now, the role of the fasciae in etiology has been underestimated. Future research should continue to investigate the complex interdependence between fascial structures and nerve entrapments.

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CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

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