Current Concepts Review

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Current concepts in diagnosis and management of common upper limb nerve injuries in children

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Abstract

Peripheral nerve injuries (PNI) of the upper limb are a common event in the paediatric population, following both fractures and soft tissues injuries. Open injuries should in theory be easier to identify and the repair of injured structures performed as soon as possible in order to obtain a satisfying outcome. Conversely, due to the reduced compliance of younger children during clinical assessment, the diagnosis of a closed nerve injury may sometimes be delayed. As the compliance of patients is influenced by pain, anxiety and stress, the execution of the clinical manoeuvres intended to identify a loss of motor function or sensibility, can be impaired. Although the majority of PNI are neuroapraxias resulting in spontaneous recovery, there are open questions regarding certain aspects of closed PNI, e.g. when to ask for electrophysiological exams, when and how long to wait for a spontaneous recovery and when a surgical approach becomes mandatory. The aim of the article is therefore to analyse the main aspects of the different closed PNI of the upper limb in order to provide recommendations for timely and correct management, and to determine differences in the PNI treatment between children and adults.

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Introduction

Peripheral nerve injuries (PNIs) in the upper limbs are common in the paediatric population, occurring after fractures and soft-tissue injuries.[1] In theory, open injuries are easier to identify than closed injuries, and injured structures should be repaired as soon as possible to obtain better outcomes. However, owing to reduced compliance of younger children during clinical assessments (influenced by younger age, pain and anxiety), the diagnosis of a closed nerve injury may sometimes be delayed. Nerve injuries have been classified into neurapraxia, axonotmesis and neurotmesis, depending on the degree of nerve damage (Table 1).

Although the majority of PNIs are neurapraxias that undergo spontaneous recovery, open questions exist about certain aspects of closed PNIs, such as when electrophysiological examinations are necessary, when and how long to wait for spontaneous recovery and when a surgical approach becomes mandatory. Therefore, the aim of this study was to analyze the main aspects of different closed PNIs in the upper limbs to provide recommendations for timely and correct management, and to determine the differences in the treatment of PNIs between children and adults.

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Localization of PNIs in the upper limbs

Open PNIs frequently occur after soft-tissue injuries in the forearm, wrist, hand and fingers. Both the median and ulnar nerves may be involved, especially in the wrist region, whereas the digital nerves are affected in hand and finger injuries. With respect to closed lesions, the majority of PNIs occur around the elbow joints,[2] most commonly after supracondylar humeral fractures, followed by humeral shaft and forearm fractures. In general, the median and radial nerves are the most commonly involved nerves, whereas the ulnar nerve is less often affected; however, the ulnar nerve can be considered the main nerve potentially involved in iatrogenic lesions, especially after medial-entry (cross-)pinning of supracondylar humeral fractures.

Median nerve injuries

Apart from lacerations caused by sharp objects such as utility knives, broken glass, scissors and household machines, most median nerve injuries occur in relation to fractures at the elbow and forearm level. The mechanism of injury is either traction, compression or nerve laceration during the primary trauma, or contusion, incarceration, laceration, or transection during fracture reduction and pinning.

Supracondylar fractures are known to be associated with a high prevalence of neurological deficits occurring with nerve injuries (10% to 20% of all cases).[3,4] Recent studies have shown that the median nerve is the most frequently damaged nerve in extension-type supracondylar fractures (Fig. 1). Approximately 60% to 70% of median nerve injuries are isolated injuries to the anterior interosseus nerve (AIN).[5] As the AIN arises distal to the elbow joint, most authors suggested compression of the posterior fibres of the median nerve at the fracture site as the triggering event for an isolated AIN injury.[6]

In a recent anatomical study, Vincelet et al[7] demonstrated that a single event is an unlikely cause. They proposed that a combination of different events (compression of the median nerve at different sites of the elbow followed by traction of the AIN) is a more plausible explanation for this injury pattern. Iatrogenic nerve injuries occurring during the treatment of supracondylar fractures are reported to have an incidence of approximately 3.5%.[8] These injuries are caused either during fracture reduction or during percutaneous pinning.

The predictive factors for an increased risk of neurovascular compromise are ecchymosis and skin puckering on the volar aspect of the elbow.[9,10] These signs of considerable soft-tissue injury usually appear after the displacement of the proximal fracture fragment through the brachialis muscle. The median nerve is not only close to the

Table 1 Different grades of nerve degeneration according to Seddon and Sunderland classification

Nerve degeneration	Seddon classification	Sunderland classification
Local myelinic damage without any other injuries	Neurapraxia	First degree
Axonal damage without discontinuity	Axonotmesis	Second degree
Axonal and endoneurium discontinuity; perineurium and epineurium intact	Axonotmesis	Second degree
Loss of continuity of axons, endoneurium and perineurium: epineurium intact	Axonotmesis	Second degree
Complete disruption of the entire nerve trunk	Neurotmesis	Third Degree

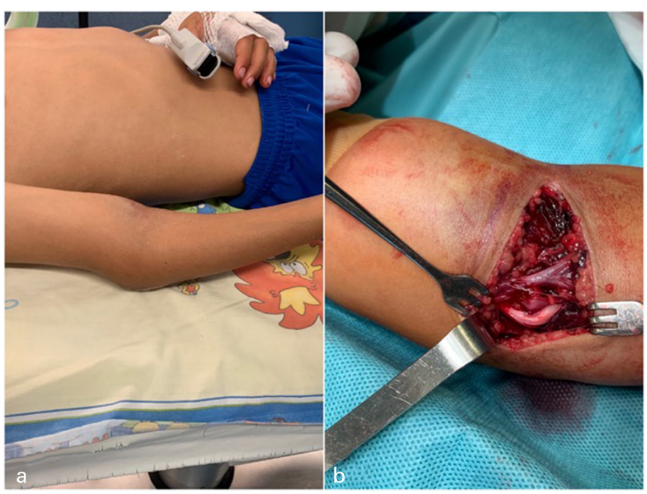

Fig. 1 Clinical features of a Gartland III supracondylar humeral fracture (a) with median nerve (b) entrapment in the fracture site.

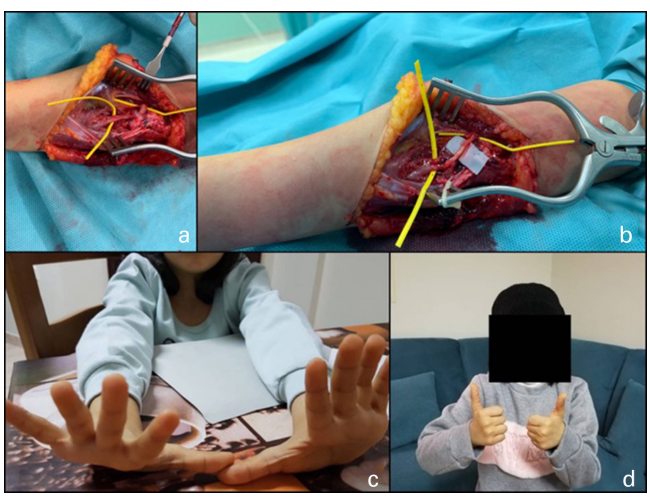

Fig. 2 Radial nerve lesion after Monteggia injury (a, b); motor recovery of the extensor communis digitorum and extensor longus pollicis after nerve repair (c, d).

proximal bone fragment but can also be tethered around the protruding bone spiculae, and thus can be incarcerated at the fracture site during reduction. A non-reducible fracture gap and the disappearance of the radial pulse are suggestive of incarceration of neurovascular structures.

Incarcerations of the median nerve have also been described in other elbow injuries. McCarthy et al[11] reported on entrapment of the median nerve in a medial condyle fracture, and several authors have published cases of intra-articular median nerve entrapment after elbow dislocation.

In the forearm, closed injuries to the median nerve tend to occur in association with radial fractures and closed injuries to the ulnar nerve occur with injuries to the ulna.

Radial nerve injuries

The radial nerve is commonly involved in closed injuries (Fig. 2). The main cause is traumatic, accounting for more than half of all cases. The most common site of injury in the paediatric population is the distal main radial trunk (56%), followed by the posterior interosseous nerve (PIN; 31%), in contrast to adults whose lesions tend to involve the main trunk at the spiral groove of the humerus.[12]

Humeral supracondylar fractures are more frequently associated with radial nerve injuries than humeral shaft fractures. Nerve injuries primarily occur because of tenting or entrapment of the nerve in the proximal fragment, whereas iatrogenic lesions occur either during closed manipulation, percutaneous fixation of fracture fragments or occasionally during open procedures.[13,14]

The frequency rate of radial nerve injuries in supracondylar humeral fractures has been reported to be between 5% and 29% of all cases, and extension fractures with posteromedial dislocation of the distal fragment are most commonly associated with radial nerve palsy.

With respect to the shaft of the humerus, the association between fractures and nerve injuries is not as clearly defined in children as in adults, and only a few studies have described the incidence and management. The risk of radial nerve injury seems mostly correlated with Holstein-Lewis fractures, although it is also associated with other humeral shaft fractures.[15] In addition, Runner et al[16] reported a case in which delayed radial nerve palsy occurred four days after the reduction of a humeral shaft fracture treated with a cast.

Forearm injuries frequently involve the PIN, which is vulnerable at several locations. Both acute and delayed PIN palsy are typically reported in the literature as complications of acute or chronic Monteggia lesions. Hirachi et al described a group of 17 irreducible radial head dislocations in Monteggia lesions, eight of which were associated with PIN palsy. Moreover, according to Wang et al,[17] type III Monteggia fracture dislocations are often complicated by PIN injury. In addition, in fractures that simultaneously involve the shafts of the radius and ulna, displacement of the radius may cause nerve stretching with a distraction force. Alternatively, the stump of the fractured radius may directly damage the nerve. Finally, Ruchelsman et al described a rare case of PIN palsy as a complication of recurrent radial head dislocation.[18,19]

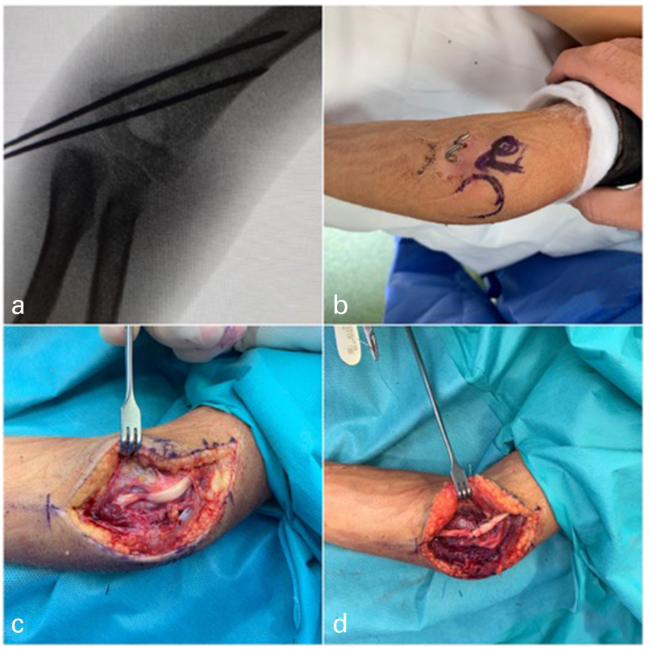

Fig. 3 Medial pinning for supracondylar humeral fractures (a, b) with ulnar nerve injuries (c, d).

Ulnar nerve injuries

A closed injury to the ulnar nerve is less common than closed injuries involving the radial and median nerves. In the elbow region, the nerve is potentially damaged after a flexion-type supracondylar humeral fracture, although this pattern accounts for only 2% of supracondylar injuries. However, the peculiar anatomy of the ulnar nerve around the elbow increases its risk for injury during or after a medial pinning, especially when swelling of the elbow hinders the easy identification of the correct pin insertion on the medial epicondyle (Fig. 3).[20,21]

The cubital tunnel, which holds the ulnar nerve, extends between the medial epicondyle and the olecranon. The nerve enters the tunnel via the medial intermuscular septum and the arcade of Struthers, and leaves it distally between the humeral and ulnar heads of the flexor carpi ulnaris. The roof of the tunnel is formed by the arcuate ligament of Osborne. Therefore, the anatomy of the cubital tunnel and the nerve hypermobility explain the increased vulnerability of the ulnar nerve during a medial pinning performed with the elbow in hyperflexion. The nerve can be transfixed or compressed by the Kirschner wire, leading to immediate or delayed palsy.[22] Some authors believe that the risk of ulnar nerve injuries may be reduced by

using prone positioning, neurostimulation, or ultrasound examination during medial pin insertion.[23,24]

Furthermore, late palsy of the ulnar nerve can occur owing to progressive compression after malunion of distal humeral fractures. The nerve can be progressively compressed after cubitus varus deformity following a supracondylar fracture of the humerus. The cause of compression in this case is kinking of the dislocated nerve at the proximal border of the flexor carpi ulnaris owing to the restriction of the cubital tunnel and the medialization of the medial head of the triceps.[25]

Another potential cause of compression is nonunion of a medial epicondyle fracture, which, after distal bone migration due to the traction of the epitrochlear muscles, leads to a secondary restriction of the cubital tunnel.

Clinical assessment and diagnostic examinations: when are they necessary?

Because all upper-limb fractures can be complicated by a nerve injury, an accurate physical examination of the motor and sensory functions must be performed both before and after treatment. However, complete neurological assessment of a child can be challenging because of pain and anxiety after trauma. Several attempts should be made to assess whether a nerve is injured or not.

The Tinel sign test and two-point discrimination (TPD) are the most common tests used to evaluate the recovery of a nerve injury. However, especially in the pre-school age, patient compliance is usually poor, making the execution of the test manoeuvres difficult to assess. Moreover, although motor recovery is relatively easy to evaluate, the same is not true for sensibility. The skin wrinkle test and an anhidrosis test should be considered because they can help surgeons determine whether sensory recovery is proceeding or not (Fig. 4).[26]

Given the difficulty in assessing discriminative sensibility, some authors exclusively focused on motor recovery, considering that functionality is more important.

Once the nerve injury has been confirmed, a 'wait-and-see' approach should be employed, considering that up to 92% of neural deficits in children are transitory.[27] The same principle should be adopted when determining the need for electrophysiological examinations.

Although neurophysiological studies can guide interventions for nerve lesions in adults, there is a paucity of studies on the healing process of nerve lesions after fractures in children. As a result, there are no reliable cut-off values for interventions. However, in the absence of motor and sensory function after two to three months, neurophysiological testing and/or thorough ultrasound visualization of the nerve may be recommended. Because there is no evidence supporting the usefulness of electrophysiological examinations in the first month after trauma due to Wallerian degeneration, the habit of requesting for such tests should be abandoned.[28,29]

Fig. 4 Skin wrinkle test; the right side had a median injury secondary to a supracondylar fracture.

If electromyography shows no signal, an ultrasound examination could reveal if the nerve is severed, tethered or caught in the fracture. The use of ultrasound in the evaluation of nerve injuries has increased in the last few decades because of its non-invasive nature, ease of use and ability to aid in the identification of patients eligible for surgery.[30,31] The advantages of ultrasound are that it provides information on the course of the nerve, its internal structure and its relationship to the surrounding structures (especially the fracture site and bone callus).

MRI is less frequently used for the evaluation of PNIs because ultrasound has a higher resolution, thus providing more information that can aid in deciding the correct management.

Nerve recovery in children and differences from adults

Children with nerve injuries have biological advantages over adults with the same injuries. One noted that in

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median and ulnar nerve repairs, the recovery of the TPD value (in mm) was directly related to the age of the patients (up to age 20 years).[32] Young et al[33] observed useful TPD in 80% of patients younger than 20 years who underwent digital nerve repairs. The excellent function observed in children has been attributed to factors such as a shorter distance from the axon repair site to the target muscle, nerve regeneration potential and superior adaptive capacity of the central nervous system (neural plasticity).

Neuroplasticity refers to a range of neural responses, from the cellular and molecular mechanisms of synapse formation to cellular re-alignment or organization of neural networks, as well as learning, memory, or behaviour. After birth, the human brain is believed to become highly dependent on and to be modified and shaped by experience. The brain undergoes maturation until at least 20 years after birth. The plasticity of the brain is believed to be maximal during critical periods, defined as maturational time periods during which some crucial experience will have its peak effect on development or learning, resulting in normal behaviour attuned to the particular environment to which the organism has been exposed. Neuroplasticity refers to the ability of neurons to change, proliferate and synapse during the developmental stage.[34] In adults, nerve regeneration proceeds at a rate of approximately 1 mm/day. Interestingly, despite the availability of numerous articles on various aspects of peripheral nerve recovery in children, there seems to be a paucity of studies on the extent to which the rate of nerve regeneration in children is higher than in adults.

Neural plasticity is considered to be the most crucial determinant of recovery, as it is believed to allow better incorporation of aberrant sensor inputs. It has also been demonstrated that the patient's age at the time of injury is an important prognostic factor. Patients who sustained injuries during early childhood were observed to have a lower risk of developing chronic pain than patients injured during adolescence.[35,36]

Treatment: timing and therapeutic options

Considering the higher neuroplasticity in children, there is a clear potential for spontaneous recovery that is higher than in adults, even in proximal injuries. The wait-and-see approach should be the main path in the management of PNIs in children.

Currently, the general consensus is that six months after a closed injury should be considered the limit for spontaneous recovery. However, the concept of nerve regeneration is related to the distance of the repair site to the affected the muscle: the longer the distance, the longer the expected recovery time. Thus, the six-month cut-off may be arbitrary and the wait time should be determined on an anatomical case-by-case basis.

Surgery is indicated for closed nerve injuries in the absence of motor recovery from six to 12 months after the injury.[37,38]

However, it is known that some children develop a dissociated or delayed return of motor or sensory functions. This raises the question of whether additional waiting time should be considered before recommending surgical revision and repair. The literature does not help in deciding the better option for this group of patients. Physicians should consider the progress of recovery of each individual patient before deciding whether surgical repair is necessary or not.

On the one hand, some children show incomplete recovery signs six months after trauma; however, these patients may not be considered for surgery because they are still expected to achieve full recovery. On the other hand, some patients who do not show any recovery signs need to undergo surgical exploration and nerve repair.

To date, several treatment options are available for paediatric traumatic PNIs, including direct repair, nerve grafting, nerve transfers, nerve conduits and tendon transfers.

The time after injury is the main factor to consider when choosing the most adequate treatment modality. Beyond 12 months after injury, tendon transfers are indicated to restore active movement. However, some authors consider the presence of muscle fibrillations in electromyographic studies as an indication for nerve repair even in injuries older than 12 months.[39] Moreover, there is no scientific evidence suggesting that neurolysis (perineural fibrous scar excision) improves nerve function, and postoperative improvements are probably attributable to the natural history of the injury itself.[40] In contrast, open injuries warrant prompt nerve exploration and repair.

Direct nerve repair is indicated in acute or subacute nerve lacerations; however, it is rarely performed for old lesions. Nerve coaptation is usually performed with epineural sutures, avoiding tension on the repair site. Another option is epi-perineural suturing, which is technically challenging but ensures a more anatomical reconstruction. However, in children, no clinical differences have been reported between epineural repair and the more time-consuming and complex epi-perineural repair. Nerve repair with fibrin glue might provide similar or better results than sutures but should be performed only if there is no tension in the coaptation site, which is unusual in nerve lacerations.

Grafting continues to be the benchmark for filling a nerve gap, especially in mixed nerves. The sural nerve is the usual source for grafting, although other sensory nerves obtained from the upper limbs, such as the medial

and lateral antibrachial cutaneous nerves, may also be used.[41] No comparative studies on donor-site morbidity and outcomes after the use of these different options are available. No solid scientific evidence exists on the use of acellular processed nerve allografts or nerve conduits in mixed nerves, and these options should not be used in children until more evidence is available. Nerve conduits, however, represent a valid alternative to grafts for digital nerves, in cases with a gap of < 2 cm to 3 cm.[42]

Moreover, conduits may be considered a spare material in cases of multiple injuries for which there are insufficient donor nerves for grafting.[43]

Nerve transfers for median, radial and ulnar nerve palsies are indicated in adults, preferentially combined with nerve grafting of the main trunk.[44] Conversely, nerve transfers are not systematically indicated in children owing to their better motor recovery. An exception might be very late cases (approximately 12 months), as recently reported for obstetrical brachial plexus palsy with a late presentation.

Tendon transfers are indicated in late presentations (> 12 months). Tendon selection should follow Brand's classical principles,[45] the most important of which are expendability, synergism for easier postoperative re-education and adequate donor muscle strength and excursion.

Conclusions

Although PNIs in the upper limbs are a common complication of paediatric trauma, the majority of patients are expected to spontaneously recover. The approach to paediatric upper-limb PNIs should differ from that in adults. The wait-and-see approach should be extended to at least two months after trauma before requiring electrophysiological studies, and every request for such examination in the first weeks after trauma should be rejected. Most expert nerve surgeons, however, make decisions based on clinical examinations alone rather than electrophysiological studies. Reconstructive surgery should be considered not earlier than six months after the trauma, and only for cases that show no signs of motor or sensory recovery. However, cases in which ultrasound shows nerve entrapment at the fracture site may be exceptions. Meticulous nerve exploration and repair could be considered in these cases in the absence of signs of recovery. Figure 5 proposes an algorithm for diagnosis and treatment based on the experience of both authors and the literature recommendations. Nerve grafting remains the benchmark for a gap in mixed nerves, whereas tendon transfers are indicated in late presentations whenever the remaining potential for further nerve recovery is insufficient.

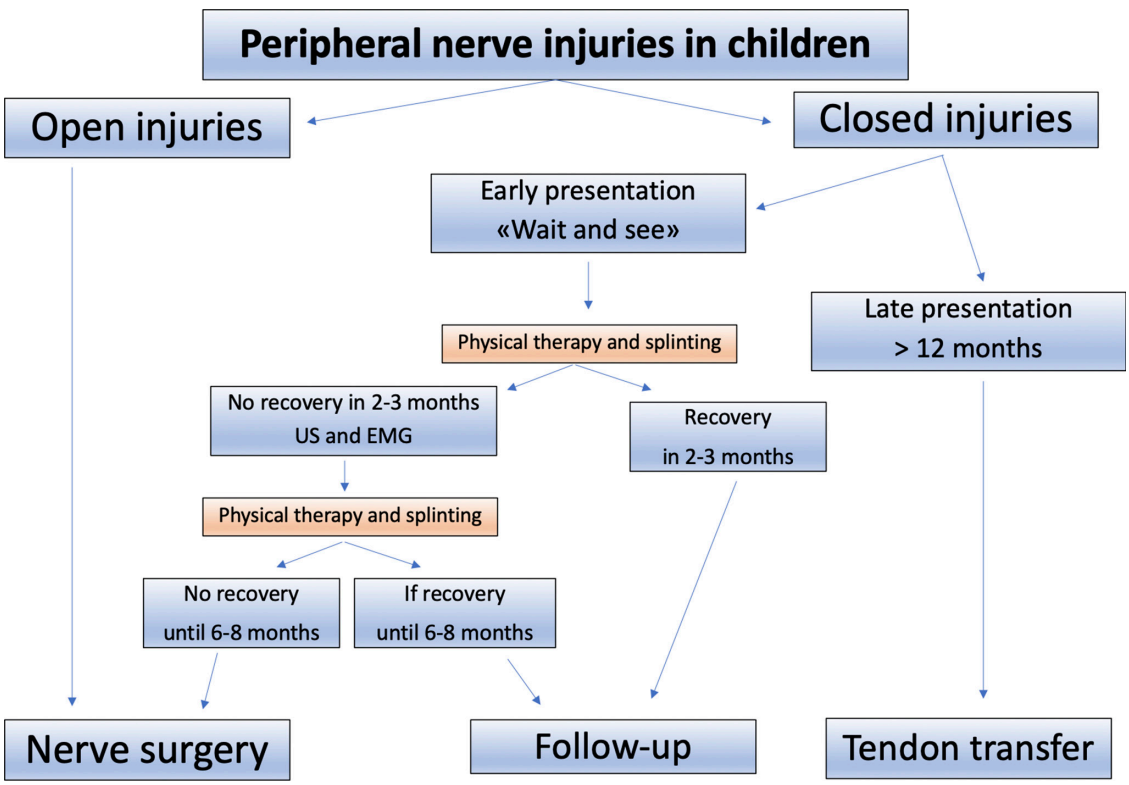

Fig. 5 An algorithm for diagnosis and treatment based on the experience of both authors and the current literature (US, Ultrasound; EMG, Electromiography)

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None declared.

AUTHOR CONTRIBUTIONS

All the authors wrote one of the sub-chapters of the paper.
NC and SF arranged and checked the final manuscript.

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