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Case Report

Management of large and diffuse distal neuroma of suprascapular nerve by nerve transfer: A case report and review of literature

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ABSTRACT

Aim: Owing to its peculiar anatomical location, circuitous pathway with acute angles and several sites of relative fixation, suprascapular nerve (SSN) is vulnerable to both traction and compression forces along its course from the upper trunk to its terminal insertion on the infraspinatus muscle. We report a case of a large neuroma in the distal part of SSN, managed by direct transfer of the distal part of spinal accessory nerve into the SSN.
Subject: A 26-year-old man presented with restricted shoulder abduction and wasting of the supraspinatus and infraspinatus muscles. Electromyography (EMG) revealed complete denervation of the infraspinatus muscle, while the deltoid and paraspinal muscles were normal. On anterior exploration all components of supraclavicular plexus appeared essentially normal. SSN, though appeared normal, failed to respond to electrical stimulation. Distal part of the SSN approach through a dorsal approach, revealed a long neuroma-in-continuity. Resection of the neuroma created a 4.5 cm nerve defect. Neuronal continuity was restored by a direct transfer of the distal part of SAN to the distal stump of SSN.
Result: At 24 months follow-up patient had restored a full range of shoulder abduction and about 70° of external rotation.

1. Introduction

Suprascapular nerve (SSN) is predominantly a motor nerve that supplies the supraspinatus and infraspinatus muscles. Anatomically the SSN is fixed at multiple sites, viz. at its origin in the upper trunk of the brachial plexus; at the suprascapular notch[1]; and also at its insertion on the infraspinatus muscle.[2] Owing to this feature, the nerve is vulnerable to various traction forces that increase the acromio-mastoid distance, thus stretching the nerve between the Erb's point and the suprascapular notch.[1,3] Bora et al[4] and Sunderland[5] observed that a nerve stretched 6% beyond its resting length leads to altered conduction, while stretching beyond 15% leads to irreversible damage. Direct trauma due to glenohumeral joint dislocation, fracture of the proximal humerus or scapula, or a penetrating injury are another common modes of SSN injury.[6,7]

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Nagano[8] noted that double or triple level injury in the SSN is not infrequent, and Mikami et al[9] documented a double lesion of the SSN in 7 out of 22 patients. Thus it is imperative that management protocol of any SSN injury should include a thorough exploration along its entire course by a combined anterior and dorsal approaches to overlook all possible sites of injury.

In this case report, we illustrate the utility of the combined anterior and dorsal approaches in the effective management of a large neuroma in the distal part of the SSN, which could have otherwise been missed by a solitary anterior dissection. The combined approach not only helped in identifying the lesion, but also ensured feasibility of SSN reconstruction by a direct transfer of the distal part of the spinal accessory nerve (SAN) into the SSN.

2. Case history

A 26-year-old man presented with loss of shoulder abduction and external rotation in the non-dominant left upper extremity, 4 months after a motorbike accident [Fig. 1]. Clinical examination revealed wasting of the supraspinatus and infraspinatus muscles [Fig. 2]. The bulk of the deltoid muscle was well maintained. Elbow flexion strength was graded M4 on the Medical Research Council (MRC) scale. Electromyography (EMG) revealed a complete denervation of the infraspinatus muscle, while the deltoid and paraspinal muscles were normal. Magnetic Resonance Imaging (MRI) study of the supraclavicular brachial plexus failed to reveal pseudomeningocele or a neuroma in the upper trunk. A complete brachial plexus exploration was performed under general anesthesia through the standard anterior supraclavicular approach. Intraoperatively all components of the plexus appeared normal. A stimulus of 2 mA, delivered by a nerve stimulator, confirmed the integrity of the supraclavicular plexus, barring the SSN, which though appeared normal, failed to respond to electrical stimulation.

Further exploration of the SSN in the retroclavicular region also did not reveal any abnormal finding. Subsequently, the patient was placed in a lateral position and the distal part of the SSN was approached through a dorsal approach.[10] This revealed a 4 cm long neuroma-in-continuity in the middle and distal segments of the SSN that was tethering the nerve to the underlying adipose tissue [Fig. 3]. Resection of the neuroma [Fig. 4] created a 4.5 cm nerve defect. Neuronal continuity was restored by a direct transfer of the distal part of SAN to the distal stump of SSN [Fig. 5].

Histopathological examination of the resected tissue revealed a non-encapsulated lesions consisting of a disordered collection of axons, Schwann cells, endoneurial and perineurial cells in a dense collagenous matrix surrounded by fibroblasts, consistent with a traumatic neuroma [Fig. 6].

Fig. 1 – Preoperative photograph showing lack of shoulder abduction.

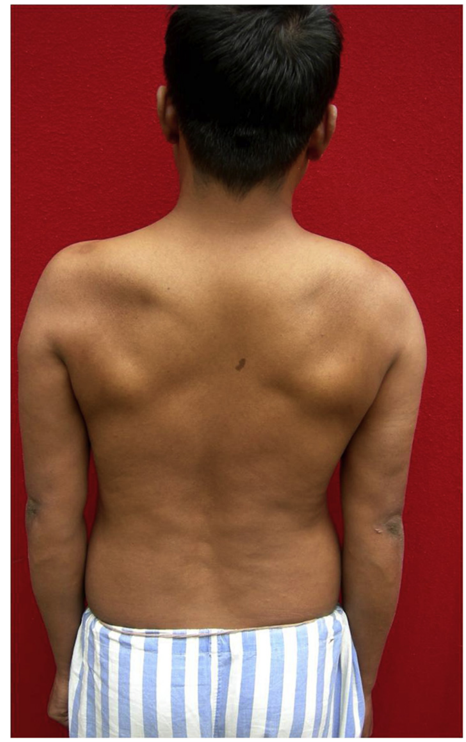

Fig. 2 – Wasting of supraspinatus and infraspinatus muscles.

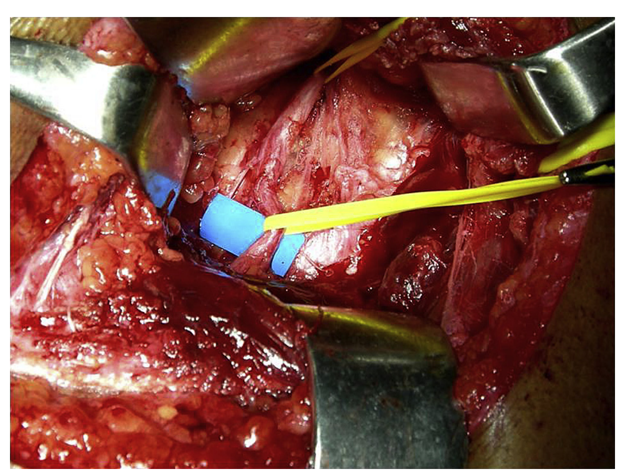

Fig. 3 – Neuroma-in-continuity in the distal part of suprascapular nerve, just proximal to the suprascapular notch.

Fig. 4 – Resected specimen of neuroma.

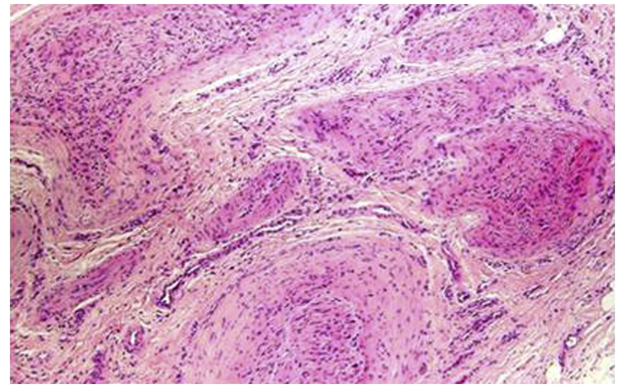

Fig. 6 – Histopathological examination of resected specimen is consistent with a traumatic neuroma.

The patient was placed on regular postoperative follow-up. The first electrophysiological sign of reinnervation of the infraspinatus muscle appeared at 22 weeks. At 24 months follow-up, he had restored a full range of shoulder abduction [Fig. 7] with 70° of external rotation. The muscle bulk of the supraspinatus and infraspinatus muscles were also restored.

3. Discussion

The SSN originates from the upper trunk and contains fibers from the C5 and C6 cervical roots. It supplies motor fibers to the supraspinatus and infraspinatus muscles as well as sensory innervations to the glenohumeral and acromioclavicular joints, the coracohumeral and coracoacromial ligaments, and the subacromial bursa. It takes an oblique and lateral course from its origin, passes deep to the trapezius and omohyoid muscles, and traverses towards the suprascapular notch beneath the suprascapular ligament. At this juncture, the suprascapular artery and vein crosses over the ligament. In the supraspinatus fossa the SSN gives one or two branches to the supraspinatus muscle and sensory twigs to the glenohumeral and acromioclavicular joints. The motor branch to the supraspinatus muscle, however may arise proximal to the suprascapular notch and course over the ligament along with the artery.[11] The nerve then continues distally, passing through the spinoglenoid notch, and entering the infraspinatus fossa, where it innervates the infraspinatus muscle.

Owing to the peculiarities of its anatomical course, the SSN is susceptible to injury at several places. Two of the most

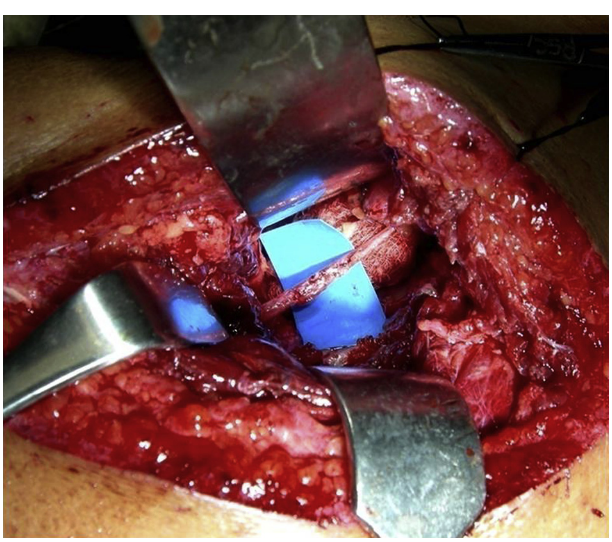

Fig. 5 – Direct transfer of spinal accessory nerve into the suprascapular nerve.

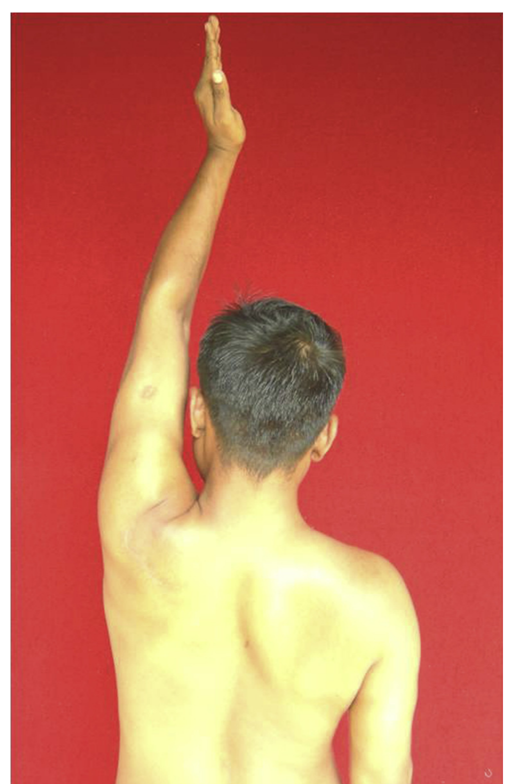

Fig. 7 – Postoperative recovery in shoulder abduction.

vulnerable sites are the supraclavicular region, near the Erb's point, and at the scapular notch.[12] Terzis et al[13] mentioned vulnerability of the SSN at three points, viz. where it leaves the upper trunk; at the suprascapular notch; and at the spinoglenoid notch where it bends around the spine of the scapula.

Rengachary et al[14] attributed the SSN injury to a "sling effect", where diverse movements of the arm leads to traction of the nerve between the Erb's point and the suprascapular notch, in addition to the friction or rubbing of the nerve against the suprascapular ligament or the notch. Other possible mechanisms of SSN injury include traction induced by stretching of the nerve and its accompanying microvasculaure; or a direct trauma leading to an intimal damage and microembolization of the vasa nervorum.[15] SSN injury may present either as a neuroma, or as an ischemic fibrosis due to the severe stretching of the nerve between its fixed points.

Proximal SSN injuries are amenable to repair by the standard supraclavicular (anterior) approach. In contrast, the retroclavicular part of the SSN has been approached via an incision along the deltopectoral groove and the distal part through a posteriorly placed incision along the spine of scapula. For a complete exposure of the SSN, Ochiai et al[16] used a saber-cut incision across the shoulder, detaching part of the trapezius and the posterior deltoid from the scapula and clavicle.

In general MRI provides multiplanar imaging to assess the various components of the supraclavicular brachial plexus and has become a standard diagnostic modality in the primary evaluation of brachial plexus injuries. In MR myelography root avulsions manifest as pseodomeningoceles. MRI also offers better evaluation of the trunks and cords. In the present case, SSN dysfunction was related to a large neuroma in the distal part of the nerve following a traction injury sustained during a motorbike accident. These distal injuries are generally not amenable to routine MRI evaluation of the brachial plexus and often remain undiagnosed. Therefore in the setting of upper brachial plexus injury, when MRI findings are eqivocal and plexus appears normal on supraclavicular exploration, it becomes mandatory to explore the entire course of SSN, especially when it is 'silent'on electric stimulation. In this case study MRI study as well as the exploratory findings of the supraclavicular plexus were normal as the neuronal injury was restricted to the distal part of SSN. Therefore to have a complete exposure of the SSN we combined the standard anterior approach with the posterior or dorsal approach.

The other unique feature of the reported case rests on its management aspect. One course of action in neuroma management would have been its excision and bridging of the nerve defect with sural nerve graft. We avoided this, as it would have resulted in two coaptation sites with loss of growing axons at each site. This constitutes the rationale for managing devastating brachial plexus injuries, where the SSN has preferentially been reinnervated by nerve transfers.

Dorsal approach provides a good exposure of the distal parts of the SSN and SAN and allows a tension-free coaptation between the two target nerves. This distal transfer of the SAN preserves the important proximal branches to the trapezius muscle and also possesses an adequate number of motor axons[10] to reinnervate the SSN.

In this case report we describe a large neuroma-in-continuity involving the distal part of SSN. To the best of our knowledge, such a large non-conducting neuroma located in the distal part of the SSN has not yet been reported in the literature. Bodily et al[17] report a 2 cm neuroma-in-continuity involving the SSN, which was located several centimeters proximal to the suprascapular ligament and was identified through the infraclavicular exposure. With extensive mobilization of the SSN and release of the suprascapular ligament, they could generate a 2.3 cm of an additional usable nerve length and thus achieve a direct end-to-end repair. This was not feasible in our case as the neuroma was extending more distally and its excision created a large nerve defect (4.5 cm) that was not amenable to direct repair.

We would also like to highlight that in devastating brachial plexus injuries, a possibility of injury to the distal part of SSN should always be excluded. Hence it is imperative to examine the nerve along its entire course, for which we recommend a routine use of combined anterior and dorsal approaches. In large nerve defects, where a direct repair is not feasible after excision of an SSN neuroma, nerve transfer utilizing the distal part of SAN is an alternative and possibly a more effective technique in restoration of shoulder movements.

Conflicts of interest

All authors have none to declare.

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