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CASE REPORT

Isolated suprascapular nerve injury to the infraspinatous following minor trauma

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Case history

A 30-year-old male banker presented with severe weakness to his shoulder. He demonstrated a striking muscular atrophy to his supraspinatous and infraspinatous muscles with severe weakness in external rotation (see Figs. 1 and 2).

All other movements were maintained. The only possible history of injury was a simple fall 8 months previously onto his scapula from which he suffered no pain at the time.

Plain radiographs of his shoulder were normal and magnetic resonance imaging of the shoulder revealed no evidence of trauma, tumour, fracture, cyst or other pathology to the shoulder or scapular regions.

He underwent EMG studies to assess the innervation of the SSN and this demonstrated that there was partial innervation to the supraspinatous muscle but there was no innervation to the infraspinatous.

Surgical exploration was advised to repair and graft the nerve, but the patient declined. He completed an aggressive shoulder physiotherapy programme and was left with the functional deficit.

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Discussion

SSN neuropathy was first reported by Kopell and Thompson[7] and since then there have been numerous authors reporting this rare injury although the true incidence is still not known.

The SSN may be injured anywhere along its course from where it begins at the upper trunks of the brachial plexus to the distal fibres which innervate the muscle. The commonest injury point is proximally where the nerve runs below the superior transverse ligament at the suprascapular notch where there is a foramen that may cause entrapment especially during abduction and cross-adduction.[13]

The nerve then has three branches to supply the supraspinatous and also the glenohumeral and acromioclavicular joints. The SSN then runs across the scapular border and over the spinoglenoid notch into the infraspinatous fossa where there are more branches to supply the infraspinatous. This notch is covered by the spinoglenoid ligament. This ligament has had some controversy regarding its occurrence, but in a recent cadaveric study was shown to be present in 100%.[11]

In the literature injuries to the SSN are split into two distinct groups of proximal neuropathy affecting both infra and supraspinatous, and a distal neuropathy affecting just the infraspinatous muscle.

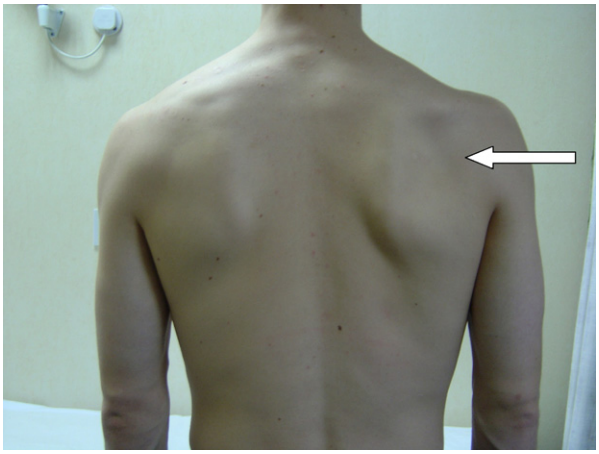

Figure 1 Photograph demonstrating the whole back of the patient. There is a marked atrophy to the infraspinatous as shown with the arrow. There is also some suprascapular and deltoid wasting noted.

The previous reported causes of entrapment neuropathy are secondary to scapular and clavicular fracture,[4] shoulder dislocation,[14] sprain and surgical injury. Other compressive lesions resulting from ganglion cysts and tumours are also reported, and if the compression is anterior to the SSN then a full paralysis of both muscles is more likely.

The SSN may be greatly affected by traction injury and shoulder movements and reported most often in volleyball players, but is also reported in basketball, volleyball and baseball players who all experience extremes of overhead motion.[2,12] A biomechanical study has shown on cadavers that extremes of motion can cause up to a 25% elongation of the nerve and the infraspinatous rather than supraspinatous is more often affected as it is less tolerant to traction.[9]

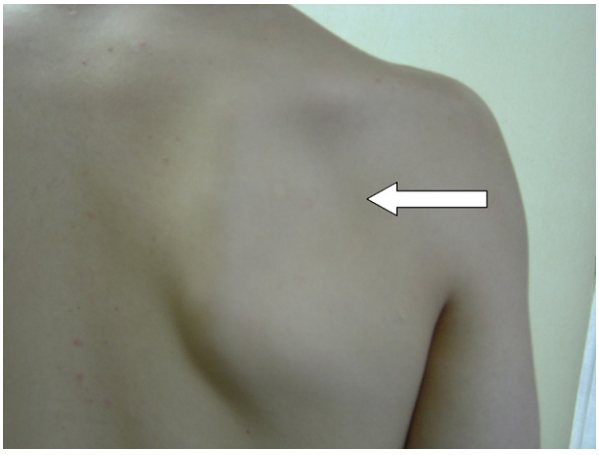

Figure 2 Close view of shoulder with infraspinatous atrophy demonstrated.

Isolated injuries to the infraspinatous branch are more rare and are usually affected at the level where the nerve passes through the spinoglenoid ligament which may become hypertrophied and entrap the nerve. They also occur following gleno-labral tears that can cause cyst formation.[10]

Because the SSN normally has no cutaneous sensory branches, it is often diagnosed late because there are no objective sensory deficits apparent in the innervated area and is not noticed until much later when there is significant muscular atrophy.[3]

Assessment of the nerve and its innervation is performed with electromyography (EMG) and whether any injury is partial or complete.[6] It is usual to measure the level of nerve conduction 10 and 20 cm from Erb's point to assess the supraspinatous and infraspinatous and measure the insertional activity at rest and during voluntary contraction.

Magnetic resonance scanning is the gold standard for ruling out compressive lesions and has a 95—100% sensitivity for diagnosis of SSN entrapment at the spinoglenoid notch via T1 and T2 weighted images showing the degree of oedema which is also a marker for denervation. MRI has been shown to be useful not only the diagnosis of compressive lesions, but also for assessing the stage of paralysis.[5,8]

The normal treatment of injuries to the SSN especially if no direct compressive cause is found should be non-operative, and precludes the halting of any aggravating sports and the completion of a shoulder rehabilitation programme. Surgery is only indicated if this fails and should be performed to decompress the nerve only after a failed period of rehabilitation and should be commenced from between 6 months to a year.

The decompression should include all of the structures potentially compressing the nerve including the spinoglenoid ligament and notchoplasty of the suprascapular notch. Results from decompression have been shown to have favourable outcome if there is a compressive cause such as tumour or cyst. If the injury is traction related or direct closed, then there has been shown to be no direct benefit from early surgery compared to non-operative treatment.[1]

Conclusions

Isolated shoulder weakness in adults may be due to a SSN neuropathy most likely at the level of the spinoglenoid ligament. It usually occurs following repetitive trauma and overhead sporting actions such as in volleyball players, but may occur following trivial injury to the shoulder as shown in this case.

The diagnosis is generally delayed as it is a painless condition presenting late with muscle loss and weakness. Diagnosis is made with magnetic resonance scans and EMG studies and will rule out fractures and compressive lesions.

Initial treatment is with an aggressive shoulder physiotherapy programme but once SSN neuropathy is established, there is little chance of improvement without surgery, and this should be commenced at 6—12 months to decompress and graft the nerve for optimal results.

It is important to be aware of this condition in those presenting following trauma with or without shoulder pain, and to always perform a thorough shoulder examination paying particular attention to any muscular atrophy which may be the first demonstrable sign.

There are no conflicts of interest and all data is genuine and has not bee submitted elsewhere. Full written consent has been obtained from the patient for publication.

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