Two cases of suprascapular neuropathy in a family

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Suprascapular entrapment neuropathy is well known in certain athletes, especially volleyball players. A brother and sister presented with right shoulder pain and wasting of the scapular muscles, particularly the infraspinatus. They had played volleyball for over six years and were forced to retire because of disability. Investigations showed involvement of the rhomboid muscles also, suggesting a probable extension of this syndrome to other nerves in the region such as the dorsal scapular nerve.

Suprascapular nerve entrapment with scapular and shoulder pain, associated with weakness and wasting of the spinati muscles, is well documented in sports people, especially volleyball players. Several mechanisms have been proposed to explain the symptoms, but excessive traction or stretching of the nerve is the most plausible pathomechanism. When this condition develops with an identical clinical profile in two siblings, both volleyball players of some repute, then some anatomical peculiarity seems logical. A brother and sister presented with weakness and wasting of the right scapular muscles and the details are reported here.

CASE REPORTS

Case 1
A 35 year old right handed male volleyball player developed right shoulder and scapular pain after a strenuous match in 1986. He was forced to rest for several weeks but the pain remained unabated. Before the onset of pain, he played volleyball regularly for almost seven years, often representing the state.

Within 2–3 months, wasting of the scapular muscles was observed. He was briefly investigated by radiography and electrophysiology, when neurogenic changes were observed in the right infraspinatus muscle. A year later, he had surgical decompression of the right suprascapular nerve at two levels: the suprascapular notch and the spinoglenoid notch. The surgical notes did not indicate the presence of any nerve atrophy, tight ligaments, or ganglions. However, he continued to deteriorate with persistent pain and progressive wasting, even after decompression, while he continued to play volleyball. He had to retire from active sport around 1990. Since then the condition has stabilised and the pain lessened. However, the pain recurred when he became active again as a volleyball coach. He has no history of right shoulder injury or dislocation.

Physical examination showed an athletic man with no systemic disease or skeletal deformities. There was a surgical scar extending from the right scapula to the upper arm.

All cranial nerves were intact; no abnormality of the facial muscles, sternomastoids, or trapezii was found. He had considerable wasting of the right infraspinatus muscle with weakness of external rotation of the right arm (fig 1). Right shoulder abduction was weak from initiation to 30°. He had mild winging of the right scapula but the power of the serratus anterior was near normal. There was no weakness or wasting of the right deltoid muscle. Other muscles were normal. No muscle fasciculations or myotonia was found.

Deep tendon reflexes were normal and symmetrical in both upper and lower limbs, and plantar responses were flexor bilaterally. All sensations were intact. The right shoulder showed a full range of passive movements. There was mild tenderness over the right scapular spine and acromioclavicular joint.

Routine haemogram, erythrocyte sedimentation rate, and blood chemistry were normal, including normal muscle enzymes. Collagen screening was negative. A radiograph of the right shoulder and cervical spine was normal.
Electromyography of the right shoulder girdle muscles showed chronic neurogenic changes from the right infraspinati and supraspinati and rhomboid muscles. Normal response was found from the right deltoid, trapezius, and serratus anterior.

Case 2
The 27 year old sister of the patient in case I, a right handed volleyball player of about seven years duration, also developed shoulder and scapular pain on the right side about five years earlier, which worsened after vigorous games or matches. Wasting slowly became evident over the right scapula with some prominence of the bone. Because of persistent pain, she too had to retire from the game about two years earlier, and since then had no further progression.

On examination, there were no skeletal abnormalities, thyromegaly, or lymphadenopathy. Demonstrable abnormality was confined to the right scapular region. There was no weakness of the right trapezius or deltoid muscle. Weakness and wasting of the right infraspinatus with weakness of the supraspinatus and rhomboid muscles was demonstrable. There was mild winging of the right scapula at rest (fig 2), with no further increase on pushing or raising the arm (serratus anterior action). There were no myotonia or muscle fasciculations. She had normal and symmetric deep tendon reflexes and flexor plantar bilaterally. She had no sensory dysfunction or muscle tenderness. Passive movements of the right shoulder showed full range and no tenderness.

Blood chemistry including muscle enzymes, haemogram, erythrocyte sedimentation rate, and thyroid function were normal. Collagen screening was negative.

Radiographic and magnetic resonance imaging of the right shoulder, scapula, and cervical spine were normal.

Electromyographic investigation showed chronic neurogenic changes from the right supraspinatit and infraspinatit and rhomboid muscles. Trapezius, deltoid, and serratus anterior were normal.

She refused further investigations and surgical exploration.

DISCUSSION
Suprascapular nerve palsy due to entrapment is common in several sports activities, particularly volleyball.\(^7\) Most of the reported cases are in professional volleyball players, and, in some, even asymptomatic atrophy of the infraspinatus muscle has been documented.\(^3\)

The suprascapular nerve is a motor sensory peripheral nerve (C5) arising as a solitary branch from the superior trunk of the brachial plexus in the posterior triangle of the neck.\(^6\) It passes through the suprascapular foramen beneath the transverse scapular ligament, supplies the supraspinatus muscle, and sends sensory fibres to the acromioclavicular joint and subacromial bursa. Then it descends through the spinoglenoid notch to supply the infraspinatus muscle.

A common proximal site of entrapment of the nerve is at the suprascapular foramen, and hypertrophy or calcification of the transverse scapular ligament and other congenital malformations are among the probable causes.\(^7\) Distally, the nerve may become compressed at the fibro-osseous tunnel formed by the spine of the scapula and the spinoglenoid ligament.\(^5\)

Suprascapular nerve entrapment with isolated paralysis of the infraspinatus muscle is uncommon, but has been reported in volleyball players.\(^4\) A larger than normal range of mobility of the shoulder joint with greater traction injury during the game has been postulated as the probable cause.\(^5\)

Shoulder pain is a common symptom in patients with suprascapular nerve entrapment and is characterised as a dull ache, exacerbated by overhead activities.\(^7\) The pain is articular in origin based on the sensory supply of the acromioclavicular joint and surrounding structures by the suprascapular nerve.

The interesting aspect of the cases reported here is the almost identical symptoms in two siblings, brother and sister, both volleyball players of repute. Both played in state teams for six or seven years before they developed shoulder pain and then muscle wasting. Thus the pattern of their disorder suggested a common anatomical peculiarity for the entrapment rather than coincidence. (There were six other siblings, three brothers and three sisters, who were not athletic and did not have this condition.)

There was clear evidence of maximal involvement of the right infraspinatus muscle in both, but the supraspinatus muscle was also involved clinically and electrophysiologically. In addition, there was slight winging of the scapula with demonstrable weakness of the rhomboid muscles. Serratus anterior muscle weakness produces prominent scapular winging, especially on pushing forward, with difficulty in lifting the arm beyond 90°. Weakness of the rhomboids may also cause mild winging of the scapula without interference with arm abduction and elevation.\(^10\) The patients reported here had no difficulty or weakness in raising the arm fully at the right shoulder with no further increase in scapular winging during such movements. The possibility of weakness of the rhomboid muscles should also be considered in the differential diagnosis as that too may produce winged scapula, without interfering with arm elevation. This problem was thus highlighted in both patients reported here. The dorsal scapular nerve (C5 nerve to the rhomboid muscles) also arises from the brachial plexus. Therefore there may be injury or entrapment of the C5 root somewhere in its course in these patients. However, this seemed unlikely, with the deltoid and other C5 root muscles being completely spared. Hence, the possibility of entrapment not only of the suprascapular nerve but also of the nerve to the rhomboids, with the suprascapular nerve dominating the clinical picture, arises. No previous report has shown involvement of the rhomboid muscles. Another possibility is an aberrant supply of the rhomboid muscles by the suprascapular nerve, but this...
Saphenous nerve injury after fasciotomy for compartment syndrome

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A case is reported of chronic exertional compartment syndrome treated by fasciotomy. The decompression procedure was complicated by injury to the saphenous nerve. The importance of accurate placement of the posteromedial incision line to avoid saphenous nerve injury is highlighted.

Compartment syndrome refers to muscle ischaemia following small vessel occlusion resulting from an increase in local tissue pressure within a closed fascial compartment (fig 1). The condition may be acute (usually secondary to trauma) or less commonly chronic, secondary to exertion. The definitive treatment is surgical decompression of the compartment by fasciotomy. We describe a case of chronic exertional compartment syndrome (CECS) treated by fasciotomy. The decompression procedure was complicated by injury to the saphenous nerve.

CASE HISTORY

A 24 year old kickboxer, with no previous medical history, presented with pain in both shins and posteromedial aspect of his calf muscles, after an increase in his training level. The pain would slowly resolve over hours on cessation of training but would recur when training was restarted. Physical examination failed to show any abnormal clinical signs. Radiographs of tibiae and fibulae were normal. An isotope bone scan excluded any stress fractures. Intracompartamental pressure measurements were made at rest and during a variety of sprinting exercises. The resting measurements were 25 mm Hg in both deep posterior compartments rising to 35 mm Hg in the left posterior compartment during exercise. The resting pressures were 35 mm Hg in both anterior compartments rising to 45 mm Hg in both compartments during exercise. This confirmed the diagnosis of CECS in both anterior and left deep compartments.1 The patient subsequently underwent bilateral compartmental decompression. His original symptoms resolved, but two weeks after surgery he developed a diffuse burning pain extending from below the knee at the end of the anterior incision, down the medial shin to the region of the arch of the left foot. The pain was persistent. The ankle and knee jerks were both present, and there was no evidence of any weakness. A repeat of the intracompartamental pressure measurements and an isotope bone scan showed no abnormality. Nerve conduction studies found no activity in the left saphenous nerve, indicating that the nerve had been severed. Tinel’s sign could not be elicited. The stump of the nerve was identified by nerve conduction studies, but identification was also aided by the presence of a small neuroma which was hypersensitive to the touch.

Figure 1 Compartments of lower limb.

REFERENCES