Radial nerve palsy in an elite bodybuilder

A B Y Ng, J Borhan, H R Ashton, A N Misra, D R M Redfern

A case is reported of high radial nerve palsy in an elite bodybuilder caused by an extrinsic mass effect of muscular hypertrophy. Surgical decompression resulted in complete clinical resolution.

Peripheral compressive neuropathies of the arm are common, and a thorough understanding of the anatomy, pathophysiology, and clinical correlation are required for diagnosis and prognosis. Nerve compressions may be acute or chronic, and may remain mild or result in considerable disability, depending on the mechanism of the injury and the extent of the insult to the nerve. The latter also dictates the prognosis of nerve recovery.

We report a unique case of high radial nerve palsy in an elite bodybuilder caused by an extrinsic mass effect of muscular hypertrophy, with complete clinical resolution after surgical decompression. We believe that this is the first report of such an injury in a bodybuilder.

CASE REPORT

A 45 year old, right handed, elite, male bodybuilder presented with a two week history of gradual onset of spontaneous right wrist drop. There was no evidence of recent trauma to the brachial plexus, upper arm, or forearm to account for the condition. Past medical history included left sided carpal tunnel syndrome, which resolved spontaneously in six months. Physical examination showed considerable wasting of the brachioradialis muscle, loss of the extensor carpi radialis and extensor carpi ulnaris, loss of digital extensors, and loss of the extensor pollicis longus and abductor pollicis longus. The triceps were preserved clinically. Plain radiography did not show any sinister bony and soft tissue lesion. A nerve conduction study showed severe acute motor denervation of muscles innervated by the right radial nerve, suggesting a lesion around the spiral groove. In view of spontaneous recovery of a previous compressive neuropathy without surgical intervention, a similar approach of conservative treatment was started with non-steroid anti-inflammatory drugs and physiotherapy. He was reviewed again after three months; no improvement was found. Magnetic resonance imaging of the right arm did not show any abnormal soft tissue mass, with a normal appearance of the humeral cortex and marrow signal. As no recovery was noted after four months, exploration of the right radial nerve was planned. The patient was placed in the lateral decubitus position, and a posterior longitudinal incision (fig 1) was made. The radial nerve was identified after entering the interval between the long and lateral heads of the triceps. The raphe was incised in the usual manner to expose the nerve. The course of the nerve was traced proximally. There was a notable extensive venous engorgement of the comitantes vessels. The nerve was dissected proximally to the inferior edge of the teres major, which appeared to be constricting the underlying nerve. The aponeurotic edge of the muscle was incised 1.5 cm proximally allowing free passage of the examining digit into the triangular space until no extrinsic compression...
was felt. The lateral head of the triceps was extremely irritable to the point of spontaneously twitching after the decompression was performed. The long head and medial head of the triceps were spared because of the high origin of the branches of the radial nerve. This finding showed that the level of entrapment was at the triangular space, which compromised the nerve supply to the lateral head of the triceps, which has a lower origin. Recovery was uneventful. He was started immediately on a physiotherapy regimen to mobilise his right arm. A review after six months showed that radial nerve function had been completely restored (fig 2).

**DISCUSSION**

Radial nerve palsy may occur with humeral shaft fractures, particularly oblique fractures at the junction of the middle and distal thirds of the bone, local trauma, and after strenuous exercise. The latter has been attributed to compression of the nerve as it passes through the distal portion of the spiral groove by a fibrous arch in the lateral head of the triceps. In addition, progressive palsy has been reported as a result from compression of the triceps in the absence of any defined fibrous arch. Radial nerve entrapment after muscular overexertion has also been reported to be caused by a fibrous arch coming from the long head of the triceps.

We believe that this is the first report of compressive neuropathy of the radial nerve in a bodybuilder. The site of compression is by a muscle previously not known to cause radial nerve palsy. The anatomy of the lesion is located above the spiral groove and in the triangular space, as the teres major forms the upper border of the triangular space posteriorly. The radial nerve runs in front of the tendon of the latissimus dorsi and then passes back through the triangular space. We believe that surgical decompression of the triceps alone would not have alleviated the patient's symptoms. Severe hypertrophy of the teres major produced an extrinsic mass effect on the underlying radial nerve and its associated neurovascular bundle. The patient had presented with serious neuropraxia which only resolved after surgical division of the inferior edge of the muscle.

**Take home message**

Management of peripheral nerve compressions requires a thorough understanding of anatomy, pathophysiology, and clinical correlation for diagnosis and prognosis. This is best undertaken by a neurophysiologist and an orthopaedic surgeon.

The patient resumed routine bodybuilding exercises six months after surgery without any ill effect. Complete clinical resolution was seen six months after surgical decompression.

**REFERENCES**