Axillary neuropathy in volleyball players: report of two cases and literature review

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Abstract

Two cases of isolated neuropathy, not consequent to acute trauma, of the axillary nerve of young volleyball players are described. Interest in the pathology derives from the rarity of such case reports and the fact that the pathogenesis may be linked to a specific sporting activity. The lesion site is thought to be in the quadrilateral space.

Keywords: axillary nerve; entrapment neuropathy; quadrilateral space; volleyball

Isolated lesions of the axillary or circumflex nerve are, in most cases, due to acute trauma and generally occur in cases of dislocation of the scapulo-humeral articulation or fracture of the surgical collar of the humerus.1 Descriptions of an isolated neuropathy of this nerve not consequent to acute trauma are rare. We describe two cases of axillary nerve neuropathy in professional volleyball players.

Case 1

A young 24 year old right handed female semi-professional volleyball player came to our attention complaining of hypoesthesia in the shoulder area innervated by the right axillary nerve associated with limited abduction of the arm and a slight wasting of the deltoid muscle. The symptoms could not be linked to any history of trauma. The patient reported that the deficit appeared in a progressive way after a period of intense sporting competition.

An EMG and electroneurography (ENG) examination showed the presence of fibrillations and slow positive potentials at the level of the anterior and middle portions of the right deltoid muscle. Study of voluntary muscle activation disclosed a pronounced reduction of the interference pattern at the middle level of the right deltoid muscle, no motor unit potentials were registered at the anterior portion of this muscle; its posterior portion presented, however, a slight reduction of the interference pattern. Supramaximal stimulation of the right circumflex nerve at the axilla evoked a weak polyphasic response at the posterior portion of the right deltoid muscle with a 3.2 ms latency and 8.6 mV amplitude; from the middle portion of the muscle, the compound muscle action potential (CMAP) showed reduced amplitude (6 mV) and a latency of 3.5 ms. No evoked response was obtained from the anterior portion. The motor conduction velocity (MCV), from Erb’s point to the axilla, was about 68 m/s. These data suggested discrete involvement, mainly axonal, of the right circumflex nerve in its distal part.

The patient abstained from competitive sport for a prolonged period. After about six months she was seen again. Her sensory disturbance had resolved and there was improvement in muscular strength.

Case 2

An 18 year old left handed woman—a major league professional volleyball player—presented with progressive wasting of the left deltoid muscle and a slight reduction in the strength of shoulder abduction associated with dysaesthesia, at times painful. Finger pressure over the left quadrilateral space provoked tenderness. The symptoms had appeared insidiously some months before, and worsened during a period of intense training. Radiography and MRI studies of the left shoulder and cervical spine and repeated visits to an orthopaedist excluded any osteoarticular pathology.

An initial EMG-ENG examination showed a distal circumflex nerve neuropathy with signs of denervation in the anterior and middle portions of the left deltoid muscle and a pronounced reduction of the interference pattern. Supramaximal stimulation at the axilla evoked a CMAP of reduced amplitude with a significant increase in latency (7.5 ms and 5 mV in the posterior part, 18 ms and 3 mV in the middle, and 15 ms and 3.8 mV in the anterior portion of the muscle). The MCV, from Erb’s point to axilla, was 50 m/s. The patient was advised to reduce all sporting activity.

A follow up EMG-ENG was carried out after about a year; it showed a disappearance of denervation activity and a consistent reduction in latency (4-9 ms posterior, 7-0 ms ante-
Discussion
The axillary, or circumflex, nerve originates from the posterior cord of the brachial plexus. Together with the posterior circumflex artery of the humerus, it passes through the quadrilateral space (figure).

The quadrilateral space is the anatomical compartment bounded by the teres major muscle inferiorly, by the long head of the triceps medially, and by the surgical neck of the humerus laterally; superiorly, it is limited anteriorly by the subscapular muscle and posteriorly by the teres minor. The axillary nerve has its anterior and posterior terminal branches in the quadrilateral space or just after it. Fibres for the teres minor depart from the posterior branch or directly from the main trunk just before or after the passage of the nerve through the quadrilateral space.2

Quadrilateral space syndrome, or more precisely, the compression of the circumflex nerve in this space, sometimes associated with varying degrees of compression of the posterior circumflex artery of the humerus, is a rare syndrome first described by Cahill and Palmer in 1983.1

Isolated neuropathy of the axillary nerve has been described as being secondary to acute trauma involving the shoulder4 or the quadrilateral space directly;5 secondary to sport activity in baseball pitchers6,7 and tennis players,8 and after the use of prosthetic devices for the upper arm using a “figure of eight” type of suspension,9 secondary to hypertrophy and steady use of contiguous muscles,4,10 and finally, secondary to fibrous bands in the quadrilateral space.11,11

The two cases of axillary neuropathy that we describe had an involvement of the terminal anterior and the posterior branches of the nerve, although the second seemed less damaged. It is therefore logical to assume that the lesion was not situated beyond the bifurcation of these two branches, in proximity of the quadrilateral space. In the second case EMG-ENG examination also showed a prolonged latency distally to the probable site of compression. This fact may well be explained by axonal distal atrophy secondary to more persistent trauma.12

In both cases, the patients were young professional volleyball players who had general muscular hypertrophy, especially in the muscles of the shoulder girdle and arm. The circumflex nerve involved was that of the dominant arm—that is, the arm used in serving and smashing, typical volleyball movements that involve significant abduction and extrarotation with flexion or extension of the arm. These types of movements can cause a reduction in the size of the quadrilateral space, possibly resulting in repeated compressions of the circumflex nerve between the surrounding hypertrophic muscles and the humerus.

A contributing factor in such a compression mechanism could be muscle strengthening exercises of the shoulder girdle; however, such exercises are commonly done in many sports.

Therefore, it is reasonable to hypothesise that muscle hypertrophy is only one factor that favours the problem. The typical movements of volleyball are the more fundamental factors that determine the neuropathy.

From the pathophysiological point of view, various hypotheses have been proposed in an effort to explain the mechanism of this neuropathy. Fibrous bands have been seen in many of the cases operated on for this syndrome; necropsy data from those not having a case history of quadrilateral space syndrome did not show such bands.13 It is reasonable to think, therefore, that the adherence bands are secondary to microtrauma that favours the establishment of scar tissue. On the other hand, the compression of the nerve or artery could be a direct consequence of entrapment between hypertrophic muscle heads and the humerus. The nerve also can be damaged by microtrauma and stretching secondary to the large and violent articular excursions often seen during sporting activity.

In the cases reported in the medical literature, the treatment was both conservative (analgesics, physical therapy, and kinetic therapy) and surgical, with various outcomes.11,14 In the two cases that we studied, the suspension or reduction of physical activity resulted in a clinical improvement with disappearance of the sensory symptoms and a discrete recovery in strength.

The fact that the suspension of the specific sporting activity was able to induce a favourable evolution supports the idea of genesis of microtrauma caused both by the type of athletic movement and muscular hypertrophy. On the other hand, the extreme rarity of similar
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case descriptions in athletes, supports the hypothesis of a predisposing genetic cause—for example, a smaller than normal quadrilateral space caused by an anomalous insertion of the muscle heads.

We thank Dr Paolo Guidoni for the anatomical drawing.