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Different patterns of neurogenic quadrilateral space syndrome: a case series of undefined posterior shoulder pain

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Abstract

Background Quadrilateral space syndrome is a painful disorder of the shoulder caused by static or dynamic entrapment of the axillary nerve and the posterior humeral circumflex artery. It was first described in 1983; however, it is an uncommon syndrome that initially presents with nonspecific shoulder pain or selective deltoid atrophy, and diagnosis is often delayed owing to its rarity. Young athletes of overhead sports are more commonly affected by this syndrome. Symptoms of quadrilateral space syndrome include silent deltoid atrophy, persistent posterior shoulder pain, paresthesias, and tenderness over the quadrilateral space. Vascular symptoms may involve thrombosis and embolisms of the upper limb. Instrumental tests and imaging are not always conclusive, leading to frequent misdiagnosis of the syndrome.

Patients and methods The aim of this study is to present a case series of four patients diagnosed with neurogenic quadrilateral space syndrome, describe different clinical presentations, and suggest tips for diagnosing this syndrome. All patients underwent a detailed medical history collection, were interviewed about the sports and hobbies they engaged in, and received a comprehensive clinical examination of the neck and shoulder. Patients also underwent diagnostic exams such as magnetic resonance imaging (MRI) and electromyography. An ultrasound-guided injection of local anesthetic was performed into the quadrilateral space.

Results All patients affected by neurogenic quadrilateral space syndrome underwent conservative treatment, which included a rehabilitation program. Only one out of four patients experienced complete resolution of symptoms and did not require surgical decompression.

Conclusions To properly treat this rare syndrome, we propose classifying it as either "dynamic" or "static," on the basis of the clinical history, MRI findings, and physical examination. The study includes a rehabilitation program that was effective for one patient, demonstrating that surgical decompression may be avoidable if the cases are promptly diagnosed and classified.

Level of evidence IV according to "The Oxford 2011 Levels of Evidence"

Keywords Quadrilateral space syndrome, Axillary nerve, Nerve entrapment syndrome, Posterior shoulder pain, Anterior deltoid atrophy, Overhead pain, Sports injury

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Introduction

Cahill and Palmer first described the entrapment syndrome of the axillary nerve (AN) and the posterior humeral circumflex artery (PHCA) within the quadrilateral space (QS) in 1983 [1]. The QS is bounded by the upper border of teres major inferiorly, the lower border of teres minor superiorly, the long head of triceps medially, and the surgical neck of the humerus laterally [1]. Within the QS, the axillary nerve divides into three distinct fascicular groups that innervate different fibers of the deltoid and teres minor. Several anatomic variants have been described [2–5]. Notably, in approximately 14% of individuals, the long head of triceps brachii is partially or entirely innervated by a branch of the axillary nerve [6, 7].

It predominantly affects young male athletes (20–40 years old) who perform shoulder abduction and external rotation (AER) movements, such as swimmers and tennis, volleyball, and baseball players [8]. Athletes are at higher risk for dynamic stress on the axillary nerve compared with the general population; the presumed prevalence in a cohort of beach volleyball athletes was estimated to be 2.2% [9, 10]. Fibrous bands or other space-occupying lesions can be responsible for the symptoms [10].

Quadrilateral space syndrome (QSS) can present with neurological signs and symptoms such as posterior shoulder pain, nondermatomal paresthesias, tenderness over the quadrilateral space, and isolated deltoid atrophy [11]. The epidemiology of QSS is not fully understood; a small number of cases have been described so far, and the heterogeneity of clinical presentation makes the diagnosis challenging even for senior clinicians, as posterior shoulder pain is frequently vague and difficult to interpret.

It is worth noting that all the patients described in our case series were competitive athletes in overhead sports. This consideration helped us in making the diagnosis and choosing the appropriate treatment. Moreover, when QSS affects the posterior humeral circumflex artery, known as "Pitcher syndrome," symptoms may include thrombosis, aneurysms, embolisms, and digital or hand ischemia [8, 10].

Pathogenesis, clinical features, and diagnosis

The pathogenesis of this syndrome is multifactorial; anatomic findings in neurologic QSS include paralabral cysts, fibrous bands, venous plexus, and space-occupying lesions such as osteochondroma, lipomas, and axillary schwannomas [8]. In vascular QSS, repetitive AER movements induce turbulent blood flow, leading to thrombotic occlusions and aneurysms. In neurogenic QSS (nQSS), the undefined shoulder pain may be aggravated by AER movements [8]

Some cases of painless QSS have also been described in beach volleyball athletes that presented anterior deltoid atrophy and weakness in forward flexion. It is frequently associated with atrophy of the teres minor, though cases with normal or hypertrophic teres minor are also observed [11].

A complete clinical assessment of all common causes of posterior shoulder pain is crucial for making a diagnosis. We suggest including the evaluation of posterior labral injury, posterior instability, and suprascapular nerve entrapment.

The Mayo Clinic classifies QSS into neurogenic and vascular types [10]. Neurogenic QSS includes silent deltoid atrophy, persistent shoulder pain, nondermatomal paresthesias, tenderness over the quadrilateral space, movement limitations unrelieved by common painkillers, and fasciculation of the posterior deltoid or long head of the triceps brachii.

Vascular quadrilateral space syndrome encompasses thrombosis and embolisms, characterized by pallor, coolness, and cyanosis of the hand and digits.

Tenderness over the quadrilateral space suggests QSS, confirmed by ultrasound-guided injection of 5 mL of 1% plain lidocaine into the quadrilateral space. If the anesthetic spreads between the infraspinatus and the teres minor, the test might not be specific, as the anesthetic could involve both the suprascapular nerve and the axillary nerve [12]. To avoid misleading examinations, the procedure should be performed by an expert ultrasound technician. Electromyography (EMG) is not always indicative of QSS because the condition is sometimes caused by a dynamic compression within the quadrilateral space muscular belly, thus the examination should be performed during AER movements. When EMG is performed at rest, it can exclude suprascapular nerve entrapment or significant axillary nerve involvement [13]. MRI is useful for evaluating cystic ganglion or space occupying lesions, and examining the muscle belly may show the first signs of denervation or nerve damage, such as muscle edema or atrophy of the teres minor and deltoid muscle. Two weeks after denervation, it is possible to observe diffused belly edema on STIR images, which sometimes are the first signs of nerve distress [14]

Patients and methods

We present a series of four clinical cases that were consecutively diagnosed and treated as neurogenic quadrilateral space syndrome (nQSS) in the last 5 years; none of the patients exhibited features of vascular QSS, which in our experience, is a very rare condition. All patients underwent a detailed medical history

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collection, were interviewed about the sports and hobbies they engaged in, and received a comprehensive clinical examination of the neck and shoulder, including static and dynamic tests for disc herniation, rotator cuff tendinopathy, glenohumeral instability, capsulolabral tears, scapular dyskinesis, and shoulder stiffness. All patients also underwent magnetic resonance imaging (MRI) and electromyography (EMG). To complete the diagnostic process, an ultrasound-guided injection of local anesthetic was performed into the quadrilateral space by clinicians well trained in shoulder ultrasound examination. After confirming quadrilateral space syndrome, all patients underwent conservative therapy, which included a rehabilitation program and the administration of painkillers.

The conservative treatment focused on strengthening the scapular stabilizing muscles, along with the posterior deltoid and restoring shoulder rotator balance, and to prevent scapular protraction and improper mobility. Reinforcement of pectoralis major and subscapularis muscles was used to compensate for the lack of anterior deltoid function. Transverse friction massages were also used to release soft tissue stiffness.

Finally, self-assisted stretching exercises for the posterior capsule were proposed.

If surgical procedure is considered necessary, we suggest the lateral decubitus position: a 5-cm surgical incision is made, centered on the posterior border of the deltoid. Using retractors to gently elevate the deltoid, the adipose tissue within the quadrilateral space is identified, and by blunt dissection, the axillary nerve is isolated in its various components as described in Fig. 1.

The intraoperative examination with an electroneuro stimulator allows us to identify the various branches of the axillary nerve and to assess the potential for partial or complete paralysis of these branches.

The rehabilitation program after surgical decompression of quadrilateral space was tailored to each patient; initially, activation of periscapular and rotator cuff muscles was encouraged to restore shoulder balance. When possible, early mobilization was recommended to limit the formation of fibrous bands. Patients who underwent surgical neurolysis were evaluated the day after surgery, as well as 1 month and 3 months postoperatively [13]. Patients receiving conservative treatment underwent strict clinical evaluation to monitor their progress. Follow-up concluded once the patient reported resolution of symptoms (visual analog score [VAS] 0–1) and complete recovery of active and passive range of motion (ROM), measured in comparison with the contralateral shoulder (Figs. 2, 3, 4, 5).

Case presentation

Case 1

A 54-year-old right-handed male tennis player presented with posterior shoulder pain, paresthesias, and limitations in daily activity, sports practice, and sleeping. The visual analog scale (VAS) was 6 at rest and 9 during AER movements. Symptoms worsened insidiously, and the patient used a sling for pain management.

Fig. 1 Surgical approach to the QS; it is possible to identify the common branch of axillary nerve (AN), which divides into the branch for anterior and middle deltoid (AB), and in the posterior branch (PB) for posterior deltoid and teres minor. In this picture, it is possible to observe the hourglass deformation of the PB, which was entrapped by fibrous bands

Fig. 2 Surgical approach to the quadrilateral space

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Fig. 3 Edematous and atrophic right teres minor on magnetic resonance imaging of shoulder

Fig. 4 Posterior labral peel-back lesion that is elevated by a palpator tool

Fig. 5 Anchors repair of posterior labral peel-back lesion

Clinical examination ruled out rotator cuff disease and cervical pain but revealed a posterior clicking and a positive Porcellini's test [15]. The pain was intensified by palpation over the quadrilateral space and pain relief after ultrasound-guided lidocaine injection (lido-test) confirmed QSS. No differences in deltoid muscle trophism were noted. He underwent electromyography, which did not detect abnormalities of the axillary or suprascapular nerves. The administration of oral nonsteroidal antiinflammatory drugs, opioids, and even pregabalin was not completely effective. The pain intensified during rehabilitation exercises for the deltoid and teres minor, and therapeutic massages did not improve the symptoms. After some sessions, the pain worsened, and physical therapy was interrupted. MRI showed no abnormalities; 2 months later, he repeated the examination, and a progressive atrophy of the teres minor was noted in the comparative view of the sagittal plane.

Four months after the onset of symptoms and relative conservative therapy, the patient underwent surgical decompression of the fibrous band entrapping the branch for the teres minor. An arthroscopic view was also performed, and posterior labral peel-back lesion was fixed with anchors. The patient reported complete pain relief the day after the surgery and began a rehabilitation program, which involved activating the periscapular muscles and resting for 3 weeks in a sling with 15° abduction pillow. After 3 weeks, passive shoulder mobilization was initiated across all planes, with internal rotation avoided for an additional 3 weeks. Following the restoration of range of motion, the patient was encouraged to begin active movements in a warm pool. Eight weeks post surgery, the patient was ready to start a strengthening program for the external rotators, deltoid, and humeral head depressor muscles. Subsequently, the internal rotator muscles were also strengthened to prevent imbalance. Only after isometric testing confirmed balanced strength between internal and external rotators did the patient return to play.

Case 2

A 17-year-old right-handed professional volleyball player presented with undefined shoulder pain and loss