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REVIEW ARTICLE

Quadrilateral space syndrome: a review



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Quadrilateral space (QS) syndrome (QSS) is a relatively **rare** condition in which the axillary nerve and the posterior humeral circumflex artery are compressed within the QS. **Fibrous bands** are most commonly implicated as the cause, with true space-occupying lesions being less common. QSS is characterized by poorly localized shoulder pain and paresthesia over the lateral aspect of the shoulder and arm in a nondermatomal pattern. These symptoms are **aggravated** by shoulder abduction and external rotation. Point tenderness is typically present over the QS; however, **diagnosis on physical examination can be difficult**. Pain relief after lidocaine block of the axillary nerve within the QS is a useful finding in the evaluation of patients with suspected QSS. No definitive diagnostic imaging exists, making diagnosis difficult, although radiographs and magnetic resonance imaging are recommended **to rule out** other pathology. **Nonoperative treatment**, including nonsteroidal anti-inflammatory drugs, activity modification, and physical therapy, for at least 6 months is recommended before pursuing operative intervention. Small case series have shown that surgical decompression of the QS has good outcomes, with resolution of pain and return to sport.

Level of evidence: Narrative Review

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Quadrilateral space (QS) syndrome (QSS) was first described by Cahill and Palmer⁶ in 1983 as compression of the posterior humeral circumflex artery and axillary nerve or 1 of its major branches in the QS. This rare syndrome can lead to poorly localized shoulder pain, discrete tenderness to palpation over the QS, and possible teres minor and deltoid denervation.^{6,22} These symptoms are typically exacerbated by abduction and external rotation or forward flexion of the shoulder.⁶ The vague symptoms and lack of definitive imaging make the diagnosis difficult and require diligence on the part of the clinician for accurate assessment.

Institutional Review Board approval was not needed for this study because it was a review of prior published articles.

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Anatomy

The QS is located in the posterior aspect of the shoulder, bounded by the long head of the triceps medially, the medial edge of the surgical neck of the humerus laterally, the teres major and latissimus dorsi muscles inferiorly, and the teres minor muscle or the glenohumeral capsule superiorly (Fig. 1, A and B). The QS contains the posterior circumflex humeral artery and the axillary nerve (Fig. 1, A and C). A study of 50 cadaveric specimens found the axillary nerve originated from the posterior cord of the brachial plexus in all cadavers, traveled obliquely over the anterior aspect of the subscapularis tendon, passed under the axillary recess of the glenohumeral joint, and entered the QS. The axillary nerve then divided into anterior and posterior branches within the QS in 88% of specimens and within the deltoid muscle in the remainder of specimens.²⁴

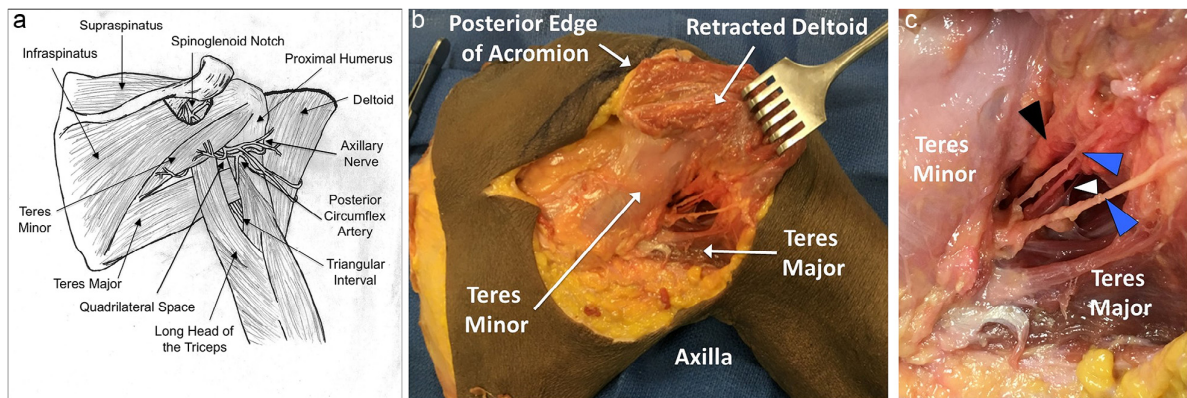


Figure 1 (A) Schematic of the quadrilateral space (QS) from the posterior view. (B) Cadaveric dissection of the QS from the posterior view. (C) Magnified cadaveric dissection of the QS and its contents, including the posterior humeral circumflex branches (blue arrowheads), axillary nerve (black arrowhead), and fibrous bands (white arrowhead). Lateral is on the right of the image.

In all specimens in 2 cadaveric studies, the anterior branch of the axillary nerve traveled with the posterior circumflex humeral vessels, with the nerve lying superior.^{24,30} The posterior branch is responsible for innervating the teres minor and the skin over the distal two-thirds of the posterior deltoid via the superolateral brachial cutaneous nerve.¹⁶ The posterior circumflex artery also divides after entering the QS into branches similar to the anterior and posterior branches of the axillary nerve. Fibrous bands are commonly found in the QS in cadaveric studies^{22,30} (Fig. 1, C). The study by McClelland and Paxinos²² found fibrous bands in 14 of 16 cadaveric shoulders originating from the thick fascial layer of the long head of the triceps and attaching to the teres major, adjacent to the axillary nerve in the QS.

Etiology

QSS can be caused by any condition that decreases space and causes compression of the QS contents. The most common reason cited for compression of the axillary nerve and posterior circumflex humeral artery is fibrous bands.^{5,19,21,24} One cadaveric study noted that the fibrous bands caused a decreased cross-sectional area of the QS when they tightened during shoulder abduction and external or internal rotation and postulated that this may cause compression of the QS contents.²² Compression of the axillary nerve within the QSS is often dynamic or positional, which can make diagnosis difficult. Space-occupying lesions in the QS have also been implicated as the cause of cases of QSS in the literature, including paralabral cysts,^{5,15,27,28} bony fracture fragments,¹ benign tumors, such as lipomas, humeral osteochondromas,¹⁰ and axillary schwannomas.¹⁸ Venous dilation has been described as a cause of QSS.²⁰ Muscle hypertrophy has been cited as a causative agent for compression in competitive overhead athletes.^{6,25,26}

The exact pathophysiology of QSS is unclear. One theory is that QSS is caused by compression or irritation of the branches of the axillary nerve (neurogenic), whereas another

common theory is that it is caused by compression leading to decreased perfusion of the posterior humeral circumflex artery (vascular). Patients with combined nerve and vascular compression confirmed with imaging and surgery have been reported.⁵ Irritation of the posterior humeral circumflex artery can cause arterial thrombosis or aneurysm, with reported emboli causing distal ischemia.⁵

Epidemiology

QSS is most commonly seen in patients aged 20 to 40 years and most commonly involves the dominant shoulder.³ Men are more commonly affected than women.⁵ QSS usually affects active patients, especially overhead athletes.³ Sports most commonly associated with QSS involve significant use of abduction and external rotation such as volleyball, swimming, and baseball.^{5,21,25} The true frequency of QSS is unknown given the difficult and often incorrect diagnosis.

History

The typical presentation of a patient with QSS is slow-onset, intermittent, and poorly localized shoulder pain without trauma. Pain has been described as posterior as well as over the lateral shoulder and arm. Paresthesias of the affected upper extremity in a nondermatomal distribution, usually over the posterior or lateral aspect of the shoulder and arm, or both, without diminished sensation to light touch are commonly seen. These symptoms are typically aggravated by abduction and external rotation of the affected shoulder.¹¹ Throwing athletes may describe pain with the late cocking phase of throwing and shoulder fatigue in the overhead position.²

Physical examination

Other causes of shoulder pain should be ruled out on physical examination, most commonly rotator cuff pathology, labral

pathology, cervical spine pathology, and thoracic outlet syndrome. Point tenderness over the QS is almost always seen and can help distinguish QSS from rotator cuff or labral pathology. Palpation of the QS sometimes causes lateral shoulder pain or paresthesias. Holding the arm in flexion, abduction, and external rotation for 1 to 2 minutes has been described as a method to recreate the patient's symptoms.² Deltoid atrophy or weakness, or both, can be seen in chronic cases but is not a common finding.²⁴ QSS should be considered when prior shoulder arthroscopy patients describe no relief of their pain compared with preoperative pain levels.

In the most extreme form of vascular QSS, aneurysm or thrombosis of the posterior circumflex humeral artery can occur. Thrombosis of the posterior humeral circumflex artery can back up into the axillary artery and then embolize distally, causing distal cyanosis, digital ischemia, loss of pulses, cold intolerance, or splinter hemorrhages.^{5,31}

Imaging

Plain radiographs are an important first step in the radiologic evaluation of possible QSS. Radiographs can not only allow the clinician to rule out fractures but also evaluate for bony masses or other radiopaque space-occupying lesions.¹⁰ When Cahill and Palmer⁶ first described QSS, they recommended that all patients with the cardinal features of QSS undergo a subclavian arteriogram with the shoulder in abduction and external rotation to assess for compression of the posterior circumflex humeral artery. This is no longer used as a diagnostic tool for QSS because a positive subclavian arteriogram has been shown to occur in both symptomatic and asymptomatic patients, thus having a high false-positive rate.²³ Dynamic arteriography can still be used but requires an arterial needle puncture, uses relatively high doses of radiation, and is expensive.

Electromyogram (EMG) has been used in diagnosis of QSS but is often negative due to the dynamic or positional nature of QSS.²⁰ However, EMG can demonstrate decreased amplitudes along the axillary nerve and evidence of teres minor or deltoid muscle denervation in some cases.^{15,24} EMG can also help rule out other neurologic causes of pain such as cervical spine or brachial plexus pathology.

Advanced imaging with magnetic resonance imaging (MRI) or computed tomography (CT) can be used to evaluate for findings associated with QSS such as teres minor or deltoid muscle atrophy (Fig. 2, A) or a space-occupying lesion in the QS. MRI can also be used to assess the contents of the QS, although the individual structures are often difficult to see (Fig. 2, B). An abnormal signal within the teres minor muscle on T2 imaging suggesting neurogenic edema may also be present in QSS.^{4,11,13,15} Atrophy of teres minor or deltoid muscle may suggest chronic QSS. However, isolated teres minor denervation seen on MRI is not uncommon on routine shoulder MRI, suggesting that it may be associated with other pathologies causing altered biomechanics such as rotator cuff injuries, prior surgical intervention, and traction injuries with glenohumeral instability.^{11,29} In addition, 3-Tesla magnetic resonance neurography is a possible modality, which has been recently described, to evaluate for axillary nerve compression.⁹ Advanced imaging can also exclude other causes of shoulder pain such as rotator cuff or labral pathology.

Concomitant QSS and intra-articular pathology can also exist.^{15,27,28} MR angiography has been shown to reveal occlusion of the posterior circumflex humeral artery during abduction and external rotation, but this occurred in both symptomatic individuals and in 80% of the asymptomatic individuals evaluated.²³ CT angiogram can also be useful in diagnosing thrombosis in the posterior humeral circumflex artery and in distal arterial lesions.⁵

Ultrasound examination of the shoulder can be performed when attempting to diagnose QSS, although this technique is sonographer dependent. Color Doppler

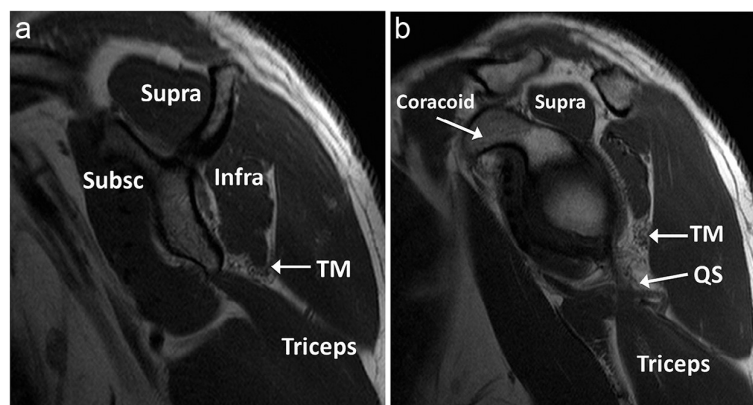


Figure 2 (A) Sagittal oblique T1 magnetic resonance image medial to the suprascapular notch shows teres minor (TM) muscle atrophy. (B) Sagittal oblique T1 magnetic resonance image in the plane of the quadrilateral space (QS). *Infra*, infraspinatus; *Subsc*, subscapularis, *Supra*, supraspinatus.

sonography can be used to visualize the posterior humeral circumflex artery both with the arm abducted 90° with the arm hyperabducted and externally rotated to assess for differences in arterial flow between the 2 positions.⁴ Ultrasound can demonstrate swelling or lesions of the axillary nerve and space-occupying lesions in the QS. Teres minor muscle size and echogenicity can also be visualized using ultrasound and compared with the contralateral side.^{4,8,24} Unfortunately, ultrasound is unable to detect the fibrous bands that are most commonly implicated in QSS.⁴

Diagnosis

The differential diagnosis for QSS is broad and should include thoracic outlet syndrome, referred pain from cervical radiculopathy, brachial plexitis or plexopathy, rotator cuff pathology, impingement syndrome, glenohumeral joint arthritis, adhesive capsulitis, glenohumeral instability, and suprascapular nerve injury. McAdams and Dillingham²⁰ described their algorithm for the assessment of patients with posterior shoulder pain before surgical decompression of the QS. Patients were sent to physical therapy to treat other possible common causes of posterior shoulder pain if the physical examination was concerning for possible QSS. An MRI of the shoulder was obtained if the patient did not improve with 6 months of physical therapy. If the MRI did not show a clear anatomic source for posterior shoulder pain, a lidocaine block test was performed in which 5 mL of 1% lidocaine was injected in the QS 2 to 3 cm inferior to the standard posterior shoulder portal. The block test was considered positive if the patient's tenderness in the QS resolved and the patient had no pain during an active throwing motion. A QS block with lidocaine, with or without ultrasound guidance, can aid in the diagnosis of QSS if the administration of the anesthetic around the axillary nerve in the QS provides significant pain relief.^{7,20} A local anesthetic block can be considered a diagnostic gold standard in musculoskeletal conditions with low specificity of physical examination maneuvers and lack of a good diagnostic study.⁷ Although there is no literature on a true gold standard for the diagnosis of QSS, a lidocaine block test seems to be the best current diagnostic tool.

Treatment

Literature on the management of QSS is limited; however, the consensus is to start with conservative treatments. Conservative management for QSS is similar to that for other shoulder pathologies, including use of nonsteroidal anti-inflammatory medication, activity modification to avoid abduction and external rotation, and physical therapy. Physical therapy modalities can include transverse friction massage and active release soft tissue massage techniques as well as traditional shoulder protocols such as shoulder range of motion exercises, posterior rotator cuff strengthening, and scapular stabilization exercises.¹⁷ Physical therapy for periscapular

strengthening can improve muscular coordination around the shoulder girdle, leading to better compression of the humeral head in the glenoid providing less microtrauma to the contents of the QS.³¹ These techniques are typically used for a minimum of 6 months before operative intervention is considered. Studies on the efficacy of physical therapy techniques are lacking.

Pregabalin combined with NSAIDs was reported to cause resolution of symptoms in a patient with a paralabral cyst compressing the QSS.¹⁵ Steroid injection has been cited as a possible treatment option³; however, no studies support its use.

Indications for surgery for QSS include a space-occupying lesion or a positive lidocaine injection test with at least 6 months of failed conservative treatment, as mentioned previously. Open surgical decompression has been described in overhead athletes, patients with a positive lidocaine injection test, and patients with a space-occupying lesion. The patient is placed in lateral decubitus position, and a longitudinal incision of approximately 4 cm is made over the posterior shoulder along Langer's lines (Fig. 3, A). The posterior border of the deltoid is identified and reflected superolaterally to expose the underlying fat in the QS between the teres minor and the teres major (Fig. 3, B and C). The deltoid fibers are not split, and the deltoid remains attached to the acromion during this exposure. The axillary nerve and the posterior circumflex humeral vessels can then be palpated as they exit the QS, and the axillary nerve can be dissected free from fibrous adhesions (Fig. 3, D). While continuing to palpate the axillary nerve, the arm is abducted and externally rotated to examine for free nerve gliding after decompression. The same maneuver is used to palpate the artery to ensure maintenance of a pulse.²⁰

This same approach can also be used for decompression of the QS occupied by space-occupying lesions. Arthroscopic débridement can be performed after open decompression in cases where there is intra-articular pathology, including paralabral cysts or labral tears that may be causing compression of the QS contents.⁵ Alternatively, shoulder arthroscopy can be done before QS decompression but must be limited and efficient to prevent significant fluid extravasation, which would make the decompression more difficult.

Treatment of vascular thrombosis or aneurysm causing distal emboli consists of thrombolytic medications and possible distal thrombectomy as well as posterior humeral circumflex ligation if the symptoms do not improve after thrombolytic medications.⁵

Postoperative rehabilitation

Immediate pendulum exercises and early physical therapy for range of motion are typically recommended after surgical decompression of the QS to prevent adhesion formation postoperatively.²⁰ Some recommend avoiding hyperextension, abduction, and external rotation for 4 weeks

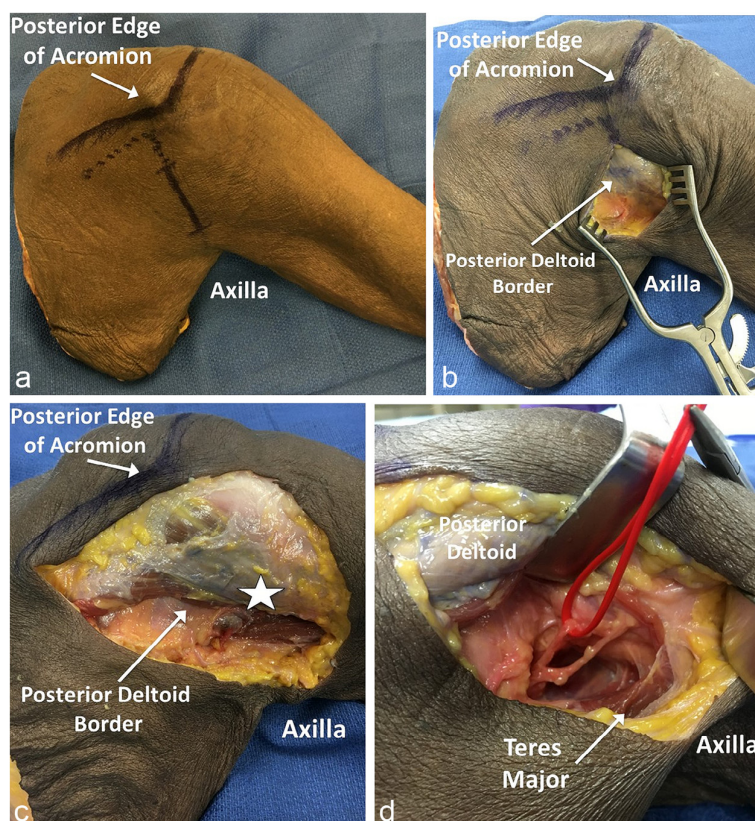


Figure 3 (A) Posterior view of a cadaveric shoulder shows the incision used for quadrilateral space (QS) decompression. The *solid line* represents the least invasive approach. The *dotted line* represents the extended approach if needed. (B) Dissection through subcutaneous tissue exposes the posterior deltoid border and the QS (*white star*). (C) Extended dissection shows the relationship between the posterior border of the deltoid and the QS (*white star*). (D) QS contents with the posterior edge of the deltoid retracted superolaterally. One branch of the posterior humeral circumflex vessel is marked with the red tubing.

postoperatively.²⁰ Sport-specific therapy programs can start as early as 6 weeks postoperatively for athletes.

Outcomes

Complete resolution of symptoms has been reported with nonoperative management, including physical therapy,¹⁷ stopping or limiting inciting activities,²⁵ NSAIDs,¹⁵ and other medications, such as pregabalin,¹⁵ as well as a combination of several modalities.⁸ Most cases resolve with conservative treatment; however, surgical decompression is an option if symptoms persist and other causes of pain have been ruled out.

Although the literature on surgical outcomes is limited, surgical decompression of the QS after failed conservative treatment with excision of fibrous bands has been described as successful, with complete or nearly complete resolution of symptoms in most reported cases.^{5,19-21} One study of 4 overhead athletes who underwent surgical decompression of QS with lysis of the fibrous bands reported that all patients had complete relief of their posterior shoulder pain.²⁰ All

patients regained full active shoulder motion and returned to sports competition by 3 months postoperatively.²⁰ One review reported that of the 23 cases reported of surgical decompression for QSS, only 2 patients (9%) had no improvement of symptoms, and the remaining had good improvement or complete resolution of symptoms.¹² Another study of 5 patients who underwent surgical decompression of the QS after shoulder trauma showed all patients had improvement in pain and nearly normal range of motion.¹⁴

Good symptom relief has been reported after resection of space-occupying lesions as well.^{1,10,18,27} Surgical intervention risks injury to the axillary nerve or its branches and to the posterior humeral circumflex vessels given the required dissection in their vicinity. Infection is also a possibility, but the rate is unknown. Continued pain¹⁹ and stiffness¹⁴ are possible, yet rare, after surgical treatment. Very few complications have been reported after surgical treatment for QSS.

Surgical outcomes of the treatment of posterior circumflex humeral artery vascular occlusion are generally good.⁸ Thrombolytic treatment has not shown good results in these situations, likely because the thrombi are chronic at the time

of accurate diagnosis.⁵ Complications from vascular access, such as iatrogenic femoral artery dissection, have been reported.⁵

Conclusion

QSS is a rare condition in which the axillary nerve and the posterior humeral circumflex artery are compressed within the QS, with fibrous bands most commonly implicated as the cause. QSS is characterized by poorly localized shoulder pain and paresthesias over the lateral aspect of the shoulder and arm in a nondermatomal pattern. The symptoms are aggravated by shoulder abduction and external rotation. Diagnosis on physical examination can be difficult, although point tenderness at the QS is typically seen. There is no definitive imaging, although radiographs and MRI are recommended. EMG, MR or CT angiogram, and ultrasound can be obtained as adjunct tests if needed. Pain relief after lidocaine block of the axillary nerve within the QS is a useful finding in the evaluation of patients with suspected QSS. Nonoperative treatment, including NSAIDs, activity modification, and physical therapy for at least 6 months, is recommended before pursuing operative intervention. Surgical decompression of the QS has been shown to have good outcomes with resolution of pain and return to sport in small case series of patients with QSS.

Disclaimer

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