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Quadrilateral Space Syndrome: The Mayo Clinic Experience With a New Classification System and Case Series

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Abstract

Quadrilateral space syndrome (QSS) arises from compression or mechanical injury to the axillary nerve or the posterior circumflex humeral artery (PCHA) as they pass through the quadrilateral space (QS). Quadrilateral space syndrome is an uncommon cause of paresthesia and an underdiagnosed cause of digital ischemia in overhead athletes. Quadrilateral space syndrome can present with neurogenic symptoms (pain and weakness) secondary to axillary nerve compression. In addition, repeated abduction and external rotation of the arm is felt to lead to injury of the PCHA within the QSS. This often results in PCHA thrombosis and aneurysm formation, with distal emboli. Because of relative infrequency, QSS is rarely diagnosed on evaluation of athletes with such symptoms. We report on 9 patients who presented at Mayo Clinic with QSS. Differential diagnosis, a new classification system, and the management of QSS are discussed, with a comprehensive literature review. The following search terms were used on PubMed: *axillary nerve*, *posterior circumflex humeral artery*, *quadrilateral space*, and *quadrangular space*. Articles were selected if they described patients with symptoms from axillary nerve entrapment or PCHA thrombosis, or if related screening or imaging methods were assessed. References available within the obtained articles were also pursued. There was no date or language restriction for article inclusion; 5 studies in languages besides English were reported in German, French, Spanish, Turkish, and Chinese.

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Quadrilateral space syndrome (QSS) arises from compression or mechanical injury to the axillary nerve (neurogenic quadrilateral space syndrome [nQSS]) and/or posterior circumflex humeral artery (PCHA) (vascular quadrilateral space syndrome [vQSS]) as they pass through the quadrilateral space (QS). The QS is bounded by the edge of the long head of the triceps medially, the medial edge of the surgical neck of the humerus laterally, the tendon of the teres major and latissimus dorsi muscles inferiorly, and the teres minor muscle or the scapulohumeral capsule superiorly[1,2] (Supplemental Figure 1,[3] available online at http://www.mayoclinicproceedings.org).

The term QSS was first coined by Cahill in 1980[4] and then again in 1983 in a subsequent article describing several patients with axillary nerve compression.[5] McCarthy et al[6] later reported partial occlusion of the PCHA in a baseball pitcher, with distal embolization to the right third digit, felt to be due to PCHA injury within the QS. Subsequently, QSS has been reported most commonly in overhead or "throwing" athletes in sports that heavily involve abduction and external rotation (AER), including volleyball,[7-16] baseball,[17-22] and swimming.[23,24] Quadrilateral space syndrome has also been associated with other activities with frequent AER, such as yoga[25] or window cleaning.[26]

Patients with QSS manifest with various symptoms (Figure 1). Neurogenic manifestations may include nondermatomal neuropathic pain, numbness, and weakness in the shoulder (usually posterior[27]), often radiating down the arm. Vascular manifestations may

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include thrombosis, microembolism or macroembolism, digital or hand ischemia, and the full spectrum of signs and symptoms associated with acute ischemia such as pain, pallor, and absent pulses (Figure 1). Vascular manifestations are frequently not reported under the term QSS, but as "PCHA injury,"[10] "compression syndrome of the PCHA,"[9] "aneurysm of the PCHA,"[8,11,25,28] "PCHA pathological lesions with digital ischemia,"[7] or "Pitcher syndrome."[25]

We report a case series of 9 patients presenting to Mayo Clinic with nQSS or vQSS; patients 2 and 3 were previously reported.[29] Each patient underwent diagnosis and treatment according to the algorithm provided in Figure 1. For a comprehensive literature review on PubMed, we used the following search terms: *axillary nerve*, *posterior circumflex humeral artery*, *quadrilateral space*, and *quadrangular space*. Articles were selected if they described patients with symptoms from axillary nerve entrapment or PCHA thrombosis, or if related screening or imaging methods were assessed. References available within the obtained articles were also pursued. There was no date or language restriction for article inclusion; 5 studies in languages besides English were reported in German, French, Spanish, Turkish, and Chinese.

ARTICLE HIGHLIGHTS

- Quadrilateral space syndrome (QSS) is a rare and underdiagnosed yet potentially debilitating neurovascular syndrome; physicians, particularly those treating elite athletes and perhaps those in sports medicine, vascular medicine, and neurology, should continue to pursue understanding of the recognition, differential diagnosis, discrimination, and prognosis of both neurogenic quadrilateral space syndrome (nQSS) and vascular quadrilateral space syndrome (vQSS).
- Fixed structural impaction of the quadrilateral space (QS) with space-occupying lesions or fibrous bands leads to nQSS with nondermatomal paresthesias and QS point tenderness.
- Repetitive mechanical injury to the posterior circumflex humeral artery (PCHA) as it passes through the tight QS and wraps around the humeral neck during abduction and external rotation leads to vQSS with PCHA thrombosis and/or aneurysm with distal embolization and digital ischemia.
- The prevalence of nQSS relative to vQSS is 1.5:1, with a male predominance (male/female ratio, 7:1) for all QSS, and an almost exclusive male population for vQSS (likely due to traditional sex distribution in overhead athletics).
- Posterior circumflex humeral artery ligation is used to surgically treat vQSS to prevent distal embolization, along with thrombolysis for acute thrombotic embolization. Surgical neurolysis and QS decompression are used to definitively treat nQSS.

CASE PRESENTATIONS

Patient 1: vQSS Due to PCHA Thrombosis and Distal Embolism

A 20-year-old right-handed female collegiate volleyball player presented with a 2-week history of paresthesias and purplish discoloration involving multiple digits of the right hand. She was a nonsmoker without known thrombophilia. Examination revealed a pale, cool right hand with splinter hemorrhages (Supplemental Figure 2, A, available online at http://www.mayoclinicproceedings.org) and absent radial and ulnar pulses. No motor or sensory deficits were noted. Arterial Doppler signals at the wrist were reduced.

Computed tomography angiography (CTA) revealed multifocal occlusion of the right ulnar and common interosseous arteries with distal reconstitution, as well as long-segment occlusion of the right radial artery. There was no evidence of thoracic outlet arterial compression or aneurysm suggestive of a more proximal embolic source. She was anticoagulated with intravenous heparin. An arch angiogram with right upper extremity runoff confirmed the CTA findings (Figure 2) and revealed a thrombosed PCHA (Figure 3).

After 48 hours of catheter-directed thrombolysis, there was minimal improvement in flow to the hand. Surgical exploration revealed acute dissection with fresh thrombus in the PCHA. The thrombus was removed, and the PCHA was ligated. Embolectomy of the radial, ulnar, and interosseous arteries was performed (Supplemental Figure 2, B-D) on 2 different occasions. She was maintained on therapeutic anticoagulation. At 3 months, she continued to work with her volleyball trainer, with gradual improvement. An iatrogenic right femoral artery dissection from her angiogram

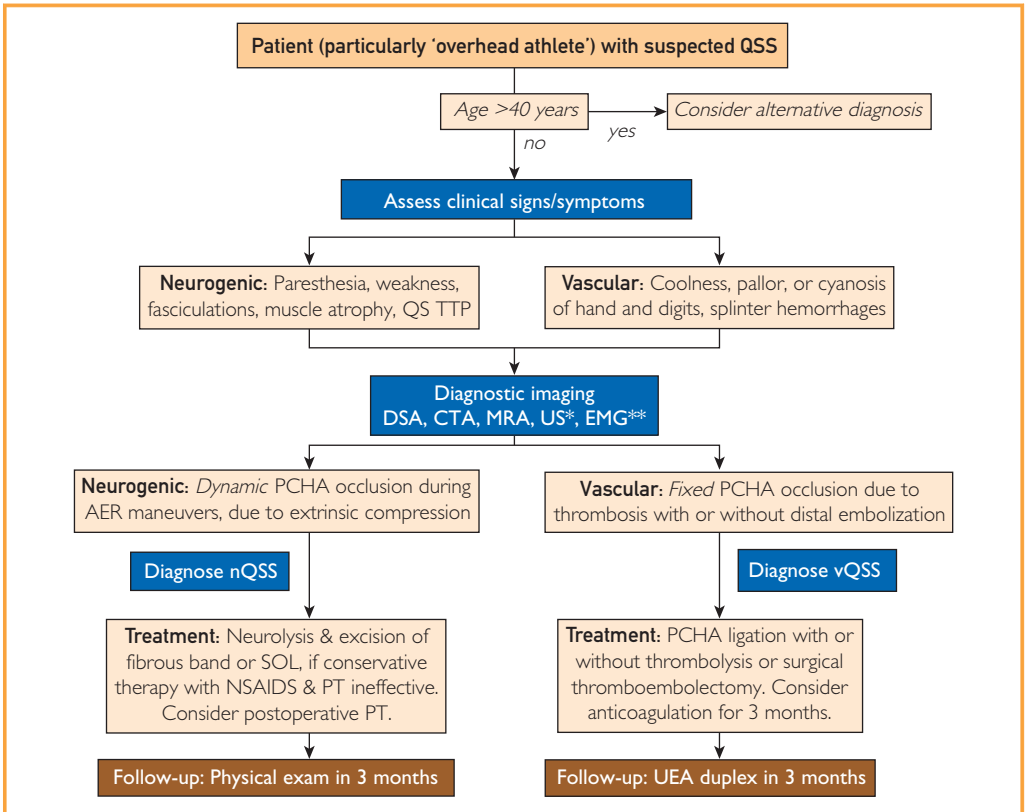

FIGURE 1. Diagnosis and treatment algorithm for patients with suspected QSS (alternative diagnosis must also be excluded). AER = abduction and external rotation; CTA = computed tomography angiography; DSA = digital subtraction angiography; MRA = magnetic resonance angiography; nQSS = neurogenic quadrilateral space syndrome; NSAID = nonsteroidal anti-inflammatory drug; Overhead athlete = any patient engaged in elite, collegial, professional, or dedicated sport or activity involving repetitive abduction and external rotation of the arm above the head; PCHA = posterior circumflex humeral artery; PT = physical therapy; QS = quadrilateral space; QSS = quadrilateral space syndrome; SOL = space-occupying lesion; UEA = upper extremity arterial; US = ultrasound; vQSS = vascular quadrilateral space syndrome. *Ultrasound imaging has been preliminarily studied for the diagnosis of nQSS; there have not yet been studies specifically assessing ultrasound use to diagnose vQSS. **Electromyography has poor sensitivity for nQSS, but can rule out alternative diagnoses.

required patch angioplasty. At 6 months, she had normal upper extremity pulses with unrevealing noninvasive vascular laboratory studies, and complete recovery from the femoral artery repair.

Patient 2: vQSS Due to PCHA Thrombosis and Distal Embolism

A 24-year-old left-handed female professional volleyball player presented with a 1-month history of blue discoloration and intermittent swelling of the fingers of her left hand, with cold intolerance. Her only thrombophilia risk factor was the use of oral contraception, which she used for irregular menses. Examination revealed cyanosis of digits 3, 4, and 5 in the left hand. There was a splinter hemorrhage in digit 3. Digits were otherwise normal. Lower extremities were unremarkable.

Cervical spine radiographs were normal without a cervical rib. Vascular laboratory studies revealed slightly abnormal ulnar artery signal on the left with a positive compression test result. The finger brachial pressure indices were also unremarkable. Arch angiography with left upper extremity runoff revealed a small PCHA aneurysm that occluded with abduction of the arm. Both the radial and

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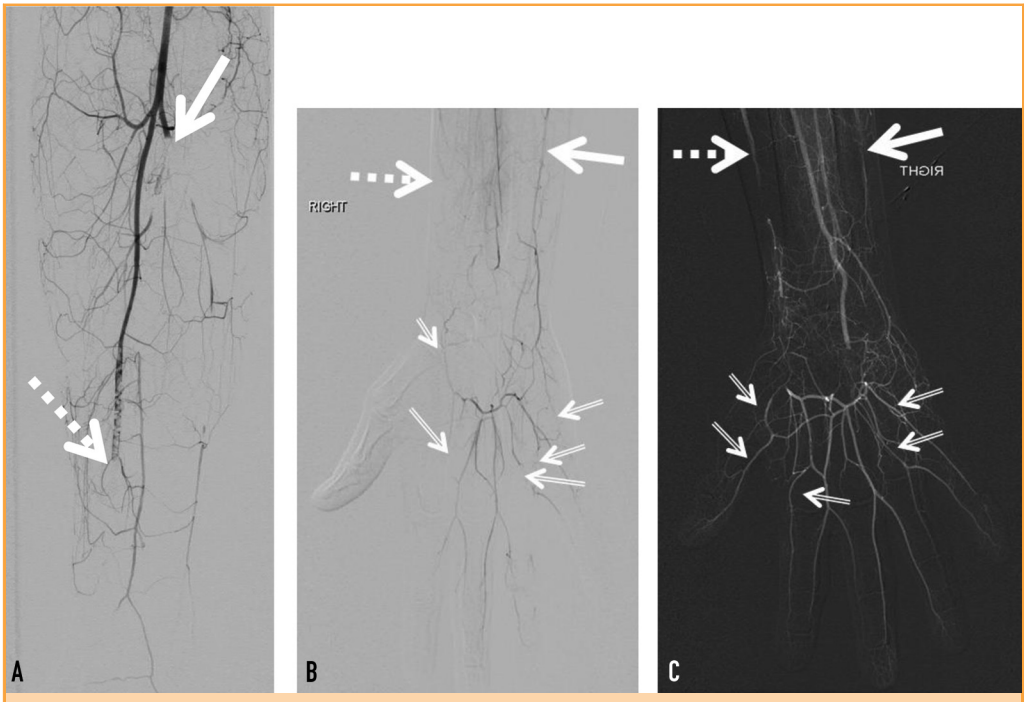

FIGURE 2. Angiography of the right hand for patient 1. A, Angiogram of the right forearm on day 1 demonstrates occlusion of the proximal ulnar artery (solid arrow) and proximal interosseous arteries with distal reconstitution. The radial artery occlusion (dashed arrow) begins in the mid forearm. B, Angiogram on day 1 demonstrates poor or absent contrast filling of the ulnar (solid arrow) and radial (dashed arrow) arteries, as well as common digital arteries (smaller double arrows) due to embolization. C, Angiogram after surgical intervention 7 days after the first angiogram (panels A and B) demonstrates overall improved flow into the hand with partial revascularization of the ulnar (solid arrow) and radial (dashed arrow) arteries, as well as common digital arteries (smaller double arrows).

ulnar arteries were occluded at the carpometacarpal joint, with multiple palmar arch and digital artery occlusions.

Surgical exploration involved resection of a small PCHA aneurysm and ligation of the PCHA, with no embolectomy. She recovered well and was discharged on antiplatelet treatment alone (325 mg aspirin daily). At 12 months, her symptoms had completely resolved and she had returned to playing competitive volleyball.

Patient 3: vQSS Due to PCHA Thrombosis and Distal Embolism

A 26-year-old right-handed female professional volleyball player presented with a 2-month history of right posterior shoulder pain, arm numbness, and paresthesias in digits 2 through 5 with associated pallor of the right hand. There was no history of nocturnal pain or ulceration. She was a nonsmoker without any risk factors for a hypercoagulable state. Examination revealed cool, pale right hand and digits with markedly delayed capillary refill and splinter hemorrhages.

Vascular laboratory studies reported radial, ulnar, and digital occlusive disease. Arch angiography with right upper extremity runoff revealed chronic-appearing thrombus involving both the PCHA and the anterior circumflex humeral artery (ACHA). Although the brachial artery was normal, there was partial occlusive embolus in the proximal ulnar artery, with complete occlusion distally. Occlusion of the distal radial artery was noted. There was also occlusion of the digital arteries of the second digit and thumb.

Thrombolysis was terminated at 24 hours with no improvement. Therapeutic anticoagulation was begun and continued until just

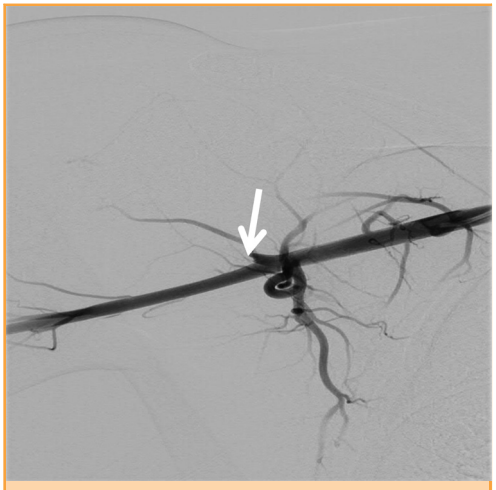

FIGURE 3. Angiography of the right shoulder for patient 1 showing abrupt occlusion of the PCHA (arrow) caused by thrombus. PCHA = posterior circumflex humeral artery.

before operation. During surgery, a PCHA pseudoaneurysm was ligated. The ACHA was noted to be normal. At 12 months, she had mild cold intolerance in digits 1 through 5 of the right hand and had successfully returned to competitive volleyball.

Patient 4: nQSS With Concomitant PCHA Compression

A 27-year-old right-handed active-duty male military sailor presented with a 6-month history of position-dependent right shoulder pain and upper arm twitching. The inciting activities included lifting the arm overhead and complete flexion of the arm above the shoulder. There was a history of antecedent trauma in the setting of performing fly dumbbell weight-lifting exercises. Examination was remarkable for tenderness to palpation of the right posterior shoulder. No atrophy of the shoulder muscles was noted, though decreased strength was present on the right, particularly with shoulder abduction. In addition, greater than 90° abduction of the shoulder produced fasciculations in the posterior deltoid.

Magnetic resonance imaging (MRI) of the right shoulder revealed no masses occupying the spinoglenoid notch or the QS. There was no substantial atrophy of the shoulder girdle musculature including the deltoid and teres minor. Electromyography (EMG) of the right upper extremity revealed chronic or old denervation to the right posterior deltoid. Empirical ultrasound-guided local anesthetic injection of the QS relieved symptoms. Right upper extremity angiogram revealed dynamic PCHA occlusion during abduction of the shoulder.

During surgery, exploration of the axillary nerve and neurolysis of a surrounding thick fascial scar all the way to the anterior margin were performed, with subsequent symptom improvement. At 12 months, he had almost complete resolution of symptoms and returned to full activity including weight training, running, and swimming.

Patient 5: nQSS With Concomitant PCHA Compression

A 33-year-old right-handed male facilities manager and recreational racquetball player presented with chronic right posterior shoulder pain and occasional "popping." Examination revealed no atrophy of the scapulothoracic or shoulder girdle muscles. He had posterior shoulder pain with anterior apprehension (passive AER) and was tender over the QS. Radial pulses were normal with no splinter hemorrhages.

Radiographs and MRI arthrogram of the shoulder, and a cervical MRI, were unremarkable. Physical therapy for rotator cuff and scapular-strengthening exercises and subacromial and scapulothoracic bursal corticosteroid injections provided no relief. A diagnostic shoulder arthroscopy was also unremarkable. Electromyography revealed increased insertional activity in the deltoid muscle, and angiography was performed to evaluate for PCHA compression with AER. The study showed dynamic compression of the PCHA with AER, without distal filling.

Open exploration of the QS revealed the axillary nerve and PCHA to be heavily entrapped by fibrous bands. Lysis of these bands was performed, and 1 month later he reported complete resolution of symptoms.

Patient 6: nQSS With Concomitant PCHA Compression

A 45-year-old right-handed female housekeeper presented with right shoulder pain

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aggravated by reaching overhead. Examination revealed posterior shoulder pain in the overhead position, marked QS tenderness, and a positive dynamic labral shear test result.

A noncontrast MRI of the right shoulder revealed a posterior labral tear with a large paralabral cyst, and atrophy of the teres minor muscle. Electromyography of the deltoid and teres minor muscles was unrevealing. An arteriogram showed dynamic occlusion of the PCHA with the arm in AER, with retrograde filling of distal branches from collaterals.

Surgical treatment was pursued, exposing the QS. Thick fibrous bands crossing between the teres minor and the teres major were resected. The axillary nerve was identified and neurolysis was performed. Arthroscopic debridement of the paralabral cyst and posterior labral repair were also performed. She underwent physical therapy for shoulder strengthening, and she had resolution of her shoulder pain.

Patient 7: nQSS With Concomitant PCHA Compression

A 45-year-old male heavy machinery operator presented with several months of posterior left shoulder pain aggravated by placing his arm in AER. Examination revealed tenderness over the posterior aspect of the glenohumeral joint, as well as reproduction of his pain with AER.

Radiographs of the left shoulder were unremarkable. An MRI scan of the left shoulder revealed atrophy of the teres minor muscle. Electromyography was performed, which revealed reduced amplitude motor responses of the axillary nerve by nerve conduction studies and decreased recruitment and large units in both the deltoid and teres minor. There was no evidence of cervical radiculopathy. An arteriogram revealed mild dilation of the proximal PCHA with the arm in the neutral position (possibly related to chronic compression distal to this area). With the arm in AER, compression of the PCHA with a distinct narrowing was identified.

The patient is awaiting surgical decompression.

Patient 8: nQSS With Concomitant PCHA Compression

A 68-year-old right-handed female administrative assistant presented with bilateral anterior shoulder pain. Physical examination findings were suggestive of rotator cuff pathology, with no posterior tenderness in either shoulder and no posterior shoulder pain with AER.

An MRI of both shoulders revealed tendinosis of the rotator cuff tendons, as well as a subluxed long head of the biceps tendon. Isolated grade 4/4 atrophy of the teres minor muscle in the left shoulder was also noted. Electromyography revealed no changes in the deltoid muscle. Arteriogram revealed PCHA compression with maximum AER.

She was given a corticosteroid injection in the bursal space above the shoulder and started on physical therapy for rotator cuff tendinosis. On follow-up, she had persistent left shoulder pain and had unfortunately been diagnosed with invasive breast cancer. She has not yet returned for further evaluation or treatment of the left shoulder.

Patient 9: nQSS With Concomitant PCHA Compression

A 45-year-old left-handed female nurse presented with progressive pain in the posterior right shoulder aggravated by AER. Examination revealed marked QS tenderness and considerable posterior shoulder pain with passive AER.

Magnetic resonance imaging of the right shoulder was unremarkable, with no teres minor atrophy. Angiogram showed compression of the PCHA with AER. Electromyography revealed enlarged motor unit potentials in several muscles innervated by C5 (deltoid, mid cervical paraspinal, and rhomboid muscles), suggesting a C5 cervical radiculopathy. Magnetic resonance imaging of the cervical spine revealed right foraminal stenosis at C3 through C6.

She was seen by colleagues in Neurosurgery, who recommended conservative measures. She underwent physical therapy, multiple nerve root injections, and 3 right cervical foraminotomies, with improvement in her neck pain and some of her upper extremity paresthesias. However, there was no improvement in her posterior shoulder pain aggravated by AER. She will undergo surgical decompression of the QS.

DISCUSSION

This report provides 9 cases of QSS (Table). Patient cases 1 to 3 (vQSS) involved PCHA

TABLE. Distribution of Published Quadrilateral Space Syndrome (Neurogenic and Vascular) Cases[a]

Demographic characteristic	The Mayo experience (n=9)	Reviewed/ previously published (n=76)
Year		
1980-1989	0 (0)	22 (29)
1990-1999	0 (0)	24 (32)
2000-2014	9 (100)	30 (39)
Neurogenic	6 (67)	46 (61)
Sex		
Female	6 (67)	6 (13)
Male	3 (33)	18 (39)
Unknown	0 (0)	22 (48)
Etiology		
Structural	6 (100)	44 (96)
Not reported	0 (0)	2 (4)[b]
Overhead athletes		
Baseball	0 (0)	2 (4)
Swimmer	0 (0)	1 (2)
Triathlete	0 (0)	1 (2)
Vascular	3 (33)	30 (39)
Sex		
Female	3 (100)	0
Male	0 (0)	26 (87)
Unknown	0 (0)	4 (13)
Etiology		
Mechanical	3 (100)	30 (100)
Overhead athletes[c]		
Volleyball	3 (100)	16 (53)
Baseball	0 (0)	12 (40)
Trapeze	0 (0)	1 (3)
Yoga	0 (0)	1 (3)

[a]Values are No. (%).
[b]Two neurogenic cases did not report surgical exploration to assess for structural causes.
[c]Overhead athletes = any patient engaged in elite, collegial, professional, or dedicated sport or activity involving repetitive abduction and external rotation of the arm above the head.

thrombosis with distal embolism. No QS fibrous bands were noted in their operative reports. Six cases involved axillary nerve injury (nQSS). Half of the neurogenic cases had no surgical confirmation and were diagnosed on the basis of clinical symptoms and imaging results. Patient 4 had fascial scarring surrounding the axillary nerve due to history of QS trauma. Fasciculations of the deltoid muscle in this fourth patient were noted on presentation, which is rarely reported in the literature. Patient 5 had thick fibrous bands compressing the axillary nerve. Patient 6 also had fibrous bands, as well as a large paralabral cyst. Patients 7 to 9 await surgical decompression and neurolysis if symptoms do not resolve, possibly owing to fibrous bands within the QS.

We suspect that the varied phenotypes with findings of compression of the axillary nerve vs thrombotic occlusion of the PCHA with distal embolization may be due to fundamental differences between underlying causes of the 2 types of QSS. Comprehensive literature review suggests that the difference lies in whether QSS is caused by (1) continuous mechanical trauma to the PCHA with resulting thrombosis due to repetitive AER movement in elite athletes such as volleyball and baseball players (patients 1-3) vs (2) the presence of fixed structural entities (eg, fibrous bands and paralabral cysts) that serve as space-occupying lesions compressing the axillary nerve (and PCHA without thrombosis) (patients 4-6 and possibly 7-9) (Table).

We therefore propose 2 categories of QSS in which symptoms are described under vQSS or nQSS, similar to the classification of thoracic outlet syndrome (TOS),[30] although of different mechanistic etiology. Quadrilateral space syndrome would therefore be described as a neurovascular syndrome that leads to multivariate pathology including peripheral nerve compression in nQSS and artery aneurysm and/or thrombosis with or without distal embolization in vQSS.[2,4,5,31-33] Formalizing a classification system will aid in recognizing and diagnosing QSS more consistently. This is in response to its prevalence, yet underdiagnosis, as a neurovascular injury particularly in "overhead" athletes.[19]

The vQSS Entity

Mechanism of PCHA Injury: Repetitive Mechanical Stress During AER. The mechanism of thrombosis of the PCHA in vQSS involves repetitive trauma to the PCHA wall during AER. Durham et al[34] describe this as a repetitive tension effect that stretches the PCHA as it travels through the tight QS and winds around the neck of the humerus, analogous to a taut stretched rubber band leading to intimal injury and weakening of the vessel wall (Figure 4, inset circle A). The repetitive pulley movement could lead to turbulent blood flow within the PCHA (Figure 4, inset

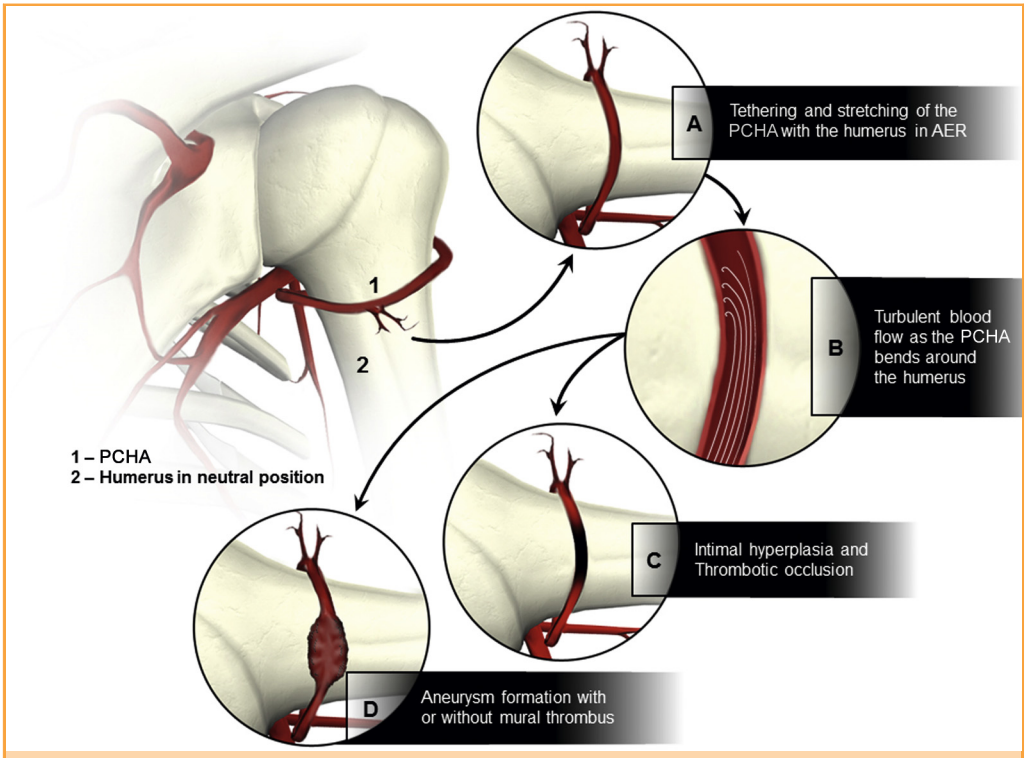

FIGURE 4. Proposed mechanism for PCHA aneurysmal degeneration and thrombosis: the PCHA stretches around the neck of the humerus analogous to a taut stretched rubber band leading to intimal injury and weakening of the vessel wall (inset circle A); the repetitive pulley movement likely leads to turbulent blood flow within the "bend" PCHA around the humeral neck (inset circle B); vessel injury from these changes leads to subsequent aneurysmal degeneration and thrombosis (inset circles C and D). AER = abduction and external rotation; PCHA = posterior circumflex humeral artery.

circle B). This is consistent with findings in arterial model studies that "bends" in arteries and other tubular structures associate with turbulent flow and atherosclerotic deposits.[35] Turbulent blood flow induces changes in nonlaminar shear stress,[36,37] which stimulate vascular endothelial cells to release varying amounts of prostacyclin, calcium, thrombomodulin, transforming growth factor-beta, and other regulators of thrombosis, coagulation, and smooth muscle cell proliferation in the vessel wall (see reviews 38-40). Vessel injury from these changes leads to subsequent aneurysmal degeneration and thrombosis[8,15,28,29,38] (Figure 4, inset circles C and D). The PCHA also undergoes repetitive taut stretching near its origin from the axillary artery before looping around the neck of the humerus.[41] This leads to aneurysms near the origin of the PCHA in the QS.[42] Thus, intimal injury to the PCHA is multifactorial, because of a combination of tethering at the axillary artery and turbulence at the humeral neck, while stretching and sliding during AER. Digital ischemia or "limb angina" due to embolic occlusion ensues from extrusion of the thrombus squeezed from the artery under pressure during repetitive AER.[8,11,25,28,29] Thrombus compression can also lead to retrograde embolism in proximal regions of the axillary artery upstream of the PCHA,[42] as is possibly the case in patient 1. The Table shows that professional or collegiate overhead athletes, such as patients 1 to 3, are more likely to develop vQSS.

PCHA Response to Fixed Extrinsic Compression. Extrinsic PCHA compression in the QS by space-occupying lesions such as thick fibrous bands in the absence of repetitive

mechanical strain does not appear to have marked clinical sequelae. This is likely due to an anastomotic blood supply to the deltoid and teres minor muscles and to the proximal humerus from 3 main arteries: the PCHA, the ACHA, and the thoracoacromial artery.[43,44] This is consistent with the absence of any reports of space-occupying lesions or fibrous bands in any of the vQSS cases.

Clinical Signs. Vessel thrombosis and distal embolization can present with coolness, cold intolerance, pallor, and cyanosis of the upper extremity digit, with or without splinter hemorrhages[7,29] (Figure 1).

Differential Diagnosis. Quadrilateral space syndrome can mimic a number of other neurovascular disorders (Supplemental Table, available online at http://www.mayoclinicproceedings.org), including those that involve thrombosis or aneurysm formation in other more proximal branches of the axillary artery,[21,28,41] such as in arterial TOS.[30] In arterial TOS, the subclavian artery is compressed between the clavicle and the first rib or anatomic abnormalities such as a cervical rib or fibrous band.[30] In hypothenar hammer hand syndrome, the ulnar artery is injured by repeated impact against the hamate bone in the wrist.[9,11] Both these conditions may cause intimal damage, aneurysm formation, thrombotic occlusion, and distal embolization of thrombus with ischemia of the hand. They may be diagnosed with ultrasound, digital subtraction angiography (DSA), CTA, or magnetic resonance angiography (MRA). Other causes of ischemic fingers such as emboli from the heart, scleroderma, vasculitis, atherosclerosis, and thromboangiitis obliterans should also be considered[41] (Supplemental Table).

Diagnostic Imaging. Imaging reveals fixed PCHA occlusion due to thrombosis with or without distal embolization[7,29] on DSA,[10] CTA,[7,25,29] or MRA[10] (Figure 1). Digital subtraction angiography provides superior spatial resolution and remains the best imaging modality for the small arteries of the hand. It has the added advantage of being able to proceed immediately with catheter-directed thrombolysis if acute thrombus is present.

Treatment. If untreated, QSS can disable the overhead or throwing athlete and threaten the patient's career and viability of the involved parts.[31,45] Thrombolysis can be used in the case of acute thrombus, although this yields limited results.[28,29] Unfortunately, chronic thrombus is often present at the time of diagnosis and will not respond well to thrombolysis. Surgery is the most definitive therapy. The pathological portion of the PCHA is ligated to prevent further embolus propagation from aneurysmal thrombus[7,10,25,28,29]; thrombectomy is also performed for distal emboli (Figure 1).[8,28] Aneurysm resection[13] and endovascular treatment with coiling[8,14,15] have also been reported. Many athletes return to practice after operation.[7,10,29] This could be supplemented by the administration of anticoagulants for 3 to 6 months; there is no clear indication for the use of antiplatelet agents.

The nQSS Entity

Mechanism of Axillary Nerve Injury: Fixed Extrinsic Compression. Various fixed anatomic anomalies can lead to nQSS, with fibrous bands being the most common.[2,5,23,32,46-51] In cadaveric shoulder dissections, fibrous bands were located between the teres major and the long head of the triceps; external rotation reduced the cross-sectional area of the QS.[2] The fibrous band often results from overt or occult repeated microtrauma to connective tissue in the QS with the formation of permanent scarring and adhesions. These fibrous bands can sometimes occur in recreational athletes such as swimmers or racquetball players (eg, patients 4 and 5). The Table shows that overhead athletes indeed can rarely present with nQSS.[17,18,23,24] This is likely after having experienced microtears to the connective tissue within the QS, with the formation of thickened fibrous bands early on in or even before beginning their career. In these athletes, this precedes the opportunity for eventual thrombosis or aneurysm of the PCHA with distal embolization. The nQSS can also result from hypertrophy of the muscular boundaries,[5,18] an atypical nerve course,[52] bone spikes,[53] or space-occupying lesions, such as glenoid labral cysts,[54,55] paralabral cysts,[3] ganglia,[56] fracture hematoma, or

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humeral osteochondroma.[57] Chronic fixed nerve compression permanently displaces internal nerve contents in transverse and longitudinal dimensions, leading to long-term damage of axons and myelin.[58]

Axillary Nerve Response to Repetitive Mechanical Stress During AER. Interestingly, the axillary nerve does not appear to sustain injury from the mechanical strain that leads to vQSS. Several authors suggest that mechanical properties of individual layers of peripheral nerves may help determine peripheral nerves' excursion and strain responses to stretch about a joint.[58,59] Excursion describes gliding of the axillary nerve relative to its nerve bed; strain is the change in axillary nerve length induced by longitudinal tensile stress.[58] Dynamic elongation of the nerve under tensile force is counteracted by transverse contraction of the nerve, which is reversible as the tensile force is relieved.[58] This provides physiological changes that allow for nerve lengthening by 6% to 8% for short durations, which seems to fit stretch ranges tolerated by individuals when experimentally probed.[58] Georgeu et al[59] suggest that the epineurum or nerve sheath, which has axial or circumferential collagen fibers, withstands more force than it transmits to the endoneurum or nerve core, which has longitudinal collagen fibers interspersed with neurons. In simplified analyses, the perineurum or interface between the nerve sheath and the core provides a shear plane that facilitates independent movement of the core (endoneurum) and the sheath (epineurum).[58,59] We suspect that the axillary nerve sheath and interface (or shear plane) protect the core from injury that could otherwise be induced by stretch involved in the pulley system that occurs during AER.

Clinical Signs. Patients with muscle atrophy occurring from nQSS present with paresis, paresthesia, poorly localized shoulder pain,[2] or tenderness with palpation of the anterior,[26,51] lateral,[60] or most often posterior[27] shoulder (over the QS[2]), corresponding to deltoid and teres minor muscle fibers (Figure 1). There is often radiation of paresthesias to the arm or the forearm in a nondermatomal distribution.[26] Fasciculation of the long head of the triceps has also been identified.[23]

Differential Diagnosis. A differential diagnosis for nQSS is presented in the Supplemental Table and includes suprascapular nerve entrapment and complex regional pain syndrome,[61] overt shoulder trauma,[62,63] and iatrogenesis.[64-69] C5 and/or C6 radiculopathy should also be ruled out, as found in the case of patient 9. Axillary nerve pathology can overlap with C5/C6 radiculopathy because the axillary nerve arises from the C5 and C6 nerve roots.[20]

Diagnostic Imaging. Imaging reveals dynamic PCHA occlusion during AER maneuvers, due to extrinsic compression, by DSA,[47] CTA,[26,70] MRA,[47] or ultrasonography[51] (Figure 1). The accuracy of vascular studies alone for confirming the diagnosis of nQSS is unclear. In an ultrasound study, 16% of asymptomatic volunteers exhibited PCHA stenosis during AER.[71] In contrast, Mochizuki et al[47] found PCHA occlusion in 80% of asymptomatic volunteers on MRA during AER. This dichotomy may be due to different imaging techniques in different patient populations, or MRA may be less specific for diagnosing nQSS. Teres minor atrophy on MRI in the absence of nQSS symptoms (Figure 1) is also nonspecific,[48,52,54,72-74] and can result from variations in the length and origin of the axillary nerve[52] or from normal variant fascial slings "tenting" the course of the axillary nerve[75] (Supplemental Figure 3, available online at http://www.mayoclinicproceedings.org). Limited data exist on EMG sensitivity after vascular imaging studies suggestive of nQSS (Figure 1)[26] because of technical difficulty with needling the teres minor. Accuracy may improve with ultrasound guidance.[27,51] QSS diagnosis therefore requires correlation of the clinical scenario and imaging results (Figure 1).

Treatment. Conservative treatment options include oral anti-inflammatory medications, physical therapy, and limiting inciting activities. Surgical decompression involves neurolysis and excision of fibrous bands or other space-occupying lesions[5,17,26,32,45,46,49,50,60] (Figure 1). Surgically treated patients return

to full activity without pain or limitation after several weeks.[5,17,29,32,45,46,49,50,60]

Common Management Considerations

Noninvasive screening during medical assessments of professional athletes in whom repeated trauma to the arm or hand is likely, such as volleyball players, has been postulated,[16,29] but remains controversial. Given the high prevalence of incidental teres minor denervation[72,73] and incidental PCHA occlusion with dynamic maneuvers,[47] screening overhead athletes may lead to a myriad of false positives based on imaging alone.[29,41] A screening questionnaire[7] could be used to triage which players are candidates for diagnostic imaging, although a high prevalence of self-reported symptoms might complicate this approach. Furthermore, all patients, particularly athletes presenting with symptoms consistent with ischemia or nerve compression, should be assessed for QSS (Figure 1). Timely diagnosis increases the likelihood of preserving the function of the affected limb.[61] Increasing familiarity with QSS and using an algorithm such as that given in Figure 1 will hopefully minimize delayed diagnoses and associated morbidity.[76] A high index of suspicion should be maintained, especially by health professionals who care for overhead athletes with repetitive AER.[13,41,61]

CONCLUSION

Quadrilateral space syndrome is a rare, yet potentially debilitating, entity. Figure 1 outlines an algorithm for diagnosing and treating QSS. Repetitive mechanical injury to the PCHA as it passes through the tight QS and wraps around the humeral neck during AER leads to vQSS, manifested as PCHA thrombosis and/or aneurysm with distal embolization and digital ischemia in elite athletes. Fixed structural impaction of the QS most often with space-occupying lesions or fibrous bands leads to nQSS, manifested by nondermatomal paresthesias and QS point tenderness.

Symptoms of nQSS have considerable overlap with other conditions that cause posterior shoulder pain, paresthesias, or weakness of the affected extremity. Severity can also vary, in that patients may have 1 or all 3 of these predominant symptoms. Unfortunately, given the low specificity of both vascular studies and electromyography in the absence of symptoms, the nQSS diagnosis should be made after excluding other conditions. In this case series of 9 patients treated at Mayo Clinic over the past decade, one-third of the patients presented with vQSS and the remaining two-thirds presented with nQSS. This suggests possibly a 2:1 prevalence of nQSS relative to vQSS. The Table catalogs a larger distribution of published nQSS[3-5,17,18,23,24,26,32,46-51,54-57,60,62] and vQSS[6-15,21,22,25,28,34,41,42] cases. Results from the Table support a 1.5:1 prevalence of nQSS relative to vQSS, with a male predominance (male/female ratio, 7:1) for all QSS, and an almost exclusive male population for vQSS (likely due to traditional sex distribution in overhead athletics). Imaging studies such as DSA, CTA, and MRA have been used to diagnose QSS, even though lacking complete specificity. The use of ultrasound has been studied as well. Magnetic resonance imaging and EMG are limited supportive methods. Posterior circumflex humeral artery ligation is used to surgically treat vQSS to prevent further distal embolization from the PCHA, along with thrombolysis for acute thrombosis. Anticoagulation might limit further clot formation, while the benefit of antiplatelet therapy is uncertain. Neurolysis and QS decompression are used to definitively treat nQSS. Physicians, particularly those treating elite athletes and those in vascular specialties, orthopedics, hand surgery, and neurology, should continue to pursue understanding of the recognition, differential diagnosis, discrimination, and prognosis of both nQSS and vQSS.

SUPPLEMENTAL ONLINE MATERIAL

Supplemental material can be found online at http://www.mayoclinicproceedings.org.

Abbreviations and Acronyms: **ACHA** = anterior circumflex humeral artery; **AER** = abduction and external rotation; **CTA** = computed tomography angiography; **DSA** = digital subtraction angiography; **EMG** = electromyography; **MRA** = magnetic resonance angiography; **MRI** = magnetic resonance imaging; **nQSS** = neurogenic quadrilateral space syndrome; **PCHA** = posterior circumflex humeral artery; **QS** = quadrilateral space; **QSS** = quadrilateral space syndrome; **TOS** = thoracic outlet syndrome; **vQSS** = vascular quadrilateral space syndrome

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