Arterial Thoracic Outlet Syndrome in the Competitive Athlete

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Abstract

Major neurovascular conditions of the upper extremity are relatively rare and often difficult to recognize in competitive overhead athletes and those involved in frequent throwing or weight lifting regimens. Understanding these conditions is particularly important for vascular and thoracic surgeons caring for athletes, as well as orthopedic surgeons, emergency room physicians, and sports medicine specialists, because they can seriously limit athletic performance and may even have limb-threatening consequences. Some of the most significant upper extremity neurovascular disorders are related to compression of the brachial plexus nerves or the axillary and subclavian vessels, and are therefore considered different forms of thoracic outlet syndrome (TOS). In this chapter, current strategies for the diagnosis and treatment of arterial TOS are highlighted, both to avoid serious complications and to promote successful treatment outcomes, including arterial TOS involving the subclavian artery, compressive lesions of the axillary artery and its branches, and some forms of the quadrilateral space syndrome. Although the treatment options to be considered for arterial TOS are similar for all patients, competitive athletes often present special circumstances that need to be taken into account in the decision-making process. With early recognition, proper initial treatment, and definitive surgical care, most competitive athletes affected by arterial TOS can return to previous levels of performance.

Critical Take-Home Points

1. Overhead athletes with arterial disease of the upper extremity often present with unilateral digital ischemia from emboli or vasospasm, and may exhibit diminished upper extremity blood flow with arm elevation that can be identified by contrast-enhanced imaging studies.
2. Arterial TOS is characterized by subclavian artery pathology that can cause thromboembolism and digital ischemia (aneurysms or occlusive lesions) in association with a bony abnormality, such as a cervical rib.
3. Other localized thromboembolic lesions in athletes include compression of the axillary artery and posterior circumflex humeral artery associated with repetitive vigorous overhead activity.

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89.1 Introduction

Major neurovascular conditions of the upper extremity are relatively rare and often difficult to recognize in competitive overhead athletes and those involved in frequent throwing or weight lifting regimens. These conditions are nonetheless quite important, because they can seriously limit athletic performance and may even have limb-threatening consequences [1, 2]. Some of the most significant upper extremity neurovascular disorders are related to compression of the brachial plexus nerves or the axillary and subclavian vessels, and are therefore considered different forms of thoracic outlet syndrome (TOS), including arterial TOS involving the subclavian artery, compressive lesions of the axillary artery and its branches, and some forms of the quadrilateral space syndrome [3]. In this chapter, current strategies for the diagnosis and treatment of arterial TOS are highlighted, both to avoid serious complications and to promote successful treatment outcomes. In most cases, early recognition, proper initial treatment, and comprehensive surgical care can allow the athlete to successfully return to previous levels of performance.

89.2 Clinical Recognition

One of the most common presenting findings in patients with arterial TOS is unilateral digital ischemia, with symptoms including numbness, tingling, cold and painful sensations, cyanotic or pale discoloration, and delayed capillary refill in the fingers [4]. The brachial, radial and/or ulnar pulses may be absent or decreased in the presence of a proximal arterial occlusion, with diminished blood pressure. Digital ischemia may also exist with normal radial and ulnar pulses if the site of obstruction is solely within the hand, which can occur with digital artery embolism from a more proximal site or with digital artery thrombosis secondary to local trauma. Digital ischemia usually coexists with and is exacerbated by local vasospasm, and in some circumstances, primary vasospasm can also result in digital ischemia in the absence of arterial thrombosis or embolism.

The presence of digital ischemia requires differentiation between proximal and distal arterial sources of thromboembolism, localized digital artery occlusion, and primary vasospasm (see Chap. 95) [4]. In most cases of upper extremity embolism a proximal artery source must be considered, which is best evaluated by contrast-enhanced arteriography, with positional views of the neck and upper arm and high-resolution views of the hand. This may also be accomplished with contrast-enhanced computed tomography (CT) or magnetic resonance (MR) imaging, but catheter-based transfemoral arteriography remains the most accurate and definitive approach [5].

89.3 Arterial TOS: Subclavian Artery

Arterial TOS is the least frequent form of TOS, representing only 1–3% of patients in most clinical series [6]. This condition is characterized by pathological changes in the subclavian artery resulting in either thrombosis or aneurysm formation, in associated with a bony abnormality, such as a cervical rib (Fig. 89.1). Positional compression of the subclavian artery during arm elevation is a frequent finding on physical examination, as well as on vascular laboratory testing and vascular imaging studies. However, in the absence of actual arterial pathology with the arm at rest, this finding does not represent a source of clinical symptoms and is not considered to reflect arterial TOS. Indeed, positional compression of the subclavian artery is frequently misunderstood when encountered in a patient with upper extremity symptoms that are more accurately attributable to neurogenic TOS [3, 6].

Subclavian artery aneurysms arise from compression at the level of the first rib with poststenotic dilatation and arterial wall degeneration, leading to ulceration, mural thrombus, and distal embolization. Once suspected, anticoagulation and anti-platelet therapy should be started to limit the extent of arterial thrombosis. Contrast-enhanced arteriography is then required to image the extent, location and character of arterial

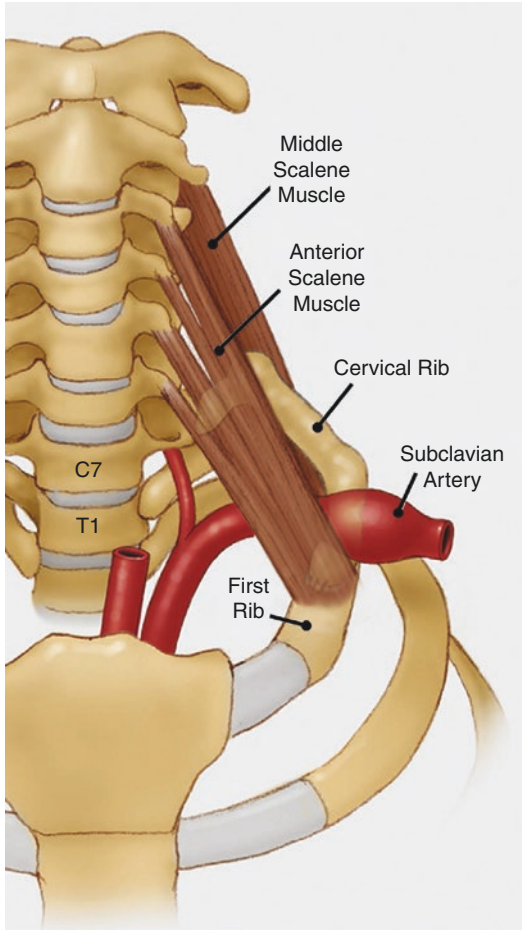

Fig. 89.1 Arterial TOS with subclavian artery aneurysm. A cervical rib originating from the transverse process of C7 is inserting into a normal first rib, compressing the subclavian artery between the rigid cervical rib and a tight anterior scalene muscle. This stenosis of the subclavian artery causes poststenotic dilation and aneurysm formation. Adapted from Illig et al. [3], with permission from Elsevier.

occlusions and to detect the presence of subclavian artery compression and aneurysm formation. Although intra-arterial thrombolytic therapy may have a role in helping to resolve acute distal thromboembolism, this approach is better used in conjunction with or after definitive operative treatment.

Surgical treatment for subclavian artery aneurysms consists of supraclavicular thoracic outlet decompression with scalenectomy and removal of the cervical and first ribs, followed by excision of the aneurysmal segment of subclavian artery [6–8]. In some cases, a second infraclavicular incision is needed to obtain distal arterial control at the level of the axillary artery. Interposition bypass graft reconstruction is performed to restore flow through the subclavian artery. Following reconstruction of the subclavian artery, intraoperative contrast arteriography may be used to define the extent and distribution of any distal emboli, and thromboembolectomy performed as necessary to clear the involved distal vessels to the extent feasible. When there is extensive digital artery embolism beyond the capabilities of surgical thrombectomy, adjunctive intra-arterial infusion of vasodilator and thrombolytic agents may help achieve optimal outcomes.

We have reviewed our experience with patients undergoing primary or reoperative treatment for arterial TOS over an 8-year period [6]. This series involved 40 patients with a mean age of 40.3 ± 2.2 years (range, 13–68 years), with over half presenting with upper extremity ischemia/emboli (n = 21) or posterior stroke (n = 2) and 8 that had required urgent brachial artery thromboembolectomy. There were also 17 (42%) patients with a non-vascular presentation, with 11 having neurogenic TOS and 6 having an asymptomatic neck mass or incidentally discovered subclavian artery dilatation. All 40 patients underwent thoracic outlet decompression, of which 75% had a cervical rib (24 complete, 6 partial), 5 had a first rib abnormality, and 4 had a clavicle fracture. Subclavian artery reconstruction was performed in 70%, but 30% had only mild arterial dilatation (<100%) that required no direct arterial reconstruction. Subclavian artery patency was 92% during mean follow-up of 4.5 ± 0.4 years, no patients had further dilatation or embolism, and chronic symptoms were present in 6 (4 post-ischemic/vasospasm, 2 neurogenic). Based on this review we concluded that early thoracic outlet decompression and surgical treatment of subclavian artery aneurysms, with restoration of direct arterial flow to the hand and digits, can lead to excellent results and a full return to function over a period of several months. Unfortunately, due to the insidious clinical presentation of subclavian aneurysms and delays in

surgical treatment, many patients with this condition exhibit residual symptoms secondary to hand or digital thromboembolism that cannot be easily resolved.

89.4 Arterial TOS: Axillary Artery and its Branches

There is a unique group of vascular lesions that appear to occur almost exclusively in elite overhead athletes, such as baseball pitchers and volleyball players [1, 2, 9–11]. These lesions involve the third portion of the axillary artery and the branch origins of the subscapular artery (SA), anterior circumflex humeral artery (ACHA), and posterior circumflex humeral artery (PCHA), which arise from the axillary artery just beyond the level of the pectoralis minor muscle. In combination with anatomic fixation of the axillary artery underneath the pectoralis minor muscle and by the fascia at the location of the subscapular and circumflex humeral branch vessel origins, these lesions are caused by arterial compression and stretching by the head of the humerus as it moves forward during the extremes of arm elevation and extension, as seen in the overhead pitching motion, even without overt shoulder joint subluxation (Fig. 89.2) [12]. The response to repetitive axillary artery injury at this location can cause intimal hyperplasia and arterial stenosis, with subsequent thrombotic occlusion, or disruption and aneurysmal degeneration, with the formation of mural thrombus and distal thromboembolism [1, 2, 4, 9–11]. Axillary artery compression may also cause arterial dissection, which may extend proximally or distally to occlude additional segments of the circulation. Lastly, repetitive arterial injury affecting the

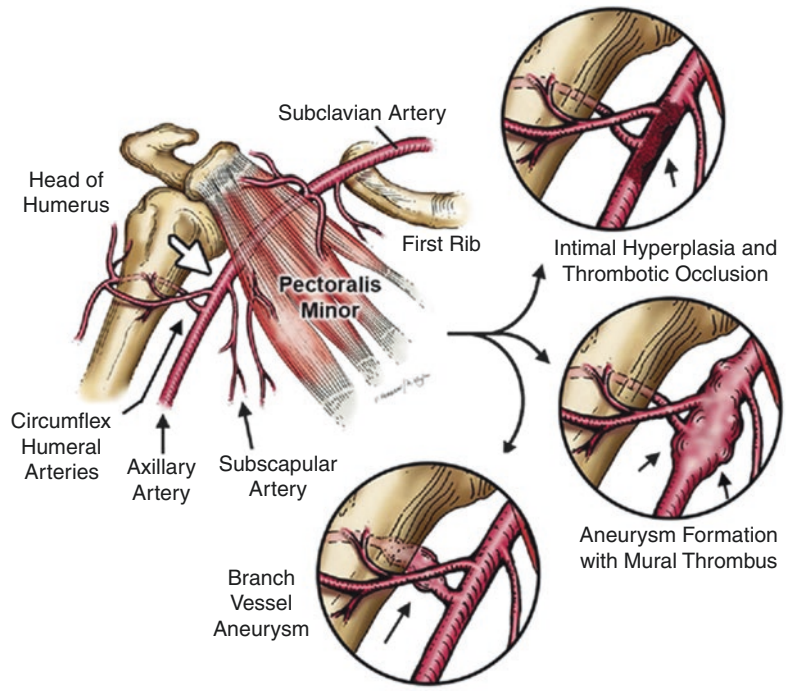

Fig. 89.2 Pathogenesis of compressive axillary artery lesions. Compression of the third portion of the axillary artery by anterior displacement of the humeral head during the overhead throwing motion. The axillary artery is relatively fixed in position at this site by the overlying fascia, branch vessel origins, and the pectoralis minor muscle. Repetitive compression can lead to intimal hyperplasia with stenosis, or aneurysm formation, with or without thrombotic occlusion. Similar lesions may also arise in the adjacent axillary artery branches (subscapular and circumflex humeral arteries). Reproduced from Duwayri et al. [11], with permission from Elsevier.

first several centimeters of the branch vessels at this location, likely due to stretch of the branch vessel origin adjacent to the axillary artery, typically leads to branch vessel aneurysm formation with thrombosis and distal embolism [11]. It is notable that this is distinct from lesions of the distal PCHA that occur in quadrilateral space syndrome, a separate condition (see below).

Exertional arm fatigue, digital cutaneous microemboli, and/or acute hand/digital ischemia are the presenting symptoms typically caused by lesions of the axillary artery and its branches [9, 11–15]. Like subclavian artery aneurysms, initial treatment includes anticoagulation and diagnostic vascular laboratory tests to evaluate arterial flow to the arm, with imaging studies being required to visualize the site, extent and character of the arterial lesion. This may be accomplished with either CT or MR angiography, but direct catheter-based arteriography is preferred [11]. If the artery is not completely occluded during arteriography at rest, overhead elevation of the arm should be used to determine if positional obstruction occurs at this location.

Medical management with thrombolysis, anticoagulation and/or antiplatelet therapy cannot assure against the recurrence of distal emboli from mural thrombus, and thrombolysis with balloon angioplasty and/or stent placement is unsatisfactory, since this does not correct the underlying cause and may introduce the potential for additional complications. Thus, surgical treatment is recommended for all lesions of the axillary artery and its branches.

The preferred operative approach initially involves anterior exposure through an axillary and upper medial arm incision and complete mobilization of the affected portion of the axillary artery to prevent compression [11]. This includes mobilizing the axillary artery from the surrounding branches of the brachial plexus but does not usually require division of the pectoralis minor muscle. For lesions directly affecting the main axillary artery, the affected segment is usually excised and reconstructed with an interposition bypass, typically using a short reversed saphenous vein graft. It is preferable to include or reimplant at least one of the three axillary artery branch vessels during construction of the distal anastomosis of the bypass graft (usually the PCHA), to maintain optimal perfusion to the humeral head and shoulder girdle musculature. For aneurysms confined to the axillary artery branch vessels, ligation and excision of the affected branch is sufficient. Intraoperative completion arteriography should be performed to assess the distal circulation in the upper extremity, and localized thrombectomy may be needed in the brachial, radial or ulnar arteries to re-establish direct arterial flow to the palmar arch of the hand. When there has been thrombosis within the palmar arch or digital arteries, intraoperative administration of thrombolytic agents and vasodilators may be valuable adjuncts. Recovery from surgical treatment is typically complete within 6–12 weeks of operation and a full return to previous levels of function can usually be expected. Nonetheless, it is recognized that in some patients a full functional recovery may remain limited by persistent digital ischemia due to the existence of pre-existing chronic thromboembolism, similar to that occasionally observed in patients with subclavian artery aneurysms.

We have previously reviewed the outcomes of surgical treatment for axillary artery pathology in a series of nine high-performance overhead athletes, of whom seven were elite baseball pitchers, with a mean age of 30.9 ± 2.9 years [11]. At angiography and surgical exploration a mean of 2.5 ± 0.8 weeks after the onset of symptoms (range 1–8 weeks), 6 had occlusion of the distal axillary artery opposite the humeral head either at rest (three) or with arm elevation (three), one had axillary artery dissection with positional occlusion, and 2 had thrombosis of a PCHA aneurysm. Treatment included segmental axillary artery repair with saphenous vein ($n = 7$; 5 interposition bypass grafts and 2 patch angioplasties), ligation/excision of PCHA aneurysms ($n = 2$), and distal artery thrombectomy/thrombolysis ($n = 2$). The mean postoperative hospital stay was 3.8 ± 0.5 days and the time until resumption of unrestricted overhead throwing was 10.8 ± 2.7 weeks. At a median follow-up of 15 months, primary-assisted patency was 89% and secondary patency was 100%, and all nine

patients had continued careers in professional baseball. This led us to conclude that repetitive positional compression of the axillary artery can cause a spectrum of pathology in the overhead athlete, that prompt recognition of these rare lesions is crucial given their propensity toward thrombosis and distal embolism, and that full functional recovery can usually be anticipated within several months of surgical treatment.

89.5 Quadrilateral Space Syndrome: Distal PCHA

The quadrilateral space is a distinct anatomical area in the posterior shoulder bounded medially by the edge of the triceps muscle, laterally by the medial edge of the neck of the humerus, inferiorly by the tendon of the teres major and latissimus dorsi muscles, and superiorly by the teres minor muscle or the glenohumeral capsule (Fig. 89.3) [16, 17]. The axillary nerve and distal PCHA pass through this space, where they can be compressed by hypertrophied muscle, fascial bands, or fibrotic tissues, particularly with dynamic positioning of the arm during overhead elevation. The clinical presentation of quadrilateral space syndrome most frequently involves sensory and motor findings related to the axillary nerve, including poorly localized neuropathic pain and weakness in the posterior shoulder and deltoid muscle, often radiating into the arm, tenderness over the posterior shoulder, and numbness in a discrete area over the deltoid ("regimental patch") [16–20]. When the PCHA adjacent to the axillary nerve is also compressed, arterial findings can include aneurysmal dilatation or thrombosis, potentially leading to thromboembolic complications in the upper extremity. Quadrilateral space syndrome is therefore part of the differential diagnosis of thromboembolism in the competitive overhead athlete [21].

Diagnosis of quadrilateral space syndrome is made by exclusion of other more common conditions and clinical examination, along with adjunctive contrast-enhanced MR or CT imaging of the related shoulder musculature (to visualize associated teres minor and/or deltoid atrophy), angiography (to identify aneurysmal dilatation or thrombotic occlusion of the PCHA), and electrodiagnostic testing [19, 20]. Injection of local anesthetic into the quadrilateral space can also be used to assess the response of local tenderness at rest and during arm activity. For patients that have not responded to physical therapy and other

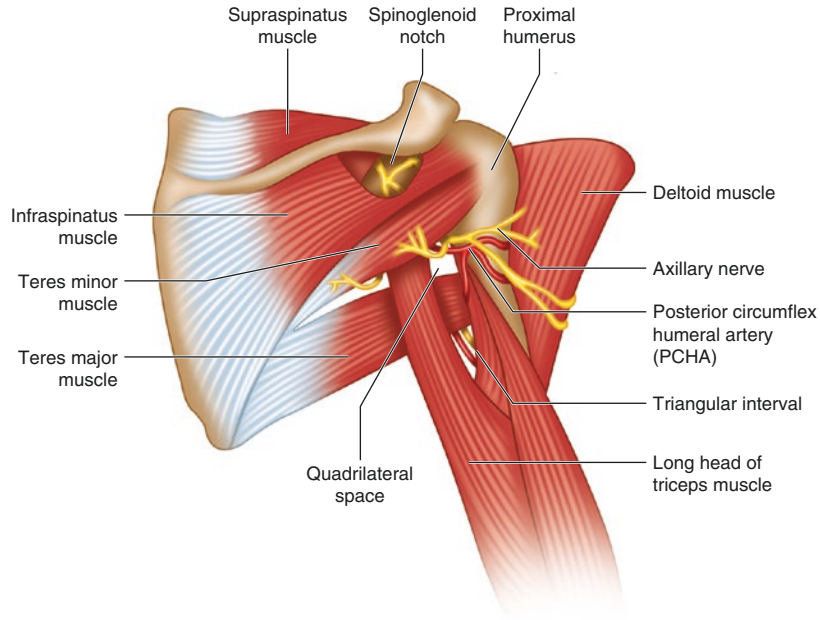

Fig. 89.3 Quadrilateral space syndrome. Anatomy of the quadrilateral space shown from a posterior view of the right shoulder, showing the quadrilateral space bounded superiorly by the teres minor muscle, inferiorly by the teres major muscle, medially by the long head of the triceps, and laterally by the proximal humerus. Reproduced from Flynn et al. [19], with permission from Elsevier

conservative measures, treatment of quadrilateral space syndrome is aimed at direct surgical decompression, most frequently through a posterior approach, to excise fascial and fibrous tissues along the path of the axillary nerve and PCHA [22]. When the PCHA is involved with aneurysmal degeneration or thrombosis, the vessel is ligated to prevent thromboembolism. This can usually be accomplished through the same posterior approach, taking care to obtain satisfactory proximal control of the PCHA as it enters the quadrilateral space. Alternatively, an anterior approach may be used similar to that for management of axillary artery lesions, to ligate the origin of the PCHA.

89.6 Special Considerations in the Competitive Athlete

The physician specializing in the management of TOS is often consulted for advice regarding the optimal treatment for a competitive athlete that has developed arterial complications. In evaluating the various diagnostic and treatment options for arterial TOS, the physician must obviously be closely familiar with the pros and cons of each strategy. In addition, it is valuable to have an understanding of the demands of the individual athlete's sport, the timing and duration of the season and off-season schedule, and the particular nuances that may accompany competition at various levels. For example, the competitive high school athlete is sometimes driven by parental pressure to achieve a level of success sufficient to earn a college scholarship, in which case the timing of medical/surgical treatment must be juxtaposed with a desired return to sport during the school year, particularly the junior and senior years. Similar considerations apply to the collegiate athlete; however, some individuals are motivated to compete in a final year or two of sports without anticipation of a professional career, and may therefore have less ambitious plans for future participation in sports. For the professional athlete, the type and timing of treatment is often dictated by the point in the season or off-season at which symptoms arise. Whether or not the team is actively competing for a championship in that particular season or is a "rebuilding" phase, the role played by the individual athlete in that team effort versus the potential substitute(s) available, and the finances and stage of an individual athlete in their professional contract, may all influence the aggressiveness desired in achieving a rapid return to play.

At times there is a desire to offer partial or temporizing treatment to allow an athlete to complete play during a given season, planning for definitive treatment once the season has ended. However, the feasibility of this approach is limited with arterial TOS, since in most sports the patient cannot compete while on anticoagulation treatment, and a resumption of athletic activity in the absence of surgical treatment can be expected to result in further thromboembolic complications. Thus, in patients who have undergone arteriographic diagnosis the best option is almost always for prompt surgical treatment to allow for the most rapid recovery and rehabilitation.

The consulting physician should be familiar with the approaches used to manage arterial TOS and be able to place these considerations into the context of the potential need for additional treatments and an anticipated return to athletics for the patient, family, athletic trainers, other team representatives, and agents (in the case of the professional athlete). It is not uncommon for the athlete, family, team and/or agent to request several different opinions; thus, it is helpful for the consulting physician to be aware of the specialists with greatest experience in treating patients with arterial TOS and to be able to facilitate additional referrals when necessary.

For the surgeon undertaking treatment of the competitive athlete with arterial TOS, it is crucial to recognize the impact that this condition has had, and will have, on the patient's participation in competitive sports, which in many cases is the patient's means to obtain or maintain a collegiate scholarship or is even their principal livelihood. An extra degree of attention is required with regard to close and open communication with the patient, family, and team representatives regarding expectations and progress at each step of care, along with close collaboration between the

surgeon and other health care personnel, particularly the physical therapy team assisting with postoperative care. With a coordinated effort and well-established protocols, the surgeon can obtain excellent results with a high degree of patient satisfaction, along with a prompt and successful return of the athlete to competitive activity.

89.7 Conclusions

Overhead athletes with arterial disease of the upper extremity often present with unilateral digital ischemia from emboli or vasospasm, and may exhibit diminished upper extremity blood flow with arm elevation that can be identified by contrast-enhanced imaging studies. Arterial TOS is characterized by subclavian artery pathology that can cause thromboembolism and digital ischemia (aneurysms or occlusive lesions) in association with a bony abnormality, such as a cervical rib. Other localized thromboembolic lesions in athletes include compression of the axillary artery and posterior circumflex humeral artery associated with repetitive vigorous overhead activity.

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