Vascular Problems in Elite Throwing Athletes

Cara M. Thorne, MD,* Baris Yildirim, MD,* Margaret C. Tracci, MD,† A. Bobby Chhabra, MD*

Continuing Medical Education

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Dawn M. LaPorte, MD, has no relevant conflicts of interest to disclose.

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All authors of this journal-based CME activity have no relevant conflicts of interest to disclose. In the printed or PDF version of this article, author affiliations can be found at the bottom of the first page.

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Dawn M. LaPorte, MD, has no relevant conflicts of interest to disclose. The editorial and education staff involved with this journal-based CME activity has no relevant conflicts of interest to disclose.

Learning objectives

Upon completion of this CME activity, the learner will:

- Recognize the presentation of vascular injuries in throwing athletes.
- Understand the pathophysiology and diagnosis of vascular injuries in throwing athletes.
- Understand the treatment options and prognosis for vascular problems in elite throwing athletes.

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There are approximately 880 and 3,600 major league baseball and minor league baseball players who currently are active in their respective leagues, with thousands of players in the collegiate, high school, and little league ranks. Although relatively uncommon, vascular injuries, such as thoracic outlet syndrome, axillary artery compression, quadrilateral space syndrome, and direct vascular trauma, can afflict these players. These career- and limb-threatening injuries can mimic often seen muscular sprains and strains in their early stages

*From the *Department of Orthopaedic Surgery and †Department of Vascular Surgery, University of Virginia, Charlottesville, VA.*

Received for publication March 22, 2022; accepted in revised form August 12, 2022.

No benefits in any form have been received or will be received related directly or indirectly to the subject of this article.

Corresponding author: Cara M. Thorne, MD, Department of Orthopaedic Surgery. University of Virginia, 2280 Ivy Road, Charlottesville, VA, 22903; e-mail: cmt3z@virginia.edu.

0363-5023/22/4801-0009$36.00/0
https://doi.org/10.1016/j.jhsa.2022.08.016

with nonspecific symptoms, such as exertional fatigue, which can delay diagnosis with disastrous sequelae, including thrombus propagation, aneurysm rupture, and ischemia from distal embolization. The goal of this review is to discuss the pathophysiology, diagnosis, and treatment of these injuries to increase awareness of sport-related vascular phenomena among the hand and upper-extremity surgery community because these players typically are seen first in the training room or a hand specialist's office. *(J Hand Surg Am. 2023;48(1):68–76.*

Key words Baseball pitchers, digital ischemia, quadrilateral space syndrome, repetitive finger trauma, vascular thoracic outlet syndrome.

CONDITIONS AFFECTING VASCULATURE ABOUT THE SHOULDER

Arterial thoracic outlet syndrome

In arterial thoracic outlet syndrome (aTOS), the subclavian artery is compressed at the thoracic outlet. This is a rare variant contributing to approximately 2% of all cases of TOS.[1] In 85% of patients presenting with aTOS, the compression is attributable to the presence of bony abnormalities, such as a cervical rib.[2] In this instance, the displaced subclavian artery is compressed between the first rib and anterior scalene muscle. However, in the athletic population, compression of the subclavian artery can be because of hypertrophy, fibrous scarring, muscle spasm of the scalene muscles, or arterial degeneration associated with repetitive motion injury. Hypertrophy of the pectoralis minor as well as extremes of shoulder position common in overhead throwing, namely hyperabduction and external rotation, also can lead to axillary artery compression, a distal analog of aTOS.[3,4] Glenohumeral instability with anterior subluxation of the humeral head in the throwing position also can cause axillary artery compression.[5] Stapleton et al[5] demonstrated a correlation between the amount of humeral head translation and the degree of axillary artery compression seen on duplex ultrasound. Typically, the third portion of the axillary artery is affected given its fixed position from branching vessels, the posterior humeral circumflex and subscapular arteries, and surrounding fascia. Although constant, long-term compression in typical aTOS results in intimal thickening, stenosis, and ultimately turbulent arterial flow contributing to poststenotic aneurysmal degeneration and the associated risk of thrombus formation, the etiology in overhead throwers is attributable to a transient, positional compression rather than the static, anatomic compression associated with aTOS in the general population.[4]

Athletes presenting with early aTOS often describe nonspecific symptoms, such as unilateral arm heaviness and weakness, and decreased endurance with pitching. Unless there is a high level of suspicion from the athletic training and coaching staffs, these patients often will experience delays in diagnosis until later sequelae of the condition manifest. These sequelae include cold intolerance, temperature difference, and acute ischemia of the hand and fingers demonstrated by progressive pain, discoloration, and ulceration if there is propagation of a thrombus or embolic event. Although vasomotor phenomena may be associated with brachial plexus compression and neurogenic thoracic outlet syndrome (nTOS), acute or chronic embolization because of arterial thrombus formation can present similarly, yet portend worse outcomes, including tissue loss. The rich anastomoses about the upper extremity can lead to deceptively normal arterial examination maneuvers despite considerable stenosis or thrombus formation, with apparently normal ulnar and radial pulses, wrist-brachial indices, and Allen test. Because of the poor reliability of physical examination in diagnosing this condition, imaging modalities are the mainstay of diagnosis.[6] Duplex ultrasound is a commonly used first line, noninvasive study, but is somewhat operator-dependent and does not offer the anatomic details afforded by computed tomography (CT) and magnetic resonance angiography (MRA) (Fig. 1). Unlike traditional angiography, these studies are noninvasive, protocoled, and although not dynamic studies, can permit positional evaluation, typically shoulder abduction in the throwing athlete, to increase diagnostic sensitivity. When embolization is suspected, it is important to rule out other causes of the patient's symptomatology, such as atrial fibrillation, which remains the most common cause for upper-extremity emboli, and valvular heart disease.[7]

If a thrower presents with symptomology consistent with aTOS without imaging evidence of arterial injury, such as intimal damage, thrombus formation, or aneurysm, it is reasonable to pursue non-operative management. This typically consists of hand or

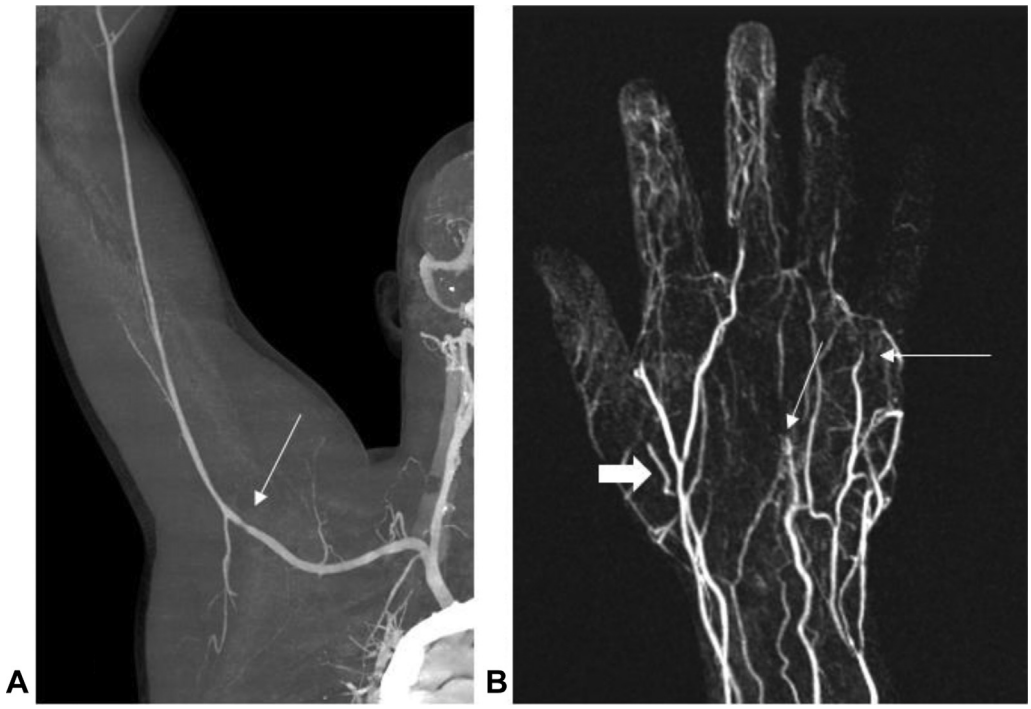

FIGURE 1: Angiography of the right upper extremity of a 19-year-old male baseball pitcher who presented with intermittent numbness and discoloration of the little finger following a throwing session. **A** CT angiogram of the shoulder and brachium demonstrating occlusion of the axillary artery (solid arrow). **B** MR angiogram of the right hand with multifocal occlusions of the princeps pollicis (thick solid arrow), and multiple common digital arteries (thin solid arrows) with resultant severely decreased perfusion to the little finger, and somewhat decreased perfusion to the thumb and ring finger.

occupational therapy working on scapular stabilization and throwing mechanics. Procedural intervention is warranted if the patient has evidence of arterial injury. It is recommended that elite throwers first undergo surgical decompression, which may consist of removal of any cervical rib and associated fibrous tissue, release of hypertrophied muscle, and potentially first rib resection. This is followed by reconstruction of the diseased segment of vasculature, most commonly by either thromboendarterectomy and patch repair or resection and interposition grafting. Autogenous saphenous vein is the preferred graft choice. Surgical release and reconstruction are preferred over purely endovascular treatment, such as balloon angioplasty with or without stent placement because of the likelihood of early failure without relief of the source of extrinsic compression and concerns about the long-term durability of endovascular stents in flexural areas.[7–11] Where there is evidence of embolization, most providers also recommend concurrent thrombectomy or intraarterial thrombolytic therapy.[8,9] After treatment for thrombosis or embolism, patients typically remain on oral anticoagulation for 6–12 weeks after surgery. After undergoing surgical intervention for aTOS, patients generally are able to resume play within 3 months.[8,9–12] The duration of therapeutic anticoagulation is a factor in the timing of a return to full contact activities. In a case series of 9 players treated surgically for aTOS, patients engaged in immediate hand therapy focusing on upper-extremity range of motion, posture, and shoulder mechanics. Their cohort resumed throwing at 10.8 ± 2.7 weeks after surgery.[8] In other case reports, patients were permitted to start a throwing program at 8 weeks and returned to their professional or collegiate teams by 3 months after surgery.[9,11,12] If concomitant nTOS is present, recovery may be prolonged. In a study of major league baseball pitchers, 10 of 13 pitchers returned to play following surgical treatment of nTOS 10.8 ± 1.5 months after surgery.[13]

Paget-Schroetter syndrome

Also known as effort thrombosis of the subclavian vein, Paget-Schroetter syndrome or venous thoracic outlet syndrome (vTOS) is related to vigorous overhead arm use and is caused by compression and repetitive injury of the subclavian vein between the clavicle and first rib. Because of the medial position of the vein in the thoracic outlet, this condition is not associated with the presence of a cervical rib. In a study of 32 overhead athletes treated surgically for effort thrombosis, the etiology of compression

included aberrant fascial bands in 41%, hypertrophic or anomalous scalene minimus muscle in 31%, or anomalous phrenic nerve in 6%.[14]

Most athlete patients present with arm heaviness, fatigue, and pain with use following games or practice. Spontaneous arm swelling, cyanosis, and distention of subcutaneous veins of the shoulder and pectoral region are possible with rare reports of dizziness and shortness of breath because of pulmonary embolism as a presenting symptom. Although patients often will undergo venous duplex ultrasound, this imaging modality has a diagnostic accuracy of 71% for Paget-Schroetter syndrome.[14] Reasons for this low diagnostic accuracy include difficulty visualizing the subclavian vein beneath the clavicle and the presence of large collateral veins. Because of these limitations, MR venography, CT venography and catheter-based contrast venography are preferred (Fig. 2).

Once Paget-Schroetter syndrome with venous thrombosis has been diagnosed, the current standard of care is surgical decompression with resection of the medial portion of the first rib, the subclavius muscle and tendon adjacent to the vein, and circumferential venolysis. Although a case series in 1987 described effective conservative management in 85% of patients, more recent studies indicate that non-operative treatment rarely results in a symptom-free patient and imposes limitations in positioning that most patients find unacceptable.[14,15] Moreover, recurrent or chronic thrombosis is likely to lead to more severe long-term changes to the subclavian vein. Widely reported protocols include catheter-directed thrombolysis followed either immediately or following a variable, anticoagulated interval by surgical decompression. Reversing this sequence and performing catheter-directed thrombectomy and thrombolysis following surgical decompression also has been reported. Stent placement in the subclavian vein, with or without decompression, is associated with high rates of restenosis and occlusion and is not recommended in this patient population.[16] Surgical decompression may be accomplished by either an infraclavicular or a transaxillary surgical approach.

If the vein remains visibly narrowed, thick-walled to palpation, or there is venographic evidence of persistent obstruction, catheter angioplasty, often with serial and/or prolonged anticoagulation of up to a year is recommended. Direct vein reconstruction by patch venoplasty or interposition grafting largely has been supplanted because of the central nature of the compression and associated venous injury seen in vTOS with full reconstruction often requiring at least partial sternotomy with considerable associated morbidity. In any case, anticoagulation typically is continued for 30–90 days after surgery, with longer durations reserved for rethrombosis or extensive residual venous disease. Interestingly, many patients with rethrombosis following decompression remain asymptomatic or nearly so when managed with a course of anticoagulation alone, presumably because of the presence of large collaterals.[17–20]

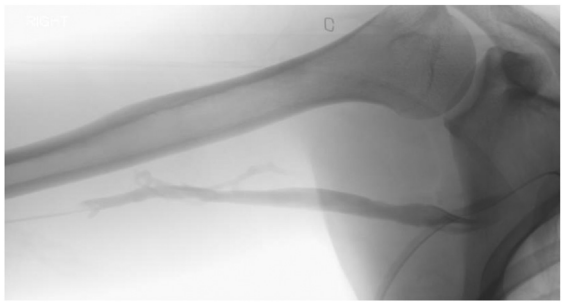

FIGURE 2: Direct contrast venography of the right upper extremity of a 17-year-old male Olympic weightlifter who presented with right chest wall swelling and arm heaviness demonstrating near-complete obstruction to venous outflow at the level of the axillary/subclavian vein junction during abduction of the arm.

In young, athletic patients, the role of prophylactic bilateral decompression for venous symptoms without frank thrombosis is debated.[21] If associated symptoms of nTOS are present, anterior and middle scalenectomy and brachial plexus neurolysis may be considered, although nTOS is more likely to present in combination with the aTOS rather than vTOS.[22]

Hand therapy consisting of shoulder range of motion can be initiated immediately after surgery, and full activities generally can be resumed 3 months after surgery.[14] Again, the resumption of full contact activities is informed by the duration of full anticoagulation with associated bleeding risks. The average time for return to play in a cohort of 32 athletes was 4.3 months in patients without surgical complications, 4.7 months in patients with surgical complications, and 4.4 months for the cohort. There were no statistically significant differences with respect to return to play between these groups.[14]

Quadrilateral space syndrome

The quadrilateral space is defined medially by the surgical neck of the humerus, laterally by the long head of the triceps, superiorly by the teres minor, and inferiorly by the teres major and latissimus dorsi.

The posterior humeral circumflex artery (PHCA) passes through this anatomic space, and when

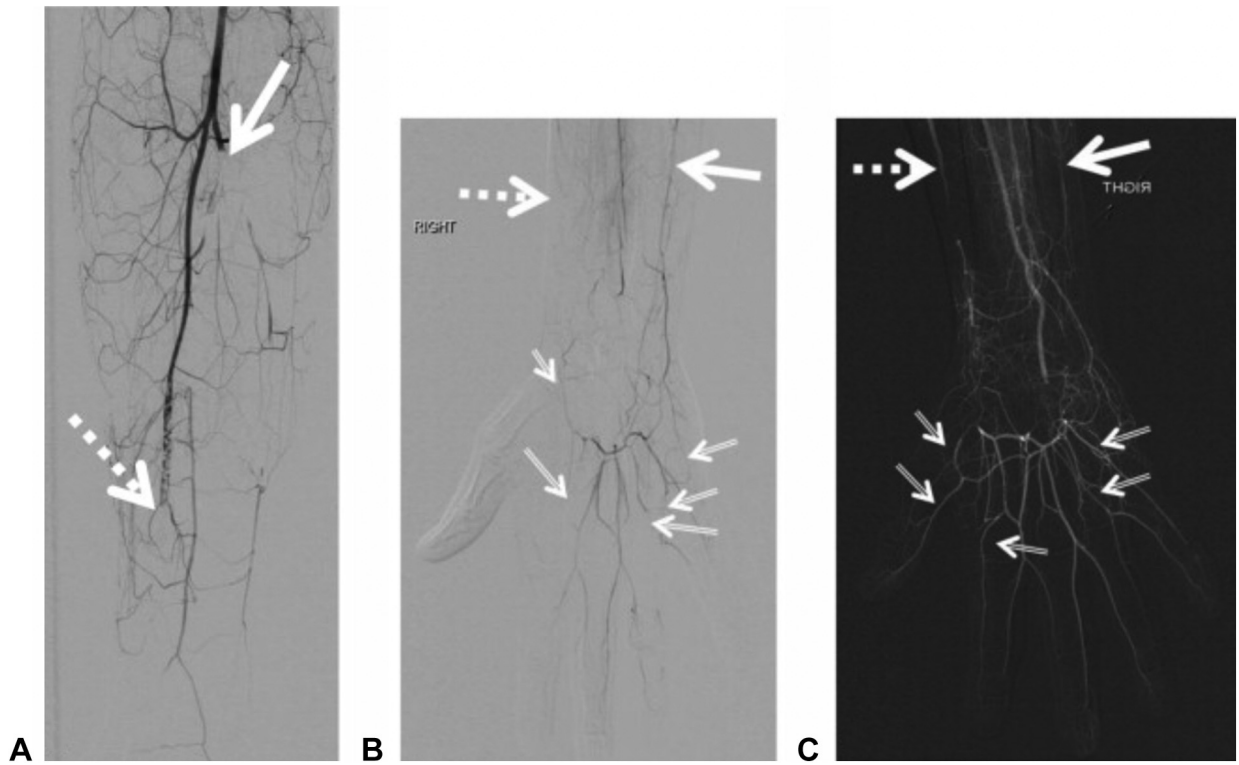

FIGURE 3: Angiography of the right hand of a 20-year-old female volleyball player who presented with purplish discoloration of multiple digits of the right hand. **A** Angiogram of the right forearm demonstrates occlusion of the proximal ulnar artery (solid arrow) and proximal interosseous arteries with distal reconstitution. The radial artery occlusion (dashed arrow) begins in the mid forearm. **B** Angiogram of the right hand 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) because of embolization. **C** Angiogram after surgical intervention demonstrates overall improved flow into the hand with partial revascularization of the ulnar (solid arrow) and radial (dashed arrow) arteries, as well as the common digital arteries (smaller double arrows). Reproduced with permission from: Brown SA, Doolittle DA, Bohanon CJ, et al. Quadrilateral space syndrome: the Mayo Clinic experience with a new classification system and case series. *Mayo Clin Proc*. 2015;90(3):382–394.[23]

compressed, produces a constellation of symptoms termed quadrilateral space syndrome (QSS). Patients can present with upper limb claudication, pallor, ischemia, discoloration and ulceration of digits, and Raynaud phenomenon. When associated with axillary nerve compression, patients may exhibit weakness in shoulder external rotation or abduction, and muscle atrophy in the teres minor and deltoid. They also may describe poorly localized pain about the shoulder thought to be secondary to axillary nerve compression rather than ischemic pain.

Athletic patients most likely to present with QSS are those who are involved in repetitive shoulder external rotation and abduction commonly seen in baseball, volleyball, and football, especially quarterbacks.[23] The PHCA is tethered by the humeral neck at its branch point from the axillary artery and is susceptible to microtrauma from extremes of shoulder motion. This can lead to intimal injury and aneurysms with increased risk for thrombus formation and subsequent embolic phenomena. Interestingly, the axillary nerve seems protected from the mechanical strain and traction injury leading to vascular QSS with neurogenic QSS because of space-occupying lesions in the quadrilateral space.[23]

Ultrasound findings, including PHCA aneurysms and reduced blood flow velocity in shoulder external rotation and abduction have proven nondiagnostic for QSS as they are present in 5% to 20% of healthy volunteers.[24,25] Similar to ultrasound, CT angiography and MRA are limited by nonspecific findings. Stenosis or occlusion of the PHCA was found in 80% of controls.[26] As such, digital subtraction angiography is often the study of choice because of its superior spatial resolution.[23]

The preferred mode of treatment for vascular QSS consists of PHCA ligation with or without thrombolysis or surgical thromboembolectomy.[23] Unlike in aTOS, the damaged artery can be ligated because it is a terminal branch and there is a rich collateral blood

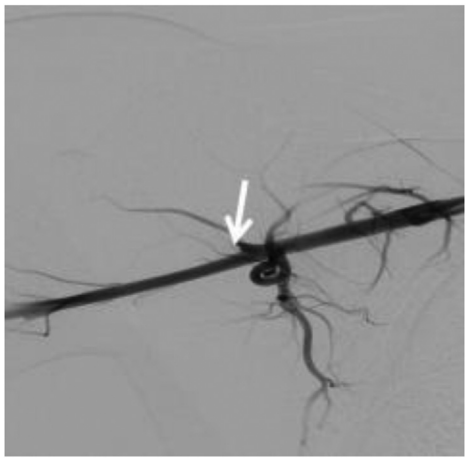

FIGURE 4: Angiography of the right shoulder of the same patient in Figure 3 showing abrupt occlusion of the posterior circumflex humeral artery (arrow) caused by thrombus. Reproduced with permission from: Brown SA, Doolittle DA, Bohanon CJ, et al. Quadrilateral space syndrome: the Mayo Clinic experience with a new classification system and case series. *Mayo Clin Proc*. 2015;90(3):382−394.[23]

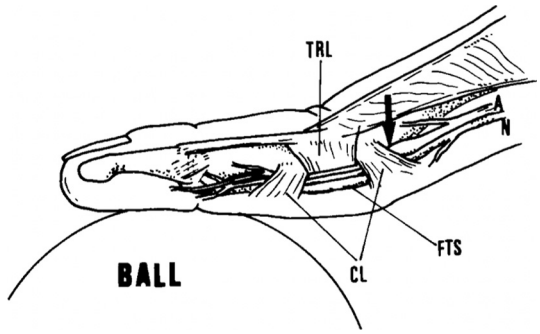

FIGURE 5: The entrapment of the neurovascular bundle in hyperextension of the proximal interphalangeal joint. FTS, flexor tendon sheath; TRL, transverse retinacular ligament; CL, Cleland's ligament. Reproduced with permission from: Itoh Y, Wakano K, Takeda T, Murakami T. Circulatory disturbances in the throwing hand of baseball pitchers. *Am J Sports Med*. 1987;15(3):264−269.[34]

supply to the glenohumeral joint, teres minor, teres major, and deltoid via the anterior humeral circumflex artery and thoracoacromial artery. Case reports of athletes with vascular QSS treated with PHCA ligation return to full, painless sport participation at 2−12 months.[23,27] If the athlete has concurrent neurogenic QSS, it can be treated with a trial of hand therapy, corticosteroid injection, and activity modification to prevent shoulder external rotation, and abduction. However, because most athletes cannot abandon this shoulder movement in their sport, concurrent disease often is treated with lysis of adhesions and axillary nerve neurolysis (Figs. 3, 4).[23,28]

CONDITIONS AFFECTING VASCULATURE ABOUT THE HAND AND WRIST

Hypothenar hammer syndrome

Because the superficial palmar branch of the ulnar artery leaves Guyon canal, it is relatively unprotected from blunt trauma to the hand causing impact against the hook of hamate. This trauma leads to intimal injury with possible aneurysmal degeneration, thrombosis, occlusion, and embolization. Athletes who use a bat, stick, or racket are susceptible to this condition. The condition itself typically is not associated with the act of throwing; however, it should remain on the differential for baseball players who present with digital pallor, discoloration, Raynaud phenomenon, paresthesias, pain, and ischemia especially when involving the ulnar digits. Ultrasound often is helpful in making a diagnosis when lack of arterial flow distal to the ulnar artery is seen; however, angiography provides a more definitive diagnosis. Treatment mainly consists of lifestyle and equipment modification. For instance, bat grip modifications or additional padding to batting gloves. Pharmacologic therapy can be helpful with calcium channel blockers and steroids to improve blood supply and sympatholytics to reduce vasospasm.[29] Operative intervention is reserved for rare cases involving critical ischemia, intractable pain, tissue loss, or failure of non-operative treatment. If the patient has a complete arch, surgical ligation or excision of the diseased segment with venous grafting are options. Catheter-directed thrombolysis also is an option if thromboembolic events have occurred. In 1 case study of a hockey player presenting with pain, paleness, and coolness of his ulnar 3 digits, the patient was able to return to play, following catheter-directed thrombolysis and stick modifications, to full sport activities in 3 months.[30]

Repetitive finger trauma

Extraordinary stresses are placed on the hands of baseball players with every swing of the bat, pitch, and catch which can lead to pain, cold insensitivity, cyanosis and ulcer formation of the fingers. In pitchers, the index and middle fingers are affected most often.

Repetitive impact is the most common cause of these findings in catchers.[31,32] The cause of repetitive finger trauma in pitchers has been associated with hyperextension of the fingers during pitching as well as hypertrophy of intrinsic hand musculature. There

is rapid flexion and extension of the metacarpophalangeal and proximal interphalangeal joints with pitching.[33] Even in asymptomatic pitchers, blood flow velocity decreases at the proximal and middle phalanges with extension of the metacarpophalangeal and proximal interphalangeal joints compared to neutral positioning. This is thought to be because of compression of the neurovascular bundle between Cleland ligament and the phalanges (Fig. 5).[34] Pitching also requires repeated contractions of the intrinsic hand muscles leading to hypertrophy of these muscles with impingement of the intermetacarpal arteries.[34]

Digital subtraction angiography is the gold standard diagnostic test, providing superior resolution and anatomic detail. With direct hand trauma, most associated with catchers, adding more webbing or padding to the glove often is successful.[31] A case series by Sunagawa et al[35] looked at whether oral antithrombotic agents would be beneficial in treating ulcerations in this population. They found that aspirin and prostaglandin E1 administration did not improve healing of patients' fingertip ulcerations. They postulated that these medications could be beneficial in prevention of ulcerations if the disease state was caught in earlier stages; however, no long-term studies have been conducted to corroborate this notion. Surgery is reserved for those with critical ischemia and centers on releasing the constricting structure, such as Cleland's ligaments or the lumbrical canal. If no constricting structure exists, the vessel may have internal fibrosis or an aneurysm. In these scenarios, the diseased segment of artery is excised and a vein graft is used to reconstruct the artery. All players who underwent surgical correction in 2 case studies, returned to prelevel function within 3−6 months.[34,35]

Distal ischemia from embolic phenomena

Multiple etiologies of fingertip ulceration, pain, and numbness have been presented in this review, namely more proximal vascular trauma, arterial degeneration (stenosis and/or aneurysm formation) and associated thromboembolic phenomena. Management of these patients consists of determining the etiology of the emboli typically with MRA or digital subtraction angiography and subsequent treatment of the cause as described above. The distal emboli can be treated with thrombectomy in the vessels of the arm or forearm or catheter-directed thrombolysis in more distal vessels; however, it is essential that the primary cause of the emboli be addressed to prevent recurrent events.

In summary, overhead athletes, such as baseball pitchers, volleyball players, swimmers, and football quarterbacks, undergo hypertrophy of the muscles about the shoulder girdle and hand because of the repetitive nature of their sport and also experience altered glenohumeral mechanics because of the extremes of shoulder positioning necessary for the overhead throwing motion. These changes can lead to traction and compression of the axillary, axillary branch, and subclavian vessels with subsequent intimal thickening, stenosis, and ultimately turbulent flow contributing to thrombus risk and aneurysm formation. More proximal arterial pathology may lead to embolization affecting distal vessels from brachial to digital locations. Distal vessels also may be injured directly by repetitive trauma to the wrist or hand. A high index of suspicion is needed to diagnose and treat these vascular pathologies in the overhead throwing athlete before the onset of irreversible ischemia and tissue loss.

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