Upper Extremity Vascular Problems in Athletes

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CHRISTOPHER K. ZARINS, M.D.

ABSTRACT Upper extremity vascular injuries are being seen with increasing frequency. These injuries particularly involve the subclavian artery and vein, the axillary artery, and the arteries of the hand. Stretch injury to the branches of the axillary artery may also occur, leading to aneurysm formation. These aneurysms may develop intraluminal thrombus, which can embolize as a result of compression of the aneurysm with repetitive activity. Noninvasive testing has been found to be very helpful in the diagnosis of these sports-related injuries. Proper management depends upon the vessel involved. With appropriate management, results have been good, and the majority of these athletes, professional and amateur, return to full athletic activity.

Keywords Upper extremity, ischemia, aneurysm


Sports, both amateur and professional, have become an expanding part of American culture. As a result, physicians are seeing an increasing number of sports related injuries. Although most athletic injuries involve the musculoskeletal system and hence are the province of the orthopedic surgeon or sports medicine specialist, injuries to the vascular system are being seen with increasing frequency. Vascular related athletic injuries involve primarily three areas: the shoulder girdle (axillary and subclavian vessels), the hip (iliac vessels), and the knee (popliteal vessels). These injuries may involve either the
arterial or venous systems and are related to either repetitive motion or stress on the associated joint. A fourth area of injury, direct injury to the arteries of the hand, may occur as a result of direct repetitive trauma.

Upper extremity vascular problems in athletes can be divided into four groups: (1) injuries to the subclavian artery and its branches as part of a thoracic outlet syndrome, (2) subclavian vein thrombosis—Paget-Schroetter syndrome, (3) injury to the axillary artery and its branches, and (4) direct injury to the arteries of the hand. Injuries to the axillary and subclavian vessels have been described in a variety of athletes but are most common in baseball players, especially pitchers, and volleyball players. Injuries to the arteries of the hand are most common in baseball catchers and handball players.

It is important that vascular surgeons be familiar with these injuries. Frequently their presentation is subtle and management decisions may be difficult. As many of these athletes are high-priced professionals, decisions regarding management may have significant consequences both for the athlete and for the team administration.

SUBCLAVIAN AND AXILLARY ARTERY INJURIES

The significance of vascular injuries in athletes was documented by Fields and colleagues in 1986 with the report of a baseball pitcher who sustained a stroke following thrombosis of his right subclavian artery with subsequent propagation of the clot into the innominate and right carotid arteries.1 Two excellent reports on axillary and subclavian artery injury in athletes have been published subsequently by the vascular surgery division at Northwestern University Medical School.2,3 Compression of the third portion of the axillary artery by the head of the humerus was first reported by Lord and Rosati in 19584 and subsequently by Rohrer and colleagues in 1990.5 These authors stressed the importance of repetitive injury to the axillary artery by the head of the humerus during hyperabduction. This was confirmed by Durham and colleagues at Northwestern.6 Nijhuis and Muller-Wiefel, in 1991, first reported embolization from a branch artery aneurysm of the axillary artery.6 Two additional cases of embolization from branch artery aneurysms of the axillary artery in professional baseball pitchers were added to the literature by the Stanford group in 1995.7 Reckers and colleagues reported aneurysms of the posterior circumflex humeral artery in two volleyball players, both of whom also sustained distal embolization from their aneurysm.8 As a result of these reports, it became well recognized that athletes were particularly prone to injury of their subclavian and axillary arteries and that these injuries could result in upper extremity ischemia.

Injury to the subclavian artery is usually a complication of thoracic outlet syndrome and results from compression of the artery by a cervical rib, an anomalous first rib or by the soft tissue structures of the thoracic outlet.
Repetitive injury to the subclavian artery may produce arterial stenosis, which can progress to arterial occlusion as in the patient of Fields et al. Subclavian stenosis also may be associated with poststenotic dilatation of the artery or frank aneurysm formation. These dilated areas may accumulate thrombus, which can be a source of distal embolization. Also, as in the case of one of our patient 3, repetitive trauma to a branch vessel of the subclavian artery against the clavicle may produce an aneurysm that can develop intraluminal thrombus, which can embolize, producing distal ischemia.

Injury to the axillary artery may be caused by compression by the pectoralis minor tendon or by compression of the third portion of the axillary artery by the head of the humerus during hyperabduction and external rotation. Stretch injury to the branches of the axillary artery—the circumflex humeral arteries and the subscapular artery—also may occur with repeated hyperabduction. Repetitive injury to these branches produces aneurysms of the proximal portion of these vessels, which may develop intraluminal thrombus. Subsequent compression of these aneurysms with athletic activity results in retrograde extrusion of thrombotic material into the axillary artery with embolization to distal arteries.

We have treated four baseball players—three pitchers and one first baseman—at Stanford University Medical Center with lesions of the axillary or subclavian arteries (Table 1). In addition, we have consulted two professional baseball pitchers with aneurysms of their circumflex humeral arteries complicated by distal embolization and digital ischemia. Initial symptoms in these athletes may be quite subtle, for example, loss of speed or fine control during pitching, coolness and palor of the fingers, and arm fatigue. Not infrequently, these athletes are believed to have lost their skill or to be suffering from arm fatigue rather than from a vascular lesion. If untreated, these

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Sport</th>
<th>Vascular Lesion</th>
<th>Treatment</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>25</td>
<td>M</td>
<td>Baseball pitcher</td>
<td>Circumflex artery aneurysm</td>
<td>Aneurysm resection</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>2</td>
<td>22</td>
<td>M</td>
<td>Baseball pitcher</td>
<td>Subscapular artery aneurysm</td>
<td>Lytic therapy/aneurysm resection</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>3</td>
<td>23</td>
<td>M</td>
<td>Baseball/1st base</td>
<td>Suprascapular artery aneurysm</td>
<td>Lytic therapy/aneurysm resection</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>4</td>
<td>32</td>
<td>M</td>
<td>Baseball pitcher</td>
<td>Axillary artery aneurysm</td>
<td>Lytic therapy</td>
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</tbody>
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patients ultimately develop significant digital ischemia with pain, discoloration, and ulceration.

Evaluation

**Noninvasive Evaluation.** All four patients managed at our institution underwent noninvasive testing in our vascular laboratory. In three of the patients, duplex scanning demonstrated the axillary artery or branch artery lesion. Figure 1 is a color duplex image demonstrating the subscapular artery aneurysm with intraluminal thrombus seen in patient 2 (Table 1). Figure 2 demonstrates the wall thickening and early aneurysmal changes seen in the orifice of the subscapular artery in patient 4 (Table 1). The one patient in whom we did not visualize the aneurysm by noninvasive testing was the first patient we evaluated, and failure to find his lesion may be attributable to our initial inexperience. Photoplethysmograph (PPG) waveforms were also obtained in all patients and were helpful in documenting digital artery occlusions. Also, follow-up PPG studies demonstrated that the waveforms returned to normal in these patients, thus confirming the efficacy of treatment (Fig. 3).

![PPG waveforms](image)

**Fig. 1** PPG waveforms from patient 1 (Table 1), a left-handed professional baseball pitcher. Initial waveforms in the left panel demonstrate diminished waveforms in the first and second fingers of the left hand. The right-hand panel demonstrates normalization of the waveforms 3 months after resection of the aneurysm. Lytic therapy was not used in this patient.
Fig. 2 Thickening of the wall of the axillary artery (+) and early aneurysmal change of the orifice of the subscapular artery (arrow) in patient 4 (Table 1).

Fig. 3 Thickening of the wall of the axillary artery (+) and early aneurysmal change of the orifice of the subscapular artery (arrow) in patient 4 (Table 1).
Invasive Evaluation. Arteriography was performed in all patients. In the first patient, a left-handed pitcher, the left upper extremity arteriogram showed multiple filling defects in the digital arteries, but an embolic source was not initially appreciated. Arteriography performed with his arm in multiple positions failed to demonstrate any compression of his subclavian or axillary artery. Also, there were no areas of aneurysmal change or poststenotic dilatation. Cardiac workup did not demonstrate any cardiac source for his emboli. Closer inspection of his arteriogram demonstrated occlusion of the posterior circumflex artery 0.5 cm distal to its origin (Fig. 4). Also, this arteriogram demonstrated the close proximity of the head of the humerus and its ability to compress the branch artery aneurysm (Fig. 4). In the second patient, arteriography helped confirm the presence of an aneurysm of the subscapular artery and demonstrated multiple digital emboli. No evidence of arterial compression in the thoracic outlet was identified. In the third patient, arteriography revealed an aneurysm of the suprascapular artery (Fig. 5). In the fourth patient, arteriography revealed evidence of early aneurysmal dilatation involving a short segment of the right axillary artery between the subscapular artery and the circumflex humeral artery. Intravascular ultrasound demonstrated thickening and irregularity of the vessel wall consistent with intimal hyperplasia, thrombus, or subintimal injury. Stress testing with the arm in abduction revealed deformation of the axillary artery by the humeral head.

![Fig. 4 Arteriogram of patient 1 (Table 1), a professional baseball pitcher. Note the occluded circumflex humeral artery, which at surgery was found to be an occluded aneurysm. Also note the proximity of the humeral head.](image)
Management Results

The management of these patients has evolved with our experience. At present, appropriate treatment of these patients involves removing as much of the embolic material as possible and resecting the source of the emboli. Three of these patients were treated with catheter-directed intraarterial urokinase to lyse the emboli. Unfortunately, the embolic lesions in these patients occur over an extended period of time and we were not successful in completely removing all occlusions in any given patient. However, in each instance where urokinase was used, it significantly improved the distal circulation. Following lytic therapy, the source of the emboli was resected in patients 1 to 3. In each case the branch artery aneurysm was resected and the artery repaired. The suprascapular aneurysm resected in patient 3 is demonstrated in Fig. 6. This picture also illustrates the thrombotic material present in these branch artery lesions. Patch grafting or interposition grafting was not required in our patients but has been reported by others. Should this be required, we would recommend the use of autogenous tissue. Experience in the literature would suggest that the saphenous vein is a durable graft in this location, although some surgeons prefer autogenous artery.

All three of our operated patients did well following surgery. Each patient has remained free of further ischemic lesions, and in each case their PPG studies returned to normal within 3 months (Fig. 3). Each of these patients has returned to full athletic activity. The fourth patient in our series had diffuse, mild enlargement of his axillary artery with wall thickening but was not anxious to undergo surgical intervention. This patient was treated with coumadin and rested from his pitching duties for the remainder of the season. He resumed pitching the following season and has not developed any further vascular problems.
SUBCLAVIAN VEIN INJURIES

Athletes may also develop subclavian vein thrombosis—Paget-Schroetter syndrome. We have treated nine athletes with subclavian vein thrombosis over the past 3 years. In five patients, their treatment was initiated elsewhere but subsequently they were referred to our institution for further management. These nine patients represent a variety of different sports but common to all is extensive use of the upper extremities or direct trauma to the shoulder girdle as in patient 5 (Table 2). Patients with Paget-Schroetter syndrome present with arm swelling, discoloration, and discomfort. These symptoms may occur following a particularly prolonged or arduous athletic activity but more frequently occur spontaneously as a complication of repetitive activity. Typically these patients develop stenosis of the subclavian vein at the level of the first rib. This obstruction to flow is a result of extrinsic compression of the vein by the rib, the clavicle, or the soft tissue structures of the thoracic outlet combined with the repetitive motion used in athletic activities.

Evaluation

Diagnosis is made by color flow duplex evaluation (Figs. 7 and 8). This study will confirm the presence of intraluminal thrombus and the obstruction to flow. Collateral vessels may be documented. We have found this to be the best screening test for patients with suspected subclavian vein thrombosis. To date, we have not noted any false-negative studies. Venography is ultimately required to demonstrate the extent of the lesion and to evaluate the status of the subclavian vein following lytic therapy if it is utilized (Figs. 9 and 10).
Table 2  Subclavian Vein Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Sport</th>
<th>Vascular lesion</th>
<th>Lytic therapy</th>
<th>Anticoagulation</th>
<th>Surgical decompression</th>
<th>Result</th>
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<tbody>
<tr>
<td>1</td>
<td>18</td>
<td>M</td>
<td>Football</td>
<td>R SC vein thrombosis</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
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</tr>
<tr>
<td>2</td>
<td>20</td>
<td>F</td>
<td>Decoy/weight lifter</td>
<td>R SC vein thrombosis</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>3*</td>
<td>26</td>
<td>F</td>
<td>Windsurfer</td>
<td>L SC Vein thrombosis</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>4*</td>
<td>24</td>
<td>F</td>
<td>Swimmer</td>
<td>L SC vein thrombosis</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
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</tr>
<tr>
<td>5</td>
<td>20</td>
<td>M</td>
<td>Hunter</td>
<td>R SC vein thrombosis</td>
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<td>Yes</td>
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<td>6</td>
<td>20</td>
<td>M</td>
<td>Baseball</td>
<td>R SC vein thrombosis</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
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<tr>
<td>7*</td>
<td>27</td>
<td>M</td>
<td>Baseball</td>
<td>R SC vein thrombosis</td>
<td>Yes</td>
<td>Yes</td>
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<td>Asymptomatic</td>
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<tr>
<td>8*</td>
<td>27</td>
<td>M</td>
<td>Baseball</td>
<td>R SC vein thrombosis</td>
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<tr>
<td>9*</td>
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<td>M</td>
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<td>L SC vein thrombosis</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Asymptomatic</td>
</tr>
</tbody>
</table>

R SC, right subclavian; L SC, left subclavian
* = Patient initially treated elsewhere

Management

The management of these patients remains controversial.9-14 As demonstrated in Table 2, our patients, especially those initially treated elsewhere, received several different treatment regimens. There is, however, general agreement that, once the diagnosis of subclavian vein thrombus is entertained, a venogram should be done and lytic therapy instituted if venous thrombosis is documented.

Our present treatment protocol based on the management of these athletes as well as other patients with subclavian vein thrombosis managed in our institution is

1. Any patient with suspected subclavian vein thrombosis is investigated with color duplex imaging. If venous thrombosis is documented, a venogram is performed promptly.
2. If the venogram confirms subclavian vein thrombosis, catheter-directed lytic therapy using urokinase is initiated. This is continued until maximum lysis is achieved but not usually for longer than 48 hours. Intravenous heparin therapy is started concomitantly, keeping the PTT in the low therapeutic range.

(Text continues on page 12)
Fig. 7 Transverse image of subclavian vein in patient 5 (Table 2). Note significant decrease in lumen of vein secondary to thrombus.

Fig. 8 Longitudinal image of subclavian vein in patient 7 (Table 2).
Fig. 9 Venogram in patient 7 (Table 2) prior to lytic therapy. Note obstruction of subclavian vein beginning at level of thoracic outlet and collaterals coursing superiorly.

Fig. 10 Complete occlusion of subclavian vein and collaterals with abduction and external rotation of the arm.
3. After maximum clot lysis, heparin is continued and the patient is started on coumadin. When the INR is therapeutic, heparin is discontinued and the patient is discharged.

4. Surgical intervention, thoracic outlet decompression, and subclavian venolysis is deferred for at least 30 days to allow the inflammation associated with thrombosis to resolve. In our experience, about one half of the patients with subclavian vein thrombosis who undergo successful lytic therapy will not require surgical intervention after this short course of anticoagulation.

5. Indications for surgical intervention include evidence of significant and persistent extrinsic compression of the subclavian vein following lytic therapy, obstruction of the collateral pathways, associated with subclavian vein obstruction, when the arm is in abduction and external rotation, and persistent symptoms of venous hypertension associated with subclavian vein obstruction. If these indications are present at the conclusion of the course of coumadin therapy, thoracic outlet decompression is recommended. We perform a first rib resection with resection of the anterior and middle scalene muscles by a suraclavicular approach. Subclavian vein venolysis is also performed, making sure that there is no residual compression of the vein. The vein is not opened or bypassed.

6. Angioplasty of the subclavian vein is reserved for cases of persistent venous obstruction after surgical decompression. Angioplasty has not been durable prior to rib removal because of continued extrinsic compression of the vein by bone that the angioplasty balloon cannot affect. Likewise, stents should not be used in this area as long as extrinsic compression of the vein can occur. Repetitive compression of the stent can lead to stent fracture.

Using this protocol, it is our experience that about one half of the patients with subclavian vein thrombosis will require surgical decompression. As is noted in Table 2, however, satisfactory results appear to be obtained by a variety of treatment regimens. Unfortunately, it is difficult to predict the result of any particular treatment for any given patient, which contributes to the present controversy regarding the management of patients with these lesions. It is important to pursue all treatment possibilities until a satisfactory result is obtained.

HAND INJURIES

Hand ischemia may occur in athletes as a result of repetitive blunt trauma. Digital artery occlusions and aneurysms of the distal ulnar artery have been reported, analogous to the hypothenar hammer syndrome. These lesions are most common in baseball catchers but have also been reported in athletes
involved in handball, hockey, lacrosse, volleyball, and karate. These patients typically present with cold hypersensitivity, digital ischemia, or a pulsatile mass in the hypothenar area. Patients with aneurysms of the ulnar artery are best treated by aneurysm resection and interposition vein grafting if required. The remainder, patients without aneurysms, may be managed nonoperatively. Treatment includes lytic therapy, vasodilator therapy, and changes in the athletic equipment, for example, more padding for the catcher’s glove. We have not had any experience with this entity, but reported results have been uniformly good with the above treatment regimen.1,16,17

CONCLUSION

Today’s athletes are susceptible to a wide range of injuries, including vascular injuries that frequently involve the upper extremities. Vascular surgeons should be aware of these lesions. Initial symptoms may be subtle. Management decisions are difficult and complicated by the desire of the athlete to continue to play and, in professional sports, by the money involved. A high index of suspicion, a thorough work-up that includes noninvasive studies and arteriography/venography, and prompt repair of appropriate lesions are important for a successful outcome in these patients.

REFERENCES

Expert Commentary

Of all forms of vascular injury, damage to arteries or veins as a result of athletic activity has received little attention. This article, containing useful diagnostic and management information, is a welcome addition to the literature on vascular injuries.

Two important features need further emphasis. First, surgeons must be aware that the humerus head can compress the axillary artery. Second, damage to the axillary artery is not confined to just the main trunk; branch arteries such as the circumflex artery (anterior or posterior) or the sub- or suprascapular artery can be affected as well. Aneurysms of these branches serve as the source of distal embolization, causing severe hand ischemia.

For diagnosis, color duplex scan is a useful initial diagnostic test, but arteriogram with positional exposure is necessary to confirm the diagnosis. In the acute phase, we also found infusion of urokinase to be helpful in relieving hand ischemia, but definitive surgical intervention is needed to eliminate the source of aneurysm or to repair the damaged artery, as outlined by the authors.

Hand ischemia as a result of direct impact to the hand, such as seen in baseball catchers, can also be found in frisbee, karate, and handball players. In addition to digital artery occlusion, aneurysm formation of the ulnar artery at the hypothenar space has been reported. Even though this aneurysm is small in size, embolization to digital arteries is known to occur, and surgical treatment is indicated. Of interest is the recent case report of ulnar aneurysm in snowboarders.1

Treatment of venous thrombosis of the subclavian-axillary vein is somewhat controversial. Most would agree urokinase infusion is the first line of treatment. Once the patency is restored, the decision to decompress the outlet by removal of the first rib remains a subject of debate. I quite agree with the current protocol outlined by the authors. We also would recommend a supraclavicular approach because the vein can be inspected under direct vision. One problem of the supraclavicular approach is the limitation of resection of the first rib at the junction with the clavicle. Compression of the subclavian vein at the junction with the innominate vein by the clavicle is a real entity. Recently, Molina proposed a new surgical approach by extending the incision medially to the sternum and superiorly to the sternal notch.2 This partial sternotomy allows a better inspection of the junction of the subclavian, internal jugular, and innominate veins. I have no experience in this approach, but the
idea is intriguing. Obviously, resection of the clavicle would be the easier way but removal of the clavicle tends to affect the shoulder motion if the player is going to resume playing activity.

Athletic vascular injury is a distinct clinical entity. Treatment of this type of injury requires a team approach, including an orthopedic surgeon specialized in sports medicine and a sound rehabilitation program with the trainer. In young athletes, expectation from family members can be high or even somewhat unrealistic. Treatment may be difficult in athletes with humerus head compression alone without structural damage to the artery. There is no effective surgical treatment for this condition unless there is evidence of subluxation of the humerus head. Perhaps, a change of pitching motion with close monitoring of the artery with color duplex scan may be the only preferred approach. In these athletes, serious consideration must also be given to the potential development of their playing ability to achieve a professional level. The first rule of medicine, to “do no harm,” may be a wiser advice to these athletes.

REFERENCES
The Last Word

Cornelius Olcott, IV, M.D.

The comments by Dr. Yao are appreciated by the authors. In fact, it was at Dr. Yao's urging that we persisted with our evaluation of our first patient with hand ischemia. This work-up ultimately led to the diagnosis of an axillary branch artery aneurysm, a diagnosis that Dr. Yao had very astutely predicted.

Dr. Yao's group at Northwestern and our group at Stanford have shared a growing interest in vascular problems in athletes. From review of his comments, it is apparent that our two groups agree for the most part, in the management of these patients.

Dr. Yao's last statement is perhaps the most important. The adage of "do no harm" is of course appropriate to all patients, but particularly important in this group of patients, who, like surgeons, rely on their upper extremities for their living.