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Bony glenoid augmentation complicated by late traumatic axillary artery pseudoaneurysm and disseminated intravascular coagulation

Daniel Bechtold, MD, Kimberly Bartosiak, MD, Alexander W. Aleem, MD*

Department of Orthopaedic Surgery, Washington University School of Medicine, St. Louis, MO, USA

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Recurrent glenohumeral instability is a common problem in the young, active patient population. Although incidence is variable depending on the activity level, this diagnosis is most commonly seen in an athletic or military individual with at least 23% of all shoulder injuries reported in the National Collegiate Athletic Association being attributed to glenohumeral dislocation.[21] Recurrence rates demonstrate similar variability, but in a young active individual can be estimated at 28.6%.[15] After recurrent traumatic anterior dislocation, bony lesions of the glenoid rim have been demonstrated in up to 90% of patients.[22] It is increasingly recognized that stabilization of these injuries must account for bony defects when present to reduce risk of treatment failure and subsequent recurrent dislocation.[3,4,14]

Bony stabilizations using coracoid transfer were introduced by Latarjet and Helfet in the 1950s and have served as an attractive option for glenoid reconstruction given the close proximity of the coracoid for bony augmentation.[11,16] The Latarjet procedure provides a 3-fold effect of increasing stability with transfer of the conjoint tendon that acts as a sling, increasing the glenoid surface area with the bone transfer, and using the coracoacromial ligament stump to reinforce the anterior capsule.[11,16,23] These procedures have long been common in Europe but have demonstrated increasing popularity in the United States, with the total number of Latarjet procedures performed nearly doubling over a 4-year period from 2007 to 2011.[9] With an estimated near 7000 patients undergoing shoulder stabilization procedures each year in the United States, it is important to define the complications of these procedures.[9] Although these procedures have been largely successful in providing anterior glenohumeral stability with recurrence rates as low as 6%,[7,12,19,20] they do alter the native anatomy in close proximity to the axillary artery and brachial plexus providing avenue for a unique and potentially devastating set of complications. In this case report, we present a case of delayed axillary artery pseudoaneurysm complicated by disseminated intravascular coagulation (DIC) resulting in limb loss and significant medical morbidity.

Patient history and diagnosis

A 45-year-old right-hand–dominant man presented to the emergency department in acute distress with new onset severe right upper extremity pain and loss of function. The patient gave history of recurrent traumatic anterior right shoulder instability throughout adolescence and early adulthood without any associated neurovascular injuries. He had subsequently undergone an uncomplicated bony stabilization procedure (Bristow) in the late 1990s, which

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*Reprint requests: Alexander W. Aleem, MD, Campus Box 8233, 660 South Euclid Ave, St. Louis, MO 63110, USA.

E-mail address: aleema@wustl.edu (A.W. Aleem).

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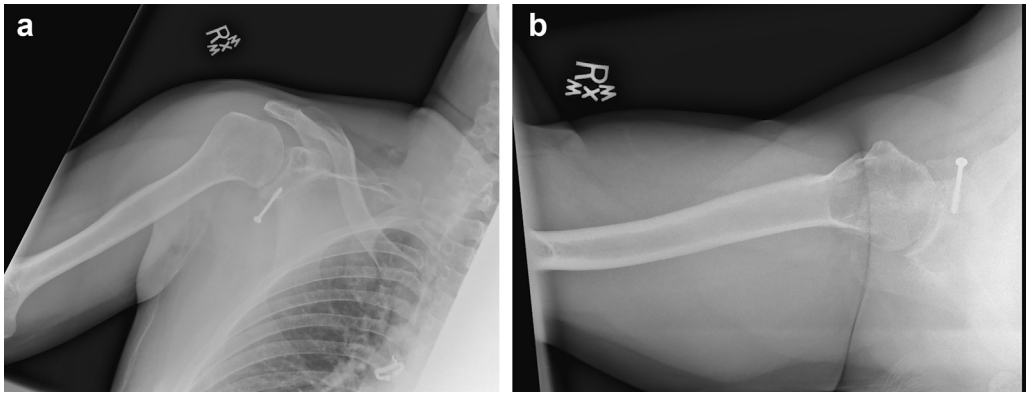

Figure 1 (**a**) True anteroposterior radiograph demonstrating a single partially threaded screw in glenoid with evidence of screw pullout. (**b**) Axillary radiograph demonstrating a single partially threaded screw in glenoid.

resolved his instability. Over the last decade, he had noted frequent pain and numbness radiating into his right hand with overhead activities, in particular with throwing a baseball. In the month before presentation, he reported increasingly frequent episodes of numbness, paraesthesias, and shooting pains that would resolve gradually without intervention. Thus, he did not seek medical attention. He had no significant past medical history.

On the day of presentation, he described atraumatic onset of severe pain in his right arm starting around 14:00. This progressed over the next several hours to the point that his arm began feeling cold to the touch and he noted color changes throughout his hand, prompting presentation to the emergency department at 19:30. On examination, the patient was writhing in pain. His hand was cold and pale with tense compartments and severe pain with passive range of motion of the fingers. His fingertips were notable for bluish coloration of all of his nailbeds and mottling of the skin. There was decreased sensation to light touch throughout median, ulnar, and radial distributions with 2-point discrimination greater than 25 mm in median and ulnar distributions. He had preserved axillary sensation. Motor function was as follows: 4/5 deltoid, 2/5 biceps and triceps, 0/5 wrist flexors, wrist extensors, intraosseous, extensor pollicis longus, flexor pollicis longus, extensor digitorum communis, and flexor digitorum profundus to digits 2-5. Notably, the patient did have a palpable radial pulse and an audible Doppler signal in his ulnar artery.

Radiographs (Fig. 1, *a* and *b*) demonstrated a single partially threaded screw from the prior bony augmentation procedure of the right glenoid that had partially backed out. A computed tomography angiogram (Fig. 2) demonstrated an intraluminal abnormality in the right mid-axillary artery immediately adjacent to the screw and brachial artery occlusion in the mid right arm with minimal to no reconstitution of the distal brachial artery, radial artery, or ulnar artery. There was apparent nonunion of the coracoid bone block. The patient was diagnosed with Rutherford IIB-III acute limb ischemia by the vascular surgery team and was taken to the operating room emergently with orthopedic surgery available for hardware removal and the hand surgery team available for possible forearm fasciotomies.

Patient treatment

The patient was taken to the operating room with the vascular surgery team for formal angiography that revealed aneurysmal change with intra-arterial material in the segment of the right mid-axillary artery in close proximity to his prior bony glenoid augmentation procedure (Fig. 3). On arrival to the operating suite, the patient was noted to be in DIC with lab values as follows: INR greater than 9, apTT greater than 150 seconds, platelet count of 64,000, D-dimer greater than 40,000 ng/mL, and fibrinogen less than 60. He then underwent

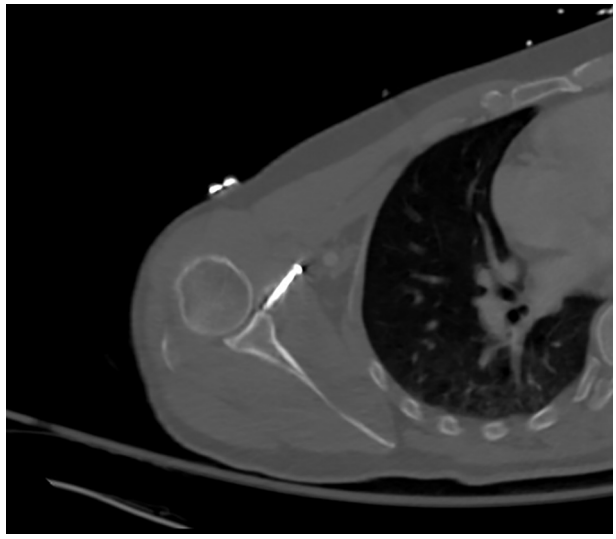

Figure 2 Axial cut of computed tomography angiogram demonstrating pseudoaneurysm of the axillary artery at the location of the screw and evidence of nonunion.

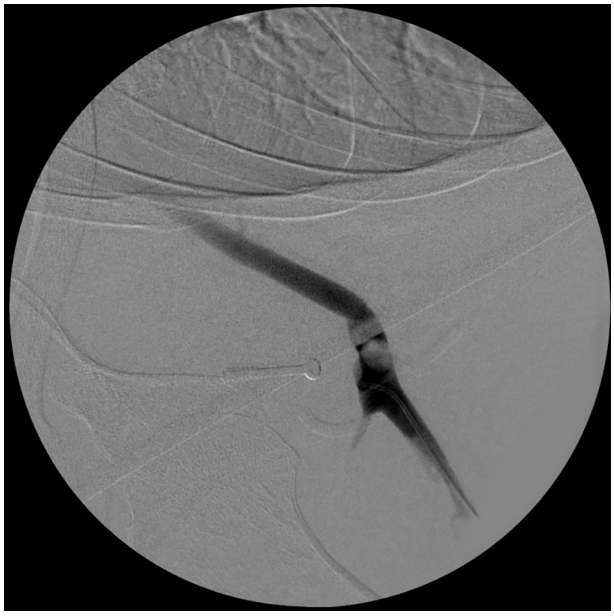

Figure 3 Intraoperative angiogram demonstrating aneurysmal change with intra-arterial material in the segment of the right mid-axillary artery immediately adjacent to the screw.

cutdown thrombectomy of the brachial and radial arteries. The vascular surgery team then turned their attention proximally to the injured axillary artery. A deltopectoral approach was used to examine the axillary artery that was deemed irreparable, and a bypass was subsequently performed from the proximal to distal axillary artery with a polytetrafluoroethylene graft.

Given the prolonged ischemic time, forearm and hand fasciotomies as well as carpal tunnel and Guyon's canal release were performed. Fasciotomy incisions were notable for unhealthy muscle that improved in color throughout the case. The deltopectoral interval used for the bypass was then used to remove the single 2.7 mm partially threaded screw that had backed out only partially from the glenoid. The transferred bone block was palpably stable. Fasciotomy wounds were all left open, whereas wounds about the shoulder were closed. Despite intraoperative resuscitation, the patient had continued evidence of ischemia with intraoperative arterial pH of 7.20 and lactate of 6.7. The patient was then transferred intubated and sedated with a palpable radial pulse to the intensive care unit on 0.1 mcg/kg/min of norepinephrine that was weaned within 1 hour of arrival to intensive care unit.

Postoperative course

On the morning of postoperative day 1, the patient was evaluated in the surgical intensive care unit (SICU). At that time his hand was cold to the touch and unperfused, and he was clinically deteriorating. He had developed hyperkalemic metabolic acidosis with acute aneuric renal failure and continued to require large quantities of cryoprecipitate due to DIC. The plastic surgery team evaluated the fasciotomies and felt that the underlying muscle was nonviable. It was determined that the patient was developing life-threatening metabolic abnormalities as a result of tissue necrosis and the decision was made to return to the operating room to evaluate the viability of the patients forearm musculature.

Intraoperatively, the muscle bellies of the forearm, biceps, and triceps were found to be grossly necrotic with no muscular response to electrocautery or bleeding of tissue distal to the deltoid. Given the patient's morbid condition and these findings, an above elbow amputation was performed at the level of the pectoralis major insertion. Pathologic specimen was sent and final report noted extensive ischemic necrosis of the deep soft tissues in the delivered specimen.

After amputation, the patient required a further stay in the SICU for medical stabilization. He was started on continuous venovenous hemodialysis in the SICU by the nephrology team and eventually required placement of tunneled venous catheter before discharge for continued outpatient dialysis. He was extubated on hospital day 3 when his metabolic status was improving. He required 3 further trips to the operating room for subsequent débridement of nonviable muscle in his wound. He was maintained on broad spectrum antibiosis until after closure of his right upper extremity surgical wound. His wound was managed with an incisional negative pressure dressing, and showed evidence of healing at the time of discharge. He was transferred out of the SICU on hospital day 7 and discharged to home on hospital day 12 in stable condition, but required dialysis for renal support. Throughout his hospital course, he was treated with subcutaneous heparin for DVT prophylaxis given his renal injury and intravascular coagulation.

Three weeks postoperatively he underwent targeted muscle reinnervation of the pectoralis major with the goal of minimizing phantom symptoms and creating a regenerative peripheral nerve interface with a goal of prosthetic placement to return to work and playing guitar. At the time of submission, his wound is healed and he has undergone prosthetic fitting with the orthotics and prosthetics service. His right upper extremity stump is sensate and he is able to fire his deltoid and pectoralis major. He has been off dialysis for 5 months with improvement in baseline creatinine but still in stage IV chronic kidney disease. Despite targeted muscle reinnervation he has developed significant phantom limb pain that disrupts his sleep and that has been difficult to medically control in the setting of renal dysfunction. His postoperative rehabilitation was slowed by a zone 1 flexor tendon injury to his left index finger; however, he reports he is now able to use his left hand to accomplish activities of daily living. He has been working with occupational therapy with the goal of being able to return to guitar playing

and was able to return to his work as an educator 4 months after his initial presentation.

Discussion

Bony glenohumeral stabilization procedures have been a widely successful procedure performed in a primarily young and healthy population. The reported complications of these procedures include damage to the axillary neurovascular bundle.[10] To our knowledge, only 5 prior cases of axillary artery pseudoaneurysm have been reported with only 1 occurring greater than a decade after the index procedure.[1,5,6,8,13] Our case is unique from the prior remote case in that our patient had no antecedent neurovascular compromise.[5] To our knowledge, this is the only reported case of loss of extremity resulting from a complication of these procedures.

Consistent with the case presented by Cappello et al,[5] it was noted that the screw used to fix the coracoid tip had migrated from its original position and was in close proximity to the pseudoaneurysm. In addition, both patients had described transient ischemic episodes in the months preceding the sentinel event. Such reports should be interpreted with caution in patients who have had bony stabilization procedures even if first developing remote to the index operation. Nonunion or fibrous union after coracoid transfer is a known complication and has been reported at 9.3%.[10] Given the risk to neurovascular structures with construct loosening and screw backout, we recommend consideration of more robust fixation constructs than a single partially threaded screw when performing these procedures.

Embolic disease in this patient set off a coagulopathic cascade that required urgent intervention by the treating team. DIC is most frequently encountered in the setting of sepsis, trauma, or obstetric complications but has previously been described as a complication of atheroembolism from the ascending aorta.[2,17] The presented case brings to light the possibility of DIC in patients with embolic disease and an ischemic limb. Identification of the process requires a high index of suspicion by the treating surgeon and anesthesia team. There is no single lab test to confirm the diagnosis; therefore, early identification of a disease process with potential to activate the intravascular clotting cascade in conjunction with prolonged clotting times, evidence of fibrin degradation, and a platelet count less than 100,000 is needed for diagnosis.[18] The treatment of DIC is focused on control of the underlying disorder; however, supportive measures including platelet and factor replacement may be necessary to allow for adequate hemostasis and safe exit from the operating theater.[17,18] Despite bleeding risk, surgeons should give early consideration to therapeutic anticoagulation in these patients to avoid further thromboembolic disease.[18]

Conclusion

This case highlights the importance of not ignoring transient upper extremity pain and numbness in a patient after the coracoid transfer procedure. In this setting, interpreting radiographs carefully for evaluation for any loss of screw fixation is critical to avoiding such devastating complications as observed in this case. We also recommend robust fixation for any open coracoid transfer procedure with at least 2 screws that traverse across entire glenoid.

Disclaimer

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