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Shoulder arthroplasty death with axillary artery and brachial plexus damage: lessons from a tragedy

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Total shoulder arthroplasty (TSA) is a very safe procedure, with an in-hospital mortality rate of just 0.09%.[21] The complications of TSA are commonly described as prosthetic loosening, glenohumeral instability, periprosthetic fracture, infection, rotator cuff tear, nerve damage, and deltoid dysfunction.[3] Very rarely are more serious complications described in the literature, such as brachial plexus injury[22] or axillary artery damage.[26] Even outside of TSA, damage to the axillary artery is rare and overwhelmingly described in the literature as a consequence of shoulder dislocation.[11] When it is discussed in conjunction with shoulder arthroplasty, most of the literature states that damage to the axillary artery is a risk during reverse shoulder arthroplasty,[10,26] with a paucity of information available about the risk during anatomic TSA.[8,26] In fact, our literature review could identify only 2 cases that described axillary artery injuries occurring during TSA.[26] Similarly, brachial plexus injuries during TSA are very rarely encountered in the literature.[22]

We present the case of a patient experiencing both iatrogenic axillary artery and brachial plexus injury during elective TSA for glenohumeral arthritis at an outside facility, most likely due to a centralizing glenoid threaded Steinmann pin. Revascularization was attempted more than 24 hours after the onset of ischemia, leading to rhabdomyolysis, multisystem organ failure, and death.

Case report

A 79-year-old female patient underwent an elective TSA for glenohumeral arthritis at a private hospital performed by a general orthopedic surgeon, with a low volume of shoulder arthroplasty procedures. The patient had a medical history significant for stroke, sick sinus syndrome with implantation of a pacemaker, insulin-dependent diabetes, right-sided breast cancer treated by a lumpectomy, hypoalbuminemia, obesity, and hypertension. Preoperative radiographs showed Walch type C glenoid morphology, so an anatomic TSA was planned. The patient received a preoperative interscalene nerve block, and the case was finished around 10:30 AM. The surgeon did not note any heavy intraoperative bleeding or other neurovascular complications intraoperatively and noted no muscular twitches during the procedure.

Postoperatively, the patient described paresthesias and loss of motor function in her right upper extremity (operative limb) but these were attributed to the nerve block, and normal postoperative care was provided with no further workup at the time. The next day, the patient continued to complain of paresthesias, and skin mottling was also noted by the family. This was brought to the attention of the surgical team around 11 AM on postoperative day (POD) 1 (25 hours after surgery), and a computed tomography (CT) angiogram of the right upper extremity was obtained. The CT angiogram showed loss of contrast in the axillary artery and a lack of reconstitution distally (Fig. 1). A right-sided pneumothorax was also noted incidentally, and a chest tube was placed. At that time, the patient was transferred to our emergency department for further management of an iatrogenic axillary artery injury and iatrogenic pneumothorax.

The patient was admitted to our facility at 2 PM and evaluated immediately for both injuries. The pneumothorax showed radiographic improvement since chest tube insertion at the outside facility. However, on physical examination of the arm, pallor, poikilothermia, paresthesias, pain, a pulseless state, and paresis were noted. No radial, ulnar, or brachial pulses could be identified through palpation or Doppler ultrasound. The vascular surgery team was emergently consulted and discussed with the family the

No institutional review board approval was required for this case report.

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Figure 1 Computed tomography arthrogram with 3-dimensional reconstruction showing lack of distal arterial flow within axillary artery.

risk of attempting revascularization more than 24 hours after the onset of ischemia. The family, patient, and surgical team decided together that revascularization should be attempted, and the patient was taken to the operating room around 3:30 PM for axillary artery exploration with possible revascularization and fasciotomies. Our orthopedic service was called at 7:30 PM to perform hand fasciotomies.

Intraoperatively, the vascular surgery team noted that the brachial artery was transected and the proximal portion of the artery was twisted around itself multiple times in a "corkscrew" fashion, along with disruption of the brachial plexus (Fig. 2). Further dissection revealed that the entire visible vessel was twisted in this fashion, suggesting this was the case all the way up until it reached the distal subclavian artery. At this time, the vascular surgery team performed a bypass procedure connecting the right common carotid artery to the distal, uninjured brachial artery. The vascular surgery team then performed a forearm fasciotomy, noting that all the muscle groups were unresponsive to electrical stimulation via electrocautery. The orthopedic service then arrived and performed hand fasciotomies, noting "gray-appearing noncontractile muscle"; the possibility of amputation was discussed, and concern was noted regarding reperfusion injury due to the prolonged ischemia. At the conclusion of the case, the radial pulse was identifiable with palpation and Doppler ultrasound.

Postoperatively, the patient experienced acute respiratory failure and remained intubated with mechanical ventilation. By 10 PM on POD 1, the patient had lactic acidosis with a lactic acid level of 5.2 and pH of 7.34. By 4 AM on POD 2, more metabolic signs of reperfusion injury were present including hyperkalemia (potassium [serum] level, 6.5 mmol/L), acute kidney injury (creatinine [serum] level, 1.68 mg/dL [baseline, 0.89 mg/dL]), hypocalcemia (calcium [serum] level, 7.8 mg/dL), and mild transaminitis (aspartate aminotransferase level, 145 units [U]/L; alanine aminotransferase level, 66 U/L). Three hours later, the patient was found to be

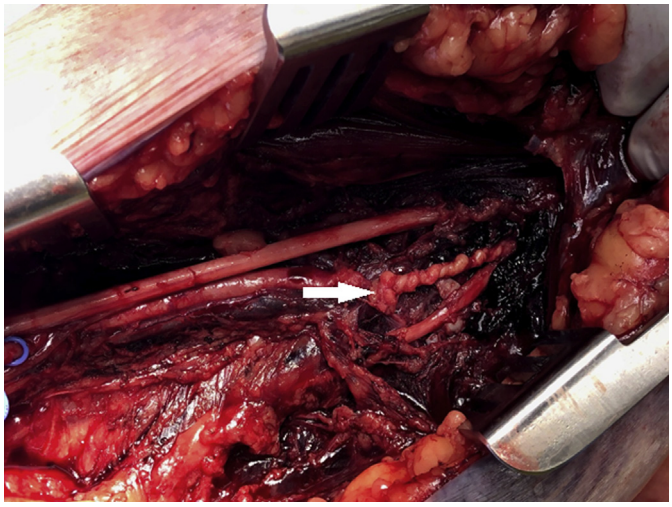

Figure 2 Corkscrew appearance of transected axillary artery (*arrow*), with surrounding injured brachial plexus.

in a state of rhabdomyolysis with a creatine kinase (CK) level of 11,636 U/L.

At noon on POD 2, hypotension developed with systolic blood pressure (SBP) in the 70s and hypoxemia. Laboratory testing at this time showed further biochemical derangements (CK level, 14,244; lactic acid level, 9.7; pH, 7.22; creatinine [serum] level, 2.05 mg/dL; potassium [serum] level, 6.4 mmol/L; calcium [serum] level, 7.7 mg/dL), and a chest radiograph showed worsened right-sided pneumothorax. Two units of packed red blood cells were given, and the chest tube was replaced. Blood pressure stabilized with SBP in the 90s, and the patient was admitted to the intensive care unit for aggressive medical management. The nephrology department was consulted for continuous renal replacement therapy, which was started shortly thereafter.

Overnight, the patient required 3 U of fresh-frozen plasma and 2 U of packed red blood cells. On the morning of POD 3, her SBP was stable in the low 100s and she was at least minimally responsive for the first time since the bypass procedure. However, the laboratory values continued to show severe abnormalities with a CK level of 15,000, and there was concern for cerebrovascular accident due to anisocoria and lack of left-sided movement in either extremity. Around 3 PM on POD 3, the patient became acutely hypotensive and was given 2 U of fresh-frozen plasma, and administration of vasopressors was started, as she was nonresponsive to fluid boluses. Within 3 hours, intensive care unit staff had a discussion with the family indicating that amputation of the extremity was the patient's only chance for survival. The family declined and withdrew further life-sustaining therapy. At 9:24 PM on POD 3, the patient died due to rhabdomyolysis leading to multisystem organ failure.

Discussion

This case demonstrates many principles of which orthopedic surgeons should remain cautious and mindful. We have considered this case in the following 4 ways: (1) Preoperative interscalene blocks may mask potentially salvageable devastating iatrogenic injuries. (2) Low-volume surgeons need greater support and input when performing certain higher-risk procedures. (3) Surgical training has used technology as a surrogate for better clinical and surgical skills. (4) Limb salvage guidelines need to be followed more strictly.

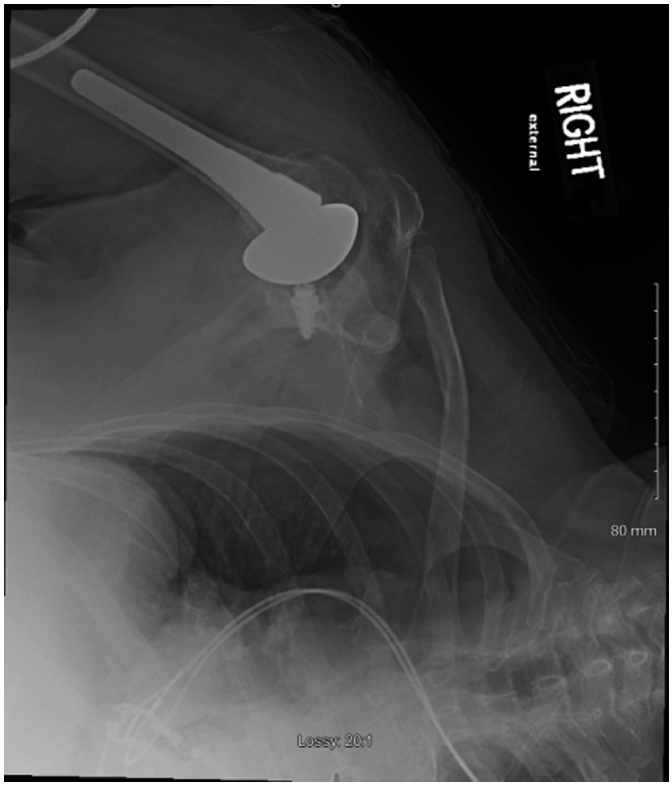

Figure 3 Postoperative anteroposterior radiograph.

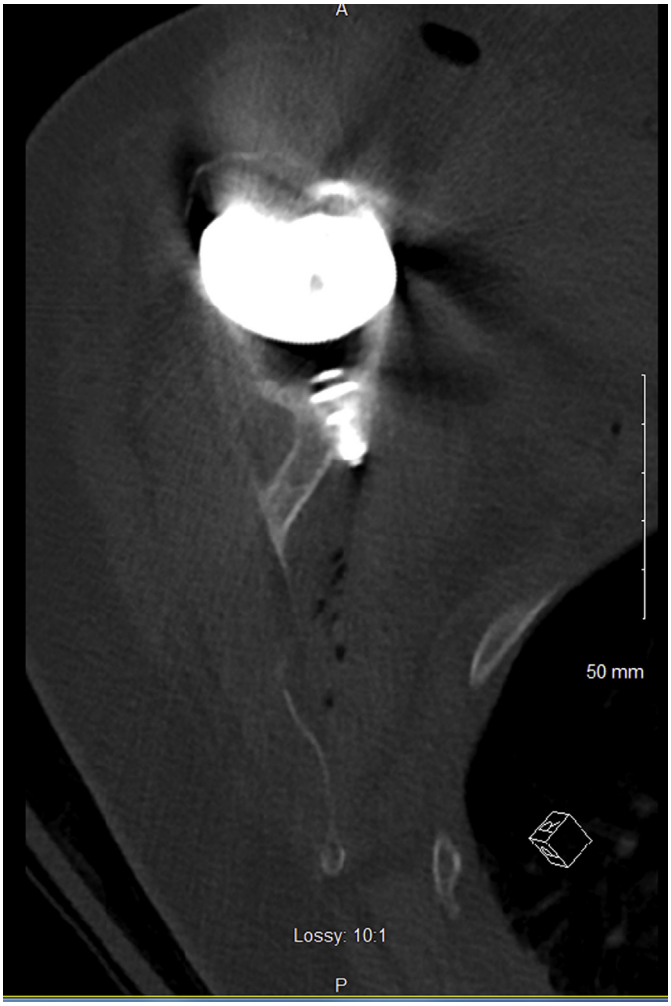

Figure 4 Axial computed tomography scan demonstrating Walch type C glenoid with central prosthetic glenoid peg transgressing anterior cortex (subsequent slices located it medial to coracoid base). *A*, anterior; *P*, posterior; *R*, right.

Regarding the first aspect, preoperative interscalene nerve blocks are commonly used to obtain postoperative shoulder arthroplasty pain control.[2] As seen in this case, symptoms of iatrogenic nerve damage may be initially dismissed as effects of a nerve block because of their similarity in the immediate postoperative period, notably loss of motor function or altered sensation.[5,10] This is an understandable early approach, as the rate of iatrogenic nerve injury during TSA is around 0.3%[18] whereas the rate of similar effects from a peripheral nerve block approaches 100% when performed correctly,[1,5,7] with a complete nerve block occurring 93% of the time.[7] The typical effects of a peripheral nerve block subside after 8 hours or more, which may also be considered 8 hours or more after an iatrogenic neurovascular injury occurs.[1] As seen in this case, the interscalene block contributed to a delay in both the diagnosis and treatment of concomitant iatrogenic neurovascular injuries, particularly as no unexpected bleeding was noted intraoperatively.

The preoperative nerve block also contributed to the occurrence of the devastating outcome in this patient as it often leads to complete motor paralysis.[1,5,7] In the absence of such a nerve block, the surgeon would have understood that he was encroaching on the brachial plexus owing to movement of the patient. This would have provided some feedback and perhaps allowed reorientation. This is a reason that the role of preoperative nerve blocks, although commonly used, should be considered a potential confounder in the postoperative neural status evaluation. The latter point must be borne in mind when considering that interscalene nerve blocks have been shown to provide no benefit for pain control after 8 hours.[1] The senior author does not use preoperative nerve blocks.

Regarding the second aspect, low-volume shoulder arthroplasty surgeons account for a significant total percentage of shoulder arthroplasties performed.[28] In contradistinction, high-volume surgeons have better clinical outcomes and lower complication rates,[13,15] although this finding is not unique to shoulder arthroplasty.[16,20] Many factors account for the 2 former issues, some of which are not documented: anatomic and surgical training of the surgeon; volumes, outcomes, and complications of individual shoulder surgeons; infrastructure afforded to a surgeon in tight fiscal environments; facility to refer difficult cases to a higher-volume surgeon; and so on. A low-volume generalist surgeon is unlikely to have detailed knowledge about less common atypical anatomy, especially in joints less routinely encountered in practice such as shoulders compared with hips and knees. Although low-volume surgeons should be wary of taking on high-risk patients or cases involving atypical anatomy, the real challenge is for such surgeons to correctly identify atypical pathoanatomy. In this case, the surgeon performing the TSA misinterpreted the preoperative radiographs and failed to appreciate the atypical glenoid morphology or the trajectory of the glenoid vault. Perhaps with better training and the institutional support to allow for patient-specific instrumentation, this would have been avoided. This misinterpretation led to the threaded Steinmann pin being drilled anteromedial to the coracoid base, a region occupied by the axillary artery and brachial plexus. As a result of the nerve block, there was no limb movement when the threaded Steinmann pin penetrated the far cortex (Figs. 3 and 4) and entered and disrupted the brachial plexus. The lack of heavy intraoperative bleeding can be explained

by the corkscrew-like appearance of the axillary artery, a result of the artery being caught and spun around the rotating threaded Steinmann pin. With the twisting arterial disruption, the endothelium may seal each end of the rupture site, thereby preventing significant overt bleeding.

A technical detail of relevance is the use of a power drill with a very sharp threaded Steinmann pin, which is used as a central glenoid guide pin. When a power drill is used, the surgeon's tactile feedback is naturally dampened so that if the far cortex, in this case the anterior glenoid, is less robust than other more common cortices encountered by the general orthopedic surgeon, such as the femur and tibia, the awareness of cortical penetration may be deficient. This lack of awareness of cortical penetration may have led to a continued anterior progression of the pin that, once tethered with coiled soft tissues, is difficult to withdraw without placing the drill in the reverse setting. The basic surgical understanding of when the far cortex was breached by the Steinmann pin was lacking, which appears to be more widespread than reported, suggesting a deficiency in how surgeons are trained.

Regarding the third aspect, surgical training has been under pressure for some time, with the productive time spent in specialty training decreasing. In addition to decreasing training time, the opportunity for surgical training is decreasing because of the productivity demands placed on attending surgeons. These 2 issues are deteriorating the skill level of trainees. To counteract this trend, more technologies such as navigation, patient-specific instrumentation, and robotic surgery are being used to bridge the skill gap in the guise of operative efficiency and precision. The use of preoperative 2- and 3-dimensional CT scans has been well described in the TSA literature, especially for patients with severe arthritis and/or abnormal glenoid morphology.[4,23] There is some evidence that 3-dimensional scans may allow the surgeon to more accurately visualize regional anatomy preoperatively.[6,9,24] Surgical navigation systems are another promising technology that may increase a surgeon's awareness of regional anatomy and allow for more accuracy and precision.[25] However, although these technologies have their place in the health care industry, they should not be a surrogate for supervised, hands-on, repetitive, high-volume training of surgical candidates.

Finally, regarding the fourth aspect, limb devitalization and revascularization guidelines are well researched and published.[12,14,19] Both tissue type and temperature have implications for acceptable delays in revascularization.[12,14,19] Skeletal muscle has been shown to have the shortest tolerable ischemic time, with periods of only 2 to 4 hours for warm ischemia and 6 to 8 hours for cold ischemia.[12,19] The recommendation is to avoid replanting or performing revascularization of the upper extremity proximal to the wrist after these periods.[19] This case demonstrates that the delay in the diagnosis of a warm ischemic arm, by a CT arthrogram, was more than 24 hours. Initial ischemia led to rhabdomyolysis and caused local accumulation of byproducts of lactic acidosis and electrolyte derangements.[12] Subsequent revascularization distributed products of rhabdomyolysis into the systemic circulation and predictably led to multi-organ failure. At this stage, aggressive medical management including fluid resuscitation and continuous renal replacement therapy cannot predictably salvage multi-organ failure. This case demonstrates the severe and potentially fatal risks of late revascularization and the potentially life-saving nature of amputation.

Conclusion

We have presented the case of a 79-year-old patient who underwent a TSA and had iatrogenic axillary artery and brachial plexus injury, from a threaded Steinmann pin, that was not recognized postoperatively for at least 24 hours. Delayed revascularization led to systemic distribution of rhabdomyolysis products, multisystem organ dysfunction, and death. This case highlights the importance of proper case selection, regional pathoanatomic knowledge, preoperative image analysis, relevant surgical training and skills, hypervigilant neurovascular monitoring after shoulder arthroplasty with interscalene blocks, and the option of amputation if early revascularization is not possible. It demonstrates the potential danger of regional nerve blocks, complex surgical procedures performed by low-volume surgeons, and deviation from well-established revascularization protocols.

Disclaimer

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