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Case Report

Occult thoracoacromial artery injury during reverse total shoulder arthroplasty resulting in a cascade of postoperative complications: A case report

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ABSTRACT

We report a case of a 78-year old female patient got the reverse total shoulder arthroplasty with a series of events. After diagnosed with anterior shoulder dislocation and coracoid process fracture at a local hospital, she received conservative care after reduction but persistent reduction loss occurred. Preoperative Magnetic Resonance Imaging confirmed underlying massive fatty infiltration and severe retraction of rotator cuff muscles. Considering underlying chronic rotator cuff arthropathy on preoperative X-ray and Computed Tomography scan with irreparable rotator cuff tears, we decided to perform reverse total shoulder arthroplasty. After unexpected vessel injury possibly due to underlying bleeding tendency or intra-operative procedure, severe complications occurred including active arterial bleeding, brachial plexus palsy and skin necrosis. Serial managements which included embolization of the artery, wound management while implant exposure status, and operative coverage of a skin defect by Latissimus Dorsi pedicled flap and Split Thickness Skin Graft were done. This series of events suggests that surgeons should be more careful than we were about possible injuries of small vessel branches that can cause unexpected complications, and keep in mind the importance of immediate cooperation with other medical professionals such as radiologists, plastic surgeons, and thoracic surgeons.

Introduction

In recent years, reverse total shoulder arthroplasty (RTSA) has become increasingly popular for improvement of functional outcome. Yet, multiple complications associated with RTSA have been reported. Intraoperative complications include glenoid fracture, implant malposition and nerve injury, and common postoperative complications are infection, shoulder dislocation, scapular notching, stress fracture of the acromion, and glenoid loosening [1–7]. We report a postoperative event that has not been reported so far. Before writing this report, we obtained informed consent from the patient and patient's son, who is a legally authorized representative.

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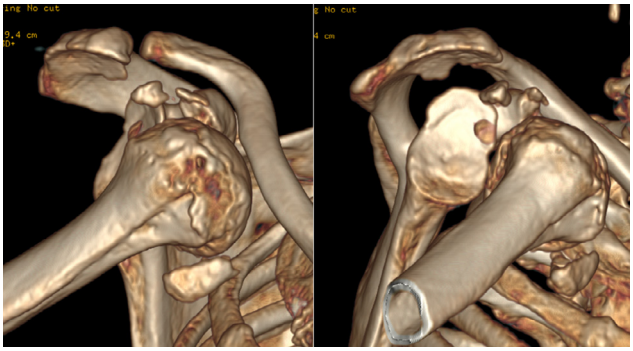

Fig. 1 – Initial 3D CT images of the coracoid process fracture and anterior dislocation.

Case Report

A 78-year-old female patient visited our hospital with a chief complaint of right shoulder pain and loss of motion. She had been diagnosed with a shoulder dislocation and coracoid process fracture at a local hospital after slipping down 1 week before the visit. She had received conservative care after reduction, but persistent reduction loss occurred, and eventually, the patient was transferred to our hospital for further treatment. The patient was taking aspirin due to a history of hypertension and atrial fibrillation. Distal motor and sensory functions were intact, the initial X-ray and computed tomography (CT) scan showed anterior dislocation of the shoulder joint and coracoid process fracture. Also, X-ray and CT scan showed underlying chronic rotator cuff arthropathy with less than 5 mm of acromiohumeral interval and acromion acetabularization which correspond to Hamada grade 3. (Fig. 1) MRI confirmed massive rotator cuff tears, periarticular fluid collection and fatty infiltration of rotator muscles. Considering advanced fatty infiltration and severe retraction of rotator cuff muscle, underlying massive rotator cuff tear was suspected on supraspinatus, infraspinatus, and subscapularis, which all correspond to Goutallier grade 4. Subscapularis also shows severe medial retraction on glenoid level but we couldn't seem to prove a direct relationship between recent repetitive anterior dislocation and subscapularis massive tear. (Fig. 2) CT and MRI were performed immediately after a reduction on the day of the visit. Considering old age, current medical status, and persistent reduction loss with irreparable rotator cuff tears, we decided to proceed with RTSA surgery.

The surgery was performed 1 week later after arrival, using the deltoid-pectoral approach, with the patient under general anesthesia and in the beach-chair position. Complete tears of the subscapularis tendon that were retracted to the level of the glenoid were confirmed. A Comprehensive Reverse Shoulder System (Biomet Inc., Warsaw, IN, USA) was implanted. The baseplate well covered the glenoid and the stem was press-fit; postoperative soft-tissue gas was found on X-ray. (Fig. 3) No intraoperative active bleeding was detected. A severely retracted subscapularis tendon was not reattached. Appropriate tension was confirmed after implant insertion and a Hemovac drain was placed into the joint cavity. Postoperative X-ray findings and neurologic symptoms were nonspecific. Her postoperative hemoglobin level was 12.2 g/dL and vital signs were stable.

Eight hours after the end of surgery, she complained of mild dyspnea and swollen operated shoulder. Her mental status was alert at that time, but her systolic blood pressure was 80 mmHg and O_2 saturation was 71%. Her right shoulder was swollen (Fig. 4), and the Hemovac drainage volume was 100 cc. Laboratory results at that time revealed a hemoglobin level of 5.4 g/dL (initial 12.2), a platelet count of 51,000 albumin level of 1.5 g/dL and prolongation of prothrombin time and activated partial prothrombin time. Massive transfusion was performed because of low blood pressure: 18 units of packed red cells, 6 units of fresh-frozen plasma, 30 units of platelet concentrate, and 20 units of cryoprecipitate were transfused for 5 days. Considering the normal ranges of prothrombin time and activated partial prothrombin time before surgery, their prolongation was considered as a temporary change caused by mass bleeding. It was normalized after transfusion of 6 units of fresh-frozen plasma. Therefore, it seems that there was no causal relationship between the persistent bleeding and prolongation of prothrombin time and activated partial prothrombin time.

On postoperative day (POD) 1, swelling and bruising were observed around the operation wound. CT angiography was done to detect any hidden bleeding focus. Although uncertain due to variant branching, the origin of active bleeding was suspected to be the 1st branch of the thoracoacromial artery. CT scan confirmed that the screw was placed correctly and the possibility of damage due to the excessive length and incorrect positon of the screws seemed small. Some arterial injury was suspected possibly due to underlying bleeding tendency or intra-operative procedure. (Fig. 5) In collaboration with a radiologist, embolization through the common femoral artery access and super selection of extravasation artery was tried but failed. (Fig. 6) The reason for the failure of super selective catheterization was that the blood vessel was too small for the guidewire to access, and several attempts to access made the vessel to contract. Serial chest X-ray showed a large hematoma around the chest wall. (Fig. 7)

On POD 5, reduced motor and sensory functions were found below the axillary level and a complete loss of brachial plexus was confirmed. This seemed to be attributable to brachial plexus compression caused by the hematoma. Skin necrosis and loss were also observed around the operation wound. Urgently, a thoracic surgeon was asked for a consultation on open drainage. A 200 cc hematoma was removed under local anesthesia. (Fig. 8) Swelling subsided after incision and open drainage with gauze packing. Since the drainage, her systolic blood pressure, hemoglobin level, and chest X-ray were normalized.

On POD 14, the patient had a reonset of active bleeding after dressing. She was drowsy, with sBP 60 mmHg and Hb 4.8 g/dL. Despite another blood transfusion, her vital signs and mental status were unstable. Embolization was reattempted and was successful on the second attempt. (Fig. 9) As before, the common femoral artery was accessed under ultrasound guidance. Contrast extravasation from a thoracoacromial artery branch was seen on the right subclavian artery

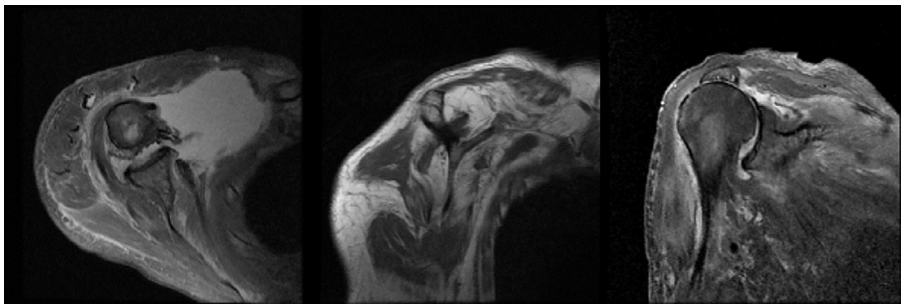

Fig. 2 – MRI images of irreparable rotator cuff tear and fatty infiltration of rotator muscles.

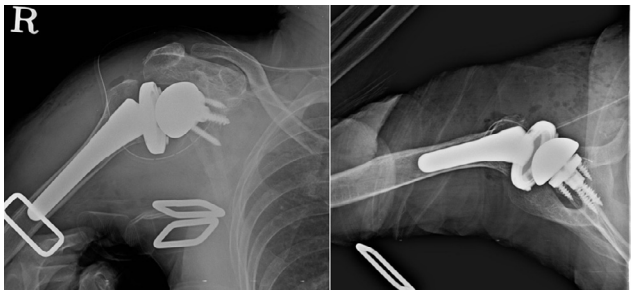

Fig. 3 – X-ray after reverse total shoulder arthroplasty.

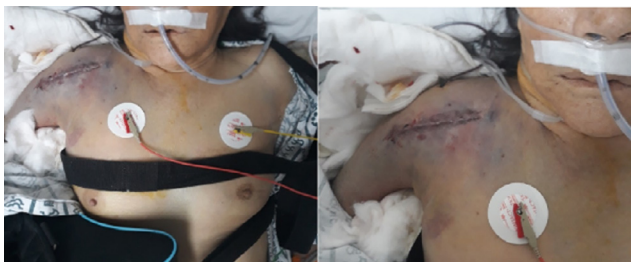

Fig. 4 – Swollen operated shoulder.

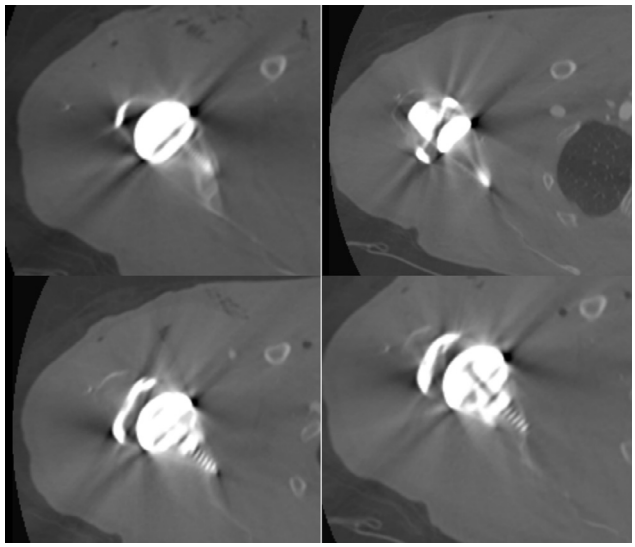

Fig. 5 – CT scan showing a well-fixed glenoid screw.

by digital subtraction angiography. After superselection using Shepherd hook technique, embolization was performed using glue (Histoacryl; B. Braun; the glue was diluted with 0.9% normal saline at a ratio 3:1). Serial follow-up digital subtraction angiography confirmed the absence of any additional contrast extravasation, and the embolization procedure was finished. Swelling around the operation site subsided, and her general condition recovered, with normalized hemoglobin level and systolic blood pressure. Despite continuous dressings, skin necrosis progressed and her brachial plexus palsy did not improve. (Fig. 10) After clear demarcation of normal tissue (POD 47), the removal of necrotic tissue and debridement was carried out in collaboration with a plastic surgeon. The

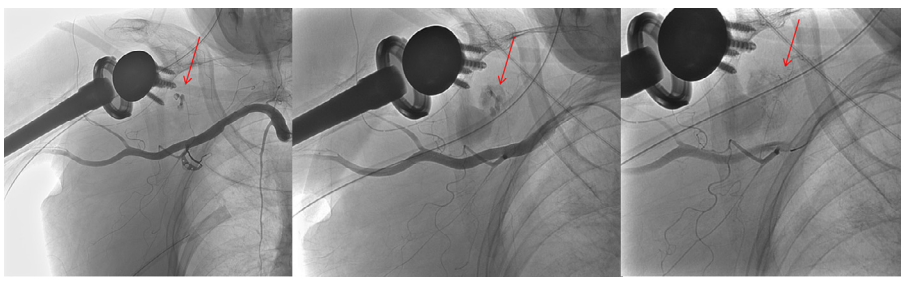

Fig. 6 – CT angiography of bleeding from the 1st branch of the thoracoacromial artery and failed embolization.

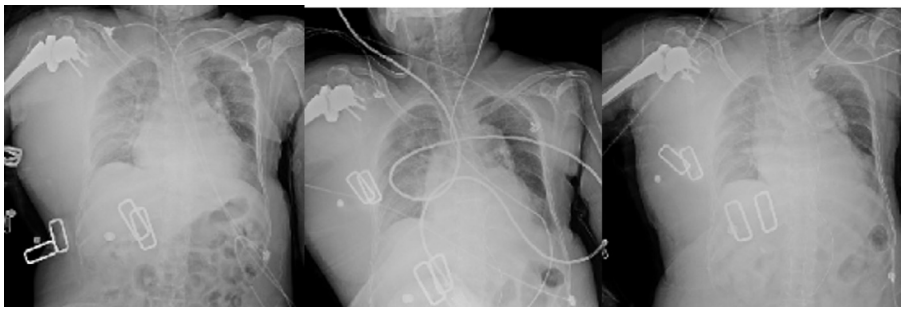

Fig. 7 – Serial chest X-ray showing a large hematoma around the chest wall.

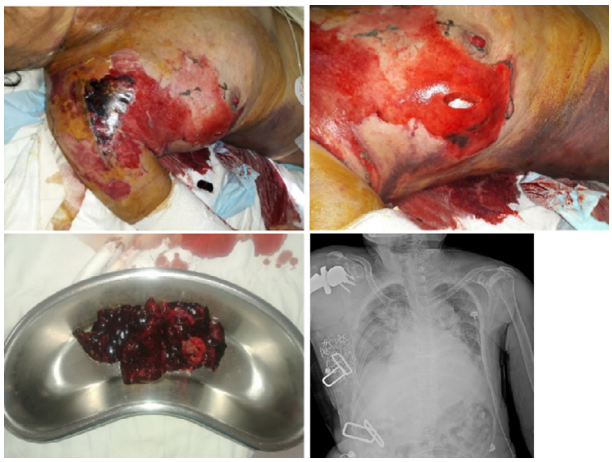

Fig. 8 – 7After the patient complained of mild dyspnea and swollen operated shoulder with brachial plexus palsy symptoms, a 200-cc hematoma was evacuated.

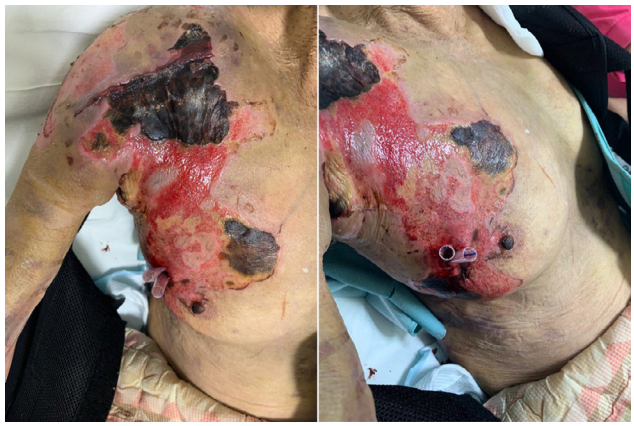

Fig. 10 – Clinical photo showing failed healing of skin necrosis despite continuous dressings.

arthroplasty implant was exposed after debridement. Fortunately, there was no sign of periprosthetic infection. (Fig. 11)

Four months after the operation, soft tissue grew around the skin defect site and no infection was observed. With the aid of a plastic surgeon, a Latissimus Dorsi pedicled flap and Split Thickness Skin Graft were performed to cover the skin defect site. (Fig. 12) The patient's wound has fully recovered and finger-tip sensitivity improved slightly during the first follow-up year. Yet, brachial plexus palsy remains.

Discussion

Although no intraoperative complications were seen in this case report, the first problem we confronted after surgery was decreased Hb levels. The Hemovac drainage volume was 100 cc, and massive transfusion was needed because

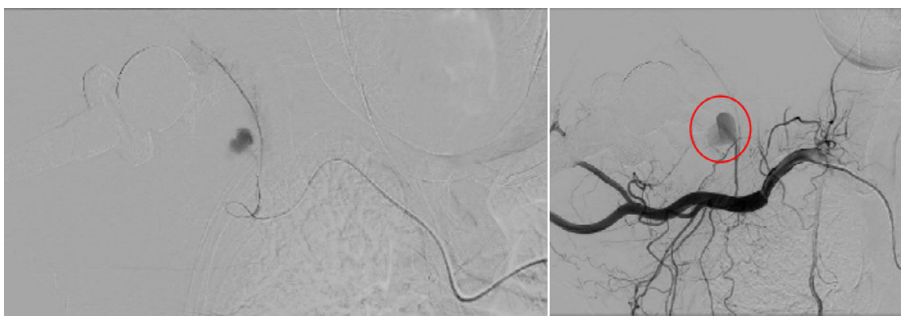

Fig. 9 – CT angiography showing successfully embolization of the 1st branch of the lateral thoracic artery on the second attempt.

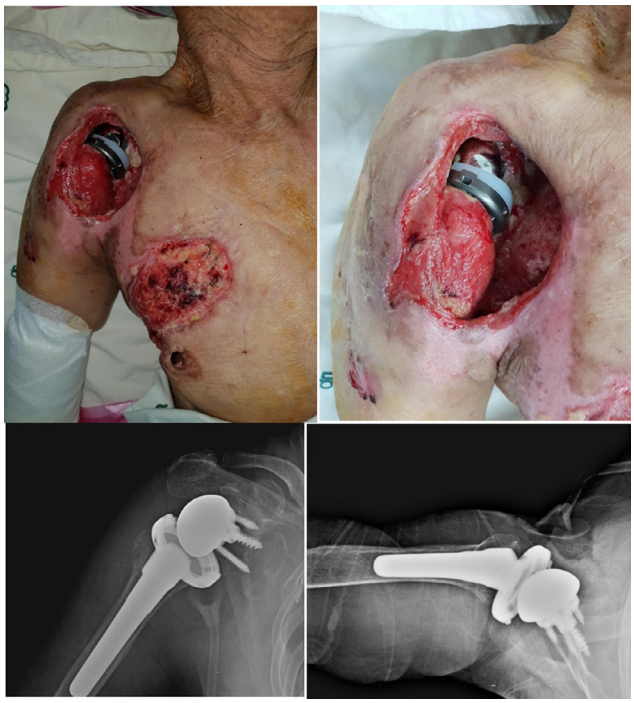

Fig. 11 – The absence of signs of infection around the operation site and X-rays showing no loosening.

of a drastic decrease in Hb concentration and unexpected swelling around the operation site. We suspected that these series of events ware caused by a vascular injury that might have occurred during the surgical procedure or due to bleeding tendency which might cause by aspirin. Considering the severity of swelling, the amount of Hemovac drainage was not too high to suspect active bleeding. A Hemovac inserted into the joint space was functioning well without blood clots. But because of severe swelling and low Hb level, we had to rule out active bleeding and angiography was necessary to accurately identify the bleeding focus. The origin of active bleeding was strongly suspected to be the first branch of the thoracoacromial artery, but could not be confirmed because of many variants associated with this branch. Unfortunately, embolization failed because of the inaccessibility of the vasospasm to wire.

We were unable to clarify the cause of postoperative arterial bleeding. When reaming the glenoid for screw fixation, we carefully penetrated the far cortex and observed no active intraoperative bleeding. However, since arterial bleeding was detected in the direction of the glenoid screw on CT angiography, we cannot exclude injury during reaming. We also suspect that minor trauma may have caused vessel injury because the patient had a heart problem and was taking aspirin. We may have failed to detect intraoperative bleeding because the active bleeding site may have been adjacent to the chest wall rather than the shoulder joint.

The second problem we confronted was brachial plexus palsy. After failure of embolization, the patient complained of sensory loss and weakness in the right upper limb on POD 5. After hematoma evacuation, swelling of the chest wall was relieved, but brachial plexus palsy still persisted at least for 1 year (time of the last hospital visit). The compression of the brachial plexus because of the hematoma might be the cause of complete palsy.

The third problem was skin necrosis. After failure of initial embolization, internal bleeding continued despite hematoma evacuation, and skin condition aggravated. Skin necrosis eventually expanded to the breast, and debridement and coverage were inevitable. Skin necrosis is thought to be a result of shearing of superficial subcutaneous tissues away from underlying fascial layers, which leads to blood filling the cavity in the prefascial plane [8,9,10]. As a result of the separation of these layers, transaponeurotic capillaries and lymphatic vessels become disrupted. These avulsed channels leak lymph and blood into the newly formed cavity and a haemolymphatic collection develops. Similar to Morel-Lavallée lesions that occur in the pelvic area, we found that this kind of lesion can also develop after shoulder surgery. Bleeding was under control after embolization succeeded at the second attempt, and the size of necrosis no longer increased.

Fortunately, infection, which is a complication that we want to avoid the most, did not occur. As the arthroplasty implant was exposed after the removal of necrotic tissue, we paid more careful attention to wound management. Clean soft tissue grew as a result of consistent aseptic dressing,

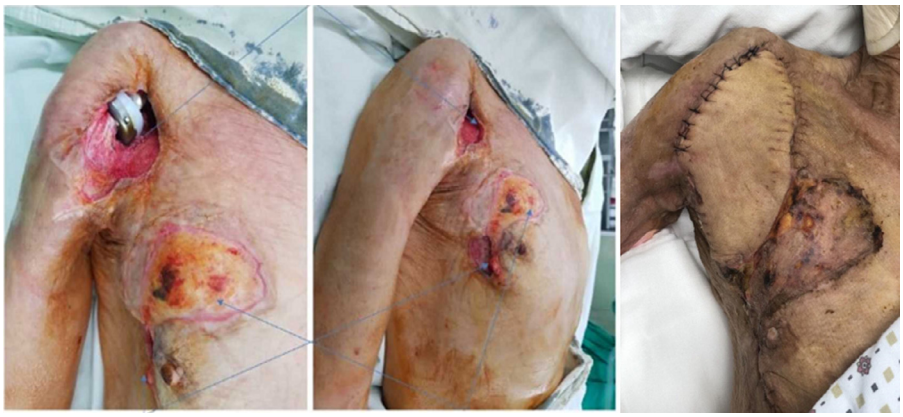

Fig. 12 – Clinical photo showing coverage of the skin defect site after LD pedicled flap and STSG.

vacuum dressing, and tissue debridement. Latissimus Dorsi pedicled flap and Split Thickness Skin Graft were successfully performed without infection. The patient's wound problem has been completely resolved.

The series of events that occurred, in this case, suggests that surgeons should be more careful than we were to avoid injury of small vessel branches during surgery. Also, surgeons should always keep in mind the importance of collaboration with a competent radiologist during management of massive bleeding after unexpected vessel injury. In this case, the role of the radiologist was crucial.

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