CASE REPORT

Anterior humeral circumflex artery avulsion with brachial plexus injury following an isolated traumatic anterior shoulder dislocation

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SUMMARY

A 70-year-old man presented to accident and emergency with an isolated anteriorly dislocated shoulder, in the absence of a concomitant fracture. There was no neurovascular deficit at presentation, and the shoulder was reduced under sedation, using the Kocher's technique. Following this, the patient developed signs of hypovolaemic shock. Clinical examination revealed an expanding fullness in the deltopectoral area, with compromise of the limb neurovascular status. CT imaging confirmed an expanding haematoma from the axillary vessels, restricting left lung expansion. Once resuscitated, the patient was transferred to theatre for exploration of the bleeding vessels. Intraoperative findings included an avulsed anterior circumflex humeral artery that was subsequently ligated. Postoperatively, the patient developed axillary, radial, median and ulnar nerve neuropraxia, which improved clinically prior to discharge. The patient was ultimately discharged home after a lengthy inpatient stay.

BACKGROUND

Anterior shoulder dislocations are a common presentation to hospital, accounting for nearly 50% of joint dislocations. While vascular injury itself is a rare entity in fracture-dislocations, it is an even rarer presentation with isolated shoulder dislocations. Identifying the pathognomonic triad for arterial disruption including a dislocated shoulder, diminished (or absent) radial pulse and palpable axillary haematoma should prompt urgent referral for an orthopaedic and vascular consult. Subsequent exploration of the vasculature should occur in theatre. Our case demonstrates a delayed presentation of an expanding haematoma resulting in hypovolaemic shock, restriction in lung expansion and subsequent neuropraxia. We discuss the reasons for arterial disruption and neurological compromise in an isolated shoulder dislocation with particular focus on vessel anatomy and associated risk factors. We hope, through this case report, to encourage serial examinations of the joint pre-reduction and post-reduction and to have a high index of suspicion for vascular injury even in the absence of a fracture.

CASE PRESENTATION

A 70-year-old man presented to accident and emergency following a fall from a two-stair height onto his left shoulder. There were no preceding medical symptoms and the fall was thought to be purely

mechanical. His medical history consisted of hypertension and previous deep vein thrombosis for which he took regular warfarin.

Examination of the joint revealed a palpable 'fullness' to the anterior aspect of the left shoulder with generalised restriction in the range of movement. Motor and sensory function along the distribution of the radial, ulnar and median nerves remained intact and there was no sensory deficit along the 'regimental patch' distribution of the axillary nerve. The radial pulse was noted to be weak on initial examination, but was confirmed with a triphasic waveform on hand-held Doppler scan. Anteroposterior and lateral radiographs of the shoulder joint were performed (figures 1 and 2), confirming an anterior shoulder dislocation in the absence of any visible fracture. The shoulder was relocated using the Kocher's method, however, the performing physician remained hesitant that the shoulder was successfully relocated due to the fullness in the shoulder and persistent restricted movement of the shoulder joint. A further radiograph was performed (figure 3), confirming relocation of the humeral head. Postreduction, no immediate distal neurovascular deficit was noted.

Within 30 min of the procedure, the patient reported of left-sided chest pain and was noted to desaturate on room air (SpO₂: 83%) having

Figure 1 Pre-reduction anteroposterior radiograph of left shoulder.

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Figure 2 Pre-reduction lateral radiograph of left shoulder.

increasing oxygen requirements. A full re-assessment was carried out on the patient. The patient had become tachycardic at 120 bpm, hypotensive at 97/65 mm Hg and tachypnoeic at 25 breaths/min. Examination revealed an expanding fullness to the shoulder, extending proximally into the left deltopectoral area with no obvious bruising to the overlying skin. Although haemoglobin levels remained stable at 12.3 g/dL, the patient's International Normalised Ratio was 2.3. In view of the hypovolaemic shock, he was immediately fluid resuscitated with crystalloid solution, and subsequent improvement in systolic blood pressure was noted. A Focused Assessment with Sonography for Trauma scan revealed a small pericardial effusion requiring no acute intervention.

In view of the rapid deterioration, a CT scan of the chest, abdomen and pelvis was requested.

INVESTIGATIONS

CT imaging (figures 4 and 5) revealed a large haematoma (white arrow) with active bleeding in the left axillary region extending along the left lateral chest wall to the level of the diaphragm. There were no associated rib fractures from the original insult and the lung parenchyma remained intact. Abdominal CT revealed no gross abnormality.

DIFFERENTIAL DIAGNOSIS

In view of the clinical and radiological findings, a preliminary diagnosis of axillary artery rupture was made. The resulting haematoma on the left lateral chest wall acted as a mechanical

Figure 3 Postreduction anteroposterior radiograph of left shoulder.

Figure 4 Transverse CT image of thorax indicating expanding haematoma.

obstruction to lung expansion, resulting in a picture of restrictive lung disease with hypovolaemic shock from the bleed.

TREATMENT

The patient was immediately referred to a vascular centre. On arrival, he was noted to have global weakness to his left arm with altered sensation peripherally. Warfarin was immediately reversed with a prothrombin complex concentrate and the patient was taken to theatre for exploration of the axillary artery.

During anaesthetic induction, the patient had a pulseless electrical activity (PEA) arrest but had return of spontaneous circulation following administration of epinephrine. Both blood loss and a potential tamponade effect of the haematoma would have been contributing risk factors for a PEA arrest, highlighting the urgency of haematoma evacuation. Therefore, an open (rather than endovascular) approach was utilised to allow for decompression of the axillary fossa by evacuating the haematoma.

Intraoperatively, the brachial artery was exposed and followed proximally into the axilla. Theatre findings revealed a completely avulsed anterior circumflex humeral artery with active bleeding. The bleeding artery was subsequently ligated and haemostasis achieved. The remainder of the axillary artery was

Figure 5 Coronal CT image of thorax indicating expanding haematoma.

deemed to be intact. The brachial plexus was also explored and no obvious damage noted. Postoperatively, a strong radial pulse was palpated.

OUTCOME AND FOLLOW-UP

The patient made a slow but stable recovery with a brief stint in the intensive care unit in the immediate postoperative period. Despite no obvious injury to the brachial plexus, he had persistent global weakness in his left arm and altered sensation distally. With time, this gradually improved and the patient benefited from additional physiotherapy. However, his symptoms did not fully normalise prior to discharge. Sensation along the distribution of the regimental patch gradually improved postoperatively. Once deemed clinically stable, he was discharged with routine follow-ups.

DISCUSSION

Shoulder dislocations account for nearly 50% of all major joint dislocations presenting acutely to emergency departments.¹⁻³ Over 90% of all shoulder dislocations present anteriorly³⁻⁵ and are usually secondary to trauma. Clinical examination of the shoulder joint is often challenging, compounded by pain and swelling of the joint. Approximately 10% of primary anterior dislocations are associated with axillary nerve neuropraxia.⁶ While isolated vascular injuries are relatively rare (1–2%), they demand immediate intervention. It is very rare for a vascular injury to occur in an anterior dislocation in the absence of concomitant fracture.⁷

Vascular damage may be obvious or relatively subtle, however, major arterial injury may co-exist with a good peripheral capillary refill time and a palpable radial pulse. This is due to the development of extensive collateral circulation in the upper limb.⁸ A monophasic radial waveform on a hand-held Doppler should also warrant further investigation and raise suspicion for arterial disruption. The pathognomonic triad for arterial disruption consists of an anteriorly dislocated shoulder, diminished (or absent) radial pulse and a palpable axillary haematoma. In addition to this, clinicians should have a lower threshold of suspicion for arterial disruption, with or without a concomitant fracture, in patients who are anticoagulated. In our case report, being on warfarin was undoubtedly a risk factor for the development of a progressive haematoma and failure of the bleed to potentially tamponade, thus requiring reversal preoperatively. As such, a thorough examination should be performed pre-reduction and postreduction of the shoulder.

The axillary artery, which represents a continuation of the subclavian artery, lies anterior and medial to the proximal humerus. It is divided into three parts based on its relationship to the pectoralis minor muscle. The first part lies proximal (suprpectoral), the second posterior (retropectoral) and the third lateral (infrapectoral) to the pectoralis muscle. As described in this case report, in an isolated anterior dislocation (without fracture), the axillary artery can be damaged in its third part by avulsion of the subscapular and circumflex humeral vessels. Additionally, it can be damaged in its second part by avulsion of the thoracoacromial trunk.⁷ The mechanism of arterial injury in isolated anterior dislocations is thought to be due to the tethering of the axillary artery to its branches (the subscapular and anterior circumflex branch) thus accounting for injuries primarily occurring in the third part.⁷⁻⁹ The pectoralis muscle is also thought to act as a fulcrum over the artery, resulting in arterial stretch when the glenoid head dislocates. In excess tension, this will subsequently result in arterial rupture.⁹⁻¹² Other contributing factors include previous injury to the shoulder resulting in fixation of the vessel

to a scarred joint capsule or atherosclerotic vessels compromising vessel compliance.⁷

Given the anatomical location of the brachial plexus, it not surprising that neurological injuries accompany traumatic anterior dislocations. A dislocated humeral head displaces the subscapularis nerve and the axillary nerve (which originates from the posterior cord of the brachial plexus) anteroinferiorly creating traction and direct pressure on the nerve. Injury to the nerves varies from neuropraxia to axonotmesis, or neurotmesis.¹⁰ In view of the improving symptoms prior to discharge, we suspect that our patient suffered from axillary neuropraxia with additional involvement of the radial, median and ulnar nerves, compounded by the accumulating haematoma. The entire brachial plexus may be involved following a traumatic dislocation. In the absence of clinical improvement, electromyography provides an objective evaluation and measure of neurological function and may be utilised 4 weeks postinjury.^{10 13}

The technique utilised for shoulder reduction is well debated. At present, there is no reduction method that has a 100% success rate.³ The Kocher's technique, although popular and effective, has fallen out of favour recently, due to complications associated with the manoeuvre and subsequent torque applied. These include humeral shaft fractures,^{14 15} axillary vessel rupture,¹⁶ and rotator cuff and pectoralis major ruptures.¹⁷ Safer techniques described in the literature include simple adaptations of the Kocher's method (external rotation method of Leidelmeier), Milch, Spaso, Stimson, 'FARES' (Fast, Reliable and Safe) method and scapular manipulation techniques.^{3 18-20} Given the pre-existing 'fullness' in the shoulder, it is likely that vessel disruption was caused prior to any manipulation of the shoulder back into joint.

Learning points

- ▶ Vascular compromise may be obvious or relatively subtle, and therefore clinicians should treat any shoulder injuries with a high index of suspicion.
- ▶ In patients who are anticoagulated, there should be a lower threshold of suspicion for arterial disruption following a shoulder dislocation, with or without a concomitant fracture.
- ▶ Careful neurological and vascular examination should be performed at presentation and repeated postrelocation of the shoulder into joint.
- ▶ The pathognomonic triad for arterial disruption (anteriorly dislocated shoulder, diminished (or absent) radial pulse and palpable axillary haematoma) should prompt an urgent orthopaedic and vascular consult.
- ▶ Apply safe reduction techniques to minimise the risk of iatrogenic procedural complications.

Competing interests None declared.

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