Axillary artery injury secondary to displaced proximal humeral fractures: a report of two cases

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ABSTRACT

We report 2 cases of axillary artery injury secondary to low-energy proximal humeral fractures. In case 1, early diagnosis based on signs of acute ischaemia of the arm enabled early treatment and a favourable outcome. In case 2, there were no signs of ischaemia or neurological deficit, resulting in delayed diagnosis and increased severity of the injury. The patient developed a false aneurysm and sepsis and eventually died. A high index of suspicion is necessary for diagnosing an axillary artery injury. We recommend that all patients with proximal humeral fractures with severe medial displacement of the shaft and a bone spike should routinely undergo Doppler ultrasound scanning to rule out vascular injuries and the presence of a false aneurysm.

Key words: axillary artery; humeral fractures; shoulder; shoulder fractures

INTRODUCTION

Proximal humeral fractures are common injuries accounting for 5% of all fractures. They are usually the result of high-energy trauma in young patients and low-energy trauma in older patients. The brachial plexus and axillary artery are near to the proximal humerus and can be damaged during shoulder injuries, although this is uncommon. Diagnosing an axillary artery injury after low-energy trauma to the shoulder may be difficult. Its presentation varies and may include acute ischaemia of the arm, an arm with palpable pulses and a normal capillary refill time secondary to the extensive anastomotic network of collateral vessels around the shoulder; brachial plexopathy secondary to swelling and compression from the injured axillary artery where they lie within a common fascial sheath. A missed or delayed diagnosis may be fatal as the false aneurysm created by the injury expands and compresses local structures and eventually ruptures. We present 2 cases of axillary artery injury following low-energy proximal humeral fractures. The presentations and outcomes highlight
the importance of having a high index of suspicion when treating patients with proximal humeral fractures.

CASE REPORTS

Case 1

In September 2006, a 41-year-old woman presented with severe pain and numbness over the left shoulder after falling onto it while sleepwalking. The entire shoulder was swollen and tender and she had a cool hand and capillary refill time of <2 seconds. Sensation over the deltoid region was decreased but no other neurological deficit was noted. The left brachial, radial, and ulnar pulses were impalpable, and hand-held Doppler ultrasound scanning revealed monophasic signals over these areas. Radiographs demonstrated a 3-part fracture of the left proximal humerus with dislocation of the gleno-humeral joint according to Neer’s classification (Fig. 1).

She underwent closed reduction within 2 hours, but the distal pulses were still impalpable and the capillary refill time increased to 4 seconds. Arteriography (through a right common femoral artery approach) revealed occlusion of the left axillary artery at the level of the fracture site by a spike of bone (Fig. 2a). Exploration of the vessel revealed local thrombosis and an intimal flap. The artery was repaired with a long saphenous vein patch, and the hand regained good circulation and all pulses were palpable (Fig. 2b). Internal fixation of the fracture was performed using Rush pins and the patient remained well with no residual neurological or vascular deficiency. At one-month follow-up, the rush pin had failed and was revised with a locked intramedullary nail. After 3 months of follow-up, she had a good functional outcome after a course of physiotherapy.

Case 2

In June 2006, a 72-year-old woman presented with severe pain on her right shoulder after slipping and falling onto it one week previously. There were no distal neurological or vascular deficiencies. Radiographs demonstrated a Neer’s 2-part displaced fracture of the proximal humerus (Fig. 3).

She underwent open reduction and internal fixation using Rush pins 6 days later. She required 2 units of whole blood prior to the procedure as her preoperative haemoglobin level had fallen to 82 g/l from 93 g/l. On postoperative day 1, she was found to have radial nerve dysfunction, which was
presumed to be secondary to neuropraxia during surgery. She underwent further blood transfusion to manage her low haemoglobin level (74 g/l). On day 2, neurological function of the right hand deteriorated, involving all 3 cords of the brachial plexus. She was provided with a splint for support in anticipation of neurological recovery. By week 2, her shoulder pain and neurological deficit were persisting and she had severe bruising around the elbow and a delayed capillary refill time. Radiographs showed loss of fixation so she was readmitted and underwent blood transfusion (haemoglobin level, 62 g/l) prior to revision surgery. Profuse bleeding was encountered intra-operatively requiring urgent transfusion, wound packing, and closure.

Postoperative nerve conduction studies confirmed a diffuse brachial plexopathy (C5–T1). Doppler ultrasound scanning and computed tomographic angiography confirmed the presence of a 15-cm false aneurysm arising from the right axillary artery extending cranially above the right shoulder and caudally below the level of the pulmonary trunk with some distal flow into the brachial artery (Fig. 4). She underwent axillary exploration and repair of the false aneurysm. It was noted intra-operatively that a bone spike had caused the injury. Further bony fixation was not feasible due to poor bone quality and impending necrosis of the overlying skin. She remained unwell and developed a wound infection with muscle necrosis. She underwent several wound debridements with washouts and vacuum dressing application for 3 weeks, but the sepsis did not resolve. Amputation was suggested but declined by the patient. She remained septic, underwent several further blood transfusions but died several days later.

**DISCUSSION**

Axillary artery injury following low-energy trauma to the shoulder is an uncommon complication, with an incidence in a supra-regional centre of 20 cases over 20 years. A total of 44 such cases associated with proximal humeral fractures have been reported in the literature. Two of them had continuous haemorrhages detected and treated 6 and 8 weeks after the initial injuries; 3 others had massive secondary haemorrhages from false aneurysms and were treated between 8 and 16 weeks after the injury. The limb salvage rate was 94% and 70% of patients regained good neurological function.

The injury mechanisms include direct injury by a bone spike, violent overstretching of the artery in hyperabduction, and avulsion or rupture of a branch at its origin. Acute injuries involve total or partial rupture of all arterial layers, or intimal damage only, producing occlusion of the lumen. Delayed injuries include false aneurysm, arteriovenous fistula or thrombosis.

Early diagnosis and treatment results in better functional outcomes. It is difficult to identify the vascular injury in those patients with no neurological or vascular deficits. Only 46% of patients presented with neurological deficits, and 68% with acute ischaemia of the arm. In the absence of acute ischaemia of the arm, clinical presentations indicating axillary arterial injury include: (1) persistent or worsening pain, despite fracture reduction and stabilisation, (2) delayed onset of nerve palsy, (3) deepening of a nerve palsy inflicted at the time of injury, and (4) local swelling or axillary
bruising. Arteriography and surgical exploration are necessary to confirm the injuries. Vascular treatments include endarterectomy, vein patch repair, and resection of the lacerated segment with end-to-end anastomosis and vein patching or grafting.

In case 2, delay in the presentation and diagnosis despite the persisting pain, shoulder swelling, and fall in the haemoglobin level were major factors contributing to her death. Although no signs of a neurovascular injury were noted initially, the presence of a medial bone spike with impingement and severe displacement of the shaft into the axilla should have created suspicion of a vascular injury. The definitive diagnosis was eventually confirmed by Doppler ultrasound scanning. We suggest that all patients with proximal humeral fractures with severe medial displacement of the shaft and a bone spike should routinely undergo Doppler ultrasound scanning to rule out vascular injury and the presence of a false aneurysm.

REFERENCES