CASE REPORT

Delayed brachial plexus compression secondary to anterior shoulder dislocation—The late consequence of an axillary artery pseudoaneurysm: A case report

Philip A. McCann*, Mohannad J. Barakat, Jon S. Wand

Department of Trauma and Orthopaedics, Cheltenham General Hospital, General Surgery, Sandford Road, Cheltenham GL53 7AA, UK

Accepted 8 June 2006

Introduction

Anterior dislocation of the shoulder is a common orthopaedic injury. Treatment is usually straightforward and performed in the Emergency Department. Various methods of reduction are used in clinical practice, and preference is dependant on personal experience and/or local hospital guidelines. However, complications arising from either the original injury or its treatment are less well reported.

Joint stiffness is almost universal postinjury. Damage to the rotator cuff muscles occurs in approximately 15–32% of cases, and is seen more commonly in the older patient. Vascular injury following orthopaedic trauma is rare, estimated at 1% and injury to the axillary or subclavian vessels accounts for only 20% of arterial injuries to the upper limb. Damage may result in a limb or life threatening scenario. Injury to the brachial plexus is also uncommon but can produce significant longstanding morbidity.

This paper describes a case of delayed neurovascular insult complicating primary anterior shoulder dislocation. The injury did not present until the axillary pseudoaneurysm compressed the brachial plexus producing extreme pain and loss of function two weeks after the initial injury.

Case report

A 63-year-old right hand dominant male presented to the Emergency Department complaining of severe pain in the right shoulder and arm following a fall directly onto the shoulder against a low wall. He was otherwise fit and well except for medication controlled hypertension. He had no previous history of injury to either shoulder.

Clinical examination found a globally reduced range of movement in the right upper limb. No neurovascular deficit was noted. Radiographic analysis confirmed a diagnosis of anterior dislocation of the shoulder (Fig. 1). This was easily reduced using Kocher’s technique with intravenous sedation and analgesia. Postreduction radiographs were satisfactory (Fig. 2). The patient had no neurovascular deficit after reduction. He was placed in a broad

* Corresponding author. Tel.: +44 8454 223245; fax: +44 8454 223644.
E-mail address: pasmccann@hotmail.com (P.A. McCann).

1572-3461 © 2006 Elsevier Ltd. Open access under the Elsevier OA license.
doi:10.1016/j.injury.2006.06.023
arm sling and admitted overnight for an associated head injury. On re-examination the following morning he was complaining of only moderate pain in the right shoulder. Motor and sensory branches of the radial, ulnar and median nerves were functioning normally.

At clinic review six days later the patient was comfortable in a broad arm sling. No axillary nerve or rotator cuff damage was noted. The joint was immobilised and he was scheduled for physiotherapy.

Two weeks after the initial injury the patient re-presented to the Emergency Department complaining of sudden onset of severe pain in the right shoulder and arm, with numbness along the radial border of the forearm. On examination he was found to have a swollen arm with reduced sensation in the distribution of the medial and radial nerves. The peripheral pulses were palpable. No axillary mass was noted. Power was difficult to formally assess due to severe pain. Radiographs of the shoulder confirmed the humeral head was in joint. There was no evidence of compartment syndrome. The initial diagnosis was of axillary vein thrombosis with possible brachial plexus injury, and the patient was admitted for analgesia, elevation, anticoagulation and further investigation. After adequate analgesia power in the right arm was found to be MRC grade 0/5 throughout, except for shoulder, elbow and wrist flexion which were 1/5. Sensate loss had now progressed to the distribution of the ulnar nerve.

First line investigation with a venogram was inconclusive, as satisfactory views of the subclavian and axillary vessels could not be achieved. The only significant finding was of a large haematoma in the subcutaneous tissues.

The patient proceeded to CT angiography. This showed an axillary artery pseudoaneurysm measuring 67 mm in diameter enclosed by a larger haematoma (Fig. 3). A 40 mm infrarenal abdominal aortic aneurysm was also noted as an incidental finding.

Surgical exploration of the pseudoaneurysm was undertaken. Proximal and distal control of the damaged axillary artery was obtained and the tense haematoma evacuated. The injured segment was excluded and bypass performed using a reverse long saphenous vein graft (Fig. 4). Postoperative recovery was unremarkable. A marginal increase in muscular power to 2/5 was noted.

At outpatient review two weeks later the patient was still suffering from pain in the right arm and paraesthesia in the right hand. Peripheral

![Figure 1](image1.jpg)  
**Figure 1** Anterior-posterior view of the right shoulder prior to reduction.

![Figure 2](image2.jpg)  
**Figure 2** Anterior-posterior view of the right shoulder post reduction.

![Figure 3](image3.jpg)  
**Figure 3** Maximum intensity projection (MIP) coronal reconstruction of the CT angiogram of the patient’s right axilla.
pulses were intact. Power had not improved beyond 2/5. He was referred for physiotherapy and nerve conduction studies. These revealed a severe axonal loss lesion involving all segments of the right brachial plexus. The posterior cord was most heavily affected, showing complete axonal degeneration. Whilst eventual recovery of the lateral and medial cords may be possible, the functional outcome of the posterior cord remains guarded.

Discussion

The majority of axillary and subclavian artery injuries occur as a consequence of high energy penetrating trauma. However, injury may also arise from low energy blunt trauma, which is more likely to lead to an underlying brachial plexus injury resulting in long term disability.

The above case highlights the serious consequences of vascular trauma associated with anterior dislocation of the shoulder. What is most striking in this case is the delayed presentation until two weeks after the initial accident. In this interval period the patient had no neurological symptoms or signs. He had complained only of pain in the shoulder region, which is of course to be expected after such an injury.

Pseudoaneurysm of the axillary artery was first described in 1975. The pathogenesis of axillary artery injury is poorly understood. It has been shown that the majority of these cases affect individuals over the age of 50 years. Atherosclerotic change in the vessels makes them less elastic and less resistant to shearing forces. Vessels of the younger patient are more elastic and compliant, being less vulnerable to injury during dislocation.

The anatomical position of the vessel is thought to play a role in its susceptibility to trauma. Initially it was suggested that injury may arise as a consequence of the position of the axillary artery, fixed between the anterior and posterior circumflex arteries and the supscapular arteries. Another theory purports that movement of the humeral head into hyperabduction compresses and distorts the vessel against pectoralis minor. The majority of injuries occur within the lower third of the artery distal to this muscle. The axillary artery displays a degree of anatomical variation in its course which may contribute further to its susceptibility to injury by enveloping structures.

In this case it is probable that transection of the intimal layer and/or medial layers of the arterial wall occurred, with temporary containment by the adventitial layer for the interval period of two weeks. Iatrogenic vascular injury is common, accounting for up to 34% of arterial trauma. However, it is impossible to know if axillary artery pseudoaneurysm is a result of the initial trauma or its treatment. It is possible that the act of reduction contributes to or even causes the vascular insult, as tractional forces are applied across the vessels during manipulation of the limb. To our knowledge this mechanism of iatrogenic arterial injury has not been mentioned in the literature. Vascular damage may be very difficult to distinguish in the acute phase due to the anastomotic networks within the upper limb. Indeed, there may be no disruption of the peripheral pulses (as occurred

Figure 4  Intraoperative images of right axilla: (A) the aneurysm during repair and (B) with reverse long saphenous vein graft in situ.
this case), even with complete transection of the vessel.\textsuperscript{5,10} Detection of neurological deficits, especially motor components, is also problematic as clinical examination may be profoundly limited due to pain.\textsuperscript{17}

As the axillary artery and brachial plexus run in a common fascial sheath, bleeding and haematoma formation will create a direct pressure effect on the nerve fibres. As true ischaemia of peripheral nerve fibres is uncommon given their generous anastomotic blood supply, extrinsic compression by an expanding pseudoaneurysm and surrounding haematoma is the most probable pathogenic mechanism leading to neurapraxia.\textsuperscript{26} Evacuation of the haematoma with surgical repair of the underlying vessel is the gold standard in restoring function to the artery\textsuperscript{26,18} in such cases.

Anterior shoulder dislocation is a clinical scenario encountered in everyday orthopaedic practice. Reduction techniques are generally easily taught and can be performed safely and efficiently.\textsuperscript{8} However, this case reminds the treating clinician to remain alert to the possibility of secondary neurovascular injury, particularly in the older patient. Prompt diagnosis and treatment are essential in the management of these potentially limb and life threatening injuries. Given the gravity of the complications associated with shoulder dislocation, the treating clinician should maintain a high index of suspicion of such problems when dealing with the patient who does not make an unremarkable recovery.

Acknowledgments

We gratefully acknowledge the assistance of Keith R Poskitt MD FRCS, JM Gibson FRCR and Fiona Court MRCS, in the preparation of this manuscript.

References