



Case Report

Axillary artery injury after an anterior shoulder fracture dislocation and “periosteal sleeve avulsion of the rotator cuff” (SARC). Case report and review of the literature☆

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ABSTRACT

We present the rare complication of an axillary artery injury associated with an anterior dislocation of the humerus and what we believe to be the first reported periosteal sleeve avulsion of the entire rotator cuff (SARC). We review the literature and discuss the cause of this unusual injury pattern.

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Case report

A 17 year old boy was involved in a high-speed trail bike accident in the countryside. He fell from his bike and hit a tree at 80 km/h and suffered a fracture/dislocation of his previously normal shoulder. He was intoxicated at the time, a known smoker and had a previous splenectomy for spherocytosis. An ambulance transferred the patient to the nearest rural centre. An attempt at closed reduction in EMD at the rural centre was attempted but failed and was transferred to a level 1 trauma centre, 5 h post-injury. On arrival in EMD gentle closed reduction was again performed and when failed, the Orthopaedic service was consulted.

On examination his upper limb was slightly hyperaemic and his shoulder was extremely swollen. His pulse remained present and was tachycardic, which was presumed to be secondary to pain. His Haemoglobin (Hb) was 100 g/dl, INR was 1.2 and platelet count was 76. His chest X-ray was normal with no pneumothorax. He was consented for an open reduction in theatre. At the time no theatre was available and a CT was performed to clarify the bony anatomy and in hind sight a large haematoma is evident posteriorly (see Fig. 1). Whilst waiting for theatre he developed a dense radial nerve palsy. An Arteriogram was not performed as theatre time became available soon after the nerve palsy occurred and was urgently taken to theatre thereafter.

A vascular and plastic surgeon were all called prior with the possibility of a vascular injury requiring repair.

A Deltpectoral approach revealed the humeral head sitting anterior to the subscapularis and behind the conjoint tendon. The humeral head was devoid of any rotator cuff muscle attachment like a cadaveric specimen and the entire rotator cuff musculature

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Fig. 1. Axial CT radiograph showing large posterior Haematoma indicated by arrows. **Fig. 3:** Photographs taken at time of surgery showing axillary artery with intimal tear indicated by arrow.

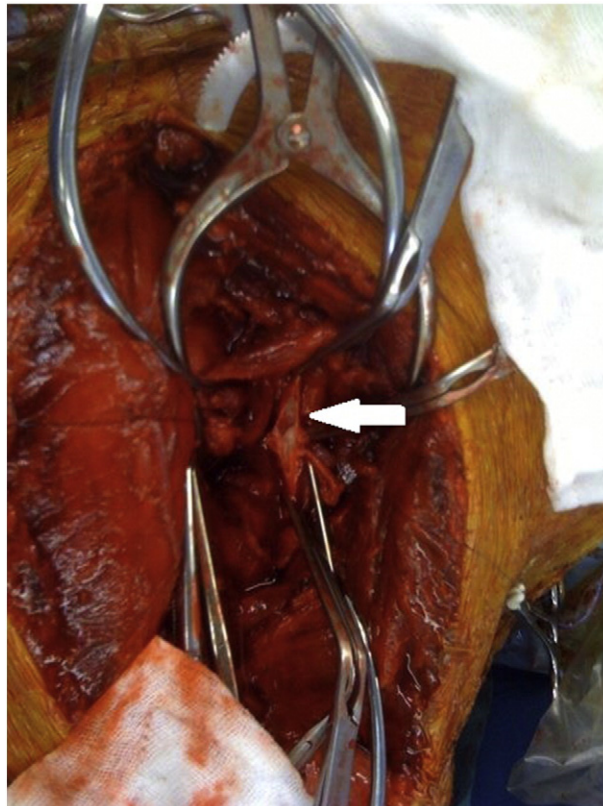


Fig. 2. Photographic picture of open reduction and internal fixation (ORIF) and accompanying vascular repair at time of surgery.

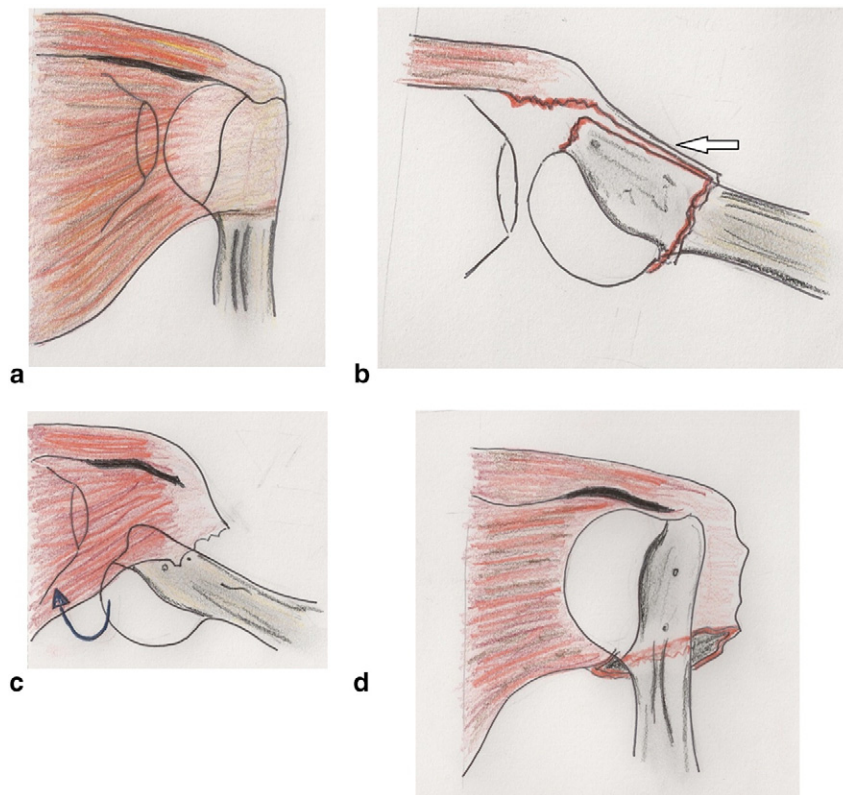


Fig. 4. a–d: Proposed mechanism of injury shown in diagrammatic sequence. a) Normal rotator cuff; b) abduction and inferior translation of humeral head. Note: avulsion of cuff; c) extreme abduction and lateral translation under sleeve; and d) final position of humerus and sleeve.

Periosteal sleeve avulsions are well described from the scapula, either an anterior labroligamentous periosteal sleeve avulsion (ALPSA) [24] or a posterior labroligamentous periosteal sleeve avulsion (POLPSA) [30].

The clavicle has a well-recognised periosteal sleeve fracture due to the coracoclavicular ligaments being stronger than the enveloping periosteum. The clavicle ruptures superiorly through the periosteum giving the false impression of an acromioclavicular joint dislocation.

This is not the case on the humeral side. The closest presentation of this type of periosteal avulsion was described in relation to an irreducible shoulder dislocation with a posterior periosteal sleeve and cuff tear [2] with no vascular injury. There have also been reports of adolescent rotator cuff tears causing irreducible shoulder dislocations in high energy trauma [28] as well as glenohumeral interposition of the cuff after skiing accidents, although no periosteal hinge were found to be intact [20].

In our unique situation the entire rotator cuff has peeled off or de-gloved from the humerus, leaving only the vessels to undergo marked stretching till failure at focal weak points. In this case, although skeletally mature, the patient still retained a very thick periosteal layer in direct continuation with the rotator cuff tendons because of his young age. It is the periosteum that is the weak point at this age rather than the ligament and tendons in adulthood. The periosteal thickness also varies throughout the humerus. Since the periosteum is quite strong in the posteromedial aspect of the proximal humerus, but relatively weak in the anterolateral aspect, the initial rupture may have begun here [29]. This is also a point of change in thickness between the thick periosteal layer of the diaphysis.

Our presumption is that the head was forced initially inferiorly (Fig. 4). This caused a rent in the weak anterolateral portion of the periosteum. This rent continued circumferentially and with the head continuing to abduct and to translate laterally the head was able to slip under the periosteal sleeve. Incorporated in the sleeve was areas of the lesser and greater tuberosities that had yet to fully consolidate with bone. The final position of the humerus was posterior to the conjoint tendon sitting anterior to the entire periosteal sleeve avulsion of the rotator cuff.

There are two main theories behind the mechanism of axillary artery rupture. Adovasio et al. [1] independently proposed that the axillary artery is exposed to direct injury by the dislocating humeral head. It relies on the artery being anchored by the subscapular branch and compressed by the humeral head and then stretched by hyper abduction. The axillary artery then ruptures at the origin of the subscapular branch. This was also confirmed by post-mortem arteriograms by McKenzie and Sinclair [23]. This is similar to our proposed mechanism of injury since the humeral head, to slip out of the cuff, would require a significant amount of lateral displacement and abduction.

The second theory was that the pectoralis minor muscle acts as a fulcrum for the artery, causing a vascular injury by kinking, shearing or by compression [5,11].

A well-defined subgroup is those with a previous or recurrent dislocation. The artery can become adherent to peri-capsular tissue by scar tissue and, when the humeral head tears the capsule of the shoulder joint, the artery is transected [16].

Specific arterial injuries described range from intimal tears and secondary thrombus formation [9,21], pseudo-aneurysms [8,13,14,17], haematoma dissecting the arterial wall [30], entrapment within the glenohumeral joint [15] and frank transection [3,5,16,19].

Dislocations represent blunt trauma and when a vessel is either compressed or stretched an intimal tear can develop. The intimal layer is the first to rupture due to its reduced elasticity whilst the adventitial layer remains intact. A subadventitial partial vessel rupture creates an intimal flap and a subsequent subintimal haematoma, obstructing flow and leading to thrombosis [22].

When this compression is chronic this can create pseudo aneurysms, seen in both volleyball, handball players and baseball pitchers [28,32].

Classically, the axillary artery, a continuation of the subclavian artery, begins at the outer border of the first rib. It is divided by the pectoralis minor muscle into three parts. The first part of the axillary artery is located between the first rib and the superior border of the pectoralis minor muscle. The second part of the axillary artery lies deep to the pectoralis minor muscle. The third part of the axillary artery distal to pectoralis minor, which is more commonly injured has three branches: the subscapular, the anterior circumflex humeral and the posterior circumflex humeral arteries.

There are also many variations in the branching patterns of the axillary artery [27]. Such variations are significant since this can influence observational injury patterns and the amount of tethering of the axillary artery.

There are sites along the axillary artery that are consistently injured. The axillary artery is damaged in its second part due to avulsion of the acromiothoracic trunk and in its third part from an avulsion of the subscapular or circumflex humeral vessels with subsequent linear tears in the artery and intraluminal thrombosis. If transection occurs, it is usually in the third part - distal to the branching arteries [7].

The varying presentations relate to the status of collateral circulation in the upper limb which is extensive anastomoses with 5 major branches.

This can mask a significant axillary artery injury due to sufficient collateral perfusion to maintain distal pulses and capillary return. Multiple authors have stressed the importance of the unreliable nature of a palpable pulse and its relationship to arterial injury [4,7,31]. It is clear that the axillary may be completely transected or non-patent yet via the periscapular collateral circulation, based on the thyrocervical trunk, can have a palpable pulse [4]. Although there are reports of presentations with frank ischaemia of the upper limb [6,12,22] there is an increasing series of missed diagnosis in the contemporary literature [13,14,33].

An important triad of axillary swelling, reduced or loss of distal pulses and a dislocation should now alert one to the potential axillary artery injury [18].

A diminished pulse in a dislocated shoulder is always a significant finding and should never be attributed to spasm and warrants further investigation by an arteriogram.

Axillary swelling in this setting is also indicative of a vascular injury; often well after the initial injury because of the development of a pseudoaneurysm. Classically an enlarging mass with a pulsation is indicative of a pseudoaneurysm yet this is not always the case resulting in the delay in diagnosis. The pathophysiology of its creation is thought to be caused by traumatic disruption of side-branch vessels of the axillary resulting in a side-hole-type injury [26].

When the pseudo aneurysm has extended into the joint it will cause an intra-articular haematoma and subsequent lateral subluxation of the glenohumeral joint [13,33].

Brachial Plexus injuries are common with axillary arterial injuries ranging from 27% to 60% and are often found due to the close proximity to the artery [10,25,31]. These are often complete lesions secondary to traction that can be the most important determinate to long term disability [19]. This is in stark contrast to plexus lesions in isolated closed dislocations that are commonly postganglionic, infraclavicular and have an excellent prognosis. This is why exploration of the plexus at the time of vascular repair is mandatory and that all axillary sheath haematomas should be evacuated to prevent delayed ischaemia of the nerve [34].

Although this is still a rare complication post-shoulder dislocation, improved awareness can improve the early diagnosis. This can allow for prompt surgical investigation and subsequent treatment. It is imperative that a thorough examination is performed after every dislocated shoulder to not only look for signs of ischaemia but also subtler hyperaemia, an abnormal pulse or axillary swelling. It is also important to recognise that young patients involved in high energy shoulder dislocations can have an intact large periosteal sleeve preventing and complicating reduction.

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