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Case Report

Amputation for upper extremity ischemia following shoulder dislocation: case report and a review of literature

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ABSTRACT

Injury of the axillary artery following glenohumeral dislocation is a very rare situation. The mechanisms for arterial injuries are lacerations, rupture, avulsion of main branches or intimal tears and pseudo aneurysm formations. In this report we present an upper extremity ischemia following shoulder dislocation resulting with loss of extremity. Our aim was to highlight the importance of the third part of axillary artery and consequences of underestimation of vascular pathologies following shoulder injuries.

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Introduction

Axillary arterial injuries following blunt shoulder trauma are rare but also well reported, of which might end up with catastrophic results, if not properly diagnosed and treated. Mechanisms of arterial injury are reported as intimal tears, artery lacerations or avulsions, penetrating trauma or tethering between fracture fragments. There are several published reports of axillary artery injury following shoulder dislocations and proximal humeral fractures [1-6].

In this report we present a misdiagnosis of an axillary arterial injury case following shoulder dislocation resulting with mid humeral amputation. Also we aim to highlight the importance of neurovascular monitorization in the treatment of shoulder injuries.

Case Presentation

A 45-year-old male was admitted to emergency department of another institution following a right anterior-inferior shoulder dislocation with tuberculum majus fracture during a motor vehicle accident. Closed reduction of glenohumeral joint was performed on emergency department under sedation and shoulder was immobilized in a Velpeau bandage. Two days after reduction patient was referred to our clinic with symptoms and signs of total paralysis of right upper extremity and ischemia. Fluoroscopic image shows a well reduced joint with minimally displaced fracture of greater tuberosity (Figure 1). Patient declared that, he was unable to use his extremity, right after initial trauma. He also stated that, there was no improvement of pain following reduction. Also, there was no

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prior history of shoulder pathology. Physical examination of the patient revealed absent radial and ulnar pulses, total paralysis below shoulder with marked limitation of passive motion, swelling and tightness of forearm and arm, with coldness and numbness of right upper extremity below elbow. There were no axillary mass or hematoma formation. Doppler study revealed absence of flow in both ulnar and radial artery. Digital Substraction Angiography (DSA) showed diminished flow at the level of tuberculum minus without signs of extravasation (Figure 2). Emergent vascular exploration was performed. Intraoperatively there was no sign of major bleeding or hematoma formation. It was observed that tuberculum majus had tackled and lifted anterior circumflex humeral artery upwards, resulting with obliteration and thrombosis of axillary artery. Macroscopically, distal musculature was noncontractile, looking pale and ischemic. Following release of axillary artery, proximal and distal embolectomy was attempted. After failure to achieve adequate blood flow after 5 consecutive embolectomy trials, resection of segment and end to end grafting was performed using saphenous graft. Multiple fasciotomies were made on arm and forearm as well. Despite all efforts viability of upper extremity could not be achieved and mid humeral amputation was made, 52 days after initial trauma (Figure 3).

Table 1. Previous reports of axillary artery injuries following shoulder trauma resulting with loss of extremity or patient.

Author	Year	History	Result
St John et al. [20]	1945	Spontaneous rupture of axillary artery with noncontact trauma, fatal hemorhage	Loss of patient
Lodding and Angeras. [21]	1988	Dislocation- Longitudinal tear of axillary artery	Loss of patient
Cook and Varley. [22]	1992	Fracture- Large hematoma- compression to trachea	Loss of patient
Modi et al. [23]	2008	Fracture- neurologic deficit- Hb. fall, muscle ischemia, sepsis. Repair- debridements- Amputation declined	Loss of patient
Mouzopoulos et al. [24]	2008	Fracture- Axillary artery rupture, compartment syndrome, rhabdomyolysis, acute renal failure	Loss of patient
McKenzie and Sinclair. [25]	1958	Dislocation- Late admission, laceration of axillary artery, thrombosis of both artery and veins, gangrene	Mid humeral amputation
Gibson. [26]	1962	Spontaneous rupture of axillary artery, failure of revascularisation, secondary bleeding	Shoulder disarticulation
McQuillan and Nolan. [27]	1968	Fracture-dislocation, laceration + distal thrombosis, recurrence of thrombosis	Mid humeral amputation
Smyth. [28]	1969	Fracture- Axillary artery thrombosis, failure of revascularisation	Loss of patient
Sathyarup et al. [29]	1988	Fracture: Axillary artery thrombosis	Forearm amputation
Ng et al. [30]	1990	Fracture dislocation, proximal laceration of axillary artery, crush syndrome with acute renal failure	Shoulder disarticulation
Syed and Williams. [31]	2002	Fracture- Late onset ischemia, pseudoaneurysm	Shoulder disarticulation

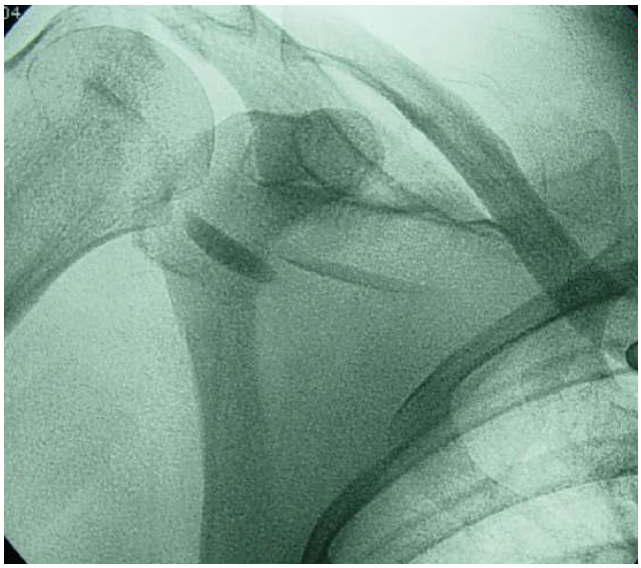

Figure 1. AP roentgenogram shows minimally displaced fracture of greater tuberosity.

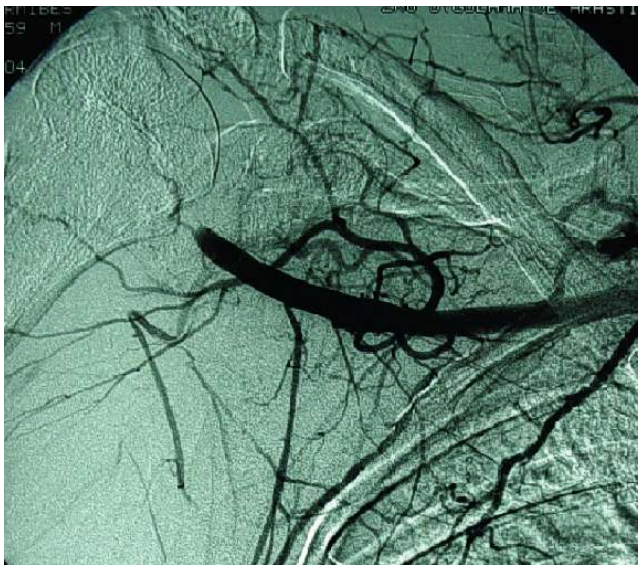

Figure 2. Diminished arteriel flow at the level of tuberculum minus at angiography (DSA) image.

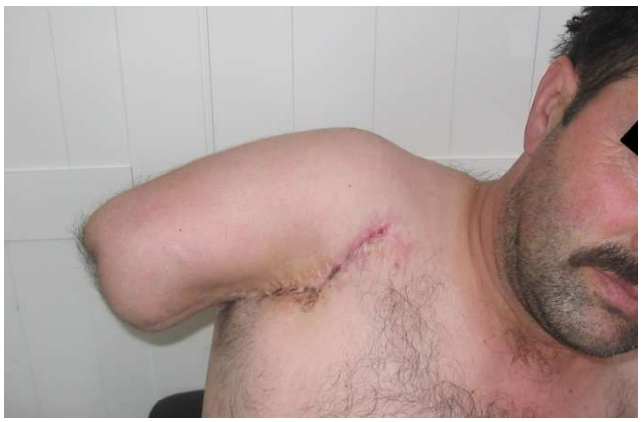

Figure 3. Mid humeral amputation of the patient.

Discussion

More than 200 cases of axillary artery injuries following blunt shoulder trauma have been reported [7]. Forty-nine cases of these reports were following shoulder dislocation. Although most of the cases had resulted with favorable results, there are reports of casualities and loss of extremity.

Axillary artery is anatomically divided into three parts. First portion lies proximal to upper edge of pectoralis minor muscle whereas second part runs deep to the pectoralis minor muscle. Third part is the distal portion of artery which runs from lower edge of pectoralis minor muscle and distal edge of teres major [8]. Third part of artery gives circumflex humeral branches and subscapular branch to opposite directions. This condition makes third part of the artery relatively inflexible, therefore more vulnerable to traction injuries. It was reported that 86 % of axillary arterial injuries were at the third part of the axillary artery. Especially just distal or proximal part of which subscapular artery arose is the common site for tear [9]. Another proposed mechanism is the pericapsular scarring of the shoulder grid. On 27% of cases of axillary artery injuries following anterior shoulder fracture/dislocations, there was a prior history of shoulder trauma. Periscapular scarring, following previous injuries (mainly recurrent dislocations), cause adhesions around the third part of the axillary artery, resulting with increased risk for traction injuries. Age is another main risk factor for axillary arterial injury following shoulder dislocations. It was reported that 86 % of cases of axillary artery injury following shoulder dislocations were older than 50 years of age [9]. Third part of the axillary artery is also a common place for atheroma plaque formation and atherosclerosis [1]. This property also decreases elasticity of the artery, thus increases risk for injury. This finding also correlates with age distribution of cases.

An enlarging mass in axilla and diminishion of pulses are common findings in axillary arterial injuries. The mass effect of hematoma may cause neurological deficit by either direct compression of nerve or ischemic neuropathy by decreased flow of the vasa nervosa [10-12]. At 95 % of cases with axillary arterial

injuries, radial and ulnar pulses were absent. Due to collateral circulation, presence of pulses doesn't rule out diagnosis of axillary arterial injury. One finding of arterial injury in presence of pulses is diminution of biphasic pattern of flow in Doppler study [1]. Persistent decreased hemoglobin also might be an indicator of axillary arterial bleeding.

Brachial plexus injuries, as much as 60 % may accompany these lesions and may be difficult to differentiate progressive muscle paralysis from traumatic plexopathy in delayed cases. As progressive ischemia cause muscle paralysis also, differential diagnosis may be difficult. In early phase electro diagnostic tests can be insufficient discriminating plexopathy from ischemia. In such cases an MRI of brachial plexus might help diagnosis of stretched or lacerated nerve roots [11-13, 22].

Axillary artery pseudo aneurysm is a late complication of vascular injury following shoulder dislocation. There are numerous published reports of late pseudo aneurysms originating from the third part of the axillary artery [13-17]. Pseudo aneurysms may present as progressive decrease of hemoglobin with large pulsatile mass at axilla, resulting with late onset ischemia with neurological symptoms.

Weakening of radial pulse or decreased pressure of involved extremity prior to reduction is a risk factor for vascular injury. Also if vascular status (pulse and flow) don't return to normal level following reduction, extension of vascular evaluation is advised. Eastcott et al. defined the condition as "imminent condition of severe irreversible acute ischemia" [18]. According to this condition danger signs were defined as: [1] waxy pallor, [2] persistent pain and numbness following reduction, [3] coldness of extremity in relation to opposite side, [4] muscle weakness or paralysis, [5] tenderness or rigidity [18]. If angiography reveals extravasation, damage or pseudo aneurysm formation, spontaneous recovery cannot be expected and early vascular intervention is indicated [16]. Although most of the cases were treated with open surgery, successful endovascular interventions have also been reported [19-21].

Since most injury types are tears, lacerations or avulsions of the axillary artery, tethering of circumflex arteries during reduction of dislocated shoulder is an unreported mechanism of injury. We hypothesized that, as the shoulder was relocated, tuberculum majus held and tethered anterior circumflex humeral artery upwards and caused tethering of axillary artery together. This resulted with total obliteration of artery at the level of circumflex branches.

Treatment options includes primary repair, synthetic or saphenous grafting, embolectomy as well as endovascular interventions. Since duration of ischemia is the main determinant of extremity viability, awareness of ischemia is the most important point of diagnosis and treatment of such injuries. Since our case was referred to our clinic two days after initial trauma, despite all efforts aiming perfusion and avoidance of reperfusion injury, extremity survival could not be achieved.

Our case is unique due to the mechanism of injury. Although one cannot predict such an arterial injury mechanism while reducing the shoulder, every surgeon should be aware of potential injuries of the axillary artery and should take precautions to prevent such devastating results.

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