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Pulseless Upper Extremity Following Closed Reduction of a Dislocated Shoulder: A Case Report and Review of the Literature

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Abstract

Anterior shoulder dislocation is one of the most common orthopaedic injuries treated in the emergency department. While associated vascular injuries are rare, they can be a potentially devastating complication, usually seen after high-energy or penetrating trauma. Therefore, a complete neurovascular exam is critical on initial presentation. We present the case of a 58-year-old male who experienced severe right upper extremity pain and numbness approximately eight hours after closed reduction of an anterior shoulder dislocation. He was ultimately found to have thrombosis of the axillary artery requiring emergent embolectomy and prophylactic fasciotomies. Although uncommon, vascular injury must be ruled out in all patients with a shoulder dislocation before and after closed reduction.

Introduction

Shoulder dislocation is one of the most commonly treated orthopaedic injuries in the Emergency Department (ED) with an incidence between 23.9 and 26.9 per 100,000 person-years [1]. It is estimated that greater than 90% of shoulder dislocations are anterior, often the result of excessive external rotation and hyperabduction of the arm [2]. Frequently, shoulder dislocations are associated with Hill-Sachs and Bankart lesions [3]. In most cases, closed reduction is indicated [4].

Vascular injury is a rare complication after shoulder dislocation and is reported in fewer than one percent of cases [5]. It is commonly due to penetrating trauma and is exceedingly rare after blunt trauma [6]. The mechanism behind blunt trauma induced vascular injury involves microvascular damage to the arterial wall, which increases the presence of clotting factors and can result in vessel thrombosis, aneurysm formation, and/or tearing of the vessel wall. The axillary artery is the most commonly damaged vascular structure after blunt trauma induced shoulder dislocation, specifically at the segment found distal to the lower edge of the pectoralis minor muscle (90%) [7]. Shoulder dislocation associated with acute vascular injury is a potential surgical emergency that requires immediate vascular surgery consultation.

We present the case of a 58-year-old male who sustained an acute anteroinferior glenohumeral joint dislocation. Upon initial presentation the Emergency Department (ED), he was closed reduced and placed in a sling. He was found to be neurovascularly intact on post-reduction exam. Approximately eight hours

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later, he re-presented with a pulseless upper extremity secondary to axillary artery thrombosis. He ultimately required operative intervention including embolectomy and multiple fasciotomies. This case highlights the importance of maintaining a high index of suspicion for vascular injury before and after closed shoulder reduction, as well as timely surgical intervention for this potentially limb threatening condition. Informed consent for the publication of this case was provided by the patient.

Case report

A 58-year-old male with HIV (unknown CD4+ count), schizophrenia, bipolar disorder, and polysubstance use disorder presented to the ED after an altercation in which he fell on his right shoulder. He was intoxicated on initial. Right upper extremity radiographs revealed an anterior shoulder dislocation with associated Hills Sachs deformity **(Figure 1)**. His distal neurovascular status was intact. He was closed reduced and placed in a sling. Repeat imaging showed an appropriately located humeral head **(Figure 2)**. Immediate post-reduction physical exam was unchanged.

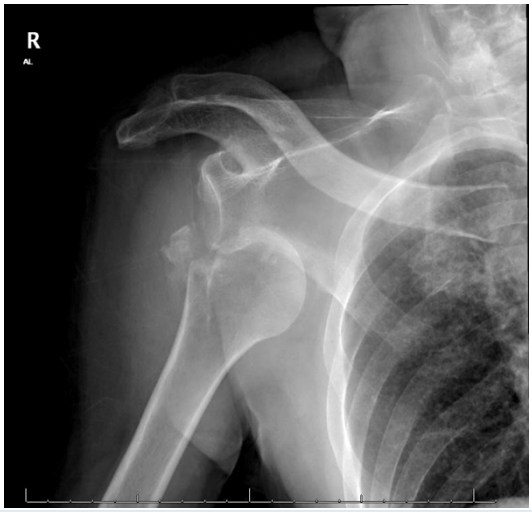

Figure 1: Pre-reduction.

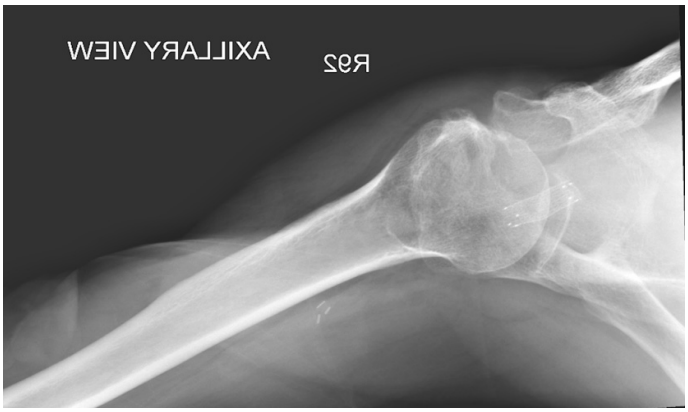

Figure 2: Post-reduction.

Approximately eight hours after initial closed reduction, he re-presented to the emergency department with severe right upper extremity pain and numbness and was found to have a pulseless extremity. On physical exam, he had sensory and motor deficits in the ulnar, radial, and median nerve distributions. His hand was cold, and his fingers were mottled. Ultrasound of the radial and ulnar arteries was performed revealing the absence of blood flow. Repeat x-rays did not demonstrate any new acute fracture or dislocation. The shoulder remained located. CT angiography revealed thromboembolic occlusion of the distal axillary artery without reconstitution distally. Vascular surgery was consulted.

Approximately six hours after re-presentation, the patient was taken to the operating theater for right upper extremity embolectomy with vascular surgery. A transverse axillary arteriotomy was made and an embolectomy catheter was introduced. The balloon was inflated and two passes of the balloon yielded significant thrombus followed by a clean pass. The distal brachial, ulnar, and aberrant radial arteries were also embolectomized, yielding no thrombus. The arteriotomy was closed with interrupted prolene sutures. Post-embolectomy doppler ultrasonography showed strong signals proximal and distal to the closure.

Due to prolonged ischemia and concern for reperfusion injury, prophylactic fasciotomies of the right forearm and hand were performed along with carpal tunnel release. Prior to closing, dopplerable radial and ulnar pulses at the distal wrist were present along with signals at the distal phalanx pulp of the right thumb and right ring finger. The index, middle, and small fingers did not have a dopplerable signals at the pulp, however, the pulp was pink with brisk capillary refill. The incisions were closed and dressed with sterile gauze, cast padding, and Ace bandage.

The patient had an uncomplicated post-operative course and was discharged home on post-op day three. Prior to discharge, he was found to have an intact distal neurovascular status consistent with his pre-injury baseline. He has followed closely in orthopaedic trauma clinic for approximately four months post-operatively. He reports some stiffness of his right shoulder and pain with overhead movements but is able to fire anterior interosseous, posterior interosseous, and ulnar motor nerves with sensation intact to light touch intact across the median, radial, and ulnar nerve distributions. He had palpable radial and ulnar pulses with brisk capillary refill in all fingers.

Discussion

The glenohumeral joint, or shoulder joint, is the most commonly dislocated joint in the human body, with studies estimating that between 15-50% of traumatic dislocations involve the shoulder [8,9]. Specifically, anterior shoulder instability is the most common type of shoulder instability, accounting for over 80% of reported cases of shoulder instability in athletes and over 90% of glenohumeral dislocations [2,10]. The glenohumeral joint displays only 30% of surface contact between the humeral head and glenoid fossa, leaving other soft tissue components such as the labrum, glenohumeral ligaments, rotator cuff, and deltoid muscles as the primary stabilizing components of the joint [11]. Specifically, the anteroinferior component of the glenohumeral ligament complex is the weakest component in an abducted and externally rotated shoulder, introducing a higher risk of anterior dislocation.

Anatomically, the glenohumeral joint is located near several important neurovascular structures. The joint receives its main vascular supply from the posterior and anterior circumflex humeral arteries, both of which originate from the axillary artery [12]. Specifically, the third segment of the axillary artery, located distally to the lower edge of the pectoralis minor muscle, is in close proximity to the anteroinferior glenoid. Innervation of the glenohumeral joint is primarily provided by the suprascapular, lateral pectoral, and axillary nerves, all of which arise from the brachial plexus in the proximal portion of the upper

extremity [12]. The axillary nerve arises from the posterior cord of the brachial plexus and courses closely to the glenohumeral joint capsule, lying an average of 12 mm from the infra-glenoid tubercle [13]. As such, patients who suffer anterior glenohumeral dislocations are at increased risk for neurovascular injury involving the axillary nerve and artery.

There are several reported complications of anterior shoulder dislocation, including damage to bony, ligamentous, vascular, and nervous tissue. Common complications include Hill-Sachs and Bankart lesions, as well as injuries to the anterior glenoid labrum. Approximately 10% of patients suffer from clinically significant injury to the axillary nerve, although studies utilizing electromyography have suggested an even higher percentage of patients may be affected [9,15]. Vascular injuries, such as the one described in the present case report, are far less common and are seen in fewer than one percent of patients with anterior shoulder dislocations. A five-year retrospective analysis performed by Sparks et al. (2000) examined 1565 patients with upper extremity dislocations, of which 931 were shoulder dislocations. Of the 931 shoulder dislocations, just nine (0.97%) experienced arterial injury. The axillary artery was affected in all nine cases, and eight patients ultimately required operative intervention [21]. Despite the low prevalence of shoulder dislocation associated vascular injury, early identification is critical to prevent permanent neurovascular injury in the setting of prolonged limb ischemia [9].

A thorough examination of the literature reveals eight case reports detailing vascular injury after shoulder dislocation. Six case reports describe upper extremity arterial thrombosis as a complication following reduction or surgical intervention [6,16-20]. All six cases occurred in males over the age of 40 (range 40-70). Five (83%) patients experienced thrombosis of the axillary artery, with the other case involving the subclavian artery. All six dislocations were managed with closed reduction, however, only three cases revealed thrombosis during the initial presentation. Of the five (83%) requiring operative intervention, three underwent prophylactic fasciotomies.

Axillary artery thrombosis following closed reduction of an anterior shoulder dislocation is rarely reported in the literature, despite the relatively high frequency of shoulder dislocation relative to other orthopedic injuries. The majority of reported cases of axillary artery injury in the setting of glenohumeral dislocation occurred in patients over the age of 50, with loss of arterial elasticity and increased atherosclerosis considered major contributing factors [22]. Arterial thrombosis, a rare subset of arterial pathologies following traumatic musculoskeletal injuries, is thought to be due to due to a combination of stasis, hypercoagulability, and endothelial damage, otherwise known as Virchow's triad [23]. In the setting of glenohumeral dislocation, the close proximity of the axillary artery to the anteroinferior glenoid likely predisposes the artery to endothelial damage following dislocation. More often, traumatic orthopedic injuries are a strong risk factor (odds ratio >10) for venous thrombosis, rather than arterial thrombosis, making the present case unique [23].

As such, it is important for physicians to recognize vascular injury as a possible complication of traumatic shoulder dislocation. "Hard" signs of significant vascular injury include pain, pallor, pulselessness, parasthesias, paralysis, pulsatile bleeding, or expanding hematoma. These signs suggest the chance of significant vascular injury to be over 90%. "Soft" signs have a relatively lower risk (30-35%) of vascular injury and include diminished pulses, nonexpanding hematoma, and peripheral nerve injury [24]. While our patient did not exhibit symptoms immediately after reduction, our findings underscore the importance of performing a post-reduction physical exam. The gold standard for diagnosis of arterial injury is CT angiography, although Doppler ultrasonography is often used at bedside as a faster, cheaper, and less invasive method for initial evaluation [25]. These methods are particularly helpful in differentiating thrombosis from other similarly presenting pathologies like arterial spasm, pseudoaneurysm, and arterial transection [16].

Conclusion

Axillary artery injury is a rare, yet devastating sequelae of glenohumeral dislocation. Failure to identify such an injury can result in severe vascular compromise, irreversible damage, and potential limb loss. Othopaedic and vascular surgeons should be immediately if vascular injury is identified. This case is an important contribution to the current literature as it describes a delayed presentation axillary artery thrombosis following anterior shoulder dislocation. The present case report emphasizes the importance of prompt recognition and intervention in patients with acute vascular injury following shoulder dislocation and/or closed reduction.

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