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Case Report

Subcoracoid Acromioclavicular Type VI Dislocation with Neurovascular Involvement: A Case Report and Literature Review

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Abstract

Type VI Acromioclavicular (AC) dislocations are rare injuries characterized by inferior dislocation of the lateral clavicle into a subacromial (type VIa) or subcoracoid (type VIb) position. There is no mention of type VIb AC dislocations with vascular involvement (axillary and subclavian vessels) in current literature. Literature review only describes possible neurological involvement and different reduction and stabilization techniques with possible ligaments repair or reconstruction. We report the case of a 20-year-old man with type-VI right AC with right axillary-subclavian artery traumatic rupture and brachial plexus damage. He underwent urgent endovascular repair and subsequent open reduction of AC dislocation and K-wires stabilization. He survived hypovolemic shock, but reported brachial plexus injury, which resulted in upper limb paralysis. No further episodes of dislocation occurred.

Keywords: Acromioclavicular Joint; Subclavian Artery; Dislocations; Coracoacromial Ligaments

Introduction

Acromioclavicular (AC) joint dislocations are 9% of all shoulder injuries [1]. They are classified into six types, according to Rockwood et al [2]. Type VI injuries are rare and characterized by the disruption of the acromioclavicular ligaments with inferior dislocation of the lateral clavicle into a subacromial or subcoracoid position. According to many authors, [3-5] supracoracoid dislocations are different from subcoracoid dislocations. The latter are provoked by with high-velocity accidents and have other significant associated injuries. Clavicle is often trapped beneath the conjoined tendon of the biceps and coracobrachialis. They are always associated with disruption of the coracoclavicular ligaments. Subacromial supracoracoid dislocations, on the other hand, are associated with lower velocity accidents and less severe concomitant injuries and coracoclavicular ligaments remain intact. Because of these differences, type VI acromioclavicular joint dislocations are subclassified into type VIa (subacromial, supracoracoid) and VIb (subcoracoid). Coracoclavicular ligaments (conoid and trapezoid), which are considered primary stabilizers of AC joint [6], are disrupted when dislocation is subcoracoid but remain intact in the subacromial type. Surgery is always needed in type VIb and conservative management before proceeding with operative intervention should be taken into consideration for type VIa [4]. The most common mechanism of injury of a type VI AC dislocation is hyper abduction and external rotation of the shoulder [7]. AC dislocation can be associated with other injuries, such as clavicle, coracoid, acromion and rib fractures but also with neurovascular lesions (brachial plexus, spinal accessory nerve, musculocutaneous nerve) (6). Type VIb AC dislocation can result in brachial plexus and axillary or subclavian vessels compression because of the narrowed subcoracoid space. In the current literature

only few cases of type VI sub coracoid AC dislocation have been reported, among which only 12 are sub coracoid. Associated neurologic lesions of brachial plexus, spinal accessory nerve [1], and musculocutaneous nerve are documented in only 5 existing case reports. There is no mention of type VIb AC dislocations with vascular involvement (axillary and subclavian vessels) in all the current existing literature. The only type VI AC dislocation with vascular complications reported in current literature is a type VIa with mention of transient blue and white discolouration of the upper limb [3]. Considering the typically high-energy mechanism of type VI AC sub coracoid dislocation, it is surprising that there is no report of vascular or persistent neurological damage in this injury. Although the presence of arterial lesions in dislocating injuries of the shoulder are rare, sometimes can represent an important factor in the overall surgical management and if not early diagnosed can led to severe complications. We report a rare case of a young adult male who presented a type VIb AC dislocation associated with an homolateral axonal injury to the upper trunk of the brachial plexus and an homolateral axillary-subclavian artery traumatic rupture.

Case Report

A 20-year-old man with no relevant medical history presented at the emergency department reporting type-VI right AC dislocation, displaced and comminuted open left tibio-fibular shaft fracture (Gustilo IA) and homolateral medial malleolar fracture, multiple rib and vertebral fractures, after being injured in a motorbike accident. The AC dislocation was provoked by high-speed impact against a road pole. All lesions were diagnosed after a total body CT scan, executed on patient arrival at the emergency department, according to hospital protocols for polytraumatized (Figure 1). The patient arrived unconscious, hypotensive with an arrhythmic heart rate. After the early first aid, a low level of haemoglobin was noticed. The clinical scenario suggested a possible hypovolemic shock and therefore a total body CT scan with contrast was immediately requested to rule it out. The CT scan showed active spreading of contrast in arterial phase from the subclavian right artery, a homolateral hematoma (13x10cm) in the subclavian region, suggesting a right axillary-subclavian artery traumatic rupture in addition to type VI right AC dislocation (Figure 2). The patient immediately underwent an endovascular repair of subclavian/axillary artery injury with a covered stent (VIABAHN® 6mmx15cm). After the endovascular repair surgery, the patient underwent orthopaedic surgery. Surgical open reduction of the AC dislocation was performed with open access to AC joint and manual manoeuvres; the joint was stabilized with 3 K wires. No coracoclavicular ligament repair was performed (Figure 3-6). The open shaft tibia fracture and medial malleolar fracture were treated respectively with external fixation (XCaliber® Orthofix) and with cannulated screw. The following days a complete upper right limb paralysis with hypo/anaesthesia of the homolateral axillary region was found. After three weeks electromyography and evoked potentials were performed which demonstrated severe nerve conduction disorder along the somatosensory pathways and axonal injury to the upper trunk of the right brachial plexus. Five weeks after the accident and after clinical and radiographic examination, the three K wires were removed from the right AC joint (Figure 7) to perform brachial plexus MRI which showed involvement of primary trunks. From the radiographic and clinical examination, the dislocation showed signs of healing. However, the paralysis of the upper limb persisted. At 2-year follow-up the patient reported complete upper limb paralysis and no signs of AC secondary dislocations.

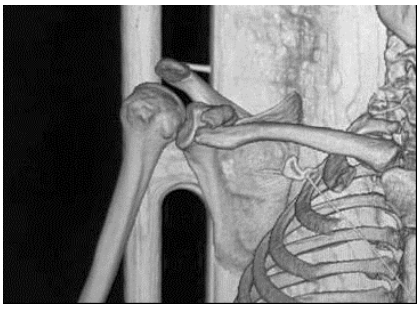

Figure 1: 3D reconstruction of total body CT scan executed on patient arrival at emergency department. Detail of right shoulder.

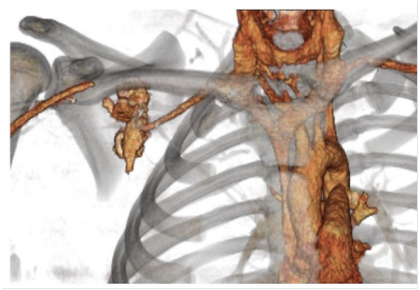

Figure 2: Active spreading of contrast from the right subclavian artery in arterial phase contrast CT scan, suggesting traumatic rupture in addition to type VI right AC dislocation.

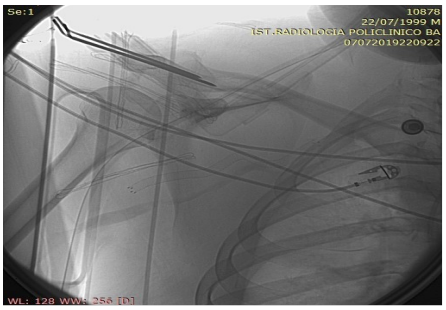

Figure 3: Intraoperative fluoroscopy after reduction and K-wires fixation of the AC joint.

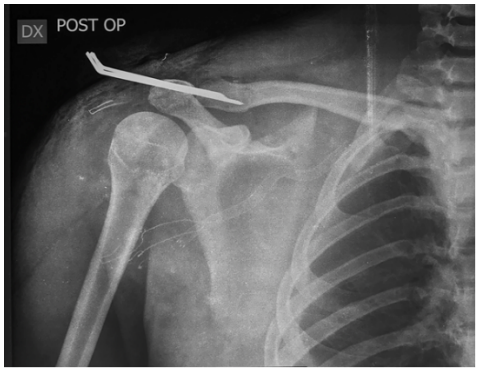

Figure 4: Postoperative X-rays.

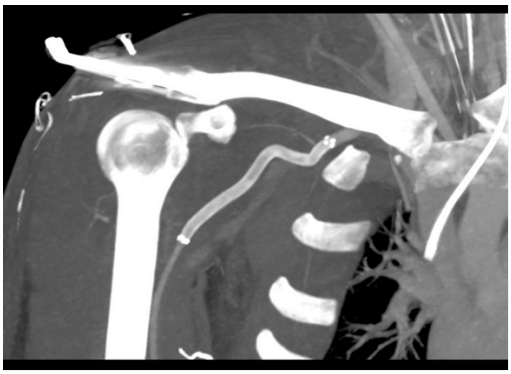

Figure 5: Postoperative CT scan showing subclavian/axillary artery repair.

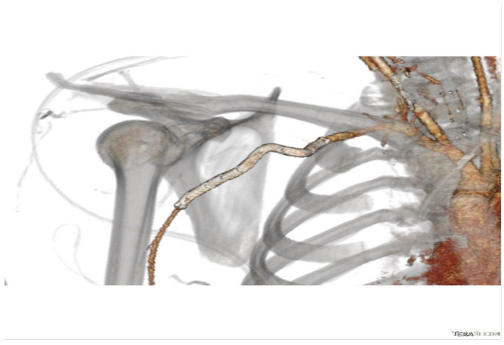

Figure 6: Absence of contrast spreading after vascular repair in the postoperative CT scan.

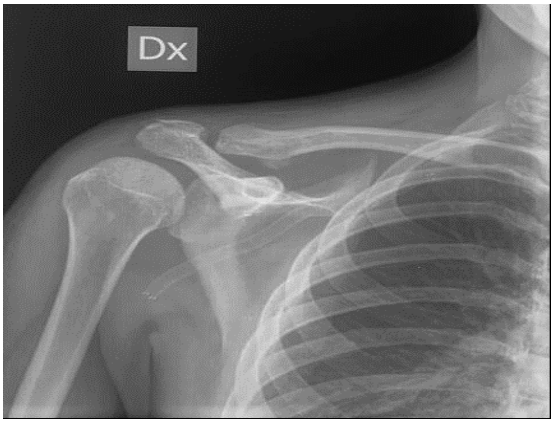

Figure 7: Postoperative X-rays after 5-week removal of K wires.

Discussion

Among the dislocating injuries of the upper limbs, type VI acromioclavicular (AC) joint dislocation is extremely rare and only few case reports are reported in current literature [4]. Type VI AC dislocation can occur with two different patterns: subacromial or subcoracoid [2]. Mechanism of injury consists of extreme abduction and subsequent external rotation of the shoulder with highly intensive load [5]. Traumatic dislocations are often associated to other injuries involving surrounding anatomical structures. In type VI AC dislocations, concomitant clavicular and coracoid fractures, ligamentous ruptures and neurovascular lesions can be observed [8]. The latter represents a surgical emergency if important arterial vessels are damaged. In such cases, a multidisciplinary approach is mandatory. In most of the analysed case-reports, diagnosis of AC dislocation is usually late. As a matter of facts, if not considered in the differential diagnosis and specifically looked for on imaging studies, the lack of specific clinical signs on physical exam combined with other distracting injuries can make it easy to miss, especially in polytrauma patients. In some cases, possible lack of initial local imaging is reported, leading to late diagnosis [9]. For this reason, even though AC joint dislocations are seldom reported, a clinical suspicion during physical exam and radiological assessment is essential to prevent missed or delayed diagnosis of this injury [9]. The commonest reported complication directly caused by late diagnosis is osteolysis of the distal end of clavicle, because of its entrapment under the coracoid process.

The most frequent symptom of presentation, apart from swelling and pain in the acute phase, is mechanical block of shoulder. In our report, clinical signs of a hypovolemic shock were essential for differential diagnosis and instrumental exams made possible to highlight the presence of the arterial lesion. Contrast CT scan was the most diriment exam for diagnosis and permitted a sudden vascular surgery repair of the damaged artery, achieved with a stent positioned via endovascular approach. The ability of the emergency department to promptly request appropriate exams was also determinant to achieve a correct early diagnosis. Although in current literature not many cases of type VI AC dislocation are mentioned, many surgical techniques are reported [3,8,10-13]. Conservative treatment is never possible for type VIb AC dislocations. It can be taken into consideration only in type VIa, also generally surgically treated in acute setting; conservative treatment is possible in chronic dislocations with no sympthoms, no ROM limitations and no distal clavicle osteolysis [4]. In one reported case the type VIa AC dislocation reduced spontaneously and no further treatment was considered [14]. When conservative treatment was attempted in type VIb dislocations [13,15], the difficulty with which the outer end of the clavicle was reduced suggests that open reduction is a best choice. Even if reduction may be possible in the early stages by closed manoeuvres, traction and hyperabduction of the arm may provoke neurologic injuries. Successful closed reduction of subcoracoid dislocations has never been reported and when taken into consideration, was immediately converted into surgical open treatment. On the other hand, if not treated, traumatic AC dislocation results in recurrent dislocation [16]. The integrity of the coracoclavicular ligaments is the greatest concern, so reduction and fixation of the clavicle is often followed by coracoclavicular ligaments reconstruction to ensure the stability of the acromioclavicular joint [3]. When type VIb AC dislocation is surgically approached, clavicle entrapped under coracoid and posterior to intact conjoint tendon is a common intraoperative finding. Coracoclavicular ligaments (conoid and trapezoid) are always disrupted, but in some cases their integrity is reported, avoiding the need for their reconstruction. AC ligaments, articular capsule and intra articular disk are often also damaged: intra articular disk damage seems to induce future AC joint arthrosis. When AC dislocation is tardively treated, osteolysis of distal clavicle is a common finding, compromising future complete shoulder ROM recovery. Clavicle reduction can occasionally be difficult [13] and when reduction is impossible, osteotomy of the coracoid process is performed to unhook the clavicle. It usually happens when surgery is delayed but it is also practiced when the surgeon is reluctant to apply more force on an already compressed brachial plexus. Distal clavicle entrapment by conjoint tendon is a further common complications to reduction. Surgical techniques usually focus on coracoclavicular ligament augmentation because they proved to be primary stabilizers of the acromioclavicular joint. It is reported the use of temporary coracoclavicular lag screw and AC K-wire, ligament repair and imbrication of the deltotrapezial fascia over the top of the clavicle [5]. Use of two Steinmann pins [12], coracoclavicular screw [13] or tension band wiring [3] to stabilize the dislocation are also reported. AC K-wire fixation is also reported. In three cases, only open reduction was performed and no implant was used; in a different case, stabilization was obtained by suturing deltotrapezial fascia and joint capsule [8,16,17]. As far as timing is concerned, treatment is recommended within 2-3 weeks [18]. Despite this, literature shows that even if diagnosis is delayed, surgical treatment can give a good outcome apart from cases in which distal clavicle osteolysis is already occurred [17]. As a matter of facts, delay in reduction of dislocation can lead to clavicle osteolysis which compromises good functional outcome [9]. During follow-up, osteolysis of clavicular distal end, calcification of the coracoclavicular ligaments and joint capsule, narrowing of the joint surface are mentioned. AC joint arthrosis is also a late complication especially when intra articular disk is ruptured and dislocated. However, despite all these radiological findings, all patients had satisfactory functional results [11]. In our case no ligament reconstruction was performed. Since the patient reported complete upper limb paralysis, no evaluation of functional outcomes was possible. The only purpose of the performed treatment was to restore anatomical continuity of the shoulder girdle and to release the neurovascular structures to avoid further damage. This is an important example of teamwork and co-operation between the emergency department, vascular and orthopaedic surgeons that allows a safe and effective management of the patient. To our knowledge this is the first case of a Type VI AC dislocation associated with traumatic rupture of the axillary-subclavian artery and severe neurological complication managed in our department. Within the limitation of this case report, we believe it is mandatory to exclude neurovascular damages prior to any surgical intervention, as this was crucial in our case.

Conclusions

Type VI AC dislocation is a rare shoulder injury, which can be presented with neurovascular damage. Correct identification of injured anatomical structures is fundamental for clinical and surgical management. Vascular repair and subsequent open reduction and fixation with 3 K-wires demonstrated to be a successful and effective technique.

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