Axillary artery injury and brachial plexus palsy as a complication of proximal humerus fractures

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1. Introduction

Proximal humerus fractures are common injuries and represent approximately 5% of all fractures. These fractures are infrequently associated with neurovascular injuries. The axillary artery is commonly ruptured in high energy injuries around the shoulder, which are associated with multiple rupture of nerves. However, the artery or its branches are also easily damaged by low-energy injuries in older people with atheromatous vessels.^{1,2}

This study describes a case of axillary artery injury associated with radial nerve palsy after proximal humerus fracture, based on the literature review, offers suggestions for the early diagnosis and effective treatment of this uncommon injury

2. Case report

An 85-year-old woman with a medical history of allergy to penicillin and acetylsalicylic acid, osteoporosis, arterial hypertension, seropositive for Hepatitis C Virus and instability of gait, fell at home on her outstretched left upper limb. Presented to the emergency room with severe pain in the shoulder area.

Examination revealed a significant deformity and mild swelling with tenderness on palpation and pain with attempted range of motion. An obvious hematoma was present on the shoulder and left side of the chest. The hand remained warm with adequate capillary refill, and no cyanosis was noted. Brachial, radial and ulnar pulses were present. Neurologic testing revealed marked

loss of function of the left upper extremity. Transcutaneous oximetry results were normal.

Initial radiography of the extremity disclosed two parts displaced fracture of the humeral neck according to Neers classification.

Blood test: HB 13.5 g/dl, Hto 39.5%, Platelets: 260,000, TTPA: 22.8 sec, Glucose: 282 mg/dl.

Operative treatment was undertaken through an extended deltopectoral groove incision. Surgical exploration revealed a two-part humeral neck fracture and large organized hematoma, which was evacuated, starting a nonstop pulsatile bleeding from axillary region. Compressive hemostasis was performed but surgeons were unable to identify the vessel source of bleeding. Vascular surgeons team were warned about the emergency. Fracture was reduced and stabilized with locking plate, while containing the bleeding with compressive measurements. Once stabilized the fracture focus, the vascular surgeon intervenes through the same surgical approach. The Axillary artery was isolated and controlled. The lesion consisted on a rupture almost complete of the axillary artery with intimal tearing proximal and distal to the lesion.

Primary repair by end to end anastomosis was dismissed due to the characteristics of the lesion. A first attempt to repair with greater saphenous vein interposition graft was practiced being not effective due to mismatch of calibers. The definitive treatment was achieved by performing a By-pass with ePTFE prostheses anastomosing proximal and distal axillary artery damage. The cords of the brachial plexus appeared uninjured.

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The patient left the operating suit with a strong palpable radial pulse and well perfused left arm. Immobilization was performed with a sling in slight external rotation.

During the immediate postoperative period, the patient presented hypovolemic Shock requiring entry in the Intensive Care Unit for stabilization, where she remained six days with concomitant Diagnostics of: Disseminated Intravascular Coagulation, Congestive Heart Failure, Bronchial Asthma and Nosocomial Infection of lower respiratory tract; until total recovery is accomplished.

The patient stayed admitted in the Orthopedics Department to control evolution until three weeks after admission were accomplished. The patient underwent intensive Physical Rehabilitation, without improvement in the neurological status. Reflexes of the left upper limb were abolished. Paralysis and anesthesia of radial, ulnar and median territories were established.

Follow up after two months from the injury showed a good general status of the patient. Although the results of vascular repair of axillary artery injury were excellent, the outcome was compromised and determined by the neurologic morbidity from concomitant brachial plexus injury. Electromyography (EMG) practiced four weeks after surgery reported a postganglionic brachial plexopathy with severe axonotmesis and affection of the three branches.

EMG practiced sixteen weeks later showed slight improvement in biceps contraction. Radial and ulnar nerve persisted without neurologic activity. The radial pulse was palpable and systolic blood pressure measurements were equal in both arms. The patient has no pain and performs her basic activity of daily life with the use of an orthosis.

3. Discussion

The vascular anatomy in the proximal humerus has an important role to these fractures, since a disruption of the vascular supply could commit not only the humerus head but the viability of entire limb. Despite the anatomic proximity of axillary artery respect to the proximal humerus, its injury is not as often as it might be expected, in the literature there are few reported cases in closed traumatism. Axillary artery along its three parts, provides five branches, which provide an important collateral circulation to the upper limb, being able to maintain the peripheral blood supply without presenting symptoms of acute ischemia, despite complete occlusion of axillary artery.³

A well perfused limb does not discard the existence of vascular lesion, however other indirect signs such as expanding hematoma, distal paresthesias or decrease in hematocrit oblige us to discard this pathology. For these reasons, it is of paramount importance to maintain a state of high suspicion of vascular injury with a proximal fracture of humerus accompanied by any of these signs.^{1,5,7}

An incidence of up to 43% of injury to the brachial plexus in patients with axillary artery lesion has been described. The proximity of these two structures predisposes the hemorrhagic lesion, to a compression of the brachial plexus and subsequent ischemia of nervous trunks because of the formation of a

hematoma or inflammation of the peripheral tissue. However, direct injury to nerve structures by the fracture focus is one of the most frequent causes of neurological injury. This neurological lesion implies the principal affection to patient's functionality outcome.⁴

Early diagnosis and treatment of vascular lesions are the basis to prevent prolonged time of limbs ischemia with devastating results as the upper limb amputation or even patient death. Repeated neurovascular evaluations, along with complementary studies as Doppler ultrasound and angiography tests methods are essential to diagnose this complication.

When the lesion is clinically evident or visible by angiogram, the gold standard consists in open reduction and internal fixation (ORIF) of the fracture followed by vascular repair. The type of vascular repair will depend on the type of arterial injury, being able to practice thrombectomy, endarterectomy, primary repair, saphenous vein interposition graft, anastomosis terminal or vascular prosthesis. The use of Endovascular techniques has spread in this type of pathology. Ischemia time determines the order and type of definitive treatment that is to be run. Vascular occlusion by itself, can produce permanent neurological damage, so minimizing this time is vital. If the time of ischemia is greater than 6–8 h, prophylactic fasciotomies of forearm is recommended or strict monitoring to detect an acute compartment syndrome.⁶

The decision of performing the osteosynthesis before vascular repair will be determined by the general condition of the patient and time since onset of the vascular lesion. It is recommended to obtain a stable surgical field through prior synthesis of the focus of fracture and thus prevent new injuries and facilitate vascular repair, however, in front of a hemodynamically unstable patient or more than 8 h of ischemia, prior revascularization and then proceed to the osteosynthesis for fracture is recommended.⁵

Conflict of interest

None.

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