



Case report

Axillary artery lesion secondary to fracturing of the proximal third of the humerus: case report[☆]



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ABSTRACT

Lesions of the axillary artery are rare in patients with fracturing of the proximal third of the humerus and may have greatly varying clinical manifestations. They are responsible for 15% and 20% of upper-limb artery injuries and the commonest mechanism is a fall to the ground, which accounts for 79% of such injuries. In some cases, the signs only appear later on. It is important to bear this association in mind, so as to make an early diagnosis and avoid serious complications. We report on a case of traumatic injury of the axillary artery secondary to fracturing of the proximal third of the humerus in an 84-year-old patient, with late evolution of clinical signs of ischemia in the limb affected. The aim here was to discuss the diagnostic difficulties and treatment.

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Lesão da artéria axilar secundária a fratura do terço proximal de úmero: relato de caso

RESUMO

As lesões da artéria axilar são raras em pacientes com fraturas do terço proximal do úmero e podem ter manifestações clínicas bastante variadas. São responsáveis por 15% a 20% das lesões arteriais dos membros superiores e o mecanismo mais comum é a queda ao solo, que representa 79% dos traumas. Em alguns casos os sinais só aparecem tardiamente. É importante lembrar essa associação, a fim de diagnosticá-la precocemente e evitar complicações graves. Relatamos um caso de lesão traumática da artéria axilar secundária à fratura do terço proximal do úmero em uma paciente de 84 anos, com evolução tardia dos sinais

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clínicos de isquemia do membro acometido. O objetivo é discutir as dificuldades do diagnóstico e do tratamento.

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Introduction

Injuries to the brachial plexus and axillary artery are rare in patients with fractures of the proximal third of the humerus (FPTH), despite the anatomical proximity of these structures.¹

Injuries to the axillary artery are responsible for 15–20% of the arterial injuries of the upper limbs²; 94% of them are caused by penetrating wounds and the remainder (6%) are due to dislocated fractures of the shoulder. The most common mechanism for the latter is falling to the ground, which accounts for 79% of such injuries.³

The aim of this article was to report on a case of injury to the axillary artery that occurred as a consequence of FPTH, along with the difficulties in making the diagnosis and performing the treatment.

Clinical case

The patient was an 84-year-old woman who was admitted to the emergency service after having suffered a fall to the ground, with right-side FPTH and cranial injury.

On physical examination, edema, hematoma and pain when moving the right shoulder were observed. Neurological examination of the right upper limb showed paresis in the hand and elbow, but this examination was impaired because of the lowered consciousness level associated with the cranial injury. On vascular examination, palpation of the distal pulse and measurement of peripheral perfusion were normal. A radiograph on the right shoulder showed a FPTH with marked medialization of the metaphysis (Fig. 1). Surgical treatment was proposed, but because of the cranial injury and comorbidities, it was not possible to operate on the patient as an emergency.

On the third day of the hospital stay, it was observed that the temperature of the right upper limb had decreased, the distal perfusion had diminished and there was no distal pulse. Emergency surgical exploration was indicated, with the suspicion of thrombosis of the axillary artery. There was no need for preoperative arteriography, since the clinical condition of ischemia was self-evident and this supplementary examination would have postponed the operation and added greater damage to the limb.

By means of the deltopectoral route, arthroplasty was performed in order to resect the humeral epiphysis, because of the severity of the situation and the patient's poor clinical condition. The vascular surgery team performed dissection of the axillary artery and found that it was intact, but with pulse present in the region proximal to the fracture and absent distally. Thromboendarterectomy was performed (Fig. 2) using a Fogarty® catheter in order to completely remove the obstruction of the arterial lumen. Intraoperative arteriography (Fig. 3)

showed another obstruction at the level of the elbow, which was also dealt with in order to achieve limb reperfusion.

In exploring the brachial plexus, we only observed signs of contusion of the median, ulnar and musculocutaneous nerves.

The patient died after the operation, 10 h after she was taken to an intensive care unit, where she had arrived intubated and presenting hemodynamic instability. Her condition progressed to bradycardia, followed by asystole, which could not be reversed. The cause of death was identified as pulmonary thromboembolism.

Discussion

Traumatic injury to the axillary artery, as a complication of FPTH, is rare. Yagubtan and Panneton³ only found 24 cases of injury to the axillary artery subsequent to FPTH described in the English-language literature. A neurological deficit was observed in 46% of the patients and 54% had injuries of the intima layer of the artery, which led secondarily to thrombosis. Vascular repair was performed in all the cases, with an upper-limb salvage rate of more than 89%.



Fig. 1 – Radiographic image of the right shoulder in anteroposterior view showing fracture of the proximal third of the humerus: note medial displacement of the humeral metaphysis.



Fig. 2 – Intraoperative image showing dissection of the axillary artery and arteriotomy: note exit of thrombus from the axillary artery through the opening (white arrow).



Fig. 3 – Image of arteriography of the right upper limb showing arterial obstruction at the level of the elbow (white arrow).

The brachial plexus presents a close relationship with the axillary artery, inside a common fascial sheath. Therefore, any damage to the artery that causes mild edema may lead to nerve compression.⁴ Sukei et al.⁵ emphasized that paresthesia is probably the most reliable symptom of inadequate peripheral circulation. Thus, vascular injury should be suspected when there is a neurological deficit associated with the fracture.

According to Mathei et al.,⁶ suspecting arterial injury is the first and most important step toward making the diagnosis. When clinical signs of ischemia of the limb are present, the diagnosis becomes easy, although in some cases the signs of ischemia may not be evident just after the injury and may only appear later on, with severe consequences for the limb.

Vascular injuries associated with fractures of the proximal region of the humerus are more common among elderly patients. The pathogenesis of these injuries consists of a combination of osteoporosis and atherosclerosis.⁵

The injury mechanisms include direct trauma due to bone spicules or excessive stretching of the artery with the arm in hyperabduction with avulsion or rupturing of the origin of one of the branches. The acute injuries range from laceration of the artery to damage only to the intima layer, which leads to occlusion of the lumen of the vessel. Injuries seen later on include pseudoaneurysm, arteriovenous fistula or thrombosis.^{7,8} Thus, the vascular clinical state should be assessed regularly on the days following the injury.⁹

In the case presented here, the injury mechanism was probably arterial contusion resulting from direct contact with the bone spicule, which led to injury of the intima layer and evolved with subsequent clinical manifestation of total obstruction of the vessel.

The clinical condition of axillary artery injury is often complex and variable. Physical examination is an excellent predictor for detecting arterial injury, with sensitivity of 96%.³ In some cases, greater signs are present, such as active hemorrhage, absence of radial pulse, altered brachial artery pressure and pulsatile hematoma.^{6,10} In other cases, only signs of risk may be present, such as alterations of the distal pulse, pain after reduction and stabilization of the fracture, muscle weakness, numbness, paralysis, stiffness, pallor or one extremity colder than that of the opposite limb.⁴ In our case, the physical examination was somewhat impaired because of the lowering of the patient's level of consciousness due to the associated cranial injury.

Modi et al.⁷ recommended that all patients with FPTH with significant medial displacement of the diaphysis or a medial bone spicule should routinely undergo ultrasonography in order to rule out vascular injuries. In our opinion, given that this examination may be inconclusive in the acute phase, because of not ruling out injury of the intima layer of the vessel, we do not agree with the indication of performing it on all patients with displaced fractures who do not present clinical signs.

This article draws attention to the association between traumatic injury of the axillary artery and cases of FPTH. Even though this association is uncommon, it may lead to disastrous complications when present. In some cases, like ours, the signs only appear later on. It is important to bear this

association in mind, so as to diagnose it early and avoid complications of greater severity.

Conflicts of interest

The authors declare no conflicts of interest.

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