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Case Report

Late recurrent peripheral upper limb ischemia after non-union of a clavicle fracture

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KEYWORDS

clavicle non-union
thoracic outlet syndrome
TOS
arterial thoracic outlet syndrome
ATOS
upper limb chronic ischemia
subclavian artery
internal fixation
plate osteosynthesis
autologous bone graft

ABSTRACT

A 74-year-old woman was referred to our hospital due to recurrent episodes of upper limb ischemia. Her past medical history included a clavicle non-union developed after a clavicle midshaft fracture that had occurred 30 years previously. After a long asymptomatic period, she started showing symptoms of chronic ischemia to the left arm that were misdiagnosed. Thoracic outlet syndrome (TOS) is a rare but possible complication of mal-union and non-union of clavicle fractures; symptoms related to arterial involvement (ATOS) amount to less than 1% of all existing forms of thoracic outlet syndrome. In case of clavicle non-union, local instability plays a key role in determining the initial injury to the vessels and the recurrence of symptoms. Restoration of local bone stability and anatomy, obtained by compression plating and autologous bone grafting, combined with an appropriate vascular surgery, is essential to achieve a clinical resolution of symptoms and to avoid the recurrence of symptomatology as seen in the herein case.

Introduction

Clavicle fractures represent 2.6 to 4% of all adult fractures and 35% of all injuries of the shoulder girdle in the adult; in 50% of cases they are displaced and from 69 to 82% they are located in the middle part of the bone [1–3].

Mid-shaft clavicle fractures are often non-surgically treated. Non-union rates are usually lower than 10%, and can be treated afterwards without serious complications.

In spite of the anatomic proximity with subclavian vessels (the mean distance of the bundle from the posterior border of the clavicle was found to be 9.2 mm) vascular injuries following clavicle fractures are uncommon. The majority of them are associated with a high-energy trauma mechanism, usually due to an acute tearing or compression exerted by fracture's fragments [5–9].

Late vascular complications are also rare but have been described in the literature; they can be secondary to a vascular compression applied by bony callus, mostly in case of mal-union, or to a chronic injury to the vessels in case of non-union [10–13].

Thoracic outlet syndrome (TOS) is a term introduced by Peet et al. [14] in 1956 to describe a wide spectrum of disorders that includes "upper extremity symptoms due to compression of the neurovascular bundle by various structures in the area just above the first rib and behind the clavicle".

Symptoms related to a TOS are mostly neurological [15,16] (more than 90% according to what has been stated by Sanders [17]), whereas symptoms related to an arterial involvement (ATOS) are rare being less than 1% of all forms of TOS.

We present a case of late arterial thoracic outlet syndrome (ATOS) due to a clavicle non-union, which occurred 30 years previously. This case is of particular interest not only because it presents symptoms related to an ATOS (the rarest form of all TOS), but mainly because the symptoms have an extremely late onset from the initial skeletal injury.

Case report

A healthy 74-year-old woman was referred to our hospital with a non-union of the left clavicle complicated by recurrent episodes of a left upper limb ischemia.

Her past medical history included a fracture of the clavicle shaft which occurred 30 years previously (1984) and which was not operatively treated and, therefore, evolved into non-union (Fig. 1).

Since then, the patient hasn't had any problem and symptom for 26 years. However, in September 2010, she started showing signs of chronic ischemia (intermittent claudication) to the left arm. The problem was misdiagnosed and was attributed to the clavicle non-union, thus it wasn't treated.

In May 2013, the patient suffered an acute episode of ischemia of the left arm. She was diagnosed with subclavian artery

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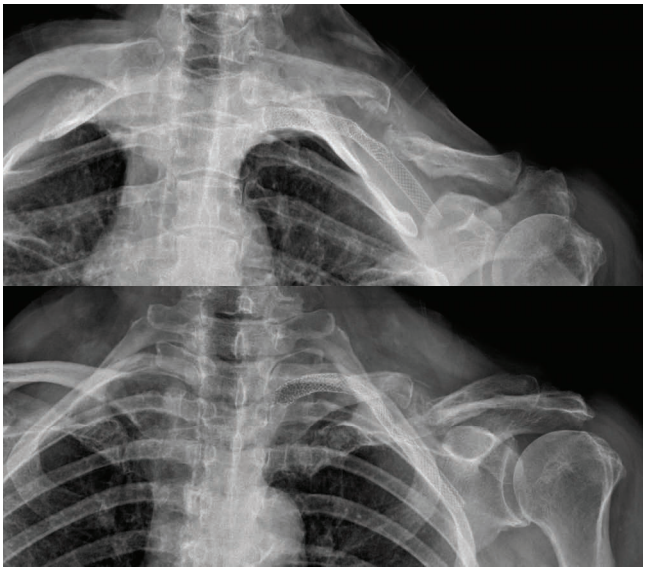

Fig. 1. Pre-operative X-rays showing non-union of the left clavicle.

thrombosis which was treated by open thrombectomy and intra-operative angiography; this procedure depicted stenosis of the artery due to extrinsic compression. A chest CT scan confirmed the clinical extrinsic compression of the subclavian artery exerted by the unstable clavicle.

Two weeks later, as soon as the patient started moving actively the arm, a new episode of acute ischemia occurred to the left arm that was due to a sudden thrombosis of the subclavian artery. She underwent an endovascular procedure of angioplasty and stenting.

However, three months later, she once again had an ischemic episode of the left arm due to the fracture of the stent that was related to the compression applied by the clavicle. She underwent further endovascular procedure of angioplasty and a replacement of the stent with a PTFE Viabahn vascular endoprosthesis. Despite this, she continued to be subjected to symptoms of chronic ischemia of the left arm until September 2013. At this point she was referred to our hospital for an evaluation and treatment of the concomitant left clavicle non-union.

Plain radiographs of the chest and a chest CT angiography showed an extrinsic compression of the subclavian artery (Fig. 2), confirmed also by the 3D reconstruction (Fig. 3).

Due to the unstable vascular situation, we decided to surgically treat the clavicle non-union in order to address both the bone disease and the vascular disease.

The clavicle non-union was treated by open reduction and bone grafting (Fig. 4).

To begin, a resection of the non-union was performed followed by a tricortical autograft harvested from the anterior iliac crest in order to be used to fill the gap left from the non-union resection. In the final phase of this operation, a compression osteosynthesis with an anatomic plate (LCP superior anterior clavicle plate, Depuy Synthes) was obtained (Fig. 5).

Following surgery and the resulted clavicle stability, the patient has been completely asymptomatic until now, without ongoing complaints of any symptoms of ischemia (follow up of 20 months) (Fig. 6).

Discussion

Trauma-related cases of TOS are rare but have specific features in terms of treatment. Sanders et al. [17] divided the different

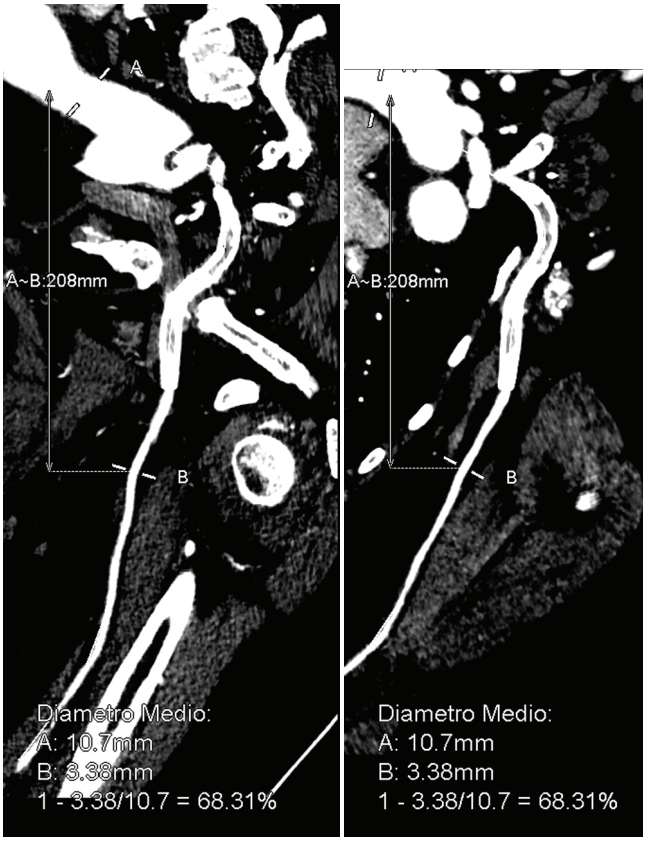

Fig. 2. CT angiography showing the close relationship between the vascular prosthesis and the bony fragments.

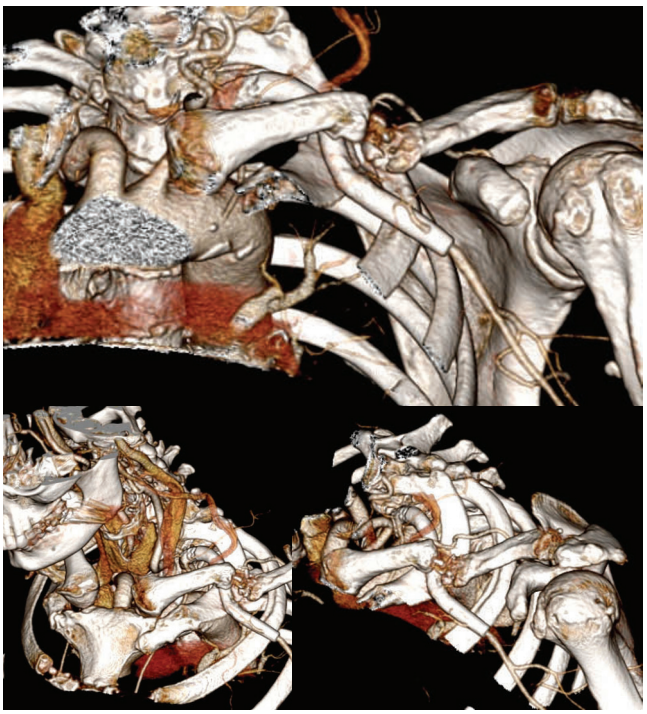

Fig. 3. CT angiography with 3D reconstruction.

forms of TOS in arterial TOS (ATOS), venous TOS (VTOS) and neurogenic TOS (NTOS) adding a modifier that specifies the structure compressed (subclavian artery, subclavian vein or brachial plexus). In the majority of cases the brachial plexus is involved (NTOS), provoking neurological symptoms. The incidence of ATOS has been found to be rare estimated to be less than 1%.