We report the case of a 59-year-old male patient who returned to the Hospital with severe pain in the right upper arm, 9 hours after reduction of an anterior dislocation of the right shoulder. A thrombosis of the axillary artery was quickly diagnosed and the rapid treatment allowed revascularisation in the arm without any major consequences.

Keywords: shoulder dislocation; arterial thrombosis; complications.

INTRODUCTION

Injury of the axillary artery secondary to an anterior shoulder dislocation is very rare. It has been described mainly in association with traffic accidents (9). The high-energy mechanisms implicated in these accidents may cause a great displacement of both humeral head and axillary artery, causing rupture of the artery or severe lesions of the inner layer and adventitia which may trigger its occlusion. In the beginning of the twentieth century, the mortality and morbidity of this rare lesion was high due to the delay in both diagnosis and treatment (2). We report the case of a patient who developed thrombosis of the axillary artery after reduction of an anterior shoulder dislocation, associated with fracture of the greater tuberosity.

CASE REPORT

A 59-year old male consulted for pain, deformity and functional incapacity in the right shoulder two hours after having been assaulted. Physical examination revealed the typical deformity of an anterior shoulder dislocation. Neurological examination was normal and distal pulses were present. Radiographs showed an antero-inferior dislocation of the right shoulder associated with a fracture of the greater tuberosity (fig 1). The reduction was achieved by the Kocher maneuver and the post-reduction radiograph was satisfactory (fig 2), without any modifications in the neuro-vascular status. The arm was immobilised in a sling and the patient was sent to his local orthopaedist for follow-up.

Nine hours later, the patient returned for severe pain in the right upper arm. Physical examination revealed local coldness, paleness, dysesthesias and absence of radial, ulnar and humeral pulse. A further radiograph was identical to the one immediately after reduction. An arteriography was performed, showing a complete contrast stop at the...
level of the axillary artery (fig 3). The patient was operated; resection of the thrombus was performed, followed by an end-to-end anastomosis of the humeral artery and prophylactic fasciotomy in the forearm. The arm was immobilised in a sling during three weeks. Three months later, the functional recovery was acceptable, with some limitation in antepulsion and in the last degrees of rotations. The distal neurovascular examination was correct and the radiograph showed union of the greater tuberosity fracture.

**DISCUSSION**

An arterial lesion secondary to a shoulder dislocation is an infrequent complication (2, 4, 7, 9). In a retrospective study of 1565 dislocations of the upper extremity, Sparks et al (8) identified only 0.97% of arterial lesions associated with a shoulder dislocation. It appears that elderly patients are more susceptible to arterial injury following a shoulder dislocation due to the loss of arterial elasticity secondary to arteriosclerosis (4). Most of the reported cases of arterial lesion occurred in patients over the age of 50.

Various mechanisms have been postulated. Adovasio et al (1) considered that the axillary artery, fixed between the subscapular and humeral circumflex arteries, can be pulled together with the humeral head and elongated. Other authors (2, 4) thought that the pectoralis minor muscle could act as a fulcrum over which the humeral head would bend and compress the artery. In patients with
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recurrent dislocations, the formation of a scar tissue can facilitate the occurrence of this lesion (3). Anyway, it is extremely important that the reduction maneuvers are done as soon as possible and in a gentle way, since these maneuvers could also be a cause of arterial injury.

Absence of distal pulses can be detected by the physical examination prior to, during or after the reduction (6, 9), as occurred in our case in which, instead of a rupture of the artery, a progressive thrombosis occurred. The clinical signs are characteristic of an acute arterial ischaemia: paleness, pain, paraesthesias and absence of arterial pulse. Even though absence of pulse is the rule, occasionally the radial or ulnar pulse can be detected. This phenomenon can be observed when a collateral artery is damaged and either a weak flow still persists through the principal artery or an extensive collateral circulation in the arm can maintain a peripheral pulse. Doppler ultrasonography can help in detecting the pulse but at the same time may it can be misleading because of the collateral circulation that could result in a detectable peripheral pulse. Paraesthesias are probably the most reliable sign of inadequate distal circulation and should raise the suspicion of a vascular injury (4). Other signs, less frequent, include an expanding axillary haematoma which may however be misdiagnosed as the haematoma from the fracture itself. The arterial injury can be associated with a brachial plexus lesion secondary to laceration, compression and elongation, but this association is more frequent in high-energy mechanisms such as traffic accidents or blunt trauma. It is the most important determinant of long-term disability (4).

Although the diagnosis is primarily clinical, arteriography can distinguish between four possible entities: arterial spasm, thrombosis, pseudoaneurism and transection of the artery (1, 3, 5, 6). Transection, partial or complete, is the less frequent lesion, and is more frequently associated with lesions of the humeral artery. In cases with a coexisting injury of the brachial plexus, electromyography offers little help in the acute phase. Nevertheless, MRI can help to distinguish between compression from a haematoma or disruption of the nervous fibers (1, 6).

Treatment of an arterial thrombosis is an emergency. Its success depends on the early diagnosis and the rapidity of the treatment. Successful cases have been published with intra-arterial treatment consisting of thrombolysis and stent application (10). Nevertheless, at present the best option is open surgery, including thrombectomy, end-to-end anastomosis, saphenous vein graft or prosthetic implant (8). Possible avulsion of collaterals should be treated by ligation. After the arterial flow is reestablished and depending on the evolution time there is a high risk that a compartmental syndrome may develop. For this reason, it is preferable to perform prophylactic fasciotomies in the forearm or at least to monitor the compartmental pressure.

CONCLUSION

Peripheral pulse evaluation before and after the treatment of a shoulder dislocation is crucial, and the index of suspicion for an arterial injury should be high. When facing suggestive clinical signs of ischaemia, an arteriography should be done in order to evaluate the characteristics and localisation of the lesion.

REFERENCES


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