CASE REPORT

Progressive neurological deficit in blunt shoulder trauma
“Beware the vascular injury”

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Introduction

Progressive sensory motor deficit of the upper limb following blunt shoulder trauma, even in the absence of fracture or dislocation, should raise the clinical suspicion of a major vascular injury. This may involve an expanding haematoma or pseudo aneurysm of the axillary artery or its branches.

Case report

A 70-year-old man tripped, and landed heavily on his left side, immediately noting severe pain in his left shoulder, radiating into the hand. On admission to the accident and emergency department it was noted that he had a pulse rate of 84, and a blood pressure of 172/97 mmHg. A tense swelling of the left shoulder region with complete sensory loss and motor paralysis of the hand was noted. No neurological deficit was noted proximal to the wrist and the radial pulse was palpable. No other injuries were noted and radiographs of the chest, cervical spine and left shoulder showed no fracture or dislocation.

One hour later the shoulder swelling had increased and there was complete sensory loss and motor paralysis distal to the elbow. The radial pulse was still palpable but sympathetic function not documented. The haemoglobin was 10.1 g/l. A venogram was performed, confirming a large haematoma in the shoulder region and occlusion of the axillary vein. A vascular opinion was sought and non operative management advised.

Twenty-four hours after admission there was a complete sensory loss and total motor paralysis involving the segments C5 to T1, i.e. now the entire forequarter. There was no neuropathic pain in the limb. The radial pulse was still palpable but sympathetic function remained undocumented.

Eight units of whole blood were transfused to maintain a haemoglobin of 10 g/l. Six days after injury, the patient became short of breath. A pulmonary computerised tomographic angiogram showed no evidence of pulmonary embolism but revealed a false aneurysm of the posterior circumflex humeral artery, 2 cm in diameter, with recent
and continuing haemorrhage (Fig. 1). This appeared to be due to a rupture of the posterior circumflex artery. The patient later underwent embolisation of the false aneurysm (Fig. 2) before being transferred, 3 weeks after his injury, to the peripheral nerve injury unit at the Royal National Orthopaedic Hospital, Stanmore.

Operative findings on exposure of the supraclavicular and infraclavicular brachial plexus:

1. Normal appearance and normal proximal conduction for the C5 to T1 nerve roots.
2. The infraclavicular plexus was compressed by a 20 cm diameter haematoma, approximately 3 l in volume, which was evacuated.
3. The entire plexus at that level was flattened, attenuated, and demonstrated no conduction, either distally or across the lesion.

Figure 1 Angiogram showing avulsion injury and false aneurysm of circumflex humeral artery.

Twelve hours after infraclavicular decompression the patient had regained some sensation in the C5 dermatome but no sign of motor recovery elsewhere. Six weeks later he had regained MRC grade 3/5 power in supraspinatus and infraspinatus but the rest of the limb was flail. He had no neuropathic pain.

Discussion

Rupture of the circumflex humeral arteries from the axillary is a recognised event following penetrating trauma, fracture or dislocation at the glenohumeral joint, particularly in the elderly.1,2 Vascular avulsion resulting from blunt trauma is much less common. In blunt injuries a brachial plexus lesion can result from a direct blow or traction injury of the nerves creating an immediate neurological deficit.4,5

Progressive neurological deficit, as illustrated by this case, indicates ongoing neural compression. With no clinical evidence for a dislocation or fracture of the shoulder girdle, this finding strongly indicates the probability of vascular injury. In this case the huge shoulder swelling and anaemia supports that diagnosis.

The following points are noteworthy

1. The presence of a progressive neurological deficit, beginning in the hand and advancing proximally suggests ongoing compression of the brachial plexus. In the absence of a dislocation or fracture, this is likely to represent an injury to the axillary artery or its branches.
2. The circumflex humeral vessels can be ruptured or avulsed from the axillary artery with huge blood loss, in spite of a good radial pulse.
3. The axillary artery may be ruptured or occluded, even though distal pulses are still present via the peri-scapula collateral circulation based on the thyrocervical trunk.3
4. Life threatening blood loss can occur with arterial injury at the shoulder, shown by increasing swelling, progressive neurological deficit and progressive anaemia.
5. If arterial injury is suspected, an emergency angiogram is indicated, not a venogram.
6. In the presence of progressive neurological deficits a non-operative expectant policy is not safe.
7. Ongoing compression of a nerve will initially cause a non-degenerative conduction block (neurapraxia). With continued compression, progressive neuronal death results in a degenerative lesion (axonotmesis, with a poorer prognosis).

Figure 2 Embolisation of false aneurysm.
8. An embolisation may be carried out to prevent further blood loss but this does not deal with the haematoma, or with the compression.

9. Therefore, when assessing progressive neurological deficits after shoulder trauma (or any neurological lesion associated with an articular injury) whether traumatic or iatropathic, it is not safe to assume that a neurappraxic (non-degenerative conduction block) lesion exists. It is unsafe to adopt an expectant policy.

Electromyographic analysis has no place in the urgent assessment of such injuries. Early referral to a specialist nerve injury and repair service invariably produces better outcomes and any delay simply worsens the prognosis.

References