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Late posttraumatic compartment syndrome in the axillary neurovascular sheath—a case report

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A previously healthy 48-year-old man with alcohol abuse fell onto his abducted left arm. Plain radiographs revealed an anterior dislocation of the shoulder joint. The dislocation was easily reduced during light anesthesia and control radiographs were normal. Distal pulses were normal. Neurological examination revealed slight numbness of the lateral aspect of the shoulder and volar side of the forearm and hand which was interpreted as partial sensory loss involving the axillary, ulnar and median nerves. Motor function of the forearm

and hand was normal. The shoulder joint was immobilized with a collar-and-cuff sling and the patient was discharged with a clinical follow-up in 2 weeks.

1 week later, the patient returned because of increasing numbness and swelling of the shoulder and arm and reported that it had started 4 days after the trauma. In addition to substantial edema of the entire arm, abduction of the shoulder joint was not possible. Flexion and extension of the elbow were weak and there was practically no motor

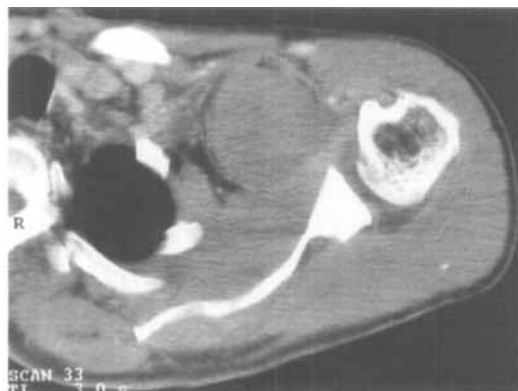
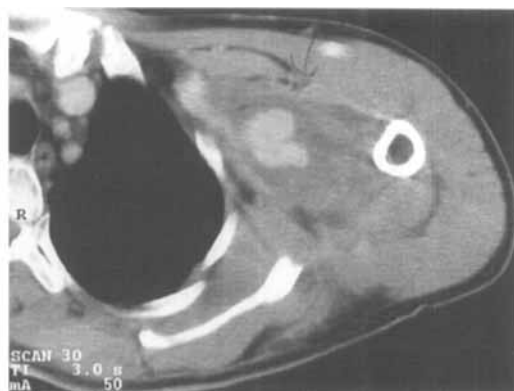


Figure 1. CT of the left shoulder at a level just below the major tubercle. The arrows indicate the limits of the large hematoma. The vascular tear was located at the junction of the circumflex humeral and axillary arteries.



CT of the left shoulder at a level approximately 10 cm below the major tubercle. Note that the hematoma dissected down into the neurovascular sheath well below the axilla. The light areas in the center of the hematoma indicate leakage of contrast fluid.



Figure 2. Venogram of the left shoulder. Note the occlusion of the brachial vein (white arrow). Venous drainage occurred via the cephalic vein (black arrow)

function in the wrist or fingers except for slight wrist extension. Little or no sensation was present in all regions supplied by the axillary, musculocutaneous, ulnar and median nerves. Distal pulses were normal, but the hand was pale with slow capillary filling.

CT-scan of the shoulder revealed a large hematoma in the axilla (Figure 1). A subsequent venogram of the left arm showed occlusion of the axillary vein at the level of the humeral head (Figure 2). Venous drainage from the arm occurred via the cephalic vein.

Compartment syndrome of the infraclavicular neurovascular bundle was suspected and the patient was operated on immediately. On opening

the neurovascular sheath, large masses of hematoma were seen and a major bleeding from the axillary artery ensued. After proximal clamping of the artery, the tear was found at the junction of the circumflex humeral artery and axillary artery. The vascular lesion was sutured.

Postoperative electromyography showed total denervation of the distal and proximal median nerve and the distal axillary, musculocutaneous and ulnar nerves. The findings suggested complete damage of the superior trunk distal to the supraclavicular nerve and partial damage of the intermediate and inferior trunks.

During the first 15 months, the patient recovered partially. According to repeated electromyography, the median nerve showed some recovery, but the ulnar nerve had only few signs of reinnervation. The patient could flex and extend the elbow almost normally, but with weak biceps function. The shoulder could be abducted only a few degrees and there was substantial atrophy of the deltoid, supraspinous and infraspinous muscles. The wrist had normal motion, but weak flexion-extension. Finger extension was normal. Finger flexion was weak in all fingers and the fifth finger had practically no flexion. Thumb opposition was weak. Sensation had returned to all parts of the arm, except the areas supplied by the ulnar nerve.

Although an alcohol-abuser, the patient showed no signs of alcohol polyneuropathy before the injury.

Discussion

Nerve lesions are common complications of shoulder dislocation and have been reported in up to one third of cases (Blom and Dahlbäck 1970, Ebel 1973, Pasila et al. 1978). The rare supraclavicular type results from pathological elongation of the nerves (Leffert 1970) in the basal neck region. The prognosis for recovery is often poor. It may occasionally result from shoulder dislocation (Jerosch et al. 1990). Most infraclavicular lesions are caused by traumatic stretching forces from closed injury to the scapula or the humerus or after dislocation of the humeroscapular joint (Leffert and Seddon 1965, Travlos et al. 1990). These nerve lesions often recover spontaneously within a few months to one year (DeLaat et al. 1994). The

commonest lesion is that of the axillary nerve (Post 1978, Depalma 1983).

Vascular damage is a rare but well known complication of shoulder injuries (Henson 1956, Mustonen et al. 1990). It takes the form of swelling and hematoma of the shoulder and axilla which are usually noted immediately (Jensen et al. 1987). The most vulnerable parts of the axillary artery have been found to be those near the branching vessels (Drury and Scullion 1980). The types of injury reported include intimal tears, avulsion of minor branches and complete transection (Drury and Scullion 1980). Although arterial injury has been frequently reported as a complication of proximal humerus fracture (van Arkel et al. 1998), late appearance of arterial injuries in conjunction with shoulder dislocation has not been reported.

Rarely, vascular damage may also present with concomitant neurologic deficiency (Leffert and Seddon 1965, Nash et al. 1984, Mustonen et al. 1990) with immediate onset of symptoms. Leffert and Seddon (1965), who reviewed 21 cases with infraclavicular brachial plexus lesions, found traumatic aneurysms of the axillary artery in 2 cases, indicating that this combination may not be rare.

Nicholson (1991) reported 2 cases with late presentation of vascular damage, one due to a 3-week-old humeral fracture and the other to anterior dislocation. In the latter case, the symptoms of ischemia, pain and paralysis presented 1 day after the trauma.

Our patient had a tear of the axillary artery, resulting in late development of a hematoma and progressive neurological symptoms with permanent nerve damage.

As pointed out by Nevasier et al. (1993), in a patient suffering the first anterior dislocation after the age of 40, who is unable to elevate the arm after reduction, rupture of the rotator cuff must take precedence over axillary nerve injury in the differential diagnosis. In our case, the patient had normal motor function of the arm, but numbness (slight sensory loss) was noticed corresponding primarily to the axillary nerve. It is therefore likely that there was a true lesion of the nerve impairing abduction. There may have been a concomitant rotator cuff rupture. The severe motor and sensory loss noticed one week later was separated

in time from this episode and was caused by the compartment syndrome in the neurovascular sheath.

In this most unusual case the lesion was located at the junction of the circumflex and axillary arteries. It was the result of the initial trauma or possibly the reduction maneuver. Obviously, the lesion was not large enough to give immediate symptoms. The result was a slowly growing hematoma. The subsequent pressure in the neurovascular bundle resulted in late symptoms. This mechanism may be called a false aneurysm (Downs 1982).

A patient with anterior shoulder dislocation presenting with a swollen shoulder and no fracture on radiography should be observed, with special attention to the circulatory and neurological functions of the arm.

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Pseudoaneurysm of the ulnar artery following use of an external fixator—a case report

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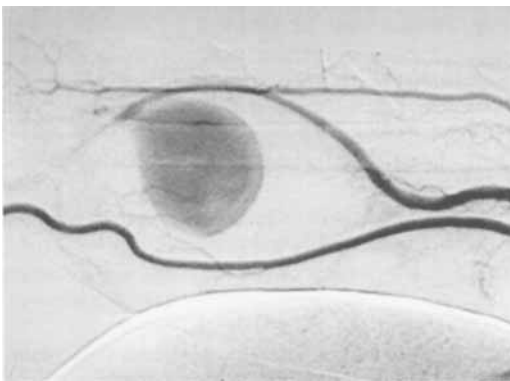
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A 70-year-old woman presented with a Colles' fracture of the right wrist, which was treated by external fixation. There were no signs of vascular or nerve damage during her hospital stay (4 days). The postoperative period was uneventful and the pins were removed after 6 weeks, the distraction having been discontinued after 3 weeks. Range of motion exercises were instituted as soon as the fixator was removed. 2 weeks after the removal of the pins, the wrist became swollen, warm and painful on mobilization. During the following weeks, the pain and swelling increased and a pulsatile mass was palpated on the palmar side of the wrist. An ultrasonic examination showed a false aneurysm with a diameter of 5 cm. Because of its

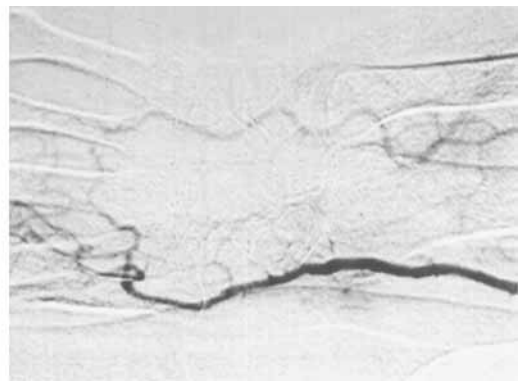
size, the echography could not determine its origin.

Arteriography confirmed the presence of a pseudoaneurysm of the ulnar artery at the site of one of the radial pins (Figure). It also showed a substantial decrease in blood flow in the ulnar artery.

The treatment consisted of manual compression and an elastic bandage. One month later, the aneurysm had disappeared clinically (i.e., absence of a mass, arterial bruit and pain). The disappearance of the aneurysm was confirmed by Doppler examination. After 6 months of follow-up, the aneurysm had not recurred.



Arteriography: aneurysm of the ulnar artery at the site of one of the radial pins.



Decrease in blood flow in the ulnar artery.