AXILLARY ARTERY DAMAGE FROM SHOULDER TRAUMA - A REPORT OF 2 CASES

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ABSTRACT

Various shoulder injuries can give rise to axillary artery complications. Two such cases were admitted to Tan Tock Seng Hospital late last year. Both were young adult males, who had developed axillary artery thrombosis secondary to shoulder injuries. The following is a short discussion on the common types of shoulder injuries, how they result in axillary artery compromise and a short section on their management and treatment.

Keywords : Vascular injuries, axillary artery thrombosis, shoulder dislocation, humeral fractures, clavicular fractures.

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INTRODUCTION

Damage to the axillary artery as a result of shoulder injuries is a serious and not infrequent complication (1-4). They commonly result from anterior dislocation of the shoulder or a fracture of the proximal end of the humerus, or a combination of both. Rarely, fractures of the clavicle can also be accompanied by a vascular injury.

Injuries to the axillary artery can be classified as either Acute or Delayed lesions (5) :

ACUTE injuries include total or partial rupture of the artery resulting in haemorrhage or thrombosis, mechanical compression from bony fragments, hematomas or other surrounding tissues, and arterial spasms.

DELAYED injuries includes false aneurysms, arterio-venous fistulas, or thrombosis of the artery after surgical repair as well as thromboembolism (6).

Below are two such cases. The first was a fracture of the left clavicle and the second a fracture-dislocation of the left shoulder. Both resulted in axillary artery thrombosis.

The first case, with prompt attention, allowed the successful preservation of the left arm. The second case, due to extensive tissue injury and time lag, resulted in a shoulder disarticulation.

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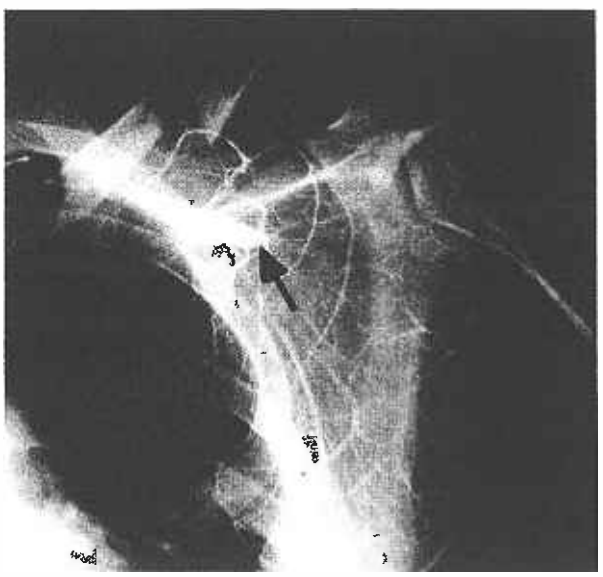
Both cases illustrate the paucity of clinical signs that can accompany arterial occlusions from thrombosis and also show the usefulness of urgent angiograms, where facilities permit, in diagnosing and managing such complications. Though arterial damage from shoulder injuries occur more frequently in older age groups, our two cases occurred in young adult males, under 25 years old.

CASE 1

A 22-year old male Chinese motorcyclist, following a road traffic accident, sustained a closed fracture of his left clavicle. Neurological examination revealed decreased motor and sensory function over the C5/C6 nerve roots. The left radial pulse was initially palpable but progressively grew weaker. An urgent angiogram revealed a left axillary artery thrombosis with collaterals from the left circumflex humeral going into the left branchial artery (Fig. 1).

Fig 1.
Angiogram showing left axillary artery thrombosis (arrow) following a fracture of the left clavicle. The artery was repaired with a venous graft.



Urgent exploration and repair of the artery was undertaken. Intraoperatively, the 2nd part of the axillary artery was seen to be macerated with thrombosis at both ends. The musculocutaneous nerve was avulsed from the lateral cord. A saphenous vein graft was used to repair the damaged artery. Post-operatively, the radial pulse returned and was of good volume.

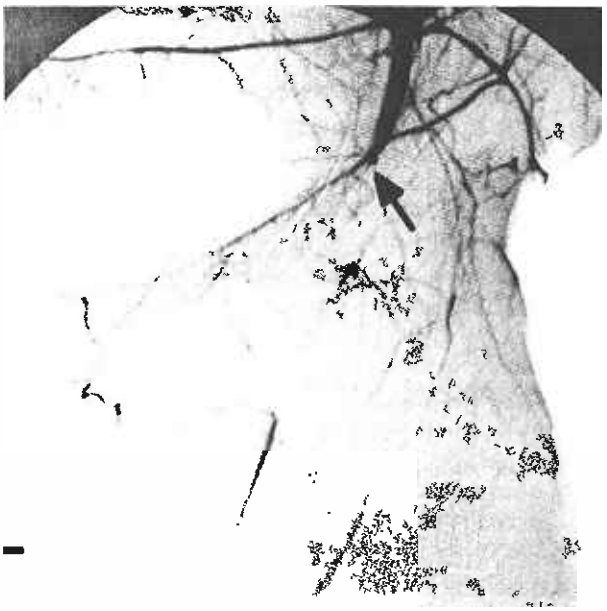
CASE 2

A 25 - year old Malay man was found unconscious on a grass verge, smelling strongly of alcohol. The left shoulder was bruised and swollen and X-rays revealed a close fracture-dislocation of the left shoulder. There were also bruises noted all along his left flank. An urgent manipulation and reduction was carried out for the dislocated left shoulder.

The whole of the left upper limb was found to be stiff and in slight flexion. There was no sensation or movement and it was also comparatively cooler. Capillary return to the fingers was poor. An urgent angiogram revealed a complete obstruction of the left axillary at its origin, with extension of the thrombosis proximally (Fig. 2).

Fig 2.

Angiogram showing complete obstruction of the left axillary artery at its origin (arrow). The arm was clinically not viable and was disarticulated.



The patient subsequently developed crush syndrome with acute renal failure. An emergency shoulder disarticulation was performed. Careful monitoring and treatment to maintain a proper fluid and electrolyte balance was instituted. The patient recovered satisfactorily from the transient renal insult. Serial monitoring showed good return of renal function.

The patient was discharged one month later, with the wound after disarticulation granulating well.

DISCUSSION

Shoulder Dislocations

Shoulder dislocations, especially anterior dislocations with axillery artery damage usually occur in adults in the second half of their 6th decade. One predisposing factor in this age group is the inelasticity of the atheromatous vessels.

During normal extension and abduction of the shoulder, the neurovascular bundle is stretched to some degree (4). Three mechanisms of injury to the axillery artery from shoulder dislocations have been proposed.

According to Milton, the axillary artery is fixed by the circumflex and subscapular arteries. This allows for little escape from damage to the artery from a shoulder dislocation.

Brown et al, suggested that the lateral edge of the pectoralis minor acts as a fulcrum over which the artery is bent. There is also some anchoring effect as the cords of the branchial plexus encircle the artery (3). This can result in an atheromatous artery being injured or avulsed. This is especially so when the arm is abducted and externally rotated in shoulder dislocations.

A third mechanism is proposed by Jardon et al (1). Fixation of the artery by scar to the pericapsular tissue may result in transection of the artery when the humerus head tears the capsule of the shoulder joint. Both their patients had previously dislocated their shoulders.

The most frequently torn site is the 3rd part of the artery, at the origin of the circumflex or subscapular branches (3). The 2nd part may also occasionally be torn.

The axillary artery is relatively fixed at two points : in its 1st part by the thoracoacromial branch as it pierces the clavipectineal fascia and in the 3rd part where the subscapular branch appears and enters the subscapular muscle. It is in this second point situated directly caudad and slightly anterior to the inferior lip of the glenoid fossa that is especially vulnerable to stretch and direct trauma during dislocation. During dislocation, the humeral head opposes on the axillary artery directly at the point where the artery gives off these branches. This is thus the suceptible area (4).

Other associated injuries involve the arterial branches (2) especially the long thoracic, the circumflex or subscapular branches. Injuries to the vein or branchial plexus are also known.

A rare type of dislocation of the shoulder resulting in axillary artery injury is an erect dislocation of the shoulder (luxatio erecta) - one such case was reported by Lev-El et al (4). The dislocation resulted from the application of a violent force to an upraised and abducted arm.

Humeral Fractures

Any one of the 3 segments of the axillary artery can be injured in such fractures. Possible mechanisms include the following (5,7):

1. Direct injury of the artery from sharp bony fragments, particularly the distal fragment of the fractured humerus.
2. Violent overstretching of the artery in the hyperabducted position especially in atheromatous vessels. Simple intimal tear with thrombosis can ensue (7). Diagnosis may be difficult as the pulses may still be intact.
3. Avulsion or rupture of a branch of the axillary artery (the most vulnerable being the subscapular and circumflex branches)

4. Arterial spasm (8)

Hayes and Van Winkle (7) also pointed out that even minimally displaced fractures of the neck of humerus can lead to major axillary artery damage. Displacement at the time of fracture can be much greater than is seen on the X-ray.

Clavicular Fractures

Clavicular fractures are usually not accompanied by axillary artery injury. This is because the neurovascular bundle is 'protected' by the trapezius and subclavius muscles in such fractures.

The distal fragment of the fractured clavicle is pulled downward and forward by the weight of the limb and the proximal part is drawn upward and backward into the belly of the trapezius. Often, other associated injuries like a fracture of the first rib combined with the fractured clavicle, is needed to cause damage to the artery (6).

MANAGEMENT

The collateral circulation about the shoulder is good and the distal circulation may remain strong after injuries to the shoulder result in axillary artery compromise (4). Drapanas (9) found palpable distal pulses in 27% of patients with major arterial injuries.

The key to a successful outcome in such injuries is in early diagnosis and treatment (7). One must always bear in mind the masking effect (10) of the collaterals on the clinical presentation. Hence, a high index of suspicion must be maintained.

Hayes and Van Winkle (7) state that parasthesia is probably the most reliable first sign of inadequate distal circulation, as studies by Parkes (1945) and Tibbs (1962) have shown (5). This sign is especially useful in a fully conscious patient.

In cases of slowly progressing or subacute ischaemia, parasthesia, lividity of the fingers and a weaker radial pulse with colder peripheries are usually the only signs and symptoms present. Furthermore, signs of ischaemia may ultimately weaken or disappear with the opening of compensating collaterals.

Delay in detecting arterial insults due to the paucity of signs may allow the injury to evolve into a more serious arterial complication. This occurs when an undetected intimal tear is followed eventually by occlusive arterial thrombosis (5).

Studies by Miller & Welch (15) have shown that if a limb has its vascularity restored within 1-6 hours of ischaemia, there is a 90% salvage rate, compared with 50% in periods lasting 12-18 hours and 20% in those with ischaemia lasting 24-36 hours (11).

Furthermore, with ischaemia continued for 12 or more hours, the return of function is always delayed and incomplete (11).

Confusion may arise when possible associated neurological damage is involved and this may mimic the motor and sensory disturbances of ischaemia. In such cases (12), the distribution of the neurological signs, the associated absent pulse, and increasingly painful and ominous flexures of the fingers are helpful as distinguishing signs.

Minimal signs should still lead one to suspect possible arterial compromise, especially if the mechanism of shoulder injury is severe or the injury extensive. It is in such equivocal cases that the angiogram comes into play. The indication for the angiogram is given on clinical grounds.

It is helpful in localising arterial damage and demonstrating damage when there is confusion in diagnosis. However, the angiogram is not an absolute criterion needed for diagnosis - especially if the injury is clinically obvious, and/or facilities are lacking or where there is a delay in obtaining such radiographic aids, as measured against the delay in allowing any suspected ischaemia to persist.

One must also bear in mind the possible hazards of angiography, including reaction to the injected media, injury to the artery or the accompanying vein and rarely tissue necrosis from extravasation of the media (13).

TREATMENT

There are essentially three elements in shoulder injuries with associated neurovascular complications - the bony, the vascular and the neurological parts.

The management of traumatic limb ischaemia is not only to reduce the risk of loss of limb but also to prevent future functional restrictions (14).

The method of arterial repair depends on the nature and extent of the damage. They include (1,2,4,5,14) :

1. Endarterectomy.
2. Grafting with a venous or prosthetic graft. Venous grafting may not be easy due to disparity in the size of the lumen and thickness of vessel wall between artery and vein. Also, the graft is easily compressible and it has occasionally been found difficult to close the wound without occluding the circulation (14).
3. Resection with an end-to-end suture. This is only possible in injuries involving small segments as it must be done without undue tension on the reanastomosed vessel.
4. Other procedures include repair of the arterial wall by a venous patch and simple ligation of the vessel. In general, ligation is used in younger patients with good collaterals (1). Even so, it is used in only exceptional circumstances (4).

In cases with arterial spasms, there is still no uniformly successful means of relieving it and the management differs (8).

Treatment ranges from using 2.5 % papaverine (Kinmonth); injecting the lumen with saline between clips, segment by segment (Mustard & Bull); instillation of heparin into the lumen below the obstruction (Bonney); and a combination of fasciotomy, instillation of 2.5 % papaverine and excision of the injured segment initiating the spasm (Seddon).

However, one must bear in mind that this complication does not usually occur in isolation, and that the associated intimal damage to the artery usually results in it being resected.

With the restoration of vascular viability to the limb, post-operative complications include:

1. Decreased function from nerve or muscle damage due to ischaemic exposure.

2. Chronic pain.
3. Infection.
4. Development of edema with restoration of arterial flow.

Factors contributing to the edema include associated venous injury and severe ischaemia itself damaging the viability of the arteries (9). Reichart has shown that its disappearance accompanies the regeneration of lymphatics across the transection.

Miller & Welch (15) have also shown a correlation between the duration of ischaemia and this edema.

In ischaemia of less than 6 hours, the edema peaks later and disappears earlier, compared to limbs exposed to a longer period of ischaemia - there is a more rapid peak and a longer persistence. With infection, its resolution is further delayed (15).

Finally, the treatment of the fractures associated with axillary artery damage is controversial. This applies especially to proximal humeral fractures. It was previously thought that rigid fixation was necessary to protect the site of vascular repair (16). Presently, studies have shown that equally good results are achieved without such fixations (17).

REFERENCES

1. Jardon OM, Hood LT, et al . Complete avulsion of the axillary artery as a complication of shoulder dislocation. J Bone Joint Surg 1973; 55A(1) : 189 - 92.
2. Antal CS, Conforty B, et al . Injuries to axillary artery due to anterior dislocation of shoulder. J Trauma 1973; 13: 564 - 6.
3. Brown FW, Navigato WJ. Rupture of the axillary artery and brachial plexus palsy associated with anterior dislocation of the shoulder. Clin Orthop 1968; 60: 195 - 9.
4. Lev-El A, Adar R, Rubinstein Z. Axillary artery injury in erect dislocation of shoulder. J Trauma 1981; 21(4) : 323 - 5.
5. Theodorides Th, deKeizer G. Injuries of the axillary artery caused by fracture of the neck of the humerus. Injury 1976; 8: 120 - 3.
6. Tse DHW, Slabaugh PB, Carlson PA. Injury to the axillary artery by a closed fracture of the clavicle. J Bone Joint Surg 1980; 62A(A) : 1372 - 3.
7. Hayes JM, Van Winkle GN. Axillary artery injury with minimally displaced fracture of the neck of the humerus. J Trauma 1983; 23(5) : 431 - 3.
8. Smyth EHJ. Major arterial injury in closed fracture of the neck of the humerus. J Bone Joint Surg 1969; 51(B) : 508 -10.
9. Drapanas T, RL Hewitt RL, et al . Civilian vascular injuries. A critical appraisal of 3 decades of management. Ann Surg 1970; 172(3) : 351 -60.
10. D Sathyarup D, Huilgol AK, Iyer KM. Axillary artery thrombosis following a fracture of the neck of the humerus. Injury 1988; 19 : 45 - 7.
11. Eastcott HHG. The management of arterial injuries. J Bone Joint Surg 1965; 47B(3) : 394 - 8.
12. Henson GF. Vascular complications of shoulders. J Bone Joint Surg 1956; 38B(2) : 528 -31.
13. Sinkler WH, AD Spencer AD. The value of periphera arteriography in assessing acute vascular injuries. AMA Arch Surg 1960; 80 : 132 - 6.
14. McQuillan WM, Nolan B. Ischaemia complicating injury. J Bone Joint Surg 1968; 50B(3) : 482 - 92.
15. Miller HH, Welch CS. Quantitative studies of time factor in arterial injuries. Ann Surg 1949; 130(3) : 428 -38.
16. Linson MA. Axillary artery thrombosis after fracture of the humerus. J Bone Joint Surg 1980; 62(A) : 1214 - 5.
17. Lim EVA, Day L. Thrombosis of the axillary artery complicating proximal humeral fractures. J Bone Joint Surg 1987; 69(A): 778 - 80.
18. James EC, Khuri NT, Fedde CW, et al. Upper limb ischaemia from arterial thromboembolism. Am J Surg 1979; 137 : 739 - 44.
19. Gerlock AJ, Mathias J, Gonclarenko V, Maravilla A. Angiography of intimal and intramural arterial injuries. Radiology 1978; 129 : 357 - 61.