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Case Report

Crutch induced axillary artery injury

B McFall, N Arya, C Soong, B Lee, R Hannon

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Chronic use of axillary crutches is sometimes associated with axillo – brachial thrombo-embolic disease. Inappropriate placement of the patient's body weight on the axillary pad of the crutch causes repetitive trauma to the axillary artery leading to stenosis or aneurysm formation. We report a case of acute occlusion of the axillary artery caused by the use of the axillary crutch.

CASE REPORT A 53-year-old female presented with sudden onset of pallor and decreased sensation of the right hand and forearm, without loss of power. She had rheumatoid arthritis for the last 30 years, and had been using axillary crutches for the last 10 years. She had undergone bilateral knee and left hip replacements. There were no other significant cardiovascular risk factors, nor any previous history of deep venous thrombosis or miscarriages suggesting a hypercoagulable state. She was a non-smoker.

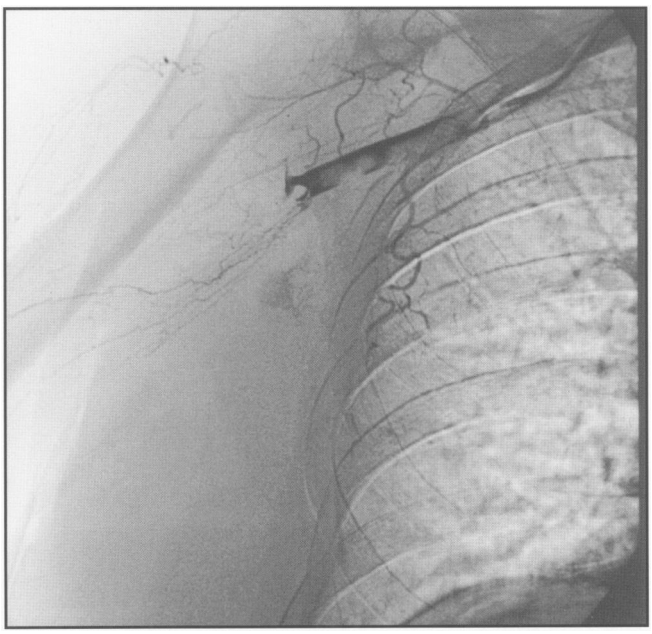

Fig 1. Arteriogram demonstrating complete occlusion of the right axillary artery.

On examination, she was in normal sinus rhythm. Both hands showed classical deformities of burnt out rheumatoid arthritis. The right hand was cold and pale, with delayed capillary return. Sensation was impaired over the fingers. Right brachial, ulnar and radial pulses were absent.

X ray of the thoracic inlet showed no evidence of bony cervical rib. ECG and echocardiogram were normal. An urgent angiogram showed an acute occlusion of the right axillary artery, with poor filling of the distal vessels (*figure*).

Because of severity and persistence of symptoms, surgical exploration of the brachial artery above the antecubital fossa was performed.

Fresh clot was retrieved from distal radial and ulnar artery with balloon thrombo -embolectomy. Further clots were obtained from the proximal axillary artery. Subsequently a good forward pulsatile flow was achieved. After closure of arteriotomy and restoration of blood flow, the hand became warm and pink, with a palpable radial and ulnar artery at the wrist. Follow up duplex scan showed no evidence of residual stenosis or thrombus in the axillary artery. No obvious aneurysm formation was demonstrated.

She was subsequently assessed and trained in the use of a rollator with wrist braces, as a walking aid.

Department of Vascular Surgery, Belfast City Hospital, 91 Lisburn Road, Belfast BT9 7AB.

B McFall, BSc, MB, BCh, Senior House Officer.

N Arya, MS, FRCS, Specialist Registrar.

C Soong, MD, FRCS, Consultant Surgeon.

B Lee, FRCS, Consultant Surgeon.

R Hannon, MD, FRCS, Consultant Surgeon.

Correspondence to Mr Soong.

DISCUSSION

Radial nerve and axillary artery are the commonest structures injured with the long term use of axillary crutch. It usually presents in the form of traumatic radial nerve palsy or a thromboembolic episode in the upper limb.[1]

Radial nerve palsy affects motor supply to the extensor muscles of the arm and forearm. Damage to it causes the forearm to adopt a characteristic position with a flexed, limp wrist. In this state, grip strength is lost since for maximum power in the digital long flexors, the wrist has to be held in extension. If the lesion occurs above the upper third of the arm, the triceps muscle is also affected and elbow extension will be absent. Sensation is tested over the area of the anatomical snuffbox. Sensation here is lost with a radial nerve lesion at any level.

Repetitive trauma due to the use of axillary crutch causes disruption and degeneration of the tunica intima and tunica media leading to the formation of an aneurysm or stenosis. Pathological examination of the axillary artery reveals fragmentation of the intima and elastica associated with a perivascular fibrous reaction.

Crutch induced aneurysms of the axillary artery was first reported as early as 1930.[2] The commonest presentation is in form of sudden ischaemia of the upper limb, with no reported case of rupture.[3,4,5] The patients are usually elderly, suffering form chronic arthritis or weakness of the lower extremities, and have used axillary crutches to mobilize for many years. Muscle wasting around the pectoralis, latissimus dorsi and shoulder joint are often seen in rheumatoid patients, and one could postulate that this muscle wasting and/or loss of body fat may make such an injury more likely.

Intimal disruption leads to thrombogenesis and repetitive trauma dislodges showers of small emboli, which gradually occlude distal vessels and may compromise the results of revascularisation during a later ischaemic episode.[1] For this reason surgical treatment is suggested on discovery of an aneurysm even if asymptomatic. Surgical alternatives include thrombectomy, axillo-brachial bypass or primary repair of the axillary artery. For non-aneurysmal disease, percutaneous thrombolysis and angioplasty may be safer, less invasive and equally efficacious.

Thrombolytic agents have been successfully used to dissolve the occluding thrombus, reconstitute blood flow, and improve the status of the tissue bed supplied by the involved vascular segment. Thrombolytic agents in clinical use are actually plasminogen activators and plasmin is the active molecule that cleaves fibrin polymer to cause dissolution of thrombus. Peripheral thrombolytic therapy is administered through a catheter-directed approach to achieve regional thrombolysis with minimal systemic fibrinolysis. However a moderate systemic proteolytic state often results, culminating in haemorrhagic complications and hence limiting their use to patients with no contraindications (*Table*). There is no overall difference in limb salvage or death between surgery and thrombolysis in the initial management of acute limb ischaemia.[6] Thrombolysis may be associated with a higher risk of continued limb ischaemia, and of haemorrhagic complications including stroke and this must be balanced against the risks of surgery in each patient.

TABLE

Contraindications to use of thrombolytic agents

Absolute Contraindications
1. Active bleeding disorder
2. Gastrointestinal bleeding within 10 days
3. Cerebrovascular event within 6 months
4. Intracranial or spinal surgery within 3 months
5. Head injury within 3 months
Relative Contraindications
1. Major surgery or trauma within 10 days
2. Hypertension (systolic >180 mm Hg or diastolic >110 mm Hg)
3. Cardiopulmonary resuscitation within 10 days
4. Puncture of noncompressible vessel
5. Intracranial tumour
6. Pregnancy
7. Diabetic haemorrhagic retinopathy
8. Recent eye surgery
9. Hepatic failure
10. Bacterial endocarditis

However, in this case no aneurysm formation was found. The lack of a local stenotic lesion raised the possibility of embolus arising from a more central source. However the absence of any previous symptoms or signs of cardiac disease, and a normal echocardiogram suggest that this was unlikely.

Therefore the presentation of acute upper limb ischaemia in this case was most probably due to axillo-brachial thrombosis secondary to chronic axillary crutch use, even though no focal stenotic lesion could be identified.

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