States 50% of such patients had prior muscle trauma.² So far the evidence for the role of immunosuppression in pyomyositis is unproven. It has been recognized in patients with diabetes, acquired immunodeficiency syndrome and haematological malignancies.⁴ *S. pneumoniae*, present here, is rare as it causes fewer than 1% of cases of pyomyositis, *S. aureus* being causative in 90%.⁵ Muscle involvement is multiple in 40–60% of cases.² The muscles of the upper leg are most frequently involved, those of the upper arm are less commonly affected.⁵

Pyomyositis is usually accompanied by pyrexia, malaise, leucocytosis, a raised ESR and blood cultures are usually sterile. The involved muscle becomes tender and the overlying skin erythematous. Fluctuation is only present at a late stage and if untreated progresses to systemic sepsis.⁴ The diagnosis is difficult because the typical features of an abscess are absent. Diagnosis, however, may be made by ultrasound scan, computed tomography scan, magnetic resonance imaging, direct aspiration or drainage. Treatment is with surgical drainage and antibiotics. Functional recovery is usually good.⁵

This case illustrates the problem of diagnosis as

the clinical signs may mimic septic arthritis. Although still rare in temperate climates, pyomyositis has to be considered in such atypical presentations, because it is vital to institute treatment early in the course of the disease to prevent progression to septic shock.

At follow-up our patient received the 23-valent pneumococcal vaccine and was advised to seek an early medical opinion if he developed any symptoms of infection. The evidence for antibacterial prophylaxis in asplenic patients is controversial and our patient did not receive such prophylaxis. In the light of this case, it is reasonable to suggest that such patients should seek early medical help when they recognize any symptoms of an infectious illness. Re-immunization with pneumococcal vaccine is not widely recommended in view of the potential reactions, however, due to the serious nature of sepsis in this case re-immunization was considered an appropriate measure.

Acknowledgements

We are grateful to Mr C. Pool and Dr P. Gillette for their help in this case.

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Cervical ribs: a cause of distal and cerebral embolism

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Summary: The thoracic outlet syndrome occurs when the neurovascular structures are compressed as they traverse the thoracic outlet. Degenerative changes can occur in the subclavian artery and the vessel may become a source of embolism with the risk of acute or chronic upper limb ischaemia. Rarely, distal thromboembolism in the thoracic outlet syndrome may be associated with retrograde flow when there is the added risk of cerebral thromboembolism.

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Accepted: 28 May 1992

Introduction

Vascular sequelae of the thoracic outlet syndrome arise as a result of the intermittent but long-term compression of the subclavian artery by cervical rib, cartilage or fibrous band.

Long-term trauma to the subclavian artery leads to degenerative changes within the vessel wall, localized atheroma and aneurysm formation. Distal thromboembolism causes acute and chronic upper limb ischaemia. Retrograde flow can occur in the thoracic outlet syndrome and lead to cerebral embolism.

We report a case of thoracic outlet syndrome secondary to cervical rib in which both distal and retrograde thromboembolism occurred.

Case report

A previously fit 41 year old storekeeper developed a painful right forearm and a cold hand. He did not seek help as the acute symptoms settled over the next few days. However, he was left with forearm claudication which led to his premature retirement from competitive squash. Two years later he developed a left hemiparesis which resolved after 4 days. A diagnosis of cerebral embolism in the distribution of the right middle cerebral artery was made. He was advised to stop smoking and commenced on low-dose aspirin.

Six months later he was referred for investigation of the right forearm claudication which had become worse. On examination, both hands were equally warm but on the right, pulses were absent distal to the brachial artery. There were no signs of cerebrovascular disease. Biochemical and haematological indices were normal. Since a plain chest X-ray demonstrated a large right cervical rib, a provisional diagnosis of thoracic outlet syndrome was reached (Figure 1). Doppler sonography demonstrated the brachial artery pressure to be reduced from 108 mmHg on the left to 93 mmHg on the right but was not reduced by shoulder abduction. Electrocardiogram and echocardiogram were carried out and were both normal.

By the time he was admitted for angiography 3 months later, the right brachial artery was no longer palpable in the cubital fossa. At digital subtraction angiography, the subclavian artery was narrowed at the thoracic outlet by the cervical rib and there was a post-stenotic saccular aneurysm (Figure 2a). Blood flow was reduced on shoulder abduction (Figure 2b). The carotid and vertebral arteries were free from atheromatous disease.

The patient underwent surgical exploration of the right thoracic outlet by a supraclavicular approach with excision of the middle third of the clavicle. The cervical rib and a 3 cm length of

Figure 1 Plain radiograph demonstrating a large right cervical rib.

subclavian artery, including the saccular aneurysm which contained thrombus, were resected. The artery was replaced with a 5 cm × 6 mm woven Dacron graft, anastomosed end to end with the host vessel. The axillary and brachial arteries were cleared of thrombus distally using a Fogarty embolectomy catheter and the brachial pulse was restored. Radial and ulnar pulses were present at one week. He was discharged on oral anti-coagulants to reduce early graft thrombogenicity.

Histology confirmed degeneration and fibrosis of the wall of the artery, maximal at the site of aneurysmal formation.

Discussion

The thoracic outlet syndrome was first recognized by a surgeon from this institution, Sir Astley Cooper in 1821. The neurological and vascular symptoms and signs of the syndrome result from the compression of the brachial plexus, subclavian artery or vein as they traverse the thoracic outlet, a space defined by the first rib inferiorly, scalenus

Figure 2a Digital subtraction angiography demonstrating subclavian artery compression by the cervical rib with a post-stenotic sacular aneurysm.

Figure 2b Digital subtraction angiography demonstrating reduced flow on arm abduction.

medius posteriorly and the scalenus anterior anteriorly. The neurovascular bundle may be impinged upon by cervical ribs or related structures (cervical transverse process, cartilage or band); anomalous insertions of muscles (scalenus anticus syndrome; hyperabduction syndrome) or local bony injury (costoclavicular syndrome).^{1–2} Cervical ribs are more commonly associated with unilateral upper limb arterial insufficiency than with neurological or venous symptoms. Those of ischaemia develop during exercise whilst the neurological manifestations of the thoracic outlet syndrome appear at the start or after exercise.^{1–4}

Patients may present with an acutely ischaemic hand or with disabling forearm claudication.^{2,5} Although arterial stenosis due to progressive fibrosis at the site of arterial compression has been suggested as the aetiology, both presentations have the same aetiology: thromboembolism originating from the damaged vessel wall or aneurysm.^{6,7}

Two years after the onset of forearm claudication, this patient developed a transient hemiparesis affecting the right side. Cerebral embolus is a rare but recognized manifestation of the thoracic outlet

syndrome and has been associated with occlusion of the subclavian artery with retrograde flow.^{8,9} In this patient, emboli from the distal arterial lesion may have refluxed into the cerebral circulation during diastole to cause the stroke.⁹

In the management of cerebral and distal thromboembolism caused by cervical ribs, the aim is to identify the arterial pathology and the extent of arterial occlusion. This can be achieved by angiography or duplex Doppler ultrasound.^{1,10} The aim must be to prevent further embolism and restore function by surgical decompression of the thoracic outlet and resection of the embolic source.^{10–12}

When a cervical rib is the cause, this must be excised.¹³ Access to neurovascular structures can be further improved by resecting the clavicle or the first rib. Removing the latter may also prevent the development of neurological symptoms.¹⁰ There are several different surgical approaches to the first rib.^{14,15} The largest series has suggested that a combined infra- and supra-clavicular approach with excision of the first rib may best facilitate arterial reconstruction and outlet decompression.

Whatever the vascular pathology – stenosis or

aneurysm – the abnormal segment of subclavian artery should also be excised. Primary anastomosis may be possible when only a short length of vessel has been removed. However, this was not possible in our case and vascular reconstruction with a prosthetic graft was necessary.

This case demonstrates that a cervical rib may cause peripheral and cerebral thromboembolism in the thoracic outlet syndrome. Urgent angiography should be used to identify the site and extent of arterial pathology. Patients need surgical decompression of the thoracic outlet with excision of the segment of abnormal artery.

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Idiopathic calcification of the basal ganglia

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Summary: Idiopathic calcification of the basal ganglia is a rare disorder characterized by neuropsychiatric abnormalities, a movement disturbance of parkinsonian and/or choreoathetoid type and dense calcification of the basal ganglia. We report a 60 year old patient diagnosed as having delusional disorder and tardive dyskinesia who was subsequently found to be suffering from idiopathic calcification of the basal ganglia.

Introduction

Idiopathic calcification of the basal ganglia (ICBG) is a rare disorder characterized by neuropsychiatric

abnormalities, a movement disturbance of parkinsonian and/or choreoathetoid type and dense calcification of the basal ganglia.¹ Its aetiology is unknown but it is familial in some cases.^{2,3} Other causes associated with calcification of the basal

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Accepted: 1 June 1992