patient it allowed control of variceal bleeding refractory to sclerotherapy, tamponade and pharmacotherapy without recourse to surgery.

Beta-blockers prevent variceal haemorrhage and reduce mortality when used in secondary prophylaxis.\textsuperscript{16} Partial decompression of the portal hypertension had been achieved by mechanical means but a significant portosystemic gradient remained. Propranolol was continued in an attempt to further reduce his risk of recurrent variceal haemorrhage. Although there are insufficient data reported on combining pharmacotherapy and TIPSS, it has been shown that combinations of different pharmacological therapies have useful additive portal hypotensive effects.\textsuperscript{1} Long-term follow-up is unavailable and the exact place of TIPSS in the management of lower risk patients and its relationship to non-operative and operative modalities needs to be defined. It should be considered as a potentially life-saving procedure in critically ill patients with variceal bleeding.

References


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Myositis due to cholesterol emboli

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Summary: Myositis due to spontaneous cholesterol embolization is uncommon and usually associated with cutaneous abnormalities at presentation. A case of myositis due to cholesterol emboli is reported. The patient presented with painful weak legs, and the diagnosis was confirmed by muscle biopsy.

Introduction

The patient presenting with leg weakness may pose diagnostic difficulties. The initial differential diag-

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nosis is wide and would include neurological disorders, primary muscle diseases, metabolic causes, systemic diseases such as anaemia and vascular causes. Cholesterol emboli to the legs presenting as muscle pain and weakness is a rare presentation of peripheral vascular disease.\(^1,2\) Associated cutaneous features, including livedo reticularis and 'purple toe', may make the diagnosis apparent. We report a case presenting with the sudden onset of myositis alone.

**Case report**

A 63 year old Caucasian male was admitted to Peterborough District Hospital with a history of sudden onset of weakness and pain in both thighs and calves. The symptoms had originally occurred whilst lifting a heavy weight and had gradually improved over the next few hours. He was a smoker of 20 cigarettes per day.

Initial examination revealed him to be afebrile with bilateral calf tenderness, but no objective muscle weakness or neurological deficit. There were no cutaneous abnormalities; however, the left great toe was noted to be cool. Blood pressure was 150/90 mmHg, all peripheral pulses were normal and there were no audible bruits. General examination including fundoscopy was otherwise unremarkable.

Laboratory investigations including full blood count, blood urea and electrolytes, creatinine, glucose, calcium and lipids were normal. The ESR was 21 mm/hour and an autoimmune profile was negative. The creatine kinase was elevated, however, with a peak rise of 1,362 U/I (normal range <150). A presumptive diagnosis of myositis was made and a muscle biopsy of the left medial gastrocnemius performed to confirm this.

The muscle biopsy showed patchy myocyte atrophy and necrosis with a surrounding infiltrate of histiocytes and occasional lymphocytes. An adjacent small artery was occluded by a cholesterol embolus (Figures 1 and 2). An abdominal ultrasound, performed to exclude an aortic aneurysm as the source of emboli, was normal. However, an aortogram demonstrated an ulcerating plaque in the distal abdominal aorta (Figure 3).

![Figure 1](https://example.com/image1.png)  **Figure 1** Muscle biopsy showing myocyte atrophy and necrosis. Boxed area shows a vessel with cholesterol emboli. × 16.

![Figure 2](https://example.com/image2.png)  **Figure 2** Higher magnification of a small artery occluded by cholesterol emboli. × 40.

![Figure 3](https://example.com/image3.png)  **Figure 3** Aortogram showing an ulcerating plaque above the bifurcation.
Over the next 2 weeks the calf pain persisted and the left great toe became progressively dusky. He subsequently underwent an aorto-bifemoral graft. Immediately postoperatively, pedal pulses were undetectable. Over a period of 48 hours his feet became clinically ischaemic and he subsequently underwent bilateral embolectomies from both femoral and popliteal approaches. Although flow was apparently restored with restoration of dorsalis pedis pulses, the condition of the feet deteriorated with progressive dry gangrene of the toes requiring bilateral below-knee amputation.

The low limbs were examined histopathologically. There was widespread necrosis of muscle in both calves with numerous cholesterol emboli occluding small vessels. The left anterior tibial and right posterior tibial artery were occluded by thrombus and atheromatous debris.

**Discussion**

Cholesterol emboli arise from ulcerating arterial plaques in major vessels; the released crystals subsequently occlude distal arterioles and capillaries of the microcirculation.\(^1\) Cholesterol embolization is a well-recognized complication of arteriography, presenting in a variety of fashions as diverse as systemic hypertension, renal failure, acute pancreatitis, peptic ulceration and organic brain disease.\(^3,4\) Cholesterol embolization is also well described following arterial surgery.\(^5\)

Autopsy studies suggest that spontaneous cholesterol embolization is common.\(^6-8\) However, since spontaneous emboli may mimic the presentation of more common disease processes, the diagnosis may be frequently missed.\(^9\) The clinical picture of muscle pain and weakness associated with an elevated creatine kinase suggests a myositis but, in the absence of cutaneous or peripheral emboli, the possibility of underlying embolic disease may be overlooked. The diagnosis was suspected in this case due to the rapidity of onset and the atypical distribution of muscle involvement. Involvement of calf and thigh muscles with upper limb sparing is characteristic. The progressive ischaemia of the great toe in the presence of normal dorsalis pedis and posterior tibial suggested possible occlusion of the microcirculation. The diagnosis may be confirmed by muscle biopsy but not excluded due to the problems of sampling error.

Medical treatment is generally ineffective. The only proven mode of therapy at present is removal of the source of emboli.\(^9\) In this case the progressive ischaemia required the insertion of an aortic inlay graft.

The initial presentation of this case implies that the atheromatous plaques are friable and prone to give rise to emboli. Although small vessel embolization is a recognized complication of aortic surgery, the extent of embolization in this case is unusual and may reflect the increased friability of the plaques. This group of patients presenting initially with emboli may be at increased risk of embolic complications postoperatively.

**References**

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doi: 10.1136/pgmj.69.818.947

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