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Case Report

Diabetic muscle infarction: an unusual cause of leg swelling in a diabetic on continuous ambulatory peritoneal dialysis

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Introduction

Diabetic muscle infarction is a rare complication of diabetes first described by Angervall and Stener in 1965 [1]. It typically arises in patients with advanced diabetic complications. Only 25 cases have been reported previously [2]. The condition has not been reported previously in the nephrological literature.

Case

A 49-year-old type 1 diabetic male commenced continuous ambulatory peritoneal dialysis in December 1995. His diabetes was complicated by both retinopathy and neuropathy in addition to renal disease, but he had no clinical evidence of large-vessel disease.

After 6 months' uneventful dialysis he presented with a tender, erythematous swollen left thigh. He was non-toxic, afebrile, with a normal white-cell count. There was no muscle wasting and no evidence of diabetic amyotrophy. Ultrasound of the ileo-femoral veins was normal, excluding venous thrombosis as a cause. The thigh was explored surgically and a tumour was thought to be present, however, a biopsy taken from the anterior compartment of the left thigh was consistent with chronically inflamed scar tissue with no evidence of malignancy. His glycated haemoglobin was 7.9% and an autoimmune screen was negative. The swelling resolved spontaneously following a period of bedrest and analgesia.

In August 1996 he developed pseudomonas peritonitis necessitating removal of his Tenckhoff catheter and a period of haemodialysis. In September 1996 he presented once more with a swollen tender right thigh (Figure 1). His C-reactive protein was 44 mg/l initially rising subsequently to 214 mg/l. A biopsy taken from the right thigh (Figure 2) showed extensive necrosis of skeletal muscle, with numerous neutrophil polymorphs (left) and florid fibrovascular proliferation in adjacent connective tissue (right). Most of the blood vessels

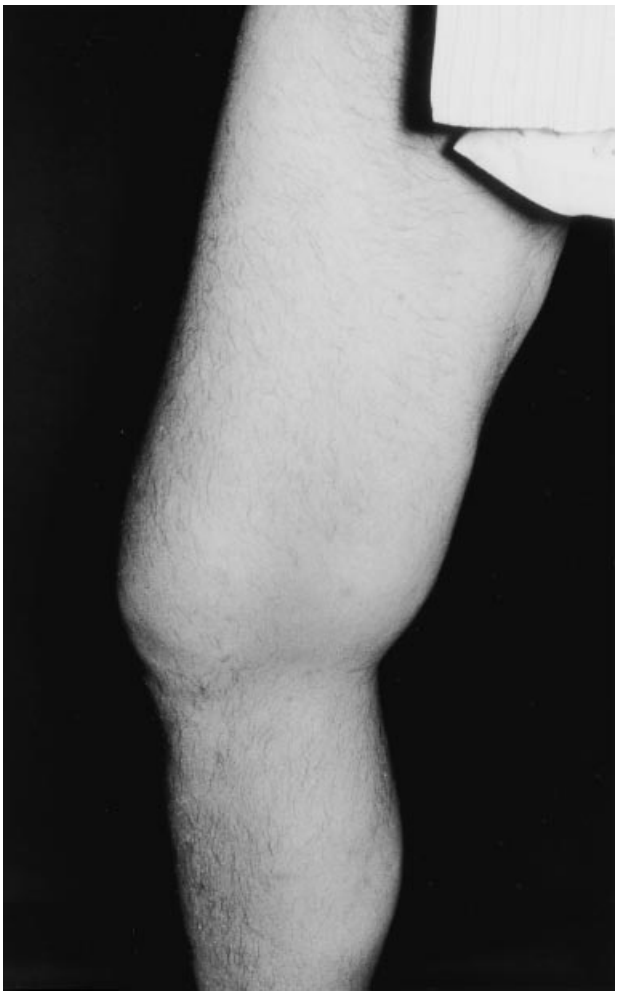

Fig. 1. Photograph showing swollen right thigh.

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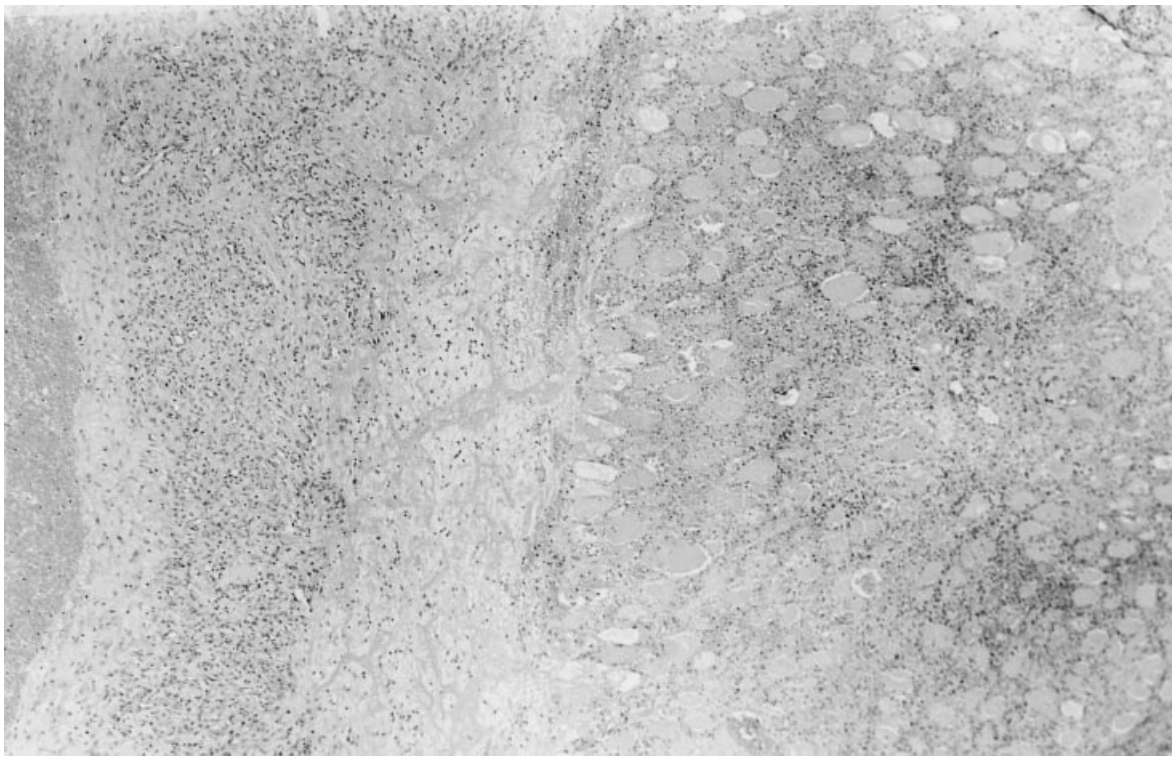

Fig. 2. Photomicrograph showing a large area of muscle necrosis associated with polymorph neutrophil infiltrate (left) and fibrovascular proliferation of adjacent connective tissue (right). Haematoxylin and eosin. Magnification × 100.

appeared normal, however, fibrin thrombi were noted in occasional small vessels.

An ultrasound examination of the right leg revealed an ill-defined inflammatory mass superficial to the distal right superficial femoral vein and artery. Magnetic resonance imaging (MRI) showed diffuse increased signal intensity and hyperaemia involving the vastus medialis muscle in T_1- and T_2-weighted images with no contrast enhancement (Figure 3).

Empirical courses of antibiotics were tried, augmentin initially then clindamycin and ciprofloxacin and finally vancomycin, rifampicin and ciprofloxacin; none of which appeared to be of benefit. The swelling resolved spontaneously once more after rest and analgesia.

He had two further similar episodes of acute painful muscle swelling, one in December 1996 involving the posterior compartment of the left thigh and one in January 1997 involving the right calf. He then received a course of prednisolone (initial dose 40 mg daily) but this was followed by a further episode involving the left calf in February 1997. There have been no further episodes since then.

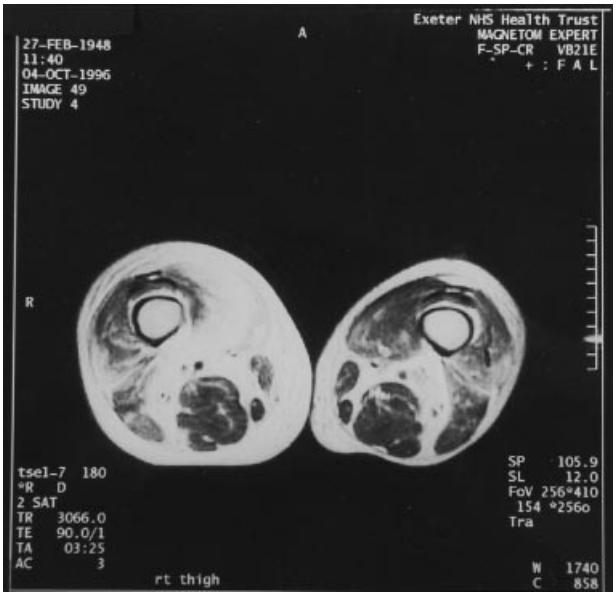

Fig. 3. MRI showing diffuse increased signal intensity and hyperaemia of the vastus medialis muscle in T_1- and T_2-weighted images.

Discussion

Diabetic muscle infarction usually presents with acute onset of thigh pain with an associated palpable tender mass and swelling of surrounding tissues [2]. The vastus lateralis, thigh adductors and biceps femoris are most commonly affected but there may be calf muscle involvement too. There is no evidence of infection, cellulitis, gangrene or deep-vein thrombosis. Muscle power is maintained without muscle atrophy and there are no sensory signs. The lesion may last several weeks before spontaneously resolving. Recurrent episodes in

the same or contralateral limb occur in 50% of patients [2].

The patients are usually long-standing insulin-dependent diabetics with a mean age at presentation of 39 ± 12 years. Diabetic control has previously been poor and most have other end-organ complications of retinopathy, nephropathy and neuropathy [2]. The creatinine kinase is frequently normal but this may be related to the timing of the test in relation to the time of muscle infarction [3]. The histological features are not specific for diabetic muscle infarction. In acute cases the muscle is necrotic with the nerves and blood vessels infiltrated by polymorphs. Chronically there is focal replacement of muscle by collagen and some regeneration of muscle fibres. The walls of small blood vessels become thickened and hyalinized with narrowed lumens [2,4].

Radiologically, MRI is the best technique to delineate the affected muscle group. T_2-weighted and short tau inversion recovery (STIR) images show the oedematous changes of affected muscle. However, the appearances are not specific for diabetic muscle infarction. MRI also gives information on the adjacent perifascial spaces and subcutaneous tissues and may be used to exclude vascular thrombosis [5,6]. Ultrasound and computed tomography may localize the region of soft-tissue involvement and enable assessment for cystic and bony changes [7].

Management of the condition is largely supportive with analgesia, rest and aggressive diabetic control. In worsening cases prednisolone or intravenous gamma globulin may be considered [8]. Anticoagulation has been suggested based on the hypothesis that a baseline hypercoagulable state may exist in diabetics because of abnormalities in coagulation and fibrinolysis and that this may contribute together with atherosclerosis and microvascular disease to the muscle infarction [9].

Conclusion

Spontaneous muscle infarction is a rare complication of diabetes which should be considered in the differential diagnosis of a painful swollen leg in such patients.

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