LESSON OF THE WEEK

Diabetic muscle infarction

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Timely diagnosis of diabetic muscle infarction helps prevent complications in a patient presenting with painful swollen extremity

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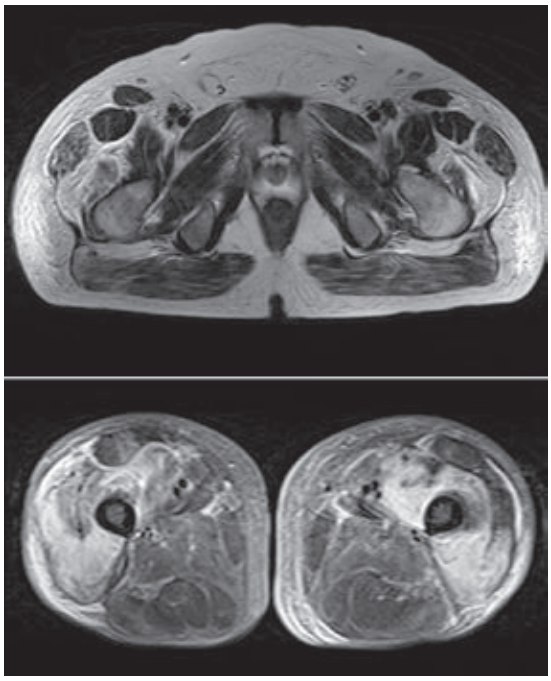
Diabetic muscle infarction is a rare complication of diabetes that occurs in patients with type 1 diabetes (70% of total cases) or in patients with poorly controlled type 2 diabetes.[1] It presents with sudden onset of a painful swelling, often of the thigh, which is bilateral in up to a third of patients, and it occurs spontaneously without a history of trauma or features of infection.[2] Diabetic muscle infarction is under-recognised and often misdiagnosed,[3 4] and treated as rhabdomyolysis or polymyositis. A high index of suspicion is needed to make a timely diagnosis and to avoid the use of steroids or surgical intervention. This report highlights the clinical investigations, laboratory tests, and imaging scans needed to establish the clinical diagnosis in a timely fashion to avoid unnecessary and possibly harmful interventions.

Case

A 38 year old man with a 10 year history of type 2 diabetes presented with severe pain and swelling of the thighs of two weeks' duration. He described a burning pain mainly at the front of both thighs that started spontaneously and became bad enough to limit his mobility. He denied any injury or heavy exertion before the onset of symptoms. Nor had he been travelling or had a recent viral or febrile illness. He had used anti-inflammatory agents with some relief. He had no pain in other muscle groups or joints or symptoms of fever, chills, or night sweats. He had no history of skin rash, but he had a history of hypertension, stage 3 diabetic kidney disease, and diabetic retinopathy with previous photocoagulation to the left eye. He had not recently taken statins.



Two representative slices of T_2 weighted magnetic resonance imaging scan: bilateral high T_2 signal corresponding to fluid or oedema affecting subcutaneous fat, quadriceps, and tensor fascia latae bilaterally

The patient did not smoke and denied using alcohol. His medications included insulin NPH 0-0-0-40 and insulin 30/70 45-0-0-0, perindopril 8 mg a day, amlodipine 10 mg a day, hydrocholorothiazide 25 mg a day, and aspirin 325 mg a day. He was on diclofenac 50 mg twice a day. He worked as a computer technician.

The patient was of heavy build, and his blood pressure was 146/70 mm Hg with regular rhythm. He had a temperature of 36.8°C. Heart sounds were normal. His lungs were clear, and the abdomen was benign. He had no ankle oedema. On examination he had diffuse induration and tenderness on the anterolateral aspect of both thighs—left more than right, with limitation of movements at knee joints (because of pain) but no effusion or erythema. There was no evidence of cellulitis or lymphangitis, but both thighs were warmer than normal. He had no hyperaesthesia, lymphadenopathy, or tenderness at either of the anterior superior iliac spines. Peripheral pulses were symmetrical. There were no muscle fasciculations, muscle atrophy, evidence of neurovascular compromise of the lower extremities, or swelling or tenderness of the spine. Neurological examination was non-focal except for limited movement at the knees.

Laboratory data showed a slightly raised white blood cell count of 11.9×10^9/l (normal range 4.0-11.0), with a normal eosinophil count, slightly low haemoglobin of 118 g/l (120-160), normal platelets of 371×10^9/l (normal range 150-400) and raised erythrocyte sedimentation rate of 66 mm/h (normal range 10-20). Blood urea nitrogen was raised at 19.5 mmol/l (1.7-8.3), as was serum creatinine at 265 µmol/l (44-106), although electrolytes were normal, with a serum potassium of 4.0 mmol/l. Glycated haemoglobin was 0.076 (0.060-0.070). Serum calcium was normal at 2.15 mmol/l (2.02-2.60), serum uric acid was raised at 544 µmol/l (202-416), as was serum phosphate at 1.72 mmol/l (0.87-1.45) and thyroid stimulating hormone at 12.89 mIU/l (0.49-4.67), whereas serum albumin was low at 32 g/l (35-50). Creatine kinase was 442 U/l (0-190 U/l) and antinuclear antibody was negative. Urinalysis showed ≥3.0 g/l of protein (normal–negative).

Cite this as: *BMJ* 2009;338:b2271
doi: 10.1136/bmj.b2271

A magnetic resonance imaging scan of the pelvis and thighs showed high T_2 signal bilaterally, corresponding to fluid or oedema affecting the subcutaneous fat that overlies the distribution of tensor fascia latae (figure). In addition, a high T_2 signal, which indicates oedema was noted within the quadriceps and tensor fascia latae bilaterally. The bones were not affected.

Bilateral involvement of the quadriceps with mostly pain rather than weakness, a small increase in creatine kinase, negative collagen work-up, and a longstanding history of diabetes with associated diabetic microvascular complications—retinopathy and nephropathy—is highly suggestive of diabetic muscle infarction. The findings on magnetic resonance imaging supported this clinical diagnosis. The patient was treated conservatively with bed rest and analgesics, and his symptoms improved over the next three weeks.

Discussion

Painful swelling in an extremity can occur for various reasons (box). However, proximal muscle pain and swelling with a mild increase in creatine kinase suggest the possibility of an inflammatory process such as polymyositis, panniculitis, pyomyositis, or, rarely, diabetic muscle infarction. In polymyositis, the prominent feature is weakness of the proximal muscles and mild pain, rather than intense pain. In this condition, electromyography would show a myopathic process in the proximal muscles, and muscle biopsy is important for establishing the diagnosis. Adenopathy and systemic signs of infection are characteristic of an infectious process such as pyomyositis, and aspiration may establish the diagnosis and guide appropriate treatment. Diabetic muscle infarction typically presents spontaneously as a localised, exquisitely painful and tender swelling of acute onset, associated with limited motility of the affected extremity, but with no systemic signs of infection. It often affects the thighs, with the quadriceps being involved in 83% of cases, and it rarely affects the calves.[1] Bilateral involvement occurs in up to 30% of patients.[5]

Diabetic muscle infarction is often associated with microvascular complications (nephropathy, retinopathy, or neuropathy) of diabetes.[6] Its pathogenesis is unclear, but it is probably secondary to a diffuse microangiopathic process and related to hypoxia-reperfusion injury.[1 6]

Differential diagnosis of painful swelling of an extremity in a patient with diabetes

Infection
Cellulitis
Soft tissue abscess
Necrotising fasciitis
Osteomyelitis with soft tissue involvement
Parasitic infestation

Inflammatory
Polymyositis or dermatomyositis
Myositis, inclusion body

Neurological
Diabetic lumbosacral radiculoplexopathy (amyotrophy)
Bruns-Garland syndrome

Rhabdomyolysis
Traumatic
Non-traumatic
Drug induced (statins)

Neoplastic
Benign: lipoma, fibroma, or leiomyoma
Malignant: liposarcoma

Vascular
Deep venous thrombosis
Haemorrhage (haematoma)
Thrombophlebitis
Arterial occlusion
Post traumatic pseudoaneurysm

Miscellaneous
Diabetic muscle infarction
Calcific uraemic arteriolopathy (calciphylaxis)

The diagnosis can be made with reasonable certainty in a patient presenting with these symptoms who has a longstanding history of diabetes—often poorly controlled with microvascular complications—and characteristic findings on magnetic resonance imaging.[5 6] Magnetic resonance imaging of the extremity is the modality of choice; on T_2 weighted sequences it shows characteristic features of extensive oedema within the muscle(s), muscle enlargement, subcutaneous and interfascial oedema, and multifocal involvement in a patchwork pattern.[5-7] Gadolinium enhancement is not needed but may show an enhanced margin of the infarcted muscle, with a central non-enhanced area of necrotic tissue.[5 6] However, gadolinium should be used with caution in patients with decreased kidney function because of the associated risk of gadolinium induced nephrogenic systemic fibrosis.[8] A muscle biopsy is often not needed because it may prolong recovery and is indicated only when the presentation is atypical, response is poor, or diagnosis is uncertain.[2] The biopsy when performed shows pale muscle on gross examination and areas of muscle necrosis and oedema surrounded by muscle fibres in various stages of degeneration and regeneration, with hyalinosis and thickening of arterioles.[9]

No evidence based recommendations are available about the management of this condition. However, a retrospective analysis supports conservative management with bed rest, leg elevation, and adequate analgesia.[10] Increased pain and swelling after stretching or exercise can occur, so these activities should be avoided during the acute phase. Tight diabetic control is important because poor control may prolong the episode. There is no evidence to support the use of steroids or surgery. Surgery may in fact worsen the outcome.[10] The short term prognosis is good, but the recurrence rate is high (40%), and recurrences may not necessarily affect the same muscle group.[10] Diabetic muscle infarction portends a poor prognosis with 10% mortality over two years.[10]

Contributors: MSP is sole author and guarantor.

Funding: None.

Competing interests: None declared.

Provenance and peer review: Not commissioned; externally peer reviewed.

Patient consent obtained.

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Accepted: 15 September 2008