

**Recurrent Diabetic Muscle Infarction Presenting With Painful Thigh Swellings Simulating  
Recurrent Deep Venous Thrombosis**  
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**ABSTRACT**

Atraumatic painful swollen lower extremity has limited differential diagnosis and in patients with risk factors for venous thromboembolism offers little challenge. Poor awareness of rare entities results in misdiagnosis and mismanagement. Diabetic muscle infarction is a rare complication of longstanding uncontrolled diabetes mellitus causing ischemic skeletal myonecrosis and similar presentation.

**Keywords:** Diabetic Muscle Infarction, deep venous thrombosis, swelling.

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## **CASE REPORT**

A 49 year-old Jordanian man with a history of hypertension and type II diabetes mellitus diagnosed 10 years ago (after a significant period of delay in seeking medical care due to insurance issues) presented with pain and swelling of the left thigh that started 4 weeks before presentation. The pain progressively affected his activity and suddenly became so severe that he could not bear his own weight and was worse with ambulation. He had no pain in other muscle groups or joints. He denied heavy exertion, injury, trauma or falls before the onset of symptoms. He had no recent viral or other febrile illnesses. The patient works as a taxi driver; he is a heavy smoker for the last 30 years (30 pack-years). He denied alcohol intake or illicit drug use, his medication at time of presentation included mixtard insulin, enalapril, amlodipine, furosemide, aspirin and multivitamins.

One year before the onset of his current left thigh presentation he underwent excision for a painful right thigh mass but he lost follow up and histopathological diagnosis was not available at time of admission.

The patient was well built with body mass index of 28.7 kg/m<sup>2</sup>. His blood pressure was 140/85 mmHg with regular rhythm. He had a temperature of 37.1°C. Heart sounds were normal. His lungs were clear to auscultation, and the abdominal examination was normal. Examination of extremities revealed diffuse induration and tenderness on the anterolateral aspect of his thigh, with limitation of movement because of pain. There was no evidence of cellulites, or lymphangitis, but the left thigh was warmer than the right one. He had no hyperaesthesia, lymphadenopathy, or tenderness at the inguinal areas. Peripheral pulses were symmetrical with no bruit. He had decreased sensation or light touch and pin prick in a glove and stocking distribution. Ophthalmologic examination revealed normal intraocular pressure, severe non

proliferative diabetic retinopathy in left eye and proliferative diabetic retinopathy as well as inferior vitreous haemorrhage in the right eye.

Laboratory data showed raised white blood cell count  $23.2 \times 10^9/L$  (normal range  $4.0-11.0 \times 10^9/L$ ), with 90% neutrophils (normal 33-75%), normal eosinophil count, normochromic normocytic anemia with haemoglobin 9.5 gm/dl (normal 12-16), raised platelet count  $740 \times 10^9$  (normal range 150-400), raised erythrocyte sedimentation rate (westgren) (ESR) 145 mm/hr (normal 0-25), and raised C reactive protein (CRP) 384 mg/ L ( normal < 6).

Blood urea nitrogen was raised to 15.1 mmol/L (normal= 2.0- 8.3), as was serum creatinine at 345  $\mu\text{mol/l}$  (normal 44-106). Initially he was hyponatremic (sodium level 124 mmol/L; normal = 135- 148) but other serum electrolytes (including potassium, magnesium and calcium) as well as uric acid were all within the normal limits. Urine analysis revealed no red cell casts or microscopic hematuria but had +3 protein, 24 hour urine collection revealed heavy proteinuria (9.62 gm / 24 hrs) and serum albumin was 23 gm/ L ( normal= 35- 50).

Glycosylated haemoglobin (HbA1C) was 9.0 % (normal  $\leq 5.7$  %), Lipid profile and Thyroid function test were normal. Creatine Kinase (CK) was slightly elevated 194 U/L (normal up to 190).

Antiphospholipid antibodies, Anti nuclear antibodies, antineutrophilic cytoplasmic antibodies, anti endomyseal antibodies, and complement were all negative. Blood cultures revealed no growth.

Patient was suspected to have deep venous thrombosis and heparin treatment was started but Venous Doppler ultrasound revealed no evidence of deep vein thrombosis (repeated twice).

Electrocardiogram and echocardiogram were normal. Non contrast CT scan of the chest, abdomen, and pelvis did not reveal any lymphadenopathy, or masses.

Magnetic resonant imaging (MRI) through both thighs was done and showed changes of muscle infarction in left thigh (Figure 1) & (Figure 2).

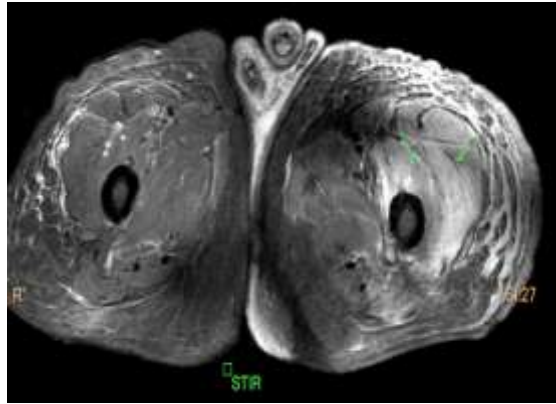


Fig. 1: Abnormal heterogeneous high signal intensity (on Short tau inversion recovery (STIR) sequences) in the quadriceps muscle compartments of the left thigh involving vastus intermedius and vastus lateralis, representing muscle oedema (arrows). Subcutaneous and skin oedema is also shown (curved arrow).

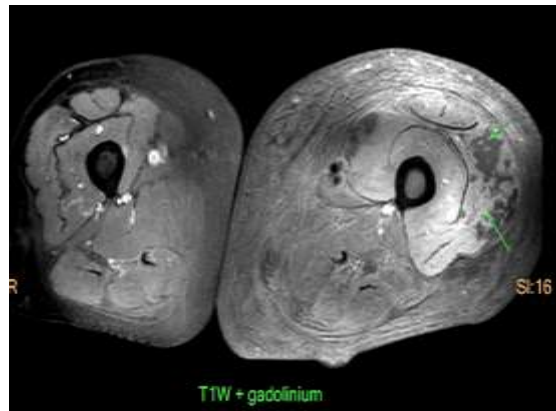


Fig. 2: Axial fat-suppressed contrast-enhanced T1-weighted image of both thighs shows diffuse enhancement involving the quadriceps compartment of the left thigh with multiple foci of central areas of low signal (necrosis) with rim enhancement in the vastus lateralis muscle (arrows) consistent with infarction.

Old records were obtained and showed similar presentation one year ago with repeatedly negative Venous Doppler ultrasound for deep venous thrombosis. A right thigh mass was excised and biopsy result showed skeletal muscle fibers exhibiting marked variation in size of fibers along with foci of nuclear bags in keeping with atrophic changes and one core showed coagulative necrosis of the fibers suggestive of ischemic muscle infarction.

Based on the above findings and after excluding other causes of painful extremity in a patient with diabetes mellitus diagnosis of recurrent diabetic muscle infarction was made. Patient was treated with bed rest and analgesics, and his blood sugar was controlled. He was given intravenous methylprednisolone 80 mg for 7 days, changed to prednisolone 30 mg daily gradually tapered over 2 weeks. His condition and his lab tests improved, he was able to walk and was discharged from the hospital after 4 weeks.

All investigations conform to standards currently applied in Jordan.

## **DISCUSSION**

Diabetic muscle infarction or spontaneous diabetic myonecrosis is a rare vascular complication of longstanding uncontrolled diabetes mellitus that refers to ischemic skeletal muscle necrosis. Due to poor awareness of this rare diabetic complication and presence of many more common conditions that have similar presentations, it is often misdiagnosed or underdiagnosed; it was first reported in 1965 by Angervall and Stener (1) and there have been fewer than 100 patients reported since 1965. DMI typically presents as a localized atraumatic severe painful and tender swelling of acute onset (but sometimes can be subacute or subtle) especially in the lower extremities usually not associated with systemic signs.

It often affects the thighs; with the quadriceps being involved in 83% of cases, and it rarely affects the calf muscles (2). Bilateral involvement occurred in one third of the patients (3). Recurrence at the same or different site occurred in about 45% of 84 patients reviewed (4). About 97% of patients had other microvascular complications including nephropathy, retinopathy or neuropathy (4). With regard to age, the mean age of onset was 42 years (5).

The pathogenesis of DMI is still unclear but a diffuse microangiopathic process possibly associated with hypoxia perfusion injury has been implicated as a cause (6-8).

Poor controlled diabetes has been strongly associated with the development of DMI; most of the known cases of DMI had HbA1C greater than 7% (5).

DMI is a rare and easily misdiagnosed condition that occurs in patients with long standing diabetes, laboratory tests are usually not specific, about three –quarters had ESR greater than 50 mm /hour. One third had leukocytosis, and half only had elevated CK (5).

The diagnosis of DMI should be included in the differential diagnosis in a patient with long standing history of diabetes (especially with microvascular complications) presenting with unexplained sudden painful localized swelling in the lower extremities.

The diagnosis can be confirmed by magnetic resonance imaging (MRI) of the affected extremity, and /or muscle biopsy of the affected muscle(s). MRI is the preferred diagnostic tool; abnormal MRI findings have been reported in all patients with DMI. T<sub>2</sub> weighted sequences shows characteristic features of extensive oedema within the muscle(s), muscle enlargement, subcutaneous and interfascial oedema. The most characteristic is multifocal areas of involvement in a patchwork pattern (6, 9, 10).

Muscle biopsy is not advisable, and often not needed for the diagnosis of DMI, as it may be associated with delayed recovery (11). The biopsy when performed shows pale muscle on

gross examination, irregular areas of haemorrhage, necrosis of myocytes with regeneration, and atrophy of surrounding fibers and hyalinosis and thickening of arterioles (1, 12, 13). In our case excisional muscle biopsy was performed in the first time because of the unawareness of this condition by treating physician, we avoided biopsy in the second time.

The infrequent presentation of this condition makes retrospective literature review the source of valuable information concerning the management of this condition. Most literature supports conservative management with bed rest, avoidance of weight bearing, analgesia, tight control of diabetes, and anti-inflammatory drugs, including steroids (5). The patient symptoms improves (as in this case) after treatment with steroids. Surgery may delay recovery (4).

The recovery period can last for 6 -8 weeks with bed rest and analgesia, with a recurrence rate of 40%. Recurrence may not affect the same muscle group (4). The short term prognosis is good. More ominous, however, is the underlying vascular disease that DMI is associated with. The cause of death were mainly due to macrovascular events such myocardial infarction, stroke, or gangrene (5). Patients with DMI should undergo evaluation for all end organ manifestation of diabetes.

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