Diabetic Muscle Infarction (Myonecrosis): Underdiagnosed or Underreported?

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Abstract
Diabetic muscle infarction is a rare complication of diabetes. It is seen more in Type 1 DM than Type 2 DM, but in both it is associated with longer duration of diabetes, poor glycemic control with or without microvascular complications. We present a case report of a 47 year diabetic male, who presented with sudden onset of painful swelling of the left thigh muscles (vastus group). The patient had microvascular complications of diabetes. Apart for mild elevation of CPK and LDH other investigations were normal. MRI findings of left thigh showed T2 hyperintensities in the involved muscles which established the diagnosis. Muscle biopsy revealed necrosis of the muscle fibres, presence of inflammatory cell infiltrates and hyalinization of the blood vessels with luminal narrowing which is characteristic of diabetic muscle infarction. The patient was treated with immobilization, analgesics and adequate blood sugar control. This case highlights the rarity of finding vascular changes as well as the poor prognosis and the occurrence of fatal complications in near future.

Introduction
Diabetic muscle infarction is a rare complication of diabetes. It is seen more in Type 1 DM than Type 2 DM, but in both it is associated with longer duration of diabetes, poor glycemic control with or without microvascular complications.

Case Report
A 42 year male, a known diabetic and hypertensive for 3 yrs, presented with complaints of generalised weakness. He had no other significant history. He was found to have high blood sugars (440mg/dl).

On examination vitals were stable, all peripheral pulses were felt. Systemic examination was essentially normal and there was no other target organ involvement, clinically. Patient was admitted to wards and put on regular insulin and supportive treatment.

The investigations (Table 1) revealed that the patient had already developed microvascular complications in the form of Grade 2 diabetic nephropathy and fundoscopy revealed NPDR.

On 4th day of hospitalization, he complained of sudden onset of pain in left thigh. There was no preceding trauma or fever. Examination revealed localized swelling of left thigh region which was warm and tender with induration. There was no clinical evidence of deep vein thrombosis.

Further investigation for the cause of swelling was undertaken. CPK was 371U/L (24-195) and LDH was 384U/L (90-200). USG of left thigh revealed a predominantly hyperechoic collection with increased peripheral vascularity, in the rectus femoris and vastus laterals muscles.

CECT of the left thigh (Figure 1) showed bulky left vastus lateralis, parts of rectus femoris and sartorius muscle with central non-enhancing regions (open arrow). These features were consistent with diabetic myonecrosis.

The MRI of the left thigh showed hyperintensities in T2 and STIR images, mainly in vastus lateralis, parts of rectus femoris and sartorius muscle (Figure 2), which became hypointense on T1 (Figure 3).

Muscle biopsy of sartorius muscle confirmed the diagnosis, it showed skeletal muscle fibres with foci of myonecrosis, macrophage phagocytosis and inflammatory cell infiltrate and granulation tissue formation (Figure 4A). Blood vessel appeared thickened and hyalinised.

Table 1: Investigations

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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<tr>
<td>Hb 14 Gms/dl</td>
<td>S. Urea- 38 mg/dl</td>
</tr>
<tr>
<td>TLC 11000</td>
<td>S. Creat- 1.1 mg/dl</td>
</tr>
<tr>
<td>(P 80%, L 18%, E 2%)</td>
<td>LFT- WNL</td>
</tr>
<tr>
<td>Platlets 2 Lacs</td>
<td>Lipid profile:</td>
</tr>
<tr>
<td>ESR 90mm/ 1st hour</td>
<td>TGs-125 mg/dl</td>
</tr>
<tr>
<td>Urine KB negative</td>
<td>T.Chol- 232 mg/dl</td>
</tr>
<tr>
<td>FBS 323 mg/dl</td>
<td>HDL- 47 mg/dl</td>
</tr>
<tr>
<td>PPBS 541 mg/dl</td>
<td>LDL- 160 mg/dl</td>
</tr>
<tr>
<td>HbA1C 10.3%</td>
<td>Urine Microalbuminuria</td>
</tr>
<tr>
<td></td>
<td>50.53 mg/dl (upto 30)</td>
</tr>
</tbody>
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with luminal narrowing suggesting arteriosclerotic changes (Figure 4B).

A diagnosis of diabetic myonecrosis was made and the patient was advised analgesics and adequate bed rest. Four weeks later the patient reported to have considerable reduction in pain, swelling and induration.

**Discussion**

Diabetic muscle infarction (DMI) or diabetic myonecrosis is a rare complication of diabetes or possibly, an underdiagnosed complication. The first case was reported by Angervall and Stener in 1965. The exact prevalence of the disease is not known. In world literature there are less than 200 cases reported and from India only 5 cases have been reported.\(^1\) Considering the number of diabetics in our country this is an extremely small number.

DMI is described in people with long-standing diabetes with a greater incidence in Type 1 diabetes (71%). However, patients with Type 2 DM also develop this complication.\(^2\)

The clinical presentation is of an acute atraumatic painful swelling of the affected muscle and a palpable mass. Commonly the thigh muscles (quadriceps (62%), hip adductors (13%), hamstrings (8%), and hip flexors (2%)\(^3\) are affected but the calf muscles can also get involved. One case of upper limb involvement has also been reported. Bilateral involvement has been reported in 8.4% cases.\(^4\)

A differential diagnosis as shown in Table 2 should be considered as these are of a more serious nature requiring active intervention. The use of USG and MRI ruled out these problems, in our case.

Although the exact pathogenesis of muscle infarction in diabetes has not been identified, severe diabetic microangiopathy leading to occlusion of blood vessels and ischemia has been proposed as the underlying mechanism that leads to spontaneous non-gangrenous and focalized muscle infarction. Other proposed theories include coagulation-fibrinolysis derangement, hypoxia-reperfusion abnormalities, and recently, the role of antiphospholipid antibodies.\(^5\)

Diabetic myonecrosis is a sporadic microvascular complication commonly seen in patients with poor glycemic control. Invariably the patient will have microvascular complications of diabetes mellitus, such as nephropathy (71%), retinopathy (56%) and neuropathy (54%).

Laboratory investigations may be unhelpful. Creatine phosphokinase levels are normal or slightly elevated in 52%; leucocytosis occurs in 14% of cases. AST, ALT, LDH levels and the erythrocyte sedimentation rate are normal most times. In our case there was marginal elevation of both CPK and LDH levels while the leukocyte count and ESR were elevated.
Sonography aids in detecting venous thrombosis, if any. In diabetic muscle infarction it shows internal linear echogenic structures coursing through the lesion; an absence of internal motion or swirling of fluid with transducer pressure; and lack of predominantly anechoic areas.

CT scan shows diffuse muscular enlargement with diminished attenuation of the affected muscle, increased attenuation of the subcutaneous fat, and thickening of the subcutaneous fascial planes and of the skin.4

MRI is the best investigation for diagnosis.4 The characteristic features of diabetic myonecrosis include increased signal from the affected muscle area in T2-weighted, inversion-recovery, and gadolinium-enhanced images and isointense or hypointense areas on T1-weighted images.

Muscle biopsy can firmly ascertain the diagnosis, however it is routinely not recommended in view of post-procedural complications that delay in wound healing. In our case, the demonstration on muscle biopsy of arteriosclerotic changes shows that diabetic microangiopathy is the cause.

Management is mainly conservative. Good glycemic control should be achieved and patients usually recover within few weeks with supportive treatment like bed rest and analgesics for pain.

The long-term prognosis of this complication is poor in the form that most patients die within 5 years of diagnosis. The main underlying difficulty is advanced generalized microvasculopathy leading to life-threatening complications. Up to 50% of cases may have a recurrence, most of them in a previously affected muscle. Almost half of these recurrent events occur in a period of two months.

Conclusion

We have reported a case of diabetic muscle infarction, with involvement of the left thigh in a Type 2 diabetic patient. Recognition of this complication is essential as it carries a poor long-term prognosis. Early intervention in the control of diabetes, can probably lead to better outcomes. We therefore, feel that DMI is an underdiagnosed problem and creating awareness among clinicians managing diabetes would go a long way in reducing the morbidity and mortality associated with the microvascular complications of diabetes mellitus.

References