

A CASE OF DIABETIC MUSCLE INFARCTION DESPITE GOOD DIABETIC CONTROL

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Case Presentation

A 52 year-old female with type 2 diabetes of 10-year duration was admitted with worsening of shooting pain and swelling of her lower extremities over the previous nine days. The pain was rated 10/10 in severity, was worse in the right side as compared to the left, radiated from her lower legs to thighs, and limited her mobility for the previous five days. She reported no history of trauma, fever, chills, skin changes, or medication noncompliance. She was managing her diabetes with two injections of premixed insulin. Her other medical problems include a history of congestive heart failure with an ejection fraction of 45%, coronary artery disease, stage 4 chronic kidney disease, and hypertension. A review of systems was remarkable for exertional dyspnea and mild ascites.

On admission, the patient was afebrile, had blood pressure 147/101 mm Hg, a regular pulse of 88 beats/minute, respiratory rate of 18 breaths/minute, and pulse oximetry of 100% on two liters of oxygen via nasal cannula. Cardiovascular exam was benign except for scattered crackles at the bases on deep inspiration. A musculoskeletal examination revealed tender, edematous lower extremities up to the top third of her thighs without any skin excoriation or ulceration. All pulses were palpable with normal pressure and volume in both extremities. Neurological exam was only significant for inability to walk secondary to pain and swelling.

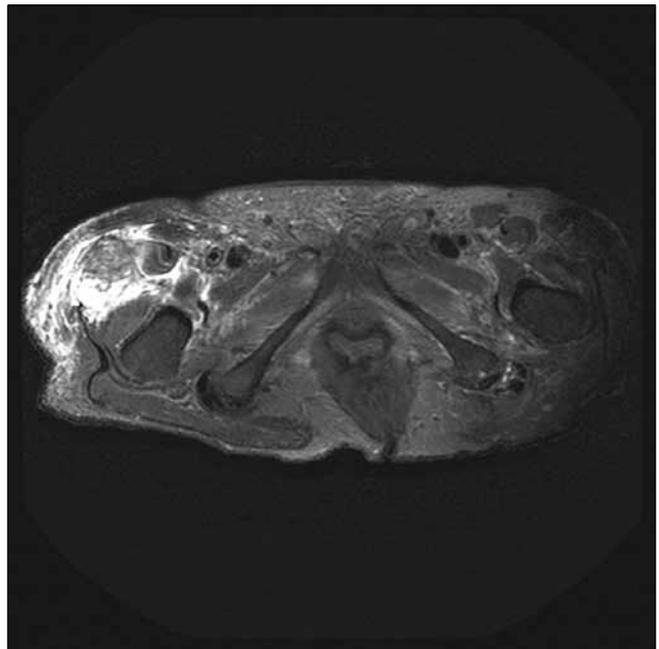
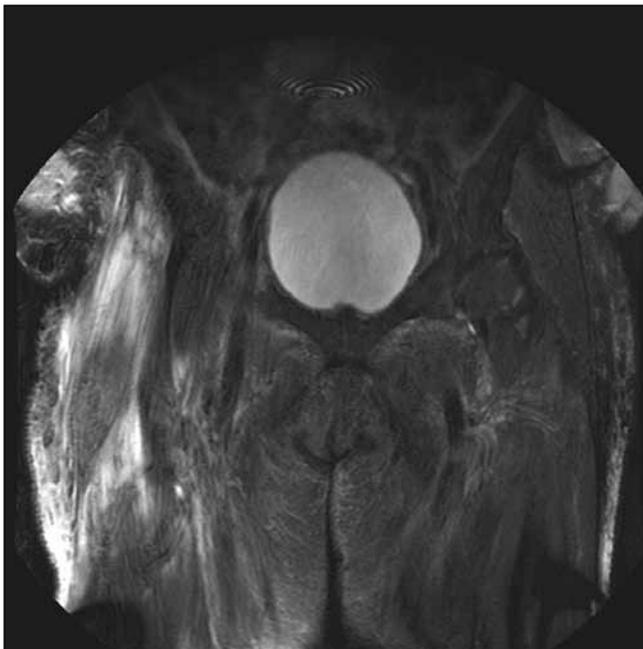
The laboratory work-up revealed a hemoglobin A_{1c} level of 6.5%, fasting blood glucose of 95 mg/dL, serum creatinine of 3.4 mg/dL, white blood count (WBC) of 17.1 B/L, and an erythrocyte sedimentation rate (ESR) of 95 mL/hr.

A diuretic was initiated to lessen lower extremity edema as it was initially thought to be due to congestive heart failure. Lower-extremity ultrasound was negative for deep vein thrombosis, and a blood culture was negative. Magnetic resonance imaging (MRI) of lower extremities revealed diffuse swelling and edema involving the tensor fascia lata with areas of muscle replaced by fluids in both legs especially in the thigh area (Figures 1 and 2).

A diagnosis of diabetic myonecrosis was made on the basis of MRI findings, raised ESR, and elevated WBC in the absence of negative blood cultures and fever. The patient was managed with supportive care and analgesia as needed.

Discussion

Diabetic myonecrosis is a rare complication of diabetes, first described in 1965 and known to affect patients with poorly controlled diabetes of long-standing duration.¹ Most common presentation of diabetic myonecrosis is acute lower extremity pain and swelling involving calf and thigh. These patients often have established nephropathy, retinopathy, and a long-standing history of poor glycemic control.



Figures 1 and 2. Coronal Image - T2 weighted with fat saturation. The image shows diffuse swelling involving the entire right tensor fascia lata with areas of muscle replacement by fluid, findings consistent with diabetic myonecrosis. There is also swelling of the left adductor magnus, which may represent diabetic myopathy versus early diabetic myonecrosis.

The pathophysiology of diabetic myonecrosis is not completely understood. The possible contributing role of athermatous emboli, thrombo-occlusive disorder, and arteriosclerosis has been implicated in some cases.²

Table 1. Differential Diagnosis of Acute Lower Extremity Swelling.

Deep Vein Thrombosis
Thrombophlebitis
Trauma
Infection
Fasciitis
Hematoma
Diabetic Myotrophy
Myositis Ossificans
Muscle Strain or rupture

Laboratory tests typically reveal leukocytosis, elevated C reactive protein, and creatinine kinase in 35%, 75%, and 45% of cases, respectively.³ Histological features of diabetic myonecrosis consist of large areas of muscle necrosis and edema.⁴ Diagnosis is primarily made through clinical suspicion and on MRI. Recurrence is common and has been reported in up to 50%

of cases. Diabetic myonecrosis is a self-limited condition, which resolves on its own within weeks to months. Treatment is supportive care with analgesia, rest, and optimal glucose control. Physical therapy is not recommended, as it may acutely aggravate necrotic muscle.

Conclusion

After reviewing the literature, this case is one of the first reports of diabetic myonecrosis in a well-controlled diabetic patient. This case demonstrates that the diagnosis of myonecrosis should be considered even in well-controlled diabetics in the setting of acutely painful, swollen lower extremities in patients with long-standing diabetes.

References

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