

Diabetic Myonecrosis: Rare And Ominous Complication Of A Common Disease

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Background

Diabetic Myonecrosis is an uncommon complication of Diabetes Mellitus (DM), often presenting with severe pain in the medial thigh. Diabetic myonecrosis is often misdiagnosed as a neoplasm, an abscess, or myositis. Significant proportions of patients do not comply with their insulin regimen and as a result have poor diabetic control for 5 to 30 years (mean, 15 years). This is reflected in the increased prevalence of diabetic end-organ damage. Interestingly, retinopathy, neuropathy, nephropathy, and hypertension are present in most of such patients. Diabetic myonecrosis is unique as it occurs in young adult (mean age, 37 years) and is more common in females. The etiology may be atheroembolism or arteriosclerosis obliterans. The pathophysiologic pathway of diabetic myonecrosis is controversial. Some investigators propose, a compartmental syndrome exacerbating ischemia versus abnormalities in the clotting cascade or in fibrinolytic pathways.

The diabetic myonecrosis patients usually present with, an abrupt onset of severe pain in the thigh and exquisitely tender swelling in the absence of trauma or fever. The quadriceps compartment is most frequently involved, followed by the thigh adductors and hamstrings, with only occasional involvement of the calf muscle. The pain is present at rest and exacerbated by movement. Muscle strength appears to be normal, with limited range of motion. Laboratory tests typically show a normal white-cell count and normal or slightly elevated levels of creatine kinase. MRI is the most sensitive diagnostic modality available for this condition. However, histopathological evaluation is confirmatory.

Short-term prognosis of patients with diabetic myonecrosis is excellent. Diabetic myonecrosis itself is of little significance, as symptoms resolve with adequate diabetic control and supportive care. Recurrence is seen in half of the cases, either in the same leg or in the opposite leg. Long-term prognosis of these patients is generally poor because of associated generalized vascular disease. Significance of diabetic myonecrosis lies not only in early detection and subsequent treatment, but also investigating the patient for target organ damage.

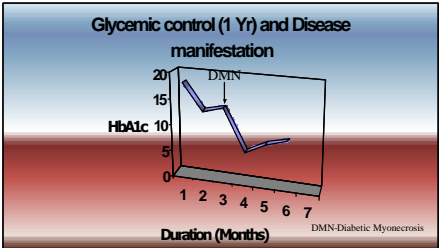
Case Report

History and Examination:

A 35-year-old African American male presented to the Emergency Department with a 2-week history of localized pain [10/10 (1 being best & 10 being worst)] and swelling in the medial side of the right thigh worsening with activity. Low-grade intermittent fever was present. The patient was a known diabetic (type 2) for 10 years, managed on insulin and oral hypoglycemics. HbA1c was 14% on admission. Past medical history was significant for alcoholic cardiomyopathy (EF 20%), hypertension, diabetic nephropathy, anemia and depression. Other medications included, Metoprolol, Risperdal, Acetyl Salicylic acid, Pepcid and Lisinopril. Family History was non-contributory.

On examination there was local warmth, tenderness and an ill-defined swelling in the medial thigh without any induration or crepitus. A grade-2/6 pansystolic murmur was present and distal pulses were intact bilaterally. CBC revealed leukocytosis and anemia. Coagulation profile was normal.

Figure 1: Graphical Representation Of Hba1c And Natural History Of Disease



Management

Investigations:

CT of the right thigh showed prominence of right vastus group of muscles (asymmetric fullness of the right vastus with loss of fascial planes). These features were suggestive of myositis with fascitis. Simultaneous real time imaging of the venous blood flow using both pulsed and color Doppler, as well as B-mode evaluation study of the lower limbs was negative for DVT. The patient was treated with broad-spectrum antibiotics for 3 days. The patient continued to have pain. Creatinine kinase and Aldolase were elevated. Multiplanar imaging of the right thigh was performed in multiple sequences before and after the administration of gadolinium. Gadolinium enhanced MRI revealed multiple nonenhancing regions within the vastus medialis with interfacial fluid suggestive of myonecrosis or pyomyonecrosis with superimposed fascitis.

During surgical exploration vastus medialis appeared to be fibrofatty, firm and woody with no contractility. It appeared to be non-viable throughout its entire extent distally. The gracilis muscle appeared to be spared. Histologically, vastus medialis showed patchy recent myocyte necrosis with active acute inflammatory cell infiltrate, predominantly composed of polymorphous nuclear leucocytes. In some areas, muscle fibers were atrophic, degenerated and show endomysial lymphocytic infiltrates. The perimysial and surrounding fibrous tissue revealed fibroblastic proliferation forming granulation tissue. All sized blood vessels demonstrated severe arteriosclerotic changes with intimal fibroplasias and narrowing of lumen. Tissue cultures were negative for fungi, bacteria and acid-fast bacilli.

Figure 1:

Gadolinium enhanced MRI of right thigh (Before and after administration) - revealing multiple nonenhancing regions (arrow) within the vastus medialis with interfacial fluid - suggestive of myonecrosis or pyomyonecrosis with superimposed fascitis

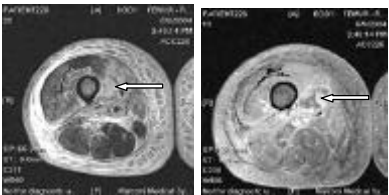


Figure 2:

Photomicrograph of Vastus medialis muscle showing diffuse myocyte necrosis (H&E, original magnification X100)

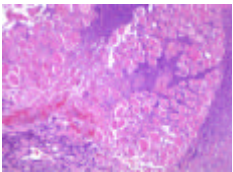
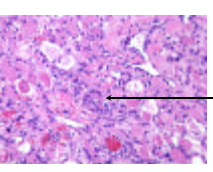


Figure 3:

Photomicrograph of Vastus medialis muscle showing severe arteriosclerosis of small blood vessel (arrow) (H&E, original magnification X400)



Differential Diagnoses:

The most important differential diagnoses considered were Deep vein Thrombosis, cellulitis, fasciitis, abscess, hematoma, pyomyositis, soft tissue sarcoma, osteomyelitis, calcific myonecrosis, myositis ossificans, diabetic amyotrophy, vasculitis, arterial occlusion, tuberculosis and amyloidosis. In the following table highlights the most important differentiating features.

Treatment and outcome:

Patient received analgesic therapy with opioids, immobilization and physical therapy. Blood glucose levels were strictly controlled with insulin. Other issues during admission included nephritic syndrome and congestive heart failure. Patient improved over the course of one week and was discharged.

Table 1:

Clinical symptoms and signs for diabetic myonecrosis and its differential diagnoses:

Sign / Symptom	Diabetic myonecrosis	Contusion	Pyomyositis	Sarcoma
History	Poorly controlled DM	±Trauma	±Trauma	Non-specific
Onset of Pain	Acute	Acute	Sub-acute	Insidious
Fever	No fever	-	+	-
Leucocytes	±	-	+	-
Creatinine				
Phosphokinase	↑	↑↑	Normal	Normal / ↑
E.S.R.	-	-	↑	-
C.T./M.R.I.	Muscle Edema	Hematoma and	Fluid / Abscess edema	Hemorrhage and edema

Conclusion

Diabetic myonecrosis is an uncommon, missed sign of underlying chronic vascular disease. Most patients reported in literature are relatively young with a mean 15 yr history of diabetes with poor chronic control. The pathophysiology is not understood yet. The most accepted hypotheses being arteriosclerosis obliterans, compartmental syndrome or abnormalities in clotting cascade. No correlation is found with trauma or local injection of insulin. MRI is diagnostic, biopsy is the "gold standard" and treatment is non-specific. Its diagnosis remains a surrogate marker of severe uncontrolled diabetes. In patients with diabetic myonecrosis, aggressive control of blood sugar is imperative because most patients die of other complications from their diabetes within 5 yr of diagnosis.

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