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Diabetic Myonecrosis: A Rare Complication of Diabetes Mellitus Mimicking Deep Vein Thrombosis

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Study Design A
Data Collection B
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Data Interpretation D
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Patient: Male, 54
Final Diagnosis: Diabetic myonecrosis
Symptoms: Calf pain and swelling
Medication: —
Clinical Procedure: —
Specialty: Internal Medicine

Objective: Rare disease

Background: Diabetic myonecrosis is an uncommon complication of long-standing poorly controlled diabetes mellitus. It presents as acute non-traumatic swelling and pain of the lower extremity, which can mimic deep vein thrombosis (DVT). The clinical course is usually self-limiting and patients respond well to supportive medical therapy.

Case Report: A 54-year-old male with past medical history of poorly controlled diabetes mellitus type II, hyperlipidemia, gastroesophageal reflux disease (GERD), and remote history of DVT presented to our emergency department with 2-week history of progressively worsening left calf pain and swelling. On physical examination, the patient had increased warmth, edema, erythema, and tenderness in the left calf, with positive Homan's sign. A lower-extremity venous Doppler was negative for DVT. His creatinine phosphokinase (CPK) level was normal, but hemoglobin A1C was 11.0%, reflective of poor glycemic control. Magnetic resonance imaging (MRI) of the left calf revealed a focus of non-enhancement in the gastrocnemius muscle along with increased enhancement of the rest of the muscle, suggestive of diabetic myonecrosis.

Conclusions: Diabetic myonecrosis is a rare complication of long-standing diabetes mellitus that can often mimic DVT. Diagnosis can be made on an MRI, and treatment involves strict glycemic control along with antiplatelet therapy and non-steroidal anti-inflammatories (NSAIDs).

MeSH Keywords: Diabetes Mellitus • Magnetic Resonance Imaging • Muscle, Skeletal • Necrosis • Ultrasonography, Doppler, Color

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Background

Diabetic myonecrosis is an uncommon complication of diabetes mellitus that occurs in patients with long-standing poorly controlled diabetes. Angervall and Stener first reported it in 1965 as focal muscular degeneration in 2 diabetic patients [1]. Since then, more than 100 cases have been reported [2]. It is also known as spontaneous diabetic muscle infarction and can occur in patients with either type I or type II diabetes. Although the pathogenesis of this disease is unclear, it is thought to be secondary to diabetic microangiopathy. Due to the rarity of this disease, it is often under-diagnosed or under-reported, resulting in unnecessary testing and delay in diagnosis. We present the case of a patient with left calf pain secondary to diabetic myonecrosis, mimicking deep vein thrombosis (DVT).

Case Report

A 54-year-old white male with past medical history of diabetes mellitus type II, hyperlipidemia, GERD, and remote history of DVT (about 15 years ago) presented to our emergency department with complaints of left calf pain and swelling. He noted swelling in his left calf about 2 weeks prior to admission, associated with calf pain while walking. He denied any recent surgery, prolonged immobilization, or trauma to his leg. He denied any other systemic complaints. His past medical history was significant for long-standing poorly controlled diabetes mellitus type II complicated by diabetic neuropathy, without any evidence of nephropathy or retinopathy. He also reported having a DVT about 15 years ago for which he was treated with warfarin for 6 months and did not have any recurrent episode of DVT. Additionally, he reported having GERD and hyperlipidemia. His home medications included insulin glargine 50 units daily, gemfibrozil 600 mg 3 times a day, Glyburide-Metformin 2.5 mg – 500 mg twice a day, aspirin 81 mg daily, pantoprazole 40 mg daily, atorvastatin 80 mg daily, and gabapentin 300 mg 3 times a day. The patient reported taking the above medications, but the medication compliance was questioned given his elevated hemoglobin A1C level.

The patient's vital signs on admission were within normal limits. On physical examination, he was noted to have increased warmth, edema, and tenderness in his left calf, with positive Homan's sign. His foot was warm to touch, with 3+ dorsalis pedis and posterior tibial pulses in both feet, which ruled out any signs of arterial occlusion or compartment syndrome. His laboratory studies, including complete blood count and comprehensive metabolic panel, were unremarkable, except for hyperglycemia with blood glucose level of 273 mg/dL on admission. The hemoglobin A1C was noted to be elevated at 11.0%, reflective of poor glycemic control. His C-reactive protein (CRP) was slightly elevated at 12.9 mg/L, but his creatinine phosphokinase (CPK) level was normal at 44 U/L. He underwent a

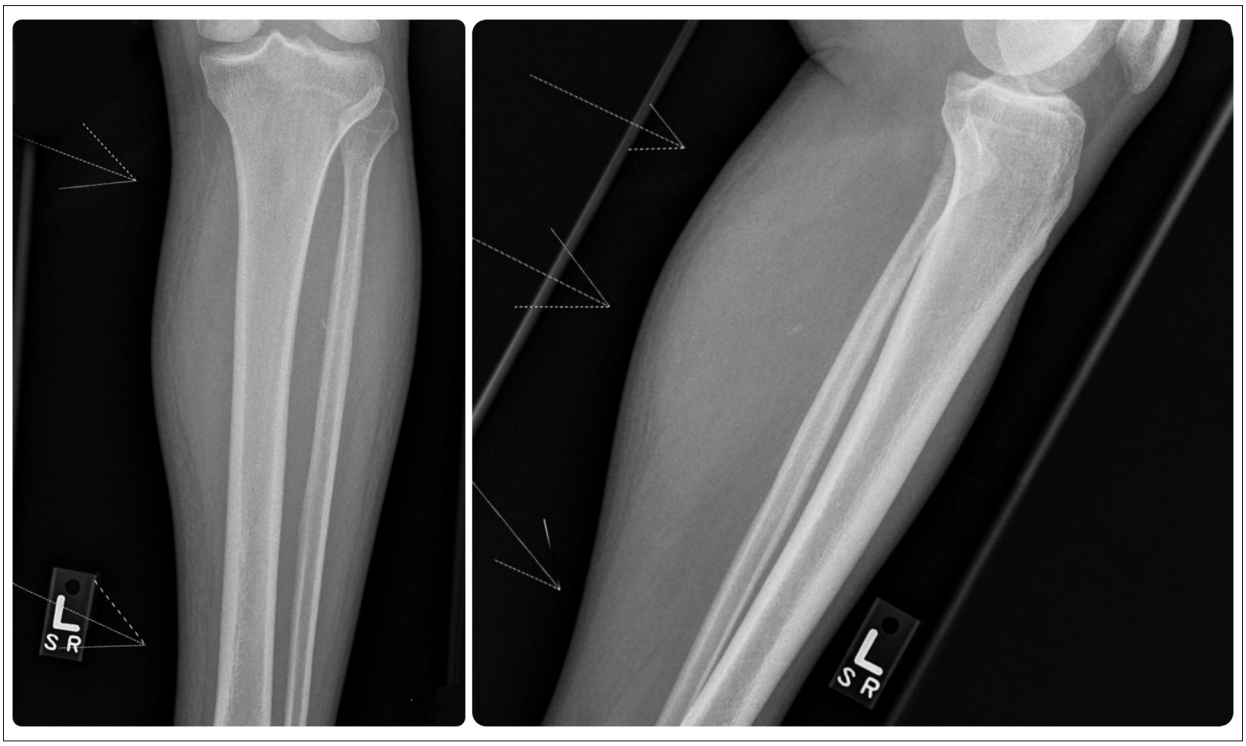

Figure 1. Left AP and lateral tibia fibula X-ray, which demonstrates soft tissue swelling of the calf and a small focus of increased density (white arrows) suggestive of dystrophic soft tissue mineralization.

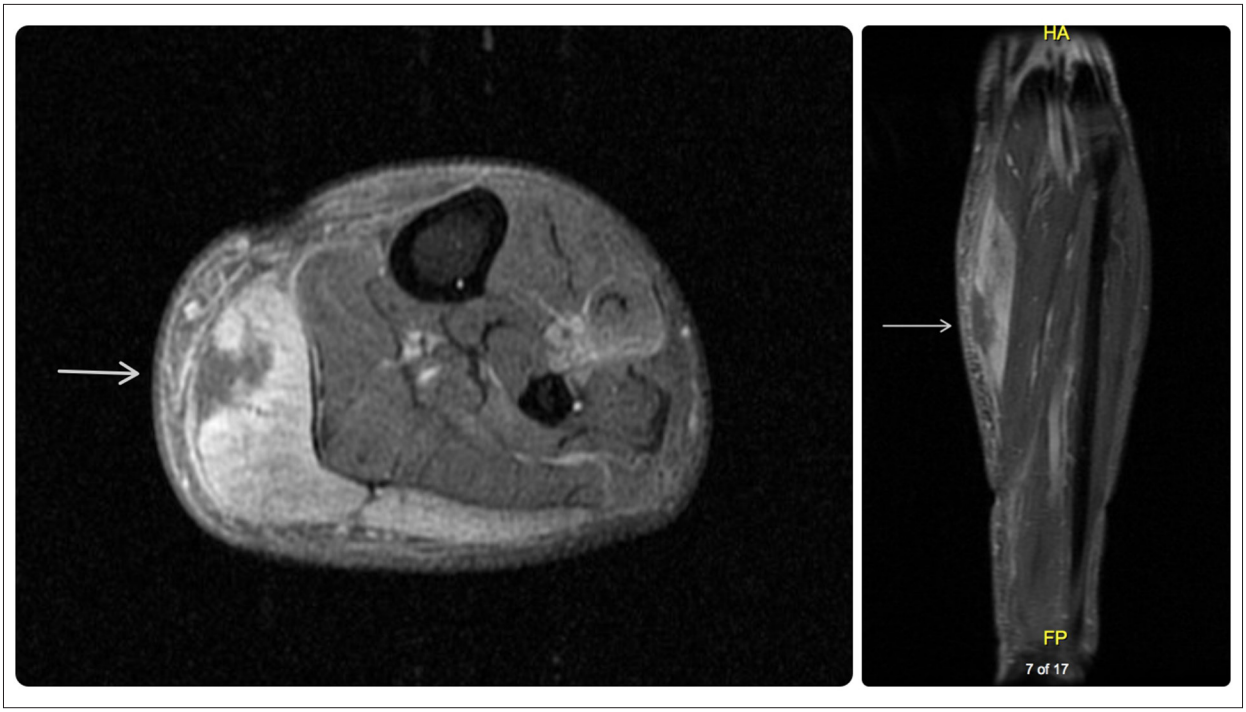

Figure 2. Left calf MRI axial and coronal FSE T1 with contrast. MRI shows a 4.7×2.1×1.6 cm focus of non-enhancement (white arrow) in the inferior medial head of the gastrocnemius muscle and edema-like signal changes with enhancement of the rest of the gastrocnemius and extensor digitorum longus muscle, suggestive of diabetic myonecrosis.

venous Doppler due to high suspicion of DVT based on clinical examination and remote history of DVT; however, the venous Doppler was negative for any thrombus or occlusion. He also underwent an X-ray of the left leg (Figure 1), which was negative for any fracture, but it did show soft tissue swelling of the calf and a small focus of increased density suggestive of dystrophic soft tissue mineralization. Given the negative venous Doppler and suspicious X-ray findings, he underwent an MRI of the calf with contrast. The MRI showed a 4.7×2.1×1.6 cm focus of non-enhancement in the inferior medial head of the gastrocnemius muscle and edema-like signal changes with enhancement of the rest of the gastrocnemius and extensor digitorum longus muscle, suggestive of diabetic myonecrosis (Figure 2). Surgical consultation occurred and a general surgeon evaluated the patient, recommending conservative treatment instead of surgery because there were no signs of sepsis or compartment syndrome. The patient was continued on low-dose aspirin and was started on IV ketorolac 30 mg every 6 hours as anti-inflammatory and pain control, which resulted in modest improvement in the calf pain and swelling over the next 2–3 days while he was in the hospital. He was started on insulin glargine, prandial lispro, and sliding scale for strict glycemic control. His repeat laboratory work-up was unchanged. He was subsequently discharged home on metformin, glipizide, Lantus, low-dose aspirin, and oral ketorolac with outpatient follow-up.

Discussion

Diabetic muscle infarction, otherwise known as diabetic myonecrosis, is a rare complication of diabetes mellitus that occurs in patients with long-standing diabetes (usually >15 years) and often along with other microvascular complications such as retinopathy, nephropathy, or neuropathy. It is more common in type I diabetics but can also occur in patients with type II diabetes [2]. Patients often present with acute-onset unilateral muscle pain, tenderness, and swelling [3]. Bilateral involvement can occur and has been previously reported, but it is rare. The most commonly affected muscle group is the thigh muscles (71.2%), including the quadriceps, hip adductors, hamstrings, and hip flexors, with calf muscles (15.3%) being the second most common site involved [4,5]. Although neuropathy is frequently seen in patients with long-standing diabetes mellitus such as our patient, there is no clear evidence at this time suggesting that neuropathy increases the risk of developing diabetic myonecrosis.

The pathophysiology of diabetic myonecrosis is not fully understood; however, several theories have been put forth, including arteriosclerosis, microangiopathy, ischemia-reperfusion injury, and an alteration in the coagulation-fibrinolysis system [6,7]. Chester and Banker suggested an atheroembolic phenomenon, but the results of their study were more consistent with arteriosclerosis obliterans, which at this time

appears to be the most widely accepted hypothesis [8]. Others proposed theories include alterations in the coagulation pathway (with increased factor VII activity and increased level of plasminogen activator inhibitor) as well as the presence of phospholipid antibodies as possible etiologies for myonecrosis, but more research is needed this field to establish a definitive cause [9,10].

Diagnosis of diabetic myonecrosis begins with a strong clinical suspicion of the disease. Most common differential diagnoses include DVT, cellulitis, hematoma, abscess, pyomyositis, fasciitis, and malignancy. Laboratory testing, including leukocyte count, CPK, and inflammatory markers, are non-specific as they can be elevated or normal in patients with diabetic myonecrosis [11]. The test of choice is MRI, with and without contrast, of the muscle group involved, which is highly sensitive in diagnosis of this disease [5,12]. Typical MRI findings include loss of intermuscular septae (most easily seen on T1-weighted images) and diffuse enlargement of the involved muscle, with subcutaneous edema (best visualized on T2-weighted images as hyperintensity of the involved muscle compared with unaffected muscle and fat) [13]. MRI images show diffuse enhancement of the affected muscle, which represents edema and inflammation, and a focal area of low intensity signal suggestive of muscle necrosis or infarction [13]. Although muscle biopsy is the criterion standard for diagnosing diabetic myonecrosis, it is reserved for atypical patients when clinical or radiologic findings are not suggestive, due to the poor wound healing and high risk of infection associated with muscle biopsy [14].

Treatment is usually conservative, with bedrest, antiplatelet agents, and non-steroidal anti-inflammatory drugs (NSAIDs) [14]. In rare cases, surgical exploration may be needed if there are signs of sepsis or compartment syndrome. Early recognition of the disease helps avoid unnecessary use of antibiotics [14]. Short-term prognosis is good, with most patients reporting resolution of symptoms in a few weeks to months. However, the recurrence rate is as high as 40%, with 2-year mortality of 10% [15]. Recovery time is shortened by prompt diagnosis and early treatment. The long-term prognosis, however, is poor due to underlying microvascular disease associated with diabetes; hence, the best treatment strategy is achieving optimum glycemic control.

Conclusions

Diabetic myonecrosis is an under-diagnosed and uncommon complication of long-standing poorly controlled diabetes, and due to its rarity is can often be mistaken for DVT or cellulitis. Physicians should have a high index of suspicion in patients with poorly controlled diabetes who present with acute-onset painful swelling of any extremity, with negative venous Doppler for DVT. MRI is the diagnostic test of choice and muscle biopsy is reserved for atypical cases. Treatment should focus on strict glycemic control along with bedrest, NSAIDs, and antiplatelet therapy.

Conflict of interest

None declared.

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