CLINICAL MANAGEMENT extra

A Wolf in Sheep's Clothing: An Unusual Presentation of Diabetic Myonecrosis

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GENERAL PURPOSE:

To provide information about the diagnosis and treatment of diabetic myonecrosis (DMN).

TARGET AUDIENCE:

This continuing education activity is intended for physicians, physician assistants, nurse practitioners, and nurses with an interest in skin and wound care.

LEARNING OBJECTIVES/OUTCOMES:

After participating in this educational activity, the participant should be better able to:

1. Cite the incidence and symptomatology of diabetic myonecrosis.

2. Identify the diagnostic tests associated with DMN.

3. Summarize the evidence-based treatments for DMN.

ABSTRACT

Diabetic myonecrosis is a rare complication of poorly controlled diabetes mellitus that presents similarly to many common conditions such as cellulitis, abscess, and fasciitis. Therefore, a high index of suspicion is required for diagnosis. Magnetic resonance imaging is the investigative test of choice. Treatment includes antiplatelet therapy, nonsteroidal anti-inflammatory agents, and glycemic control.

KEYWORDS: aseptic myonecrosis, cellulitis, diabetic myonecrosis, diabetic muscle infarction, diabetic complications, muscle necrosis, necrotizing soft tissue infections

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INTRODUCTION

Diabetic myonecrosis (DMN) is a spontaneous infarction of skeletal muscle and an acute complication of poorly controlled, long-standing diabetes mellitus (DM). This rare disease has fewer than 200 cases reported in the literature.[1] The presentation of DMN may mimic cellulitis, abscess, deep vein thrombosis (DVT), hematoma, pyomyositis, fasciitis, or malignancy;[1,2] overlapping symptomatology such as pain and erythema can easily lead to misdiagnosis. This report describes a case of DMN that was initially diagnosed and managed as cellulitis. Subsequent radiologic imaging led to the revised diagnosis of DMN.

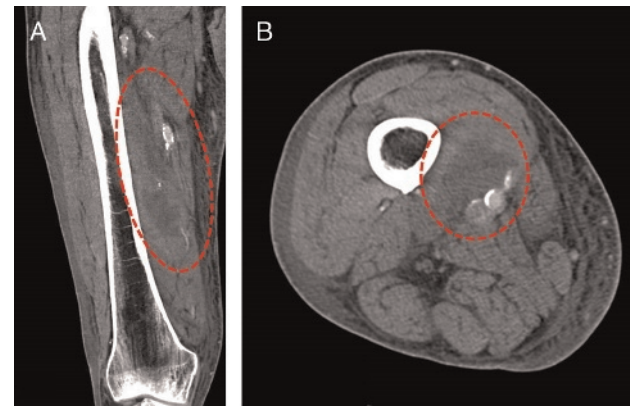
CASE REPORT

A 56-year-old white man with poorly controlled type 2 DM presented to the emergency department (ED) with right inner thigh pain that began 3 days prior to presentation. He initially attributed the symptoms to a pulled muscle; however, worsening pain, swelling, and redness prompted his visit to the ED. He denied any history of trauma, recent travel, prolonged immobilization, fever, or chills. His medical history was significant only for poorly controlled DM for the last 40 years.

In the ED, his blood pressure was elevated (151/92 mm Hg). On physical examination, the entire right medial thigh was noted to be edematous, erythematous, warm, and tender with no palpable crepitus. He was also noted to have an open, 3-cm grade 3 ulcer present over his right Achilles tendon, which he reported had been present for the last several months secondary to trauma from wearing new shoes. The right dorsalis pedis and posterior tibial pulses were 2+. Laboratory findings were significant for leukocytosis, 14,400/L; sodium, 132 mEq/L; blood glucose, 406 mg/dL; hemoglobin A1c, 10.9%; C-reactive protein, 114 mg/L; and erythrocyte sedimentation rate, 70 mm/h. In light of the physical examination and laboratory findings, the patient was admitted with a diagnosis of right medial thigh cellulitis and was started on cefazolin, insulin glargine, and insulin aspart with local wound care for the right heel ulcer.

A bilateral lower extremity venous duplex was performed and showed no evidence of a DVT. A computed tomography (CT) scan was then performed to evaluate for soft tissue infection and showed 2 fluid collections in the vastus medialis at the level of the mid to distal femur, reflecting a developing abscess versus myonecrosis (Figure 1). The CT also revealed nonspecific edema within the abductor magnus muscle and diffuse subcutaneous soft tissue edema without gas. Based on the radiologic findings, antibiotic coverage was broadened to vancomycin and meropenem. An ultrasound of the right thigh showed an amorphous, intramuscular, complex fluid collection within vastus medialis muscle. Magnetic resonance imaging (MRI) was then performed, which revealed a 13.2 × 5.5 × 4.0-cm irregular area within the vastus medialis and adductor muscle groups with peripheral enhancement most consistent

Figure 1.
COMPUTED TOMOGRAPHY OF THE RIGHT THIGH



Coronal view (A) and transverse view (B) with red outline illustrating the areas of fluid collection in the vastus medialis.

with myonecrosis (Figure 2). Based on this information, superimposed infection was still a possible diagnosis. The patient was given enoxaparin for DVT prophylaxis during his admission. The patient was continued on antibiotics with subsequent clinical improvement and was discharged home with intravenous daptomycin and ertapenem, in addition to insulin and antihypertensive medication for newly diagnosed hypertension.

The patient was seen repeatedly in the wound care outpatient clinic for follow-up and reported resolving pain of his right thigh, with complete resolution 6 weeks after discharge from the hospital. The patient was also followed by the infectious disease department and completed a recommended 6-week course of minocycline. Repeat MRI was performed 1 month after discharge and showed resolving DMN (Figure 3). An additional MRI repeated 6 weeks later showed further resolution of the intramuscular fluid collection and a decrease in surrounding reactive edema (Figure 4).

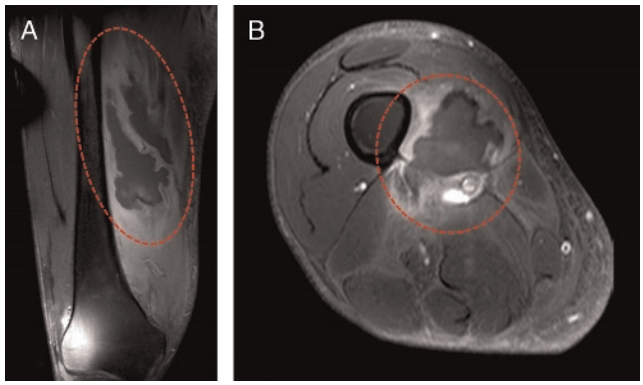
DISCUSSION

Diabetic myonecrosis can occur in both types 1 and 2 DM, with mixed reports about which type is more prevalent.[1,3] The first reported DMN cases were 2 patients with DM who presented with painful, circumscribed, elongated swelling in the thigh that was initially thought to be tumors.[4] Biopsy revealed hemorrhagic necrosis surrounded by homogenous muscle fibers with regressive changes separated by wide edematous spaces.[4] A 2015 systematic review reported 126 cases published in English; 68 cases (54%) were females, and the mean age at DMN presentation in all patients was 44.6 years (range, 20–67 years).[1] Although short-term prognosis is good, long-term prognosis is poor because of underlying microvascular disease with a 2-year mortality of 10% and recurrence in approximately half of patients.[2,5] Of note, recurrence is typically at a different site.

The exact etiology of DMN is unknown.[1] However, it is thought to be associated with vasculopathy such as diabetic microangiopathy and atherosclerosis.[1] Arteriosclerosis obliterans is another possible contributor suggested by Chester and Banker.[6] Umpierrez et al[7] suggested vasculitis phenomenon associated with fibrinoid necrosis in these patients. Other theories include alteration in the coagulation fibrinolysis system.[8] Gargiulo et al[9] and Palmer and Greco[10] suggested antiphospholipid antibodies as causative factors. Galtier-Dereure et al[11] pointed to a high association of antiphospholipid antibodies in types 1 and 2 DM patients.

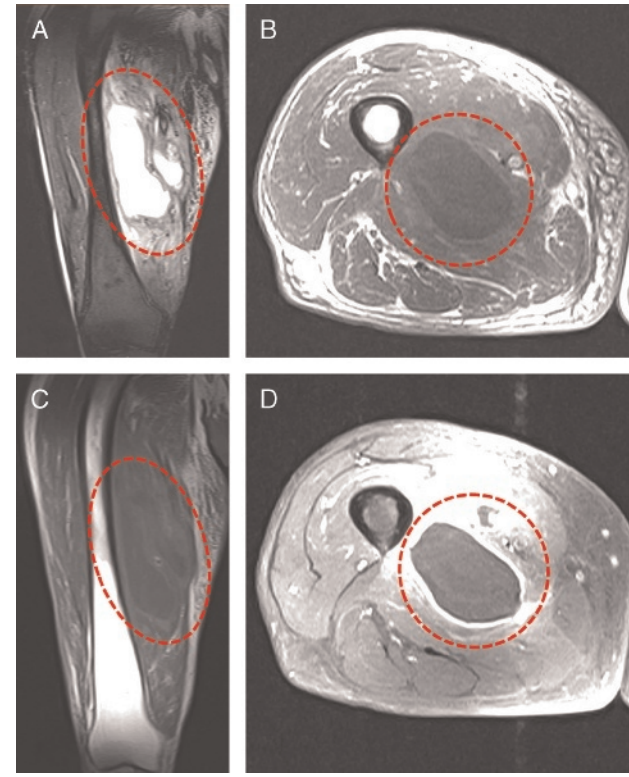
Diabetic myonecrosis usually presents as subacute to acute pain and swelling of the affected area, with or without accompanying induration and erythema. It is an atraumatic, noninfectious process, often with no systemic signs and symptoms of infection. However, an inflammatory reaction to muscle infarction may be present, and a few cases are described as having mild fever.[1,3,7] It also generally presents as unilateral muscle pain,

Figure 2.
POSTCONTRAST MAGNETIC RESONANCE IMAGING OF THE RIGHT THIGH



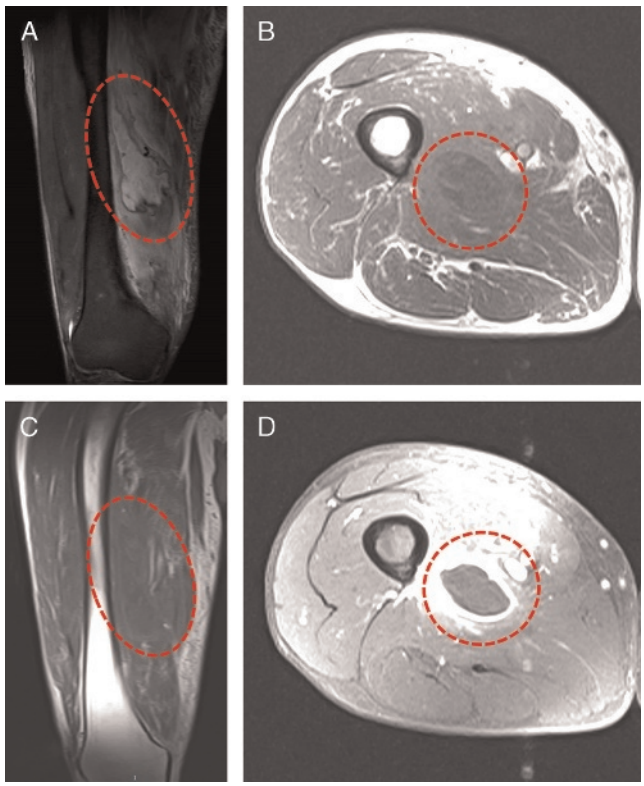
T1 fat-saturated coronal view (A) and cross-sectional view (B) with red outline illustrating the areas of fluid collection in the vastus medialis.

Figure 3.
MAGNETIC RESONANCE IMAGING OF THE RIGHT THIGH



T2 fat-saturated coronal view (A), T1 axial view (B), T1 coronal view (C), and T1 contrast fat-saturated cross-sectional view (D) with red outline illustrating the areas of resolving fluid collections in the vastus medialis.

Figure 4.
MAGNETIC RESONANCE IMAGING OF THE RIGHT THIGH



T2 fat saturated (A), T1 axial (B), T1 coronal (C), and contrast T1 fat-saturated (D) imaging with red outline illustrating the areas of fluid collection in the vastus medialis.

although bilateral involvement can occur.[3] The anterior thigh is the most commonly affected location (55%), followed by the calf and posterior thigh (15.3%), anterior upper extremities (5%), and posterior forearm (<5%). Within the thigh, the most commonly affected muscle groups are the quadriceps (65%), hip adductors (13%), hamstrings (8%), and hip flexors (2%).[1]

Radiographically, MRI, CT, and ultrasound are useful tools for diagnosis of DMN, and MRI is the test of choice.[1] Most often, MRI findings include loss of intermuscular septae, especially on T1-weighted images. T1-weighted images are characterized by tissues with high fat content appearing bright and fluid appearing dark. In DMN, T2 images show diffuse enlargement of involved muscle, subcutaneous edema, and hyperintense muscle. On T2-weighted MRI images, fluid-filled compartments appear bright, and tissues with high fat content appear dark.[2,12]

Ultrasound and CT can assist in differentiating between diagnoses that may present in a similar fashion. Ultrasound can be used to diagnose intramuscular hematoma, venous thrombosis, and intramuscular abscess.[13] A CT scan can identify localized abscess, tumor, and bone destruction and may demonstrate muscle swelling.[14] Ultrasound in DMN usually has nonspecific findings of hypoechoic regions (regions less reflective of sound waves that appear darker than neighboring structures). Internal linear structures (lines) coursing through these regions represent muscle fibers, and there is an absence of fluid motion with pressure from the transducer.[15] In contrast, abscesses on ultrasound are typically seen as anechoic (regions that do not reflect sound waves at all and appear black on ultrasound) or hypoechoic, with posterior acoustic enhancement (regions that appear lighter than surrounding structures and are usually present behind a structure that does not transmit sound waves), and possibly have intrinsic motion of fluid with pressure from the transducer.[15]

Patients with DMN typically have a history of uncontrolled DM and present with pain but do not usually have signs or symptoms of sepsis. However, in DMN, imaging will not reveal a well-circumscribed fluid collection as with an abscess.

Laboratory values, such as white blood cell count, creatinine phosphokinase, and inflammatory markers (eg, erythrocyte sedimentation rate, C-reactive protein), are considered nonspecific as they can range from normal to elevated in DMN patients.[1] One review included a discussion of laboratory values obtained for DMN patients and noted that inflammatory markers were elevated in most cases but found no consistency in diagnostic usefulness.[1]

Muscle biopsy is reserved for atypical patients with an unclear diagnosis after radiologic imaging. The main pathologic findings of biopsies are muscle necrosis and edema.[1] Biopsies are usually avoided because of the risk of infection and poor wound healing.[1] When a biopsy is performed, MRI guidance should be used for its ability to localize the lesion.[12]

Management options for DMN include a conservative approach with bed rest, antiplatelet therapy, and nonsteroidal anti-inflammatory drugs (NSAIDs),[16] which counteract the endothelial damage, platelet-associated imbalance, and inflammatory reactions that create the prothrombotic state of DMN.[1] However, tight glycemic control is the mainstay of the treatment.[17] In contrast with the aggressive management of cellulitis and necrotizing soft tissue infections, early recognition of DMN avoids antibiotic use in the absence of superimposed infections, surgery, and biopsy.[1,16] Surgery and biopsy prolong recovery time, whereas NSAIDs decrease recurrence.[1] The recurrence risk associated with surgery is 50%, and the mean time to resolution with biopsy is 60.8 days compared with 29.5 days without biopsy.[1]

CONCLUSIONS

Diabetic myonecrosis is an uncommon complication of poorly controlled, long-standing DM that can present in a similar

fashion to many other diagnoses such as cellulitis, abscess, DVT, hematoma, pyomyositis, fasciitis, and malignancy. Therefore, a high index of suspicion is required by providers for prompt and accurate diagnosis. Magnetic resonance imaging is the diagnostic test of choice, and treatment includes bed rest, antiplatelet therapy, NSAIDs, and, most importantly, tight glycemic control. As illustrated in the case presentation, early diagnosis can help prevent unnecessary surgical intervention. Diagnostic acuity can also eliminate unwarranted use of antibiotics. Future studies, possibly using animal-based models, are needed to further delineate the exact cause of DMN in order to improve awareness and prevention.

PRACTICE PEARLS

- Diabetic myonecrosis is an uncommon complication of poorly controlled DM. The etiology is unknown but is thought to be associated with vasculopathy.
- A high index of suspicion is required because DMN presents in a similar fashion to cellulitis, abscess, DVT, and hematoma.
- Magnetic resonance imaging is the diagnostic test of choice.
- Treatment includes bed rest, antiplatelet therapy, NSAIDs, and glycemic control.
- Antibiotics and surgical intervention are not indicated as treatment options. Operative management has been shown to double the time to healing.

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