Diabetic Myonecrosis: Likely an Underrecognized Entity

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abstract

Diabetic myonecrosis is a rare complication of long-standing diabetes mellitus that presents as acute onset of swelling and pain of the affected muscles. The differential diagnosis includes cellulitis/pyomyositis, necrotizing fasciitis, neoplasm, and deep venous thrombosis (DVT). Missed diagnoses can lead to unnecessary invasive diagnostic procedures and inappropriate treatment. The diagnosis is established by the clinical presentation and findings on magnetic resonance imaging (MRI) scan. A 30-year-old African-American man presented with a painful mass affecting the medial aspect of the right thigh for several months. Initial laboratory studies showed white blood cell count of 8800 cells/mm$^3$, D-dimer value of 0.55 µg/mL, HgbA1c level of 15.1%, glucose level of 352 mg/dL, erythrocyte sedimentation rate of 22 mm/h, and C-reactive protein level of 222 mg/L. An MRI scan was obtained, and diabetic myonecrosis was diagnosed and treated. One year later, the patient had similar symptoms of pain in the contralateral thigh. Repeat workup and MRI scan were obtained. The MRI abnormalities originally seen in the right thigh 1 year earlier were present in the left thigh, with complete resolution of the abnormalities seen in the right thigh. Treatment with bed rest and analgesics resulted in symptom resolution. Patients with diabetic myonecrosis typically have no fever, normal white blood cell count, mildly increased erythrocyte sedimentation rate, and elevated C-reactive protein level in 50% of cases. They lack the radiologic signs of fascial enhancement or well-defined, rim-enhancing collections that are seen in necrotizing fasciitis and pyomyositis/abscess. The onset of severe pain and the lack of mass effect on imaging differentiate diabetic myonecrosis from tumor-like conditions such as vascular malformations or soft tissue tumors. Normal D-dimer levels and ultrasound Doppler examination of the extremity help to rule out DVT. The typical MRI scan findings and clinical presentation can lead to the diagnosis of diabetic myonecrosis, allowing the physician to avoid invasive tests, such as muscle biopsy, and to reassure patients that this condition is self-limiting with appropriate treatment.
Diabetic myonecrosis is a rare complication of long-standing diabetes mellitus that commonly presents as acute onset of swelling and pain in the affected muscle. The differential diagnosis includes conditions such as cellulitis/pyomyositis, necrotizing fasciitis, neoplasm, and deep venous thrombosis (DVT). The diagnosis can be confidently established based on clinical presentation and magnetic resonance imaging (MRI) scan findings. The authors report a case of diabetic muscle infarction and review its etiology, pathogenesis, clinical presentation, and imaging findings.

**Case Report**

A 30-year-old African-American man presented to the orthopedic clinic with a painful mass affecting the medial aspect of the right thigh. On 3 occasions in the month before presentation he went to the emergency room because of this pain. Computed tomography scan performed in the emergency room showed a mass involving the medial aspect of the thigh, and a referral to orthopedics was made.

The patient reported intermittent pain in the thigh for several months, but did not notice the mass until approximately 4 to 5 weeks before presentation, when the pain became more severe. The patient reported losing approximately 15 to 20 lb over 6 months and also had intermittent bilateral pedal and ankle swelling. He had no specific history of trauma, injury, surgery, or injection in the area. He had no gastrointestinal problems, fever, nausea, vomiting, or diarrhea. The patient had type 1 diabetes mellitus, but denied any syndrome or previous history of a neoplastic process.

Physical examination of the lower extremities showed normal range of motion of the hips and knees, except for tightness in the adductor, or medial thigh, region that limited abduction of the right hip. The patient had mild peripheral neuropathy, but no focal neurovascular deficits.

An MRI scan with contrast of the right thigh showed diffuse T1 isointense to hypointense and T2 hyperintense signal within the adductor magnus muscle and minor involvement of the gracilis and adductor longus muscles (Figure 1). Overlying perimuscular fascial edema was also seen, without significant subcutaneous edema. Postcontrast images showed diffuse muscular enhancement with small focal nonenhancing foci, likely representing areas of necrosis. No fascial enhancement or marrow signal abnormality was noted. Initial laboratory tests showed white blood cell count of 8800 cells/mm$^3$, D-dimer level of 0.55 µg/mL, HgBA1c level of 15.1%, glucose level of 352 mg/dL, erythrocyte sedimentation rate (ESR) of 22 mm/h, and C-reactive protein (CRP) level of 222 mg/L.

A diagnosis of diabetic myonecrosis was made, based on the clinical presentation and imaging findings. The patient was treated with bed rest and analgesics, and symptoms resolved several weeks later.

Approximately 1 year later, the patient presented with similar symptoms of pain in the contralateral thigh. A similar workup was performed, and repeat MRI scan was obtained (Figure 2). The MRI findings in the left thigh were similar to those originally seen in the right thigh 1 year earlier. The patient had complete resolution of the signal abnormalities seen in the right thigh 1 year previously. He was treated with bed rest and analgesics, and the clinical signs resolved.

**Discussion**

Diabetic myonecrosis, or diabetic muscle infarction, is a rare sequela in patients with poorly controlled, long-standing diabetes mellitus. It was first described by Angervall and Stener in 1965 as tumoriform focal muscular degeneration. The exact pathogenesis of diabetic muscle infarction is not clear, but it is believed to be caused by vascular occlusion from mechanisms such as arteriosclerosis obliterans, embolization of arteriosclerotic plaques, thrombosis related to an altered coagulation-fibrinolysis system, or antiphospholipid antibodies.
At least 63 cases of diabetic muscle necrosis or infarction have been reported in the literature; however, the actual incidence may be underreported because of difficulty in diagnosis. At presentation, patients generally have other associated microvascular complications of diabetes, such as neuropathy, nephropathy, or retinopathy. They usually present with acute onset of severe pain in the thigh or leg, with or without a palpable mass. Involvement is most commonly unilateral, but can be bilateral. The thigh muscles (especially the quadriceps) are the most commonly affected regions, followed by the calf muscles (seen in only 20% of patients) and rarely (1%) the upper-limb musculature. Patients are often afebrile, without systemic signs of infection. On examination, the affected portion of the extremity is swollen and tender. Occasionally, a firm mass is felt. Laboratory tests are helpful in the differential diagnosis. Patients with diabetic myonecrosis typically have a normal total white blood cell count, with mildly increased ESR and elevated CRP level in 50% of cases.

Typical of patients with diabetic myonecrosis, the current patient’s physical examination findings included firm, tender swelling over the affected area, without erythema or cellulitis. Like other patients with diabetic myonecrosis, the patient was afebrile and had a normal white blood cell count (8800 cells/mm³), a mildly elevated ESR (22 mm/h), and a highly elevated CRP level (222 mg/L). The HbA1c level was 15.1% (normal, <6.0%), with glucose 300 to 400 mg/dL, indicating poor glycemic control. This patient also had the typical MRI findings of diffuse swelling and increased T2 signal intensity within the affected adductor muscles. This constellation of clinical findings suggests diabetic myonecrosis as the most likely etiology.

The differential diagnosis of diabetic muscle pain includes pyomyositis with abscess formation, necrotizing fasciitis, rhabdomyolysis, other forms of myositis, vascular malformations, and soft tissue tumors. Clinical signs of infection, such as fever, elevated white blood cell count, and radiologic signs of fascial enhancement or well-defined, rounded rim-enhancing collections, are absent in patients with diabetic myonecrosis, unlike in necrotizing fasciitis and pyomyositis/abscess. The thigh is the most common site affected in diabetic myonecrosis, whereas polymyositis involves multiple muscle groups.

Lesions of diabetic myonecrosis occasionally are mistaken for vascular malformations or soft tissue tumors. Diabetic myonecrosis is clinically characterized by sudden onset of severe pain, which differentiates it from tumor-like conditions that often have an insidious onset. Tumors also typically present as mass lesions on MRI scan, with disruption of the normal architecture, unlike the MRI findings in diabetic myonecrosis.

Although the thigh is the most common location for diabetic myonecrosis lesions, they present in the calf in 20% of patients. Therefore, it is important to differentiate its presentation from DVT. Patients with diabetic myonecrosis usually have a normal D-dimer result and negative findings on Doppler ultrasound of the extremity. These findings are elevated or positive, respectively, in DVT.

An MRI scan is typically diagnostic. The most commonly affected muscle group within the thigh is the quadriceps, followed by the hamstring and adductor muscles. The affected muscles appear isointense on T1 and hyperintense on all fluid-sensitive sequences, such as T2, short-tau inversion-recovery, and proton density fat suppression, suggesting edema and inflammation. Occasionally, areas of T1 hyperintensity, suggesting hemorrhage, are seen within the affected muscles. Gadolinium enhancement is not essential for diagnosing diabetic muscle infarction. However, it can be per-
formed when pyomyositis, which is commonly seen in diabetic patients, is a strong differential consideration. Postcontrast images show diffuse enhancement of the involved muscles, with nonenhancing necrotic areas. Diabetic muscle infarction can be confidently diagnosed in most patients on the basis of the distinctive clinical and MRI features.

Muscle biopsy is not indicated in diabetic myonecrosis and is done only in cases of atypical clinical presentation or when findings on MRI scan are highly suggestive of infection or malignancy. Histopathologic examination of the biopsy specimen during the acute phase shows areas of muscle infarction, with infiltration of polymorphonuclear cells. Small arteries within the infarcted area may have thickened hyalinized walls and may be occluded with fibrin or calcium fragments. Later, the necrotic muscle fibers undergo phagocytosis and granulation tissue repair. Eventually, necrotic muscle fibers are replaced by fibrous tissue.

Treatment of diabetic myonecrosis is largely symptomatic and includes bed rest, analgesics, physical therapy, and good control of diabetes. During the acute phase, physical activity is restricted and analgesics and anti-inflammatory medications are given. Gentle physical therapy is started once the acute phase is over. The condition is self-limiting. Pain and swelling resolve spontaneously within a few weeks. Recurrence has been reported either in the same group of muscles or in the contralateral limb. The short-term prognosis is good; however, the long-term prognosis is guarded because most patients with diabetic muscle infarction already have associated systemic end-organ complications of diabetes at presentation.

**Conclusion**

Diabetic myonecrosis is a rare complication of diabetes mellitus that can be easily missed if clinical suspicion is not present. Careful coordination between orthopedists and radiologists can play a critical role in diagnosing the condition. Typical MRI scan findings, when interpreted in light of the characteristic clinical presentation, can often lead to the diagnosis. This allows the physician to avoid invasive tests, such as muscle biopsy, and to reassure patients that the condition is self-limiting.

**References**