Diabetic Myonecrosis: Lessons in Recognizing and Treating a Rare Complication

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Abstract

Diabetic myonecrosis is a rare complication of poorly controlled diabetes that presents as spontaneous limb pain and swelling. It is associated with other microvascular diabetic complications such as nephropathy or retinopathy and is frequently misdiagnosed given its resemblance to infectious and vascular complications. We present a case of a 49-year-old male with poorly controlled type 2 diabetes and a history of recurrent thigh pain. This was initially treated as cellulitis, but the patient experienced persistent severe pain despite multiple rounds of antibiotics. Imaging with MRI ultimately confirmed a diagnosis of diabetic myonecrosis. The patient was successfully managed with a combination of aspirin, insulin therapy, and a multimodal pain regimen, leading to significant clinical improvement. This condition poses a diagnostic challenge due to its rarity and nonspecific presentation, often leading to delays in appropriate treatment. Prompt diagnosis with exclusionary testing and imaging, followed by appropriate management, can prevent severe complications. Additional research is needed to establish a standardized protocol for treating this condition.

Keywords

diabetes complications, limb pain, myopathy, microvascular disease

Introduction

Diabetic myonecrosis, also termed diabetic muscle infarction, is a commonly under-recognized cause of spontaneous lower extremity pain in patients with poorly controlled diabetes. While clinicians are aware of the macrovascular complications of longstanding diabetes, such as heart and cerebrovascular diseases, the microvascular pathogenesis of diabetic myonecrosis often mimics other infectious and musculoskeletal conditions.[1] In diabetic patients presenting with acute, severe limb swelling and muscle pain, several steps must be taken to exclude conditions including thrombosis, muscular hematoma, calciphylaxis, myositis, and necrotizing fasciitis.[2] Moreover, this is considered a relatively uncommon diagnosis, with fewer than 200 cases reported in available literature since this condition was discovered over 50 years ago.[3] Given its rare occurrence and challenging diagnosis, there are limited evidence-based treatment guidelines available for management. We present a case of a patient with severe, recurring thigh pain that was mistakenly treated as a soft tissue infection. Once diagnosed with MRI, his condition significantly improved through antiplatelet and insulin therapy, as well as a multimodal pain regimen. Our goal in presenting this case is to help readers expand their differential diagnosis for acute limb pain in diabetic patients and increase awareness of diabetic myonecrosis.

Case Presentation

A 49-year-old male with untreated type 2 diabetes and a history of cocaine use presented to the emergency department with right anteromedial thigh pain at the request of his primary care physician. He had visited the emergency department days prior with a 1-week history of severe right thigh pain, chills, nausea, and vomiting. Examination revealed a localized area of nonfluctuant swelling in the right mid-thigh along with a low-grade fever. Bedside ultrasound revealed an indeterminate echogenic lesion but no evidence of a deep venous thrombosis. Laboratory testing was deferred as it was

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Table 1. Laboratory Results During Hospitalization (Days 1-3).

Laboratory Test	Day 1	Day 2	Day 3	Reference range*
White blood cell count ($\times 10^3/mm^3$)	8.3	11.6	10.3	4.5–11
Hemoglobin (g/dL)	12.6	12.6	12.1	14–18
Platelets ($\times 10^3/mm^3$)	549	556	493	150–400
Sodium (mEq/L)	135	136	133	132–146
Potassium (mEq/L)	4.6	4.5	4.1	3.5–5.5
Chloride (mEq/L)	96	95	97	99–109
CO_2 (mEq/L)	30	28	26	20–31
Blood urea nitrogen (mg/dL)	13	15	10	9–23
Creatinine (mg/dL)	0.96	1.04	0.80	0.6–1.6
Glucose (mg/dL)	345	340	202	74–106
Creatine kinase (U/L)	572	673	1150	32–200

*Reference ranges reflect institutional laboratory values at the time of hospitalization.

Table 2. Additional Laboratory Results from Hospital Day 2.

Laboratory Test	Day 2 results	Reference range
Erythrocyte sedimentation rate (mm/h)	57	0–21
C-reactive protein (mg/dL)	13.4	0–0.5
Hemoglobin A1c (%)	14.9	4–6

Table 3. Available Laboratory Studies 5 Months Prior to Hospitalization.

Laboratory Test	Results	Reference range
Blood urea nitrogen (mg/dL)	17	9–23
Creatinine (mg/dL)	1.0	0.6–1.6
Glucose (mg/dL)	413	74–106

not expected to alter management. He was prescribed a 5-day course of oral antibiotics for a presumed soft tissue infection, though his symptoms continued to worsen.

Upon returning to the emergency department, the patient was afebrile and hemodynamically stable with inguinal lymphadenopathy, right medial thigh erythema, and significant tenderness to palpation. Bedside ultrasound during this visit demonstrated fluid tracking along the vastus medialis. For better visualization, a CT with contrast was obtained, showing possible infective myositis but no definitive intramuscular abscess. The patient received a single dose of vancomycin for empiric treatment of a soft tissue infection. Due to severe pain and inability to ambulate, he was admitted to the academic internal medicine service for further evaluation.

Laboratory studies were significant for thrombocytosis and elevated creatine kinase (Table 1). Additional labs collected on hospital day 2 showed an elevated erythrocyte sedimentation rate (ESR), c-reactive protein (CRP), and hemoglobin A1c of 14.9% (Table 2). Blood cultures showed no growth. His renal function as well as fasting glucose levels from a hospital visit 5 months earlier were also available for comparison (Table 3).

Extensive history taking revealed the patient had experienced recurrent episodes of pain affecting both thighs over the past 6 months. This included a visit to the emergency department 5 months earlier for left thigh pain without additional signs of acute illness. At that time, the physical exam noted a "muscle spasm" in the left posterior thigh, and the patient received Tylenol and a muscle relaxant. He was also previously treated with antibiotics and steroids at an outside urgent care, with the pain resolving after a few weeks. He reported discontinuing his insulin therapy a few years ago after incarceration and was lost to follow-up with his primary care physician. Given his elevated inflammatory markers and recurrent thigh pain, a contrast-enhanced MRI of the right thigh was ordered. MRI revealed heterogeneously increased signal in multiple thigh musculature in T2-weighted fat-suppressed images suggesting ischemia predominantly in the vastus medialis muscle, and to a lesser degree vastus intermedius, rectus femoris, sartorius, adductor longus, and vastus lateralis muscles. Areas of non-enhancement in the vastus medialis muscle, and to a lesser degree sartorius muscle, were consistent with myonecrosis (Figure 1A–D). Diffuse subcutaneous and intermuscular edema was appreciated along with anterolateral subfascial fluid collection, likely reactive in nature.

Antibiotics were discontinued upon confirmation with MRI. His condition improved with low-dose daily aspirin and a multimodal pain regimen. Insulin therapy was also reinitiated during his hospitalization with a weight-based basal-bolus regimen. A low-dose SGLT2 inhibitor was started on hospital day 3 after ensuring stable glucose levels and the absence of an anion gap acidosis. A prior authorization was also sent for a GLP1 agonist. The patient was discharged home, following up with the resident continuity clinic and diabetic pharmacist where he continues to undergo routine lab monitoring and titration of his antihyperglycemic regimen.

Discussion

Diabetic myonecrosis generally presents as spontaneous, subacute limb pain, and swelling in patients with poorly controlled diabetes. This often involves the anterior thigh with frequent recurrence in the contralateral muscle groups. It may also rarely affect the calves and upper extremities.[4,5] This is consistent with our patient's presentation, as he previously had a similar episode in his left anterior thigh which was misdiagnosed as cellulitis. Diagnosis is currently based on the exclusion of inflammatory myositis, infection/abscess formation, and vascular thrombosis with the clinical picture of longstanding diabetes and confirmational MRI with contrast. MRI currently serves as the most definitive diagnostic tool. T2-weighted fat-suppressed MR images show enlargement and heterogeneously increased signal in multiple muscle compartments, usually bilaterally, which represent muscle ischemia. Edema in subcutaneous tissue and intermuscular fascia is commonly

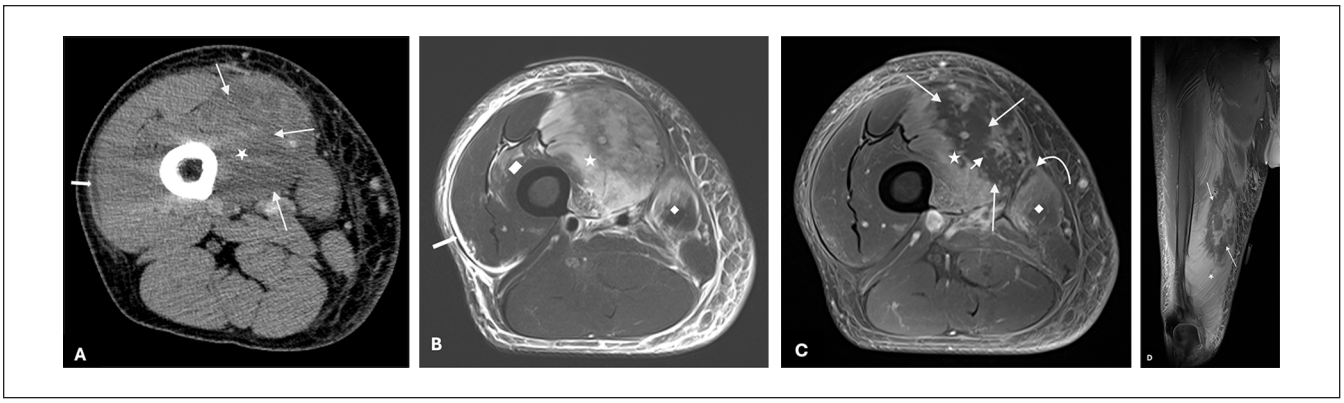

Figure 1. Diabetic myonecrosis in the right thigh. (A) Axial contrast-enhanced CT scan image of mid-thigh demonstrates enlargement of the vastus medialis muscle *(star)* with areas of non-enhancement *(thin long arrows)*. There is also a small amount of subfascial fluid collection in the anterior lateral compartment (*thick arrow*) and mild subcutaneous edema which is likely reactive. (B) Axial T2-weighted fat-suppressed MR image demonstrates heterogeneously increased signal/edema in the vastus medialis muscle *(star)* and to a lesser degree sartorius muscle *(diamond)*, vastus intermedius *(rectangle)* and other thigh muscles (not shown) which may represent muscle ischemia. Diffuse subcutaneous and intermuscular edema with anterior lateral subfascial thin fluid collection noted *(thick arrow)*. (C) Mid-thigh axial and (D) anterior thigh coronal T1-weighted fat-suppressed post-contrast MR images demonstrate diffuse swelling and enhancement in vastus medialis *(star)* and sartorius *(diamond)* with areas of non-enhancement in vastus medialis *(thin long arrows)* and sartorius (curved arrow) muscles containing some stipple enhancements (*short arrow*) consistent with myonecrosis. No sizable peripherally enhancing organized fluid collection to suggest an abscess.

seen along with subfascial fluid collection. T1-weighted imaging may show areas of increased signal due to hemorrhagic infarction. Post-contrast images usually demonstrate diffuse enhancement in the affected muscles which may suggest partial viability with areas of non-enhancement consistent with myonecrosis. Rim enhancement around necrotic muscles can be seen, especially in the subacute phase, making it difficult to differentiate from an intramuscular abscess,[6] therefore clinical and laboratory correlation is necessary. Enhanced CT scans may show a similar (but less conspicuous than MRI) pattern of enlargement and enhancement with the absence of enhancement in the necrotic areas. Attention must also be paid to the patient's diabetic nephropathy before administering IV contrast. A bedside ultrasound may reveal echogenic linear structures/muscle fibers running within the focal hypoechoic infarcted muscle area with probable adjacent hyperemia by Doppler US. It can be helpful to exclude an abscess, which would be seen as an anechoic/hypoechoic pocket of fluid with internal moving debris and posterior acoustic enhancement.[7] Biopsy is not required unless imaging remains inconclusive, which shows muscle necrosis, edema, and occlusion of capillaries.[8] Additional tests recommended include a complete blood count (CBC) with differential, partial thromboplastin time/prothrombin time (PTT/PT), creatine kinase (CK), CRP, ESR, hemoglobin A1c, blood cultures, venous doppler, and a plain radiograph of the affected limb.[9] In our patient, the progressive rise in creatine kinase, elevated inflammatory markers, negative blood cultures, and absence of leukocytosis, supported the diagnosis of diabetic myonecrosis rather than an infectious process. Patients should also be evaluated for additional microvascular diabetic complications including eye and sensory exams in the outpatient setting.

Pathogenesis for this condition is not fully understood, however, it often involves microvascular ischemia surrounding large muscle groups via fibrin-mediated occlusion. Prevalence is equal among patients with both type 1 and 2 diabetes.[10] According to a recent systematic review, an estimated 75% of cases are diagnosed in the setting of nephropathy.[11] Our case is notable as the patient maintained normal renal function both prior to and during hospitalization.

Our patient also had a history of cocaine use, a drug well known for its vasoconstrictive properties, leading to endothelial damage and platelet aggregation with regular use.[12] This mechanism, along with the underlying endothelial dysfunction associated with poorly controlled diabetes, likely contributed to the pathogenesis of diabetic myonecrosis. Given these risks, counseling patients on the importance of cocaine cessation is essential for preventing further vascular complications.

Although treatment data remains limited, a multimodal approach with insulin management, pain control, and daily aspirin is often the most effective. Given the microvascular dysfunction from uncontrolled diabetes, lifelong aspirin is advisable. Daily clopidogrel is also an acceptable antiplatelet medication if aspirin cannot be tolerated. In addition to insulin therapy, the decision was made to begin an SGLT2 inhibitor during his hospitalization. While the American Diabetes Association guidelines do not recommend routine inpatient use of this class of antihyperglycemic therapy, the patient's stable glycemic levels, absence of acidemia, and lack of acute systemic illness or hemodynamic instability supported its safe initiation.[13] With appropriate management, most cases of diabetic myonecrosis are self-limited and resolved with rest and analgesics; however, severe muscle infarction may

require surgical excision.[14] Patients also remain at high risk for other vascular complications including myocardial infarction, stroke, and peripheral artery disease, requiring close follow-up and care.

Conclusion

Evaluation and diagnosis of diabetic myonecrosis is particularly challenging given its nonspecific presentation and extensive differential diagnosis for acute limb pain in a diabetic patient. Having a strong clinical suspicion early in its presentation can expedite patient recovery, avoiding unnecessary testing and treatment. Like the microvascular insults seen in diabetic retinopathy, neuropathy, and nephropathy, diabetic muscle infarction is a concerning downstream consequence of untreated diabetes. This case highlights the substantial need for patient education on glucose control and substance use cessation in diabetes management. We hope to also encourage additional research on its management to establish standards of treatment and prevention of recurrence.

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Ethics Approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed Consent

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References

1. Banker BQ, Chester CS. Infarction of thigh muscle in the diabetic patient. *Neurology*. 1973;23(7):667-677. doi:10.1212/wnl.23.7.667
2. Rocca PV, Alloway JA, Nashel DJ. Diabetic muscular infarction. *Semin Arthritis Rheum*. 1993;22(4):280-287. doi:10.1016/0049-0172(93)80076-r
3. Angervall L, Stener B. Tumoriform focal muscular degeneration in two diabetic patients. *Diabetologia*. 1965;1(1):39-42. doi:10.1007/BF01338714
4. Jalali Z, Sharif SK. Upper-limb diabetic myonecrosis: atypical presentation of a rare complication. *Am J Case Rep*. 2019;20:1268-1272. doi:10.12659/AJCR.917030
5. Khanna HK, Stevens AC. Diabetic myonecrosis: a rare complication of diabetes mellitus mimicking deep vein thrombosis. *Am J Case Rep*. 2017;18:38-41. doi:10.12659/ajcr.900903
6. Jelinek JS, Murphey MD, Aboulafia AJ, Dussault RG, Kaplan PA, Snearly WN. Muscle infarction in patients with diabetes mellitus: MR imaging findings. *Radiology*. 1999;211(1):241-247. doi:10.1148/radiology.211.1.r99ap44241
7. Delaney-Sathy LO, Fessell DP, Jacobson JA, Hayes CW. Sonography of diabetic muscle infarction with MR imaging, CT, and pathologic correlation. *AJR Am J Roentgenol*. 2000;174(1):165-169. doi:10.2214/ajr.174.1.1740165
8. Ganokroj P, Boonchaya-Anant P. Diabetic muscle infarction: rare complication with a distinct clinical manifestation. *BMJ Case Rep*. 2019;12(4):e228480. doi:10.1136/bcr-2018-228480
9. Lawrence L, Tovar-Camargo O, Lansang MC, Makin V. Diabetic myonecrosis: a diagnostic and treatment challenge in longstanding diabetes. *Case Rep Endocrinol*. 2018;2018:1723695. doi:10.1155/2018/1723695
10. Yong TY, Khow KSF. Diabetic muscle infarction in end-stage renal disease: a scoping review on epidemiology, diagnosis and treatment. *World J Nephrol*. 2018;7(2):58-64. doi:10.5527/wjn.v7.i2.58
11. Horton WB, Taylor JS, Ragland TJ, Subauste AR. Diabetic muscle infarction: a systematic review. *BMJ Open Diabetes Res Care*. 2015;3(1):e000082. doi:10.1136/bmjdrc-2015-000082
12. Bachi K, Mani V, Jeyachandran D, Fayad ZA, Goldstein RZ, Alia-Klein N. Vascular disease in cocaine addiction. *Atherosclerosis*. 2017;262:154-162. doi:10.1016/j.atherosclerosis.2017.03.019
13. ElSayed NA, Aleppo G, Aroda VR, et al. 16. Diabetes care in the hospital: standards of care in diabetes-2023. *Diabetes Care*. 2023;46(Suppl 1):S267-S278. doi:10.2337/dc23-S016
14. Zhang D, Janssen SJ, Tarabochia M, von Keudell A. Factors associated with myonecrosis at time of fasciotomy in acute leg compartment syndrome. *Eur J Orthop Surg Traumatol*. 2020;30(6):1089-1095. doi:10.1007/s00590-020-02662-x