Diabetic Myonecrosis: A Case Report of Acute Muscle Infarction in a Patient with Long-Standing Diabetes Mellitus

Journal of Health and Rehabilitation Research (2791-156X)
Volume 4, Issue 3
Double Blind Peer Reviewed.
https://jhrlmc.com/
DOI: https://doi.org/10.61919/jhrr.v4i3.1575
www.lmi.education/
LINK MEDICAL INTERFACE

Yahya Ur Rehman[1], Nadia Hashmi[2], Rabia Umar[3], Syeda Mubeena Nahri[3]

Correspondence
Yahya Ur Rehman
yahyarehman2001@gmail.com

Affiliations
1 Liaquat University of Medical and Health Sciences, Jamshoro, Pakistan
2 Medical Officer, The Kidney Centre, Karachi, Pakistan
3 General Practitioner, Saudi German Hospital, Dubai, UAE

Keywords
Diabetic myonecrosis, diabetes complications, muscle infarction, MRI diagnosis, acute thigh pain, long-standing diabetes, diabetic muscle infarction, microvascular complications.

Disclaimers

Authors' Contributions	All authors contributed equally to the study's conception, data collection, and manuscript preparation.
Conflict of Interest	None declared
Data/supplements	Available on request.
Funding	None
Ethical Approval	Respective Ethical Review Board
Study Registration	N/A
Acknowledgments	N/A

ABSTRACT

Background: Diabetic myonecrosis is a rare complication of long-standing, poorly controlled diabetes mellitus, often associated with microvascular complications and diagnostic challenges. Early recognition and appropriate management are crucial to avoid unnecessary interventions and optimize patient outcomes.

Objective: To report a case of diabetic myonecrosis in a patient with poorly controlled diabetes and highlight its diagnostic approach and management.

Methods: A 49-year-old male with a 30-year history of uncontrolled type 1 diabetes presented with acute pain and swelling in the right thigh. Laboratory investigations included complete blood count, ESR, CRP, and HbA1c. Imaging modalities such as computed tomography (CT) and magnetic resonance imaging (MRI) were used to evaluate muscle involvement. Conservative management included optimized insulin therapy, aspirin, and analgesics.

Results: Laboratory findings showed elevated ESR (121 mm/hr), CRP (3.98 mg/dL), and HbA1c (15.2%). MRI confirmed diffuse muscle edema in the right adductor muscles. Following treatment, pain severity decreased from 8/10 to 2/10, and mobility significantly improved over a two-week period.

Conclusion: Early diagnosis and conservative management of diabetic myonecrosis can prevent complications. Clinicians should consider this condition in patients with acute musculoskeletal pain and poorly controlled diabetes.

INTRODUCTION

Diabetic muscle infarction (DMI), also referred to as diabetic myonecrosis, is a rare but serious complication that predominantly occurs in patients with long-standing, poorly controlled diabetes mellitus. Although diabetic complications such as nephropathy, neuropathy, and retinopathy are widely recognized, diabetic myonecrosis remains underdiagnosed, often leading to significant diagnostic delays (1, 2). First described in 1965 by Angervall and Stener, this condition is typically associated with advanced microvascular complications in individuals with type 1 diabetes mellitus, although it has also been observed in patients with type 2 diabetes (1). The pathophysiology of DMI is not yet fully understood, but it is thought to involve microangiopathy leading to compromised perfusion of the muscle tissue, ischemia, and subsequent infarction. This ischemic process may be exacerbated by hypoxia-reperfusion injury, which triggers a cascade of inflammatory responses, resulting in muscle necrosis and edema (2, 3). Clinically, patients present with acute-onset, severe muscle pain and swelling, most commonly affecting the lower extremities, particularly the thigh and calf muscles. These manifestations often mimic other more common conditions such as cellulitis, pyomyositis, deep vein thrombosis, or compartment syndrome, further complicating the diagnostic process (3, 4).

Magnetic resonance imaging (MRI) is the preferred diagnostic modality due to its high sensitivity in identifying muscle edema and infarction, which appear as hyperintense signals on T2-weighted images and hypointense or isointense signals on T1-weighted images (4). In contrast, computed tomography (CT) scans are less sensitive, and routine laboratory investigations, including creatine kinase (CK) levels, may not consistently indicate muscle injury (5). Muscle biopsy, while considered the gold standard for diagnosis, is typically avoided due to its invasive nature and the risk of poor wound healing in diabetic patients (5, 6). Due to these diagnostic challenges, diabetic myonecrosis is often diagnosed late, leading to inappropriate or delayed treatment. Early recognition is crucial, as unnecessary interventions such as surgical debridement, typically indicated in conditions like necrotizing fasciitis, may exacerbate tissue damage and worsen outcomes in patients with DMI (4, 6).

Management of diabetic myonecrosis is primarily conservative, focusing on optimizing glycemic control, providing adequate pain management, and advising rest to reduce the mechanical stress on the affected muscles (6). Strict blood glucose management is essential, as persistent hyperglycemia is a key factor in the pathogenesis of diabetic microvascular complications. In the acute phase, non-steroidal anti-inflammatory drugs (NSAIDs) or opioids may be required for effective pain relief. Although no standardized treatment protocol exists due to the rarity of

the condition, some evidence suggests that antiplatelet agents such as aspirin may be beneficial in preventing further microvascular events (5). Prognosis is generally favorable with conservative management, but recurrence is not uncommon, especially in patients with suboptimal glycemic control and persistent microvascular complications (6).

This report aims to highlight a case of diabetic myonecrosis in a 49-year-old male with a long-standing history of uncontrolled diabetes mellitus, presenting with acute thigh pain and swelling. The patient's presentation, diagnostic workup, and management are discussed, emphasizing the need for increased clinical awareness of this rare but serious complication to facilitate early diagnosis and appropriate treatment strategies. Comprehensive management, including stringent glycemic control and adequate pain management, remains the cornerstone of therapy for preventing recurrent episodes and improving patient outcomes (6). Further research is needed to better understand the pathophysiological mechanisms underlying diabetic myonecrosis and to develop targeted treatment strategies aimed at reducing morbidity in this vulnerable patient population.

MATERIAL AND METHODS

The patient was a 49-year-old male with a 30-year history of type 1 diabetes mellitus, who presented to the hospital with progressively worsening pain and swelling in his right thigh over the past few days. Upon admission, a comprehensive clinical assessment was conducted, including a detailed history and physical examination. Laboratory investigations were performed, including a complete blood count, renal function tests, glycated hemoglobin (HbA1c), and inflammatory markers such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). These tests were aimed at evaluating the patient's overall condition and ruling out potential infectious or inflammatory etiologies. Imaging studies were also conducted to aid in diagnosis. A computed tomography (CT) scan of the affected thigh was initially obtained to exclude conditions such as necrotizing fasciitis or pyomyositis. Subsequently, magnetic resonance imaging (MRI) was performed to assess the extent of muscle involvement and to identify characteristic findings consistent with diabetic myonecrosis, including muscle edema and infarction (3, 4).

The clinical diagnosis was made based on the patient's history, laboratory results, and MRI findings, which demonstrated diffuse edema in the right adductor muscles, predominantly affecting the adductor magnus, without evidence of abscess formation or hematoma. Biopsy was considered but ultimately not performed due to the invasive nature of the procedure and the risk of delayed wound healing in this diabetic patient. The diagnosis was supported by the exclusion of other possible conditions such as deep vein thrombosis, cellulitis, and compartment syndrome through clinical evaluation and imaging studies (5).

The patient provided written informed consent for the diagnostic workup and subsequent treatment in accordance with the ethical guidelines outlined in the Declaration of Helsinki. The patient's confidentiality was strictly maintained throughout the study, and no personal identifiers were disclosed. Management was initiated with a conservative approach, focusing on optimizing glycemic control and providing adequate pain relief. The patient was started on a tailored insulin regimen, including rapid-acting insulin before meals and long-acting basal insulin at night, to improve glycemic control. Analgesic therapy consisted of nonsteroidal anti-inflammatory drugs (NSAIDs) and opioid-based medication as needed for severe pain. Aspirin 81 mg daily was also administered for cardiovascular protection, given the patient's history of long-standing diabetes and increased risk of microvascular complications (6).

Data from the patient's clinical presentation, laboratory investigations, imaging findings, and therapeutic response were compiled and analyzed to support the case description. Descriptive statistics were used to summarize the patient's laboratory and imaging results. All clinical data were anonymized before analysis. Data analysis was conducted using SPSS version 25. The primary aim was to document the clinical presentation, diagnostic challenges, and management strategies for diabetic myonecrosis in a patient with poorly controlled, long-standing diabetes mellitus. Ethical approval was sought and obtained from the institutional review board before commencing the study. As this was a single case report, a formal power calculation and sample size determination were not applicable. The findings were reviewed in the context of existing literature to provide insights into the pathophysiology, clinical course, and management of this rare complication of diabetes (2, 4).

RESULTS

The patient presented with acute pain and swelling in the right thigh, which progressively worsened over a few days.

Table 1. Baseline Laboratory Investigations of the Patient

Parameter	Value	Reference Range
Hemoglobin (Hb)	12.5 g/dl	13.0 – 17.5 g/dl
White Blood Cell Count (WBC)	12,000 cells/μL	4,000 – 11,000 cells/μL
Hematocrit	40%	38% – 50%
Platelet Count	320,000/μL	150,000 – 450,000/μL
Neutrophils	9,360 cells/μL	1,500 – 8,000 cells/μL
Lymphocytes	2,160 cells/μL	1,000 – 4,800 cells/μL
Erythrocyte Sedimentation Rate (ESR)	121 mm/hr	<20 mm/hr
C-Reactive Protein (CRP)	3.98 mg/dL	<0.5 mg/dL
HbA1c	15.2%	<6.5%

Laboratory investigations revealed significant elevations in markers of inflammation and poor glycemic control, as shown in Table 1. His HbA1c was notably elevated at 15.2%, reflecting long-term poor glycemic management. Additionally, inflammatory markers such as ESR and CRP were markedly elevated, indicating an active inflammatory process.

The patient's imaging studies supported the diagnosis of diabetic myonecrosis. A computed tomography (CT) scan of the right thigh ruled out abscess formation and necrotizing fasciitis but showed diffuse swelling. Magnetic resonance imaging (MRI) findings confirmed extensive edema in the right adductor muscles, predominantly affecting the adductor magnus, with no evidence of hematoma or focal abscess (Figure 1).

Table 2. Imaging Findings

Imaging Modality	Findings
CT scan	Diffuse swelling in the right thigh, no abscess formation or necrotizing fasciitis.
MRI	Diffuse edema in the adductor muscles (adductor magnus), no hematoma or abscess formation. MRI showed hyperintense signal on T2-weighted images and hypointense signal on T1-weighted images, consistent with muscle infarction.

The patient was managed conservatively with a focus on improving glycemic control and providing adequate pain management. His insulin regimen was optimized, and analgesic therapy, including NSAIDs and opioids, was initiated to manage pain. Aspirin was added for cardiovascular protection. Over a two-week follow-up period, the patient reported significant improvement in pain and mobility, as shown in Table 3.

Table 3. Clinical Outcome at Follow-Up

Parameter	Baseline	Follow-Up
Pain (Numeric Pain Rating Scale)	8/10	2/10
Swelling (on physical examination)	Moderate	Mild
Mobility	Restricted	Improved
Glycemic Control (HbA1c)	15.2%	12.8%

Overall, the patient's clinical symptoms showed marked improvement with conservative management, although strict glycemic control remained challenging. The final outcome emphasized the importance of early recognition and non-invasive management of diabetic myonecrosis to prevent complications and recurrence.

DISCUSSION

Diabetic myonecrosis is a rare yet serious complication predominantly observed in patients with long-standing, poorly controlled diabetes mellitus. The condition often remains underrecognized due to its nonspecific presentation and overlap with other musculoskeletal disorders, such as cellulitis, pyomyositis, and deep vein thrombosis. The present case report aligns with previous studies that have highlighted the diagnostic challenges posed by diabetic myonecrosis and the tendency for delayed recognition (2, 4). Similar to findings reported by Iyer et al., the patient in this study presented with severe pain and swelling in the thigh, a common manifestation of this condition, which led to initial diagnostic considerations such as necrotizing fasciitis or abscess formation (5). This case reaffirms the need for heightened clinical suspicion in patients with a history of long-standing diabetes and microvascular complications when they present with acute musculoskeletal pain.

Magnetic resonance imaging (MRI) played a pivotal role in confirming the diagnosis, consistent with the observations made by Goswami and Baruah, who emphasized the utility of MRI in visualizing muscle infarction and differentiating it from other similar conditions (3). The characteristic findings of muscle edema on T2-weighted images and hypointense signals on T1-weighted images, along with the absence of hematoma or abscess, were critical in establishing the diagnosis. Although muscle biopsy is considered the gold standard, it was not performed in this case due to the risks associated with invasive procedures in diabetic patients, such as delayed wound healing and potential for secondary infections (6). This approach aligns with the recommendations from previous literature that advocate for non-invasive diagnostic methods whenever possible to minimize patient morbidity (5, 6).

The pathophysiology of diabetic myonecrosis remains unclear, but microangiopathy leading to ischemia and subsequent infarction is widely accepted as the primary mechanism. Additionally, ischemia-reperfusion injury may contribute to the inflammatory response and muscle necrosis, as suggested by Choudhury et al. (4). The present case supports these hypotheses, given the patient's significant history of microvascular complications, including diabetic nephropathy and retinopathy, which are common in cases of diabetic myonecrosis. Strengths of this report include the use of comprehensive imaging modalities to establish the diagnosis and the clear documentation of the clinical course and response to conservative management. However, the study has limitations, including the absence of histopathological confirmation, which, while not essential, could have further substantiated the diagnosis. Furthermore, the inability to achieve optimal glycemic control during the follow-up period underscores the challenge of managing such complex cases in resource-limited settings.

Conservative management remains the cornerstone of treatment for diabetic myonecrosis, focusing on optimizing

glycemic control, providing pain relief, and advising rest to promote muscle recovery. This is consistent with existing literature, which recommends avoiding aggressive surgical interventions that are more suited to other conditions like necrotizing fasciitis (4). In this patient, significant clinical improvement was achieved with tailored insulin therapy, cardiovascular protection with aspirin, and pain management using NSAIDs and opioids, as evidenced by the marked reduction in pain and improved mobility at follow-up. However, as highlighted in a review by Horton et al., recurrence is a significant concern, particularly in patients with suboptimal glycemic control (2). Thus, stringent monitoring and long-term management strategies, including patient education on the importance of adherence to insulin therapy, are recommended to prevent future episodes.

The case also underscores the importance of multidisciplinary management, involving endocrinologists, radiologists, and primary care physicians to ensure early diagnosis and appropriate treatment. Recommendations for future research include larger, multi-center studies to better define the epidemiology, pathophysiology, and optimal management strategies for diabetic myonecrosis. Additionally, studies exploring the role of novel therapeutic interventions, such as antiplatelet or anti-inflammatory agents, in preventing recurrence are warranted. In conclusion, diabetic myonecrosis should be considered in patients with long-standing diabetes presenting with acute muscle pain, and a high index of suspicion, combined with appropriate imaging, is essential for timely diagnosis and effective management.

CONCLUSION

Diabetic myonecrosis is a rare but critical complication in patients with long-standing, poorly controlled diabetes mellitus, often leading to significant diagnostic delays due to its nonspecific presentation and overlap with other musculoskeletal conditions. Early recognition, accurate diagnosis through non-invasive imaging modalities, and conservative management focusing on glycemic control and pain relief are essential to prevent complications and optimize outcomes. From a broader healthcare perspective, this case highlights the need for heightened clinical awareness and a multidisciplinary approach to managing complex diabetes complications, reinforcing the importance of comprehensive diabetic care and patient education to prevent the onset of such debilitating conditions.

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