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Case Report

Diabetic myonecrosis: a rare complication of poorly controlled diabetes

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ABSTRACT

Diabetic myonecrosis is a rare complication of poorly controlled diabetes that can be overlooked. This case report describes a 58-year-old man with type 2 diabetes who presented with severe pain and swelling in his right anterolateral thigh for one month. Despite antibiotics, his symptoms persisted. Laboratory tests revealed a normal white blood cell count, elevated blood glucose levels, an elevated erythrocyte sedimentation rate, and elevated C-reactive protein levels. Magnetic resonance imaging revealed diffuse oedema in the anterior thigh muscles, strongly suggesting muscle necrosis. The patient was diagnosed with diabetic myonecrosis based on clinical presentation, basic investigations and imaging findings. He was managed with rest, pain control, and improved glycemic control. This case highlights the importance of considering diabetic myonecrosis in patients with poorly controlled diabetes who present with acute muscle pain and swelling. Early recognition and appropriate management are crucial for avoiding unnecessary interventions and improving outcomes. Increased awareness of this complication is needed among clinicians to ensure prompt diagnosis and treatment.

Keywords: Diabetic myonecrosis, Diabetes complications, MRI diagnosis, conservative management

INTRODUCTION

Diabetic myonecrosis (DMN), also known as diabetic muscle infarction, is a rare but important microvascular complication of diabetes mellitus. First described by Angervall et al in 1965, DMN typically occurs in patients with long-standing, poorly controlled diabetes.[1] It predominantly affects women, with an average age of 42.5, and involves the large muscles of the lower limbs, particularly the quadriceps, adductors, and hamstrings.[2,3]

The affected muscle feels hard and tender, making diagnosis difficult, as it may be mistaken for other conditions, such as deep vein thrombosis, cellulitis, myositis, haematoma, or a ruptured muscle.

The underlying cause of diabetic myonecrosis is unclear, but it appears to be associated with hypoxia reperfusion injury, atherosclerotic occlusion, or vasculitis with thrombotic vascular changes in the muscle of long-standing, poorly controlled diabetic patients.[4]

It is often underdiagnosed, with unknown prevalence. Diagnosis poses a challenge due to lack of definitive criteria and relies on pattern recognition, confirmed by MRI with appropriate clinical and laboratory findings. For clinicians treating diabetes, early recognition prevents unnecessary investigations and improves patient outcomes. While basic understanding exists, gaps remain in disease comprehension and optimal diagnostic and treatment approaches.

We analyzed the current management of diabetic myonecrosis. This case highlights the clinical relevance of this condition in patients with long-standing diabetes and poor glucose control.

CASE REPORT

A 58-year-old male patient, who was diagnosed with type 2 diabetes four years ago and initiated insulin therapy one year ago due to inadequate glycemic control, presented with severe, progressively worsening pain in the right anterolateral aspect of his thigh, persisting for one month. Walking exacerbated the pain, which was alleviated upon rest. The patient had no history of ischemic heart disease or stroke. He reported no symptoms of weight loss, altered vision, frothy urine, or gustatory sweating but did complain of stocking-type sensory loss extending up to his ankle. The patient's home blood sugar levels occasionally ranged from 250 to 300 mg/dl. Two months' prior, the patient experienced bilateral lower limb cellulitis, which had resolved completely. Eight years ago, he underwent a left nephrectomy for a renal tumour. Currently, the patient requires 35 U of mixed insulin daily, 25 U of nocte daily, and 200 mg of pazopanib for his renal cell carcinoma. He is an entrepreneur and a non-smoker and has occasionally consumed alcohol since the age of 30. He had no other known comorbidities.

A week before the presentation, the patient's ultrasound scan had revealed myositis, and he was prescribed antibiotics by a general practitioner. However, as his symptoms did not improve, he was subsequently hospitalised.

General medical examination

The patient presented with a blood pressure reading of 130/90 mmHg and a pulse rate of 96 beats per minute. He was found to be afebrile. Upon physical examination, a swelling was observed on the right lateral aspect of the patient's thigh.

The thigh was then examined in greater detail, revealing an elongated swelling with faint redness but no signs of ulceration, bruising, or ill-defined contours. The swelling was located 7 cm above the right knee joint, measured 6 cm by 11 cm, and was oval. It was firm, faintly red, tender, warm to the touch, and attached to the muscle. There were no fluctuations evident in the mass. Despite the presence of the swelling, the patient was able to walk independently but had an antalgic gait. The muscle tone was normal, but the range of movement in the right hip and knee joints was limited, and power on the affected side was difficult to assess. The reflexes were intact.

Investigations

The complete blood count (CBC), fasting plasma glucose (FPG), HbA1c and Biochemical parameters are shown in Table 1.

Table 1: CBC, FPG, HbA1c and biochemical parameters.

Variables	Values
Fasting blood glucose / mg/dl	193
White blood cells / $*10^9$/l	6.7
Red blood cells / $*10^{12}$/l	5.1
Hemoglobin / g/l	163
Hematocrit (%)	48
HbA1c (%)	10.5
ESR / mm/h	80
C-reactive protein / mg/dl	19
Creatinine / mg/dl	0.8
Creatine phosphokinase / U/l	85
Sodium / mmol/l	136
Potassium / mmol/l	4.3
Albumin / g/dl	3.3

Imaging studies

Abdominal and pelvic ultrasound examinations revealed a slightly enlarged right kidney with increased cortical echogenicity. Additionally, an ultrasound scan of the right thigh indicated potential myositis affecting the vastus muscles, along with edema in the perimyosial connective tissue, subcutaneous tissue, and dermis.

The MRI scan showed widespread muscle oedema in the lateral, intermediate, and medial rectus tertiaries status muscles, extending from the proximal thigh to the knee level. Additionally, there is subcutaneous fat oedema but no subfascial or intramedullary collections. The non-enhancing focal area in the distal portion of the vastus lateralis, measuring 4.2 × 2.5 × 2.6 cm, suggested muscle necrosis with intermediate signals in TW and no peripheral enhancement.

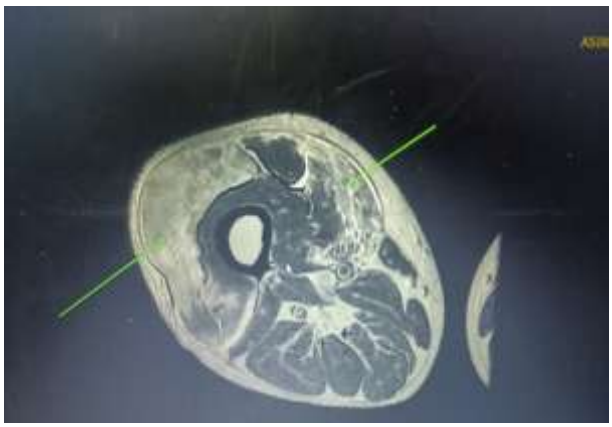

Figure 1: Presents an axial T2-weighted image, utilizing a proton density (PD) fat-saturated MRI sequence. The image reveals diffuse muscle oedema in the vastus lateralis, vastus medialis, and sartorius muscles, extending from the proximal thigh to the knee level. Arrows indicate the areas of necrosis.

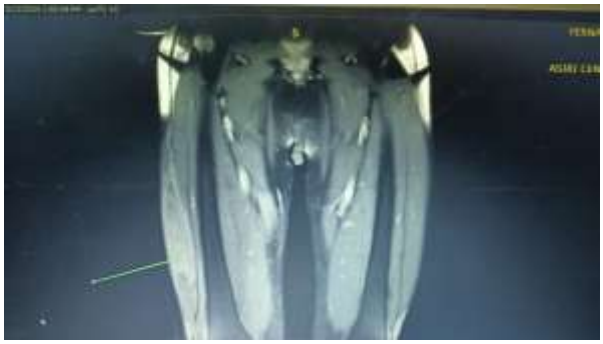

Figure 2: Coronal T2-weighted MRI showing focal necrosis within the vastus lateralis.

Management and outcome

The patient received treatment consisting of bed rest, analgesics, and stringent glycaemic control. Antibiotic therapy was discontinued after the infection was excluded. Physiotherapy was not administered during the acute phase. The patient's pain progressively improved over a period of five days, leading to discharge. A follow-up ultrasound conducted four weeks later indicated partial resolution without the presence of an abscess. The patient remained asymptomatic after three months, with no recurrence observed.

DISCUSSION

DMI, also referred to as diabetic myonecrosis, is a rare complication of diabetes that was initially described in 1965. It is usually associated with poor control and microvascular disease.[4] Both type 1 and type 2 diabetes mellitus patients can develop DMI. The occurrence of this disease is higher in females (54%) compared to males.[4] Although DMI has a favourable short-term prognosis, recurrence rates are high (43.9%).[5] At the time of the patient's diagnosis, the mean HbA1c value was very high at 10.5%, which is a known risk factor for developing DMI.[3] DMI is typically observed in the advanced stages of diabetes, with 46.6% of patients having concurrent acute retinopathy, acute nephropathy, or acute neuropathy and 65% having at least two of these complications.[3] However, our patient did not have evidence of such microangiopathy-related complications

There is uncertainty regarding the exact pathophysiology of diabetic muscle infarction (DMI). While some attribute it to ischemia-reperfusion injury, vasculitis with thrombosis, atherosclerosis, or diabetic microangiopathy, the definitive cause remains undetermined.[6] Individuals with uncontrolled diabetes presenting with acute proximal leg muscle pain and swelling should be evaluated for DMI. The thighs are typically the primary pain site, although pain in the calf and upper arm occurs less frequently.[3] The pain presents as an acute, unilateral, severe ache in the muscle, with swelling and a palpable, tender area. However, alternative presentations may occur, with a subacute onset showing weakness and inability to walk, as observed in our patient.[6]

In cases of DMI, laboratory results may indicate mild to moderate leukocytosis, a slight elevation in CPK levels, and an increase in erythrocyte sedimentation rate in 8%, 48.2%, and 52.8% of cases, respectively.[4]

Laboratory tests for DMI are generally non-specific (1). In a study of 113 cases, a normal white blood cell count (WCC) was observed in the majority of instances (56.6%), as was the case with our patient. Conversely, 42.5% of cases exhibited elevated levels (1). The normal white cell count and CPK levels in our patient suggest that the likelihood of an abscess or pyomyositis is reduced.

Exclusion of mimicking conditions

Several differential diagnoses were considered and excluded: pyomyositis or abscess: The patient was afebrile with a normal white cell count. MRI showed no rim-enhancing fluid collection or gas, necrotising fasciitis: No fascial gas or systemic toxicity, deep vein thrombosis: The clinical assessment did not support this diagnosis, haematoma: no trauma or anticoagulant use; MRI showed no layering or bleeding, drug-related necrosis: pazopanib-related myopathy was considered, but the focal pattern, imaging features, and resolution with conservative management favored diabetic myonecrosis.

The ultrasound characteristics of diabetic muscle infarction (DMI) include linear structures resembling muscle fibers traversing the lesion.[4,5,7] It may show heterogeneous, mass-like echogenic changes, indicating muscle swelling from disrupted myofascial interfaces. However, these findings can be non-specific. The ultrasound features in our patient matched some typical characteristics, though they suggested possible myositis as a differential diagnosis. The features of DMI and absence of infection indicators help differentiate it from an intramuscular abscess.[4]

Rationale for not performing a tissue biopsy

MRI demonstrates high sensitivity and specificity for diagnosing DMI. T2-weighted images display bright signals and consistent T1-weighted signals from the affected muscle, with surrounding tissue swelling.[8] MRI with clinical evaluation suffices for diabetic myonecrosis diagnosis, supporting conservative treatment. Muscle biopsy is discouraged due to longer recovery (60.8 vs 29.5 days without biopsy; $p<0.001$), reserved only for atypical cases or failed management.[4] For patients with characteristic findings—such as an afebrile course, a normal leukocyte count, and no rim-enhancing collections on MRI—noninvasive diagnosis and conservative management are recommended.

Currently, only symptomatic treatment is available, but its effectiveness remains uncertain. Patients should receive

optimal diabetic control, pain management, and rest and may also receive low-dose aspirin.[6] Physiotherapy should be avoided during acute illness, as it may prolong symptom resolution.[9]

The DMI outlook is positive and resolves within weeks; however, its high recurrence rate (45-47%) within the first few years is concerning.[10] Recurrences occur more in other regions (39%) than in the original site (8%).[8] DMI indicates microangiopathy in diabetic patients, emphasising the need for aggressive vascular risk management.

CONCLUSION

In patients with diabetes who present with acute limb pain and swelling, diabetic myonecrosis should be considered, especially when infection and vascular occlusion have been ruled out. Magnetic resonance imaging (MRI) is the preferred diagnostic tool, and conservative treatment typically results in a favourable prognosis. It is imperative for clinicians to be aware of this condition to avoid misdiagnosis and unnecessary medical interventions.

Declaration

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REFERENCES

1. Angervall L, Stener B. Tumoriform focal muscular degeneration in two diabetic patients. Diabetologia. 1965;1(1):39-42.
2. Trujillo-Santos AJ. Diabetic muscle infarction: an underdiagnosed complication of long-standing diabetes. Diabetes Care. 2003;26(1):211-5.
3. Kolfenbach JR. Endocrine-associated arthropathies. Rheumatology Secrets E-Book: Rheumatology Secrets E-Book; 2019: 386.
4. Horton WB, Taylor JS, Ragland TJ, Subauste AR. Diabetic muscle infarction: a systematic review. BMJ Open Diabetes Res Care. 2015;3(1):e000082.
5. Taylor JS. Muscle infarction in patients with diabetes mellitus. MR imaging findings. Radiology. 1999;211:241-7.
6. Rocca PV, Alloway JA, Nashel DJ. Diabetic muscular infarction. In Seminars in arthritis and rheumatism WB Saunders. 1993;22:280-7.
7. Grigoriadis E, Fam AG, Starok M, Ang LC. Skeletal muscle infarction in diabetes mellitus. J Rheumatol. 2000;27(4):1063-8.
8. Botero Suarez CS, Matos M, Suryanarayanan S. Diabetic Muscle Infarction: An Uncommon Diabetic Complication With a Lack of Standardized Treatment. JCEM Case Reports. 2003;1(2):18.
9. Chester CS, Banker BQ. Focal infarction of muscle in diabetics. Diabetes Care. 1986;9(6):623-30.
10. Yong TY, Khow KSF. Diabetic muscle infarction in end-stage renal disease: a scoping review on epidemiology, diagnosis and treatment. World J Nephrol. 2019;7(2):58.

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