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Caso Clínico

# Diabetic Muscle Infarction in a 45-Year-Old Male

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A B S T R A C T

Diabetic muscle infarction is a rare complication of diabetes mellitus, affecting patients with long-standing disease and poor glycemic control.
A 45-year-old male, with history of longstanding (15 years) type 2 diabetes, presented to the Emergency room with bilateral muscle leg pain and gait impairment. Neurological examination showed no neurological focal signs or impaired muscle strength.
Urgent ultrasound examination of both thighs was compatible with ischemic muscle changes in the lateral left and medial right thigh muscles. Thigh magnetic resonance revealed bilateral infarction of the vastus medialis and left vastus lateralis muscles. The patient was started on anti-inflammatory drugs and low-dose aspirin. Pain resolved two weeks after admission and there was no gait limitation at discharge.
Current data is limited on which therapeutic and management approaches should be indicated and future studies should further investigate the role of anti-inflammatory drugs in diabetic muscle infarction, as well as the impact of glycemic control on recurrence rates.

## Enfarte Muscular Diabético num Homem de 45 Anos de Idade

R E S U M O

O enfarte muscular diabético é uma complicação rara da diabetes, em casos de doença prolongada e mal controlada.
Um homem de 45 anos, com história de diabetes tipo 2 de longa data (15 anos de evolução), apresentou-se no serviço de urgência com dor muscular bilateral nas pernas e comprometimento da marcha. O exame neurológico não mostrou sinais neurológicos focais ou diminuição da força muscular.
A ecografia das coxas foi compatível com alterações musculares isquémicas. A ressonância magnética das coxas revelou enfarte bilateral dos músculos vasto medial e vasto lateral esquerdo. Foi iniciado tratamento com anti-inflamatórios e aspirina em baixa dose. A dor resolveu em duas semanas após a admissão e não havia limitação da marcha à alta.
Os dados atuais são limitados sobre quais abordagens terapêuticas mais indicadas e estudos futuros devem investigar melhor o papel dos anti-inflamatórios no enfarte muscular diabético, bem como o impacto do controlo metabólico nas taxas de recorrência.

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## Introduction

Despite not being the most frequent constellation of symptoms at the emergency department, lower limb pain and gait impairment are frequent complains that lead to an urgent consultation.

These symptoms may have multiple causes, from systemic infection and medication toxicity to auto-immune diseases, polyneuropathy and inflammatory myopathies. Localized lower limb myalgias have, however, a narrower list of differential diagnosis and diabetic muscle infarction, although rare, must be thought of if the presentation is acute or subacute and there is past history of longstanding and poorly controlled diabetes mellitus.

Existing evidence on diabetic muscle infarction or spontaneous diabetic myonecrosis is based on case reports and analyses of the findings described in each of them, so its prevalence is difficult to determine. This condition was first described in 1965, and since then less than 200 cases have been reported.[1] It is considered to be a complication of diabetes mellitus although there is no consensus on the pathogenic mechanisms responsible.[2] Microangiopathic changes, vasculitis or ischemia-reperfusion injury are some of the mechanisms proposed.[2-4] Clinically, diabetic muscle infarction is characterized by acute or subacute pain, swelling and tenderness of thigh or calf, unrelated to trauma or exercise and evolving over days to weeks. It occurs in both type 1 and type 2 diabetes, and most of the patients have other associated microvascular complications.[2,5]

In a 2015 systematic review, mean age at presentation was 45 years-old and bilateral presentation occurred in about eight percent of the cases analysed.[2] Routine laboratory tests are often unrevealing, with normal to mildly elevated leukocyte counts, CK levels and erythrocyte sedimentation rates.[2] Measurements of glycated hemoglobin are usually high (mean 9.3% in the 2015 systematic review).[2] Diagnosis is made combining clinical data and imaging results. Ultrasound and magnetic resonance imaging (MRI) are useful diagnostic imaging options, with MRI yielding the most specific results.[2,6,7] Muscle biopsy is reserved for when the diagnosis remains unclear despite clinical, laboratory and imaging data.[8]

Management options include bed rest, symptomatic relief, non-steroidal anti-inflammatory drugs (NSAIDs), low-dose aspirin and glycemic control as these strategies appear to be associated with the shortest recovery times and lower recurrence rates, although limited data exist on these approaches.[2]

Mean time to symptom resolution ranges from 28.5 to 81.6 days, depending on treatment option, with physiotherapy and surgery being associated with longer recovery times.[2,3] Recurrence rates reported are high, ranging from 10% to 50%. Treatment with NSAIDs has been found to relate to a lower recurrence risk (10%).

## Case Report

We describe the case of a 45-year-old male with past medical history of ill-controlled type 2 diabetes mellitus (with 15 years of evolution) who presented to our ER with bilateral thigh myalgias and gait disturbance. These symptoms had started one week before and evolved to unbearable and constant muscle pain on both thighs, partially ameliorated when standing. There was no history of trauma or intense exercise and the patient denied pain in any other muscle group. Diabetes mellitus diagnosis was made when he was 30 years old and, despite no specialist follow-up, he had no diagnosed micro- or macrovascular complications. Current patient medication included oral anti-diabetics (metformin 1000 mg qd + vildagliptin 50 mg qd and gliclazide 30 mg qd), insulin basal-bolus scheme (although the patient admitted to a weak compliance with his insulin administration scheme), sinvastatin 20 mg qd and sertraline 50 mg qd. None of these drugs had been introduced or changed in the last year.

Neurological examination had no evidence of neurological focal signs, namely no motor or sensitive deficits. Thigh movements showed normal muscle strength bilaterally. Bilateral thigh active or passive movement and muscle palpation were extremely painful. There was no apparent muscle swelling or hypertrophy nor associated skin changes. Gait was difficult due to pain, with incapacity to walking more than 2-3 consecutive steps without help, but there was no ataxia. Physical examination was otherwise unrevealing.

The patient had a high glucose level at admission (>300 mg/dL), was hemodynamically stable and afebrile. Blood tests revealed mildly elevated CK levels (322 U/L), no leukocytosis and normal D-dimers, erythrocyte sedimentation rate and C-reactive protein values. Blood cultures were obtained in order to exclude infectious muscle necrosis.

Thigh ultrasound showed increased thickness and echogenicity of thigh skin bilaterally with associated muscle hyper-echogenicity. Ultrasonographic structure was heterogeneous in the lateral left and medial right thigh regions, with hypoechoic regions interspersed, suggesting muscle infarction in these topographies. Venous walls appeared thickened, but there were no signs of venous thrombosis. Arterial Doppler ultrasound showed no relevant stenotic or occlusive lesions on both legs. These findings were further characterized by thigh MRI which revealed subcutaneous edema on both thighs, and hyperintense signal of the right vastus medialis and left vastus lateralis muscles on T2- weighted images. These muscles appeared hypointense on T1-weighted images with interspersed areas of T1 hyperintensity, suggesting hemorrhagic transformation areas inside infarcted muscles (Fig. 1).

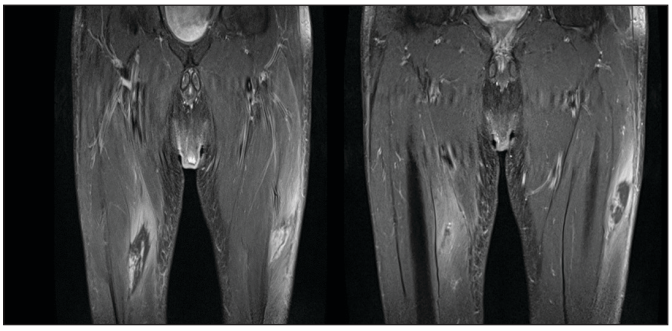

*Figure 1.* Bilateral diabetic muscle infarction.
Thigh MRI hyperintense signal of the right vastus medialis and left vastus lateralis muscles on T2- weighted images.

The patient was admitted and started on NSAIDs (ibuprofen 400 mg 3 id) and low-dose aspirin (100 mg id). Glycemic control was achieved during hospital stay mainly due to better compliance to insulin therapy. Further laboratory tests revealed a glycated hemoglobin of 14%. Serum creatinine levels were 1.49 mg/dL at admission but normalized during hospital stay. Auto-immunity markers, including antinuclear antibody (ANA) and anti-neutrophil cytoplasmic antibody (ANCA) status, and myopathy antigen antibodies were negative, as were the blood cultures. Coagulation

studies showed no abnormalities. No signs of compartment syndrome or other complications were detected during hospital stay.

Bed rest was preferred is the first week, but mild physical activity was encouraged as soon as severe pain subsided. Rest and movement associated muscle pain resolved in two weeks after admission. At discharge from in-hospital care there was no gait limitation. Mild thigh tenderness persisted. Aspirin was maintained and follow-up consultations scheduled for vigilance of glycemic control, other vascular risk factors and recurrence signs.

## Conclusion

Diabetic muscle infarction is a rare but potentially serious complication of diabetes mellitus, affecting mainly young adults with longstanding and poorly controlled diabetes. Symptoms and signs may be interpreted in the context of other (more frequent) causes of leg pain, and this may result in delays in diagnosis and treatment or lead to the use of invasive and unnecessary diagnostic tests. Therefore, a high clinical suspicion is the key to identify this condition, and to correctly choose complementary diagnostic techniques and initiate treatment shortly after presentation. Current data is limited on which therapeutic and management approaches should be indicated, although existing evidence supports the use of NSAIDs and aspirin (providing that there are no contra-indications) as these strategies seem to be associated with shorter recovery times. Rigorous glycemic control should be targeted in order to prevent, not only the more common micro- and macrovascular complications of diabetes, but also recurrence of diabetic muscle infarction.

In this clinical case, early recognition of diabetic muscle infarction allowed for early treatment and risk factor control, which resulted in a relatively short recovery time. Ultrasound and MRI were crucial for confirming diagnosis. Treatment strategy chosen (with aspirin, NSAIDs and rest) was based on the existing evidence from case reports and small case series and absence of contra-indications for the use of these medications. Future studies should, however, further investigate the role of NSAIDs and other therapeutic approaches in diabetic muscle infarction, as well as the impact of these strategies and glycemic control on recurrence rates and prognosis of this condition.

## Contributorship Statement / Declaração de Contribuição:

TC: Conceptualization, data collection, writing original draft, and final approval.
LS: Conceptualization, data collection, review and final approval.
RM and PF: Conceptualization, methodology, supervision, review and final approval.

## Responsabilidades Éticas

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