IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 23, Issue 2 Ser. 2 (February. 2024), PP 58-61
www.iosrjournals.org

Diabetic Muscle Infarction, A Rare Complication Of Type 2 Diabetes Mellitus

Behera Govind R[1], Roy Ayan[2], Hui Pratisruti[3]
[1](Endocrinology And Metabolism, Aiims Kalyani, India)
[2](Endocrinology And Metabolism, Aiims Kalyani, India)
[3](Radiology, Aiims Kalyani, India)

Abstract:
Diabetic muscle infarction, a rare and under recognized complication seen in poorly controlled diabetes mellitus. Currently, there isn't a universally agreed-upon set of guidelines for treating diabetic muscle infarction (DMI), a rare and often overlooked complication linked to poorly managed diabetes. This condition manifests as sudden, intense muscle pain and swelling, particularly in the lower limbs. Magnetic resonance imaging (MRI) using Short TI Inversion Recovery (STIR) sequences is the preferred diagnostic tool. Treatment typically involves using non-steroidal anti-inflammatory drugs (NSAIDs), ensuring rest, and maintaining good control of blood sugar levels, which usually suffice for managing most cases. We are hereby reporting a case of DMI from a tertiary care setup which raised considerable diagnostic dilemma.

Key-words: *Complications of Diabetes, Muscle Infarction, Diabetic Myonecrosis*

Date of Submission: 29-01-2024 Date of Acceptance: 09-02-2024

I. Introduction

Diabetic muscle infarction (DMI), also referred as diabetic myonecrosis, is a rare but dreadful complication of poorly controlled diabetes mellitus. A systematic review of all cases published in the English language since the initial description of the condition identified reports of 170 records of DMI. Its pathogenesis remains unclear as it arises spontaneously without trauma or infection signs, often masquerading as cellulitis, deep vein thrombosis, or necrotizing fasciitis. Misdiagnosis is common due to insufficient suspicion. Here, a case is presented involving DMI affecting the quadriceps muscles in both lower limbs of a diabetic individual.

II. Case Report

A gentleman in his 50s with a history of uncontrolled type 2 diabetes mellitus for 7 years and pre-existing peripheral neuropathy, started to develop complaints of difficulty walking due to excruciating pain in both the thighs, from the fall of 2022. There was no history of fever, trauma preceding the symptoms. Over the next few months, the insidious onset of bilateral thigh pain was accompanied by progressive proximal muscle weakness, culminating in substantial gait disturbances and an inability to ambulate. In final month of 2022, the patient initiated analgesic agents for pain relief. However, by early 2023, he experienced a sudden exacerbation in the symptoms and an inability to stand or walk properly, with a history of frequent falls, leading to multiple hospital admissions. In second calendar month of 2023, the patient presented to our hospital with sudden severe pain (intensity of 8/10 on the numerical pain scale) in quadriceps group of muscles of both the thighs with muscle weakness, also diabetic bullae over the fingers of both hands and mild cellulitis of the left foot near the great toe. The patient's vital signs were recorded as follows: blood pressure of 146/92 mm Hg, heart rate of 88 per minute, respiratory rate of 20 breaths per minute, oxygen saturation level of 98% on room air, and body temperature of 36.6 degree Celsius. BMI was 20.1 kg/m2 and mild features of cachexia was observed. His physical examination, exhibited with power of 1/5 in both the proximal anterior thigh compartment, diabetic bullae over the fingers of both hands, mild cellulitis of the left foot, and warm-to-touch with tenderness over anterior thighs, with the left side being more affected, than the right, but sparing inguinal and perineal regions. Muscle girth measurement of left thigh was noted to be 5cm more than the contralateral right thigh. There were no signs of critical limb ischemia, and pulse was palpable on both lower extremities, suggesting no peripheral arterial disease. However, he had florid features of peripheral sensory neuropathy. But retinopathy was absent. There were initial concerns for deep vein thrombosis and cellulitis. Therefore, a duplex ultrasound of both lower extremities was performed, ruling out deep vein thrombosis and blood cultures were remained negative. With vascular pathology eliminated, electromyography was employed to assess the muscular component. Electromyography findings revealed active and chronic denervation changes in multiple muscles, including the

left abductor pollicis brevis, right first dorsal interosseous muscle, bilateral tibialis anterior, left rectus femoris, and right vastus medialis. These neurophysiological abnormalities strongly suggested an underlying neuro-muscular process, prompting further investigations to elucidate the precise ethology. Anecdotal evidence suggests that open biopsy prolongs recovery secondary to poor wound healing and also carries risk of complications such as hematoma and infections as was seen in our patient. So, to explore the structural integrity of the affected muscles, MRI of the left thigh was conducted. Findings including subcutaneous oedema, fat stranding, areas of restricted diffusion, and peripheral contrast enhancement in the adductor group of muscles, pectineus, rectus femoris, vastus lateralis, vastus medialis, and vastus intermedius. The MRI findings were consistent with muscle inflammation, oedema, and infarction, which were in line with the suspected diagnosis of diabetic muscle infarction (Figure A, B, C). Also, there was increment in CPK (376 U/L). To assess kidney function and potential kidney involvement, the Urine Albumin to Creatinine Ratio (UACR) was measured, and the result was alarmingly high at 1441 mg/g (reference range: 30-300 mg/g). This elevated UACR signified macro albuminuria, indicating renal involvement and diabetic nephropathy. Then further treatment plan involved rest, elevation and immobilisation of the affected limb and low dose Aspirin was added and continued. Regular follow up were scheduled to monitor glycaemic control and assess the patient's progress. Serum Procalcitonin level was 0.09 ng/ml (bacterial infection is unlikely). Thyroid panel, D-dimer, antinuclear antibody (ANA), ANA Profile, APLA (anti-phospholipid antibody) profile, Vasculitis screen was unremarkable.

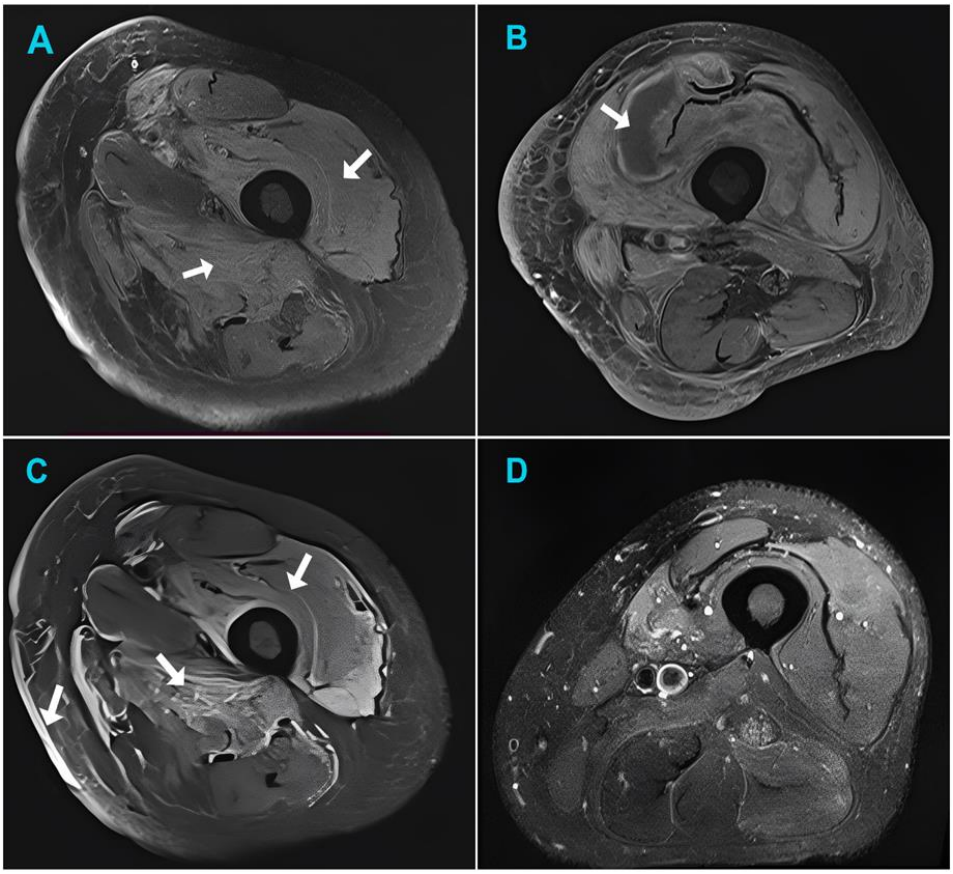
III. Discussion

The first documented cases of DMI were reported in 1965 by Angervall and Stener. Since then, approximately 228 cases have been reported in the Embase database[1]. A systematic review of the literature identified 126 initial cases of DMI[2]. DMI was more frequently observed in patients with long standing poorly controlled T2DM (50%) than in those with T1DM (41.7%). Nephropathy was present in 75% of DMI cases[2]**.** In cases of sudden onset acute muscular pain with a palpable mass that is firm, tender, and localized in an extremity, DMI should be considered, especially in diabetic patients. The most commonly affected site is the front thigh (55% of cases), followed by the calf (15%) and back thigh[2,10]. The involvement can be unilateral or bilateral, affecting a single muscle or an entire muscle compartment[9]**.** The affected extremity shows no signs of infection, and the overlying skin appears intact or may exhibit mild erythema. Usually, history of trauma or fever is absent in DMI. Pain worsens at rest and with movement, and functional impairment is a result of muscle damage and local swelling[11]. Spontaneous smooth muscle or myocardial necrosis in diabetes mellitus has not been reported in the literature. DMI is considered a very late-stage and terminal complication of diabetes[12]. Inflammatory markers like C-reactive protein (CRP) or erythrocyte sedimentation rate (ESR) are often elevated, while white blood cell (WBC) counts typically remain within normal limits in most cases[2,9], same is with our case. Radiological imaging plays a crucial role in the diagnosis of DMI. Magnetic resonance imaging (MRI) is considered the imaging modality of choice, particularly when gadolinium contrast is administered. Axial short tau inversion recovery (STIR) and fat-suppressed T2-weighted images reveal increased signal intensity in the affected muscle and ring-enhancing patterns around the area of necrosis. Other characteristic MRI findings include an isointense to hypointense signal on T1-weighted images, associated subfascial fluid, and subcutaneous or interfascial oedema[11,13,14]. Sonographic findings in DMI include a well-marginated, hypoechoic intramuscular lesion with internal linear echogenic structures coursing through it, ruling out deep vein thrombosis (DVT), abscesses, or necrotic tumours[11,14]. Contrast-enhanced CT may reveal a low-attenuation lesion with ring-enhancing margins within the affected muscle[7,11,15]. Muscle biopsy can provide a definitive diagnosis, but it is not strongly recommended due to the risk of complications such as bleeding, prolonged recovery, and infection[16]. While DMI itself is a relatively benign condition, a comprehensive and vigilant differential diagnosis is necessary to exclude more serious conditions such as DVT, abscesses, cellulitis, osteomyelitis, benign and malignant tumours, pyomyositis, dermatomyositis, hematoma, focal myositis, ruptured Baker's cyst, exertional muscle rupture, diabetic lumbosacral plexopathy, or diabetic amyotrophy. The pathophysiology of diabetic myonecrosis remains uncertain. One theory suggests arteriosclerosis obliterans is the primary factor leading to DMI[17]. Other theories link DMI to vasculitis, atherosclerosis, or diffuse microangiopathy associated with hypoxia-reperfusion injury[4]**.** The optimal treatment for DMI is still undetermined. Current recommendations focus on bed rest, analgesia, and achieving adequate glycaemic control. A study by Onyenemezu and Capitle[16] analysed the impact of different supportive care modalities on the recovery time and recurrence risk of DMI. The study considered three main techniques: surgery, physiotherapy, and bed rest. In the short term, the prognosis for DMI is generally good, with most patients recovering within a few weeks to six months and minimal functional impairment. However, recurrence is common, and the long-term prognosis is generally unfavourable for patients with microvascular complications[9,15]. In conclusion, we describe a rare complication of diabetes i.e., diabetic muscle infarction triggered by poor glycaemia in an adult patient who had other features of complications like nephropathy and

neuropathy. MRI imaging can be diagnostic in this setting and the outcome with conservative therapy remains satisfactory but a long-term follow up is required.

Laboratory Test (Units):	Reference Range:	On Admission:
C-reactive protein (CRP)(mg/L)	<= 10	108
Total leukocyte count (10^3/uL)	4-10	6.16
Total protein (g/dL)	6.4-8.3	5.7
Sodium (mEq/L)	137-145	136
Potassium (mEq/L)	3.5-5.1	3.0
Aspartate aminotransferase (AST) (U/L)	15-46	17
Alanine aminotransferase (ALT) (U/L)	<50	12
Creatinine (mg/dL)	0.6-1.2	1.22
Urine Albumin to Creatinine Ratio (UACR) (mg/g)	30-300	1441
Thyroid Stimulating Hormone (TSH) (uIU/mL)	0.27-4.20	4.97

Magnetic resonance imaging of the left thigh revealed subcutaneous oedema and fat stranding along the left thigh. T2 short tau inversion recovery images showed hyperintensities and areas of diffusion restriction noted in the adductor group of muscles, pectineus, rectus femoris, vastus lateralis, vastus medialis, vastus intermedius with induration s/o oedema with areas of necrosis; which shows peripheral contrast enhancement (Figure A, B, C).



Figures: A) Axial post contrast fat suppressed T1 weighted image shows enhancement of the affected muscles. B) Axial post contrast fat suppressed T1 weighted image shows areas of non-enhancement in vastus medialis and intermedius which represent myonecrosis. C) Axial fat suppressed T2 weighted image showing diffuse subcutaneous and intramuscular oedema in the anterior and medial compartment. D) Axial T2 STIR image showing reduction in hyper intensities in the muscles after five months of follow up.

IV. Conclusion

- Diabetic muscle infarction is a relatively rare and also under recognised complication that healthcare professionals need to be aware of due to the rising prevalence of diabetes mellitus.
- Diabetic muscle infarction manifests as sudden onset of intense muscle pain and noticeable swelling, especially in the lower limbs.

- Muscle biopsy can provide definitive confirmation, it is advisable to reserve this invasive procedure for cases where the presentation is unclear or when alternative diagnoses need to be ruled out.
- MRI is imaging study of choice.
- Non-Steroidal anti-inflammatory drug therapy is linked to quicker recovery periods and reduced likelihood of recurrence.

Acknowledgement

The authors would like to thank Dr. Pratisruti Hui from the Department of Radiology, All India Institute of Medical Sciences, Kalyani for her assistance in CT and MRI results analysis and interpretation.

References

[1]. Angervall L, Stener B. Tumoriform Focal Muscular Degeneration In Two Diabetic Patients. Diabetologia. 1965 Aug;1:39-42
[2]. Horton Wb, Taylor Js, Ragland Tj, Subauste Ar. Diabetic Muscle Infarction: A Systematic Review. Bmj Open Diabetes Research And Care. 2015 Apr 1;3(1):E000082
[3]. Banker, B.Q.; Chester, C.S. Infarction Of Thigh Muscle In The Diabetic Patient. Neurology 1973, 23, 667–677
[4]. Silberstein, L.; Britton, K.E.; Marsh, F.P.; Raftery, M.J.; D'cruz, D. An Unexpected Cause Of Muscle Pain In Diabetes. Ann. Rheum. Dis. 2001, 60, 310–312
[5]. Aboulafia Aj, Monson Dk, Kennon Re. Clinical And Radiological Aspects Of Idiopathic Diabetic Muscle Infarction: Rational Approach To Diagnosis And Treatment. The Journal Of Bone And Joint Surgery. British Volume. 1999 Mar;81(2):323-6
[6]. Verjee Ma, Abdelsamad Na, Qureshi S, Malik Ra. Diabetic Muscle Infarction: Often Misdiagnosed And Mismanaged. Diabetes, Metabolic Syndrome And Obesity: Targets And Therapy. 2019 Mar 5:285-90
[7]. Morcuende Ja, Dobbs Mb, Buckwalter Ja, Crawford H. Diabetic Muscle Infarction. The Iowa Orthopaedic Journal. 2000;20:65
[8]. Cabrera So, Michel R, Lu A, Dimitrov V. Diabetic Muscle Infarction With Complications. Int J Case Rep Images. 2013 Jun 1;4:303-7
[9]. Yong Ty, Khow Ks. Diabetic Muscle Infarction In End-Stage Renal Disease: A Scoping Review On Epidemiology, Diagnosis And Treatment. World Journal Of Nephrology. 2018 Mar 3;7(2):58-64
[10]. Chason Dp, Fleckenstein Jl, Burns Dk, Rojas G. Diabetic Muscle Infarction: Radiologic Evaluation. Skeletal Radiology. 1996 Feb;25:127-132.
[11]. 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 Jan 1;174(1):165-169.
[12]. Wong Ty, Li Pk. Unusual Muscle Pain In Two Patients With Diabetic Renal Failure. Hong Kong Med J. 2002 Oct;8(5):368-371.
[13]. Jawahar A, Balaji R. Magnetic Resonance Imaging Of Diabetic Muscle Infarction: Report Of Two Cases. The Iowa Orthopaedic Journal. 2014;34:74.
[14]. Huang Bk, Monu Ju, Doumanian J. Diabetic Myopathy: Mri Patterns And Current Trends. American Journal Of Roentgenology. 2010 Jul;195(1):198-204.
[15]. Trujillo-Santos Aj. Diabetic Muscle Infarction: An Underdiagnosed Complication Of Long-Standing Diabetes. Diabetes Care. 2003 Jan 1;26(1):211-215.
[16]. Onyenemezu I, Capitle Jr E. Retrospective Analysis Of Treatment Modalities In Diabetic Muscle Infarction. Open Access Rheumatology: Research And Reviews. 2014 Jan 10:1-6.
[17]. Chester Cs, Banker Bq. Focal Infarction Of Muscle In Diabetics. Diabetes Care. 1986 Nov 1;9(6):623-630..