# Diabetic myonecrosis, an uncommon presentation of diabetes mellitus in tropical area: a case report

**Ziryab Imad Taha Mahmoud¹'², Yassin Abdelrahim Abdalla³'⁴, Salih Boushra Hamza³'⁴, Ali Ibrahim Elsiddig Ahmed², Sami Ahmed Abd Algadir², Sohep abdalla osman²**

¹University of Bahri, Khartoum, Sudan

²Sudanese Medical Specialization Board, Khartoum, Sudan

³Department of Internal Medicine, Faculty of Medicine and Health Sciences, Omdurman Islamic University, Khartoum, Sudan.

⁴PSO\_Research Unit

**Corresponding author:** Salih Boushra Hamza, MBBS, Department of Internal Medicine, Faculty of Medicine and Health Sciences, Omdurman Islamic University, Khartoum, Sudan. E-mail: salihboushra@gmail.com. phone: +249999253222

**Abstract:** Diabetic myonecrosis is uncommon complication related to long standing poorly controlled diabetes. 33-year-old Sudanese male patient with type one diabetes presented with progressive, severe bilateral thigh pain with low grade fever. Lab results show hyperglycemia with ketonuria and elevated creatine kinase but normal white cell blood count. The patient was diagnosed initially as diabetic ketoacidosis with pyomyositis and receive analgesic and insulin the patient partially improved. after second evaluation bilateral thigh MRI was requested and shows diffuse edema involving medial muscle group of upper third of right side with intramuscular facial edema, appearing as low signal in T1 and high signal in T2 and fat suppression images with no evidence of collection or abscess. Diagnosis of diabetic myonecrosis was made. The patient managed conservatively and discharge on aspirin with full recovery.

**Keywords**: diabetes, tropical area, muscle infarction, myonecrosis, edema

## Introduction

Diabetic myonecrosis is an uncommon complication of diabetes mellitus that occurs in patients with long-standing poorly controlled diabetes. Angervall and Stener first reported it in 1965 as focal muscular degeneration in 2 diabetic patients [1].

Since then, less than 200 cases have been reported [2].Pathogenesis of this rare entity is poorly understood. Literature reports role of microangiopathy, atherosclerotic plaquing of microvessels, superimposed vasculitis, ischemic-reperfusion injury and thrombosis of microvasculature, along with causative role of coagulation fibrinolytic cascade with hypercoagulable state secondary to low antithrombin-III levels [3–5].

The diagnosis of DMN is often very challenging and difficult. The diagnosis is often missed or the condition is misdiagnosed unless physician or radiologist is well aware of the clinical presentation and diagnostic clues.

Diabetic myonecrosis most commonly affects the thigh and usually presents with acute muscle pain, edema, and erythema in the absence of trauma or fever (6).

Physical exam reveals swollen and tender muscle, mimicking deep venous thrombosis (DVT) and almost all the time presumptive diagnosis of DVT is made and often examination remains suboptimal due to fear of throwing thrombus. However, if examination done properly, muscle may feel indurated that can suggest muscular etiology. At time diagnosis of lymphangitis/cellulitis is considered due to presence of subcutaneous edema and fluid. Other differential include abscess, necrotizing fasciitis, compartment syndrome, osteomyelitis, polymyositis, and dermatomyositis, drug induced myositis and superficial thrombophlebitis. However, absence of overlying erythema and classic constitutional symptoms should make physician to consider diagnosis of DMN in long standing poorly controlled diabetes with leg pain [3].

Blood work classically shows normal to mildly increased WBC, normal to elevated inflammatory markers (ESR, CRP), normal or mild elevation of muscle enzymes, like CPK [5]. Ultrasonography (US) is the initial imaging study that should be performed to rule out venous thrombosis, superficial thrombophlebitis,

Underlying abscess or localized fluid collection and necrotizing fasciitis [7]. Magnetic resonance imaging (MRI) is next modality of choice with sensitivity of T2-weighted MRI approaches 90% for picking up active muscle disease however specificity for muscle infarction only 43% [8].

## Case presentation

33 years old Sudanese male, self-employer who is known case of type one diabetes mellitus for six year presented with bilateral thigh pain for four days with low grade fever for two days.

The pain was localized to medial aspect of both thighs, gradual in onset which became very severe and interfere with his daily activity, increase by movement and activity, decrease by rest and staying static. It is associated with generalized fatigability. There is no other group of muscles affected.

Fever was low grade intermittent, not documented and response well to oral paracetamol.

Systematic review was unremarkable apart from poly-urea, nausea and vomiting, urine was clear with normal color.

On reviewing his relevant past history, the patient was diabetic 6 years on mixtard insulin 15-10 but Poor control, less adherence to medications with frequent hospitalizations, no ICU admission no other chronic illnesses. No past history of upper respiratory tract infection, bloody diarrhea neither watery diarrhea.His family history was positive for D.M, negative for rheumatological disorder. No chronic medication apart from insulin.

.

The patient was on severe pain, febrile, tachypnic and tachycardic with regular radial pulsation. Peripheral lower limb pulsation was intact. There was severe tenderness on medial aspect of both thigh with hotness and swelling. The patient cannot move his legs. Other neurological examination was normalwith no upper limb or cranial nerve abnormality.

The patient had high RBG and HbA1c. His inflammatory markers were raised with normal white cell count. CK was elevated (900U/L) U&E and Liver enzyme was both within normal limits (Table 1). Doppler U/s was done and was negative for DVT.

**Table 1: Investigation done in first evaluation in hospital**

|  |  |
| --- | --- |
|  |  |
| Hb | 13.5 g/dl |
| TWBC | 9700mg |
| Random blood glucose | 250 mg/dl |
| HbA1C | 14% |
| Serum urea | 42mg/dl |
| Cretinine | 0.7mg/dl |
| Uric acid | 4 |
| Acetone (urine sample) | +++ |
| S.CK | >900 |
| S.Ca | 7.2mg/dl |
| S.k | 4.46mEq/L |
| CRP | 200 |
| ESR | 25 |

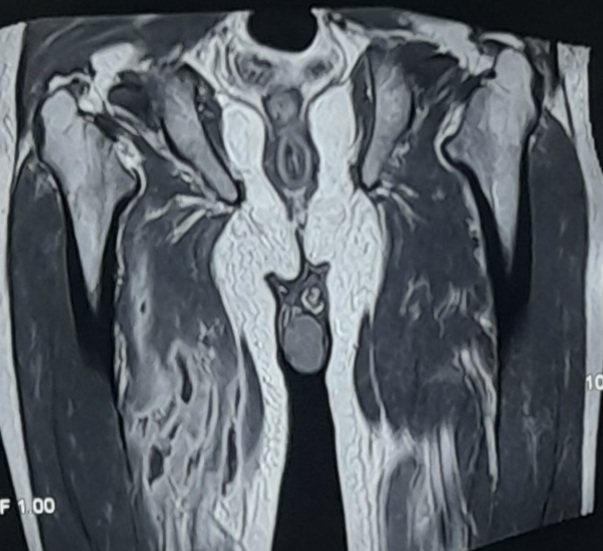
The patient diagnosed as diabetic ketoacidosis with polymyositis and admitted to hospital for six day where he received .IV fluid, analgesia, insulin, and antibiotic; the patient recovered from DKA with partial decrease in thigh pain and discharged on lantus 16 units with soluble insulin 8 units before each meal. Then the patient referred to rheumatology clinic.

On second evaluation, the patient was still have bilateral thigh pain with nausea and vomiting and unable to walk and afebrile. Creatine kinase level normalizes (25.6 U/L) and CRP level decreased but still high (25mg/L) CBC and LFT was all within normal.

Bilateral thigh MRI with I.V was done, it shows diffuse edema involving medial muscle group of upper third of right side with intramuscular facial edema, appearing as low signal in T1 and high signal in T2 and fat suppression images. The size of affected right thigh is larger than left. There is no enhancing abscess or free fluid collection, which is consistent with diabetic myonecrosis (Figure 1).

The diagnosis of diabetic myonecrosis was made and patient received analgesia. His blood glucose was optimized and underwent physiotherapy sessions. The pain gradually improved the patient was discharged on aspirin in good condition.

**Figure 1: MRI of the thigh**



B

A

A: T1-weighted coronal view show lowsignals

B: T2-weighted coronal view show high signals

## DISCUSSION

Diabetic myonecrosis (DMN) is rare presentation in long standing poorly controlled complicated diabetes it is often associated with misdiagnosis[9,10]. Although treatment is conservative, invasive procedure as biopsy and inappropriate management as anticoagulation associated with increase morbidity and delay recovery [10, 11,14].

The most common affected muscle is thigh muscle which consistent with our case [11]. surprisingly as oppose to classical age of presentation and diabetes status our case was relatively young with diabetes onset less than 15 years with no identifiable target organ complications. Although it is rare; but bilateral involvement has been reported in 10% of cases [10, 11]. The examination finding of hot, tender swollen muscle is common finding and was elicited during assessment. Some literatures report weakness and even sensory disturbance and so our patient has weakness but intact sensation. Lab result of normal white cell count and mildly elevated CK with poor glycemic index (RBG and HbA1C) was same as previous literatures [12, 13].

Diagnosis of diabetic myonecrosis was challenging in this patient; as low grade fever (although not documented) with DKA especially in tropical setting put the possibility of pyomyositis which is devastating disease that require early surgical intervention. MRI is useful guidance for differentiation.

## Disclosure

The authors report no conflicts of interest in this work.

## Support

No financial support.

## Authors contribution

Dr. Ziryab Imad Taha: writing paper, editing, diagnosis, management

Dr. Yassin Abdelrahim Abdalla: writing paper, editing, follow-up.

Dr: Salih Boushra Hamza: writing paper, editing, follow-up.

Dr: Ali Ibrahim Elsiddig: reviewing image.

Dr: Sami Ahmed Abdelgadir: reviewing image.

Dr:Sohep Abdalla Osman: editing,follow-up.

Consent:

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

References:

1. Angervall L, Stener B: Tumoriform focal muscular degeneration in two diabetic patients. Diabetologia. 1965;1: 39–42.

2. Trujillo-Santos AJ: Diabetic muscle infarction. Diabetes Care. 2003;26:211–15

3. P. Mukhopadhyay, R. Barai, C.A. Philips, J. Ghosh, S. Saha, An unusual case of myonecrosis, Case Rep. Endocrinol. 2011.

4. A. Rastogi, S. Bhadada, U. Saikia, A. Bhansali, Recurrent diabetic myonecrosis: a rare complication of a common disease, Indian J. Med. Sci. 2011;65(7): 311.

5. G.W. Palmer, T.P. Greco, Diabetic thigh muscle infarction in association with antiphospholipid antibodies, Semin. Arthritis Rheum. 2001;30(4)272–280.

6. World Health Organization, Global Report on Diabetes, World Health Organization, 2016.

7. A. Nagdev, M. Murphy, C. Sisson, Bedside ultrasound for the detection of diabetic myonecrosis, Am. J. Emerg. Med. 2008;26(8)969-e3.

8. J.A. Morcuende, M.B. Dobbs, J.A. Buckwalter, H. Crawford, Diabetic muscle infarction, Iowa Orthop. J. 2000;20:65.

9. Baker JC, Demertzis JL, Rhodes NG. Diabetic Musculoskeletal Complications and Their Imaging Mimics. 2012;1959–75.

10 . Gupta S, Goyal P, Sharma P, Soin P, Kochar PS. Recurrent diabetic myonecrosis – an under-diagnosed cause of acute painful swollen limb in long standing diabetics. Ann Med Surg [Internet]. 2018;35:141–5.

11. Stevens AC. Diabetic Myonecrosis : A Rare Complication of Diabetes Mellitus Mimicking Deep Vein Thrombosis. 2017;38–41.

12.Storandt M, Thondapi C, Matta A. of Case Reports in Diabetic Myonecrosis : An Uncommon Complication of a Common Condition. EJCRIM. 2020;7(3).

13. Jelinek JS, Murphey MD, Aboulafia AJ et al: Muscle infarction in patients with diabetes mellitus: MR imaging findings. Radiology. 1999;211(1):241–47.

14.Kapur S, Brunet JA, McKendry RJ. Diabetic muscle infarction: case report and review. J Rheumatol. 2004;31(1):190–194.