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Commentary

Diabetic myonecrosis: A review

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Abstract

Diabetic Myonecrosis is a rare, under-diagnosed complication of long-standing diabetes mellitus. The condition often mimics several other conditions which results in delay in diagnosis and treatment. It usually affects muscles of the lower limbs especially thighs, presenting with acute onset pain and swelling. It is often associated with long term diabetic complications like nephropathy, retinopathy, neuropathy and others. Although a rare manifestation, we must keep in mind the rising prevalence of diabetes mellitus and hence the expected rise in such uncommon complications in the population, making it essential for physicians to identify the condition promptly and implement efficient interventions. Herein, we discuss the prevalence, etiopathogenesis, clinical features, evaluation and the treatment aspect for DMN.

Introduction

The prevalence of diabetes mellitus (DM) is 830 million worldwide in 2022, which is 14% of the adults aged 18 and older. It is quite high when compared to 200 million of the adults affected with DM in 1990 according to WHO [1]. This increase in prevalence in DM can also show a probable continuation in surge in atypical complications like Diabetic Myonecrosis (DMN), as 116 cases diagnosed in 2003 has risen to 200 in 2015 [2,3]. The earliest description of DMN was given by Angervall and Sterner in 1965 [4]. He described as tumoriform focal degeneration. Other descriptions include spontaneous diabetic muscle infarction, aseptic myonecrosis, and ischemic myonecrosis. DMN occurs as a result of long standing, poorly controlled diabetes. It is described in both in Type 1 DM(T1DM) and type 2 DM(T2DM) with age of presentation at 42.6–44.5 years [5]. DMN is reportedly more common in T1DM with higher incidence in females [6]. The mean duration of DM with muscle infarction was 18.9 years in T1DM and 11 years in T2DM [5]. Studies show an average HbA1c of 9.3% at the time of diagnosis of DMN with the presence of coexisting micro- and macro-vascular complications in diabetic patients due to persistently elevated blood sugars, to be present, such as congestive heart failure, renal failure (diabetic nephropathy, end stage renal disease on dialysis), hypertension, atherosclerotic diseases, peripheral vascular diseases, diabetic neuropathy, and retinopathy [7–9].

Etiopathogenesis

The occurrence of DMN is seen to be more frequent in T1DM and in women [6,10]. The pathogenesis although unknown, suggests a vascular origin likely because of nonenzymatic glycosylation of proteins in the walls of microvasculature resulting in ischaemia, and a further trigger of an inflammatory cascade producing ischaemic necrosis. Subsequent reperfusion generates reactive oxygen species and inflammatory cytokines causing further damage to the vasculature [11]. This hypothesis is supplemented by the commonly seen presence of microvascular complications, specifically, advanced diabetic retinopathy and renal disease. However, there have been cases of DMN in patients without vascular complications of diabetes, one such case report being that of a woman with T1DM without any micro- and macro-vascular complications of DM, although she

presented with long standing altercating hyper- and hypo-glycaemia from several years [12]. The pathogenesis suspected here is elevated HIF-alpha and Rac-1 expression during hyperglycaemia, producing a state of pseudohypoxia, and hypoglycaemia (due to probable insulin overdose) producing hypophosphatemia and resultant rhabdomyolysis secondary to hypophosphatemia; all in all, causing muscle infarction in this case [13–15].

Clinical Features

Patients presents with acute pain and swelling which is non-traumatic origin of the affected muscle group. It usually affects lower limbs especially anterior thigh region and is unilateral. The typical clinical manifestations include acute severe localised pain out of proportion to the physical signs and rapidly progressive over days in the affected muscles with localised swelling and induration. Pain is continuous, exacerbated by movement and also present at rest. Local examination reveals tenderness, warmth, and induration. Ambulation is difficult due to pain when lower extremities is involved. Examination of adjacent joints may be normal with normal range of motion without neurological deficits at the site of involvement. Quadriceps is commonly affected muscle with an incidence rate of 62%. With decreasing in frequency DMN affects hip adductors (13%), hamstrings (8%) and hip flexors (2%) [5,6,16], however any muscle group can be affected. Upper limb involvement is rarely described. Bilateral occurrence simultaneously is seen in 10% of patients [17,18]. Recurrence rate may be up to 34.9% usually involving contra-lateral limb within 6 months [6,19]. Recurrence in the same or opposite limb is in about 50% of the cases over a period of time. Systemic signs of infection such as fever is absent in majority. Rarely if the extent of infarction is severe enough to affect adjacent nerves such as DMN of the hand with resultant Acute Carpal Tunnel Syndrome causing both sensory and motor deficits on examination [20]. Bilateral reflexes will be symmetrical and normal as well as bilateral pulses of extremities involved will be normal and palpable. Most cases show no history of recent trauma, surgery, muscular injections/drug use or recent travel that helps physicians to rule out differentials such as compartment syndrome post trauma or post-surgical infections, probable abscess formation due to intra-muscular injections or other infectious aetiology. Deep venous thrombosis, pyomyositis, soft tissue abscess, necrotizing fasciitis, dermatomyositis, proliferative myositis, focal myositis/ nodular myositis, primary lymphoma of muscle, benign tumours or sarcomas of the muscle, diabetic amyotrophy, osteomyelitis, exertional muscle rupture, and ruptured Baker's cyst are commonly considered differential for DMN [6,21]. In patients with chronic renal disease, calciphylaxis also needs to be considered [22].

Investigations

Routine investigations include complete blood count, with most cases showing a normal Haemoglobin, white blood cell count, platelets, and biochemical parameters in majority of the cases. Some show non-specific elevation in total count, serum creatinine kinase, C-reactive protein, Serum. Lactate Dehydrogenase, and ESR. Elevated ESR, CRP, and creatinine phosphokinase is not directly proportional to the severity of myonecrosis, thus delaying the diagnosis. Serum osmolarity, with presence or absence of acidosis should be checked for, as ketosis as a causation for myopathy must be ruled out. Serum electrolytes including serum phosphate and serum calcium should be done, as dys-electrolytemia common in hyperglycaemic states, is a differential to be considered. Iron profile, thyroid profile, liver functions including enzymes (aspartate and alanine transaminases, glutamyl transferases), coagulation profile, serum cortisol and lipid profile must be sent for, to rule out other probable causes for myopathy. Infectious profile including *Trichinella*, *Taenia solium*, *Toxoplasma*, CMV, HIV-1/2, HTLV-1/2 must also be evaluated for. Micro and macrovascular complications of DM should be evaluated simultaneously.

Ultrasound can be initial non-invasive imaging modality that can be used to rule out abscess, DVT, or subcutaneous edema. Venous and arterial doppler should always be done to rule out DVT and peripheral arterial disease. DMN on ultrasound appears as a well marginated hypoechoic intramuscular lesion. The additional sonographic features include: Internal linear structures that are compatible with muscle fibres coursing through the lesion; the lack of a predominantly anechoic region; and an absence of motion or swirling of fluid with transducer pressure [23]. Predominantly anechoic areas are seldom seen. Posterior acoustic enhancement may be seen if the lesions are not in close proximity to the bone cortex. Doppler study may not demonstrate increased flow in the lesions. CT of the involved muscle may not give much additional information but contrast enhanced CT may show low attenuation lesions with ring enhancing margins localized to group of muscles involved [23].

Magnetic resonance imaging (MRI) is a very effective diagnostic tool to diagnose DMN (**Figures 1** and **2** represents typical findings).

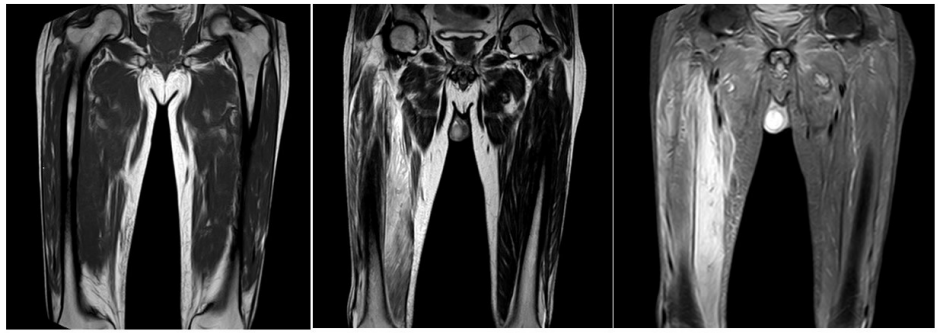

Figure 1. Coronal images of Vastus medialis muscle which shows hypointense on T1W and hyperintense in the T2W and STIR.

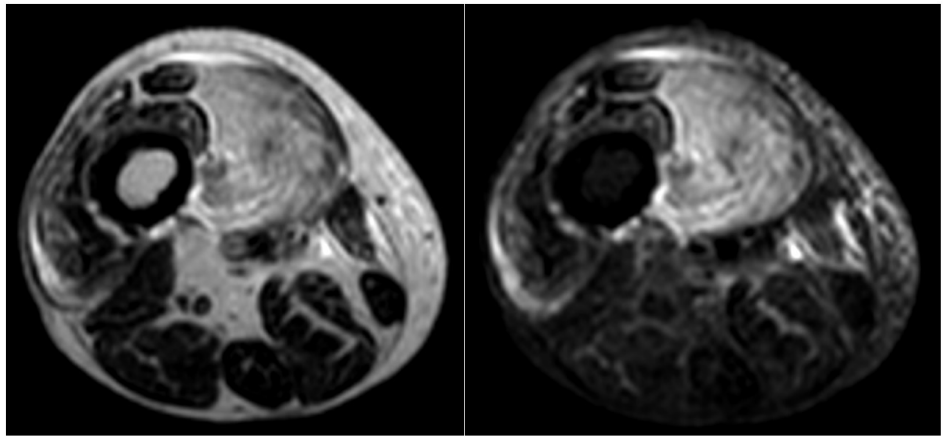

Figure 2. Axial images - T2W and STIR images show increased signal intensity of vastus medialis muscle belly. Mild fascial edema and subcutaneous edema can also be appreciated in the STIR images in both the images.

MRI, with IV Gadolinium as contrast is the choice for imaging. It shows well defined regions with iso to low signal intensity on T1-weighted images and predominantly high signal intensity on T2 weighted STIR or Proton density weighted with fat saturation images [6,19,24-26]. Subcutaneous edema, Subfascial hyperintensity and fluid are present in majority. Administration of IV contrast demonstrates peripheral rim enhancement in the affected regions. Biopsy is the gold standard, although it is not considered routinely due to its invasiveness and several negative effects such as delayed tissue healing, hematoma formation, and superimposed infections in diabetic patients with poorly controlled sugar levels [27]. When performed, it reveals necrosis, edema, lymphocytic infiltration, and thrombosis of microvasculature and surrounding areas of fibrosis [28]. The necrotic muscle is replaced with fibrous tissue followed by lymphocytic infiltration and muscle regeneration in later stages. It is considered when the levels of suspicion of DMN are very low and other diagnostic and empirical therapeutic interventions have failed to alleviate the condition. However, with the MRI findings, infectious myositis or pyomyositis, necrotizing fasciitis, or focal inflammatory myositis must continue to be kept in mind [29]. Focal inflammatory myositis can be ruled out with a negative screen for anti-nuclear antibodies, ANCA, anti-DNA abs, anti-smooth muscle abs, and anti-phospholipid abs.

Previous Studies of DMN (Table 1)

One of the earliest reviews by Trujillo *et al.* included 115 patients with 166 episodes from 47 references from the literature [6]. Fifty-nine percent had T1DM with mean age at the time of presentation was 42.6 years of which and mean duration of diabetes was 14.3 years. Micro-vascular complications of diabetes like nephropathy (71.1%), retinopathy (56.6%), and neuropathy (54.5%) were also present. In this series also DMN most frequently affected the thigh (83.7%). Quadriceps muscles were the most commonly affected of which vastus lateralis (24%) and the vastus medialis (22%) were most frequently affected. While the calf involvement was reported in 32 cases (19.28%) and bilateral in 14 cases (8.4%). Only one case involving upper limb (forearm) was reported.

Horton *et al.* did a recent systemic review of 126 cases of DMN from 87 articles [3]. The mean age of presentation was 44.6 years (20–67) and it was 35.9 years for T1DM and 52.2 for T2DM. The mean DM duration at diagnosis was 18.9 (5–33) years for T1DM and 11.0 (1–25) years for T2DM. The mean HbA1c in 51 cases was 9.34% (5–21). Of the 126 cases, 117 had other vascular complications with most common was nephropathy which was present in 75% of cases. Fifty-five cases (46.6%) had concurrent retinopathy, nephropathy, and neuropathy. Thigh pain/swelling was most commonly reported presenting symptom in 79 cases (71.2%), Calf pain/swelling and upper arm pain/swelling were noted in 17 (15.3%) and in six (5.4%) cases respectively. Thirty-four point nine percent of recurrence were noted amounting to 44 episodes of which 61.4% were noted at different location/muscle group other than the initial presentation.

Yong *et al.* systematically reviewed the cases of DMN in end stage renal disease [22]. Forty-one patients of CKD were included which were described in 24 articles. The mean age at presentation was 44.2 years and 22 were women (53.7%). T2DM was present in 53.7% of patients. Of the 41 patients, 60.1% were on hemodialysis, 21% on peritoneal dialysis, and 12.2% had renal transplantation. The proximal lower limb musculature was the most commonly affected site with muscle pain and swelling being the most frequent manifestation. Sixteen cases (39.0%) underwent muscle biopsy. More than 60% had HbA1c of greater than 7.

Meher *et al.* described six cases of DMN of which four patients were women [23]. The mean age at presentation was 51.5 ± 7.2 years (range 37–56 years) All patients had T2DM with mean duration of diabetes of 10.16 ± 8.23 years (range 1–25 years). Neuropathy and dyslipidaemia were present in all cases. Nephropathy was present in five cases, and retinopathy in four cases. All six patients had involvement of the quadriceps, and one had additional calf muscle involvement. One each had bilateral involvement and recurrence in the opposite thigh after 2 years initial presentation.

Nalloor *et al.* described seven episodes in six patients. They had a median age of 55 years (range 43 – 71 years) [30]. All had T2DM with median duration of 10 years (range 10–20 years). The median HbA1c was 7.3 (6.3-8.8%). They presented with acute, non-

Table 1. Previous studies of DMN.

	Santos *et al.* [6]	Horton *et al.* [3]	Yong *et al.* [22]	Meher *et al.* [23]	Nalloor *et al.* [30]
Type of article	Literature review	Literature review	Literature review	Case series	Case series
No. of patients	115	126	41 with ESRD	6	7
Mean age at the time of presentation (years)	42.6	44.6	44.2	51.5±7.2	54
Mean duration of DM (years)	14.3	T1 DM- 18.9 T2DM- 11	T1 DM- 17.3 T2DM- 15.7	10.16±8.23	11.8
Type of Diabetes Mellitus	Type 1 > Type 2	Type 2 > Type 1	Type 2 > Type 1	Type 2	Type 2
Mean HbA1c value	-	9.34%	7.2%	-	7.4%
Mean CRP mg/dl	-	15.6	15.36	-	32.34
Vascular complications	Nephropathy (71.1%), Retinopathy (56.6%), Neuropathy (54.5%)	Nephropathy Retinopathy Neuropathy	Nephropathy (100%), Retinopathy (58.5%), Neuropathy (56.1%)	Neuropathy + diabetic dyslipidemia, Retinopathy, Nephropathy	Nephropathy Retinopathy Neuropathy
Bilateral involvement	8.4%	-	-	16.6%	-
Recurrence	47.82%	50% post surgery	43.9%	16.6%	14.2%

traumatic pain with swelling affecting the thigh region. Microvascular complications in the form of either retinopathy, nephropathy, and/or neuropathy were noted in all patients. Creatinine and ESR levels were raised in all 7 cases. Five of them were known cases of chronic kidney disease and another 2 were newly detected. Recurrence was present in 1 patient in the contra-lateral limb within a year of first episode. None of them had upper limb involvement. All cases were treated conservatively with bed rest, analgesics, glycemic control, diuretics with supportive measures without any surgical intervention. Two weeks was the mean time taken for recovery.

Management and Prognosis

Diabetic myonecrosis is a self-limiting condition that resolves spontaneously over a period of time usually 2-4 weeks in most of the cases. Patients should be treated conservatively along with supportive care with good glycemic control. NSAIDs such as aspirin can be used for pain relief. As the possible explanation for DMN is microvascular pathogenesis, anti-platelet therapy is theoretically feasible [31]. Kapur's three treatment strategy is supportive (bed rest, analgesics), medical (anti-platelet therapy, steroids), and surgical treatment may be options [32]. The meantime recovery for the 3 strategies were 5.5, 8.1, and 13 weeks, making surgery an unfavourable modality [32]. Hence, the study by Kapur *et al.* also concluded excisional biopsy, early debridement, mobility, to all have led to complications and delayed recovery. Thus, the leading treatment is simply supportive. Surgical intervention must be limited if the condition is complicated with abscess formation or peripheral arterial compromise. Although DMN has a good short-term prognosis, there is a high chance of recurrence either in the same limb or the other. The likelihood of recurrence in the same muscle or contralateral limb is greater than 50%, with as many as 1 to 2 episodes per year after the initial event. Long term survival in patients who are known to have micro/macrovascular complications is usually poor.

Novel Concept—Sirtuins and DM Myonecrosis [33–38]

Sirtuins are a family of conserved, NAD+-dependent protein deacetylases that play a role in various biological processes. Their involvement in various cellular processes include metabolism, stress resistance, genomic stability, DNA repair, aging, and disease. In the context of skeletal muscle, their roles have gained considerable attention owing to involvement in metabolic regulation, developmental processes, and implications in numerous muscular disorders. Mammals have seven sirtuin isoforms, SIRT1–7, each with unique functions and subcellular localization. Sirtuins, especially SIRT1, play a role in maintaining cellular homeostasis and are implicated in various diabetic complications. Studies have shown that SIRT1 is often downregulated in diabetic conditions, and its activity is reduced in diabetic nephropathy. It has also been linked to the prevention of muscle loss and the maintenance of muscle function during aging. Sirtuins, especially SIRT1, are involved in regulating glucose uptake, fatty acid oxidation, and mitochondrial dynamics within skeletal muscle.

Measuring plasma SIRT1 levels in patients with diabetic myonecrosis is crucial for several reasons: it can help in early diagnosis and management of the condition, predict the progression of the disease, and potentially guide treatment strategies to prevent muscle necrosis and reduce the risk of metabolic complications. Measuring plasma SIRT1 levels in patients with diabetic myonecrosis can provide insights into the pathogenesis of the disease and help in early diagnosis and management. It may also guide to strategize the treatment to prevent muscle necrosis and reduce the metabolic complications.

Conclusion

DMN has a good short time prognosis with recovery in many cases within 3-4 weeks, albeit physicians must keep alert to identify this under-diagnosed condition at the earliest to prevent morbidity. Given its rarity, our knowledge of pathogenesis and causes of DMN and so the optimal treatment for it, remains unclear to this date, and therefore further studies are essential, to enhance our understanding of this uncommon complication of diabetes. The specific role of sirtuins in diabetic myonecrosis is not well-established. It needs further studies to look into metabolic complications of diabetes including micro/macrovascular complications and skeletal muscles.

References

1. Diabetes [Internet]. [cited 2025 Mar 13]. Available from: https://www.who.int/news-room/fact-sheets/detail/diabetes
2. Buell KG, Ajmal SR. Diabetic Myonecrosis: A Poorly Recognized Complication in an Adult With Type 2 Diabetes. Can J Diabetes. 2020 Apr;44(3):213–5.
3. Horton WB, Taylor JS, Ragland TJ, Subauste AR. Diabetic muscle infarction: a systematic review. BMJ Open Diabetes Res Care. 2015 Apr 24;3(1):e000082.
4. Angervall L, Stener B. Tumoriform focal muscular degeneration in two diabetic patients. Diabetologia 1965; 1:39–42.
5. Lawrence L, Tovar-Camargo O, Lansang MC, Makin V. Diabetic Myonecrosis: A Diagnostic and Treatment Challenge in Longstanding Diabetes. Case Rep Endocrinol. 2018 Aug 12;2018:1723695.
6. Trujillo-Santos AJ. Diabetic muscle infarction: an underdiagnosed complication of long-standing diabetes. Diabetes Care. 2003 Jan;26(1):211–5.
7. Ivanov M, Asif B, Jaffe R. Don't Move a Muscle: A Case of Diabetic Myonecrosis. Am J Med. 2018 Nov;131(11):e445–8.
8. Differentiation of Diabetes by Pathophysiology, Natural History, and Prognosis | Diabetes | American Diabetes Association [Internet]. [cited 2025 Mar 14]. Available from: https://diabetesjournals.org/diabetes/article/66/2/241/35290/Differentiation-of-Diabetes-by-Pathophysiology.
9. Noor S, Zubair M, Ahmad J. Diabetic foot ulcer--A review on pathophysiology, classification and microbial etiology. Diabetes Metab Syndr. 2015 Jul-Sep;9(3):192–9.
10. Choudhury BK, Saikia UK, Sarma D, Saikia M, Choudhury SD, Bhuyan D. Diabetic myonecrosis: An underreported complication of diabetes mellitus. Indian J Endocrinol Metab. 2011 Jul;15(Suppl 1):S58–61.
11. Rocca PV, Alloway JA, Nashel DJ. Diabetic muscular infarction. Semin Arthritis Rheum. 1993 Feb;22(4):280–7.
12. Virally M, Laloi-Michelin M, Médeau V, Meas T, Kévorkian JP, Mouly S, et al. Muscle infarction in a young woman with brittle type 1 diabetes. Diabetes Metab. 2007 Dec;33(6):466–8.
13. Marfella R, D'Amico M, Di Filippo C, Piegari E, Nappo F, Esposito K, et al. Myocardial infarction in diabetic rats: role of hyperglycaemia on infarct size and early expression of hypoxia-inducible factor 1. Diabetologia. 2002 Aug;45(8):1172–81.
14. Tofade TS, Liles EA. Intentional overdose with insulin glargine and insulin aspart. Pharmacotherapy. 2004 Oct;24(10):1412-8.
15. Singhal PC, Kumar A, Desroches L, Gibbons N, Mattana J. Prevalence and predictors of rhabdomyolysis in patients with hypophosphatemia. Am J Med. 1992 May;92(5):458–64.
16. Joshi T, D'Almeida E, Luu J. Diabetes myonecrosis - A rare complication. Diabetes Res Clin Pract. 2015 Sep;109(3):e18–20.
17. Mukhopadhyay P, Barai R, Philips CA, Ghosh J, Saha S. An unusual case of myonecrosis. Case Rep Endocrinol. 2011;2011:624020.
18. Rastogi A, Bhadada SK, Saikia UN, Bhansali A. Recurrent diabetic myonecrosis: a rare complication of a common disease. Indian J Med Sci. 2011 Jul;65(7):311–5.
19. Gupta RD, Haobam SS, Krishna A, Ramchandran R, Satyaraddi A, Shetty S, et al. Clinico-radiological characteristics and not laboratory markers are useful in diagnosing diabetic myonecrosis in Asian Indian patients with type 2 diabetes mellitus: A 10-year experience from South India. J Family Med Prim Care. 2018 Nov-Dec;7(6):1243–7.
20. Ahmad F, Michalski J, Winterton M, Simcock X, Wysocki RW. Spontaneous Diabetic Myonecrosis Presenting as Acute Carpal Tunnel Syndrome. J Hand Surg Glob Online. 2021 Nov 18;4(1):53–6.
21. Nigwekar SU, Kroshinsky D, Nazarian RM, Goverman J, Malhotra R, Jackson VA, et al. Calciphylaxis: risk factors, diagnosis, and treatment. Am J Kidney Dis. 2015 Jul;66(1):133–46.
22. Yong TY, Khow KSF. Diabetic muscle infarction in end-stage renal disease: A scoping review on epidemiology, diagnosis and treatment. World J Nephrol. 2018 Mar 6;7(2):58–64.
23. Dayanidhi Meher, Vivek Mathew, Raiz Ahmed Misgar, Satinath Mukherjee, Subhankar Chowdhury, Jyothi Chowdhury; Diabetic Myonecrosis: An Indian Experience. Clin Diabetes 1 April 2013; 31 (2): 53–8.
24. 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;174(1):165–9.
25. Weisman BN, Wong MW, Smith DN. Imaging interpretation session:1996. Radiographics. 1997;17:243–67.
26. Khoury NJ, el-Khoury GY, Kathol MH. MRI diagnosis of diabetic muscle infarction: report of two cases. Skeletal Radiol. 1997 Feb;26(2):122–7.
27. Nagdev A, Murphy M, Sisson C. Bedside ultrasound for the detection of diabetic myonecrosis. Am J Emerg Med. 2008 Oct;26(8):969.e3–4.
28. Bhasin R, Ghobrial I. Diabetic myonecrosis: a diagnostic challenge in patients with long-standing diabetes. J Community Hosp Intern Med Perspect. 2013 Apr 17;3(1):20494.
29. Schmid MR, Kossmann T, Duewell S. Differentiation of necrotizing fasciitis and cellulitis using MR imaging. AJR Am J Roentgenol. 1998 Mar;170(3):615–20.
30. Nalloor S, Pooja RP, Prasad BNS, Vijay HV, Chandrashekar P. Diabetic Myonecrosis: A Single-Center Experience. Indian Journal of Rheumatology. 2023;18(4):p 330–5.
31. Khan S, Al-Riyami D, Al-Ghailani M, Al-Dhuhli H. An Under-recognized Complication: Diabetic Myonecrosis. Oman Med J. 2009 Jul;24(3):228–30.
32. Kapur S, McKendry RJ. Treatment and outcomes of diabetic muscle infarction. J Clin Rheumatol. 2005 Feb;11(1):8–12.
33. Imai S, Guarente L. NAD+ and sirtuins in aging and disease. Trends Cell Biol. 2014 Aug;24(8):464–71.
34. Martins IJ (2017) Single Gene Inactivation with Implications to Diabetes and Multiple Organ Dysfunction Syndrome. J Clin Epigenet. 3:24.E
35. Martins IJ. Evaluation of diagnostic tests in human health and disease. J Clin Path Lab Med. 2018;2(1):13–5.
36. Morigi M, Perico L, Benigni A. Sirtuins in Renal Health and Disease. J Am Soc Nephrol. 2018 Jul;29(7):1799–809.
37. Gibril BAA, Xiong X, Chai X, Xu Q, Gong J, Xu J. Unlocking the Nexus of Sirtuins: A Comprehensive Review of Their Role in Skeletal Muscle Metabolism, Development, and Disorders. Int J Biol Sci. 2024 Jun 3;20(8):3219–35.
38. Kume S, Uzu T, Kashiwagi A, Koya D. SIRT1, a calorie restriction mimetic, in a new therapeutic approach for type 2 diabetes mellitus and diabetic vascular complications. Endocr Metab Immune Disord Drug Targets. 2010 Mar;10(1):16–24.