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Endocrinology

DIABETIC MUSCLE INFARCTION: THE GREAT IMPERSONATOR

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ABSTRACT Till date, there is no clear consensus guideline on how to treat diabetic muscle infarction (DMI), a rare and under-recognized complication associated with poorly managed diabetes mellitus, till date. It presents as sudden onset muscular pain and swelling, especially in inferior extremities. Magnetic resonance imaging (MRI) with Short TI Inversion Recovery (STIR) sequences is investigation of choice. Non-steroidal anti-inflammatory drugs, rest and adequate control of blood sugar serve the purpose on most occasions. Invasive interventions for diagnostic or therapeutic purpose should be avoided and long term prognosis is malicious. We are hereby reporting a case of DMI from a rural based tertiary care setup which raised considerable diagnostic dilemma.

KEYWORDS : Diabetic muscle infarction, MRI, STIR sequences, diagnostic dilemma

INTRODUCTION:

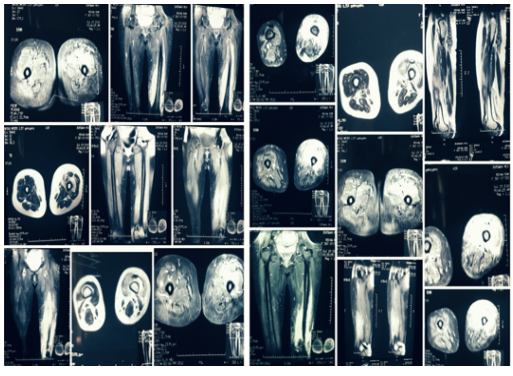
Diabetic muscle infarction (DMI) or myonecrosis is a rare (less than 200 cases have been reported in literature) microangiopathic complication of poorly controlled long standing diabetes. DMI is usually associated with the other components of the ominous octet of diabetes. It is frequently misdiagnosed clinically and treated as diabetic cellulitis, polymyositis, pyomyositis. But with characteristic clinical manifestations and imaging, it can be accurately diagnosed and treated, thus avoiding unnecessary investigations and interventions. [1] A case of DMI involving the left quadriceps group of muscles in a diabetic patient is being reported here.

CASE REPORT:

An-38- year old type-2 diabetic lady presented with excruciating pain and swelling of lower portion of left thigh in the out-patient department which started almost two weeks back and was gradually intensifying until the previous night when it got worst and made her to wake up. The pain was severe enough to cause mobility restriction. She denied any fever, trauma or administering insulin injections into her thighs. On examination, she was afebrile normotensive. On local examination left thigh was swollen (difference in circumference of 10 cm between left and right) maximum on anterolateral aspect without any erythema and cellulitis but was warmer than right thigh. The area was indurated and was pretty tender to touch. Fundus examination showed background diabetic retinopathy changes (non- proliferative type). Peripheral pulses were equally and symmetrically palpable. There were no muscle fasciculations and atrophy. There was no evidence of neurovascular compromise. Neurological examination did not reveal any abnormality suggestive of distal symmetrical polyneuropathy and amyotrophy.

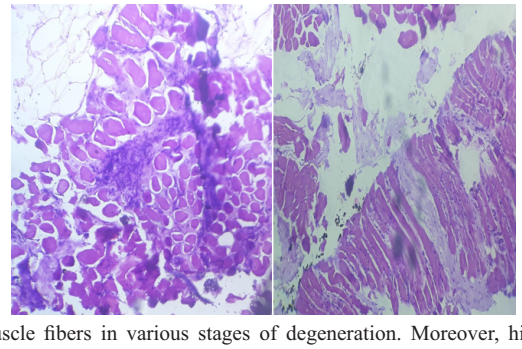
Based on this history and clinical findings, a list of differential diagnoses were made: Diabetic Cellulitis, Soft-tissue abscess, Pyomyositis, Necrotizing fasciitis, Osteomyelitis with soft-tissue extension, Diabetic muscle infarction, Dermatomyositis, Deep venous thrombosis (DVT), Compartment syndrome, vasculitis, primay muscle neoplasms. However, from the history and clinical examinations that revealed abrupt onset unbearable pain and swelling without weakness in the thigh, any signs of local and systemic inflammation, any evidence of neurovascular compromise, increment in CPK, in a long-standing, poorly-controlled diabetic woman having underlying micro vascular complications, we kept diabetic muscle infarction as the most tentative clinical diagnosis.

Her complete metabolic panel revealed no abnormality except noticeably high levels fasting and post-prandial blood glucose and mixed dyslipidemia. HbA1c level was 13.4 %. Repeated Urinary Albumin-Creatinine Ratio (UACR) reports were normal. X-Ray of left thigh excluded bony neoplasm and osteomyelitis. Compressive Ultrasonography of left thigh was suggestive of inflammatory changes in deeper muscle plane in absence of any cellulitis and abscess or neoplastic entity. Colour Doppler study of bilateral lower limb vessels excluded possibilities of deep venous thrombosis (DVT) on more than one occasion. MRI of left thigh showed (just below)



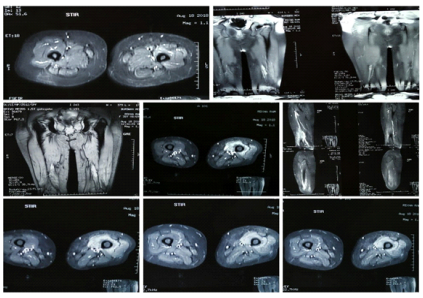
high T2 signal and hypodense signal on T1 images of left thigh characteristic of fluid affecting subcutaneous and quadriceps with maximum involvement of vastus lateralis muscle of left thigh and. On i.v. gadolinium there was uniform enhancement of the muscle with central non-enhanced area of necrotic muscle. Repeated body fluid cultures negated possibilities of infectious etiology. 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 were unremarkable. Interestingly enough, levels of serum creatine phosphokinale (CPK), Aldolase were never increased.

A Trucut biopsy was done which showed (just below)



muscle fibers in various stages of degeneration. Moreover, highly characteristic MRI findings further support the clinical diagnosis. The patient was treated conservatively with bed rest, analgesics and

insulin. She started showing improvement and was discharged after 45 days and the follow up MRI (see below) documented near-total resolution of the lesion.



DISCUSSION:

DMI first diagnosed by Angervall and Stener in 1965, is a very uncommon complication of diabetes and predominately occurs in type 1 diabetes (70% of cases) or long standing poorly controlled type 2 diabetic patients. [2], [3] It usually presents acutely as a confined exquisitely painful swelling of the involved muscle, associated with restricted range of motion without redness and systemic signs of infection. [4] DMI most commonly affects quadriceps (involved in 84% of cases, particularly Vastus lateralis) and calves (in about 19% of cases). [3] Bilateral involvement of thighs is seen in 8 to 30% of cases. [3],[4],[5] DMI occurs more frequently in poorly controlled diabetic women having other underlying micro vascular complications. [6] Albeit exact pathogenesis is unknown, a diffuse microangiopathic process resulting into hypoxia-reperfusion injury seems most likely etiology. [3],[6]

However, inflammatory conditions such as polymyositis and pyomyositis present with proximal muscle pain and swelling and raised creatine kinase are the most common clinical entities to be differentiated from DMI. [7] The diagnosis of diabetic muscle infarction is clinico-radiological. The clinical possibility should be entertained in a long standing poorly controlled diabetic patient who presents with abrupt onset of exquisitely painful and tender swelling of thighs without erythema and cellulites with mild or no rise of creatine kinase. [3],[7] In radiology, MRI of the extremity is the modality of choice, characteristically shows an increased signal from affected muscle (intramuscular and perimuscular area) and subcutaneous fat on T2 weighted images and isointense or hypointense areas on T1 weighted images, highly suggestive of fluid from edema and inflammatory changes. [5],[6],[8] And Gadolinium enhancement characteristically show enhanced margin of the infracted muscle with central nonenhanced area of necrotic tissue. [5],[6] Muscle biopsy may prolong the recovery it is not needed routinely, it indicates only if diagnosis is uncertain, response to treatment is poor and presentation is atypical. [4], [9] The biopsy when done shows an areas of muscle necrosis and edema surrounded the muscle fibers, which are in various stages of degeneration and regeneration with hyalinosis and arterioles thickening. [2],[6] Conservative management in the form of bed rest, leg elevation and good analgesia is the mainstay of treatment as no evidence based recommendation is available. [10] Exercise during acute phase may increase pain and swelling, hence should be avoided. Poor diabetic control prolongs the recovery, strict diabetic control is important. No evidence based recommendation support the use of steroid. [7] There is no role of surgery in diabetic muscle infarction. In fact surgery may worsen the outcome, should be avoided. [7] In acute phase the recovery is good but the recurrence is high (48%), which may affect other muscle in 39% of cases. [10] Long term prognosis is not good in diabetic muscle infarction, with 10% mortality over next two years. [10]

To conclude, diabetic muscle infarction is an extremely rare but noteworthy complication of diabetes mellitus with an overall sinister long-term outcome. With the increasing prevalence of diabetes mellitus in our country, the incidence of diabetes muscle infarction can be expected to increase. Clinicians need to have a high index of suspicion to recognize this rare condition. With a good clinical history and timely imaging studies, the diagnosis of this rare condition can be made with confidence leading to cost-effective appropriate intervention and better outcomes.

REFERENCES:

1. Kiers l. Diabetic muscle infarction: magnetic resonance imaging avoids the need for biopsy. Muscle nerve 1995;18:129-30.
2. Angervall l, Stener b. Tumoriform focal muscular degeneration in two diabetic patients. Diabetologia 1965;1:39-42.
3. Trujillo-Santos. Diabetes muscle infarction: an under diagnosed complication of long standing diabetes. Diabetes care 2003;26:211-5.
4. Aboulafia aj, monson dk, kennon re. Clinical and radiological aspects of idiopathic diabetic muscle infarction. J bone joint surg br 1999;8:323-6.
5. Jelinek js, murphey md, aboulafia aj, dussault rg, kaplan pa, snearly wn, et al. Muscle infarction in patients with diabetes mellitus: mr imaging finding. Radiology 1999;211:241-7.
6. Grigoriadis e, fam ag, staok m, ang lc. Skeletal muscle infarction in diabetes mellitus. Rheumatology 2000;27:1063-8.
7. Parmar ms. Diabetic muscle infarction: lesson of the week. Bmj 2009;339:171-2.
8. Gee r, munk pl, o'counnell jx, chan lp, keogh c, luck l, et al. Diabetes muscle infarction. Can assoc radiol j 2003;54:296-8.
9. Chester cs, banker bq. Focal infarction of muscle in diabetes. Diabetes care 1986;9:623-30.
10. Kapur s, mackendry rj. Treatment and outcome of diabetic muscle infarction. J clin rheumatol,2005;11:8-12.