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DIABETIC MUSCLE INFARCTION (MYONECROSIS)

General Medicine

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ABSTRACT

Diabetic muscle infarction is a rare complication of diabetes. It is seen more in Type 1 DM than Type 2 DM, but in both it is associated with longer duration of diabetes, poor glycemic control with or without microvascular complications.

KEYWORDS

INTRODUCTION

Diabetic muscle infarction is a rare complication of diabetes. It is seen more in Type 1 DM than Type 2 DM, but in both it is associated with longer duration of diabetes, poor glycemic control with or without microvascular complications.

CASE REPORT

A 4 2 years old male patient , a k n o w n diabetic and hypertensive for 3 yrs, presented with complaints of generalised weakness. He had no other significant history. He was found to have high blood sugars (440mg/dl). On examination vitals were stable, all peripheral pulses were felt. Systemic examination was essentially normal and there was no other target organ involvement, clinically. Patient was admitted to wards and put on regular insulin and supportive treatment. The investigations revealed that the patient had already developed microvascular complications in the form of Grade 2 diabetic nephropathy and fundoscopy revealed NPDR. On 4th day of hospitalization, he complained of sudden onset of pain in left thigh. There was no preceding trauma or fever. Examination revealed localized swelling of left thigh region which was warm and tender with induration. There was no clinical evidence of deep vein thrombosis. Further investigation for the cause of swelling was undertaken. CPK was 371U/L (24-195) and LDH was 384U/L (90-200). USG of left thigh revealed a predominantly hypere choic collection with increased peripheral vascularity, in the rectus femoris and vastus laterals muscles. CECT of the left thigh showed bulky left vastus lateralis, parts of rectusfemoris and sartorius muscle with central nonenhancing regions (open arrow). These features were consistent with diabetic myonecrosis. The MRI of the left thigh showed hyperintensities in T2 and STIR images, mainly in vastus lateralis, parts of rectus femoris and sartorius muscle, which became hypointense on T1. Muscle biopsy of sartorius muscle confirmed the diagnosis, it showed skeletal muscle fibres with foci of myonecrosis, macrophage phagocytosis and inflammatory cell infiltrate and granulation tissue formation. Blood vessel appeared thickened and hyalinised with luminal narrowing suggesting arteriosclerotic changes

Investigation

Hb 14 Gms/dl TLC 11000 (P 80%, L 18%, E 2%) Platlets 2 Lacs, ESR 90mm/ 1st hour ,Urine KB negative FBS 323 mg/dl PPBS 541 mg/dl HbA1C 10.3% S. Urea- 38 mg/dl S. Creat- 1.1 mg/dl LFT- WNL Lipid proflie: TGs-125 mg/dl T.Chol- 232 mg/dl HDL- 47 mg/dl LDL- 160 mg/dl Urine Microalbuminuria 50.53 mg/dl (upto 30)

Diagnosis and treatment

A diagnosis of myonecrosis was made and the patient was advised with insulin for strict controlled of sugars, analgesics and adequate bed rest. Four weeks later the patient reported to have considerable reduction in pain, swelling and induration.

DISCUSSION

Diabetic muscle infarction (DMI) or diabetic myonecrosis is a rare complication of diabetes or possibly, an underdiagnosed complication. The first case was reported by Angervall and Stener in 1965. The exact prevalence of the disease is not known. In world literature there are less than 200 cases reported and from India only 5 cases have been reported.1 Considering the number of diabetics in our country this is an extremely small number. DMI is described in people with long-standing diabetes with a greater incidence in Type 1 diabetes (71%). However, patients with Type 2 DM also develop this complication.2 The clinical presentation is of an acute atraumatic painful swelling of the affected muscle and a palpable mass. Commonly the thigh muscles (quadriceps (62%), hip adductors (13%), hamstrings (8%), and hip flexors (2%) 3 are affected but the calf muscles can also get involved. One case of upper limb involvement has also been reported. Bilateral involvement has been reported in 8.4% cases.4 A differential diagnosis should be considered as these are of a more serious nature requiring active intervention. The use of USG and MRI ruled out these problems, in our case. Although the exact pathogenesis of muscle infarction in diabetes has not been identified, severe diabetic microangiopathy leading to occlusion of blood vessels and ischemia has been proposed as the underlying mechanism that leads to spontaneous nongangrenous and focalized muscle infarction. Other proposed theories include coagulation-fibrinolysis derangement, hypoxia-reperfusion abnormalities, and recently, the role of antiphospholipid antibodies.5 Diabetic myonecrosis is as poradic micro vascular complication comm only seen in patients with poor glycemic control. Invariably the patient will have microvascular complications of diabetes mellitus, such as nephropathy (71%), retinopathy (56%) and neuropathy (54%). Laboratory investigations may be unhelpful. Creatine phosphokinase levels are normal or slightly elevated in 52%; leucocytosis occurs in 14% of cases. AST, ALT, LDH levels and the erythrocyte sedimentation rate are normal most times. In our case there was marginal elevation of both CPK and LDH levels while the leukocyte count and ESR were elevated. Sonography aids in detecting venous thrombosis, if any. In diabetic muscle infarction it shows internal linear echogenic structures coursing through the lesion; an absence of internal motion or swirling of fluid with transducer pressure; and lack of predominantly anechoic areas. CT scan shows diffuse muscular enlargement with diminished attenuation of the affected muscle, increase dattenuation of the subcutaneous fat, and thickening of the subcutaneous fascial planes and of the skin.4 MRI is the best investigation for diagnosis.4 The characteristic features of diabetic myonecrosis include increased signal from the affected muscle area in T2- weighted, inversion-recovery, and gadolinium-enhanced images and isointense or hypointense areas on T1 -weighted images. Muscle biopsy can firmly ascertain the diagnosis, however it is routinely not recommended in view o f post - procedural complications that delay in wound healing. In our case, the demonstration on muscle biopsy of arteriosclerotic changes shows that diabetic microangiopathy is the cause. Manage ment is mainly conservtive. Goodglycemic control should be achieved and patients usually recover within few weeks with supportive treatment like bed rest and analgesics for pain. The long-term prognosis of this complication is poor in the form that most patients die within 5 years of diagnosis.6 The main underlying difficulty is advanced generalized microvasculopathy leading to lifethreatening complications. Up to 50% of cases may have a recurrence, most of them in a previously affected muscle. Almost half of these recurrent events occur in a period of two months.

CONCLUSION

Recognition of this complication is essential as it carries a poor long-term prognosis. Early intervention in the control of diabetes, can probably lead to better outcomes. We therefore, feel that DMI is an underdiagnosed problem and creating awareness a m o n g c l i n i c i a n s m a n a g i n g diabetes would go a long way in reducing the morbidity and mortality associated with the microvascular complications of diabetes mellitus.

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