Case Report

SPONTANEOUS MUSCLE INFRACTION IN DIABETES MELLITUS

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INTRODUCTION :

This case report describes a middle aged female having a late onset of type I diabetes mellitus and presenting with pain and swelling in left thigh due to spontaneous skeletal muscle infraction.

CASE HISTORY

A 49 year old housewife presented with history of pain and swelling of left thigh of two months duration, gradual in onset. The pain was cramp like, present at rest but was worse on attention to move, stand or walk. External pressure caused excruciating tenderness. She required to assist herself with her left hand to bend the thigh or lift the leg. She had fever five days duration, mainly in the evening with rigors lasting for two to three hours. Appetite was poor. She complained of tiredness. There was no cough. There was no burning or painful urination. She gave no history of injury or any injections in the left thigh.

She was known diabetic of 11 years duration, poorly controlled (blood sugar levels most of the time more than 200 mg/dl) with various hypoglycemic agents and ayurvedic preparations. She was also being treated for hypertension in the last three months. She has not live in an area endemic for filariasis. She was a mother of two children, both delivered through a lower segment caesarian section. Menstrual cycle was regular 4-5/28 days, moderate flow.

PHYSICAL EXAMINATION

Height : 145 cms, Weight : 47 kgs Pulse : 86/min, BP 170/80mm of Hg, pallor present, febrile, no icterus, no cyanosis. There was no lymphadenopathy in the neck or groin. Heart, lugs, abdomen and central nervous system were all normal.

The left thigh was swollen with warmth of the overlying skin. There was no erythema. There was excruciating tenderness all over the swelling. The skin was indurated on medial and posterior aspects. The circumference of left thigh 15 cms above tibial tubercle was 41 cms, compared to 31 cms of the right thigh. There was mild edema of the left leg and a healed blister on the left great toe. Arterial pulsations including dorsalis pedis were felt bilaterally. No varicosities were observed. On pelvic and rectal examination no abnormality was detected.

INVESTIGATIONS :

Creatine phosphokinase 114 units, Hb 9 g%. Total WBC count 14,400 c.mm, Band 5%, P 85% (Neutrophils) l 8% M 2%, ESR 150m/1st hour, Urine Analysis : Albumin ++, No acetone. Peripheral smear showed hyersegmented polymorphoneutrophils with toxic granulation, consistent with macrocytic anemia.

Material parasite and filarial parasite were not seen in the peripheral blood smear. Blood urea nitrogen, serum creatinine, lipid profile, clotting time, bleeding time, partial thromboplastin time and platelets were all normal. Liver function test was unremarkable. S. calcium, phosphorus and acid phosphatase were all normal. Random blood glucose on admission was 390 mg%.

Mantoux test was negative after 48 hours, Chest X-ray was normal, X-ray left thigh:no abnormality detected, Ultrasonography of abdomen was reported as : nephropathy Grade-1, cholelithiasis and fibroid uterus. Ultrasonography of left thigh showed thickness of soft tissue with no definite collections. Arterial and venous Doppler were normal.

Magnetic resonance imaging (MRI) was reported as "T1 weighted images show vastus medialis, intermedius, lateralis, rectus femoris, sartorius and tensor fasciae lata to be bulky. They appear iso to hypointense on T 1 weighed images and turn hyperintense on T2 weighed inversion recovery fast spin echo (IRFSE) images. The subcutaneous tissue is also edematous appearing hyperintense on T2 weighed images." (figure 1 and 2).

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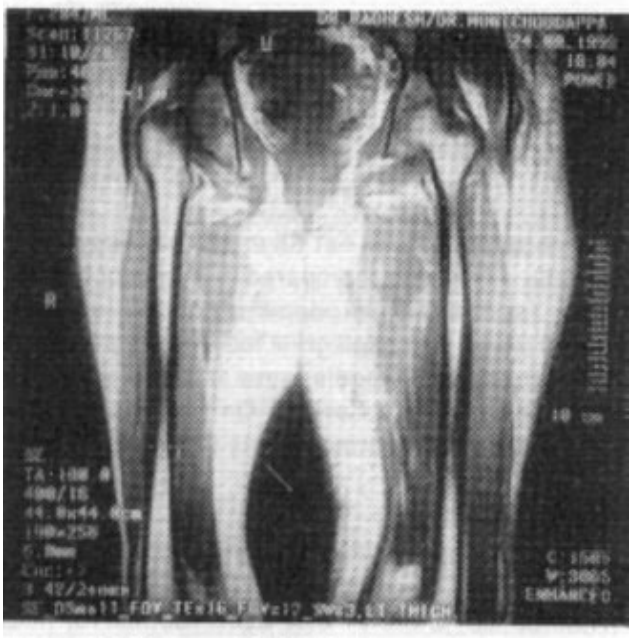

Fig 1 : MRI Scan of left thigh (saggital)

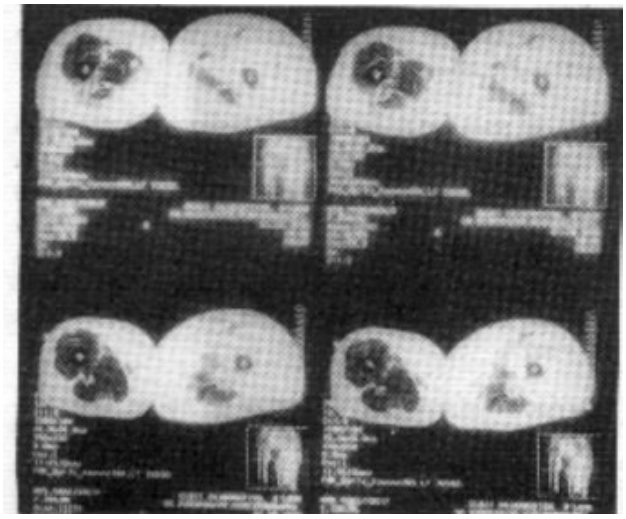

Fig 2 : MRI Scan of left thigh (Transverse)

HOSPITAL COURSE :

Fever subsided with ciprofloxacin. Diabetes was reasonably under control with pre-breakfast, pre-lunch short acting human insulin and pre dinner mixture of short and intermediate acting insulin. She was also started on corticosteriods (prednisolone) 20 mg three times daily with adequate insulin coverage.

The swelling and pain of the left thigh started diminishing. She was able to walk and was discharged. A week after her discharge the swelling and pain were considerably reduced. She was able to walk normally. The circumference of left thigh was reduced by 2.5 cms. Six weeks after starting corticosteroids and a week after stopping it, the left thigh circumference was 33 cms (8 cms less than intial). She was able to walk and was free from pain. She had gained 3 kgs, in weight. She was continued on insulin, haematinic's and antihypertensives.

DIFFERENTIAL DIAGNOSIS

Diagnosis of deep vein thrombosis, cellulitis, lymphoedema, osteomyelitis, primary or metastatic tumour and deep seated abscess were considered and excluded on clinical grounds and by relevant investigations.

DISCUSSION

Necrosis of skeletal muscle should be considered when a diabetic patient presents with a painful mass involving the skeletal muscles without wasting. This condition was first discribed as tumoriform focal muscle degeneration in two patients in 1965 (1). So far 25 such cases have been reported (2,3).

Muscle infarction is usually seen in type 1 diabetes mellitus of long duration with a female preponderance, offen associated with poor glycemic control and multiple microvascular complications (4). The thigh muscles are more frequently involved than the calf muscles. Recurrence on the same or the opposite side is noted in about half of the patients reported.

The cause for skeletal muscle necrosis is not clear. Embolisation from an atheromatous plaque in aorta or atherosclerotic vascular obstruction as a result of extensive arterial occlusive disease in skeletal muscle and other organs have been suggested to be responsible for muscle necrosis (2, 5). Hypercoaguable state is another suspect (6, 7).

The diagnosis is best established by MRI, (8, 9). A markedly increase signal intensity on T2 weighed images and hypointensity in T1 weighed images is characteristic. However MRI changes are not specific for diabetic muscle infarction (3). Autoimmune myopathies, some infection or inflammatory process may have identical signal intensity changes on MRI.

Muscle biopsy has shown hemorrhagic necrosis of skeletal muscle with connective tissue proliferation, small vessel occlusion, inflammatory cell infiltration and often degenerated peripheral nerves (2). However, biopsy should be avoided whenever possible, because of risk of producing further damage(2).

Treatment comprises, as in the case reported, of glycemic control, antibiotics and if necessary analgesics and corticosteroids. The painful lesion

typically resolves with this line of treatment after several weeks or months (10). Because of possible role hypercoaguable state, aspirin and anticoagulants are recommended but generally not accepted.

ANTERIOR COMPARTMENT SYNDROME

This is a related entity with devastating consequences. The anterior compartment syndrome could be unilateral or bilateral. It usually occurs as a result of trauma. There will be severe muscle ischemia leading to swelling within a closed fascial space (11, 12). A rise in tissue hydrostatic pressure results in compromised muscle blood flow, muscle nercrosis and limb dysfunction.

The affected muscle will become tense and is associated with serve pain. This situation demands emergency surgical fasciotomy.

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