Diabetic myonecrosis: a case report
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Patient: 55 years, male

Clinical History:

The patient was referred as a painful right thigh swelling increasing in size in the last 3 weeks. He is a known insulin dependant diabetic for the last 15 years. There was however no history of recent injection in the thigh and no history of previous malignancy.

Imaging Findings:

A 55 y.o. man presented with an increasing painful swelling in his right anterior thigh. He had diabetes mellitus for 15 years, treated by insulin injections, but not at the site of the swelling. He did not have a history of previous malignancy. Physical examination showed a tender swelling, mobile and not fixed to the underlying bone. The peripheral circulation was clinically normal. Laboratory data were normal, except a modestly elevated ESR (25 mm/h). The plain radiograph demonstrated slight displacement of the calcified femoral artery, but no bony abnormality. The US exam (Fig.1a&b) showed a focal mixed echogenicity muscle lesion with undefined margins gradually blending with more normal appearing muscle, and increased vascularity in the proximal aspect, suggestive of an inflammatory lesion; no frank fluid collection was detected. On the MR scan (Fig. 2a&b), the affected rectus femoris appeared swollen and showed a focal poorly defined area of increased signal intensity on T2-weighted and fat suppressed images with enhancement on the post contrast images (Fig.3a&b). There was a central reduced signal area, without enhance on the post contrast images in keeping with an area of necrosis. A small amount of peri-muscular fluid was detected. An open biopsy of rectus femoris muscle was performed. Histology revealed areas of chronic skeletal muscle necrosis replaced by collagenous tissue and also the evidence of recent fibrinoid necrosis, without evidence of pyogenic infection or granulomatous inflammation. The combination of clinical diabetes, imaging features and histology are in keeping with a diagnosis of diabetic myonecrosis.

Discussion:

Diabetic skeletal muscle infarction (DMI) is rarely reported in the literature as one of the complications of diabetes mellitus. Studies [1] proposed the hypothesis of ischemia due to arteriosclerosis and microangiopathy. Subsequent studies demonstrated significant occlusive peripheral vascular disease in the affected muscles (“arteriosclerosis obliterans”), which led to a mild compartment syndrome and worsening ischemia. Bjornskov et al. [2] demonstrated normal vascularity histologically in their cases and they hypothesized that the interaction of endothelial damage from microangiopathy, an activated coagulation cascade, and impaired fibrinolysis may collectively account for spontaneous infarction in patients with diabetes. As regards the laboratory examinations [6], the WBC is usually normal and helps the clinician to differentiate DMI from pyomyositis. Measurement of the serum muscle enzyme, creatine kinase (CK), yields conflicting results, and even a normal CK level does not exclude the diagnosis of DMI. Frequently the ESR is elevated. Though plain radiography, ultrasound and CT [3] scan serve to locate the site and extent of the lesion, the imaging features of these modalities are not specific. The sonographic features of DMI consist of a well-marginated hypoechoic intramuscular lesion with internal linear structures that are compatible with
muscle fibres coursing through the lesion. There is a lack of a predominately anechoic region and an absence of motion or swirling of fluid with transducer pressure. The presence of these three sonographic findings helps exclude a necrotic mass or abscess; but they are not absolutely specific [4]. In the literature [5], investigators have proposed that MR imaging, especially with administration of IV gadolinium, because of its excellent soft tissue contrast [4], is the study of choice to diagnose DMI. In most cases, on MRI, the affected muscles appeared enlarged and showed uniform increase in signal intensity on T2-weighted and IR images; this is the result of increased interstitial fluid content. On T1-weighted images the affected muscles have either normal or decreased signal intensity and muscle swelling is sometimes less appreciated on this sequence. Gadolinium-DTPA enhanced studies showed in some cases a uniform enhancement of affected muscles and in others rim-enhancing areas, probably representing necrosis. Perifascial and subcutaneous oedema can also be seen in patients with DMI and is best assessed on IR or T2-weighted fat-suppression sequences but it is a rather uncommon finding and, when present, it is usually minimal. The differential diagnosis of DMI based on these MR changes is limited to an infectious process, i.e. muscle abscess or pyomyositis and a biopsy may often be necessary. MRI is also useful in directing biopsy to the site with highest diagnostic yield. Treatment [7] consists of analgesics and short-term immobilization. Exercise and physical therapy exacerbates pain and extends infarction and thus is discouraged. Rest may be followed by gentle physical therapy. Myonecrosis alone is of little significance; risk of recurrence in the same or opposite leg is high. Diabetic myonecrosis is a reminder that aggressive diabetic control is essential in all patients to delay the progression of vascular disease and to avoid end-organ damage, morbidity, and death.

**Differential Diagnosis List:** Diabetic Myonecrosis

**Final Diagnosis:** Diabetic Myonecrosis

**References:**

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