Radiology for the surgeon

Musculoskeletal case 34

Presentation

A 49-year-old male patient with no history of trauma complained of pain around his left knee joint and general weakness in his legs. His medical history was remarkable only for long-standing inflammatory bowel disease, for which he had been taking courses of high-dose corticosteroids.

A physical examination revealed marked muscle wasting bilaterally in his lower limbs, but was otherwise unremarkable. After plain radiographs revealed no notable abnormality, magnetic resonance images were ordered.

T_1-weighted coronal images of his left side showed serpiginous areas of abnormality in the distal femur and proximal tibia, with hypointense rims (Fig. 1, Fig. 2). Although fast short-tau inversion-recovery (FSTIR) images confirmed these findings, they showed the surrounding rims as hyperintense (Fig. 3, Fig. 4). Axial T_1-weighted imaging confirmed marked muscle wasting, manifesting as decreased muscle bulk with interpositioning of high-signal fat in the muscle substance (Fig. 5).

What is the diagnosis?

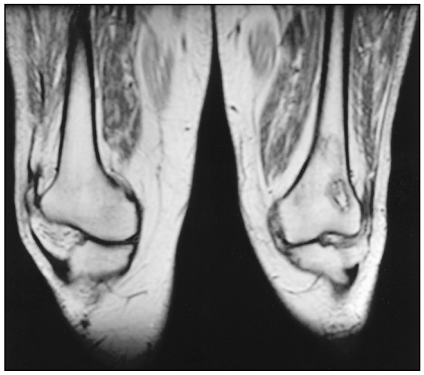

FIG. 1.

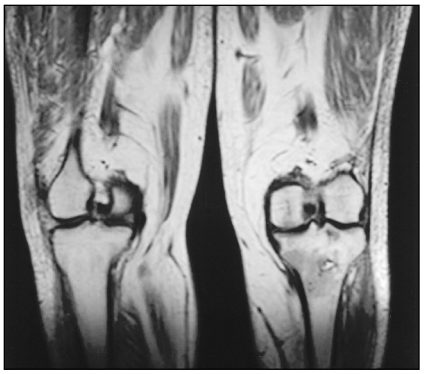

FIG. 2.

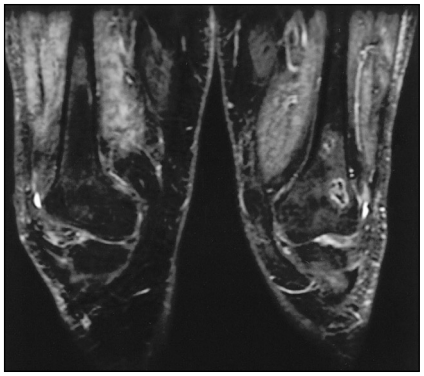

FIG. 3.

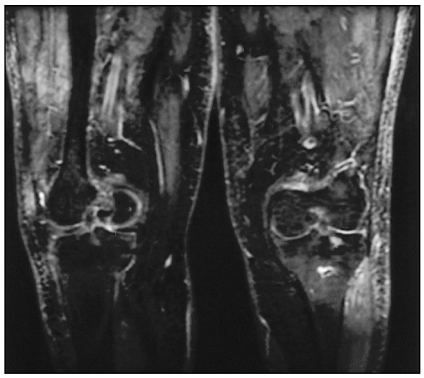

FIG. 4.

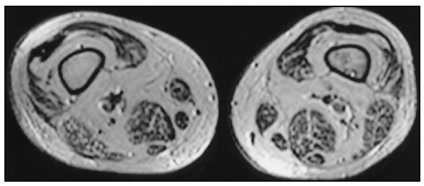

FIG. 5.

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Diagnosis

The side effects of corticosteroid agents are protean and well documented.[1] They include osteonecrosis and myopathy of skeletal muscle. Steroid-induced osteonecrosis most commonly affects the femoral head, but has also been well described in the distal femur and proximal tibia.[2]

The precise etiology of the condition remains unclear, although theories of fat embolism, intraosseous hypertension and increased blood coagulability have been advanced. Individual susceptibility is highly variable and no consistent correlation with dose has been observed.[3]

Symptoms of osteonecrosis generally precede even the earliest plain-film findings by several months. Isotope bone scanning provides an earlier indication of the disease process, but MRI is the gold standard both for early diagnosis and for more precise delineation of the process: MR scans often indicate a far more extensive area of infarction than demonstrated on plain film.[3]

Treatment of extensive disease such as that present in this case is likely to involve total joint replacement, but options for less extensive disease would include arthroscopic debridement, curettage or drilling of the lesion, bone grafting, high tibial osteotomy and osteochondral allograft.[4]

Steroid-induced myopathy is thought to arise primarily from muscle atrophy. Apoptosis of differentiated skeletal muscle cells has been demonstrated at a microscopic level, along with selective degeneration of myosin filaments and loss of thick myofilament.[5,6] Biopsy of affected muscle groups shows increased variation in muscle-fibre diameter, angular atrophic fibres and diffuse necrotic and basophilic fibres. There is increased connective tissue between fibres, reflecting atrophy. Special stains show that fibre atrophy predominantly affects fast fibres.[7]

The characteristic MR findings of reduced muscle bulk and markedly increased intermuscular fat reflect the atrophic process.

Competing interests: None declared.

References

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Correction

In our December issue, several references in the Quill on Scalpel item by Drs. Mark Bernstein, Joseph Bampoe and Abdallah S. Daar (Ethical issues in molecular medicine of relevance to surgeons. *Can J Surg* 2004;47:414–21) have been updated, as follows.

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