

Steroid-induced Extensive Bilateral Femoral and Tibial Bone Infarcts in a Patient with Ulcerative Colitis

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Citation

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Abstract

Osteonecrosis is a common complication of chronic or repetitive steroid treatment. A 55 year-old black man with a history of ulcerative colitis for 30 years, treated with multiple courses of steroids is presented in this clinical vignette. Multifocal extensive osteonecrosis was detected on imaging studies.

INTRODUCTION

Osteonecrosis is ischemic death of the cellular elements of the bone and bone marrow. In general, bone infarct refers to lesions occurring in the metaphysis and diaphysis of bone and lesions in the epiphysis are called avascular necrosis. Considerable lack of uniformity in terminology exists and these terms have been used interchangeably in the literature. [1]

Osteonecrosis has multifactorial etiology. Many pathological processes or conditions such as trauma, Legg-Calve-Perthes disease, sickle cell disease, renal transplantation, Cushing syndrome, Gaucher disease, Fabry disease, systemic lupus erythematosus, rheumatoid arthritis, scleroderma, infection, pancreatitis, pregnancy, gout, diabetes, use of immunosuppressant and other drugs (steroids, indomethacin, phenylbutazone), alcohol use, diving (dysbaric osteonecrosis), radiation therapy and arteritis may cause or predispose to osteonecrosis.

Here, we present a case of extensive bilateral femoral and tibial osteonecrosis, with a history of bilateral femoral head avascular necrosis diagnosed previously.

CASE PRESENTATION

A 55 year-old black man with a history of ulcerative colitis for 30 years was admitted to our acute inpatient rehabilitation unit after left total knee replacement. In the past, he was treated multiple times with steroids for flairs of his ulcerative colitis. Review of the preoperative images revealed extensive bilateral femoral and tibial bone infarcts involving epiphyses, metaphyses, and diaphyses. (Fig. 1, 2

and 3) He was also diagnosed with bilateral femoral head avascular necrosis a few years back by magnetic resonance imaging. Further investigation did not reveal history of sickle cell disease or any other predisposing conditions for osteonecrosis. Laboratory studies showed normal mean corpuscular volume.

X-RAY FINDINGS

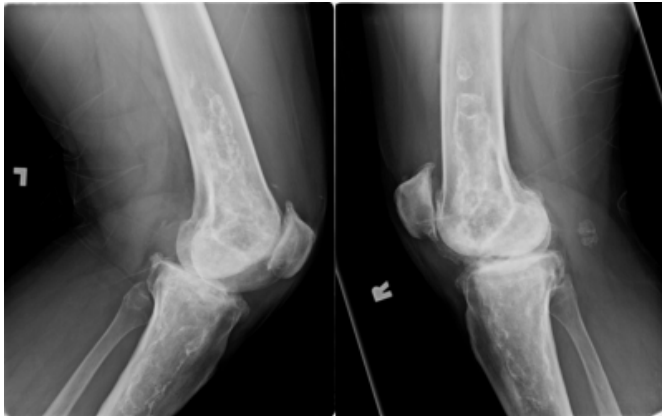
Figure 1

Fig. 1 Plain radiography. Anterior-posterior view of bilateral knees. Note the irregular areas of lacy and serpentine calcific deposits in bilateral distal femurs and proximal tibias typical for bone infarcts.



Figure 2

Figs. 2 and 3 Plain radiography. Lateral view of bilateral knees. Note the characteristic changes as described in Fig. 1



DISCUSSION

Avascular necrosis of bone induced by cortisone was first recognized in 1957 and is one of the most disabling complications of that treatment. [2] The femoral head is most often affected, although other areas of the skeleton such as humeral head, knees and ankles have been described as well. Even short courses of high dose corticosteroids may be associated with the development of osteonecrosis of bone. In some cases, osteonecrosis may develop years after steroid treatment.

Plain radiographic findings are characteristic in established osteonecrosis. In the epiphyseal region, areas of patchy loss on bone opacity are intermingled with sclerotic areas and bone collapse. [1, 2] Secondary osteoarthritis develops after

structural failure and collapse. In the diaphyseal region, a sheetlike lucency of varying size is usually surrounded by shell-like sclerosis and/or calcification and periostitis. [1, 2, 3]

Magnetic resonance imaging and bone scan are more sensitive than plain radiography in the early stages of osteonecrosis and are imaging of choice if plain radiography is non conclusive and suspicion for osteonecrosis is high. [4, 5] Computed tomography can help in diagnosing bone infarction earlier than conventional radiography, however, sensitivity is lower than that of magnetic resonance imaging and bone scan.

Awareness, timely diagnosis and treatment of this potentially crippling complication may prevent or decrease disability related to osteonecrosis.

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