Extensive bone infarcts in an asymptomatic patient with systemic lupus erythematosus

Sirs,

Bone infarcts (BI) or bone osteonecrosis is a condition that frequently affects patients with systemic lupus erythematosus (SLE). However, there are few reports on extensive radiological BI involving more than three anatomical sites, in asymptomatic SLE patients.

A 60-year-old female patient with SLE visited us with complaints of knee stiffness and difficulty walking for four weeks. She was diagnosed with SLE 40 years ago with kidney involvement and received high doses of corticosteroids, along with immunosuppressive drugs. She is now in sustained clinical remission, receiving prednisone 5 mg/day, mycophenolate mofetil, and hydroxychloroquine. Past medical history revealed no evidence of dyslipidaemia, diabetes mellitus, thrombophilia, or trauma. She does not have antiphospholipid syndrome (APS). She does not smoke and consumes alcohol occasionally. Routine laboratory tests showed no abnormalities, and the acute phase reactants were within normal limits. On clinical examination, a mild crepitus sensation on knee palpation was noted, while flexion and extension of the knees revealed no pain; neither effusion nor synovitis was observed. Conventional radiographs of the knees showed large, well-defined serpiginous sclerotic medullary bone lesions affecting the distal femur and proximal tibia symmetrically. These findings represent BI (1). In addition, a mild narrowing of the medial joint space and chondrocalcinosis were noted (Fig. 1a). The lateral view of the knees shows osteonecrosis in the diaphysis of the femur and tibia bones. There are also irregular calcific deposits with a spike-like pattern typical of BI. Similar features are seen in both patellae (Fig. 1b). BI is a term that refers to osteonecrosis within the metaphysis and diaphysis of a bone, leading to bone damage (2). BI results from ischaemia, which can destroy bony architecture, causing abrupt pain in the affected bone and loss of function. BI has numerous causes, among them SLE, APS, alcoholism, pregnancy, blood dyscrasias, corticosteroid therapy, cytotoxic drug intake, trauma, and others (3-5). Our patient had three major potential causes of BI: SLE, high dose of steroids, and cytotoxic agents used. The risk of BI is very high in patients with SLE and early high doses of steroid therapy, as in our patient. Most patients with BI present abrupt and severe pain in the involved bones. However, in some patients, the disorder follows a mild clinical course or is asymptomatic (4), but the radiographic findings are impressive, as in the patient presented above.

Thus, physicians treating SLE patients need to be aware of this complication, and as such, close follow-up and monitoring are necessary.

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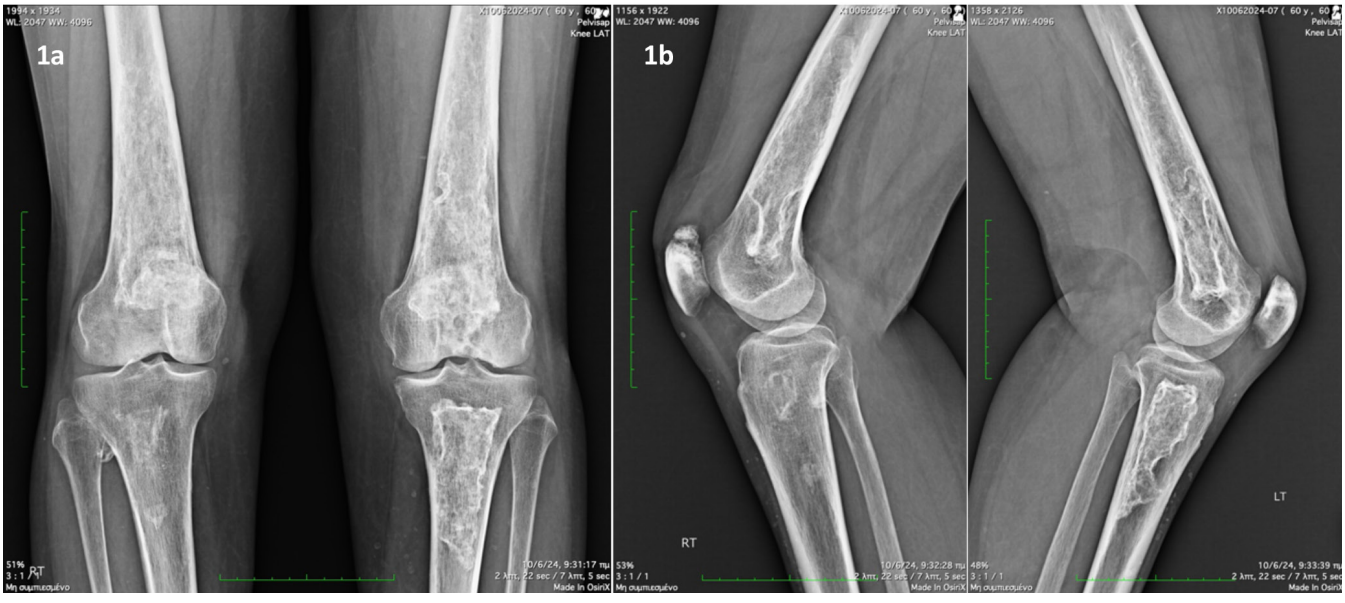

Fig. 1a. Conventional radiographs frontal views of both knees. Note large sclerotic areas, showing serpentine patterns in distal femoral and proximal tibias. A mild medial femoral joint space narrowing with chondrocalcinosis are shown.

Fig. 1b. Conventional radiographs lateral views of the knees. Medullary lesions of sheet-like central lucency are shown surrounded by shell-like sclerosis with serpiginous borders. Patellas are also involved.