MULTIPLE BONE INFARCTS OF THE LEFT FEMUR AND TIBIA

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Background: A 52-year-old female, known with history of multiple sclerosis and breast carcinoma, was referred to the hospital because of knee complaints. Patient was treated with methylprednisolone for multiple sclerosis. Following treatment for keratous acanthoma, a chronic cutaneous infection developed at the medial side of the knee.
Work-up

Conventional radiograph of the left knee (Fig. 1) shows intramedullar, well-defined lesions, composed of areas of patchy bone loss and sclerotic areas, at the epiphysis of the distal femur and the proximal tibia. The sclerotic borders of the lesions are serpiginous in configuration. In the tibia the lesions extend from the epiphyseal to the diaphyseal level.

MRI of the left knee (Fig. 2) includes a coronal T2 TSE SPIR-image (A) showing well-defined lesions in both femur condyles and proximal tibia and outlined by a rim of high signal intensity. Axial T1 TSE-image through the femur condyles (B) demonstrates moderate low intensity of the lesions, which sharply hypo-intense margins at the periphery. On axial T2 TSE-Image at the level of the femur condyles (C), intermediate intensity of the bone lesions with a hyper-intense margin is seen. Axial T1 TSE-image at the level of the proximal tibia (D) shows sharply demarcated bone lesions with hypo-intense margins. The centre of this lesion is hypo-intense to fatty tissue, characterizing the changes in the marrow of the bone. On axial T2 TSE-image through the proximal tibia (E), a well-circumscribed lesion of intermediate intensity but with hypo-intense margins is visible.

Radiological diagnosis

Based on the clinical history and the typical radiological findings, multiple bone infarcts of the left femur and tibia were diagnosed. In this patient the osteonecrosis had developed in several bony structures as a result of corticosteroid use.

Discussion

Osteonecrosis results from a reduction or obliteration in the vascular supply (arterial, capillary, sinusoidal or venous) due to intravascular obstruction, vascular compression or physical disruption. Predisposing factors or diseases are trauma, renal transplant, steroid use, collagen vascular disease, pancreatitis, alcoholism, Gaucher’s disease and arthritis.

There are two hypotheses for the pathogenesis of bone infarct as a result of the use of steroids. The first one suggests a mechanical cause in which steroid-induced osteoporosis leads to microfractures and osseous collapse. The second theory assumes that vascular compression arises as a result of accumulation of fat in bone marrow, fat embolization from steroid-induced fatty liver, vasculitis or hyperviscosity of blood.

Pathologically four zones can be distinguished in bone infarct: a central zone of cell death surrounded by a zone of ischemic injury, a zone of active hyperemia and finally a zone of normal tissue.

Repair of the osteonecrotic area begins along the junction between the ischemic zone and the area with intact circulation. The reactive interface encompasses the ischemically injured zone. The changes in ischemic bone do not appear directly. The most important changes occur in the matrix or the stressbearing areas as a result of anoxia. Any alteration in bone density is an indication of osteoblastic or osteoclastic cell activity and is perceived in the bone or marrow surrounding the osteonecrotic area.

Besides meticulous investigation and treatment of the cause, the treatment of bone infarct ranges from analgesics and exercise to surgical core decompression or bone grafting to prevent collapse. A minimal follow-up of two years is indicated in order to evaluate the changes in adjacent joints. If there is progressive degeneration of the joint, total replacement of the affected joint is indicated.

On conventional radiographs, bone infarction in the epiphysis presents as a subchondral lucent lesion with or without areas of patchy bone loss, mixed with sclerotic areas. In the diaphysis, a sheenlike lucency surrounded by shell-like sclerosis and/or calcific cation or periostitis occurs. CT scan shows a serpiginous outline and increased density of bone.

To evaluate bone infarction, MRI is the imaging modality of choice. Most useful are T1- and T2-weighted and Short Tau Inversion Recovery (STIR) sequences.

T1-weighted MR-images show serpiginous lines of decreased signal intensity. T2-weighted MR-images show a “double line” sign of decreased peripheral signal intensity with adjacent hyperintense inner border, which is characteristic for osteonecrosis. Proton density (PD) images show the same characteristics as T2-images, but are less sensitive for demonstrating edema. The PD-images are useful for the evaluation of overlying chondral surfaces and subarticular infarcts. STIR-images show a high intense periphery of the infarcts, because of bone edema in the hyperemic zone, but spatial resolution is limited compared to FS PD FSE.

Bibliography