Letter to the Editor

Stress fractures can be classified into two groups. Fatigue fractures may occur when repetitive muscular forces or stresses are applied to a normal bone, and are most common in adolescents, athletes and military recruits.

In contrast, insufficiency fractures are found when physiological forces are applied to a weakened bone in patients with diseases like osteomalacia, fibrous dysplasia, metabolic disease and osteoporosis [1, 2].

Patients with juvenile idiopathic arthritis (JIA) accumulate a number of risk factors for the development of osteoporosis, such as inactivity and treatment with steroids or methotrexate (MTX).

Problems in the diagnosis of stress fractures may arise from the absence of radiographic abnormalities in the early phase after the fracture has occurred.

In arthritis patients, diagnosis can be delayed further because symptoms of the underlying disease might mimic complaints caused by the fracture. In this report, we present two JIA patients with stress fractures.

A 10-yr-old girl with polyarticular JIA and a 14-yr-old boy with systemic onset of JIA presented with complaints of pain, the boy in the left extremity and the girl in both lower extremities, at a regular follow-up visit in our pediatric rheumatology clinic.

At that time, both patients were treated with non-steroidal anti-inflammatory drugs and low-dose steroids, the girl also receiving MTX. Neither patient had a history of trauma.

Physical examination revealed localized tenderness below the knee without other signs of inflammation. Because of the discrepancy between the severe localized pain and the lack of further signs of arthritis, X-radiography was performed in both patients and MRI was also carried out for the girl.

Radiography demonstrated a left-sided proximal tibia stress fracture in the boy (Fig. 1a•) and bilateral fractures in the girl. In the girl these findings were confirmed by MRI (Fig. 2•).

The fractures were treated by avoidance of weight-bearing in both children. The girl had to have cast immobilization due to the bilateral involvement.
FIG. 1. X-ray of the male patient. (a) Stress fracture of the left proximal tibia with sclerotic transformation of the bone and formation of callus. (b) Left tibia after complete restitution.

FIG. 2. Fat-saturated T2-weighted coronal MRI of the female patient. The horizontal linear low-signal bands, with adjacent bone marrow oedema (high signal intensity) and periostal swelling (left more than right) are diagnostic of bilateral stress fractures of the proximal tibias.

Because of the ongoing JIA disease activity, steroid and MTX therapy could not be reduced or discontinued. However, the fractures healed without complications, as shown by clinical improvement and radiography after 8 weeks (Fig. 1b•).

Stress fractures are considered to be the result of repetitive forces that exceed the bone’s ability to react with hypertrophy. However, these forces are too weak to cause a complete traumatic fracture.

In children as well as in adolescents and young adults, the typical and most common location for a stress fracture is the posterior cortex of the proximal third of the tibia [3–5].

Bilateral stress fractures of the tibia and stress fractures in children younger than 8 yr are uncommon [3,6].

Risk factors for stress fractures are pathological conditions of bone metabolism, such as osteoporosis, osteomalacia and fibrous dysplasia, or underlying metabolic or rheumatoid diseases or joint prosthesis [7, 8].

The risk of stress fractures is known to be increased by steroid or MTX treatment and irradiation [6], and by an increasing level of activity after a long period of relative inactivity with resulting osteopenia, which is often seen in the remission phase of chronic diseases.

The clinical presentation of stress fractures is usually characterized by localized pain, tenderness and swelling. Pain is relieved by rest and aggravated by exercise [5].

These symptoms are not very different from those described in active arthritis. Initial X-rays may be normal, but stress fractures can later develop dense callus and cause new periosteal bone formation.

Although not always seen in children, linear cortical lucency may become visible. The differential diagnosis needs to include osteomyelitis, eosinophilic granuloma, osteogenic sarcoma and Ewing sarcoma [9].

Whereas in earlier reports the standard diagnostic procedure consisted of X-radiography in two planes and triple-
phase bone scanning, more recent studies have preferred MRI for the second stage.

MRI is sensitive in the detection of subtle osteochondral, non-displaced fractures, which may be difficult to detect on radiographs [7].

In the stress stage, CT scanning usually shows increased bone marrow density, endosteal and periosteal new bone formation and soft tissue oedema [4]. However, CT scanning may rarely show fracture lines that are not seen on plain radiography [8]. Like MRI, bone scintigraphy is non-specific, but it is less sensitive.

However, results are often positive even before radiographic changes are visible. Furthermore, the entire skeleton can be visualized in order to rule out multifocal disease [6].

If the stress fracture is not dislocated, the treatment usually consists of avoidance of weight-bearing. After the full range of motion without pain is achieved, weight-bearing is gradually reinstated, as tolerated by the patient.

Cast immobilization is used for patients in whom weight-bearing cannot be controlled [10]. If the condition is untreated, pseudarthrosis and chronic pain is likely to develop.

Patients with JIA are at high risk of developing stress fractures. Their underlying disease, treatment with steroids or MTX and inactivity may result in osteoporosis.

In these patients the diagnosis of the stress fracture may be a pitfall, as findings typical of stress fractures in the patient’s history and examination might be falsely perceived as being caused by the underlying disease.

To avoid stress fractures, it is necessary to prevent osteoporosis.

Notes

Correspondence to: J. B. Kueggerle-Deschner, Universitätskinderklinik Tübingen, Hoppe-Seyler-Strasse 1, 72076 Tübingen, Germany.

References


[Web of Science][Medline]


