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Multifocal Osteonecrosis Caused by Traumatic Pancreatitis in a Child

A Case Report

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The main factors contributing to the development of secondary osteonecrosis are trauma, the habitual use of alcohol and steroid drugs, and medical conditions such as diabetes mellitus and hyperlipidemia^{1,2}. Osteonecrosis is frequently seen in association with pancreatitis complicated by the alcohol factor but is rarely associated with uncomplicated pancreatitis^{1,4}. We are aware of only one report of symptomatic osteonecrosis secondary to trauma-induced pancreatitis⁴.

We treated a child in whom multifocal osteonecrosis developed following traumatic pancreatitis. The patient's family

was informed that data concerning the case would be submitted for publication and they consented.

Case Report

A ten-year-old girl was injured in a fall from an iron fence surrounding a swing set. She sustained a severe impact to the abdomen and was taken to our hospital. Her family history and medical history were unremarkable. On admission, the blood pressure was 120/87 mm Hg, the pulse was 82 beats/min, and the body temperature was 36.7°C. She reported



Fig. 1-A



Fig. 1-B

Figs. 1-A and 1-B Coronal magnetic resonance imaging scans of the knees. Multiple nodular and macular lesions are evident, primarily in the femoral condyles and the proximal parts of the tibiae. **Fig. 1-A** T1-weighted image. **Fig. 1-B** T2-weighted image.

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Fig. 2-A



Fig. 2-B

Figs. 2-A and 2-B Radiographs made two months after injury. Multiple radiolucent lesions and sclerotic areas are visible in the distal parts of the femora, the proximal parts of the tibiae and fibulae, and the diaphyses of the metacarpals.

Fig. 2-A Anteroposterior radiographs of both knees. **Fig. 2-B** Anteroposterior radiograph of the hand.

intense abdominal pain and nausea. Upper abdominal muscle guarding was observed, and an abdominal computed tomography scan showed pancreatic trauma. The serum amylase level was 296 IU/L, and the white blood-cell count was $14,200/\mu\text{L}$ ($14.2 \times 10^9/\text{L}$). She was diagnosed with traumatic pancreatitis. Peripancreatic drainage was performed, and conservative treatment consisting of fasting (including water), fluid management, administration of FOY (gabexate mesilate), and administration of antibiotics was initiated.

Approximately ten days after the injury, polyarthralgia developed in both elbow joints, both knees, and the right ankle joint and gradually intensified. The patient had intermittent fevers of between 39°C and 40°C , but no heat or swelling was detected locally in the painful joints. The radiographic findings were unremarkable, but $\text{Tc}^{99\text{m}}$ bone scintigraphy showed high levels of tracer accumulation in the distal parts of both femora, in the proximal parts of both tibiae, and in the distal part of the right tibia. Magnetic resonance imaging showed a mixture of (1) extensive band-shaped area(s) with low signal intensity on both T1 and T2-weighted images encompassing large areas of both femora and both tibiae and (2) slightly high-intensity region(s) containing multiple nodular or macular lesions, which, in part, suggested the possibility of fatty marrow being replaced by osteonecrosis (Figs. 1-A and 1-B). Fat-suppressed T2-weighted (short tau inversion recovery [STIR]) images showed lesions with high signal intensity in the bone marrow, suggesting bone marrow edema and inflammation in response to osteonecrosis.

Even with the conservative care described above, the patient continued to experience high fever and abdominal pain. The serum amylase level rose to 8140 IU/L, and the P-amylase level, to 5117 IU/L. Diagnostic imaging revealed gradual enlargement of a pancreatic pseudocyst, which reached a diameter of 13 cm, and two months after the initial trauma a gastropancreatic pseudocyst was decompressed endoscopically. Immediately after the surgery, the abdominal pain and fever were reduced, and the amylase values decreased in parallel to a reduction in the size of the pseudocyst. Because of the patient's severe condition and the need for fluid management, including intravenous hyperalimentation, the bone lesions were treated with symptomatic therapy and in-bed muscle-strengthening training for the limbs. Subsequently, radiographs showed radiolucent lesions and sclerotic areas around both the knee and the elbow joints and in the metacarpal bones, which gradually became more pronounced (Figs. 2-A and 2-B). Periosteal callus formation was also noted. The musculoskeletal symptoms decreased gradually with the reduction in the amylase levels. One year after the accident, the patient was able to run without pain and experienced no interference with daily activities.

Discussion

Traumatic osteonecrosis often develops as necrosis of the femoral head subsequent to a femoral neck fracture. Non-traumatic osteonecrosis generally involves etiologic factors such as alcohol abuse or long-term steroid use and is rarely caused by uncomplicated pancreatitis alone¹⁻⁴. We found only one case of

multifocal osteonecrosis secondary to trauma-induced pancreatitis in the English-language literature⁴.

Reports in the literature suggest that fat necrosis and fat embolism within the bone marrow are major factors contributing to the onset of pancreatitis-induced osteonecrosis^{1,2,4-7}. The lipolytic enzymes released by the pancreatic tissue, such as phospholipase A and lipase, break down fatty tissue to form fatty droplets that occlude the arterioles and capillaries in the bone tissue (lipid embolism within the bone marrow). The anatomical characteristics of the nutrient vessels could lead to this condition in the metaphyseal and epiphyseal portions of the long bones of the limbs. In addition, the free fatty acids that are released into the blood promote the production of prostaglandin E1, eliciting edema within the bone marrow with increased intraosseous pressure. Increased norepinephrine and low insulin levels also can accelerate localized intravascular coagulation, giving rise to intraosseous fat necrosis and osteonecrosis⁸. However, the cases reported to date have occurred almost exclusively in adults and have been complicated by other factors, with very few reports of osteonecrosis associated with uncomplicated pancreatitis¹⁻⁴. Thus, the mechanism of action described above remains a hypothesis, and the risk of osteonecrosis developing by this mechanism is not high. Our patient was a completely healthy child with no etiologic factors other than traumatic pancreatitis, conclusively demonstrating that the symptomatic osteonecrosis was induced solely by the mechanism described. The patient also had a higher number of loci of osteonecrosis, in all four limbs, than was seen in the other pediatric case, reported by Allen and Jinkins⁴.

According to L'Hirondel et al.⁹, magnetic resonance imaging of pancreatitis-induced osteonecrosis commonly shows areas of extensive nodular or macular lesions with low signal intensity on both T1 and T2-weighted images. Early magnetic resonance imaging of our patient showed similar findings before the lesions could be detected with radiography. Such findings are mostly seen in steroid-induced multifocal osteonecrosis and may prove to be characteristic of pancreatitis-induced osteonecrosis. When a patient with traumatic pancreatitis reports joint pain in the limbs, the possibility of secondary osteonecrosis should be considered, and early magnetic resonance imaging can provide valuable diagnostic information. ■

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