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Idiopathic Transient Osteoporosis of the Feet: A case report

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Idiopathic transient osteoporosis is a self limiting, reversible and uncommon condition. There is usually an overlap between various clinical syndromes, which include reflex sympathetic dystrophy, and infectious etiology. We report a case of idiopathic transient osteoporosis involving both feet. The diagnosis was confirmed by clinical examination and supported by biochemistry, blood investigations and radiographs. A 45 year-old man was treated conservatively with bed rest, non-steroidal anti-inflammatory drugs, protected weight-bearing and physiotherapy. This patient made a full clinical and radiographic recovery.

Key Words: Idiopathic transient osteoporosis

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Idiopathic transient osteoporosis does not seem to be a syndrome that is widely known, even though reported cases of this condition during the last few years suggest that it may not be very uncommon.[8,9] In general, it is not difficult to diagnose provided the physician is aware of its characteristic features. The absence of abnormal laboratory tests and the self-limited course of the syndrome differentiate it from systemic diseases or local infection or neoplasia. During the last few years, the syndrome of idiopathic transient osteoporosis, first described in 1966, has received only limited recognition.[10]

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A patient with this syndrome is an adult who seeks medical advice because of pain in the foot. There is no apparent reason for the pain. The pain may be rapid or gradual in onset and it is mainly felt on weight-bearing. It usually progresses within a few weeks and becomes severe enough to cause a limp. The range of joint movement is only slightly restricted. There are no other symptoms or signs and all hematological tests are within normal limits. Radiographs show the constant and distinctive feature of the condition is rarefaction of the bones which is easily recognized and is usually severe.

The disease usually lasts from two to six months and the patient almost always recovers completely. As the pain gradually diminishes, the limp disappears and the foot recovers its full range of motion. Radiographs show a progressive restoration to normal of the density of the bones and the knee eventually regains its normal appearance within a few months after clinical recovery. The disease is self limited and disappears regardless of treatment.

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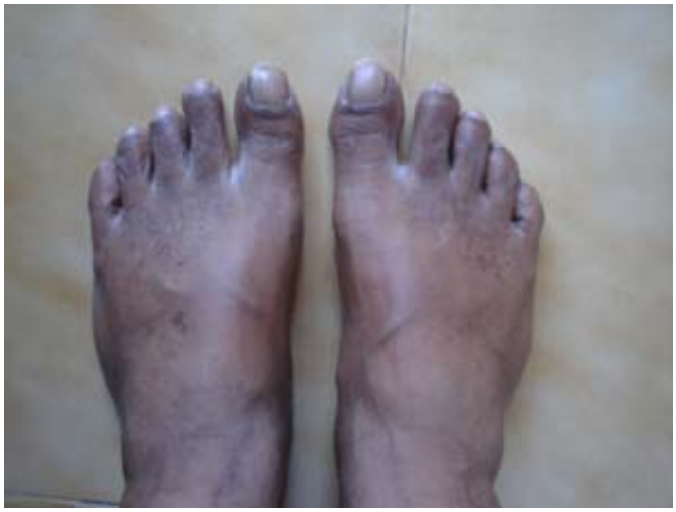

Figure 1 Signs of minimal inflammation is seen clinically. The patient complained of pain to both feet.

Case Report

A 45 year-old man came to our casualty department with complaints of pain to both feet for 7 days duration. There were no recognizable factors which might have precipitated the pain in the joint, and the symptoms seemed to appear spontaneously. No definite history of antecedent trauma or pre-existing disease in the joint was obtained. The episode of pain began gradually. The pain became more severe over a period of 2 weeks to a month. The pain increased, and it became more diffuse involving the foot and ankle. He walked with a limp because of the pain, and most of the time used a cane. Morning stiffness was not present. Signs of inflammation were present but were minimal. (Fig. 1) Mild swelling, involving the ankle and most of the foot rather than being confined to a specific portion of the tarsal and metatarsal joints was present. (Fig. 2)

The swelling was pitting and tended to become worse toward the end of the day. The synovial membrane was not thickened. The feet were tender on palpation, but the skin was not cold, clammy, moist, cyanotic, or atrophic. Passive joint motion was slightly limited. Active motion was moderately limited because of pain.

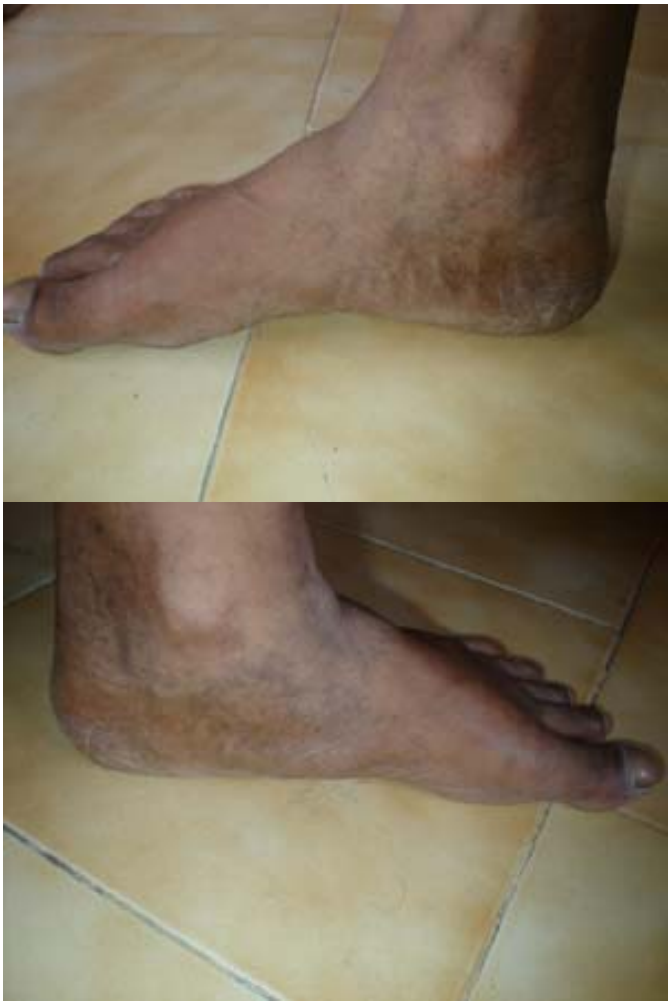

Figure 2 Mild swelling involving both ankles and most of the foot is observed. The swelling is not confined to a specific region of the tarsus or other joints of the foot.

Mild atrophy of the muscles surrounding the ankle and feet was seen and tended to disappear as the symptoms lessened. Radiographs of the feet showed decreased bone density but no clear evidence of destruction of the joint or fracture (Fig. 3). In the early stage, the demineralization tended to be diffuse and minimum but as it progressed, it was patchy or mottled. The degree of osteoporosis was striking during the period of most severe involvement. (Fig. 4)

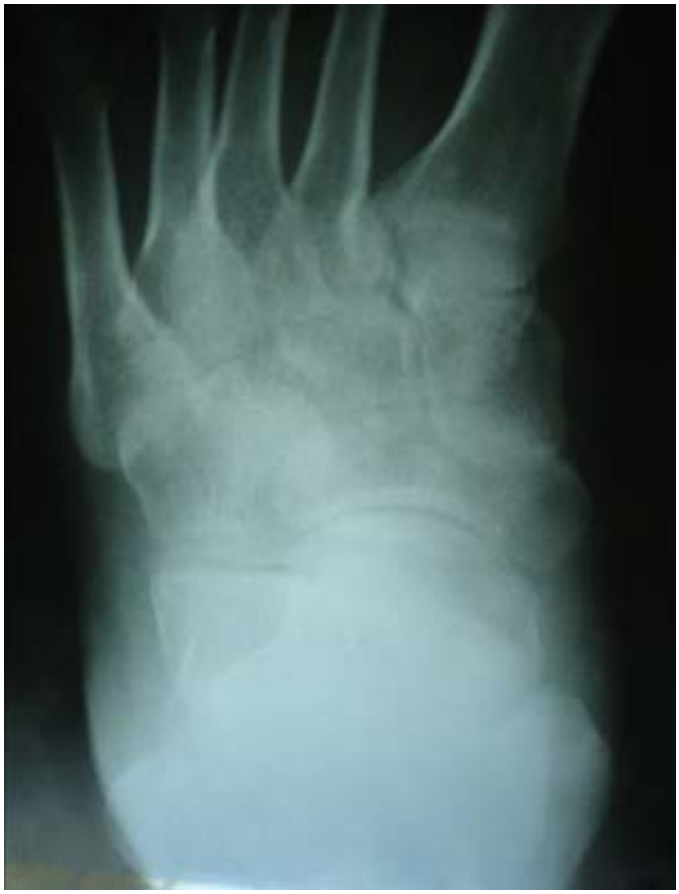

Figure 3 Radiograph shows spotty demineralization, diffuse osteoporosis and mottled appearance of the bone.

Diffuse osteopenia, generalized and spotty demineralization were the characteristic radiological features seen in our patient. The bone cortex was only faintly seen and the cortical margin could not be definitely discerned. The distal end of the tibia and fibula near the ankle joint also became osteoporotic but usually to a lesser degree than the bones of the foot. His random blood sugar was 106 mg/dl. No past history suggestive of ankylosing spondylitis, rheumatoid arthritis, thrombo-embolic events in the past or any chronic illness of long duration. Erythrocyte sedimentation rate was 78mm/ hour (normal <14). His blood parameters revealed hemoglobin 11.2gms, TLC 7,500cu\mm, serum calcium 9.0mgs, phosphorus 3.5 mgs, serum alkaline phosphatase 100IU\L, which were within normal limits.

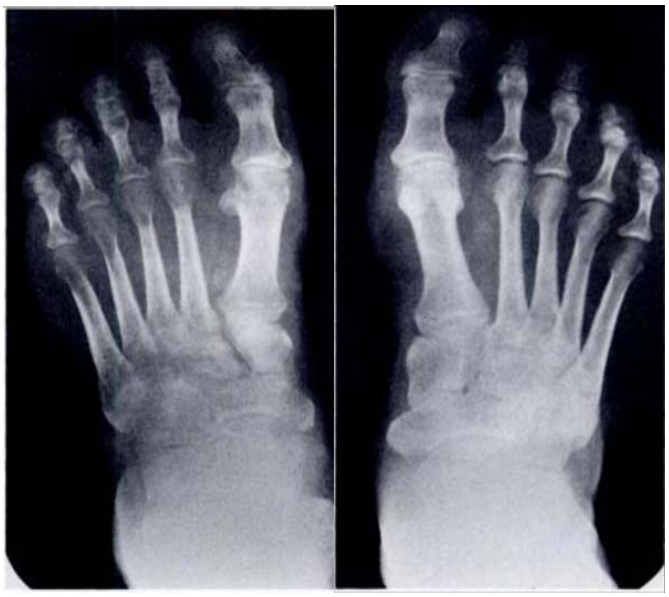

Figure 4 In the period of most severe involvement, there is striking osteoporosis and mottling of the bone. The most characteristic features include diffuse osteopenia with generalized and spotty demineralization.

C-reactive protein, liver function tests, creatinine, electrolytes, and thyroid function test and protein electrophoresis were normal. Tests for anti-nuclear body, rheumatoid factor and HLA- B27 were negative.

The patient was treated with non-steroidal anti-inflammatory drugs, protected partial weight-bearing, ankle exercises and regular physiotherapy. The physiotherapy included active and passive range of motion exercises for 4 weeks. After 6 weeks, the patient came again to our orthopedic outpatient department with complete relief in his foot pain. He was then walking with one stick. Four weeks later his symptoms were greatly relieved, and he could walk with minimum pain and a slight limp. After six months there was no pain, no swelling and movements were full. Radiographs showed a general increase in bone density and an intact cortex. (Fig. 5) The patient could walk normally with full weight bearing, no limp and walking aid.

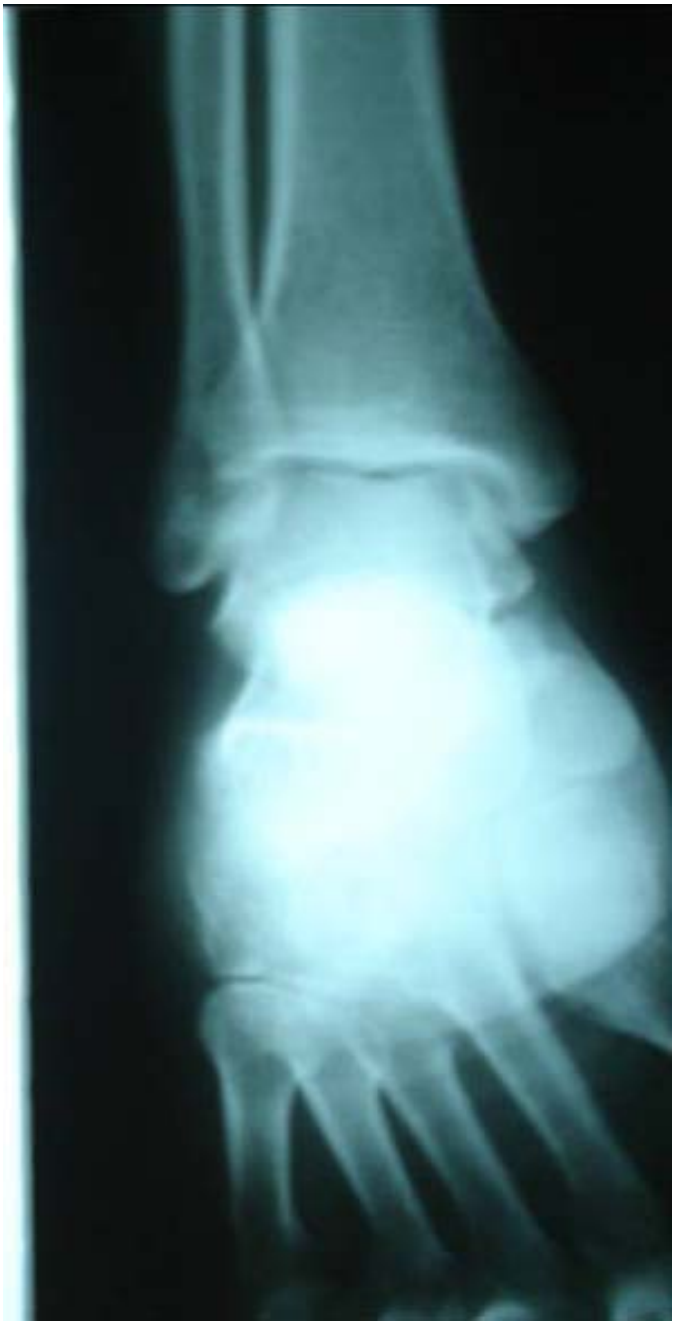

Figure 5 After treatment including non-steroidal anti-inflammatory drugs, protected weight bearing, ankle exercises and regular physiotherapy, the patient showed complete recovery of symptoms. Radiographs showed general increase in bone density without signs of bone mottling or spotty osteoporosis.

Discussion

The etiology of idiopathic transient osteoporosis remains uncertain. Most authors speculate that transient osteoporosis is a variant of Sudeck's bone dystrophy. Others have suggested neural compression, obstruction of venous return, transitory capsulitis or transitory inflammatory but no data are available to support these explanations.[8]

Even though the pathogenetic mechanisms responsible for the episodes of painful osteoporosis remain obscure, the findings manifested by these patients are similar and appear to form a recognizable clinical syndrome. These features include the following: episodes of osteoporosis in the bones of a joint of the extremity, pain with use of the joint (especially weight-bearing), and gradual spontaneous recovery. Although episodes varied in duration, invariably each was symptomatically self-limited.[8]

The most common theory is that microvascular injury causes tissue ischemia, bone marrow edema and limited cell death.[1,5,6] Although these also occur in avascular necrosis, the presence of bone marrow edema, reactive bone formation, osteoclastic resorption in the absence of necrosis suggests that idiopathic osteoporosis is different from avascular necrosis.[5]

Classically, transient osteoporosis starts with spontaneous, acute or gradually increasing joint pain, worse on weight bearing. On examination there is generalized effusion, tenderness, muscle wasting. The range of movement is moderately limited and sometimes painful at extremes. The symptoms usually subside after three to nine months.[3,4] The discrepancy between functional impairment and limited clinical findings is characteristic.[2] The diagnosis of reflex sympathetic dystrophy requires the presence of regional pain and sensory changes following minor trauma and associated with abnormal skin color, temperature changes, abnormal sudomotor activity with edema and significant impairment of motor function.

Patients with transient osteoporosis appeared to recover without permanent injury regardless of the treatment, whereas patients with reflex dystrophy may have permanent disability, especially if not adequately treated.[2,9]

The differential diagnosis included a number of conditions in which there was pain in a joint and osteoporosis. Monoarticular arthritis was considered initially in some part of our management, as were rheumatoid arthritis, gout, sarcoidosis, and chronic sepsis. The lack of morning stiffness, definite synovial thickening on examination, cortical erosions, or joint narrowing in the presence of marked osteoporosis tended to exclude synovitis, as in rheumatoid arthritis, as the primary pathological process. The gradual onset and long duration of the episodes and normal serum uric acid values were incompatible with crystal synovitis. There was no evidence of sarcoidosis, infection, or a systemic process. It seemed unlikely that a general disturbance of calcium metabolism should cause such localized bone changes. The serum calcium, phosphorus, and alkaline phosphatase values were normal. Massive osteolysis, which has been described as disappearing bone disease, is progressive and irreversible, unlike the osteoporosis in the present reversible episodes.[4]

In the early phase, plain radiographs revealed no significant finding. A delay between the onset of symptoms and findings of the osteopenia in plain radiographs is the rule. Second phase radiograph reveals variable focal loss of bone density and blurring of trabecular structure with poorly defined cortical borders, sometimes with complete effacement of the subchondral cortex. The joint space is always preserved and osseous erosion or subchondral collapse never occurs.[1,2,7] Treatment of transient osteoporosis should consist of a conservative program of physical therapy with gradual ambulation. Some protection against full weight-bearing is advisable because of the possibility of bone damage during the period of severe osteoporosis.

Reassuring the patient that the episode is self-limited is helpful. In severe cases, corticosteroids may be considered for pain relief, but it has not yet been shown that such treatment shortens the course of the episode.[9] We feel it is important to include transient osteoporosis in a differential diagnosis of unusual feet pain. High level of suspicion makes diagnosis easier and avoids unnecessary interventions. The availability of clinical findings supported by blood investigation and radiographs helps to rule out more serious conditions and remains the modality of choice for detection.

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