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Bone-marrow oedema syndrome

In 1959 Curtiss and Kincaid described a clinical syndrome characterised by pain and transient osteoporosis of one or both hips which affected women in the last trimester of pregnancy. This was distinguished from other types of 'secondary' osteoporosis by the absence of any obvious triggering factor or underlying hip pathology. It regressed spontaneously, with restoration of normal function and bone density over a period of 6 to 12 months. It is now known that the condition may occur in patients of either sex and at all ages from late adolescence onwards.

The combination of pain, limp, restricted mobility, regional osteoporosis and increased activity on radioscin-tigraphy suggests a range of possible diagnoses, which include septic arthritis, tuberculosis, monarticular rheumatoid disease, pigmented villonodular synovitis and covert bone lesions such as osteoid osteoma or subacute osteomyelitis. In the knee or ankle, these findings would suggest algodystrophy. In all of these conditions there are leads to the diagnosis in the history or radiographic or laboratory investigations, but transient osteoporosis comes and goes like a cloud across the landscape, leaving no clue as to its origin and no mark of any lingering abnormality.

The pathogenesis of transient osteoporosis remains a mystery although some of its features suggest a local neurovascular disorder, possibly related on the one hand to reflex sympathetic dystrophy and on the other to non-traumatic ischaemic necrosis (Resnick and Niwayama 1988).

When clues run dry, try MRI. In 1988, Wilson et al reported ten patients with clinical and radiological features typical of transient osteoporosis. All showed similar abnormalities on MRI: low signal intensity on T1-weighted images with matching high signal intensity on T2-weighted images which extended from the femoral head to the intertrochanteric region and corresponded to the areas of increased scintigraphic activity. They attributed these abnormalities to bone-marrow oedema; all ten patients recovered spontaneously.

Neither the clinical nor the imaging features in these cases suggested the presence of ischaemic necrosis, and biopsy specimens from three patients showed normal marrow and bone. A series of nine patients (ten hips) with what appears to be the same disorder is reported by Hofmann et al in this issue of the Journal (p. 210). In these cases bone biopsies showed histological changes consistent with bone-marrow oedema; here again, however, there were no convincing signs of bone death. The patients in this series were treated by core decompression of the femoral head and neck, with rapid and complete relief of symptoms and a return to normal MRI signal patterns.

Is this, as the authors suggest, a very early (and reversible) stage of ischaemic necrosis? There will be much argument on this issue. Supporters of the idea will point to the fact that in cases of proven avascular necrosis the typical focal changes in MRI are sometimes accompanied by diffuse abnormalities that are characteristic of bone-marrow oedema. Sceptics will argue that this is not surprising, since the reparative phase of avascular necrosis is associated with inflammation, hyperaemia, marrow congestion, a decrease in marrow fat and an increase in interstitial fluid. In early avascular necrosis the 'oedema signal' in MRI appears at the periphery of the necrotic segment, where vascular ingrowth and new bone formation are commencing (Mitchell et al 1987). More difficult to refute is the report by Turner et al (1989) of five patients who presented with hip pain and MRI evidence of marrow oedema and then subsequently developed focal changes of osteonecrosis or were shown on biopsy to have marrow and bone necrosis.
Whatever the answer to this puzzle, it is important to recognise the difference between bone-marrow oedema without osteonecrosis and that with osteonecrosis. The former is a hypervascular, usually self-limiting disorder whereas the latter is unequivocally an ischaemic disorder which may go on to bone collapse and articular distortion. The paper by Takatori et al on page 217 of this issue of the Journal gives a clear lead in this matter. These authors have identified various patterns (or stages?) of MRI changes in avascular necrosis. Only one of these, type A with an abnormal MRI fat signal confined to the medial anterosuperior part of the femoral head, is potentially reversible and likely to respond to core decompression.

With present knowledge, it would seem to be sensible to regard uncomplicated bone-marrow oedema syndrome as a transient condition which usually requires no active intervention. If MRI shows focal abnormalities, it is important to distinguish between the 'favourable' signal patterns of limited anterosuperior necrosis (for which core decompression is a feasible option) and the 'unfavourable' signal patterns which presage femoral head collapse and a likely need for reconstructive surgery.

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Fractures of the calcaneum

Severe fractures of the calcaneum present a considerable challenge: the patient is frustrated by prolonged treatment and slow progress and those who treat him have to select methods of treatment without any clear-cut indications. This issue of the Journal includes three papers which add to our knowledge. Eastwood, Gregg and Atkins (p. 183) describe a new CT classification which aids the selection and planning of the operation described in their second paper (Eastwood, Langkamer, and Atkins, p. 189). In the third paper the long-term results of a similar type of operative treatment are presented by Leung, Yuen and Chan (p. 196).

The choice of treatment for severe, displaced fractures involving the joints remains controversial because varied results have been described. For many such injuries the initial fracture deformity is accepted and conservative treatment is advised with the aim of restoring early function. The results of such treatment for the more severe fractures have been described (Nade and Monahan 1972; Pozo, Kirwan and Jackson 1984). Pozo et al showed that only 19% of such patients had 'significant' pain after a mean follow-up of more than 14 years. All the involved feet were of abnormal shape, but every patient could wear normal shoes. Three-quarters of the patients could run, and walk without restriction on flat ground, but 80% had less than half the normal range of subtalar joint movement. There had been improvement for at least two years after injury, and frequently for up to six years. Most patients had a relatively normal lifestyle and 80% had returned to their original work. The other 20% had poor results.

The important question is whether surgery can improve on these levels of temporary morbidity and permanent disability. This is best considered for each of the individual features which may cause disability.

The subtalar joint is disrupted in all serious calcaneal fractures. Loss of movement at this joint does not appear to correlate with the overall quality of result, but there is a direct relation between late symptoms and the degree of disruption of its posterior facet. Crosby and Fitzgibbons (1990) found that all patients with serious residual damage of the subtalar joint had persistent disability. They showed, by multiple regression analysis, that the state of this joint was the most important indicator of prognosis. Disruption of the calcaneocuboid joint was also associated with poor results.

Increase in the lateral width of the heel due to distortion and loss of vertical height is known to lead to impingement symptoms which may be very troublesome. Collapse of the foot with flattening of the arch and the heel due to a varus, valgus or complex deformity within the calcaneum may also produce symptoms. Finally,