but this is not helpful as B27 is frequently present in healthy people. It is important to distinguish it from other back problems, for if neglected ankylosing spondylitis may lead to permanent stiffness and disability.

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Regular Review

Early diagnosis and treatment of steroid induced avascular necrosis of bone

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Steroid induced avascular necrosis of bone is found in patients with renal transplants, lupus erythematosus, asthma, glomerulonephritis, peripheral neuritis, sinusitis, pemphigus, Guillain-Barré syndrome, head injuries, and those given combination chemotherapy. Progressive pain and restriction of joint movement may be present for several months before radiological evidence of avascular necrosis appears.[1] Once radiographic changes are present the joint usually collapses, resulting in severe dysfunction and disability.[2 3] Until recently the standard treatment has been total prosthetic replacement. Now, however, new techniques for detecting the very early preradiographic changes of avascular necrosis have made biologically oriented joint sparing surgical procedures a practical alternative.

Avascular necrosis of bone induced by cortisone was first recognised in 1957[4] and is one of the most disabling complications of treatment. The femoral head is most often affected, but the process may also damage the head of the humerus, the femoral condyles, the tibial plateau, the talus, and the capitellum. Ihde and De Vita[5] estimated that 1·3% of patients treated with combination chemotherapy would develop avascular necrosis of bone 20 to 42 months from the onset of treatment having received a total dosage of between 2·1 g and 26 g of prednisolone. Thorn *et al*[6] reviewed the records of 3500 patients with malignant lymphoma and found 12 with avascular necrosis of bone, mostly affecting the hip joint. All but one had received combination chemotherapy including steroids, and the total dosage of steroid varied from 1·4 to 14·95 g of prednisolone.

Deaths in the early months after renal transplantation are usually due to infection—which may be attributable largely to the high steroid dosage.[7] To assess the effects of reducing the cumulative steroid dose, Morris carried out a randomised controlled trial of 72 patients, 39 receiving the usual high dose regimen and 33 a new low dose regimen of oral prednisolone, both in combination with azathioprine.[7] Patients were followed for at least two years after transplantation. Patient and graft survival was identical in the two groups, and the morbidity associated with steroids was impressively lower in patients receiving a low steroid dose: eight patients on the high dose regimen had developed avascular necrosis of the femoral head after two years compared with only one patient on the low dose regimen. There seemed little justification for continued use of high doses of oral steroids with azathioprine after cadaveric renal transplantation.

Even short courses of high dose corticosteroids may be associated with the development of avascular necrosis of bone. McCluskey and Gutteridge[8] reported three patients who developed bilateral avascular necrosis of the femoral and humeral heads seven to 22 months after taking high doses of dexamethasone for 18 to 37 days as prophylaxis against oedema of the central nervous system. Though short courses of high dose dexamethasone are of proved value in patients with malignant brain tumours,[9] their value in patients with stroke[10] and head injury[11] is less certain.

Many theories have been advanced to explain why treatment with corticosteroids should predispose to avascular necrosis of bone: fat embolism,[12] hypercoagulability,[13] increased intraosseous pressure,[14] and fat cell swelling[15] have all been implicated, but the question remains unresolved.

Early diagnosis

Techniques for early diagnosis and treatment of avascular necrosis have developed from our improved understanding of

the circulation of blood through bone. Arterial blood enters the medullary cavity through the nutrient arteries and passes longitudinally and centrifugally through a capillary bed to the venous channels in surrounding muscles or to an anastomotic venous plexus. Within the bone blood flow follows the principles of a Starling resistor—a rigid canister through which passes a flexible thin walled tube.[16] Any increase in the pressure within the canister will result in a proportional decrease in the flow within the flexible tube so long as the driving pressure remains unchanged. Thus bone marrow pressure directly affects vascular resistance and therefore bone blood flow, which may be completely independent of any primary pathological changes within the vessels.[17] This is likely to be the mechanism of the formation of a sequestrum in osteomyelitis and of bone infarction in conditions which cause a rise in the intramedullary pressure. Wang and others have shown raised marrow pressures in rabbits given steroids.[15] Another possible mechanism may parallel the accumulation of cerebroglycosides in the reticuloendothelial cells of patients with Gaucher's disease, which appears to reduce the size of capillaries within the bone,[18] reducing intramedullary circulation. Possibly similar changes occur in response to steroid treatment; again, intramedullary fat cells do grow larger in rabbits given steroids.[15] Possibly, therefore, a compartment syndrome may develop within the bone requiring early decompression analogous to a soft tissue compartment syndrome treated by urgent fasciotomy to prevent further cell damage and death.

Studies of the early stage of avascular necrosis of bone have been possible because idiopathic avascular necrosis of the femoral head is bilateral in up to two thirds of cases.[3 19] Radiographic changes in the second hip may postdate those in the first hip by a matter of years. Radiological stage one avascular necrosis has been defined[1] as that in which plain radiographic changes are absent apart from occasional osteoporosis; it may be recognised with the newer techniques described below. In stage two, abnormal or irregular bone density is seen in the femoral head with a linear subchondral zone of lucency, often best seen on the lateral film, occurring roughly 2 mm from the joint surface.[20] Stage three is an intermediate stage, with irregular bone density and obvious radiographic collapse of the femoral head and the appearance of sequestrum. Stage four changes show severe deformity of the femoral head with associated osteoarthritic change and extensive destruction of the joint. These changes may affect the whole or merely a segment of the femoral head. Radiographic stages three and four are recognised easily. Stage two, which coincides with attempted revascularisation of the necrotic area, may be seen only on high quality radiographs which show the bone trabeculae clearly. A high definition screen film combination is recommended with a focal point of 0·6 to 1·2 mm. Probably the greatest error leading to failure of diagnosis of stage 2 is underpenetration. An increased penetration by as much as 5-10 kilovolts is recommended.[21] A true increase in radiological density of the affected bone occurs after revascularisation, when new bone is laid down on the dead trabeculae, while a relative increase in density may be seen when the surrounding healthy bone is affected by disuse osteoporosis.

Radionuclides were first used in diagnosing disease of the femoral head, including avascular necrosis, by Tucker in 1950.[22] Avascular necrosis might be expected to diminish uptake, but in fact large series report only 1% of cases showing decreased uptake when scanned soon after bone infarction before any reparative response is mounted.[23] Other series[24] found no cases with demonstrable "cold" lesions, possibly either because the initial size of the avascular segment is below the limits of resolution of the detector system or because the cold site is obscured by increased uptake of the normal reactive bone surrounding the avascular segment. Three per cent of scans show no abnormality,[23] but in the remainder an area of increased uptake may be seen, centred on the femoral head. The characteristic image found in about a quarter of these cases is of a ring or crescent shaped epiphysial focus of increased uptake inferolaterally[25] with an upper medial area of decreased uptake. This is most likely to be seen in radiological stages one and two. More extensive abnormalities of the head, neck, shaft, and acetabulum are seen as the condition progresses through stages three and four. The highest uptake ratios of ^{99m}Tc diphosphonate correspond to osteonecrosis complicated by osteoarthritis, but scintigraphy then no longer has the same value since the radiological diagnosis of avascular necrosis is evident by stage two. Gregg has shown in studies on rabbits that the bone scan result can become positive three weeks after a vascular insult.[26]

Certainly bone scans may be useful in the diagnosis of early avascular necrosis, but increased activity remains a non-specific change and is not of itself sufficiently diagnostic to indicate surgical intervention in the preradiological stages of avascular necrosis of bone. What other diagnostic aids are available? The bone marrow or intraosseous pressure is consistently raised up to three or four times above normal in patients with stage one avascular necrosis of bone.[27] The pressure may be measured through a rigid needle inserted percutaneously through the lateral femoral cortex at the level of the greater trochanter. A five minute recording is the base line measurement. Five ml of physiological saline is then injected, and the pressure five minutes later is referred to as the stress pressure. Both these measurements are raised in patients with stage one avascular necrosis of bone.[28] The normal bone marrow pressure is less than 30 mm Hg and the stress pressure does not provoke intraosseous hypertension. Patients with avascular necrosis, however, have a base line pressure above 50 mm Hg with a hypertensive response to saline loading.[18]

Intraosseous venography or osteomedullography can be performed using the same needle. Eight ml of soluble contrast material is injected intraosseously with radiographs taken at the completion of the injection and then five minutes later. The injection is uncomfortable for patients with normal hips and excruciatingly painful for patients with avascular necrosis, so sedation or a general anaesthetic is required. A normal joint will show rapid filling of the extraosseous drainage routes and rapid clearance of the intraosseous contrast with the metaphysial veins clearly shown. In stage one avascular necrosis, however, there is outflow obstruction. Five minutes after injection intramedullary stasis may be seen, with poor filling of the metaphysial veins and diaphysial reflux.[14] Bone marrow pressure recordings and intraosseous venography are of comparable accuracy with core biopsy and are now established as diagnostic of avascular necrosis of bone.[29]

The first comparison of the sensitivity of bone scans with radiographs using the independent monitor of bone marrow pressure was performed by Conklin[28] using patients with systemic lupus erythematosus having steroid treatment. Radiographs were found to be 41% sensitive in diagnosing avascular necrosis compared with a 92% sensitivity of bone scans. It appears that bone scans diagnose an earlier stage of avascular necrosis than radiographs.

Koshino carried out arthroscopic examination of eight patients with spontaneous necrosis of the knee and stated that abnormalities in the articular cartilage which were not seen radiographically could be observed in detail.[30] In the early stages of osteonecrosis the usual findings were flattening and fissuring of the articular surface with or without the formation of a cartilage flap.

Treatment of early lesions

It is, therefore, now possible to diagnose avascular necrosis of bone before irreversible joint changes occur. This clearly alters the whole emphasis of management in these patients in favour of biological methods of joint preservation in preference to waiting until joint collapse occurs to justify total prosthetic replacement. Treatment may be medical or surgical. Medical management[31] is designed "to protect the joint from all forces until it has revascularised itself and restored its original strength so that collapse and disintegration of the femoral head will not occur." This generally means six months' non-weight bearing and six months' limited weight bearing combined with analgesics and anti-inflammatory drugs. Once radiographic changes have occurred, however, such non-operative management is unsatisfactory.[2 3]

No consensus has emerged on the most effective methods of surgical treatment, which began with the observation that after removal of a core of bone from the head and neck for histological diagnosis of stage one avascular necrosis some patients showed rapid relief of symptoms and arrest of the disease process.[32] Core decompression appears logical on the basis of the known abnormalities in medullary pressure. A 1 cm core of cancellous bone is removed through a drill hole in the metaphysis of the proximal femur for the hip or distal femur for the condyles. The specimen is used for histological confirmation of the diagnosis. Postoperative management consists of protective weight bearing for a minimum of six weeks to allow the bone to adapt to the created defect. Weight bearing is increased for a further four to six weeks until unrestricted weight bearing is allowed at 10 to 12 weeks.

By analogy with anterior tibial compartment decompression, if the compartment is decompressed during the early ischaemic phase one may expect normal function to be restored. If the compartment is decompressed only after extensive tissue necrosis the condition is unlikely to be reversed. In one series 11 out of 12 patients with stage one and two avascular necrosis remained symptom free for between four and 45 months after decompression.[33] Even in stage three disease the early results of decompression have been impressive with reduction of symptoms and arrest of radiological progression on short term (12 months) follow up. The average age of these patients was 28·3 years, so that any treatment that may at least postpone the need for total joint replacement for a number of years would be a great benefit. Hungerford compared conservative treatment as described with core compression. In stage two disease only one of eight joints treated surgically remained symptomatic with radiological evidence of progression compared with 21 out of 22 treated medically which remained symptomatic while 19 showed radiological progression.

An alternative surgical approach to joint preservation is designed to promote revascularisation of the necrotic femoral head. Judet[34] described the muscle pedicle bone graft, and this procedure was subsequently modified by Meyers.[35] Lee[36] followed up 10 patients with radiological stage one and two necrosis treated by muscle pedicle bone grafting; seven showed radiological signs of healing and revascularisation and remained symptom free. In three patients the condition progressed, possibly owing to removal of excess bone from the head followed by subsequent collapse of the articular surface. Meyers reviewed 24 patients with muscle pedicle graft procedures and recommended the procedure for early asymptomatic stage two avascular necrosis but found poor results in stages three and four, which is only to be expected.[35]

The use of frozen osseous and osteochondral allografts was introduced by Lexer in 1908[37] and there have since been a few reports of the use of small fresh allografts.[38] Gross reported 20 patients who had received allografts for osteonecrosis and had been followed up for an average of 40 months (range 12-60).[39] Eight patients under the age of 50 with traumatic osteonecrosis achieved the best results, but results in steroid induced avascular necrosis were poor, only one of three patients improving. This may be because the allografting procedure has very little effect on the underlying pathological process so that the recipient area of bone rejects the graft just as it generated the initial avascular segment.

Judet took his approach a stage further, using free fibular graft and a microvascular technique to anastomose its nutrient vessel to the anterior circumflex vessel.[40] He believed that this procedure was suitable for young patients with advanced avascular necrosis in whom the only alternative would be total hip replacement, but the long term results remain unknown. Smith reviewed 38 patients with avascular necrosis of 56 hips managed with tibial bone grafting.[41] He noted, however, that patients should ideally be young with a good life expectancy. This rules out many patients with necrotic lesions of the femoral head who are likely to die prematurely from the disease which initiated the avascular necrosis.[42] Smith's ideal patients would also have minimal collapse and degenerative change and a satisfactory preoperative hip rating equal to or greater than 80 on the Iowa hip rating scale.[43]

The third surgical approach is osteotomy. An avascular segment in the femoral head is quite often located in the anterolateral aspect of the head and, by rotating the head and neck anteriorly on the longitudinal axis of the neck between 45° and 90°, this damaged area may be removed from the weightbearing area and replaced by intact cartilage and bone. Clearly this procedure must be done before the head collapses completely. A transtrochanteric anterior rotational osteotomy was described in 41 patients by Sugioka, who stated that it prevented progression of the collapsed segment and preserved the joint surfaces.[44] He recommended a rotational osteotomy in patients in whom less than two thirds of the diameter of the head was affected at its junction with the neck. Kotz reviewed 17 patients with idiopathic avascular necrosis over an average of 25 months of follow up, finding that the Sugioka technique gave better results than an intertrochanteric osteotomy with muscle release.[45] The subtrochanteric abduction or adduction osteotomy is not effective for extensive lateral lesions,[2 3] but in 61 patients with 80 avascular femoral heads the results in early necrosis showed complete relief of pain in 17 patients with apparent arrest of joint collapse but no increase in the range of movement of the joint. In addition, rather surprisingly, 11 patients with grade 3 and 4 necrosis derived complete or fairly complete pain relief after subtrochanteric osteotomy but their range of movement was diminished. Osteotomy may itself have a beneficial effect on the vascularity of the femoral head.

In the treatment of spontaneous osteonecrosis of the knee a high tibial osteotomy has been described as both mechanic-

ally correcting the varus deformity and stimulating the formation of new bone in the avascular segment.[46] Koshino reported on 36 patients with avascular necrosis of the femoral condyles affecting 37 knees; surgery (osteotomy with drilling and grafting of the avascular segment of the femoral condyle) was performed on 23 knees.[46] The necrotic lesion became radiographically undetectable in 13 and improved in 17. These results are similar to those described by Rozing.[47]

Clearly some patients will present too late for joint preserving biological procedures to be undertaken. In these instances total joint replacement is extremely effective for hip disease, and two patients have had hip arthroplasties after cardiac transplantation.[48]

At present, therefore, stage one avascular necrosis of bone may be regarded as a compartment syndrome affecting the intraosseous compartment, depriving the bone of its blood supply, and demanding early decompression. It may not be entirely clear why the intraosseous pressure increases, but none the less the relief of pain and arrest of radiological progression that follow core decompression appear to justify its use.

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