SEQUENTIAL MAGNETIC RESONANCE IMAGING IN PERTHES' DISEASE

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Forty-eight images using magnetic resonance imaging (MRI) in 16 hips with Perthes' disease were evaluated over a mean period of two years. MRI depicted exactly the infarcted zone in the femoral head before typical radiological changes were evident. Early determination of the extent of the infarcted bone on MRI benefits those patients who require treatment. Follow-up MRI scans at six-monthly intervals, reflected the chronological stages of the repair process in each group classified according to Catterall.

In Perthes' disease an ischaemic process is responsible for avascular necrosis of the capital epiphysis (Dolman and Bell 1973; Sanchis, Zahir and Freeman 1973; McKibbin and Rališ 1974; Inoue et al 1976; Jensen and Lauritzen 1976; Catterall et al 1982). The extent of the involvement, which later may include the metaphysis, determines the early disability, the growth disturbance and deformity of the femoral head. Therefore, early diagnosis and definition of the infarcted bone is of paramount importance for successful treatment. The earliest radiographic signs are a small dense epiphysis, and a subchondral fracture line (Waldenström 1938; Caffey 1968). In ischaemic necrosis, considerable loss of trabecular bone is required before marrow abnormalities become radiographically evident. Scintigraphy has been the only diagnostic method used to demonstrate early vascular disturbance of the femoral head (Sutherland et al 1980; Calver et al 1981; LaMont et al 1981; Bensahel et al 1983), but it is non-specific and cannot determine the extent of epiphyseal necrosis (Paterson and Savage 1986). The extent of necrosis is usually determined by conventional radiographs (Catterall 1971, 1982). Recently, magnetic resonance images (MRI) have demonstrated detailed marrow changes in the femoral head damaged by ischaemia (Scoles et al 1984; Toby, Koman and Bechtold 1985; Lang et al 1988; Mitchell et al 1989).

MRI evaluates all bone marrow components directly. The basic constituents of bone marrow that determine the signal intensities of an image are fat, haemopoietic cells, water and mineral. Mineral contributes in a negative way to the signal intensity due to lack of mobile protons. Images in the epiphysis of the femoral head are derived from haemopoietic cells and especially fat. As a rule epiphyseal and apophyseal ossification centres can be characterised as haemopoietically inactive, yellow marrow (Kricun 1985). This is characterised by high-signal intensities on T1-weighted images. Thus, when the amount of fat diminishes, one might expect the signal intensity to diminish. The consecutive MR manifestations in Perthes' disease showed a constant pattern.

Our prospective study examines MRIs in the early phase of Perthes' disease and records the changing signal patterns in correlation with radiographic changes.

PATIENTS AND METHODS

Fifteen consecutive patients (14 boys and one girl) with 16 affected hips were studied by serial radiography and MRI. All patients had their first clinical and radiographic examination, including anteroposterior and Laenenstein lateral views, within one month of the first symptoms. The average age of the patients at the onset of symptoms was five years 10 months (range three years three months to 10 years one month).

Follow-up radiographs and MRI were made within two weeks of each other, approximately every six months. The study was continued at least until radiographs confirmed that epiphyseal resorption was complete. Every patient had at least three MRIs. A total of 48 MRI examinations were made. The mean follow-up time was two years (range one year six months to two years six months). The hips were definitively grouped at the end of the resorptive stage of the disease according to

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Figure 1a – Sagittal T1-weighted MRI. A subchondral low intensity fracture line is visible in the anterior and middle parts of the femoral epiphysis.
Figure 1b – Six months later the anteroposterior radiograph shows loss of epiphyseal height, a dense area in the dome of the epiphysis, with a considerable medial and small lateral fragment. A metaphyseal lesion is visible in the anteromedial area.
Figure 1c – Corresponding coronal T1-weighted MRI showing the repair tissue interface, which is subdivided into an outer cup-shaped low-intensity band (arrow) and an inner zone of intermediate signal intensity. A sequestrum is visible as a central dark structure in the dome of the epiphysis. A metaphyseal lesion of intermediate signal intensity is surrounded by a low-intensity rim.
Figure 1d – Corresponding coronal T2-weighted MRI. The inner area of the repair tissue interface is now characterised by high signal intensity (arrow). The size and the signal intensity (low) of the sequestrum are similar. The joint capsule is distended inferiorly due to fluid effusion (asterisk). The signal intensity of the central area of the metaphyseal lesion is high.

Figure 1e – One year later, the radiograph reveals further loss of epiphyseal height, a large sequestrum, a considerable viable medial fragment, a small viable lateral fragment and a diffuse metaphyseal lesion.
Figure 1f – Corresponding coronal T1-weighted MRI of the right hip. There is more epiphyseal flattening. The sequestrum is smaller. The inferomedial capsule is still distended with fluid (asterisk).
Catterall’s classification (1971), one hip in group I, five in group II, eight in group III and two in group IV.

MRI was not used to determine treatment. In cases of serious restriction of hip movement, bed-rest with skin traction was employed for at least three weeks. When considered necessary, treatment was initiated as soon as the radiological classification and at-risk signs described by Catterall (1971, 1982) could be identified. The period of treatment ranged from four to 18 months (mean 12 months). The hips in group I and group II and three hips in group III were not given treatment. Four hips in group III were treated in an abduction brace for a mean period of six months. Surgery was performed in one hip of group III, after a period of treatment in an abduction brace, and in the two hips in group IV. The surgical procedure included an acetabular shelf-plasty in these three hips.

**Magnetic resonance imaging.** Coronal and sagittal planes were examined; transverse planes were not used as the physis and infarcted bone give a similar low-intensity signal. MRI was performed on a 0.5 T Gyroscan superconductive system (Philips, Shelton, Connecticut, USA). A flexible ‘receive only’ surface coil was used in all examinations. Relatively T1-weighted spin echo (SE) sequences were made with a repetition time (TR) of 550 ms and an echo time (TE) of 30 ms. T2-weighting was emphasised by using SE sequences with TR of 1500 ms and a dual echo technique (TE 50 and 100 ms). Thick slices (5 mm) were made with the T1-weighted sequence in the coronal and sagittal planes. The relatively T2-weighted sequence was made in the coronal plane with 7 mm thick slices. The slice intergap was 0.1 mm for all sequences. The acquisition matrix was 178 × 256, the display matrix varied from 256 × 256 to 512 × 512.

Figure 2a – Coronal T1-weighted MRI six months after the first symptoms, revealing the repair tissue interface as a thin low intensity semilunar band (arrow) in the epiphysis of the right hip, surrounding an area of intermediate intensity. The medial and lateral aspects of the epiphysis are uninvolved. Figure 2b – Corresponding sagittal T1-weighted MRI of the right hip. The subchondral fracture remains visible as a low-intensity line. Only a small part of the posterior aspect of the epiphysis is uninvolved. Figure 2c – One year later, the sagittal T1-weighted MRI shows further healing of necrotic bone. This is reflected by the forward spread of a normal signal intensity.

Two to four measurements per data line were used. A two-dimensional Fourier reconstruction technique was used in all instances.

**RESULTS**

In the initial phase of the disease a subchondral low-intensity line was visible in all hips on both T1- and T2-weighted MRI (Fig. 1). Except in the only hip in group I, the second set of MRIs showed that the repair process started distally. This was characterised on T1-weighted images by a semilunar or cup-shaped low-intensity band with its convexity directed distally in the femoral epiphysis or metaphysis, and an inner zone of intermediate signal intensity (Figs 1 and 2). This has previously been described with histological correlation in studies of femoral head necrosis in adults, as the repair tissue interface (Lang et al 1988; Vogler and Murphy 1988; Mitchell et al 1989). It corresponds with thickened trabecular bone, and fibrous repair tissue. The signal intensity of the inner portion of the repair tissue interface, which correlates with radiolucent areas, increases on the T2-weighted images. This has been ascribed in previous studies to increased water content in hyperaemic granulation tissue (Lang et al 1988; Mitchell et al 1989). The repair tissue interface separates the intact trabeculae from the necrotic area.

Dead trabecular bone, either intact or fractured, that had not been reached by the invading repair tissue, remains visible as a core-shaped structure of low signal intensity on T1- and T2-weighted images in the dome of the epiphysis (Fig. 1). This sequestrum was reabsorbed at a later stage.
The outer zone of the repair tissue interface on MRIs (low intensity on T1- and T2-weighted images) appears to be similar to the sclerosis at the periphery of the lesion seen on radiographs (Fig. 1). This is defined as new bone formation, also known as 'creeping substitution' (Pheimister 1930). The metaphysis and joint-space are represented on the second and third MRI by a signal of intermediate intensity on T1-weighted images and by a high-signal intensity on T2-weighted images in hips of group III and IV, consistent with oedema and fluid accumulation in the joint-space (Figs 1 and 3).

Group I (one hip). The only hip in group I was without symptoms whilst the contralateral hip had the typical signs of Perthes' disease (group IV). An osteolytic area in the superior part of the epiphysis on the radiograph corresponded with a low-signal intensity area on the T1- and T2-weighted MRIs (Fig. 3). The signal intensity of the underlying epiphysial bone did not change during the course of the disease and gave the same signal as subcutaneous fat. The infarcted area in the dome of the epiphysis became smaller and finally showed high-signal intensity on T1-weighted images, consistent with new bone formation (Fig. 3). No metaphysial changes were evident on radiographs or MRIs.

Group II (five hips). In the initial phase, all the radiographs demonstrated an increased density of the affected epiphysis. Only in three hips did a subchondral fracture line extend from the anterior to the mid-superior area on the radiographs. On coronal MRIs a subchondral fracture appeared as a low-intensity line covering the lateral half of the epiphysis, sparing an area of the lateral part of the epiphysis. The MRIs in the sagittal plane showed a fracture line covering the complete anterior half of the epiphysial bone. Epiphysial bone appeared on T1- and T2-weighted images as an intermediate signal intensity in three hips, whereas in two hips the signal intensity of the whole epiphysis appeared to be normal.

Radiographs taken six months later showed resorption of the central epiphysial area with slight loss of epiphysial height. The corresponding MRIs revealed the characteristic semilunar shaped repair tissue interface in the epiphysis on T1-weighted coronal images (Fig. 2). This interface demarcated living bone medially and laterally from the central area of dead bone in all hips. The sagittal T1-weighted images revealed that the posterior part of the epiphysis was not involved (Fig. 2).

Another six months later still, the radiographs showed resorption of the central area of the epiphysis, indicating healing. The corresponding coronal and sagittal T1-weighted MRIs displayed an increase of normal signal intensity, indicating healing. In the anterosuperior part of the epiphysis, immediately under the articular cartilage, a small core-shaped signal-void structure was persistently seen on T1- and T2-weighted images, consistent radiographically with unresorbed necrotic bone. On the T1-weighted sagittal MRIs the extent of the low-signal area in the anterior aspect of the epiphysis was reduced, indicating that during the process of healing the semilunar band had moved in a cranial and anterior direction in all five hips (Fig. 2). In all hips cyst-like changes in the metaphysial area were represented by intermediate signal intensities on T1- and by high-signal intensity areas on T2-weighted images, surrounded by a band of low intensity. Articular cartilage appeared to be thickened on the medial, dorsal and lateral aspects of the femoral head.

Group III (eight hips). In the initial phase of the disease a subchondral fracture line, extending from the anterior to the posterior surface, was visible on the radiographs of five hips, while MRIs in all hips demonstrated that a small part of the lateral and posterior aspect (Fig. 1a), and a larger part of the medial aspect of the epiphysis, was not affected by the subchondral fracture line. The femoral capital epiphysis was represented by a high (unchanged) signal intensity on MRIs in three hips and by an intermediate signal intensity in five hips.

Radiographs examined six months later showed a large dense area in the central part of the epiphysis with a sizeable viable medial fragment and a smaller viable lateral fragment (Fig. 1b). The corresponding MRIs showed a characteristic repair tissue interface on coronal T1- and T2-weighted images (Figs 1c and d). The central area of the interface reached the growth plate, demarcating a rather large unaffected medial and a small unaffected lateral part of the epiphysis. A centrally situated oval core, of low intensity on T1-weighted coronal images, represented dead trabecular bone (sequestrum). The whole epiphysis then had an intermediate signal intensity. The femoral head appeared to be flattened, due to a loss of height in the epiphysis. In the metaphysial area separate lesions were visible in all hips, characterised by a low intensity outer circle on T1- and T2-weighted images (Figs 1c and d). The area within these lesions was characterised on T1-weighted images by an intermediate signal intensity and on T2-weighted images by a high-signal intensity, consistent with repair tissue.

After a further six months the radiographs showed more loss of epiphysial height, a viable medial fragment, resorption of the affected area, and a large central sequestrum (Fig. 1e). T1- and T2-weighted MRIs revealed that the size of the sequestrum had decreased and that the femoral head was flattened (Fig. 1f). Articular cartilage appeared to be thickened particularly on the medial and lateral aspects of the femoral head.

Group IV (two hips). In the earliest phase of the disease the radiographs showed a subchondral fracture line covering the whole epiphysis in one hip, and increased density of the whole epiphysis in both hips. MRIs at this stage showed a subchondral, low-intensity fracture line covering the complete epiphysis, on the coronal and sagittal T1-weighted images. The underlying bone of the epiphysis was characterised by an intermediate signal intensity on T1- and T2-weighted images in both hips.

After six months, the radiographs showed a total...
collapse of the epiphysis, widening of the joint space, diffuse metaphyseal involvement and lateral uncovering of the femoral head. On MRIs the outer low-intensity band of the repair tissue interface was visible in the metaphysis to such an extent that the growth plate was situated within this line. The small epiphysis was represented by a broken line of low intensity.

Six months later still, the resorption appeared to be complete on the radiographs (Fig. 3a). New bone formation was visible in the medial and lateral parts of the epiphysis. There was severe flattening of the femoral head. The MRIs showed thickened articular cartilage over the whole femoral head and an increased intensity of the epiphysis and metaphysis on T1- and T2-weighted images (Figs 3b and c).

DISCUSSION

There have been many reports of MRI with histological correlation of ischaemic necrosis of the femoral head in adults (Bassett et al. 1987; Markisz et al. 1987; Lang et al. 1988; Mitchell et al. 1989), but few studies in Perthes' disease (Scoles 1984; Pinto, Peterson and Berquist 1989).

Salter and Thompson (1984) stated that in the very early phase of Perthes' disease the subchondral fracture line reveals the extent of the ischaemic zone. Unfortunately, however, the subchondral fracture line is a transient radiographic phenomenon seen only in 25% of cases (Catterall 1982). In our patients the subchondral fracture line was visible on the radiographs of 10 of the 15 affected hips, but on all MRIs and well beyond the early phase of the disease. The normal signal intensity of the underlying capital epiphysis had not changed in six hips on the first MRIs, whereas in nine hips it had changed to an intermediate or low intensity on T1- and T2-weighted images, consistent radiographically with thickened trabecular bone (Fig. 1). Therefore a consistently decreased signal intensity, as seen in 10 hips, does not necessarily represent the early stage of cell death in the epiphysis (Genez et al. 1988). On the other hand, early necrosis may display normal signal intensities.

Catterall assessed the extent of epiphyseal involvement on radiographs and therefore the precise degree of involvement could not be determined before the process of resorption was complete (Hardcastle et al. 1980; Van Dam et al. 1981). On MRIs the repair tissue interface can indicate whether and to what extent the infarcted bone includes the growth plate. The second set of MRIs, six months after the onset of the disease, precisely delineates the extent of the infarcted zone below the subchondral fracture line. This means that MRIs show the true extent of necrosis sooner than radiographs. In the later stages of the disease the MR findings and radiographs correlated more closely.

In group II the extent of the necrosis was restricted to the epiphysis with considerable viable bone medially, laterally and posteriorly, separated by an interface of repair tissue. The growth plate was not involved. These
hips did not suffer from loss of epiphyseal height and did not run the risk of growth disturbance.

In group III the growth plate was not involved in the infarcted bone area, but the extent of the epiphyseal necrosis appeared to be greater. These hips suffered the most loss of epiphyseal height at follow-up. As the repair tissue interface in group IV was located in the metaphysis beyond the growth plate, substantial loss of epiphyseal height is to be expected.

The concept that total ischaemia of the epiphyseal head precedes a subchondral fracture (Salter and Thompson 1984) can be confirmed on MRIs in groups III and IV, but not in those of groups I and II. In group II the medial, lateral and posterior aspects of the epiphysis show persistent normal signal intensities during the disease.

We concluded from this study that MRI will allow the orthopaedic surgeon to diagnose Legg-Calve-Perthes disease at an earlier stage than radiography. Also, the extent of the infarcted bone can be determined sooner and more precisely. Therefore, MRI will facilitate classification and can assist in treatment decisions at an earlier stage. We anticipate better results of treatment, especially in groups III and IV.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

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