The cause of subchondral bone cysts in osteoarthrosis

A finite element analysis

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Background **The etiology of subchondral bone cysts in arthrotic joints is unclear.**

Materials and methods **We used two-dimensional finite element analysis to evaluate the hypothesis that subchondral bone cysts in the osteoarthrotic hip joint may be the result of microfractures caused by localized cartilage defects or a thinned layer of cartilage. We evaluated the equivalent bone stress (von Mises (VM) stress) in the cancellous bone as an indicator of potential microfractures and further development of cystic lesions.**

Results **Cartilage defects induced stress peaks in the subchondral bone. This peak stress distribution corresponded to the clinical observation of development of acetabular and femoral subchondral cysts in a "kissing" position. A femoral subchondral bone cyst induced a stress peak at the corresponding acetabular site, whereas subchondral acetabular cysts did not increase stress in the femoral head. Acetabular cysts showed an increased level of stress at the lateral and medial border of the lesion which was much higher than the stress levels in the femoral head, indicating a tendency to faster growth.**

Interpretation **Our study supports the theory that stress-induced bone resorption may cause development of subchondral bone cysts in osteoarthrosis.**

Despite the fact that subchondral bone cysts are a common finding in osteoarthrosis, their etiology is still being debated (Plewes 1940, Resnick et al. 1977). Freund (1940) found evidence that pressure-induced intrusion of synovial fluid may cause enlargement of cystic lesions, but this does not explain the presence of subchondral cysts without joint contact (Freund 1940, Landells 1996). Bone necrosis due to "violent impact" between opposing joint surfaces without cartilage shelter has been suggested (Rhaney and Lamb 1955). This mechanical theory was further investigated by Ondrouch (1963) who showed that the induction of subchondral mechanical stress by unevenness of the articular surface caused bone cysts in his photoelasticity model. Our theory was that stress-induced microfractures of the subchondral bone may be primary events in the development of subchondral bone cysts in osteoarthrosis. We used a finite element model of the hip joint to study stress distribution in relation to cartilage destruction.

Materials and methods

We constructed a two-dimensional axisymmetric finite-element model (Figure 1). It consisted of the acetabulum, the joint cartilage on both sides and the femoral head. Each bone was modeled with a cortical layer, the cancellous bone and the cartilage. Linear, homogenous and isotropic material properties were used to simplify the model (Schoenfeld et al. 1974, Reilly and Burstein 1975, Athanasiou et al. 1995). Vertical shifting was prescribed at the acetabulum, and the femoral head was kept in a fixed position. The maximum load used was

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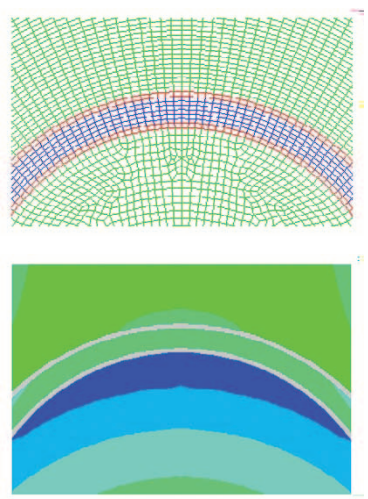

Figure 1. Stress distribution in a finite-element model of the hip joint with undestroyed cartilage. With an evenly calculated cartilage thickness of 2.8 mm, no stress peaks are visible.

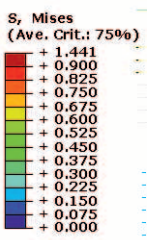

Figure 2. Color scale of the von Mises stress (MPa) resulting in the cancellous bone after loading.

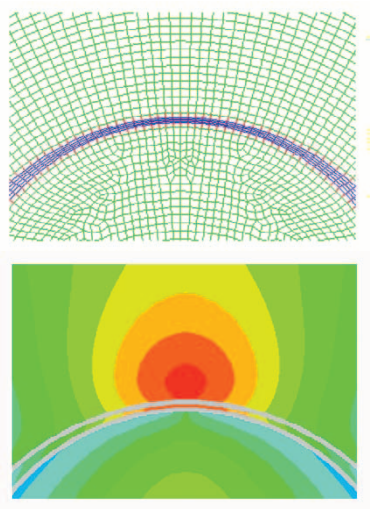

Figure 3. Stress distribution in a finite-element model of the hip joint with a continuously thinned cartilage layer. There is a clear stress peak in the center of the acetabulum, whereas the femoral head shows no signs of increased stress distibution.

chosen to result in a reaction force of 1.13 kN. At least this force is achieved by individuals with more than 58 kg of body weight, assuming an average hip load of twice the body weight. Different models were calculated: (1) an intact cartilage; (2) an increasingly thinned cartilage layer; (3) a local cartilage defect on the acetabular site, the femoral head or on both; (4) total loss of cartilage, and (5) cystic lesions either in the acetabulum or the femoral head.

As an indicator of potential microfractures and further development of cystic lesions, we evaluated the equivalent bone stress (von Mises (VM) stress) in the cancellous bone. The stress distribution was scaled in such a way that all figures showed the same color scale (Figure 2) and the local differences in the cancellous bone were kept visible. As a consequence, the cortical stress distribution is not visualized. All figures are mirrored at the rotational axis to enhance the overview.

In local cartilage lesions, the contact problem between the cortical layer was taken into account. The models had between 4266 and 8560 degrees of freedom. The finite-element system ABAQUS was used.

Results

An undestroyed cartilage could distribute the applied load evenly without any peaks of stress (Figure 1). If the cartilage layer was continuously thinned to the center of the model, a high stress peak was visible in the central region of the acetabulum (Figure 3). Remarkably, this kind of defect did not cause a major increase in stress on the femoral head. This situation changed with the creation of a localized full-thickness cartilage defect in the center of the model (Figure 4). Now the subchondral cancellous bone of the femoral head showed a high stress peak just beneath the lesion. In addition, the acetabular stress increased at the rims of the defect. This peak stress distribution corresponds to the clinical observation that acetabular and femoral subchondral cysts are likely to develop next to each other. If the cartilage defect was calculated on the femoral or the acetabular site only, the resulting stress had a lower peak level, especially in the femoral subchondral bone, but there were no differences in stress distribution. Smoothing of the rim borders also reduced the peak levels. If the cartilage layer was totally removed (Figure 5), a dramatic increase in stress resulted, especially in the subchondral acetabular bone.

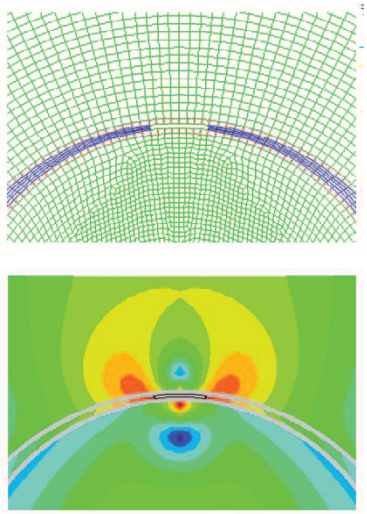

Figure 4. Stress distribution in a finite-element model of the hip joint with a central full-thickness cartilage defect. A localized high increase in stress distribution was calculated in the femoral head just beneath the defect, with additional peaks in the acetabulum at the rims of the lesion.

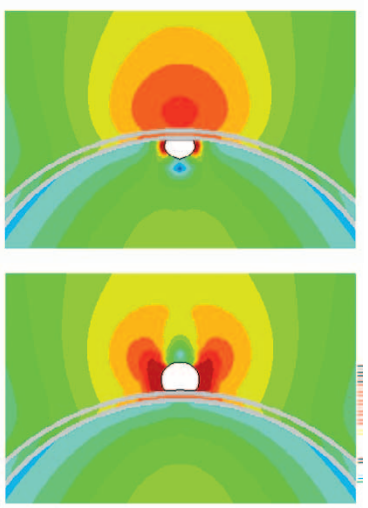

Figure 6. Stress distribution in a finite-element model of the hip joint with a central subchondral cyst in the femoral head or the acetabulum. There is increased stress above the femoral cyst in the acetabulum, an effect not observed in the acetabular cyst. As a secondary effect, a major increase in stress at the lateral rims of the cystic lesions, more pronounced at the acetabulum, is visible.

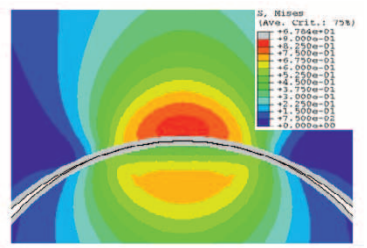

Figure 5. Stress distribution in a finite-element model of the hip joint with a total loss of cartilage. A maximum stress peak is visible in the center of the acetabulum.

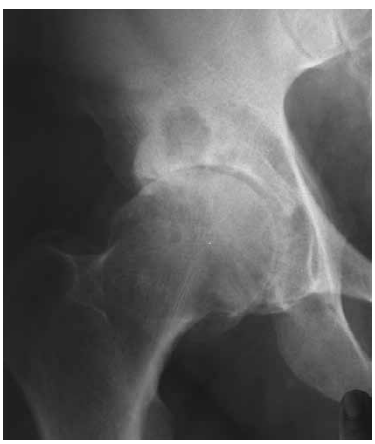

Figure 7. Radiograph of the right hip in a 60-year-old patient with coxarthritis, showing a large acetabular cystic lesion.

To simulate the effect of subchondral bone cysts, we simulated such lesions in the subchondral acetabular or femoral bones (Figure 6). Interestingly, a femoral subchondral bone cyst induced a stress peak at the corresponding acetabular site, whereas subchondral acetabular cysts did not increase the stress in the femoral head. As expected, acetabular cysts also showed an increased level of stress at the lateral and medial borders of the lesion, much higher than those in the femoral head, indicating a tendency to faster growth (Figure 7).

Discussion

Some subchondral bone cysts, for example in rheumatoid arthritis, may be induced by invasion of pannus tissue (Magyar et al. 1974, Ginsberg et al. 1975, Rennell et al. 1977). The origin of secondary subchondral cystic lesions, for example

ganglion cyst or synovial cyst, is less clear. Most authors propose that these develop from a defect in the joint surface with either protrusion of fluid or an active synovial proliferation into the bone or the cyst (Hicks 1956, Crane and Scarano 1967, Willems et al. 1973). This mechanism may occur secondary to mechanical breakage of the thin sclerotic subchondral rim in a preformed cyst. The intrusion of fluid or synovial tissue that follows may then cause rapid enlargement, often seen in ganglion bone cysts (Schmalzried et al. 1997). This mechanism has been proposed as the etiology behind subchondral cystic changes in osteoarthrosis (OA), but there is no convincing evidence of a regular communication between the cysts and the joint. Most authors assume that detailed histological sectioning would reveal such a communication, i.e. that a tissue plug has terminated fluid intrusion (Milgram 1983). Ondrouch (1963) rejected this theory, mainly because defects of the subchondral bone are often found in healthy joints without cyst formation (Ekholm and Norbäck 1951). Moreover, Ondrouch stated that this theory does not explain the paired, or so-called kissing, position on both sides of the joint and the formation of a soft-tissue plug in the communicating channel. This plug cannot stand pressure conditions that should be able to cause the breakage of the cancellous subchondral bone itself.

The theory of Ondrouch and our finite element analysis may explain the development of subchondral bone cysts in osteoarthritis. As shown in Figure 1, an intact cartilage layer distributes the applied load evenly across the subchondral bone region. As soon as the cartilage thins or totally deteriorates, increased circumscript stress will develop, especially on the acetabular side (Figures 3-5). Shape and location of the stress peaks represent the patterns found in osteoarthrotic cysts (Ondrouch 1963). The peak levels of the calculated von Mises stress are in the region of those found experimentally as ultimate compression strength in cancellous bone (0.51–5.6 MPa), and may result in microfractures (Rohl et al. 1991).

Our findings suggest that stress-induced microfracture may be the first step in the development of subchondral bone cysts in OA. Secondary effects, such as osteoclast resorption, then induce the cystic lesion itself (Sabokbar et al. 2000). The fibrous tissue often seen in these lesions shows an upregulation of cytokines such as prostaglandin E2, which may then enhance further expansion of the cyst (von Rechenberg et al. 2001).

In conclusion, our study supports the theory of Ondrouch that stress-induced bone resorption may be the cause of the development of subchondral bone cysts in osteoarthrits. Cartilage defects, either localized or a general thinning, are the primary lesions inducing stress peaks in the subchondral bone. If a subchondral cyst exists in the femoral head, it has a tendency to grow through raised lateral and medial stresses and induce stress peaks, followed by cyst development on the opposite acetabular side. In contrast to cysts on the femoral head, acetabular cysts have a greater tendency to become enlarged due to high stresses in their walls, but do not significantly increase subchondral femoral head stress and may therefore remain solitary for longer periods of time.

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