Osteonecrosis in retrieved femoral heads after failed resurfacing arthroplasty of the hip

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We present the histological findings of bone retrieved from beneath the femoral components of failed metal-on-metal hip resurfacing arthroplasties. Of a total of 377 patients who underwent resurfacing arthroplasty, 13 required revision; for fracture of the femoral neck in eight, loosening of a component in three and for other reasons in two. None of these cases had shown histological evidence of osteonecrosis in the femoral bone at the time of the initial implantation.

Bone from the remnant of the femoral head showed changes of osteonecrosis in all but one case at revision. In two cases of fracture which occurred within a week of implantation, the changes were compatible with early necrosis of the edge of the fracture. In the remaining six fractures, there were changes of established osteonecrosis. In all but one of the non-fracture cases, patchy osteonecrosis was seen.

We conclude that histological evidence of osteonecrosis is a common finding in failed resurfaced hips. Given that osteonecrosis is extensive in resurfaced femoral heads which fail by fracture, it is likely to play a role in the causation of these fractures.

Resurfacing arthroplasty as a treatment for arthritis of the hip is an attractive option since it conserves bone on the femoral side. Previous series using a metal femoral resurfacing component articulating with a polyethylene acetabular component showed promising early results, although the risk of early failure from fracture of the femoral neck was noted. Subsequently, in the medium term there have been unacceptably high rates of failure related to polyethylene wear.1 Throughout this period, the potential concern of osteonecrosis of the femoral head was expressed, but this was generally dismissed by the proponents of the technique.

With the advent of improved metallurgy and manufacturing techniques, the concept of hip resurfacing using metal-on-metal bearings has become popular,3-5 although fracture of the femoral neck continues to be documented as a complication.5 While mechanical and technical factors are possibly involved in the aetiology of these fractures, concern persists about the possible role of osteonecrosis of the remnant of the femoral head.

In order to determine whether osteonecrosis is involved in the pathogenesis of these fractures, we have followed all patients with metal-on-metal hip resurfacings performed in our unit since 1998. We have considered the histological results of a retrieval analysis of femoral components from patients in whom revision of the femoral side was required, regardless of the mode of failure.

Patients and Methods

Since 1998, 377 metal-on-metal hip resurfacings have been performed by consultants and appropriately supervised trainees at our unit for the treatment of end-stage arthritis. The implants used were 358 Birmingham hip replacements (MMT, Birmingham, UK) and 19 Cormet 2000 replacements (Corin, Cirencester, UK). We have considered them as one group since the differences, mainly in the metallurgy and design of the acetabular component, were unlikely to substantially affect the risk of osteonecrosis as a consequence of the surgery. Fifteen of these patients required revision surgery, eight for fracture of the femoral neck, five for loosening of a component, one for culture-negative inflammation and one for persistent pain. Two cases of acetabular loosening, in which the femoral head was retained and the cup only revised, were excluded since there was no femoral bone to review. The details of the 13 cases are presented in Table I.

In all cases, the standard technique described in the manufacturers’ instructions was followed by all surgeons, including the use of the
Table I. Details of the patients and indications for surgery

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yrs)/gender</th>
<th>Primary indication*</th>
<th>Mode of failure</th>
<th>Weeks to revision</th>
<th>Revision procedure</th>
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<tbody>
<tr>
<td>1</td>
<td>59/M</td>
<td>OA</td>
<td>Fracture</td>
<td>4</td>
<td>THA</td>
</tr>
<tr>
<td>2</td>
<td>52/M</td>
<td>OA</td>
<td>Fracture</td>
<td>6</td>
<td>THA</td>
</tr>
<tr>
<td>3</td>
<td>59/M</td>
<td>SUFE</td>
<td>Fracture</td>
<td>1</td>
<td>THA</td>
</tr>
<tr>
<td>4</td>
<td>56/M</td>
<td>DDH</td>
<td>Fracture</td>
<td>8</td>
<td>THA</td>
</tr>
<tr>
<td>5</td>
<td>45/M</td>
<td>OA</td>
<td>Fracture</td>
<td>21</td>
<td>THA</td>
</tr>
<tr>
<td>6</td>
<td>60/M</td>
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<td>1</td>
<td>THA</td>
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<tr>
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<td>47/F</td>
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<tr>
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<td>56/M</td>
<td>OA</td>
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<td>22</td>
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<tr>
<td>9</td>
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<td>THA</td>
</tr>
<tr>
<td>10</td>
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<td>Inflammation</td>
<td>78</td>
<td>THA</td>
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<tr>
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<td>36/F</td>
<td>SC</td>
<td>Persistent pain</td>
<td>105</td>
<td>THA</td>
</tr>
<tr>
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<td>OA</td>
<td>Cup loose</td>
<td>171</td>
<td>THA</td>
</tr>
<tr>
<td>13</td>
<td>59/F</td>
<td>OA</td>
<td>Femoral loosening</td>
<td>25</td>
<td>THA</td>
</tr>
</tbody>
</table>

* OA, osteoarthritis; SUFE, old slipped upper femoral epiphysis; DDH, old developmental dysplasia of the hip; AS, ankylosing spondylitis; SC, synovial chondromatosis

results

The patients' clinical records and radiographs were reviewed for evidence of possible mechanical factors predisposing to fracture, including the position and seating of the femoral component and evidence of notching of the femoral neck, as well as for any known risk factors for avascular necrosis documented before either the initial or revision procedures. The mean time to failure of the arthroplasty was nine weeks (1 to 69) for those with a fracture and 77 weeks (8 to 171) for those with other causes. At the time of revision the femoral component in all those who had a fracture was found to be secure.

In two specimens (cases 3 and 6) the fracture occurred within seven days of the operation. Subchondral bone in these cases showed necrotic and degenerative changes, haemorrhage into the fatty marrow and reparative fibrous and granulation tissue formation. None of these cases showed a loss of osteocyte nuclei from lacunae within bone trabeculae. These changes are seen both in early osteonecrosis and after a recent fracture.

In cases 1, 2, 4, 5, 7 and 8, in which fracture occurred after the implant had been in situ for between four and 69 weeks, there was also evidence of a reparative response to recent fracture. In addition, there was loss of osteocyte nuclei from lacunae within thickened cancellous bone trabeculae, and formation of appositional new bone was seen on the surface of necrotic bone trabeculae (Fig. 1). These changes are indicative of established osteonecrosis.

In five cases, failure of the implant occurred after it had been in situ for between eight and 105 weeks and was not associated with an obvious fracture, although the femoral component was revised. Variable changes were found in the bone of the femoral head although in all but one of these cases there was histological evidence of bone necrosis. In case 13, in which the implant had been in situ for 25 weeks, necrotic and degenerative changes and fibrosis were seen in marrow fat. In case 9, in which the implant had been in situ for eight weeks, similar changes as well as a patchy chronic inflammatory cell infiltrate, composed mainly of lymphocytes and macrophages, were noted in the fatty marrow. Most of the bone trabeculae in this case appeared to be viable, but extensive remodelling was seen on the surface of many cancellous bone trabeculae, some of which showed focal loss of osteocyte nuclei from lacunae. In case 10, in which the implant had been in situ for 78...
weeks, most cancellous trabeculae in the femoral neck were viable, but focal areas of osteonecrosis with loss of osteocyte nuclei from lacunae and the formation of appositional new bone were also noted. Case 11, in which the implant had been in situ for 105 weeks, also contained areas of viable and necrotic bone. In the necrotic bone, trabeculae were thickened and there was widespread loss of osteocyte nuclei from lacunae with extensive formation of appositional new bone (Fig. 2). Changes of osteonecrosis in bone trabeculae were seen in both superficial and deep areas of the recovered femoral heads. Only in case 12, in which the implant had been in situ for 171 weeks, did the sampled bone appear to be viable with no changes typical of osteonecrosis.

Notching of the cortex of the neck and failure to cover all the prepared cancellous bone of the femoral neck have been associated with fractures. Radiographs of hips which fractured were inspected for these features. In case 7, the femoral component was incompletely seated leaving exposed prepared cancellous bone. There was a superior notch of 5 mm noted in case 1. An inferior overhang of the femoral component was seen in cases 5 and 6, measuring 3 mm and 4 mm, respectively, although in neither case was a notch noted at the time of implantation or on post-operative radiographs. Two patients showed no radiological 'at-risk' features. In the two cases in which the fracture occurred before the initial post-operative check radiograph, analysis of the radiological features could not be carried out.

Discussion
We have found evidence of post-operative osteonecrosis in bone sampled from 12 of 13 retrieved femoral heads obtained at revision surgery after failure of a hip resurfacing arthroplasty. Since none of the patients in our series had any histological evidence of osteonecrosis in the femoral bone removed at the time of implantation, we conclude that this occurred after the resurfacing surgery and was probably attributable to it. There were three broad patterns of osteonecrosis seen in the bone sampled.

In the two patients in whom the fracture had occurred before the post-operative check radiograph, there was necrosis of the marrow reparative fibrous and granulation tissue formation. These histological changes are seen after recent fracture and in early osteonecrosis. Since the changes seen in these two conditions are similar, it is not possible to state categorically whether the osteonecrosis contributed to the fracture or whether the histological changes of early osteonecrosis merely reflected the presence of a recent fracture. In both of these cases, the patients had undergone bilateral simultaneous resurfacing and the first side to be operated on sustained the fracture. In these cases, we speculate that the fractures may have occurred for mechanical reasons, perhaps associated with impaction of the cup during the resurfacing of the second side.

In the remaining six patients who had a fracture of the femoral neck occurring after the implant had been in place for more than four weeks, there was prominent loss of osteocyte nuclei from lacunae in cancellous bone and formation of appositional new bone. These changes were seen in addition to those of a recent fracture. Since loss of osteocyte nuclei from lacunae does not usually occur earlier than seven days after the blood supply to bone has been interrupted,6,7 this suggests that the fracture in these cases took place against the background of established bone necrosis (i.e. osteonecrosis was present for more than seven days and so predated the fracture).
In four of the remaining five cases, in which revision was not performed for fracture, there was evidence of osteonecrosis in the bone from the retrieved femoral head. In these cases, there were patchy areas of established osteonecrosis with loss of osteocyte nuclei and of viable bone in variable proportions. The changes were less uniform than in the delayed fracture group.

Since none of the hips examined had evidence of osteonecrosis at the time of implantaion, it seems probable that the resurfacing surgery is related to the osteonecrosis. The extensive surgical approach required disrupts the extraosseous circulation during capsulectomy and the intraosseous circulation is disrupted as a result of mechanical and thermal injury during preparation of the femoral head, insertion of the component and curing of the cement. This combination could cause osteonecrosis.

The differing histological patterns of established osteonecrosis suggest that bone necrosis occurs commonly after a resurfacing arthroplasty and that it is likely to play a role in the aetiology of post-operative fracture. We believe that patients who sustain a widespread vascular insult at the time of resurfacing are placed at an increased risk of failure by fracture of the femoral neck, particularly when the neck has additionally been weakened mechanically. The interval required for resorption of avascular bone would explain the timing of the fractures. When the vascular insult is less extensive, the patient is likely to go on to long-term success, unless the implant fails for another unrelated cause, in which case the coincidental patchy osteonecrosis can be detected histologically.

Previous authors have reached different conclusions about the significance and cause of the osteonecrosis which is seen in bone after a resurfacing arthroplasty. Howie, Cornish and Vernon-Roberts felt that necrotic changes were secondary to loosening of the component. However, in our study, the femoral components were secure in 12 of the 13 cases, yet osteonecrosis was a common finding. Campbell, Mirra and Amstutz suggested that osteonecrosis may result from thermal injury after polymerisation of cement. However, in our study, histological changes of osteonecrosis were found in bone samples taken both at the cement-bone interface and remote from this site. These findings would argue against this explanation. Other authors have shown that resurfacing of the head can cause necrosis of the bone in the subcapital region and that this may precipitate post-operative fracture of the femoral neck. Our findings support this view, but should be seen in the context of our whole series of implants, with post-operative fracture seen in 1.9% of cases and a rate of dislocation of 0.3%. This suggests that resurfacing substitutes the risk of femoral neck fracture for that of dislocation seen in conventional arthroplasty. The histological evidence of osteonecrosis of the femoral head and neck occurring after resurfacing arthroplasty of the hip is presented as a factor which predisposes to fracture. More work is required in order to identify exactly where and how the blood supply to the head and neck is interrupted.

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References