BONE LYSIS IN WELL-FIXED CEMENTED FEMORAL COMPONENTS

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We have reviewed 25 cases of focal femoral osteolysis in radiographically stable, cemented femoral implants. In three hips retrieved at post-mortem from two patients, we have been able to make a detailed biomechanical and histological analysis.

The interval between arthroplasty and the appearance of focal osteolysis on clinical radiographs ranged from 40 to 168 months, and in over 70% of the cases this did not appear until after five or more years. Few had significant pain and there was no relation to age, sex or original diagnosis. The most common site for osteolysis were Gruen zones 2 and 3 on the anteroposterior radiograph and zones 5 and 6 on the lateral radiograph. In 15 cases (60%), the area of osteolysis corresponded to either a defect in the cement mantle or an area of very thin cement. The rate of progression of these lesions was variable, but to date only one has progressed to gross loosening of the femoral component.

The back-scatter scanning electron microscopic examination of serial sections and biomechanical testing of the post-mortem specimens demonstrated focal cement fracture around implants that were otherwise rigidly fixed. In eight cases from which tissue was available, histology showed a histiocytic reaction with evidence of particulate polymethylmethacrylate. We consider that this local fragmentation was the stimulus for local osteolysis in an otherwise stable cemented femoral component.

It is well recognised that bone lysis may occur in loose cemented femoral components; its cause in aseptic cases is probably multifactorial, including foreign-body reactions to particulate polymethylmethacrylate and polyethylene, enzymes such as collagenase, implant motion and hypersensitivity to implant materials.

Osteolysis around well-fixed cemented femoral components is much less common. We have previously reported four such cases (Jasty et al 1986), but the aetiology was not clear. We now provide follow-up data on the four initial cases, add 21 more cases, and report extensive biomechanical and histological data from three

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0301-620X/90/6019 2.00

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Radiograph of hip 9 showing osteolysis adjacent to a thin area of cement mantle in an otherwise well-fixed femoral component.
Table I. Details of 25 hips showing focal lysis about the femoral components

<table>
<thead>
<tr>
<th>Hip</th>
<th>Sex</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Cemented implant</th>
<th>Interval before lysis (months)</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>F</td>
<td>50</td>
<td>Old CDH</td>
<td>Harris type 1</td>
<td>63</td>
<td>Pain, acetabulum loose</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>34</td>
<td>Osteoarthritis</td>
<td>Trapezoidal 28</td>
<td>127</td>
<td>Mild pain</td>
</tr>
<tr>
<td>3*</td>
<td>F</td>
<td>50</td>
<td>Old CDH</td>
<td>Harris micromini</td>
<td>93</td>
<td>Pain, acetabulum loose</td>
</tr>
<tr>
<td>4*</td>
<td>F</td>
<td>57</td>
<td>Old CDH</td>
<td>Trapezoidal 28</td>
<td>60</td>
<td>No pain</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>51</td>
<td>Osteoarthritis</td>
<td>HD-2</td>
<td>80</td>
<td>No pain</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>69</td>
<td>Osteoarthritis</td>
<td>CAD</td>
<td>48</td>
<td>No pain</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>60</td>
<td>Failed THR</td>
<td>HD-2</td>
<td>103</td>
<td>Slight pain</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>69</td>
<td>Failed THR</td>
<td>HD-2</td>
<td>63</td>
<td>No pain</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>64</td>
<td>Osteoarthritis</td>
<td>CAD</td>
<td>141</td>
<td>No pain</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>64</td>
<td>Osteoarthritis</td>
<td>HD-2</td>
<td>52</td>
<td>No pain</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>59</td>
<td>Osteoarthritis</td>
<td>HD-2</td>
<td>51</td>
<td>No pain</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>68</td>
<td>Osteoarthritis</td>
<td>HD-2</td>
<td>127</td>
<td>Mild pain</td>
</tr>
<tr>
<td>13</td>
<td>F</td>
<td>47</td>
<td>Rheumatoid arthritis</td>
<td>CAD</td>
<td>149</td>
<td>Slight pain</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>49</td>
<td>Osteoarthritis</td>
<td>CAD</td>
<td>149</td>
<td>No pain</td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>48</td>
<td>Inflammatory arthritis</td>
<td>CAD</td>
<td>136</td>
<td>No pain</td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>71</td>
<td>Failed THR</td>
<td>CAD</td>
<td>134</td>
<td>Severe polyarticular arthritis</td>
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<tr>
<td>17</td>
<td>F</td>
<td>63</td>
<td>Post-traumatic arthritis</td>
<td>CAD</td>
<td>71</td>
<td>No pain</td>
</tr>
<tr>
<td>18</td>
<td>M</td>
<td>62</td>
<td>Acetabular fracture</td>
<td>Harris type 1</td>
<td>168</td>
<td>Pain, acetabulum loose</td>
</tr>
<tr>
<td>19</td>
<td>M</td>
<td>64</td>
<td>Osteoarthritis</td>
<td>Aufranc-Turner</td>
<td>102</td>
<td>Slight pain</td>
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<tr>
<td>20</td>
<td>F</td>
<td>66</td>
<td>Osteoarthritis</td>
<td>CAD</td>
<td>117</td>
<td>No pain</td>
</tr>
<tr>
<td>21</td>
<td>F†</td>
<td>39</td>
<td>AVN</td>
<td>HD-2</td>
<td>40</td>
<td>No pain</td>
</tr>
<tr>
<td>22</td>
<td>F†</td>
<td>70</td>
<td>Osteoarthritis</td>
<td>CAD</td>
<td>69</td>
<td>No pain</td>
</tr>
<tr>
<td>23</td>
<td>F</td>
<td>70</td>
<td>Hip fracture</td>
<td>Moore</td>
<td>42</td>
<td>No pain</td>
</tr>
<tr>
<td>24</td>
<td>F</td>
<td>42</td>
<td>CDH</td>
<td>Micromini</td>
<td>60</td>
<td>No pain</td>
</tr>
<tr>
<td>25</td>
<td>F</td>
<td>29</td>
<td>Post-traumatic arthritis</td>
<td>Harris type 1</td>
<td>96</td>
<td>No pain</td>
</tr>
</tbody>
</table>

* original case (Jasty et al 1986) † autopsy specimen

The clinical data of all 25 hips (24 patients) are summarised in Table I. Although 15 of the patients in this series had bilateral cemented femoral components, only one had bilateral localised femoral osteolysis (hips 22 and 23 in Table I). The initial diagnosis leading to the total hip replacement was osteoarthritis in 11 hips, congenital dysplasia in four, avascular necrosis in three, post-traumatic arthritis in two and inflammatory arthritis in two. The other three hips were revision arthroplasties for previous aseptic loosening of the femoral component. The implants were either stainless steel (3) or cobalt-chromium alloy (22), and all were fixed with Simplex P bone cement.

In 19 of the hips, the patients had reported either no pain or slight, occasional discomfort (Harris 1969). Two hips gave mild thigh pain, and four hips in four patients gave significant pain. Of these, however, there had loose acetabular components and one had generalised arthritis with diffuse joint pain.

The time interval between arthroplasty and the radiographic appearance of localised osteolysis ranged from 40 to 168 months. Contact between the stem of the implant and cortical bone was evident in 10 cases, usually in zones 3 or 5 (Gruen, McNeice and Amstutz 1979). In 11 other cases, the cement mantle was seen to be less than 1 mm thick, usually in zones 2, 3, 5 and/or 6. The most common sites for osteolysis were zones 2 and 3 on the anteroposterior radiograph and zones 5 and 6 on the lateral radiograph. In 15 hips (60%), the area of osteolysis corresponded either to the location of a mantle defect or to an area of thin cement (Fig. 1).

In the 11 hips for which there were comparable serial radiographs more than one year apart (range 1 to 7.5 years, mean 3 years), eight demonstrated progression of the lesion (Fig. 2), but at a variable rate. Only one component had become grossly loose. In one patient (not in our own series), who was referred to the senior author,
a femoral fracture had been caused by relatively minor trauma 10 years after a total hip replacement (Fig. 3).

Three of our four original cases (Jasty et al 1986) had revision surgery at which biopsies were taken. Two hips (1 and 2) had new cemented components which have remained stable at five and six years after revision, with no evidence of bone lysis. The third (3) had a resection arthroplasty with allografting of the femur and no new bone lysis was visible after six years. The fourth hip in the original series (4) and two other hips (20 and 25) had biopsy only. In hip 4, the biopsy site healed and follow-up radiographs at three years showed no bone lysis (Fig. 4). Hips 20 and 25 were biopsied only recently.

All the cultures from nine aspirations or surgical biopsies were negative and the histology in eight hips also showed no evidence of infection. Although the radiographic appearance of all lesions was similar, the histological appearance varied with the extent of the lesions radiographically.

In three cases with lesions less than 2 cm in diameter, the lytic area consisted of a cyst, lined by a thin layer of fibrous tissue. There were occasional macrophages, but particulate debris from implant material was rare or not identified. Immediately adjacent to the fibrous membrane, there was active osteoclastic resorption of the cortex. In some areas, the bone was necrotic and the haversian canals contained macrophages similar to those found in the region of the cyst lining.

In the three cases with more extensive lysis, the membrane was thicker. The surface layer adjacent to the cement was lined by large, polygonal, synovial-like cells.

Immediately underneath this layer were sheets of granular histiocytes, scattered foreign-body giant cells and particulate polymethylmethacrylate. Identical tissue filled the intertrabecular spaces. In some areas, macrophages abutted the endosteal surface while in other areas, osteoclasts were found in active resorption sites.

The material obtained at post-mortem examination showed some macrophages and occasional foreign-body giant cells. There was also necrotic bone with evidence of osteoclastic bone resorption. No case showed the synovial-like lining previously reported around loose cemented total hip replacements (Goldring et al 1983). Particulate polyethylene was not found in any specimen.

In all three cases studied biomechanically at post-mortem (hips 21, 22 and 23), the prostheses were remarkably stable, the maximum axial micromotion at loads simulating gait being only 10, 13 and 6 μm. At 150 inch-pounds of torque, simulating stair climbing, the rotational micromotion was only 88, 13 and 18 μm respectively and the cement–bone interface was completely intact with no intervening fibrous tissue except in the areas of lysis. In hip 21, multiple fractures were noted through multiple small voids in the cement mantle at the tip of the stem (Fig. 5). This local cement failure had generated particulate debris and there was endosteal bone lysis adjacent to the local cement fragmentation. In hip 22, there was a small defect in the mantle at the tip of the stem. The prosthesis had separated from the

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**Fig. 2a**
Radiographs of hip 19 showing osteolysis in zone 3 at 102 months and 32 months later. Progression of lysis is apparent.

**Fig. 2b**

**Fig. 3**
Femoral fracture through a lytic lesion at the tip of the prosthesis in an otherwise well-fixed femoral component.
cement at that level but no membrane was found between the cement and the metal, though there were multiple cement fractures with local cement fragmentation (Fig. 6).

DISCUSSION

We initially reported only four cases of local lysis from a series of over 3000 total hip replacements. However, the incidence of this complication is higher than we initially believed. The earliest bone lysis was found at 40 months following surgery; over 70% of cases did not occur until later than five years. In a recent review of 106 cemented total hip replacements with a minimum 10-year follow-up, seven cases of this lesion were noted (Mulroy and Harris 1990). Five of these had appeared for the first time between six and 11 years postoperatively.

Our findings indicate that lysis is caused by a foreign body reaction to particulate polymethylmethacrylate (Willert, Ludwig and Semlitsch 1974; Charnley 1975; Harris et al 1976; Miller et al 1978; Willert, Mueller and Semlitsch 1979; Freeman, Bradley and Revell 1982;
Scott, Riley and Dorfman 1985; Huddleston 1988). No cases had evidence of infection. Implant motion is not a likely aetiology. None of the five cases having re-operation or the three post-mortem specimens had loose femoral components. Detailed biomechanical analysis of the post-mortem specimens showed that these implants were as rigidly fixed as recently cemented implants in fresh cadaver specimens. Moreover, the histological specimens showed no evidence of the synovial transformation which is often seen at the cement–bone interface of grossly loose implants. This change is probably secondary to gross movement of the whole implant. We found no particular polyethylene.

Focal cement fracture can be expected in areas where there is an inadequate cement mantle, especially at the tip of the stem where stresses are high (Lewis et al 1984). The significance of this is supported by the association of lysis with mantle defects and thin cement both in our series and in other reports (Carlsson, Gentz and Linder 1983; Huddleston 1988). Once local cement fracture has occurred, particulate debris is generated; this results in a foreign-body response. This theory is supported by animal studies, which show that bulk methylmethacrylate is well tolerated but that particulate methylmethacrylate leads to a foreign-body response (Goldring et al 1986; Paiement et al 1986; Goodman, Fornasier and Kei 1988).

The rate of progression of these lesions is variable. Although Huddleston (1988) reported that lysis invariably leads to implant loosening, this has not been our experience. Only one femoral component in our series has progressed to loosening. However, some lesions enlarge fairly rapidly and all such cases should be followed regularly. As mentioned above, one case referred to us had a pathological fracture, and others have been reported (Pazzaglia and Byers 1984).

Our belief is that this process, in stable reconstructions, is an early, limited and focal example of the more extensive bone erosion seen in grossly loose cemented femoral components (Harris et al 1976; Goldring et al 1983). Small amounts of particulate methylmethacrylate appear to stimulate endosteal erosion and localised osteolysis. This study was supported by the William H. Harris Foundation, Boston, Massachusetts.

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.

REFERENCES


