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Alumina-Debris-Induced Osteolysis in Contemporary Alumina-on-Alumina Total Hip Arthroplasty

A Case Report

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A ceramic-on-ceramic bearing coupling, because of its qualities of reduced friction and wear, is an attractive alternative bearing surface in total hip arthroplasty. The use of such bearing couplings is likely to reduce problems related to polyethylene wear debris. Osteolysis has only rarely been reported in association with ceramic-on-ceramic bearing couplings, and reports are generally limited to cases involving early generation ceramic bearings or loosened prostheses. Recently, several cases of osteolysis have been reported after total hip arthroplasty with use of a contemporary alumina bearing, but these reports only described the femoral scalloping seen on radiographs and did not present any histological evidence that ceramic particles were the causative factor. We present the case of a patient who had formation of a large amount of osteolysis about both the acetabulum and the proximal part of the femur, induced by ceramic wear particles from a well-functioning contemporary alumina-on-alumina total hip prosthesis. The patient was informed that data concerning the case would be submitted for publication, and she consented.

Case Report

A sixty-three-year-old woman underwent bilateral total hip arthroplasty in June 1998 for the treatment of corticosteroid-induced osteonecrosis. The arthroplasties included the use of cementless implants (PLASMACUP SC-BICONTACT; Aesculap, Tuttingen, Germany) that incorporated 28-mm alumina femoral heads and alumina acetabular inserts (BIOLOX forte; CeramTec, Plochingen, Germany). The PLASMACUP had a roughened titanium plasma-sprayed exterior and a machined interior that accepted an alumina insert with a self-locking press-fit taper. The BiCONTACT stem was a tapered, rectangular, titanium-alloy implant, the proximal one-third of which was surface-treated with titanium plasma spray. The

Fig. 1
Anteroposterior radiograph of the right hip, showing expansive, geographic osteolytic lesions about the acetabular cup and greater trochanter (arrows) eight years after alumina-on-alumina total hip arthroplasty.

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alumina head was secured to the stem by means of a tapered cone. After surgery, the patient did not present for regular follow-up visits and did not revisit our clinic until June 2006. At the time of that visit, although she had no hip symptoms or discomfort, plain radiographs showed expansive, osteolytic lesions about the acetabular cup and greater trochanter of the right hip (Fig. 1). These osteolytic lesions measured approximately 39 × 18 mm in the acetabulum and 15 × 27 mm in the greater trochanter on the anteroposterior radiograph. There was no evidence of osteolysis in the left hip. Radiographic evidence of implant loosening was not found in either hip. The erythrocyte sedimentation rate was 17 mm/hr (normal range, 0 to 9 mm/hr), and the C-reactive protein level was 1.65 mg/dL (16.5 mg/L) (normal range, 0 to 0.5 mg/dL [0 to 5 mg/L]). Routine bacterial cultures of bloody fluid (5 mL) aspirated from the greater trochanteric lesion under ultrasonographic guidance were negative.

Intraoperative findings included a black discoloration of the bearing surface of the alumina liner, which was most prominent posterosuperiorly (Fig. 2). This black discoloration was also observed on the alumina femoral head but was not as extensive. There was no gross evidence of alumina bearing wear, fracture, or crack formation. In addition, there was no evidence of scratching of the metal surface or any sign of impingement between the acetabular cup and the neck of the stem. After the liner was retrieved, curettage and allogeneic bone-grafting were performed to treat the osteolytic lesions in the acetabulum and greater trochanter and implantation of a new polyethylene liner was carried out. The alumina femoral

Fig. 2
Photograph demonstrating black discoloration, which is most prominent posterosuperiorly, on the bearing surface of the retrieved alumina liner. No evidence of alumina wear, fracture, or crack formation is evident on gross observation.

Fig. 3
Photomicrograph showing abundant alumina wear particles and a diffuse macrophagic-lymphoplasmacytic infiltrate in the osteolytic tissue from the acetabulum. Alumina wear particles appear as granule-like yellowish-brown particulates (<1 µm to 5 µm) within macrophages (arrows) and in intercellular tissue (arrowheads) (hematoxylin and eosin; original magnification, ×400).
head was left in place because there was no evidence of damage on gross inspection. No evidence of acute infection or inflammation was observed during the operation.

Abundant ceramic wear particles and a diffuse macrophagic and lymphoplasmacytic infiltrate were revealed on histologic analysis of the osteolytic tissues after staining with hematoxylin and eosin. Ceramic wear particles were visualized as granule-like yellowish-brown particulates (≤5 µm) and were noted within the cytoplasm of macrophages and in intercellular tissue (Fig. 3). Immunohistochemical staining with monoclonal antibodies against cluster-designation (CD) 3 (Dako, Carpinteria, California), 4 (NeoMarkers, Fremont, California), 8 (NeoMarkers), 20 (Dako), and 68 (Dako) suggested that the majority of these cells were macrophages (i.e., they showed a diffuse positive staining pattern to CD 68). Focal infiltrations of T and B lymphocytes (i.e., a focal positive staining pattern to CD 20, 3, 4, and 8) were also observed.

Elementary mapping analysis of the osteolytic tissue, conducted with an electron probe microanalyzer (EPMA model JXA-8900R; JEOL, Tokyo, Japan) with an accelerating voltage of 15 kV, a probe current of 100 nA, and a probe diameter of 0.5 µm confirmed that the granular particles were composed of alumina. Furthermore, mapping for alumina particles of >0.5 µm resulted in bright green and red coloration throughout the evaluation field, indicating that high levels of alumina particles were diffusely present in tissue (see Appendix). Electron probe microanalysis demonstrated a dark blue color in the other field of the same tissue (which had been shown to be free of particles by light microscopy), indicating that a low level of alumina particles or no alumina particles were present in this control tissue (see Appendix).

Scanning electron microscopic (SEM; SM-900, Topcon, Tokyo, Japan) analysis of the retrieved alumina liner demonstrated progressive, disruptive wear of bearing and edge surfaces11 (Figs. 4-A and 4-B). An intergranular fracture with multiple craters (<1 µm to 5 µm in size) had caused near total loss of bearing surface integrity; wear was most prominent on the anteroinferior aspect of the bearing surface (Fig. 4-A). An intergranular fracture and grain pullout with micrometer-sized or submicrometer-sized crater formation was also observed on the edge surface, mainly in the anterosuperior aspect (Fig. 4-B). There was no evidence of wear or damage on the back-side surface of the alumina liner. Energy-dispersive x-ray spectrophotometric analysis (EDS, Voyager-3055; Thermo Fisher Scientific, Waltham, Massachusetts) confirmed that the black discoloration on the bearing surface of the alumina liner was a titanium metal stain (see Appendix).

**Discussion**

Short and midterm results of total hip arthroplasty with alumina-on-alumina bearings have been encouraging8,9,12,13. Nevertheless, problems related to the wear and osteolysis of ceramic bearings in vivo remain major concerns, and many authors have shown that wear debris can accumulate near alumina bearing surfaces7,14,15. However, many of these studies have been performed on the periprosthetic interfacial membranous tissues of loosened implants rather than on osteolytic tissues7,14,15. Furthermore, we are aware of only one series in which osteolysis was reported as a prominent problem, and the osteolysis in that series involved a loosened Autophor hip7 (Osteo AG, Selzach, Switzerland), which was a prosthesis that was known to have a suboptimal design and quality as compared with current standards. Foreign-body responses described in association with alumina-on-alumina bearings are

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**Figs. 4-A and 4-B** Scanning electron microscopic findings of the bearing surface of the retrieved alumina liner. **Fig. 4-A** Disruptive wear, characterized by intergranular fracture with total loss of bearing-surface integrity, is observed on the bearing surface (original magnification, ×2000). **Fig. 4-B** Intergranular fracture and grain pullout with crater formation are also observed on the edge surface (original magnification, ×2000).
related to a large amount of alumina wear debris generated by abnormal contact, such as edge-loading of the alumina head or impingement of the neck on the liner, after a long period of implant loosening. To the best of our knowledge, the present case report is the first to describe substantial osteolysis caused by ceramic wear particles generated from a well-functioning contemporary alumina-on-alumina total hip prosthesis.

It is believed that the wear rates of well-functioning alumina-on-alumina bearings are extremely low and that such wear rates are insufficient to cause osteolysis. Nevertheless, if their size, shape, and volume are optimal, alumina particles can induce biologic responses similar to those induced by polyethylene. In the case of our patient, extensive alumina grain pullout and micrometer-sized or submicrometer-sized crater formation, indicating wear and microscopic damage of the surfaces, were observed on both the bearing and edge surfaces of a retrieved alumina liner (Figs. 4-A and 4-B). We postulate that this wear and microscopic damage of the alumina liner generated sufficient phagocytosable alumina particles to elicit a macrophage response. We found abundant alumina wear particles within the macrophages that were involved in inflammation and osteolysis. In addition, most of the alumina particles were <5 µm in diameter, which is a particle size suitable for phagocytosis by macrophages.

Black discoloration, such as that observed on the alumina liner and head in the present case, is not an uncommon finding. At revision surgery, a dark metallic-appearing stain may be observed on ceramic bearings, and it has been previously reported that this is the result of metal transfer from the edge of the acetabular shell. This transfer occurs even after relatively minor contact, and its severity is related to increased bearing surface roughness. In the past, we had observed that when the contact force increases, scratching of the alumina surface can occur in vitro, although alumina is harder than titanium (unpublished data). Moreover, transferred metallic debris probably increases bearing surface wear via an abrasive or a third-body wear mechanism.

In the case of our patient, metal transfer might have occurred intraoperatively when the head was scratched by the metal shell during reduction. These findings suggest that caution is required to avoid contact of the alumina head with metallic materials and that the surgeon should reduce the artificial joint under direct visualization during total hip arthroplasty when using an alumina bearing coupling.

In the case of our patient, we did not replace the alumina femoral head. Although metal transfer to the alumina head was suspected, it was moderate in extent and no evidence of wear or damage was observed on gross inspection. However, in retrospect, after reviewing the histologic evaluation of the osteolytic tissues and the microscopic findings of the retrieved alumina liner, we regret not replacing the alumina femoral head. We therefore recommend that all bearing surfaces should be exchanged with new implants when treating osteolysis after total hip arthroplasty with an alumina-on-alumina bearing.

On the basis of this case, we recommend close follow-up of patients who have had total hip arthroplasty with contemporary alumina-on-alumina bearings, with special regard given to the possibility of osteolysis.

Appendix

Figures showing the results of electron probe microanalysis of the osteolytic tissues and energy-dispersive x-ray spectrometry analysis of the liners are available with the electronic version of this article, on our web site at jbjs.org (go to the article citation and click on “Supplementary Material”) and on our quarterly CD-ROM (call our subscription department, at 781-449-9780, to order the CD-ROM).

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