

# DIAGNOSTIC FEATURES OF PELVIC OSTEOLYSIS ON COMPUTED TOMOGRAPHY: THE IMPORTANCE OF COMMUNICATION PATHWAYS

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**Background:** Progressive periacetabular osteolysis following total hip arthroplasty may require revision surgery. The purpose of this study was to use computed tomography scans of hemipelvises retrieved at autopsy from patients who had had a total hip arthroplasty, to define the radiographic characteristics that differentiate clinically important osteolytic lesions from osteoarthritic bone cysts.

**Methods:** We analyzed forty-four hemipelvises that had been retrieved at autopsy at a mean of eight years after a total hip arthroplasty with an uncemented acetabular component. Computed tomography images were analyzed to identify the location, volume, and presence of cortical erosion and/or communication pathways with the joint space for all periacetabular bone defects. Lesions that were not present on preoperative or immediate postoperative plain radiographs were defined as new lesions. These new lesions were compared with those that were present on preoperative or immediate postoperative plain radiographs, which were defined as preexisting lesions.

**Results:** Forty-six lesions were identified on computed tomography, and sixteen of them were preexisting lesions. The mean volume of the preexisting lesions was  $1.5 \pm 1.5 \text{ cm}^3$ , which was significantly smaller than the mean volume of  $5.6 \pm 11.4 \text{ cm}^3$  of the thirty new lesions ( $p = 0.034$ ). Twenty-eight of the thirty new lesions had a clear communication pathway with the joint space, while thirteen of the sixteen preexisting lesions demonstrated no communication pathway. New lesions were significantly more likely to communicate with the joint space than were preexisting lesions ( $p < 0.001$ ). Cortical erosion was seen in sixteen of the thirty new lesions; none of the sixteen preexisting lesions exhibited cortical erosion ( $p < 0.001$ ).

**Conclusions:** The most important difference between osteolytic lesions and preexisting bone defects was the presence of a communication pathway to the joint space. Lesions that did not have an identifiable communication to the joint space were smaller and were not associated with cortical erosion. Lesions with communication to the joint through multiple pathways or through a central dome hole were larger and more likely to be associated with cortical erosion.

**Clinical Relevance:** Periacetabular lesions that are not present on perioperative plain radiographs and that have a communication pathway with the joint space and associated cortical erosions as seen on computed tomography are likely to be osteolytic lesions.

Uncemented cup fixation is commonly used in primary total hip arthroplasty and has shown excellent mid-term and long-term results<sup>1-5</sup>, but pelvic osteolysis is one of the major obstacles threatening the long-term success of these uncemented acetabular cups<sup>6-9</sup>. Pelvic osteolysis has

been observed with use of computed tomography in association with as many as 52% of total hip replacements<sup>10,11</sup>. We have observed, in our clinical practice, that it is difficult to determine on computed tomography whether a lesion represents total hip arthroplasty-induced osteolysis or the remnants of a preoperative osteoarthritic cyst, especially if the preoperative and immediate postoperative radiographs are not available for comparison with the computed tomography scan.



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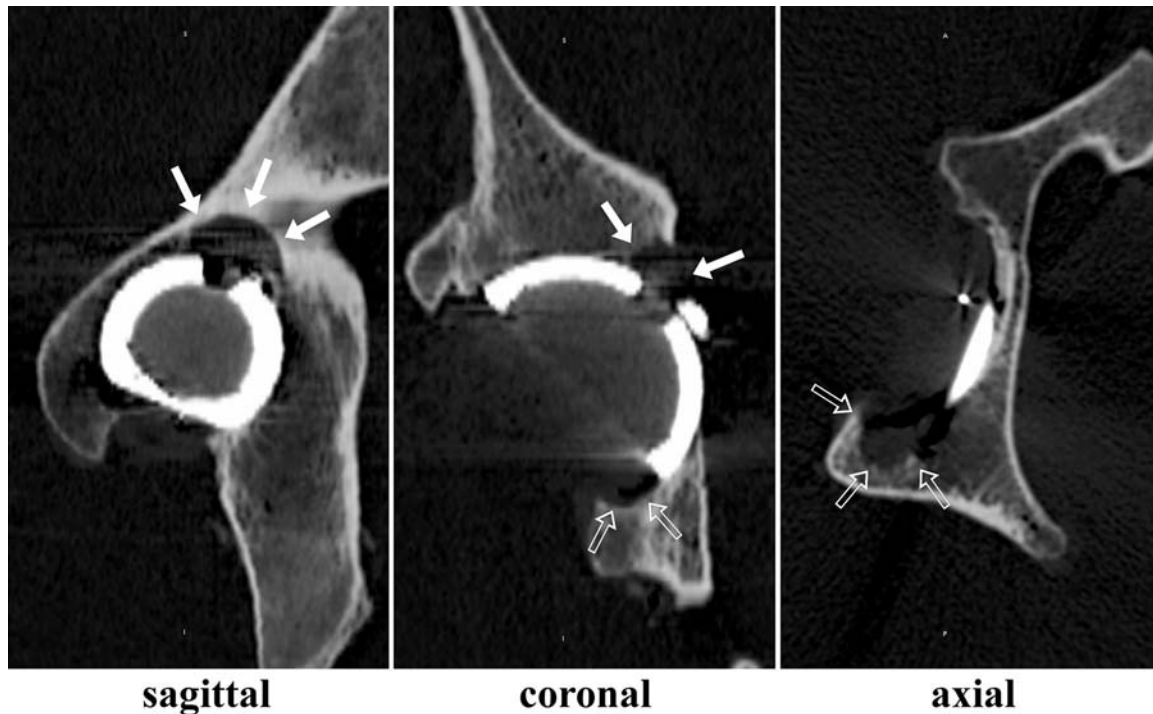


Fig. 1

Sagittal, coronal, and axial computed tomography slices demonstrating pelvic osteolysis in a right hemipelvis retrieved post mortem. The solid white arrows on the sagittal and coronal images show a lesion communicating through the central dome hole with perforation of the medial wall. The outlined white arrows on the coronal and axial images show a lesion with an erosive pattern communicating around the rim of the acetabulum. In this hip, the hole eliminator has separated from the central dome hole.

The purpose of this study was to use computed tomography scans to define the characteristics that could be used to differentiate between clinically relevant osteolysis and other bone defects, such as osteoarthritic bone cysts, that existed prior to a total hip arthroplasty. We hypothesized that new lesions would have larger volumes, would more frequently communicate with the joint space, and would be more likely to be associated with cortical bone erosion.

### Materials and Methods

We examined forty-four hemipelves containing un cemented titanium acetabular components with a modular polyethylene liner. All of the acetabular components had been implanted at our institution with the same press-fit method<sup>12</sup>. All of the hemipelves were retrieved from individuals who had provided appropriate consent for the use of their autopsy material in research studies. The index arthroplasties had been performed because of primary osteoarthritis in thirty-nine hips, rheumatoid arthritis in three hips, osteonecrosis in one hip, and posttraumatic arthrosis in one hip. None of the hips had a prior infection or had undergone structural bone-grafting.

The implanted acetabular components were of six different designs: eighteen were Duraloc 100 cups (DePuy, Warsaw, Indiana), five were Duraloc 1200 cups (DePuy), eleven

were Triloc cups (DePuy), eight were Arthropor cups (Joint Medical Products, Stamford, Connecticut), one was a PSL cup (Osteonics, Allendale, New Jersey), and one was a Harris-Galante II cup (Zimmer, Warsaw, Indiana). Screws were used with the Arthropor cup design to secure the polyethylene liner, but they were not used with any of the other designs. The implanted femoral components were all cast chromium-cobalt anatomic medullary locking stems (AML; DePuy)<sup>13</sup>. The femoral head was chromium-cobalt in thirty-five retrieved specimens and ceramic in nine. Twenty-four femoral heads were 32 mm in diameter, and twenty femoral heads were 28 mm in diameter.

The forty-four hemipelves were from twenty-two men and twenty-two women with a mean age at the time of surgery of 69.9 years (range, forty-two to eighty-seven years). The mean age at the time of death was 77.9 years (range, forty-seven to ninety-five years). The acetabular components were in situ for a mean of 8.1 years (range, 1.7 to 15.9 years). All acetabular and femoral components had stable fixation and had been functioning well at the time of the patient's death.

### Plain Radiographs

One author (C.A.E.), who was blinded to the results of the computed tomography scans, examined the preoperative and immediate postoperative radiographs for preexisting bone

defects. The anteroposterior pelvic and iliac oblique radiographs for each hip were placed in random order, and the reviewer assessed each of the radiographs independently of one another. The sclerotic border of all cystic bone defects was outlined for later comparison with the three-dimensional computed tomography reconstruction derived from the postmortem computed tomography images.

All clinical follow-up radiographs were also reviewed. Although we had excellent-quality preoperative and immediate postoperative radiographs of the hips in this study, we did not have an adequate number of annual radiographs to perform a serial analysis of lesion growth. Only twenty-eight hips had one or more radiographs that had been made more than three years postoperatively, and only three hips with new lesions had clinical radiographs with visible lesions at more than two time-points. Additionally, the time-intervals between the last clinical radiograph and the postmortem analysis (specimen radiographs or computed tomography images) were too variable to allow a study of the rate of lesion growth. The time-interval between the last clinical radiographs and the deaths of the patients ranged from 0.3 to

9.4 years (mean [and standard deviation],  $3.0 \pm 2.4$  years).

#### *Computed Tomography*

The use of retrieved specimens enabled us to optimize the image quality of the computed tomography scans since we were able to image each hemipelvis separately after we removed the femoral head and stem, which often generate most of the image artifacts associated with clinical computed tomography scans.

Each retrieved specimen was scanned in 1-mm axial slices in standard mode (GE HiSpeed Advantage; General Electric, Waukesha, Wisconsin; and Somatom Sensation 4; Siemens, Munich, Germany) at 120 kV and 220 mA. Coronal and sagittal images were reconstructed from the axial images (Fig. 1). The original data were automatically segmented on the basis of statistical properties, and an experienced orthopaedic surgeon reviewed the raw and segmented data to identify all periacetabular bone defects. For this study, we defined osteoarthritic cysts and other bone defects that existed prior to total hip arthroplasty as "preexisting lesions." Expansile regions devoid of bone that were not visible on preoperative and/or

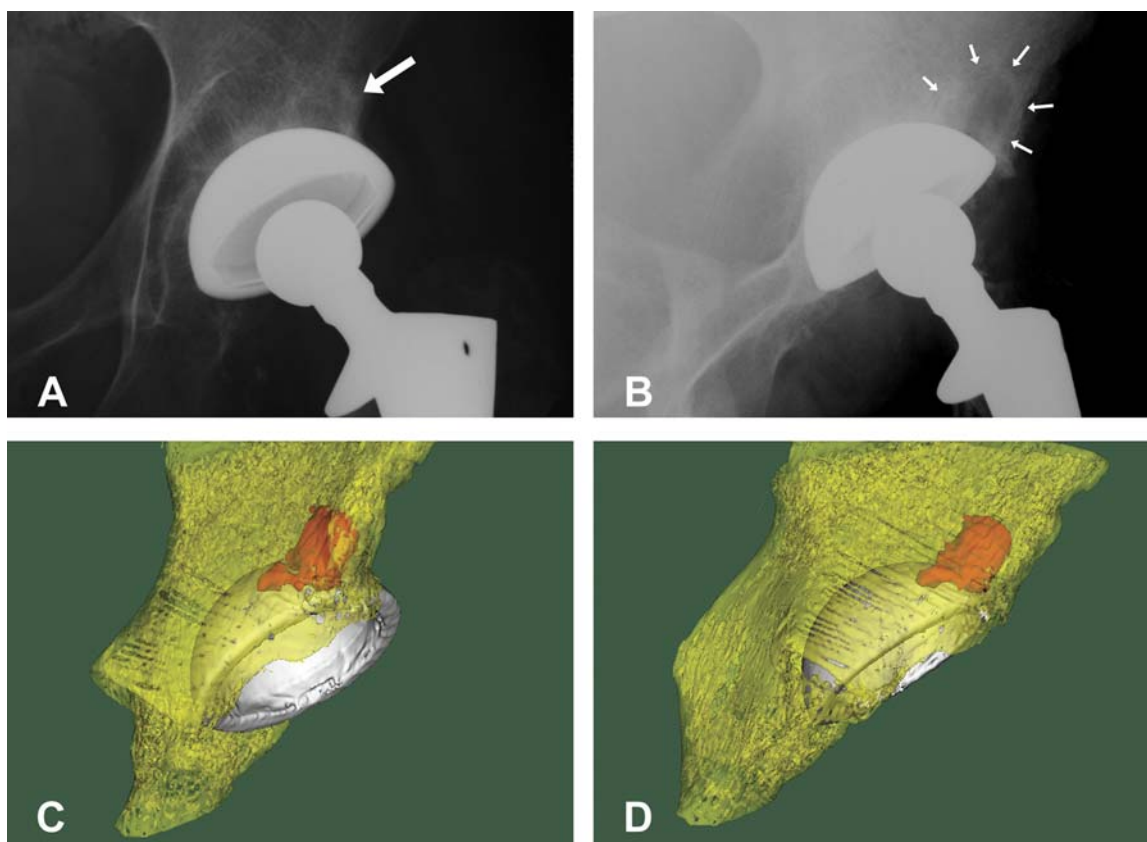


Fig. 2

Anteroposterior pelvic and iliac oblique plain radiographs of a left hip made immediately postoperatively and the three-dimensional computed tomography model of the same hip. A cystic bone defect (solid arrows) was suspected on the anteroposterior radiograph (A) and was clearly seen on the iliac oblique radiograph (B). The three-dimensional computed tomography model was oriented in the same planes. The anteroposterior view (C) and the iliac oblique view (D) of the three-dimensional model clearly show the lesion.

immediate postoperative radiographs were subsequently referred to as “new lesions.”

A three-dimensional computed tomography model was made of each specimen that had one or more bone defects. This model included the acetabular component, the bone defects, and the periacetabular bone, which was rendered translucent, allowing us to identify the defect location through the bone. The three-dimensional model was oriented to match the contour and size of the bone as seen in the radiographs (Fig. 2). We then defined a bone defect as a “pre-existing lesion” if it had the same appearance and location on both the postmortem computed tomography and the preoperative or immediate postoperative plain radiographs. The three-dimensional volume of each lesion was calculated with use of a computer-aided imaging program (VirtualScopics, Rochester, New York) (Fig. 3). The three-dimensional location of each lesion was determined by its presence in one or more of the five zones that comprise the cup surface: anterior, posterior, superior, inferior, and central (Fig. 4). The diameter of the central zone (Zone 5) was defined as one-half of the cup diameter on a two-dimensional projection of the cup surface. The peripheral zones (Zones 1 through 4) were equally divided by four lines drawn  $\pm 45^\circ$  from the cup center with respect to the superior-inferior central axis through the most proximal and distal points of the intersection of the coronal plane and the edge of the cup. A lesion was associated with a particular zone if it occupied  $>40\%$  of the cup surface within that zone or if half of the total area of involvement of the lesion on the cup surface fell within that

zone. If a lesion spanned more than three zones and did not occupy  $>40\%$  of the cup surface within any zone or if half of the total area of involvement of the lesion on the cup surface did not fall within any zone, it was assigned to a zone with the most cup surface area involvement.

The computed tomography images were examined to determine the presence and pattern of communication between the bone defect and the joint space. We also evaluated whether the lesions exhibited cortical thinning or perforation and measured the maximum width of the cortical perforation. A cortical perforation was defined as a defect wider than 2 mm. All other instances of cortical erosion were classified as cortical thinning.

#### Data Analysis

A Mann-Whitney U test for two independent samples was used to compare the volume of preexisting and new lesions. Chi-square analysis was used to determine whether the prevalence of preexisting and new lesions differed among locations. Chi-square analysis was also used to determine whether the prevalence of preexisting and new lesions differed on the basis of communication pathways to the joint space and the presence of cortical erosion. A Kruskal-Wallis nonparametric test for multiple independent samples was used to assess whether the lesion volume differed among cases depending on the communication pathway. Statistical analysis was performed with use of SPSS statistical software (version 8.0; SPSS, Chicago, Illinois). Probability values of  $<0.05$  were considered to be indicative of significance.

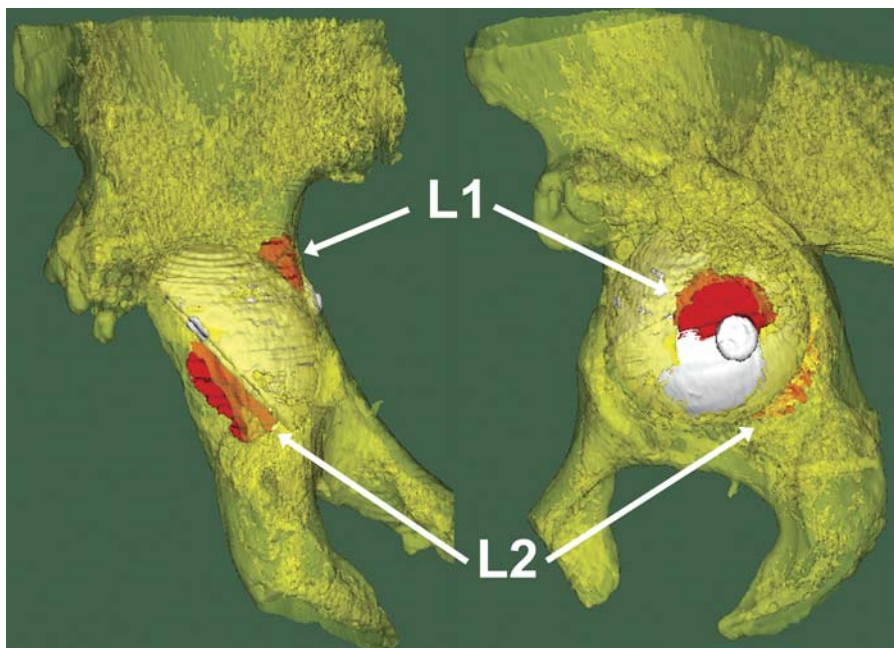


Fig. 3

Anteroposterior (left) and intrapelvic (right) views of the three-dimensional reconstruction of the acetabular component and pelvic bone defects. The size of the lesion communicating through the central hole (L1) was  $1.6 \text{ cm}^3$ ; the lesion communicating with the rim (L2) was  $1.4 \text{ cm}^3$ .

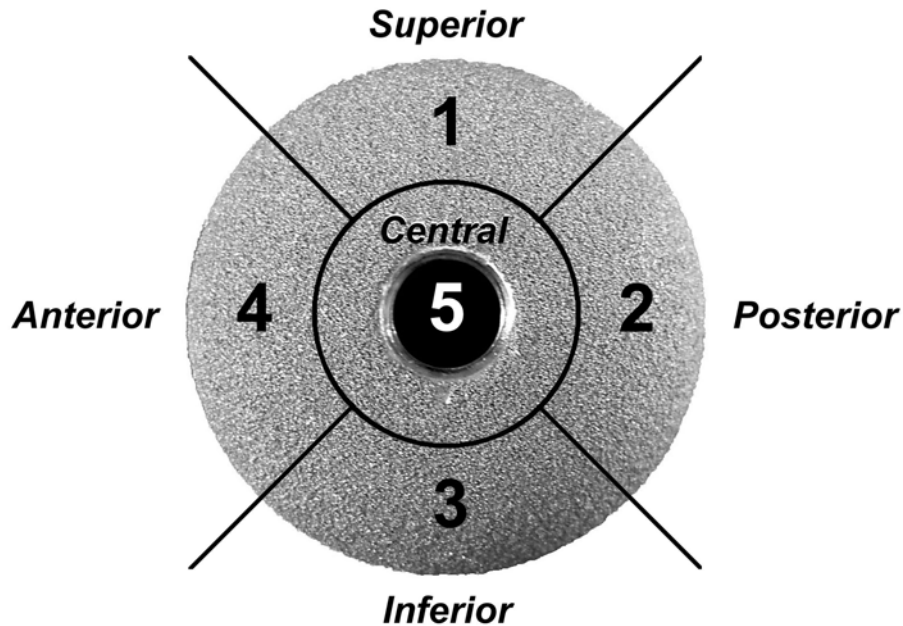


Fig. 4

The three-dimensional location of each lesion was defined by dividing the acetabular component into five zones: anterior, posterior, inferior, superior, and central. A lesion was assigned to a particular zone if it occupied >40% of the cup surface within that zone or if half of the total area of involvement of the lesion on the cup surface fell within that zone.

## Results

### Periacetabular Bone Defects Identified on Computed Tomography

A total of forty-six lesions were identified in twenty-eight of the forty-four hemipelves (Fig. 5). The mean volume (and standard deviation) of the bone defects was  $4.2 \pm 9.4 \text{ cm}^3$  (range, 0.3 to  $54.6 \text{ cm}^3$ ). The lesion volume was  $<1 \text{ cm}^3$  in twenty lesions (44%) and was  $>5 \text{ cm}^3$  in five lesions (11%).

By matching the three-dimensional computed tomography models with the perioperative clinical plain radiographs, we identified sixteen lesions that had existed prior to total hip arthroplasty (preexisting lesions). The mean volume of the preexisting lesions was  $1.5 \pm 1.5 \text{ cm}^3$  (range, 0.3 to  $4.9 \text{ cm}^3$ ). The mean volume of all other defects (new lesions) was  $5.6 \pm 11.4 \text{ cm}^3$  (range, 0.3 to  $54.6 \text{ cm}^3$ ). The difference in size between the preexisting lesions and the new lesions was significant ( $p = 0.034$ ).

### Lesion Location

Thirteen of the sixteen preexisting lesions appeared in the superior zone, while only eight (27%) of the thirty new lesions were present in the superior zone (Fig. 6). Preexisting lesions were more likely to be identified in the superior zone ( $p < 0.001$ ).

### Communication Pathways Between the Lesions and the Joint Space

We were able to observe a clear communication pathway be-

tween the lesions and the joint space in thirty-one (67%) of the forty-six lesions. Twenty-eight of the thirty-one lesions were new lesions. We identified four different communication pathways between the lesions and the joint space: around the rim (nine lesions), through a central hole (six lesions), in association with a screw or screw-hole (eight lesions), and through multiple pathways (eight lesions) (Fig. 7). Of the six lesions that definitely communicated through the central hole of the acetabular shell, three lesions also had potential communication through a gap between the bone and implant surface (Fig. 7). We could not unambiguously identify a communication pathway for fifteen lesions. The mean volume of lesions that had a definite communication pathway was  $5.6 \pm 11.2 \text{ cm}^3$  (range, 0.3 to  $54.6 \text{ cm}^3$ ), which was significantly larger than the mean volume of  $1.3 \pm 1.4 \text{ cm}^3$  (range, 0.3 to  $4.9 \text{ cm}^3$ ) for the lesions that did not have a clearly identifiable communication pathway ( $p = 0.012$ ).

Thirteen of the sixteen preexisting lesions did not have a communication pathway to the joint space. Among the three preexisting lesions that did communicate with the joint space, two of the lesions had openings located at the rim of the acetabulum and peripheral to the rim of the acetabular component. The other lesion was located at the site of a screw-hole in the acetabular cup. Twenty-eight (93%) of the thirty new lesions had at least one apparent communication with the joint space. The two new lesions (7%) without communication were 0.4 and  $1.2 \text{ cm}^3$  in size. Compared with preexisting lesions, the new lesions were significantly more likely to communicate with the joint space ( $p < 0.001$ ).

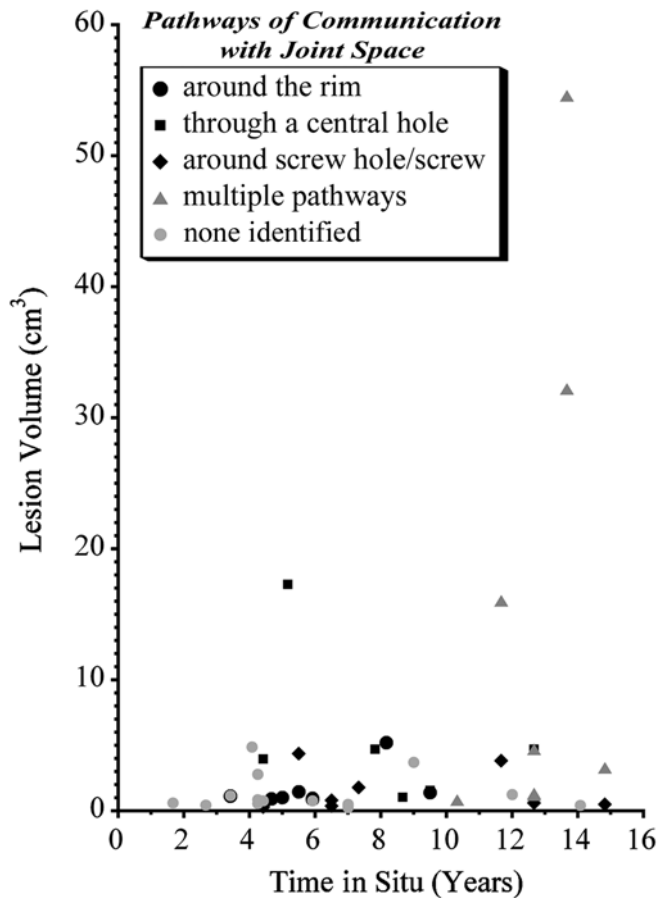


Fig. 5  
Lesion volume versus time in situ for forty-six lesions.

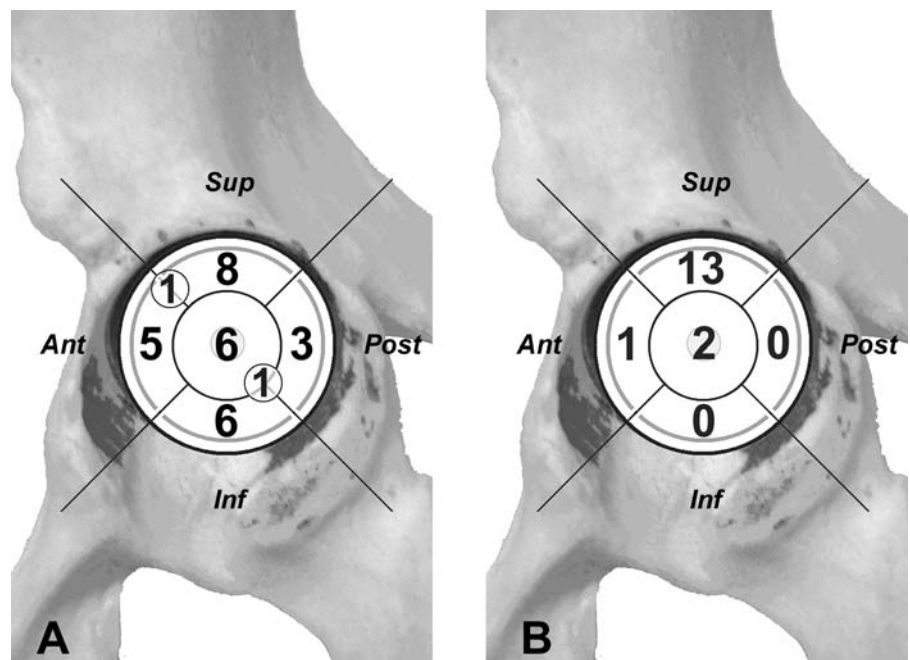
#### *Lesion Volume Compared with the Type of Communication Pathways*

For the twenty-eight new lesions that had communication with the joint space, a significant difference was detected in lesion volume depending on the type of communication pathway ( $p = 0.019$ ). The eight lesions that communicated through multiple pathways (mean lesion volume,  $14.3 \pm 19.6 \text{ cm}^3$ ) and the six lesions that communicated only with the central dome hole (mean lesion volume,  $5.6 \pm 6.0 \text{ cm}^3$ ) tended to be larger than the seven lesions that communicated only through screw-holes (mean lesion volume,  $1.2 \pm 1.3 \text{ cm}^3$ ) and the seven lesions that communicated only around the rim (mean lesion volume,  $1.7 \pm 1.6 \text{ cm}^3$ ).

#### *Cortical Erosion Around the Lesions*

None of the preexisting lesions demonstrated cortical perforation or cortical thinning. Sixteen of the thirty new lesions were associated with cortical perforation. Twelve of the sixteen new lesions perforated the acetabular rim, while four others perforated the medial wall. Three other lesions had cortical thinning. All of the new lesions communicating through multiple pathways were associated with cortical perforation. Four of the six lesions communicating through the central dome hole were associated with cortical perforation or thinning of the medial wall. Two of the seven lesions communicating around the screw-hole had cortical thinning. Five of the seven lesions communicating around the rim had cortical perforation adjacent to the cup. The prevalence of cortical erosion was higher for new lesions than for preexisting lesions ( $p < 0.001$ ).

Fig. 6  
A: The locations of the new lesions stratified by cup zone. Two of the thirty lesions spanned multiple zones. B: The locations of the preexisting lesions. Such lesions were more likely to be located in the superior zone ( $p < 0.001$ ).



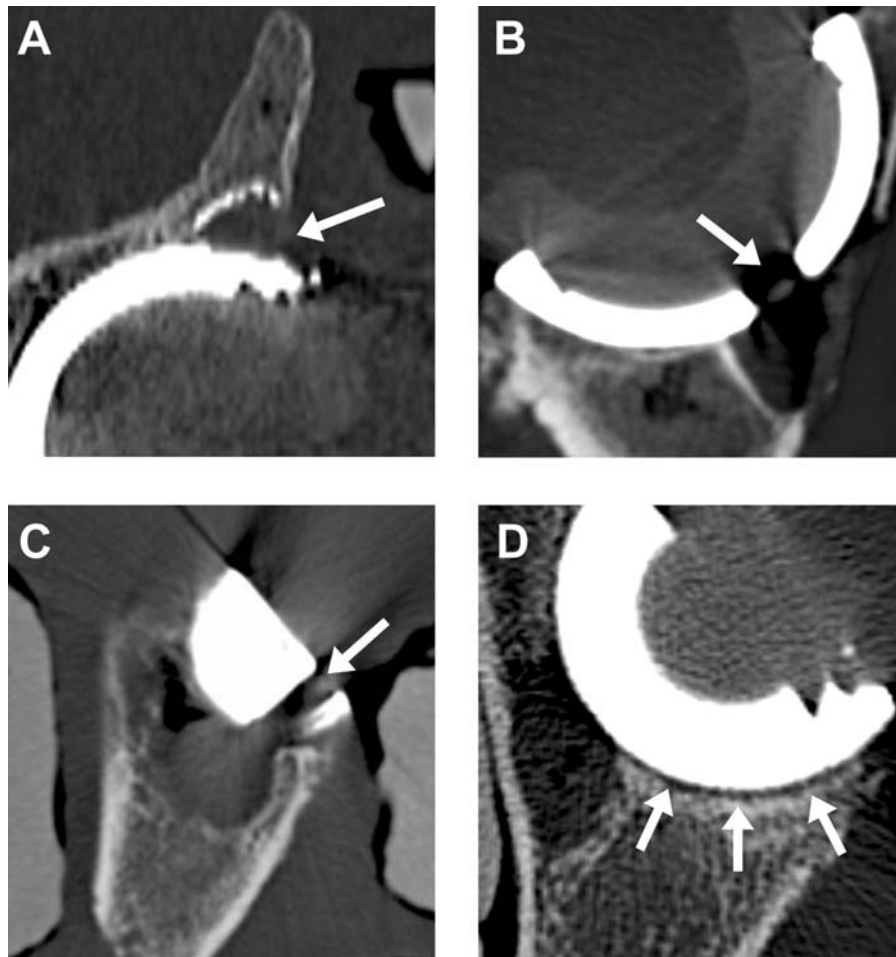


Fig. 7

Examples of four different communication pathways between the osteolytic lesions and the joint space seen on computed tomography: around the rim (A), through the central hole (B), around a screw-hole (C), and through a gap (D).

## Discussion

For more than ten years, we have retrieved postmortem specimens containing primary uncemented titanium acetabular shells and modular polyethylene liners. We recently began to analyze these specimens to better understand the patterns of osteolysis associated with uncemented implants. Removal of the femoral head and stem allowed us to eliminate the primary sources of beam scatter and computed tomography image artifact. This, in turn, allowed us to examine more carefully the characteristics of different bone defects and to determine how osteolytic defects differ from preexisting osteoarthritic cysts. From this analysis, we found that the most important difference between new lesions and preexisting ones was the presence of a communication pathway to the joint space. We identified four communication pathways, including the rim, central dome hole, peripheral screw-holes, and implant-bone gaps. Our results led us to conclude that, if a communication pathway cannot be found when cystic bone lesions are examined, there is a high probability that the bone

lesion is not an osteolytic one resulting from the implantation of the total hip replacement. Rather, lesions with no communication pathways likely existed prior to the total hip arthroplasty. In this study, thirteen of the fifteen lesions without communication pathways on computed tomography were visible on the preoperative or immediate postoperative radiographs. Moreover, our results suggest that detecting the type of communication pathway may also be an important factor in predicting future lesion size. We found that lesions with multiple communication pathways or communication through a central dome hole were larger than those with communication through a screw-hole or around the rim.

It has been postulated that a communication pathway to the joint space is important to the pathogenesis of both osteoarthritic cysts and osteolytic lesions<sup>14-16</sup>. In this study, all lesions that were  $>5 \text{ cm}^3$  had a communication pathway to the joint space. The average volume of preexisting lesions was  $1.5 \text{ cm}^3$ , and the largest preexisting lesion was  $4.9 \text{ cm}^3$ . In view of the lesion volumes illustrated in Figure 5 and the absence of

reports in the literature describing complications associated with osteoarthritic cysts, we regard bone defects with a volume of  $\geq 5 \text{ cm}^3$  and communication with the joint space as probably osteolytic and, therefore, clinically important. We believe that when small lesions are observed on computed tomography, one must be particularly careful to determine whether they are truly osteolytic lesions or preexisting bone defects. Puri et al. reported a 52% prevalence of osteolysis in fifty hips at a mean of 7.6 years with use of computed tomography<sup>10</sup>. However, only 14% of the cups in their study had osteolytic lesions of  $>5 \text{ cm}^3$ . On the basis of the findings in the current study, if the lesions are  $<5 \text{ cm}^3$ , do not erode cortical bone, and do not have a communication pathway to the joint, there is a high probability that they do not represent osteolysis and thus are not of clinical concern.

We acknowledge several constraints and limitations of the present study. First, we were restricted to a relatively small number of specimens because we analyzed donated postmortem retrieved specimens from a select, rather elderly population. While we are certain that the bone defects identified on plain radiographs made prior to the total hip arthroplasty do not represent osteolysis, we cannot be certain that all defects appearing in the postmortem computed tomography images represent total hip replacement-induced osteolytic lesions. It remains possible that some of the new lesions identified in this study represent preexisting lesions that we were unable to identify on plain radiographs. We suspect that this is the case for the two "new" lesions without communication pathways. Theoretically, an immediate postoperative computed tomography image would definitively identify any preexisting bone defects, but such was not available for these cases.

Second, we recognize that there are many factors that may contribute to the development of periacetabular osteolysis, such as the type of polyethylene, wear rates, sterilization techniques, cup orientation, time in situ, and cup design. However, we did not analyze this information for two reasons: (1) the limited number of hips with osteolytic lesions in this study precluded a meaningful statistical analysis with regard to these variables, and (2) the purpose of this study was not to correlate wear with osteolysis or to clarify the mechanism of the osteolysis but, rather, to demonstrate that not all localized areas of periacetabular bone loss represent total hip replacement-induced osteolysis. The correlation of osteolysis with these and other variables remains an important area for future study.

Third, we caution that this postmortem study does not demonstrate that routine clinical computed tomography scans can accurately differentiate between osteolytic lesions and preexisting bone defects. Metal artifact from the femoral components in a clinical scan could hinder the ability to visualize osteolytic lesions. However, we believe that the advent of new technologies and the use of optimum settings will likely mitigate future problems associated with metal artifact.

Finally, we acknowledge that, while the determination of the pattern and rate of the radiographic growth of periacetabular bone defects is very important and of interest to clinicians, the lack of annual follow-up radiographs precluded a serial analysis of the rate of lesion growth in these hips.

Clinicians should be aware that computed tomography may lead to an overdiagnosis of osteolysis, particularly in the absence of perioperative imaging studies that can document the existence of preexisting bone defects. Osteolysis is currently defined as a periprosthetic region devoid of bone with a well-defined sclerotic border. In view of our findings, we propose that the definition of osteolysis on computed tomography be expanded to include the presence of a communication pathway to the joint space. ■

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In support of their research or preparation of this manuscript, one or more of the authors received funding (a general institutional grant) from Inova Health System, Alexandria, Virginia. None of the authors received payments or other benefits or a commitment or agreement to provide such benefits from a commercial entity. A commercial entity (Inova Health System) paid or directed, or agreed to pay or direct, benefits to a research fund, foundation, educational institution, or other charitable or nonprofit organization with which the authors are affiliated or associated.

doi:10.2106/JBJS.D.02882

## References

1. Archibeck MJ, Berger RA, Jacobs JJ, Quigley LR, Gitelis S, Rosenberg AG, Galante JO. Second-generation cementless total hip arthroplasty. Eight to eleven-year results. *J Bone Joint Surg Am.* 2001;83:1666-73.
2. Clohisy JC, Harris WH. The Harris-Galante porous-coated acetabular component with screw fixation. An average ten-year follow-up study. *J Bone Joint Surg Am.* 1999;81:66-73.
3. Della Valle CJ, Berger RA, Shott S, Rosenberg AG, Jacobs JJ, Quigley L, Galante JO. Primary total hip arthroplasty with a porous-coated acetabular com-

ponent. A concise follow-up of a previous report. *J Bone Joint Surg Am.* 2004; 86:1217-22.

4. Kim YH, Kim JS, Cho SH. Primary total hip arthroplasty with a cementless porous-coated anatomic total hip prosthesis: 10- to 12-year results of prospective and consecutive series. *J Arthroplasty.* 1999;14:538-48.

5. Udomkiat P, Dorr LD, Wan Z. Cementless hemispheric porous-coated sockets implanted with press-fit technique without screws: average ten-year follow-up. *J Bone Joint Surg Am.* 2002;84:1195-200.

- 6.** Harris WH. Wear and periprosthetic osteolysis: the problem. *Clin Orthop Relat Res.* 2001;393:66-70.
- 7.** Maloney WJ, Herzurm P, Paprosky W, Rubash HE, Engh CA. Treatment of pelvic osteolysis associated with a stable acetabular component inserted without cement as part of a total hip replacement. *J Bone Joint Surg Am.* 1997;79:1628-34.
- 8.** Maloney WJ, Galante JO, Anderson M, Goldberg V, Harris WH, Jacobs J, Kraay M, Lachiewicz P, Rubash HE, Schutzer S, Woolson ST. Fixation, polyethylene wear, and pelvic osteolysis in primary total hip replacement. *Clin Orthop Relat Res.* 1999;369:157-64.
- 9.** Manley MT, D'Antonio JA, Capello WN, Edidin AA. Osteolysis: a disease of access to fixation interfaces. *Clin Orthop Relat Res.* 2002;405:129-37.
- 10.** Puri L, Wixson RL, Stern SH, Kohli J, Hendrix RW, Stulberg SD. Use of helical computed tomography for the assessment of acetabular osteolysis after total hip arthroplasty. *J Bone Joint Surg Am.* 2002;84:609-14.
- 11.** Stulberg SD, Wixson RL, Adams AD, Hendrix RW, Bernfield JB. Monitoring pelvic osteolysis following total hip replacement surgery: an algorithm for surveillance. *J Bone Joint Surg Am.* 2002;84 Suppl 2:116-22.
- 12.** Engh CA, Hopper RH Jr, Engh CA Jr. Long-term porous-coated cup survivorship using spikes, screws, and press-fitting for initial fixation. *J Arthroplasty.* 2004;19(7 Suppl 2):54-60.
- 13.** Engh CA Jr, Culpepper WJ 2nd, Engh CA. Long-term results of use of the anatomic medullary locking prosthesis in total hip arthroplasty. *J Bone Joint Surg Am.* 1997;79:177-84.
- 14.** Freund E. The pathological significance of intra-articular pressure. *Edinburgh Med J.* 1940;47:192-203.
- 15.** Landells JW. The bone cysts of osteoarthritis. *J Bone Joint Surg Br.* 1953;35:643-9.
- 16.** Schmalzried TP, Jasty M, Harris WH. Periprosthetic bone loss in total hip arthroplasty. Polyethylene wear debris and the concept of the effective joint space. *J Bone Joint Surg Am.* 1992;74:849-63.