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LETTER TO THE EDITOR

Letter to the Editor

Association of Osteonecrosis and Failure of Hip Resurfacing Arthroplasty

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To the Editor:

We read with interest, the article titled "Association of Osteonecrosis and Failure of Hip Resurfacing Arthroplasty" by Zustin et al. [12]. The authors discussed the findings in femoral head remnants retrieved from failed resurfacings with particular emphasis on the role of osteonecrosis, induced by the total hip resurfacing, in causing failure of surface arthroplasty.

The role of osteonecrosis in causing failure of resurfacing has been debated among arthroplasty surgeons. Osteonecrosis may be caused by insult to the vascular supply [1, 11] or by thermal damage [6, 8]. However, postoperative radionuclide imaging studies have revealed no evidence of reduction in vascularity [5, 10]. Implant retrieval analyses after failed resurfacings have shown evidence of osteonecrosis of varying prevalence. Bradley et al. found evidence of osteonecrosis in two of 25 failed resurfacings [3], Howie et al. saw evidence in one of 72 [7], Little et al. saw evidence in 12 of 13 [9], and Campbell et al. saw evidence in seven of 98 [4].

We have some concerns regarding the study by Zustin et al. [12]. First, the assumption that histologic evidence of osteonecrosis observed in the retrieved specimens is induced by the resurfacing procedure is questionable in cases with periprosthetic fractures of the femoral neck. The osteonecrosis may be secondary to the femoral neck fracture [7]. Zustin et al. did not specify whether this issue was addressed. Histologic evidence of advanced osteonecrosis alone is insufficient as the time between the fracture and the revision surgery is not known and might be long enough to cause histologically advanced osteonecrosis after the periprosthetic fracture. Bogoch et al. [2] observed a biologic reaction at the bone-cement interface, which included a foreign body reaction to the polymethylmethacrylate, as evidenced by histiocytes, giant cells, and crystals of polymethylmethacrylate which are birefringent in polarized light. This reaction was observed to occur if the remnant head remains viable after resurfacing and would be absent if osteonecrosis was induced by the procedure. A remnant head retrieved after a periprosthetic fracture and showing a biologic reaction at the bone-cement interface along with evidence of osteonecrosis would suggest that the femoral head remained viable after the resurfacing procedure but underwent osteonecrosis after fracture of the femoral neck. Zustin et al. did not mention whether they analyzed the interface for any such evidence.

Second, in cases with femoral loosening, areas adjacent to the interface may show evidence of superficial osteonecrosis [7]. Howie et al. advocate using sections from the central area of remnant heads to differentiate this from the generalized osteonecrosis caused by resurfacing [7]. Zustin et al. did not specify any precaution that they have taken to avoid this error.

Third, patients with posttraumatic arthritis might have had posterior dislocation or posterior fracture-dislocation as the initial traumatic event or could have undergone surgery through the posterior approach and osteonecrosis

(Re: Zustin J, Sauter G, Morlock MM, Rüther W, Amling M. Association of osteonecrosis and failure of hip resurfacing arthroplasty. *Clin Orthop Relat Res*. 2009 Jul 14. [Epub ahead of print]. DOI 10.1007/s11999-009-0979-3).
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developed before the resurfacing procedure. It would be interesting to know if such patients were included.

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