

Reply to Letter to the Editor

Association of Osteonecrosis and Failure of Hip Resurfacing Arthroplasty

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We thank Doctors Kannan and Malhotra for their letter regarding our manuscript, Association of Osteonecrosis and Failure of Hip Resurfacing Arthroplasty [12]. They have challenged the evidence we presented suggesting osteonecrosis (ON) was associated with substantial failures of hip resurfacing arthroplasty [12]. More specifically, they questioned if (1) ON could not occur secondary to a periprosthetic fracture, (2) we analyzed the central section from the femoral remnant according to Howie et al. [5] to confirm the occurrence of superficial and generalized ON, and (3) patients with posttraumatic arthritis did not have ON develop before the resurfacing procedure.

We explained thoroughly the criteria for diagnosis of ON lesions in our study. We defined advanced ON lesions microscopically as the presence of trabeculae without stainable osteocytes, disorganized bone marrow, and bordering fibrosis, and by contact radiography of the specimens because of the observation of bordering sclerosis. Qui et al. [9] showed that remnants of apoptotic

osteocytes may remain in situ for several weeks or months, recognizable by pyknotic nuclei using conventional light microscopy, but eventually the remnants become undetectable, leaving an apparently empty lacuna. This morphologic finding is considered a reliable sign that the osteocyte originally present has died [9]. Similarly, ON becomes recognizable radiographically the first several weeks or months after the fracture [6, 11]. Our definition of an advanced ON lesion also included the absence of an interface histiocyte reaction to cement as described by Bogoch et al. [1]. Therefore, we suggested ON probably was a cause of the specific type of periprosthetic fractures after a hip resurfacing arthroplasty (51 hips of a total of 85 failures associated with fracture) which occurred acutely in all cases with valid clinical data. Kannan and Malhotra correctly pointed out that previous retrieval analyses have shown varying rates of evidence of ON. Little et al. [7] and Steffen et al. [10] reported even greater occurrences of ON in hips revised for periprosthetic fractures after a resurfacing arthroplasty, however, the total number of retrieved hips was smaller. They suggested ON played a major role in periprosthetic fracture after the current generation hip resurfacing arthroplasty. In the study by Campbell et al. [3], the majority (70 of 98 hips) of revisions in their cohort were not attributable to fractures, and they detected ON in all five hips with an in situ time longer than 6 months, however, they did not discuss in detail the occurrence of ON in fractures that occurred within a short time. Based on our observations, we proposed that ON led to failure of the resurfacing arthroplasty predominantly within the first few months after implantation. Postnecrotic fractures in our study cohort were revised with a mean in situ time of 5.3 months. Although Forrest et al. [4] and McMahan et al. [8] radiographically analyzed well-functioning hips after a followup longer than 18 months and their cases were not

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analyzed morphologically, the comparison with our retrieval study does not seem to be reasonable. Furthermore, we also consider the comparison with the second-generation hip resurfacing arthroplasty not quite appropriate. There are several substantial differences in design and clinical outcome of the second as compared with the current generation hip resurfacing arthroplasty. Howie et al. [5] analyzed 72 cases mainly with intermediate- and long-term failures (none was revised within the first 6 months after implantation) and focused primarily on a possible association of ON with loosening. Bradley et al. [2] found two of 25 resurfaced hips revised for fracture had substantial regions of ON, and Bogoch et al. [1] found ON of the entire femoral head in three of four fractures. Therefore, our observations are consistent with those in the literature.

Owing to design of the femoral component in the current generation hip resurfacing arthroplasty, it is impossible to analyze sections from the central areas as did Howie et al. [5]: the current generation femoral components are stemmed and the tissue from the central part of the femoral remnant is removed at implantation surgery. Nonetheless, we analyzed the tissue present between the superficial and central areas of the femoral remnant. By examining sections from three quadrants of each femoral remnant in their full length with a standardized sampling procedure, we believe we adequately addressed the question of sampling: the extent of ON was determined from the entire remnant rather than only the superficial and control portions. All ON lesions we observed were connected with the proximal surface of the bone remnant. We are not aware of any report of an isolated ON lesion after a hip resurfacing arthroplasty in the central or distal part of the femoral remnant without including its proximal areas. In addition, to avoid any possible misdiagnosis in cases with primary avascular necrosis of the femoral head, we excluded hips with this preoperative diagnosis.

Drs Kannan and Malhotra correctly pointed out that in cases with posttraumatic arthritis, morphologic changes related to the initial traumatic event or its subsequent therapy might have led to ON before implantation. Preoperative biopsy information from hips before implantation was not available and could have been available only from the removed central region where the stem was located. As we examined only seven hips with a preoperative diagnosis of posttraumatic arthritis, this specific issue remained unanswered by our study. However, we found ON lesions in each hip from this group. Moreover, three of the six hips with posttraumatic arthritis with failed results attributable to periprosthetic fractures after the resurfacing arthroplasty were considered as having postnecrotic fractures. Because we did not have a control

group (eg, examining for ON in patients without implant failure and/or femoral heads removed for conventional total hip arthroplasty), any statements regarding possible relationships would be highly speculative. This question, however, might be adequately answered by clinical and radiographic studies.

Based on our observations, we believe it is reasonable to presume the presence and the extent of ON after a resurfacing arthroplasty may be linked to complications from the arthroplasty.

Again we thank Drs Kannan and Malhotra for their insightful comments.

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