Are there Differences in Wear Modes, Debris Type, and Tissue Response between Anatomic and Reverse Shoulder Arthroplasties?

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INTRODUCTION: Little attention has been given to potential long-term complications due to tissue reactions to implant debris, and potential loosening of the implant due to particle-induced osteolysis in total shoulder arthroplasties (TSA). Recent studies have shown that implant wear with subsequent immune cell response does occur in TSAs. Here we examine a series of retrieved anatomic (aTSAs) and reverse (rTSAs) designs to determine extent of implant damage and to characterize the nature of the corresponding periprosthetic tissue responses and how these relate to implant loosening.

METHODS: TSA components and periprosthetic tissues were retrieved from 63 patients (35 aTSA, 28 rTSA). None of the implants in this study were removed for infection. Damage to the implants was characterized using light microscopy. Head/stem taper junction damage was graded 1-4 as minimal, mild, moderate, or marked. Metal bearing surfaces was graded 1-3 (mild, moderate, marked). Damage on polyethylene (PE) was given a score based on the summation of scoring for extent of wear scar, presence of polishing, 3-body wear, rim damage, delamination, and grooves and scratches for a maximum score of 14. H&E stained sections of periprosthetic soft tissues were evaluated for the extent and type of cellular response. A semi-quantitative system was used to score (1=rare to 4=marked) the overall number of particle-laden macrophages, foreign body giant cells, lymphocytes, plasma cells, eosinophils, and neutrophils. Implant damage and histopathological patterns were compared between the two TSA groups using the Mann-Whitney and Spearman tests.

RESULTS: PE bearing surface scores were greater in the aTSA group (8.6±2.4, 4.8±1.7, $p \leq 0.000$). The PE bearing surfaces of aTSAs were dominated by 3-body wear and plastic deformation, whereas the rTSA PE components exhibited mainly polishing and scratching. Cases with severe rim damage occurred mainly due to delamination and plastic deformation in aTSA, and in rTSA due to notching (8 of 28 cases) (Figure 1). Metal surface damage occurred in a few cases in both groups. Only one aTSA case exhibited marked taper corrosion. Metal particles were seen in 77% of aTSAs and 93% of rTSAs. More bone cement and suture debris was seen in the aTSA group compared to the rTSA ($p \leq 0.004$). In both groups the primary nature of the inflammatory response was a moderate to marked macrophage response to wear particles (86% of cases). The particle-laden macrophages tended to occur in broad sheets and contained metal, PE, bone cement, and suture debris. There was no difference in the cellular response between aTSA and rTSA.

DISCUSSION AND CONCLUSION: The principal difference in the design, fixation, and biomechanical loading of the two TSA types resulted in different wear mechanisms and modes, and subsequently, different types and sizes of implant debris. Both groups exhibited a strong macrophage response to a combination of these different types of debris—PE, metal, bone cement, and suture. Particularly, the difference in PE particles size between the groups may be due to different acting wear mechanisms that are dependent on the design of the implant, and also on the occurrence of rim damage, all of which may also affect the extent of macrophage response.

Figure 1A: Severely delaminated aTSA glenoid component with extreme wear-through at rim; B: Excessive rim damage in a rTSA component due to scapular notching which occurs when the PE of the humeral component is in repetitive contact with the inferior scapular neck.