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CORR Insights®: Can Wear Explain the Histological Variation Around Metal-on-metal Total Hips?

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Where Are We Now?

The need for revision arthroplasty for septic and aseptic hardware failure remains a central problem for the component engineer and orthopaedic surgeon alike. Total hip prostheses have undergone several design iterations, beginning with first generation metal-on-metal (MoM) articulations, which were plagued by failure mechanisms including frictional locking and corrosion [1]. These early designs were largely supplanted by prostheses with other bearing surfaces (polyethylene or cera-

mic). However, polyethylene wear debris itself is a known cause of aseptic loosening, and polyethylene, depending on molecular weight and crosslink properties, wears (both linearly and volumetrically) faster than steel alloys. An interest in MoM prostheses therefore was rekindled, especially for younger patients. When compared to historical data [2], overall usage of MoM devices has likely declined, though these implants will probably represent an increasingly important share of revision cases. A subset of these implants is prone to early failure through mechanisms thought related to

local immunologic response to metal debris/metal ions (cobalt, chromium, molybdenum, nickel) – probably Type-IV reactions involving native proteins and hapten formation, though this is not universally accepted, especially given the largely nonspecific histopathologic inflammatory cellular infiltrate (lymphocytes and plasma cells) about failed MoM prostheses. These reactions have variously been referred to as “metal hypersensitivity reaction,” “pseudotumor,” and “aseptic lymphocyte-dominated vasculitis-associated lesion” (ALVAL) [8]. The latter term has gained favor as it references the single histologic feature not seen in other types of prosthetic failure, namely a perivascular lymphocytic infiltrate [8, 9]. Unfortunately, the exact factors leading to or predictive of ALVAL formation remain elusive.

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Where Do We Need To Go?

The current manuscript adds a valuable contribution to the ALVAL literature in that its negative association between linear wear depth and

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ALVAL score (a scoring system for roughly qualifying/quantifying histopathologic findings associated with failed MoM prostheses) [4] illustrates the multifactorial nature of the problem. While low metallic wear rates (on the order of two to three microns per year) make it inherently difficult to establish correlation (much less causation) between metallic wear and histologic response, there is both confirmative [6] and equivocal/dissenting literature [7]. Notably, it does appear that pseudotumor/ALVAL formation is possible in the presence of low wear, and it is thus highly probable that individual host response plays a critical role in this pathologic process in conjunction with the myriad variables associated with implant design (alloy composition, corrosion properties and debris particle size, weight bearing axes). Fundamentally, we should ask whether the ALVAL score (of which there are actually variations) is adequately nuanced to capture the underlying biological complexity of failed orthopaedic prostheses given the inherent complexity of immunologic cellular and molecular signal transduction. For example, though elevated chromium and cobalt levels have been demonstrated in some patients with loose prostheses [3], this finding does not definitively correlate with ALVAL formation. Moreover, metal sensitivity skin patch testing, though positive at

greater frequency in patients with metal implants, is not necessarily predictive of pseudotumor development.

How Do We Get There?

One is left to believe that metal debris/corrosion products are central to the failure of at least some subset of orthopaedic prostheses, likely in conjunction with highly variable individual host immunologic response mechanisms. Given the nature of these complex systems, in the true mathematical sense of the term “complex,” should we look only to detailed biologic/biochemical mechanisms of implant failure or should we pragmatically consider a broader perspective and search for patterns among the complexity? For example, through DNA microarray technology, might we be able to “fingerprint” host parameters predictive of MoM implant failure even without necessarily understanding the subtle underlying mechanistic details? Might we combine such a fingerprint with measurables such as metal skin sensitivity testing to preoperatively identify patients at risk for implant complications? Alternatively, through the development of lower-wear materials, we may ultimately avert, or at least significantly reduce, the need for ALVAL evaluation. In those patients where preoperative testing is

ineffective, imaging techniques, such as MRI with magnetic artifact reduction sequences, may also allow earlier diagnosis of pseudotumor formation [5], perhaps facilitating preemptive, minimally-invasive intervention that obviates the need for revision arthroplasty.

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