

Metallosis: The battle of the five armies

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Metals and their alloys have been widely used as implantable materials such as artificial joint prosthesis; internal fixation nails, plates and rods; dental implants and stents. Metallosis including adverse reactions to metal debris (ARMD) in hip arthroplasty is an aseptic fibrosis, local necrosis, inflammation, or loosening of an implanted device secondary to metallic corrosion and release of wear debris. The mechanism of inflammation in metallosis is controversial due to the complexity of the morphology, composition of the wear particles and cell/tissue responses involved. Metal allergy can partially explain some clinical cases but the causes of many are still unknown. We have recently analyzed 285 cases of metallosis from hip replacement. It is identified that five

key factors have played an important role in metallosis: (1) wear metal nanoparticles; (2) metal corrosion products; (3) macrophages; (4) master cells; and (5) lymphocytes. In the cases of hip arthroplasty, there are distinct differences between wear metal particles and metal corrosion products, both are recognized as foreign body invasion and induce a battle led by host macrophages, master cells and lymphocytes. Macrophages respond to both types of metal materials by phagocytosis and engulfment in all cases with metallosis. Master cells are seen in some cases and located between macrophages/metal particles and blood vessels. Lymphocytes are dissociated with metal particles but with more severe clinical manifestation following their infiltration. Consequently, it results in large area of cell apoptosis and necrosis, inflammation, fibrosis or pseudotumor formation and failure of the implants.

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