Greater Trochanteric Fragmentation After Failed Metal-on-Metal Hip Arthroplasty

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abstract

Adverse reaction to metal debris (ARMD) involving the hip joint has emerged as an important reason for failure and revision among patients with metal-on-metal (MOM) hip arthroplasty. To the authors’ knowledge, there are no reports of adverse radiographic sequelae in the greater trochanter subsequent to revision for ARMD. The authors describe clinical and radiographic findings in 2 patients who developed greater trochanteric fragmentation 1 to 2 years after conversion of their failed MOM hips to polyethylene bearings. Both patients had solid pseudotumors with tissue necrosis. Several reports describe various clinical features of ARMD. Although poor outcomes have been demonstrated after some MOM revisions, to the authors’ knowledge, no reports document greater trochanter fragmentation in ARMD. The current patients highlight the fact that tissue damage occurring with MOM bearing hips can involve bone in addition to soft tissue even after a pseudotumor has been removed and serum metal levels have decreased to normal levels after revision. Unlike the greater trochanteric fractures historically associated with polyethylene wear and osteolysis, no evidence of bone cysts or lesions was found prior to the fractures and neither fracture healed with conservative treatment. For these 2 patients, the authors believe the tissue necrosis included both soft tissue and bone. The necrotic bone resorbed gradually after removal of the MOM bearing, resulting in bone fragmentation with ongoing symptoms. These patients emphasize and remind us that damage is not only limited to soft tissues, but also includes bone. Surgeons should be aware of this radiographic finding and the associated clinical symptoms. [Orthopedics. 2015; 38(5):e447-e451.]

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A dverse local tissue reaction involving the hip joint has emerged as an important reason for failure and revision among patients who have undergone metal-on-metal (MOM) total hip arthroplasty (THA). A variety of nomenclatures describe this local tissue reaction, including adverse reaction to metal debris (ARMD), aseptic lymphocyte dominated vasculitis-associated lesion, pseudotumor, and metallosis. 1-4 Adverse reaction to metal debris is a new umbrella term defined as aseptic fibrosis, local necrosis, or loosening of a prosthesis secondary to metallic debris. 2

Failure of MOM modular THA has been well documented. Pseudotumors and masses that may be solid or fluid-filled have been reported with these failures. 3, 6 The reported incidence of ARMD ranges from 0% to 6.5% of MOM THAs. 5 The authors’ institute has previously reported a 0.3% incidence of patients with ARMD in a series of MOM THA. 5, 7 Several reports in the literature describe the clinical features of ARMD. 4, 8-11 The authors are not aware of reports of adverse radiographic sequelae in the greater trochanter subsequent to revision for ARMD.

In the current report, the authors describe the clinical and radiographic findings in 2 patients who had greater trochanter fragmentation approximately 1 year after revision of their MOM hip, which had a solid pseudotumor. To the authors’ knowledge, this complication has not been previously reported.

These cases highlight the fact that tissue damage occurring with MOM bearing hips can involve bone in addition to soft tissue. The bone damage can also have clinical implications and radiographic findings that evolve 1 to 2 years after revision with exchange of the bearing surface. The patients were informed that data concerning their case would be submitted for publication, and they consented.

Case Reports
Patient 1
A 62-year-old man who had undergone a right primary MOM THA 8 months previously due to arthritis of the hip was referred to the authors’ institute for the evaluation and treatment of pain in the groin and front of his hip. Initially, he had done well after the primary surgery. The arthroplasty was performed using an articular surface replacement cup, a 47-mm femoral head, and a titanium alloy proximally coated stem (US Food and Drug Administration–approved implants). The cup abduction angle was 40° and the antversion angle was 15°. Radiographs showed the stem and cup in good position (Figure 1A). The杯 abduction angle was 40° and the antversion angle was 15°. Radiographs showed the stem and cup in good position (Figure 1A). The erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were 46 mm/h (normal, 0-20 mm/h) and 6.1 mg/L (normal, 0-4.9 mg/L), respectively. Serum chromium and cobalt were 0.7 µg/L (normal, 1.4 µg/L) and 9.8 µg/L (normal, <0.5 µg/L), respectively. Magnetic resonance imaging revealed joint capsule distention, osteolysis, and abductor disruption (Figure 2). A hip aspiration was culture-negative in 3 samples. The manual cell count was 2 white blood cells, 7000 red blood cells with a differential of 50% polymorphonuclear cells, and 50% lymphocytes. A large amount of acellular debris was observed, which prompted the manual count because the automated machine count would be inaccurate secondary to the debris being counted as cells by the machine.

The patient underwent an acetabular revision and ball exchange. The acetabular component was loose. A modular revision cup with supplemental screws, a polyethylene liner, and a 40-mm ceramic head were used. Intraoperatively, a large solid pseudotumor was noted encompassing the capsule and protruding anteriorly with necrosis of the iliopectas tendon sheath and muscle. Corrosion material was found around the taper junction. The retrieved ball was scored as moderate corrosion (3 of 5 points) according to a published score for grading taper corrosion. 12 Histopathologic examination revealed extensive necrosis consistent with a reaction...
to metallic wear and corrosion products with tissue necrosis. Serosanguineous fluid obtained from the hip joint at the time of revision was sent for cultures and sensitivities. Culture-negative periprosthetic joint infection was excluded based on the patient’s synovial cell count from the preoperative hip aspiration (2 white blood cells, large amounts of cellular debris, and 50% polymorphonuclear cells), 3 negative cultures with no bacterial growth from the preoperative hip aspiration, the presence of a pseudotumor and corrosion at the trunnion, intraoperative frozen sections and permanent pathology diagnosis demonstrating no acute inflammation but consistent with chronic inflammation and perivascular lymphocytic infiltrates with scattered eosinophils and foreign body reaction, and 3 intraoperative synovial fluid and tissue cultures that showed no bacterial growth.

The patient had 2 postoperative posterior dislocations at 6 weeks and 3 months postoperatively that were treated with closed reduction. At subsequent follow-up, he had groin and trochanteric discomfort. At 1.5 years after the revision, he had increased right hip pain and trochanteric tenderness. He subsequently developed a Trendelenburg gait and positive Trendelenburg’s sign. Radiographs revealed a greater trochanteric fragmentation that progressed over 2.5 years (Figures 1B-E). At that time, the ESR and CRP were normal. Serum cobalt was not detected (normal, <0.9 µg/L) and serum chromium was 0.3 µg/L (normal, 0.1-2.1 µg/L). The laboratory results seem to rule out infection and the possibility that continued corrosion were causes of the progressive greater trochanteric fragmentation.

He was treated with nonsteroidal anti-inflammatory drugs and rest followed by physical therapy. At the most recent follow-up, he could walk without assistance but had weak abductors and trochanteric tenderness. He continues to have moderate pain. During follow-up, the patient has never taken antibiotics or had issues with wound drainage. Based on the collective data from the preoperative presentation, intraoperative findings, and follow-up observations, the patient’s clinical course is consistent with ARMD as opposed to culture-negative periprosthetic joint infection. The authors believe there was bone necrosis at the time of the revision and that the bone was gradually resorbed, resulting in the greater trochanteric fragmentation.

**Patient 2**

A 54-year-old woman with osteoarthritis arthritis of the hip who had undergone bilateral MOM THAs presented for evaluation 3 years after her left THA. The arthroplasties were done with a Pinnacle cup (DePuy, Warsaw, Indiana), a 36-mm femoral head, and a Prodigy stem (DePuy) (US Food and Drug Administration–approved implants). Her hip replacements relieved her pain, and she was satisfied with the surgery. Radiographs showed stable cementless femoral and acetabular components without osteolysis. The cup abduction angle was 45° on left side (Figure 3A).

She had new onset of pain and swelling in her left buttock and greater trochanter 3 years postoperatively. The ESR was 38 mm/h (normal, 0-20 mm/h), CRP was 1.3 mg/dL (normal, 0-0.8 mg/dL), serum chromium was 4 µg/L (normal, ≤1.4 µg/L), and serum cobalt was 31 µg/L (normal, <0.5 µg/L). Magnetic resonance imaging revealed marked distention of her left hip capsule with an 11×11×8–cm mass, consistent with an adverse local tissue reaction. Aspiration demonstrated a milky type, low viscosity fluid. Analysis of the synovial fluid found 1,320 white blood cells, 20,000 red blood cells, 2% polymorphonuclear cells, 62% lymphocytes, 19% mononuclear cells, 17% eosinophils, and 0% other cells. The manual cell count also demonstrated significant acellular debris. Three synovial fluid specimens demonstrated no organisms and no bacterial growth from the cultures. A left hip revision was performed 39 months after her primary THA. During the revision, a solid mass involving the posterior capsule, the posterior one-third of the hip abductor, and external rotator was removed. Corrosion on the ball and stem were seen. The retrieved ball was scored as severe corrosion (4 of 5 points) according to a published corrosion score. The modular metal liner and ball were changed to a 36-mm metal-on-polyethylene bearing surface. Histopathologic examination revealed necrotic tissue with chronic inflammation and perivascular lymphocytic infiltrates consistent with a MOM reaction.

The patient had 2 dislocations that required closed reduction at 2 and 12 months after revision. Thirteen months after revision, fragmentation of the greater trochanter was noted radiographically (Figures 3B-E). Fourteen and 19 months after revision, she had febrile episodes associated with pain and swelling of her hip. Her serum chromium and cobalt ion level had decreased to 1.6 and 3 µg/L, respectively. The ESR and CRP were 35 mm/h (normal, 0-30 mm/h) and 14 mg/L (normal, <8 mg/L), respectively. An aspiration was negative for infection. The patient sustained a third dislocation, and
the decision was made to convert her to a constrained liner 24 months after her initial revision. At her most recent evaluation, the patient had a painless range of motion, a negative Trendelenburg’s sign, and no limp.

DISCUSSION

The authors present 2 patients with greater trochanter fragmentation and worsening symptoms at 1 year after conversion of failed MOM bearings to polyethylene bearing surfaces. Intraoperatively, pseudotumors were observed surrounding the joint capsule, as well as corrosion of the stem-ball taper junction. Adverse reaction to metal debris was the cause of failure given the painful MOM THA with a solid pseudotumor.

Several reports in the literature describe various clinical features of ARMD, including discomfort in the groin, lateral aspect of the hip or buttock, sensation of mass or visible swelling around the hip, a clicking sensation, late dislocation, hip instability, and localized osteolytic reaction. Although poor outcomes have been demonstrated after some MOM revisions, to the current authors’ knowledge, there are no other reports documenting greater trochanter fragmentation in ARMD. In a prior report that included 9 cases of greater trochanter fractures resulting from osteolysis, all of the hips demonstrated radiographic evidence of greater trochanteric osteolysis prior to fracture and most of the fractures healed in situ without operative fixation. Unlike prior reports, both of the current cases had a rapid progression of fragmentation and no evidence of bone cysts or osteolysis in the region of the greater trochanter at the time of revision and neither fracture healed with conservative treatment even though the pseudotumor was removed and the serum metal level had decreased to normal levels after revision (Figure 1 and Figure 3).

The authors suspect that there was greater trochanteric necrosis present at the initial revision and that the radiographic fragmentation was the result of gradual resorption of the necrotic bone. An alternative hypothesis is that the corrosion seen at the time of both revisions was ongoing after surgery and contributed to continued necrosis with fragmentation. However, the current authors believe this is unlikely because both patients had dramatic reductions in serum cobalt and chromium levels after conversion to a polyethylene bearing surface.

The current patients demonstrate that progressive resorption of necrotic bone secondary to a MOM pseudotumor can contribute to a poor clinical outcome and that the need for further surgery after conversion to polyethylene eliminated the metal debris generation. It has been believed that the consequences of ARMD might regress after revision surgery along with the trend for decreases in serum cobalt and chromium levels. In the current patients, ARMD had bone consequences that progressed even though the pseudotumors were removed and the serum metal level had decreased to normal levels after revision. The current patients still had persistent pain and inflammation of the hip 1 or more years after revision that was, in part, related to the greater trochanteric fragmentation.

When persistent inflammation is present, it is important to rule out periprosthetic joint infections that may be culture negative. Because periprosthetic joint infections cannot be ruled out based on a single test, the current authors rely on a series of tests and observations, including CRP, ESR, hip aspiration synovial cell counts and differential (manual not automated, looking for the presence of significant acellular debris), synovial fluid characteristics and appearance, chromium and cobalt metal levels, constitutional symptoms, synovial fluid and tissue culture results on greater than 3 samples, intraoperative findings consistent with the presence of corrosion, tissue necrosis and/
or pseudotumor, and frozen and permanent tissue pathology. For the 2 patients in the current report, the results of these tests and observations indicated ARMD as opposed to infection. Both patients also had hip dislocations, which might be attributed to soft tissue necrosis around the hip joint. Although dislocation is a relatively common complication after revision surgery, with a reported incidence typically ranging from 5% to 10%,<sup>18-20</sup> although it can be as high as 25% in some patient populations,<sup>21</sup> the current authors have not previously encountered trochanteric fragmentation after a dislocation as was seen in these 2 cases. The current patients, like other patients in the literature who had revisions for ARMD, had outcomes significantly worse than patients who had revisions for other reasons.<sup>12,22</sup>

**Conclusion**

These patients emphasize the fact that tissue necrosis associated with some failed MOM bearing hips is not limited to soft tissues. In these patients, the bone necrosis did not heal and manifested as greater trochanteric fragmentation that became radiographically evident 1 to 2 years after revision. Surgeons should be aware of this radiographic finding and the associated clinical symptoms. Infection should be ruled out. The authors recommend nonoperative treatment for greater trochanter fragmentations but close follow-up of patients due to the high risk of dislocation.

**References**


