



Case report

Adverse reaction to metal debris with concomitant incidental crystalline arthropathy in hip arthroplasty

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ABSTRACT

Adverse reaction to metal debris (ARMD) is a known cause of failed metal in hip arthroplasty. Diagnosis of this type of prosthesis failure may be difficult, and the hallmark is an abnormally elevated serum cobalt level. Concomitant diagnoses may also be present, such as infection, instability, and loosening, and this may confuse interpretation of abnormal laboratories. We present here, for the first time, 2 patients with ARMD and crystalline arthropathy. In each case, the patient chose surgery for ARMD, with resolution of symptoms and no recurrence of the crystalline arthropathy. We present these cases to alert the orthopaedist that crystalline arthropathy may be present at the same time as ARMD, but is likely not the primary cause of symptoms.

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Introduction

Hip arthroplasty (HA) is the most effective treatment to relieve pain and increase function for end-stage arthritis of the hip [1]. Unfortunately, between 1% and 2% of HA, patients will develop an early complication requiring surgical revision [2], and about 10% will require revision over a longer period [3]. A cornerstone of successful management of implant failure is early and accurate diagnosis of the exact cause or causes, which allows appropriate treatment.

Current recommendations for patients with otherwise unexplained pain after HA includes assessment for infection [4]. More recently, adverse reaction to metal debris (ARMD) after hip resurfacing of metal-on-metal (MoM) hips and adverse local tissue reaction secondary to mechanically assisted crevice corrosion (MACC) have been recognized as emerging concerns for failure in

contemporary HA, and should also be considered [5,6]. A less common cause of hip pain and synovitis after HA is crystalline arthropathy such as gout or pseudogout. Reports of crystalline arthropathy in a total joint replacement alone [7–9] or in conjunction with infection [10] have been previously reported.

Crystalline arthropathy and infection have been noted to coexist in 1.5% of 265 native joint aspirations based on a retrospective review of arthrocentesis samples [11]. To our knowledge, there has never been a published case of ARMD after HA with concomitant crystalline arthropathy. We present 2 cases in which crystalline arthropathy of the artificial hip was diagnosed by joint fluid analysis before revision surgery for ARMD. Neither case had a deep infection, and in both cases, repeat aspiration at the time of surgery was negative for crystals. Both patients provided written informed consent for the print and electronic publication of this case report.

Case histories

Case 1

A 65-year-old male patient presented with acute left hip pain 14 months after a left total hip arthroplasty (THA) with a cobalt-chromium alloy femoral head, titanium stem, cross-linked polyethylene acetabular bearing surface and tantalum acetabular shell

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(Zimmer, Inc., Warsaw, IN). His pain was rated at 3 or 4 out of 10, and was primarily in the groin area. He noted that this pain caused difficulty with daily tasks such as putting shoes and socks on, as well as walking up stairs. Pain occurred with the startup of activity, and the discomfort abated as he moved around. The patient denied having any postoperative pain before this acute situation, and was diligent in following his physical therapy protocol postoperatively. He denied other joint pain, fevers, chills, or systemic symptoms. Past medical history was negative, specifically for any history of gout or pseudogout, kidney disease, alcohol abuse, or dietary restrictions. On physical examination, palpation over the greater trochanter elicited no pain. The surgical incision was well healed. Hip flexion was 115° and range of motion was painful at extremes. He had a positive Stinchfield sign [12]. There was no loss of sensation or muscle strength. Deep tendon reflexes were 2+ bilaterally.

Radiographs demonstrated excellent fixation of the left THA, with no signs of osteolysis, loosening, or polyethylene wear (Fig. 1a and b). The component position appeared satisfactory and there was no prominence of the anterior acetabular component on the direct lateral radiograph to suggest iliopsoas impingement. Laboratory tests demonstrated normal serum white blood cell (WBC). Serum C-reactive protein (CRP) was elevated at 1.67 mg/dL (normal, ≤ 1.0 mg/dL). Erythrocyte sedimentation rate was elevated at 22 mm/h (normal, 0–20 mm/h). Serum cobalt level was elevated at 5.5 ppb (abnormal, ≥ 1.0 ppb), and chromium was in acceptable range at 0.2 ppb (abnormal, ≥ 0.3 ppb). A metal artifact reduction sequence magnetic resonance imaging (MRI) scan was unremarkable, with no signs of pseudotumor or synovitis (Fig. 2). The patient's symptoms in conjunction with the elevated serum cobalt level and inflammatory markers suggest an ARMD in association with MACC.

Synovial fluid was aspirated and analyzed to confirm MACC and rule out concomitant diagnoses. The fluoroscopy-guided aspiration yielded purulent material, with intracellular and extracellular monosodium urate crystals observed (negative birefringence under polarized light microscopy), confirming a diagnosis of gout. The sample was reviewed by 2 experienced laboratory technicians, including a supervisor to confirm diagnosis. The technician-counted synovial WBC count was 1488/cmm. Polymorphonuclear leukocytes were elevated at 92%. Gram stain was negative for bacteria and culture showed no growth at 10 days, ruling out infection.

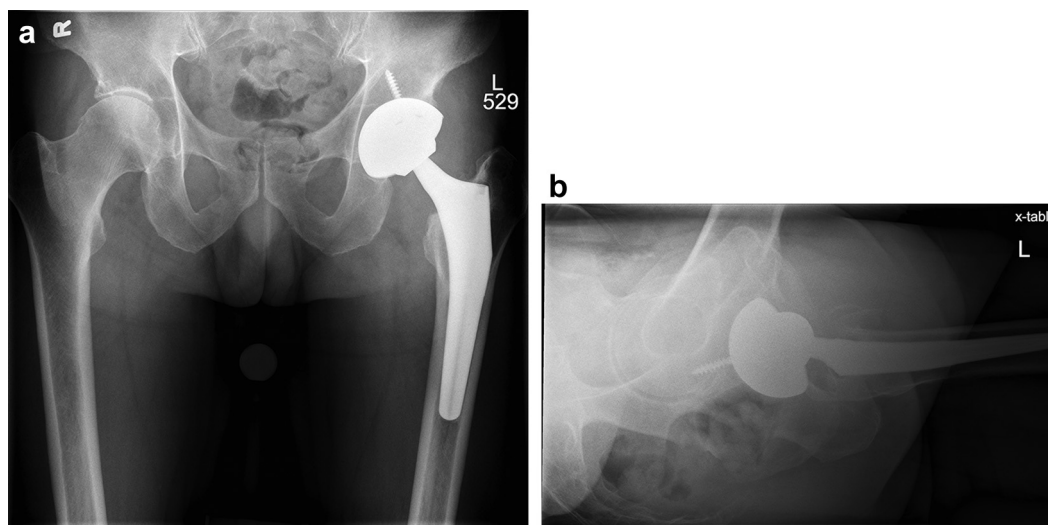


Figure 1. (a) Patient 1, Anteroposterior (AP) radiograph at presentation shows no signs of osteolysis, loosening, or polyethylene wear of left THA. (b) Patient 1, lateral radiograph showing excellent fixation of left THA

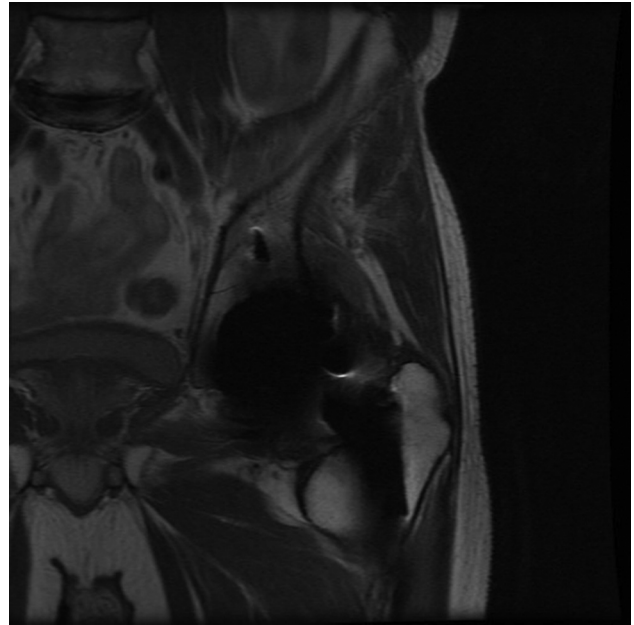


Figure 2. Patient 1, metal artifact reduction sequence (MARS) MRI at presentation is unremarkable, showing no signs of pseudotumor or synovitis.

A methylprednisolone dose pack was prescribed for gout in conjunction with MACC, and on subsequent follow-up appointments, there was a gradual diminution in hip symptoms. The patient was monitored for increases in cobalt levels and inflammatory markers, as the elevated serum cobalt levels were likely due to corrosive products [5]. Indications for revision for MACC remain controversial, but have been proposed to include ongoing symptoms, abnormal MRI, or progression of elevation in serum ion levels [13].

Approximately 4 months after the hip aspiration, serum elevation in cobalt was found to be 9.6 ppb, and the patient reported gradual return of symptoms. A left hip revision was offered after shared decision-making, including acetabular polyethylene exchange and revision of the Co alloy femoral head to a BIOLOX delta head with a revision titanium sleeve (CeramTec, Plochingen, Germany). At surgery, aspiration was repeated, and no intracellular

or extracellular urate crystals were found, suggesting resolution of the gout. The patient recovered without complication and is under regular follow-up; he has not experienced any similar attacks of pain or other joint involvement. Serum Co has returned to 0 ppb, and Cr continues to decrease (0.9 ppb at 6-month follow-up).

Case 2

A 54-year-old female patient presented with acute-on-chronic left hip and groin pain 7 years after bilateral hip MoM resurfacing arthroplasties (ASR, DePuy, Warsaw, IN), which were performed in New Zealand. Her pain was sharp, 10 out of 10, and was in both the groin and lateral hip. She reported that she recently began working a job that required standing for prolonged periods. Furthermore, she stated that the pain caused an inability to move her left hip freely, as well as difficulty with daily tasks such as putting shoes and socks on, and walking up stairs. The patient had low-grade trochanteric pain since her surgery, and knew that she had a fibrous union of the trochanteric osteotomy. She denied other joint pain, fevers, chills, or systemic symptoms. Past medical history was negative, specifically for any history of gout or pseudogout, kidney disease, thyroid disease, alcohol abuse, or dietary restrictions. On physical examination, palpation over the greater trochanter elicited moderate pain. The surgical incision was well healed. Hip flexion was 105° and range of motion was painful at extremes. She had a positive Stinchfield sign [12]. There was no loss of sensation or muscle strength, other than 3/5 strength of hip abduction against gravity. Deep tendon reflexes were 2+ bilaterally.

Radiographs demonstrated satisfactory fixation of the left resurfacing arthroplasty (RA), with fibrous union of the prior trochanteric slide osteotomy [14] and associated screw breakage (Fig. 3a and b). The component position appeared satisfactory. Laboratory tests demonstrated normal serum WBC. Serum CRP was elevated at 1.87 mg/dL (normal, ≤ 1.0 mg/dL). Erythrocyte sedimentation rate was normal at 11 mm/h (normal, 0–20 mm/h). Serum cobalt and chromium levels were elevated at 8.4 ppb and 4.2 ppb, respectively. There is no known completely agreed on normal metal level with bilateral MoM resurfacing arthroplasties, but these levels are significantly elevated [15]. A metal artifact reduction sequence MRI scan was abnormal, with no obvious fluid collection, but significant soft tissue and bony edema, which was consistent with ARMD. The MRI also showed evidence of nonunion and hardware migration associated with the prior left trochanteric osteotomy (Fig. 4). The patient's symptoms, in conjunction with the

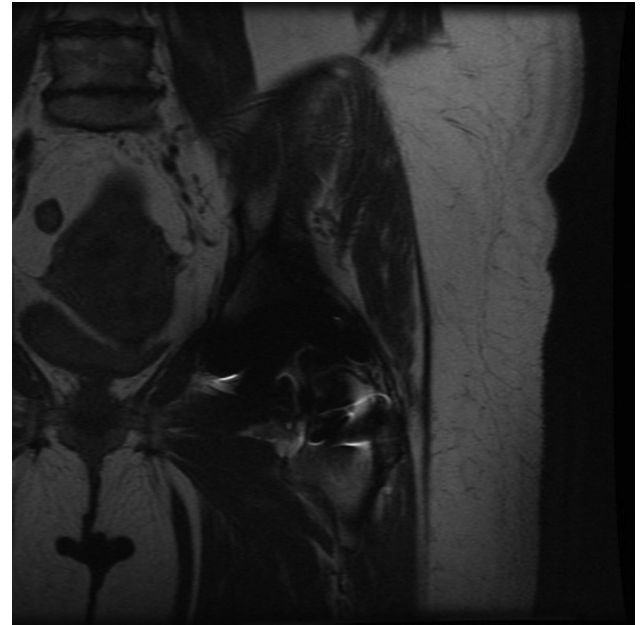


Figure 4. Patient 2, MARS MRI at presentation showing evidence of nonunion and hardware migration associated with prior left trochanteric osteotomy.

elevated serum Co, Cr, CRP, and MRI suggest an ARMD in association with recalled MoM RA.

Synovial fluid was aspirated and analyzed to confirm ARMD and rule out concomitant diagnoses. The fluoroscopy-guided aspiration yielded bloody synovial fluid, with rhombus-shaped extracellular calcium pyrophosphate crystals (weak positive birefringence under polarized light microscopy), consistent with pseudogout. The sample was reviewed by 2 experienced laboratory technicians, including a supervisor, to confirm the diagnosis. The technician-counted synovial WBC count was 1363/cmm. Polymorphonuclear leukocytes were elevated at 87%. Gram stain was negative for bacteria and culture showed no growth at 10 days, ruling out infection.

The patient's pain stabilized and she was monitored over the next year for inflammatory markers and changes in serum metal ion concentrations. She had been offered surgical intervention, but deferred until 1 year later when she presented with severe hip pain. Her serum chromium and cobalt were continuing to elevate, at

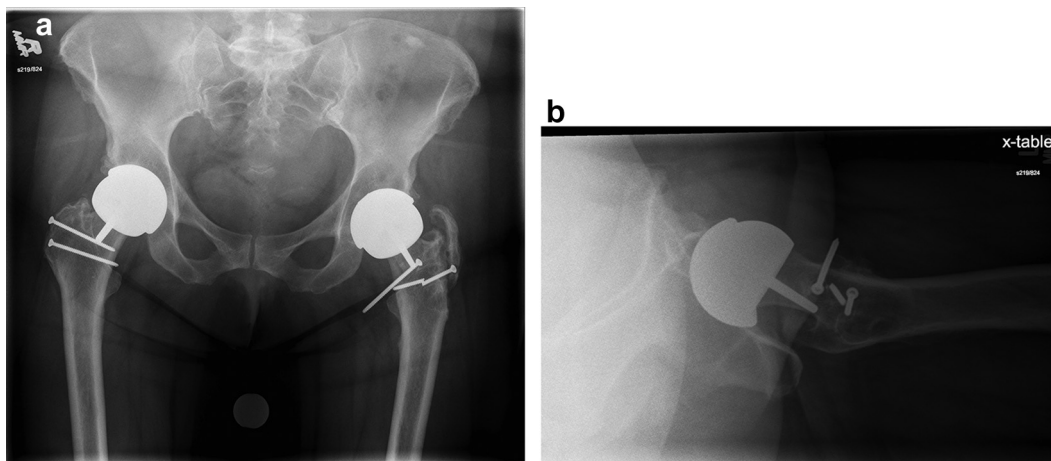


Figure 3. (a) Patient 2, AP radiograph at presentation demonstrates fibrous union of trochanteric slide osteotomy and associated screw breakage of left RA. (b) Patient 2, lateral demonstrates satisfactory fixation of left RA, with screw breakage.

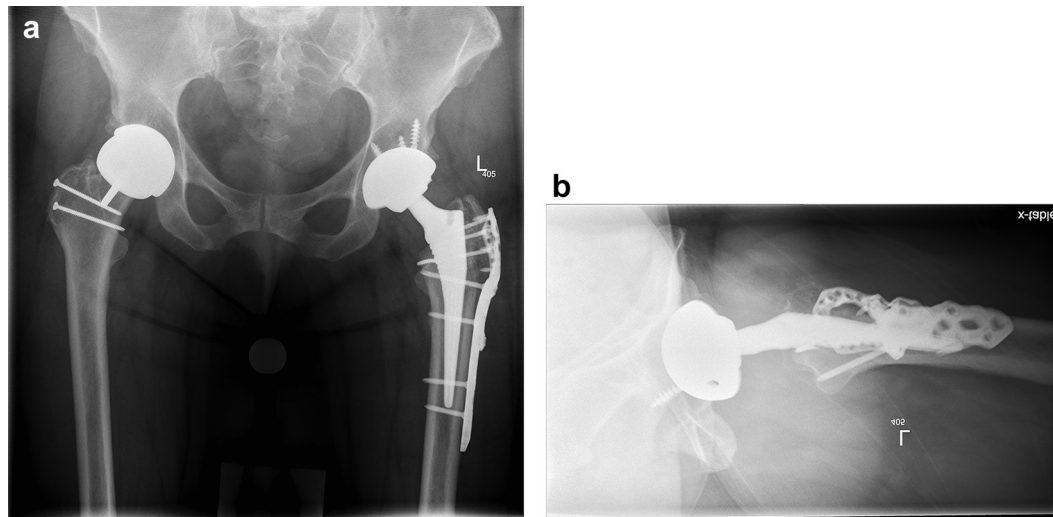


Figure 5. (a) Patient 2, AP radiograph status after revision showing left THA after revision, including open reduction internal fixation of trochanteric nonunion with an autologous bone graft, and removal of fractured screw. (b) Patient 2, lateral radiograph showing left THA after revision.

25.5 ppb and 18.0 ppb, respectively. At this point, the patient's preliminary diagnoses were: (1) failed left resurfacing THA with a recalled prosthesis, (2) trochanteric nonunion, and fracture of retained hardware, and (3) concomitant pseudogout of the left hip.

A revision of both components of her left THA was performed, including an open reduction internal fixation of the trochanteric nonunion with an autologous bone graft, and a partial removal of the fractured screw (Fig. 5a and b). During the surgery, another synovial fluid aspiration was performed, and no crystals were found. The patient continued to improve in subsequent follow-up appointments. Serum metal ion levels have continued to drop since revision surgery. The patient healed well and has yet to have another similar attack of pain.

Discussion

Crystalline arthropathy, including gout and pseudogout, may present acutely with severe pain, stiffness, and dysfunction, with overlying erythema around the associated joint. The underlying pathogenesis involves the deposition of monosodium urate crystals (gout) or calcium pyrophosphate crystals (pseudogout) within the articular and periarticular structure of one or more joints. Monoarticular involvement in crystalline arthropathy is most common, but polyarticular episodes can occur [16]. In gout, the first metatarsophalangeal joint is most commonly involved, with other frequently involved joints including the midfoot, ankle, and knee [17]. Conversely, in pseudogout, the most often involved joints are the knee and wrist, but cases have also been found in the elbow, ankle, metacarpophalangeal joints, sternoclavicular joints, and the hip [17–19]. Gout and pseudogout have also been described in conjunction with HA [20].

The presence of urate crystal deposition can occur after precipitating events such as trauma, surgery, psoriasis flares, chemotherapy initiation, diuretic therapy, infection, IV contrast media, acidosis, and alcohol consumption [21,22]. However, hyperuricemia is the single most important risk factor for developing gout [23]. The risk factors for pseudogout include older age, previous joint damage, osteoarthritis, loop diuretics, and certain metabolic diseases, such as hemochromatosis, hypophosphatemia, hypomagnesemia, hyperparathyroidism, and hypothyroidism [16].

In both cases presented, the only risk factors were ARMD because of metal and/or corrosion products locally.

Following a synovial fluid aspiration in suspected ARMD cases revealing concomitant crystalline arthropathy, physicians cannot assume that all symptoms are due to crystal deposition. Synovial fluid WBC counts can frequently be confusing and should be performed manually to ensure accurate results [24]. Ruling out periprosthetic joint infection using cultures in addition to subsequent pathology analysis obtained at the time of surgery is imperative.

In considering possible causes of crystal precipitation in these cases, our theory is that joint pH changes in MACC and ARMD within the joint capsule may predispose to crystal deposition. It is well-known that acidosis is an important risk factor for hyperuricemia, and also promotes calcium pyrophosphate and uric acid crystal transformation to the insoluble form [25]. Fretting interfaces in MACC lead to the accumulation of hydrogen ions within the implant interfaces [26]. This resultant drop in pH may explain the conditions within the joint capsule that predispose patients with MACC to developing gout or pseudogout.

Acute crystalline arthropathy in HA is a considerably rare occurrence, with only a few cases reported in the literature. Historically, it has been important to consider crystalline arthropathy when a patient presents with a painful HA, as this can mimic the presentation of both infection, with elevated inflammatory markers, local erythema, and acute pain. Failure to recognize the symptoms as gout or pseudogout can result in unnecessary and inappropriate care. On the other hand, we have demonstrated that, although present in both patients in this report, this was not the primary cause of their hip failures. The crystalline arthropathy may have contributed to their presenting symptoms, but resolved before revision. Since revision surgery, the patients have been followed closely and no symptoms of crystalline arthropathy have recurred. We conclude that in contrast to patients with HA and no MACC or ARMD, crystalline arthropathy is a secondary cause of pathology in metal reaction THA failures, and should not be treated in isolation. These patients must be revised for the failed HA.

Summary

We present here 2 patients who presented with acute hip pain after primary THA. These patients were subsequently diagnosed

with ARMD and concomitant crystalline arthropathy. In each case, the patient chose revision surgery for ARMD, with resolution of symptoms and no recurrence of the crystalline arthropathy. We report these cases to alert the orthopaedist that crystalline arthropathy may be present at the same time as ARMD, but is likely a secondary cause of symptoms. Serum and synovial laboratory tests may be abnormal because of the crystals, but revision for ARMD resolved the symptoms in our patients.

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