



# Metallosis in metal-on-metal hip resurfacing: An unusual presentation

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## ABSTRACT

A case of a 75-year-old gentleman with an unusual presentation of metallosis 3 years after a left hip resurfacing arthroplasty is presented. This report illustrates the atypical appearances of metallosis using CT and other imaging modalities, which has been only sparsely described in the literature.

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## 1. Introduction

Metallosis is a rare complication of arthroplasty. It is defined as the infiltration of peri-prosthetic soft tissue and bone by metallic debris resulting from the wear of arthroplasties [1]. This diagnosis can be made radiographically by the detection of cloudy radiodensities in periprosthetic tissue [2] or hyperdensities around the joint space [3]. However, cystic collections caused by inflammatory activation in response to metallic debris can sometimes be missed without cross-sectional imaging. We describe the radiographic and CT features of a case of metallosis in the hip occurring 3 years post-resurfacing arthroplasty.

## 2. Case report

A 75-year-old male, with a past medical history of ischaemic heart disease, underwent a left hip resurfacing arthroplasty in May 2007 for advanced osteoarthritis of the left hip. The prosthesis used was a DePuy ASR<sup>TM</sup> Hip Resurfacing System (size 51 ASR head and

58 ASR cup) and was fitted using ASR<sup>TM</sup> computer navigated instruments. The operation was uncomplicated, with excellent fixation and a stable hip joint being achieved. Postoperative plain X-rays of the left hip revealed the prosthesis in a satisfactory position and the patient initially did well.

He presented 2 years later (January 2009) with intermittent lumbar back pain, radiating down the lateral aspect of the left thigh. Clinical examination confirmed no left hip joint tenderness or signs of inflammation, and excellent range of hip movement. Plain film radiographs of the left hip did not show any abnormality (Fig. 1). He was referred subsequently to the neurosurgeons as the problem was thought to be originating from the spine. An MRI scan of the lumbar spine (1.5 T, Siemens, Germany) revealed degenerative disc protrusion from L2–4 with lateral canal stenosis. He went on to have a L3/4 lumbar laminectomy and undercutting of L2/3 and L4/5 in August 2009. This appeared to relieve some of the presenting symptoms.

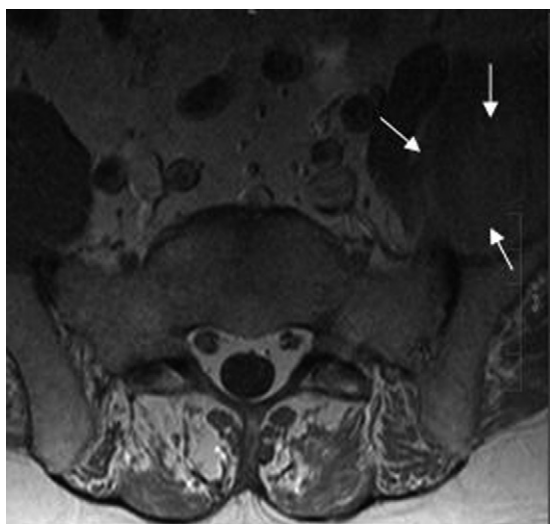
He re-presented in January 2010 with worsening left sided lower back pain radiating to the left gluteal region and to the posterolateral aspect of the left thigh. The range of movements in the left hip remained satisfactory in all directions. White cell count (WCC) was  $11.8 \times 10^9 \text{ L}^{-1}$  and C-reactive protein (CRP) was raised at 375 mg/L. Repeat MRI of the lumbosacral spine confirmed that the marked spinal stenosis had been relieved by the laminectomy, and there was no evidence of further impingement on the nerve roots. However, it did show an abnormal inflammatory T2W signal in the left ileopsoas (Fig. 2). To clarify, a CT scan of the abdomen and pelvis (64 slice Aquilion, Toshiba Medical Systems, Japan) was undertaken in

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**Fig. 1.** Plain film AP radiograph of left hip. The arthroplasty is satisfactory and no abnormalities are evident.



**Fig. 2.** Axial T2W MRI section through the upper pelvis showing an asymmetric enlargement of the left iliopectineal bursa with an abnormal high signal. This prompted further investigations.

February 2010. This showed asymmetrical enlargement of the left iliacus with some stranding in the anterior fat around the paracolic gutters in the left iliac fossa. Two fluid collections were seen (1) a slightly hypodense collection 3.1 cm in diameter within the body of the left iliacus (Fig. 3a) and (2) a trilobulated collection in the left groin, the largest of which measured 6.3 cm × 4 cm and was related to the anterior aspect of the femoral neck (Fig. 3b). These appeared to communicate. There was no osteomyelitis and the arthroplasty itself appeared concentric and normal. The loculated lesions were shown to be unrelated to the previous laminectomy. Given the communication, a clinical suspicion of metallosis was raised. To prove this, the periarticular and iliacus collections were separately aspirated under ultrasound guidance, and approximately 10 mL and 20 mL of straw coloured fluid was aspirated from the collections, respectively (Fig. 3c). The fluid was sent for culture and sensitivity and analysis for urates/crystals and metalloids. Using the same needle in the iliacus collection, 15 mL of dilute, non-ionic, iodinated contrast (Niopam 300, Bracco UK Limited) was injected into the iliacus collection and a limited CT of the pelvis was performed. This showed communication between the iliacus and the periarticular

collection (Fig. 3d and e). Given the communication, the main differential diagnosis at this point was metallosis secondary to the left hip resurfacing arthroplasty.

The cytological specimen was prepared using thin prep liquid base cytology and stained with the Papanicolaou technique. Examination revealed a paucicellular preparation with scattered histiocytes together with sparse mixed inflammatory cells. Within the cytoplasm of some of the histiocytes there was an accumulation of particulate black pigmented metallic fragments (Fig. 4). No crystals were seen on polarised light microscopy. This was in keeping with a diagnosis of metallosis.

The patient underwent a revision left total hip arthroplasty (with conversion of the hip resurfacing into uncemented arthroplasty) in March 2010. He is currently making excellent progress, and is able to fully weight bear on his left leg with no pain.

### 3. Discussion

Hip resurfacing techniques were first described in the 1970s, but initially rejected due to high failure rates [4]. Over time with increased understanding of new materials available and implant techniques, they were re-introduced in the 1990s [4]. The Birmingham Hip is an example of metal-on-metal hip resurfacing (MMHR). However, recent studies have reported increasing prevalence of complications requiring revision surgery [5]. MMHR is commonly performed and is the choice of prosthesis for young patients. It enables easier revision surgery, unless complications including metallosis occur [6].

Common complications of hip resurfacing include fracture of the femoral neck, avascular necrosis, and collapse of the femoral head, femoral impingement and aseptic loosening of a component [4,5]. In addition, other studies have indicated biological reactions around MMHR resulting in an immunological response leading to osteolysis, tissue destruction and high levels of cobalt, chromium and molybdenum [6].

The term metallosis is defined as an infiltration of peri-prosthetic soft tissue and bone by metallic debris resulting from the wear of arthroplasty [1]. At a cellular level it has been shown that there is perivascular infiltrate of T- and B-lymphocytes and plasma cells, high endothelial venules, massive fibrin exudation, accumulation of macrophages with droplike inclusions with eosinophil infiltration leading to necrosis [7]. Previous studies have referred to this presentation as 'pseudotumours', where there is a non-infective solid and or cystic mass related to the metal on metal hip resurfacing. These have become known as aseptic vasculitis associated lesions [8]. This is a recognised, but rare complication of arthroplasty.

A number of reasons have been postulated to explain the cause of metallosis. These include femoral head position against the weight bearing surface of the acetabular component [9] or incorrect positioning during surgery resulting in impingement on the acetabular cup [10]. Risk factors include female gender, a small femoral component, obesity and a high abduction angle [11]. Several recent studies have illustrated the incidence of metallosis causing failure and requiring subsequent revision surgery [12]. This ranges from 0.3 to 3.1% over 3–5 years [11–13]. A large review of 418 cases of total hip arthroplasty identified a failure rate due to metallosis of 5.3% [13]. Patients almost universally present with pain in groin and buttocks, loss of function and a soft tissue mass [6]. Diagnosis is normally made on clinical suspicions supplemented by radiographic evidence and surgery [14].

The role of diagnostic radiology is less clear. On plain films, radiographic signs have been documented including the 'cloud' and

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