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Non-ischaemic cardiomyopathy associated with elevated serum cobalt and accelerated wear of a metal-on-metal hip resurfacing

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Non-ischaemic cardiomyopathy associated with elevated serum cobalt and accelerated wear of a metal-on-metal hip resurfacing

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SUMMARY

A man in his late 30s developed non-ischaemic cardiomyopathy due to systemic cobalt toxicity associated with accelerated bearing surface wear from metal-on-metal hip resurfacing implanted in the previous 6 years. Following revision arthroplasty, the patient regained baseline cardiac function. Cobalt-induced cardiomyopathy is a grave condition that deserves early consideration due to potentially irreversible morbidity. We present this case to increase awareness, facilitate early detection and emphasise the need for research into the diagnosis and management of at-risk patients.

BACKGROUND

The Birmingham hip resurfacing (BHR, Smith and Nephew, Memphis, Tennessee, USA) has reported implant survival rates of 98% at 15 years.[1] This contrasts most metal-on-metal total hip arthroplasties that have been removed from the market due to local and systemic complications.[2–5] While the use of metal-on-metal arthroplasty components is minimal in current practice, many patients with existing implants are at risk of severe sequela, including cardiovascular disease. We present a case of non-ischaemic cardiomyopathy due to systemic cobalt toxicity from BHR implant wear to aide in understanding the pathophysiology and diagnosis of this comorbid condition.

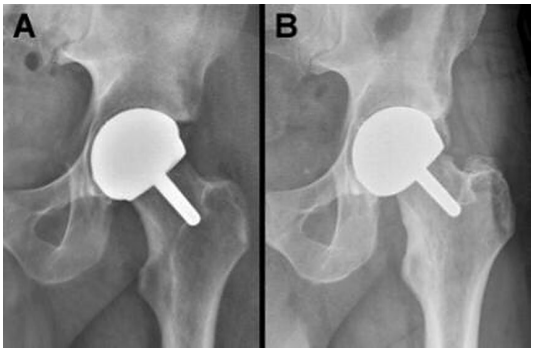

Figure 1 (A) Six-week postoperative and (B) prerevision (7 years after index surgery) left hip anteroposterior radiographs. There was no evidence of implant loosening or migration on the prerevision radiographs compared with the initial postoperative films. CT scan prior to revision surgery measured 51° acetabular inclination and 48° acetabular anteversion.

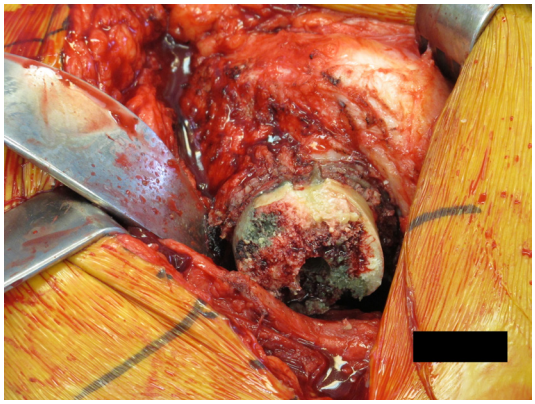

Figure 2 Patchy, dark staining of the cancellous bone was observed after removal of the resurfacing component.

CASE PRESENTATION

A man in his early 30s with history of left hip septic arthritis, treated 3 years previously with surgical debridement and antibiotics, presented to the senior author's clinic complaining of left hip pain. On exam, the patient had limited motion. Radiographs demonstrated bone-on-bone arthritis, and infectious workup was negative. Due to the patient's age, he opted for BHR. A couple of months later, the BHR was completed using size 52 cup and 44 femoral head, with a routine perioperative course.

Six years later, the patient presented to the emergency department with shortness of breath. Workup showed a severely reduced left ventricular ejection fraction (EF) of 25% and large pericardial effusion. Cardiac catheterisation ruled out cardiac ischaemia and pericardiocentesis yielded transudate. The patient was stabilised and discharged on diuretics and beta blockers with a diagnosis of non-specific cardiomyopathy. His symptoms improved over the next 2 months and repeat echocardiogram showed an EF of 60%. However, several months later, he returned to the cardiologist for shortness of breath. Inotropes were prescribed. Four months later, he was admitted for acute cardiac decompensation. His EF was 30%.

INVESTIGATIONS

At the time of the patient's acute cardiac decompensation, autoimmune and infectious workups for non-ischaemic cardiomyopathy were negative. The patient was stabilised with intravenous inotropes.

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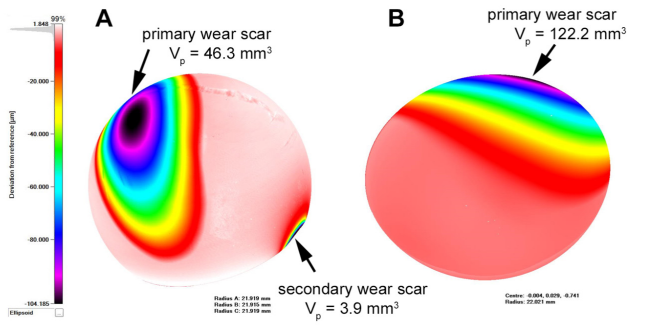

Figure 3 Optical coordinate measuring machine heat maps of the femoral head (A) and acetabular liner (B) bearing surfaces show primary and secondary wear scars with corresponding volumetric wear penetration, indicative of edge loading. Illustration and model were completed by DJH and RP.

Given the atypical clinical presentation, cardiology considered metal-induced cardiotoxicity and ordered specific metal labs. Serum cobalt was significantly elevated at 178.7 ng/mL and a repeat confirmatory lab was 141.8 ng/mL (normal <1.0 ng/mL; ARUP Laboratories, Salt Lake City, Utah, USA). Worsening cardiac function prompted admission to the cardiac critical care unit and chelation was initiated with acetylcysteine. Within 72 hours of chelation, serum cobalt decreased to 38.1 ng/mL. Myocardial biopsy performed at this time showed interstitial fibrosis and vacuolar changes consistent with prior reports of cobalt-associated cardiomyopathy.[6 7]

TREATMENT

Orthopaedics was consulted for evaluation of the BHR. The patient was medically optimised for revision surgery with 2 weeks of acetylcysteine. Prerevision radiographs showed no implant loosening (figure 1). CT of the abdomen/pelvis demonstrated an acetabular component with 51° of inclination and 48° of anteversion. Ultrasound and MRI showed an intra-articular effusion that communicated with the iliopsoas bursa.

Intraoperatively, after making the arthrotomy, there was straw-coloured synovial fluid with no signs of infection. The acetabular and femoral components were both well fixed. On implant removal, patchy areas of black-stained cancellous bone were noted (figure 2). The hip was successfully revised to a ceramic-on-polyethylene total hip arthroplasty.

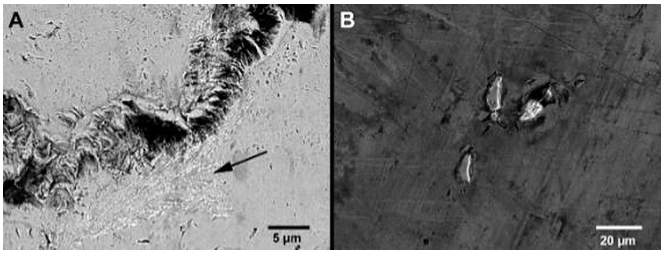

Figure 4 (A) Backscattered scanning electron microscope image of the CoCrMo femoral head bearing surface with a large chatter mark (trail left behind by hard particles trapped between the bearing surfaces) and indents indicating abrasive third body wear. Mixed hard phases (arrow) are also seen on the surface. (B) Embedded carbide particles within the surface of the femoral head.

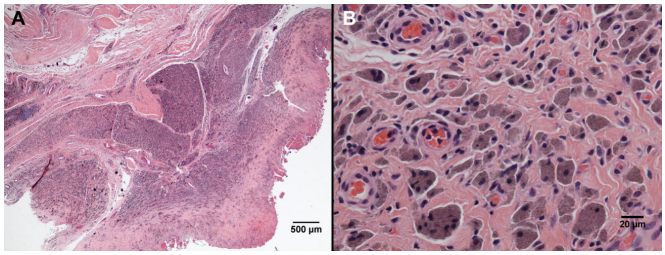

Figure 5 (A) The joint pseudocapsule was dominated by a marked macrophage response. The synovial surface (at right) was replaced by an organising fibrin exudate. (B) Greyish, particle-laden macrophages were seen throughout the tissue. Larger, dark particles were discernible within the cytoplasm. A few scattered plasma cells are also present. (H&E; A, ×20; B, ×400).

OUTCOME AND FOLLOW-UP

Postoperatively, echocardiography showed an EF of 30% on intravenous inotropes, and serum cobalt level was 63.1 ng/mL. Two months after surgery, the patient's EF was 49% and the patient reported he was back to his functional baseline. The patient was gradually weaned off intravenous cardiac inotropes over the next 2 months and was transitioned to only taking oral medications. By 4 months postoperatively, his serum cobalt level had decreased to 4 ng/mL. Two years after revision surgery, the patient continues to take maintenance oral cardiac medications and regularly follows up with his cardiologist. He has sustained normal cardiac function on follow-up echocardiograms. At the time of writing, the patient is alive and continues to functionally improve.

Retrieval analysis

Methodology

Both head and liner were measured with an optical coordinate-measuring machine (OrthoLux, RedLux) for three-dimensional reconstruction of the original bearing surface and computation of volumetric wear. Bearing surfaces were also viewed in a scanning electron microscope (SEM) to examine wear features.

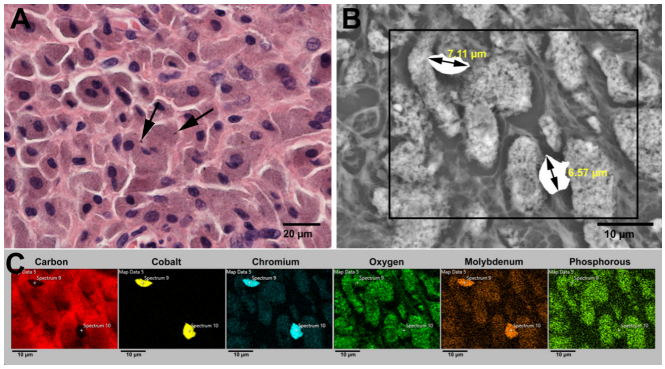

Figure 6 (A) High-power micrograph of particle-laden macrophages within the joint pseudocapsule. A few large particles are also visible (arrows) (H&E, ×600). (B) Backscattered scanning electron microscope image of a comparable area in an adjacent section to (A), showing densely packed macrophages. Two large (7.11 and 6.57 µm) extracellular particles are also present as denoted by the arrows along the long axis of the particles (×2200). (C) EDS elemental maps of the area outlined in (B) demonstrate that the intracellular particles within the macrophages are chromium phosphate and the two extracellular particles are CoCrMo alloy. EDS, energy-dispersive x-ray spectroscopy.

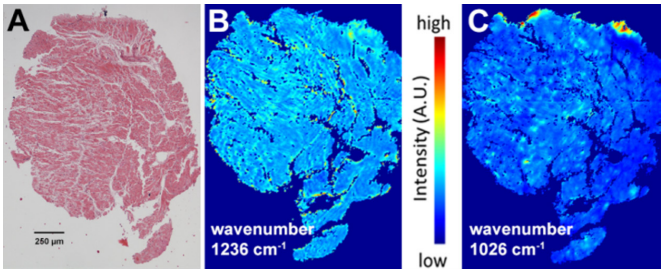

Figure 7 (A) Cardiac biopsy adjacent to tissue samples shown in (B) and (C) (H&E, ×40). (B) FTIR-I heat map imaged at the amide III-specific wavenumber (1236/cm) showing abnormal collagen expansion and confirming interstitial fibrosis. (C) FTIR-I heat map imaged at 1026/cm, indicating granular glycogen deposition. FTIR, Fourier transform infrared spectroscopy.

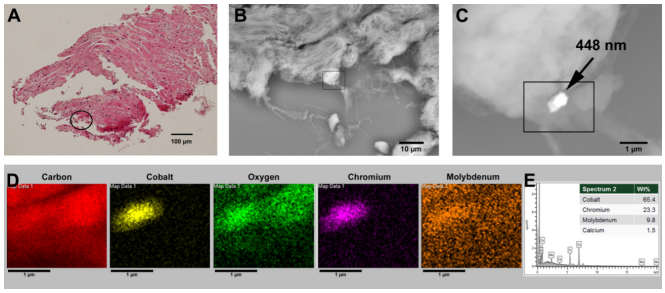

Figure 8 (A) Cardiac biopsy section adjacent to tissue samples shown in (B) and (C) (H&E, ×100). (B) Backscattered scanning electron microscope image of biopsy tissue (×1500). (C) Higher magnification of area outlined in B (×17 000) showing a 448 nm particle in the tissue. (D) EDS elemental maps of the area outlined in (C) demonstrates the particle to be CoCrMo alloy. (E) Quantitation of the corresponding EDS spectrum of the particle shows the elemental composition percentages of CO, Cr and Mo to be similar to the percentages seen in the implant alloy. EDS, energy-dispersive x-ray spectroscopy.

Tissue analysis was performed on joint pseudocapsule and heart biopsy tissues. Tissue samples were assessed via light microscopy for histopathological analysis of H&E-stained sections, SEM analysis (JEOL, IT500HR-LV) with energy-dispersive x-ray spectroscopy (EDS, Oxford Instruments) for localisation of wear debris within tissue and chemical mapping and quantification of implant metals, and Fourier transform infrared spectroscopy (FTIR) for chemical analysis of periprosthetic and cardiac tissue.[8]

RESULTS

Wear features on head and liner were consistent with edge loading (figure 3). SEM analysis revealed multiple grooves and scratches, indicating the acting wear mechanism was abrasion. Additionally, previously described mixed-coarse hard phases—characteristic for as-cast cobalt-chromium-molybdenum alloy—within the alloy had fractured from the surface (figure 4).[9] The total wear volume was 168.5 mm^3 (head: 46.3 mm^3, cup: 122.2 mm^3), resulting in a wear rate of 24.4 mm^3/year (head: 6.7 mm^3/year, cup: 17.7 mm^3/year).

The joint pseudocapsule tissue was dominated by a marked macrophage response (figure 5), with only mild to moderate perivascular infiltrate of lymphocytes and plasma cells. Diffuse lymphocytes and plasma cells were also mildly present. The macrophages throughout the tissue were laden with greyish particles. Formations of particle-induced granulomas with necrosis were also observed. The synovial surface consisted of fibrin exudates with some areas organising and containing dense particle-laden macrophages.

The SEM/EDS analysis of the pseudocapsule revealed large, mostly extracellular CoCrMo particles ranging in size from 1 µm to 7 µm (figure 6). Intracellularly, mostly chromium phosphate was observed as indicated by EDS mapping and confirmed by a strong characteristic phosphate ($\nu(PO_4^{3-})$: 1200–900/cm) absorbance peak in their infrared spectra.[10] FTIR univariate imaging of cardiac tissue demonstrated interstitial fibrosis and glycogen deposition (figure 7), which are hallmarks of cardiomyopathy pathology described by Mathlouthi and Koenig and Colagar *et al*.[11 12] Surprisingly, CoCrMo particles ranging in size from 0.16 μm to 15.9 μm were found within the heart tissue (figure 8). Eighteen particles were found within two sections of the same biopsy sample. No direct association of macrophages or other cell types with CoCrMo particles was observed.

DISCUSSION

This case demonstrates a rare, systemic complication of metal-on-metal hip replacement/resurfacing. Cobalt-induced cardiomyopathy secondary to metal-on-metal hip arthroplasty/resurfacing is an increasingly investigated complication in the literature, but most of these cases are associated with total hip arthroplasty

Table 1 Summary of select cases illustrating non-ischaemic cardiomyopathy in patients with metal-on-metal hip replacements

Case	Demographic	Patient presentation	Maximal cobalt (ng/mL)	Cardiac function	Intervention	Sequelae
1[14]	49-year-old man with MoM Articular Surface Replacement System (DePuy)	2 years after revision due to hip pain, tinnitus, neuropathy	83	Diastolic dysfunction noted on echocardiogram	Hip joint revision surgery	Not documented
2[15]	58-year-old rural woman with right MoM resurfacing system	10 years after arthroplasty with symptoms of heart failure	169	NYHA class III non-ischaemic dilated cardiomyopathy	Right hip revision with evidence of metallosis and osteolysis	Improved echocardiogram measures
3[21]	46-year-old Caucasian man with bilateral MoM THA (ZimmerBiomet)	3 years after arthroplasty with dyspnoea, ascites; evaluated for cobalt toxicity 6 years later	156	EF=20%	LVAD placement and then bilateral hip revision with evidence of metallosis. Excessive anteversion noted.	Recovered EF to 38%
4[22]	69-year-old woman with bilateral MoM THA (DePuy)	2 years after arthroplasty with hip pain	192	EF=10%–15%	Bilateral hip revision with evidence of metallosis	Death from haemorrhagic stroke 7 days post-revision
5[7]	54-year-old man with bilateral MoM THA (Biomet)	Symptoms of heart failure	189	Left ventricular dysfunction noted on echocardiogram and cardiac MRI	Revision of bilateral hips	Lack of improvement with permanent myocardial damage

EF, ejection fraction; LVAD, left ventricular assistance device; MoM, metal on metal; NYHA, New York Heart Association; THA, total hip arthroplasty.

instead of hip resurfacing.[6 13] Two prior cases of cardiomyopathy associated with hip resurfacing and elevated cobalt levels have been reported, and in both cases, cardiac function improved after revision arthroplasty.[14 15] In both instances, retrieval analysis demonstrated acetabular component edge loading. Neither of these previous cases provided a detailed analysis of the wear pattern or volume, and therefore quantitative comparisons were not possible. Our analysis revealed that the BHR had a malaligned acetabular component with excessive inclination and anteversion, which subsequently may have produced high wear due to edge loading. Based on normal wear rates determined by Koper *et al* (head: <1 mm^3/year, cup: <1 mm^3/year), our patient had 6× and 17× normal head and cup wear rates, respectively.[16] Gill and Pourzal *et al* have described that edge loading causes a shift in wear mechanism from mild surface fatigue to severe abrasion.[17 18] Under these abrasive conditions, larger wear particles (100 nm to 2 µm) are generated that maintain the alloy composition of CoCrMo, whereas during surface fatigue, smaller particles (<100 nm) mostly consisting of chromium oxide are generated.[19] Therefore, the finding of large particles within the periprosthetic tissue is consistent with edge loading. EDS and FTIR analysis further suggest that macrophages alter these particles chemically through phagocytosis, resulting in chromium–phosphate as the primary corrosion product. Interestingly, CoCrMo alloy particles were also found within cardiac tissue. The particles having the exact elemental composition as the alloy suggests that these particles were not yet phagocytosed by macrophages prior to transport to the heart. Larger CoCrMo particles are likely to corrode slowly due to protective surface passivation. However, in the capsular tissue, we observed that phagocytosis is associated with $CrPO_4$, which may also result in the release of ionic Co. While we observed numerous particles within the cardiac biopsy, we were not able to identify any interaction with resident macrophages. However, if particles within the heart were phagocytosed by macrophages, the result may be a high local concentration of Co which, coupled with high circulating levels of blood Co, may potentiate a cardiomyopathy.

A literature review of cardiomyopathy cases associated with metal-on-metal hip arthroplasty showed that some patients presented with hip symptoms, while others had isolated cardiomyopathy (table 1). Patients in these cases differed in the extent of cobalt toxicity, with cobalt levels ranging from 83 ng/mL to 192 ng/mL. While all patients in the cases underwent revision to non-metal-on-metal bearing reconstructions, outcomes varied with permanent myocardial damage and subsequent death in some cases but improved cardiac function in others. Some authors have argued that systemic complications typically occur at cobalt levels of >100 ng/mL and are most likely once concentrations reach 300 ng/mL.[6 20] However, given our patient had serum cobalt levels less than 300 ng/mL, it is evident blood cobalt levels must be considered with patient-specific risk factors, clinical presentation and analysis of periarticular and cardiac tissue.

Patient's perspective

My story started back in *-censored year-* when I had injured my finger doing construction work. My finger ultimately got infected and the infection spread through my blood to my hip, which destroyed all my cartilage. My wife, who worked in our local healthcare system, referred me to my orthopedic surgeon who gave me the option of hip resurfacing, while many others had no attractive solutions since I was so young. I was not able to even walk at this point and decided to proceed with the resurfacing surgery.

I honestly did not have any problems with my hip after the first surgery aside from some muscle tightness since I had not walked normally for 2 years before my hip resurfacing. I had no pain or mobility issues.

Eventually, I started having trouble with getting around and I could not even go up and down the stairs without shortness of breath. My wife knew that something serious was happening since she worked in the cardiology department. She got me set up with my cardiologist and things just started to escalate. When I was initially put on medications, I started to feel better. However, a couple months later, things became significantly worse. For a whole year, it was unclear what was happening. I was terrified because I was so young and did not know what was happening. I did not want to leave my young children so early in life. Not once during that year did I think that the issue was ultimately my hip.

At the beginning of *-censored year-* after being on IV heart medications and multiple cardiac catheterizations, did we consider there may be an issue with metal toxicity from my hip. When I had heard that the problem may be coming from my hip, I had gotten a feeling of relief; that I could be treated and feel better. I went into my revision surgery hopeful, despite how sick I was at that time. Two months after the surgery, I was getting back to normal. I was able to go on vacation with my family and even walk 15 miles in a day without much issue.

Fast forwarding to today, I am now able to do things without limitations; I am able to go biking with my kids and go hiking with my family. I have no issues with my hip and do not experience shortness of breath. In fact, my mobility with my hip is even better than before. I still have to take oral medications for my heart, but I continue to improve and am grateful.

There is nothing I think the medical providers could have done differently. It came down to getting the metal out of me and getting on the right medications. My biggest supporter was my wife throughout this process. I believe that my condition often goes underdiagnosed because many patients with metal hips are older and heart failure is common, therefore there is no consideration of heavy metal toxicity. I think this problem is more prominent than we think. I hope no one has to go through something like me and I hope this research provides clarification for other doctors and patients.

Learning points

- Cobalt toxicity-induced cardiomyopathy is a rare but grave complication following metal-on-metal total hip resurfacing.
- Hip arthroplasty component malpositioning may increase the risk of cobalt toxicity in the context of metal-on-metal bearing surfaces due to altered wear mechanics.
- Cellular responses and systemic transport of metal alloy particles likely play a critical role in cobalt-induced cardiotoxicity.
- Prompt consideration and diagnosis of a metal-induced cardiomyopathy in at-risk patients can allow for early chelation and revision surgery to non-metal-on-metal hip replacement components, ultimately preventing irreversible cardiac damage.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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