Case report

Arthroprosthetic cobaltism associated with cardiomyopathy

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A B S T R A C T
Systemic cobaltism related to metal-on-metal total hip arthroplasty has been published in case reports and series with effects on the cardiac, neurologic, endocrine, and immunologic systems. This case report presents a 46-year-old male who underwent bilateral metal-on-metal total hip arthroplasty and subsequently developed cardiomyopathy requiring left ventricular assist device implantation. Intervention with bilateral revision to non-cobalt-containing implants resulted in improved cardiac function. This case report will alert clinicians to the presentation of this rare but devastating complication while also displaying improvement following revision total hip arthroplasty. It is our hope this case will aid in early recognition and intervention of this condition.

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Introduction

Metal-on-metal (MoM) total hip arthroplasty (THA) implants were first described more than 70 years ago and were introduced in 1966 with the McKee-Farrar prosthesis [1,2]. As improvements were made in cobalt-molybdenum alloys, their popularity increased due to benefits related to the properties of the alloy. It was thought that these implants would have reduced volumetric wear, resulting in decreased osteolysis. MoM implants also offered increased femoral head sizes allowing for improved stability [3,4].

As these implants became widely used in the mid-1990s, national registry data reported a 2- to 3-fold increase in revision rate when compared to conventional bearing surfaces [5]. Contrary to the proposed promises of MoM implants, this increase in revision rate has since been attributed to volumetric wear between the metal bearing surfaces causing release of metallic particles and ions [6]. This release of particles creates macroscopic necrosis, osteolysis, sterile joint effusions, and pseudotumors in the surrounding soft tissue [7-9].

Individuals without abnormal cobalt exposure have a serum level <0.2 ppb whereas hypercobaltemia is defined as a level exceeding 1 ppb. A recent survey of 498 patients with MoM hip prostheses reported a serum cobalt >4 ppb in one-third [10]. Systemic concerns with cobalt alloy implants have been reported including cardiac dysfunction, neurologic sequelae (hand tremor, headache, cognitive decline, incoordination, depression visual disturbance, and optic nerve atrophy), thyroid dysfunction, and hematologic/immune disturbance [11-14]. The patient described in this case report had laboratory-proven hypercobaltemia with new-onset cardiomyopathy following bilateral THAs, which showed improvement following the removal of the MoM implants.

Case history

The patient is a 46-year-old Caucasian male who first presented at the age of 34 with bilateral (left worse than right) hip pain due to idiopathic avascular necrosis. He underwent left hip core decompression in 2004 (Table 1). His symptoms and radiographic findings of avascular necrosis progressed, and he underwent left THA with MoM implant in August of 2005 with a Biomet 12.5 lateral offset Taperloc femoral stem, size 54-mm cup, and a plus 9-mm length, 28-mm-diameter cobalt-chrome head (ZimmerBiomet, Inc., Warsaw, IN). Stability was noted to be excellent at resolution of the case and no complications were noted. One year later, he again underwent MoM THA on the right side (Fig. 1). The patient was doing well until December of 2007 when he was admitted to the cardiology service with dyspnea on exertion and increased
abdominal girth. He was subsequently diagnosed with idiopathic cardiomyopathy of unknown cause with left ventricular ejection fraction (LVEF) 20% on echocardiogram. Endomyocardial biopsy failed to show myocarditis or amyloidosis. The patient’s clinical function continued to deteriorate with multiple admissions secondary to congestive heart failure exacerbations. During an admission in 2013, he was evaluated for cobaltemia and found to have a level of 156 ppb (normal level <0.2 ppb). Chelation therapy was deferred at this time. He was admitted again 2 months later and underwent repeat cardiac biopsy which was negative for cobalt. Due to continued deterioration of his cardiac function (LVEF 25%-30%), the patient was identified as candidate for heart transplant vs left ventricular assist device (LVAD) implantation. The patient elected for the latter, and in May of 2013, an LVAD was implanted.

The patient presented to our clinic 1-year status after placement of his cardiac device as a referral from his cardiologist, who suspected that his cardiac dysfunction was secondary to prolonged cobaltemia. During his first visit, he endorsed he had had continued pain in both hips, left worse than right, for the past 6 years. He had not sought intervention as he had been able to live with the pain. Upon presentation, his cobalt level was 114 ppb and LVEF 25%. The patient was determined to be a good candidate for left revision THA to ceramic head on polyethylene liner components (Fig. 2). Preoperative chromium levels were ordered; however, the patient did not return to the laboratory before surgery to obtain these laboratory values. Intraoperatively, a large pseudotumor was noted just beneath the gluteus maximus extending posteriorly toward the sciatic nerve, which was debrided back to healthy tissue. The abductors were noted to be intact. Upon dislocation of the hip and removal of the femoral head component, there was significant metallosis within the joint as well as corrosion between the head and the taper. It was noted that the acetabular component was excessively anteverted and had more than 45° of inclination. Attention was then turned to the acetabulum as there was retroacetabular osteolysis noted on preoperative radiographs. After explantation of the acetabular component, large cavitory acetabular defects and large areas of metallosis were noted. Metallic debris was curetted back to healthy bone, defects filled with bone graft, and a trabecular metal acetabular component was fixed with screws. The patient previously had a plus 9-mm head in place. This length was restored with plus 6-mm head and a plus 3.5-mm liner. The hip was noted to be stable in all standard tested positions with final components in. At 2-week follow-up, the patient was doing well with well-fixed components and his serum cobalt level had decreased to 27.2 ppb.

The following 6 months the patient had no hospitalizations and serum cobalt measurements continued to downtrend. He had no pain in his left hip at 2-month follow-up. Due to ongoing right hip pain and good result with the contralateral side, the patient decided to undergo right revision THA in September of 2014 to ceramic head with polyethylene liner (Fig. 3). Significant metallosis was noted again beneath the fascia lata and within the hip joint, although no pseudotumor was evident on this side.

During the following 18 months, the patient remained pain free and had no complications with respect to his hardware. His serum
cobalt level decreased to 1.5 ppb, and he recovered cardiac function as illustrated by his most recent LVEF of 38%. He has experienced continued dilatation of the right ventricle, however. His cardiologists are closely monitoring for return of cardiac function with the goal of explanting his LVAD when safe.

**Discussion**

This case presents an example of cardiomyopathy associated with systemic hypercobaltism after bilateral MoM THAs. Although causation is suspected, our patient’s cardiac biopsy was improperly processed for presence of cobalt. Positive biopsy is not a requirement for diagnosis, although other case reports of cardiomyopathy associated with occupational [15] and prosthetic cobalt exposure [16] have shown positive cobalt deposition in the myocardium. In our patient’s case, the patient had a precipitous decline in serum cobalt levels and significant improvement in cardiac function following his revision operations, supporting the hypothesis that his cardiomyopathy was associated with elevated serum ion levels. The acetabular component was placed in excessive anteversion and inclination during his primary THA on the left, which is likely to have contributed to his excessive wear pattern and increased cobalt levels. It is possible this patient could have avoided such a grave decline in cardiac function if his condition had been recognized and/or intervened upon earlier in its course. Guidelines released by the FDA in early 2013 to address MoM implants would have indicated investigation of his hips with advanced imaging, such as magnetic resonance imaging with metal artifact reduction, due to his increased ion levels in addition to having continued pain in his left hip at the time. Imaging at this time may have detected his large pseudotumor and have led to earlier intervention with revision arthroplasty [17].

**Systemic cobaltism following MoM THA has been published in previous case reports and series [12,18-25].** A systematic review of 25 patients identified several organ systems involved with the cardiovascular system (60%) being the most frequently affected, followed by central nervous system. The mean time to presentation or revision in this population was 41 months, and the mean serum cobalt level 324 ppb, although they reported symptoms in patients with levels as low as 20 ppb [11].

The aim of this case report is to alert physicians to the association of cardiomyopathy with cobalt MoM total hip implants and raise awareness of this rare yet devastating complication. Frequent monitoring of patients with MoM implants and cardiac dysfunction should be performed to ensure prompt intervention. Patients with renal impairment are considered at increased risk of hypercobaltism-associated cardiomyopathy due to reduced cobalt clearance [11]. There is also an observed dose-response relationship between serum cobalt level and severity of symptoms, which may make this a good screening tool with levels >7 ppb, warranting observation of cardiac and neurologic function and levels >20 ppb as an indication for revision surgery to alternative bearing surfaces [11,26].

**Table 1**

| Timeline of events. |
|---------------------|
| **March 2005**      |
| Left THA with MoM implant |
| **2006**            |
| Right THA with MoM implant |
| **2007**            |
| Hospital admission with diagnosis of idiopathic cardiomyopathy, LVEF 20% |
| **January 2013**    |
| Hospital admission for congestive heart failure (CHF) exacerbation. Cobalt level first measured at 156 ppb |
| **May 2013**        |
| Left ventricular assistive device implanted |
| **March 2014**      |
| Left revision THA to ceramic on polyethylene. Cobalt level 114 ppb; LVEF 25% |
| **April 2014**      |
| Cobalt level 27.2 ppb |
| **September 2014**  |
| Right revision THA to ceramic on polyethylene. |
| **March 2015**      |
| LVEF 38% |
| **June 2015**       |
| Cobalt level 1.5 ppb |
Summary

Arthroprosthetic cobaltism is a rare but devastating known consequence of MoM THA. It is both locally and systemically harmful causing osteolysis of the effective joint space along with the potential for systemic complications including the cardiac, neurologic, endocrine, and hematologic systems, as well as others. The possibility of this diagnosis should always be in the forefront of clinicians’ minds when confronted with a patient with MoM THA and systemic symptoms that cannot be attributed to other more common systemic diseases. This case displays the devastating cardiomyopathy that can occur as a sequelae and the potential recovery that can be made after explant of these implants.

References

[1] McKee GK, Watson-Farrar J. Replacement of arthritic hips by the McKee-Farrar prosthesis. J Bone Joint Surg Br 1966;48(2):245.
[2] Scales JT. Arthroplasty of the hip using foreign materials: a history. Symposium on lubrication and wear in living artificial human joints. London: Institution of Mechanical Engineering; 1967. p. 63.
[3] Fisher J, Jin Z, Tipper J, Stone M, Ingham E. Tribology of alternative bearings. Clin Orthop Relat Res 2006;453:25.
[4] Smith TM, Berend KR, Lombardi Jr AV, Emerson Jr RH, Mallory TH. Metal-on-metal total hip arthroplasty with large heads may prevent early dislocation. Clin Orthop Relat Res 2005;441(441):137.
[5] Bolognesi MP, Ledford CK. Metal-on-metal total hip arthroplasty: patient evaluation and treatment. J Am Acad Orthop Surg 2015;23:724.
[6] Macdonald SJ, McCalden RW, Chess DG, et al. Metal-on-metal versus polyethylene in hip arthroplasty: a randomized clinical trial. Clin Orthop Relat Res 2005;406:282.
[7] Ralow A, Schoon J, Dienele A, et al. Influence of particulate and dissociated metal-on-metal hip endoprosthesis wear on mesenchymal stromal cells in vivo and in vitro. Biomaterials 2016;58:31.
[8] Pandit H, Glv-Jones S, McLardy-Smith P, et al. Pseudotumours associated with metal-on-metal hip resurfacings. J Bone Joint Surg Br 2008;90(7):847.
[9] Rona G, Chappel CI. Pathogenesis and pathology of cobalt cardiomyopathy. Recent Adv Stud Cardiac Struct Metab 1973;2:407.
[10] Van Der Straeten C, Van Quickenborne D, Pennynck S, et al. Systemic toxicity of metal ions in a metal-on-metal hip arthroplasty population. Bone Joint J 2013;95-B(suppl 34):E17.
[11] Gessner BD, Steck T, Woeber E, Tower SS. A systematic review of systemic cobaltism after wear or corrosion of chrome-cobalt hip implants. J Patient Saf 2015 [Epub ahead of print].
[12] Tower SS. Arthroprosthetic cobaltism: neurologic and cardiac manifestations in two patients with metal-on-metal arthroplasty. A case report. J Bone Joint Surg Am 2010;92(17):2847.
[13] Catalani S, Rizzetti MC, Padovani A, et al. Neurotoxicity of cobalt. Hum Exp Toxicol 2012;31:421.
[14] Gill HS, Grammatopoulos G, Adshead S, Tsialogianni E, Tsiridis E. Molecular and immune toxicity of CoCr nanoparticles in MoM hip arthroplasty. Trends Mol Med 2012;18(3):145.
[15] Barbiorik M, Dusek J. Cardiomyopathy accompanying industrial cobalt exposure: case report. Br Heart J 1972;34:113.
[16] Martin JR, Spencer-Gardner L, Camp CL, Stulak JM, Sierra RJ. Cardiac cobaltism: a rare complication after bilateral metal-on-metal total hip arthroplasty. Arthroplasty Today 2015;1(4):95.
[17] Metal-on-metal hip implants: FDA safety communication. Fda.gov; 2013. http://www.fda.gov/MedicalDevices/Safety/AlertsandNotices/ucm335775.htm [accessed 31.08.16].
[18] Cheung AC, Banerjee S, Chervian JJ, et al. Systemic cobalt toxicity from total hip arthroplasties: review of a rare condition part 1 – history, mechanism, measurements, and pathophysiology. Bone Joint J 2016;98-B(1):6.
[19] Mosier BA, Maynard L, Sotereanos NG, Sewecke JJ. Progressive cardiomyopathy in a patient with elevated cobalt ion levels and bilateral metal-on-metal hip arthroplasties. Am J Orthop 2016;45(3):E132.
[20] Oldenburg M, Wegner R, Baur X. Severe cobalt intoxication due to prosthetic wear in repeated total hip arthroplasty. J Arthroplasty 2009;24:825.e15.
[21] Roessler PP, Witt F, Efe T, Schmitt J. Arthroprosthetic cobaltism and pseudotumour also occur in patients with small diameter femoral ball head metal-on-metal hip arthroplasty. BMJ Case Rep 2014;2014.
[22] Sotos JC, Tower SS. Systemic disease after hip replacement: aeromedical implications of arthroprosthetic cobaltism. Aviat Space Environ Med 2013;84(3):242.
[23] Tower SS. Arthroprosthetic cobaltism associated with metal on metal hip implants. BMJ 2012;344:e430.
[24] Machado C, Appelbe A, Wood R. Arthroprosthetic cobaltism and cardiomyopathy. Heart Lung Circ 2012;21(11):759.
[25] Zywiel MG, Cherian JJ, Banerjee S, et al. Systemic cobalt toxicity from total hip arthroplasties: review of a rare condition part 2. Measurement, risk factors, and step-wise approach to treatment. Bone Joint J 2016;98-B(1):14.
[26] Tower SS. Arthroprosthetic cobaltism: identification of the at-risk patient. Alaska Med 2010;52:28.