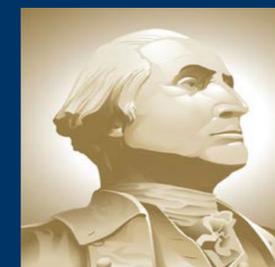




Cobalt, Cardiomyopathy, and Chelation: A Fatal Case of Cobalt Metallosis in a Patient with a Metal-on-Metal Hip Implant

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Introduction

- Metal-on-metal (MoM) joint prostheses can deteriorate over time, releasing free cobalt ions into the blood stream. In rare instances, systemic cobalt toxicity may result.
- Systemic symptoms attributed to cobalt toxicity include cardiomyopathy, hypothyroidism, impaired renal function, and neuro-psychological changes.
- In otherwise healthy people, blood cobalt levels < 300 mcg/L are not associated with symptomatic illness.
- We present a fatal case of a 63 year-old-man with a MoM hip implant who developed systemic illness consistent with cobalt toxicity and a peak serum cobalt concentration of 202 mcg/L.



<http://www.nih.gov/news/health/dec2011/niams-22.htm>

Case Report

A 63-year-old man underwent a total hip arthroplasty for degenerative joint disease, receiving a MoM prosthesis. Three years later, the patient developed progressive shortness of breath and exercise intolerance. After an extensive work up (including cardiac catheterization and biopsy), he was diagnosed with non-ischemic cardiomyopathy, with an ejection fraction (EF) of 20%.

Later the same year, he presented to the hospital with decompensated heart failure (EF 15%). Serum cobalt concentrations were obtained. The peak serum cobalt concentration was 202 mcg/L (0.1-2 mcg/L), and was felt to be contributory to the development of the patient's cardiomyopathy. Orthopedic surgery was consulted for removal of the prosthesis, but the patient had developed renal failure (serum creatinine, 2.0 mg/dL) and *S. aureus* sepsis and was not stable for surgery.

Chelation therapy with succimer was administered for two weeks. N-acetylcysteine (21 hours intravenous infusion) and thiamine were also administered. After completion of chelation therapy, the measured serum cobalt concentration decreased to 11 mcg/L.

The patient developed an arterial thrombus requiring surgical removal and fasciotomy. Due to refractory hemodynamic instability, the patient was placed on extracorporeal membrane oxygenation (ECMO) and a bi-ventricular assist device was placed; however, patient expired one month after admission to the hospital.

Limitations/Discussion

- This single case should not be generalized to the population of patients who have received MoM prosthesis.
- The optimal management strategy for patients with MoM prosthesis has yet to be determined.
- Current treatment of MoM prosthesis-induced cobalt toxicity consists of supportive care, removal of the deteriorating device, and in rare instances, chelation.
- Indications for chelation are end-organ toxic effects such as acidosis and cardiac failure.
- Management should be individualized based on the clinical status of the patient.

Conclusions

- Despite a relatively low serum cobalt concentration, our case demonstrates a catastrophic outcome associated with a MoM prosthesis.
- Chelation therapy, while useful for reducing total body burden of cobalt, may not lead to clinical improvement or reduction in mortality, as the damage resulting from cobalt exposure may be irreversible.

The authors do not have any conflicts of interest to disclose.

