

CASE REPORT

# A 61-year-old woman develops polyuria after hip revision

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## Abstract

A 61-year-old previously healthy woman developed progressive hearing and visual loss over a period of 2–3 months prior to admission. Her medical/surgical history was remarkable for a left hip arthroplasty 11 years ago requiring revision approximately 6 months prior to admission. After dislocating the revised hip, she re-presented to her surgeon and underwent a closed reduction. Several weeks following the reduction, the patient began having polyuria and symptoms of hearing and vision loss along with numbness in her extremities and abdominal region.

## INTRODUCTION

A 61-year-old previously healthy woman underwent a left hip arthroplasty 11 years ago requiring revision approximately 6 months prior to admission. After dislocating the revised hip, she re-presented to her surgeon and underwent a closed reduction. Over 8–10 weeks prior to admission, she began complaining of blurred vision, difficulty hearing and numbness in her extremities. A serum cobalt level was obtained and was found to be grossly elevated. She was admitted to the hospital for explant of her hip. During admission, she developed polyuria, dehydration and lactic acidosis.

## CASE REPORT

A 61-year old woman with a surgical history significant for a left hip metal-on-metal arthroplasty in 2004 presented to her

orthopedic surgeon in May 2015 after experiencing recurrent spontaneous subluxations. A hip revision was performed in which the original Stryker polyethylene acetabular cup (4.5 × 4.5 × 2.5 cm, 625-OT-32E, Size E) and Stryker cobalt femoral head component (3.3 × 3.3 × 2.8 cm, 04-102234 A1203) were noted to be shattered and were replaced. The patient tolerated the procedure well and had an uneventful postoperative clinic visit 2 weeks later.

Subsequently, the patient dislocated her left hip while participating in physical therapy as an outpatient. She underwent a left hip closed reduction under general anesthesia for this acute posterior dislocation. About 2 months after the operative reduction, the patient presented to her orthopedic surgeon with new-onset complaints of difficulty sleeping, decreased appetite and post-prandial nausea and vomiting. She additionally reported an unintentional 30-lb weight loss since surgery. Her symptoms were originally attributed to her known history

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Figure 1: Preoperative left hip x-ray performed prior to explant and debridement. Soft tissue densities around previous hip implant.

of anxiety and depression, and she was started on medication for these conditions. However, the patient's symptoms escalated until she was reporting blurred vision, hearing loss and abdominal numbness. The patient was referred to an ophthalmologist who suspected that the patient may be suffering from cobalt toxicity from the hip implant. Serum cobalt levels were drawn and were found to be grossly elevated (2128 mcg/l, normal range <0.9 mcg/l).

A left hip X-ray was performed preoperatively and showed myriad soft tissue densities (Fig. 1). These were suspected to be ceramic debris from her original hip implant. It was suspected that the residual ceramic debris from her first hip implant had ground into the lining of the replacement cobalt-based femoral head, causing cobalt to be released into the bloodstream. The patient was transferred to our institution for a planned resection arthroplasty and further management of her cobalt toxicity. A CT scan was performed at our institution, which confirmed extensive local tissue reaction (8.7 × 3.5 × 25 cm) around the left hip prosthesis (Fig. 2).

On arrival, the patient had a serum cobalt level of 1254 mcg/l (normal: 0–0.9 mcg/l). Both renal replacement therapy and then plasmapheresis were attempted as initial maneuvers to decrease the serum cobalt levels. However, a definitive and continued reduction of the patient's serum cobalt level was only obtained once the cobalt-bearing hip implant was removed on hospital Day 6. During the revisional arthroplasty, the original ceramic-based acetabular was noted to be in minimal anteversion and vertical position. The removed specimen consisted of an intact metallic ball of 35 mm in diameter and a metal and plastic socket with an outer diameter of 55 mm and

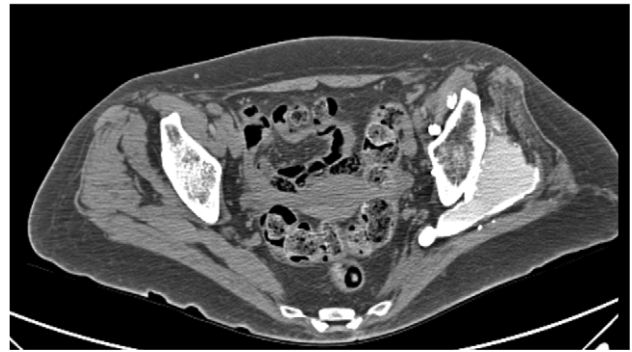


Figure 2: Preoperative left hip x-ray performed prior to explant and debridement. Image shows extensive local tissue reaction (Hounsfield units 167, 8.7 × 3.5 cm in the axial plane with involvement extending 25 cm in craniocaudal dimension) around the left hip prosthesis. There is significant replacement of the left gluteus medius muscle posteriorly with the abnormal soft tissue tracking inferiorly in the trochanteric bursa and fascial planes deep to the iliobial tract.



Figure 3: POD 7 X-ray.

an inner diameter of 35 mm. Two metallic screws compromised the remainder of explant. The previous implant was replaced with a Stryker Trident PSL acetabular component (50 mm with 36 mm polyethylene liner) and a femoral head component (Stryker Cobalt chrome, 36 mm) with a titanium adapter sleeve with Delta ceramic material. Patient was sitting upright by postoperative Day 2 (POD 2) and walking with assistance with physical therapy by POD 4 (Fig. 3). Of note, over a 3-day period prior to her hip explant, she developed a progressive lactic acidosis due to hypovolemia and required massive crystalloid repletion to maintain euvoolemia.

On hospital Day 6 (POD 0–1), the patient returned from her revisional arthroplasty appearing clinically volume-depleted with a lactic acidosis and required vasopressors to maintain a mean arterial pressure  $\geq 60$  mm Hg. Her urine output was approximately 400–500 ml/hour and over the next 2 days went as high as 800–1000 ml/hour. Urine and serum osmolalities were sent on POD 2 and were 202 mOsmol/kg (normal range 300–900 mOsmol/kg) and 288 mOsmol/kg (normal range 275–295 mOsmol/kg), respectively. A presumptive diagnosis of partial central diabetes insipidus (DI) was suspected, and the patient was treated initially with intranasal and then intravenous desmopressin (DDAVP). Within 1–2 hours of administration, her urine output decreased to approximately 100 ml/hour and her urine osmolality increased to 329 mOsmol/kg. Her volume status stabilized enabling a rapid wean in vasopressors.

The patient was successfully discharged to a rehabilitation center 2 weeks after her initial admission. On follow-up visits, she has not experienced further subluxations and is able to ambulate without difficulty. Her visual and auditory losses are still significant although she is showing a slight improvement in these senses and continues to be followed closely as an outpatient.

## DISCUSSION

Cobalt toxicity is a rare but increasingly recognized complication of prosthetic hip implantation [1]. Signs and symptoms of the entity known as prosthetic hip-associated cobalt toxicity (PHACT) include cardiac and neurologic dysfunction [2]. High clinical suspicion is necessary to make the diagnosis and can be confirmed with serum cobalt testing. The exact mechanism of cobalt neurotoxicity is as yet unknown. Suggested hypotheses include disruption of mitochondrial oxidative phosphorylation, neurotransmitter modulation and direct neuron cytotoxicity [3–6].

Although PHACT has been widely reported in the literature, we believe our experience is the first to report recognition and successful treatment of central DI in a patient with this condition.

DI results from either a deficiency in antidiuretic hormone (ADH) secretion (central) or a tubular resistance to the effects of ADH (nephrogenic), both of which result in the production of a large volume of dilute urine. *In vitro* data suggest that the pro-inflammatory cytokine interleukin 2 (IL-2) is part of the pathway of ADH release from the hypothalamus [7–8]. *In vivo* studies in female mice have suggested cobalt–chromium particles antagonize the effects of IL-2 [8]. This antagonism of IL-2 may in turn impair the release of ADH. In the case of our patient,

we suspect her high serum cobalt levels impaired ADH secretion and contributed to her development of central DI [9]. Synthetic vasopressin, in the form of DDAVP, aided in the patient's recovery from this condition and helped her regain normal urine output and urine osmolality.

Metallosis from prosthetic hip implantation is rare. Understanding the manifestations of cobalt toxicity is key to establishing the diagnosis in a timely manner. While lithium-induced nephrogenic DI is well described [10], we report the first case of cobalt-induced central DI.

## CONFLICT OF INTEREST STATEMENT

None declared.

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