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Intracranial hypertension associated with arthroprosthetic cobaltism?

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ABSTRACT

Purpose: The purpose of this case report was to detail a unique patient with proven systemic cobaltism from metal-on-metal prosthetic hip articulation who then presented with clinical and radiographic signs of increased intracranial pressure.

Observations: A 59-year-old man with a past medical history of degenerative joint disease of the hip that status post total hip arthroplasty with cobalt-chromium implant presented with clinical and radiographic signs of increased intracranial hypertension. He underwent a revision arthroplasty with local debridement and removal of the cobalt-chromium implant and his serum cobalt level was elevated at 0.9 microg/L (normal range 0.1–0.4microg/L). One year after removal of the implant, the patient was asymptomatic and stable on acetazolamide.

Conclusions: To our knowledge, this is the first such report in the English literature to associate increased intracranial hypertension with systemic cobaltism, and publication of this case report would make clinicians aware of the potential neurologic and neuro-ophthalmic presentation of metal-on-metal orthopedic prosthetics.

1. Introduction

Metal-on-metal (MoM) total hip arthroplasty was initially introduced to decrease orthopedic volumetric wear, osteolysis, aseptic loosening, and other wear related failure. However, the use of MoM THA has declined due to multiple complications secondary to the unintentional release of metallic particles and ions generated by MoM articulations.[1]

Arthroprosthetic cobaltism (APC) encompasses both local and systemic complications that can occur from the generation of cobalt and chromium metal ions during MoM joint articulation. Locally, the release of metallic particles can induce necrosis, osteolysis, sterile joint effusions, and soft tissue pseudotumor formation. Reported systemic complications include cardiac, thyroid, hematologic, immunologic, and neurologic disorders (e.g., visual disturbances, optic nerve atrophy, cognitive decline, tremor, headache).[1,2]

Although visual disturbances have been reported with APC previously, to our knowledge this is the first case of APC associated intracranial hypertension in the English language, ophthalmic literature.

2. Case history

A 59-year-old man was referred for bilateral optic disc edema and nonspecific headaches for the past few weeks. Body mass index (BMI) was 45 kg/m^2. The patient had degenerative joint disease of the hip that required total hip arthroplasty with cobalt-chromium implant 10 years prior. This implant was subsequently recalled by the manufacturer. The patient then presented with worsening right hip pain, stiffness, and weakness. He had reduced right quadriceps function and was diagnosed with hip arthroplasty loosening with the formation of localized inflammatory pseudotumor in his right prosthetic hip. He underwent a revision arthroplasty with local debridement and removal of the cobalt-chromium implant. Histopathology showed fibroconnective tissue with focal hemorrhage, macrophages, and chronic inflammation with focal multinucleated giant cells. Serum cobalt level was elevated at 0.9 microg/L (normal range 0.1-0.4microg/L), with normal serum chromium level. Levels of cobalt in the cerebrospinal fluid was not measured.

Neuro ophthalmic examination revealed visual acuity of 20/20 in

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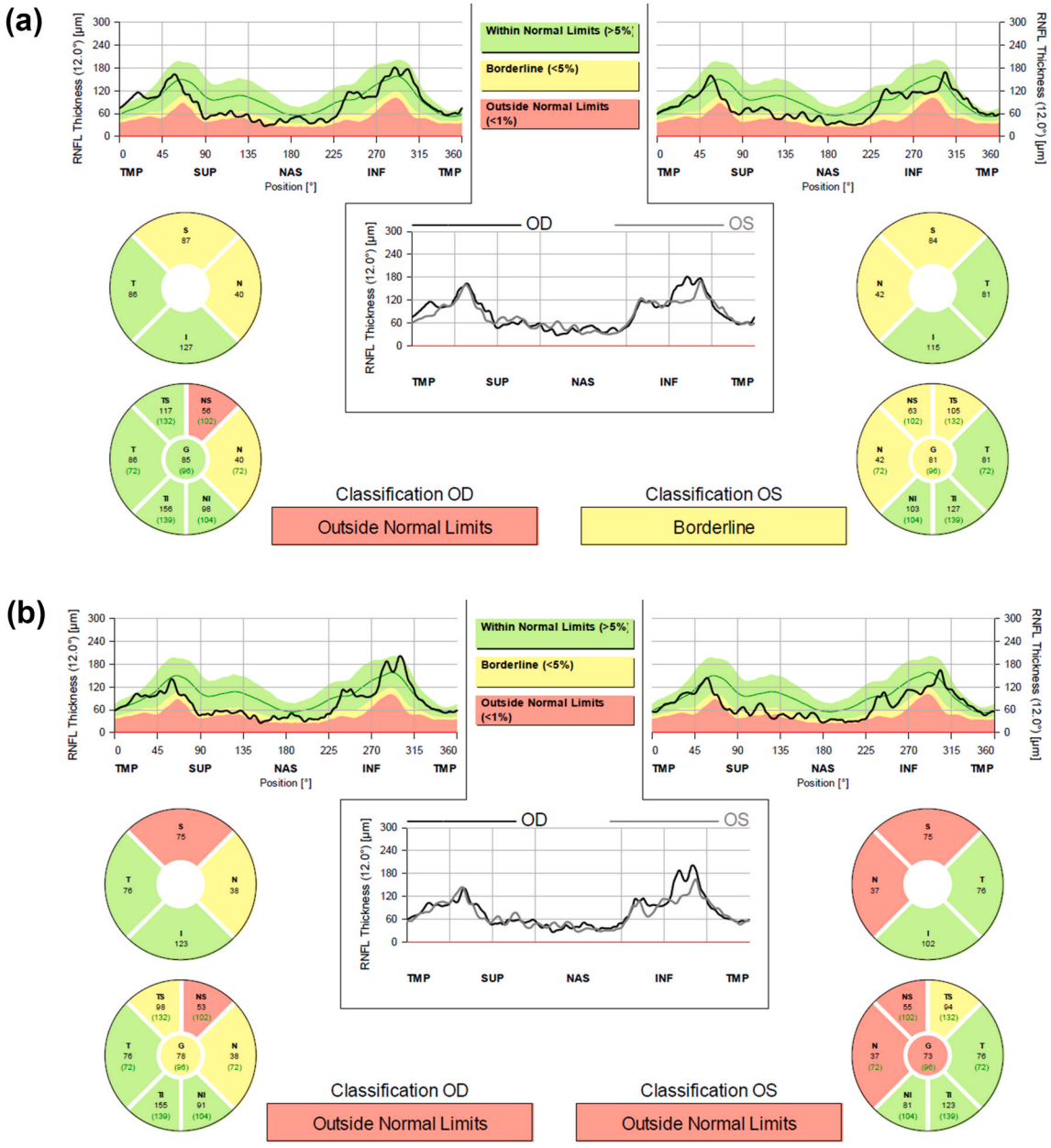

Fig. 1. Optical Computed Tomography (OCT). (a) OCT showed average retinal nerve fiber layer thickness of 90 µm in the right eye (OD) and 85 µm in the left eye (OS) at the first visit and (b) 78 µm OD and 73 µm OS consistent with mild optic nerve atrophy at the second visit.

both eyes (OU). His pupils were isocoric without a relative afferent pupillary defect. External and slit lamp examinations were normal. Intraocular pressure measurements were 16 mm Hg OU. Dilated fundus examination showed nasal elevation of the optic disc margins OU (Frisen grade 1 optic disc edema). Humphrey visual fields 24-2 were normal, with a mean deviation of −1.72dB OD and −1.44dB OS. Optical coherence tomography (OCT) showed average retinal nerve fiber layer thickness (RNFL) of 90 µm in the right eye (OD) and 85 µm in the left eye (OS) (Fig. 1a). Magnetic resonance imaging (MRI) of the head and orbits showed increased fluid around the optic nerve sheaths OU (Fig. 2). Orbital ultrasound also confirmed fluid in the optic nerve sheaths and a positive 30-degree test. Lumbar puncture revealed normal cerebrospinal fluid content and a borderline elevated opening pressure of 22 cm H_2O. The patient was commenced on acetazolamide 500 mg twice daily for a presumed diagnosis of idiopathic intracranial hypertension. Subsequent workup with sleep study was diagnostic for obstructive sleep apnea (OSA).

One year after removal of the MoM implant, the patient was asymptomatic, had lost more than 30 pounds, and was stable on acetazolamide. Visual acuity remained 20/20 OU. Repeat OCT showed mild RNFL thickness loss to 78 µm OD and 73 µm OS consistent with mild optic nerve atrophy (Fig. 1b). Humphrey visual field 24-2 showed a normal visual field with a mean deviation of −1.39dB OD and −1.22dB OS.

3. Discussion

Idiopathic intracranial hypertension (IIH) is a relatively common cause of optic disc edema and predominantly affects young, obese females. The diagnosis of IIH is established by the modified Dandy criteria and requires negative neuroimaging and normal CSF contents with an elevated intracranial pressure. Our case is unique because the clinical and radiographic features of intracranial hypertension occurred in the setting of metal-on-metal (MoM) arthroprosthetic cobaltism (APC) that

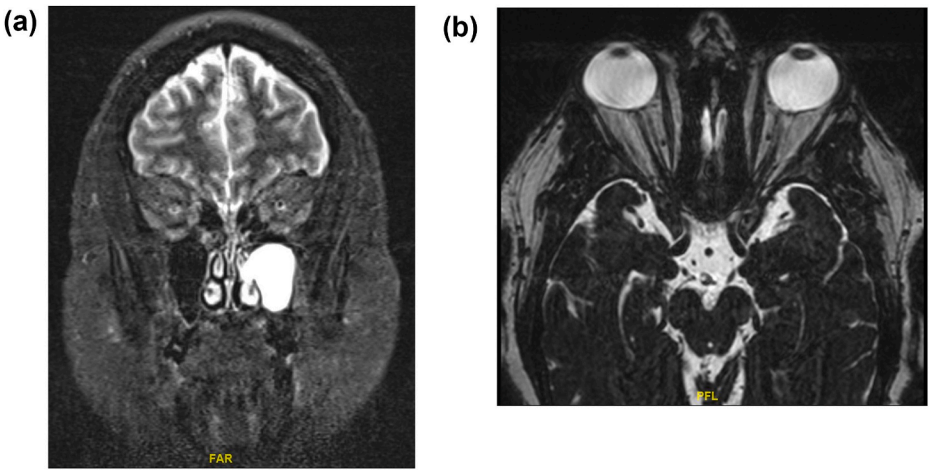

Fig. 2. Magnetic Resonance Imaging of the orbits. (a) Coronal Short-TI Inversion Recovery (STIR) sequence and (b) axial T2 constructive interference in steady state (CISS) shows increased fluid around the optic nerve sheath, suggestive of increased intracranial pressure.

required explanation. Systemic cobaltism from MoM articulations has previously been associated with cardiac, thyroid, hematologic, immunologic, and neurologic dysfunction but intracranial hypertension has not yet been reported.[1,2] The only neuro-ophthalmic symptoms that have been reported are visual impairment and optic atrophy.[1,2] The previously observed cobalt levels needed for systemic toxicity was at or below the level observed in our patient.

Our patient demonstrated both residual increased blood cobalt levels and signs of local metallosis after the removal of the implant. The patient, however, had predisposing risk factors for IIH including increased BMI and OSA.

APC can cause systemic and local symptoms through multiple, ill-defined mechanisms including heavy metal toxicity, reduced cellular viability, increased DNA damage, increased apoptosis, dysregulated cell mediated immunity, and deranged metabolism.[1–4]

Currently, the only definitive treatment of APC is the revision and removal of the MoM implant to eliminate the source of the metal ions. Studies have shown that the decrease of cobalt and chromium levels in the blood after MoM hip revision follows an exponential decay curve with a half-life of approximately 50 days. However, elevated levels of blood metal ions, as in our case, can persist for years after revision, especially in patients with high levels of initial exposure.[5]

In our patient, his cobalt levels drawn 18 months after his MoM hip revision were still elevated, suggesting even higher cobalt levels prior to surgical treatment.[5] The cause of IIH remains idiopathic and elusive. Whether APC in our case was causal or coincidental cannot be established from this case report, but to our knowledge, this is the first such report in the English language ophthalmic literature. Further study may be appropriate to determine the role of systemic cobaltism from APC in cases of atypical IIH. Clinicians should be aware of the potential neurologic and neuro-ophthalmic presentations of MoM orthopedic implants.

4. Patient consent

Consent to publish this case report has been obtained from the patient in writing. This report does not contain any personal identifying information.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Declaration of competing interest

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