CASE REPORT

Optic Neuropathy from Cobalt Toxicity in a Patient who Ingested Cattle Magnets

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ABSTRACT

Cobalt is a widely used in the industrial production of hard metals. Cobalt ingestion has been reported to cause widespread systemic toxicity, but its effects on vision have been sparsely reported. The authors report the case of a patient who ingested cattle magnets, which remained in his stomach for an unknown duration of time. These magnets largely consist of cobalt that gradually leached into his blood stream, resulting in protean systemic manifestations, which included optic atrophy.

KEYWORDS: cobalt; optic neuropathy; toxic

INTRODUCTION

Cobalt is a metal that has been known, if ingested, to produce a reticulocytosis and other adverse reactions, which include gastrointestinal disturbance, nerve damage, renal dysfunction, hypothyroidism, cardiomyopathy, and pulmonary fibrosis.¹ There have been two published case reports of cobalt induced optic atrophy—Licht et al.² described the case of a 32-year-old Jewish male with pancytopenia and a hypercellular bone marrow who was treated with four courses of cobalt chloride over the span of 3 years with a total dose of 73 g of cobalt chloride and subsequently developed decreased vision in both eyes (OU). His dilated funduscopic examination (DFE) revealed bilateral optic disc pallor and fluorescein angiography demonstrated irregular choroidal perfusion around the optic nerve head. Following the cessation of therapy, there was no further deterioration in visual function, supporting the toxic effect of cobalt therapy. The second report published by Meecham and Humphrey¹ is the case of a 48-year-old male who developed optic atrophy and bilateral deafness with tinnitus following industrial exposure to raw cobalt powder for 20 months. His DFE showed temporal optic disc pallor

and blood levels of cobalt were 234 µg/L (normal 0.5–3.9 µg/L). We report a very unusual case of optic neuropathy following the accidental ingestion of metal objects containing cobalt in a teenager.

CASE REPORT

A 16-year-old male was in his usual state of health until October 2008 when he developed generalised malaise. Evaluation by his paediatrician revealed hypothyroidism with a noticeable goitre, tachycardia, and polycythaemia. Despite evaluation by endocrinology, cardiology, and haematology, this constellation of symptoms could not be explained. In the initial work-up, the patient underwent routine vision screening by his local optometrist where his vision was noted to be 20/40–3 in the right eye (OD) and 20/60–2 in the left eye (OS) best corrected with a normal slit-lamp examination (SLE) and DFE. He was referred to a local ophthalmologist who performed Humphrey Visual Field testing, which revealed a central defect and early nasal step OD and generalised central depression OS. Neuroimaging was requested to investigate the possibility of a structural defect but the magnetic resonance imaging scan was terminated because the patient suffered an episode of acute abdominal pain during the study. Abdominal plain films were obtained, which showed two 9-cm magnets in his stomach (Figure 1). The radiologist reported that these magnets were

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pitted and corroded, indicating that they were likely to have been present in the patient's stomach for a prolonged period of time, although the patient denied any recollection of having swallowed them. The patient was subsequently admitted to hospital for foreign body removal in February 2009. A 24-hour urine collection ordered post-surgery yielded cobalt $473.2 \mu\text{g/L}$ (normal $0.1\text{--}2.2 \mu\text{g/L}$), nickel $73.8 \mu\text{g/L}$ (normal $0\text{--}5.2 \mu\text{g/L}$), and aluminum $10 \mu\text{g/L}$ ($0\text{--}7 \mu\text{g/L}$), and a blood cobalt level yielded $112.4 \mu\text{g/L}$. The patient received 21-hour *N*-acetylcysteine (NAC) chelation therapy on the recommendation of the regional poison control centre. Upon completion of the chelation therapy protocol, the patient was subsequently discharged and seen in the ophthalmology clinic 5 days later.

On examination, visual acuity was measured as 20/50 + 2 OD and 20/100 – 1 OS and colour vision was 7/11 OD and 8/11 OS by Ishihara pseudoisochromatic colour plate testing. DFE showed temporal disc pallor OU (Figures 2, 3). Intraocular pressure was normal at 16 and 14 mm Hg in the right and left eyes, respectively, by Goldmann applanation, and the remainder of the SLE was unremarkable. An optical coherence tomography scan was obtained, which did not demonstrate any macular pathology. Electrodiagnostic testing including electroretinogram (ERG), electrooculogram (EOG), and visual evoked responses (VERs) were ordered to help distinguish between retinal and optic nerve disease. The patient was referred to neuro-ophthalmology

in March 2009, at which time visual acuity was unchanged, although colour vision had improved to 10/11 OD and 11/11 OS. Temporal disc pallor was once again appreciated in both eyes. The patient's subsequent visit to the neuro-ophthalmology clinic 2 months later demonstrated greatly improved visual acuity at 20/25 + 3 OD and 20/25 – 1 OS with colour vision 10/10 OU. Goldmann Visual Fields were performed, which showed bilateral nasal steps (Figure 4). VER showed a delayed p100 wave at 137.3 ms OS and 114.7 ms OD, consistent with optic neuropathy OU. Also notable was a low Arden ratio of the EOG at 1.2 OU (normal >1.80), indicating dysfunction at the interface of the retinal pigment epithelium.

DISCUSSION

The objects found in the patient's stomach were determined to be cattle magnets; cattle magnets protect these animals from metal objects that are inadvertently

FIGURE 1 Abdominal plain film showing cattle magnets.

FIGURE 2 Temporal pallor OD.

FIGURE 3 Temporal pallor OS.

FIGURE 4 Goldmann visual field OD demonstrating nasal step and caecocentral scotoma.

ingested while the animals are grazing by sequestering them and preventing them from perforating the bowel wall. The magnet remains in the stomach of the animal for its lifetime. The patients' father, who is a farmer, confirms possession of these magnets. These particular magnets are typically 1 cm × 8 cm in size and contain 5–38% cobalt, 14–27% nickel, and 7–12% aluminum. There have been no reports of the other two metals of which these particular cattle pellets are comprised (nickel and aluminum) causing ocular toxicity; whereas cobalt chloride has been shown in both in vivo and in vitro animal models as a hypoxia-mimicking agent resulting in selective photoreceptor cell degeneration followed by total retinal damage at excessive dosages.³

Although cobalt ingestion is typically treated with chelation therapy with ethylenediaminetetraacetic acid (EDTA), it has been shown that NAC is also effective in increasing urinary excretion and decreasing the

concentration of cobalt in end organs and should be considered as a possible alternative due to decreased associated toxicity in comparison to EDTA.⁴

The case we describe is the first known case in the literature where accidental ingestion of magnets that are widely used in the dairy industry has caused ocular toxicity. Although the patient has not returned to baseline, his visual acuity and visual fields are continuing to improve, as are his symptoms of hypothyroidism, tachycardia, and polycythaemia for which he is being followed by the relevant specialists.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

Note: Figures 2–4 of this article are available in colour online at www.informahealthcare.com/oph.

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