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3.14 Bilateral optic neuropathy confirmed with VEP in a case of chronic chrome/cobalt intoxication

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Purpose: Optic neuropathy is a common condition in daily practice and diagnosis often remains difficult due to multiple nosologic contexts. Toxic optic neuropathy is a common cause of bilateral, progressive visual loss associated with a normal optic disc appearance at early stages and a caeco-central defect on the visual field. For the diagnosis of toxic optic neuropathy,

a thorough investigation is required to finally lead to the identification of the causal agent.

Methods: We report the case of a 76 year-old female who consulted her general ophthalmologist for the first time in November 2017 complaining about painless, bilateral, rapidly progressive visual loss. She was referred to our regional hospital for complementary investigations. We received the patient in March 2018. A thorough clinical examination included static automated perimetry, VEP recording and optical coherence tomography (OCT) of the macula and the optic nerve.

Results: The medical history revealed a right hip joint replacement in 2009 with clinical right leg metallosis features leading to ablation surgery of her right hip prosthesis in 2015. Right visual acuity (VA) was 0.6 logMAR and left VA was 0.3 logMAR. IOP was normal in both eyes (10 mmHg). The anterior segments were normal except for discrete bilateral cortico-nuclear cataracts. Fundus examination revealed a few bilateral serous drusen and a normal appearance of optic discs and vessels. On static perimetry, bilateral central scotomas were found. OCT RNFL was normal in both eyes. VEPs revealed bilateral axonal dysfunction: no P100 response could be identified. Laboratory testing for blood levels of chrome and cobalt are in progress to confirm our suspicion of bilateral toxic neuropathy.

Conclusions: Altered VEP responses confirmed the diagnosis of toxic optic neuropathy in this patient with bilateral visual field impairment and normal findings on both OCT and angiography. Cr/Co systemic toxicity is known to cause hypothyroidism, dilated cardiomyopathy, pericardial effusion, dyspnoea, hearing loss, tinnitus, cognitive trouble and peripheral sensori-motor neuropathy. Several cases of toxic Cr/Co induced bilateral optic neuropathy with central and caecocentral scotoma or homonymous quadrantanopsia have been reported. A partial recovery of symptoms after reducing blood levels of the causal agents has been described. The definitive treatment remains surgical prosthesis ablation, but some chelation studies have shown promising results with sodium-calcium edetate, acetylcysteine or dimercaptosuccinic acid. Recent studies suggest that the major toxicological risk comes from the insertion of a Cr/Co component concomitant with residual ceramic debris.