Methanol poisoning: documentation of optic disc changes

INTRODUCTION

Methanol is a clear, flammable, and volatile liquid, present in several commercial and consumer products, such as paints, glues, antifreeze for radiators, and alcoholic beverages. The main incidents occur due to the contamination of ethanol with methanol and improper distillation. Ingestion, inhalation, and dermal contact may be the route of entry of this substance into the body, which after metabolism generates formic acid—the main substance related to systemic toxicity of this alcohol. Among the clinical manifestations, confusion, ataxia, visual impairment, metabolic acidosis, brain lesions, and death may occur. Ocular toxicity is aggravated by metabolic acidosis, and the consequent optic atrophy seems to result from progressive demyelination of the nerve fibers.

KEYWORDS:
Toxic optic neuropathy; Methanol; Optic nerve Diseases.

ABSTRACT
Methanol poisoning is considered as a rare condition and can occur as a consequence of ingestion, inhalation, and dermal contact. Individuals at risk include young children, alcoholics, and those with suicide attempts, with most cases related to contaminated ethanol and improper distillation. Various systemic manifestations can occur, from irreversible visual damage to death. This study reported the case of a patient with toxic optic neuropathy secondary to the ingestion of methanol from a gas station.

RESUMO
A intoxicação por metanol é considerada uma condição rara e pode ser consequente à ingestação, inalação e contato dérmico. Indivíduos em risco abrangem crianças pequenas, alcoólatras e tentativa de suicídio, sendo a maioria dos casos relacionado ao etanol contaminado e à destilação imprópria. Várias manifestações sistêmicas podem ocorrer, desde danos visuais irreversíveis até a morte. Relatou-se neste estudo o caso de um paciente com neuropatia óptica tóxica secundária à ingestão de metanol de posto combustível.

INTRODUÇÃO

Methanol é um líquido claro, inflamável e volátil, presente em vários produtos comerciais e consumidores, como tintas, cola, anticongelante para radiadores e bebidas alcoólicas. Os principais incidentes ocorrem devido à contaminação de etanol com metanol e destilação imprópria. A ingestão, inalação, e contato dérmico podem ser os modos de entrada deste substância no corpo, que após metabolização gera ácido formal—o principal substância relacionada à toxicidade sistêmica deste álcool. Entre as manifestações clínicas, confusão, ataxia, deficiência visual, acidose metabólica, lesões cerebrais, e morte podem ocorrer. A toxicidade ocular é agravada pela acidose metabólica, e o consequente atrofia óptica parece resultar da progressiva desmielinização dos nervos.
CASE REPORT

A 28-year-old male patient was admitted to the emergency room due to sensory motion disorder. On admission, the use of crack cocaine and consumption of approximately 500 ml of ethanol from the gas station were mentioned. After 48 hours, the patient showed slight improvement in his sensory condition and was referred for ophthalmologic evaluation to assess bilateral visual acuity loss. Ophthalmologic examination showed absence of light perception in eyes (OU), mydriatic pupils, and absence of direct and consensual pupillary light reflex in OU. Fundoscopy revealed a pink optic disc with well-defined margins, excavation of approximately 0.2 and presence of intraretinal hemorrhage in the bilateral upper nasal region (Figure 1). On this occasion, two weekly doses of B-complex vitamin (5,000 mcg) were prescribed intramuscularly. In the reassessment a month later, he presented visual acuity of hand movement in OU, isochoric pupils, direct pupillary light reflex and consensual of 4+/4+ in OU, without relative afferent pupillary defect. In the fundoscopy examination, a diffuse pallor of the optic nerve and bilateral excavation of 0.9 were observed, with a generalized defect in the layer of nerve fibers (Figure 2). In this context, the ophthalmological findings along with the anamnesis are suggestive of toxic optic neuropathy secondary to the ingestion of ethanol contaminated with methanol.

DISCUSSION

Methanol gets rapidly absorbed after ingestion and oxidized in the liver to formaldehyde and formic acid. The accumulation of this substance causes metabolic acidosis and leads to cytochrome oxidase inhibition, interfering in the adenosine triphosphate production (ATP) by the mitochondria. The tissue hypoxia generated induces the death of axon cells, impacting on retinal ganglion cells that are affected by the degeneration of their axons and myelin, espe-

Figure 1. Retinography of both eyes (OU) on ophthalmologic admission.

Figure 2. Retinography of both eyes (OU) a month after intoxication.
Methanol poisoning can be fatal and lead to blindness. Photophobia, blurred vision, and eye pain on movement may be the first symptoms. In the acute setting, fundoscopy may show hyperemia and edema of the optic disc associated with dilation of retinal veins. Visual field defects, such as central and cecocentral scotomas, may be present. The acute phase may result in complete remission of the condition or evolve to optic atrophy with increased excavation, being part of the differential diagnoses of optic neuropathies with excavated disc. In addition, due to the significant bilateral visual acuity loss, neuromyelitis optica can be considered an important differential diagnosis. However, in this condition, the most common optic nerve alteration is the optic disc pallor without significant increase in excavation.

REFERENCES

AUTHOR’S INFORMATION

João Victor Fernandes Fabricio
http://lattes.cnpq.br/2831366551722164
https://orcid.org/0000-0003-2048-7074

Lucas de Oliveira Marques
http://lattes.cnpq.br/7901454901389457
https://orcid.org/0000-0003-0451-0027

Ana Laura de Araujo Moura
http://lattes.cnpq.br/0931275371609775
https://orcid.org/0000-0003-1768-6080