Unilateral Blindness Following Acute Methanol Poisoning

Jenny J. Lu · Ejaaz A. Kalimullah · Sean M. Bryant

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A 47-year-old man with history of alcoholism presented to the emergency department with altered mentation and “blurry vision.” He was tachypneic and minimally responsive to

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J. J. Lu (✉)

Department of Emergency, Division of Toxicology,
Cook County-Stroger Hospital,
1900 West Polk St., 10th floor,
Chicago, IL 60612, USA
e-mail: jlu@ccbh.org

J. J. Lu

Toxikon Consortium,
Chicago, IL, USA

E. A. Kalimullah

Department of Emergency Medicine,
Advocate Christ Medical Center,
Oak Lawn, IL, USA

E. A. Kalimullah

University of Illinois College of Medicine,
Chicago, IL, USA

S. M. Bryant

Department of Emergency Medicine, Division of Toxicology,
Cook County-Stroger Hospital,
Chicago, IL, USA

S. M. Bryant

Toxikon Consortium,
Chicago, IL, USA

S. M. Bryant

Illinois Poison Center,
Chicago, IL, USA

external stimuli, requiring endotracheal intubation. Vital signs were temperature 97.8°F, blood pressure 216/139, pulse 140/min, respiratory rate 26/min, and 98% oxygen saturation on room air. The pre-intubation venous pH was 6.79. Notable initial labs were sodium 135 mEq/L, potassium 3.7 mEq/L, chloride 95 mEq/L, total CO₂ 4 mEq/L, blood urea nitrogen (BUN) 11 mg/dL, creatinine 1.7 mg/dL, anion gap 36, serum osmolality 361 mOsm/kg, ethanol <10 mg/dL. An ethanol infusion, NaHCO₃ drip, folic acid 1 mg, thiamine 50 mg, and pyridoxine 50 mg were initiated. The patient was dialyzed for 6 h until resolution of the anion and osmolar gaps. The initial methanol level was 123 mg/dL with a post-dialysis level of 17 mg/dL. Ethylene glycol was undetectable. Following extubation, the patient, who had no prior ophthalmologic history and did not wear corrective lenses, was evaluated for persistent visual complaints. On exam, he had 20/20 acuity in the left eye, but only hand motion detection from the right eye. A right afferent pupillary defect and right optic disc pallor were also noted. Due to the unusual deficit, a magnetic resonance imaging (MRI) of the brain and orbits was obtained, revealing bilateral putaminal necrosis and edema at the retrobulbar and intracranial portions of only the right optic nerve (Fig. 1). MRI findings and cerebral spinal fluid (CSF) analysis did not provide alternative explanations for the ophthalmologic abnormalities. The patient was discharged on corticosteroids. On follow-up five months later, he had no visual improvement and remained ataxic and dysarthric.

Discussion

Acute methanol poisoning characteristically causes metabolic acidosis as well as neurologic and ocular toxicity. The manifestations of ocular toxicity may vary, but commonly

Fig. 1 MRI (T1 weighted) of the brain and orbits revealing enhancement of right optic nerve

include visual disturbances ranging from blurry or cloudy vision to total vision loss. Alterations in light, color, and depth perception may also occur [1, 2]. Retinal and optic nerve damage is believed to be due to the accumulation of the formic acid metabolite and to the effects of edematous and ischemic insults on these structures. The extent and permanence of ocular complications are likely affected by the severity of poisoning as well as the duration of exposure to formic acid [3, 4]. As with most, if not all, metabolic and toxicologic disorders, neurologic deficits are generally global or diffuse in nature while unilateral findings are unusual. Our

patient displayed neurologic and radiographic findings consistent with methanol-induced basal ganglia injury. However, while asymmetric visual evoked potentials and optic nerve injury patterns following methanol poisoning have been reported [5], blindness in one eye with normal visual acuity in the other eye is rare. It is possible that a structural anatomic variation could provide one explanation for this discrepancy. We are aware of only one previous cursory mention of unilateral optic neuropathy from methanol poisoning [6] and present here, in more detail, this uncommon occurrence.

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