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Ethylene Glycol Toxicity: Chemistry, Pathogenesis, and Imaging

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The ingestion of ethylene glycol results in toxicity with characteristic chemical, pathological, and imaging findings. In the case presented, magnetic resonance imaging demonstrated bilateral symmetric hyperintensity within the basal ganglia, thalami, and brainstem. Ethylene glycol toxicity also resulted in restricted diffusion within the white matter tracts of the corona radiata, a finding not previously described in the literature. In the acute clinical setting, ethylene glycol toxicity is an important differential consideration of the pathologies involving the deep grey matter nuclei.

Introduction

The ingestion of ethylene glycol, an organic solvent used in common household products such as automotive antifreeze and paints, results in toxicity with characteristic chemical, pathological, and imaging findings.

Case Report

A 20-year-old male presented to the emergency department following increasing lethargy and emesis over a twelve hour period. At presentation, the patient's Glasgow coma scale score was 6. Arterial blood gas demonstrated a pH of 6.96, pCO2 25 mm Hg, pO2 225 mm Hg and HCO3 5 mmol/L. The serum osmolality was 337 mmol/L and the osmolality gap was markedly elevated at 32 mmol/L. Microscopic examination of the urine demonstrated cigar and envelope shaped crystals characteristic of calcium oxalate crystals (Figure 1). Based on the clinical picture, laboratory findings and microscopic examination of the urine, the diagnosis of ethylene glycol toxicity was confirmed.

The patient was transferred to the intensive care unit for mechanical respiratory support, fluid management, and hemodialysis. Arterial blood gases were monitored from initial presentation through normalization. pO2 levels ranged from 225 mm Hg (elevated due to supplemental oxygen administration as part of resuscitation) to 84 mm Hg (normal at 36 hours into management). No hypoxic or anoxic periods were demonstrated during hospitalization. Due to clinical concern for seizure activity, head computed tomography (CT) was performed which showed diffuse hypodensity within the bilateral basal ganglia and thalami with loss of differentiation with the subjacent white matter,

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Abbreviations: ADC, apparent diffusion coefficient; CT, computed tomography; DWI, diffusion-weighted images; FLAIR, fluid attenuation inversion recovery; MRI, magnetic resonance imaging; T2WI, T2-weighted images

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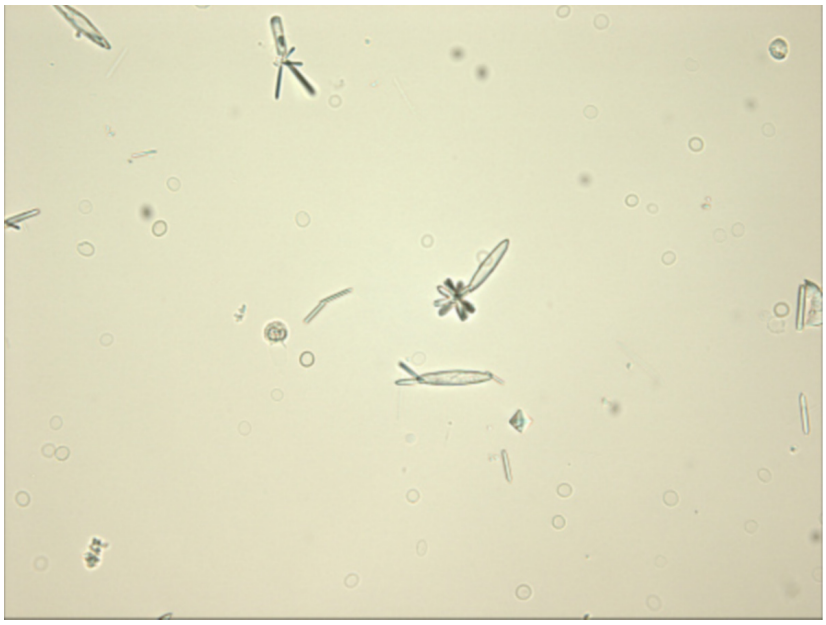

Figure 1. Urine microscopy demonstrates "cigar" and "envelope" crystals, a pattern characteristic of calcium oxalate monohydrate and calcium oxalate dihydrate crystals respectively.

compatible with edema (Figure 2). For further evaluation, magnetic resonance imaging (MRI) was performed (day one of hospitalization) which demonstrated increased signal intensity on T2 and fluid attenuation inversion recovery (FLAIR) sequences within the basal ganglia, thalami, amygdala, hippocampus, and brainstem bilaterally (Figure 3). There was restricted diffusion within the white matter tracks of the corona radiata bilaterally (Figure 4) on diffusion weighted (DWI) and apparent diffusion coefficient (ADC) imaging. On completion of the medical treatment seven days later, the patient's minimental status exam was normal with a score of 27. As the patient's symptoms had resolved, no further imaging was clinically indicated.

Discussion

Ethylene glycol is a common organic solvent, which is found in antifreeze as well as numerous other household products (paints, lacquers and polishes). Small quantity ingestions, either accidental or intentional, can produce toxicity, which is characterized by a severe anion gap metabolic acidosis, osmolar gap, and calcium oxalate crystals in the urine.

Chemistry

Ethylene glycol is metabolized in the liver through a series of enzymes. The intermediate metabolites of the pathway (in order) are: glycoaldehyde, glycolic acid, and glyoxylic acid. Ultimately, glyoxylic acid is converted to oxalic acid, which precipitates in the presence of calcium as calcium oxalate crystals. The most clinically significant metabolite in the pathway is glycolic acid which is primarily responsible for the metabolic acidosis [1,2]. This pathway assists in explaining our patient's laboratory and urine microscopy findings.

Pathogenesis

Ethylene glycol toxicity can affect multiple organs systems but predominatly involves the central nervous, cardiopulmonary and renal systems. Multiorgan damage is mainly due to the various toxic metabolites which have numerous deleterious effects at the cellular level including on the electron transfer chain, oxidative phosphorylation, cellular respiration, glucose metabolism, and DNA replication [1]. The deep grey matter nuclei of the basal ganglia being metabolically more active than the remaining brain parenchyma are affected first by these metabolites, as well as by the associated hypoxia and acidosis. Additionally, human autopsy studies have demonstrated calcium oxalate crystal deposition within the walls of the cerebral blood vessels and accompanying perivascular edema and inflammation [3,4]. The deposition of calcium oxalate crystals within the vasculature likely add up to produce further edema and damage to the deep grey matter nuclei and adjacent white matter.

Imaging

CT in the acute stages of ethylene glycol toxicity reveals edema without signficant mass effect with an affinity for the basal ganglia. Edema may also involve the temporal basal regions and brainstem [5,6]. Although the CT scan findings of ethylene glycol toxicity have been documented, there are only a few prior case reports with MR images published in the setting of ethylene glycol intoxication. Morgan et al. reported bilateral putaminal necrosis by

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