Acute Renal Toxicity After Ingestion of Lava Light Liquid

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A 65-year-old man with a history of alcohol abuse and seizure disorder presented to the emergency department with altered mental status, increased anion gap acidosis, phenytoin toxicity, and acute kidney failure. The patient had ingested the liquid contents of a Lava light, which contained chlorinated paraffin, polyethylene glycol (molecular weight 200), kerosene, and microcrystalline wax. Gas chromatography–mass spectrophotometry of the patient's blood produced results consistent with the same analysis of the Lava light contents. After 3 days of declining mental status and worsening kidney function, the patient required hemodialysis. After a prolonged hospitalization, the patient was discharged home with residual renal insufficiency. Although multifactorial, the associated renal toxicity was most probably related to the low molecular weight polyethylene glycol content of the lamp's liquid contents.

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INTRODUCTION

Decorator Lava lights were commonly displayed in households during the 1960s and 1970s, and they have also had a recent resurgence in popularity. Despite the popularity of these lamps, ingestion of the liquid contents has never been reported in the medical literature. We report a case of acute renal toxicity after ingestion of Lava light liquid.

CASE REPORT

A 65-year-old white man presented to an urban emergency department with a history of mental status change. The patient was alert but oriented to name only and was described as lethargic. Vital signs were blood pressure, 134/84 mm Hg; pulse, 110; respirations, 32; and temperature, 38.6°C. His physical examination was remarkable

for equal and reactive pupils with horizontal nystagmus, poor dental hygiene, and no oral mucosal burns or edema. The pulmonary, cardiovascular, and gastrointestinal examinations were normal. There was no costovertebral angle tenderness. Neurologic examination demonstrated no focal deficits.

In the ED, the arterial pH was 7.32, partial pressure of carbon dioxide was 22 mm Hg, and partial pressure of oxygen was 84 mm Hg. The serum sodium was 131 mEq/L; potassium, 4.0 mEq/L; chloride, 104 mEq/L; and bicarbonate, 12 mEq/L (anion gap of 15). Kidney function tests revealed a blood urea nitrogen (BUN) level of 17 mEq/L, and a creatinine level of 5.7 mg/dL. Five months previously the patient had had a BUN of 4 mEq/L and a creatinine level of .9 mg/dL. The serum glucose was 121 mEq/L. A complete blood count demonstrated a WBC count of 6.3/mm^3, hemoglobin of 14.1 g/dL, and platelets, $202 \times 10^3/mm^3$. The measured osmolality was 275 mOsm/L, with an osmolal gap of 1. The initial creatine phosphokinase (CPK) was 122 U/L. Urinalysis revealed a specific gravity of 1.020, with more than 100 WBCs per high-power field, more than 100 RBCs per high-power field, many bacteria, and positive ketones but without oxalate crystals or myoglobin. Urine toxicology screen for routine drugs of abuse was negative. Serum levels of acetaminophen, salicylates, ethanol, and the toxic alcohols (ethylene glycol, methanol, isopropanol) were zero. The serum phenytoin level was 49 µg/mL.

The ECG demonstrated sinus tachycardia with no ectopic beats or ischemic findings. The chest radiograph and computed tomographic scan of the head were normal. A renal ultrasound study demonstrated normal kidneys.

The patient was admitted to the ICU with a preliminary diagnosis of phenytoin toxicity and alcoholic/starvation ketoacidosis. A secondary provisional diagnosis was urinary tract infection, rule out pyelonephritis. A lumbar puncture was negative. Appropriate doses of intravenous ampicillin and gentamicin were begun. The urine culture grew gram-negative *Escherichia coli*, and blood cultures were negative.

Despite IV fluid hydration, the patient became oliguric 48 hours after admission, with an increasing BUN level of 30 mEq/L and a creatinine level of 7.9 mg/dL. On the third day, despite large doses of IV furosemide, the patient became anuric, with worsening kidney function (BUN, 66 mEq/L; creatinine, 11.3 mg/dL) and declining mental status. The nephrology service elected to subject the patient to hemodialysis. A repeat phenytoin determination made before dialysis revealed a level of 39 µg/L.

At this juncture, the family revealed that the patient had ingested the cooled liquid contents of a red Lava light, as an alcohol substitute, before admission. The time of ingestion was estimated to have been 12 to 24 hours before presentation. The family denied knowledge of other ingestions (which was later confirmed by the patient). The lamp's contents comprised water (38%), chlorinated paraffin (36%), polyethylene glycol (PEG) of molecular weight 200 (13%), kerosene (7%), and microcrystalline wax (6%). The liquid's pH was 6.5.

Gas chromatography–mass spectrophotometry (GC-MS) was performed on the patient's blood obtained from the day of admission. The separation of the aromatics (naphthalene, xylene) of higher boiling fractions (boiling point, 200° to 280°C) of kerosene have been described for kerosene's distillate.[1] Our approach entailed chloroform extraction of Lava lamp liquid, blood, and urine, drying of extracts at 55° to 60°C, and resolubilization in methanol. The instrument was a Finnigan ITS 40-EI operated in full scan 70 to 396 mass-to-charge ratio; the gas chromatography column was a 15 m×.25 mm, 95% dimethylsiloxane, 5% phenyl (DB-5) (JW Scientific). Run time was 10 minutes, with a temperature ramp program of 140° to 280°C. The results of blood analysis demonstrated peaks consistent with the tracing of the same analysis of the lamp's liquid contents. We could find no pattern in any of the four urine samples comparable to the blood or lamp liquid content.

The patient's subsequent clinical course was complicated by acute ethanol withdrawal and aspiration pneumonitis. He was discharged home after almost 3 months with residual oliguric renal insufficiency (BUN, 40 mEq/L; creatinine, 3.0 mg/dL).

DISCUSSION

Initially, the development of acute kidney failure in this patient appeared to be multifactorial. However, the coincidental ingestion of Lava light liquid implicates it as a primary causative agent. Although the initial starvation ketosis, dehydration, and prerenal azotemia were expected to resolve promptly with adequate fluid and glucose replenishment, this result did not occur. The fact that the initial serum creatinine level was markedly increased only 12 to 24 hours after ingestion may be attributable to the presence of acetone, which can interfere with creatinine determinations.[2] In our patient, however, the serum creatinine levels continued to increase after the ketones were cleared. Uncomplicated urinary tract infections and acute pyelonephritis typically do not produce detectable

alterations in BUN and creatinine levels.[3,4] Nitrogen retention is usually seen only in chronic pyelonephritis or end-stage kidney disease.[4,5] Our patient received appropriate aminoglycoside therapy after impaired kidney function was detected. Nonetheless, this potentially nephrotoxic antibiotic could have exacerbated the patient's kidney failure.

Renal toxicity following acute phenytoin toxicity has been reported, but it is usually caused by seizure activity or hypersensitivity reactions with ensuing rhabdomyolysis.[6,7] As evidenced by the normal CPK and lack of myoglobinuria, however, the renal damage suffered by this patient was not caused by acute rhabdomyolysis.

The Lava light liquid comprises water, chlorinated paraffin, microcrystalline wax, kerosene, and PEG of molecular weight 200. Limited data exist concerning the toxicity of chlorinated paraffins. However, both percutaneous and oral absorption in the rat model have produced minimal toxicity with no evidence of renal impairment.[8] Ingestion of microcrystalline wax is considered nontoxic.

Petroleum distillates such as kerosene are poorly absorbed from the gastrointestinal tract, and no significant complications are thought to occur as a consequence of absorption through this route.[9,10] The classic toxic manifestations of hydrocarbon ingestion are aspiration pneumonitis and central nervous system depression.[11]

Acute kidney failure subsequent to ingestion of hydrocarbons is thought to be caused by injury to the proximal tubule.[9] Tubular necrosis and acidosis are well documented with glue and toluene inhalation.[12,13] Acute rhabdomyolysis was reported in an adult female who sustained a cardiopulmonary arrest and hemolysis associated with prolonged exposure to concentrated vapors from mineral spirits.[14] Isolated case reports of acute kidney failure associated with dermal and gastrointestinal exposure to diesel fluid and refined petrol have been documented.[10,15] Kerosene has not been implicated as a cause of renal effects after oral ingestion.[9]

In any alcoholic patient with mental status changes, metabolic acidosis and kidney failure, ethylene glycol should be suspected.[16] In our patient, the lack of an osmolal gap, urinary calcium oxalate crystals, or a detectable ethylene glycol level ruled out ethylene glycol toxicity.

High molecular weight PEG-electrolyte solutions, such as Golytely (molecular weight 3,550) are considered nontoxic and cause minimal net water and electrolyte shifts.[17] These solutions are used for decontamination (whole-bowel irrigation) after certain types of toxic ingestion.[17] They are safe and well tolerated, and they do not cause electrolyte or osmotic imbalances. Animal studies addressing long-term ingestion of high molecular weight PEG solutions have documented little or no toxicity.[18]

On the other hand, renal tubular necrosis with oliguria, azotemia, and metabolic acidosis has been reported after IV or oral administration of low molecular weight (200 to 400) PEG.[19,20] High anion gap metabolic acidosis and kidney failure have been reported in burn patients receiving topical low molecular weight PEG dressings.[21,22] Although potentially multifactorial, the kidney failure in our patient was most likely linked to and exacerbated by the low molecular weight PEG content of the ingested liquid. The GC-MS documentation of Lava light contents in the blood strengthens the argument for this as a contributing cause.

The manufacturers of these lamps have recently improved the safety seal, making access to their contents more difficult and child-resistant. In addition, the contents have been reformulated to contain less toxic components. Nonetheless, many of the older lamps are still accessible, and the risk of renal toxicity resulting from ingestion remains.

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