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Hemodialysis Following Butoxyethanol Ingestion

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ABSTRACT

Case Report: This case report describes a 19-year-old male who ingested a product containing butoxyethanol, propylene glycol, and monoethanolamine. Neurotoxicity, acidosis, and butoxyacetic acid persisted after hemodialysis. Hemodialysis was used to treat the acidosis, but the half-life of butoxyacetic acid did not appear to have been significantly altered. Fomepizole, a recently approved alcohol dehydrogenase inhibitor for ethylene glycol intoxication, was not available at the time of this case. Residual neurological deficits persisted after recovery from this severe intoxication by a glycol ether.

INTRODUCTION

Butyl glycol ether, butoxyethanol (BE), ingestions have been rarely reported.[1,2] Toxicity includes coma, metabolic acidosis, and anemia secondary to hemolysis. The acidosis results from the metabolism of the BE to butoxyaldehyde by alcohol dehydrogenase and then to butoxyacetic acid (BAA) by aldehyde dehydrogenase.[3] The hematotoxicity has been shown to result from the BAA rather than the parent compound, BE. Because of limited clinical and scientific experience, treatment recommendations have been primarily anecdotal.[3] Beyond general supportive measures, antidotal administration of alcohol dehydrogenase inhibitors have been recommended.[4] Hemodialysis has been performed in the treatment of an adult patient.[2] We now describe another case of severe BE intoxication that was treated with hemodialysis.

Case Report

A 19-year-old male ingested an estimated 20 to 30 ounces of Spitfire®. This product contained BE 25-35%, propylene glycol 15-25%, monoethanolamine 5-10%, and potassium hydroxide 1-3%. The product was considered an alkaline corrosive, pH 13.

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This patient's past history is significant for mental retardation, depression, attention deficit hyperactivity disorder, and posttraumatic stress disorder secondary to child abuse. The patient resided in a group home. The ingestion was intentional following a violent outburst. His current medications were chlorpromazine, benztropine, lithium, propranolol, and vasopressin.

Twenty minutes after his ingestion, he arrived in the Emergency Department. Vital signs were heart rate 80 bpm, respiratory rate 24/min, blood pressure 114/60 mm Hg, and temperature 99.8°F. The patient was vomiting and became lethargic within an hour of the ingestion. His initial physical exam was also positive for rhonchi on chest auscultation. The blood pressure dropped to 85/64 mm Hg. He became comatose requiring mechanical ventilation. Gastric lavage and instillation of activated charcoal was performed within 1 hour of ingestion. The patient was begun on dopamine and transferred to the Toxicology Service at The Penn State Geisinger Health System.

On arrival in the intensive care unit 3½ hours after ingestion, the patient was deeply comatose. He had fixed, dilated pupils, no corneal reflexes, and positive Babinski's reflexes bilaterally. Tonic-clonic seizure activity was treated with lorazepam and phenytoin. His arterial blood gas on 100% oxygen was pH 7.36, Pco_2 19 mm Hg, Po_2 54 mm Hg, bicarbonate 11. Disseminated intravascular coagulation was evident: prothrombin time >60 sec, partial thromboplastin time >120 sec, fibrinogen 112 mg/dL, and a positive D-dimer. The electrolytes were sodium 139 mEq/L, potassium 5.2 mEq/L, chloride 116 mEq/L, and bicarbonate 13 mEq/L. Calcium was 1.5 mEq/L. The lactate level was 5.0 mmol/L. A chest X ray demonstrated a "white out" of the left lung with right-sided changes consistent with a severe aspiration pneumonia. Severe hypotension, systolic BP 58 mm Hg, required dopamine, epinephrine, and norepinephrine in addition to calcium replacement. An immediate esophagogastroscopy demonstrated mild inflammatory changes.

Ethanol therapy was considered as an adjunct that may have inhibited the metabolism of the BE, but because the patient was so critically ill, this theoretic therapy was withheld. Over the next 24 hours, the patient's condition improved significantly. All pressors were weaned and oxygen requirements decreased to 50%. The patient remained acidotic and therefore hemodialysis was performed. Although the patient's acidosis improved, BAA did not appear to be influenced by hemodialysis performed between 29 and 33 hours after the ingestion (Table 1).

Hematuria developed on the second day. The hematocrit fell from 43.1% to 23.0% on the fourth day. His platelet count fell to 88 K/UL on the fifth day postingestion. There was no evidence of renal or hepatic toxicity. A computerized axial tomographic scan of his head demonstrated mild diffuse cerebral edema on the second hospital day. The pulmonary findings and X ray improved dramatically over 2 days.

The patient had a significant recovery despite neurologic sequelae. He had choreoathetoid movements which resolved within 2 months. He occasionally has difficulty with fine motor skills. While he makes sounds, he does not speak. He did so prior to the intoxication. However, he comprehends language and uses a communication board. He attends social functions and otherwise interacts appropriately. In addition, he no longer appears to need psychotropic medications.

DISCUSSION

This case had the characteristic features of coma, acidosis, and hematuria, which were previously reported following BE ingestion. The etiology of the coma may have been multifactorial. The cointoxicants in the product, propylene glycol and monoethanolamine, may have contributed to the central nervous system depression. BAA may also induce or contribute to coma. In methoxyethanol ingestions, agitation and confusion were progressive, suggesting a correlation with the accumulation of the metabolite, methoxyacetate.[5] Our patient has been slowly recovering from severe neurologic deficits. Although the initial hypotension and hypoxia may be sufficient to explain the neurologic injury of our patient, BAA-induced neurologic injury should also be considered. Encephalopathy and neurologic impairments have followed the chronic exposure to glycol ethers.[4]

Pulmonary toxicity was another feature in this case, consistent with aspiration pneumonitis. BE was an ingredient in the aerosol leather protectors that produced pulmonary edema.[6] Ethanolamines also have produced occupational asthma.[7]

Table 1

Laboratory Data Before, During (29–33 hours), and After Hemodialysis

Hours Postingestion	Propylene Glycol (mg/dL)	Lactate (mmol/L)	Bicarbonate (mEq/L)	Butoxyacetate (mg/dL)
4		5.0	13	
9		6.6	11	
15	43	4.6	10	170
25	7	4.6	12	120
29	2	4.6	16	94
41	ND		20	51
48	ND	3.9	18	37
65			23	

The persistent metabolic acidosis correlated with the BAA levels. Propylene glycol is metabolized by alcohol dehydrogenase to lactate.[8] From 15 to 29 hours after the ingestion, the lactic acidosis was mild, despite the initially severe hypotension and hypoxia. Hemodialysis was performed to treat the refractory acidosis. It did not appear to significantly alter the clearance of BAA, although the overall acidosis did improve after dialysis. This is the first report to contain serial BAA levels. In the previous case that also reported hemodialysis, BAA was only measured in the urine.

This case occurred before the FDA approval of fomepizole (4-methylpyrazole, 4-MP). Alcohol dehydrogenase inhibitors in a rat model prevented toxicity from BE. There was a marked increase in the glucuronide and sulfate conjugates compared to BAA. The use of fomepizole may be warranted for the treatment of future BE intoxications.

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