Ethylene Glycol Poisoning following Ingestion of Brake Fluid

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ABSTRACT

A 32-year old male, with a history of depression and previous suicide attempts, was brought to hospital comatose after ingestion of brake fluid. He developed severe metabolic acidosis with an increased anion gap, hypotension, seizures and mild renal impairment. He required intensive care treatment for ventilatory and inotropic support. The clinical features, diagnosis and treatment of this unusual poison are discussed.

Envenenamiento con Glicol de Etileno Tras la Ingestión de Líquido de Frenos

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RESUMEN

Un sujeto masculino de 32 años de edad, con una historia de depresión y previos intentos de suicidio, fue llevado en estado comatoso al hospital, luego de haber ingerido líquido de freno. El paciente desarrolló una acidosis metabólica severa con aumento del gap aniónico, hipertensión, convulsiones, e insuficiencia renal moderada. Requirió tratamiento mediante cuidados intensivos con apoyo ventilatorio e inotrópico. El trabajo analiza las características clínicas, el diagnóstico y el tratamiento de este envenenamiento inusual.

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INTRODUCTION

Ethylene glycol is a toxic alcohol used in antifreeze, brake fluid, coolants and chemical solvents (1, 2). It has been implicated in both accidental and intentional poisoning and patients requiring hospitalization often need intensive care. There have been no reported cases of ethylene glycol poisoning from the English-speaking Caribbean.

CASE REPORT

A 32-year old unresponsive male, was brought to the Emergency Department of the University Hospital of the West Indies approximately one hour after ingesting brake fluid. He was known to have borderline personality disorder, major depression and alcohol dependence. He had four hospital admissions previously for suicide attempts.

He reportedly ingested approximately 200 ml of brake fluid, became confused shortly thereafter and collapsed, sustaining an injury to the side of his head on impact with the floor. On arrival at hospital, he had a Glasgow Coma Score (GCS) of 3/15 and gasping respiration. His pulse was 86/minute and low volume, his blood pressure was 100/80 mm Hg, the pupils were 5 mm and unreactive to light. He was intubated and manually ventilated. He subsequently became hypotensive (55/39 mmHg) and did not respond to fluid boluses. A dopamine infusion was commenced.

Investigations revealed a metabolic acidosis with a bicarbonate level of 14 mmol/L, base excess -7 mmol/L and lactate level 10.1 mmol/L. Electrolytes were normal and his creatinine level was 138 mmol/L. The calculated anion gap was 28 mmol/L. A computed tomography scan of the brain was normal.

He was admitted to the Intensive Care Unit for ventilatory and inotropic support. The metabolic acidosis was treated with sodium bicarbonate (200 mEq in 24 hours). He had several tonic-clonic seizures and was treated with a loading dose of 1g phenytoin followed by 100mg eight hourly. He received 600 mls ten per cent ethanol over four hours *via* nasogastric tube, followed by an infusion of 160 ml/hr. Intravenous thiamine and pyridoxine (Pabrinex I and II) were administered.

Within 48 hours, his metabolic acidosis had improved (HCO_3 = 20.4 mmol/l, BE = -5.9 mmol/l), as well as his GCS score (11/15). He was weaned off inotropes and his urine output was satisfactory, but his creatinine level increased to 200 mmol/L. The ethanol infusion was decreased to 80 ml/hour. On day 3, he was extubated uneventfully and maintained excellent peripheral oxygen saturations. His GCS

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score was now 15 and he had no neurological deficits. His metabolic acidosis had resolved (HCO_3 25 mmol/l; BE 0.1 mmol/L) and the ethanol infusion was discontinued. He was dis-charged to the medical ward and then home six days later with normal creatinine levels (74 mmol/L).

DISCUSSION

Ethylene glycol is water-soluble, cleared by the kidney, and has a half-life of 7 to 10 hours (1). It has little endogenous toxicity, but is metabolized by hepatic alcohol dehydrogenase to glycoaldehyde, glycolic and oxalic acids which are highly toxic (2, 3). These remain in the body for several days.

Early symptoms of poisoning include confusion, ataxia, hallucinations, slurred speech and coma. Patients present similarly to those with ethanol intoxication, but without an ethanol odour. This is followed by metabolic acidosis and renal involvement may develop within 24 to 72 hours (1, 3). Nausea, vomiting, tetany and seizures may occur. If untreated, severe ethylene glycol toxicity is usually fatal within 24 to 36 hours (3).

Quantitative serum levels of ethylene glycol are difficult and time consuming to obtain. A severe metabolic acidosis with an increased anion and/or osmolar gap should raise suspicion of this diagnosis.

Treatment of ethylene glycol poisoning includes decontamination, general supportive care, correction of the metabolic acidosis and correction of electrolyte disturbances (hypocalcaemia). Decontamination (such as gastric lavage and activated charcoal) has limited efficacy as ethylene glycol is rapidly absorbed in the stomach (3). Supportive care includes securing the airway and providing ventilatory support, if required. Fluids should be administered to maintain adequate urine output (4, 5). Supplemental thiamine (vitamin B1) and pyridoxine (vitamin B6) 100 mg daily are recommended because they act as cofactors in the conversion of glycolic acid into nontoxic metabolites. (1, 3, 6). Parenteral calcium may be necessary for treatment of tetany or seizures caused by hypocalcaemia.

Sodium bicarbonate is given to correct the metabolic acidosis. Ethanol is a competitive inhibitor of hepatic alcohol dehydrogenase and decreases the formation of toxic metabolites (6). If this therapy is initiated early, renal failure may be avoided. The recommended loading dose is 8–10 ml/kg of a 10% ethanol solution over 30 minutes intravenously, followed by a maintenance dose of 1.4–2.0 ml/kg/hour (3, 6). An oral dose was not seen in the literature. Oral ethanol (at the dose recommended for intravenous administration) was used because an intravenous preparation was not available and gut absorption is excellent. Administration was safe in view of already established airway protection with a cuffed endotracheal tube. The authors were unable to measure blood ethanol levels, so his response was monitored by frequent assessment of the metabolic acidosis. It is believed that the favourable outcome of this patient was due to prompt diagnosis and institution of appropriate management. Proper long term psychiatric care will be necessary to prevent a recurrence of his primary problem.

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