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Forensic Science International 155 (2005) 179–184
Forensic Science International
www.elsevier.com/locate/forsciint

Ethylene glycol poisoning

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Received 13 September 2004; received in revised form 10 November 2004; accepted 10 November 2004
Available online 21 January 2005

Abstract

Ethylene glycol (EG) can be found in many agents, such as antifreeze. Ingestion of EG may cause serious poisoning. Adults are typically exposed when EG is ingested as a cheap substitute for ethanol or in suicide-attempts. Children may be exposed by accidental ingestion caused by decantation of EG to unlabeled bottles. EG has in itself a low toxicity, but is in vivo broken down to four organic acids: glycoaldehyde, glycolic acid, glyoxylic acid and oxalic acid. The metabolites are cell toxins that cause central nervous system depression, and cardio-pulmonary and renal failure. Glycolic acid causes severe acidosis, and oxalate is precipitated as calcium oxalate in the kidneys and other tissues. We present five case reports of fatal EG-poisoning, and review the literature concerning clinical presentation and diagnosis, pathological findings, treatment and prevention.

Keywords: Ethylene glycol; Antifreeze; Poisoning

1. Introduction

Ethylene glycol (EG) is a bitter-sweet-tasting dihydric alcohol (HO–CH_2–CH_2–OH) that leaves a slightly warm sensation in the mouth after ingestion. It is odor- and colorless, water-soluble and has antifreeze properties. It can be found in many agents, such as antifreeze, brake fluids, and industrial solvents. Ingestion of EG is uncommon, but may cause serious poisoning. It is readily absorbed from the GI-tract, and the maximal blood-concentration is reached within 1–4 h, and the half-life is 3–8 h [1]. Absorption through the skin or lung is minimal. The estimated lethal dose of 100% EG is approximately 1.4 ml/kg [2]. Individuals who have ingested as much as 1–2 l have survived if treated within an hour [3]. EG has an inebriating effect similar to ethanol. It has in itself a low toxicity, but is in vivo broken down by the liver enzyme alcohol dehydrogenase to four organic acids: glycoaldehyde, glycolic acid, glyoxylic acid and oxalic acid [4]. The metabolites are cell toxins that suppress the oxidative metabolism causing central nervous system depression, and cardio-pulmonary and renal failure [5,6]. The rate-limiting step in the metabolism of ethylene glycol is the conversion of glycolic acid to glyoxylic acid. This results in an accumulation of glycolic acid in the blood. Glycolic acid causes severe acidosis, and oxalate is precipitated as calcium oxalate in the kidneys and other tissues [7,8].

We present five case reports of fatal EG-ingestion in order to illustrate the lethal potential of this common and widely available product, and some of the settings in which such poisonings may occur. This is followed by a literature review of the most important clinical and pathological features of EG-poisoning.

2. Case reports

Patient 1, 1970: A 29-year-old man was found sitting somnolent on a chair with a 3/4 empty bottle containing 50%

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doi:10.1016/j.forsciint.2004.11.012

ethylene glycol, on the table in front of him. He was under medical treatment for depressions, but had no alcohol abuse. He was admitted to hospital, and on arrival was somnolent and vomiting. He had metabolic acidosis, and developed generalized seizures. The treatment consisted of bicarbonate infusion, calcium, diazepam, penicillin, diuretics and hemodialysis. Heart arrest occurred 12 h after admission.

The medico legal autopsy showed swollen kidneys (total weight 440 g) with pale cortex and contrasting dark medulla. There was edema of lungs and brain, generalized congestion of the internal organs and fatty degeneration of the liver. Toxicology showed ethylene glycol in the stomach content but not in the blood, and at microscopy crystals with the typical morphology of calcium oxalate was found in the renal tubules, liver and cerebral vessels. The mode of death was assumed to be suicide.

Patient 2, 1985: A 39-year-old man was found unconscious by his mistress in her home. He had for three years lived a ''double life''—shared between his wife and his mistress, as his wife had refused to divorce him. He had no alcohol abuse. According to the mistress he had complained of feeling ill when he arrived to her home late in the evening. He said that he had had only one bottle of beer, but felt as if he had been drinking 20. He was, as often before, a little depressed. He went to bed and slept uneasily, and was very depressed and crying next morning. He felt ill and was vomiting, but refused to receive a visit from a doctor. In the morning he became unconscious and was admitted to the hospital. He had a severe metabolic acidosis with pH 6.84, and was treated with bicarbonate infusion. Hundred grams was aspirated from the ventricle. Blood-samples were negative for ethanol, methanol, propanol and methylethylethane. He developed hypocalcemia with generalized seizures and renal failure. He was transferred to the county hospital where he developed cerebral incarceration and died 4 days after first hospital-admission.

The medico legal autopsy showed swollen kidneys (total weight 490 g) with pale cortex and contrasting dark medulla. There was cerebral edema with incarceration, generalized congestion of the internal organs, fatty degeneration of the liver, bronchopneumonia and mucupurulent tracheobronchitis. Microscopy revealed crystals in the kidney tubules proved to be oxalate by silver-rubeanate staining. EG crystals were also found in cerebral vessels. There was edema, ischemic and probably toxic changes in the brain tissue. Toxicology showed that the ventricle aspirate from the hospital contained ethylene glycol, but no other poisons. Toxicological examination of blood and liver tissue was negative. Ethylene glycol or poisons like strychnine was not found in the half emptied beer bottle from the home of the mistress. Homicide was considered, but an extensive police investigation made suicide a more likely option. The deceased had probably ingested ethylene glycol in his car before visiting his mistress. Ethylene glycol stains were found on samples cut from the driver's seat and corresponding floor mats from the deceased's car. An EG-bottle was never found.

Patient 3, 1988: A 64-year-old man with a previous history of alcoholism, depressions and three previous suicide-attempts was found dead on a settee bed. On the floor next to the bed was an almost empty bottle labeled ethylene glycol. A liqueur glass containing yellow fluid of the same color as the content of the ethylene glycol bottle was found on a chest nearby, together with several suicide notes.

The medico legal autopsy showed severe decomposition with mould growth on the face. The right hand fingers were yellow decolorized with the same color as the content of the ethylene glycol bottle. The kidneys were decomposed (weight 248 g) and with a light color with small darker spots. Microscopy revealed many crystals with the typical morphology of calcium oxalate in the renal tubules and in the liver tissue. The toxicological examinations revealed ethylene glycol in urine in a concentration of 0.3%. There was no ethanol in the blood or urine. The bottle and the liquor glass contained ethylene ethanol in a concentration of 86%. The mode of death was assumed to be suicide.

Patient 4, 2001: A 41-year-old sailor with a severe alcohol abuse is put into ''the dry dock'' by the captain. He developed severe withdrawal symptoms including generalized seizures, and was transferred to a hospital on shore. On arrival he was almost unconscious and hyperventilating. He had metabolic acidosis, hypocalcemia and leucocytosis. CT-scan of the brain raised suspicion of cerebral thrombosis. He was transferred to the county hospital, but developed fatal intractable ventricular fibrillation shortly after arrival.

The medico legal autopsy showed swollen kidneys with pale cortex and contrasting dark medulla (total weight 454 g). There was edema of lungs, generalized congestion of the internal organs, acute heart dilatation, degenerative changes of the liver and petechiea and eccymoses in several internal organs. There was no cerebral thrombosis. He had several tattoos including the drawing of a full-rigged ship and the inscription ''sailor's grave''. Microscopy revealed crystals with the typical morphology of calcium oxalate in the renal tubules, severe fatty degeneration of liver tissue with fibrosis, slight acute inflammation of the leptomeninges and athrophia of the cerebellar vermis. A serum sample was negative for ethylene glycol. There was no ethanol in blood or urine.

The cause of death was ethylene glycol poisoning. The mode of death was assumed to be an accident.

Patient 5, 2002: A 42-year-old unconscious man with a previous history of serious alcoholism and drug addiction was admitted to hospital in a Danish provincial town at 18.30 h. The deceased had on an earlier admission to the hospital ingested alcohol used for hand disinfection.

On physical examination, blood pressure was 228/145, pulse rate 116 beats per minute, temperature 38.0 °C, and he had Kussmauls respiration. Blood specimens showed metabolic acidosis with arterial pH 6.92 and S-lactate 20 mmol/l. Ethyl alcohol concentration was below the limit of detection. CT-scan of the cerebrum was normal. He was treated with bicarbonate infusion, diuretics and general support. Renal