Case Report

Ethanol 50% as Antidote in Propylene Glycol Intoxication Due to Antifreeze Ingestion in General Hospital, Bali, Indonesia: Case Report

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ABSTRACT

Propylene glycol intoxication is a medical emergency requiring immediate intervention. It is metabolized by alcohol dehydrogenase into toxic metabolites, such as lactate and pyruvate, which can lead to metabolic acidosis and organ failure. The primary antidote strategy involves inhibiting alcohol dehydrogenase to prevent the formation of these harmful metabolites. This case report highlights a 53-year-old man who accidentally ingested a propylene glycol radiator coolant. He presented with symptoms of nausea, vomiting, and metabolic acidosis, confirmed through laboratory tests. With no fomepizole available—the preferred antidote—the patient was administered ethanol (gin) at a concentration of 50% over 10 minutes until intoxication occurred. Ethanol competes with propylene glycol for alcohol dehydrogenase, thereby preventing the formation of toxic lactate metabolites. The patient recovered without complications after two days of being given oral ethanol and was discharged. Although ethanol use as an antidote for propylene glycol poisoning is limited, this case underscores its potential effectiveness in reducing morbidity and mortality when fomepizole is unavailable. This approach demonstrates the importance of timely intervention in cases of toxic alcohol exposure.

Keywords: Propylene Glycol Intoxication, Alcohol Dehydrogenase, Antifreeze, Ethanol.

Introduction

Radiator fluid typically contains traditional glycol-based antifreeze agents, including ethylene glycol, propylene glycol, 1,3-butanediol, glycerol, and other alcohols. Among these, propylene glycol is one of the most commonly utilized antifreeze substances in the automotive industry. The Food and Drug Administration has classified propylene glycol as generally recognized as safe (GRAS). While the components found in radiator fluid are generally non-toxic, the body can metabolize them into highly toxic byproducts, such as lactic acid.[1,2]

Propylene glycol (PG) is a commonly used organic compound in pharmaceuticals, cosmetics, food production, and antifreeze manufacturing. Although it is generally considered safe at low concentrations, excessive exposure or buildup in the body can lead to adverse effects. These effects may include metabolic acidosis, central nervous system depression, acute kidney injury, arrhythmias, hypotension, hemolytic anemia, hepatotoxicity, and respiratory depression, and in pregnant individuals, it could result in premature birth.[1,3]

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Exposure to propylene glycol (PG) in humans can occur through various sources, including pharmaceutical use, consumer products, food, and occupational environments. In the general population, PG exposure is relatively low and typically does not cause toxicity.[4] However, intentional or unintentional cumulative exposure can occur in individuals who intensively use PG-containing products. In the Intensive Care Unit (ICU), PG exposure often arises from medications, where approximately 30-40% of patients receiving intravenous lorazepam show elevated blood PG levels, which can lead to toxicity if not properly monitored.[5]

Propylene glycol intoxication is a critical condition necessitating prompt recognition and immediate antidote. Its metabolism produces excessive lactic acid and pyruvate, leading to metabolic acidosis characterized by a high anion gap. The accumulation of lactic acid increases hydrogen ion concentration in the blood, lowering pH and causing acidosis. Additionally, during its metabolism, there is heightened production of reactive oxygen species (ROS), such as superoxide radicals, hydrogen peroxide, and hydroxyl radicals, contributing to cellular and organ damage. Propylene glycol poisoning has been associated with organ failure, including hypotension, heart failure, kidney failure, and even death. Moreover, glycol compounds are linked to acute kidney injury, which may result in irreversible kidney damage and severe neurological complications.[1,4]

The standard antidote approach generally includes using fomepizole or hemodialysis to block toxic metabolic processes and eliminate toxins from the body. Both fomepizole and intravenous ethanol are commonly employed to compete for the active site of alcohol dehydrogenase, thereby minimizing the production of harmful metabolites.[1] However, in our facility, fomepizole or intravenous ethanol is not consistently accessible, leading us to utilize ethanol as an alternative antidote.[2,4]

This case report describes the successful antidote of propylene glycol intoxication with simple antidote, such as administering ethanol, so that this report can be an experience that can be shared in treating emergency patients with propylene glycol intoxication.

Case Illustration

A 53-year-old male driver accidentally consumed approximately 300 ml of radiator water containing propylene glycol. About an hour after ingestion, he began experiencing nausea, greenish vomit, and headaches. He denied any symptoms of blurred vision, blood in vomit (hematemesis), or black, tarry stools (melena). Additionally, he reported no issues related to his respiratory, cardiovascular, or urinary systems. Upon arrival at the emergency room, his vital signs were within normal ranges: blood pressure was 130/80 mmHg, heart rate was 81 beats per minute, respiratory rate was 18 breaths per minute, oxygen saturation (SpO2) was 98% in room air, and body temperature was 36.9 °C. During the physical examination, the only notable finding was tenderness in the epigastric region. The patient also had a medical history of hypertension, for which he was being treated with amlodipine.

Laboratory tests revealed the following results: a complete blood count showed hemoglobin at 14.3 g/dL, leukocytes at 9.96 x 10^3/µL, and platelets at 261,000 x 10^3/µL. Renal function tests indicated a blood urea nitrogen level of 12.9 mg/dL and a creatinine level of 0.99 mg/dL. Electrolyte levels were within normal ranges, with sodium at 143 mmol/L and potassium at 3.44 mmol/L. Serum osmolarity was measured at approximately 282 mOsm/kg. Blood gas analysis indicated metabolic acidosis, with a pH of 7.31, PCO2 of 46 mmHg, HCO3 of 23.2 mmol/L, and a base excess of -3.1 mmol/L. The anion gap was calculated at 8.8 mEq/L. Random blood glucose was 95 mg/dL. Additional chemical laboratory tests, including liver function and albumin levels, were all within normal limits. A chest X-ray was also performed, showing no abnormalities. The patient has no prior history of diabetes mellitus or metabolic acidosis. Furthermore, alternative acidosis causes

were excluded through clinical examination and laboratory tests.

The patient was admitted to the emergency room in less than an hour. A naso-gastric tube was applied, and a gastric lavage with 100 ml milk was given for 90 minutes. Immediately, in this case, oral an ethanol antidote was started at 0.5 grams/kg, and the patient received 50% ethanol (Gin) at a dose of 100 ml over 10 minutes and was closely monitored for therapeutic intoxication. In this case, ethanol was administered five times until the patient became intoxicated. When the patient experienced intoxication, the administration of ethanol was stopped, and it was considered that the therapeutic dose had been achieved.

During the administration of the oral ethanol antidote, close monitoring of the patient's clinical condition and laboratory parameters was conducted to minimize the risk of potential side effects from the oral ethanol antidote. However, blood ethanol levels could not be measured in this case because they are unavailable in laboratory facilities. If tests are conducted at another laboratory center, the ethanol level results may take days and not reflect the current condition. So, the focus of the monitoring was on clinical signs of ethanol toxicity, such as low blood pressure, arrhythmias, slow breathing, and other symptoms like worsening nausea, vomiting, or signs of gastric irritation after ethanol administration. In this case report, no signs of ethanol toxicity were observed during the given antidote.

One day after initiating the oral ethanol antidote, the patient was reevaluated. Clinically, symptoms such as nausea, vomiting, and abdominal pain had significantly improved. A repeat arterial blood gas analysis revealed a pH of 7.34, PCO2 of 40 mmHg, HCO3 of 21.6 mmol/L, and a base excess of -4.2 mmol/L. After two days of hospitalization, the patient fully recovered and was discharged without complications.

Discussion

This case shows a successful emergency rescue of propylene glycol by immediate ethanol administration during the golden period of this agent intoxication. Since it could be neutralized in the blood circulation, it did not produce further toxic effects in the cells and organs. This condition may explain why the patient does not progress to a worse condition and immediately recovers.

Propylene glycol is widely used in antifreeze solutions for vehicles such as cars, aeroplanes, and ships, accounting for approximately 16% of its total usage. Additionally, it plays a significant role in the production of polyester resins and various plasticizers, making up about 27% of its overall applications.[6,7]

According to this case report, propylene glycol can be accidentally consumed due to its resemblance to water, a clear, liquid-like substance. The lethal dose considered dangerous varies depending on the route of administration and blood concentration. When ingested orally, a lethal dose is approximately 20 g/kg, with blood levels exceeding 100 mg/dL linked to the onset of symptoms. In adults, the half-life of propylene glycol ranges from 1.5 to 5 hours.[8]

Propylene glycol intoxication can lead to symptoms similar to those caused by ethanol. Exposure to high doses has been associated with toxic effects, including lactic acidosis (due to the production of both D- and L-lactate), hypoglycemia, seizures, coma, hypotension, heart rhythm abnormalities (dysrhythmias), and cardio-vascular collapse.[9]

Clinical complaints arise due to the metabolic products propylene glycol produces. Propylene glycol metabolism is similar to ethylene glycol metabolism, which requires alcohol dehydrogenase compounds in the liver. Metabolic products will produce D-lactate and pyruvic acid. The metabolic product D-lactate will cause lactic acidosis, an increase in the anion

gap, and kidney failure due to the slow clearance of D-lactate in the kidneys.[9,10]

Since many hospitals do not evaluate propylene glycol levels, it may be challenging to diagnose propylene glycol intoxication clinically. Nonetheless, propylene glycol intoxication typically results in elevated osmolarity, anions, and lactate testing. As in this case report, exposure to propylene glycol, clinical examination, and blood gas analysis were used to make the diagnosis, which showed metabolic acidosis.[6,8]

Antidote for propylene glycol intoxication requires quick action because delays in given antidote can be fatal and lethal. The inhibition of alcohol dehydrogenase compounds is one of the most important antidotes for preventing the formation of toxic agents such as D-lactate and pyruvate. Agents that can inhibit alcohol dehydrogenases, such as fomepizole and ethanol, are commonly used in the antidote of propylene glycol intoxication.[2,6,11]

Fomepizole is a drug that plays a competitive role in alcohol dehydrogenase, which is difficult to find, especially in Indonesia. Therefore, in propylene glycol intoxication, it is recommended to use ethanol, which is effective in treating propylene glycol intoxication. Because ethanol competes with and is a better substrate than alcohol dehydrogenase, it inhibits the metabolism of propylene glycol. Ethanol has adverse effects like hypoglycemia, hepatotoxicity, and additional central nervous system depression, making the management of ethanol in ethanol-intoxicated patients critical. Depending on the amount consumed and the patient's sensitivity. Some morbidity which dwells on the use of ethanol could also create a dilemma in the successful treatment response. The initial dose should be 600–1000 mg/kg, with maintenance dosing from 1000–1500 mg/l to maintain target ethanol levels and ensure sufficient saturation of the alcohol dehydrogenase to prevent further conversion of propylene glycol to toxic metabolites. Patients are treated with an ethanol antidote and require intensive care, which entails monitoring the ethanol and glucose levels in the blood every few hours.[1,10]

Ethanol remains a critical antidote option due to the limited availability of fomepizole. However, the administration of ethanol in propylene glycol intoxication is still limited. In general, the antidote for propylene glycol, apart from administering an antidote, can include decontamination of propylene glycol through gastric lavage, administration of sodium bicarbonate if metabolic acidosis is present, and hemodialysis, which is the last option if it causes complaints in the form of kidney failure.[9,10]

Ethanol is a well-established antidote for propylene glycol (PG) toxicity, particularly in cases where fomepizole (the preferred antidote) is unavailable. Ethanol works by competitively inhibiting ADH, thereby preventing the formation of these toxic metabolites. With ADH inhibited, propylene glycol is excreted unchanged in the urine, reducing systemic toxicity.[4,12]

Both fomepizole and ethanol are antidotes that bind more strongly to alcohol dehydrogenase (ADH) than toxic alcohols. However, fomepizole has a much greater affinity for ADH, being over 8,000 times stronger than ethanol. Fomepizole functions by competitively attaching to the enzyme's active site, effectively preventing the conversion of toxic alcohols into harmful metabolites. In contrast, ethanol only temporarily competes for this active site because ADH also metabolizes it.[4,13]

This difference in binding affinity makes fomepizole a more effective antidote than ethanol. Nonetheless, the possibility of conducting randomized controlled trials to assess their effectiveness is minimal due to ethical issues, the infrequency of poisoning incidents and outbreaks, and the lack of adequate research facilities in developing countries where such outbreaks are more common.[12,13]

In addition to its greater effectiveness in binding to enzymes, fomepizole has fewer side effects and does not require the same level of monitoring as ethanol. Unlike ethanol, which necessitates ongoing observation, fomepizole can be administered without constant oversight.[14] Although oral ethanol can serve as a useful antidote, it comes with several drawbacks, including the necessity for regular blood tests to monitor ethanol levels, continuous supervision during given antidote, a higher likelihood of adverse effects, and the requirement for multiple doses to sustain therapeutic levels.[4,5,15]

The limitation of this case report is due to the lack of equipment to test blood ethanol levels at the site, which means that ethanol level testing was not included in the monitoring process. Additionally, it would take days for the test results to be obtained from an outside laboratory, making it hard to match the patient's current state. This case report can be a useful resource for handling propylene glycol toxicity even in the absence of blood ethanol level measurement. The patient's clinical condition served as the basis for toxicity monitoring, and the therapeutic dose was deemed reached when the patient showed symptoms of intoxication and the ethanol administration was stopped. Throughout the patient's intoxicated state, continuous clinical monitoring and vital sign observations were conducted, with no signs of ethanol-induced toxicity observed.

Establishing guidelines for managing propylene glycol intoxication is crucial as a reference. Additionally, research should be undertaken to compare fomepizole and oral ethanol use, especially in Indonesia. However, the feasibility of this research may be limited by ethical considerations because research involving ethanol may conflict with cultural norms in Indonesia, particularly in communities where alcohol consumption is taboo. This trial can affect participant recruitment and acceptance of the study.

Since the availability of fomepizole in medical facilities, especially in Indonesia, tends to vary, the availability of fomepizole is expected to increase. Ethanol is often used as a substitute because it is cheaper and more accessible, even if it is less effective and needs to be monitored more closely. This situation highlights the critical importance of implementing strategic actions to facilitate access to fomepizole, including collaboration between healthcare professionals for distribution, financial assistance, and local manufacturing.

Moreover, research also needs to include other more available and affordable antidotes. The present study must focus primarily on effectiveness and safety, which must be maximized whenever possible in treating poisoning. It is also important to educate health-care workers on the treatment of propylene glycol intoxication with both antidotes of ethanol and fomepizole. Enhancing this understanding can lead to faster delivery of antidotes and correct use when necessary.

Conclusion

Propylene glycol intoxication is an emergency that requires prompt antidote to prevent morbidity and mortality. This successful management of propylene intoxication is because of an immediate decontamination band followed by ethanol as an alcohol dehydrogenase competitor agent.

Declarations

Competing interests

The authors declare no conflict of interest.

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None.

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