

Suicide by Ingestion of Household Phenol Disinfectant: A Case Report

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ABSTRACT

Phenol, also known as carbolic acid is one of the oldest antiseptic agents, which is rarely used today due to its toxicity. They are weaker in local action and better absorbed into the systemic circulation and can cause detrimental health effects. Phenol poisoning can occur by skin absorption, vapor inhalation, or ingestion. Gastrointestinal and dermal absorption can result in significant morbidity and mortality. The present case illustrates the potentially severe toxicity of suicidal oral administration of household phenol.

Key words : phenol; phenol poisoning; phenol burns; household disinfectant.

INTRODUCTION

Organic acids are weaker in local action and better absorbed into the systemic circulation¹. Majority of the phenol derivatives are important as they are active components in numerous antiseptics; and it is popular as a hospital and household disinfectant². Strong corrosive effect of phenol accounts for its toxicity. Phenol poisoning can occur by skin absorption, vapor inhalation, or ingestion, and, regardless of route of exposure, can result in detrimental health effects³. Although many cases of phenol poisoning were reported in the past, acute oral overdoses of phenol-containing solutions are relatively uncommon today. The present case-report is to illustrate the toxicity seen in a suicidal case of household phenol ingestion.

CASE-REPORT

A 41-year old agriculture laborer, who is a chronic alcoholic, found lying unconscious in bedroom by his wife. A toilet cleaner bottle is found lying in the room. He was rushed to nearby local Hospital, given supportive treatment and referred to our centre. He presented with vomiting and altered sensorium with a Glasgow coma scale of E2 V2 M4 on admission. He was hypotensive (80/60mmHg) and tachypneic with bilateral constricted pupils. He succumbed next day morning hours. On autopsy his face appears congested with black

discoloration of lips and chin (Fig. 1). Rigor Mortis was present throughout the body. Post mortem Lividity seen over the back and fixed. No other external injuries seen. Mucosa of esophagus and larynx appeared grey in color (Fig. 2 A,B). Stomach contained 200ml of grey color fluid with partially digested food particle with peculiar odor of phenol. Mucosa appeared congested with areas of corrosion in the fundus region (Fig. 3). Kidneys appear congested with well demarcation of cortico-medullary junction. Histopathological examination of kidney showed glomerular congestion and acute tubular necrosis (Fig. 4). Liver showed periportal steatosis and lymphocytic infiltrates accounts for the chronic alcoholism (Fig. 5). A sample of urine drawn showed greenish grey color (Fig. 6). The chemical analysis of routine viscera reports detection of phenol in stomach, intestine and its contents, liver and kidney but absent in the blood sample. The cause of death was opined as death due to acute tubular necrosis following phenol poisoning.

DISCUSSION

Phenol, also known as carbolic acid is one of the oldest antiseptic agents, which is rarely used today due to its toxicity. It has been replaced by various phenolic derivatives which are currently used as disinfectants, nail cauterizer, also as a component in lotions, ointments and gargles⁴. Symptomatic phenol poisoning can result from the intentional ingestion, occupational exposure,

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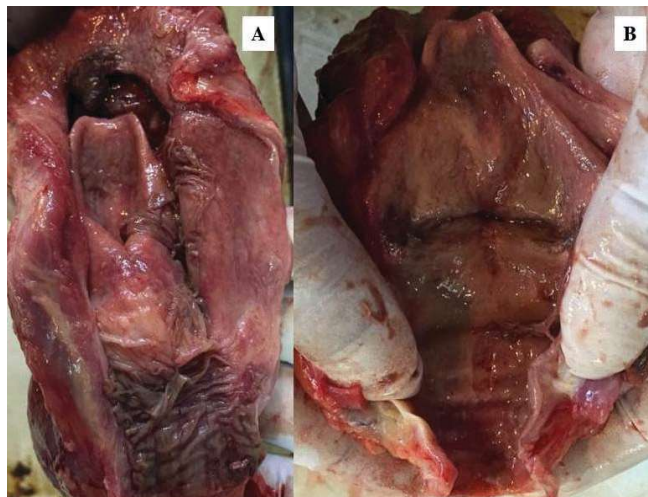
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Fig 1: showing blackish discoloration around the lips and chin (marked Circle) caused due to the Phenol burns.



parenteral administration and dermal contact. Gastrointestinal and dermal absorption can result in significant morbidity and mortality and have a fatal outcome of fifty percent in all reported cases. Systemic toxicity causing death within minutes to hours from severe dermal burns from phenol has been reported⁵. Phenols after penetration to the cell undergoes active transformation with cytochrome P450 and it leads to toxicity by the formation of electrophilic metabolites which can bind and damage DNA or enzymes⁶. Systemic manifestation includes central nervous system (CNS) causing CNS depression, seizure, lethargy and coma and cardio vascular system (CVS) like arrhythmias and hypotension. Metabolic acidosis, meth-hemoglobinemia, hypothermia also can get manifested. Rabbit syndrome characterized by fine rapid repetitive movements of the perioral musculature is observed due to extrapyramidal effect as a result of increased acetylcholine and decreased dopamine has been reported^{7,8}. Phenol causes skin necrosis as it reacts with amino acids contained in the keratin and collagen of skin and leads to brown discoloration of skin and white patches in the oral cavity as seen in this case⁹. Phenol levels in urine and blood can get elevated following ingestion and skin exposure and give a greenish blue color¹⁰. Sulfate group of enzymes have high affinity for phenol in low doses to form sulfate ester and in high doses detoxification is carried out by glucuronidation.

Fig 2A,B: showing greyish discoloration of mucosa of esophagus and larynx.



The unconjugated phenol can get excreted through kidney causing damage to glomeruli and renal tubules as in this case it leads to tubular injury¹¹. Cutaneous decontamination can be done with a low-molecular weight polyethylene glycol solution, isopropyl alcohol or high flow water^{3,12}. Management of systemic poisoning includes managing hypotension, arrhythmias and convulsions along with assisted ventilation if necessary. Emesis and gastric lavage are not recommended generally but activated charcoal can be administered by small bore nasogastric tube¹³.

CONCLUSION

Even though a diverse group of disinfectants, antiseptics are available, the more toxic chemicals like phenols, chlorates are still available and can cause life threatening injuries on exposure either accidental or suicidal. Prolonged exposure to phenol may result in major absorption and a long elimination half-life and can cause significant morbidity and mortality.

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Fig 3: showing the congested mucosa of stomach with areas of mucosal corrosions in the fundus.



Fig 4: showing areas of glomerular- congestion with the features of renal tubular necrosis (arrows). (H&E staining; 400x)

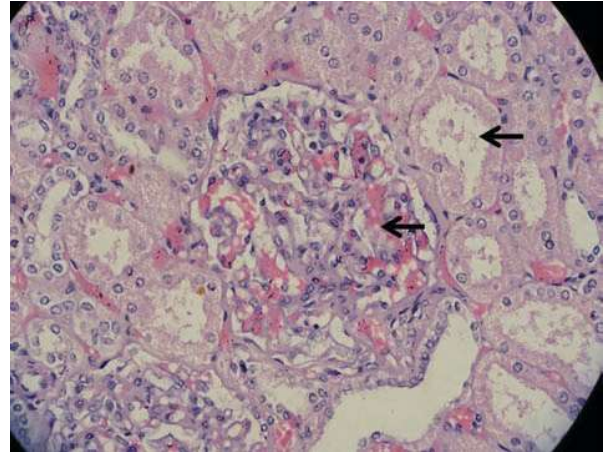


Fig 5: Showing periportal steatosis and lymphocytic infiltration of hepatic parenchyma.(H&E staining; 100x)

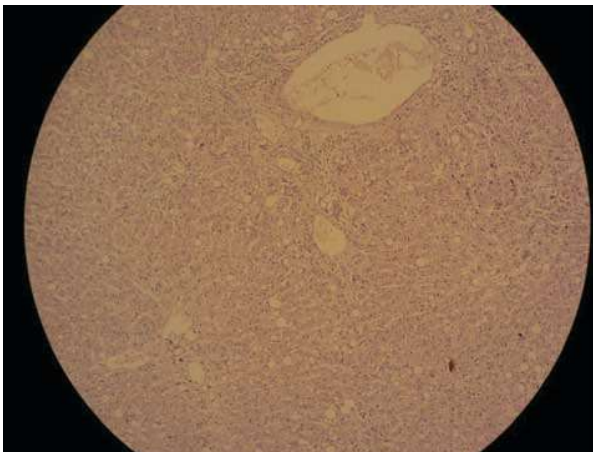


Fig 6: Showing Greenish-grey discolored urine collected at autopsy after about 12 hours of death.



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