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CASE REPORT

Methemoglobinemia and hemolytic anemia due to phenol ingestion

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Abstract

Background: Phenol is a highly toxic and corrosive compound frequently used as a disinfectant. Ingestion, whether accidental or intentional, can result in multi-system toxicity. Hematological complications, including methemoglobinemia and intravascular hemolysis, though rare, may present in a delayed fashion.
Case Presentation: We report a case of a 34-year-old female who ingested phenol with suicidal intent. Initially treated at a local facility, she presented to our emergency department two days later with nausea, vomiting, generalized weakness, and dark red urine. Clinical evaluation revealed severe anemia (Hb 4.6 g/dL), indirect hyperbilirubinemia, elevated lactate dehydrogenase, and methemoglobinemia (4.8%). Peripheral smear showed features of intravascular hemolysis. Despite hemoglobinuria, renal function remained preserved. She was managed with packed red cell transfusions, methylene blue, and supportive care. Her condition gradually improved, and she was discharged in stable condition.
Discussion: Phenol ingestion can lead to delayed hematological complications such as hemolytic anemia and methemoglobinemia, often appearing after initial stabilization. Mechanistically, oxidative damage to red blood cells and impaired oxygen delivery due to methemoglobin formation explain these findings. Unlike many reported cases complicated by acute kidney injury, this patient maintained preserved renal function despite hemoglobinuria, emphasizing the variability of presentations. Early recognition of delayed hematological effects and timely therapy with methylene blue and transfusion are critical for recovery.
Conclusion: Phenol remains a readily available toxic agent, and ingestion carries high risk of multisystem complications. Clinicians should maintain vigilance for delayed hematological manifestations and institute prompt supportive and antidotal treatment to reduce morbidity and mortality.

Keywords: Phenol, Poisoning, Intravascular Haemolysis, Hemolytic Anemia, Methemoglobinemia, Acute Kidney Injury

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INTRODUCTION

Phenol, also known as carbolic acid, is an aromatic hydroxy compound extensively used in industrial manufacturing, chemical laboratories, and as a disinfectant in household and healthcare environments [1]. It is a highly corrosive and toxic substance that can be absorbed through oral ingestion, inhalation, or skin contact. Accidental and intentional ingestions are not uncommon, especially in regions where phenol-based disinfectants are easily accessible. In India, phenol poisoning continues to be reported among adults, often in the context of suicide attempts [2].

Epidemiological data on phenol poisoning are limited but suggest sporadic cases across both developed and developing countries. In low-resource settings, under-regulation of industrial chemicals and easy over-the-counter access to phenol-based cleaners contribute to their misuse [3]. Case series from Southeast Asia and Africa indicate that phenol poisoning constitutes a small but significant percentage of chemical ingestion-related emergencies [4].

Phenol exerts its toxicity through multiple mechanisms. It is rapidly absorbed and primarily metabolized in the liver by conjugation with sulfate and glucuronic acid [5]. However, free phenol and its quinone metabolites can cause oxidative damage, leading to direct cytotoxicity. The estimated lethal dose in adults ranges between 3 to 30 grams, although even smaller amounts (as little as 1 gram) may prove fatal depending on the route of exposure and promptness of treatment [6].

Clinical manifestations depend on the route and quantity of exposure. Ingestion typically causes immediate

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oropharyngeal and gastrointestinal irritation—manifested by oral burns, vomiting, abdominal pain, and CNS depression. Systemic absorption can result in cardiovascular collapse, seizures, hepatic and renal dysfunction, and hematologic effects such as methemoglobinemia and hemolysis [7,8]. Methemoglobinemia occurs due to the oxidation of ferrous iron in hemoglobin to the ferric state, which impairs oxygen delivery to tissues and leads to cyanosis unresponsive to supplemental oxygen [9]. Hemolytic anemia is believed to result from oxidative injury to red blood cell membranes and may present several hours to days after the initial exposure [10].

Management of phenol poisoning is supportive and time-sensitive. Decontamination (e.g., dermal irrigation), maintenance of hemodynamic stability, and monitoring for organ dysfunction are key principles. Activated charcoal is generally avoided due to phenol's corrosive nature. Methylene blue is the antidote of choice for symptomatic methemoglobinemia and works by accelerating the NADPH-methemoglobin reductase pathway [11]. Blood transfusions may be necessary in severe hemolysis. Renal protection is crucial in cases of hemoglobinuria to prevent pigment nephropathy [12].

Despite extensive systemic toxicity, delayed hematologic manifestations such as methemoglobinemia and hemolytic anemia remain under-reported in medical literature. We report a case of delayed-onset methemoglobinemia and intravascular hemolysis following suicidal ingestion of phenol, underscoring the importance of prolonged observation and targeted therapy even after initial stabilization.

CASE PRESENTATION

A 34-year-old female presented to the emergency department with complaints of nausea, multiple episodes of vomiting, weakness, and dark red colour urine for one day (figures 1 and 2). She had ingested phenol two days back with suicidal intent, following which she was admitted to another centre and was discharged in stable condition. On general examination, she was found to have pallor, icterus with pulse rate 112/minute, blood pressure 80/50mmHg, oxygen saturation (SpO2) 88% on room air. Oral cavity examination revealed slight corrosion at the angle of the mouth and the inner aspect of the lips, with slight swelling of the tongue. Arterial blood gas (ABG) revealed hyperoxia (pO2 416mmHg), severe anaemia (Hb 4.9 g/dl), along with methemoglobinemia (4.8 %). Blood workup revealed severe anaemia (Hb 4.6 g/dl) with leucocytosis (TLC 15.09 x 109) and indirect hyperbilirubinemia (total bilirubin 6.1 mg/dl, Indirect bilirubin 5.6 mg/dl) with transaminitis (SGOT 109 U/L, SGPT 20 U/L, ALP 91 U/L, GGT 13 U/L). Renal function test was normal (urea 40.7 mg/dl, creatinine 0.7 mg/dl). Urinalysis revealed proteinuria with 3 RBC/HPF. She raised serum lactate dehydrogenase (LDH 1455 IU/L) and creatine phosphokinase (CPK 399 U/L), reticulocyte count (13.5%), peripheral smear showed marked anisocytosis, normocytic normochromic forms, admixed with macrocytes, microspherocytes, nucleated RBCs (2/100WBC), agglutination seen, suggestive of haemolytic anaemia. Urine-free haemoglobin was positive. She was

Figure 1. Gross appearance of the patient's urine on day 2 of presentation, showing dark reddish-brown discoloration due to hemoglobinuria following fenol ingestion

Figure 2. Subsequent urine sample demonstrating persistent red discoloration, indicative of ongoing intravascular hemolysis associated with phenol-induced methemoglobinemia

managed with PRBC transfusion along with empirical antibiotics and other supportive measures. USG whole abdomen, X-ray chest PA view, and 2D ECHO were done, which were normal. Serial monitoring of arterial blood gases, blood counts, liver function, and renal profile was done. She had persistent methemoglobinemia for which injection of methylene blue was given 1 gm/kg/day for a total of 3 doses. Gradually, she was started on clear liquids followed by a soft liquid diet as tolerated by her. She was discharged in stable condition after 5 days and is in stable condition on follow-up after 7 days.

DISCUSSION

Phenol is a corrosive also known as carbolic acid and its poisoning is known as "carbolism" [13]. The lethal dose is between 3 and 30g, but may be as little as 1 g. [14] Oral, inhalation, and dermal exposure to phenol are readily absorbed and widely distributed. The gastrointestinal tract, lungs, liver, and kidneys are the major sites of metabolism [5], producing toxic effects on the cardiac, respiratory, renal, and cranial nervous system [7] leading to multiple organ failure. Ingestion of a significant dose of phenol leads to intense burning of the mouth and throat, followed by necrosis of the skin and mucous membrane of the throat, along with gastrointestinal irritation, nausea, vomiting, sweating, and abdominal pain [8]. However, our patient had nausea, vomiting, and abdominal pain, symptoms consistent with gastrointestinal irritation, but there was no necrosis seen in the oral mucosa. Hypotension with shock observed in our case could be attributed to dehydration. Acute kidney injury (AKI) had been reported previously [15], presenting as darkening of the urine and glycosuria [6]. Various mechanisms causing AKI have been proposed, including unconjugated phenol excretion through urine, which damages glomeruli and renal tubules, or renal ischemia, or cast formation due to haemoglobin precipitation and glutathione depletion [12,16]. Although she had darkening of urine and free haemoglobin was present in urine, but she continued to have good urine output and her serum creatinine remained within normal range.

CNS manifestation includes depression, seizure, lethargy, and coma [13]. Cardiovascular effects include bradycardia, arrhythmia, and shock reported within 6 hours of exposure [8,13]. Phenol affects the respiratory system by suppressing the respiratory centre, leading to an increase in respiratory rate initially, followed by a decrease in rate and magnitude, leading to respiratory failure [17]. Haematological effects include haemolytic anaemia due to intravascular haemolysis [10], methemoglobinemia, and metabolic acidosis [13]. With an increase in the level of methemoglobinemia, Spo2 gradually decreases till it reaches a plateau of 85%, as was seen in our cases [9]. In the present case, methemoglobinemia and haemolytic anaemia due to intravascular haemolysis were seen, similar to those reported earlier. Management should be focused on decontamination as soon as possible to minimize absorption including contaminated clothes removal along with careful observation for complications and its prompt management. The toxic effect of phenol and its derivatives develops later, presenting with darkening of urine and methemoglobinemia, which could be fatal if left untreated, so it should be kept under observation.

CONCLUSION

Phenol or carbolic acid is a commercially available antiseptic and disinfectant, which is easily procurable, so it is a very common cause of poisoning. Phenol ingestion could lead to severe life-threatening complications and have high mortality and morbidity due to toxic effects on the gastrointestinal, CNS, and hemolysis, leading to acute kidney injury. There is sparse literature on the toxic effect of Phenol and its derivatives and complications occurring later, leading to improper management, which in turn leads to high mortality and morbidity, which could be prevented with proper knowledge and information.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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