

## Phenol and Its Toxicity: A Case Report

Mahesh Chand Meena<sup>\*1</sup>, Rahul Band<sup>1</sup>, Girish Sharma<sup>1</sup>

Received: 15.08.2014

Accepted: 28.09.2014

### ABSTRACT

**Background:** Phenol and its derivatives like dinitrophenol and pentachlorophenol (carbolic acid) are widely used as insecticides, but they are very toxic substances. Phenol is a general protoplasmic poison with corrosive local effects that denature proteins. Poisoning with phenol compounds may occur by ingestion, inhalation, and absorption through skin. In this report we presented the toxicity effects of Phenol and its derivatives like dinitrophenol and pentachlorophenol on humans.

**Case report:** A 27-year-old married female was found unconscious at her residence in September 2013. She was expired after hospitalization in Lady Hardinge Medical College and its associated hospital on the same day after six hours. On examination, corrosion of skin, at angel of mouth and chin, and brown discoloration in mucosa of the esophagus were seen. Histological examination showed exfoliation of esophageal mucosa and coagulative necrosis of gastric mucosa. In toxicological analyses, carbolic acid was detected.

**Conclusion:** Strict precautionary measures are advised when using this compound.

**Keywords:** Corrosion, Phenol, Toxicity.

IJT 2015; 1222-1224

### INTRODUCTION

Phenol is a hydroxybenzene which is used commercially as a disinfectant. Phenol and its derivatives like dinitrophenol and pentachlorophenol are very toxic substances with a toxicology rating of 4 [1]. Exposure may occur by inhalation, cutaneous adsorption, or oral ingestion. Phenols denature and precipitate cellular proteins and thus may rapidly cause poisoning [2]. They are metabolized by conjugation and oxidation [3]. Both local and systemic effects have been reported following ingestion of phenol, including cardiovascular effects, respiratory distress, metabolic acidosis, renal failure, neurological effects, shock, coma, and death [4]. Intense burning of the mouth and throat may occur following swallowing a significant concentrated dose of phenol, leading to necrosis of the skin and mucous membranes of the throat, as well as abdominal pain and gastrointestinal irritation, nausea, vomiting, sweating, and diarrhea. Phenol is considered to be very toxic to humans through oral exposure. Ingestion of 1 g phenol is reported to be lethal with symptoms, including muscle weakness and tremors, loss of coordination, paralysis, convulsions, coma, and respiratory arrest [5].

### CASE REPORT

A 27-year-old married female was found unconscious at her residence in September 2013. She was expired after hospitalization in Lady Hardinge Medical College and its associated hospital on the same day after six hours. On external examination of the body, hypostasis was fixed and rigor mortis was complete. Corrosion of skin at angel of mouth and chin was seen (Figure 1). Corrosions were initially white but turned brown in color. Signs of splashing were also noted. On internal examination, the tongue was white and swollen. Mucous membrane of mouth, throat, and lips was sodden with ash gray. The mucosa of the esophagus was tough and grayish-white (Figure 2). Mucosa of the stomach was also tough, white or gray, corrugated and arranged in longitudinal folds, and looking leathery (Figure3). The gastrointestinal mucosa was congested and hemorrhagic, with bluish discoloration of mucosa. All internal organs were congested. The stomach contained about 300 ml of phenol odor emitting fluid (Figure4). During autopsy, samples of biologic fluids were taken for toxicological examination. Histological examination showed exfoliation of esophageal

1. Department of Forensic Medicine and Toxicology, Lady Hardinge Medical College, New Delhi, India.

\*Corresponding Author: E-mail:drmahe2012@gmail.com

mucosa and coagulative necrosis of gastric mucosa. In Toxicological analyses, carbolic acid was detected. On this report was conducted ethics compliance with the carcasses of the human body.



**Figure 1.** Corrosion of skin (brownish) over mouth and chin.



**Figure 2.** Mucosa of the esophagus (tough and greyish-white).



**Figure 3.** Mucosa of stomach (tough, corrugated and arranged in longitudinal folds and looking like leather).



**Figure 4.** Stomach content (about 300 ml of phenol-odor-emitting fluid).

## DISCUSSION

Phenol absorption is really fast (about 30 min). It is quickly excreted into urine [6]. The toxicity of conjugated forms of phenol is less than that of free compounds. Therefore, the free phenol concentrations in the blood can be regarded as an index of poisoning [7]. The average fatal dose is 25-50 ml of household phenol.

Derivatives of phenol, such as cresol, are endogenously produced in normal subjects. They may also be present in urine at concentrations of 20-200 mg/ml, mainly in the conjugated form. Endogenous phenol concentration in urine is about 5-10 mg/ml on average [3]. There are many reports on toxic injuries with phenols including cases with fatal outcome [1, 7, 8]. Intense burning of the mouth and throat may occur following swallowing a significant dose of concentrated phenol, leading to necrosis of the skin and mucous membranes of the throat, as well as abdominal pain, gastrointestinal irritation, nausea, vomiting, sweating, and diarrhea. Storage of energy in the form of adenosine triphosphate is prevented, thereby leading to a compensatory increase in the basal metabolic rate which is responsible for most of the principal clinical features of the toxicity of this substance.

In the present case, hypotension with shock was observed which can be ascribed to the combined effect of gastrointestinal bleeding and dehydration. Phenol is depressant of the nervous system, especially the respiratory centre. Respiratory effects are often characterized by an initial increase in respiratory rate followed by a decrease in both its rate and magnitude, leading to respiratory failure, which is the most common cause of death following acute ingestion of phenol [9]. Cardiovascular effects such as bradycardia, have been reported following phenol ingestion [4]. However, following ingestion of one ounce of 89 % phenol, respiratory arrest within 30 minutes and ventricular tachycardia within 60 minutes are expected, which subsequently may progress to supraventricular and ventricular dysrhythmias. The signs of GI irritation as esophagitis and GI bleeding occur within one week of exposure [9]. Dark urine may be produced following ingestion of phenol. Acute renal failure may also occur [4].

Death has been reported within 10 minutes of ingestion of 4.8 g phenol. Other cases have been reported in which death has occurred within hours after ingestion of 10–20 g phenol. In the latter case, tachypnea was initially observed, followed by dyspnea. On autopsy, pulmonary edema is reported [9]. The lowest dose at which death has occurred in humans is 140 mg/kg body weight.

Santa *et al.* have reported a case of hemolytic anemia following accidental ingestion of phenol in 2003 [10]. Intravascular hemolysis after inhalation of phenol compounds has been reported as well [11].

Phenol vapors are irritating to the upper respiratory tract and produce wheezing. Other effects associated with phenol inhalation include anorexia, weight loss, headache, vertigo, salivation, and dark urine. Phenol is corrosive and can also cause severe ocular damage including corneal opacification [4].

Dermal exposure produces dermal inflammation, erythema, and painless blanching. However, once pain becomes evident, serious burns, corrosion, and necrosis have already occurred. Effects are worse if the affected sites are bandaged [4, 9].

Cardiovascular shock, cardiac arrhythmias and bradycardia, as well as metabolic acidosis have been reported within 6 hours after skin peeling procedures with phenol [9]. Hyperventilation, acute renal failure, and methemoglobinemia have also been reported.

## CONCLUSION

Phenol ingestion can lead to severe and life-threatening effects, with high mortality in severe cases due to the combined effect of gastrointestinal bleeding and dehydration, hypotension, and shock. Strict precautionary measures are, therefore, advised when using this compound.

## ACKNOWLEDGEMENTS

We would like to thank all the physicians and staff of the Department of Forensic

Medicine, Lady Hardinge Medical College, India, for their timely support.

## REFERENCES

- Gosselin R, Hodge H, Smith R, Gleason M. Clinical Toxicology of Commercial Products: Acute Poisoning. Baltimore, MD: Williams and Wilkins Co. 1976.
- Monma-Ohtaki J, Maeno Y, Nagao M, Iwasa M, Koyama H, Isobe I, et al. An autopsy case of poisoning by massive absorption of cresol a short time before death. Forensic science international. 2002;126(1):77-81.
- Moffat A, Jackson J, Moss M, Widdop B. Clarke's isolation and identification of drugs. The Pharmaceutical Press, London. 1986;2:936-8.
- Dell Acqua C, Pronczuk J. Poisons information monograph: brodifacoum. Poisons Information Monograph: IPCS; 1999.
- Agency for Toxic Substances and Disease Registry (ATSDR), Toxicological Profile for Phenol, US Public Health Service, US Department of health and human services, Atlanta, GA, 1989.
- Subrahmanyam B. Modi's medical jurisprudence and toxicology: Butterworths; 1999.
- Boatto G, Nieddu M, Carta A, Pau A, Lorenzoni S, Manconi P, et al. Determination of phenol and o-cresol by GC/MS in a fatal poisoning case. Forensic science international. 2004;139(2):191-4.
- Armstrong RW, Eichner ER, Klein DE, Barthel WF, Bennett JV, Jonsson V, et al. Pentachlorophenol poisoning in a nursery for newborn infants. II. Epidemiologic and toxicologic studies. The Journal of pediatrics. 1969;75(2):317-25.
- Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Phenol. US Department of Health and Human Services. Atlanta, US, 1998.
- Santa N, Bhagyabati S, Jeetenkumar T, Motu T, Lokeshwar K, Singh MK. Phenol-induced Haemolytic Anaemia. 2003; 4(2): 174-5.
- Hassan AB, Seligmann H, Bassan HM. Intravascular hemolysis induced by hexachlorophenol. Br Med J. 1985; 291: 21-2.