Case Report

Case of Phenol Poisoning with Severe Intravascular Hemolysis with Hemoglobinuria

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ABSTRACT

Phenol is one of the oldest antiseptics. Currently it is used as a pesticide, disinfectant, chemical intermediate and nail cauterizer. Phenol is a general protoplasmic poison (denatured protein) with corrosive local effects. Phenol derivatives are less toxic than pure phenol. Poisoning with phenol compounds may occur by ingestion, inhalation, and absorption through skin. Because of its oxidative properties, apart from the local corrosive effects, many systemic side effects are reported with phenol poisoning. Haemolytic anaemia following phenol poisoning is rarely reported in the literature. Although no definite treatment is available, keeping an eye open for this complication and providing early supportive treatment can save the life of the patient

Introduction :

Phenol is a hydroxybenzene obtained from coal tar oil by fractional distillation. It is a potent insecticide, herbicide, and fungicide. Phenol and its derivatives like chloroxylenol (Dettol), dinitrophenol and pentachlorophenol are very toxic substances with a toxicology rating of 4[1]. Poisoning may occur by ingestion, inhalation, and absorption through the skin. Although phenol is considered as a corrosive, many systemic side effects are reported. Systemic side effects can involve all organ system including the cardiovascular, respiratory, renal, central nervous system and hematological[2]. Strict precautionary measures, therefore, should be taken when handling these substances. We report a patient who developed haemolytic anaemia, cyanosis and metabolic acidosis following ingestion of phenol. Cases of intravascular haemolysis through inhalation of phenol compounds have been reported[3], but a case of haemolytic anaemia following accidental ingestion of phenol is rarely reported in the literature[4].

Case Report :

A 24 years old previously healthy male was admitted to Department of Medicine for investigation of excessive weakness, nausea, and pain upper abdomen. Three days prior, he had ingested a phenol preparation. There was no history of taking any drugs or other substances than the phenol preparation. On examination he had icterus, cyanosis and was afebrile with a heart rate of 108 beats per minute; blood pressure was 110/70 mmHg and cola coloured urine. Although patient had cyanosis, he was not dyspnoeic and his SpO_2 level was maintained around 95%. The rest of clinical examination did not reveal any abnormality

On investigation the Hb concentration was 7.8 gm/dl, white cell count 23,300/mm3, platelets 1,80,000; reticulocyte count of 1.5%, peripheral blood smear showed normochromia and anisocytosis. Total bilirubin was 11mg/dl with an indirect fraction of 10.0 mg/dl. SGOT (AST), SGPT (ALT) levels were 78 and 63IU/L respectively and alkaline phosphatase was 190 IU/L, and INR was 1.09, urine was positive for haemoglobin. ABG pH7.27; PO_2 67 mm of Hg ; HCO_3 9.0 mmol/L. Blood urea was 83 mg/dl and serum creatinine was 2.0 mg/dl.G6PD levels were normal.

Based on clinical examination and investigations a diagnosis of phenol induced intravascular hemolysis (hemoglobinuria, anisocytosis, elevated indirect bilirubin) with metabolic acidosis (ABG) with methhemoglobunemia (cyanosis, normal SpO_2, normal arterial Po_2) with AKI (deranged renal function tests) was made. Methhemoglobulin levels cannot be done due to financial constrains.

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Patient was started on antibiotics (injection cefotaxim, injection metronidazole), proton pump inhibitors, antiemetic's, diuretics, blood transfusions and intravenous fluids. He was also given injection methylene blue presuming significant methhemoglobunemia was present. Injection sodium bicarbonate was given to counteract acidosis. Although his cyanosis decreased, his general condition deteriorated and he developed MODS. His liver function test become further deranged and patient went into respiratory failure for which he was intubated and kept on ventilatory support. Inspite of above supportive measures, we were not able to save the patient. Patient expired on 6th day after admission.

Discussion :

This patient presented with severe intravascular hemolysis with hepatic injury, metabolic acidosis and methhemoglobinemia after phenol consumption. There are many reports on the toxic injury with phenols including cases with fatal outcome[1,5]. Acute toxicity causes intense burning sensation in mouth, throat, and stomach. Systemic side effects can involve all organ systems. Phenol is rapidly absorbed into the blood and there may be hyper- or hypo-thermia, tachycardia, tachypnoea, pronounced general weakness, dizziness, nausea, CNS depression, ARDS, hemolysis, renal dysfunction and shock leading to death[8]. The average fatal dose is 2g[6]. It is excreted chiefly in the urine and also by the liver, lungs, and skin[6].

Very little is known about the metabolic effects of crude phenol, but it may share the ability of its derivatives like dinitrophenol, pentachlorophenol to interfere with the oxidative phosphorylation in cells[1]. 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.

The main source of energy in red blood cells is anaerobic glycolysis. Energy is stored in the molecules of adenosine triphosphate, a process that might be prevented by the toxic effect of the phenols. Due to shortage of energy, red blood cells cannot continue to perform their vital functions like preventing the osmotic equilibrium across the cell membranes, the cation pump and cell deformability. This metabolic handicap may lead to premature lysis of the cells causing haemolysis[2]. Also, phenol may produce Heinz bodies and contribute to haemolysis[7]. No definitive treatment other than supportive measures is available at present. Therefore, we recommend that utmost precautions should be taken while using this potent and widely used chemical.

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