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Hyperbaric Oxygen Therapy in Phenol Poisoning with Methemoglobinemia - A Case Report

Dr. Kuldeep Chittora[1*], Dr. Mansi Choudhary[2], Dr. Shabbar H.K. Joad[3]

[1]DNB- Anesthesia attending consultant Dept. of critical care medicine Fortis Escorts Hospital, Jaipur, Rajasthan, India
[2]MBBS DNB Resident- internal medicine Dept. of internal medicine Fortis Escorts Hospital, Jaipur, Rajasthan, India
[3]FNB – Critical Care Head of department – Critical care Dept. of critical care medicine Fortis Escorts Hospital, Jaipur, Rajasthan, India

***Corresponding author**
Dr. Kuldeep Chittora

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Abstract: "Pheneol" (containing coal tar acid, phenolic compounds and coal tar oil) is a common household floor cleaner (pesticide / bactericide / fungicide) used in India, with number of reports concerning its human toxicity. We present a case of middle aged woman presented with suicidal oral ingestion of phenol, manifested with Methemoglobinemia, followed by administration of hyperbaric oxygen therapy and rapid recovery.

Keywords: Pheneol, Household floor cleaner, Methemoglobinemia, Hyperbaric oxygen therapy.

INTRODUCTION

Pheneol (fig. 1) household floor cleaner (bactericide, pesticide, fungicide) containing coal tar acid, phenolic compounds and coal tar oil are very commonly used in India[1]. Phenol (C_6H_5OH) derivatives are important as they are active components in numerous antiseptics, hospital and household disinfectants. Phenol poisoning can occur by skin absorption, vapour inhalation or ingestion. Phenol is extremely corrosive leading to detrimental health effects. We present a case of Methemoglobinemia following deliberate ingestion of pheneol during suicidal attempt. With rapid identification of the cause and early administration of hyperbaric oxygen therapy, this type of acutely intoxicated patient can be managed successfully with a good prognosis.

CASE REPORT

A 43yr old female with a history of depressive disorder was brought to our emergency department by her family. She had ingested about 50 ml-100 ml of household floor cleaner (containing coal tar acids, phenolic compounds and coal tar oil) during an attempt of suicide 1 hour back; the ingestion was followed by vomiting and drowsiness.

On arrival in emergency of our hospital, she was dull and drowsy but following verbal commands. Her temperature was 36.8 °C, pulse rate was 84 beats/minute, respiratory rate was 24 breaths/minute, and her blood pressure was 90/60 mmHg. Room air SpO_2 was 92%, Random Blood Sugar 201mg/dl. On physical examination, pallor was present. The pupil size was 2.0 mm symmetrically, and both pupils had a good light reflex. The patient's skin was dry. Oxygen was immediately supplied through nasal prongs at 4 lt/min. Vomiting, with the content having a pesticide odor, was noted at the emergency department, so gastric lavage was done with 1.5 L Normal saline.

Patient was immediately shifted to medical ICU. Skin decontamination with body wash and scrub was done. Clothes were changed.PR: 84beats/min, RR: 20/min, B.P. 80/60 mm Hg, SpO2: 82%. Oxygen supplementation was continued. Initial blood and urine investigations comprising CBC, LFT, RFT, ABG, chest x-ray and 12 lead ECG were ordered.

Laboratory examination disclosed a white cell count of 8.9×10^9 /L, a hematocrit of 39.8%, a platelet count of 289×10^9 /L, Sr. creatinine of 0.93mg/dl, sodium of 134 mmol/L, and potassium of 3.74 mmol/L.

An arterial blood gas analysis revealed a pH of 7.436, $PaCO_2$ of 40 mmHg, PaO_2 of 145 mmHg and HCO_3^- of 26.0 mmol/L; FMethHb 15.5%, FO_2Hb 78.8% on nasal prongs with O_2 delivery of 4L/min. Chest radiography showed bilateral exaggerated lung markings.12 lead ECG was normal.

Patient was assessed for airway, breathing and circulation. Initial fluid resuscitation with normal saline was started at 120ml/hr. Empirical broad spectrum antibiotic therapy initiated as per suspected aspiration. Gastroenterologist team reviews the patient and emergency endoscopy revealed grade I erosive injury (fig. 2). Proton pump inhibitor infusion (Esmoperazole) was started at the rate of 8 mg/hr. Patient was catheterized with no. 14 foley's catheter; dark brown colour urine output of around 300 ml was noted just after 30 mins of catheterization. However, respiratory distress developed soon after admission to the intensive care unit, and reduction in SpO_2 to 75% was noted. Immediately ABG was done that showed pH of 7.475, $PaCo_2$ of 35.3 mmHg, PaO_2 of 192 mmHg, HCO_3^- of 26.0 mmol/L, and FO_2Hb of 75.1% with MethHb 19.6%. Patient was still consciousness and following verbal commands but the response was dull.

Patient was initially taken on Bipap support to manage respiratory distress. In view of high MethHb level, patient was planned for antidote administration. However patient was on antipsychotics- anti depression drug therapy, so in view of high risk of serotonin syndrome with administration of antidote (methylene blue) and middle age female sex was considered, secondly availability of hyperbaric oxygen therapy in our institution was considered and finally hyperbaric oxygen therapy administration for 2 hours was planned.

After 2 hrs of therapy, patient gradually started to show signs of improvement, and the SpO_2 increased to 86–89%. MethHb level drop down to 16.2%. Weaning from BiPap support started. A repeat hyperbaric oxygen therapy was given next day for 2 hour twice in a day and the SpO_2 improved further to 95–98%. After the second session of the hyperbaric oxygen therapy, MethHb level was found to have dropped to 14.8%. Urine output improved and colour started to lighten. Serial ABG examination (Table 1) showed improvement in level of SpO2 and decrease in level of MethHb. Hyperbaric oxygen therapy was continued for 3 days with successive monitoring of MethHB. In total, 6 sessions of hyperbaric oxygen therapy were given.

In addition, a psychiatrist was consulted in order to carry out a suicide risk assessment. Psychiatric clinic follow-up was advised. On day 4, the patient was transferred to a general ward in a stable condition, with no respiratory distress, urine output normal in amount and colour, weaned from oxygen therapy, antibiotics de-escalated and shifted on oral medications. She was discharged from hospital after 3 days in stable condition.

Table-1: Serial ABG Examinations

Time of ABG	pH	pCO_2 (mmHg)	PO_2 (mmHg)	HCO_3 (mmol/l)	sO_2 %	FO_2Hb %	FMethHb %	FCOHb %	Lactate (mmol/l)
At admission	7.346	40.5	145.8	26.6	96.8	78.8	15.5	3.1	1.4
After 2 hrs	7.475	35.3	192.7	25.4	95.5	75.1	19.6	1.8	2.77
After 1st HBOT	7.424	33	300.4	22.5	95.8	79.9	16.2	0.4	1.49
After 2nd HBOT	7.508	36.5	125.4	28.1	97.3	82.7	14.8	0.3	1.16
After 4th HBOT	7.471	40.5	114.7	28.9	98.6	85.8	8.6	0.4	0.55

Fig-1: Poison

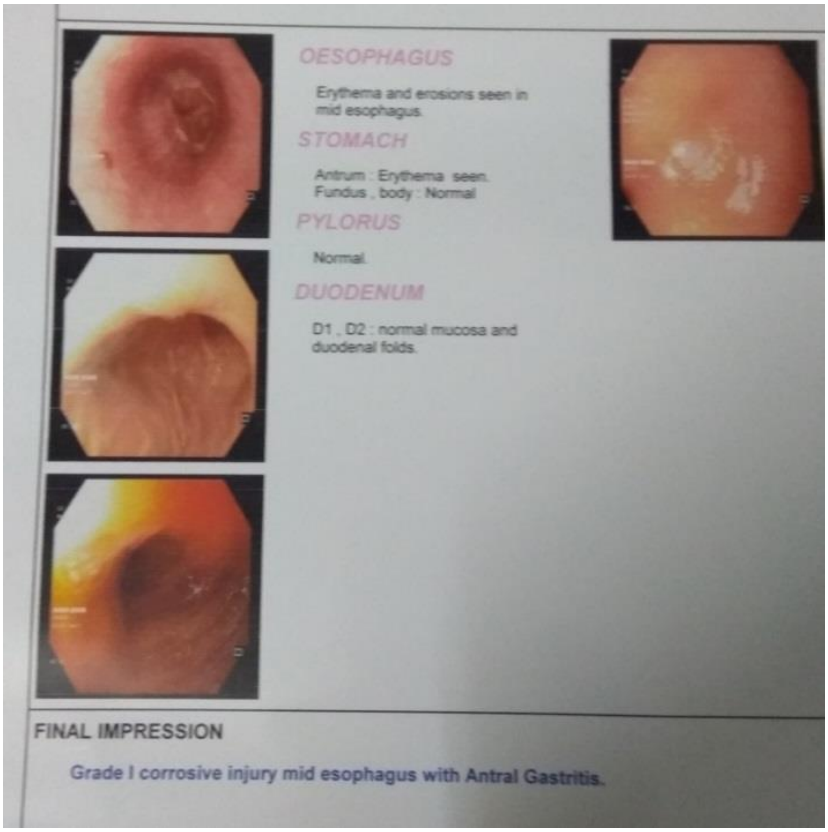

Fig-2: Endoscopy report

DISCUSSION

Symptomatic phenol poisoning can result from the intentional ingestion, occupational exposure, parenteral administration and dermal contact. Gastrointestinal and dermal absorption can result in significant morbidity and mortality. Phenols after penetration to 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 enzymes [2].

Systemic manifestation includes CNS depression, seizure, lethargy, coma, arrhythmias and hypotension. Metabolic acidosis, methemoglobinemia, hypothermia also can get manifested. The unconjugated phenol can get excreted through kidney causing damage to glomeruli and renal tubules leading to tubular injury.

Management of systemic poisoning includes management of hypotension, arrhythmias and convulsions along with assisted ventilation if necessary. There is no antidote for phenol. Patients should be checked for methemoglobinemia. Symptomatic patients should be treated. Methylene blue is the primary emergency treatment for documented symptomatic methemoglobinemia [3]. It is given in a dose of 1-2 mg/kg (up to a total of 50 mg in adults, adolescents, and older children) as a 1% solution in IV saline over 3-5 minutes. Administration may be repeated at 1 mg/kg every 30 minutes as necessary to control symptoms. Methylene blue is itself an oxidant at doses greater than 7 mg/kg and thus may cause methemoglobinemia in susceptible patients; hence, careful administration is essential. Methylene blue is contraindicated in patients with G6PD deficiency. Because it requires G6PD to work, it is ineffective in G6PD-deficient patients with methemoglobinemia. Additionally, methylene blue administration may cause hemolysis in these patients [4]. The US Food and Drug Administration (FDA) warn against using methylene blue concurrently with serotonergic psychiatric drugs, unless such usage is indicated for life-threatening or urgent conditions [5]. Methylene blue may increase central nervous system (CNS) serotonin levels as a result of monoamine oxidase (MAO)-An inhibition, thus increasing the risk of serotonin syndrome [6].

Exchange transfusion (which replaces abnormal hemoglobin with normal hemoglobin) may be considered for G6PD-deficient patients who are severely symptomatic or unresponsive to methylene blue.

Hyperbaric oxygen treatment is another option for situations where methylene blue therapy is ineffective or contraindicated. This approach permits tissue oxygenation to occur through oxygen dissolved in plasma, rather than through hemoglobin-bound oxygen [7]. Therapeutic mechanisms of action for hyperbaric oxygen therapy are based on elevation of both the partial pressure of inspired O_2 and of the hydrostatic pressure [8].

N-acetylcysteine has been shown to reduce methemoglobin in some studies but is not currently an approved treatment for methemoglobinemia.

CONCLUSION

Prolonged exposure to phenol may result in major absorption and a long elimination half-life and can cause significant morbidity and mortality. This case report highlights the importance of considering the possibility of methemoglobinemia in cases of exposure to phenolic compounds and its early recognition and management. Consideration of serotonin syndrome and use of hyperbaric oxygen therapy was unique in our case. Fortunately, the patient was successfully treated with hyperbaric oxygen therapy before catastrophic results had occurred. Limitation of availability of hyperbaric oxygen therapy units is major limitation in such cases.

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