Fatal poisoning caused by oral ingestion of a hair dye
R Verma, N Tewari, S Jaiswal, V Rastogi, D Singh, A Tiwari

Citation

Abstract
We have presented a case of a 20-year-old female who ingested about 100ml of a hair dye (Vasmol 33™) with suicidal intent. Information on the systemic effects of hair dye in acute poisoning after oral ingestion is limited. Hair dyes mainly contain PPD and resorcinol.

The important clinical and laboratory findings were, unconsciousness, severe cervical and facial edema, respiratory failure that required mechanical ventilation, generalized tonic-clonic convulsions, severe metabolic acidosis, intravascular hemolysis, rhabdomyolysis, methemoglobinemia and oliguria, culminating in renal failure. The patient died following cardiac arrest.

INTRODUCTION
Super Vasmol 33™ is an emulsion based hair dye commonly used in India. The main ingredients of the dye are, paraphenylenediamine(< 4%), resorcinol, propylene glycol, liquid paraffin, cetostearyl alcohol, sodium lauryl sulfate, EDTA sodium, herbal extracts and preservatives and perfumes.

Some of these ingredients are known toxicants with multi-organ effects, while the toxicity profiles of others are not known.

The cosmetic trade in oxidizing hair dyes uses Paraphenylenediamine (PPD); it is also commonly used to intensify the color of henna (Lawsonia Alba). Though rare in the West, ingestion of PPD is frequently reported from Africa and Asia. Accidental or suicidal ingestion of PPD causes systemic toxicity, manifested by severe edema of neck and face and laryngeal edema with respiratory distress frequently requiring emergency tracheotomy and mechanical ventilation. It also causes rhabdomyolysis and acute renal failure, culminating in death if not treated aggressively.

Resorcinol is a phenolic chemical used in photography, tanning and cosmetics (hair dye) industry. It is also a pharmaceutical agent used topically in skin diseases. Resorcinol is a moderately toxic and corrosive chemical. After oral administration, resorcinol is readily absorbed from the gastrointestinal tract, metabolized, and excreted by male and female rats, indicating little potential for bioaccumulation in animal tissues. It is known to cause eye, skin, oral and gastrointestinal injuries. Systemic toxicity is manifested as vomiting, dyspnea, methemoglobinemia, hypothermia, tachypnea, pallor, profuse sweating, hypotension and tachycardia. Here we report a case of suicidal ingestion of this hair dye, resulting in rapidly developing massive edema of neck and face, respiratory distress, seizures and death due to renal failure.

CASE REPORT
A 20-yr-old girl was admitted to the emergency department of our hospital at 4 pm, alleged to have ingested about 100 ml of hair dye (Super Vasmol 33™), an hour ago. She was conscious at the time of admission, responding to verbal commands. Gastric lavage was performed and activated charcoal was given in the emergency department.

The patient was shifted to ICU at 6.00 pm, with severe face and neck swelling, respiratory distress, convulsions and loss of consciousness. She was gasping and developed bradycardia. She was intubated and 0.6 mg of atropine was given IV.

The patient was put on ventilator in SIMV mode. Her pupils were normal in size and reacting to light. She was afebrile with pulse rate 132/min, BP 118/80 mm Hg, SpO2- 96% (FiO2= 80%). Auscultation of lungs and heart revealed no abnormality.

The patient soon developed generalized tonic-clonic
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convulsions and her deep tendon reflexes were absent. ECG showed normal sinus rhythm with a pulse rate of 120.

The first arterial blood gas analysis (immediately after starting ventilation) showed, pH 7.03, pCO₂ 52 mm Hg, pO₂ 180 mm Hg (FiO₂ 80% ), HCO₃ 13.4 mmol/L, base excess (BE) -17.1 mmol/L. Serum electrolytes were, Na 134 meq/L, K 3.3 meq/L, Ca/iCa 1.06.

Laboratory data revealed, hemoglobin 12.4 g/dl, TLC 17700/ml, P 71%, L 25%, E 2%, M 2% ; platelet count was 310, 000/ml. Blood urea was 34.6 mg/dl and serum creatinine, 1.2 mg/dl. Liver function tests showed serum bilirubin 1 mg/dl, SGOT 438 IU/L, SGPT 214 U/L, Alkaline phosphatase 144 U/L.

Total serum protein was 6.6g%, serum albumin 4.4g%. Blood glucose and uric acid were normal. Routine urinalysis reported, (pH 6.6, protein 28 mg/dL, and no ketones, blood, bilirubin and nitrite). CPK was not elevated and urine was negative for myoglobin.

Mechanical ventilation was continued. For treatment of generalized clonic-tonic seizures, midazolam was given IV. Due to recurrent seizures even after midazolam, a loading dose of phenytoin was followed by continuous infusion. To alleviate metabolic acidosis, NaHCO₃ infusion was given under arterial blood gas control.

A central line was established. Foley's catheter was inserted to monitor urine output. At the time of catheterization, urine output was 1200ml; in the next 24 hours total urine output was 700 ml. The patient gradually developed oliguria, reflected by the decreasing urine output on second and third day(850ml and 360 ml respectively).

Further treatment comprised intravenous fluids, broad-spectrum antibiotics, bicarbonate replacement and H₂ blockers. Since the patient did not respond to phenytoin infusion, sodium valproate was administered, which was also was ineffective. Ultimately, the seizures were controlled by thiopentone infusion.

In view of the gradually developing oliguria and increasing serum urea and creatinine levels, the patient was put on peritoneal dialysis on the third day. The patient also developed pulmonary edema and hypotension and was supported with ionotropes. Despite vigorous treatments, metabolic acidosis persisted; the patient could not maintain normal SaO₂ even after ventilation with 100% oxygen.

Cardiac arrest occurred and despite cardiopulmonary resuscitation, the patient died on the morning of 4th day.

Autopsy revealed diffuse pulmonary edema, renal congestion, an eosinophilic substance in renal cortical tubular lumen and hyperemia in all abdominal organs. Renal collecting tubule and distal tubule epithelium was seen to be necrotic. The blood showed methemoglobinemia (10%).

DISCUSSION

Though hair dye poisoning is rare in western countries, it is common in eastern Africa, Middle East countries and Indian sub continent. Here we have presented the key clinical manifestations of systemic hair dye poisoning. Common hair dye ingredients used are, hydrogen peroxide, PPD, resorcinol or aminophenols. But the manufacturer of Vasmol claims that no hydrogen peroxide or ammonia were added. Although there are a few studies that indicate systemic toxic effects of PPD on humans, information on the effects of resorcinol in acute poisoning after oral ingestion is limited. In our report, the patient developed symptoms common to both resorcinol and PPD poisoning.

Resorcinol is of moderate acute toxicity. Studies have reported rapid absorption of resorcinol from GI tract following its oral ingestion. It is rapidly metabolized and excreted.

Death, following resorcinol ingestion is associated with, convulsions, salivation, dyspnea, emaciation and hyperemia of the GI tract. The lowest lethal dose (LDL) of resorcinol in humans has been reported as 29 mg/kg body wt. Studies report that systemic manifestations of resorcinol poisoning may include nausea, dyspnea, methemoglobinemia, tachypnea, pallor and profuse sweating, with hypotension and tachycardia. Pulmonary edema and bronchospasm may also occur. Since respiratory arrest occurred in our patient 1hr 30 min after ingestion, she had to be intubated, pulmonary edema was later seen in autopsy findings.

Resorcinol is also neurotoxic and its acute exposure effects range from seizures, followed by CNS depression to lethargy, coma and death. Our patient developed generalized tonic-clonic convulsions. She was unresponsive to major antiepileptic drug infusions, and the seizures were finally controlled only with sodium thiopentone. In the blood sample taken at the autopsy, a high level of methemoglobin was found.

Paraphenylenediamine (PPD) is a key ingredient of hair
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dyes, used for color enhancement. Only few studies have been done on systemic PPD poisoning. Common clinical manifestations of systemic toxicity are, cervicofacial edema, chocolate brown colored urine, upper airway tract edema, oliguria, muscular edema and shock. The biological results were dominated by rhabdomyolysis, metabolic acidosis, acute renal failure and hyperkalemia.

On arrival, our patient showed characteristic face and neck swelling, dyspnea, following which she was intubated. She presented diagnostic chocolate brown colored urine and gradually developed oliguria due to impaired renal function.

She suffered from severe metabolic acidosis and had increased level of potassium in blood. Treatment with hydrocortisone, antihistaminic drugs, broad spectrum antibiotic and metabolic acidosis correction did not prove effective. The patient died after 72 hours.

The patient presented with the mixed symptoms of resorcinol and PPD poisoning, as shown by clinical presentations, lab and autopsy findings. Thus clinical manifestations of hair dye poisoning were associated with respiratory, neurological, renal and hematologic symptoms.

CORRESPONDENCE TO
Dr. Sushil Jaiswal, M.D., Junior Resident, Dept. of Anesthesiology Institute of Medical Sciences Banaras Hindu University, Varanasi 221005 Phone 0542- 2309310 , Cell: 09889566818,09889284878) E-mail: drsushiljswl@rediffmail.com

References
Author Information

Ravi Verma, M.D.
Department of Anesthesiology, Institute of Medical Sciences, Banaras Hindu University

Nidhi Tewari, M.Sc.
Department of Anesthesiology, Institute of Medical Sciences, Banaras Hindu University

Sushil Jaiswal, M.D.
Department of Anesthesiology, Institute of Medical Sciences, Banaras Hindu University

Virendra Rastogi, M.D.
Department of Anesthesiology, Institute of Medical Sciences, Banaras Hindu University

Dinesh Kumar Singh, M.D.
Department of Anesthesiology, Institute of Medical Sciences, Banaras Hindu University

Arun Tiwari, M.D.
Department of Anesthesiology, Institute of Medical Sciences, Banaras Hindu University