

Case Article**Hair Dye Poisoning: Case Report and Review of Literature**

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ABSTRACT

Background: Hair dye poisoning, with main toxic component paraphenylene diamine, is a medical emergency. It is on increasing trend in developing countries due to wide availability and low cost of hair dyes. It carries a high mortality and does not have any specific antidote.

Case Report: Twenty three years old patient presented with breathlessness along with difficulty in speaking for one hour after consuming hair dye. He developed cervicofacial edema followed by rhabdomyolysis, myoglobinuria and acute kidney injury, classical of hair dye poisoning. Laboratory investigations revealed blood urea of 100 mg/dl and serum creatinine of 3.8 mg/dl. The serum creatinine phosphokinase (CPK) levels were 1230 U/L and urine myoglobin was positive. He was treated conservatively for acute kidney injury and discharged in stable condition.

Conclusion: Early diagnosis and treatment of poisoning due to hair dyes leads to improved prognosis, so widespread awareness is needed about this emerging form of poisoning.

Keywords: Acute Kidney Injury, Hair Dye, Para-Phenylene Diamine, Rhabdomyolysis.

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INTRODUCTION

Hair dye poisoning is an emerging etiological factor for suicides in developing countries. Main toxic component of hair dye is paraphenylene diamine (PPD). Majority of hair dye poisoning are suicidal in nature [1]. A strong suspicion for hair dye poisoning should be kept when clinical picture includes a triad of cervicofacial edema, rhabdomyolysis and acute renal failure. The characteristic chocolate brown colour of the urine and confirmation of hair dye in urine by thin layer chromatography could be confirmative evidence of hair dye poisoning due to PPD [2].

CASE REPORT

A 23-year-old married male, not a known case of any chronic illness but disturbed family life presented in Accident & Emergency Department with history of suicidal consumption of 100 ml of hair dye along with approximate 200 ml of country alcohol four hours back. He had shortness of breath along with difficulty in speaking for last one hour. Relatives also complained of swelling of the face and neck of the patient. There was no history of altered sensorium or seizure at the time of presentation. No history of discoloured urine, decreased urine output, vomiting or disturbance in the vision. Clinical

examination revealed had facial swelling with edema of lips, swollen neck and tongue. Stridor was present.

The pulse rate was 108/min, blood pressure was 110/80 mmHg, and SpO₂ was 98% with oxygen by mask. The cardiovascular system and respiratory system examinations were essentially normal. Per abdomen examination revealed mild epigastric tenderness without any organomegaly. There was no neurological deficit. In view of stridor and facial puffiness patient was treated with injection hydrocortisone 100 mg every 8 hourly along with pantoprazole and antihistamines for angioneurotic edema. Patient got relieved of symptoms within next 6 hours. On procuring fresh packet the content of hair dye was para phenylene diamine, propylene glycol resorcinol, sodium lauryl sulphate liquid, light liquid paraffin, herbal extracts along with preservative and perfumes.

On the following day, the patient developed dark coloured urine with a decreased urine output and pedal edema. The laboratory investigations revealed blood urea of 100 mg/dl and creatinine of 3.8 mg/dl. The serum creatinine phosphokinase (CPK) levels were 1230 U/L and urine myoglobin was positive. The liver function tests were normal and peripheral smear did not reveal evidence of hemolysis. Blood gas analysis showed mild metabolic acidosis with raised anion gap.

Ultrasonography of abdomen showed normal size and echo texture of both kidneys. The chest x-ray and electrocardiogram were also normal. Patient was haemodynamically stable and treated on conservative lines for acute kidney injury along with forced alkaline diuresis. His renal function improved on 5th day and was discharged in stable condition.

The authors committed to the Helsinki Convention at all stages of the investigation. An informed consent form was taken from the patient.

DISCUSSION

The art of modifying hair colour or hair dyeing can be traced back to ancient times when dyes used were obtained from plants and other natural products like henna, indigo, senna and amla. However, use of synthetic hair dyes started from 1860s when reactivity of PPD with air was discovered [2]. Normally henna takes 6 – 8 h for dyeing. PPD accelerates this process and deepens the colour when mixed with henna. PPD accelerates the dyeing process. Hair dye poisoning is an emerging etiological factor for suicidal attempts in developing countries like India. The factors leading to its increased use in suicide attempts are its easy availability and low cost.

First case of PPD poisoning was reported in 1924 in a hair dresser due to accidental exposure [3]. Hair dyes generally are a blend of various chemicals like paraphenylene diamine, resorcinol, polyethylene glycol, bismuth citrate, lead acetate etc. The main toxic component is PPD which is a coal tar derivative. Highly mutagenic and toxic bondrowski's base is produced on oxidation of PPD. This base is responsible for the toxic effects of PPD [4]. It is also speculated that PPD causes free radical damage and reduced glutathione stores [5]. It can cause local manifestations like skin irritation, contact dermatitis, chemosis and lacrimation. This compound is also known to cause various systemic manifestations like mild angioneurotic edema to rhabdomyolysis, intravascular hemolysis, and acute kidney injury. The lethal dose of PPD is about 3 gm [6].

Early phase of PPD poisoning includes cervico-facial and laryngeal edema followed later by rhabdomyolysis and acute kidney injury. PPD leads to leakage of calcium from smooth endoplasmic reticulum and promotes calcium release thereby leading to persistent muscle contraction and irreversible changes in the muscle

followed by rhabdomyolysis [7]. Rhabdomyolysis causing myoglobinuria is the main cause of acute renal failure. Occurrence of renal failure suggest poor prognosis. However, hypovolemia and direct toxic effects also contribute to acute kidney injury. Histologic findings are suggestive of acute tubular necrosis [8]. PPD also causes inflammatory edema of larynx and tongue, due to toxic effects on mucosa, leading to asphyxia. PPD ingestion can also lead to myocarditis and arrhythmias [9]. Polyethylene glycol also causes hyperosmolality, acute tubular necrosis and anion gap metabolic acidosis contributing further to the renal injury [10]. Resorcinol, a phenol derivative, contributes to neurotoxicity caused by hair dye poisoning [11]. Lead acetate and bismuth citrate lead to chronic kidney disease and acute interstitial nephritis respectively [10].

Hair dye poisoning should be strongly suspected when presentation includes a triad of angioedema causing asphyxia, rhabdomyolysis with evidence of myoglobinuria and acute renal failure. In a study conducted on 19 patients with PPD poisoning, clinical picture was dominated by cervicofacial edema and chocolate brown colored urine [12]. In another study out of 150 studied patients with PPD poisoning, 60% developed acute renal failure requiring dialysis [13]. Our patient presented with cervicofacial edema followed by rhabdomyolysis, myoglobinuria and acute kidney injury.

Hair dye poisoning is a medical emergency. PPD has no antidote and is not dialyzable [14]. Treatment is symptomatic. Initial resuscitation involves securing the airway by endotracheal intubation/tracheostomy if laryngeal edema develops. Alkaline diuresis and haemodialysis has an important role on treatment of rhabdomyolysis. Mortality ranges from 0.3% to 60% [5].

CONCLUSION

Hair dye poisoning is a common form of poisoning in India due to its wide availability and low cost. It carries a high mortality and no specific antidote is available. Lack of specific diagnostic tests and antidote emphasizes the importance of early diagnosis. Strong suspicion, early diagnosis and treatment lead to improved prognosis so widespread awareness is needed about this emerging form of poisoning in developing countries.

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