

## CASE REPORT

# Methylprednisolone in hair dye poisoning

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## ABSTRACT

Poisoning due to hair dye consumption is relatively rare in India. Compound responsible for hair dye toxicity is paraphenylenediamine (PPD). Hair dye (PPD) poisoning has high morbidity and mortality and its incidence has increased dramatically in the past 4 years. Prompt recognition and effective management lead to complete recovery. Diagnosis is made solely on the basis of the history given by the attendants and symptoms of cervicofacial edema, black colored urine and muscular pain. The key to successful management is detection and avoidance of triggers, early recognition of attacks, and aggressive airway management when warranted. Initial treatment in a patient presenting with most forms of angioedema includes antihistamines and glucocorticoids if required. Epinephrine should be administered if there is a concern for laryngeal edema.

We present here a case report of hair dye poisoning where patient presented as cervicofacial edema not responding to conventional treatment, but responded to methylprednisolone intravenously.

**Key words:** Angioedema; Hair dye poisoning; Paraphenylenediamine; PPD; Methylprednisolone

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## INTRODUCTION

Hair dye is available in several forms and the commonest cheap form is stone hair dye which is available in 20 gm pack. Other branded hair dyes like 'Godrej', Kesh kala, colour mate etc. are available in powder or liquid forms. The concentration of active substance para phenyldiamine, varies from 70 to 90% in stone hair dye and 2 to 10% in branded dyes, which are used for coloring the grey hair black. The stone hair dye is very cheap and freely available, making it an attractive option for suicidal intent. It is brownish to black colored solid which is partially soluble in water and easily soluble in hydrogen peroxide. PPD is a good hydrogen donor and is metabolized by electron oxidation to an active radical by cytochrome P<sub>450</sub> peroxidase to form a reactive benzoquinone diamine. This is further oxidized to a trimer known as Brandowaski's base, a compound reported to cause anaphylaxis as well as being strongly mutagenic.

Oral ingestion of PPD causes mainly two types of toxic effects; the first manifestation is angioneurotic edema presenting as rapid development of severe edema of the face, neck, pharynx, tongue and larynx with respiratory

distress sometimes requiring emergency tracheostomy. The time of onset of symptoms after ingestion was about 4 to 6 h and at the time of admission, majority of the patients are presented with acute angioneurotic edema of the head and neck and wooden hard swollen protruded tongue with swelling of pharynx and larynx, and these are typical diagnostic feature of acute hair dye poisoning. Initial insult is due to local irritation of the mucous membrane and the skin, causing intense edema. Edema of the eyes may be very severe and may cause exophthalmos. Muscle pain was the next most common presentation of hair dye poisoning following angioneurotic edema. Limbs are usually swollen, tender and stiff. Muscle pain typically occurs about 10 to 12 h after ingestion of hair dye.

Acute hair dye poisoning leads to rhabdomyolysis and muscle necrosis. Patients pass chocolate brown colour urine which along with typical orofacial swelling is diagnostic of acute hair dye poisoning specially in those cases where history of dye ingestion is lacking. This chocolate brown colour of urine is due to the presence of myoglobin and hemoglobinuria. In the later phase, rhabdomyolysis and acute tubular necrosis supervene. The myoglobin is released because of rhabdomyolysis and muscle necrosis

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which reach the renal tubules and clog them, leading to pathologic features of acute tubular necrosis responsible for oliguria and acute renal failure. Acute renal failure was seen as the late cause of death in acute hair dye poisoning cases. Tubular obstruction by myoglobin casts is regarded as the principal mechanism for producing ARF.

There is no antidote for PPD poisoning. If the poisoning is not recognized early, it has a very high mortality. We report a patient who suffered from hair dye poisoning. She presented with cervicofacial and lingual edema and recovered within a week.

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An 18 years old female weighing 50 kg with the history of hair dye ingestion presented to casualty department with complaints of respiratory distress and agitated behaviour. General physical examination revealed a pulse rate of 130/min, BP 100/50 mmHg and bilateral conducting sounds on chest auscultation. Gastric lavage was done. Within ½ an hour, patient developed facial and periorbital edema with stridor. Patient was intubated immediately using 6.5 mm cuffed endotracheal tube (Figure 1). At that time her physical examination revealed a pulse rate of 146/min and BP 70/36 mmHg. Diagnosis of angioedema with anaphylactic shock was made. She was given inj. chlorpheniramine 50 mg, inj. hydrocortisone 300 mg, inj. dexamethasone 8 mg and inj. adrenaline 1 mg subcutaneously stat and inj. dopamine infusion was started at 6 µg/kg/min. The patient was immediately shifted to RICU and put on ventilatory support with SIMV mode, tidal volume 380 ml/min, frequency 12/min, FiO<sub>2</sub> 60%.

Standard monitoring for electrocardiography, heart rate,



**Figure 1: Patient on arrival with gross facial, periorbital edema**

oxygen saturation and non-invasive blood pressure was applied. Nebulization, using 0.25 ml 1:100 adrenaline added to 2 ml isotonic saline, was done 6 hourly. Volume resuscitation was done using colloid and crystalloid solutions. She was catheterized and forced diuresis was done. Under aseptic precautions, a triple lumen central line was inserted in right subclavian vein and CVP guided fluid given. She was put on inj. hydrocortisone 100 mg 8 hourly, inj. metrogyl 100 ml 8 hourly, inj. ranitidine 12 hourly, inj. furosemide 20 mg 12 hourly, inj. chlorpheniramine 50 mg 8 hourly and inj. sodium bicarbonate 20 meq 8 hourly. Slowly inj. dopamine infusion was tapered off and inj. dobutamine infusion was started at 5 µg/kg/min. Even after 18 hours of treatment, her facial, periorbital edema did not resolve, so she was put on inj. methylprednisolone 125 mg IV 6 hourly and nebulisation was done every four hours. She responded to treatment, so dobutamine support was tapered off gradually and stopped. Edema of her facial, periorbital and neck soft tissues reduced. After 36 hours patient started responding to verbal commands, maintaining her vital signs at HR- 106/min and BP 104/60 mmHg. Her chest remained clear bilaterally. She was extubated on 3<sup>rd</sup> day and monitored for next 12 hours. She did not develop any further complications. Her urine output was adequate. Urine color was initially dark chocolate colored, and it became clear by the end of 3<sup>rd</sup> day. She was shifted back to the ward on 4<sup>th</sup> day.

### DISCUSSION

Compound responsible for hair dye toxicity is PPD. PPD imparts black colour to hair dye. It is also used in tattoos and (black) henna. PPD is used because it is cheap, has high temperature stability, high strength, chemical and electrical resistance. Poisoning due to PPD is reported in northern Africa and Arabia due to local custom of using henna (black). A few cases are reported from north India. This poison can be Poisoning can be accidental, suicidal or homicidal. It is also used as abortifacient.

Systemic toxicity occurs in two phases. Phase 1 is an acute presentation with edema of the neck, airway obstruction, gastritis and severe vomiting while phase 2 is a subacute presentation with acute renal failure, rhabdomyolysis and hemolysis.

PPD applied locally can cause contact dermatitis. Transcutaneous absorption of PPD can be rapid and may lead to systemic effects including angioedema, gastrointestinal disturbances, tremors, drowsiness, convulsions, dyspnea, liver atrophy, acute renal failure, cardiac arrest and death in few cases. However, systemic effects develop following chronic topical application. Chronic dermal exposure can cause lethargy, myalgia, purplish discoloration of gums and teeth, anorexia, GIT disturbances, liver and spleen enlargement, subacute atrophy of the liver, jaundice,

CRF, progressive neurological symptoms and coma. Poisoning due to PPD has high mortality. Therefore, early recognition can be life saving. There is no specific antidote. The most important aspect of management is early recognition of poisoning by this compound, supportive measures that include gastric lavage with 2% soda bicarbonate and alkalinization of urine. Asphyxia is the major early challenge, which may require ventilatory support. Drugs used include hydrocortisone, antihistaminics and vasopressors. Renal support in the form of dialysis is required in ARF. Supportive management is life saving if instituted early in course as little delay is disastrous leading to death. Diagnosis is easy to make but requires a higher degree of suspicion as clinical features are quite distinctive that is, orofacial edema, chocolate brown urine and history of PPD intake. Till now, no definite guideline of management has been given, despite exhaustive literature search. As a result, we formulated our own line of management. After a quick clinical examination, special attention was given to vital parameters. As the immediate cause of death was hypoxia, airway patency was maintained by oral airway, endotracheal intubation and emergency tracheostomy. Circulatory volume and pressure were maintained by giving appropriate fluid therapy. The treatment was based on the following principles:

1. Since no antidote is available against PPD, management was basically supportive.
2. Gastric lavage was done with activated charcoal and tap water.
3. Cases where angioedema progresses rapidly should be treated as a medical emergency as airway obstruction and suffocation can occur. In severe cases, stridor of the airway occurs, with gasping or wheezy inspiratory breath sounds and decreasing oxygen levels. Tracheal intubation is required in these situations to prevent respiratory arrest and risk of death. Asphyxiation due to laryngeal edema yields a 3-40% mortality rate. Airway swelling can make intubation difficult; the risk is increased for vocal cord damage during intubation. Of angioedema presented in the emergency room, 10-25% of cases are considered to be

life threatening.

4. Intravenous hydrocortisone (300 mg stat dose then 100 mg every 6 h) is used till angioneurotic edema subsides. This is the mainstay of treatment for angioneurotic edema which decreases mortality and morbidity later on. But this case did not respond to intravenous hydrocortisone rather responded to methylprednisolone 125 mg 6 hourly as IV infusion for 5 days. It has shown promising results and leading to decreased morbidity and mortality as evidenced by early subsidence of angioneurotic edema.
5. Sodium bicarbonate 20 meq 8 hourly was administered to prevent myoglobin precipitation in kidney along with loop diuretics (furosemide or torsemide) to maintain adequate urine volume.
6. Chlorpheniramine maleate (25-50 mg IV every 8 h) was used till orofacial edema subsided (average 3-5 days).
7. Vasopressors (dopamine and/or dobutamine) were used till hypotension persisted, despite adequate fluid therapy.

Time elapsed in seeking medical care and early management influence the mortality and morbidity. Early clinical diagnosis and early intervention are the cornerstones of management. Respiratory failure mainly determined the short term prognosis, whereas long term prognosis was affected by the importance of muscular and renal damage. Good results with methylprednisolone in marked cervicofacial edema were seen. It reduced edema markedly and improved associated respiratory distress. Preventing renal failure was a very important goal in systemic PPD intoxication, since its occurrence was associated with high mortality and an increase in ICU stay. Prevention was based on abundant fluid infusion, alkalinization of urine and the correction of the hemodynamic disturbances. Cases who consumed up to 10 gm of PPD usually survived if they are presented to hospital within 4 hours of dye ingestion in whom proper management can be delivered in the form of methyl prednisolone and other supportive measures. Severe edema of face, neck and floor of mouth, renal failure and myocarditis are poor prognostic factors.

It is important that medical fraternity should be aware of this poison because the poison is available quite freely and used extensively.

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