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Hair Dye Poisoning: a Case Report

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Abstract

Hair dye ingestion is frequently reported from Africa, the Middle-East, and the Indian subcontinent. The main component of hair dye causing toxicity is Paraphenylenediamine (PPD). This compound causes contact dermatitis in susceptible individuals,but when ingested it has been found to cause angioneurotic edema, rhabdomyolysis and renal failure. We report a case of paraphenylenediamine poisoning.

Key Words : Paraphenylenediamine, hair dye poisoning

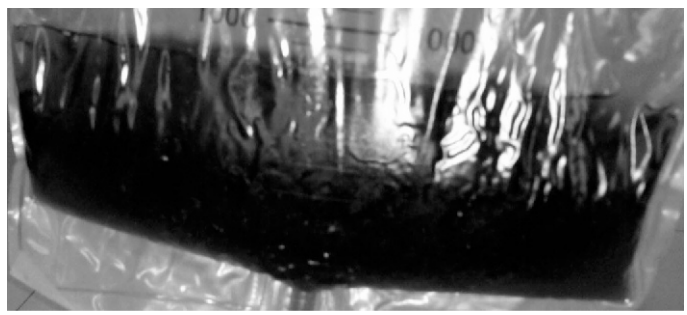
Introduction : Paraphenylenediamine(PPD), is a derivative of paranitroanailine.[1] Complex reaction takes place and several intermediates are produced on oxidation of PPD. The symptoms are considered to be dose related and patients with ingestion of larger amounts of PPD have higher morbidity and mortality. The toxicity of PPD usage includes skin irritation, contact dermatitis, chemosis, lacrimation, exophthalmos, or even permanent blindness, due to local contact. But ingestion of PPD produces two types of toxic effects. The first consists of rapid development of severe oedema of the face, neck, pharynx, tongue, and larynx with respiratory distress, often requiring tracheostomy[2]. In the later phase, rhabdomyolysis and acute tubular necrosis can occur.Vomiting, gastritis, hypertension, vertigo, tremors, and convulsions have been reported. We report a case of intentional ingestion of hair dye.

Case Report : A 21 year old lady had consumed around 40-50ml of hair dye-Super Vasmol 33 with the intention of self harm. Following that she developed pain abdomen and vomiting. She was initially taken to a local hospital, where she had cardiopulmonary arrest and was successfully resuscitated. This was followed by status epilepticus. She was then referred to our hospital for further management. At admission she had continuous generalized tonic-clonic seizures.

On examination whole face was oedematous, pulse was 130/ minute, BP was 124/60 mm Hg with ionotropic support(noradrenaline). Crepitus was present over neck and anterior part of chest. GCS was E1M1Vt .Other systemic examination were normal.Urine was chocolate brown.Blood investigations showed leukocytosis (44,100), hyperkalemia (5.2meq/l) mild conjugated hyperbilirubinemia (Total bilirubin 0.7mg/dl and Direct ,bilirubin 0.4mg/dl), grossly raised liver enzymes (SGOT-1192.5 and SGPT-883.9), prolonged INR(1.5), hypocalcemia(7.0 mg/dl), raised CPK(3342 U/L), normal levels of Hemoglobin, random blood sugar, urea, creatinine, serum sodium, magnesium, albumin and globulin.

Arterial blood gas analysis showed compensated mild metabolic acidosis.

Urinalysis showed chocolate brown urine which tested positive for blood,nitrates and protein.



Chest X ray revealed surgical emphysema over neck. CT Brain showed diffuse cerebral oedema.

Status epilepticus got controlled with intravenous phenytoin and levetiracetam. Angioneurotic edema responded well to intravenous hydrocortisone. Intravenous correction of calcium gluconate was given. The patient was well hydrated. During the stay she continued to be in shock and required continuous ionotropic and ventilatory support. There was no improvement in sensorium. The patient again had cardiopulmonary arrest on the sixth day of hospital stay and despite of our best efforts she couldn't be revived.

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Discussion :

The contents of this hair dye mainly includes PPD , sodium ethylene diamine tetra acetic acid(EDTA), resorcinol, propylene glycol and liquid paraffin.[3]

In 1924, Nott described the first case of systemic toxicity with PPD. Another report from Sudan described a series of cases of acute hair dye poisoning. Sood *et al* and Chugh *et al* have reported cases from India. The lethal dose of PPD is not known; estimates vary from 7 – 10 grams.[4] The renal involvement caused by PPD varies from transient protienuria to oliguric acute kidney injury. Passing of chocolate brown urine and acute renal failure is a consistent feature of PPD poisoning. The PPD has a direct toxic effect on kidney due to its aromatic structure, and hence high concentration in tubules. It can also cause acute tubular necrosis as a result of myoglobinuria due to rhabdomyolysis and hemoglobinuria due to hemolysis.Other reported features include anaemia, leukocytosis, haemoglobinaemia, and haemoglobinuria. Another hallmark is angioedema, well-recognized non-pitting edema involving the deeper layers of the skin, subcutaneous tissue, and mucosa that can lead to life threatening airway obstruction.[5] Liver necrosis has also been reported. Hypertension is generally seen, but presence of hypotension/shock is a poor prognostic indicator. The main toxic features include methemoglobinaemia, hoarseness of voice, cardiac toxicity, hepatitis, convulsions, coma and sudden cardiac death.[1] Diagnosis is by identifying the characteristic combination of clinical features of angioneurotic oedema of face, neck, and chest along with muscle tenderness, chocolate coloured urine, and acute renal failure. Confirmation can be obtained by gas chromatography mass spectrometry analysis of patient's urine.

The propylene glycol component can cause high anion gap metabolic acidosis and acute renal failure[6]. Also, the resorcinol component can cause methemoglobinemia and renal toxicity[7].

Our case is interesting because along with oedema of face and neck, she also had neurological manifestation and she did not have renal failure. Neurological manifestations like seizures, was similarly reported recently in a study conducted in New Delhi.[1] Our patient had hypocalcemia and status epilepticus. The cause of the hypocalcemia was thought to be sodium EDTA component of hair dye,and not due to rhabdomyolysis in this case. Seizures that developed initially can be due to toxins in the dye or hypocalcemia.

Since there is no specific antidote, the management is mainly supportive treatment.[8] The major early challenge is preservation of an open airway (by emergency tracheostomy, if needed). Maintenance of hydration, oxygenation, and a good urine output are absolutely essential. In the phase of ATN, haemodialysis is the modality of choice, although the toxin is not removed by dialysis. Renal dialysis is life saving when oliguria develops. Intravenous corticosteroids and antihistamines have been tried, but controlled studies are needed. Shock not responding to ionotropes worsens the prognosis.

Derivatives containing PPD is used extensively in our country and easily available so there may be many cases of accidental and intentional poisoning. Hence emergency physicians should be aware of various manifestations of this poisoning and high index of suspicion helps in early recognition and early intervention improving the outcome.

References :

1. Chugh KS. Malik OH. Singhal Pc. Acute renal failure following paraphenylene diamine(hair dye) poisoning: report of two cases Med 1982;13:131-7.
2. Ashraf W, Dawling S, Farrow LJ. Systemic paraphenylenediamine (PPD) poisoning: a case report and review. Hum Exp Toxicol 1994:13:167-70.
3. Bhargava P,Mathew P. Hair dye poisoning. J Assoc Physicians India 2007;55:871-2
4. Sumeet Singla, Sanjeev Miglani, A K Lal, Pulin Gupta, A K Agarwal. Paraphenylene diamine(PPD) poisoning.JIACM 2005; 6(3): 236-8
5. Barna IS. Frable MA. Life threatening angioedema. Otolaryngol Head Neck Surg 1990:95-8.
6. Sampathkumar K,Yesudas S. Hair dye poisoning and the developing world. J Emerg Trauma Shock 2009;2:129-31.
7. Blondet P, Roux P, Schandelong A, Legueuelt LC, Laine G. Methemoglobinemia and acute toxic hemolytic anaemia caused by benzocaine and resorcinol Press Med.1985;14:17-57.
8. Abdulraheem M, Hamdouk M, Ziijlstra E. Paraphenylene diamine Hair dye poisoning in children.Arab J Nephrol Transplant.2010;3:39-43.

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