Case Report

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Super Vasmol 33 (Hair Dye) Poison: A Case Report

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Abstract

Poisoning is one of the common modes of suicide in India. Hair dye poisoning is an uncommon form of poisoning in the west; however, in some parts of the world such as East Africa and Indian sub-continent it is not uncommon. The active ingredients causing toxicity includes paraphenylenediamine (PPD), propylene glycol, liquid paraffin, cetostearyl alcohol, sodium lauryl sulfate and resorcinol. There is no specific antidote for hair dye poisoning and treatment is mainly supportive.We report a case of suicidal ingestion of hair dye that presented with angioneurotic edema and rhabdomyolysis. Our patient improved completely with symptomatic management.

Keywords: Paraphenylenediamine (PPD); Angioneurotic Edema; Rhabdomyolysis; Symptomatic Management.

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Introduction

Acute, deliberate self-poisoning has reached epidemic proportions in parts of the developing world where highly toxic poisons and sparse medical facilities ensure a high fatality rate.[1] Hair dye poisoning is common in developing countries like India. [2.] The hair dye is extremely cheap and freely available, making it an attractive option for suicidal intent. It is available both in liquid (Super Vasmol 33) and powder (Super Vasmol) form. Both of these forms contain substances like paraphenylenediamine (PPD), resorcinol, ethylene diamine tetra acetic acid (EDTA), propylene glycol, liquid paraffin, cetostearyl alcohol, sodium louryl sulphate liquid, herbal extracts, preservatives, almond protein and perfume.[3]

PPD is the most toxic active ingredient among the above substances. The concentration of PPD varies from, 2 to 10% in branded dyes. PPD accelerates the dyeing process and thus used in hair dye formulation. Systemic features of PPD poisoning occur in three phases.

- Phase 1 is an acute presentation with edema of neck, airway obstruction, gastritis and severe vomiting.
- Phase 2 is a subacute presentation with acute renal failure (ARF), rhabdomyolysis and hemolysis.
- Phase 3 progresses to multiorgan failure.

Mainstay of management is early recognition and supportive measures as there is no specific antidote[4] We hereby report a young female who presented to us with features of angioedema and rhabdomyolysis after ingesting super vasmol 33, which was treated successfully.

Case Report:

A 31-year-old female presented to our Emergency Department within 30minutes after suicidal consumption of super vasmol 33 hair dye about 100 ml in the midnight. She initially had 2 episodes of vomiting, mild puffiness of face, edema of lips. There was no history of dark colored urine, decreased urine output, breathlessness or seizures at presentation.

On examination, pulse rate was 90/min, blood pressure was 110/70 mmHg and SpO2 was 95% at room air, blood glucose was 120mg/dl. Her systemic examination was normal.

Gastric lavage was given to the patient and in view of edema of lips and facial puffiness (stage 1, see Table 1) she was treated with injection hydrocortisone 100mg IV and antihistamines.

Complete hemogram with peripheral smear, liver and renal function tests, serum electrolytes including calcium, PT, INR, aPTT, CPK- Total, CK-MB, LDH, ECG, chest X-ray were ordered at the time of admission and was within normal limits.

Within the next one hour she developed stridor, difficulty in speaking and soft palate edema (stage 2, see Table 1), hence immediate rapid sequence intubation was done and was shifted to the ICU.

On next day morning, she was passing dark-colored urine. Her RFT, LFT, coagulation profile were within normal range and ABG was not showing any acidosis. However, serum phosphorus and magnesium were found to be low and was corrected. Nephrology opinion was taken immediately and she was treated with forced alkaline diuresis with prompt monitoring of serum potassium levels and urine pH. ENT opinion was sorted in view of persisting angioedema (stage 4) and X-ray of neck was taken and steroids were continued (inj.hydrocortisone 100mg q6h).

On Day 2 her CPK- Total, CK- MB and liver enzymes started to rise with normal serum bilirubin level and RFT. Medical gastroenterologist opinion was taken and hepatoprotective drugs like ursodeoxycholeic acid and S-Adenosyl methionine was started and USG abdomen was done which was normal. Forced alkaline diuresis was continued and adequate hydration was maintained with strict intake – output chart.

On Day 3 her liver enzymes continued to rise but there was a fall in CPK- Total and CK- MB levels. Her urine output was adequate and color looked normal. Subsequent days both liver enzymes and CPK- Total and CK- MB levels continued to fall down as shown in table2. Forced alkaline diuresis was stopped on Day 4

Her laryngeal edema was gradually reducing and she was put on T- piece trial on day 4 and was extrubated on day 5 and ENT review was obtained and video laryngoscopy was done which showed adequate airway and congested epiglottis and arytenoids.

Patient was then shifted to ward where her hemodynamic and laboratory parameters were monitored. Psychiatry opinion was obtained and counseling done. The patient improved with conservative management and was discharged on 10th day. At follow up after 15 days she was asymptomatic.

Table 1: Stages of Angioedema.

Stage 1	Facial and lip edema
Stage 2	Soft palate edema
Stage 3	Lingual edema
Stage 4	Laryngeal edema

Table 2: Relevant Laboratory Parameters During Hospital Stay

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 8
SGOT	20	258	433	983	477	251
SGPT	11	1293	1267	470	421	157
CK-Total	99	33,332	29759	18039	6551	3330
CK-MB	16	455	287	168	75	51
Potassium	3.8	3.7	4.0	4.1	3.4	2.8
Calcium	8.8	8.1	8.2	8.3	8.5	8.5
Phosphorus	1.7	2.2	2.2	2.5	3.1	3.0
Magnesium	1.7	2.5	2.2	2.2	2.1	2.0
Creatinine	0.6	0.5	0.4	0.4	0.4	0.5

Discussion

Hair dye ingestion leading to a wide variety of complications has been described in literature. Super vasmol 33 is one of the widely used hair dyes in India. It has a potent nephrotoxic cocktail-containing PPD, propylene glycol and resorcinol. The exact concentration that causes toxicity is unknown. But 3gms is sufficient to cause systemic complications. Fatal dose is 7–10 gm. Fatal period is within 24 hr of consumption. It has high mortality rate (47%).[5]

Usually, high doses of this dye ingestion can cause

- *Cervico facial:* Laryngeal oedema—most dominating presentation, frequently requiring emergency tracheostomy.[6, 7] It is immunologically mediated.
- *Musculo skeletal:* Rhabdomyolysis
- *Renal:* Impaired renal function–occurs due to a combination of hypovolemia, toxic injury and myoglobinuria.
- *Hepatic:* raised liver transaminase enzymes fluid and electrolyte imbalance
- *Cardiac:* Myocarditis (due to rhabdomyolysis of myocardium) causing ventricular arrhythmias, sudden cardiac death
- *Neurological:* Convulsions, brain death, rare due to anoxia.

The respiratory syndrome following the ingestion of PPD is represented by asphyxia and respiratory failure secondary to inflammatory edema involving cricopharynx and larynx[8]

The mechanism of rhabdomyolysis is due to leakage of calcium ions from the smooth endoplasmic reticulum, followed by continuous contraction and irreversible change in the muscle's structure. Rhabdomyolysis is the main cause of ARF and the morbidity and mortality are high once renal failure develops. Early recognition of rhabdomyolysis is crucial, since intravenous bicarbonate and saline have been shown to ameliorate the development of ARF and prevent requirement of dialysis.[4] CK levels are the most sensitive indicator of myocyte injury.

This case highlights the major toxicities of hair dye. In our patient early intubation was done which has prevented the complications of difficult airway and the need of emergency tracheostomy. Rhabdomyolysis in our patient was managed with forced alkaline diuresis and strict intake output monitoring which has prevented acute renal failure and the need for dialysis. The patient responded well to the symptomatic treatment and was discharged on Day 10 of hospital admission.

Conclusion

Super-Vasmol 33 contains a combination of potentially dangerous toxins which can result in multiorgan dysfunction and death. Early, effective and aggressive management like ET tube insertion for respiratory distress, forced alkaline diuresis along with adequate hydration for rhabdomyolysis prevents acute renal failure minimizing the need for dialysis and correction of electrolyte imbalances may be live saving in these cases.

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