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Case Report

Hair Dye Poisoning - An Emerging Problem in Urban Area: A Case Report

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1. ABSTRACT

Ingestion of hair dye is in trend as a major source of suicidal poisoning following pesticides because of its easy availability and low cost. Super Vasmol 33 is an emulsion based hair dye commonly used in India. Supervasmol-33 is emerging as a poison in urban area. The main constituent is paraphenylenediamine (PPD). Common clinical manifestations of super vasmol poisoning are cervicofacial edema, chocolate brown colored urine, oliguria, muscular edema, and shock. Quick recognition and early supportive treatment improves the clinical outcome. We report a case which highlights the toxic effects upon super vasmol ingestion.

Keywords: Super vasmol poison, Paraphenylenediamine (PPD), Supportive treatment.

2. INTRODUCTION

Poisoning is one of the preferred means of suicide. The most common poisoning encountered is ingestion or exposure to insecticides and pesticides. Ingestion of hair dye is in trend as a major source of suicidal poisoning following pesticides because of its easy availability and low cost. [1] Accidental and intentional causes of hair dye poisoning have been reported from various parts of India. [2] Self-poisoning tendency was predominant in young females of lower and middle socioeconomic class. It is usually ingested to threaten the family members if their demands were not met. The hair dye is extremely cheap and freely available, making it an attractive suicidal option. Super Vasmol 33 is an emulsion based hair dye commonly used in India. Supervasmol-33 is emerging as a major source of poison in urban area. The constituents of this hair dye include paraphenylenediamine (PPD) (4%), resorcinol, propylene glycol, ethylenediaminetetraacetic acid (EDTA), sodium, liquid paraffin, cetostearyl alcohol, sodium lauryl sulphate, herbal extracts, preservatives, and perfumes. [3] Some of these ingredients are known toxins with multi organ effects, while the toxicity profiles of others are not known. The combined effect of the individual compounds may be responsible for its significant morbidity and mortality. PPD is present in most hair dye brands like ‘super vasmol 33’, ‘Godrej’, Keshkala, colour mate etc. which are available in powder or liquid forms. The concentration of PPD varies from, 2 to 10% in branded dyes. The features of poisoning were observed with consumption of even lower volumes such as 25 mL. There was a threefold increase in the values of the markers of rhabdomyolysis and hepatitis upon consumption of larger volume suggesting its dose-dependent toxicity. [4] With large volumes, there was an increase in morbidity such as patients needing ventilator support, duration of hospital stay and mortality. [5]

The classical features of acute poisoning were seen within 3-6 hrs. Common clinical manifestations of PPD are cervicofacial edema, chocolate brown colored urine, oliguria, muscular edema, and shock. [5] Hypocalcaemia may occur in the setting of severe rhabdomyolysis or due to sodium EDTA. Patients can develop seizures, which may be due to toxins in dye or as a result of hypocalcaemia. Poisoning due to PPD has a high mortality. Therefore, early recognition can be life saving. As there is no specific antidote, the management of poisoning is supportive therapy. [5] Respiratory distress is the major early challenge, which may require ventilator support. Renal support in the form of dialysis is required in acute renal failure. [5]

3. CASE REPORT

A 20-year-old female presented to our casualty with history of suicidal consumption of super vasmol hair dye 100ml, 3 hours before admission. She had puffiness of face and neck, edema of lips and tongue, stridor, and difficulty in speaking and breathing. There was no history of dark colored urine, decreased urine output, or seizures at presentation. On examination, pulse rate was 100/min, blood pressure was 130/80 mmHg and SpO2 was 92% at room air. Her systemic examination reveals trismus and severe cervicofacial edema. Oxygen 6 litre/min given through face mask and Patient was immediately shifted to intensive care unit. Call over given to ENT specialist and emergency tracheostomy done and connected to mechanical ventilator. Gastric lavage was given to the patient and in view of stridor and facial puffiness she was treated with injection hydrocortisone 100 mg t.i.d. and antihistamines for angioneurotic edema, which resolved completely within next 12 hours. On day 1 complete hemogram, creatinine phosphokinase (CPK), serum electrolytes, liver function test, renal function test, E.C.G, chest X-ray were normal.

On day 2, she complained of passing dark-coloured urine with a decreased urine output. Her complete hemogram with peripheral smear, liver and renal function tests, ECG and chest X-ray were within normal range. However, blood urea and serum creatinine raised to 68 mg/dL and 2.8 mg/dL, respectively on Day 2 of admission. Her serum electrolytes were normal, urine myoglobin came to be positive and creatinine phosphokinase (CPK) 1500IU/dl. Nephrology opinion was taken immediately and she was treated with forced alkaline diuresis. Her ultrasonography (USG) abdomen was normal in size and echotexture. By day 3, her urine output came down to 400 mL over 24 hours. The serum creatinine phosphokinase (CPK) levels were 3500 U/L, serum calcium 10 mg/dl, blood urea 120 mgs/dl, serum creatinine raised to 5.3 mg/dL and serum potassium 5.5mEq/L. Arterial blood gas analysis showed severe metabolic acidosis. She was then hemodialysed for three sittings on Days 3, 4 and 6 till urine output improved. Her renal parameters and urine output became normal on day 7. On day 7 her tracheostomy tube was removed. On day 8 she was shifted to step down unit and then moved to general medicine ward. She was then referred to psychiatrist for counseling and discharged on 10th day. At the time of follow up she was completely normal.

TABLE: 1 BLOOD INVESTIGATION

DAY	T.WBC count (Cell/cu.mm)	S.creatinine (mgs/dl)	Blood urea (mgs/dl)	CPK (IU/L)	S.pottasium (mEq/L)
DAY 1	8,000	1.0	32	100	3.6
DAY 2	16,000	2.8	68	1500	4.5
DAY 3	15,000	5.3	120	3500	5.5
DAY 5	12,000	2.4	60	400	4.6
DAY 7	10,000	1.0	30	50	4.0

4. DISCUSSION

Poisoning is one of the preferred means of suicide. Accidental and intentional causes of poisoning have been reported from various parts of India. [1] The first artificial dye was synthesized in the laboratory in 1856. [2] Since 1883 PPD has traditionally been used for dyeing as a fresh preparation mixed with hydrogen peroxide. [2] PPD is a well know skin irritant allergic mutagenic and highly toxic reported to be carcinogenic in animals. [2] Hair dye consumption is not an uncommon means of deliberate self-harm. It is being increasingly reported in the developing countries due to easy availability and low cost. But it is uncommon in the west. Both accidental & intentional ingestion of PPD is frequently reported from Africa, Middle - East, Sudan, Morocco & Indian subcontinent. Lethal dose of PPD is not known. [3] Toxic effects of PPD are dose related. The degree of the tissue damage is related to the dose of the poison. The exact concentration that causes toxicity is not known. 3g PPD cause systemic poisoning & 7 to10gm is lethal dose. Ingestion of 100ml (12g of PPD) of super vasmol 33 dye can lead to severe complications like Laryngeal edema, Acute Renal Failure and Rhabdomyolysis. [3] The treatment is mainly supportive and depends on clinical presentation. Antihistamines and steroids are commonly used in the management of airway edema because of the possibility of a hypersensitivity reaction to PPD but there is no evidence to support this mode of treatment [3] alkaline diuresis using isotonic saline, sodium bicarbonate, and diuretics are used in the management of myoglobinuria. [4] There is no specific antidote for PPD, and trials of PPD removal using hemoperfusion and hemodialysis had variable results. The toxin is not dialyzable [5] and dialysis in only supportive therapy.

Our patient presented with cervicofacial edema of upper airway and on day 3 of poisoning developed rhabdomyolysis and acute renal failure with severe metabolic acidosis. After emergency tracheostomy, she received treatment with hydrocortisone, antihistamines, antibiotics, proton pump inhibitor, nebulisation with salbutamol-budicortisone, and chest physiotherapy for first 5 days. The acute renal failure was managed with forced alkaline diuresis and hemodialysis. The patient responded well to treatment and was discharged on 10^{th} day of hospital admission.

5. CONCLUSION

Clinical results depend on early recognition, quick referral, and supportive therapy. Early diagnosis of rhabdomyolysis and acute renal failure with institution of appropriate supportive management will have a better outcome in hair dye poisoning. And also the time of development of renal failure following PPD intoxication is uncertain and hence all patients should be monitored in hospital for development of renal complications. Common domestic products are being used incorrect manner in the society. Educational programme about the hair dye toxicity at various levels should be conducted. It is the responsibility of the health authorities to prevent the trade and use of PPD in marketplace.

6. REFERENCES

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