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Case Report

Clinical presentation and Renal dysfunction in Supervasmol 33™ poisoning

Rupesh Kumar Naik,* Ambika Prasad Patra**

*- Department of Forensic Medicine and Toxicology, SVIMS Sri Padmavathi Medical College For Women Tirupati.
**- Department of Forensic Medicine and Toxicology, JIPMER, Puducherry

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Corresponding author: Dr. Rupesh Kumar Naik, Assistant Professor, Department of Forensic Medicine and Toxicology, SVIMS Sri Padmavathi Medical College For Women Tirupati.
Email: rupeshnaik.bukkya@gmail.com

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Abstract

Super vasmol 33^{Tm} an emulsion based hair dye containing paraphenylene diamine and various other ingredients. A 20 year old female was admitted to the hospital as emergency case to our hospital with difficulty in speech, decreased urine output, facial swelling, and bilateral stiffness of lower limbs. Laboratory investigations revealed renal failure. Expired at 5th day of admission in morning hours 6.00 am after constant resuscitative efforts. On autopsy all visceral organs were congested. Here we are reporting a case of intentional ingestion of super vasmol 33 ^{Tm} hairdye.

Keywords: Hair dye, Paraphenylene diamine, Acute tubular necrosis.

Introduction

Supervasmol 33^{Tm}, an emulsion based hair dye commonly used in India. The common ingredients of the dye are paraphenylene diamine, resorcinol, propylene glycol, Sodium ethylene diamine tetra acetic acid (EDTA), preservatives, and perfumes. The main compound responsible for the toxicity is paraphenylenediamine (PPD). Though uncommon in the west, both accidental and intentional ingestion of PPD is frequently reported from Africa, the Middle-East, and the Indian subcontinent. Where PPD is commonly mixed with henna, which is traditionally applied to colour the palms of hands and to dye the hairs. As per our knowledge a few cases are reported in literature. Here we are presenting a case report of suicidal ingestion of supervasmol hair dye.

Case history

We report a case of intentional ingestion of about 100 ml of Supervasmol 33^{Tm} hair dye in a 20 year old unmarried female admitted to the hospital as emergency case in the mid noon 01.00 pm. She had difficulty in speech, decreased urine output, facial swelling, and bilateral stiffness of lower limbs. On admission the pulse rate was 101/min, blood pressure was 110/80 mmHg, and SpO_2 was 97% with oxygen by mask. The cardiovascular system and respiratory system examinations were normal. Laboratory investigations were as in table 1.

Table 1. Renal function profile during the hospitalization period

Parameter	Day 1 #	Day 2 #	Day 3 #	Day 4 #	Day 5 #
Blood urea (20-40 mgdl)	52	120	180	200	220
Sodium (135-145 meq/L)	135	139	145	136	140
Potassium (3.5-4.5 meq/L)	4.5	6.6	6.9	7.8	8.0
Creatinine (05-1.5 meq/L)	1.4	4.0	3.5	2.4	3.0
CPK* (<5 IU/L)	135	1560	1050	2000	2300
Urine myoglobin	Positive	Positive	Positive	Positive	Positive

*CPK- Creatinine phosphokinase # days she received dialysis.

On examination, angioedema, neck swelling, and chocolate brown coloured urine were recorded. On enquiring further, she belongs to poor socioeconomic status family, illiterate and stays in home working in fields. There was no history of previous suicidal attacks and depression. The amount of consumption was not known.

She was treated with injection hydrocortisone and antihistamines intravenously to bearing in mind stridor and respiratory distress. These resolved subsequently 2 days of treatment. On day 2 of poisoning, she developed rhabdomyolysis and acute renal failure. Her blood parameters urea, creatinine was elevated and arterial blood gas analysis displayed severe metabolic acidosis. She was managed initially with forced alkaline diuresis and taking into account unembellished metabolic acidosis and hyperkalaemia she received five sittings of haemodialysis. She succumbed to the complications of renal failure on 5th day of admission and body sent for autopsy after police inquest. *Autopsy Findings*: There were no external injuries over body. Internal Findings (Figure 1-3), larynx and tracheal mucosa was congested. Brain was congested and oedematous on cut section no abnormality detected. Right lung was collapsed, stomach-intact, on cut section 100 ml of dark coloured fluid present without any typical smell and the mucosa was hemorrhagic. Kidney on gross examination was intact with well-maintained renal capsule and on cut section appeared deeply congested. The rest of the viscera like liver, spleen, urinary bladder all were congested. Viscera reported negative for any poison and histopathology of kidney showed extensive necrosis of tubular cells along proximal tubule suggesting acute tubular necrosis. The final cause of death was given based on case sheet findings, statement of deceased (statement of the deceased whether suicidal or homicidal), autopsy findings and histopathology report as “Acute tubular necrosis, rhabdomyolysis as a complication of supervasmol poisoning”.

Discussion

The common ingredients used in a hair dye are hydrogen peroxide; PPD, resorcinol or aminophenols. [1] Hair dye contains mainly PPD a derivative of paranitroaniline which on oxidation produces several intermediates, of which Bondrowski's base is most allergenic, and highly toxic.[2] It is readily absorbed even with dermal contact. In 1924, Nott[3] described the first case of PPD with systemic toxicity in the owner of a hair salon. Sood et al [4]. and Chugh et al [5]. have reported cases from India.

The toxicity effects depend on the dosage. PPD is used for colour enhancement. Commonly seen systemic manifestations are cervico-facial oedema, dyspnoea, Chocolate brown coloured urine, oliguria these manifestations later will be dominated by metabolic acidosis, acute renal failure and rhabdomyolysis.

The mechanisms of kidney injury subsequent hair dye poisoning are sundry. The PPD has a direct toxic effect on kidney because of its aromatic structure, which is responsible for its easy reabsorption and concentration in tubule and leading to ARF.[6] It can also cause rhabdomyolysis with the deposition of myoglobin cast within the renal tubules and hemolysis with resultant hemoglobinuria causing acute tubular necrosis and ARF.[7]

Crispal A et al done a retrospective study in a referral hospital, South India. He noted Eleven of the thirteen patients were women and the mean was 27.2 years. Clinical features like cervico-facial oedema and pain, cola-coloured urine and oliguria are prominent. Laboratory studies revealed elevated hepatic transaminases (100%), leucocytosis (92.3%), elevated creatinine phosphokinase (92.3%), metabolic acidosis (84.6%), hypocalcaemia (61.5%), hyperphosphataemia (46.2%) and renal failure (38.5%). Eight of the them were recovered completely. The main reasons behind poor outcome given as following: delay in presentation at our centre; no gastric lavage given at the primary-care centre; those patinets requiring tracheostomy/ intubation: patinets with low Glasgow Coma Score or seizures; renal failure; and those who require intensive care.[8]

Classical features were seen in our case after 4-6 hrs of ingestion, with in this period appropriate medical care should be given. Kallel et al. observed Cervicofacial oedema (79%) as the first symptom to develop but its exact cause remains unclear. Along with classical symptoms, rhabdomyolysis and ARF due to the supervasmol poisoning were seen in our case similar to other studies.[9]

Conclusion

Since PPD based hair dyes is easily available and cheaper than organophosphorous compounds;

hence, it may become an alternative. Public awareness is needed and restricts its sale by the government. In order to decrease mortality, early recognition and early treatment should be initiated. Since there is no specific antidote, gastric lavage, early ET intubation, and alkalinisation of the urine, renal dialysis with ventilator support are the appropriate management protocols.

Conflicts of interest/Competing interests: None

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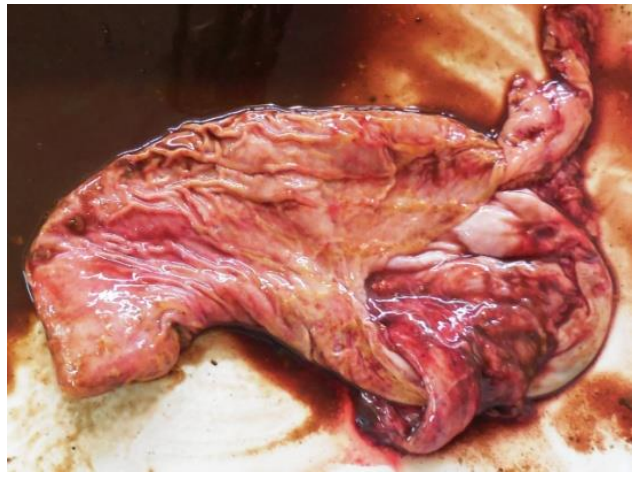

Figure 1: Showing confluent areas of submucosal haemorrhages in the stomach

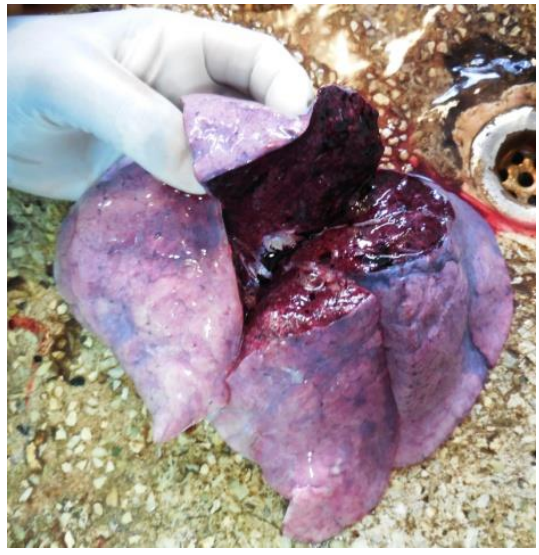

Figure 2: Showing congested and edematous lungs.

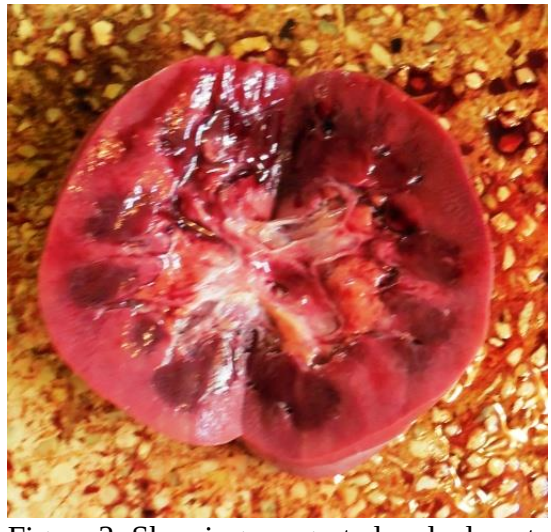

Figure 3: Showing congested and edematous kidneys with features of acute tubular necrosis - loss of cortico-medullary differentiation.