CASE REPORT

AN ANALYSIS OF THE TREND OF RISING IN POISONING DUE TO HAIR DYE BRAND (SUPERVASMOL 33 KESHKALA)

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ABSTRACT: Hair dye ingestion as a means of deliberate self-harm is well reported[1] and a growing trend is observed among rural Indian population[2]but rarely encountered in children. Hair dyes are being used extensively by the general population in India, and several brands are available in the market. There are increasing reports of suicidal ingestion of hair dye from various parts of the country because of easy availability and high lethality. Human poisoning due to hair dye has been reported from third world countries. Case reports on hair dye poisoning are getting published from various parts of India. We have conducted this study in view of the increased suicidal attempts with supervasmol 33 (Kesh Kala) reported at our hospital.We report 18 cases of super vasmol 33 (keshkala) poisoning admitted in M.G.M. Hospital from June 2005 to May 2006.
KEYWORDS: Supervasmol 33, ParaphenyleneDiamine, Hair dye poisoning.

INTRODUCTION: ParaphenyleneDiamine (PPD) is an aromatic amine not found in nature. It is used in a variety of industrial products and in different hair dye formulations. It is well known that PPD is an allergen that may cause contact dermatitis, erythematous urticarial papules and eczema in susceptible individuals. However, the major systemic problem occurs when it is ingested accidentally, for purposes of suicidal intent or during attempted murder.

Super Vasmol 33® is a popular emulsion-base permanent hair dye, containing mainly paraphenylenediamine (4%), with liquid paraffin, cetostearyl alcohol sodium lauryl sulphate, EDTA disodium, resorcinol, propylene glycol, herbal extracts, and permitted preservatives and perfume. The effects of PPD when ingested are serious and include cervicofacial edema, mucosal injury, respiratory distress, acute renal failure, rhabdomyolysis, and myocardial injury [3]. These dyes are available in stone, powder, or liquid forms. While the liquid forms are more often ingested with suicidal intentions, mortality is higher with the stone forms [4].

MATERIALS AND METHODS: The present study was carried out in view of the trend towards increased consumption of hair dye as a means of suicidal attempt especially in young adults. 18 cases of suicidal poisoning due to hair dye admitted in our hospital during the period June 2005 to May 2006 were included in our study. Every patient completed a detailed questionnaire providing information about amount of poison consumed.Routine blood investigations were carried out in all the patients within hours following admission. Examination of other systems (lungs, liver and kidney) was carried out. Patients breathing / respiratory pattern, colour of the urine, other signs of renal failure (oliguria, pedal oedema) were recorded. Patients were enquired about related symptoms of hair dye poisoning like muscle pain, diarrhea, episodes of vomiting, pain abdomen and

burning micturition. Levels of serum creatinine, blood urea, serum potassium, liver functional tests, chest radiographs were carried out in all patients.

Treatment protocol opted for the patients included – stomach wash, use of activated charcoal, oxygen inhalation, high doses of steroids, I.V diuretics along with routine symptomatic management. Patients who presented with complaint of dyspnoea (secondary to angioneuroticoedema) were candidates for emergency tracheostomy. Dialysis was opted for patients who failed to respond to conservative management of renal failure. Psychiatric counseling was given to all recovered patients at the end of their treatment regimen.

RESULTS: Out of the 18 case of hair dye poisoning admitted in our hospital 14 patients were of female sex and 4 were males. Reports of routine blood investigations like complete blood picture, ESR, Hb, RBS were all within normal limits. Average levels of serum creatinine in these case was around – 6.5mg/dl, Blood urea – 80- 100 mg/dl, serum potassium > 7.5mmol/lt. There was a moderate rise in the levels of liver functional test. Chest radiography performed in these patients showed normal study. 14 out of the 18 patients developed angioneurotic edema which predisposed to development of dyspnoea. Emergency tracheostomy was performed for such patients. Among the 14 tracheostomised patients, two of them died and one patient developed tracheo-oesophageal fistula. 10 out of 14 Tracheostomised patients or 10 out of 18 cases super vasmol 33 (Keshkala) poisoning developed renal failure. 5 out of 10 cases that have developed renal failure were recovered with conservative management. Remaining 5 patients were kept on dialysis. 4 out of 5 cases those who received dialyzed survived. While 1 out of 5 cases who were subjected to dialysis died.

DISCUSSION: Paraphenylenediamine (PPD) is a derivative of paranitroaniline. Chemically, it is an aromatic diamine related to aniline. However, the major product formed is Bondrowski's base, which is allergic, mutagenic and highly toxic [5].Resorcinol is a toxic phenolic derivative, and a corrosive chemical. It is known to cause irritation to the eyes, skin, and GI mucosa [6].

This study comprised 18 victims, of which 14 were females and 4 were males, all suicidal in nature, and mostly belonged to the 18 - 35 year age group. Rural, young poor women are the common subjects for whom this agent is inexpensive and easily available[7]. The brand Super Vasmol 33 is an emulsion containing 4 g of PPD in 100mL costing only Rs. 35/-. Hair dyes could be perceived as "not bad enough to kill" by the vulnerable victims who may be taking it just with an intention of threatening the family. Unlike the other commonly used organophosphates, hair dye can be bought without raising suspicion of suicidal intentions, particularly in small villages with closed communities. PPD is thought to have abortive effect in rural Africa.

PPD is the permanent black colouring agent applied with ammonia and hydrogen peroxide in hair dying[8]. It is also added to henna (Lawsoniaalba) and used in the popular tattooing for its darkening effect. PPD is shown to cause rhabdomyolysis in rats by promoting leakage of calcium ions from the smooth endoplasmic reticulum resulting in prolonged muscle contraction and irreversible change in muscle structure.

The diagnosis of PPD intoxication is largely dependent on clinical manifestations. The clinical features are rather unique and in the absence of laboratory facilities in many developing countries the angio-edema of the face and neck together with the hard protruding tongue and the chocolate-

brown color of the urine are used for clinical diagnosis [9]. Organ damage may be assessed by appropriate tests for rhabdomyolysis, and kidney and liver involvement. The urine can be tested for PPD using thin layer chromatography which is essential for medico-legal purposes [10]. However, this test is not routinely available and there is a need for a rapid test to demonstrate PPD in blood or urine.

The effects of resorcinol in acute poisoning after oral ingestion are limited. Resorcinol ingestion is associated with convulsions, salivation, dyspnea, emaciation and hyperemia of the GI tract. The lowest lethal dose (LDL) of resorcinol in humans has been reported as 29 mg/kg body weight. Systemic manifestations of resorcinol poisoning may include nausea, dyspnea, methemoglobinemia, tachypnea, pallor and profuse sweating, with hypotension and tachycardia. Resorcinol is also Neurotoxic and its acute exposure effects range from seizures, followed by CNS depression to lethargy, coma and death. Common clinical manifestations of systemic toxicity due to PPD are cervicofacial edema, chocolate brown or COLA colored urine, upper airway tract edema, oliguria, muscular edema and shock. The biological results were dominated by rhabdomyolysis, metabolic acidosis, acute renal failure and hyperkalemia.

Treatment is mainly supportive depending on clinical features at presentation. Tracheostomy is a life saving measure for an obstructed airway, and some patients may need endotracheal intubation. Antihistamines and steroids are commonly used because of the possibility of a hypersensitivity reaction to PPD but there is no evidence to support this mode of treatment. Alkaline diuresis using isotonic saline, sodium bicarbonate and diuretics is used in the management of myoglobinuria with variable results. There is no specific antidote available, and trials of PPD removal using hemoperfusion and hemodialyis had variable results. However, dialysis is an effective supportive measure in case of oliguric or anuric AKI.

Management of the patients depends on the amount of poison consumed and time lag between poison consumption and onset of treatment. Dialysis should be performed in patients with severe acute renal failure not responding to conservative treatment. Injection calcium gluconate is useful patients with severe muscle cramps and hypocalcaemia. Injection of paracetamol and tramadol is warranted for patients with severe myalgias. Psychiatric counseling given to all recovered patients.

CONCLUSION:PPD intoxication is a life threatening condition. Clinical outcomes rely on early recognition, prompt referral, and aggressive supportive treatment in collaboration with different specialties. Health authorities should call for the prevention of the use and trade of PPD in the market. Awareness programs about its toxicity should be implemented at different levels.

Hair dye (Super Vasmol 33) poisoning is emerging as a suicidal poison that is available quite freely and extensively. There is no specific antidote for PPD and treatment is supportive. Early treatment can prevent renal failure. However, therapeuticdialysis and supportive therapy can result in complete recovery in those who developed renal failure.

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