HAIR DYE POISONING

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Abstract
Hair dye is becoming an emerging suicidal agent in the developing world with increased morbidity and mortality. The use of hair dyes can be traced back to 4000 years before the Christian dye and was a common practice among men and women. Today hair dyes are commonly used by population. The main content of hair dye is paraphenylenediamine (PPD), which is highly toxic thus making life threatening. PPD poisoning may cause multiorgan failure and ultimately death. We report a case of 18 years old female of PPD poisoning with typical features of angioneurotic edema, myoglobinuria, rhabdomyolysis and acute tubular necrosis. Management is only supportive and helpful if started early. A high degree of suspicion is needed to make the diagnosis.

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Key Words: Hair dye poisoning; Paraphenylenediamine; Toxic effects

Introduction
Hair dye is used as a suicidal agent in the developing world and the incidence is on rise [1]. Its use can be traced back to 4000 years before the Christian age and the art of dying hair with henna indigo (which was extracted from the plant Indigo feratinctoria) and vegetable dyes was known already at that time. Today millions of consumers use hair dyes[2]. Its main ingredient is paraphenylenediamine (PPD) which after consumption causes toxicity in the form of agioneuroticedema, airway obstruction, hoarseness of voice, methemoglobinemia, acute renal failure, gastritis, rhabdomyolysis, hemolysis, myocarditis, multiorgan failure and death[3]. Hair dye is available in powder and liquid forms, freely available in the market and cheap resulting in suicidal tendencies. We report a case of PPD poisoning, which had the characteristic features of angioneurotic edema, acute tubular necrosis and rhabdomyolysis.

Case Report
An 18 year old female working as maid servant in the home, was brought to the emergency within 5 hours of consuming about 100ml of hair dye. She was not paid for the wages by her landlord. She presented with swelling of face, tongue and neck, recurrent vomiting and difficulty in breathing. On examination she was conscious with Glasgow coma scale of 13/15, Blood pressure 120/80 mmHg, Pulse rate 60 beats/min, Respiratory rate of 24/min and oxygen saturation 90% with supplement oxygen. Examination of central nervous system, cardiovascular, respiratory systems was unremarkable. There was no cyanosis and jaundice. She had marked swelling of face, tongue and neck with an inspiratory stridor with no edema over any other part of body. But her limb muscles were tender on palpation. All her emergency investigations were normal. After 3 hours of admission, her urine output started decreasing and the colour of urine became dark chocolate brown. Creatinine phosphokinase (CPK) level was 1600 IU/L (normal 150 IU/L) with urine showing myoglobin and haemoglobin by electrophoresis. Urine examination revealed urinary casts. Her urine output progressively decreased over next 2 days from 900 ml on first day to 250 ml on the 4th day. But her blood urea and serum creatinine remained unaltered. She was managed with Inj adrenaline (1:1000), Inj avil, Inj ceftriaxone, Inj pentoprezole, Inj Lasix, I/V fluids and endotracheal intubation. The tracheostomy tube was removed on 3rd day with disappearance of edema and colour of urine and CPK level coming to normal. Psychiatric consultation was also taken and she was discharged in stable condition by medicine department on 8th day with no fresh complaints.

Discussion
The main content of hair dye is PPD which is used for colour enhancement. It is not found in nature and many industrial companies produce it commercially from coal tar. It is available in the form of white crystals when it is pure and rapidly changes to brown colour on exposure to air. It is available in many forms and the most common cheap form is stone hair dye which is available as 20 gm pack. Other branded hair dyes like Godrej,

Supervasmol, 33 Kesh Kala, L'oreal, Garnier, Colourmate etc are available in liquid or powder form. The concentration of PPD in hair dye formulation varies from 70% to 90% in stone hair dye and 2-10 % in branded dyes, which are used for giving black colour to hair[4]. The major oxidant product of PPD is Bondrowski's base, which is allergic, mutagenic and highly toxic. It is also added to henna (Lawsoniaalba) and used in popular tattooing for its darkening effect. The first case of PPD poisoning was reported in 1924 in a hair dresser[5]. Cases of hair dye poisoning as suicidal agent are not uncommon in developing countries probably due to easy availability and low cost. 150 cases of hair dye poisoning over 10 years have been reported from Khartoum, Sudan alone[6]. Many case reports have been published from India especially from Andhra Pradesh[7]. The pathophysiological mechanisms could be due to increased free radical formation, skeletal and cardiac muscle necrosis (scattered coagulation necrosis), formation of highly nephrotoxic quinonediamine (an oxidation product of PPD metabolites), renal tubular occlusion due to myoglobin casts and acute tubular necrosis. The toxicity of PPD includes angioedema of face, neck and tongue, dysphagia, slurring of speech, hoarseness of voice, methemoglobinemia, cardiac toxicity, rhabdomyolysis, acute renal failure, limb edema, muscle tenderness, hepatitis, convulsions, hypotension, exophthalmos, blindness and death[3,8]. The toxic effects are dose related[9]. Diagnosis is made on the basis of history of ingestion of hair dye or if hair dye is not available, then there has to be high index of suspicion. Hair dye poisoning is commonly suicidal as in our case but it may rarely be accidental or homicidal[3]. The angioedema of face, neck and tongue occurs due to increased permeability of blood vessels while brown black urine and oliguria results from rhabdomyolysis and acute tubular necrosis respectively in this case. There is no specific antidote for PPD and the treatment is mainly supportive[10]. Our patient was timely and successfully managed with adrenaline, antihistamines, I/V fluids and endotracheal intubation. Dehydration needs to be avoided. Dialysis is indicated in acute renal failure. The most consistent predictiors of mortality are amount of hair dye ingested, hyperkalemia, hypocalcemia and hypophosphatemia and mortality rate varied between 0.03% to 60%[11]. There is limited information on the systemic effects and outcome of hair poisoning in pregnant mother and children[12].

Conclusion

Hair dye poisoning is a life threatening emergency. It is not uncommon in India unlike in the west since it is cheap, freely available and used extensively by the people. It has no specific antidote. Early recognition, timely intervention with early intubation or trecheostomy and supportive therapy can be life saving. There is a strong recommendation for the regulation and restriction of sale of PPD.

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