A REVIEW ON HAIR DYE POISONING

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**ARTICLE INFO**

<table>
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<tr>
<td>Received 12/01/2015</td>
<td>Available online</td>
</tr>
<tr>
<td>30/01/2015</td>
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</tbody>
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**Keywords**

Hair dye poisoning
Methemoglobinemia, Rhabdomyolysis, Homicidal Cause.

**ABSTRACT**

Hair dye is an emulsion based cosmetic agent commonly used in India as an intentional, accidental and homicidal cause of poisoning having deleterious effects with multisystem involvement. The main objective of the study is to review effects of hair dye poisoning .the common clinical manifestations are air way obstruction with severe edema of face, neck, pharynx and larynx following respiratory distress. The urine turns to chocolate brown color due to methemoglobinemia, rhabdomyolysis, and acute tubular necrosis. If this proceed further it may lead to fatal complications such as arrhythmias and intravascular haemolysis. Most common cause of death is air way obstruction if not relieved by intubation. No specific antidote is available till today, but symptomatic treatment can be given with continuous monitoring.

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*Please cite this article in press as Dr.s.Chandrababu et al. A Review on Hair Dye Poisoning. Indo American Journal of Pharm Research.2015:5(01).*

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INTRODUCTION

Suicide is one of the common modality of death all over the world. It is ranked as the third leading cause of death in the age group 15–44 years. Suicide rates have increased by 66% in the past 50 years.[1] Most of the house hold products (hair dye, kerosene, washing agents), cosmetics (nail polish), and pesticides and tablets are using as suicidal agents.

Hair dye has been reported as an intentional, accidental and rarely homicidal cause of poisoning from developing countries in Asia [2,3,4,5] and Africa [6,7,8,9,10]. As means of deliberate self-harm is well reported and a growing trend is observed among rural Indian population[11].

Hair dye is an emulsion based dye commonly used for dyeing hair, which contains potential toxins including paraphenylene diamine, resorcinol, sodium ethylene diamine tetra acetic acid, liquid paraffin, propylene glycol, cetosterol alcohol, sodium lauryl sulphate, preservatives and perfumes[12]. These dyes are available in stone, powder and liquid forms while the liquid forms are more often ingested with suicidal intentions mortality is higher with the stone forms[13].

Main ingredients of the dye is PPD which is a derivative of para nitroaniline that is available in form of white crystals then pure and rapidly turns to brown when exposed to air.[10] PPD is an aromatic amine used in a variety of industrial products such as textile, or for dyes dark coloured cosmetics, temporary tattoos, photographic development, lithographic plates, photo copying printing inks, black rubber, oils, greases and gasoline[10].

The first artificial dye was synthesized in the laboratory in 1856.[1] Since 1883 PPD has traditionally been used for dyeing as a fresh preparation mixed with hydrogen peroxide[10].

Toxic effects of PPD are dose related [15]. The degree of tissue damage is related to the dose of poison [3] (3gm PPD cause systemic poisoning and 7-10 gms is lethal dose). Ingestion of 100ml (12gms of PPD) of hair dye can lead to severe complications like laryngeal edema, acute renal failure, and rhabdomyolysis[13].

Local contact of PPD results in skin irritation, contact dermatitis chemosis, and lacrimation, exophthalmos or blindness[16]. Ingestion of PPD results in poisoning resulting in multisystem involvement of CNS, CVS, renal and musculoskeletal.

Manifested by severe edema of neck, face and laryngeal edema with respiratory distress requiring emergency intubation, tracheotomy and mechanical ventilation[8]. Rhabdomyolysis, and intra vascular haemolysis with hemoglobinuria cumulative in acute renal failure[1,10], oliguria, acute tubular necrosis[17], focal glomerular sclerosis[4], Cardiac toxicity resulting in myocarditis[2] and fatal arrhythmias causing sudden death. Rhabdomyolysis by promoting leakage of Ca2+ ions from smooth endoplasmic reticulum results in prolonged muscle contraction and irreversible change in muscle structure[18]. Anaemia, leucocytosis, liver necrosis[24], metabolic acidosis and hyperkalaemia[8].

The effects of resorcinol in acute poisoning after oral ingestion are limited. Resorcinol ingestion is associated with convulsions, salvation, dyspnea, emacination and hyperemia of gastrointestinal tract. The lowest lethal dose of resorcinol in humans has been reported as 29 mg/kg body weight systemic manifestation of resorcinol poisoning may include naeusa, dyspnea, methamoglobinemia, tachypnea, pallor, and profuse sweating with hypotension and tachycardia. Resorcinol is also neurotoxic and it exposure effects range from seizures followed by CNS depression to lethargy coma and death[19].

Cetosteary alcohol can produce allergic and utricarial reactions and sodium lauryl sulphate causes irritation to skin[12].

Case series on this Hair dye poisoning have been reported from Khartoum[17, 20, 21] Casablanca[22] and morocco[23]. Numerous papers published from India[7,8,9,10,24,25,26,27]. In India from AP[8,9] and from Tamilnadu (Vellore)[7,11].

CLINICAL MANIFESTATIONS:

Within four to six hours; causes laryngeal edema due to direct corrosive effect of PPD on mucous membranes rapid development of severe edema of face, neck, pharynx and larynx with respiratory distress. Tongue is dry and wooden hard and swollen due to edema.[11,15]

Within days to week: chocolate brown colored urine due to methemoglobinemia, rhabdomyolysis, acute tubular necrosis, arrhythmias and intravascular haemolysis, pain abdomen, vomiting, gastritis, hypertension, vertigo, tremors and convulsions[6,15]. Most common cause of death is airway obstruction and acute renal failure.[16]

Toxicity of PPD was first discovered in hair dresser in 1924 following exposure due to occupational handling[28]. A 13 yrs old girl was noted to have chemosis striking facial edema extending to the neck she was tachycardia with a gallop continuous bladder drainage was placed with drained cola colored urine[29]. A 29 yrs old male consumed 100ml of hair dye presented with complaints of breathing difficulty from half an hour swelling of face, tongue and neck[30]. A 23 yrs old married female presented with complaints of facial swelling with edema of lips, swollen neck and tongue.[31]. A 16 years female complaints of inability to open her mouth and cramps in her legs. On examination, she was found to have trismus. On day 2 She had deranged liver and renal parameters. Her urine was of black color. Investigations obtained revealed myoglobinuria and hyperkalaemia. Arterial blood gas analysis showed metabolic acidosis. She improved and discharged.

A case from RIMS Kadapa that 28 yrs old wife intentionally consumed super vasol and noted to have survice facial edema so tracheostomy was done and connected to a ventilator. Chocolate brown colored urine was observed, that rise in creatinine phosphokinase and potassium levels was observed.[33]. A 20 yrs old girl complaints with severe face and neck swelling, respiratory distress, convulsions and loss of consciousness. She was gasping and developed bradycardia. Patient soon developed generalized tonic clonic convulsions and her deep tendon reflexes were absent. Cardiac arrest occurred and despite cardiopulmonary resuscitation, the patient died on the morning of 4th day. A 34yrs female complaints neck swelling and puffiness of face, dyspnea, excessive sweating, and haematuria. Despite all efforts patient developed cardiac arrest. Thus, the patient was declared dead on 4th day of admission. Heart showed mild cardiomegaly, Kidneys were externally normal but cut-section showed – mild cortical medullary disruption,
congestion and grayish yellow necrotic areas. Lungs showed edema and congestion with minimal inflammation. The liver showed focal fatty change, inflammatory cell infiltrates, congested and dilated blood vessels. Kidneys showed numerous abortive tubules, surrounded by peritubular acute inflammatory cell infiltrate. The patient was declared dead on 4th day of admission[34]

**GENERAL APPROACH WITH POISONING CAUSED BY ORAL INGESTION OF HAIR DYE[35]**

No specific ANTIDOTE is available till today; management is symptomatic in these cases.

Prophylactic intubation is indicated in patients who consumed large quantity with increased risk of airway obstruction; this procedure prevents need for emergency tracheostomy.

Cricothyroidotomy is emergency procedure with advanced airway obstruction in less time to save patient or where intubation is not possible.

Percutaneous tracheotomy is in severe airway obstruction when patient is maintaining good oxygen saturation levels but progressive obstruction where endotracheal intubation is not possible because of edema of oropharynx.

Early resuscitation by tracheostomy will prevent hypoxic encephalopathic death.

If patient develops a renal failure dialysis is needed

Hypocalcaemia and metabolic acidosis are the common symptoms associated with the Hair dye poisoning. For treating the hypocalcaemia by IV calcium gluconate and calcium citrate and other sources of calcium supplements. If patient develop any seizures or convulsion it can be treated with midazolam, phenytoin, valporic acid and thiopentone can be effective in treating the seizures. For increasing the urine output in the patient Furosemide (LASIX), Torsemide, thacrynic acid can be give IV dose. For the treating the metabolic acidosis patient should be kept on continuous infusion of dextrose other electrolyte balances should be maintained. Renal output should be monitored as the chance of developing acute renal failure, rhabdomyolysis and oliguria.

**CONCLUSION**

On review and observation it had been concluded that hair dye is most commonly employed suicidal agent in India having multisystem effects that results in serious, fatal complications based on the amount ingested by the person. Public should be made aware about this poisoning and make sure that the persons receive medical assistance as soon as possible. The community should be educated not to do such activities which endanger their life. Recommended for future aspects of the study. Clinical pharmacist team has to do the psycho-social evaluation by: finding out the reason of using up, letting the patients vent their feelings and discussing with them altered aspects of their situations giving them related paradigms.

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