Hair Dye Poisoning-A Clinicopathological Approach and Review

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ABSTRACT
Hair dye poisoning, super vasmol 33, a commonly used liquid hair dye, cheap, freely available in the market and easily consumable. So it is a major cause of suicidal poisoning in India. The major life threatening compound in this hair dye is para-phenylene- diamine (PPD). Oral consumption of Super vasmol 33, a four month prospective study was conducted and 264 cases registered in the causality from January 2010 to April 2010, in RIMS Govt. Hospital, Kadapa. The majority of poisoning was intentional (99%) with a female predominance (65%). Maximum incidence observed in 2nd & 3rd decades. The characteristic triad of clinical features encountered is angioneurotic edema with strider, rhabdomyolysis with chocolate colored urine and acute renal failure. Leukocytosis with neutrophilia, hemoglobinuria, myoglobinuria, increased cpk- mb observed. No specific antidote for PPD, hence management is only symptomatic & supportive with immediate tracheostomy.

Key words:
Hair dye poisoning, Laboratory investigations, Triad of clinical features.

1. INTRODUCTION
Super vasmol 33 is an emulsion based hair dye commonly used in India. The main ingredient of the dye is PPD1. PPD has been used internationally as a key ingredient in different hair dye formulations to produce a variety of shades depending on its concentration2. Concentration of PPD in different hair dyes range from 0.2% to 3.75% which give color from golden blond to black color3.

In India popular hair dyes contain PPD along with other ingredients3. PPD commonly mixed with henna which is traditionally applied to color palms of hands & to dye the hairs4. PPD is produced in Germany, Japan & UK. PPD available in the form of white crystals when it is pure and rapidly turns to brown when exposed to air3. PPD accelerating the dyeing process(1 to 2 hours)4. Application of henna alone take several hours (2 to 12 hours )to give desired color. Mixture of henna & PPD called black henna2. In Sudan PPD is mixed with henna, leaves of labwsonia Alba, which is a nontoxic herb used to color their hair, decorate the hands & feet in special social events such as wedding ceremonies3,2. In Morocco, a nontoxic herbal extract from the gallnut of athl-pine (Tamarix aphyla ) is traditionally used to dye hair. This natural extract in locally known as “takast” 3. Among the Egyptians, their hair dressers as early as 5000 years BC, who knows the art of dyeing hair. The first artificial dye was synthesized in the laboratory in 18565. Since 1883 PPD has traditionally been used for dyeing as a fresh preparation mixed with hydrogen peroxide2. PPD is an oxidation hair dye product5. It is a derivative of paranitroaniline widely used in industrial products such as textile or fur dyes dark colored cosmetics, temporary tattoos, photographic development, lithography plates, photocopying and printing inks, black rubber, oils, greases & gasoline3.

In 1924 Nott described the first case of systemic toxicity with PPD in the owner of a
hair salon\(^4\). PPD is a well known skin irritant allergic mutagenic and highly toxic reported to be carcinogenic in animals\(^6\). 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\(^7\). But it is uncommon in the west. Both accidental & intentional ingestion of PPD is frequently reported from Africa, Middle - East, Sudan, Morocco & Indian subcontinent.

The European Union restricted the concentration of PPD in hair dye formulation to a maximum of 6% in Sudan it is available in its pure form of 97%\(^3\). Lethal dose of PPD is not known. Toxic effects of PPD are dose related\(^4\). The degree of the tissue damage is related to the dose of the poison\(^8\). The exact concentration that causes toxicity is not known. 3g PPD cause systemic poisoning & 7 to 10gm is lethal dose\(^3\). Ingestion of 100ml (12g of PPD) of Super vasmol 33 dye can lead to severe complications like Laryngeal edema, Acute Renal Failure and Rhabdomyolysis\(^9\).

Ingredients of Super vasmol 33 \(^8,10\):
- Para phenylene diamine (<4%)
- Propylene glycol
- Light liquid paraffin
- Ceto stearly alcohol
- Sodium lauryl sulphate liquid
- EDTA disodium
- Resorcinol
- Herbal extracts
- Preservative and perfumes
- Water

These ingredients are toxicants with multi organ effects\(^1\).

Propylene glycol - is a phenol derivative and commonly used as a solvent\(^8\).

Resorcinol - being a phenol is corrosive and also causes methemoglobinemia & renal toxicity\(^11\).

EDTA causes hypocalcaemia\(^8\).

2. MATERIALS AND METHODS

Total numbers of cases admitted in causality in a four months period were 264. Out of them male were 93 and female were 171 with a female preponderance of 65% (Table -1). Out of 264 admissions 18 patients were died in 24 to 48 hours. 46 patients referred to tertiary hospitals for dialysis.

Reasons for ingestion mainly suicidal in 261 patients. Only three admissions are due to accidental consumption. Approximately 50 to 100 ml of Super vasmol 33 consumed.

<table>
<thead>
<tr>
<th>Reason</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major family problems</td>
<td>49%</td>
</tr>
<tr>
<td>Failure in examinations</td>
<td>15%</td>
</tr>
<tr>
<td>Depression disorders</td>
<td>8%</td>
</tr>
<tr>
<td>Cause not identified</td>
<td>28%</td>
</tr>
<tr>
<td>Common age group effected</td>
<td>15 to 35 years</td>
</tr>
</tbody>
</table>

Edema of face, neck and dysphagia are the predominant symptoms. Tongue is dry & wooden - hard & swollen due to edema (Figure: 1). Chocolate brown color urine was formed in 158 cases (Table -2). Leukocytosis with Neutrophilia in 93% of cases, hemoglobinuria & myoglobinuria recorded in 60% of cases. Urea and creatinine levels increased in 28% cases with CPK rise in 42% cases majority of these renal symptoms landed into renal failure and referred to higher centers. ECG changes observed were tachycardia, T wave inversion ST segment elevation and fibrillations in 41% cases.

Tracheostomy done in 89% cases & Tracheal intubation in 11% cases.
Table -1:

Distribution of cases according to Age and Sex

<table>
<thead>
<tr>
<th>AGE GROUP</th>
<th>MALE</th>
<th>FEMALE</th>
<th>TOTAL</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>15 – 25</td>
<td>23</td>
<td>67</td>
<td>90</td>
<td>34</td>
</tr>
<tr>
<td>26 – 35</td>
<td>68</td>
<td>98</td>
<td>166</td>
<td>63</td>
</tr>
<tr>
<td>36 – 45</td>
<td>2</td>
<td>6</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>&gt;45</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>93</td>
<td>171</td>
<td>264</td>
<td>100</td>
</tr>
</tbody>
</table>

Table - 2:

Distribution of cases according to Clinical Features

<table>
<thead>
<tr>
<th>SYMPTOMS/SIGNS</th>
<th>NUMBER OF CASES</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edema of face and neck</td>
<td>195</td>
<td>74</td>
</tr>
<tr>
<td>Dysphagia</td>
<td>187</td>
<td>71</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>137</td>
<td>52</td>
</tr>
<tr>
<td>Muscle Pain</td>
<td>82</td>
<td>31</td>
</tr>
<tr>
<td>Chest Pain</td>
<td>79</td>
<td>30</td>
</tr>
<tr>
<td>Chocolate Brown colored Urine</td>
<td>158</td>
<td>60</td>
</tr>
<tr>
<td>Hemoglobinuria &amp; Myoglobinuria</td>
<td>160</td>
<td>60</td>
</tr>
<tr>
<td>Dyspnoea</td>
<td>55</td>
<td>21</td>
</tr>
<tr>
<td>Oliguria</td>
<td>74</td>
<td>28</td>
</tr>
<tr>
<td>ARF</td>
<td>55</td>
<td>21</td>
</tr>
<tr>
<td>Albuminuria</td>
<td>119</td>
<td>45</td>
</tr>
</tbody>
</table>

Fig.1:

Picture showing hard & swollen Tongue due to edema.
3. DISCUSSION

Globally suicide rates have increased by 60% in the past 50 years. Suicide is one among the three leading causes of death in the age group between 15 to 44 years. Recently hair dye poison becomes one of the important etiological factor5. PPD poisoning is a common health problem in the Middle East, especially Sudan and Morocco. It is also common in India but rare in the west. An eleven year 1992 to 2002 retrospective study from Morocco described 374 cases of PPD poisoning, majority of patients (54%) were 15-24 years age group and children contributed 11.5%. In Sudan, over a 10 year period (1995 to 2005) 3159 patients were reported PPD poisoning, among 18% of children below the age of 14 years. So most reported cases were from adolescents & adults, but a significant number of cases occur among children3. A retrospective study of 25 cases over period of 7 years (2001 to 2008) in Egypt6,12. In the present four month prospective study 264 cases analyzed. Maximum incidence observed in 2nd & 3rd decades which is correlating with PK Jain et al and other studies.

There were female preponderance of 77% as per Ayoub Filali et al14, 80.7% as per M. Hamdouk2 and 74.86% PK Jain et al15. It was 65% in the present study.

Suicidal in 78.1% as per Ayoub Filali et al14 and were 87% as per M. Hamdouk2. It was 99% in our study which was tallying with PK Jain et al. Accidental ingestion was reported in 38.7% and suicidal were 32% in children3.

Clinical Features are unique, starting with abdominal symptoms after consuming PPD. Local contact of PPD (7 to 10 days) results in skin irritation, contact dermatitis chemosis, lacrimation, exophthalmos or permanent blindness.

Ingestion of PPD results in Acute Poisoning with major systemic problem. It results in multisystem involvement of CNS, CVS, Renal & Musculoskeletal3. The onset of effects is usually within four to six hours after ingestion. The more severe the poisoning the earlier the onset of effects16.

Phase I symptoms: - within 4 to 6 hours10,13

5 to 10 ml Super vasmol 33 can cause laryngeal edema due to direct toxic effect of PPD on mucous membranes8. Rapid development of severe edema of the face, neck, pharynx & larynx with respiratory distress. Tongue is dry & wooden - hard & swollen due to edema, Often requires tracheostomy1,4,13.

Phase II symptoms: - within days to weeks. Chocolate brown colored urine due to methemoglobinemia. Rhabdomyolysis, acute tubular necrosis, arrhythmias and intra vascular hemolysis. Pain abdomen, vomiting, gastritis, hypertension, vertigo, tremors & convulsions4,13. Most common cause of death is acute renal failure and also due to rhabdomyolysis8.

Direct trauma to the tissues by chemicals and causes dyspnoea and asphyxia Intense inspiratory effort secondary to laryngeal edema which might have contributed to over distension of the alveoli. Development of pneumothorax reported following laryngeal edema17.

The sequences of clinical features in the present study (Table -2) are similar as that of PK Jain et al and other studies15.

Investigations are normal at the time of admission4,8,15.

Pulse rate - 102/min, BP - 110/70mm/hg, Resp. rate - 20/min.
After four hours - TC - increased, DC - neutrophilia, Platelets normal, Peripheral smear - Normocytic normochromic anemia

After fourteen hours - Black color (cola colored) urine and output decreased 600 to 1200ml. Albumin ++, RBC 2 to 4 per hpf. Myoglobin + ABG – metabolic acidosis, mild hypoxia Elevated potassium levels serve as prognostic indicators of rhabdomyolysis, Blood Pressure increased – 190/110 Ventricular Fibrillation Blood urea and creatinine increased

48 hours to 10 days oliguria with increased CPK - Rapid increase in blood urea and creatinine. Increased serum LDH indicates intravenous Hemolysis and Rhabdomyolysis. Death after 10 days due to acute tubular necrosis. Elevated renal parameters AST, ALT, CPK & LDH suggested the development of acute kidney injury due to rhabdomyolysis. In the current study we found tachycardia, ECG changes were observed T wave inversion ST segment elevation and fibrillations in 41% cases. Leukocytosis with Neutrophilia in 93% of cases, hemoglobinuria & myoglobinuria recorded in 60% of cases. Urea and creatinine levels increased in 28% cases with CPK rise in 42% cases PPD can be detected in urine using thin layer chromatography.

There is no antidote for PPD Poisoning. Fallowed symptomatic approach of treatment.

1. Supportive - flash with lots of water or milk immediately.
2. Tracheostomy.
3. Hemodialysis – In ATN it is modality of choice. Though toxin is not removed by dialysis.

4. O₂ inhalation.
5. I.V Calcium correction – for hypocalcaemia which may be due to sodium EDTA.

High Mortality noticed with this poisoning. 42% of Mortality occurs within 24 hours of diagnosis. Mortality rate was 21.1% as per Ayoub Filali and it was 6.8% only in this study which depends on the quantity of consumption. Corroboration of different specialties required for ideal management that is ENT, Anesthesia, Medicine & lab Medicine. Mortality rate was lowered by 12% possibly because of availability of dialysis facilities and early therapeutic intervention and collaboration with ENT departments. It is important that medical personnel should be aware of this poisoning so early therapeutic intervention and can avoid fatality.

4. CONCLUSION

Some of the countries considered PPD to be great hazard and its use in hair dyes was banned. Germany banned in early 1900’s, subsequently France and 1964 in Sweden. However in Japan & India it is still common component in hair dyes. In view of high incidence of poisoning in some parts of India it requires banning.

5. REFERENCES

[16]. Chugh KS, Malik GH,Singhal PC. Acute renal failure following paraphenylenediamine(hair dye) poisoning :report of two cases.