Hair Dye Poisoning in Bundelkhand Region (Prospective Analysis of Hair Dye Poisoning Cases presented in Department of Medicine, MLB Medical College, Jhansi)


Abstract

**Aims of study:** Hair dye (Paraphenylene di-amine, PPD) poisoning has high morbidity and mortality and its incidence has increased dramatically in the past 4 years. A prospective study was planned to assess the clinical profile and outcome with different treatment approaches.

**Material and methods:** The material comprised of 1020 cases admitted in Medicine Department of MLB Medical College, Jhansi, U.P. from July 2004 to March 2009. Out of 1020 cases 697 cases were of stone hair dye poisoning and 323 cases were of other branded hair dyes (powdered form containing less amount of Paraphenylene diamine). Diagnosis was made solely on the basis of the history given by cases/attendant and symptoms of neck swelling, black coloured urine and muscular pain. The cases were thoroughly studied for different complications (renal, hepatic and cardiac etc) and were treated accordingly.

**Results:** Out of 1020 cases studied, majority were females in the age group of 15-45 yrs (n=734) while the rest were males. A total of 244 (23.92%) cases expired during treatment.

Neck swelling, respiratory distress and whole body muscular pain were most common symptoms at presentation, oliguria, chest pain, palpitation, presyncope / syncope, pain in abdomen, nausea with vomiting and dysphagia were other common symptoms.

**Conclusion:** Paraphenylene diamine is highly toxic. Cases who consumed up to 10 gm of PPD usually survived if they presented to hospital within 4 hour of dye ingestion. Severe edema of face, neck and floor of mouth, renal failure and myocarditis were poor prognostic factors. No deaths occurred in cases of Branded Hair dye ingestion.

Introduction

Hair dye is available in several forms and the commonest cheap form is Stone Hair Dye which is available in 20 gms pack. Other branded hair dyes like ‘Godrej’, Kesh kala, Colourmate etc. are available in powder or liquid forms. The concentration of active substance i.e. paraphenyl diamine varies from 70-90 % in Stone Hair Dye and 2-10% in branded dyes which are used for giving black colour to hair. The Stone Hair Dye is extremely cheap and freely available, making it an attractive option for suicidal intent.

The chemical used in Hair Dye is a derivative of paranitroaniline and is called paraphenyl-diamine (PPD). It is brownish to black coloured solid which is partially soluble in water and easily soluble in hydrogen peroxide (H₂O₂). PPD is a good hydrogen donor and is metabolized by electron oxidation to an active radical by cytochrome P450 peroxidase to form a reactive benzoquinone diamine. This is further oxidized to a trimer known as Brandowaski’s base, a compound reported to cause anaphylaxis as well as being strongly mutagenic. It is also traditionally used for dying palm and soles along with henna.

**Material and Methods**

Prospectively collected data of 1383 cases was studied. The study comprised of 1383 cases, 67 cases were brought dead who had history and features suggestive of stone hair dye poisoning. 83 cases died within first 5 hrs. of admission after gastric lavage, drugs and I/V fluid therapy (Table 1). Out of 1233 remaining cases 167 cases of dye ingestion did not have any feature of toxicity and were discharged or absconded in the first 12 hrs. 41 cases of stone hair dye who had relatively mild disease did not opt for investigations and were treated with I/V fluids and drugs were excluded from this study. 3 cases of known cardiac and 2 cases of known renal diseases were excluded from the study. Finally study comprised of 1020 cases who were thoroughly investigated and treated. In 631 cases (61.86%) out of 1020 cases ECG was done at 1st hour after admission and then cardiac monitoring was done in ICU.

It was realized later in the study that cases of stone hair dye who died suddenly, were developing ventricular tachyarrhythmias.

Therefore in the latter half of the study after admission in emergency ward cases were immediately shifted and managed...
Fig. 1: A 26 years old girl presenting with facial edema after hair dye ingestion

### Results

A total of 1020 cases were studied. 734 were female and 286 were male cases (Table 2). The reason for ingesting drug was mainly suicidal (97.84%). The remaining were accidental (1.86%) and homicidal (0.29%). Symptomatology was directly related to the dose of PPD ingested.

Two types of presentation were seen. One in whom neck swelling occurred predominantly and others in whom myocarditis developed. It seems that cases who after putting the dye in the mouth thought whether to ingest or not, developed prominent edema of throat, tongue lower jaw, eye hypocalcaemia subsided).

7. Vasopressors (intravenous Dopamine and/or noradrenaline) were used if hypotension persisted despite adequate fluid therapy.

8. Dialysis- Hemodialysis or Peritoneal dialysis was used for cases with renal shut down and resistant hyperkalemia.

9. Intravenous amiodarone and defibrillation for ventricular tachyarrhythmia management.

### Method

The data was analysed from the following points:-

1. Demographic profile
2. Clinical profile
3. Morbidity and mortality pattern
4. Outcome with different management strategy given in the hospital

PPD can be detected in urine by thin layer chromatography on silica gel; solvent system benzene; ethyl acetate (50:50) or hexane; acetone (90:60) and sprayed with 0.2% solution of potassium dichromate as a chromogenic reagent to give a pinkish brown spot. In this study this test was not performed due to lack of this facility at MLB Medical College, Jhansi, U.P.

### Management Plan

After a quick clinical examination special attention was given to vital parameters. As the immediate cause of death was hypoxia. Airway patency was maintained by Google’s airway and emergency tracheostomy (done in 7 cases only).

Circulatory volume and pressure were maintained by giving appropriate fluid therapy.

The treatment was based on the following principles-

1. Since no antidote is available against PPD management was basically supportive.
2. Gastric lavage was followed by intravenous methylprednisolone 1 gm/day for 5 days or hydrocortisone 100 mg 8 hourly for 7 days.
3. Oxygen was administered for Hypoxic cases.
4. Sodium bicarbonate was administered to prevent myoglobin precipitation in kidney (average dosage was 1 ampoule containing 22.5 meq. in 500 ml normal saline every 8 hourly) along with loop diuretics (furosemide or torsemide) to maintain adequate urine volume.
5. Chlorpheniramine maleate one ampoule IV every 8 hrly till cervicofacial edema subsided (average 3-5 days).
6. Calcium gluconate was given to counteract hypocalcemia. (10% calcium gluconate 10 ml every 8 hourly till

Table 2: Distribution of cases according to age and sex

<table>
<thead>
<tr>
<th>Age groups (years)</th>
<th>Male</th>
<th>Female</th>
<th>Total No.</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-25</td>
<td>102</td>
<td>349</td>
<td>451</td>
<td>44.21</td>
</tr>
<tr>
<td>26-35</td>
<td>85</td>
<td>325</td>
<td>410</td>
<td>40.20</td>
</tr>
<tr>
<td>36-45</td>
<td>79</td>
<td>32</td>
<td>111</td>
<td>10.88</td>
</tr>
<tr>
<td>&gt; 45</td>
<td>20</td>
<td>28</td>
<td>48</td>
<td>4.71</td>
</tr>
<tr>
<td>Total</td>
<td>286</td>
<td>734</td>
<td>1020</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Table 3: Distribution of cases according to serum creatinine level

<table>
<thead>
<tr>
<th>S. Creatinine (mg/dl)</th>
<th>No. of cases</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (0.6-1.4)</td>
<td>759</td>
<td>79</td>
</tr>
<tr>
<td>1.5-3.0</td>
<td>102</td>
<td>21</td>
</tr>
<tr>
<td>3.1-4.5</td>
<td>39</td>
<td>7</td>
</tr>
<tr>
<td>4.6-6</td>
<td>37</td>
<td>11</td>
</tr>
<tr>
<td>6.1-7.5</td>
<td>48</td>
<td>20</td>
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</table>

in intensive care unit with cardiac monitoring.
lids, conjunctiva and neck. While the cases who immediately swallowed sufficient quantity of dye developed myocarditis and renal failure later on.

Severe edema of face, neck and tongue was present in 73.03% (n=745) cases (Fig. 1) and lead to respiratory distress, hypoxia and dysphagia (Table 3).

Muscle pain was the next most common presentation seen in about 47.05% (n=480) cases. Limbs were swollen, tender and stiff (Fig. 2). PPD poisoning leads to rhabdomyolysis and muscle necrosis.

Dysphagia was present in most of the cases presenting with severe edema of face and neck (71.17%).

Nasal twang of speech was found in 59 cases (5.78%) while nasal regurgitation was observed in 25 cases (2.45%) only (Table 3).

Chest pain, palpitation and presyncope / syncope was another common manifestation. These cases were at high risk of developing cardiac complication.

238 cases were having strong suspicion of myocarditis on the basis of clinical features and ECG changes. The clinical features were fatigue, dyspnoea on exertion/rest, chest pain, palpitation, presyncope / syncope with positive ECG changes were seen in 238 cases changes were tachycardia, T wave inversion, ST segment elevation or depression, Bundle branch blocks, intra ventricular conduction defects (Fig. 3), atrial and ventricular premature complexes, atrial fibrillation and 21 cases developed ventricular tachyarrhythmia (Fig. 4) during therapy. Troponin T was positive in 136 cases of positive ECG changes (more than 0.1 ng/ml was positive). Transthoracic Echocardiographic (TTE) done in 238 cases. The findings were regional wall motion...
abnormality and decreased left ventricular ejections fraction (LVEF≤35%) in 126 cases on day 2 to day 5 which subsequently improved on follow up in cases survived. Cardiac dilatation in the absence of regional coronary artery disease and evidence of rapid recovery of ventricular function during follow up in 98 cases out of 238 cases was observed

Patient developing suspicion of myocarditis as per ECG changes and clinical features were having high morbidity and mortality (67 out of 238 expired) and those who developed ventricular tachyarrhythmias, 21(8.82%) were having further bad prognosis and 12 cases expired out of 21 despite standard medical management.

Chocolate brown colour urine (Figs. 5 & 6) was found in 549 (53.82%) cases specially in those cases who had marked orofacial swelling, limb edema and muscle tenderness (Table 3). This chocolate brown colour of urine was due to the presence of myoglobin and hemoglobinuria.

Decreased urine output was found in 130 cases (12.74%), and 46 cases(4.51%) had anuria. Mortality was much higher in anuric cases (63.04%).

Albuminuria was observed in 376 cases (36.86%). Albuminuria reflects impaired absorption and processing of filtered proteins by injured proximal tubules. Myoglobinuria was present in 549 cases (53.82%) suggestive of rhabdomyolysis. Renal biopsy was done in 10 cases only which showed acute tubular necrosis in all the cases.

In this study a total 261 (25.58%) cases had increase blood urea and creatinine levels. This increment was noticed on 2nd day onwards associated with oliguria or anuria in 130 cases. This increase in blood urea and creatinine was strongly associated with high level of CPK which is due to rhabdomyolysis.

521 cases (51.08%) showed raised serum CPK levels. Maximum value of serum CPK level was reported as 2,81,000 Iu/l. This raised CPK level was associated with rhabdomyolysis. Maximum value of serum CPK level was reported as 2,81,000 Iu/l. This raised CPK level was associated with rhabdomyolysis.

In the earliest report of PPD intoxication from India two cases were reported by other workers.9,10 Other workers also have reported the similar results.8 Myoglobinuria (53.82%) suggestive of rhabdomyolysis is one of the key complication of PPD intoxication, similar findings are reported by other workers.9,10

Clinical features in the present study were typical cervicofacial edema (73.03%), dysphagia (71.17%), chocolate brown color urine (52.82%) pain and or rigidity of limbs (47.05%), respiratory difficulty (22.45%), hypotension (14.61%), decreased urine output (12.75%), increase in BP (7.84%) and convulsion (2.06%) are comparable to the study by H Kallel H Chelly et al (1994-2000) whose cases were dominated by cervicofacial edema (79%) chocolate brown coloured urine (74%), upper airway tract edema (68.4%) oliguria (36.8%) muscular edema (26.3%) and shock (26.3%).

In the present study, renal failure was observed in 261 (25.58%) cases with 130 cases (12.75%) showing anuria or Oliguria. Anuria showed a poor prognosis with more electrolyte disturbances, 10 cases showed acute tubular necrosis on biopsy. In the earliest report of PPD intoxication from India two cases had renal biopsy proven acute tubular necrosis by Chugh KS, Malik GH, Singhal PC (1982).6

Sumit Singhla, Sanjeev Miglani, AK Lal, Pulin Gupta, AK Agarwal (2005)7 reported a case of PPD poisoning with characteristic features and biopsy proven ATN with Oliguria. Other workers also have reported the similar results.8

Myoglobinuria (53.82%) suggestive of rhabdomyolysis is one of the key complication of PPD intoxication, similar findings are reported by other workers.9,10

Very few cases of myocardial damage have been reported due to PPD intoxication. Zigzagha AA, et al11 reported TTE proven case of myocarditis with left ventricular apical thrombus. The finding of thrombus not observed in our cases.

Brahmi et al12 reported a case of myocarditis with myocardial

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A total of 392 cases (38.43%) showed hypocalcemia. A total of 68 cases had marked hypocalcemia (S. calcium <6mg%) and it was observed that 21 cases (30.88%) out of these severely hypocalcemic cases(n=68) developed classic features of hypocalcemic tetany along with seizures.

S. bilirubin was raised in 62 cases whereas SGOT/SGPT was raised in a large no. of cases i.e. 685 cases (67.16%). It appears that increase in SGOT/SGPT was indicative primarily of muscle injury and not liver necrosis. It was observed that SGOT was higher as compared to SGPT. In 206 cases SGOT levels were >1000 IU/L.

Total 311 cases (22.48%) died after consuming hair dye poison. A pilot study done in department revealed that 5 days duration of inj. methyl prednisolone thereby produced optimum results, the duration of steroid was guided primarily by oro-facial edema, swallowing difficulty and change in colour of urine. The morbidity and mortality decreased significantly from 27.77% to 14.02%.

**Discussion**

The present study showed that hair dye poisoning is more common among females (74.86%) and younger age group 15-25 yrs. (44.21%), maximum number of cases (97.84%) were of suicidal intent and 23.92% mortality. Most of the clinical symptoms and signs reported by us are similar to those reported earlier.1-5

Ayoub Filali Liham Semlali et al (2006)3 in there eleven years (1992-2002) study of over 374 cases showed female preponderance (77 %) with the majority of poisoning being intentional (78.1%) and their population was also young (15 – 35 yrs. old age groups accounting for 69.5%), 21.1% of poisoning cases was fatal and the source / route of poisoning was by ingestion (93%).

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Brahmi et al12 reported a case of myocarditis with myocardial
infarction induced by PPD which was confirmed by angiography that showed septoapical hypokinesia due to spasm of the anterior interventricular coronary artery. OP Jatav et al\(^1\) reported a case of myocardial damage in hair dye poisoning with similar findings.

In present study clinical manifestations of acute myocarditis ranged from asymptomatic to fatal. ECG features as multiple ventricular and supraventricular ectopics, ST-T wave changes, bundle branch blocks, ventricular tachyarrhythmias positive troponin-T and decreased left ventricular ejection fraction (< 35%) and regional wall motion abnormality in large number of cases with subsequent improvement in LVEF further supports the cardiac myolysis. Earlier studies by Lauer B, Hiedraic C et al\(^2\) have used Troponin-T level in clinical suspected cases of myocarditis.

**Summary and Conclusion**

PPD poisoning is common in females and in younger age group with maximum number of suicidal intent. Route of poisoning was ingestional in all cases.

Clinical features were typical cerviofacial edema, dysphagia, chocolate brown color urine, pain and / or rigidity of limbs, respiratory difficulty, hypotension, decreased urine output, rise in blood pressure and convulsion were major clinical manifestations.

Commonly seen abnormal laboratory investigation were raised liver enzymes, myoglobinuria, raised CPK, hypocalemia, albuminuria, raised serum creatinine, hyperkalemia, hyperbilirubinemia, deranged PT and aPTT and acute tubular necrosis on renal biopsy.

Cardiac complications were in the form of suspected myocarditis leading to ventricular tachyarrhythmia.

Late mortality was mainly due to renal failure and its related complications. Preventing renal failure with abundant fluid infusion, alkalinization of urine and the correction of hemodynamic disturbances was a very important goal. Results of hemodialysis were found to be better than peritoneal dialysis though the toxin is not removed by dialysis. Intravenous methyl prednisolone given in high doses showed rapid reduction of cerviofacial edema and symptomatic improvement.

The mortality was seen in 22.48% cases. 37.62% of total mortality was in first 24 hrs. out of which 26.69% death were seen in first 6 hrs. early therapeutic intervention is essential if full recovery is to be expected. It is important that medical fraternity should be aware of this poison because the poison is available quite freely and used extensively.

**References**