Paraphenylenediamine ingestion: An uncommon cause of acute renal failure

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ABSTRACT
Paraphenylenediamine (PPD) is a major component of hair dyes. The aim is to study the renal manifestations and outcome of PPD consumption. During a four-year period from 2002 to February 2006, 10 persons were admitted to our Institute after consuming a hair dye in a suicidal bid. The percentage of ARF due to PPD at our Institute was 0.95%. Seven patients out of 10 (70%) who consumed PPD developed ARF. All 10 patients, including the patients who had normal renal function had features of rhabdomyolysis. Two patients required ventilator support for respiratory distress and two more required tracheostomy due to upper airway tract edema. One patient has expired after two sessions of dialysis. Renal biopsy in two patients (one, postmortem) showed acute tubular necrosis along with presence of casts in tubules due to myoglobin.

KEY WORDS: Acute renal failure, paraphenylenediamine

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Case Report

The aim is to study the renal manifestations and outcome of paraphenylenediamine consumption.

Paraphenylenediamine, (PPD) a coal-tar derivative, is a chemical substance that is widely used as a permanent hair dye. The major oxidation product of PPD is Bondrowski’s base, which is allergenic, mutagenic and highly toxic. The first case of PPD poisoning was reported in a hairdresser in 1924 following exposure due to PPD dye handling.[1] It is consumed to commit suicide in the Telengana region of Andhra Pradesh, India, because of its easy availability. An 11-year (1992-2002) retrospective study of PPD poisoning reported to the Poison Control Centre of Morocco has revealed 374 cases with a female predominance (77%), of which 21.1% of poisoning cases were fatal.[2]

Case History

During a four-year period from 2002 to February 2006 10 persons were admitted to our Institute after consuming a hair dye in a suicidal bid. All the patients were evaluated with hemogram, liver and renal function tests and ultrasound abdomen. Renal biopsy, including one postmortem biopsy was performed in two patients.

Ten persons were admitted to our Institute after consuming a hair dye. Eight were males and two girls. The mean age was 23.2 ± 7.6 years (14 to 34 years). All save three patients (70%) presented on the day of consumption of the poison.

At admission, six (60%) patients had cervicofacial edema, all 10 (100%) patients had chocolate brown-colored urine and seven (70%) had oliguria. Body pains were complained by seven patients (70%), one patient (10%) had muscular edema and proximal muscle weakness.

The percentage of ARF due to PPD at our Institute was 0.95%. All 10 (100%) patients, including the patients who had normal renal function had features of rhabdomyolysis. Two (20%) patients required ventilator support. Four (40%) more patients underwent tracheostomy, due to upper airway tract edema, but have not required any ventilator support. Respiratory care was required in these six patients before they developed renal failure. Seven (70%) patients, who recovered from ARF, required 3.5 ± 2.3 (range: 1 to 6) sessions of hemodialysis and the serum creatinine normalized in four weeks. Two (20%) patients required ventilator support for 10 ± 5 days. The results of biochemical investigations are depicted in Table 1.

In one patient renal biopsy was done, as there was undue delay in recovery. It showed acute tubular necrosis along with presence of casts in tubules due to myoglobin.

The electrocardiogram was normal in nine patients. One patient had ventricular extra systoles only on the first day of admission. It was normal in the patient who expired. Echocardiogram was done only in four patients. It was normal in all.

One (10%) patient expired after two sessions of dialysis, while
**Table 1: Laboratory parameters of patients who consumed paraphenylenediamine**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. Creatinine</td>
<td>5.7 ± 4.9 mg/dL</td>
</tr>
<tr>
<td>B. Urea</td>
<td>140.3 ± 69.1 mg/dL</td>
</tr>
<tr>
<td>S. Calcium</td>
<td>7.4 ± 1.6 mg/dL</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>6.9 ± 2.4 mg/dL</td>
</tr>
<tr>
<td>SGOT</td>
<td>2761.18 ± 2615.1 IU/L</td>
</tr>
<tr>
<td>SGPT</td>
<td>847.8 ± 760 IU/L</td>
</tr>
<tr>
<td>Serum ALP</td>
<td>195.4 ± 80.45 U/L</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>12.2 ± 2.84 g/dL</td>
</tr>
<tr>
<td>CPK</td>
<td>12872.6 ± 9548.8 IU/L</td>
</tr>
<tr>
<td>CPK –MB</td>
<td>416 ± 206.71 IU/L</td>
</tr>
<tr>
<td>Total serum proteins</td>
<td>6.2 ± 0.26 g/dL</td>
</tr>
<tr>
<td>S. Albumin</td>
<td>3.2 ± 0.58 g/dL</td>
</tr>
<tr>
<td>LDH</td>
<td>1866 ± 288.27 IU/L</td>
</tr>
</tbody>
</table>

on ventilator. The cause of death as evidenced by postmortem (done for legal purpose) was acute respiratory distress syndrome (ARDS) as there was organized fibrin material in the alveoli. The microscopic sections of the kidney showed myoglobin casts in tubules.

**Discussion**

Our results show that clinical manifestations of systemic PPD intoxication are dominated by cervical and upper respiratory tract edema, muscular edema, rhabdomyolysis and acute renal failure. In the Kallel et al. study the frequency of cervical and upper respiratory tract edema and acute renal failure was 68.4% and 47.4% respectively. In that study oral-tracheal intubation was performed in 68.4% of patients and emergency tracheostomy in 15.8% of patients.

The characteristic angioedema of the face and neck, on initial presentation with difficulty in breathing, secondary to upper respiratory tract edema and chocolate brown color of the urine could be a confirmative evidence of PPD poisoning in the absence of laboratory facilities and when history is lacking in case of emergency. Other reported features are rigidity and tenderness of limbs secondary to rhabdomyolysis and ARF, leukocytosis, anemia secondary to hemolysis, hemoglobinemia and hemoglobinuria. Sudden death appears to be due to myocarditis and arrhythmia. Myocardial rhabdomyolysis and shock have also been described. Hypotensive shock is recognized and is associated with poor prognosis.

In the earliest report of PPD from India, two patients had renal biopsy proven acute tubular necrosis. Renal biopsies were also performed, as inferred from an abstract presented at the Indian Society of Nephrology, 2004, (unpublished data), in four patients of PPD intoxication and revealed acute tubular necrosis (ATN) in three patients and acute interstitial nephritis in one patient. Rhabdomyolysis has not been described in both the studies. The pathogenesis of ATN, independent of rhabdomyolysis appears to be due to the aromatic structure of PPD making it easily reabsorbed and concentrated in tubules. When the toxicity was in small doses like hair dye, membranous nephropathy has been reported owing to in situ formation of immune complex. Rhabdomyolysis, was, however confirmed in postmortem biopsy in one patient who died of asphyxia following PPD intoxication. While rhabdomyolysis was reported to occur in all patients who consumed PPD, ARF was variable from 47.4-100%. Death is mainly due to acute respiratory distress and so rapid has been its onset that tracheostomy at presentation is recommended. The major challenge to life in later stages is renal failure and myocarditis.

PPD consumption is an uncommon cause of ARF due both to ATN and rhabdomyolysis. It also causes respiratory embarrassment due to cervical and upper respiratory tract edema requiring immediate respiratory care. The majority of patients recover with early respiratory care and dialytic support.

**References**


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