

**Original article:**

## **Acute Kidney Injury in Ingestional Hair Dye Poisoning**

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### **Abstract**

**Introduction:** PPD poisoning has high mortality rate due to airway edema and acute kidney injury.

**Aim:** Aim of this study to evaluate the clinical course of Acute Kidney Injury in Ingestional Hair Dye Poisoning.

**Methods:** Prospective cross sectional observational study in Patients admitted with a history of hair dye ingestion with classical symptoms of neck swelling, muscle pain and dark colored urine.

**Results:** In 46 patients, 27 had acute kidney injury, out of them 9 required dialysis. Out of 9 patients underwent dialysis, 6 of them expired.

**Conclusion:** Adequate hydration and alkalization of the urine can prevent acute kidney injury in the setting of PPD poisoning.

**Keywords:** PPD poisoning, hair dye poisoning, renal failure, mortality

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### **Introduction**

The overall suicide rate due to self poisoning was about 31% in South India<sup>(1)</sup>. The highest rates of poisoning was due to household agents, drugs, insecticides, chemicals, animal or reptile bites in the descending order of frequency<sup>(2)</sup>. Among the household articles used for self harm - drugs (prescribed for other medical or surgical conditions for self or for others), chemicals (lavatory cleaners, hair dye) are noteworthy. Permanent hair coloring is done by the use of oxidation dyes. These are composed of para phenylene diamine, a coupling agent and an oxidant. Oxidizing agents are primarily hydrogen peroxide. Coupling agents are usually derivatives of aniline. The mechanism of coloring of hair involves three steps i.e., oxidation of p-phenylene diamine derivative to the quinone state, reaction of the resultant compound with a coupler, oxidation of the

resulting compound to the final dye<sup>(3)</sup>. In the acute phase, which is about four to six hours, ppd causes local effects such as burning sensation and numbness in the mouth, burning and choking sensation in the throat, abdominal pain predominantly in the epigastric region, vomiting which may cause dehydration and aspiration, dysphagia, dysphonia due to swelling of the tongue, sublabial structures, and of the larynx and pharynx which may ultimately lead to airway obstruction, respiratory failure with resultant hypoxia, cyanosis leading to altered sensorium, neurological manifestation like seizures mainly due to hypoxia and even death due to respiratory arrest and neurological complications if unattended. Exophthalmos, optic neuritis and permanent blindness has also been reported in some studies. Intervention in this acute phase is crucial in the outcome of the poisoning. The second phase

following the acute phase which manifests itself in days to weeks is characterized by muscle pain, muscle swelling and tenderness due to rhabdomyolysis which usually manifests by 10 to 12 hrs after ingestion of hair dye<sup>(4)</sup>. Rhabdomyolysis causes muscle necrosis and myoglobinuria, with the myoglobin clogging the renal tubules and causing acute tubular necrosis leading to acute renal failure. Urine discoloration to brown or black due to excretion of the dye, which should be picked up early and treated vigorously with immediate and liberal intravenous fluids to flush out the dye and myoglobin should be carried out to prevent renal damage. Additionally the urine can be alkalized with the help of sodium bicarbonate to make the myoglobin and dye excretion easier should be carried out<sup>(5)</sup>. Kidney damage occurs not only due to rhabdomyolysis with ensuing pigment nephropathy but is also caused/aggravated by hypoxia, dehydration, intravascular hemolysis and a possible direct toxic effect of the dye on the kidney. Renal replacement therapy i.e., dialysis may be necessary in some patients in whom these measures fail to prevent or revert renal failure. Urine examination may show hemoglobinuria, albuminuria, myoglobinuria, proteinuria; p-phenylenediamine can be detected in urine by thin layer chromatography on silica gel, sprayed with 0.2% solution of potassium dichromate as a Chromogenic reagent to give a pinkish brown color<sup>(4)</sup>.

#### **Aim**

Aim of this study to evaluate the clinical course of Acute Kidney Injury in Ingestional Hair Dye Poisoning.

#### **Materials and Methods**

A prospective cross sectional observational study conducted in Government Rajaji Hospital, Madurai,

Tamilnadu. Institutional Ethics committee approval and informed consent from the patients/relatives were obtained. Patients admitted with a history of hair dye ingestion with classical symptoms of neck swelling, muscle pain and dark colored urine were included in the study. Demographic details include age, gender, education, occupation of the individual. Clinical details include cervicofacial edema, limb pain and swelling, discolored urine, oliguria, dyspnea, palpitation, syncope, voice change, pulse and blood pressure data and oxygen saturation recorded in the bedside using a finger pulse oximeter. Laboratory data to be collected include urine analysis for protein, deposits; blood total count, blood urea, creatinine, sugar, serum sodium, potassium, serum total CPK, serum SGOT, SGPT, electrocardiogram, serum cardiac troponin T for patients in whom there are ecg changes and/or symptoms and signs of myocarditis like tachypnea, tachycardia, hypotension. Treatment details to be collected include airway management requiring tracheostomy or endotracheal intubation, ventilatory support, the dose, duration and type of steroids used, whether alkaline diureses used, dialysis details if done, the use of vasopressor, antiarrhythmics or cardioversion. Cervicofacial edema, discolored urine, muscle pain and swelling were noted in the first 24 hours of admission. Oliguria, dyspnea, palpitation, syncope, seizures etc were taken into consideration when present anytime during the hospital stay. Urinalysis, blood urea, creatinine, serum electrolytes, serum cpk were taken on admission, the second day and periodically once in one or two days thereafter. The second – third day values and discharge day values are taken into consideration for the study. ECG was taken for all patients during admission and thereafter only if patient has persistent tachycardia, electrolyte

abnormalities, hypotension, dyspnea, tachypnea, chest pain. If ECG changes were present, cardiac troponin T was done.

**Results**

Among 52 patients who were admitted with hair dye ingestional poisoning, 46 patients were included in the study. Majority of them were young.

**Table 1 Age distribution of Study Patients n=46**

Age group (years)	Total	Percentage
15 to 25	22	47.8%
26 to 35	16	34.7%
36 to 45	5	10.8%
> 45	3	6.5%

**Table 2 Clinical Profile of Study patients**

Sl no.	Symptoms/sign	No. of cases	Percentage (%)
1	Cervicofacial edema	38	82.60
2	Muscle pain and/or swelling	37	80.43
3	Brown black colored urine	37	80.43
4	Oliguria	32	69.56
5	Tachypnea, dyspnea	05	10.86
6	hypotension	07	15.21
7	Seizures	01	02.17

82.60% cervicofacial edema and features of rhabdomyolysis such as muscle pain, tenderness and discolored urine in 80.43%. 69.56% of patients had oliguria and tachypnea, dyspnea and seizures were also seen. 43.47% of patients had proteinuria, Serum total Creatine Phosphokinase levels were elevated in 78% of the patients. 27 out of 46 patients had elevated creatinine level, 7 patients had more than 4.5 mg%.

**Table 3 Renal Profile of Study Patients**

Serum Creatinine	No. of Patients
Less than or equal to 1.5 mg%	19
1.6 to 3.0 mg%	16
3.1 to 4.5 mg%	4
More than 4.5 %	7

Hemodialysis was required in 9 of the 27 patients who had elevated renal parameters. 18 patients were managed conservatively with high rate of fluid infusion along with forced alkaline diuresis to facilitate/accelerate the clearance of myoglobin. 9

patients, who had rising serum creatinine values, not decreasing by conservative measures, had to undergo hemodialysis. Out of the 9 patients, 6 patients died. One of them had developed myocarditis during the course of hospital stay.

**Table 4 Mortality due to renal failure**

Serum Creatinine	No. of Patients	Death
Less than or equal to 1.5 mg%	19	-
1.6 to 3.0 mg%	16	-
3.1 to 4.5 mg%	4	1
More than 4.5 %	7	5

All patients who expired had elevated serum creatinine levels. Five of them were on renal replacement therapy (hemodialysis). Majority of them recovered with conservative management i.e., liberal parenteral fluids and alkaline diuresis within one to two days of admission.

**Discussion**

PPD causes pigment nephropathy AKA occurs due to myoglobin. Myoglobin is more toxic to the kidney in the setting of acidemia and hypovolemia<sup>(6)</sup>. There is an initial decrease in GFR and low fractional Na excretion in rhabdomyolytic renal injury. Volume expansion when given after 18 hours of onset of rhabdomyolysis is unlikely to help because of the possible tubular necrosis that has set in by the time. In the presence of acidic tubular fluid, myoglobin is converted to ferri heme which is directly toxic to tubules<sup>(7)</sup>. The heme moiety is the nephrotoxic factor in myoglobin uric renal injury. Heme sensitizes the tubular cells to damage by Phospholipase A2. Also, heme is thought to deplete cellular energy stores<sup>(6)</sup>. The mere presence of myoglobinuria may not cause acute renal failure; other co existing conditions like hypovolemia and acidic urine is required to cause the

same<sup>(6)</sup>; this gives a rationale for early infusion of large volume of fluids and alkalization of urine to hasten recovery/ prevent renal injury in rhabdomyolysis. Tubular obstruction by cellular debris and pigmented casts causes tubular epithelial injury<sup>(8)</sup>. Both hypovolemia and acidemia accelerate this process. The rise of serum creatinine compared to serum blood urea nitrogen is more in cases of renal failure due to rhabdomyolysis than due to other causes. This is because muscle injury releases creatine which is converted to creatinine<sup>(6)</sup>. Serum Creatinine was elevated in 58.69% patients in our study. The rate was 25.6% in JCMR study<sup>(4)</sup>, 60% in Mohamed SS et al study<sup>(9)</sup>. Serum Cpk was elevated in 78.2% in our study and it was 58.3% in PK Jain et al study<sup>(4)</sup>. Of the total six cases who expired, all of them had angioedema requiring tracheostomy; all had muscle pain, elevated creatine kinase and discolored urine; all of them had oliguria and elevated renal parameters, five of them were on dialysis, one patient had myocarditis. Ventilatory support was required for two patients. The mortality rate was almost similar in males and females in our study (12.5% and 16.67%

respectively). Overall mortality was 13.05% in our study and 22.48% in PK Jain et al study.

### Conclusion

PPD Ingestion causing acute kidney injury having high mortality rate can be prevented by early

presentation and identification of the poisoning. Adequate hydration and alkalization of the urine can prevent acute kidney injury in the setting of PPD poisoning.

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