# **Paraphenylene diamine poisoning**

A. C. Jesudoss Prabhakaran Department of Pharmacology, MAHER University, Enathur, Kancheepuram, Tamil Nadu, India

#### Address for correspondence:

Dr. A. C. Jesudoss Prabhakaran, Department of Pharmacology, MAHER University, Enathur, Kancheepuram 631552, Tamil Nadu, India. E-mail: jesudossacp@gmail.com

#### Abstract

The commonest constituent of all hair dyes is paraphenylene diamine (PPD) being used by the people to color their hair all over the world. Hair dye poisoning is emerging as one of the emerging causes of intentional self-poisoning to commit suicide. In this article, the importance of clinical manifestations and of hair dye poisoning is discussed due to the lack of specific diagnostic tests. Since there is no specific antidote for PPD poisoning, the early supportive treatment modalities are discussed.

Key words: Hemoglobinuria, hemodialysis, hair dye, paraphenylenediamine, renal failure

## **INTRODUCTION**

As the global suicide rate increases by 60% in 50 years in the underdeveloped and developing countries, paraphenylenediamine (PPD) poisoning is emerging as an important etiological factor reported from India.<sup>[1]</sup> PPD poisoning was the number one cause of poisoning in Morocco in 1990.<sup>[2]</sup> PPD ingestion is a very common form of poisoning in the Indian subcontinent as this compound is a component of hair dyes and is easily available.<sup>[3]</sup> PPD ingestion causes symptoms arising from involvement of different organs. Chemically, it is a derivative of paraphenylaniline. It is brown or black colored solid substance easily soluble in hydrogen peroxide and not in water. It is a good hydrogen donor and metabolized by electron oxidation to an active radical by cytochrome P450 peroxidase to form a reactive compound called benzoquinone diamine. This can be further oxidized to a trimer known as Brandowaski's base, a well-known compound reported to cause anaphylaxis and mutation. It is traditionally used for dyeing palm, sole along with henna, and to dye the hairs. PPD accelerates the dyeing process. Ingestion of PPD causes rapid development of edema of

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the face, neck, pharynx, tongue, and larynx initially and rhabdomyolysis later. Finally, acute renal failure supervenes as renal tubular necrosis occurs due the deposits of the toxic metabolites of PPD.<sup>[4]</sup>

### **CASE REPORT**

A 24-year-old young man who had ingested PPD pellets (exact amount could not be calculated) with suicidal intention. He developed a generalized itching and intermittent lacrimation for a few days and had a symptomatic treatment from a nearby medical practitioner concealing the history of consumption of PPD pellets. He presented after 6 days with complaints of pain and stiffness of both lower limbs and passage of chocolate colored urine followed by anuria. He had swelling of feet and facial puffiness. His blood pressure was recorded as 190/110 mmHg. The general examination revealed bilateral pitting pedal edema and facial puffiness. Cervicofacial edema was absent. On investigation, his blood urea and serum creatinine were 210 mg/dl and 12.4 mg/dl, respectively. He also had hyperkalemia, hypocalcemia, and hyperphosphatemia. Arterial blood gas analysis revealed metabolic acidosis. Serum LDH was 1098 U/l and the reticulocyte count was 7.8%. Peripheral smear examination showed schistocytes suggestive of intravascular hemolysis. Urine examination revealed proteinuria, hemoglobinuria, and hemosiderinuria. His creatinine kinase (total) was 824 U/l. Ultrasound revealed normal-sized kidneys with increased cortical echogenecity but maintained corticomedullary differentiation. He

was managed symptomatically with diuretics, phosphate binders, sodium bicarbonate, oral calcium, and alkalization of urine.<sup>[5]</sup> In view of his persistent oliguria and deranged metabolic parameters, he was started on hemodialysis.<sup>[6,7]</sup> The patient required 12 sessions of hemodialysis and his urine output and metabolic parameters improved. He was observed for 1 month in the hospital for any recurrence of the symptoms.

#### DISCUSSION

PPD poisoning can lead to many symptoms. Myocarditis, myocardial rhabdomyolysis, and shock have also been described in PPD poisoning.<sup>[8]</sup> Although the cervicofacial edema is also a common manifestation, it was not a prominent clinical feature in this case. The alteration in the prominent clinical features may be due to the symptomatic management in the initial stages. However, the awareness must be created to know that the major cause of mortality is respiratory distress due to edema, complications related to myocarditis, and renal failure to make a protocol towards the management. The renal failure manifests as acute tubular necrosis along with rhabdomyolysis and hemoglobinuria. The cause of acute tubular necrosis in PPD poisoning, independent of rhabdomyolysis is due to concentration of PPD in the renal tubules. This occurs due to the aromatic structure of PPD which makes it readily absorbable and concentrated in the tubules.

With regards to treatment, there is no specific antidote available.<sup>[9]</sup> Rinsing the mouth with water and ingesting milk may alleviate the gastric symptoms. Gastric lavage with 2% sodium bicarbonate is also effective. Due to low molecular weight and hydrophilic nature of PPD, it has low adsorbability on activated charcoal. A mild case of respiratory involvement can be managed with chlorpheniramine and hydrocortisone. Severe respiratory distress requires ventilatory support. Alkalization of urine is effective when hemoglobinuria is detected. Acute renal failure is managed with adequate input and output charting, treating metabolic complications due to renal failure such as hyperklemia, hypocalcemia, hyperphosphatemia, and metabolic acidosis by hemodialysis. Renal biopsy can be taken when there is undue delay in recovery of renal function.

The PPD is found in almost all brands of commercially available hair dyes. Apart from intentional or accidental excessive exposure to PPD which results in above manifestations, the routine use of this compound on the regular basis for a long time can also lead to toxic effects. <sup>[10]</sup> Membranous nephropathy has also been reported when toxicity occurs in smaller doses. It occurs due to formation of immune complexes. Contact allergy to this compound is a well-known recognized problem.<sup>[11]</sup>

Association between the personal hair dye use and non-Hodgkin's lymphoma, multiple myeloma, leukemia and other bladder cancer have been reported but have not been consistently observed.<sup>[12]</sup> However, the United States environmental Protection Agency has not classified PPD as a carcinogen. Therefore, no warnings of toxicity have been printed on boxes of hair dye. However, in view of easy availability and potential toxicity, awareness about the toxic effects of this compound may be useful.

# CONCLUSION

In the prospective study in future, the triad of strider due to upper airway edema, rhabdomyolysis and acute renal failure has to be considered for PPD poisoning. Then, the early airway intervention, alkaline diuretics and hemodialysis are the three strategies in the management of PPD poisoning may be helpful in reducing the mortality.

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