CASE REPORT

Paraphenylenediamine: Blackening more than just hair

Dilip Gude, Dharam Pal Bansal[1], Rahul Ambegaonkar, Jayaram Prajapati
Internal Medicine/Critical care, Princess Durru-Shehvar Children's and General Hospital, Purani Haveli, [1]Pulmonology and Critical care, Medwin Hospital, Nampally, Hyderabad, Andhra Pradesh, India

Paraphenylenediamine is an important constituent of hair dye toxicity of which one could herald fatal complications such as rhabdomyolysis, renal failure, angioneurotic edema, and respiratory failure. We present a case of hair dye poisoning that presented with respiratory distress due to laryngeal edema and later developed trismus, subclinical tetany, apnea, and conduction abnormality on electrocardiogram. This case report highlights the need for a thorough toxicological review of the components of any ingested substance.

Key words: Hair dye poisoning, Paraphenylenediamine, renal failure

INTRODUCTION

Paraphenylenediamine (PPD—a common ingredient in hair dyes) poisoning, although a rare form of intoxication in the West, is fairly common in some parts of the world such as East Africa, Indian subcontinent, and Middle East countries.[1] Literature search via Google scholar revealed that a study in northern India reported 323 cases over a span of about 5 years (July 2004–March 2009) and another study in Morocco reported 374 cases over a 11 yr study period (1992–2002).[2,3] PPD poisoning poses considerable risk with mortality rates ranging from 12% to 42%.[1] We bring forth a case of PPD poisoning which developed severe edema of the face and neck requiring emergency tracheostomy, followed by rhabdomyolysis, and respiratory failure requiring mechanical ventilator support but recovered after prompt management.

CASE REPORT

A 23-year-old female, school teacher by occupation, was brought to the emergency department, Medwin Hospital, Hyderabad, India in June 2010 with alleged history of consumption of hair dye (200 ml of 4% PPD-based emulsion-type hair dye) owing to suicidal ideation a day prior to presentation. She had been taken to a local hospital where an emergency tracheostomy was done for dyspnea and stridor. She was then referred to our hospital for further management. At presentation she had complaints of inability to open mouth, difficulty in swallowing, and cramps in hands in addition to generalized body pain. On examination she had trismus, calf tenderness, abdominal tenderness, guarding and rigidity, and both chvostek's and trousseau's signs were positive. Her urine was cola colored. Investigations obtained revealed hypocalcaemia (corrected calcium = 7.2 mg/dl) with normal sodium, potassium, creatinine, and phosphorus values. Intravenous correction with calcium gluconate (2 ampoules of 10% calcium gluconate with 100 mg elemental calcium in each ampoule slow intravenous and another 2 ampoules over 6 h) was given and cramps in hands subsided gradually. The quantity of PPD ingested was calculated to be 8 gm, which is considered toxic. As rhabdomyolysis is a well-known complication of PPD poisoning, her creatinine phosphokinase (CPK) was done which was 1 62 795 IU/L (normal 60–400 IU/L). Hydration was maintained to avoid renal failure secondary to rhabdomyolysis. Patient had intermittent apnea with hypoxia, which required mechanical ventilator support. Electrocardiogram (ECG) showed significant ST-segment depression in inferolateral leads and right bundle branch block with right axis deviation. Pre-existing underlying cardiac conditions, if any, were excluded by 2D-echocardiography. She was started on parenteral nutrition because of trismus. By day 7, her CPK was 286 IU/L; trismus improved, and endoscopic guided nasogastric tube placed which revealed severe

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Address for correspondence: Dr.Dilip Gude, Intensivist & Research Coordinator Department of Internal Medicine/Critical care, Princess Durru-Shehvar Children's and General Hospital, Purani Haveli, Hyderabad, Andhra Pradesh, India. E-mail: letsgo.dilip@gmail.com
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erosive pan gastritis. By day 10, tracheostomy tube was decanulated and tracheostomy stump was closed. Later ECG was normalized except for an intraventricular conduction defect, which did not require any active medical therapy intervention. She was started on enteral feeds, which she tolerated well. She was counseled by psychiatrist prior to discharge. At follow up, she was completely asymptomatic.

DISCUSSION

The first documentation of systemic PPD poisoning in 1924 described the case of a hairdresser who developed toxicity from handling the dye.[4] PPD, a derivative of paranitroaniline, on oxidation produces several intermediates, of which Bondrowski's base is most allergenic, mutagenic, and highly toxic.[5] PPD is readily absorbed even with dermal contact. The symptoms have an onset of 4–6 h and are dose related (8 gm being the toxic dose in our case). A three-phase evolution can be seen in PPD intoxication with inflammatory stress characterized by a relative immunodepression in the first three days, proinflammatory state (due to rhabdomyolysis) from third to sixth day and immunomodulative action (due to oxidative metabolism) from the sixth day. It is a systemic inflammatory reaction specific to a cytotoxic cell support. The pathophysiologic mechanisms could be the increased free radical formation,[6] skeletal and cardiac muscle necrosis (scattered coagulation necrosis),[5] formation of highly nephrotoxic quinonediamine (an oxidation product of PPD metabolites), renal tubular occlusion due to myoglobin casts, and acute tubular necrosis.[7] Methemoglobinemia, hoarseness of voice, cardiac toxicity, hepatitis, hypotension, convulsions, coma, and sudden cardiac death are on the toxic end of the spectrum.[5]

The angioedema of face and neck on initial presentation (probably from allergic or hypersensitive reaction associated with the increased permeability of mast cells) with chocolate colored urine is characteristic in most cases[8] and might point to PPD poisoning especially when the labs and history are inconsistent. Stigmata of rhabdomyolysis (rigidity and tenderness of limbs) and acute renal failure (from hemolysis, rhabdomyolysis, hemoglobinuria methemoglobinemia, and direct tubular toxicity), leukocytosis, anemia secondary to hemolysis, are the usual accompaniments and rarely exophthalmos and blindness may also be seen. In a study of PPD poisoning spanning 7 years, cervicofacial and laryngeal edema was the dominating presenting manifestation in 72% of the cases, rhabdomyolysis in 100%, impaired renal functions in 80%, hyperkalemia in 75%, elevated liver transaminases in 76%, and fatal ventricular arrhythmias in16%.[9] A case of myocardial lysis in a fetus expelled (nonviable) by a 22-year-old mother after apparent PPD ingestion of an unknown amount of PPD was reported which on histopathology showed heart and lung congestion (interstitial edema and inflammation at the base of the lingua), in addition to a chorionic villus thrombosis and abruptio placentas.[10] Another report of two cases of PPD poisoning showed diffuse myocarditis in one and septo-apical myocardial infarction in the other.[11] Rarer complications of PPD, such as chronic kidney disease, severe myocardial rhabdomyolysis leading to cardiogenic shock and death, severe aplastic anemia, severe contact dermatitis, and optic atrophy have also been reported. As there is no specific antidote available, the treatment is mainly symptomatic and supportive and asphyxia (early on) and renal failure (later) are the most life threatening factors that need emergent addressing with tracheostomy and/or hemodialysis, respectively, when required. Our patient also had apnea with hypoxemia and fascicular conduction defect, probably because of direct toxic effect of PPD.

Early diagnosis, medical treatment with steroids and antihistaminics, and if airway compromise is not improving, endotracheal intubation or tracheostomy and in cases of refractory oliguria and rhabdomyolysis, dialytic support are the key strategies in the management of PPD poisoning.[12]

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