Case Report

Hair Dye Poisoning - A Case Report

Ramulu B,[*] Swarnalata G[**], Krishnan S[***]

ABSTRACT

A case is reported of an 18 year-old female who ingested a toxic quantity of a hair dye containing paraphenylenediamine (PPD), and presented with severe metabolic acidosis, rhabdomyolysis leading to acute renal failure, and toxic myocarditis leading to myocardial dysfunction. Haemodialysis, cardiac supportive treatment, and ICU care resulted in complete recovery over a 1-month period.

Key Words: Paraphenylenediamine, Rhabdomyolysis, Acute tubular necrosis, Toxic myocarditis

Introduction

A 18 year-old female was admitted with altered sensorium to the ICU of our hospital, with a history of consuming a popular brand of hair dye with the intention of commiting suicide. The patient was unconscious, not responding to stimuli, severly dyspnoeic, and tachypnoeic, with a pulse rate of 140 beats/min, and respiratory rate of 36 /min. Her blood pressure was 90/60 mm Hg. Her face appeared puffy. She was anaemic, with no evidence of clubbing, cyanosis, or pedal oedema. Heart sounds were normal, though the rate was high. The lungs revealed evidence of fine basal crackles on auscultation. Abdominal examination was essentially normal. Nervous system examination revealed the following: coma, normal-sized pupils and normal response to painful stimuli. Her ABG revealed severe metabolic acidosis. A provisional diagnosis of hypotensive shock and severe metabolic acidosis was made. She was treated with oxygen, IV sodium bicarbonate, inotropic support, antibiotics, IV fluids, nasogastric aspiration, and supportive care.

The patient slowly regained consciousness after acidosis was corrected. Blood pressure normalized (110/70 mm of Hg) with inotropic support. She was oliguric, and passed 50 ml of dark, chocolate coloured urine in 24 hours. Hypertention and acute renal failure developed over the next 2-3 days. Her serum creatinine levels gradually worsened, and she developed hyperkalaemia and generalised seizures (metabolic). Methhaemoglobin was 6%, and CPK was very high at 4,97,800 u/l. Urinary abnormalities worsened, and she developed proteinurea, and haematuria. The patient was taken up for requlr dialysis, and intensive supportive therapy, along with antibiotics, electrolyte correction, blood transfusion, and nutritional support. At the end of three weeks, renal biopsy was done as the patient was oliguric, and the renal failure did not improve.

Renal histopathology revealed renal coticomedullary tissue with upto 12 glomeruli. The glomeruli appeared normal. Many of the tubules had a denuded epithelial lining, and contained RBC's pigment casts. Occasional tubules contained granular casts. The intestitium was oedematous and widened, with diffuse, moderatly dense lymphomononuclear infiltrate. Blood vessels were nor-

[*]Senior Consultant, Medicine;
[**] Chief Pathologist
[***](*Author for correspondence*) Senior Consultant, Nephrology. Email: poojakrish54@yahoo.com Apollo Hospitals, Rajiv Gandhi Marg, Vikram Puri, Secunderabad 500003, Andhra Pradesh, India

mal. The whole picture was consistent with acute tubular necrosis (ATN), with possibility of superimposed pigment nephropathy.

The patient developed tachycardia, S_3 gallop, and hypotension in the first week. Echocardiogram showed global LV dysfunction with poor EF (27%), and L-carnitine and dobutamine infusion were added to the treatment, since myocardial rhabdomyolysis was suspected. With haemodialysis and supportive treatment, the patient's general condition improved, the heart rate and blood pressure normalized, and S_3 gallop disappeared. Serum CPK declined to normal levels, and muscle pains subsided. Urinary abnormalities improved, the urine colour gradually normalised, and volume increased to more than 2 litres per day (diuretic phase) over a 4-week period. Her proximal weakness persisted for over 4 weeks. Nerve conduction velocities in both lower limbs were normal. It required another two weeks of physiotherapy for complete improvement.

Discussion

The acute intoxication by paraphenylene diamine (PPD) used for dyeing, caused a syndrome of asphyxia due to laryngeal oedema and respiratory distress, severe metabolic acidosis, and hypotension. It caused rhabdomyolysis with acute renal failure (acute tubular necrosis, ATN), and cardiac rhabdomyolysis with toxic myocarditis, producing LV dysfunction. The patient improved significantly after correction of metabolic acidosis, haemodialysis, cardiac supportive treatment, electrolyte correction and symtomatic management. The ATN recovery was very slow reflecting the severity. Residual tubular dysfunction was likely. Muscle breakdown as evidenced by very high CPK levels and myocardial dysfunction were noteworthy. PPD is commonly used in several industrial processes such as dyeing furs, photochemical procedures, tyre vulcanization, oxidisable hair dye production, etc. Chemically, PPD is an aromatic diamine (coal tar derivative) related to aniline. Though its allergic effect is well established, its systemic toxicity is not well studied. Several cases of PPD poisoning - accidental or intentional - have been reported from Asian and African countries where it is traditionally mixed with henna to colour the palms and soles.

Many questions have been raised about the safety of using hair dye. Besides allergies, hair dye has come under scrutiny in recent years due to a possible link to various types of cancer. While it is known to cause skin irritation, dermatitis, chemosis, and ocular irritaion, it also has significant systemic toxicity.

Oral ingestion causes oedema of pharynx, larynx, and severe respiratory distress; rhabdomyolysis and acute tubular necrosis; and rarely seizures. Suicidal ingestion has been on the rise due to easy availability.

The Bureau of Indian Standards has specified the content of PPD, the main colouring agent in powder hair dyes, at not more than 30 per cent in the powder form and not less than 3 per cent after dilution. The lethal dose of PPD is probably around 5 gm. The other ingredients such as ammonia, peroxide or diaminobenzene are also harmful as systemic toxins. Complications of propylene glycol intake include haemolysis and haemoglobinuria.

In conclusion, it is suggested that the manufacturers of this hair dye add a clear note of caution on the containers indicating its toxic nature - if ingested.

REFERENCES

1. Singhla S, Aggarwal AK. Paraphenylene diamine poisoning - A case report. J Indian Acad Clin Med. 2005; 6: 236-238.
2. Sood AK, Yadav SP, et al. Hair dye poisoning. J Assoc Physicians India 1996; 44: 69.
3. Gabow PH, Kachny WD, Kelleher SP. The spectrum of rhabdomyolysis. Med 1982: 6: 14-52.
4. Filai A, Semlali I, Soulaymani R. A Retrospective study of acute systemic poisoning with paraphenylenediamine in Morocco. Afr J Traditional Med. 2006; 3: 142-149.
5. Chugh KS, Malik GH, Singhal PC. Acute renal failure following paraphenylenediamine (Hair dye) poisoning - Report of two cases. J Med. 1982; 13: 131-137.