Short Communication

Hair Dye Poisoning: An Emerging Epidemic

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ABSTRACT

In view of the increasing number of case reports of hair dye poisoning it is important to sensitise medical professionals and toxicologists about its potentially lethal toxicity, and guidelines pertaining to management. Most permanent hair dyes contain paraphenylenediamine, which is nephrotoxic.

The main aim of this communication is to create awareness amongst the medical fraternity regarding hair dye poisoning, so that cases of accidental or deliberate ingestion can be readily diagnosed and effectively treated.

Key Words: Hair dye; Paraphenylenediamine

Introduction

Intentional hair dye ingestion is an emerging poisoning epidemic. Hair dye is one of the most commonly used cosmetic products in India, and most brands are cheap and easily available. Paraphenylenediamine (PPD) is an aromatic diamine used in most of the hair dyeing preparations. It is also used in tattoos, photocopying, printing ink, photographic developing solutions, tyre vulcanization industry and for dyeing furs.¹

Paraphenylenediamine (PPD) accelerates the dyeing process and intensifies colour, imparts a natural look, provides permanent results, and the hair can be rinsed without losing colour. PPD mixed with henna has traditionally been used on auspicious occasions to colour palms and soles in Africa, Middle East, and Asian countries.

In recent years, physicians are witnessing a number of cases presenting with hair dye ingestion. Allergenic potential on dermal exposure, angioedema, rhabdomyolysis and acute renal failure on ingestion are the typical features. Of late, tattoos containing PPD mixed with henna and hair colour are very popular among young people in the West.

The first case of systemic poisoning by handling hair dye was reported in a hairdresser by Nott in 1924.³ The allergenic potential of PPD is well known, and it was chosen by American Contact Dermatitis Society as the 2006 "Allergen of the Year", in order to increase awareness of exposure.⁴

What is PPD?

Branded PPD hair dyes are usually available as powder, or in liquid form. The latter is usually available as two separate ingredients, one containing the PPD dye, and the other a developer. PPD is a substance that requires oxygen for it to become coloured. It is the partially oxidized state (intermediate) that may cause allergy in sensitive individuals. Fully oxidized PPD does not cause allergy. In the hair dyeing process, the peroxide is used to break down the natural hair pigment (melanin), which is then replaced by PPD.

Toxicity on Dermal or Mucosal Contact

PPD is a powerful allergen responsible for local or generalized dermatitis.⁵ Reactions may range from mild to severe. There may be reddening and swelling of the skin

(contact dermatitis), urticaria, etc. Eye exposure can cause lacrimation, chemosis and blindness. Severe allergy can lead to anaphylaxis. Permanent pigmentary changes and scars to temporary tattoos by PPD have been reported. Patients with a history of dermatitis to henna tattoos should avoid PPD hair dyes. Type1 hypersensitivity reactions comprising urticaria, angioedema, and anaphylaxis with lethal outcome have been reported. Temporary tattoos are a vehicle of contact with PPD that is relevant to children. Measures should be taken to discourage unnecessary exposure amongst children.

Toxicity on Ingestion

The typical clinical symptoms on presentation of a case of hair dye ingestion include angioedema of face and neck, chocolate-brown coloured urine, and tender muscles. Other features that may be encountered include leucocytosis, anaemia secondary to haemolysis, myocarditis, and renal failure. ^{2,10-12}.

Angioedema – It is a type 1 hypersensitivity reaction that may range from mild to severe. It is immunologically mediated and anatomically limited oedema that can be life threatening. Ishoo et al proposed a staging system by which airway risk may be predicted from the anatomic site¹³:

Stage I - Facial rash, facial oedema and lip oedema

Stage II - Stage I + palate oedema

Stage III - Lingual oedema

Stage IV - Laryngeal oedema

Stages I and II can be managed in the outpatient department or the common ward, but stages III and IV warrant ICU admission. Airway intervention was necessary in 7% of stage III and 24% of stage IV cases in one study. ¹⁴ Airway risk in angioedema may be predicted by anatomic site of presentation, allowing appropriate triage with preparation for airway intervention in selected cases.

Myocarditis – Clinical manifestations of acute myocarditis may vary from asymptomatic to fatal. Myocardial myolysis may lead to ST-T wave changes, conduction defects, and ventricular and supraventricular ectopics. ^{14,15} Positive Troponin-T confirms the diagnosis. ¹⁶

Myositis – Severe generalized bodyache, with inability to move the limbs due to pain, tender muscles and depressed reflexes can be attributed to rhabdomyolysis, and confirmed by elevated creatine phosphokinase level.

Intravascular Haemolysis – This leads to haemoglobinaemia, haemoglobinuria and anaemia. Intravascular haemolysis can be confirmed by the presence of schistocytes on a peripheral blood film, a low haptoglobin level, and elevated lactate dehydrogenase.

Acute Renal Failure – It is a delayed manifestation of PPD poisoning. While rhabdomyolysis occurs in all patients, acute renal failure is variable. PPD ingestion leads to acute renal failure both due to acute tubular necrosis and rhabdomyolysis. Hypovolaemic and direct toxic effect of PPD, or its metabolite on kidney also contributes to this.^{2,10-12} Histological changes of acute tubular necrosis have been described in PPD poisoning.¹⁷ The pathogenesis of acute tubular necrosis independent of rhabdomyolysis appears to be due to easy reabsorption and concentration in tubules.

Effect on Pregnancy – Intentional ingestion may lead to abortion.¹⁸

Treatment

There is no antidote for PPD. The major challenge is preservation of airway. Early clinical diagnosis and prompt interventions such as tracheotomy for upper airway tract obstruction in those with stage 3 and 4 angioedema, and ventilation in appropriate cases remain the cornerstone of management.

Gastric lavage can be considered, and activated charcoal administered to all patients. Hydration should be maintained by intravenous fluids. Diphenhydramine, epinephrine, and steroids should be considered, especially in the presence of angioedema.

Continuous haemodynamic and ECG monitoring to detect arrhythmias is absolutely essential. All the patients should be catheterized for two reasons. Firstly, to see the colour of urine, which after ingestion of dye becomes choclate-brown coloured, especially in the initial stages, ¹⁹ and secondly, to monitor the urine output. Patients with acute renal failure need frequent dialysis support.

It is advisable to follow up a case of PPD ingestion for at least one month following ingestion, to look for delayed complications.

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