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Case Report

Homicidal poisoning with para-phenylenediamine hair dye: a case report

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ABSTRACT

Paraphenylenediamine (PPD) is a widely used chemical in various hair dye formulations. Due to its cheap price and easy availability, it emerges as a major cause of suicidal poisoning. It carries high morbidity and mortality and does not have any specific antidote. We reported an unusual type of poisoning of a 44-year-old man with PPD and how he was managed with an excellent outcome.

Keywords: Paraphenylene diamine, Hair dye, Stridor, Homicide, Renal failure

INTRODUCTION

Henna is used in the decoration of the palms of the hands, soles of the feet, nails, and tattooing. Because it's a lengthy and tedious procedure, paraphenylenediamine (PPD), the most popular component of hair dye products may be added to the mixture to enhance the process, darken it, and add more precision to the design.

Nowadays, thousands of consumers use hair dyes in developing countries particularly North East Africa like Sudan, Morocco, and the Indian sub-continent due to easy availability and affordability. PPD is considered to be an emerging etiological factor for suicides as it is rapidly absorbed into the blood through mucous membranes of the digestive tract after its oral intake.[1] Majority of hair dye poisoning are suicidal in nature, moreover, PPD may be ingested accidentally leading to high morbidity and mortality rate. PPD when applied to the skin has been observed to cause contact dermatitis, erythematous urticarial papules, eczema as well as systemic effect.[2]

CASE REPORT

A 44-year-old male came to the otolaryngology hospital in Khartoum, Sudan, with shortness of breath, cervicofacial edema, and dark-colored urine. He later devolved severe inspiratory stridor, cyanosis, and oliguria, a typical scenario of hair dye poisoning. The patient gave a history that he assumed someone had poisoned him by adding hair dye to his coffee drink. Fortunately, his relatives found him in an altered state and brought him to a nearby primary hospital, where he was subsequently referred to our tertiary hospital for emergency management. Few hours following his arrival oxygen saturation fell down to 82%. His tongue was getting more stiff and his neck was edematous. He had cyanosis, rapid pulse (120 B/M) and his blood pressure was 100/60.

Urgent therapeutic interventions and investigations

Anesthetists attempted to intubate him, and it was difficult. An urgent tracheostomy was done. The patient was admitted to the ICU where he was placed on

mechanical ventilator. Laboratory investigations revealed blood urea of 100 mg/dl and serum creatinine of 3.2 mg/dl. He was treated with renal dialysis for acute kidney injury.

Follow-up and outcome of interventions

Following tracheostomy, the patient was seen on the same day, cervicofacial and tongue edema started to subside, urine became normal in volume and less dark, and after 3 days cervicofacial edema completely subsided, the 4th day of admission, tracheostomy tube was removed following a successful trial of the tracheostomy tube weaning, and the wound was cleaned and dressed. The renal profile returned to its normal values, and the patient was discharged in good condition. One week later, the patient came to the otolaryngology refer clinic for follow-up and had no complaints.

DISCUSSION

Epidemiology and clinical features

PPD poisoning is emerging as an important etiological factor reported from Sudan, Morocco, the Indian subcontinent, and Egypt.[3]

There is a wide variety of clinical presentations regarding gender. PPD was found prevalent more in females than males.[4] Psychologic approach is curial since poisoning with PPD for suicide is noticed to be among the middle-aged population.[5] Hairdressers with regular exposure to PPD observed with a high prevalence of renal impairment. Accidental ingestion was observed among children. PPD intoxication varies from trivial skin lesions to severe systemic involvement involving the cardiopulmonary, nervous, muscular, and renal systems.[6]

Shortness of breath, cervicofacial and tongue edema were the dominating presenting manifestations, in acute severe poising patients usually presented with severe stridor due to angio-neurotic edema, in untreated cases this complicated with respiratory failure and subsequent death.[4] Myocarditis is a fatal and commonly overlooked complication of PPD poisoning, due to lack of awareness of this complication among doctors and rarity of data in the medical literature.[7] T-wave inversion and ventricular ectopic beats were observed in electrocardiographic tracings of most patients with PPD intoxicated, moreover, bradycardia followed by cardiac arrest was reported.[8]

Acute PPD poisoning is known to cause acute renal failure manifested with oliguria or anuria and this requires early referral to nephrology units for urgent dialysis.[9] Rhabdomyolysis is the main cause of acute renal failure and the morbidity and mortality are high once renal failure develops. Hypovolemia, hyperkalemia, and metabolic acidosis are well-known complications.[1] Dark coffee brown colored urine is pathognomic of PPD.[10] The enzyme creatinine kinase (CK) is elevated in majority of the patients with PPD poisoning. Hypocalcemia is also reported in this poisoning. Biochemistry revealed elevated liver transaminases and bilirubin. Liver function tests were elevated of aspartate and alanine aminotransferases up to maximum levels.[10] Acute poising with PPD was observed to cause neurotoxicity including drowsiness, paraplegia, paraparesis unconsciousness, generalized tonic-clonic convulsions, and deep coma.[11] Following ingestion of PPD, numbness and burning of the mouth and throat, epigastric pain, gastritis, and persistent vomiting occur, leading to dehydration and dysphagia.[12]

Muscle pain, protruded woody tongue and trismus were reported following poisoning with PPD.[7] Also, carpopedal spasm and both Chvostek's and Trosseau's signs were observed positive. Acute PPD was reported to cause acute live failure.[13]

PPD when applied locally may cause contact dermatitis, erythematous urticarial papules and eczema. Transcutaneous absorption of PPD can be rapid and may lead to systemic effects.[14]

Diagnosis and treatment

A high index of suspicion for hair dye poisoning should be kept when clinical features include severe pharynx and larynx angioedema accompanied by cervicofacial edema, rhabdomyolysis, and acute renal failure.[15]

Treatment in mainly supportive as there is no specific antidote for PPD, thus rapid identification of the unfortunate cases reduces mortality and morbidity rates.[16]

Emergency measures should include gastric lavage, early aggressive fluid therapy with crystalloids is mandatory to avoid the acute kidney injury associated with rhabdomyolysis.[17] Furosemide and mannitol are diuretics used to convert anuric to oliguric renal failure.[18] Intensive medical treatment with hydrocortisone, chlorpheniramine maleate (antihistaminic drug), and penicillin cover was life-saving.[19] Intravenous correction with calcium gluconate is required to correct hypocalcemia leading to rapid improvement in tetany.[20] Acute renal failure is better treated with either hemodialysis or peritoneal dialysis in the renal department.[21]

Acute PPD necessities ICU admission. Patients should be monitored for respiratory distress and endotracheal intubation has to be performed early if laryngeal edema develops.[22] Tracheotomy procedure is crucial for cases with severe cervicofacial edema with stridor to save the compromised airway. while endotracheal intubation is mandatory in mild cervicofacial edema.[23]

CONCLUSION

Hair dye poisoning carries high morbidity and mortality in underdeveloped countries, and no yet known antidote

is available. Due to its easy availability and low cost, it rises as a common type of self-harm, but it may occur as a criminal intention. Awareness of clinical features and early intervention improves the outcome.

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