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Review Article

Poisoning of paraphenylene diamine hair dye: review article

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ABSTRACT

Hair dye poisoning (Paraphenylenediamine-PPD) is a crucial cause of both intentional self-harm and accidental poisoning in the developing world. paraphenylene-diamine is a synthetic chemical that can cause local as well as systemic manifestation, of these are cervicofacial edema, laryngeal edema with stridor, rhabdomyolysis, severe metabolic acidosis and acute renal failure. Intervention earlier at the right time has been shown to improve the outcome. In this article reviewed the epidemiology, clinical features and treatment modalities for hair dye poisoning.

Keywords: Hair dye poisoning, Paraphenylenediamine, Stridor, Rhabdomyolysis, Myocarditis

INTRODUCTION

At least 4000 years back, hair dyes usage was known. Hair of Egyptian mummies was discovered to be dyed with henna (Lawsonia inermis) which is a dark green powder mixed with oil or water. In the days of the Roman Empire, leaden combs sink in vinegar were routinely used to darken greying hair.[1] Furthermore, henna used in decoration of palms of the hands and soles of the feet, nails and tattooing, and because its lengthy and tedious procedure, para-PPD, the most popular hair dye products and the main compound of permanent hair dye responsible for the toxicity, may be added to the mixture to enhance the process, to darken, and to give more precision to the design.[2]

Nowadays, thousand of consumers use hair dyes, in the developing countries particularly North, East Africa particularly in Sudan, Morocco and Indian sub-continent due to easy availability and affordability, where it considered to be an emerging etiological factor for suicides through ingestion, since PPD is rapidly absorbed into the blood through mucous membranes of the digestive tract after its oral intake.[2-5] Majority of hair dye poisoning are suicidal in nature Moreover PPD may ingested accidently leading to high morbidity and mortality rate. PPD when applied to skin observed to cause contact dermatitis, erythematous urticarial papules, eczema as well as systemic effect.

High index of suspicion for hair dye poisoning should be kept when clinical features includes severe angioedema of the pharynx and larynx accompanied with cervicofacial edema, rhabdomyolysis and acute renal failure. Acute PPD poisoning causes a swollen, dry, wooden tongue with stridor and this necessitate urgent tracheostomy.[6]

The confirmative evidence of hair dye poisoning due to PPD is the distinguish coffee brown color of the urine, moreover, the thin layer chromatography in the urine is diagnostic. Hypocalcaemia may occur in the setting of severe rhabdomyolysis or due to sodium EDTA. Patients can develop seizures, which may be due to toxins in dye or as a result of hypocalcaemia.

Treatment in mainly supportive as there is no specific antidote for PPD, thus rapid identification of the

unfortunate cases reduces mortality as well as morbidity rates.[7]

This review serves to highlight the epidemiology, clinical features and management of hair dye poisoning.

EPIDEMIOLOGY

Paraphenylenediamine (PPD) poisoning is emerging as an important etiological factor reported from Sudan, this was reported by Yagi et al in a series of 31 Sudanese children between 1984 and 1989.[8] Again Yagi et al reported an 18 case acute (PPD) poisoning.[9] In a general nephrology referrals clinic at Kaballo et al reported 89 case on a retrospective study.[10] PPD poisoning was the number one cause of poisoning in Morocco, this was reported by Filali et al in a 374 cases in a retrospective study in 2006.[5]

PPD ingestion is a very common form of poisoning in the Indian subcontinent, this was reported by Kondle between 2008 to 2011 in retrospective study of 50 case in Nellore.[11] Retrospective study was conducted over 7 years (2001-2008) in Egypt with acute PPD intoxication.[12]

There are a wide varieties in clinical presentation, regarding gender , PPD was found prevalent more in female than males, this was reported by Mohamed et al in a retrospective study for ten years at Sudan.[13] Psychologic approach is curial since poisoning with PPD for suicide noticed to be among middle age population, this was observed in a case report study in Sudan by Lamis et al and in India by Verma et al.[3,14] Again a cinema actor reported to had some quarrel with his parents about his marriage ingested PPD.[15] Hair dresser with regular exposure to PPD observed with high prevalence of renal impairment.[16] Accidental ingestion was observed among children, a 17 children with PPD intoxication were admitted to the paediatric nephrology unit in Sudan.[17] PPD intoxication varies from trivial skin lesion to severe systemic involvement involving cardiopulmonary, nervous, muscular and renal system.

CLINICAL FEATURES

Pulmonary system manifestations

Shortness of breath, cervicofacial and tongue edema were obviously the most common presenting manifestation, in acute severe poising with oral ingestion, patients usually presented with moderate to severe stridor due to angio-neurotic edema involving supraglottic and glottic region, in untreated cases this situation may be complicated with respiratory failure and subsequent death.[9-12,18] Pneumonia and pulmonary edema were reported in a 3 case study series.[4] Moreover spontaneous pneumothorax was observed on two females who gave history of consuming 150 to 200 ml of dissolved PPD.[19]

Cardiovascular system manifestations

Myocarditis is an urgent medical condition and a commonly overlooked complication of PPD poisoning, due to lack of awareness of this complication among doctors and rarity of data in the medical literature. T-wave inversion and ventricular ectopic beats were observed in electrocardiographic readings of large numbers of patients with PPD intoxication, moreover bradycardia that may be ended with cardiac arrest was reported, in the contrary The electrocardiogram was normal in 90% of patients enrolled in a retrospective study in India.[1,10,14,20,21]

Renal system manifestation

Acute PPD poisoning is well known to cause acute renal failure manifested with oliguria or anuria and this require early referral to nephrology units for urgent hemodialysis or peritoneal dialysis.[17,22] Rhabdomyolysis is approved to be the main cause of acute renal failure and the morbidity and mortality are high once renal failure develops. PPD causes rhabdomyolysis by facilitating calcium release, escape of calcium ions from the smooth endoplasmic reticulum, followed by repeated contraction and irreversible destruction in the muscle's component, which finally results in trapping of myoglobin cast over the renal tubules and hemolysis leading to acute tubular necrosis and acute renal failure.[10.23] Hypovolemia, hyperkalemia and metabolic acidosis are well known complication following PPD ingestion.[14] Dark coffee to brown colored urine is pathognomic of PPD poisoning. The enzyme creatinine kinase (CK) was reported to be above laboratory standard in majority of the patients with the PPD poisoning.[10,12,24] Proteinuria and hematuria are common complication following prolonged topical application with PPD.[16] Marked hypocalcemia with normal sodium, potassium, creatinine and phosphorus values were observed in case report series.[25]

Liver manifestation

On a case report series, lab investigations revealed elevated liver transaminases and bilirubin.[4] Another case report series in Nigeria observed liver function tests were rising of aspartate and alanine aminotransferases up to high levels.[20]

Nervous system manifestation

Acute poising with PPD observed to cause neurological toxicity including drowsiness, paraplegia, paraparesis, altered level of consciousness, generalized tonic-clonic seizure and deep coma.[4,14] It was observed that PPD is potentially toxic to human lens. Furthermore, eye protrusion and permanent blindness due to optic nerve atrophy following PPD intoxication were reported.

Gastrointestinal manifestation

It was assumed that the toxic effect of the PPD might be produced by the transformation of the PPD on mucus membranes to its oxidation product quinondimine, the later, is found to be the main cause of vigorous local irritation, therefore, in early hours following ingestion of PPD, numbness and burning of the mouth and pharynx, epigastric pain, persistent vomiting leading to dehydration, and dysphagia were observed symptoms and signs moreover anorexia and abdominal bloating were reported.[15,26]

Musculoskeletal system manifestation

Musculoskeletal pain, protruded woody tongue and trismus were reported following poisoning with PPD.[11] Again carpopedal spasm and both Chvostek's and Trosseau's signs were reported positive.[25] Chronic topical exposure to PPD observed to cause general malaise.[26]

Skin manifestation

PPD when applied to skin may cause contact dermatitis, erythematous urticarial papules and eczema. Transcutaneous absorption of PPD can be rapid and may lead to systemic effects. Chronic dermal exposure can cause lethargy, myalgia, purplish discoloration of gums and teeth, anorexia, GIT disturbances, liver and spleen enlargement, subacute atrophy of the liver, jaundice, chronic renal failure, progressive neurological symptoms and coma.

DIAGNOSIS AND MANAGEMENT OF HAIR DYE POISONING

Ingested PPD have a high morbidity and mortality rate and early immediate diagnosis and clinical management proved to be life saving in both moderate and severe poisoning.[9,10]

Even in the absence of laboratory facilities and when history is lacking in case of emergency situations, diagnosis of PPD intoxication is mainly with the obvious clinical features of cervicofacial edema, laryngeal edema, dark colored urine and renal impairment, moreover, diagnosis is established by the demonstration of myoglobin in urine and elevated levels of creatine phosphokinase and aldolase in the serum. Marked hypocalcemia and hyperuricemia during the oliguric phase and hypercalcemia during the diuretic phase are features of this condition.

There is no, yet, an antidote for hair dye poisoning. Thus, treatment is only conservative to save life, and to prevent some serious complications.[20,22,27] Patient age, amount of dye consumed and time between ingestion and hospital arrival were curial factors determining the outcome of PPD poisoning.[24] In early few hours of poisoning, emergency measures should include gastric lavage, while emesis induction is contraindicated because when PPD get in contact with oropharyngeal mucosa it causes severe edematous airway reaction.

Prompt abundant fluid therapy with crystalloids is mandatory to avoid the acute kidney injury associated with rhabdomyolysis.[27] Furosemide and mannitol are prescribed as diuretic helpful in conversion of anuric to oliguric renal failure. Comprehensive medical treatment with hydrocortisone, chlorpheniramine maleate (antihistaminic drug) and penicillin were reported as life saving.[9,11] Intravenous calcium gluconate is mandatory to correct hypocalcemia, leading to fast recovery from tetany.[25] Acute renal failure may be treated with either hemodialysis or peritoneal dialysis in renal department.[26,27]

Acute PPD necessities intensive care unit (ICU) admission. Patients should be monitored for respiratory distress and endotracheal intubation has to be performed early if laryngeal edema develops. Tracheotomy procedure is crucial for cases with severe cervicofacial oedema with stridor to safe the compromised airway.[11,17] While in endotracheal intubation is mandatory in mild cervicofacial oedema.[24] Following weaning off tracheostomy tube and hospital discharge subsequent referral to the psychiatry department for counselling is mandatory.[25]

CONCLUSIONS

The characteristic features of cervicofacial edema, stridor due to upper airway edema, rhabdomyolysis, and acute renal failure, put PPD poisoning as a provisional diagnosis. Since there is no specific antidote yet, early intubation/ tracheostomy, IV fluids, diuresis, gastric lavage and renal dialysis are the main management policies beneficial in this disease.

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