CASE REPORT

Homicidal Kala Pathar/Paraphenylene Diamine Poisoning

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ABSTRACT

Kala pathar commonly used as Hair dye and in Henna, poisoning is an uncommon form of poisoning in the west, however, in some parts of the world such asEast Africa and Indian Sub-continent it is not uncommon. The main component of kala pathar causing toxicity is Paraphenylenediamine (PPD). This compound has been found to cause angioneurotic edema, rhabdomyolysis and renal failure. We present a case of kala pathar poisoning who presented with respiratory distress due to laryngeal edema.This case report highlights the use of Kala pathar in Homicidal acts.

Keywords: Phenylene diamine, Homicide, Poisoning.

INTRODUCTION

Para-phenylenediamine, a derivative of paranitroaniline, has been used for dyeing furs,photochemical measurements, accelerating vulcanization and azo-dye manufacturing, as well as for oxidising hair dyes. Chemically, it is an aromatic diamine related to aniline. Though uncommon in the west, both accidental and intentional ingestion of PPD is frequently reported from Africa, the Middle-East, and the Indian subcontinent[1-3], where PPD is commonly mixed with henna, which is traditionally applied to colour the palms of hands and to dye the hairs. PPD accelerates the dyeing process. The toxicity of PPD includes skin irritation, contact dermatitis, chemosis, lacrimation, exophthalmos, or even permanent blindness, due to local contact. Ingestion of PPD produces two types of toxic effects. The first consists of rapid development of severe oedema of the face,neck, pharynx, tongue, and larynx with respiratorydistress, often requiring tracheostomy. In the laterphase, rhabdomyolysis and acute tubular necrosissupervene[4]. Vomiting, gastritis, hypertension, vertigo,tremors, and convulsions have been reported[5].

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CASE REPORT

A 55 year old female was admitted in ICU with 02 hour history of facial swelling and shortness of breath, and 15 minutes history of altered level of consciousness. On examination she was dyspnec, tachycardiac and cyanosed, her face, lips, tongue and larynx were swellen. There was decreased air entry in her chest. GCS was 06/15. There was no history of any drug use. She took morning tea along with her grand daughter 2 and a half hour back made by her daughter in law. Her grand daughter , 6-year old girl was brought dead in ICU, CPR was done but not revived. Diagnosis of Kala pathar /PPD poisoning was made.

However old lady was intubated and was put on mechanical ventilation. She was on respiratory support for 03 days, then she regained consciousness. Then she told that she had taken tea made by her daughter in law. This was second

marriage of her daughter in law with her son. She also told that her daughter in laws first husband and his brother died because of same symptoms due to intake of tea.

She developed renal and hepatic failure. On 10th post admission day her son took her home without medical advice. She was kept at dargah Saeedi Moosani and died there 3 days later.

We read in newspapers that her daughter in law was arrested and is under trial after killing 04 peoples with sweat tea poisoned with Kala pathar.

This case is important as this was the first noticed case where Kala pathar was used for homicide, which lead to killing of 04 peoples. In this context government should take action to ban selling of Kal Pathar for hair dying, as many precious lives are lost due to its suicidal and homicidal use.

DISCUSSION

PPD, a coal-tar derivative, on oxidation produces Bandrowski's base, which is allergic, mutagenic and highly toxic. PPD produces local as well as systemic toxic effects when applied topically and/or ingested. It is highly toxic when taken by mouth and the outcome depends mainly on the dose taken. The lethal dose of PPD is not known; estimates vary from 7-10 g[6].

Early manifestations of oral PPD intake (usually within 4 to 6 hours of ingestion) are numbness and burning of mouth and throat, vomiting, swelling of upper GI tract leading to dysphagia, and respiratory distress due to swelling of upper airway and angioedema. If the patient survives this acute phase, a late phase sets in usually after 12 hours of ingestion. It may last from few days to several weeks. Rhabdomyolysis, intravascular hemolysis, oliguria/anuria, and acute tubular necrosis/acute renal failure are delayed complications in some patients who survive the late phase. Rhabdomyolysis is the main cause of renal failure, morbidity and mortality[7]. Although our patient had ingested the lethal dose, she luckily did not develop renal failure probably because of early recognition of the poison and proper conservative management.

Cardiotoxicity and hepatic necrosis due to PPD have also been described, but very few reports in the literature have mentioned myocarditis[8,9,10], myocardial infarction[11], ventricular thrombus formation 10 and cardiac arrhythmias[11]. Clinical manifestations can vary from asymptomatic myocarditis to fatal arrhythmia. Electrocardiographic manifestations include ventricular and supraventricular ectopics, bundle branch block and ST-T changes. Increased serum levels of cardiac troponin-T provides evidence of myocyte injury in patients with clinically suspected myocarditis more sensitively than does conventional determination of cardiac enzyme levels[9]. Our patient had cardiac involvement as evidenced by ECG changes, which reverted to normal on the seventh day of admission. Myocardial involvement as evidenced by ECG changes is a rare manifestation of PPD poisoning mentioned in the literature.

PPD poisoning is a medical emergency and has high mortality if not recognized and intervened early. There is no specific antidote but the most important aspect of management is early recognition and supportive measures that include gastric lavage, normal saline and sodium bicarbonate infusion. Respiratory distress, myocarditis and cardiac arrhythmias are the major early challenges, which require vigilant monitoring to prevent early deaths. Intubation and ventilator support may be required for asphyxia and all modalities of dialysis have been found useful in renal failure[7].

During emergency particularly when patient history and good laboratory facilities are lacking, the characteristic angioedema of the face and neck with difficulty in breathing and acute renal failure manifesting as chocolate brown-colored urine could be suggestive of PPD poisoning. Vigilance must be maintained regarding cardiac manifestations and other late complications of PPD poisoning for at least 1 month in all cases.

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