CASE REPORT

ACUTE AIRWAY OBSTRUCTION DUE TO KALA PATHAR (PARAPHENYLENE DIAMINE) POISONING: A CASE REPORT

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ABSTRACT

Kala pathar (Paraphenylenediamine) is a commonly used chemical for hair dyeing in rural areas of Pakistan. It is a highly toxic substance which when ingested, accidentally or intentionally, causes cervicofacial edema and multiple organ failure. Here we present the case of a young female who was brought at Pakistan Air Force Hospital Jacobabad with typical clinical features of Kala pathar poisoning. Emergency tracheostomy was done to relieve the acute airway obstruction followed by symptomatic treatment which led to the full recovery of the patient. A high index of suspicion helped in the management of the case.

Keywords: Paraphenylenediamine, Rhabdomyolysis, Tracheostomy.

INTRODUCTION

Paraphenylenediamine (PPD) is an aromatic amine alanine derivative and is commonly known as "Kala pathar" in Pakistan. On oxidation it turns black from solid white state and is commonly used as hair dye[1]. PPD when ingested accidentally or intentionally is highly fatal however is readily available in our country[2]. It is the third leading cause of death in 15 to 44 years age group and is becoming a common source of intentional self harm in developing countries. PPD when ingested is highly toxic for the respiratory, muscular, cardiac, renal and hepatic systems. Its toxicity is mainly due to inhibition of cellular oxidation[3]. The main clinical features include cervicofacial edema, rhabdomyolysis, acute renal failure, sinus tachycardia and haemodynamic instability[4]. Treatment is essentially supportive as there is no antidote however endotracheal intubation (in cases which present early) or tracheostomy are lifesaving and can prevent mortality by relieving the upper air way obstruction[2].

CASE REPORT

A 20 year old female was brought in the emergency department of Pakistan Air Force (PAF) hospital Jacobabad by her father with history of sudden inability to speak and severe difficulty in breathing. On examination, the most predominant feature was a massively swollen and protruding tongue (fig-1). The face and neck were also edematous and the patient was in extreme distress (tossing and turning). There was also edema of the lower limbs. Stridor was present however she was maintaining blood oxygen saturation above 90%.

The history of onset was ambiguous initially, however, on probing the attendants reluctantly confessed the probable ingestion of Kalapathar (Paraphenylenediamine) by the patient. The patient was given injection solucortef (Hydrocortisone), injection avil (Pheniramine maleate) and high flow oxygen but the condition of the patient did not improve. A diagnosis of angioedema secondary to PPD poisoning was made. The attendants were explained the grave nature of the situation and high risk consent for emergency tracheostomy was obtained. The patient was shifted to the operation theatre where on passing of Foley catheter, dark brown urine collected in the urine bag. Emergency tracheostomy was done in local and intravenous anaesthesia. After successful tracheostomy the PaO2 was 99%. Antiseptic dressing was done, nasogastric tube was passed and the patient was shifted to the

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intensive care unit (ICU). In the ICU the patient was kept nil per oral till further orders and given Intravenous (I/V) fluids 3000 ml/24hrs, I/V antibiotics, injection Dexametha-sone 8mg 8hourly and Omeprazole infusion twice daily.

The patient was afebrile. Her pulse rate was 143/min. Her white cell count was 31.9 x 10^9/L, Alanine transaminase (ALT) was 238U/L, creatinine kinase was 74000U/L, Lactate dehydrogenase (LDH) was 11500U/L indicating severe rhabdomyolysis. Renal function tests were within normal limits. Electrocardiogram showed supraventricular tachycardia and early *p* wave depolarization. Dexamethasone was tapered slowly in the subsequent days. Within the next 24 hours the cervicofacial edema started subsiding. Input/output chart was maintained. Initially urine output was 900ml/day which improved to 1450ml on the second post operative day. Clear fluids were started via nasogastric tube on the second post operative day, followed by milk and egg flip. Thrombo embolic deterrent (TED) stockings were used for the lower limbs. Urine output became adequate on the third postoperative day and the cervicofacial as well as the lower limb edema was now almost gone. Care of tracheostomy tube was done throughout this period. Foley catheter was removed on the 5th post operative day as by now the urine colour was also normal. On the 6th post operative day nasogastric tube was also removed and the patient was shifted to ward. Trial for tracheostomy tube removal was given followed by removal on the 7th post operative day. The patient was discharged on the next day (fig-2) and was called for weekly follow up. All laboratory investigations were within normal limits after 4 weeks.

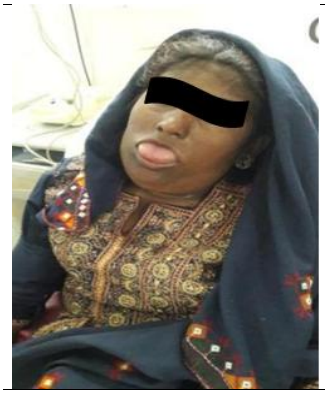

Figure-1: The patient on presentation showing cervicofacial edema.

Figure-2: The patient on discharge.

DISCUSSION

Paraphenylenediamine (PPD) is a life threatening chemical and is used as hair dye. In developing countries it is being used as a suicidal agent and is associated with high mortality. According to a study its use has female preponderance with an average age of 15 to 45 years and a mortality rate of 7.9%[5]. In another study by Khuhro *et al* 87.5% patients were females with a mean age of 25.7 years. Suicidal intention was found in 75% of the cases and most patients belonged to rural areas with low socioeconomic status suffering from social and family conflicts[6]. In a study by Khaskheli *et al* 66% of the patients were females, the intent of poisoning was suicidal in 98.94% cases and mortality rate was 31.67%[1].

The earliest documentation of PPD poisoning is as early as 1924 in the case of a barber. The symptoms have an onset of 4 to 6 hours after ingestion and are dose related. Gude *et al* have mentioned that the major complications include angioneurotic edema (72%), impaired renal functions (80%), rhabdomyolysis (100%), elevated liver enzymes (76%) and fatal cardiac arrhythmias (16%). All these are related to a systemic inflammatory reaction due to cytotoxic effects. The free radicals formed lead to break down of skeletal and cardiac muscles. In addition there is formation of highly nephrotoxic quinonediamine. There is renal tubular occlusion due to myoglobin casts and acute tubular necrosis. The cervicofacial/laryngeal edema and chocolate coloured urine are pathognomonic in most cases. As there is no antidote available treatment is mainly symptomatic and tracheostomy or haemodialysis may be required as per the condition of the patient[7]. In a case report by Prabhakaran the patient also had edema of the feet in addition to facial puffiness[8]. In a study by Balasubramanian *et al* it was seen that the mortality rate was

highest in the group that had cervicofacial edema and did not undergo tracheostomy due to delayed presentation. The mean age of patients was 24 years. The most frequent features were orofacial edema (68.8%), dark coloured urine (61.4%), muscle tenderness (56.8%), oliguria (6.4%), carpopedal spasms (4.8%) and 2.4% required dialysis[9]. According to a study by Akram *et al* a very large number of PPD ingestion cases present in hospitals in Pakistan and as there is no antidote for it, early arrival with immediate recognition and management could be lifesaving. Tracheostomy was done in all patients to prevent airway obstruction in the study and the author recommends that it should be done as early as possible[2]. Hence angioneurotic edema, rhabdomyolysis and acute renal failure should arouse suspicion of PPD poisoning[10]. In our case report similar signs and symptoms were noted and emergency tracheostomy was done. PPD poisoning is thus a medical emergency with high mortality if not recognized in time and needs high vigilance and index of suspicion[11].

CONCLUSION

Public awareness regarding the fatal effects of PPD should be emphasized along with measures at the government level so that it is not readily available.

CONFLICT OF INTEREST

This study has no conflict of interest to be declared by any author.

REFERENCES

1. Khaskheli MS, Shaikh S, Meraj M. Parapheny lenediamine poisoning: clinical features, complications and outcome in a tertiary care. Anaesth Pain & Intensive Care 2018; 22(3): 343-47.
2. Akram A, Shahid RA, Tariq M. Kala Pathar (Paraphenylene Diamine) poisoning; Role of tracheostomy: Our experience at DHQ Hospitals. Pak J Med Health Sci 2018; 12(2): 865-67.
3. Shafiq M, Maqbool F, Iqbal A, Baqai HZ. Kala Pathar poisoning. JRMC 2015; 19(1): 98-99.
4. Ishaque S, Haq A, Jurair H, Siyal H. Kala Pathar (Paraphenylene Diamine) poisoning and angioedema in a child: An unusual encounter. J Clin Toxicol 2016; 6(2): 294-99.
5. Lohano AK, Yousfani AH, Malik AA, Arain KH. Hair dye crucial threat to paraphenylenediamine poisoning and its mortality rate associated with laryngeal edema; a cross sectional study. RMJ 2017; 42(1): 60-63.
6. Khuhro BA, Khaskheli MS, Shaikh AA. Paraphenylene diamine poisoning: Our experience at PMC Hospital Nawabshah. Anaesth Pain & Intensive Care 2012; 16(3): 243-46.
7. Gude D, Bansal DP, Ambegaonkar R, Prajapati J. Paraphenylenediamine: Blackening more than just hair. J Res Med Sci 2012; 17(6): 584–86.
8. Prabhakaran AC. Paraphenylene diamine poisoning. Ind J Pharm 2012; 44(3): 423-24.
9. Balasubramanian D, Subramanian S, Shanmugam K. Clinical profile and mortality determinants in hair dye poisoning. Ann Nigerian Med 2014; 8(2): 82-86.
10. Punjani NS. Paraphenylene diamine (Hair Dye) poisoning leading to critical illness neuropathy. J Neurol Disord 2014; 2(5): 180-86.
11. Chaudhary SC, Sawlani KK, Singh K. Paraphenylenediamine poisoning. Niger J Clin Pract 2013; 16(2): 258-59.