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Hair Dye Poisoning with Refractory Arrhythmia & Interstitial Nephritis Rare Presentation

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Abstract

A 45year old female with no comorbid illness brought to emergency room with alleged history of consumption of 100ml of hair dye at 10am at her residence. She was initially treated at government hospital and brought to our hospital at 1:45pm. On arrival patient EIVIM1 in gasping state, with cervicofascial edema. In view of impending respiratory arrest patient was intubated with 6 size ET tube. After 5min she had Ventricular Tachycardia without pulse defibrillated with 200J, Chest compression given @100/min, after 2min, monitor shows Ventricular Fibrillation, defibrillated with 200J, Chest compression given @100/min Inj Adrenaline1mg, after 2min monitor shows Ventricular Fibrillation --- debrillated with 200J, Chest compression given @100/min Inj Adrenaline1mg given. Inj Amiodarone 300mg given after 3rd shock repeated with 150 mg after 2 minutes followed by infusion, in view of sustained Ventricular Fibrillation for 1hour continued treatment as per ACLS protocols. Inj Mgso4 2grams over 30min given in 100ml NS, Inj calcium Chloride 1gram Given, Inj soda bicarbonate 100meq given, during resuscitation.

INTRODUCTION

Hair dye poisoning is not rare, but becoming common in India. The main ingredients of hair dye are propylene glycol, resorcinol, sodium ethylene diamine tetra acetic acid (EDTA), paraphenylene diamine, preservatives, and perfumes. Compound responsible for the toxicity is paraphenylenediamine (PPD). Common clinical manifestations of PPD are cervicofacial edema, chocolate brown colored urine, rhabdomyolysis, oliguria, and shock.¹

Accidental and incidental causes of poisoning are increasing reported from underdeveloped and developing countries of Africa and Asia including india². Paraphenylene diamine (PPD) [C₆H₄(NH₂)₂] is an aromatic amine not found in nature and it is produced commercially by many industrial companies. It is a derivative of paranitroaniline that is available in the form of white crystals when pure and rapidly turns to brown when exposed to air.³ It is widely used in industrial products such as textile dyes, colored cosmetics, temporary tattoos, lithography plates, photocopying and printing inks, black rubber, oils, greases and gasoline.⁴ Paraphenylene diamine produces local toxic effects in the form of skin irritation, contact dermatitis, chemosis, exophthalmos and permanent blindness.

CASE REPORT

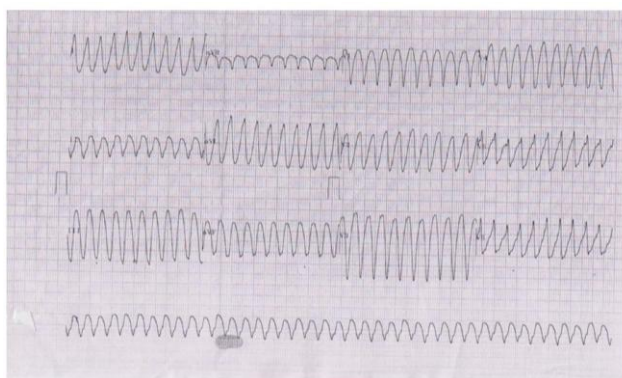
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government hospital and brought to our hospital at 1:45pm. On arrival patient E1V1M1 in gasping state, with cervicofascial edema. In view of impending respiratory arrest patient was intubated with 6 size ET tube. After 5min she had Ventricular Tachycardia without pulse defibrillated with 200J, Chest compression given @100/min, after 2min, monitor shows Ventricular Fibrillation, defibrillated with 200J, Chest compression given @100/min Inj Adrenaline1mg, after 2min monitor shows Ventricular Fibrillation --- debrillated with 200J, Chest compression given @100/min Inj Adrenaline1mg given. Inj Amiodarone 300mg given after 3rd shock repeated with 150 mg after 2 minutes followed by infusion, in view of sustained Ventricular Fibrillation for 1hour continued treatment as per ACLS protocols. Inj Mgso4 2grams over 30min given in 100ml NS, Inj calcium Chloride 1gram Given, Inj soda bicarbonate 100meq given, during resuscitation.

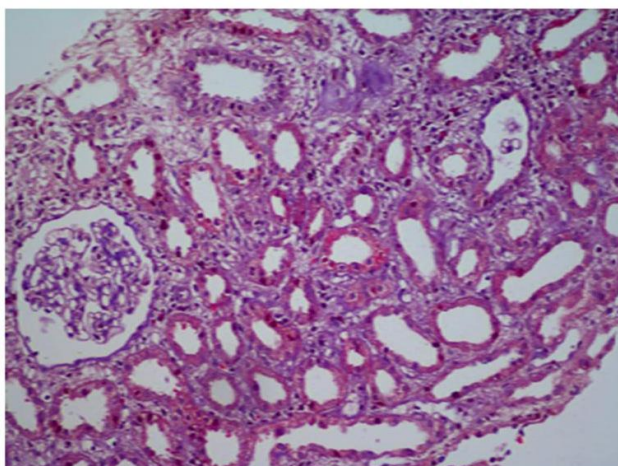
Return of spontaneous circulation attained after 1hour of Resuscitation with HR @140bpm, BP@ 90/60. ABG revealed met acidosis with respiratory alkalosis, patient shifted to Intensive care unit with Amiodarone and Dopamine infusion. Routine investigations revealed leucocytosis, calcium is 7.1, albumin 3.2(corrected calcium is 7.34), magnesium is 1.8, Elevated CPK, LDH, deranged Liver function tests and raised renal parameters , Troponin T done is Positive.2DEcho revealed normal with EF of 64%, USG abdomen reveals bilateral renal cortical echoes.

Patient was treated with IV fluids, steroids, PPI, antibiotics, antihypertensive. Nephrologist opinion was sought and dialysis was advised. Patient improved gradually was exubated on 3rd day. She continued to be oliguric haemodialysis done on alternative days. Psychiatrist opinion was sought and counselling given. Repeat leucocytes decreased. Nephrologist reviewed and continued Hemodialysis thrice a week. In view of persistent elevated creatinine levels, kidney biopsy was done which revealed Acute Interstitial Nephritis (AN). Patient recovered after treating with oral steroids for one month.

A) ECG Observation



B) Kidney Biopsy showing Acute Interstitial Nephritis.



DISCUSSION

The toxicity of paraphenylene diamine was first described in a hairdresser in 1924 following exposure due to occupational handling⁵. Paraphenylene diamine is a key ingredient in hair dyes used for colour enhancement. It is widely used in industrial products such as textile dyes, colored cosmetics, tattoos, photographic development, and gasoline. Paraphenylene diamine is used in its raw form for cosmetic purposes in Africa, Middle East, and Indian subcontinent. The toxic effects of paraphenylene diamine on the mucous membranes cause severe oedema of the face and neck, which frequently require assisted ventilation/emergency tracheostomy⁶, this is followed by rhabdomyolysis and AKI, which often culminate in death if not treated aggressively.⁷ Hypocalcaemia may occur in the setting of severe rhabdomyolysis⁸. The paraphenylene diamine aromatic structure has a direct toxic effect on kidney which makes its easy reabsorption and concentration in tubule, can cause Acute Tubular Injury.⁹ The paraphenylene diamine toxic effect on muscles results in rhabdomyolysis, which in turn contributes to AKI and the increased likelihood of sudden cardiac death¹⁰. Cardiac toxicity causing arrhythmia, heart block and sudden death was also reported in some studies. Methemoglobinemia, hypotension, hoarseness of voice, hepatitis, convulsions, coma, and sudden cardiac death are the toxic end of the spectrum¹¹. Sudden death after acute paraphenylene diamine intoxication appears to be due to myocarditis and arrhythmias¹². It is the

common cause of death in children and adults with paraphenylene diamine poisoning. Cardiac toxicity is mainly caused by the direct toxic effect of paraphenylene diamine on the heart, rhabdomyolysis of the cardiac muscle causing severe damage and hyperkalemia^{13, 14} Myocardial rhabdomyolysis and shock have also been described. These findings were also confirmed by Singh et al. reported a case of fatal myocardial damage due to paraphenylene diamine, with ECG features of multiple ventricular and supra ventricular ectopics with ST-T changes and positive cardiac troponin – T test due to cardiacmyolysis.¹⁵ On the other hand Olson proposed that critical hyperkalemia (resulting from rhabdomyolysis) causes atrioventricular (AV) block, ventricular fibrillation and cardiac arrest.¹⁶ Myocardial rhabdomyolysis due to PPD intoxication was also confirmed by postmortem microscopic examination of cardiac sections done by Ibrahim et al who found focal fragmentation and degeneration of cardiac muscle fibers with congestion and edema in between.¹⁷

CONCLUSION

Hair dye toxicity observed in south India especially in women of Andhrapradesh, which has become alternative to pesticide poisoning due to low cost. The toxicity of hairdye is dependent on the volume they ingested. Antidote availability has become a challenging except for symptomatic management in hair dye poisoning. In our case, patient had arrhythmia secondary to cardiacmyolysis and rhabdomyolysis leading to

acute kidney injury. Patient recovered from cardiacmyolysis within 48 hours, and persisted with acute kidney injury, hence Biopsy was performed which revealed as Acute Interstitial Nephritis, which responded to short course of oral steroids.

Disclosure

None of the authors have received any financial benefit from any source.

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