Clinical Profile and Complications of Hair Dye Poisoning

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Abstract- This is a prospective study of 31 cases of hair dye poisoning containing paraphenylene diamine. The clinical profile is dominated by development of angioedema, rhabdomyolysis and renal failure. Inotropic support, emergency airway management, alkaline diuresis and renal replacement therapy were the treatment modalities followed.

Index Terms- Paraphenylene Diamine poisoning(PPD), angioedema, acute renal failure.

I. INTRODUCTION

Suicide is a preventable public health problem, resulting in one million fatalities every year worldwide, increasing by 60% over the last 50 years especially in developing countries.1 Poisonsing is a preferred method of suicide and is one of the major problems encountered in emergency departments of hospitals.7

Poisoning with hair dye containing paraphenylene diamine is a new trend of intentional self harm in various developing countries of Asia and Africa,9 and is associated with high mortality.1

PPD is an active ingredient of ‘Kala Pathar’. It is crushed and mixed with henna and used as hair dye for enhancing its color.2 PPD ingestion causes symptoms involving different organs. Chemically, it is a derivative of para-phenylaniline, brown or black colour solid substance, easily soluble in hydrogen peroxide and not in water. It is a good hydrogen donor and metabolized by electron oxidation to an active radical by cytochrome P450 peroxidase to form a reactive compound called benzoquinone diamine. This can be further oxidized to a trimer known as Brandowaski’s base, a well known compound, reported to cause anaphylaxis and mutation. Ingestion of PPD causes rapid development of edema of the face, neck, pharynx, tongue and larynx initially and rhabdomyolysis followed by acute renal failure(ARF) as renal tubular necrosis occurs due to the deposits of the toxic metabolites of PPD.8

The compound PPD is highly toxic. When taken orally death occurs within the first 6-24 hours due to angioneurotic edema.2 Smaller doses cause angioneurotic edema and hepatitis while moderate doses cause acute renal failure within the first week. Despite the high frequency of cases and mortality, no antidote is available for this poisoning.6

As there has been a recent increase in frequency of hair dye poisoning, this study was done to study clinical profile of Paraphenylene diamine containing hair dye poisoning and outcome.

II. MATERIAL AND METHODS

This study was done in Osmania general hospital, Hyderabad during the period November 2011 – October 2013. Patients who were admitted in emergency department with alleged hair dye ingestion were taken up for study. Clinical history, complaints, physical examination, investigations, treatment modalities followed, clinical progress and outcome were recorded. Routine Investigations done at regular intervals include:

Complete blood picture, Random blood sugar, Renal function tests, Liver function tests, Serum electrolytes and Creatinine Phosphokinase(CPK) levels were done. Complete urine examination, Chest X-ray, Electrocardiogram, Arterial blood gas (ABG) analysis, Ultrasonography of abdomen and 2D-Echocardiogram (depending on patients clinical status) were done.

Gastric lavage was done in 20 patients in whom it was possible. All patients were treated with steroids and intubation was done in 9 patients; tracheostomy in 8 patients. Mechanical ventilatory support was needed for 3 patients. In patients with hypotension, inotropic support was given. Sufficient quantities of oral fluids were given whenever possible and intravenous fluids to maintain hydration. Diuresis / alkaline diuresis was done to avoid acute kidney injury secondary to rhabdomyolysis. In patients with acute renal failure, renal replacement therapy was instituted.

III. RESULTS

The frequency of hair dye ingestion was more in females (80.64%) than in males (18.75%). The tendency to commit suicide was more in the age group 21-30 years in both males and females. Majority of patients (96%) had burning pain in mouth, throat and abdomen. Angioedema was seen in 48% of patients with varying severity. 80% of patients had vomiting and dysphagia. Nearly 58% of patients had dark colour (cola colour) urine after ingestion of the dye. Almost all patients developed generalized muscle pains. Dyspnoea was seen in 74% of patients, of whom 29% had stridor and 45% had wheeze. 19% patients developed oral erythema and erosions. 16% patients developed pedal oedema, oliguria was seen in 12% of the patients. Other less frequent symptoms observed were weakness 16%, seizures
12%. Hypertension developed in 15% of patients, hypotension in 6.4% of patients.

Burning pain in mouth/throat/abdomen, vomiting were the earliest symptoms in almost all patients. Hypotension developed in duration of 6 hrs, angioedema, dysphagia, dyspnoea, dark colour urine, myalgias developed within 6 - 24 hours, Hypertension in 4 – 5 days. Oliguria appeared within a mean duration of 5 – 6 days, Pedal oedema with or without anasarca appeared within a mean duration of 7 - 10 days.

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Raised total count was seen n=13, elevated blood urea in n=11, raised creatinine n=13, increased bilirubin n=2, raised CPK levels above 300IU /lt n=25 patients and raised potassium in n= 8 patients respectively. Chest x ray showed features of pulmonary edema in 3%, pleural effusion and consolidation in 6% of patients.

ECG abnormalities like sinus tachycardia, non specific T wave inversions, QTc prolongation, and sinus bradycardia were seen in 38% of patients.

Renal biopsy was done in 3 cases of acute kidney injury, among 3 biopsies 2 showed acute tubular necrosis and 1 showed pigment nephrosis.

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28.5% of patients with CPK levels more than 10,000 IU/L developed ARF. Hypocalcaemia was observed in 51% of patients, 2 developed carpopedal spasms which responded to calcium administration.
ARF was seen in 19.3% of patients who ingested more than 50 ml of dye and was not seen in patients who ingested less than 50 ml of dye. The correlation was statistically significant.

About 58% of patients were discharged from hospital in good general health condition after nearly 10 days (ranging from 4 – 21 days). 25% patients left against medical advice. 12% patients expired due to complications like pneumonia, sepsis and ARF.

**DISCUSSION**

Super-Vasmol, a cheap, freely super available hair dye is emerging as a major cause of suicidal poisoning in India. It contains potential toxins including paraphenylenediamine, sodium ethylene diamine tetra acetic acid and propylene glycol which can result in multiorgan dysfunction.[2]

Prevalence was more in the age group of 21– 30 years with female preponderance of 80.64% . Akber MH[3] and Anugrah Chrispal et al[4] identified similar age group of 20.5±4.65 years with female predominance.

The mean amount of dye consumed was found to be 52.31± 29.56 ml in our study (range 10 – 100 ml). The toxic effects depend on the dosage.[4]

Angioedema developed in 48.38% of the patients with varying severity in our study with a mean duration of 3.7 ± 1 hours (range 3 – 6 hours). In 09(28%) patients with severe angioneurotic edema and stridor emergency tracheostomy was done. Suliman et al[5] observed a tracheostomy rate of 15.8% in his patients, a study at Multan[1] showed this rate to be 60% while 87.5% of our patients required this procedure.

Rhabdomyolysis was noted in 80.9% of patients. Kallel et al also noted rhabdomyolysis in 47.4% of patients in his study.[5]

Acute Renal Failure (ARF) developed in 19% of our patients requiring renal replacement therapy. ARF occurred in 37.5% of patients by Akber et al).

Mortality was 12.9% in our study, as compared to Akbar et al at Multan which was 20%[1]

As there is no specific antidote for hair dye poisoning and burden of cases has been increasing, there is need for bringing awareness among the public regarding the toxic effects of this hair dye. Primary care physicians, intensive care physicians need to be aware of its clinical manifestations and management. One should be vigilant of the anticipated complications and be prompt in instituting good supportive management to minimize the morbidity and mortality.

**REFERENCES**


**AUTHORS**

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