

Original Research Article

Role of methyl prednisolone in reducing mortality and morbidity in hair dye poisoning

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ABSTRACT

Background: Hair dye containing paraphenylenediamine (PPD) is widely used in India because of its free availability and low cost. PPD produces local as well as systemic toxic effects when applied topically and/or ingested. This study was undertaken to find the role of methyl prednisolone in reducing mortality and morbidity in hair poisoning and to study the cardiac complications of hair dye poisoning.

Methods: The present study was carried out among 1000 cases admitted in the Department of Medicine, MLB Medical College, Jhansi with history/characteristic and clinical features of hair dye poisoning. The complication and mortality profile was studied. Statistical significance was drawn with the help of Microsoft excel 2003.

Results: Mortality in acute hair dye poisoning cases was 23.20%. Majority of deaths (50.04%) occurred within 24 hours of hair dye ingestion and were because of asphyxia and respiratory failure. Mortality in 44.96% cases was after 24 hours and were mainly due to renal failure and its related complications hyperkalaemia and cardiac complication. Lower mortality was observed in group A (8.40%) as compared to group B (8.0%) and C (4.0%) in association with ST and T wave changes.

Conclusions: PPD is available quite freely and used extensively. Awareness regarding its poisonous effects should be increased among the medical health care workers.

Keywords: Hair poisoning, Methyl prednisolone

INTRODUCTION

Most popular types of hair dyes are the permanent oxidation dyes. Basically, these are oxidized 'paradyes' such as paraphenylenediamine (PPD). Hair dye containing paraphenylenediamine (PPD) is widely used in India because of its free availability and low cost. PPD produces local as well as systemic toxic effects when applied topically and/or ingested.¹

PPD is a good hydrogen donor and metabolized by one electron oxidation to a cation free radical by cytochrome P450-peroxidase to form a reactive benzoquinonediimine. This a further oxidized to a highly toxic trimer known as

Bandrowski's base, a compound reported to cause anaphylaxis. It causes violent local irritation of the mucous membranes and skin of sensitive individuals, then acetylated to dactyl derivative. The characteristic oedema production is said to be due to increased permeability of the blood vessels.

PPD poisoning also causes rhabdomyolysis and acute tubular necrosis. Rhabdomyolysis cause hyperkalemia and it is exacerbated by coexistent metabolic acidosis. This study was undertaken to find the role of methyl prednisolone in reducing mortality and morbidity in hair poisoning and to study the cardiac complications of hair dye poisoning.

METHODS

This study has been carried out among patients who presented with history and/or clinical features of hair dye poisoning in emergency department of MLB Medical College, Jhansi after taking permission from the institutional ethical committee. Patients of age above 15 years with history or /and characteristic clinical features of acute hair dye poisoning were included. Patients with history of mixed poisoning, those with history of liver, renal disease, cardiac disease were excluded. Informed consent was taken from all the study participants. Besides taking full history and detailed clinical assessment routine investigations were performed according to clinical scenario. No antidote is available against PPD so management is basically supportive. The patients were divided into 3 groups. Group allocation was done on lottery basis and treatment was given accordingly. Patients were followed up throughout hospital stay and thereafter.

Group A

Inj. Methylprednisolone 1gram in NS 100ml I/V slowly over 1 hour per 24hours along with other supportive treatment.

Group B

Inj. Hydrocortisone 100mg I/V every 8 hourly along with other supportive treatment.

Group C (control)

No steroid was given only supportive treatment given. In all the 3 groups, incidence and mortality with cardiac complications in hair dye cases was observed and the effect of steroid on morbidity and mortality in patients of hair dye poisoning with cardiac complication and renal failure was seen.

RESULTS

This study had been carried out in 1000 patients admitted in the hospital with history of hair dye poisoning and/or characteristic clinical feature of hair dye toxicity. Out of 1000 cases, maximum no. of cases (990 cases; 99.0%) were of suicidal intent, while 8 cases (0.8%) were accidental and 2 cases (0.2%) were of homicidal intent.

Table 1: Distribution of cases according to age and sex.

Age groups (years)	Male	Female	Total No.	Percentage
15-25	102	339	441	44.1
26-36	85	315	400	40.0
36-45	79	32	111	11.1
>45	20	28	48	4.8
Total	286	714	1000	100

Maximum number of patients were in the age group of 15-25 years (44.1%) and were females (71.4%). The most prominent clinical feature was characteristic severe edema of face and neck. 745 cases (74.5%) were presented with this typical facial appearance. In 17 cases, there was distinctly marked oedema over eyes. Dysphagia which was present in 726 cases (72.6%). The severity of dysphagia was significantly higher in cases with marked oedema of face and neck. Out of 726 cases, 8 cases had marked dysphagia even after disappearance of swelling of face and neck. Upper GI endoscopy were performed and benign stricture of oesophagus in one case and grade 1 oesophagitis were found in 2 cases.

Table 2: Mortality pattern in hair dye cases in different groups with renal failure.

Renal function test	Group A (n=119)		Group B (n=84)		Group C (n=58)	
	No.	%	No.	%	No.	%
> 1.5-3.5	14	11.7	11	13.0	6	10.40
3.1-4.5	4	3.30	4	47.0	4	6.90
4.6-6.0	5	4.20	5	5.90	4	6.90
6.1-7.5	4	3.30	3	3.50	3	5.10
>7.6	6	5.00	5	5.90	5	8.60
Total	3	27.5	28	33.33	22	37.93

P = 0.405 (NS)

Other clinical features of hair dye poisoning were chocolate brown colour urine (549 cases; 54.9%), pain and rigidity of limbs (480 cases; 48.0%), decreased urine output (130 cases; 13.0%), rise in blood pressure (80 cases; 8.0%), respiratory difficulty (229 cases; 22.9%), nasal twang of voice (59 cases; 5.9%), convulsion (21 cases; 2.1%), nasal regurgitation (7 cases; 2.94%), chest pain (141 cases; 14.1%), decrease in BP (149; 14.9%), palpitation (139 cases; 13.9%), tachycardia (214 cases; 21.4%), presyncop/syncope (47 cases; 4.7%).

Mortality in acute hair day poisoning cases was 23.20%. Majority of death (50.04%) occurred within 24 hours of hair dye ingestion and were because of asphyxia and respiratory failure secondary to the upper respiratory tract and cardiac oedema. Mortality in 44.96% cases was after 24 hours and were mainly due to renal failure and its related complications hyperkalemia and cardiac complication. Mortality was higher in Group A and B as compared to Group C despite medical management and dialysis.

Table 3: Mortality pattern associated with ST and T wave changes in different groups.

Outcome	Group A		Group B		Group C	
	No.	%	No.	%	No.	%
Recovery of ECG changes	218	91.6	92	92.0	64	91.43
Expired	20	8.40	28	8.00	6	8.57

P = >0.1 (Not significant)

Nonspecific ST and T wave changes were present in all the 408 cases in the form of T wave inversion in all the chest and limb leads, ST segment elevation or ST segment depression.

Table 4: Other ECG changes observed in subjects.

ECG changes	No. of patients	Percentage
Atrial fibrillation	86	21.07
Extra systoles	136	33.33
Bundle branch block	64	15.69
Tachycardia	214	19.36
1 ^o and 2 ^o AV block	79	33.82
Complete AV block	59	14.46
Ventricular tachyarrhythmia	38	9.31

Transthoracic echocardiographic abnormality was observed in patients with suspected myocarditis. Regional wall motion abnormality was seen in 35.78% patients. LVEF <35% in 45.59%, LVEF 35-50% in 26.41%, LVEF >50% in 27.94%. Cardiac dilatation in absence of regional coronary artery disease was seen in 30.8% cases. Troponin T was positive (more than 0.1 ng/ml) in 364 (89.21%) cases with positive ECG changes.

Out of 408 cases of suspected Myocarditis, 364 cases (89.22%) were Troponin-T positive and 44 cases (10.78%) were Troponin-T negative.

DISCUSSION

Out of 1000 cases in the present study, a higher proportion of cases were females (71.4%) and were from the younger age group (15-25 years) (44.1%). Filali A et al also made similar observations in their retrospective study of PPD poisoning cases reported to the poison control centre of Morocco.² It revealed 374 cases with a female predominance (77%). The group most prone to PPD poisoning were the young population (15.1-25 and 25.1-35 years old age groups accounting for 54.3% and 15.2% respectively).

In another study by Kallel H et al among 19 cases of systemic PPD intoxication admitted in the ICU of Habib Bourguiba University Hospital (Sfax, Tunisia) over 6 years, the average age SD was 27±16.8 years and the sex ratio (M/F) was about 0.58.³

In present study, all 1000 cases were due to ingestion of hair dye poison. Out of 1000 cases, maximum number of cases (990 cases; 99.0%) were of suicidal intent, while 8 cases (0.8%) were accidental and 2 cases (0.2%) were of homicidal intent.

Filali A et al, also made similar observations. They observed that commonest source route of poisoning was by ingestion (93%) and among majority of subjects poisoning was intentional (78.1%).² In another study by

Radhika et al majority of poisoning was also intentional (99%).⁴

Kallel H et al also observed that the reason for ingestion was attempted suicide in 73.7% of patients and accidental ingestion in 26.3% of patients.³

In this study, the most prominent clinical feature was characteristic severe edema of face and neck. 745 cases (74.5%) were presented with this typical facial appearance. This feature is so characteristic of the hair dye poisoning that it leads to the diagnosis in cases where history of hair dye ingestion was not present. In 17 cases, there was distinctly marked oedema over eyes.

Another common feature, in this study was dysphagia which was present in 726 cases (72.6%). The severity of dysphagia was significantly higher in cases with marked oedema of face and neck. Out of 726 cases, 8 cases had marked dysphagia even after disappearance of swelling of face and neck. Upper GI endoscopy were performed and benign stricture of oesophagus in one case and grade 1 oesophagitis were found in 2 cases.

Other clinical features of hair dye poisoning were chocolate brown colour urine (549 cases; 54.9%), pain and rigidity of limbs (480 cases; 48.0%); decreased urine output (130 cases; 13.0%), rise in blood pressure (80 cases; 8.0%), respiratory difficulty (229 cases; 22.9%), nasal twang of voice (59 cases; 5.9%), convulsion (21 cases; 2.1%), nasal regurgitation (7 cases; 2.94%), chest pain 141 cases (14.1%). Decrease in BP 149 (14.9%), Palpitation (139 cases, 13.9%), tachycardia (214 cases, 21.4%), presyncop/syncope (47 cases 4.7%).

Reason of nasal twang of voice and nasal regurgitation during recovery period in patients of acute hair dye poisoning was not clear. On laryngoscopy, no abnormality was found excepts oedema of arytenoid cartilage. These symptoms improved by giving corticosteroids for an average of 7±2 days.

Our findings are comparable to Yagi et al who studied series of 18 cases of acute hair dye (paraphenylenediamine) poisoning reported sporadically to accident and emergency department of Khartoum North Teaching Hospital (Sudan) over a period of two years.⁵ They noticed acute paraphenylenediamine poisoning face (characteristic edema of head and neck and wooden hard swollen protruding tongue), dysphagia, typical chocolate brown colour urine, respiratory difficulty, vomiting, drowsiness and exophthalmos with permanent blindness in one case.

Kallel H et al, also studied retrospectively 19 cases of systemic PPD intoxication.³ They found that at admission, clinical symptoms were dominated by cervicofacial edema (79%), chocolate brown colour urine (74%), upper respiratory tract oedema (38.4%) oliguria (36.8%), muscular oedema (26.3%) and shock (26.3%).

Razik H et al, also studied 20 cases of death due to PPD intoxication and observed clinical features dominated by cervicofacial oedema (65%), acute respiratory distress (38.5%) cardiogenic shock (30%), neurological disorders with major coma (35%) and early anuria (15%).⁶

Yagi et al reported one cases of acute systemic PPD toxicity in which the patient developed optic atrophy with resultant blindness.⁵

Out of 408 patients studied, 238 (47.6%) from group A, 100 (32.4%) cases from group B and 70 (35%) cases from group C were having suspicion of myocarditis on the basis of electrocardiographic changes and clinical features.

Transthoracic echocardiography was done in all these cases the findings were regional wall motion abnormality in 146 (35.78%) and decreased left ventricular ejection fraction (LVEF 35%) in 186 (45.58%) patients. On day II to Vth which subsequently improved on follow up in cases survived.

Cardiac dilatation was present in 126 (30.88%) cases in the absence of regional coronary artery disease and evidence of rapid recovery of ventricular function during follow-up in 98 (77.7%) cases was observed. with positive ECG changes which further supported the diagnosis.

Troponin T was positive (more than 0.1 ng/ml) in 364 (89.21%) cases with positive ECG changes which further supported the diagnosis.

Cases developing suspicion of myocarditis as per clinical features, electrocardiography, troponin T and transthoracic echo cardio-graphy with 54 (13.23%) out of 408 cases and those who developed ventricular tachyarrhythmia 38 cases (9.3%) were having further bad prognosis 29 (76.3%) cases expired out of 38 despite standard medical management.

Zeggwagh AA et al, reported a case of myocarditis induced by PPD.⁷ An 18 years old female was admitted to the hospital with asphyxia and rhabdomyolysis, ECG showed ventricular extrasystole and negative T wave. A transthoracic echocardiography showed significant left and right ventricular hypokinesia (LVEF=35%) and a left ventricular apical thrombus. Anticoagulation treatment with heparin was initiated. A follow up Echo performed on 15th day, showed normalization of ventricular function disappearance of the thrombus without any embolic event. Echocardiography is indicated in the myocarditis induced PPD poisoning to prone the ventricular dysfunction as well as the presence of thrombus. Thrombus was not observed in any of the cases in our study in which echocardiography was done.

Brahmi et al reported a case of myocarditis with myocardial infarction induced by PPD which was

confirmed by angiography that showed septoapical hyperkinesia due to spasm of the anterior interventricular coronary artery.⁸

Jatav OP et al, also reported a case of myocardial damage in PPD poisoning.⁹

In the present study mortality in acute hair day poisoning cases was 23.20%. Majority of death (50.04%) occurred within 24 hours of hair dye ingestion and were because of asphyxia and respiratory failure secondary to the upper respiratory tract and cervical oedema. Mortality in 44.96% cases was after 24 hours and were mainly due to renal failure and its related complications hyperkalemia and cardiac complication.

PPD ingestion led to a respiratory, a muscular, renal and cardiac syndrome. Respiratory failure mainly determined the short-term prognosis, whereas long term prognosis was affected by the importance of muscular renal and myocardial damage.

Other studies also showed similar mortality rate. Razik H et al, observed in their study that mortality was 70% in less than 48 hrs.⁵ Yagi and H kallel et al reported in that emergency tracheotomy and oral tracheal intubation in all serious cases improved the prognosis and reduced the mortality because of asphyxia and respiratory failure.^{3,4} Deaths among adults in most of the series were mainly due to renal failure.

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