Original Research Article

A prospective study on clinical manifestations of paraphenylene diamine containing hair dye poisoning

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Abstract

Introduction: In the developing countries like ours Pesticide poisoning is one of the leading contributors to this preventable tragedy. Recently, Hair dye poisoning is emerging as an important etiological factor. Hair dye consumption is not an uncommon means of intentional self-harm. Hair dye is available in various forms. It's cheap and easily available form is Hair dye with chief constituent chemical Para-phenylenediamine (PPD). **Aims:** To study various clinical manifestations of Paraphenylene diamine containing Hair dye poisoning and to observe the temporal sequence of symptoms, Laboratory profile in the patients and relative frequency of complications and their outcome. **Materials and methods:** It is Prospective Observational Study done in Medical wards and acute medical care unit. Study done on Clinical presentations, Complications, Laboratory profile, Mortality and Treatment of cases of paraphenylene diamine hair dye ingestion were noted. Patients of alleged hair dye ingestion were taken up for study after the exclusion criteria were ruled out. **Results:** It was observed that the tendency of poisoning by hair dye was more in females than in males and was more in the age group of 15 – 30 years, as with any other poisoning. The symptoms and signs that were observed in this study are burning pain in mouth / throat / abdomen, vomiting, cervicofacial edema, dysphagia etc. The order of chronology of development of these symptoms was burning pain in mouth / throat / abdomen with vomiting within first 40 – 60 minutes maximum upto 120 minutes, followed by development of cervicofacial edema, dyspnea and dysphagia after few hours. A significant statistical correlation was found to exist between development of AKI and the levels of CPK in blood, rhabdomyolysis. Antihistamines, steroids and maintenance of good hydration along with diuresis form the mainstay of therapy. **Conclusion:** As the burden of handling hair dye cases has been increasing in this part of the state also, primary care physicians, intensive care physicians and nephrologists 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.

Keywords: Paraphenylene diamine, Clinical manifestations, Complications.

Introduction

Globally suicide rates have increased by 60% in the 50 years. Suicide is now ranked among the three leading causes of death in the age group between 15 and 44 years[1]. In the developing countries like ours Pesticide poisoning is one of the leading contributors to this preventable tragedy. Recently, Hair dye poisoning is emerging as an important etiological factor. Hair dye consumption is not an uncommon means of intentional self-harm. It has been reported around the world, more so in the underdeveloped and developing countries. Hair dye is available in various forms. It's cheap and easily available form is Hair dye with chief constituent chemical Para-phenylenediamine (PPD). Though uncommon in the west, both accidental and intentional ingestion of PPD is frequently reported from Africa, the Middle-East, and Indian subcontinent[1].

Paraphenylene diamine poisoning was the number one cause of poisoning in Morocco during the 1990s. Many case reports have been published since then from African, Middle-East and Indian Subcontinent, describing various manifestations of PPD containing hair dye poisoning. Hair dye poisoning containing Paraphenylene diamine has high mortality and morbidity. Its incidence has increased dramatically over the past 5 years. There have been many sporadic reports of the poisonings with PPD hair dyes, but there has been no large prospective studies regarding its clinical presentations and outcomes[2,3].

Different and varied manifestations have been reported by various authors. Manifestations like skin irritation, chemosis on local contact, on ingestion edema of face, neck, pharynx, tongue, and larynx with respiratory distress, often requiring tracheostomy. In later phases, rhabdomyolysis and acute tubular necrosis supervene. Vomiting, gastritis, hypertension, vertigo, tremors, convulsions, myocarditis and death were also reported. Since, as PPD hair dye poisoning as such a varied manifestations and there has been a recent increase in frequency of these poisonings presenting to our hospital Gandhi Hospital, this study was taken up to observe the clinical profile of Paraphenylene containing hair dye poisonings and its outcome.

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Materials and methods

It is Prospective Observational Study done in Medical wards and acute medical care unit of the Department of Medicine, Gandhi Hospital, Secunderabad. Study done on Clinical presentations, Complications, Laboratory profile, Mortality and Treatment of cases of paraphenylene diamine hair dye ingestion were noted

Inclusion Criteria

All cases of PPD containing hair dye ingestion poisoning getting admission in Acute medical unit in our hospital.

Exclusion Criteria

Hair dyes not containing PPD are excluded from the study and Poisonings other than Hair Dye, Patients with significant Renal and Cardiac disorders and those who were on some form of medical or radiation therapy or surgical intervention within past 3 months of admission.

This study was carried out during the period July 2018 – 2020. Patients of alleged hair dye ingestion were taken up for study after the exclusion criteria were ruled out. Informed consent was obtained from every patient or patient's relatives. Clinical history, complaints, physical examination findings, investigation reports, treatment modalities followed, clinical progress and outcome were all recorded on the prepared proforma.

The basic investigations were carried out at regular intervals during the hospital stay and also ENMG(when patients developed weakness or sensory symptoms), 2D-Echocardiogram (depending on patients clinical status and ECG findings), ABG analysis and USG abdomen

Patients with symptoms of generalised body aches with / without generalised weakness with serum CPK levels more than 1500IU/L (more than 5 times the upper limit of normal) were diagnosed to have Rhabdomyolysis. RIFLE criteria were followed to classify patients as to those who have developed AKI.

Treatment and supportive care as Gastric aspiration through nasogastric tube was done whenever possible. All patients were treated with steroids and antihistamines to manage allergic manifestations. Activated charcoal and antacids were given in all possible conditions. Those who presented with vomiting were treated with intravenous antiemetics.Emergency Airway management was done was done in all cases that presented with stridor, wheeze with falling oxygen saturations. Endotracheal intubation, tracheostomy was done as per patient clinical status. Mechanical ventilation was given whenever required. In cases presenting with hypotension, inotropic support was administered. Intravenous fluids of approximately 2 – 3 litre / day were administered in all the patients along with sufficient quantities of oral fluid intake whenever possible to maintain hydration.

Diuresis with / without alkaline diuresis was done to avoid Acute Kidney Injury secondary to rhabdomyolysis to maintain high urine output. Patients with acute renal failure, renal replacement therapy was instituted. Patients with symptomatic hypocalcaemia were treated with oral calcium with / without IV calcium gluconate infusion dependent on clinical status.

Results

Table01: Demographic distribution of patients

Age Group(Years)	Number of Patients	Percentage
≤ 20	11	26.20
21 – 30	27	64.30
Above 30	4	9.50
Total	42	100.0
Gender		
Male	10	23.80
Female	32	76.20
District of residence		
Hyderabad	10	24
Ranga Reddy	10	24
Nizamabad	8	19
Adilabad	3	7
Nalgonda	6	14
Karim Nagar	5	12
Symptom		
Pain Oral Cavity	40	95%
Vomitings	38	90%
Angioedema	36	85%
Dysphagia	36	85%
Sign		
Dyspnea	32	76
Myalgia	32	76
Dark Urine	34	80
Oliguria	4	9.5
Seizures	4	9.5
Weakness	5	11.9
Oro-Facial Edema	36	85.7
Icterus	0	0
Pedal Edema	5	11.9
Oral Erythema	10	23.8
Hypertension	5	11.9
Hypotension	2	4.7
Stridor	12	28.5
Wheeze	19	45.2
Epigastric Tenderness	22	52.3

The relative frequency of hair dye ingestion was more in females (76 %) than males (24%). It was further observed that the tendency to commit suicide was more in the age group 21 -30 years in both the males and females. Nearly 70% males and 62.5% of female patients were of the age group 21 – 30 years. Nearly 48% of patients that presented with hair dye poisoning came from Hyderabad and Ranga Reddy districts which are in proximity to Gandhi Hospital, whereas Nizamabad accounted for 19% of the patients and Adilabad had 3 cases presenting to our hospital. Majority of patients (95%) had burning pain in mouth, throat and abdomen. Also, angioedema was seen in 85% of patients with varying severity. 90% of patients had vomiting and dysphagia. Nearly, 80 % of patients had dark colored mostly cola colored urine after ingestion , patients had also developed generalized muscle pains. Dyspnea was seen in 76% of patients, of whom 28.5% developed stridor and 45.2% developed wheeze. 24% patients developed oral erythema and occasional erosions due to ingestion of the dye. 11.9% patients developed pedal edema, of those in whom oliguria was seen in 9.5% of the patients. Other less frequent symptoms observed are weakness 11.9%, seizures 9.5%. Hypertension had developed in 11.9% of the patients, hypotension was seen in 4.7% of patients.

Table 2: Average time of development of symptoms and signs since the consumption of the dye

Symptom	Time of development in hours		
	Mean	SD	Range
Burning Pain	48.33(min)	22.99(min)	30 – 120 (min)
Vomiting	27.87(min)	19.77(min)	10 – 90 (min)
Angioedema	3.71	1.01	2 – 4
Dysphagia	4.2	0.80	3 – 6
Dyspnea	4.13	0.76	4 – 6
Myalgia	9.44	3.30	6 – 16
Dark Urine	11.97	3.28	7 – 18
Oliguria	132	79.60	48 – 240
Seizures	192	58.79	120 – 240
Weakness	210	36	168 – 240
Oro-Facial Edema	4.02	0.88	2 – 7
Pedal Edema	192	29.39	168 – 240
Hypertension	67.2	10.73	48 – 72

Burning pain in mouth/throat/abdomen, vomiting were the two symptoms which developed within a mean duration of less than one hour. The mean duration of development of angioedema, dysphagia and/ or dyspnea, dark colored urine, myalgias was within 24 hours (less than a day). Pedal edema with or without anasarca and seizures and weakness appeared within a mean duration of 7 - 10 days. Oliguria appeared within a mean duration of 5 – 6 days. Hypertension was seen within mean duration of 4 – 5 days, hypotension with a mean duration of 6 hrs.

Table 3: Symptoms and signs in patients by gender

Symptom	Males(%) N=10	Females(%) N=32	Statistical Significance
Burning Pain	10(100)	30(93.75)	P = 1.0; Ns
Vomiting	8(80)	28(87.5)	P = 0.61; Ns
Angioedema	8(80)	27(84.37)	P = 1.0; Ns
Dysphagia	9(90)	5(15.67)	P = 0.00003; S
Dyspnea	7(70)	24(75)	P = 1.0; Ns
Myalgia	7(70)	25(78.12)	P = 0.67; Ns
Dark Urine	8(80)	26(81.25)	P = 1.0; Ns
Oliguria	0	4(12.5)	P = 0.55; Ns
Seizures	1(10)	3(9.37)	P = 1.0; Ns
Weakness	1(10)	3(9.37)	P = 1.0; Ns
Oro-Facial Edema	8(80)	28(87.5)	P = 0.61; Ns
Pedal Edema	1(10)	4(12.5)	P = 1.0; Ns
Oral Erythema	4(40)	6(18.75)	P = 0.21; Ns
Hypertension	1(10)	4(12.5)	P = 1.0; Ns
Hypotension	0	2(6.25)	P = 1.0; Ns
Stridor	1(10)	11(34.37)	P = 0.23; Ns
Wheeze	4(40)	15(46.87)	P = 0.73; Ns
Epigastric Tenderness	5(50)	17(53.12)	P = 1.0; Ns

Significant correlation between gender and symptom was found only for development of dysphagia which was seen in 90% of males and 15.67% of females with a p-value of 0.00003.

Table 4: Findings in important body systems

System	Findings	Number of subjects	Percentage
Respiraory System	Normal	10	23.80
	Stridor	12	28.57
	Wheeze	19	45.24
	Crackles	4	9.52
Cardiovascular System	Normal	42	100
	Murmurs	-	
	Others	-	
Gastrointestinal System	Normal	10	23.80
	Oral Erythema/Erosions	12	28.57
	Epigastric Tenderness	22	52.38

Central Nervous System	Normal	38	90.47
	Weakness	4	9.52
	Dtr - Areflexia	4	9.52
	Decreased Fine Touch	2	4.76

Respiratory system abnormalities that included stridor / wheeze/ crepitations were seen in nearly 83% of patients. Central nervous system examination revealed diminished DTRs in 10% of patients. All these patients had consumed dye > 50 ml and associated with creatinine phosphokinase levels of more than 50,000 U.Oral erythema / erosions were seen in 28% of the patients, and Epigastric tenderness was present in 52% of patients on GIT examination.

Table-5: Routine investigative findings

Routine investigations	Mean	SD	Range
Total Count	11652.38	3269.01	7200 - 24000
Polymorphs(%)	73.39	9.23	56 - 92
Lymphocytes(%)	22.02	8.40	4 - 40
Eosinophils(%)	4.26	1.79	1 -6
Monocytes(%)	1.74	0.71	0 - 3
Blood Urea	55.57	40.73	20 - 150
Serum Creatinine	1.60	1.38	0.6 – 6.2
Serum Bilirubin	0.97	0.50	0.6 – 3.3
SGOT	67.38	51.77	28 – 234
SGPT	73.26	46.16	17 – 224
CPK	18182.14	24375.55	108 - 74880
Sodium	135.97	5.32	124 – 147
Potassium	4.63	0.63	3.2 – 6.2
Calcium	6.36	1.95	3 - 9

Mean values of SGOT and SGPT had considerable increase in their levels. Raised mean value of Serum Creatinine phosphokinase was seen. Decrease in mean serum Calcium levels was seen.

Table-6: Investigative Abnormalities in present study

Investigative Abnormality		No. Of Patients	Percentage
Raised Total Count (>11,000/cu mm)		17	40.47
Raised Blood Urea (>40mg %)		14	33.33
Raised Serum Creatinine (>1.2mg%)		11	26.19
Raised Serum Bilirubin (>2.0mg%)		4	9.52
Raised SGOT (>40)		24	57.14
Raised SGPT (>40)		25	59.52
Raised CPK (>300)		36	85.71
Raised Potassium (>5.5meq/l)		1	23.80
Serum Calcium (<8mg/l)		26	61.90
ABG			
Normal		13	30.95
Met Acidosis		19	45.24
Respiratory Alkalosis		10	23.80
CUE on microscopy			
Nil		32	76.19
Pigment Casts		10	23.80
Level			
Normal		12	28.57
+		13	30.95
++		9	21.43
+++		8	19.04
Chest X - Ray	Normal	34	80.95
	B/L Pulmonary Edema	1	2.3
	Pleural Effusion	2	4.76
	Consolidation	2	4.76
ECG	Normal	30	71.42
	Pathological Changes	12	28.57
ENMG	Normal	38	90.47
	Motor – Sensory Neuropathy	4	9.52

ABG analysis was done in every patient at presentation, finding suggestive of metabolic acidosis was seen in 45% of patients. Respiratory alkalosis was seen in 24% of patients.

Highest grade of protein urine were seen to be 3+ grade 19% of patients, 2+ in 21% of the patients and 1+ in 31% of patients. 29% of others had only traces or no protein in urine. On microscopic examination, pigmented casts was found in 24% of patients.

Chest x ray showed features of pulmonary edema in 2%, pleural effusion and consolidation in 4% of patients. ECG abnormalities were seen in 28% of patients, mostly the abnormailities were sinus tachycardia, non specific T wave inversions, QT_c prolongation, and sinus bradycardia in one patient were observed.

ENMG was done in patients with weakness and areflexic paraparesis / Quadriparesis showed Demyelinating and axonal motor – sensory neuropathy.

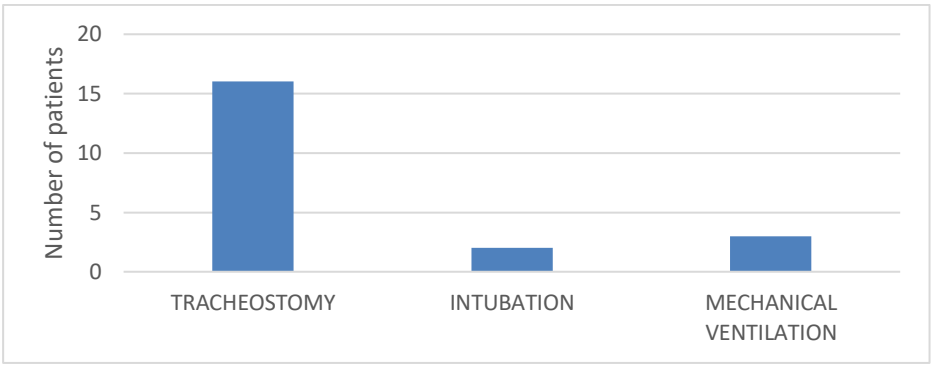

Fig 1: Interventions among patients

About 38% (16 patients) required tracheostomy, while 2 patients were managed with Endotracheal intubation. Mechanical ventilation was required in 7% of patients with modes of ventilation modified as per requirement. Other patients were managed with 'T- piece' with oxygen inhalation, and continuous $SpAO_2$ monitoring.

Table-7: Complications among patients

Complication	No. Of Patients	Percentage
Angioedema	36	85.7
Rhabdomyolysis (CPK>1500IU/L)	34	80.9
Acute Renal Failure	8	19.0
Ventilator associated Pneumonia, ARDS	2	4.76
Hypocalcemia	26	61.9

Majority of patients 85% of them developed angioedema, though of varying severity. The next most frequent complication observed was Rhabdomyolysis (CPK >1500 IU/L) which was seen in 80.9% of patients. 19% of total patients had developed Acute Kidney injury requiring hemodialysis. Hypocalcaemia was observed in 62% of patients, of whom 2 patients developed carpopedal spasms which responded to calcium administration.

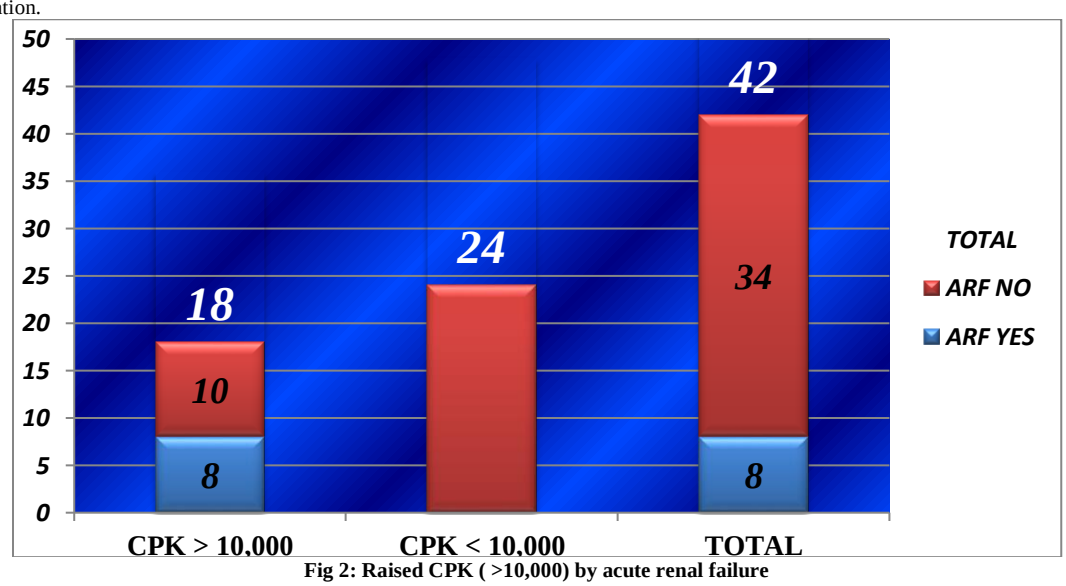

Fig 2: Raised CPK (>10,000) by acute renal failure

Chi square Phi coefficient: +0.56

Fischers Exact Probability Test P two – tailed = 0.0003; statistically significant.

28.5% of patients with CPK levels more than 10,000 IU/L developed ARF. ARF was not seen in patients with CPK levels less than 10,000 IU/L. This difference was statistically highly significant.

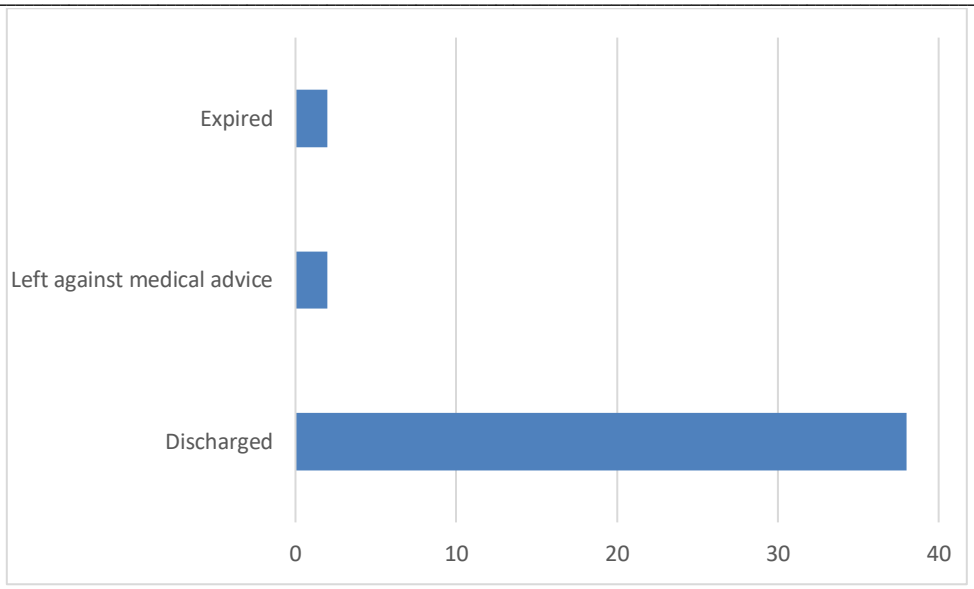

Fig 3: Outcome among patients

About 90% of patients were discharged from hospital in good general health condition after a mean duration of hospital stay of nearly 10 days (ranging from 4 – 21 days). 4.7% of patients left against medical advice. 2 (4.7%) patients expired due to respiratory complications with pneumonia, ARDS, sepsis and ARF.

Discussion

Poisoning by hair dye ingestion was predominant in the African & the Middle East countries. One study mentioning 150 cases in 10 years duration was reported from Khartoum, Sudan, between 1983 _ 93[4]. Another study mentioning 374 cases during 1992 – 2002 was reported from Morrocco[4]. Jain et al study mentioning 1020 cases in Uttar Pradesh done during a period of 5 years[5]. Poisoning by ingestion of hair dye had been increasing in this part of state for the past few years. There were 42 cases admitted into our hospital over the past two years. Yadavendra Reddy et al reported 225 cases during 2006 – 2008 from RIMS, Kadappa of Andhra Pradesh[6]. There were 30 cases reported by them in 2006, 45 cases in 2007 and 150 cases up to May of 2008. This surge may be attributed to the oral and media propaganda within the public and its cheap and easily availability in almost all local general stores.

The present study includes 42 cases of Paraphenylene diamine containing hair dye poisoning. Almost all the patients in the study 39 cases consumed the hair dye available in the market with the trade name "Super Vasmol 33", another dye "Godrej hair Dye" was consumed by 2 patients, and "Indica Hair Dye" by 1 patient. Our study has female preponderance with 76% of patients being females. The studies of Ayoub Filali et al[7], Suliman SM et al[4], Yadavendra et al[6] and Jain et al[5] also showed a female preponderance with 77%, 80%, 65% and 74.86% respectively. The mean age of the patients was 24 ± 6 (ranges 18 – 45). Manisha sahay et al[8] had patients with a mean age of 27 ± 5 years in their study. In the study of Kallel et al[9] 27.9 ± 16.8 years was the mean (range 18 – 40). The prevalence was noted to be more in the age group of 15 – 30 years in our study (90%) with almost 90% of male patients (9 of 10) and 90% of female patients (29 of 33) falling in this age group. The study of Ayoub Filali et al[7] also mentioned preponderance in this age group accounting for 70% cases . The study of Yadavendra Reddy et al[6] also observed the same. In Jain et al study 15 – 25 years age group had 44.21% and 25 – 35% had 40.20% preponderance.

The mean amount of dye consumed was found to be 52.31 ± 29.56 ml in our study (range 10 – 100 ml). In the study by Manisha Sahay et al[8] , the mean amount dye consumed was reported to be 79.5 ± 22.45 ml (range 50 – 100ml).On ingestion of dye, almost majority of the patients (95%) had developed burning pain in mouth, throat and abdomen within a mean duration of 26 ± 20 minutes ranging from (10 – 90 minutes). Vomiting occurred in 90% of the patient within mean duration of 42 ±27 minutes (30 – 120 minutes). Suliman SM et al[4]found such G.I.T symptoms in 40% of patients in their study[3]. Such symptoms were also described by Sumeet Singla et al[10] and Sharma A et al[11] in their case reports.

Angioedema developed in 85% of the patients with varying severity in our study with a mean duration of 3.7 ± 1 hours (range 3 – 6 hours) . It was described in 60% of patients by Ram et al[12]. Kallel et al[9] observed cervico – facial edema in 79% of their patients. Manisha Sahay et al[8] reported that 20% of patients had cervico – facial edema. In the study of Yadavendra Reddy et al[6], angioedema was found in 70% of patients. They also described that it was the earliest sign in such cases. Suliman et al[4] described it in 100% of their patients. Almost all case reports consistently described this feature. Jain et al[5] found angioedema in 73.03% of their patients. As complications of the angioedema, Dysphagia was observed in 85% of patients within a mean duration of 4 ±1 hours (range 4 – 6 hours), stridor was seen in 28.5% of patients with a mean duration of 6±2 hours. Kallel et al[9] described the symptoms of the upper airway tract edema in 68% of patients. Yadavendra Reddy et al[6] described laryngeal edema features in 35% of their patients. Dysphagia was seen in 71.17% of patients in Jain et al[5].

Those who presented with severe angioneurotic edema with stridor underwent emergency tracheostomy. Tracheostomy was done in 16(38%) of patients, 2 patients (4%) required Endotracheal intubation, 3(7%) patients required mechanical ventilation for respiratory support. Yadavendra Reddy et al[6] described tracheostomy done in 13% of their patients[5]. In the study of Manisha Sahay et al[8] tracheostomy was done in 20% of patients. 10% of their patients required mechanical ventilation. Kallel et al[9] have described 84% of their patients requiring respiratory support with orotracheal intubation / tracheostomy with mechanical ventilation. Ram et al[12] reported that 40% of their patients underwent tracheostomy and 20% required mechanical ventilation[6]. Sachin sony et al[13] described that 80% of their patients required emergency airway and 70% required ventilation.

Black colored urine was observed in 80% of our study population, with majority of these patients developing generalized muscle pains. It was observed within a mean duration of 12 ± 3 hours (range 7 – 18 hours). Ram et al described this feature in 100% of their patients[6]. Suliman et al[4] also observed that the first voided urine after ingestion of the dye was black in color in all 100% of their patients

that later got less intensified[3]. Kallel et al[9] described this feature in 73.3% of their patients. High colored urine was observed in 10% of patients by Manisha Sahay et al[8] who presented to them at the stage of ARF. Dark urine / chocolate brown urine was seen 53.82% of patients in Jain et al[5] study Generalized myalgias were complained by 76% of the patients. Myalgias were complained after a mean duration of 9.4 ± 3.3 hours(range 6 – 16 hours). In the study of Ram et al[12], muscle pains were complained by 70% and weakness by 10% of their patients. Yadavendra Reddy et al[6] noted severe myalgias in 35% of their patients[5]. 47.05% of patients had myalgias in Jain et al[5] study.

12% of patients developed pedal edema with / without anasarca and 9.5% of patients developed Oliguria in our study. It took a mean duration of 192 ± 29.3 hours (range 168 – 240 hours) for pedal edema and mean duration of 132 ± 79 hours (range 48 – 240) hours for Oliguria in our study. Oliguria was observed in 60% of patients by Sachin Sony et al[13]. 9% of patients developed these features in the study of Yadavendra Reddy et al[6]. Ram et al[12] noted these features in 70% of the patients[6]. Oliguria was seen in 37% of patients in the Kallel et al[9] study. The study by Manisha Sahay et al constituted all the patients with Oliguria and fluid overload[8]. Oliguria was seen in 12.54% in Jain et al series[5].

Hypertension was noted in 11.9% of patients in our study after a mean duration of 67.2 ± 10.7 hours (range 48 – 72hours). Hypertension was observed in 33.33% of patients by Manisha Sahay et al[8].4.7% patients presented with shock in our study. Kallel et al[9] reported shock in 26.3% of their patients. Sachin Sony et al[13] reported 30% frequency of shock in their study. Hypotension was observed in 14.61% of patients in Jain et al series[5].

Other less frequent symptoms and signs observed in our study were seizures seen in 10% of the patients. Encephalopathy and seizures were noted in 30% of patients in the study by Manisha Sahay et al[8]. Convulsions were also described in a case reported by Ravi varma et al[14], Trismus with carpopedal spasm with positive Chovstek's sign was described in a case report by Bhargava et al[15]. One patient in our study had carpopedal spasm. Dermatitis and itching was noted in 6.6% of patients by Suliman et al[4]. But no person in our study population had features of dermatitis. Seizures were seen in 2.25% of patients in Jain et al study[5]. Clinical examination revealed respiratory system abnormalities in 83% of the patients that included stridor, wheeze and crackles. Epigastric tenderness was observed in 52% of patients. Weakness with diminished deep tendon reflexes were found in 10% of patients. Sensory abnormalities were found in 4.57% of patients.

In our study, blood investigations revealed increased levels of SGPT in 59.52% of patients, SGOT in 57.14% of patients, Blood Urea in 33.33%, Serum Creatinine in 26.19%, total WBC count in 40.47% of the investigated patients. Hypocalcaemia was noted in 61.90% of patients. Almost, 83.33% of the investigated patients had increased Serum Creatinine phosphokinase levels, and 42.8% of them had levels more than 10,000IU/L. Significant increase in mean values were noted in CPK levels of the study population. ABG analysis revealed metabolic acidosis in 45% of the investigated patients and respiratory alkalosis in 24% of the investigated patients. Kallel et al[9] noted increased CPK level in 100% of patients (with a mean 77,762). Metabolic acidosis was reported in 100% of patients.

In the study of Manisha Sahay et al[8], rise in CPK was noted in 20% of the patients and rise in SGPT was observed in 10% of patients. Of the 30 ARF patients they studied, a mean blood urea level of 166.46 ± 62 mg/dl, a mean Creatinine level of 8.57 ± 1.85 mg/dl was noted. In the study of Jain et al[5], SGOT/SGPT was elevated in 67.16% of cases, 38.43% of patients showed Hypocalcaemia, 25.58% had an increase in Serum Creatinine and blood urea levels, 51.08% of patients showed raised CPK levels. In our study, proteinuria was seen in 71% of the patients. Microscopic examination of urine revealed pigmented casts in 23.80% of the patients. Proteinuria was observed in 36.86% of patients by Jain et al[5].

Though arrhythmias and cardiac abnormalities were reported in few case reports, only transient ECG changes that were sinus tachycardia, non specific T wave inversions, QT_c prolongation, and sinus bradycardia in one patient were observed in our study. Regarding complications, 85% of patients developed varying severity of angioedema. Rhabdomyolysis was noted in 80.9% of patients. Ram et al noted Rhabdomyolysis in 100% of their patients[12]. It was observed in 35% of patients by Yadavendra reddy et al[6]. Sachin Soni et al[13] observed it in 60% of patients. Kallel et al[9] noted this feature in 100% of patients. Manisha Sahay et al[8] reported this feature in 20% of their patients.

In our study, Acute Renal Failure (ARF) had developed in 19% of our patients requiring renal replacement therapy. In the study Kallel et al[9], the frequency of ARF was 47.4%, with dialysis requirement in 26.3%. Sachin Soni et al[13] reported ARF in 80% of their study group with dialysis requirement in 70% . Suliman et al[4] reported the frequency of ARF in their study group was 80% and dialysis requirement was 60%. In the study of Yadavendra Reddy et al[6], ARF developed in 15% of patients and dialysis requirement was in 9% of them. Ram et al[12] reported 70% of ARF in their study group and dialysis as required by all 70%. Dialysis was required by 8.62% of cases in Jain et al series[5].

In our study a significant statistical correlation between rhabdomyolysis and ARF, CPK levels of more than 10,000 IU/L were found. Mortality was 4.7% in our study population. 2 patients who expired had undergone tracheostomy, requiring ventilator support; developed Ventilator associated pneumonia and needed renal replacement therapy. Ram et al[12] had 10% mortality in their study. Mortality in the study of Yadavendra Reddy et al[6] accounted for 10.5%[5]. There was a mortality of 60% in the study of Sachin Soni et al[13]. Mortality of 31.6% was reported by Kallel et al[9]. Manisha Sahay et al[8] reported 26.6% mortality in their study group. Mortality of 22.48% was observed by Jain et al[5].

Table-8: Comparison with other studies

	Kallel et al[9] Study	Suliman et al[4] study	Manisha Sahay[8] Study(ARF)	Yadavendra Reddy et al[6] study	Ram et al[12] Study	JAIN et al[5]	Our Study
Study Population	19	150	30	225	10	1020	42
Age (Mean ± SD) in years	27.9 ± 16.8	40 ± 6.89	26.9 ± 4.95	26.8 ± 7.34	23.2 ± 7.6	-	24.6 ±6.2
Female : Male	11:8	4:1	13:7	4:1	3:2	3:1	3.2:1
SYMPTOMS AND SIGNS							
G.I.T Symptoms	60%	40%	-	-	-	71.17%	95%
Cervico – Facial Edema	79%	100%	20%	70%	60%	73.03%	85%
Stridor/wheeze/Crackles	68%	-	-	35%	-	22.45%	83%
Dark Urine	73.3%	100%	10%	80%	100%	53.82%	80%
Myalgia	60%	10%	-	35%	70%	47.05%	76%
Oliguria	37%	60%	100%	9%	70%	12.75%	9.5%
Hemodynamic Shock	26.3%	30%	-	-	-	14.61%	4.7%

INVESTIGATION							
High CPK levels	100%	--	20%	-	-	51.08%	83.33%
High SGPT levels	-	-	10%	-	-	67.16%	59.5%
Metabolic Acidosis	100%	-	80%	-	-	-	45%
COMPLICATIONS							
Angioedema	79%	100%	20%	70%	60%	73.03%	85%
Rhabdomyolysis	100%	68%	20%	35%	100%	51.08%	83%
Acute Renal Failure	47.4%	80%	100%	15%	70%	8.62%	19%
OUTCOME							
Mortality	31.6%	-	26.6%	10.5%	10%	22.48%	4.7%

Conclusion

PPD containing hair dye has emerged as a potential suicidal poison. The clinical profile of hair dye poisoning is marked by respiratory, muscular, hemodynamic and renal syndromes. Severe angioneurotic edema and acute kidney injury occurrences testifies to the severity of intoxication and predicts morbidity and mortality. There is no specific antidote for hair dye poisoning. The most important aspect of management is prompt initiation of supportive measures. Antihistamines, steroids and maintenance of good hydration form the mainstay of therapy. Depending on complications, measures like inotrope support, emergency airway management, alkaline diuresis and renal replacement therapy are warranted. As the burden of handling hair dye cases has been increasing in this part of the state also, primary care physicians, intensive care physicians and nephrologists 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

1. Sampath Kumar K, Sooraj YS. Hair dye poisoning and the developing world. J Emerg Trauma Shock 2009; 2(2);129-31
2. Motaouakkil S, Charra B, Hachimi A, Ezzouine H, Guedari H, Mejmi H et al. Rhabdomyolysis and PPD poisoning. Ann Fr Anaesth Reanim 2006;25:708-13.
3. Bourquia a, Jabrane AJ, Ramdani B, Zaid D. [Systemic toxicity of Paraphenylene diamine : 4 cases]Presses Med 1988; 17(35): 1798 – 800.
4. Suliman SM, Fadlalla M, Nasr ME, Beliela MH, Fesseha S, Babiker M et al. Poisoning with hair dye containing Paraphenylene diamine: Ten years experience. Saudi J Kidney Dis Transpl 1995;6;286-9
5. PK Jain, N Agarwal, P Kumar, NS Sengar, Nutan Agarwal, Asif Akhtar. Hair Dye poisoning in Bundelkhand region(Prospective analysis of hair dye poisoning cases presented in Department of Medicine, MLB Medical College, Jhansi). JAPI 2011 July; 59:415 – 19
6. Yadavendra reddy KB, Chandrababu, Venkata subbiah. A study of vasmol poisoning. Abstract of free paper presentation in proceedings of annual conference of association of physicians of India from Jan 29, 2009 to Feb 1, 2009 at Greater Noida, India.
7. Filali A, Semlali I, Ottaviano V, Soulyamani R, Furnari c, Corradini D. A retrospective study of acute systemic poisoning of paraphenylene diamine (occidental takawt) in Morocco. Afr J Trad CAM 2006;3:142-9.
8. Manisha Sahay, Vani R, Valli S. Hair Dye Ingestion – An uncommon cause of Acute Kidney Injury. J Assoc Physicians India 2009; 57:35-8
9. Kallel H, Chelly H. Dammak H, Bahloul M, Ksibi H, Hamida CB. Clinical manifestations of systemic PPD intoxication. J Nephrol 2005; 18(3):308-11
10. Sumeet Singla, Sanjeev M, Lal AK, Pulin Gupta, Agarwal AK. Paraphenylene diamine poisoning. J Ind Ac Of Clin Med 2005; 6(3):236 – 8
11. Sharma A, Mahi S, Sharma N, Suryanarayana BS, Bhalla A, Suri V et al. Intravascular hemolysis and acute renal failure following hair dye poisoning. In the proceedings of the 7th International Congress of the Asia-Pacific Association of Medical Toxicology at Chandigarh, India.
12. Ram R, Swarnalathe G, Prasad N, Dakshina Murthy KV. Paraphenylene diamine ingestion : An uncommon cause of acute renal failure. J of POST Grad Med 2007; 53:181-2
13. Sachin S, Nagarik AP, Manjunath D, Gopal Krishnan A, Anuradha. Systemic toxicity of paraphenylene diamine. Indian J Med Sci 2009; 63(4): 164-6
14. Ravi Verma, Nidhi Tiwari, Sushil Jaiswal, Virendra R, Dinesh K Singh< Arun Tiwari. Fatal Poisoning caused by oral ingestion of a hair dye. The Internet Journal Of Emergency and Intensive care Medicine 2008: 11(1):9
15. Bhargava P, Matthew P. Hair dye poisoning. J Assoc Physicians India, 2007; ;871-2

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