Original Research Paper

Volume-8 | Issue-12 | December-2018 | PRINT ISSN No 2249-555X

Medicine

CLINICAL FEATURES AND COMPLICATION FOLLOWING PARAPHENYLENEDIAMINE POISONING: A PROSPECTIVE CLINICAL STUDY

Manish Dudani — Ex Resident, Gajara Raja Medical College, Gwalior, Madhya Pradesh, India

OP Jatav — Professor & Head Department of Medicine, Gajara Raja Medical College, Gwalior, Madhya Pradesh, India

Jitendra Agrawal* — Associate Professor Department of Anaesthesiology, Gajara Raja Medical College, Gwalior, Madhya Pradesh, India *Corresponding Author

ABSTRACT

BACKGROUND: Poisoning is the main cause of High suicide rate in India and poisoning by hair dye is commonly seen, and in few places, it comprises a huge part of the poisoning cases.

Objective: To study the clinical features, electrocardiographic (ECG) changes, complications and outcome in patients presenting with hair dye poisoning.

MATERIAL AND METHODS: This prospective study comprises of 50 indoor patients admitted to poison ward of Department of Medicine, GR Medical College and JA Group of Hospitals, Gwalior, Madhya Pradesh, India. The parameters studied in all the patients were cause of ingestion, compound involved, time elapse between ingestion and admission to the hospital, duration of hospital stay, need for assisted ventilation, and cardiac manifestations at the time of presentation and during hospital stay. Routine investigations such as renal function, liver function tests,along with creatinine phosphokinase (CPK) levels, and cardiac marker test (CPK-MB) were done. ECG was done for all the patients daily to observe rate, rhythm, ST/T abnormalities, conduction defects, and P-R and Q-T intervals disturbances.

RESULT: Out of 50 patients included in the study, 38 (76%) patients were between the age group of 15–25 years, and there was a female predominance (60%). Twenty-eight (56%) patients reached hospital within 6 h of hair dye ingestion. Angioneurotic edema was the most common clinical finding. Renal functions were deranged in 38% and liver functions in 18% and CPK-MB was raised in 82% patients. ECG changes were observed in 90% patients of which ST/T changes were most common. Ten (20%) patients died in this study.

CONCLUSION: PPD poisoning was more common in females (60%) for suicide and is associated with effects on vital organs resulting in derangements of renal, hepatic and cardiac functions. ECG changes and elevated CPK-MB are found in majority of the patients with an overall mortality rate of 20%.

KEYWORDS : Hair dye, paraphenylene-diamine, poisoning, mortality.

INTRODUCTION

Suicide is a mental health concern, associated with psychological factors such as the difficulty of coping with depression, inescapable suffering or fear, or other mental disorders and pressures. For suicide, poisoning is thought to be a desired method and it is a chief problem faced in emergency department of any hospital.[1]Hair dye consumption is not an uncommon means of suicide or intentional self-harm. It has been reported around the world, more so in the underdeveloped and developing countries.

Paraphenylenediamine (PPD) is an aromatic amine, alanine derivative locally known as 'kala pathar' (black stone). It is solid and white in physical appearance but on oxidation quickly changes to a black color. PPD has been used in industry and cosmetics, for dyeing furs, photochemical measurements, accelerating vulcanization and azo-dye manufacturing however, its main use is in hair dyes and in combination with henna.[2,3]

PPD is mostly found in "kala pathar." For making hair dye, it is crushed and mixed with henna to improve its color.[1]It is a good donor of hydrogen molecules and metabolized by cytochrome P450 peroxidase to an active radical, forming a reactive compound called benzoquinone diamine. Clinical reports have shown that contact with PPD leads to irritation of skin, dermatitis, arthritis, asthma, conjunctivitis, chemosis, exophthalmos, lacrimation, and even permanent blindness. Toxicity mainly occurs by oral ingestion or percutaneous exposure.[4] Myocarditis[9] owing to hair dye poisoning is fatal and sometimes neglected complication. Oral ingestion of PPD in doses exceeding 10 g can result in extensive myocardial damage, which can lead to reduction in blood pressure, lethal life-threatening arrhythmias such as ventricular tachycardia (VT), or ventricular fibrillation. Echocardiography (ECG) shows different changes in the form of sinus tachycardia, bundle branch blocks, intraven-tricular conduction defect, atrial and ventricular premature complexes, atrial fibrillation, ventricular tachyarrhythmia, ST segment elevation or depression, and T wave inversion.[5]Cardiac biomarkers such as troponin T/I (>0.1 ng/mL), and creatinine phosphokinase isoenzyme-MB fraction (CPK-MB) are elevated in many cases. It can cause rhabdomyolysis and acute kidney injury, flaccid paralysis, severe gastrointestinal manifestations.[10]

MATERIALS AND METHODS:

In this prospective study 50 patients admitted with history of hair dye (PPD) poisoning who were admitted in Poison Ward in Department of Medicine at Gajra Raja Medical College and JA Group of Hospitals, Gwalior, Madhya Pradesh, India were included. Formal Ethical Committee approval was obtained from institutional committee. All these patients showed a history of alleged ingestion of hair dye containing PPD and were of different age groups. Parameters like demographic details, clinical features, management, and history of ingestion with reason, laboratory tests results and complication were noted.

A detailed history of amount and time of ingestion, complaint of breathlessness, and difficulty in swallowing, swelling over neck and lips, and reduction in urine output was taken. All the patients underwent routine investigations such as complete blood profile, random blood sugar, renal function tests; liver function tests (LFTs), serum electrolytes, CPK levels, and cardiac marker test (CPK-MB). These tests were repeated at regular intervals or as indicated. Other parameters such as cause of ingestion, time elapse between ingestion and admission to the hospital, duration of hospital stay, need for assisted ventilation, and cardiac manifestations at the time of presentation and during the hospital stay were recorded in the predesigned pro forma. Electrocardiogram of all the patients was done daily and repeated as required to observe rate, rhythm, ST/T abnormalities, conduction defects, and P-R and Q-T intervals measurements. All the patients were managed with symptomatic and supportive therapy including adequate hydration, inotropic support, steroids, endotracheal intubation, tracheostomy and renal replacement therapy, and ventilator support if needed.

RESULTS:

Of these 50 patients, 38 were between the age group of 15–25 years, and there was a female predominance (60%). Fifty-five of male subjects were from the rural area, and 93.33% female subjects belonged to urban area. Of 30 female hair dye poisoning patients, 27 (90%) were housewives and 3 (10%) students, while, of 20 male hair dye poisoning patients, 13 (65%) patients were in jobs and 7 (35%) students. Educational status of the patients showed that 27 (54%) were

educated up to intermediate pass, 11 (22%) to graduation, and 3 (6%) to postgraduation, and 9 (18%) were illiterate. It was also observed that 56% patients were admitted to hospital within 6 h of hair dye ingestion, 30% patients were admitted between 6 and 12 h, and 14% reached hospital after 12 h of poisoning. In clinical presentation, 21 patients showed angioneurotic edema, and 19 patients showed decreased urine output. Twelve patients underwent tracheostomy. The ECG changes were seen in 45 (90%) of patients [Tables 1,2and 3].

Of 19 patients with impaired renal functions, which was confirmed by blood urea and serum creatinine, 7 patients showed impaired RFT within 24 h, 8 patients in-between 24 and 48 h and 5 patients deranged renal parameters after 48 h. Blood urea and serum creatinine levels were significantly (**p** = 0.001) increased in hair dye poisoning patients. In patients who showed impaired hepatic profile (9), one patient showed deranged LFT between 24 and 48 h, 3 each in-between 48–72 h and 72–96 h, while 2 patients exhibited it after 96 h of admission. Of 50 patients, CPK-MB level was significantly high in 41 patients, and it was more marked in patients with hypotension. Of 50 patients of hair dye poisoning, 10 (20%) expired owing to different cardiac reasons [Table 3]. Tracheostomy was done in all these patients.

Table 1: Clinical Features of hair dye patients

S.No.	Clinical presentation	No. of patients
1.	Angioneurotic edema	21
2.	Decreased urine output	19
3.	Hypotension*	6
4.	Hypertension**	3
5.	Tachycardia ***	8
6.	Bradycardia****	1
7.	Icterus	9

Table 2: ECG findings of acute hair dye poisoning

Manifestation	Number (%)
Normal	5 (10)
ST-T change	21 (42)
Bundle branch block	4 (8)
AV Block	7 (14)
Bundle branch with AV block	2 (4)
Normal	5 (10)
ST-T change	21 (42)
Arterial fibrillation	3 (6)
Arterial flutter	2 (4)
Ventricular tachycardia	1 (2)
APC	1 (2)
EAT	1 (2)
Ventricular ectopic	1 (2)
Supraventricular tachycardia	1 (2)
VPC	1 (2)
Total	50 (100)

ST-T, ST and T wave; AV, atrioventricular; APC, atrial premature complexes; EAT, ectopic atrial tachycardia; VPC, ventricular premature complexes; ECG, electrocardiographic.

Table 2: Distribution of various ST-T changes

S. No	Name	No. of patients (%)
1	ST segment elevation	10 (47.61)
2	ST segment depression	3 (14.28)
3	ST segment elevation with T wave inversion	1 (4.76)
4	ST segment elevation with AV block and bundle branch block	2 (9.52)
5	ST segment depression with AV block and bundle branch block	1 (4.76)
6	ST segment elevation with ST segment depression	3 (14.28)
7	T wave inversion	1 (4.76)

AV, atrioventricular

Table 3: Various parameters in expired patients owing to hair dye poisoning

Case	Delay in bringing (h)	ARF	CPK-MB (0–25 U/L)	ECG findings
1	7	No	910	Atrial fibrillation
2	7	No	228	Ventricular tachycardia
3	3	No	878	ST elevation in the inferior leads
4	4	No	94	Sinus rhythm with normally conducted APCs (2nd, 5th, 9th, and 14th ORS complexes in the rhythm strip)
5	3.5	No	2868	ST elevation in leads II, III, aVF and leads V2–V6
6	3	No	22	Normal ECG
7	4	No	84	ST segment elevation in the anterior leads
8	13	No	84	ST segment elevation in leads I, II and AVL, V2–V6
9	8	No	92	ST segment elevation in leads II, III, aVF, and V3–V6
10	4	No	140	Atrial flutter

CPK-MB, creatinine phosphokinase isoenzyme-MB fraction; ARF, acute renal failure.

DISCUSSION:

PPD (C6H8N2) is the commonest and cheapest form of dye available in North Africa and the Middle East, known as stone dye, and contains the highest concentration of PPD (from 70 to 90%).[11,12] Other branded hair dyes contain lesser concentrations of PPD, typically from 2 to 10%.[13] The formation of oxide derivatives of PPD such as benzoquinone diimide is responsible for destruction of muscle cells by a mechanism of membrane lipid peroxidation which leads to muscle necrosis and also produces fatal effects on various organ by causing angio-neurotic edema, myocarditis and rhabdomyolysis. Due to its improper handling, easy availability and low cost, it becomes a common mode of self-poisoning in rural areas of India. Of 50 cases, 60% were female subjects of young age, who presented late to the hospital. Similar study done by Balasubramanian et al.[6]showed that hair dye poisoning is more common in female subjects and more in the age group of 20–30 years. Hair dye is known to cause angioneurotic edema, acute renal failure (ARF), and cardiotoxicity[5].This study confirmed these pres-entations. The angioneurotic edema was less when com-pared with that seen in the study by Hasim et al[7]; it may be because of the difference in amount of substance ingested or earlier presentation as 28 patients were hospitalized within 6 h of ingestion. Acute poisoning causes rhabdomyolysis and ARF resul-ting in death if not treated aggressively.[6] Renal functions showed derangement in our 38% of patients. In this study, hepatotoxicity was more when compared with a previous study.[4]Hair dye is a potent cardio toxin and its ingestion leads to myocardial damage. Owing to myocardial damage, there is release of cardiac specific markers such as Trop T, Trop I, and CPK-MB.[8] In this study, CPK-MB was found to be elevated in 82% patients. The ECG in myocarditis shows transient changes, which are usually nonspecific and occur in many other cardiac diseases; 90% of our patients showed one or other ECG changes varying from ST-T changes to VT. The mortality in our study was 20%, which is similar to other workers. Our study has very well documented the hepatotoxicity, nephrotoxicity, and cardiotoxicity of hair dye poisoning. However, the limitation of our study is the small subject population. Moreover, absence of specific antidote is also a matter of concern regarding its fatal outcomes.[14,15]

CONCLUSION:

The hair dye poisoning is becoming a common suicidal poisoning. Its effect on the vital organs is responsible for morbidity and mortality. The ECG manifestations in hair dye poisoning have been previously reported but not very frequently. In spite of best of the treatment in form of tracheostomy, endotracheal intubation with or without assisted ventilation, and dialysis for renal failure, mortality remains high in hair dye (PPD) poisoning.

REFERENCES:

1. Sakuntala P, Khan PM, Sudarsi B, Manohar S, Siddeswari R, Swaroop K. Clinical profile and complications of hair dye poisoning. Int J Sci Res Publ 2015;5(6):1–4.
2. Sumit Singla et al Para-phenylenediamine (PPD) Poisoning , JIACM 2005;6(3):236-8
3. Abdel MA. Acute Toxicity by Hair Dye in Upper Egypt. Int J Forensic Sci Pathol. 2017 Jan 10;5(1):305-11. [Free full text] doi: http://dx.doi.org/10.19070/2332-287X-1700069
4. Varadhraj PG, Sasikala M, Cheemalkonda R, Banarjee R, Padaki NR, Reddy N. Hairdye-induced hepatitis: an unusal cause of acute hepatitis. Saudi J Gastroenterol 2009;15(2):138–9.

5. Suganthakumar MN, Ganesh R. Hair dye—an emerging suicidal agent: our experience. Otolaryngol Online J 2012;2:1–11.
6. Balasubramanian D, Subramanian S, Shanmugam K. Clinical profile and mortality determinants in hair dye poisoning. Ann Nigerian Med 2014;8(2):82–6.
7. Hasim S, Hamza Y, Yahia B, Khogali F, Sulieman G. Poisoning from henna dye and para-phenylenediamine mixtures in children in Khartoum. Ann Trop Pediatr 1984;12:3–6.
8. Jain PK, Sharma AK, Agarwal N, Sengar NS, Siddiqui MZ, Kumar SA, et al. A prospective clinical study of myocarditis in cases of paraphenylenediamine (hair dye) poisoning in northern India. J Toxicol Environ Health A 2012;4(7):106–16.
9. Jedidi M, Hadj MB, Masmoudi T, Adelkarim SB, Mlayeh S, Dhiab MB, et al. Fatal toxic myocarditis induced by Paraphenylene Diamine. A case report. Rom J Leg Med. 2016 Mar 1;24:17-20.
10. Naqvi R, Akhtar F, Farooq U, Ashraf S, Rizvi SA. From diamonds to black stone; myth to reality: Acute kidney injury with paraphenylene diamine poisoning. Nephrology. 2015 Dec 1;20(12):887-91. [PubMed] doi: 10.1111/nep.12534
11. Khan MA, Akram S, Shah HB, Hamdani SA, Khan M. Epidemic of kala pathar (paraphenylene diamine) poisoning: an emerging threat in southern Punjab. J Coll Physicians Surg Pak. 2018 Jan;28(1):44-47. doi: 10.29271/jcpsp.2018.01.44. [PubMed]
12. Jain D, Mittal A. Hair Dye Poisoning: Case Report and Review of Literature. Iranian Journal of Toxicology. 2016 Oct 14; 10(6):51-53 [Free full text]
13. Patra AP, Shaha KK, Rayamane AP, Dash SK, Mohanty MK, Mohanty S. Paraphenylenediamine containing hair dye: an emerging household poisoning. Am J Forensic Med Pathol. 2015 Sep;36(3):167-71. doi: 10.1097/PAF.0000000000000165
14. Beshir L, Kaballo B, Young D. Attempted suicide by ingestion of hair dye containing p-phenylenediamine: a case report. Ann Clin Biochem. 2017 Jul;54(4):507-10. [PubMed] DOI: 10.1177/0004563216685117
15. Perumal S, Ayyavu S, Anandan H. Clinical Profile of Ingestional Hair Dye Poisoning: A Prospective Study. Int J Sci Stud. 2016 Aug 1;4(5):154-6. [Free full text] DOI: 10.17354/ijss/2016/450