ORIGINAL ARTICLE

Paraphenylene diamine poisoning: Our experience at PMC Hospital Nawabshah

Bashir Ahmed Khuhro*, Muhammad Saleh Khaskheli**, Abrar Ali Shaikh***

*Assistant Professor Medicine, **Assistant Professor Anesthesiology, ,SICU & Pain Management, ***Assistant Professor Community Medicine, Peoples University of Medical and Health Sciences (PUMHSW), Nawabshah (Pakistan)

Peoples University of Medical and Health Sciences (PUMHSW), Nawabshah (Pakistan)

Correspondence: Dr Bashir Ahmed Khuhro, Assistant Professor, Medical Unit-I, Peoples University Of Medical and Health Science(PUMHSW), Nawabshah (Pakistan); Email: drbashirkhuhro@gmail.com

ABSTRACT

Objective: The aim of this descriptive, case series study was to study demographics, clinical features and outcome of paraphenylene diamine (PPD) (commonly known by local people as 'kala pathar') poisoning admitted to our intensive care unit (ICU) from June 2009 and May 2012.

Methodology: All cases of PPD poisoning admitted to ICU of the Peoples Medical College Hospital, Nawabshah, between June 2009 and May 2012 were included in this study. Demographic features, clinical features and outcome of patients were recorded.

Results: A total of 16 poisoning cases were admitted to the ICU. The mean age was 25.87 ± 5.59 years; a majority of the patients were young females (21-30 years) and belonged to a low socioeconomic class. The main cause was intentional suicidal ingestion. Cervicofacial edema, throat pain, dysphagia, dysphonia, and stridor were the earliest clinical findings. Rhabdomyolysis, hepatitis and acute renal failure dominated the clinical picture during the later course of poisoning. Active pharmacological intervention, elective tracheostomy and assisted ventilation were the therapeutic measures required for survival. A high mortality rate (37.5%) was observed in the study.

Conclusion: Paraphenylene diamine (PPD) poisoning is associated with high morbidity & mortality.

Keywords: Paraphenylene diamine poisoning; Cervicofacial edema

Citation: Khuhro BA, Khaskheli MS, Shaikh AA. Paraphenylene diamine poisoning: Our experience at PMC Hospital Nawabshah. Anaesth Pain & Intensive Care 2012:16(3):243-246

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 Poisoning is a preferred method of suicide and is one of the major problems encountered in emergency departments of hospitals.² In the developed countries, an overdose of sedatives, hypnotics or narcotics is commonly employed to achieve it, whereas in developing countries agricultural pesticides are used.³⁻⁴

Poisoning with PPD is a new trend of intentional self harm in various developing countries of Asia and Africa,⁵ and is associated with high mortality.⁶ PPD is an active ingredient of 'Kala Pathar'. It is crushed and mixed with henna and used as hair dye or for enhancing the color of henna. Its use as a hair dye has been on an increase in our area. The compound PPD is highly toxic when taken orally and death occurred within the first 6-24 hours due to angioneurotic edema or cardiotoxicity which lead to fatal arrhythmias.⁷ Smaller doses, or if the patient vomits most of the dye, will usually present as angioneurotic edema and hepatitis. A moderate dose will cause acute renal failure within the first week.⁸ Despite the high mortality and frequency of cases, no antidote is available for this poisoning ⁸ and is managed conservatively.

The aim of the study was to share our experiences regarding this chemical and to document the clinical presentation, laboratory findings, and outcomes of hair dye poisoning at ICU of Peoples Medical College Hospital (PMCH) Nawabshah.

METHODOLOGY

This study was conducted at the 8-beded ICU of PMC Hospital Nawabshah, a tertiary care hospital in Sindh

province of Pakistan, between July 2009 and June 2012. During this period a total of 16 patients with hair dye poisoning were admitted in the ICU through emergency and medical departments. The ethics committee of said institute has approved the study protocol for publishing the results.

In this study, a pro forma was used to collect data including demographic features (age, sex, marital status, socio economic status), clinical features (especially cervicofacial edema and color of urine), laboratory findings (complete blood count, liver function test, CK, LDH, glucose, urea, creatinine, electrolytes and ECG), mode of intoxication (accidental or suicide) and route of intoxication (gastrointestinal system, skin). The diagnosis of PPD poisoning was based on clinical findings and information taken from the patient's family and friends. Toxicology screening or postmortem could not be preformed due to social restrictions. All patients received gastric lavage, antihistamines, parenteral steroids, sodium bicarbonate, dextrose and saline via IV. Forced diuresis was used to augment elimination of renally excreted toxins. Tracheostomies were preformed in 10 patients because laryngeal edema made intubation impossible. Synchronized intermittent mandatory ventilation and pressure support mode (pressure-controlled or volume-controlled) were started. The positive end expiratory pressure was initially applied as 5 cm H2O and then titrated to keep O, saturation above 94%. Weaning for mechanical ventilation was carried out with pressure support weaning and T-tube trials. Hospitalization time, morbidity and mortality rates were also recorded. Attendants were counseled and recovered patients were referred to psychiatry department for psychiatric assessment.

Statistical analyzes was done by using SPSS for windows release 15 (SPSS) software. Continuous data were presented as mean and standard deviation, whereas categorical data were presented in numbers and percentages.

RESULTS

Of the 16 patients admitted, 14(87.5%) were females and 2(12.5%) were males. The mean age was 25.87 ± 5.59 years and majority of the patients (68.8%) were 21-30 years of age. Suicidal intention was identified in 75% of the cases and 4(25%) were declared accidental. Social conflicts formed the basis of 80% of intoxications. All cases were from the rural areas. The poison was taken orally in 13(81.3%) cases and by transdermal route in 3(18.8%) cases. Demographic features are summarized in Table 1. Table 1: Demographic characteristics of the patients

| Parameter | Value* | |
|------------------------|---------------------|-------|
| Age (Mean <u>+</u> SD) | 25.87 <u>+</u> 5.59 | |
| Gender | | |
| Male | 02 | 12.5% |
| Female | 14 | 87.5% |
| Age (year) | | |
| 12-20 | 03 | 18.8% |
| 21-30 | 11 | 68.8% |
| 31-40 | 02 | 12.5% |
| Marital status | | |
| Single | 07 | 43.8% |
| Married | 09 | 56.3% |
| Economical status | | |
| High | 01 | 00% |
| Middle | 01 | 6.3% |
| Low | 15 | 93.8% |
| Mode of intoxication | | |
| Suicidal | 12 | 75% |
| Accidental | 04 | 25% |
| Mode of Transmission | | |
| Orally | 13 | 81.3% |
| Trans-dermal | 03 | 18.8% |

*Data expressed as N(%) unless specified

Table 2: Clinical Features and outcome of Kala Pathar poisoning

| Clinical Features | N (%) |
|--------------------------------|-----------|
| Pain in Throat | 16 (100) |
| Oral Erythema | 16 (100) |
| Cervicofacial Edema | 16 (100) |
| Dysphagia | 16 (100) |
| Dysphonia | 16 (100) |
| Difficulty in Opening of Mouth | 16(100) |
| Muscle Aches/Rigidity | 10 (62.5) |
| Dark urine | 13 (81.3) |
| Rhabdomyolysis | 09 (56.3) |
| Oliguria/Anuria | 05 (31.3) |
| Acute Renal Failure | 06 (37.5) |
| Hyperkalemia | 03 (18.8) |
| Hepatitis | 14 (87.5) |
| Hemodynamic shock | 03 (18.8) |
| Sinus bradycardia | 03 (18.8) |
| Sinus tachycardia | 13 (81.3) |
| Outcome | N (%) |
| Tracheostomy | 14 (87.5) |
| Ventilator | 12 (75) |
| ICU stay (days) | 6.43±3.61 |
| Mortality | 06 (37.5) |

The clinical features of hair dye poisoning (pain in throat, oral erythema, cervicofacial edema, dysphagia and dysphonia) were present in all patients (100%).

Evidence of rhabdomyolysis (muscle aches/tenderness, muscle edema, cola-colored urine, raised creatinine phosphokinase, myoglobinuria) was present in 56.3% of the cases. Hemodynamic shock, sinus bradycardia and T-tenting were detectable in 18.8% while sinus tachycardia was noted in 81% of the patients. Oliguria/anuria was reported in 5(31.3%), while acute renal failure was inferred in 37.5% of the cases. Stridor was observed in 8(50%) cases (Table 2). Classical features of poisoning such as cervicofacial edema, dark-colored urine, and hepatitis were observed within six hours of poison intake. Regarding laboratory investigation, the mean \pm SD of TLC, SGPT and CPK was 10375 \pm 4731.1, 851.19 \pm 1604 and 28.43 \pm 13.20 respectively [Table 3.

Table 3: Laboratory parameters

| Laboratory parameters | Mean <u>+</u> SD | Mode/Range |
|--------------------------|-------------------|-----------------|
| TLC (1000 cells/mm3) | 10375 ± 4731.1 | 6000/5000-20000 |
| CPK (U/Lin 1000) | 28.43 ± 13.20 | 24/1.00-60 |
| SGOT (U/L) | 1365.18 ± 1186.28 | 1500/119-5247 |
| SGPT (U/L) | 851.19±1604 | 100/20-6550 |
| Serum creatinine (mg/dL) | 1.98±2.97 | 1.00/0.50-13 |

During the hospital stay all patients received hydrocortisone, 14(87.5%) needed an emergency tracheotomy, 12(75%) patients required ventilator support for airway compromise and 2 patients (12.5%) developed acute renal failure (ARF) after 72 hours of poisoning. The mean ICU stay was 5.76 ± 3.05 days (1–20). 6 (37.5%) patients expired out of a total of 16.

DISCUSSION

PPD poisoning in the form of compound hair dye known as 'kala pathar' is emerging as a new trend in suicidal poisoning in our setting because of easy availability, low cost and salty taste rather than bitter. The constituents of 'kala pathar' include 4% PPD, resorcinol, propylene glycol, ethylenediaminetetraacetic acid (EDTA), sodium, liquid paraffin, cetostearyl alcohol, sodium lauryl sulphate, herbal extracts, preservatives, and perfumes.⁹ Some of these are known toxins with systemic effects, while the toxicity profile of others is not known. The toxic effects depend on the dosage.¹⁰

Like many earlier studies, the majority of the patients in our study were young females (25.87 ± 5.59 years). Akber MH⁶ and Anugrah Chrispal et al¹² identified similar age group with female predominance, 27.75 years and 20.5 ± 4.65 years respectively. Social conflicts may be the reason of poisoning in this age group. All of our patients belonged to rural area and low socioeconomic status and the purpose of ingestion of this compound was suicidal in majority of cases (70%). It is usually ingested to threaten the family members if the demands are not met.

Classical features of poisoning occurred within four to six hours of ingestion. It is very crucial to reach appropriate health care facility within this time period, during which most of the deaths occur. Cervicofacial edema was the first symptom to develop as observed in studies by Anugrah Chrispal et al¹² (69.2%) and Kallel et al (79%),¹¹ but its exact cause remains unclear.¹² Respiratory failure is the main threat to life; endotracheal intubation, tracheostomy and assisted ventilation are crucial and lifesaving measures. Suliman et al¹¹ observed a tracheostomy rate of 15.8% in his patients, a study at Multan⁶ showed this rate to be 60% while 87.5% of our patients required this procedure. This needs further explanation; the amount of poison ingested and the sample size may explain the difference. Coma/unconsciousness is another important feature of PPD poisoning which was observed in 6(37.5%) of our cases, while the figure was 20 and 26.3% in studies by Akber and Kallel et al.^{6,13} Hypovolemia, sepsis and myocarditis might be the underlying cause. We observed hyperkalemia in 12.5% patients, which has been identified as one of the factors predictive of mortality due to PPD poisoning.¹⁴ Hyperkalemia was noted to be 20% and 26.3% patients in the study by Kallel et al¹³ and in a local study respectively.6 Rhabdomyolysis and ARF may be cause of hyperkalemia. We observed 75% patients in our study had evidence for rhabdomyolysis, Kallel et al also noted rhabdomyolysis in all of patients in his study. ARF occurred in 37.5% of patients (47.4% by Kallel et al and 40 % by Akber et al).^{6, 13} We also found that the markers of hepatitis were significantly higher in our patients, 40% in local study 6 and 100% in international study. Consumption of small amount of PPD, as low as 25ml results in hepatitis.^{8, 10}

A study at Nellore India⁸ showed that the increase in morbidity, e.g. need of ventilatory support, duration of hospital stay and mortality was dependant on the dose taken by the patient but we didn't record the actual amount consumed by the patients due to lack of sources. Most of these patients expired so we assumed that they had taken large doses of the poison. We observed a high mortality rate (68.8%) as compared to other studies, 20% at Multan,⁶ 4% at India, 21% by Benslama et al.¹⁶ This difference might be due to late recognition and late arrival of patients in our hospital. Therefore, early recognition and early arrival can be life saving. As there is no specific antidote, the management of poisoning includes gastric lavage, antihistamines, parenteral steroids especially hydrocortisone, and alkalinization of the urine.⁸ Respiratory distress is the major early problem which may require ventilatory support. Renal support in the form of dialysis is required in acute renal failure,⁸ but our patients did not need dialysis because they were not fit for dialysis and expired early. could be a warning to the Asian countries and emerging as alternative to organophosphorus poisoning because of its easy availability, low cost and bitterness. We recommend public awareness regarding this toxin and sale of Kala Pathar should be legally restricted by government.

DISCLOSURE

None of the authors received any financial benefit from any source while conducting this study.

CONCLUSION

Paraphenylene diamine (PPD) (Kala Pathar) poisoning

REFERENCES

- WHO World Suicide Prevention Day 2008; WHO Statement; 2008. Available online:http://www.who.int/mental_health/prevention/suicide/wspd_2008_statement.pdf (accessed on 11 July 2012)
- Lee HL, Lin HJ, Yeh STY, Chi CH, Guo HR. Presentations of patient's of poisoning and predictor of poisoning -related fatality: findings from a hospital-based prospective study. BMC Public Health 2008;8:7.
- Michel K, Ballinari P, Bille-Brahe U, Bjerke T, Crepet P, De Leo D, et al. Methods used for Para suicide: results of the WHO/EURO Multicenter Study on Para suicide. Soc Psychiatry Psychiatr Epidemiol. 2000;35:156–63.
- Gunnell D, Ho D, Murray V. Medical management of deliberate drug overdose: a neglected area for suicide prevention? Emerg Med J 2004;21:35–8.
- Suliman SM, Homeida M, Aboval OI. Paraphenylene Diamine Induced Acute Tubular Necrosis Following Hair Dye Ingestion. Hu-

man Toxicol 1983;2:633-5.

6.

- Akbar MA, Khaliq SA, Malik NA, Shahzad A, Tarin SM, Chaudhary GM. Kala pathar (paraphenylene diamin) intoxication; a study at Nishtar hospital Multan. Vol 2, No 4• October December 2010
- Bowen DAL. A case of phenylenediamine poisoning. Medicine Sci Law 1963;3:216-9.
- Kondle R, Pathapati RM, Saginela SK, Malliboina S, Makineedi VP. Clinical Profile and Outcomes of Hair Dye Poisoning in a Teaching Hospital in Nellore. ISRN Emergency Medicine, vol. 2012, Article ID 624253, 5 pages, 2012. doi:10.5402/2012/624253
- Nohynek GJ, Fautz RF, Benech-Kieffer, Toutain H .Toxicity and human health risk of hair dyes. Food and Chemical Toxicology 2004;42(4):517–543.
- Hou FQ, Lin XH, Yu YY. Wang TL and Wang GQ. Severe liver injury induced by repeated use of hair dye. Chinese Medical Journal 2009;122 (7):875-77.

- Suliman SM, Fadlalla M, Naser Mel M, et al. Poisoning with Hair Dye Containing ParaphenyleneDiamine: Ten Years' Experience. Saudi J Kidney Dis Transpl 1995;6:286-9.
- Chrispal A, Begum A, Ramya, Zachariah A. Hair Dye Poisoning- an Emerging Problem in the Tropics: an Experience from a Tertiary Care Hospital in South India. Trop Doct 2010;40:100-3.
- Kallel H, Chelly H, Dammak H, Bahloul M, Ksibi H, Hamida CB, et al. Clinical Manifestations of Systemic ParaphenyleneDiamine Intoxication. J Nephrol. 2005;18:308.
- Soni S, Nagarik A, Anuradha GK. Supervasmol 33 Poisoning- Abstract Presented at 38th Annual Conference of Indian Society of Nephrology;2007
- Bensalama A, Moutaouakkil S, Mjahed K, El Moknia M, Lahbil D, Fadel H. Syndrome intermediairelorsd'une intoxication aigue par le malathion. Press Med. 1998;27:713-5.