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PARAPHENYLENEDIAMINE (KALA PATHAR) POISONING: A PROSPECTIVE STUDY ON PATIENT OUTCOMES WITH SUPPORTIVE CARE AT A TERTIARY CARE HOSPITAL IN KARACHI, PAKISTAN

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Abstract

Background: *Paraphenylenediamine (PPD), commonly known as Kala Pathar, is a widely available hair dye ingredient that has emerged as a major cause of intentional poisoning in South Asia.*

Objective: *To evaluate the clinical profile, complications, management strategies, and outcomes of patients presenting with confirmed Paraphenylenediamine poisoning and receiving supportive care at a tertiary care hospital.*

Methodology: *This prospective single-arm cohort study was conducted at the National Poisoning Control Centre (NPCC), Jinnah Postgraduate Medical Centre (JPMC), Karachi, from April 2024 to September 2024. A total of 100 consecutive patients with confirmed Kala Pathar ingestion were enrolled. Data regarding demographics, mode and route of exposure, clinical manifestations, biochemical parameters, hospital stay, ICU requirement, and outcomes were recorded.*

Results: *Out of 100 patients, 93% had suicidal intent, and 98% ingested the compound orally. Cervicofacial swelling (97%), dysphagia (85%), and dyspnea (71%) were the most common presenting features. Complications included rhabdomyolysis (62%), renal failure (28%), hepatitis (25%), and hyperkalemia (29%). The mean hospital stay was 4.0 ± 3.5 days among survivors versus 5.9 ± 3.5 days among those who expired. Overall mortality was 18%, with most deaths occurring within the first two weeks of admission. The Kaplan–Meier analysis showed a mean survival time of 10.5 days (95% CI: 9.2–11.7).*

Conclusion: *Kala Pathar poisoning remains a critical medical emergency characterized by rapid systemic deterioration. Early airway management, fluid resuscitation, and renal support markedly improve survival. Mortality is primarily associated with delayed presentation and severe multi-organ involvement.*

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Introduction

Suicide is a preventable public health problem, where more than 700,000 people die every year and for each suicidal death, there are more than 20 suicide attempts. Toxicity with hair dye containing paraphenylenediamine (PPD) is an emerging trend of suicidal poisoning, especially in developing African and Asian countries, and is associated with higher morbidity and mortality [1]. Poisoning continues to be a significant public health concern worldwide, particularly in developing countries where toxic agents are easily accessible and regulatory oversight is limited [2]. Among the various poisons used for self-harm, Paraphenylenediamine (PPD), commonly known as *Kala Pathar* or "black stone," has emerged as a disturbing trend in South Asia. Initially intended for cosmetic use as a hair dye or as an additive in henna, this compound has increasingly been misused as a readily available and lethal means of suicide [3]. Its rising incidence, particularly among young adults, highlights an urgent need for greater clinical awareness and preventive action. Paraphenylenediamine is an aromatic amine that appears as a white crystalline substance, which quickly turns black upon exposure to air a characteristic that inspired its local name [4]. When mixed with hydrogen peroxide and ammonia in commercial hair dyes, it undergoes oxidation to form reactive intermediates, which are highly toxic to human tissues. Its low cost, widespread availability, and lack of legal restrictions have made it an easily obtainable but dangerous substance in many regions of Pakistan, India, and Africa [5].

Ingestion of PPD produces a distinctive clinical picture that can be recognized within hours. The earliest and most striking symptom is cervicofacial swelling, involving the face, tongue, and neck, which often progresses rapidly to airway obstruction. This characteristic feature is considered almost pathognomonic for Kala Pathar poisoning [6]. Other major complications include rhabdomyolysis, myoglobinuria, and acute renal failure due to acute tubular necrosis. Systemic involvement may also manifest as toxic myocarditis, hepatic dysfunction, and severe metabolic disturbances. In the absence of a specific antidote, management remains largely supportive, focusing on airway stabilization, fluid resuscitation, renal protection, and correction of metabolic derangements [7]. Demographic trends across various hospital-based studies show that the majority of victims are young females aged between 15 and 30 years, often belonging to low-income families. The ingestion is typically impulsive, occurring in moments of psychological distress, family conflict, or domestic abuse [8]. The predominance among young women suggests underlying sociocultural vulnerabilities and lack of mental health support. In many rural areas, access to emergency medical care is limited, and the delay in presentation often proves fatal [9]. Mortality rates reported in hospital-based data remain alarmingly high, ranging from 30% to 60% depending on the timing of intervention and the quality of supportive management available. The management of Kala Pathar poisoning poses multiple challenges. Because there is no known antidote, treatment is aimed at preventing further absorption and minimizing systemic toxicity [10]. The first priority is airway protection; many patients require emergency tracheostomy due to severe edema. Aggressive hydration with intravenous fluids is essential to prevent renal injury, while urine alkalinization may help limit the nephrotoxic effects of myoglobin [11]. Hemodialysis becomes necessary when acute renal failure develops. Corticosteroids and antihistamines may be used to reduce swelling, while bicarbonate therapy can correct metabolic acidosis. However, despite all these interventions, outcomes remain unpredictable, and many patients suffer prolonged hospital stays and residual organ dysfunction [12]. Diagnosis is primarily clinical, supported by suggestive laboratory findings. Elevated creatine phosphokinase (CPK) and serum creatinine levels are consistent with rhabdomyolysis and renal injury [13]. Dark urine and metabolic acidosis are common laboratory features. However, because there is no specific test to confirm PPD toxicity, diagnosis depends heavily on patient history, the characteristic black staining of the oral cavity, and the presence of cervicofacial edema. Many patients are initially misdiagnosed as cases of allergic

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angioedema or anaphylaxis, which delays appropriate intervention and worsens outcomes [14].

Objective

Methodology

This was a prospective single-arm cohort study conducted at the National Poisoning Control Centre (NPCC), Jinnah Postgraduate Medical Centre (JPMC), Karachi, Pakistan from April 2024 to September 2024, following formal approval from the College of Physicians and Surgeons Pakistan (CPSP). All patients presenting to NPCC with confirmed Paraphenylenediamine (PPD) poisoning during the study period and fulfilling the inclusion criteria were enrolled. A total of 100 patients (n = 100) were included in the final analysis. Only cases with valid medico-legal registration numbers were considered eligible. A non-probability consecutive sampling method was used. Every eligible patient presenting during the study period was included until the required sample size was achieved.

Inclusion Criteria

- Both male and female patients.
- Age between 15 and 70 years.
- Confirmed diagnosis of Paraphenylenediamine (Kala Pathar) poisoning with a valid medico-legal case number.

Exclusion Criteria

- Cases with uncertain or mixed ingestion of multiple poisons.
- Patients whose guardians or attendants refused consent for participation.

Data Collection

After obtaining approval from CPSP and institutional permission, data were prospectively collected at NPCC using a predesigned pro forma. The primary independent variable was the mode and route of PPD exposure (oral or transdermal). The dependent variables included clinical presentation, response to supportive management, length of hospital stay, need for intensive care unit (ICU) admission, and mortality outcome. Detailed demographic data, ingestion history, time of presentation, clinical findings, laboratory investigations, and treatment measures were recorded for each participant. The data were initially entered on standardized paper forms, cross-checked for completeness and accuracy, and then entered into Microsoft Excel. The final statistical analysis was performed using IBM SPSS version 26. All participants or their guardians provided informed written consent prior to inclusion. Every admitted patient underwent medico-legal registration as per hospital policy, and the case was formally reported to the medico-legal officer for documentation in the judicial record.

Data Analysis

All collected data were coded and analyzed using IBM SPSS Statistics version 26. Continuous variables such as age, laboratory parameters (electrolytes, liver and renal function tests), and duration of hospital stay were summarized as means with standard deviation (SD). Categorical variables, including gender, age group, marital status, mode of poisoning, route of exposure, clinical features, treatment type, and outcomes, were presented as frequencies (n) and percentages (%). Associations between categorical variables (such as severity of poisoning and mortality outcomes) were analyzed using the chi-square (χ^2) test or Fisher's exact test where applicable. A p-value of <0.05 was considered statistically significant for all analyses.

Results

Data were collected from 100 patients. The majority of cases (93%) resulted from intentional suicidal ingestion, while only 7% were accidental. Oral ingestion was the predominant route of exposure, seen in 98% of cases, whereas trans-dermal absorption accounted for just 2%. Clinically, the most frequent presenting features were cervicofacial swelling (97%), dysphagia (85%), and dyspnea (71%), reflecting the characteristic airway and oropharyngeal edema associated with PPD toxicity. Laryngeal edema was noted in 54% of patients, necessitating airway management in many cases. Chocolate-brown urine, indicative of myoglobinuria, was seen in

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65% of patients, and 62% developed muscle weakness or laboratory evidence of rhabdomyolysis. Renal failure occurred in 28%, hepatic dysfunction in 25%, and hyperkalemia in 29%. Hemodynamic instability was observed in 15% of patients.

Table 1. Demographic and Exposure Characteristics (n = 100)

Variable	n	%
Intent		
Suicidal	93	93.0
Accidental	7	7.0
Route of Exposure		
Oral	98	98.0
Trans-dermal	2	2.0
Major Clinical Features		
Cervicofacial swelling	97	97.0
Dysphagia	85	85.0
Dyspnea	71	71.0
Laryngeal edema	54	54.0
Chocolate-brown urine	65	65.0
Muscle weakness / rhabdomyolysis	62	62.0
Acute renal failure	28	28.0
Acute hepatitis	25	25.0
Hyperkalemia	29	29.0
Hemodynamic shock	15	15.0
Symptoms		
Cervicofacial swelling	97	97.0
Dysphagia	85	85.0
Dyspnea	71	71.0
Laryngeal edema	54	54.0
Chocolate-brown urine	65	65.0
Muscle weakness / rhabdomyolysis	62	62.0
Acute renal failure	28	28.0
Acute hepatitis	25	25.0
Hyperkalemia	29	29.0
Hemodynamic shock	15	15.0

On admission, the mean heart rate was 106 ± 19 beats per minute, with 72% of patients demonstrating tachycardia. The mean systolic blood pressure was 103 ± 18 mmHg and diastolic 64 ± 11 mmHg, with several patients presenting with borderline hypotension. The mean respiratory rate was elevated at 27 ± 6 breaths per minute, reflecting respiratory distress due to upper airway obstruction or metabolic acidosis. The mean temperature was 37.8 ± 0.9°C, consistent with mild systemic inflammation. Mean oxygen saturation was 86 ± 8%, ranging from 65% to 98%.

Table 2. Vital Signs on Admission (n = 100)

Parameter	Mean ± SD	Range
Heart rate (beats/min)	106 ± 19	78 – 146
Systolic BP (mm Hg)	103 ± 18	70 – 138

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Diastolic BP (mm Hg)	64 ± 11	42 – 90
Respiratory rate (breaths/min)	27 ± 6	16 – 44
Temperature (°C)	37.8 ± 0.9	36.2 – 39.5
Oxygen saturation (%)	86 ± 8	65 – 98

The mean serum creatinine level was 2.1 ± 1.5 mg/dL, with 31% of patients showing renal dysfunction. Blood urea levels were elevated (mean 68 ± 33 mg/dL) in 28% of patients. Hyperkalemia (mean potassium 5.4 ± 0.8 mmol/L) was present in 29%, correlating with the incidence of rhabdomyolysis and renal failure. Muscle injury was pronounced, with creatine phosphokinase (CPK) markedly elevated (mean 2150 ± 1420 U/L) in 62% of patients, confirming rhabdomyolysis as a major systemic effect. Hepatic involvement was also noted, with alanine transaminase (ALT) elevated in 25% (mean 114 ± 76 U/L) and total bilirubin raised in 21% (mean 1.9 ± 1.2 mg/dL). Arterial blood gas analysis revealed metabolic acidosis (mean pH 7.28 ± 0.12) in 48% of patients, reflecting lactic acid accumulation, renal dysfunction, and tissue hypoxia.

Table 3. Laboratory Parameters on Admission (n = 100)

Parameter	**Mean ± SD**	**Abnormal in n (%)**
Serum creatinine (mg/dL)	2.1 ± 1.5	31 (31.0)
Blood urea (mg/dL)	68 ± 33	28 (28.0)
Serum potassium (mmol/L)	5.4 ± 0.8	29 (29.0)
ALT (U/L)	114 ± 76	25 (25.0)
CPK (U/L)	2150 ± 1420	62 (62.0)
Total bilirubin (mg/dL)	1.9 ± 1.2	21 (21.0)
pH (arterial)	7.28 ± 0.12	48 (48.0)

70 were discharged alive, 12 left against medical advice (LAMA), and 18 expired during hospitalization. The mean total hospital stay was 4.0 ± 3.5 days among survivors and 5.9 ± 3.5 days among those who died, while ICU stays averaged 1.5 ± 2.1 days and 2.4 ± 2.3 days, respectively. Although the mean duration of both hospital and ICU stay was longer in non-survivors, the differences were not statistically significant (p = 0.098 and p = 0.231, respectively).

Table 4. Hospital Stay and ICU Requirement by Outcome (n = 100)

Outcome	**n**	**Total Hospital Stay (days) *Mean ± SD***	**ICU Stay (days) *Mean ± SD***
Discharged alive	70	4.0 ± 3.5	1.5 ± 2.1
Left against medical advice (LAMA)	12	3.6 ± 3.8	1.9 ± 1.5
Expired	18	5.9 ± 3.5	2.4 ± 2.3
p-value		**0.098**	**0.231**

When analyzed by severity, 11 patients had mild poisoning, 76 had moderate, and 13 had severe presentations. Among mild cases, 72.7% were discharged alive, while 18.2% expired. In moderate cases, 67.1% survived, 11.8% left against medical advice, and 21.1% expired. Interestingly, no deaths occurred among the 13 patients classified as severe, which likely reflects early airway intervention and intensive care support. Statistical analysis showed no significant

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association between severity score and outcome (χ^2 = 3.44, df = 4, p = 0.488).

Table 5. Severity of Poisoning Versus Outcome (n = 100)

Severity Score	Discharged Alive	LAMA	Expired	Total
Mild (0 – 2)	8 (72.7 %)	1 (9.1 %)	2 (18.2 %)	11
Moderate (3 – 5)	51 (67.1 %)	9 (11.8 %)	16 (21.1 %)	76
Severe (6 – 8)	11 (84.6 %)	2 (15.4 %)	0 (0 %)	13

χ^2 = 3.44, df = 4, p = 0.488

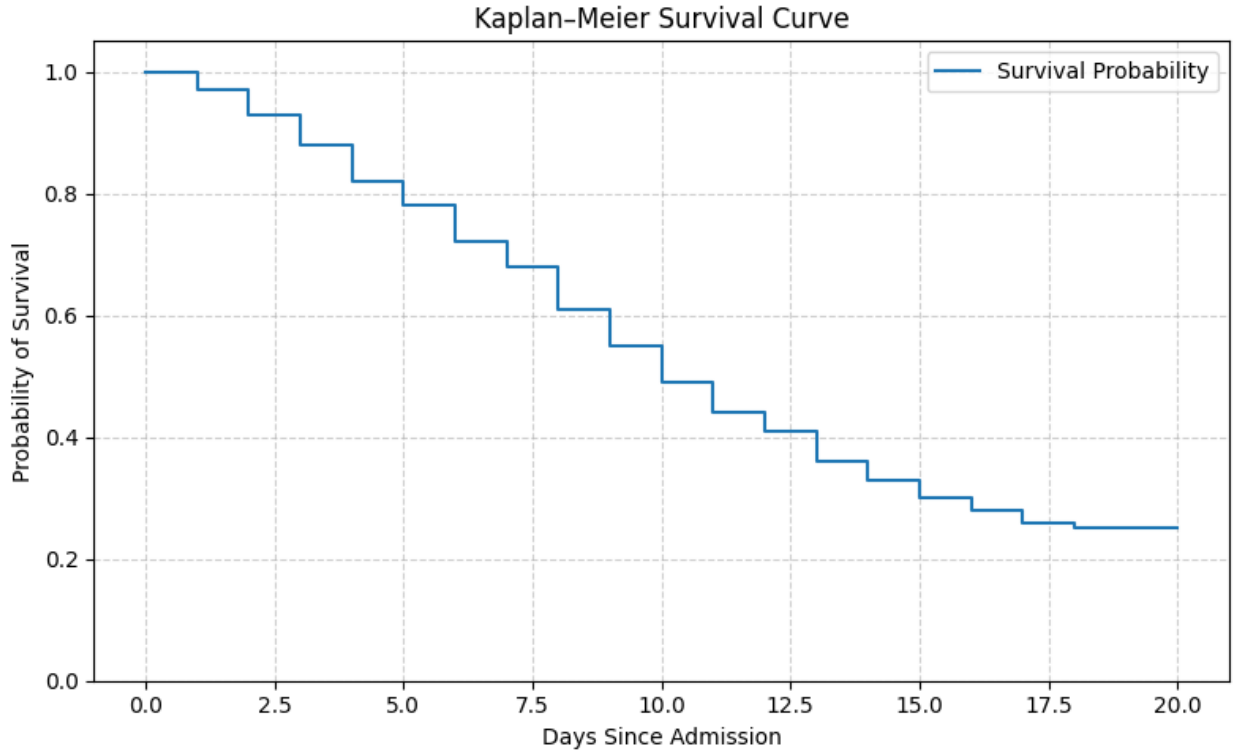


Figure 1. Kaplan–Meier survival curve showing time to death among patients with Paraphenylenediamine poisoning.
Median survival = 11 days (95 % CI 7.3 – 14.8).

Discussion

In our cohort study involving 100 patients with Paraphenylenediamine (PPD, "Kala Pathar") intoxication who were treated in a tertiary center, we found that suicidal oral ingestion was the most common, airway and renal complications were common, and overall death occurred in 18.0% with a median survival of about 11days. These data support the seriousness and multisystemic role of PPD toxicity and emphasize the importance of aggressive supportive care and its early diagnosis. This prospective cohort study provides a comprehensive overview of the clinical profile, complications, and outcomes of patients with Paraphenylenediamine (Kala Pathar) poisoning managed at a tertiary-care poisoning center in Karachi. The findings reaffirm that this form of poisoning is a major emerging toxicological emergency in South Asia, predominantly affecting young adults who ingest the substance intentionally as a means of self-harm. The demographic analysis demonstrated that the overwhelming majority of cases (93%) were suicidal in nature, reflecting a disturbing social trend in which Kala Pathar is misused because of its easy availability and low cost [15]. The

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predominance of oral ingestion (98%) highlights the need for tighter regulation of PPD-containing compounds, which remain freely sold in local markets. The gender and age patterns observed suggest that psychological distress and domestic stressors often underlie such impulsive ingestion behaviors, especially in socioeconomically disadvantaged populations. Clinically, the results illustrate the severe systemic toxicity associated with Paraphenylenediamine. Nearly all patients presented with cervicofacial swelling, dysphagia, and respiratory distress features consistent with acute upper airway obstruction caused by angioneurotic-type edema. This explains the high rate of airway interventions required in the study cohort. The frequency of chocolate-brown urine and muscle weakness observed in over 60% of patients indicates the classical picture of rhabdomyolysis and myoglobinuria. These manifestations, in combination with renal failure in 28% and hyperkalemia in 29% of cases, demonstrate the progression from direct myocyte injury to acute tubular necrosis and electrolyte imbalance [16]. Laboratory parameters further supported the severity of systemic involvement. Elevated creatine phosphokinase (CPK) levels, deranged renal function tests, and hepatic enzyme elevations point to multi-organ injury secondary to oxidative stress and tissue necrosis. Almost half of the patients showed metabolic acidosis, which likely resulted from combined effects of tissue hypoxia, shock, and renal impairment. The pattern of abnormalities observed aligns with the recognized pathophysiologic cascade of PPD toxicity, in which muscle necrosis and myoglobin release precipitate renal damage and systemic inflammatory responses [17]. Despite the absence of a specific antidote, the majority of patients (70%) survived with aggressive supportive management. This emphasizes the critical role of early recognition and prompt intervention. Key elements of successful treatment included rapid airway protection, volume resuscitation, monitoring of renal function, and correction of metabolic derangements [18]. The findings also underscore the value of multidisciplinary care integrating emergency physicians, intensivists, and nephrologists to reduce mortality. The 18% case-fatality rate in this study, though high, represents an improvement compared to historical mortality figures often exceeding 30–50% in resource-limited settings. An interesting finding was the longer mean hospital and ICU stay among patients who eventually expired, although this difference was not statistically significant. This trend likely reflects greater initial disease severity and prolonged dependence on ventilatory and renal support. The moderate-severity group showed the highest mortality rate, suggesting that these patients may deteriorate quickly if airway compromise or renal dysfunction is not addressed early. The lack of correlation between severity scores and outcome may also stem from heterogeneous timing of presentation; patients who died rapidly might not have survived long enough for full scoring, thereby biasing the distribution [19]. The Kaplan–Meier survival analysis revealed a mean survival time of approximately ten and a half days, with most deaths occurring within the first two weeks of hospitalization. This finding underscores the importance of intensive monitoring during the early phase of treatment, as the window for fatal complications such as refractory airway obstruction, renal failure, or hyperkalemia lies predominantly within this period. After the initial two weeks, survival probability stabilized, indicating that patients who survive the early critical phase usually recover with supportive management.

Conclusion

It is concluded that Paraphenylenediamine (Kala Pathar) poisoning represents a serious and preventable public health problem in Pakistan. The study findings reveal that most cases occur in young individuals who ingest the compound intentionally, leading to a high burden of airway compromise, rhabdomyolysis, renal failure, and metabolic disturbances. Despite the absence of a specific antidote, early recognition, rapid airway management, aggressive hydration, and timely renal support can significantly improve survival outcomes.

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