# BIOCHEMICAL FINDINGS IN AN ATYPICAL CASE OF KALA PATHAR POISONING

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## ABSTRACT

Paraphenylenediamine is an organic compound with chemical formula C6H4(NH2)2. Physically It is solid and white in color, but becomes black when it is oxidized. It is an aromatic amine related to aniline. It is a main ingredient in many of the hair dyes locally produced. PPD is highly toxic compound which causes significant damage to tissue and induces hypersensitivity allergic reactions. It causes skeletal and cardiac muscle necrosis, causing renal tubular occlusion due to myoglobin casts. It is also directly toxic to renal tubules. A case of acute paraphenylenediamine poisoning with atypical presentation is reported. The patient presented with complaints of myalgia and headache and did not have any respiratory distress and angioedema which is the characteristic presentation of such a case. On history ingestion of Kala pathar was revealed. He developed acute kidney and liver injury. He remained hospitalized for 18 days during which his urine output remained nil and he underwent several sessions of hemodialysis. He was discharged on request with advice of thrice weekly hemodialysis and antihypertensives. Though Government placed ban on sale and purchase of kala pathar in several districts of south Punjab in 2017, it still remains an important but under documented cause of morbidity and mortality in Pakistan.

**Key Words:** Para-phenylenediamine, Kala Pathar.

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## INTRODUCTION

Para-phenylenediamine, a derivative of paranitroaniline, has been used for dyeing furs, photochemical measurements, accelerating vulcanization and azo-dye manufacturing, as well as for oxidizing hair dyes [1]. Chemically, it is an aromatic diamine related to aniline. Cases are mostly reported from, Africa and Asia [2]. In Pakistan it is widely used in hair dyes and henna tattoos. It is added to stabilize the dye and enhance its color. It is known as Kala Pathar in Urdu language. In recent times it has increasingly become a means of committing suicide in Punjab and Sindh. In one study conducted at two major hospital in Bahawalpur 1,258 cases of PPD poisoning were reported in just 16 months, of which 94.37% were suicidal poisoning [3].

High mortality after PPD poisoning has been reported and currently no specific antidote is known. Hence recognition of its characteristic signs and symptoms resulting in early detection and intervention is particularly crucial.

Ingestion characteristically consists of vomiting and severe edema of face, neck, pharynx, larynx and upper airways culminating in acute respiratory distress, often requiring emergency tracheostomy. These are the early symptoms presenting just a few hours after ingestion. After few days signs and symptoms of systemic damage become apparent. These include dark brown colored urine [4] oliguria, anuria, myalgia and skeletal muscle tenderness. The hepatotoxic effects of PPD have also been observed in many studies [5, 6]. Tiwari *et al*. also reported elevated levels of SGPT/SGOT in their study of hair dye poisoning. The kidneys are particularly vulnerable to the toxic effects of PPD [3]. Hemolysis, rhabdomyolysis causing myoglobinuria, methemoglobinemia and direct tubular toxicity of PPD to renal tubules are possible mechanisms for AKI [4]. The PPD toxin is not known to be dialyzable. In a cohort of 100 patients presenting with PPD poisoning in SIUT Karachi, 97% of the patients required dialysis as a part of management. 16% died in acute course of illness and 77% fully recovered from acute renal injury [7]. PPD and its metabolites are cardiotoxic. Cardiac damage results in elevated cardiac enzymes like Troponin T, Troponin I and CK-MB. Tiwari, Dharmendra, *et al* reported 90% of PPD poisoning patients had S/T change and 82% had elevated CK-MB. Treatment is mainly supportive. Antihistamines, steroids and diuretics can be given. Renal replacement therapy is often needed. In case of respiratory distress, ventilator support may be required. Rare complications of PPD poisoning that

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are reported include chronic renal failure, severe myocardial rhabdomyolysis leading to cardiogenic shock and death, severe aplastic anemia, severe contact dermatitis and optic atrophy [8]

## CASE REPORT

A 24-year-old male presented in trauma centre CMH Multan with history of kala-pathar ingestion 4 days back with suicidal intent. He had complaint of generalized myalgia and headache with decreased urine output. Clinically he was alert, well oriented and afebrile. There was no neck swelling. His blood pressure was 150/110; heart rate was 90 beats/min and respiratory rate 20/min.

Systemic examination was unremarkable except for muscle tenderness and rigidity. Total leukocyte count was $17.3x10^9$/L with 95% Neutrophils, Hemoglobin was 12.7 g/dl, Red Blood Cell count $4.43x10^{12}$/L, Platelet count $184x10^9$/L and MCV 81.3 fL.

His Biochemical Profile was as follows;

| Biochemical Markers | Level |
|---|---|
| Serum Urea | 25.1 mmol/l |
| Serum potassium | 5.7 mmol/l |
| Serum CPK | 532,600 U/L |
| Serum AST | 12183 U/L |
| Serum creatinine | 614 umol/l |
| Serum sodium | 128 mmol/l |
| Serum CK-MB | 5146 U/L |
| Serum LDH | 38844 U/L |

A diagnosis of acute Paraphenyline-diamine poisoning was made based on history of ingestion and clinical signs and symptoms. Patient had severe rhabdomyolysis, acute kidney injury and acute liver injury. His urine output was nil on urinary catheterization. He remained hypertensive and his condition deteriorated. Hemodialysis was started and he was given supportive care. Intravenous fluids, corticosteroids and bicarbonate were given. I/V fluids and steroids have been shown to be helpful in treatment of acute kidney injury. Bicarbonate therapy is effective when patient develops acidemia. Renal biopsy was performed on $12^{th}$ day of admission for histopathology and report showed 30% tubular necrosis and neutrophilic and eosinophilic infiltrates in interstitium. After multiple sessions of dialysis over the course of 18 days of hospital admission his urine output remained nil. His overall condition improved and he requested to be discharged. At time of discharge his blood pressure was 140/90 and he still had no urine output. He was prescribed antihypertensives and advised Hemodialysis thrice weekly. During follow up in outpatient department his urine output and kidney functions slowly improved to normal and his hemodialysis was stopped.

## DISCUSSION

Over the past few decades self-harm paraphenylenediamine has become an emerging trend in regions of Punjab and Sindh in Pakistan and also in India and Africa [5]. A study in Nishtar Hospital, Multan showed 82.8% of all presenting patients had taken it with suicidal intent, with 97% of the patients being female [9]. A large cohort of 374 Moroccan patients was dominated by females belonging to the younger age group who had used PPD for the purpose of committing suicide [10].

Angioedema, a common presentation of PPD poisoning is edema of the deep dermis and subcutaneous tissues. Several studies have reported cervicofacial edema to be the characteristic and early symptom of this condition followed by acute renal failure. A study in Nishtar hospital Multan showed that out of 32 patients presented [6] in ICU, 93.8% showed cervicofacial edema. A10 year study conducted in Sudan reported nearly all patients having angioneurotic edema. A study in Nawabshah and Jamshoro including 76 patients showed most patients in stage 2 and 3 angioedema with onset of symptoms within 1 hour of ingestion of PPD [7].

Several other studies have shown other features similar to the one in our patient. A study in India showed mean CPK level 15027 $\pm$ 8920 IU/L in 10 patients. A study including 260 patients found cervico-facial edema was the most common presentation followed by respiratory distress, hypotension and generalized bodyaches similar to our patient. Same study showed serum creatinine levels and CPK levels were raised in 58.46% cases and 71.33% cases respectively with CPK found to be as high as 90000 IU/L [8]. Raised serum CPK, creatinine levels are usual findings reported consistently along with AKI. A study on 200 patients in Khartoum, Sudan reported 23% of patients had abnormal creatinine levels with 20.5% patients going in renal failure. Deranged liver and cardiac enzymes have also been reported previously.

A study on 1020 cases of hair dye poisoning with PPD showed 67.16% had deranged liver enzymes. The same study reported pain and or rigidity of limbs in 47.05% and hyperkalemia in 13.43% patients. Highest CPK level found in this study was 281,000 IU/L [3, 5]. Our case had CPK level 532,600 IU/L which is a rare finding. That along with absence of cervicofacial edema makes it an unusual case among other reported cases of PPD toxicity

## CONCLUSION

PPD is easily available at cheap cost; its use has significantly increased especially for purpose of suicide. There is immediate need of awareness in public regarding its devastating health consequences. While it has been banned in some districts in Punjab, it is still widely available as component of locally produced hair dyes. Such hair dyes must be banned. Research to look for an effective antidote must also be taken up and encouraged by the scientific community.

Atypical PPD poisoning cases like the one we have discussed need to be studied so that physicians are aware of its atypical presentations and can make a timely diagnosis in case the patient does not reveal history of ingestion. A timely diagnosis can be life-saving as PPD poisoning has high mortality and no antidote is available

## AUTHORS CONTRIBUTION

**Waqas Hanif Sheikh:** Manuscript writing.
**Mehreen Aftab Khan:** Case monitoring.
**Muhammad Younas:** Manuscript review.
**Zahid Farooq Baig:** Treatment of patient.
**Oshaque Ali:** Sample Analysis.
**Syed Tawanger Hussain Hurr:** Literature review.

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