Case Report

Journal of Emergency Medicine Case Reports

Intoxication Caused by Paraphenylenediamine After Henna Ingestion

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Abstract

Introduction: Henna, has been applied in eastern cultures for many years, as a hair dye and tattoo. Paraphenylenediamine (PPD) is a kind of aromatic amine added to henna. The formulation formed when PPD added known to be highly toxic.

Case report: Here, a 16-year-old patient admitted to an emergency clinic with severe angioedema, rhabdomyolysis, and acute renal failure caused by PPD after henna ingestion for suicidal purposes, was presented. An emergency tracheotomy was performed to the patient because of severe respiratory distress, and the patient was followed up by connecting to the mechanical ventilator in intensive care.

Conclusion: PPD poisoning is a life-threatening condition, and there is no specific antidote for PPD intoxication. Early intervention to the patient is essential because it is life-saving.

Key Words: Acute renal failure, Angioneurotic edema, Henna dye, Para-phenylenediamine, Rhabdomyolysis, Tracheotomy

Introduction

Henna, an extract of the Lawsonia plant, has been used in many societies for many years to dye hair, hands, and feet. It is popular in some East African countries, the Middle East, and India. Paraphenylenediamine (PPD) is an aromatic amine commonly used in various industrial products, and it usually is not found in nature[1]. PPD is added to intensify the henna color and speed up the dyeing process. Topical application of PPD sensitive people to dermatitis; may cause an increase in lacrimation, persistent blepharoconjunctivitis, and permanent blindness in local eye contact[1-3]. In 1924, the first case of PPD poisoning was reported in a hairdresser[4]. Ingestion of PPD results in severe facial, neck, tongue, and laryngeal edema with respiratory distress, which often requires urgent tracheostomy[2]. Henna intake containing PPD has a high mortality rate [up to 31%] caused by rhabdomyolysis and kidney failure[5]. The authors reported systemic poisoning with PPD, leading to angioedema resulting in tracheostomy and acute renal failure.

Case Report

A sixteen-year-old female patient was admitted to the Nyala Sudan-Turkey Training and Research Hospital emergency clinic complaining of not breathing. It was learned that the patient had ingested henna in an attempt at suicide two hours before. Diffuse edema was seen in the patient's eyes and around her face and neck. Her face and neck were swollen, and the airway was obstructed. On examination, the patient was agitated and had severe respiratory distress. The patient was cyanotic and vital signs; the pulse was 130 / min, blood pressure 160/95 mmHg, and the breathing rate was 35 / min. The oxygen saturation level of the patient was at 85% during follow-up and continued to decrease. We performed an emergency tracheotomy to keep the patient's airway open (Figure 1). Taking the intensive care unit, we connected her to a mechanical ventilator. Gastric lavage was performed by gently applying a nasogastric tube, and toxic stomach contents were removed.

The patient's head and neck edema increased by six to eight hours after starting to follow in the intensive care unit. Henna, which caused acute kidney failure due to the PPD it contains, caused the chocolate brown color urine outflow (Figure 2). The patient's potassium, Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), Creatinine Kinase (CK), and creatinine values increased (Table 1).

The patient was managed with intravenous corticosteroid [methylprednisolone 1 mg/kg], rehydration and alkaline diuretic therapy for two days. On the third day, the edema of the patient's face and neck began to decrease distinctly. The dark urine color turned light, and urine output started to increase. Serum creatinine values continued to decrease without the need for dialysis (Table 1). The patient was taken off the mechanical ventilator at the end of the third day, and then she

Corresponding Author: Fatih Öner **e-mail:** fatihoner.ent@gmail.com
Received: 26/11/2020 • **Accepted:** 28/12/2020
DOI: 10.33706/jemcr.832026

DOI: 10.33706/jemcr.832026
Journal of Emergency Medicine Case Reports 2021; 12(1):31-33

Table 1. Biochemical values of the patient

	Creatinine (IU/L*) (mean 0-1.3)	CK** (IU/L*) (mean 0-145)	Potassium (mEg/l‡,) (mean 3.5-5.5)	ALT† (IU/L*) (mean 0-45)	AST†† (IU/L*) (mean 0-35)
1st day	0,9	1453	5.7	1657	1350
2nd day	3.4	2237	5.9	1309	1232
3rd day	7.8	1807	5.3	705	549
5th day	6.7	761	4.9	578	345
7th day	4.4	323	5.0	234	141
10th day	1.1	165	4.4	41	15

**IU/L: International unit/Liter, **CK; Creatinine Kinase, ‡mEg/l: Milliequivalent per Liter, †ALT; Alanine aminotransferase, ††AST; Aspartate aminotransferase*

was taken to the clinic from the intensive care unit on the fifth day. On the seventh day, the patient was decannulated, and the tracheostomy site was closed. After mobilizing, she continued to recover more quickly, and all metabolic values returned to normal. The patient started oral intake of liquid foods and was discharged from the hospital on the 11th day.

Discussion

PPD is an allergen that can cause contact dermatitis, erythematous urticarial papules, and eczema in susceptible individuals after contact or application and the frequency of PPD allergies in the general population has increased over the years[3,6]. PPD is a highly reactive substance and has a half-life of a few hours on human skin. Skin exposed to PPD has been shown to cause extensive transcriptomic modifications, including tight connectivity and down-regulation of stratum corneum proteins, even in the nonexistence of clinical manifestations[7]. The most significant problem arises when PPD is taken orally, whether accidentally or for attempted suicide or murder. Systemic effects include angioedema, especially in the face and throat, kidney and rhabdomyolysis, and heart toxicity. While accidental intake is seen in children, among adolescents and women, as in this case, it is often drunk for suicide purposes[8].

Figure 1. The patient has severe angioedema during the initial admission to the emergency clinic

While the most common cause of tracheotomy in children used to be infections [epiglottitis, laryngitis], today, it has changed to prolonged intubation. Bezgin et al. reported that the most common tracheotomy indications in children were neurological deficits and cardiopulmonary disease[9]. As in our case, airway obstruction due to acute toxicity is much rarer. PPD begins to form diffuse angioneurotic edema in the oral mucosa, tongue, face, head, and neck regions within two hours of oral intake. Rapidly progressive tissue destruction and edema reach life-threatening levels in a short time. If emergency airway safety cannot be achieved, death will be caused by asphyxia. When our case entered the emergency department in the second hour, her level of edema required an urgent tracheotomy. Laryngeal edema and pulmonary congestion were detected in a postmortem autopsy of another suicide case[8].

Chugh et al first described kidney failure due to PPD poisoning in 1982[2]. The development of kidney failure is related to the dose taken. The beginning of oliguria after oral intake is a known sign that kidney failure will develop. Chocolate- or coffee-colored urine of the patients is a characteristic finding of PPD intoxication. All patients develop rhabdomyolysis, and approximately 80% of patients develop acute renal failure. Dialysis may be needed in the management of patients[6]. Dialysis is for support purposes only, as the toxin is not dialyzable. Kidney failure is thought to be due to myoglobinuria and the primary toxic effects of

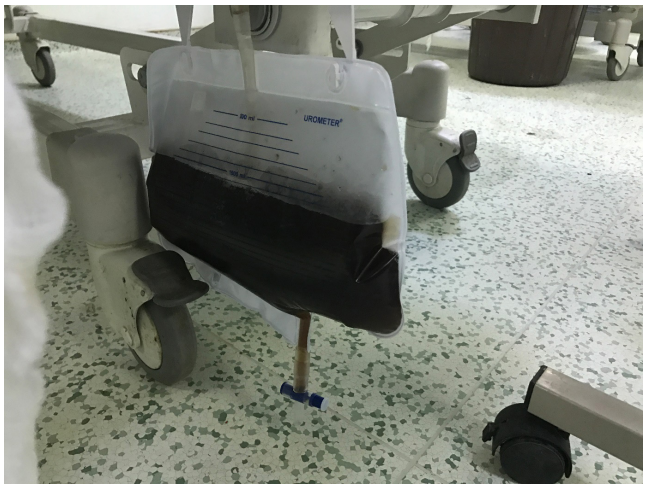

Figure 2. Typical chocolate brown urine

PPD. Acute tubular necrosis was found in a kidney biopsy[10]. Renal failure causes death in patients with airway safety effects. Electrolyte imbalances due to kidney failure lead to cardiac arrhythmias. This situation increases hospitalization time, morbidity, and mortality. Since there is no specific antidote for PPD intoxication, hydration and alkaline diuretic therapy should be started immediately upon diagnosis to protect the kidneys[2,6].

It is most important to diagnose a patient early and keep the airway open by endotracheal intubation or tracheotomy[6]. Intensive supportive treatment should be carried out. Afterward, ventilation should be provided with a mechanical ventilator in intensive care conditions. Gastric lavage should be undertaken with a gentle nasogastric tube application. It is recommended to use steroids on the first day to reduce edema in the head and neck area. Progression of kidney failure can be prevented with urgent and abundant fluid infusion therapy. To determine whether dialysis is needed, electrolyte values should be closely monitored. Alkaline diuretic therapy can be given to offset the metabolic acidosis that occurs. The use of diuretics should be started with hydration and discontinued after the first 24 hours. The use of vasopressor agents may be required[2].

Conclusion

PPD poisoning is a life-threatening condition. The condition is associated with a high risk of multisystem involvement, tracheostomy requirement, prolonged hospital stays, morbidity, and cardiac arrhythmias. The severity of symptoms is directly related to the dose of PPD taken. It is crucial to start supportive therapy immediately since there is no specific medicine known for PPD intoxication. Urgent supportive therapy can be life-saving in cooperation with early recognition, rapid referral, and appropriate specialties. We presented this case because it is rare in our country, and we wanted to draw the attention of the clinicians to this urgency.

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