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CASE REPORT

# Effective Approach to Potassium Permanganate Poisoning: Case Report and Review of Literature

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## Abstract

*Background:* Potassium permanganate (PP) is a highly corrosive and deadly agent with a reported lethal dose of 10 g in adults. In this report, successful treatment of a patient poisoned with three times the lethal dose of PP is presented, and effective and early approach to such emergency toxicologic condition is discussed.
*Case Presentation:* A 24-year-old woman presented to emergency room of our hospital following PP ingestion (30 g) one hour earlier. She had swollen and stained (dark brown / black) oral cavity, tongue, face, neck and palms. As the patient had stridor, labored respiration, and obstructive swollen upper airways in the emergency room; surgical bedside tracheotomy was performed and later she was transferred to intensive care unit for further treatments. Later, a 20-hour regimen of intravenous N-acetyl cysteine injection was started for her. The patient was also treated with proton pump inhibitor, broad spectrum antibiotics, steroid, analgesic and IV fluids. She was decannulated on the 7th day and discharged home on the 13th day post-admission.
*Discussion:* No specific antidote is available for this poisoning and so the mainstay of treatment is supportive. Airway management is of utmost importance since PP exposure can cause upper airway edema leading to respiratory compromise. Intensivists should be prepared for difficult intubation, emergency cricothyrodotomy or surgical tracheostomy to secure airway. Steroids might be useful for reversing airway
*Conclusion:* This case report mainly emphasizes the significance of early management of difficult airway during resuscitation of patients with ingestion of highly caustic agents, namely PP in this report, and timely supportive care for the improved ultimate outcome.

**Keywords:** Acetylcysteine; Laryngeal Edema; Poisoning; Potassium Permanganate; Tracheostomy

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## INTRODUCTION

Potassium permanganate (PP) is an odorless, crystalline, water soluble, powerful oxidizing agent with chemical formula of $KMnO_4$ (1). It has been used formerly for various purposes such as abortion induction, urethral irrigation for gonorrhea, gastric lavage for poisoning with alkaloid agents and local remedy for the bite site of snake attacks (2). Nowadays, it is most often used in baths and wet bandages as a dermal antiseptic and antifungal especially for patients with exudative eczema (3).

This substance is a highly corrosive and deadly agent with a reported lethal dose of 10 g in adults (1). Accidental or suicidal ingestion can produce wide range of complications (both local and systemic) leading to death (4). Although this poisoning is rare, the frequency of case reports related to PP ingestion for deliberate self-harm has shown an increasing trend in recent years (1-5). In this paper, successful treatment of a patient poisoned with three times the lethal dose of PP is presented and effective and early approach to such emergency toxicologic condition is discussed.

## CASE PRESENTATION

A 24-year-old woman presented to emergency room of our hospital following PP ingestion one hour earlier according to the history given by the patient's relatives. The relatives found 5 empty packets of PP powder in vicinity of her and they declared that they could not find any evidence of other drugs. According to her family, there was no previous personal or family history of psychiatric illness.

On examination, she had swollen and stained (dark brown / black) oral cavity (Figure 1A), tongue, face, neck and palms. The patient was confused, agitated, afebrile with heart rate of 120 beats/min, respiratory rate of 32 breaths/min and normal blood pressure (120/80 mmHg). The arterial oxygen saturation could be established within normal limits ($SpO_2$ = 95%) with 8 liters of oxygen by face mask. Arterial blood gas analysis showed partly compensated metabolic acidosis (pH = 7.3, $PCO_2$ = 29 mmHg, PO2 = 84 mmHg, $HCO_3$ = 15 mEq/L). On auscultation, crepitation was heard on right basal region. Cardiovascular and gastrointestinal systems were normal. Except sinus tachycardia, electrocardiogram was normal.

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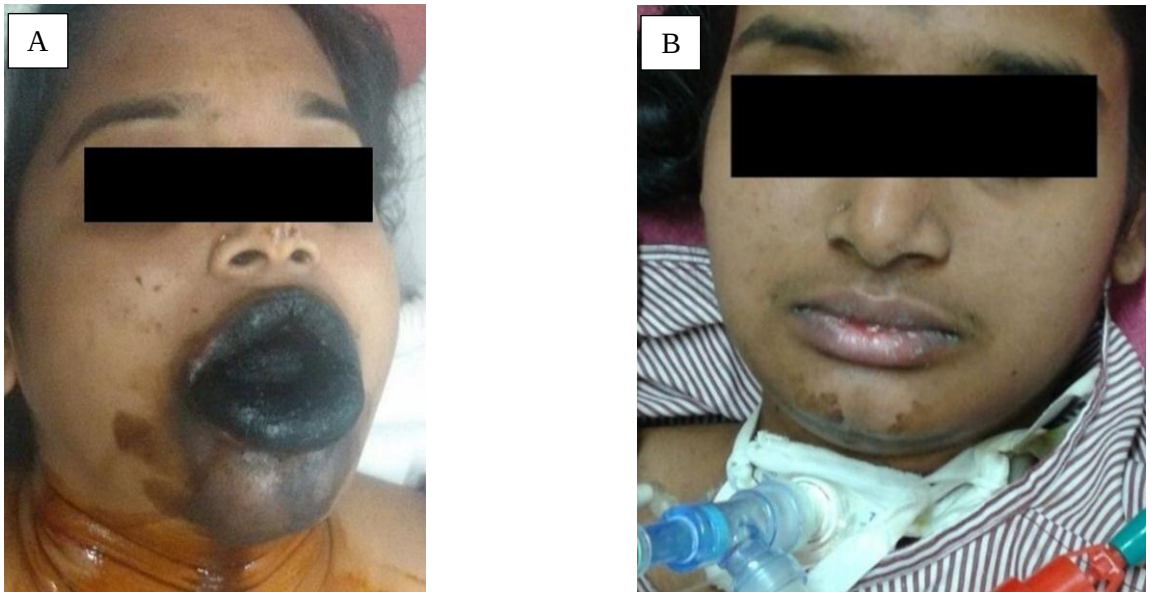


**Figure 1.** A) Swollen and black-brown stained lips and oral cavity of the patient with potassium permanganate ingestion, B) The patient on the sixth day post-admission (disappearance of swelling and discolorations of the oral cavity)

As the patient had stridor, labored respiration, and obstructive swollen upper airways in the emergency room; surgical bedside tracheotomy was performed for her using 7.0 tracheostomy tube, and later she was transferred to intensive care unit (ICU) for further treatments.

A 20-hour regimen of intravenous (IV) N-acetyl cysteine (NAC) injection (150 mg/kg bolus over 15 minutes + 50 mg/kg infusion over 4 hrs + 100 mg/kg infusion over 16 hrs diluted in 500 mL of 5% dextrose in each phase) was empirically started for the patient. SpO2 and PaO2 levels remained in normal limits during NAC treatment. Although methemoglobinemia can occur with PP poisoning, her methaemoglobin level was in normal levels (2%), and so methylene blue was not administered and the occurrence of methemoglobinemia was monitored during her stay in the ICU. The patient was also treated with proton pump inhibitor, steroid, analgesic and IV fluids. Broad spectrum antibiotics in view of risk of perforation and peritonitis were used and after ruling out these complications, they were stopped.

Her initial laboratory findings including complete blood count, liver function test, serum creatinine, serum electrolytes, coagulation profile, serum amylase and lipase were in normal limits. Chest X-ray was normal. Upper gastro-intestinal endoscopy revealed brown-black discoloration of oropharynx and esophagus along with laryngeal edema, small esophageal erosions and small erosions in fundus of stomach.

Liver function status, coagulation profile, chest X-ray and serum creatinine were monitored up to 72 hours, and none of them showed any deterioration or irregularity. Total parenteral nutrition was administered for 4 days, and later feeding jejunostomy tube was placed for enteral nutrition. The patient was slowly weaned from mechanical ventilation as her readiness for extubation was ascertained by T-piece trial. Her facial and tongue edema subsided slowly along with disappearance of brown-black discoloration from oral cavity (Figure 1B), and palms. Subsequently, she was decannulated on the 7$^{th}$ day. Oral feeding was encouraged on the 10$^{th}$ day. The patient was transferred to internal medicine ward on the 11$^{th}$ day and discharged home on the 13$^{th}$ day post-admission. Before discharge she was given a psychiatric counseling when she declared suicidal ideation. Ethical committee approval was obtained from the institution and informed consent from the patient for publishing the patient's data. The patient admitted consumption of five 6-gram packets of PP. She was in good condition in follow-up visits.

## DISCUSSION

PP exposure can cause wide range of complications. Coagulation necrosis occurs when tissue is exposed to PP (1). The corrosive effects of PP ingestion on the gastrointestinal tract and the eyes may be secondary to the formation of potassium hydroxide, a strong alkaline corrosive agent (2). Alkaline corrosives cause liquefaction necrosis, allowing deep penetration into mucosal tissue as cells are successively destroyed (2). Systemic effects of PP exposure are due to free radicals-induced oxidative injury (2,4,6,7). The lethal dose of PP in adults has been reported to be about 10 g (1,2), while our patient could resist 3 times higher doses than this dose (~ 30 g).

Complications of PP exposure ranging from local complications such as edema and ulceration of upper airways to multi-organ dysfunction are summarized in table 1 (1-10). Our patient developed discoloration and edema of oral cavity, tongue and larynx causing stridor and marked difficulty in breathing for which emergency bedside surgical tracheostomy was required in the emergency room. Early upper gastrointestinal endoscopy within 24 hours of ingestion has been advocated to look for the extent of mucosal injuries which further helps clinicians make decision on choosing the suitable treatment (11,12). PP is an acid in nature, and thus it is responsible for high-grade caustic damages firstly in stomach and then in esophagus (12). Our patient had

**Table 1.** Complications related to potassium permanganate ingestion (1-10)

| System organ | Complications |
|---|---|
| Oral cavity and upper airways | Edema and ulceration of lips, tongue, larynx and oral cavity |
| Gastrointestinal tract | Acute: nausea, vomiting, esophageal / gastric perforation, ulceration of esophagus and stomach, peritonitis, massive hemorrhage<br>Late-onset: esophageal strictures, pyloric stenosis |
| Cardiovascular system | Tachycardia, heart block, hypotension, shock, cardiac arrest |
| Respiratory system | Pulmonary infiltrations, acute respiratory distress syndrome |
| Kidneys | Subcortical and papillary hemorrhage |
| Liver | Fatty change and necrosis |
| Pancreas | Hemorrhagic pancreatitis |
| Hematologic | Methemoglobinemia, DIC |

DIC: Disseminated intravascular coagulation

esophageal and gastric erosions. Development of acute hemorrhagic pancreatitis was reported in a patient with 20 g PP ingestion (9). In our patient; however, serum amylase and lipase levels were normal and no pancreatitis was detected.

Potential therapeutic measures to reverse PP poisoning effects are summarized in table 2 (1-10). No specific antidote is available for this poisoning and so the mainstay of treatment is supportive (3,4,10). The gastric lavage may be harmful due to risk of perforation and the role of activated charcoal is controversial (1). Airway management is of utmost importance since PP causes upper airway edema leading to progressive obstruction and stridor (4). Intensivists should be prepared for difficult intubation, emergency cricothyrodotomy or surgical tracheostomy to secure airway during initial resuscitation (4).

Since PP can induce liver injuries like paracetamol overdose, early administration of NAC in the same dose as in paracetamol poisoning, has been advocated to treat or reduce the free radicals induced hepatic injury (6,13,14). Based on the past evidence, we administered NAC for our patient during the early stage of admission. For our patient, NAC

**Table 2.** Potential treatments for potassium permanganate poisoning (1-10)

| Therapeutic approach | Indications | Contraindications | Potential side effects |
|---|---|---|---|
| Activated charcoal | Controversial, might be helpful if used within the first hour post-exposure | Unprotected airway, GI strictures, GI perforation, need for endoscopic visualization | Aspiration, peritonitis, interaction with endoscopic imaging |
| Gastric lavage | Controversial, many references emphasize that gastric lavage should not be performed for caustic ingestions | Caustic ingestions | Aspiration, peritonitis |
| Endotracheal intubation | Stridor, non-advanced airway edema | Severe airway trauma or obstruction, cervical spine injury, class 3 or 4 of potential difficult airway* | Miss-placed intubation, oropharyngeal trauma, broken teeth or dentures |
| Cricothyrodotomy or emergency surgical tracheostomy | Difficult airway, obstructive airway edema, oropharyngeal hemorrhage, uncontrollable emesis | Children younger than 12 years, inability to identify landmarks (cricothyroid membrane), tumor, infection, or abscess at site of incision, lack of operator expertise | Aspiration, subglottic stenosis, laryngeal stenosis, hemorrhage, hematoma, Esophageal/tracheal laceration, mediastinal emphysema, vocal cord injury<br>Exclusively for needle cricothyroidotomy: Inadequate ventilation, posterior tracheal wall perforation, subcutaneous emphysema, thyroid perforation |
| Upper GI endoscopy | Early upper GI endoscopy is preferred within the first 24 hours post-exposure | Hemodynamic instability, peritonitis, mediastinitis | Infection, hemorrhage, GI perforation |
| Broad spectrum antibiotics | Laryngeal edema, peritonitis due to perforation | No sign or possibility of infection | Variable |
| N-acetyl cysteine | Increased liver enzymes, acute liver failure | Allergy to the drug | Nausea, vomiting, increased bronchial secretions |
| Methylene blue, vitamin C | Symptomatic methemoglobinemia (> 25 % methemoglobin) | Glucose-6-phosphate dehydrogenase deficiency (for methylene blue) | For methylene blue: shortness of breath, tachypnea, chest discomfort, burning sensation in the mouth and stomach, paresthesia, restlessness, apprehension, tremors, nausea and vomiting, dysuria, and excitation, pulse oximeter interaction, hemolytic anemia (in high doses) |
| Steroids | Upper airway edema | --- | --- |
| Acid reducers** | Protection against caustic damages on GI mucosa (for all patients) | --- | --- |

GI: gastrointestinal
* According to Mallampati classification
** Histamine antagonists (H2 blockers) or proton pump inhibitors

treatment might be described as an empiric practice, because no evidence of hepatic injury was available on admission. Nonetheless, to save the critical time, it would be wise to administer this treatment to PP poisoned patients as proposed in several articles (6,13,14).

For counteracting methemoglobinemia, methylene blue and vitamin C has been proposed (3,15). Our patient had normal methemoglobin, SpO2 and PaO2 levels, and so methylene blue was not given. Nonetheless, the patient was closely monitored during the ICU admission for any episode of methemoglobinemia. The role of corticosteroids is controversial, some scientists believe it can reduce edema and suppress inflammatory response (4). We used it for our patient and there was adequate decrease in edema during her ICU stay.

## CONCLUSION

This case report mainly emphasizes the significance of early management of difficult airway during resuscitation of patients with ingestion of highly caustic agents, namely PP in this report, and timely supportive care for the improved ultimate outcome.

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