Case Report

Potassium permanganate toxicity: A rare case with difficult airway management and hepatic damage

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Abstract

Potassium permanganate ($KMnO_4$) is rarely used for suicidal attempt. Its ingestion can lead to local as well as systemic toxicities due to coagulation necrosis and damage, caused by free radicals of permanganate. We recently managed a case of suicidal ingestion of $KMnO_4$ in a lethal dose. She had significant narrowing of upper airway leading to difficult intubation as well as hepatic dysfunction and coagulopathy as systemic manifestation. We suggest to keep ourselves ready to handle difficult airway with the aid of fiber optic bronchoscope or surgical airway management in such patients. Upper gastrointestinal (GI) endoscopy should be done at the earliest to determine the extent of upper GI injury and further nutrition planning.

Keywords: Coagulopathy, difficult airway, emergency endoscopy, hepatic dysfunction, potassium permanganate

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Introduction

Potassium permanganate ($KMnO_4$), is a powerful oxidizing agent, which is readily available over the counter. It is a highly corrosive and water-soluble oxidizing agent. Tissue contact produces coagulation necrosis. Lethal consequences of oral ingestion are well described, with morbidity and mortality caused by airway edema and obstruction or circulatory collapse. Recently we managed a case of suicidal ingestion of the lethal dose of $KMnO_4$ presenting in ER with hypoxia and upper airway obstruction and later on development of acute hepatic toxicity and coagulopathy as systemic manifestations.

Case Report

A 30-year-old housewife presented to hospital emergency with ingestion of around 15 g of $KMnO_4$ powder with a glass of water as suicidal attempt. No other drugs were ingested. Patient was happily married with no psychiatric illness in the past. She reached the hospital within 15 min of ingestion of the substance. On arrival in an emergency, she was drowsy with hypoxia and stridor. Her oxygen saturation on monitor was 85%, and initial arterial blood gas (ABG) revealed pH 7.32/pO_2 52.1/pCO_2 46/HCO_3 22/SpO_2 86.1%. She had tachycardia with heart rate of 116/min and normal blood pressure (110/70 mmHg) and temperature of 98.2°F. There were multiple patches of blackish-brown stain on face and hands. She was immediately shifted to medical intensive care unit for management. Her oral cavity examination revealed complete brownish black staining and copious secretions leading to very poor differentiation between structures of the oral cavity. Her vocal cords were swollen with almost complete obstruction of airways. Percutaneous tracheostomy was planned as the first choice in view of significant airway obstruction. During preparation for percutaneous tracheostomy attempt of intubation was taken in view of impending respiratory failure. Intubation was very difficult with stained and edematous pharynal and laryngeal structures and sloughed debris present in posterior pharynx. Small size (6.5 mm high volume low pressure cuffed) endotracheal tube was placed under fiber optic bronchoscopic (OLYMPUS BF type 1T150 video bronchoscope) guidance and intermittent positive pressure ventilation was initiated. Patient was managed with invasive ventilatory support along with broad spectrum antibiotics, steroids in view of significant edema and

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proton-pump inhibitor. Intravenous fluids were started to maintain hydration. With mechanical ventilation, her oxygentation and ABG improved (ABG pH 7.37, pO_2 113, pCO_2 33.9/HCO_3 19.2/SpO_2 98.1%). Her sensorium also improved.

Upper gastrointestinal (GI) endoscopy was done on day 1, which revealed a diffuse ulceration and necrotic areas in esophagus and fundus of the stomach. Bronchoscopy was performed for assessing the airway, which revealed edematous and inflamed mucosa and hemorrhagic patches in entire tracheo-bronchial tree until the segmental bronchus. Her initial renal and hepatic parameters, electrolytes and complete hemogram were normal. Her methemoglobin level was also normal. Patients liver function test became deranged on 2nd day with rising serum bilirubin. In next 24 h her total serum bilirubin increased to 4.5 mg/dl and serum glutamic pyruvic transaminase to 354 IU. Her coagulation profile was also deranged with INR of 2.06 and decreased platelet counts (80,000/cumm). Her clinical picture was suggestive of acute hepatic necrosis. On day 4 of hospital admission, her coagulation profile started improving but serum transaminase levels were still elevated. Oral cavity examination on day 4 revealed significant reduction in staining of mucosa as well as edema of upper airway structures. Vocal cords swelling reduced, and the patient was extubated after cuff leak check. Postextubation, she had copious oral secretions and difficulty in swallowing as well as breathing, therefore, she was reintubated and percutaneous tracheostomy was done on the same day. Patient was weaned off the ventilator next day and later shifted to ward with a tracheostomy tube *in situ*. Total parenteral nutrition was started on day 5 for nutrional support her condition gradually improved, and she started accepting clear liquids orally on day 8. Her liver functions and coagulation profile also improved slowly. Later patient was decannulated on day 9 and discharged in good health condition. Follow-up after 3 months was normal.

Discussion

Potassium permanganate can react with tissues to produce coagulative necrosis owing to its powerful oxidative potential. The lethal adult dose of $KMnO_4$ is 10 g or the equivalent of 1.5 teaspoons of crystals.[1] Possible pathogenesis of the systemic toxicity with $KMnO_4$ is due to oxidative injury from free radicals generated by the absorbed permanganate ion. Its clinical course closely resembles severe paracetamol overdose.[2] The type and severity of symptoms varies depending on the amount of chemical involved and the nature of the exposure. Our patient seemed to have absorbed the $KMnO_4$ from the GI tract as revealed by the brownish black staining in the oral cavity and necrotic and ulcerated areas in the esophagus and fundus of the stomach on upper GI endoscopy. GI erosions can lead to massive hemorrhage. Pyloric stenosis and esophageal strictures[3] are reported as late complications. Systemic toxicity of $KMnO_4$ can cause acute respiratory distress syndrome, pancreatitis,[4] hepatic and renal damage,[2,5] methemoglobinemia[6] and disseminated intravascular coagulation. Increased level of serum transaminase (alanine transaminase) at 345 IU, serum bilirubin at 4.5 mg/dl and normal alkaline phosphatase values were suggestive of hepatocellular damage in our case. Ong *et al*.[1] reported a fatal case of $KMnO_4$ poisoning with severe fatty changes and necrosis of the liver, lung consolidation, and subcortical and papillary hemorrhages of the kidney. Involvement of kidney and pancreas were, fortunately, absent in our case. Cardiovascular depression including heart block, rapid heart rate, hypotension, shock and cardiac arrest have also been reported with potassium permanganate toxicity.[7]

Treatment of $KMnO_4$ is mainly supportive. The immediate priority is to secure the airway. Due to acute laryngeal edema and airway obstruction, early intubation is necessitated which was done in our patient. Stridor is characteristic of upper airway obstruction and suggests a significant reduction in airway diameter. Its presence, together with an inability to swallow secretions, should be a warning of severe airway compromise, and critical care team should be ready for difficult intubation. In an expected difficult intubation, an awake fiber optic intubation may be indicated but the presence of secretions, including blood, may make this technically difficult. In the event of a failed intubation, emergency tracheostomy or crico-thyrodotomy may be required. Dhamrait reported a case of deliberate ingestion of $KMnO_4$ crystals leading to macroglossia and hypoxia. Patient responded to nebulized epinephrine (5 ml, 1:1000). Author also suggested role of inhalational induction with sevoflurane in oxygen allowing the intubation in a spontaneously breathing patient.[8]

Although gastric lavage has been recommended by some, it is potentially hazardous as there is a danger of perforation. The effectiveness of activated charcoal[9] is not known, thus its administration is controversial. Indeed, there has been a documented case of fatal poisoning when charcoal was mistaken for $KMnO_4$.[10] Drinking of milk or water may have a diluting and neutralizing effect.[9] Early esophagoscopy is useful to determine the extent of upper GI injury, preferably with in 24 h.[7] Korkut *et al*. reported case of $KMnO_4$ ingestion, which stuck at the posterior wall of the gastric corpus leading to necrotic area and

bleeding on endoscopic removal with forcep. Bleeding was controlled by argon plasma coagulation.[11]

The use of corticosteroids[9] is controversial although it has been postulated that they minimize tissue edema and the pathological inflammatory response. Broad spectrum antibiotics are advised in view of the risk of perforation and subsequent peritonitis. Other supportive treatment includes the administration of methylene blue and Vitamin C for methemoglobinaemia.[6] Useful investigations for monitoring include liver and renal function tests, methemoglobin level, serum amylase, and serum manganese level.

Conclusion

Potassium permanganate is mainly used as an antiseptic agent. It is a frequent culprit in accidental consumption, typically by children. Suicidal attempt by $KMnO_4$ is rarely reported. Being highly corrosive and oxidizing agent, it can produce local as well as systemic toxicity. Upper airway injuries due to oral ingestion can be lethal because of the difficult airway and respiratory failure. We should keep ourselves ready to handle difficult airway with the aid of fiber optic bronchoscopy or surgical airway management in such patients. GI damage due to ingestion of $KMnO_4$ is again uncommon situation. Upper GI endoscopy should be considered at the earliest to determine the extent of upper GI injuries and nutrition planning.

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