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Case Report

Suicidal Ingestion of Potassium Permanganate Crystals: A Rare Encounter

Ravikanti Karthik, Hari Prasad Kanakapura Veerendranath, Siddraj Wali, Murali N T Mohan, Praveen A. C. Kumar[1], Gaganam Trimurty

Departments of Emergency Medicine, and [1]Medical Gastroenterology, Vydehi Institute of Medical Sciences and Research Centre, Bangalore, Karnataka, India

ABSTRACT

Potassium permanganate poisoning is not common. Although Symptoms of potassium permanganate ingestion are gastrointestinal and Complications due to ingestion of potassium permanganate include cardiovascular depression, hepatic and renal damage, upper airway obstruction, bleeding tendency and methemoglobinemia. Gastric damage due to potassium permanganate has rarely been reported previously. We are reporting a 34-year old female patient who presented to our Emergency Department after suicidal ingestion of potassium permanganate crystals. After treatment, the patient was discharged home on the 8th day after admission. So we conclude that Emergency endoscopy has a significant role in diagnosis and management of potassium permanganate ingestion.

Key words: Emergency department, emergency endoscopy, gastric damage, potassium permanganate crystals

INTRODUCTION

Potassium permanganate is a powerful oxidizing agent. It is an odorless, crystalline substance available in powder or tablet form and is readily accessible without prescription. It is used clinically as an antiseptic and antifungal agent.[1,2] Historically its uses have been interesting: It has been used as an abortifacient, as a urethral irrigation fluid fortreatment of gonorrhea, as a fluid for stomach washout in cases of alkaloid poisoning, and inthe solid form as a local remedy for snake bite.[3] Formulations include ready-to-use solutions, pellets, tablets, crystals, and powder. At room temperature, potassium permanganate exists as dark purple or bronze-colored, odorless, sweetish, astringent tasting crystals, that are described as having a blue, metallic sheen. It is readily soluble in water; aqueous solutions are pink to violet in color and slowly deposit manganese dioxide. The corrosive effects of potassium permanganate ingestion on the gastrointestinal (GI) tract and the eye may be secondary to the formation of potassium hydroxide, a strong alkaline corrosive. Alkaline corrosives cause liquefaction necrosis, allowing deep penetration into mucosal tissue as cells are destroyed.[4] Potassium permanganate poisoning is not common. Symptoms of potassium permanganate ingestion are GI (such as dysphagia, odynophagia, nausea, and vomiting, which are a result of GI edema, burns, and ulcerations), respiratory, and circulatory.[5] Complications due to ingestion of potassium permanganate include hepatic, renal damage, upper airway obstruction, bleeding tendency, and methemoglobinemia. Major causes of deaths for severe potassium permanganate poisoning are cardiovascular depression and collapse, upper airway obstruction, hemorrhagic shock owing to massive GI bleeding.[1,6,7] Gastric damage due to potassium permanganate has been rarely reported previously. Herein we describe a case of suicidal ingestion of potassium permanganate and early use of emergency upper GI endoscopy to assess the severity and to guide management.

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Address for correspondence: Dr. Ravikanti Karthik, 3rd Year Pg in Department of Emergency Medicine, Vydehi Institute of Medical Sciences and Research Centre, #82, Epip Area, Whitefield, Bangalore - 560 066, Karnataka, India. E-mail: karthik.ravikanti@yahoo.in

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CASE REPORT

A 34-year-old woman presented to emergency medicine department an hour following suicidal ingestion of half a teaspoon of potassium permanganate crystals (approximately 3.33 g) [Figure 1] following which she had two episodes of vomiting, burning pain in the throat, and epigastrium. Past medical history was insignificant except for type II diabetes mellitus for which she was on medication with tab.metformin 500 mg once a day. On arrival, she was conscious but agitated. Her vital parameters were as follows: Heart rate (HR): 110 bpm, blood pressure (BP): 150/90 mmHg, respiratory rate (RR): 22 cpm, random blood glucose: 210 mg/dl, and oxygen saturation (SpO_2) was 98% on room air. Electrocardiogram (EKG) showed normal sinus rhythm and arterial blood gas was normal. Her Glasgow Coma Scale (GCS) was 14 (E4V4M6). On physical examination, her oral cavity showed reddish brown stains over buccal mucosa, teeth, lower vermilion border, and hard palate. Her airway was patent and no stridor was present. Her breathing and circulation was normal. Systemic examination was unremarkable except formarked epigastric tenderness. All of her laboratory values were normal except for an elevated total leukocyte count of 17,720/μL; chest radiogram was normal.

Neither orogastric tube insertion nor gastric lavage was done. Gastroenterologist consultation was taken and upper GI endoscopy was performed 6 h after arrival to hospital. Upper GI endoscopy revealed normal esophagus and gastro-esophageal (GE) junction [Figure 2], below GE junction upper two-third of stomach more towards greater curvature, mucosa is inflamed with potassium permanganate crystals embedded over the inflamed mucosa [Figure 3]. Pyloric opening was normal, duodenum bulb and D_2 were normal [Figure 4]. Because of wide area of inflammation associated with crystals deposition and increased risk of perforation irrigation, and debridement was not performed in our patient.

In view of anticipated complications associated with potassium permanganate poisoning, the patient was admitted in intensive care unit for observation and thorough psychiatric evaluation was done. Rest of her hospital stay was uneventful. The patient was discharged on 8th day after admission after confirming the serum manganese report (0.8 μg/dl) as normal. Patient was asked to review after 3 weeks for follow-up endoscopy which showed a linear gastric ulcer [Figure 5], but the patient was asymptomatic post-discharge.

DISCUSSION

Potassium permanganate is a highly corrosive, water-soluble oxidizing antiseptic for cleansing and deodorizing suppurative eczematous reactions and wounds, used in baths and wet bandages.[8] It is a simply obtainable, over the counter oxidant. The strong oxidizing action of potassium permanganate causes burns depending on the concentration and amount of local irritation[9] and it causes liquefaction necrosis.[4] Formulations include ready-to-use solutions, pellets, tablets, crystals, and powder.[4] Ingestion of concentrated solutions or dry crystals of potassium permanganate can cause swelling and bleeding of lips and tongue, pharyngeal edema, and swelling of the larynx. Our patient had inflamed mucosa associated with burns and ulcerations over buccal mucosa, lower vermilion border, and hard palate with swelling of lips with brownish stained potassium permanganate crystals in oral cavity secondary to ingestion. Burns and ulceration of the mouth, esophagus, and stomach are due to the action of potassium permanganate.[6]

Ingestion of potassium permanganate may result in damage to the upper GI tract. Manifestations of the GI symptoms of potassium permanganate ingestion include nausea and vomiting. Ingestion of potassium permanganate can cause GI complications similar to acid and alkali ingestion. Effect of potassium permanganate on the GI tract is alkaline. Burns and ulceration of the mouth, esophagus, and stomach are due to the action of potassium permanganate.[6] Correlating well with the literature our patient had moderately severe gastritis of fundus and body of stomach (upper two-thirds of stomach more towards greater curvature below gastroesophageal junction) with embedded potassium permanganate crystals over the inflamed mucosa and epigasric burning pain following two episodes of vomiting was the main complaint. Necrotic ulcers developed as a result of potassium permanganate consumption may lead to perforation. Esophageal stricture and pyloric stenosis are late complications that are reported in the literature.[10] Our patient developed a linear ulcer over body of stomach as a late complication secondary to caustic effect of potassium permanganate.

Systemic effects secondary to ingestion of potassium permanganate crystals do not usually manifest due to poor absorbance. However; ingestion of potassium permanganate may cause systemic toxic effects such as tachycardia, hypotension, hallucinations, methemoglobinemia and cyanosis, metabolic acidosis, hemolysis, adult respiratory distress syndrome, coagulopathy, acute hepatic failure, acute renal failure, pancreatitis, and even death in severe cases. Some effects can be delayed up to 36 h post ingestion including disseminated intravascular coagulation, cardiac failure, and hepatorenal failure.[8] The systemic toxicity is believed to be due to oxidative injury.[1,5] Our patient did not have any systemic toxic effects except for local gastrointestinal effects or complications. The reported lethal adult dose of potassium permanganate is 10 g[6] or the equivalent of 1.5 teaspoons of crystals;[11] and the dose

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Figure 1: Potassium permanganate crystals ingested by our patient

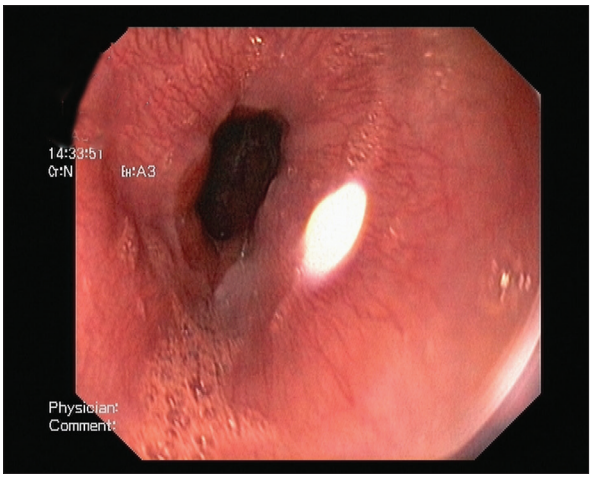


Figure 2: Normal esophagus and gastroesophageal junction on upper gastrointestinal endoscopy in our patient

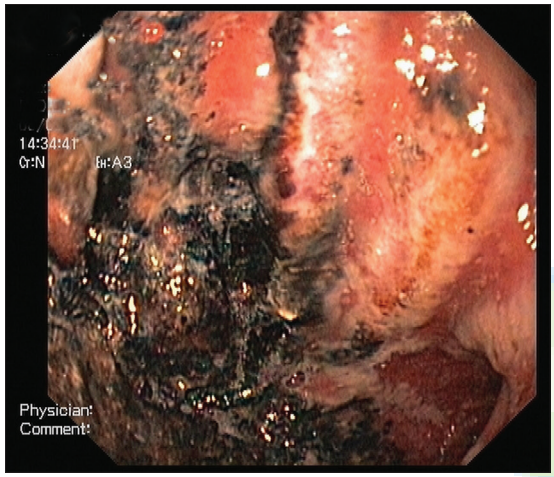


Figure 3: Body of stomach with moderately severe gastritis on upper gastrointestinal endoscopy with embedded potassium permanganate crystals

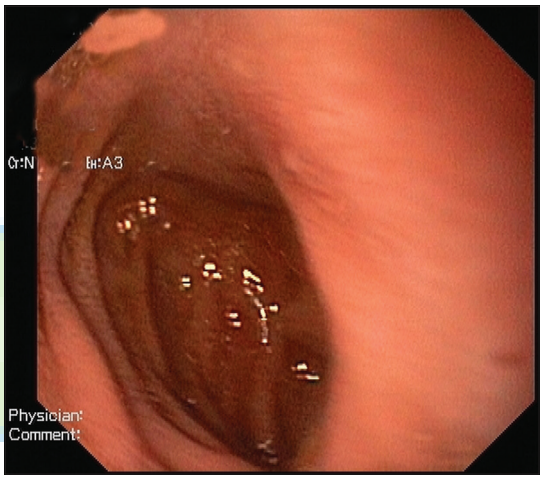


Figure 4: Normal duodenal bulb and D2 on upper gastrointestinal endoscopy in our patient

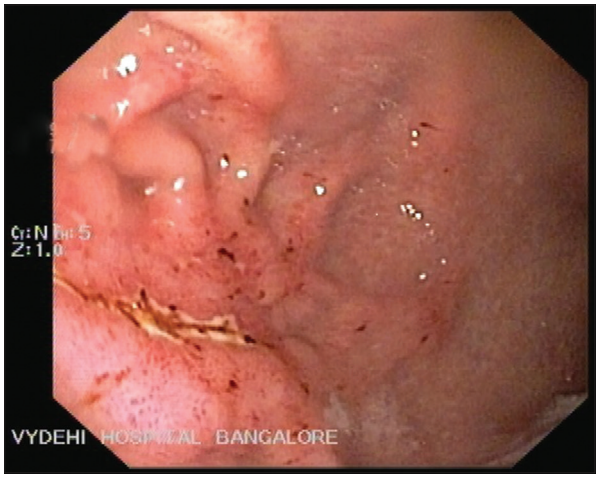


Figure 5: A linear ulcer over the body of stomach on follow-up upper gastrointestinal endoscopy at 3 weeks of ingestion

which was taken by our patient was lower (3.33 g potassium permanganate) than the toxic dose.

The mainstay in the treatment of potassium permanganate is supportive,[4,6,8] and the immediate priority is to secure the airway.[6]

The immediate concern after the ingestion of potassium permanganate is the threat of acute laryngeal edema[12] and this necessitates early endotracheal intubation or even emergency surgical airway. Our patient did not have any symptoms or signs suggestive of airway obstruction at presentation and during the course of stay in the hospital. There is a risk of perforation in insertion of nasogastric tube; however because of the potential for severe systemic toxicity, insertion of a small, flexible nasogastric tube for aspiration of gastric contents should be considered after large ingestions, at the discretion of the clinician.[4] However, a nasogastric tube insertion or gastric lavage was not done in our case because of the risk of perforation. Emetic and acidic agents are contraindicated.[13] Induced vomiting, nasogastric application, or giving of neutralizing agent

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should be avoided.[14] The effectiveness of activated charcoal is not known in potassium permanganate poisoning, thus its administration is controversial.[15,16] In our case, activated charcoal was not administered as performing emergency endoscopy was planned.

Emergency endoscopy is an important tool used to evaluate the location and severity of injury to the esophagus, stomach, and duodenum after caustic ingestion. Emergency endoscopy is not only useful to assess the severity of damage but also to guide management.[5] Patients with signs and symptoms (vomiting, drooling, stridor, or dyspnea) of intentional ingestion should undergo endoscopy within 12–24 h to define the extent of the disease. Endoscopy performed too early may miss the extent or depth of tissue injury.[17] Delayed endoscopy increases the risk of perforation.[18] Because of this follow-up exams should also be avoided between days 5 and 15.[19] In our patient, endoscopy was performed after 6 h post-ingestion and follow-up endoscopy was done 21 days post-ingestion showing a linear gastric ulcer as a late complication secondary to caustic effect of potassium permanganate.

Useful investigations in potassium permanganate poisoning include liver andrenal function tests, methemoglobin level, serum amylase, and serum manganese levels. Liver and renal functions were within normal limits as was serum amylase. In our patient we did not measure methemoglobin levels as there were no clinical signs suggestive of methemoglobinemia and arterial blood gas was normal. We did in fact measure serum manganese levels at 12 h post-ingestion and were normal (0.8 μg/dl). The use of corticosteroids is controversial, although it has been postulated that they minimized tissue edema and the pathological inflammatory response in one patient.[16] We did not administer any corticosteroids to decrease inflammatory edema in our patient.

Suicidal ingestions of potassium permanganate were rarely reported. GI damage due to ingestion of potassium permanganate crystals is an uncommon situation. In our case, corrosive damage was seen only in the stomach. Early endoscopy should be considered to determine the extent of upper GI damage in patients with suspected injury to the GI tract.

CONCLUSION

Emergency endoscopy has a significant role in diagnosis and management of potassium permanganate ingestion. Hence, early emergency endoscopy should be considered to determine the extent of upper GI damage in the Emergency Department.

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