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Case Report
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A Rare Case of Poisoning: Potassium Permanganate Toxicity

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Abstract

Potassium permanganate (PP) toxicity causes serious morbidity and mortality, though it is rarely observed clinically. Supportive treatment is essential because there is no specific antidote. Gastrointestinal (GI) damage rarely occurs due to the ingestion of PP. A 66-year-old visually impaired patient was admitted to the intensive care unit of our hospital due to toxicity following the intake of 20 PP pills in a suicide attempt. Upper GI system endoscopy was performed at the 20th hour of hospitalisation. Ulcero-necrotic corrosive gastritis was found to have developed in the stomach corpus. In this case report, we aim to discuss the current diagnostic and treatment approaches in PP poisonings in lieu of the previous literature.

Keywords: Endoscopy, gastric damage, intoxication, potassium permanganate

Introduction

Potassium permanganate (PP) is clinically used to provide antiseptic, disinfectant, antimycotic and deodorising effects in abscesses, wound cleaning, mouthwashes, eczema and skin diseases. It is also used to disinfect vegetables and fruits that are eaten uncooked. In the past, there have been reports of the use of PP in interesting indications, such as abortion, treatment of snake bites and gonorrhoea and prevention of sexually transmitted diseases (1-3). The symptoms of potassium permanganate (PP) ingestion can be gastrointestinal (GI), respiratory and/or circulatory. Gastric symptoms of PP poisoning include dysphagia, odynophagia, nausea and vomiting, which result from GI oedema and ulcerations (4, 5). Early diagnosis and treatment are life-saving (6). PP, which has corrosive and systemic toxic effects, is sold as an over-the-counter agent in many countries throughout the world. Therefore, access to the drug is directly possible without a prescription. Although PP is very easy to access, the occurrence of toxicity is rare and usually occurs after oral intake. Herein, we describe a case of a suicide attempt with PP intake that caused gastric damage.

Case Presentation

A 66-year-old man who was blind by birth was admitted to the emergency service at our hospital with complaints of nausea and vomiting 2 hours after taking 20 PP pills in a suicide attempt. According to the patient's history, he had a discussion with a relative, after which he ingested a total of 5 g of PP (250 mg PP per pill x 20 pills) and vomited 3 times on the way to hospital. He had previously been instructed to take PP to treat eczematous lesions on his feet. The general condition of the patient was good; he was conscious and was fully oriented and cooperative. No pathology was detected in the physical examination. His laboratory values were as follows; blood glucose: 110 mg dL^{-1}, Hb: 13.2 g dL^{-1}, WBC: 5000 µL, PLT: 241.000 µL and INR: 1.1. His hepatic function parameters were normal (AST: 20 µL, ALT: 9 µL and GGT:18 µL). No significant changes were observed in the control laboratory values. He had no vomiting, melena, or haematemesis. An upper GI system endoscopy was performed at the 20th hour of hospitalisa-

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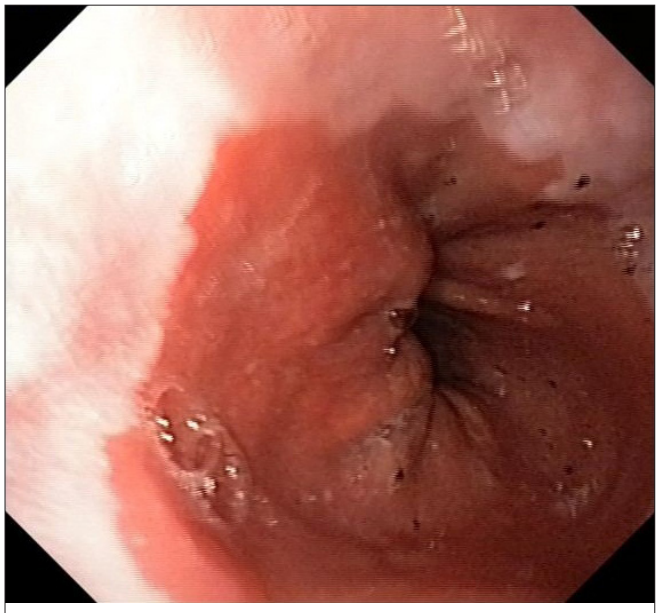

Figure 1. Ulcerations in the lower end of the oesophagus

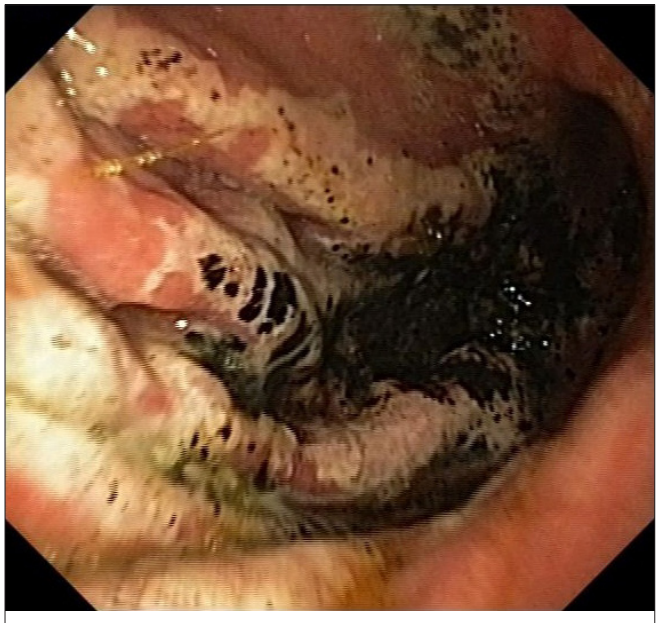

Figure 2. Ulcero-necrotic corrosive gastritis in the large corpus curvature of the stomach

tion in the intensive care unit. In the oesophago-gastro-duodenoscopic examination, the oesophageal mucosa and lumen appeared to be normal, however, starting from the entrance to the stomach, necrosis was observed in the centre of the ulcerations and the corpus major curvature, which were thickened locally. Necrotic corrosive gastritis was identified in the corpus (Figure 1 and 2). The patient was given oral regimen 1 (water), hydrotalcide (3 g day^{-1}), sucralfate suspension (12 g day^{-1}), misoprostol (600 µg day^{-1}), N-acetylcysteine (1.800 mg day^{-1}), ranitidine (75 mg day^{-1}) and pantoprazole (80 mg day^{-1}), ceftriaxone (2 g day^{-1}) and methylprednisolone (40 mg day^{-1}), while intravenous therapy was started by gastroenterology. The patient was transferred to the gastroenterology service on his 2nd day in the intensive care unit. After discharge, the patient was scheduled for a clinical and endoscopic follow-up because of possible gastric stricture and obstruction. Written informed consent was obtained from the patient.

Discussion

Potassium permanganate toxicity is rarely observed clinically and there are a limited number of such cases in the literature. PP-associated toxicity cases have been reported both in children and adults. Children often seem to suffer from accidental exposure, while adults are usually suicidal. PP is generally used as an antiseptic and antifungal. It causes coagulation necrosis in tissues with its corrosive and oxidative properties (7, 8).

When our patient used the drug orally for the purpose of suicide, the toxic effects of PP were encountered, and the case was accepted as a toxicity case. It has been reported that the lethal dose is 10 g (3). In our patient, gastric ulceration developed due to the caustic effect of PP. Gastric ulceration due to PP poisonings has been rarely reported previously (4, 9). No other clinically significant findings were found. The reason for this may be that the previous patients have taken PP at a dose of 5 g, causing vomiting and the associated pharmacodynamic processes with vomiting to occur within 10 minutes of taking the drug.

The most effective way to observe the effects of PP in the gastrointestinal tract is to perform upper gastrointestinal endoscopy (4, 9, 10), however, the time of the endoscopy is important. Early endoscopy leads to the skipping of developing lesions, and late endoscopy leads to stricture development. Some researchers are of the opinion that the ideal time for endoscopy application is between 6 hours and 24 hours in toxicity cases (5). The fact that our patient underwent endoscopy at the 20th hour is compatible with the endoscopy time suggested in the literature.

There is no specific antidote defined for PP toxicity, therefore, supportive treatment is usually preferred. Since the application of activated charcoal is generally not recommended, we did not administer activated charcoal to our patient. We started broad-spectrum antibiotics because they are recommended in cases where there is a risk of perforation and peritonitis (11).

PP toxicity is clinically similar to paracetamol toxicity. Therefore, it is suggested that the antioxidant N-acetylcysteine should be given in the early stages of PP toxicity because of its protective and hepatic damage-reducing effects (8, 12). We gave N-acetylcysteine to our patient for this purpose. Al-

though their role in reducing therapeutic effects and complications has not been proven, anti-acids, H2 receptor blockers and proton pump inhibitors provide symptomatic relief (2, 13). Despite conflicting views on the administration of corticosteroids, there is a general positive trend towards the administration of these drugs. They are thought to reduce tissue oedema and kerb the pathological inflammatory response (2, 6, 11), and we administered the same to our patient. At the same time, we preferred to use a proton pump inhibitor and an H2 receptor blocker in our treatment as well.

Conclusion

Although PP toxicity is rarely observed, it can lead to very important clinical outcomes. In our study, ulcero-necrotic corrosive gastritis developed in the gastrointestinal tract due to the ingestion of 5 g of PP in our patient, causing significant systemic complications in the gastrointestinal tract, which is an indication of the high toxicity of PP. Due administering immediate treatment because of the patient's admittance to the emergency department, the toxic effects of PP were successfully managed. Early application of endoscopy contributes to early diagnosis, identification of the lesion site, deciding the direction of treatment and shortening of the treatment period. Therefore, we conclude that upper GI endoscopy plays an important role in the diagnosis and treatment of potassium permanganate poisoning.

Informed Consent: Written informed consent was obtained from patient who participated in this case.

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