

Case Report

Suicidal corrosive ingestion in diabetes mellitus patient: what should we do?

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ABSTRACT

The corrosive agent is often used for suicide plans, and it can cause severe damage, including various gastrointestinal pathologies such as strictures, perforation, and bleeding. In more severe cases, it can lead to multiorgan system failure, disseminated intravascular coagulation, and sepsis. These was a reported case of corrosive ingestion with diabetes mellitus as a comorbidity. Prompt, appropriate, and immediate initial management by healthcare professionals is crucial to reduce complications from corrosive agents.

Keywords: Corrosive agent, Alkali agent, Suicidal, Diabetes mellitus

INTRODUCTION

Approximately 200,000 cases of caustic poisonings occur each year, usually involving acids and alkaline chemicals that are frequently used in homes for cleaning, according to American association of poison control.¹ The corrosive agent is also used for suicide. There is not enough data to indicate the number of corrosive agents that can cause death, but it is known that the use of corrosive agents can cause severe damage like various gastrointestinal pathologies such as strictures, perforation, bleeding, and in more severe cases, multiorgan system failure, disseminated intravascular coagulation, and sepsis. Patients also typically require longer treatment.^{1,2} Appropriate management must be implemented to reduce deterioration. Therefore, this case report aims to remind healthcare professionals about management of corrosive ingestion cases with comorbid diabetes mellitus.

CASE REPORT

A 56 years-old man came to the emergency room complaining of vomiting. The patient also complained of nausea. Shortly before coming to the emergency room, the patient drank corrosive agent mixed with soda drink, about

1 glass. Afterward, the patient started feeling a burning sensation in the throat, chest, and stomach, followed by vomiting. The patient said earlier that he drank corrosive agent because he just wanted to die. He mentioned feeling sad and stressed, with a lot on his mind for about the past 6 months. He mentioned thinking about his children. He said for last 6 months, they have often felt sad, pessimistic, withdrawn, and decreased appetite. As for sleep patterns, the patient tends to sleep a lot during day and has difficulty sleeping at night. Patient has a history of diabetes mellitus with treatment of metformin and glibenclamide.

Physical examination revealed that patient was conscious and general condition was found moderately ill, with blood pressure 110/70 mmHg, respiratory rate 20 breaths/min, temperature 36°C, and oxygen saturation 98%. Laboratory result show white blood cell 14.72 u/l, Hb 15.6 g/dl, platelets 285.000/uL, urea 27 mg/dl, creatinine 1.1 mg/dl, SGOT 27 U/L, SGPT 21 U/L, and blood sugar 283 mg/dl.

Patient was treated without gastric lavage and nasogastric tube placement. Patient should be kept fasting. The treatment included NaCl 0.9%: D5% at 30 drops per minute, ceftriaxone 1 gram bid, esomeprazole 40 mg bid, ondansetron 4 mg tid, methylprednisolone 62.5 mg bid (for 3 days), and insulin injections 8-8-8 IU. During treatment,

patient's blood sugar fluctuated (Table 1). Fasting blood sugar (06.00), 2 hours post prandial blood glucose (10.00), and random blood sugar were measured in patient, and the result of that influences adjustment of insulin dosage.

After 9 days of treatment, the patient's condition improved. There was no found nausea and vomiting. Patient was also referred to a psychiatric specialist for management of his physiologic symptoms, and got therapeutic for its.

Table 1: Patients blood sugar fluctuated.

BS	Day 1 st	Day 2 nd	Day 3 rd	Day 4 th	Day 5 th	Day 6 th	Day 7 th	Day 8 th	Day 9 th
06.00 (mg/dl)	361	291	294	321	292	443	319	302	254
10.00 (mg/dl)	-	-	291	257	306	444	324	248	206
22.00 (mg/dl)	221	223	231	-	-	-	193	152	-
Aspart (IU)	16 TID	20 TID	20 TID	20 TID	20 TID	Drip insulin	15 TID	17 TID	17 TID
Lantus (IU)	-	4	4	4	6		10	12	12

DISCUSSION

According to the report of the American association of poison control there are about 200,000 caustic poisonings annually, most frequently with acid and alkaline agents that are used as cleansing substances in the households.^{1,3} Corrosive drugs do not appear to be utilized as a successful suicide strategy, however the damage and impairment it inflicts are serious and. Many gastrointestinal damages, including strictures, perforations, bleeding, and, more rarely, multiorgan system failure, disseminated intravascular coagulation, and sepsis, can arise after ingesting a caustic substance.²

The length of contact, the chemical agent's corrosive power (pH for acids and bases or oxidative potential), and the chemical concentration all affect the degree and location of gastrointestinal lesions that result from ingesting corrosive agenyys. Because more viscous chemicals may stick to the GI tract wall and increase contact length, viscosity may also influence the severity of chemical harm.²

Acids induce coagulation necrosis, which may limit the substance's ability to penetrate deeper and cause more injuries by forming eschar. On the other hand, alkalis penetrate deeper into tissues and trigger liquefying necrosis and saponification once they interact with tissue proteins.⁴ Once alkali agent is consumed, the patient usually suffers more damage. Alkali agents rapidly establish bonds with tissue proteins upon contact, which frequently causes the esophagus to become corrosive. On the other hand, acidic agents often cause injury to the stomach lining. Acidic agents tend to have lower surface tension, which allows them to avoid the esophagus and cause less severe or frequent esophageal injury.⁵ Long-term sequelae of alkali ingestion include strictures of the esophagus, odynophagia, dysphagia, malnutrition, and even cancer.^{5,6}

Acute clinical symptoms of corrosive ingestion include vomiting, dysphagia, odynophagia, chest pain, hoarseness, drooling, abdominal pain, hematemesis, and stridor and for acute signs, can be found fever, tachypnea, tachycardia,

hypotension, mucosal burn, and abdominal tenderness, and maybe subcutaneous emphysema. In laboratory results can be found high white blood cell count.^{2,7}

The cornerstone of all caustic ingestions is airway and hemodynamic stabilization.⁸ It is not recommended of using gastric lavage or activated charcoal since there is a possibility that the corrosive substance could be re-exposed to the esophagus, leading to more injury. Although controlled studies have not demonstrated the effectiveness of milk or water, it is proposed that they be advantageous during the acute period (the first one to three hours). Use of milk might risk urgent esophago-gastroduodenoscopy, and heat produced by the chemical reaction might result in further post-corrosive damage.⁹

A large number of authors think that neutralization is contraindicated because, in order to be effective, it must be done within the first hour after ingestion of a caustic agent.¹⁰ Antibiotic usage in cases of acute caustic poisoning is still topic of discussion. Antibiotics are not often recommended in the treatment of caustic poisoning because there aren't enough controlled trials to support their usage. Nevertheless, carefully monitored experiments conducted on animals have demonstrated that bacterial infiltration of mucosa that has been corrosively injured and intense inflammation cause tissue granulation, which in turn causes tissue fibrosis. Because of this, certain authors defend the use of a broad spectrum of antibiotics, most notably those in the penicillin group.¹¹

For late post-corrosive complication, the most serious outcome is stricture formation. Steroid use has been shown in previous research to either prevent or lessen stricture formation. It is still up for debate whether corticosteroids should be used for acute caustic poisonings. Studies including 361 patients revealed that patients getting corticosteroid treatment had 19% esophageal and stomach stenosis, while patients not receiving corticosteroids had 41% stenosis. These individuals received a daily dose of either 1 mg/kg of dexamethasone or 2 mg/kg of prednisolone.¹² The length for steroids usage is administered for short period (3 days).¹³

It is no longer advised to do routine nasogastric intubation in order to evacuate any residual corrosive agent. This is because it may cause retching or vomiting, which could expose esophagus more by causing the residual intragastric corrosive agent to reflux. Additionally, inserting a foreign body in an emergency could serve as a nidus for infection, delaying the healing of the mucosa.¹⁴ Upon admission, patient also should be kept fasting for 10-12 h. Esophageal rest can last up to day 10 after ingestion or, according to some authors, up to day 15, until the first endoscopic follow-up. Other authors recommend introduction of liquids 48 h after ingestion if the patient can swallow its own saliva. Some authors consider that optimal approach and treatment of critically ill patients is fast and early enteral nutrition over parenteral nutrition with no nutritional support. Enteral nutrition results in reduction of initial morbidity and reduces the length of stay in hospital.¹⁵ Gastric acid suppression with H₂ blockers or intravenous proton pump inhibitors are often initiated to allow faster mucosal healing and to prevent stress ulcers.¹⁶

In this case, the patient with alkali agent intoxication received prompt, appropriate, and immediate initial management. Gastric lavage was not performed, and instead, the patient was kept fasting, antibiotics were administered for infection prevention, steroids to reduce the occurrence of late complications such as strictures, and a proton pump inhibitor to expedite mucosal healing. Nasogastric tube placement was also not performed.

Diabetes mellitus is a comorbidity in these patients, so the patient's blood sugar levels need to be monitored regularly, and insulin was administered during treatment. Inflammation and the use of steroids in the patient can lead to hyperglycemia.¹⁷ Therefore, strict observation and adjustment of insulin doses are necessary to manage blood sugar levels effectively in this case. The patient's condition continued to improve day by day, and after nine days of observation in the hospital, the patient was discharged.

CONCLUSION

Corrosive agents often used for suicide. Alkaline agents can cause more severe damage compared to acids. Complications such as perforations, bleeding, and, more rarely, multiorgan system failure, disseminated intravascular coagulation, and sepsis, can arise after ingesting a caustic substance. Therefore, health care professionals need to be aware of appropriate management strategies such as avoiding gastric lavage, administering antibiotics, short-term steroids, and PPIs to reduce the occurrence of strictures. Patients with comorbid diabetes mellitus require closer monitoring during inflammation process and steroid use.

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