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Case Report Open Access

Management of Acid Toilet Bowl Detergent-Induced Chemical Injuries

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Abstract

The main component of acid toilet bowl detergents used in Japan is 9.5% hydrochloric acid and cationic surfactant. Similar toilet bowl detergents are sold worldwide, and they are easily available. However, if taken in the wrong way, they prove very harmful, even a small amount. There is no specific therapy in cases of ingestion of acid toilet bowl detergent. Therefore, symptomatic treatment and general management such as controlling breathing and circulation are provided for the patient. Here, we presented a 75-year-old man attempted suicide by ingesting a large amount of acid toilet bowl detergents. In this case, we could diagnose in acute phase with careful questioning and the emergency crew activity at the scene of the accident. On the basis of the diagnosis, we provided symptomatic treatment and general management, but we were necessary for a treatment for a very long term. It was difficult to establish enteral nutrition due to chemical injury to digestive tract. That might be one of the causes of repeated infections such as bacterial translocation. The possibility of a temporary enterostomy should have been considered to establish early nutrition as part of intensive care.

Keywords: Chemical injury; Acid toilet bowl detergent; Hydrochloric acid; Cationic surfactant; Corrosive esophagitis

Introduction

Major acid toilet bowl detergents used in Japan consist of 9.5% hydrochloric acid and cationic surfactant. Toilet bowl detergents with similar compositions are commercialized all over the world and are readily available even though they are hazardous because even a small amount can cause serious illness [1]. Corrosive injury caused by acid intake often occurs as accidental ingestion by a child or as a suicide attempt by an adult [2-5].

For chemical injuries caused by contact with corrosive materials, symptoms such as oropharyngeal pain, dysphagia, abdominal pain, hematemesis, and dyspnea occur during the acute phase. Serious cases may lead to laryngeal edema, pulmonary edema, shock, gastrointestinal perforation, mediastinitis, acidosis, electrolyte abnormality, or Disseminated Intravascular Coagulation (DIC) [2,6-11]. During the chronic phase, stenosis is found in the esophagus or stomach [2,10]. Several reports showed these symptoms, but pathophysiology was uncertain in some symptoms.

It is difficult to deduce the intake of acid toilet bowl detergents from the viewpoint of toxidrome. Therefore, a thorough medical history and field therapy information obtained from the emergency services are important clues [12]. However, even if it turns out that the substance ingested is acid toilet bowl detergent, specific therapy does not exist. Some therapies such as dilution, Early Esophagogastroduodenoscopy (EGD), vitamin E, steroids are recommended, but there are no strong evidences. Therefore, general management and symptomatic treatment are provided for the patient [2,12]. These treatments do not have explicit criteria.

Here, we describe our experience with a patient who had consumed a large quantity of acid toilet bowl detergent. The patient's condition was serious during the acute phase. Later, the patient required long-term hospitalization because of difficulties with establishing enteral nutrition due to chemical injury to the digestive tract. We think that caused repeated infections such as bacterial translocation.

This case was important because it made us realize that not only respiratory or circulatory management but also early establishment of enteral nutrition is a critical part of general management for a poisoned patient.

Case Report

A 75-year-old man who collapsed and was found by his neighbors was transferred to our emergency medical care center because of respiratory discomfort. He informed us that he had consumed 200–300 mL of acid toilet bowl detergent as an attempt to commit suicide. The acid toilet bowl detergent container was found at the location.

The patient had a history of hypertension and cerebral infarction and had been treated with oral medicine. Although he did not have a mental disorder, he was prescribed hypnotics (Zolpidem) for insomnia.

Upon admittance, he was agitated (Glasgow Coma Scale (GCS) 13, E4V3M6) and presented with blood pressure of 159/108 mmHg, pulse rate of 114 beats/min, respiratory rate of 25 breaths/min, body temperature of 36.0°C, and oxygen saturation of 91% on 100% O_2. He had myosis, pupil diameter was 1.5 mm in both eyes, marked full-body sweating, and heavy secretions from the nasal and oral cavities. He was vomiting repeatedly. Emergency tracheal intubation was deemed appropriate. However, easy bleeding in the oral cavity, along with laryngeal-pharyngeal swelling, would have made intubation difficult. Therefore, the cricothyroidotomy was performed.

Monitor electrocardiogram showed repetition of arrhythmia. A chest X-ray showed decreased transparency of the lung field. There were no clear signs of digestive tract perforation in a chest-abdominal

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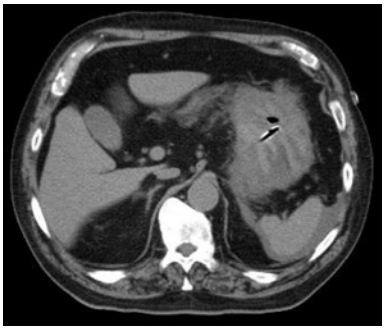
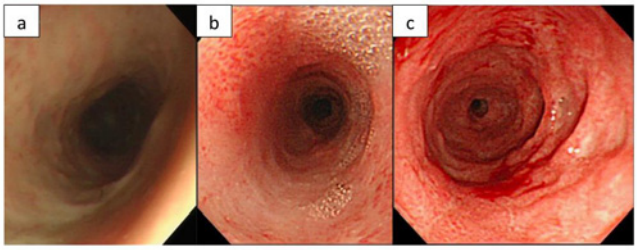


There were no signs of digestive tract perforation, but there were signs of inflammation of the adipose tissue surrounding the stomach.

Figure 1: Chest-abdominal CT scan.



a. On day 11, the EGD showed generalized ulceration of the esophagus and multiple gastric ulcers

b. On day 32, the EGD showed advanced esophageal stenosis after corrosive esophagitis

c. On day 37, the EGD showed advanced stenosis where passage was difficult even with a thin transnasal endoscope 41 cm from the incisor

Figure 2: Esophagogastroduodenoscopy.

Computed Tomography (CT) scan. However, there were signs of gastric wall thickening and inflammation of the adipose tissue surrounding the stomach. (Figure 1) Blackish-brown drainage was observed flowing continuously from a nasogastric tube.

Blood tests showed that his White Blood Cell (WBC) count had increased to 12500/μL and his Creatine Kinase (CK) level had increased to 2282 IU/L. Blood gas analysis revealed a pH of 7.29 and acidosis. There was no electrolyte imbalance. No other apparently abnormal findings were observed.

On the basis of the patient's testimony, evidence from the emergency crew, and clinical findings, he was diagnosed with chemical injury to the respiratory tract, gastrointestinal mucosa damage, pulmonary edema, and arrhythmia caused by ingestion of acid toilet bowl detergent.

After entering the Intensive Care Unit (ICU), artificial respiration control using positive end-expiratory pressure (PEEP), a large transfusion, and circulation support with catecholamine were performed. Additionally, milk, a mucosal protectant, and Proton Pump Inhibitor (PPI) were administered.

The patient showed metabolic acidosis, cardiac arrhythmia, and a high serum CK level, indicating that he may have taken other medicines. Therefore, urine alkalinization with sodium bicarbonate was also performed.

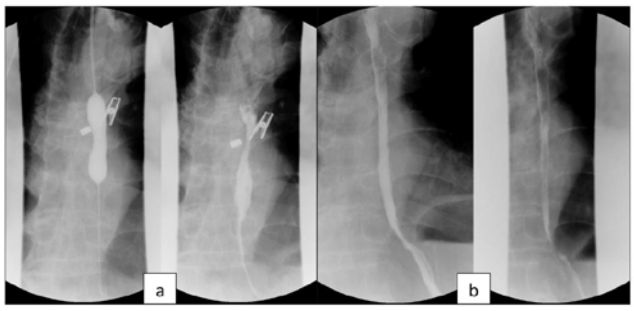
Regarding nourishment, the patient was not fed immediately and intravenous nutrition was started on day 4, when his hemodynamic became stable. After approximately 1 week in the ICU, the patient's respiration and circulation improved, and since the blackish-brown drainage from the nasogastric tube had decreased, Glutamine injection from a stomach tube was initiated on day 8, and an EGD was performed on day 11.

The EGD showed generalized ulceration of the esophagus and multiple gastric ulcers. (Figure 2a) Administration of PPI and intravenous feeding were continued, and the nasogastric tube was removed. On day 32, a second EGD was attempted but advanced esophageal stenosis prevented the endoscope from passing through. (Figure 2b) Difficulty was also experienced in passing through a thin transnasal endoscope. (Figure 2c) On day 40, upper gastrointestinal series was performed, with ballooning on the area of stenosis. (Figures 3a and 3b) However, because pyloric stenosis prevented oral feeding, (Figure 4) a gastrojejunostomy and Roux-Y reconstruction were performed on day 75.

Thereafter, the uninhibited flow of contrast media was confirmed through upper gastrointestinal series. Intake of solid food was, nevertheless, still difficult.

The elastic bougie was used on multiple occasions for the recurrence of esophageal stenosis. However, oral intake continued to be difficult even after elimination of stenosis. Thereafter, the patient had mainly been maintained on a fluid diet up until his discharge on day 127.

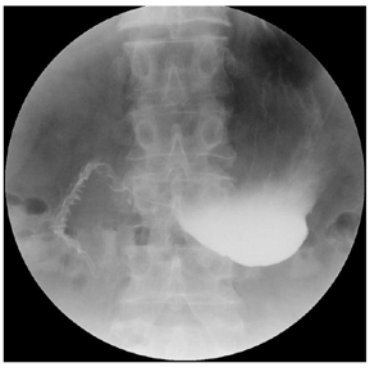
In this case, the patient developed repeated infections which were thought to be caused by bacterial translocation.



a. On day 40, ballooning on the area of stenosis was performed

b. On day 45, upper gastric series showed improved esophageal stenosis

Figure 3: Upper gastrointestinal series (Esophagus).



On day 45, upper gastrointestinal series showed advanced pyloric stenosis

Figure 4: Upper gastrointestinal series (Stomach).

Discussion

Although acid toilet bowl detergent is hazardous, it is easily available for ordinary household cleaning purposes. Most physicians are rarely confronted with acid ingestion, but they should know about the management of acid ingestion because it can occur anytime.

In some cases, several symptoms such as laryngeal edema, pulmonary edema, shock, gastrointestinal perforation, acidosis, electrolyte abnormality, or DIC may occur. However, specific therapies for acid ingestion does not exist, and general management by controlling breathing and circulation and treatment based on the symptoms is provided for patients [2,12].

There is contranersy in the therapy such as diluting acid, because it is considered that not only concentration but also amount or contact time affects the severity of chemical injury of acid. Dilution is often hesitated because it may extend the injury.

Several studies reported that milk was better than water for diluting the acid or protecting the gastrointestinal mucosa and that gastric lavage using milk was effective. However, these studies were not of high quality and the efficacy of these treatments is unknown [3-15]. In addition, definite criterion for a practical treatment method is not available.

For this patient, we performed general management and removed the gastric contents through a stomach tube in order to protect the gastrointestinal mucosa from injury. Milk (50 mL) was administered 6 h after the patient had first ingested the acid, but it failed to prevent sequential pyloric stenosis. To our knowledge, no previous study has assessed the effects of milk administration on a chemical injury caused by acid consumption. Our results suggest that 50 mL (1 mL/kg of body weight) of milk was not adequate for this patient and that the timing of administration (6 h after acid ingestion) was too late. Because this patient also developed esophageal stenosis, it was thought that oral administration would have been the ideal administration route.

Several reports have indicated that the decision regarding an early treatment strategy based on the results of initial endoscopy is effective for managing disorders in digestive organs caused by acid ingestion [2,6,9,12,16,17]. However, the validity of these results is uncertain because these were not high quality studies.

Although we aimed for conservative treatment without surgery, surgery was required for this patient. In addition, the establishment of enteral nutrition was impossible for this patient until an extended period had elapsed.

The patient could not eat solid foods even after morphological stenosis was eliminated by performing several sequences of esophageal dilatation. The patient might have developed functional abnormalities due to nerve and muscle alterations caused by corrosion of the esophagus [18].

Furthermore, the patient developed pyloric stenosis with esophageal stenosis. Because severe esophageal stenosis made it difficult to evaluate the stomach adequately, the diagnosis and treatment of pyloric stenosis was delayed.

On the basis of these results, we believe that enterostomy should be performed early in order to establish enteral nutrition, [19] regardless of the need for surgical treatment of stenosis of the esophagus and the stomach.

In this case, the patient required long-term hospitalization. It was difficult to establish enteral nutrition, and the difficulty in establishing nutrition might have caused repeated infections such as bacterial translocation. Many studies have shown the importance of early establishment of enteral nutrition for preventing infection [20-25].

Early establishment of enteral nutrition is an indispensable part of general management.

Conclusion

For chemical injury caused by ingesting acid toilet bowl detergent, the fundamental treatment is general management such as controlling breathing and circulation as well as symptomatic treatment. The early establishment of enteral nutrition is also important as a part of general management. Consequently, regardless of the requirement of radical surgery for stenosis of the esophagus and the stomach, performing enterostomy early should be considered.

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