Esophageal mucosa exfoliation induced by oxalic acid poisoning: A case report

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Abstract. This study reports the case of a 44-year-old woman with oral oxalic acid poisoning. As the illness progressed, the patient exhibited severe metabolic acidosis, large-area esophageal mucosa injury and acute kidney injury, which required dialysis. A guide wire slipped out of position during the process of hemodialysis and moved back and forth in the veins, but was removed successfully by interventional endovascular treatment. However, the patient's esophageal mucosa exfoliated, which lead to severe benign esophageal stenosis and dysphagia. Balloon distention was conducted twice in the upper digestive tract using X-ray location in combination with a dumb-bell bladder and interventional wire. The patient exhibited convulsions, shock, embolism and loss of consciousness while undergoing the second balloon distention procedure. Following symptomatic treatment, the patient eventually remained in a stable condition, the digestive tract expansion procedure was not resumed and a jejunostomy was performed in order to facilitate enteral nutrition, which was administered via the jejunum and had little stimulatory effect on the pancreas. Following various treatments, the patient's condition improved markedly, with renal function returning to normal.

Introduction

Oxalic acid (OA), otherwise known as ethane diacid, is the most simple binary acid. OA exerts marked corrosive and toxic effects, and is commonly used in industry for metal polishing, cleaning and bleaching. OA can cause burns and poisoning, and although industrial cases are extremely rare, OA poisoning is being recognized as an emerging epidemic in certain rural communities as it is a component of widely produced household laundry detergents (1). The toxic effects of OA are primarily described in associated with ethylene glycol poisoning (2), as OA is a metabolite of ethylene glycol. As a final metabolic product, OA is ubiquitously present in plants, fungi and animals. Previously reported cases of isolated OA poisoning involve the consumption of food, medications and plants that contain the compound, such as star fruit and ascorbic acid (3). As OA is the primary component of certain domestic cleaning products, oral OA poisoning cases are not uncommon. Direct intoxication with OA is a relatively frequent occurrence, due to OA being a primary component of some household laundry detergents, and reports of the toxicological effects of OA poisoning in humans are not uncommon, including gastrointestinal effects, hypocalcemia secondary to calcium oxalate crystal formation and renal toxicity (4-6). However, it is relatively uncommon for exfoliation of the esophageal mucosa to be caused by OA poisoning. The present study reports a case of oral OA poisoning in a woman that developed large-area esophageal mucosa injury and acute kidney injury following self-ingestion of OA. The study was approved by the ethics committee of Qilu Hospital of Shandong University (Jinan, China), and informed consent was obtained from the patient.

Case report

A 44-year-old woman oral consumed ~500 ml 70% OA aqueous solution in an attempt at suicide following a domestic dispute. After consuming the OA, the patient immediately experienced severe abdominal pain and vomited repeatedly. The patient was transferred to a local hospital by her family 1 h later. Following a gastric lavage, the establishment of venous access and conventional glucose and saline infusion treatment, the patient was transferred to the regional hospital. On arrival, 5 h after ingestion, the patient underwent hemodialysis to remove remaining toxic compounds and to prevent acute renal failure provoked by calcium oxalate crystal formation and renal toxicity, with a pulse rate of 98 bpm and a blood pressure (BP) of 116/77 mmHg. The peripheral capillary oxygen saturation (SpO_2) of the patient was 97%, her body temperature was 36.5˚C and respiratory rate was 23 breaths/min. Immediate volume resuscitation was conducted, and sucralfate

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Figure 1. Strip of necrotic tissue collected from patient vomit (length, ~25 cm).

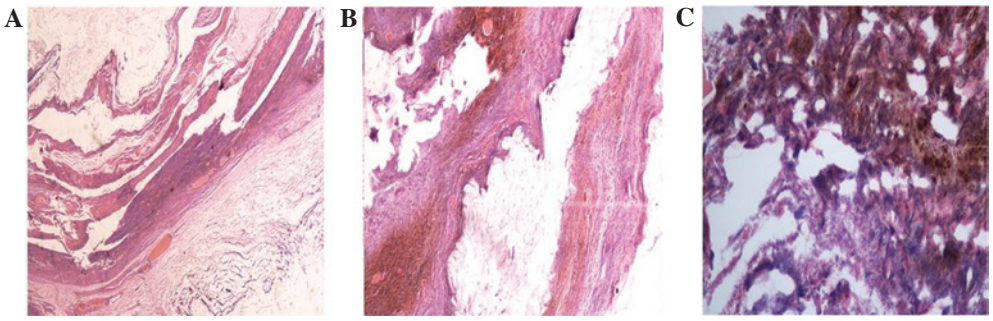

Figure 2. Histopathological examination of the stripped esophageal mucosa revealed (A) fibrinoid necrosis, (B) hemangiectasis and significant (C) inflammatory cellular infiltration composed of neutrophils, lymphocytes and a limited number of eosinophils (stain, hematoxylin and eosin; magnification, x200).

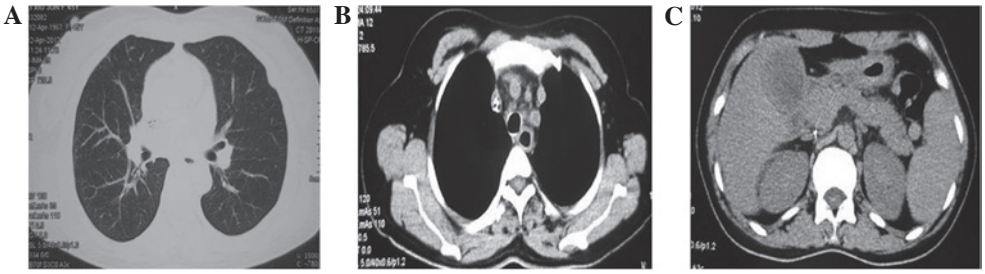

Figure 3. Computed tomography of the chest and abdomen revealed (A) pneumonia, (B) a left-sided superior vena cava and (C) twin pipe line-like high density shadow in the precava and one pipe line-like high density shadow in the postcava, in addition to (B) fatty liver and cholecystitis.

and montmorillonite were administered to provide preventive and therapeutic activity against the acute esophageal mucosal injury induced by OA. The patient continued to vomit violently for the remainder of the day, with blood observed in the vomit on a number of occasions. Over the following days basic normal urine output was maintained. On day 9 following the injury, the patient's condition was significantly exacerbated, the entire body exhibited edema and the urine volume decreased significantly. A computed tomography (CT) scan of the lung indicated an inflammatory response in the right inferior lobar bronchus, in addition to bilateral hydrothorax. The patient continued to experience nausea and vomiting. Eventually, the vomit contained blood clots and a long narrow strip of necrotic tissue was expelled (Fig. 1), which was subsequently identified as the avulsed mucosa of the esophagus. The patient was transferred to Qilu Hospital for further clinical treatment on day 29 following poisoning. During the physical examination conducted during patient admission, the basic vital signs of the patient were as follows: Temperature, 37.9˚C; pulse, 98 bpm; respiratory rate, 22 breaths/min; BP, 116/77 mmHg; and SpO_2, 98%. The patient experienced nausea, mild shortness of breath, obvious generalized edema and crepitations were audible during double-lung auscultation. The levels of alanine transaminase and aspartate transaminase were 172.6 and 52.4 U/l, respectively. There was a gradual elevation in the levels of serum creatinine (SCr, 269 μmol/l) and blood urea nitrogen (BUN, 9.9 mmol/l) levels, with a serum potassium level of 4.36 mmol/l, an erythrocyte sedimentation rate of 66 mm/h and a white blood cell (WBC) count of $11.47x10^9$/l. Furthermore, the patient was positive for urinary protein, hematuria and urine casts. Histopathological examination of the necrotic tissue strip was performed on day 30, and revealed fibrinoid necrosis, significant inflammatory cellular infiltration and hemangiectasis (Fig. 2). The patient underwent pulse methylprednisolone therapy and was treated with potassium-sparing diuretics, hepatic protectants, antioxidants and anticoagulants on the following days, and demonstrated symptomatic improvement. Urine output increased and the SCr level returned to normal. The patient was prescribed antacids and gastric mucosa protectant agents and the upper gastrointestinal symptoms were attenuated. However, the patient's temperature continued to increase and the patient experienced pain in the right ear, right eye and head after day 29. Symptomatic treatment was administered, but produced no apparent effect. CT scanning of the chest and abdomen revealed pneumonia, left superior vena cava, twin pipe line-like high density shadow

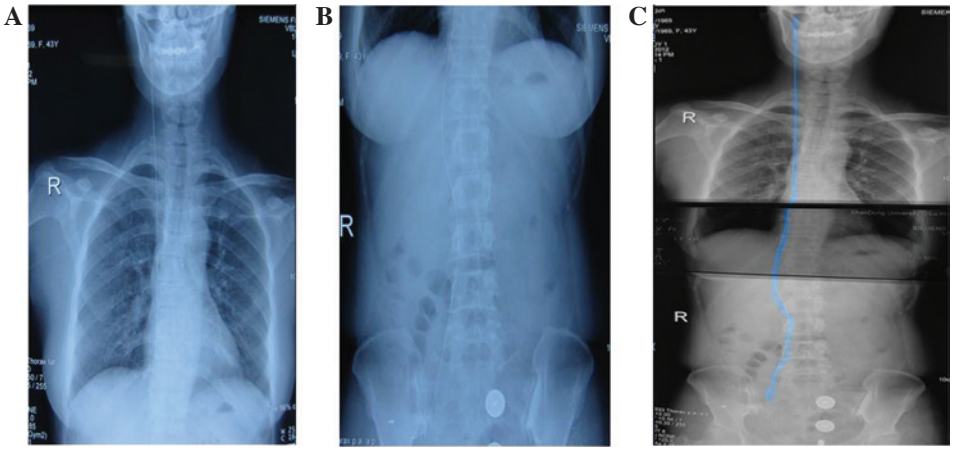

Figure 4. (A and B) A guide wire was present in the precava and the postcava. (A-C) A thick-lined high-density shadow is visible in the precava and the postcava; its upper end is in the retromandibular vein and its inferior extremity is in the right common iliac vein.

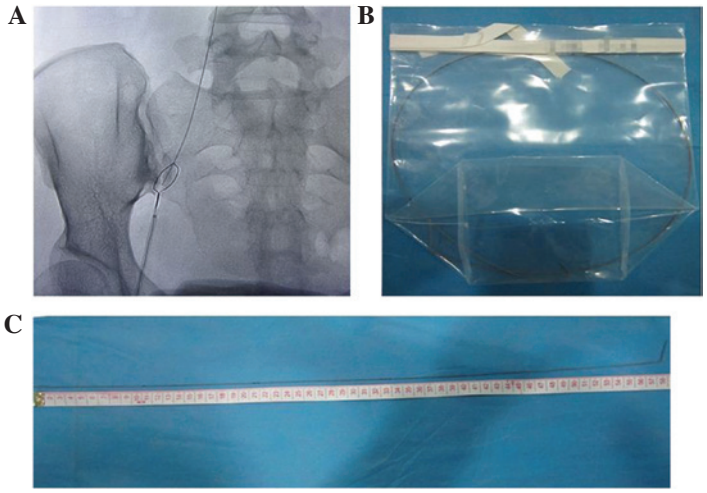

Figure 5. A guide wire of ~59 cm was removed from the femoral vein. (A) X-ray inspection prior to surgery. (B and C) Metal thread removed by surgery.

in the precava and one pipe line-like high density shadow in the postcava, in addition to fatty liver and cholecystitis (Fig. 3). The abdomen and chest X-ray revealed a thick-lined high-density shadow running through the precava and the postcava, the upper end of which extended into the retromandibular vein and its inferior extremity was in the right common iliac vein (Fig. 4). On day 32, the patient accepted intravascular foreign body extraction and a guide wire of ~59 cm was removed from her vein (Fig. 5). Analysis of the abdomen and chest X-ray affirmed that the guide wire, which had remained in the patient's vein since the process of catheter insertion in the regional hospital, was removed. The painful sensations in the patient's right eye and right ear gradually decreased. On day 40, the blood test showed mild normochromic normocytic anemia (hemoglobin, 96 g/l) with hypoproteinemia (serum albumin, 29 g/l), and normal levels of WBCs and platelets. However, the patient continued to exhibit dysphagia, and chemical burns were observed in her pharynx and esophagus via TV fibrolaryngoscope. Furthermore, upper gastrointestinal opacification and gastroscopy confirmed scarring from the chemical burn. Pathological damage included hyperemia of the esophageal mucous membrane, edema, increased susceptibility to hemorrhage and ulcers, and contracture that resulted in stricture of the esophagus. The patient underwent a balloon distention operation in the upper digestive tract, using X-ray location, a dumb-bell bladder and an interventional wire (Fig. 6). This procedure did not improve the dysphagia caused by atrophy of the gastric mucous membrane, as had been expected. The patient underwent a second procedure to stretch the esophagus, via endoscopic dilation, 4 months later. During this procedure the stimuli of dilation provoked convulsions with opisthotonos and sudden lapses of consciousness. The procedure was stopped immediately and effective emergency measures were required. No evident abnormalities were detected in the brain by CT scan, and a brain magnetic resonance imaging (MRI) scan showed that the normal signal of the left parietal lobe of the frontal cortex had been replaced by abnormally high signal intensity, which indicated embolization. Following the administration of dehydrants, cranial hypertension-reducing treatment, neuro-nutritional drugs and parenteral nutrition, the patient regained consciousness and her vital signs stabilized. Subsequently, the patient's cough worsened, and a chest CT scan revealed double pneumonia, pulmonary parenchymal exudation and consolidation in the inferior lobe of the right lung (Fig. 7). Abdominal CT scanning showed no evident abnormalities. The patient was administered

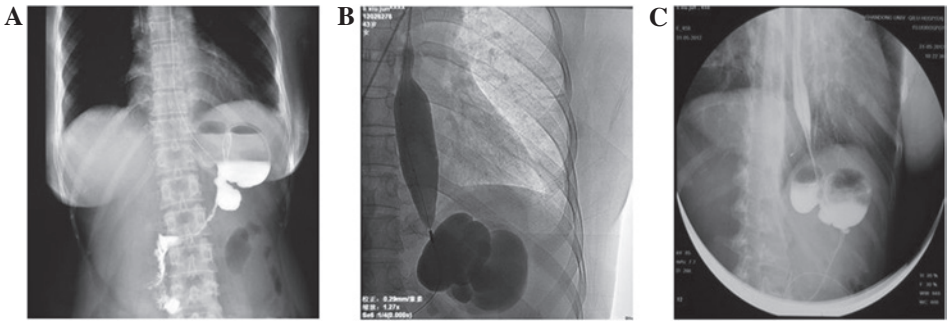

Figure 6. Results of upper gastrointestinal barium examination (A) prior to, (B) during and (C) following the gastric dilatation. The stricture did not observably improve.

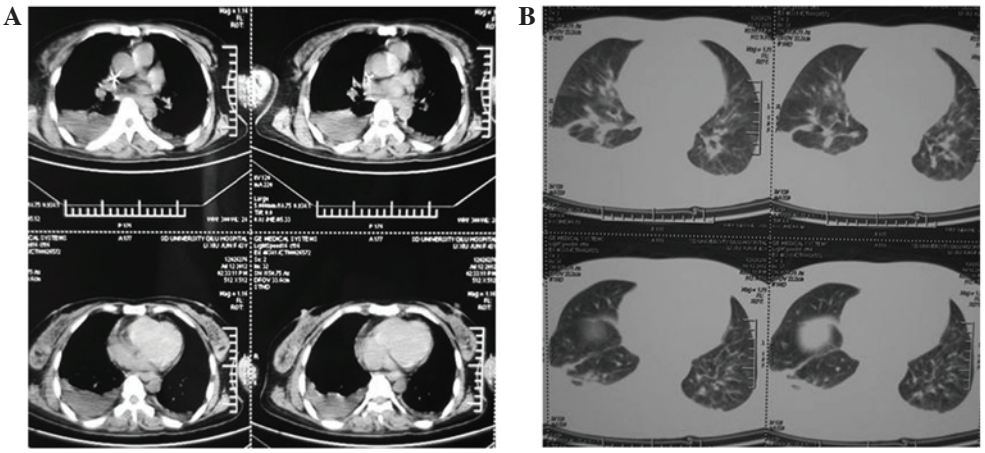

Figure 7. Bilateral pneumonia. Bilateral pulmonary multiple inflammation, pulmonary parenchymal exudation and consolidation in the right lung inferior lobe. (A) Mediastinal window of lung computed tomography (CT) scan. (B) Pulmonary window of lung CT.

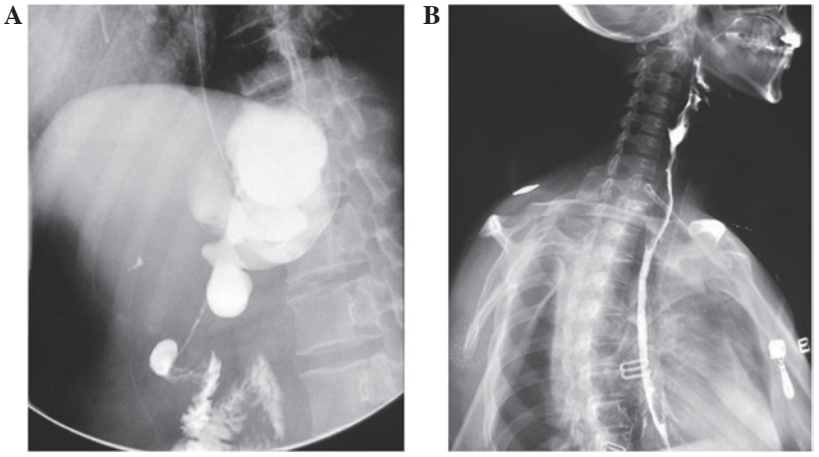

Figure 8. Repeated upper gastrointestinal barium examination revealed a number of severe stenoses in the (A) esophagus, (B) gastric body and sinuses ventriculi, in addition to severe (B) stomach contracture.

anti-inflammatory, anticoagulant and thrombolytic drugs, and her cough gradually decreased. However, the digestive symptoms continued and the repeated upper gastrointestinal barium examinations indicated numerous severe stenoses in her esophagus, gastric body and sinuses ventriculi, in addition to severe stomach contracture (Fig. 8). Metallic stent implantation is considered an effective treatment for patients with such symptoms; however, in the present case the dilating catheter was not able to pass through the stenotic area of the esophagus. Finally, the patient received a percutaneous endoscopic jejunostomy for the management of delayed gastric emptying. A 14 F nutrition tube was inserted between the jejunum and the abdominal wall, which was required to prevent the patient from succumbing to her injuries. Postoperative enteral nutrition later effectively improved the patient's nutritional status, and her general condition improved. The patient was readmitted to the local hospital and subsequently discharged a number of days later.

Discussion

Suicide by the consumption of poison is not uncommon in certain rural areas of China. Numerous cases of poisoning involve the ingestion of diazepam, paraquat and organophosphorus pesticides (7). The prevalence of self-poisoning cases is 315-364 per 100,000 individuals per year (8). In recent years, easily obtained household cleaning products have been increasingly used by individuals attempting to commit suicide. A number of these cleaning products contain oxalic acid (OA) as a primary component. Oral OA is not readily absorbed, and has a bioavailability of 2-5%. OA is excreted in an unmodified form via urine. The lethal dose of oral OA poisoning for an adult human is 15-30 g, although in a previously reported case the ingestion of 5 g OA was sufficient to result in patient mortality (9). OA can cause corrosive damage to the eyes, oral mucosa and gastrointestinal tract. However, following absorption, OA and calcium ions may reaction to form insoluble calcium oxalate crystals. Calcium oxalate precipitation in the renal proximal tubule may cause renal tubular epithelial cell necrosis and kidney failure (10). The pathophysiology of oxalate renal tubular damage includes energy depletion at the cellular level, cell swelling, the inflow of calcium, intracellular acidosis and enzyme activation (11). In addition, oxalate crystals may block the renal tubule, resulting in further renal damage. The clinical response to acute renal failure caused by oxalate poisoning is typically comprehensive symptomatic treatment, with a minority of reported cases requiring dialysis. The renal biopsy results of patients with OA poisoning suggest the occurrence of acute tubulointerstitial nephritis (1). Chronic hyperoxaluria cases exhibit pathological changes associated with interstitial nephritis (12,13). In such cases, the results may be attributable to oxalate itself, although the patients were typically treated with omeprazole, a proton pump inhibitor, simultaneously due to gastrointestinal symptoms. Omeprazole is known to cause interstitial nephritis (14). Absorbed OA can form calcium oxalate, which may cause hypocalcemia directly. However, in the present case there was no biochemical or electrocardiographic evidence of hypocalcemia and the serum calcium level of this patient was normal. However, the recorded state of shock with bradycardia at presentation at the local hospital may have been a manifestation of hypocalcemia. In cases of ethylene glycol poisoning, ethylene glycol is metabolized via aldehyde metabolites to generate OA, which is generally assumed to be the cause of the acute renal failure caused by ethylene glycol poisoning (15). The damage to the central nervous system and heart may be caused by aldehyde metabolites. In the present case, OA caused acute renal failure; however, no significant injury to the central nervous system or cardiovascular system was detected.

In conclusion, OA poisoning may result in acute renal impairment, which may result in mortality. Therefore, the safe and informed use of household cleaners should be encouraged, and household cleaning products containing OA should include clear identification and warnings regarding the potential harm associated with OA poisoning.

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