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Corrosive Injury Due to Edible Vinegar

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Vinegar is a very popular ingredient used in many cuisines. It is also known for its beneficial health, beauty and possible weight-loss properties. The authors report on a patient who presented to the emergency department with unstable vital signs complaining of generalized abdominal pain after ingestion of 450 ml of apple cider vinegar.

We documented a case of corrosive gastrointestinal injury with persistent metabolic acidosis occurring after ingesting apple cider vinegar with an acetic acid concentration of 12~14%. Toxic damage to the liver and kidney were also observed, peaking on post-ingestion day 3. The patient received supportive care and hemoperfusion for three days without much clinical improvement and died in the seventh day of intensive care due to disseminated intravascular coagulation and multi organ failure.

Edible vinegar, when taken in large amounts, is capable of inducing corrosive injuries of the GI tract as well as severe systemic toxicities, such as metabolic acidosis. Safety precautions regarding vinegar deserve more public attention and clinicians also should be astute enough to recognize the potential damage accompanying vinegar ingestion.

Key Words: Vinegar, Intoxication, Corrosive injury, Metabolic acidosis

Introduction

Apple cider vinegar is a brownish-yellow colored vinegar made from cider or apple. It often is sold unfiltered and unpasteurized as a natural product. It is also advertised in the press and internet for treatment of a variety of conditions, including aging, weight loss, hemorrhoids, high blood pressure, arthritis, sore throat, and indigestion[1]. Because of its acidity however, apple cider vinegar may be very harsh, even burning, to the throat and gastrointestinal tract if taken straight without dilution. Edible vinegar in general contains 6~7% of acetic acid, but double concentrated vinegar, with twice as much acetic acid, has also become available to the public. Acetic acid in the concentration of 12~14% is capable of inducing gastrointestinal injuries when taken in large amounts. We have encountered a case in which a patient expired after ingesting a large amount of double concentrated edible vinegar, due to gastrointestinal hemorrhage and persistent metabolic acido-

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sis, and would like to report this case, with review of existing literature.

Case

An 82 year old female presented to the emergency department (ED) complaining of whole abdominal pain and tremor of both hands. Her son explained that he had parted with her mother about five hours ago to go to sleep. He woke up to the sound of the patient groaning in pain in the living room, where he discovered her rolling on the floor, with an empty bottle of vinegar rolling nearby. He recalled that the bottle of vinegar was a double concentrated apple cider vinegar, with an acetic acid concentration of 12-14%. An interview with the patient confirmed ingestion of roughly 450 ml of the undiluted vinegar, five hours prior to visiting the ED. A sour scent was indeed noted around the patient. She confessed to having planned suicide, due to chronic lower abdominal pain and dysuria symptoms that had been tormenting her for the last three months. A dipstick test done on the remaining drops of vinegar the son had brought along suggested a pH value far below 5.

She was negative for any history of hypertension, diabetes, tuberculosis, thyroid diseases, hepatitis, nor any surgeries. Although quite agitated and uncooperative, she was communicable. Physical examination of the patient was unrevealing except for dehydrated tongue, decreased skin turgor, and tenderness in the epigastric and periumbilical area. Bowel sound was hypoactive and no peritoneal sign was observed. No neurologic abnormality was observed. Breathing sounds were clear, and heart sound was regular, with no definite murmur. On presentation, her vital signs were blood pressure 70/40 mmHg, pulse rate 80 beats/minute, respiration rate 24 breaths/minute, and body temperature 36°C.

The patient was immediately started on fluid resuscitation, with emergency blood and urine samplings, electrocardiogram (EKG), and simple x-rays of the chest and abdomen.

The EKG showed a normal sinus rhythm. Arterial blood gas analysis (ABGA) taken at room air thirty minutes after presentation revealed a pH of 7.354, PCO_2 of 27.4 mmHg, PO_2 of 216 mmHg, HCO_3^- of 14.9 mmol/L, base deficit of -10.7 mmol/L, and SaO_2 of 99.4%. Initial blood sampling results are as follows: Hb 13.8, Hct 41.5%, WBC 14600, PLT 320000, $Na^+/K^+/Cl^-$ 136/3.8/104 mmol/L, AST/ALT 76/26 IU/L, PT 102%, INR 0.99, aPTT 29.2 sec, BUN/Cr 14.9/1.00 mg/dL, amylase 275 IU/L, osmolality 298 mOsmol/kg, LDH 839 IU/L, CPK 82 IU/L. UA was clear, with pH of 5.5 and SG of 1.004. Urine drug screening was positive for acetaminophen and opiate. The patient had taken medications for common cold during the previous three days.

Initial x-rays of the chest and neck were unrevealing. Simple abdomen suggested mild paralytic ileus, with edema of the gastric and intestinal walls (Fig. 1).

We could not rule out the possibility of corrosive gastrointestinal tract injury due to vinegar ingestion, and computed tomography (CT) scan of the abdomen was ordered for further work up. In the meantime, the patient' s blood pressure was fluctuating between 90/60 mmHg and 70/40 mmHg. Proton pump inhibitor had been injected intravenously, but the patient was still complaining of abdominal pain. Follow up abdominal physical examination however, had not changed. Roughly three liters of normal saline had been infused by three hours, but hypotension persisted, with deterioration of the patient' s condition.

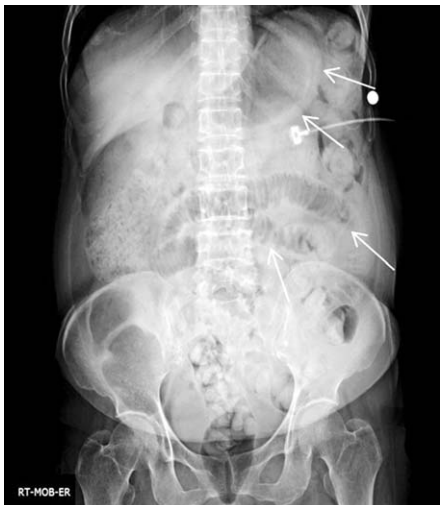

Fig. 1. Simple abdomen. Arrows indicate edema of the gastric and intestinal walls.

Several hours after arrival to ED, the patient started complaining of dyspnea, and SaO_2 gradually started to fall. Eventually SaO_2 fell to 85%, and the patient was showing an acute decline in mental status. Fifteen minutes later, the patients' rhythm had degenerated to bradycardic pulseless electrical activity (PEA), and cardiopulmonary resuscitation (CPR) was initiated. Return of spontaneous circulation (ROSC) was observed after ten minutes of CPR. ABGA sampled before initiating CPR showed an exacerbation in metabolic acidosis, with pH of 7.062, PCO_2 of 41.8 mmHg, PO_2 of 171 mmHg, HCO_3^- of 11.6 mmol/L, base deficit of -18.7 mmol/L, and SaO_2 of 98.7%. Follow up blood sampling done with the start of CPR revealed the following results: HB 12.6, Hct 37.5%, WBC 27300, $Na^+/K^+/Cl^-$ 140/3.5/111 mmol/L, BUN/Cr 22.8/1.30 mg/dL, AST/ALT 93/37 IU/L, amylase 911 IU/L, lipase 130 IU/L, total bilirubin 0.99 mg/dL, osmolality 315 mOsmol/kg, PT 62%, INR 1.38, aPTT 36.6 sec. Follow up urine analysis further revealed blood and protein on dipsticks, as well as RBCs and granular casts. Urine SG was 1.025, and pH was 5.5. Central venous pressure after ROSC was checked at 10.5 mmHg.

Fresh hematochezia, in total amounting up to 800 cc, was noted oozing shortly after achieving ROSC. We had not inserted a nasogastric tube previously, as corrosive injury was on our differential diagnosis. At this point however, a nasogastric tube was inserted to decompress the stomach, and fresh blood was also drained through the nasogastric tube.

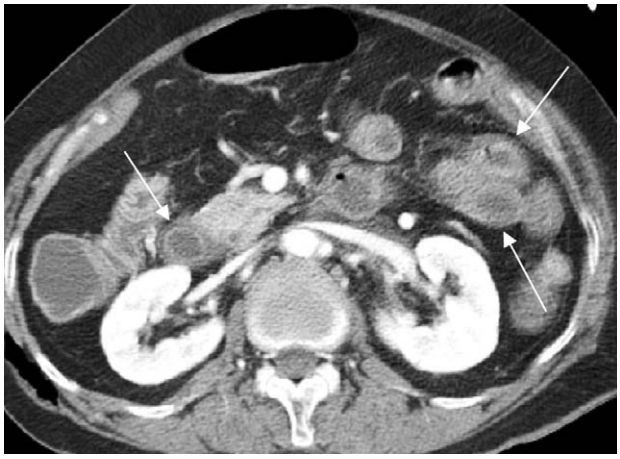

Fig. 2. Abdominal CT. Arrows indicate edema of small intestine and colon walls.

Abdominopelvic CT scan taken after stabilization revealed edema of the stomach body all the way down to the intestines, but no signs of bowel perforation were observed (Fig. 2).

Eight hours after presentation to the ED, metabolic acidosis was persistent as ever, with pH 7.26, PCO_2 36.7 mmHg, PO_2 139 mmHg, HCO_3^- 16.4 mmol/L, BE -10.6 mmol/L, and SaO_2 98%. The patient was admitted to the intensive care unit for continuous renal replacement therapy.

The patient received continuous renal replacement therapy for three days, without much improvement. She was provided with supportive measures including hydration and antibiotics, and expired on the seventh day of admission due to disseminated intravascular coagulation and multiorgan failure.

Discussion

Vinegar is an acidic liquid produced through fermentation of a variety of sources including carbohydrates and sugars. Ethanol is first produced as a result of fermentation of sugars. Ethanol is then oxidized to acetic acid by the acetic acid bacteria. Natural vinegars, in addition to acetic acid, may also contain small amounts of tartaric acid, citric acid, and other acids. Vinegar has been used since ancient times and is an important element in European, Asian, and other cuisines.

The pH of table vinegar ranges from 2.4 to 3.4, and may be higher if diluted[1]. The acetic acid concentration typically ranges from 4% to 8% by volume for table vinegar and up to 18% for pickling vinegar[1]. Many vinegar products available in market however, show inconsistency and inaccuracy in labeling product descriptions and instructions.

Hill et al[1] examined various apple cider vinegar products available in the market, and noted that there were strongly acidic products with pH ranging from 2.9 to 5.7. The concentration of acetic acid also was diverse, ranging from 1.04% to 10.57%. There were significant discrepancies between these findings and descriptions stated on product labels.

Considerable variability was also found between the brands in pH, component acid content, and label claims[1]. The vinegar our patient had ingested had also been advertised in the market as an alkali, even though the pH, measured with dipstick, suggested an acidity far lower than 5. While it may be true that most edible apple cider vinegar would be safe to drink, overdosage and inappropriate use of some highly concentrated acidic vinegar will indeed result in toxic symptoms.

Ingestion of acidic household products may result in corrosive injuries of the gastrointestinal tract. The severity would depend on the particular substance and its' acidity, concentration, and osmolarity, as well as the amount ingested, duration of gastrointestinal tract contact with the offending agent, the presence of food material within the gastrointestinal tract, and the functioning of the pyloric sphincter[2].

Chung[3] reported of a 39-year-old woman who drank one tablespoon of household rice vinegar containing 4~5% of acetic acid in order to soften crab shell stuck in her throat. Endoscopy revealed inflammation of the oropharynx and second-degree caustic injury of the oesophagus extending to the cardia, confirming that vinegar could cause ulcerative injury to the oropharynx and oesophagus. The patient in our case had ingested 450 ml of double concentrated vinegar with an acetic acid concentration of 12-14% on an empty stomach. The pH, as measured with dipstick had suggested an acidity lower than 5. She had not eaten well the previous few days due to common cold symptoms. She had presented to the ED about five hours after ingestion without vomiting even once, and we believe she would have sustained a significant degree of corrosive injury to the gastrointestinal tract.

Such corrosive injury due to acidic substances may also rarely involve the lower gastrointestinal tract in addition to the upper gastrointestinal tract[4]. Edema of the stomach and bowel wall was evident in simple x-ray and CT scan of our patient. Also, in addition to hematochezia, nasogastric tube insertion led to drainage of fresh blood, suggesting corrosive injury to both the upper and lower gastrointestinal tracts. Furthermore, serum amylase checked seven hours after vinegar ingestion had risen to 911 IU/L, suggesting pancreatitis. BUN/Cr level was 22.8/1.30 mg/dL, and we believe this rise in amylase level was due to direct injury by the vinegar rather than impaired renal function.

Vinegar ingestion may also cause systemic toxicity in addition to direct gastrointestinal tract injury. Davids et al[5] reported cases of highly concentrated acetic acid solution ingestion in three women, who suffered damage to the esophagus and the stomach, with progressing respiratory and renal insufficiency as well as haemolysis. The patient in our case was no exception. Metabolic acidosis was present on presentation, and the patient was dehydrated with inadequate urine output. Reports of metabolic acidosis ensuing vinegar ingestion have not yet been published. However, metabolic acidosis is a well described systemic presentation following poisoning with acidic substances. Similarly, the patient in our case is presumed to have been hyperventilating to compensate for metabolic acidosis. Also, metabolic acidosis most likely would have accounted for the change in mental status, as well as breakdown of the gastrointestinal wall integrity resulting in dehydration, ultimately leading to a decrease in effective circulating volume and a state of shock. We presume these various mechanisms have acted in concert, eventually resulting in cardiac arrest.

Kamijo et al[6] performed an autopsy in a patient who had expired following ingestion of concentrated (90%) acetic acid, noting corrosive injuries in the upper gastrointestinal tract and massive hepatic necrosis in a periportal distribution without significant inflammation. A direct effect of the noxious agent on hepatocytes involving the portal circulation was suggested[7]. We believe the impairments in renal and liver function in our patient, as demonstrated by the elevated BUN/Cr and liver function tests in samples taken after the cardiac arrest, may be attributed to both the direct injury caused by vinegar, as well as indirect complication of the cardiac arrest.

No known antidotes exist to date. However, Boseniuk et al[7] have suggested treatment strategies

in a report on a female patient who attempted suicide by drinking 400 ml of 25% acetic acid. They emphasized the importance of immediate treatment of the haemolysis and detoxification with plasma separation with the immediate substitution of blood and clotting factors. After acid absorption, plasma separation is claimed to be the quickest and most effective way of detoxication and removal of the products of haemolysis. In cases of severe haemolysis, exchange transfusion may also be necessary. In addition, careful management of the acid-base status is recommended. Disseminated intravascular coagulation or anemia may develop, and low dose heparin, erythrocyte transfusion and antithrombin III substitution may be necessary. Haemodialysis was recommended in secondary renal failure.

When acute intoxication has been treated, attention should be paid to fluid management and calorie intake. Care must be taken to exclude injury or stricture of the esophagus or stomach. Boseniuk et al[7] claim that despite initial deterioration, the initiation of therapy may lead to rapid improvement in circulation and renal function. The patient in our case had been hydrated with a total of three liters of normal saline after arrival to ED, before cardiac arrest occurred. Hypovolemic shock is a potentially lethal complication that should be anticipated in vinegar ingestion cases with high possibility of corrosive injury to the gastrointestinal tract. Therefore, in addition to vital sign monitoring, attention should be paid to the adequacy of urine output and central venous pressure to maintain appropriate effective circulating volume. Such aggressive management may help prevent renal injury and secondary complications of shock, and also improve the prognosis of the patient.

Edible vinegar is safe to drink in most cases. Ingestion of large amounts however, may induce direct corrosive injury to the gastrointestinal tract in addition to fatal systemic manifestations, such as refractory metabolic acidosis, renal impairment, and hypovolemic shock[8]. This calls for intensive monitoring of systemic circulation and aggressive intervention of early severe symptoms. Equally important would be efforts to provide accurate information regarding the product and instructions to the public. Thus we could reduce damage caused by inappropriate use of vinegar.

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