Accidental ingestion of 35% hydrogen peroxide

Sean Pritchett MD\textsuperscript{1}, Daniel Green MD\textsuperscript{2}, Peter Rossos MD\textsuperscript{2}


Hydrogen peroxide is a commonly used oxidizing agent with a variety of uses depending on its concentration. Ingestion of hydrogen peroxide is not an uncommon cause of poisoning, and results in morbidity through three main mechanisms: direct caustic injury, oxygen gas formation and lipid peroxidation. A case of a 39-year-old man who inadvertently ingested 250 mL of unlabelled 35% hydrogen peroxide intended for natural health use is presented. Hydrogen peroxide has purported benefits ranging from HIV treatment to cancer treatment. Its use in the natural health industry represents an emerging source for accidental poisonings.

**Key Words:** Caustic injury; Hydrogen peroxide; Portal vein gas

Hydrogen peroxide is a commonly used oxidizing agent with a variety of uses depending on its concentration. Although historically it has been used for medicinal purposes such as wound irrigation, its toxic effects preclude its routine use. Ingestion of hydrogen peroxide is not an uncommon cause of poisoning, and results in morbidity through three main mechanisms: direct caustic injury, oxygen gas formation and lipid peroxidation. The growing naturalpathic health industry has promoted the use of hydrogen peroxide in treating a wide variety of medical conditions. A case of a 39-year-old man who inadvertently ingested 250 mL of unlabelled 35% hydrogen peroxide intended for natural health use is presented.

**CASE PRESENTATION**

A healthy 39-year-old man presented to the emergency department following the accidental ingestion of one-half of a 500 mL container of 35% hydrogen peroxide. The hydrogen peroxide was in an unlabelled container in a friend's refrigerator and was mistaken for water. Immediately after ingesting the hydrogen peroxide, he realized it was a caustic substance and drank 500 mL of water to induce vomiting. A small amount of hematemesis was noted. He experienced mild epigastric pain without shortness of breath or oropharyngitis.

On arrival to the emergency department, he was hemodynamically stable and in no respiratory distress. The oral cavity and oropharynx were slightly erythematous and the abdominal examination revealed mild epigastric tenderness but no distension, and normal bowel sounds.

Intravenous (IV) fluids and IV pantoprazole, 40 mg twice per day, were initiated. Bloodwork was normal except for mild leukocytosis; the chest x-ray was normal. Esophagogastroduodenoscopy was performed to assess the severity of the caustic injury. This revealed a normal oral cavity and normal esophagus. The stomach revealed grade 2 diffuse caustic mucosal injury of the entire stomach, including the cardia, characterized by superficial erosions with exudates and edema (Figure 1). The duodenum only revealed a few scattered superficial erosions in the first and second parts (Figure 2).

A computed tomography scan of the abdomen revealed portal venous gas (Figure 3), extravascular gas in the region of the gastrohepatic ligament and possible pneumatosis of the stomach and duodenum.

On subsequent history, the patient indicated that the 35% hydrogen peroxide was used for natural health purposes, including aiding in digestion. It was kept in the refrigerator to help prolong its shelf-life. The patient recovered well, and was discharged from hospital on the third day without complications.

**DISCUSSION**

Hydrogen peroxide is a clear, colourless, odourless oxidizing agent found in concentrations ranging from 3% to 90%. Three per cent solutions are used as common household disinfectants, and are therefore a common source of accidental poisonings, especially in children. In one study (1) of more than 95,000 toxic exposures reported to a poison control centre over three years, 0.34% were due to hydrogen peroxide and of these, 60% occurred in children younger than six years of age, and 85% occurred through ingestion. Fatalities in both adults and children have been reported with ingestion of 35%
hydrogen peroxide (1). A concentration of 35% is techni-
cal grade or food grade hydrogen peroxide, and can be pur-
b�ed in many health food stores because it is reputed to have 
multiple health benefits including being useful in the treat-
ment of HIV, cancer, chronic obstructive pulmonary disease
and Alzheimer’s dementia (3). Higher concentrations of up to
90% are used as rocket fuel.

Hydrogen peroxide is relatively unstable and will rapidly
decompose, through an exothermic reaction, into water and
oxygen in the presence of alkali, metals and the enzyme
catalase, which is found in mucous membranes, liver, kidney,
red blood cells and bone marrow (1). There are three main
mechanisms of toxicity from hydrogen peroxide: caustic injury,
oxogen gas formation and lipid peroxidation (1).

Ingestion of 3% hydrogen peroxide usually results in only
mild gastritis, unless ingested in large quantities (2). Ingestion
of more concentrated forms including 35% hydrogen peroxide
can produce severe gastrointestinal erosion, ulceration and
perforation (1). Hydrogen peroxide enteritis with 3% solution
can cause instant bubbling on the mucosal surface followed by
a whitening of the mucosa termed the ‘snow white’ sign (4).

This phenomenon is thought to be secondary to absorption of
hydrogen peroxide into the epithelial interstices and capillar-
ies, and the subsequent blanching caused by formation of
microbubbles of molecular oxygen (4). Caustic injury can also
occur in the airway if aspirated or inhaled, and can lead to sub-
glottic stenosis and laryngospasm requiring intubation and
mechanical ventilation (1).

The volume of oxygen liberated from the decomposition of
hydrogen peroxide can be considerable, with 30 mL of
35% hydrogen peroxide yielding 3.5 L of oxygen (1). This
rapid release of oxygen can lead to hollow viscous perforation
(1). If the amount of oxygen exceeds the maximum solubility
in blood, it may lead to gas embolism particularly in the portal
venous system, gastric wall (5) and brain (6). Gas embolism in
the brain presents similar to ischemic stroke and has been
successfully treated with hyperbaric oxygen therapy (6).

Lipid peroxidation from ingested hydrogen peroxide can
lead to direct cytotoxic effects (1).

Despite the potential for severe injury from hydrogen per-
oxide, it has been used extensively throughout medical history.
One per cent to 3% solutions were used for wound irrigation
and as rectal enemas, but gas embolism and rectal ulceration
limited its use (1). A dilute IV version of hydrogen peroxide
was administered for the treatment of AIDS, but resulted in
hemolysis and eventually death (1). The use of hydrogen per-
oxide as a natural remedy is generally based on the premise
that it is a concentrated source of potential oxygen. It is
administered orally, through IV injection and through inhalation,
for conditions such as COPD to help oxygenate the lung
and in diabetes mellitus to improve glucose utilization (3).
Possible mechanisms of action include vasodilation, strength-
ening the immune system and stimulating prostaglandin
synthesis (3). Thirty-five per cent hydrogen peroxide is
available from commercial health food stores, is typically
stored in a refrigerator or freezer to retard decomposition, and
is diluted to achieve the desired concentration. Despite the
little evidence on its benefits and well-documented accidental
poisonings, there is continued use and availability of hydrogen
peroxide for such health applications.

Management of hydrogen peroxide exposures depends on
the severity of ingestion and includes airway management,
frequent monitoring, and diagnosis and therapy of associated complications. Due to the rapid decomposition, gut decontamination is not required. In the present case, the immediate ingestion of 500 mL of water following the hydrogen peroxide may have prevented more serious oropharyngeal and esophageal injury. Both X-ray and computed tomography are useful for ruling out viscous perforation and air embolism. Endoscopy should be performed in all patients (except if perforation is suspected or the patient is hemodynamically unstable) because signs and symptoms do not consistently correlate with the extent of injury, making endoscopy the only reliable method to assess for injury (7). Endoscopic assessment of injury severity within 24 h can be used to risk stratify patients because the severity of mucosal injury strongly correlates with the risk of death and systemic complications (8). Strictures occur most frequently in the esophagus and generally develop between six and 12 weeks (8). Consequently, routine follow-up endoscopy may be indicated in patients with esophageal injury. One trial (2) reported using IV cimetidine when there was evidence of gastric erosions or ulcers, but none have documented using proton pump inhibitors. In the present case, IV pantoprazole, 40 mg twice a day, was used, and the patient had resolution of all abdominal pain symptoms. For cerebral gas embolism, hyperbaric oxygen therapy has been used successfully (6).

Most exposures to hydrogen peroxide are with the 3% solution of hydrogen peroxide, which results in little or no morbidity. However, the storage and use of 35% hydrogen peroxide for natural health benefits results in an emerging source for more serious ingestions. Thirty-five per cent hydrogen peroxide can be lethal when ingested, and needs to be treated with caution and stored appropriately. Public awareness and regulation of the use of this substance is required.

REFERENCES