HYDROGEN PEROXIDE

Hydrogen peroxide, H₂O₂, is a clear, colorless, odorless liquid that is available in a dilute form of 3% for household use and in an industrial concentrated form of greater than 30%. It is used in the home as an antiseptic, disinfectant, mouth gargle and enema. It may also be applied to remove cerumen and bleach hair. Industrial uses of hydrogen peroxide include the bleaching and cleaning of textiles, wool, fur and paper, as well as in the production of foam rubber and rocket fuel. (1)

Hydrogen peroxide is also used illegally in a 35% solution as a milk sterilizer and to preserve and lengthen the shelf life of refrigerated perishables. (2) Recently “food grade” hydrogen peroxide has become available in health food stores and by mail, where it is marketed as “hyperoxygenation therapy” with claims of beneficial results in patients with AIDS and cancer. However, the FDA has not approved the use of hydrogen peroxide for these conditions. (1,2)

TOXICITY

Hydrogen peroxide is a powerful oxidizing agent and has two mechanisms of toxicity: gas formation and local tissue injury. When hydrogen peroxide interacts with tissue catalase the products released include oxygen, water and heat. One ml of 3% hydrogen peroxide can release 10 ml of oxygen. Local toxicity is mediated by the hydroxyl free radical of hydrogen peroxide and also by the heat released during the reaction. (1)

The potential toxicity of hydrogen peroxide is dependent on the route of exposure. The majority of exposures are oral ingestions. Ingestions of 3% hydrogen peroxide are generally benign and can cause nausea, vomiting and diarrhea. Ingestions of more concentrated solutions (35%) can cause severe irritation, inflammation and burns of the alimentary tract; hollow organ distention and rupture; seizures, cerebral infarcts and gas embolism; and finally respiratory arrest. Inhalation of hydrogen peroxide vapors, mist or aerosol can cause upper airway irritation, inflammation, hoarseness, shortness of breath, burning and chest tightness. Higher concentrations can cause severe mucosal congestion of the trachea and bronchi. While dermal contact of dilute solutions is mildly irritating to the skin and mucous membranes, contact with higher concentrations can be severely irritating and corrosive. Such contact can temporarily bleach the skin or cause burns and blisters. (3)

Morbidity and mortality secondary to hydrogen peroxide exposure is rare. In 2001 there were 7,934 exposures to hydrogen peroxide, 44% of which involved children less than six years old. Only three had a major outcome defined as a life-threatening sign or symptom, and there were no reported deaths. (4) Dickson et al. performed a retrospective study of all hydrogen peroxide exposures reported to a regional poison control center over a thirty-six month period and found 325 exposures. Over 70% of the cases involved pediatric patients, and over 80% were ingestions. Ocular and dermal exposures both occurred at a rate of approximately 6% each. There was a trend toward less severe outcomes in patients with exposures to concentrations of less than 10%. The one death that occurred in a pediatric patient was secondary to a related gas embolism. (5)

The first reports of hydrogen peroxide toxicity came from its use in closed spaces or body cavities. Oxygen embolization to mesenteric and portal veins has been reported in infants following intestinal irrigation to remove meconium plugs. These embolisms have been associated with gangrenous and perforated bowel. (6) Facial emphysema has also been described following irrigation with hydrogen peroxide during endodontic treatment. (6) Additionally, there was a
report of a near fatal systemic oxygen embolism secondary to
a wound irritation with hydrogen peroxide.

There are multiple case reports in the literature of pediatric deaths following hydrogen peroxide ingestion. One case report describes a three year old who ingested an unknown amount of 40% hydrogen peroxide. The child rapidly developed excessive salivation in the mouth, loss of consciousness, pulselessness and cyanosis before dying. An autopsy revealed pulmonary edema and small gastric erosions, with the probable cause of death listed as a mechanical obstruction of the respiratory tract. Another case report describes a one year old who ingested an unknown amount of 30% hydrogen peroxide. The child developed secretions in the mouth and nose with subsequent fatal respiratory failure within one hour. Cima et al. reported another pediatric death in a sixteen-month old child who ingested approximately eight oz. of 3% hydrogen peroxide and was found dead in bed. The probable cause of death given was respiratory failure.

There have been two additional reported cases of gastric erosions and ulcers secondary to hydrogen peroxide ingestion. One occurred in a twenty-six month old child who ingested a mouthful of 35% hydrogen peroxide. The other involved a three year old that ingested two-four oz. of 3% hydrogen peroxide. Both patients had the erosions and ulcers diagnosed with upper endoscopy.

Giberson et al. reported on the first survivor of a 35% hydrogen peroxide ingestion. A 33 year old unintentionally ingested one pint of hydrogen peroxide. She vomited, collapsed and began having tonic/clonic seizures. She was intubated after being assessed to have a Glasgow Coma Scale of six, and medicated with diazepam, naloxone, thiamine and dextrose. Upper endoscopy showed diffuse esophageal erythema and gastritis; a head CT revealed bilateral cerebral hemisphere swelling. She was ultimately discharged home with residual upper and lower extremity weakness, which was greater on the right than the left.

There have also been multiple case reports in the literature of oxygen embolisms after hydrogen peroxide ingestion. Rackoff et al. reported the first portal venous embolism in a two year old child who ingested an unknown amount of 3% hydrogen peroxide. An abdominal x-ray showed air within the liver and the child was discharged home after observation. Luu et al. later reported on an adult patient who ingested two mouthfuls of 35% hydrogen peroxide and also had x-ray evidence of air in the portal venous system. Christensen et al. reported on a fatal case of oxygen embolism after hydrogen peroxide ingestion. A two year old child became unresponsive, cyanotic and bradycardic after ingesting four-six oz. of 35% hydrogen peroxide. A chest x-ray showed air in the right ventricle, the mediastinum and the portal venous system.

More recently there have been reports of arterial cerebral gas embolisms. The first reported case was in an eighty-four year old male who ingested 30 ml of 35% hydrogen peroxide as a remedy for arthritis. He presented with multiple neurological deficits consistent with the MRI, which showed multiple cerebral and cerebellar infarcts in the anterior, middle and posterior vascular territories. Ijichi et al. also describe a case of cerebral artery embolism following ingestion of 35% hydrogen peroxide. Mullins, et al. reported the first case of successful treatment of arterial cerebral gas embolism with hyperbaric oxygen therapy. The patient received hyperbaric treatment approximately twenty hours post ingestion. The treatment lasted for forty-nine minutes and resulted in rapid and complete resolution of the neurological deficits.

**LABORATORY/TESTS**

Routine laboratories for hydrogen peroxide ingestion depend on the presentation of the patient, but may include a complete blood count, glucose and electrolytes levels, arterial blood gas saturation, upright chest x-ray, and an abdominal series. There is a screening test for the presence of hydrogen peroxide if the ingestant is unknown and hydrogen peroxide is suspected. Add one drop of 15% titanium chloride to an acidified mixture of equal parts gastric contents and ethyl ether. If the aqueous layer turns yellow to deep orange it is positive for peroxide. This assay is only sensitive for fresh gastric contents and may be read as a false negative if the hydrogen peroxide has degraded or has been completely absorbed by the stomach.

**TREATMENT**

Treatment for hydrogen peroxide exposure begins with basic decontamination. Clothing that may have been exposed should be removed. Exposed skin should be flushed with water for at least five minutes, then washed with soap and water. If the eyes have been exposed they should be irrigated with saline for at least fifteen minutes. If the substance has been ingested, emesis should not be induced. Asymptomatic patients who have ingested small amounts of 3% hydrogen
peroxide may be observed at home. Symptomatic patients with persistent vomiting, bloody emesis or abdominal discomfort should be referred to the emergency department. For a conscious patient, milk or water may be recommended to dilute the hydrogen peroxide, although there are no studies to support this practice. Activated charcoal should not be given as it will not absorb hydrogen peroxide and its presence will interfere with potential upper endoscopy. A nasogastric tube may be placed for gastric decompression and hyperbaric oxygen therapy may be considered in patients with severe embolization. Consultation with a medical toxicologist and/or regional poison control center is also recommended.

CONCLUSION

While exposures to household dilutions of hydrogen peroxide may present few problems, the exposure of any tissue to more concentrated industrial solutions may cause significant toxic effects that pose serious health risks.

Lisa Cabral, MD
Boston Medical Center

REFERENCES

2) Henry MC: Clinical Toxicology 1996; 34: 323-327.
3) Agency for Toxic Substances and Disease Registry (www.atsdr.cdc.gov)