American Journal of Emergency Medicine 35 (2017) 809.e5–809.e8

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Hyperbaric oxygen therapy for the prevention of arterial gas embolism in food grade hydrogen peroxide ingestion☆

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ARTICLE INFO

Article history:
Received 2 December 2016
Accepted 11 December 2016

Keywords:
Hyperbaric oxygen
Venous gas embolism
Arterial gas embolism
Hydrogen peroxide

ABSTRACT

Food grade hydrogen peroxide ingestion is a relatively rare presentation to the emergency department. There are no defined guidelines at this time regarding the treatment of such exposures, and providers may not be familiar with the potential complications associated with high concentration hydrogen peroxide ingestions. In this case series, we describe four patients who consumed 35% hydrogen peroxide, presented to the emergency department, and were treated with hyperbaric oxygen therapy. Two of the four patients were critically ill requiring intubation. All four patients had evidence on CT or ultrasound of venous gas emboli and intubated patients were treated as if they had an arterial gas embolism since an exam could not be followed. After hyperbaric oxygen therapy each patient was discharged from the hospital neurologically intact with no other associated organ injuries related to vascular gas emboli. Hyperbaric oxygen therapy is an effective treatment for patients with vascular gas emboli after high concentration hydrogen peroxide ingestion. It is the treatment of choice for any impending, suspected, or diagnosed arterial gas embolism. Further research is needed to determine which patients with portal venous gas emboli should be treated with hyperbaric oxygen therapy.

1. Introduction

Hydrogen peroxide (H_2O_2) is an easily accessible, clear, odorless liquid. It is used for a variety of household and industrial purposes. In low concentrations of 3%–9%, it has been used as an antiseptic or hair bleaching agent. In high concentrations, often termed "food grade" of 35% or higher, it has been used as an industrial-strength cleaning agent [1]. Additionally, some groups have advocated the consumption of highly concentrated H_2O_2 with claims of anticipated health benefits including the treatment of cancer, the reduction of bacterial burden, and others [2,3]. Contrary to these purported benefits, prior literature and case reports have demonstrated adverse effects from the ingestion of hydrogen peroxide related to its oxidizing and tissue-damaging effects (lipid peroxidation) and the vascular gas emboli liberated after ingestion [4,5]. Patients ingesting food grade hydrogen peroxide have been shown to develop varying degrees and severity of esophagitis, gastritis, gastric ulcers, perforated visci, pneumatosis, pneumobilia, venous portal gas and arterial gas emboli (AGE) [1,6]. The latter is associated with higher morbidity and mortality from multiple potential causes such as acute stroke, coronary artery occlusion, and ischemia in the vascular beds of other vital organs [7]. (See Figs. 1–6.)

Hydrogen peroxide (H_2O_2) is converted by the mucosal surface and RBC enzyme catalase to water and oxygen. It has been estimated that 1 mL of 35% H_2O_2 could liberate more than 100 mL of gaseous oxygen; thus even a tablespoon of food grade H_2O_2 would rapidly liberate 1500 mL of gaseous oxygen into the gastric mucosa if swallowed [7]. Rapid liberation of oxygen may result in gastric distention, emesis, and potentially perforation of viscera. Gastrointestinal absorption into the mesenteric and portal venous system prior to its decomposition in the stomach or intestine may result in the liberation of oxygen and venous gas emboli [6]. The presence of venous gas increases the risk for AGE via a right to left intracardiac shunt such as a patent foramen ovale (PFO). The overall incidence of PFO at autopsy has been found to be 27.3% in the general population [8]. Additionally, excessive venous gas could overwhelm the pulmonary vascular filter, transposing gas emboli to the arterial circulation with devastating consequences for patients. Indeed, AGE from H_2O_2 has been reported in multiple cases to cause acute cerebral stroke [7,9,10]. With such rapid liberation of oxygen, even an accidental ingestion of a single mouthful of food grade H_2O_2 represents a significant danger.

We present a case series of four patients who accidentally ingested concentrated H_2O_2 resulting in venous gas emboli, who were successfully treated with hyperbaric oxygen (HBO) therapy. (See Table 1.)

☆ **Support**: We received no funding or grants for this project. This work has not been previously presented.

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http://dx.doi.org/10.1016/j.ajem.2016.12.027
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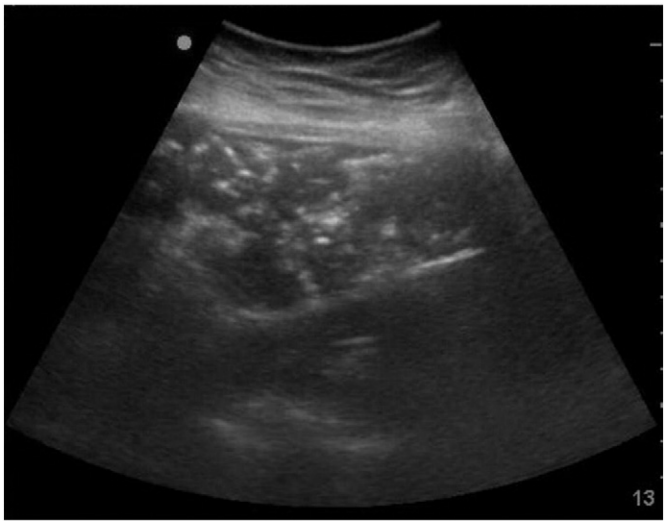

Fig. 1. Right upper quadrant bedside ultrasonography.

2. Case series

2.1. Case 1

A 33 year-old female restaurant employee accidentally drank an estimated 250 mL of 35% H_2O_2 mistaking it for water. Shortly after the ingestion, she developed nausea, abdominal pain, and hematemesis. On arrival to the emergency department (ED), she was intubated for airway protection. Her blood pressure was 126/91 mm Hg; her heart rate was 97 beats/min. No obvious oropharynx or laryngeal caustic injury was noted during the intubation. An abdominal bedside ultrasound exam visualized dynamic movement of gas bubbles in the portal venous system. A non-contrast CT scan of the head was normal. A CT scan of the chest, abdomen and pelvis demonstrated portal venous gas, pneumatosis of the stomach and esophagus, and gas in the right ventricle and pulmonary artery. Her neurologic exam was without deficit prior to rapid sequence induction and intubation. HBO therapy was provided with 100% oxygen at 2.8 ATA for 40 min, followed by a staged decompression given the possibility of a PFO and inability to follow neurologic exams after intubation. The following day an EGD was performed that demonstrated Grade Ia esophagitis and IIa gastritis. Extubation occurred one day after HBO therapy, and the patient's neurologic exam was normal at that time. A repeat abdominal ultrasound and a CT imaging performed after HBO therapy, demonstrated complete resolution of gas. The patient was discharged without significant sequela on hospital day 3.

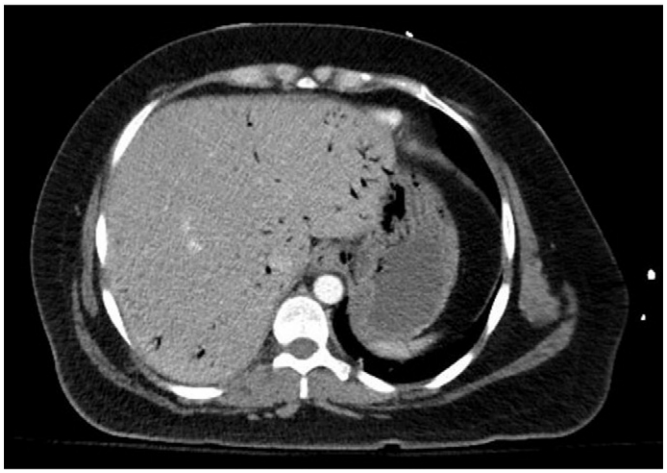

Fig. 2. CT coronal image of pneumobilia in patient number I.

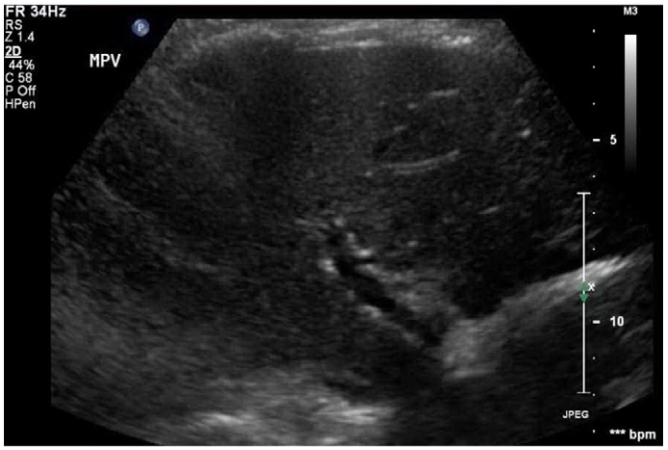

Fig. 3. Ultrasound image of the hepatic system after hyperbaric oxygen treatment of the patient in Case 1.

A bedside ultrasound still image obtained of Case 1 at presentation (see Fig. 1), demonstrated extensive portal venous gas and pneumobilia. The bright line separating the heterogeneous hyperechoic gas filled liver on the top of the image from the relative hypoechoic non gas filled kidney inferiorly is Morrison's pouch, a potential space where fluid and blood can collect in trauma. Note the pneumobilia in the hepatic system causing deflection and refraction of ultrasound waves and creating the heterogenic liver seen in this image.

A CT of the chest abdomen and pelvis with contrast was performed at presentation (see Fig. 2) which also demonstrated pneumobilia seen on the ultrasound of the patient. A post HBO therapy formal (non ED bedside) ultrasound image of the hepatic system (see Fig. 3) demonstrated resolution of the pneumobilia. Note the relative homogenous nature of the liver compared to Fig. 1 with pneumobilia.

2.2. Case 2

A 75 year-old old woman accidentally consumed approximately 3 mouthfuls of 35% H_2O_2, mistaking it for a colonoscopy preparation. Her husband obtained the product for use in homeopathy. Immediately after the ingestion, she vomited and then collapsed with loss of consciousness. She was intubated at the scene and was stabilized at a tertiary facility. A CT demonstrated portal venous and mediastinal gas (see Fig. 4). Head imaging was unremarkable, and the patient was transferred for HBO therapy. Upon arrival, the patient's blood pressure was 109/55 mm Hg, and her heart rate was 88 bpm. Sedation was continued, and the patient underwent emergent HBO therapy at 2.8 ATA for 60 min

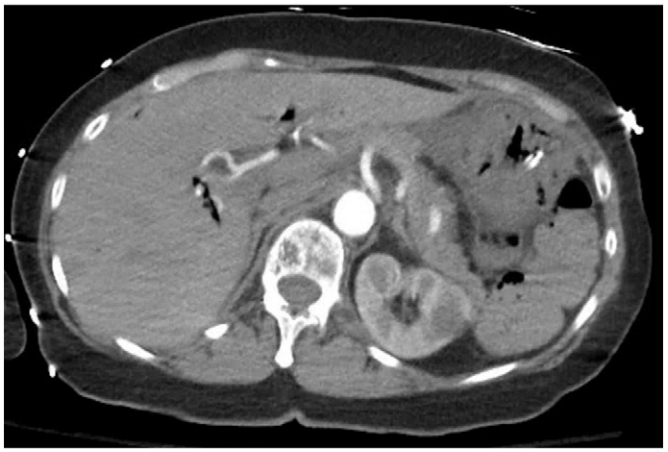

Fig. 4. CT coronal image of pneumobilia in patient in Case 2.