



Caustic Ingestion of Hydrogen Peroxide and Hepatic Portal Venous Gas: A Case Report and Literature Review

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Introduction

A commonly used over-the-counter solution that is both odorless and colorless, hydrogen peroxide (H₂O₂) and its ingestion may lead to severe medical outcomes including death. Toxicity secondary to H₂O₂ ingestion involves caustic damage, direct lipid peroxidation, and gaseous oxygen production that may create portal venous gas and air embolization with subsequent risk of cerebral infarction and mesenteric ischemia with mortality rate reaching 75-90% [1-4].

A rare radiological phenomenon diagnosed via abdominal x-ray, ultrasonography, or Computed Tomography (CT) scan, He-

Abstract

Background: Hydrogen peroxide (H₂O₂) ingestion is associated with caustic injury and gaseous oxygen production that may cause Hepatic Portal Venous Gas (HPVG) and air embolism leading to mesenteric ischemia and CNS infarction with ensuing encephalopathy, seizures, and even death.

Case report: A 38-year-old male presented with periumbilical pain after accidental ingestion of wood bleach with 30% H₂O₂ concentration, with Computed Tomography (CT) scan demonstrating left HPVG and upper endoscopy evaluation revealing diffuse moderate gastritis. Patient was managed conservatively with subsequent resolution of left HPVG on repeat CT scan the following day. He was discharged home in stable condition and lost to follow-up.

Conclusions: Following exclusion of serious intraabdominal pathology that may require surgical intervention, HPVG following substantial ingestion of concentrated 30% H₂O₂ ingestion may be treated conservatively with subsequent resolution of HPVG.

patric Venous Portal Gas (HPVG) is defined by the abnormal presence of gas in the portal system and remains most commonly associated with underlying intestinal ischemia and infarction with poor prognosis and high mortality rates [4]. However, the prognosis associated with HPVG and subsequent recommended treatment course is dependent on the pathophysiology itself, as mortality rates approximate respectively 20% and 0% in inflammatory processes and incidental cases [5]. We present a case of a 38-year-old male with accidental ingestion of hydrogen peroxide who was found to have HPVG on abdominal CT scan and who was successfully managed conservatively.



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Case report

A 38-year-old male presented to our tertiary care center with burning periumbilical pain after accidentally ingesting an estimated 250cc of unlabeled wood bleach with a concentration of 30% hydrogen peroxide in the erroneous belief that this was water. Patient also experienced shortness of breath closely following ingestion that had since resolved and a right-sided headache without any other focal neurological symptoms.

Upon presentation, vitals were normal. Patient was neurologically intact with frequent checks, and his initial abdominal exam was remarkable for tenderness to palpation at midline under the umbilicus without rebound or guarding. Laboratory studies including complete blood count and comprehensive metabolic panel were within normal limits except for a bicarbonate level of 19 mmol/L and aspartate aminotransferase of 47 U/L.

Admission CT abdomen/pelvis without IV contrast revealed portal venous gas in the left hepatic lobe, in addition to incidental fatty liver, hepatomegaly, and severe rectus diastases of multiple ventral hernias (Figure 1). He was given a 1L bolus of normal saline, carafate, and famotidine before undergoing Esophago Gastroduo Denoscopy (EGD) evaluation which was performed on the day of admission. The EGD demonstrated diffuse moderate gastritis characterized by erythema and superficial mucosal sloughing, but no esophageal injury or duodenopathy (Figure 2). Patient was started on a proton-pump inhibitor and clear liquid diet that was soon advanced to a regular diet.

Repeat CT abdomen/pelvis without IV contrast the following day revealed resolution of left hepatic portal venous gas, but interval development of thick-walled edematous stomach consistent with gastritis and remaining few dots of free intraperitoneal air by the gastric pylorus (Figure 3). Given resolution of hepatic portal venous gas and benign abdominal exam, patient was discharged home in stable condition with an eight-week course of proton pump inhibitor. The patient was advised to follow up with PCP.

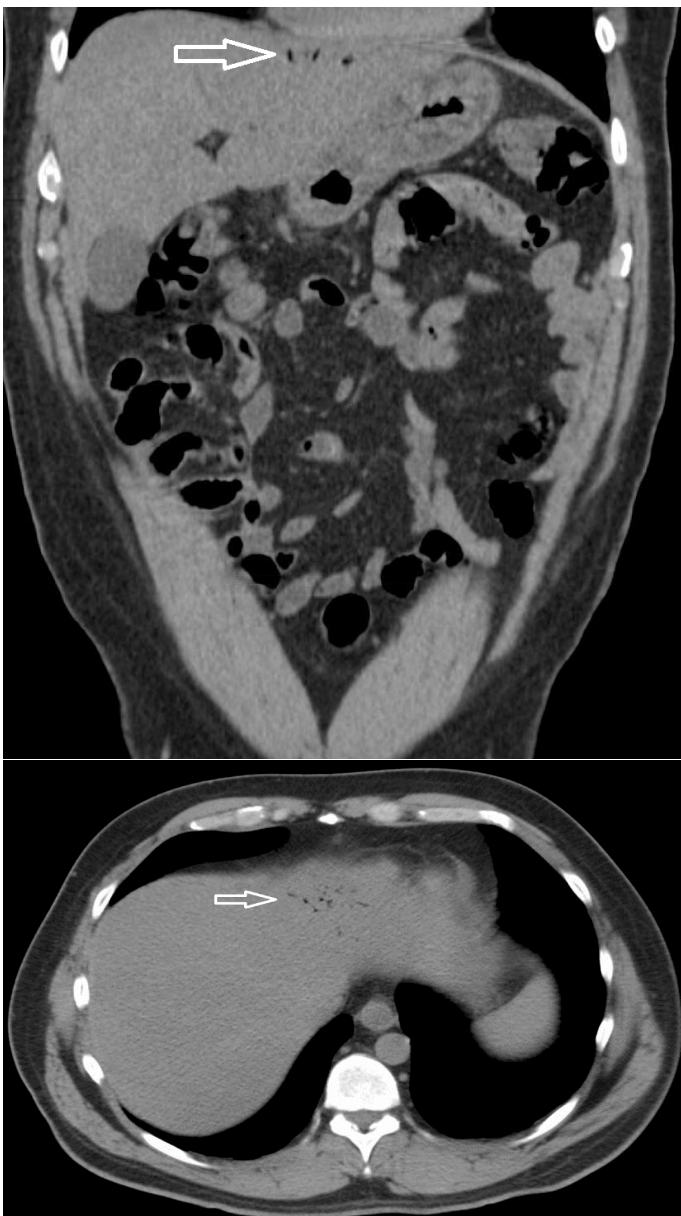


Figure 1: Computed tomography abdomen/pelvis without contrast demonstrating left hepatic portal venous gas, as well as incidental fatty liver and hepatomegaly.

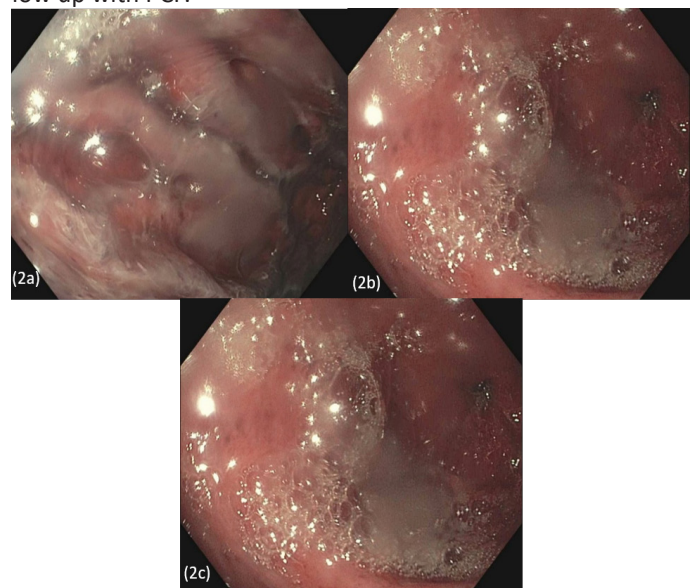


Figure 2: Esophagogastroduodenoscopy demonstrating diffuse moderate gastritis in the gastric body (2a & 2b) and pre-pyloric stomach (2c).



Figure 3: Computed tomography abdomen/pelvis without contrast demonstrating resolution of left hepatic portal venous gas, but interval development of thick-walled edematous stomach consistent with gastritis and remaining few dots of free intraperitoneal air by the gastric pylorus.

Discussion

The consequences of H₂O₂ ingestion encompasses corrosive injury, cytotoxicity via lipid peroxidation, and oxygen gas formation [1]. Caustic ingestion of concentrated H₂O₂ causes gastrointestinal tract irritation that may lead to nausea, vomiting, and hematemesis, and grade I mucosal injury is the most common and often spontaneously resolves without sequelae, as our patient demonstrated [1,6]. On the other hand, as H₂O₂ is degraded via catalase in erythrocytes and hepatocytes into water and oxygen, 1 mL of 35% H₂O₂ rapidly releases approximately 100 mL of oxygen [7]. Such rapid production of oxygen in closed body cavities may result in mechanical distension and even perforation [1]. However, mortality associated with H₂O₂ ingestion tends to involve the formation of arterial air embolism that may arise through 3 mechanisms: 1) pulmonary gas emboli from aspiration of hydrogen peroxide into the respiratory tract, 2) venous gas embolization that can transit through a patent foramen ovale or atrial septal defect, or 3) absorption of H₂O₂ across the gastrointestinal tract, subsequent catabolism into oxygen within the arterial vasculature, and the formation of air embolism when the amount of oxygen surpasses its maximum solubility in the blood [1,2,8]. Air embolisms may cause mesenteric ischemia, and CNS infarction with ensuing encephalopathy, seizures, and even death [2,3]. There have been previous reports of ischemic strokes from air emboli treated with hyperbaric oxygen therapy resulting in reversal of clinical symptoms and radiologic abnormalities [9]. As such, the presence of neurologic deficits consistent with cerebrovascular accident is a definitive indication for hyperbaric oxygen therapy, but the role of hyperbaric oxygen therapy in the presence of hemodynamic instability or elevated troponin suggestive of pulmonary or cardiac emboli remains unclear [3,7].

On the other hand, the pathogenesis of HPVG is attributed to 2 mechanisms: 1) bacterial: The presence of gas-producing bacteria and the passage of gas into the portal venous system or 2) mechanical: Damage to the intestinal mucosa from etiologies such as caustic ingestion, inflammatory bowel disease, intestinal ischemia, intestinal obstruction, intraabdominal abscess, or gastrointestinal neoplasms that allows intraluminal gas to enter the portal venous system [10]. Whereas HPVG was previously regarded as a poor prognostic indicator with high mortality of 75% due to its associations with ischemic bowel in a review of 64 cases in 1978, recent estimations of mortality rates have declined to 39% due to early diagnosis and the advent of CT scans empowering recognition of more benign or incidental causes of HPVG that are not associated with intestinal necrosis or pneumatosis Intestinalis [11–13].

As demonstrated in our patient, substantial ingestion of approximately 250mL of highly concentrated 30% H₂O₂ leading to HPVG is generally benign. Indeed, most cases of H₂O₂ ingestion causing HPVG often resolve on repeat CT scan after several days of conservative treatment [14,15]. To our knowledge, guidance regarding the management of H₂O₂ ingestion with development of HPVG relies entirely on case series/reports and a retrospective review. Management generally involves close observation using telemetry, hemodynamic monitoring, and frequent neurological checks, supportive care, upper endoscopic evaluation to evaluate the extent and depth of injury, and CT or MRI imaging to assess for formation of air embolism.[1,7] Hyperbaric oxygen therapy is also indicated in the presence of neurological symptoms after cerebral CT or MRI study given the danger of cerebral emboli [16,17]. The

role of hyperbaric oxygen therapy in the presence of HPVG without neurological symptoms is unclear [7]. After excluding serious intraabdominal pathology such as intestinal perforation or necrosis that may require emergency surgical intervention, HPVG following H₂O₂ ingestion may be treated conservatively in a clinically stable patient without neurological symptoms with subsequent resolution of HPVG [14,15,18].

Author contributions

ET supervised the study, performed literature review, and drafted the manuscript. JY performed literature review and critically revised the manuscript for important intellectual content. CT critically revised the manuscript for important intellectual content. MN supervised the study and critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

References

1. Watt BE, Proudfoot AT, Vale JA. Hydrogen Peroxide Poisoning. *Toxicological Reviews*. 2004; 23.
2. Shaw A, Cooperman A, Fusco J. Gas Embolism Produced by Hydrogen Peroxide. *New England Journal of Medicine*. 1967; 277: 238-241.
3. Mullins ME, Beltran JT. Acute Cerebral Gas Embolism from Hydrogen Peroxide Ingestion Successfully Treated with Hyperbaric Oxygen. *Journal of Toxicology: Clinical Toxicology*. 1998; 36.
4. Wiesner W, Mortelé KJ, Glickman JN, Ji H, Ros PR. Portal-venous gas unrelated to mesenteric ischemia. *European Radiology*. 2002; 12: 1432-1437.
5. Hernando Sanz A, Díaz-Tobarra M, González Álvarez G. Hepatic portal venous gas. Should it be considered as an ominous radiologic sign? *Revista española de enfermedades digestivas : organo oficial de la Sociedad Española de Patología Digestiva*. 2020; 112: 75.
6. Tohda G, Sugawa C, Gayer C, Chino A, McGuire TW, Lucas CE. Clinical evaluation and management of caustic injury in the upper gastrointestinal tract in 95 adult patients in an urban medical center. *Surgical Endoscopy and Other Interventional Techniques*. 2008; 22: 1119-1125.
7. Hatten BW, French LK, Horowitz BZ, Hendrickson RG. Outcomes After High-Concentration Peroxide Ingestions. *Annals of Emergency Medicine*. 2017; 69: 726-736.e2.
8. French LK, Horowitz BZ, Mckeown NJ. Hydrogen peroxide ingestion associated with portal venous gas and treatment with hyperbaric oxygen: a case series and review of the literature. *Clinical Toxicology*. 2010; 48.
9. Rider SP, Jackson SB, Rusyniak DE. Cerebral air gas embolism from concentrated hydrogen peroxide ingestion. *Clinical Toxicology*. 2008; 46: 815-818.
10. Li Z, Su Y, Wang X, Yan H, Sun M, Shu Z. Hepatic portal venous gas associated with colon cancer: A case report and literature review. *Medicine (United States)*. 2017; 96.
11. Liebman PR, Patten MT, Manny J, Benfield JR, Hechtman HB. Hepatic-Portal Venous Gas in Adults: Etiology, Pathophysiology and Clinical Significance.
12. Huang CY, Sun JT, Tsai KC, Wang HP, Lien WC. Hepatic Portal Venous Gas: Review of the Literature and Sonographic Implications. *Journal of Medical Ultrasound*. 2014; 22: 66-70.
13. Kinoshita H, Shinozaki M, Tanimura H, et al. Clinical Features and Management of Hepatic Portal Venous Gas Four Case Reports

and Cumulative Review of the Literature. Vol 136; 2001.

14. Zengin S, Al B, Genç S, Yarbil P, Yilmaz DA, Gulsen MT. A rare case of portal vein gas: Accidental hydrogen peroxide ingestion. *BMJ Case Reports*. Published online 2012.
15. Ghai S, O'Malley ME. Portal Vein Gas Resulting from Ingestion of Hydrogen Peroxide. *American Journal of Roentgenology*. 2003; 181: 1719-1720.
16. Byrne B, Sherwin R, Courage C, et al. Hyperbaric Oxygen Therapy for Systemic Gas Embolism after Hydrogen Peroxide Ingestion. *The Journal of Emergency Medicine*. 2014; 46: 171-175.
17. Hendriksen SM, Menth NL, Westgard BC, et al. Hyperbaric oxygen therapy for the prevention of arterial gas embolism in food grade hydrogen peroxide ingestion. *The American Journal of Emergency Medicine*. 2017; 35: 809.e5-809.e8.
18. Pritchett S, Green Md D, Rossos P. Accidental Ingestion of 35% Hydrogen Peroxide. Vol 21; 2007.