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Case Report

White spirit poisoning: An unusual cause of hepatic portal venous gas ☆

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ABSTRACT

Traditionally, the presence of air within the hepatic portal venous system has been considered a rather ominous sign as it has been associated with conditions of increased associated morbidity and mortality such as bowel ischemia and intraabdominal sepsis. However, benign conditions, not requiring any particular intervention, have been implemented in the etiology, as well. In the present report, we present the case of the accidental ingestion of white spirit as a rather unusual cause of hepatic portal vein gas. A 32-year-old, otherwise healthy, male was admitted to the emergency department following the accidental ingestion of a "sip," approximately 15 ml, of white spirit. The patient was complaining of nausea and upper abdominal pain that started soon after the ingestion of caustic agent. An imaging investigation with a computed tomography scan (CT) of the abdomen revealed the presence of hepatic portal vein gas along with a diffuse edema of the gastric wall at the site of the lesser curvature. A follow-up CT, 2 days after the admission, revealed no evidence of hepatic portal venous gas. Based on the patient's good general condition, an expectant management was decided. No intervention was required, oral feeding was recommenced after 6 days of fasting and the patient was discharged 8 days after the admission. Hepatic portal venous gas is a very impressive imaging finding with remarkably diverse etiology and prognostic correspondence. Irrespective of the cause, an approach of managing patients with hepatic portal venous gas according to their clinical condition appears reasonable.

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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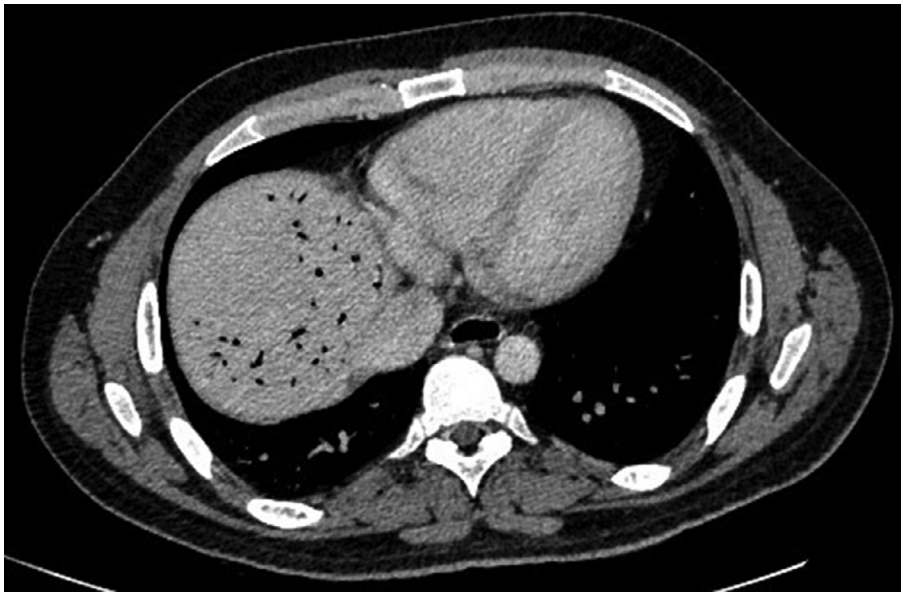

Fig. 1 – Computed tomography image showing the hepatic portal venous gas with its characteristic peripheral distribution.

Introduction

The first description of hepatic portal venous gas (HPVG) was made in 1955, by Wolfe and Evans, in infants with necrotizing enterocolitis [1]. From the pathophysiology viewpoint, the air within the hepatic portal venous system could be originated either from the lumen of the gastrointestinal tract when a damaged mucosal barrier allows gas to move from the intestinal lumen to the mesenteric veins or it could be produced directly within the blood stream should gas producing organisms circulate within the portal venous system [2,3].

Several conditions have been associated with this imaging finding. Bowel ischemia, necrotizing enterocolitis, inflammatory bowel diseases, intra-abdominal septic conditions such as acute diverticulitis, acute appendicitis and acute cholangitis have been all implemented in the etiology of the condition [3]. However, besides these relatively common causes of HPVG, the condition has been reported, as well, rarely in association with acute necrotizing pancreatitis [4], after extracorporeal shock wave lithotripsy for obstructive pyelonephritis [5], gastrojejunal anastomotic leak after laparoscopic gastric bypass [6], percutaneous endoscopic gastrostomy tube placement [7], and in the superior mesenteric artery syndrome with massive gastric dilatation [8].

Since its first description, the presence of gas within the hepatic portal venous system has been considered a poor prognostic sign as it has been linked, mainly, with conditions of high associated morbidity and mortality [2]. An urgent laparotomy is usually the final step of the treatment algorithm aiming to treat the intra-abdominal pathology. However, there are reports of successful non-operative management, as well [9]. Patients with functional gastric dilatation [10], blunt abdominal trauma [11] or inflammatory bowel disease [12,13] have been successfully treated without any interventions. In general, in patients with inflammatory bowel disease HPVG has been documented occurring either spontaneously during the course of the disease or after an endoscopy [12,13]. A review by Sebastia et al. suggested that 15% of cases of HPVG were idiopathic [14].

Recently, with the widespread use of imaging modalities, the diagnosis of HPVG associated with more benign or conditions not requiring particular intervention is much more common. For example, HPVG has been diagnosed secondary to ingestion of hydrogen peroxide [15,16]. Caustic ingestion such as bleaching agents has been highlighted as a cause of HPVG, as well [17]. In the present study, we report the rare case of a 32 years old male diagnosed with HPVG after ingestion of approximately 15 ml of white spirit.

Presentation of case

A 32-year-old, otherwise healthy, male was admitted to the emergency department following the accidental ingestion of a “sip,” approximately 15 ml, of white spirit. The patient was complaining of nausea and upper abdominal pain that started soon after the ingestion of caustic agent. Clinical examination revealed notable tenderness during the palpation of the mid epigastrium. An imaging investigation with a computed tomography (CT) scan of the thorax and abdomen was then conducted. While there were no remarkable findings from the lungs and the mediastinum, the findings of the CT abdomen were particularly impressive. More specifically, HPVG was revealed with its characteristic peripheral, subcapsular distribution (Figs. 1 and 2). A diffuse edema of the gastric wall at the site of the lesser curvature was additionally noted (Fig. 3).

An upper GI endoscopy, on an emergency basis, was ruled out at the fear of an iatrogenic perforation. Based on the patient’s good clinical condition and on the absence of imaging evidences of perforation, an expectant management was

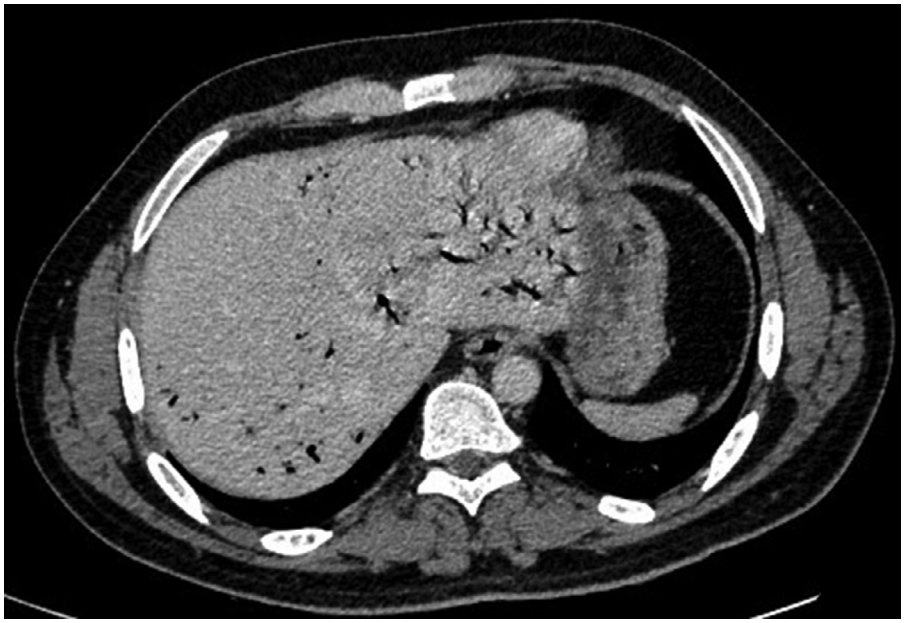

Fig. 2 – Computed tomography image showing the hepatic portal venous gas with its characteristic peripheral distribution in both hepatic lobes.

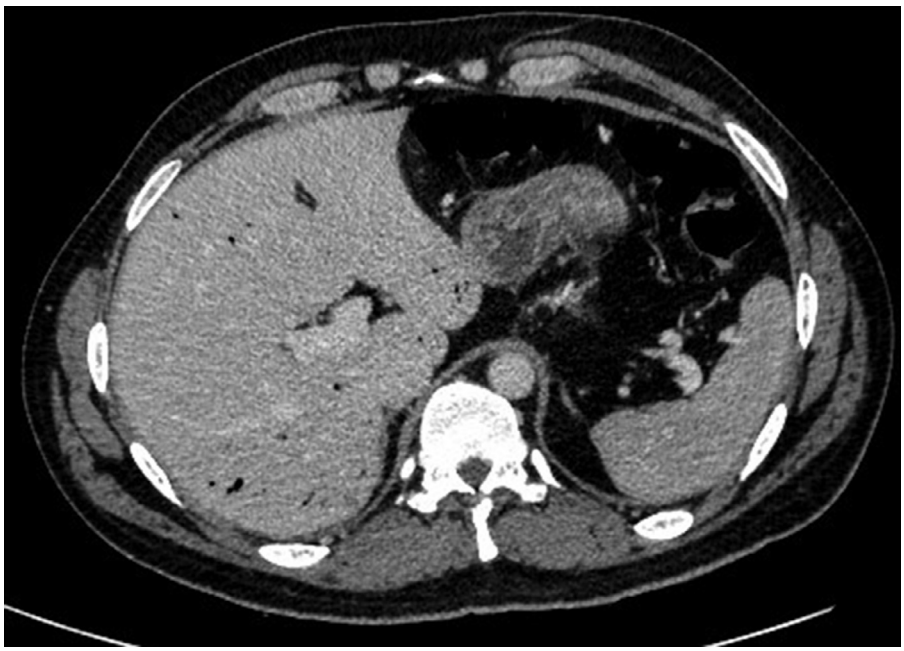

Fig. 3 – Computed tomography image showing the gastric wall edema.

decided. The patient was kept fasting with the administration of intravenous fluids and proton pump inhibitors. Two days after the admission, a follow-up CT scan abdomen was conducted in order to reassess the initial imaging findings that confirmed the spontaneous disappearance of HPVG. However, a notable aggravation of the gastric wall edema was noted (Fig. 4). After 6 days on conservative management, the upper GI endoscopy, which was deferred on admission, was conducted. It revealed a well-defined area of inflammation and edema onto the gastric wall without endoscopic evidences of perforation (Fig. 5). Then, oral feeding was recommenced and the patient was discharged 8 days after the admission.

Discussion

HPVG is an imaging diagnosis. The presence of air inside the hepatic portal vein system can be highly suspected in plain abdominal x-rays and can be further confirmed by more

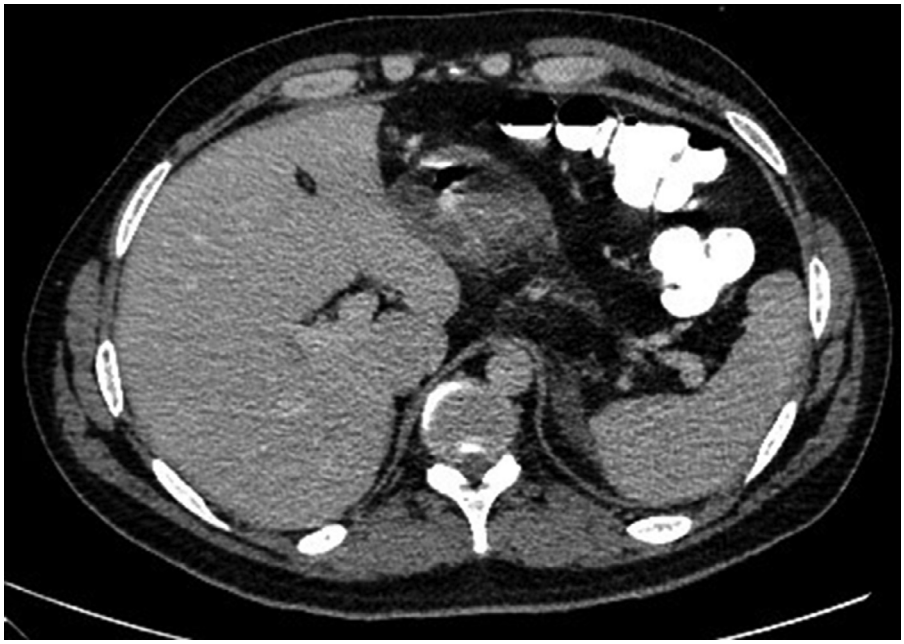

Fig. 4 – Follow-up computed tomography image with no evidence of hepatic portal venous gas but with an aggravation of the gastric wall edema.

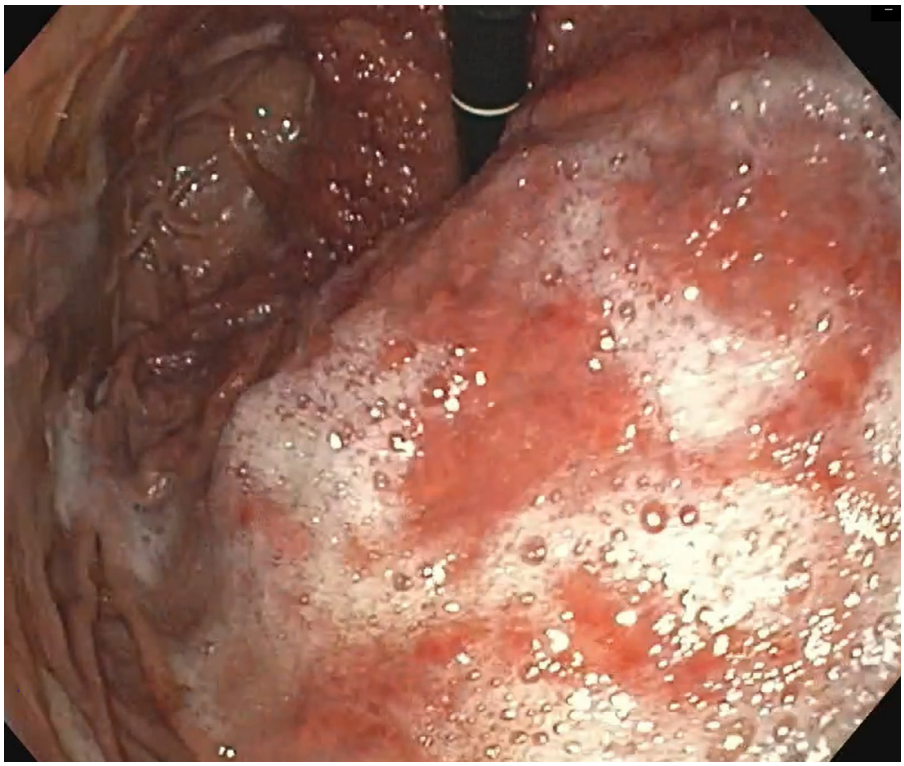

Fig. 5 – Endoscopic image of the corrosive damage onto the gastric mucosa.

elaborative imaging modalities such as ultrasonography, color Doppler flow imaging, or computed tomography (CT) scan [18]. Indeed, ultrasonography, color Doppler flow imaging, and CT scan have been reported to be superior to plain x rays in identifying HPVG [19]. In regards to ultrasonography, it is considered quite sensitive for the identification of HPVG and can be especially useful as an initial screening exam [19]. On the other hand, CT has a higher sensitivity and has the additional advantage of delineating the concomitant intra-abdominal pathology [20].

However, air within the liver parenchyma is not always synonymous with HPVG. Pneumobilia is a condition that is characterized by the presence of air within the biliary tree and should be included in the differential diagnosis. The distinction between these 2 entities i.e. HPVG and pneumobilia is based on the distribution of the air within the liver parenchyma. A peripheral gas distribution, usually within 2 cm of the liver capsule, characterizes HPVG because of the centrifugal blood flow pattern within the liver parenchyma while centrally located air is pathognomonic of pneumobilia because of the centripetal flow of bile, towards the porta hepatis [21].

In the present report, we present the case of 32-year-old male diagnosed with HPVG after the accidental ingestion of white spirit. White spirit is a paraffin-derived organic solvent, composed of a mixture of saturated aliphatic and alicyclic hydrocarbons [22]. White spirit is known by a number of different names. In the United States and Canada, for example, it is most commonly known as mineral spirits, while in Australia and New Zealand it is known as mineral turpentine. As it is used as a household solvent, present in paint thinner and remover, exposure of humans becomes common resulting in significant toxicity [23]. One important element in regards to the toxicity is the route of the exposure. Ingestion occurs most often accidentally, usually in children, while inhalation can be accidental in exposed workers or intentional in adolescents and adults. Finally, skin contact can cause toxic effects as it has been described following accidental exposure in industrial workers [24]. Perianal skin exposure has also been reported in the literature after massive intentional ingestion of white spirit [24].

In regard to ingestion, either intentional or accidental, common complaints include nausea, vomiting, diarrhea, and abdominal pain. Ingestion of large amounts can also cause complications related to the central nervous system, cardiac arrhythmias, renal and hepatic complications [25]. However, the mostly affected system after ingestion is the pulmonary system, since white spirit has a low viscosity and high volatility and even very small doses are sufficient to cause aspiration pneumonia. Direct and prolonged contact of white spirit with the skin and mucosae after ingestion has been reported to cause corrosive lesions [26].

In our case, HPVG was diagnosed during the work up of a patient presented to the emergency department due to the accidental ingestion of a sip of white spirit. The patient was in a good general condition but, to our surprise, the CT abdomen confirmed the presence of HPVG along with a notable edema of the gastric wall consistent with a probable corrosive damage. There were no evidences of free perforation. Based on the patient's condition, a conservative management was decided. The HPVG resolved spontaneously as the follow-up CT abdomen, performed 2 days later, showed no evidences of air within the liver parenchyma. An upper GI endoscopy, which was performed several days later, confirmed the, suspected by the imaging investigation, corrosive damage on the gastric wall. In an attempt to explain the unexpected finding of HPVG in this patient, we hypothesized that the ingestion of this caustic agent provoked a mucosal damage onto the gastric wall which, in turn, acted as an entrance gate to the portal venous system for this high volatile agent.

In conclusion, HPVG is a very impressive imaging finding with remarkably diverse etiology and prognostic correspondence. Irrespective of the cause, an approach of managing patients with HPVG according to their clinical condition appears reasonable.

Patient consent

I, Athina Samara, the corresponding author of the manuscript entitled "White spirit poisoning: An unusual cause of hepatic portal venous gas" confirm that the patient has signed an informed consent to publish the case.

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