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Case Report
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A Rare Case of Hepatic Portal Venous Gas and Its Management in the Emergency Department Discussed in Light of the Current Literature

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Abstract

Hepatic Portal Venous Gas (HPVG) is a rare condition. It is not a specific disease, but a diagnostic radiological finding in patients with underlying acute abdominal pathology. This report is intended to discuss, in the light of the current literature, a patient brought to the emergency department due to impaired consciousness and in whom rare HPVG was determined. An 83-year-old woman was brought to the emergency department due to impaired consciousness. The patient was bedridden, and nutrition had been provided by means of Percutaneous Endoscopic Gastrostomy (PEG) for the previous two years due to functional swallowing disorder. At physical examination, the abdomen was distended, and bowel sounds were hypoactive. Formed stool was observed at rectal examination. Non-specific gas shadows were present at abdominal X-ray. Abdominal USG could not be effectively evaluated due to diffuse gas artifacts. Abdominal Computerized Tomography (CT) revealed HPVG, perihepatic free fluid collection, and intra-abdominal free air values in the walls of the splenic flexure, in the splenic vein and the superior mesenteric vein, and around the splenic flexure. These findings were interpreted in favor of intestinal ischemia. The general surgery department was consulted, and the patient was taken for emergency surgery. We think that Multiple Detector CT (MDCT) angiography, a diagnostic radiological imaging technique, is the gold standard for the immediate detection of life-threatening primary pathologies in patients with confirmed or suspected HPVG, for prompt transferal of these to the operating room for emergency intervention and for increasing minimization of mortality rates.

Keywords: Hepatic portal venous gas; HPVG; Radiological finding; Diagnosis

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Introduction

Hepatic Portal Venous Gas (HPVG) is a rare condition. It is not a specific disease, but a diagnostic radiological finding in patients with underlying acute abdominal pathology. Its causes vary, depending on age, and it exhibits a wide etiological spectrum. It is generally associated with severe or potentially fatal conditions requiring emergency diagnosis and probably surgical intervention [1,2]. This report discusses, in the light of the current literature, a patient brought to the emergency department due to impaired consciousness and in whom rare HPVG was determined.

Case Presentation

An 83-year-old woman was brought to the emergency department due to impaired consciousness. On arrival her general condition was poor, she was unconscious, arterial blood pressure was 90/60 mmHg, heart rate was 148/min and arrhythmic, respiration rate was 26/min, and body temperature was 35.7°C. The patient was tachypneic, and oxygen (O_2) saturation was 92%. Chronic Atrial Fibrillation (AF), left hemiplegia due to previous cerebral infarct, bilateral carotid artery disease, Hypertension (HT) and Diabetes Mellitus (BM) were present in her medical history. The patient was bedridden, and nutrition had been provided by means of Percutaneous Endoscopic Gastrostomy (PEG) for the previous two years due to functional swallowing disorder. She had a history of insulin and coumadin drug use. Her Glasgow Coma Score (GCS) was 11 (E2,V4,M5). At physical examination, her abdomen was distended, and bowel sounds were hypoactive. Formed stool was observed at rectal examination. The distal half of the left tibia was

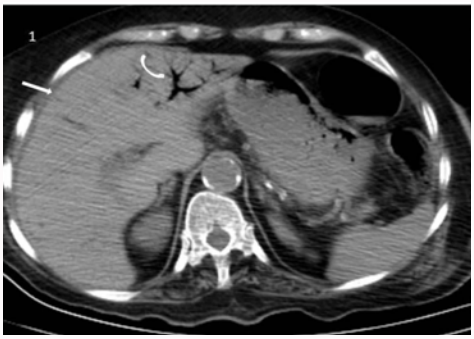

Figure 1: Linear air densities compatible with portal venous gas exhibiting branching, typically predominantly in the peripheral region in the left hepatic lobe (curled arrow) and perihepatic free fluid collection (long arrow).

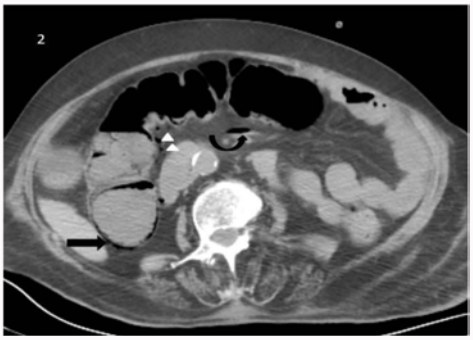

Figure 2: Linear air densities in the splenic flexure (long arrow), air densities in the superior mesenteric vein (curled arrow) and free intraabdominal air densities neighboring on the splenic flexure (arrow heads).

hyperemic and edematous. Electrocardiography (ECG) revealed AF rhythm with rapid ventricular response. At blood tests, glucose was 427 mg/dl, urea 187 mg/dl, creatinine 1.9 mg/dl, ALT 74U/L, AST 88U/L, sodium (Na) 125 mEq/L, chloride (Cl) 88 mEq/L, CK 256 U/L, CK-MB 3.5 U/L, troponin I 27.96 ng/L (range 0 to 11.6), INR 1.9, and CRP mg/L (range 0 to 5). Other biochemical parameters were within normal ranges. Her platelet count was 490 103/uL (range 142 to 424) and her leukocyte count was 13,630/mm^3 (range 3980 to 10,200/mm^3), with neutrophils predominating. At blood gas analysis, O_2 saturation was 88.2%, carbon dioxide (CO_2) pressure 38.9 mmHg, pH 7.49, and lactate 2.85 mmol/L (range 0.5 to 1.6). Non-specific gas shadows were present at abdominal X-ray. No flow was observed in the arteria tibialis posterior and arteria dorsalis pedis at Doppler ultrasonography of the left lower extremity.

Abdominal USG could not be effectively evaluated due to diffuse gas artifacts. Non-contrast abdominal Computerized Tomography (CT) revealed free air in portal vein tracts, particularly in the peripheral part of the hepatic left lobe, in the main portal, splenic, and superior mesenteric vein, occasionally in fine venous structures of the intestinal ansae in both lower quadrants, in the ascending colon splenic flexure wall, and neighboring the ascending colon inside the abdomen. Free fluid collection in the perihepatic region and focal dilation, 7 cm at the widest point, were observed at the level of the cecum in the ascending colon. These findings were interpreted in favor of intestinal ischemia. The general surgery department was consulted, and the patient was taken for emergency surgery. During the operation, punctate segmental necrosis was observed in the cecum, hepatic flexure, splenic flexure, sigmoid colon, and terminal ileum.

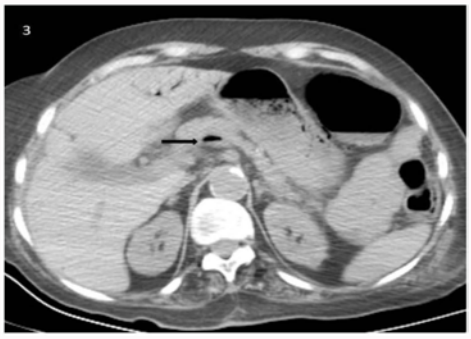

Figure 3: Air densities inside the splenic vein.

Following surgery involving hemicolectomy and ileum resection, the patient was admitted to the intensive care unit, but mortality occurred approximately 3 h subsequently.

Discussion

HPVG is a very rare radiological finding. Due to the absence of any prospective studies on the subject and the limited number of retrospective studies, we found no objective prevalence data. Sebastian et al. examined the abdominal CT scans of 13.927 adult patients taken over a four-year period and reported HPVG findings in 17, giving some idea of the prevalence [3]. Due to the anatomical nature of the hepatic venous structures, there is a greater tendency to development in the left lobe [3]. In our case, too, HPVG was in the left lobe. The prevalence is equal among males and females [2].

Various mechanisms have been proposed in the pathogenesis of HPVG. Gas is thought to pass from the venous system and lymphatics of the intestinal mucosa into the portal veins under the effect of this intraluminal dilation and increased pressure deriving from mechanical obstruction of the Gastrointestinal System (GIS).

Another proposed mechanism is that impairment of the intestinal mucosa and the epithelial barrier becoming permeable as a result of necrosis may permit gas-forming bacteria to pass to the intestinal. In addition, it has also been reported that HPVG may develop in the presence of inflammations caused by gas-forming micro-organisms of portomesenteric pylephlebitis [4].

The most common etiology, at a rate of 40% to 70%, is intestinal ischemia, followed by GIS inflammations at 8% to 16%. Causes of GI dilation, sepsis, invasive GIS procedures, pancreatobiliary infection, iatrogenic factors (such as barium enema, cardiopulmonary resuscitation, gastrojejunostomy leak, hemodialysis, hepatic artery embolization, intra-aortic balloon pulsation, liver transplantation, and percutaneous liver biopsy), colchicine toxicity and some chemotherapeutic drugs, bronchopneumonia, caustic ingestion, cryptosporidium, cystic fibrosis, diabetic ketoacidosis, emphysematous pyelonephritis, hyperbaric decompression, and seizures have also rarely been reported to lead to HPVG [2,5,6].

Our bedbound patient had a PEG inserted via a GIS invasive procedure. The presence in his history of chronic AF, previous cerebrovascular infarct, and bilateral carotid artery disease, and determination of peripheral artery disease in the left lower extremity, suggested that the basic problem was a thromboembolic event, despite coumadin use and an INR value of 1,9. We consider that

impairment of the intestinal mucosa and the epithelial barrier becoming permeable as a result of necrosis may lead to the passage of gas into the portal veins from the venous system and lymphatics of the intestinal mucosa.

Symptoms and physical findings on presentation to the emergency department vary depending on the underlying etiological cause, and HPVG is generally diagnosed *via* abdominal radiography, Ultrasonography (USG), color Doppler flow imaging, or CT. HPVG is difficult to identify using abdominal radiography, and can easily be overlooked. The technique is not sensitive to early stage intestinal ischemia in particular. However, HPVG being seen indicates intestinal infarction and poor prognosis. HPVG can be identified with USG and color Doppler imaging, but these are insufficient for determining the etiological cause. The technique with the greatest sensitivity in determining HPVG and identifying the underlying pathology is abdominal CT [7-9]. HPVG could not be determined through radiography in our case. It could also not be effectively evaluated with abdominal USG. Contrast material could not be applied due to impairment of kidney functions, and non-contrast abdominal CT revealed HPVG and perihepatic free fluid deposition and free air in the splenic flexure walls, inside the splenic vein and the superior mesenteric vein, and neighboring on the splenic flexure.

Radiologically, HPVG may be confused with pneumobilia. In pneumobilia, the air tends to be more central and in the direction of the splenic flow, and the number of branches is lower. In contrast, in HPVG it extends both to the area of the hepatic hilus and toward Glisson's capsule, in the opposite direction to the splenic flow, and the peripheral branches is tree-like in appearance [10].

Treatment and prognosis are associated not with the presence of HPVG, but with the underlying pathology. Conservative treatment or the decision to operate is based on careful analysis with laboratory and radiological findings. A conservative approach is recommended in case of benign etiological causes not requiring surgical treatment [6].

The largest study on this study to date involved a retrospective examination of 1590 adult patients with HPVG between 1 July, 2010, and 31 March, 2015, in all health institutions across Japan. An in-hospital mortality rate in patient with HPBG of 27.3% was reported in that study. The rate of HPVG with accompanying intestinal ischemia was 53%. GI system obstruction or dilation, GI perforation, GI infection, sepsis and in-hospital mortality rates were 26.8%, 31.1%, 33.3%, 13.6% and 56.4% were reported. Surgery was performed on 32.2% of patients with intestinal ischemia, and the in-hospital mortality rate was 16.5% [11]. Mortality rates for patients with HPVG findings have been reported of 75% in 1978 and 39% in 2001 [2,12]. The rate of accompanying Pneumatosis Intestinalis (PI) was reported at 70% to 80% in 2001 [13]. This significant decrease in mortality and PI is due to improvements in imaging techniques in recent years, resulting in the earlier detection and rapid treatment of potentially fatal pathologies such as intestinal ischemia, and to the reporting of several non-fatal etiological factors in parallel with larger numbers of cases being detected [14,15]. Multiple Detector CT (MDCT) angiography is currently the technique of choice for the detection of HPVG and its underlying cause [16].

Conclusion

Due to its wide etiological spectrum, the diagnosis of HPVG, which largely accompanies fatal pathologies, relies on rapid evaluation of clinical, laboratory and radiological findings. We think that among all the diagnostic radiological imaging techniques, MDCT angiography is the gold standard for the immediate determination of life-threatening primary pathologies in patients with suspected or confirmed HPVG, for being able to refer these for emergency surgical intervention without loss of time, and for the increasing minimization of mortality rates.

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