Computed Tomography Identifies Hepatic Portal Venous Gas Caused by Severe Diarrhoea

J-X Li*, Q-G Cui*, P-Y Gong

ABSTRACT

Aim: *To use computed tomography (CT) to diagnose the reasons for hepatic portal venous gas (HPVG) in the case of an elderly male patient.*

Methods: *This is a case study of an elderly male patient who suffered acute, obvious abdominal pain accompanied with stop of exhaust defecation following three days of diarrhoea, abdominal distention and emesis. The patient also developed asthma, which gradually became severe. The patient was admitted to the hospital where he underwent a physical examination and a CT scan.*

Results: *The CT results confirmed that the patient was suffering from HPVG caused by severe diarrhoea. The CT scan showed obvious expansion and pneumatosis in the enteric cavity and subcutaneous emphysema in the intestinal wall. Also, the intrahepatic portal branches and small branches of veins in the mesentery were filled with a high density of gas. The combination of many factors led to HPVG. Gastrointestinal mucosa and pressure accompanied with intestinal septic infection were the main factors. The case report revealed that gas in the enteric cavity went into the submucosa, then into the small branches of veins in the mesentery and finally into the intrahepatic portal vein system.*

Conclusions: *Computed examination revealed the imaging features of HPVG. Hepatic portal venous gas suggested the growth of enteric cavity pressure, the damage of intestinal mucosa and intestinal infection, providing references for clinical diagnosis.*

Keywords: Computed tomography, diagnosis, hepatic portal venous gas, treatment

La Tomografía Computarizada en la Identificación de Gas en el Sistema Venoso Portal Hepático Causado por Diarrea Severa

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RESUMEN

Objetivo: *Utilizar la tomografía computarizada (TC) para diagnosticar las razones para el gas en el sistema venoso portal hepático (GVPH) en el caso de un paciente de edad avanzada.*

Métodos: *Se trata del estudio de caso de un paciente de edad avanzada que padecía de dolor abdominal agudo, evidente, acompañado con distensión abdominal, vómitos, e incapacidad para expulsar gases después de tres días de diarrea. El paciente también desarrolló asma, que poco a poco se convirtió en severa. El paciente fue ingresado en el hospital donde fue sometido a un examen físico y una exploración de TC.*

Resultados: *Los resultados de la TC confirmaron que el paciente sufría de GVPH causado por diarrea severa. La exploración mediante TC mostró evidente expansión y pnematosis en la cavidad entérica, y enfisema subcutáneo en la pared intestinal. Además, las ramas portales intrahepáticas y las pequeñas ramas de las venas en el mesenterio se encontraban llenas con una alta densidad de gas. La combinación de muchos factores llevó a la formación de GVPH. La presión y la mucosa gastrointestinales, y la acompañadas de la infección séptica intestinal fueron los principales factores. El reporte de caso reveló que el gas en la cavidad entérica penetró en la submucosa, luego en las pequeñas ramas de las venas en el mesenterio, y finalmente en el sistema venoso portal intrahepático.*

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Conclusiones: *El examen computarizado reveló las imágenes características de GVPH. El gas del sistema venoso portal hepático indicó el aumento de la presión en la cavidad entérica, el daño de la mucosa intestinal y la infección intestinal, ofreciendo así referencias para el diagnóstico clínico.*

Palabras claves: Tomografía computarizada, gas en el sistema venoso portal hepático, diagnóstico, tratamiento

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INTRODUCTION

This is a case study of an elderly male patient who suffered hepatic portal venous gas (HPVG) by severe diarrhoea. The case report reveals that gas in the enteric cavity went into the submucosa, then into the small branches of veins in the mesentery and finally into the intrahepatic portal vein system. Computed tomography (CT) examination revealed the imaging features of HPVG and the presence of HPVG strongly suggests growth of enteric cavity pressure, damage of intestinal mucosa and intestinal infection.

CASE REPORT

After three days of diarrhoea, abdominal distention and emesis, an elderly male patient suffered acute, obvious abdominal pain at night that was accompanied with cessation of exhaust defecation. The patient also developed asthma, which gradually became severe; he was admitted to the hospital for examination and treatment. The patient was unconscious at presentation and physical examination revealed abdominal enlargement. The abdomen was soft, with no pressing pain or mass reported upon palpation. Percussive tympani was heard, whereas there were no bowel sounds. After admission, an endotracheal intubation was performed, and the patient was put on a respirator to alleviate his dyspnoea. Initially, the blood pressure was 80/50 mmHg, the heart rate was 56 beats per minute and the respiration rate was 39 breaths per minute. After administration of a large dose of norepinephrine, the blood pressure remained in the range 91–101/45–58 mmHg. The white blood cell was 29×10^9/L ($4.0 – 10.0 \times 10^9$/L), neutrophil 91%, blood sodium 121.8 mmol/L (137–147 mmol/L), potassium 5.16 mmol/L (3.5–5.3 mmol/L), serum chloride 84.9 mmol/L (99–110 mmol/L), blood fat low-density lipoprotein cholesterol (LDL-C) 3.14 mmol/L (0.4–1.7 mmol/L; for renal function: blood urea nitrogen (BUN) was 23.3 mmol/L (2.1–7.5 mmol/L) and creatinine 206.1 μmol/L (45–124 μmol/L).

According to the abdominal CT examination, a high density of gas was observed in the left hepatic portal vein and its branches, the hepatic portal vein, the splenic vein and the superior mesenteric vein (SMV) and its branches (Figs. 1–3). The patient was diagnosed with portal inflammation induced by severe abdominal infection [hepatic portal venous gas (HPVG)] and multiple organ failure. Positive anti-inflection and fluid infusion were performed to maintain the acid-base balance and protect the organs. During the treatment, the patient showed rapid disease deterioration and had short-term infectious shock, acute renal failure and respiratory failure. Small doses of hormone were administered as supporting treatment for shock. Severe infectious shock led to the deterioration of blood circulation, so large doses of vasoactive drugs were administered to maintain the blood pressure. In spite of this treatment, the blood pressure remained at 50/30 mmHg. The patient died three days after admission.

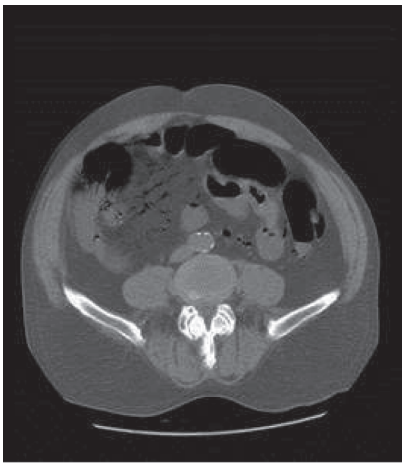

Fig. 1: Subcutaneous emphysema of the intestinal wall.

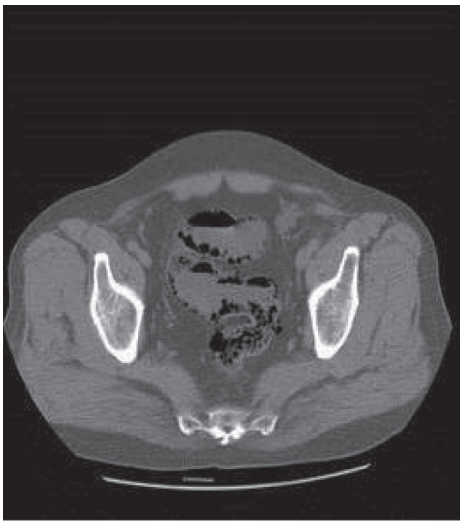

Fig. 2: Pneumatosis of branches of superior mesenteric vein.

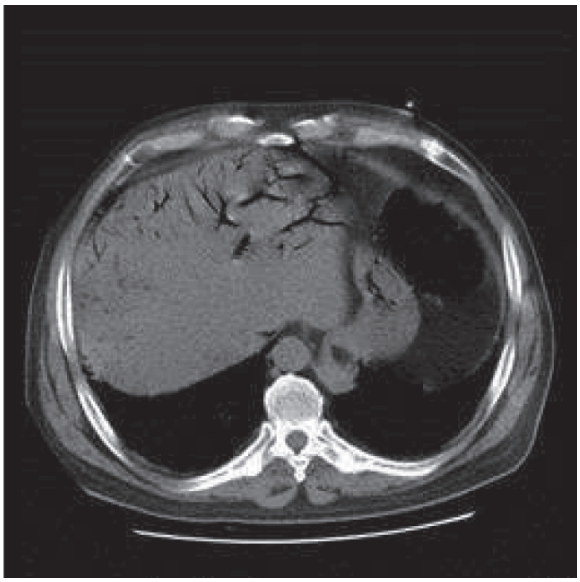

Fig. 3: Portal venous gas.

DISCUSSION

Ito *et al* reported that peritoneal dialysis caused HPVG in a patient with advanced stage diabetes (1). According to Yuda *et al*, *Enterobacter aerogenes* infection following an allogeneic stem cell transplant also led to HPVG (2). However, most researchers conclude there are three mechanisms involved in the development of HPVG (3–6). One mechanism involves barrier damage to the gastric intestinal mucosa. According to the intestinal mucosal barrier damage theory, endoscopic procedures and conditions like ulcerative colitis and Crohn's disease lead to intestinal mucosa damage and therefore an increase in permeability. This increase in permeability allows the gas in the enteric cavity to enter into small vessels of the intestinal wall and then into the circulation of the portal veins, thereby inducing HPVG.

The second mechanism involves gastrointestinal expansion and pressure increase. Intestinal obstruction and abdominal trauma to the intestinal mucosa cause luminal dilatation and increased pressure in the enteric cavity. Intestinal mucosal oedema and even ischaemia and necrosis lead to subtle damage of the intestinal mucosa, which allows gas to enter the vessels, thereby giving rise to HPVG. The third mechanism involves septic infection. Inflammation of the portal vein system, sepsis and *Enterobacter aerogenes* infection all may contribute to HPVG. The gas produced by intestinal bacteria fermentation is absorbed into the portal vein system. Mesenteric abscess, abnormal paths of peritoneum and the penetration into the region under the mesocolon come into the portal vein system in various conditions including liver transplantation, septic phlebitis, abdominal abscess and cholangitis. These three mechanisms are likely to induce HPVG, and damage to gastrointestinal mucosa and intestinal dilatation are the most frequently reported factors. In many cases, these factors appear to contribute in combination (7).

In our report, the combination of many factors led to HPVG. Gastrointestinal mucosa damage and pressure increase accompanied with intestinal septic infection were the main factors. In terms of pathophysiology, the intestinal canal in the abdominal cavity was significantly dilated and filled with gas, leading to the pressure increase in the intestinal canal and intestinal co-infection. In this case, intestinal mucosa was damaged, allowing gas in the intestinal canal to enter the vessels under the intestinal mucosa. Intestinal submucosal emphysema was significant in this patient. The gas entered the small branches of mesenteric veins and portal veins *via* blood circulation. Due to the dynamic impact of blood circulation, the gas was evenly distributed throughout small branches of portal veins under the hepatic capsular. Our case report provides a detailed description of the HPVG process from dilatation and pneumatosis of the enteric cavity, to intestinal emphysema, to mesenteric venous gas and to portal vein gas. Hepatic portal venous gas should be differentiated from pneumobilia. Deadwood-shaped gas density in the liver has a typical CT expression, becoming thinner from porta hepatis to the far end. Gas density was also found in the portal vein trunk and its branches. Gas in intrahepatic ducts was mainly due to the history of biliary tract surgery. Computed tomography results showed relatively scattered gas density, and gas was often found in extrahepatic ducts. In addition, as the direction of the portal vein flow and centrifugation was contrary to that of bile, HPVG was mostly located in liver parenchyma 2 cm under the liver capsular. In contrast, gas in bile ducts was often in the centre of liver parenchyma (8).

Hepatic portal venous gas is mostly considered a supporting image to reveal intestinal ischaemia, mucosa rupture and gastrointestinal infection (9–10). Early detection of HPVG and the identification of the resultant diagnosis have facilitated the administration of timely and targeted treatment. Rather than being an indication for surgery, the presence of HPVG should be considered a diagnostic sign. Features of the underlying cause may also be evident, such as marked pneumatosis intestinalis, gastrointestinal oedema and dilatation or paucity of luminal gas. In particular, dilatation and inflammation of the digestive tract, intraperitoneal abscess, and features of bowel ischaemia, such as pneumatosis intestinalis, may be demonstrated.

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