

CASE REPORT

# Hepatic Portal Venous Gas due to Acute on Chronic Gastric Ischaemia

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## ABSTRACT

Bowel ischemia is the commonest cause of hepatic portal venous gas and is associated with mortality in excess of 50 percent. Here we report a case of hepatic portal venous gas due to gastric ischemia. Retrospective case note review was carried out. A 81 year old gentleman, with a long standing past history of recurrent abdominal pain, presented with sudden onset of severe epigastric pain. On computerized tomography scan imaging, he was found to have hepatic portal venous gas along with pockets of air layers in the posterior gastric wall, suggestive of gastric ischemia. Patient was managed conservatively with antibiotics and proton pump inhibitor. He made an uneventful recovery. Although associated with high mortality rate, hepatic portal venous gas per se is not an indication for emergency surgery in all case. Wait and watch policy may be adopted in selected patients having acute on chronic gastric ischemia without haemodynamic compromise. However one should have very low threshold to proceed for exploratory laparotomy.

**Key words:** Portal vein, Ischemia, Gastric, Gas

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## ÖZET

### Kronik Gastrik İskemide Akut Seyreden Hepatik Portal Venöz Hava Bulunması

Bağırsak iskemisi hepatic portal vende hava görülmesinin en yaygın sebebidir ve %50'yi aşan bir mortaliteyle seyreder. Burada gastrik iskemiyeye bağlı hepatic portal vende hava görülen bir olguyu raporladık. Olgu retrospektif olarak yürütülmüştür. Karın ağrısı uzun zamandır devam eden 81 yaşındaki erkek hasta, ani başlayan şiddetli epigastrik ağrı ile başvuruyor. Bilgisayarlı tomografi incelemesinde gastrik iskemiyeyi düşündüren mide arka duvarında hava cepleri ile birlikte hepatic portal vende hava olduğu tespit edildi. Hastaya konservatif olarak antibiyotik ve proton pompa inhibitörü tedavisi başlandı. Hasta sorunsuz bir iyileşme gösterdi. Hepatic portal vende hava tespit edilmesi, yüksek mortalite ile ilişkili olmasına rağmen her olguda acil cerrahi müdahale gerektiren bir durum değildir. Akut ağrısı gelişen kronik gastrik iskemili, hemodinamik riski olmayan seçilmiş hastalarda, bekle ve izle politikası uygulanabilir. Ancak eksploratif laparotomiye geçmek için eşik değeri çok düşük olmalıdır.

**Anahtar kelimeler:** Portal ven, İskemi, Gastrik, Hava

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## INTRODUCTION

Bowel ischemia is the commonest cause of hepatic portal venous gas (HPVG) and is associated with mortality in excess of 50 percent. Here we report a case of HPVG due to gastric ischemia.

## CASE REPORT

A 81 year old gentleman from Indian ethnic origin presented with sudden onset of severe epigastric pain of few hours duration. This was associated with some nausea and vomiting. There were no other bowel symptoms of note. His past history includes long standing ischemic heart disease, recurrent post prandial abdominal pain, hypertension and glaucoma. He used to get crampy abdominal pain following large meal for varying length of time. With small meals he used to get some vague epigastric discomfort. Very occasionally, he suffered from nausea and vomiting in addition to mentioned symptoms. There was no history of recent loss of appetite or weight. He did not have past history of malignancy, bowel disease, gall stone or pancreatitis. His medications include calcium and vitamin D3, anti-hypertensive and glaucoma eye drops (Latanoprost). He did not report use of alcohol, smoking or illicit drugs.

Apart from slight tachycardia, his vital signs were within normal limits. His abdominal examination showed tender epigastrium without peritonism. There were active bowel sounds. There was no lump, distension or organomegaly. Hernias orifices, renal angles and genitalia were within normal limits. Other systemic examinations including respiratory and cardiovascular examinations were within normal limits.

Chest X-ray did not show any free gas under diaphragm. Abdominal X-ray, ECG and urine examination did not reveal any abnormality. Blood examination displayed deranged liver and renal function tests along with lactic metabolic acidosis and very high amylase level. Blood inflammatory markers were mildly raised. Initially thought to be a case of acute pancreatitis, on computerized tomography (CT) scan imaging, he was found to have HPVG along with layers of air bubbles in the posterior gastric wall, suggestive of gastric ischemia (Figure 1,2). There was a calcified plaque at the origin of the celiac axis which could be the reason behind the recurrent postprandial abdominal pain (Figure 3).

After discussion with the senior consultant gastrointestinal surgeons at tertiary care centre, decision was taken to closely observe and monitor the patient while transfer to higher centre for possible surgery

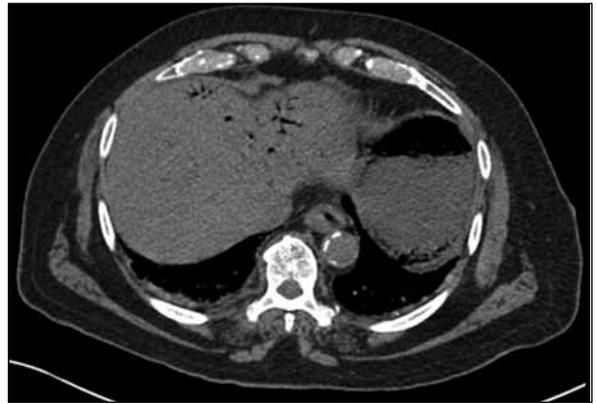


Figure 1. Hepatic portal venous gas in non-contrast CT image (HPVG-1).

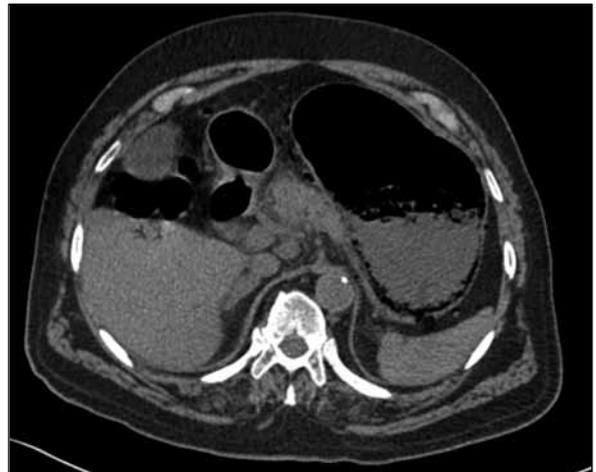


Figure 2. Layering of air bubble along the posterior wall of stomach suggestive of gastric ischemia (HPVG-2).

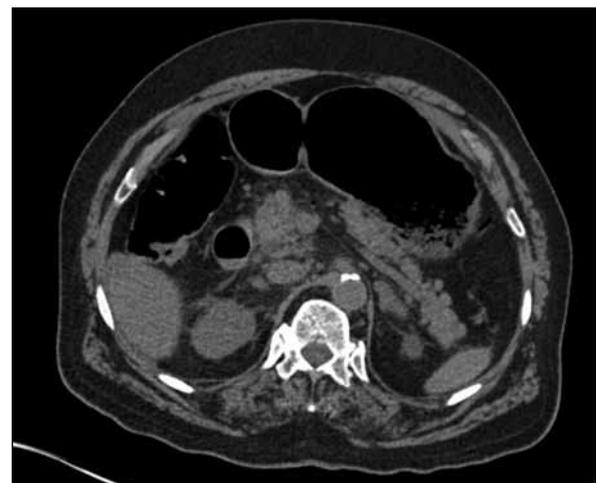
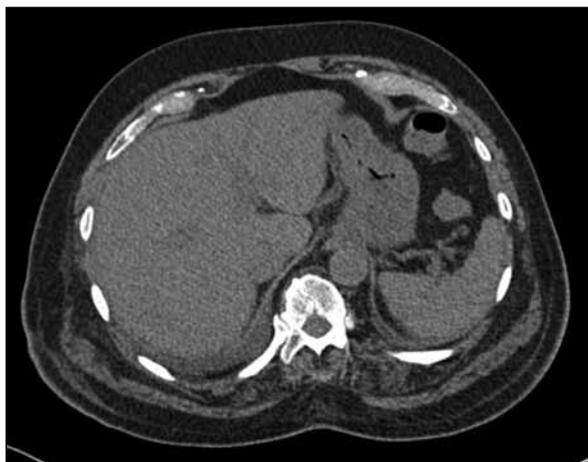


Figure 3. Calcified plaque at the origin of Celiac Axis (HPVG-3).



**Figure 4. Resolution of hepatic portal venous gas and gastric ischaemic change after three days of conservative treatment (HPVG-4).**

was set aside as a contingency plan. Patient was managed conservatively with antibiotics and proton pump inhibitor (PPI). His tachycardia continued for couple of days and then settled. His symptoms, abdominal signs and blood picture improved with time. The CT scan done three days later showed resolution of the HPVG and gastric air bubbles (Figure 4). With his recovery, patient was discharged.

#### DISCUSSION

HPVG was first reported in infants by Wolfe and Evans in 1955. Susman and Senturia described this finding in adult in 1960<sup>[1]</sup>. In 1965 Lazar et al. reported the first survivor with HPVG<sup>[2]</sup>. In 1978 Liebman et al. reviewed 64 cases of HPVG and reported a mortality rate of 75%<sup>[3]</sup>. At that time mere radiologic finding of HPVG warranted laparotomy, except for patients with ulcerative colitis, to assess the extent of bowel necrosis<sup>[4]</sup>.

Now we have growing number of case reports with this condition due to more sophisticated imaging tools. Although ultrasound scan can be used to identify and prognosticate<sup>[5]</sup>. CT scan is found to be the best imaging modality to diagnose HPVG along with the underlying disease condition<sup>[5]</sup>. Years later people started questioning the necessity to explore the abdomen<sup>[6]</sup>. The mortality statistics have improved with time. In 2001, Kinoshita et al. reported the mortality figure of 39% after reviewing 64 cases<sup>[7]</sup>. In 2009, Hussain et al reviewed 275 cases of HPVVG and reported bowel ischemia and mesenteric vascular pathology (61%) as the single most important cause of this condition followed by inflammation of the gastroin-

testinal tract (16%), obstruction and dilatation (9%), sepsis (6%), iatrogenic injury and trauma (3%) and cancer (1%)<sup>[8]</sup>.

Now there is growing evidence from the literature that clinicians should not be merely distracted the by a mere radiologic finding of HPVG as this may occur in severe gastrointestinal diseases, intestinal pseudo obstruction, inflammatory bowel disease and also following endoscopic procedures, nasogastric intubation and barium enema<sup>[9]</sup>. Sometimes this is an incidental radiologic finding<sup>[10]</sup>. Nonetheless, the presence of HPVG mandates further investigations to rule out intra-abdominal catastrophe. The prognosis of such case is determined by the underlying condition causing HPVG. Aaron et al. developed an ABC approach to HPVG management recommending urgent laparotomy (aggressive management) for patients in whom HPVG is detected by CT with concurrent signs of bowel necrosis or ischemia (like lactic acidosis and increased anionic gap) and with mortality approximated at 75%<sup>[11]</sup>.

Stenosis or obstruction of the celiac axis is known to cause gastric ischemia. Risk factors for atherosclerosis are usually present. It can manifest as chronic postprandial abdominal pain which was the case in our patient. Some of the patients develop food phobia due to pain and consequently lose weight. Many patients develop recurrent gastric ulcers which are usually slow to heal. Our patient had an upper gastrointestinal endoscopy six weeks following discharge but this did not reveal any abnormality. Gastric ischemia is known to produce air bubble in the stomach wall<sup>[12]</sup>.

HPVG is an ominous radiological sign of intra-abdominal disaster. Exploratory laparotomy is recommended in patients having hepatic portal venous gas with bowel and gastric ischemia. It is well known from literature that rarity of acute gastric ischemia often delays the diagnosis. Patients initially presenting with mild symptoms can rapidly progress to acute peritonitis, septic shock, and death. Hence patients with acute gastric ischemia should proceed for laparotomy with an aim for partial or total gastrectomy<sup>[13]</sup>. Complete resolution of signs and symptoms in this patient with expectant treatment is quite intriguing and throws light on the reversible nature of gastric ischemia in this case. This could be due to better development of collateral circulation in acute on chronic gastric ischemia cases leading to better prognosis compared to those presenting acutely.

Stomach has abundant vascular supply through five major vessels (right and left gastric, right and left gastroepiploic, and short gastric) abundant minor and collateral supply. Schein et al. showed in cadavers that one patent major artery is sufficient to produce complete gastric wall vascular filling<sup>[14]</sup>. Somervell demonstrated that gastric necrosis in animal models would necessitate the ligation of the right and left gastric arteries, right and left gastroepiploic arteries, and 80% of collaterals<sup>[15]</sup>. Babkin et al. reported that severing most of the arterial supply to dogs' stomachs did not produce in any ischemic changes which were demonstrated with every single case of both venous and arterial occlusion<sup>[16]</sup>.

Stomach being such a vascular organ, a wait and watch policy may be adopted in selected subset of patients having acute on chronic gastric ischemia without haemodynamic compromise. However one should have very low threshold to proceed for exploratory laparotomy if patient fails to improve or shows any sign of deterioration.

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