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Case Series

Computed tomography-detected portal venous gas managed conservatively: a case series emphasising clinical-radiological correlation

Yifan Liu[1,2]*, Geoffrey Stieler[1,3], Michael Auld[1,2]

[1]University of Queensland, Brisbane, Queensland, Australia
[2]Department of Surgery, Ipswich Public Hospital, Ipswich, Queensland, Australia
[3]Department of Medical Imaging, Ipswich Public Hospital, Ipswich, Queensland, Australia

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***Correspondence:**
Dr. Yifan Liu,
E-mail: liu.mbbs.1990@gmail.com

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ABSTRACT

Portal venous gas (PVG), historically considered a critical radiological finding associated with life-threatening conditions such as mesenteric ischemia and sepsis, and often prompting urgent surgical intervention, is increasingly recognised in a wider spectrum of clinical scenarios with the advent of high-resolution computed tomography (CT). This case series describes three patients in whom PVG was identified on CT and successfully managed without surgery, with underlying causes including sepsis from a urinary source with gas-forming organisms, gastric distention with pneumatosis, and suspected ischemic colitis in the setting of preserved hemodynamic stability. All patients were treated conservatively with intravenous antibiotics and demonstrated favourable outcomes, underscoring the importance of interpreting PVG as a radiological sign rather than an automatic surgical indication. Clinical context, hemodynamic stability, laboratory findings, and adjunctive investigations remain central to guiding management, and recognition of benign or iatrogenic causes is essential to avoid unnecessary operative interventions.

Keywords: Computerized tomography, Venous gas, Clinical-radiology

INTRODUCTION

Portal venous gas (PVG) refers to the abnormal presence of gas within the portal venous system. Historically, it has been considered an ominous radiological sign, often associated with sepsis and mesenteric ischemia.[1] Consequently, PVG was linked to poor prognosis, with mortality rates reported as high as 75%.[2] However, with increasing accessibility and resolution of modern CT imaging, PVG is now identified in a broader range of conditions, some of which may not necessitate urgent surgical intervention.

This article presents three cases where PVG was identified on imaging and successfully managed non-operatively in the acute surgical unit, underscoring the importance of clinical correlation in management decisions.

CASE SERIES

Case 1

A 68-year-old male was admitted to the intensive care unit with sepsis originating from cellulitis and a urinary tract infection. Examination revealed extensive cellulitis of the left lower limb extending to the thigh, but no signs of abdominal tenderness or peritonism.

CT of the abdomen and pelvis revealed bilateral staghorn calculi as well as mesenteric and peripheral intrahepatic PVG without evidence of ischemic bowel (Figure 1).

His medical history included atrial fibrillation, poorly controlled type 2 diabetes mellitus, chronic kidney disease, hypertension, and morbid obesity. Laboratory investigations demonstrated a white cell count of 29×10^9/l, CRP of 259 mg/l, and lactate of 2.7 mmol/l. Blood cultures grew pan-sensitive group G *Streptococcus* species, and urine cultures yielded pan-sensitive *Proteus mirabilis*. The patient was treated with ceftriaxone and metronidazole to cover for potential sepsis and recovered without surgical intervention. Conservative management was chosen because there were no clinical or radiological signs of bowel compromise, and the markedly elevated white cell count and CRP were thought to be more consistent with bacteraemia and urosepsis. The patient was reviewed in the surgical clinic four weeks after discharge and remained well. It was hypothesised that the PVG may have resulted from gas-forming *Proteus mirabilis* translocating into the portal system.

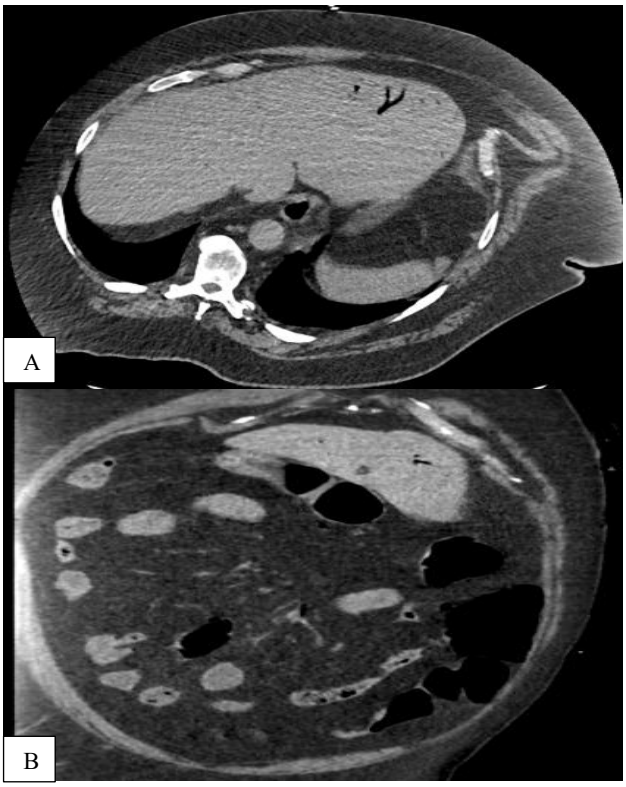

Figure 1 (A and B): Axial slice image (left) and coronal slice image (right) of the abdominal CT with portal venous contrast showing PVG in segment III of the liver.

Case 2

An 82-year-old male presented to the emergency department with a left intertrochanteric fracture. His medical history included hypertension and no history of smoking. On day two of admission, he experienced multiple episodes of vomiting and abdominal distention, without tenderness. CT of the abdomen and pelvis, performed due to concerns for bowel obstruction, showed gastric pneumatosis with associated PVG, suspected to be secondary to gastric distention (Figure 2).

The patient remained hemodynamically stable. Laboratory investigations showed a white cell count of 17×10^9/l and lactate of 3.2 mmol/l; CRP was unavailable. Upper gastrointestinal endoscopy showed gastric inflammatory changes near the gastro-oesophageal junction, with no ulceration or ischemia. An endoscopic photo is shown in Image 2.2. He was managed with a proton pump inhibitor and intravenous ceftriaxone and metronidazole to cover for potential sepsis. Conservative management was chosen because the patient remained clinically well, with no evidence of gastric compromise on examination or gastroscopy. Orthopaedic repair via intramedullary nail was performed on day three, and the patient was transferred to rehabilitation on day 18. He remained stable while in rehabilitation following discharge from the surgical ward.

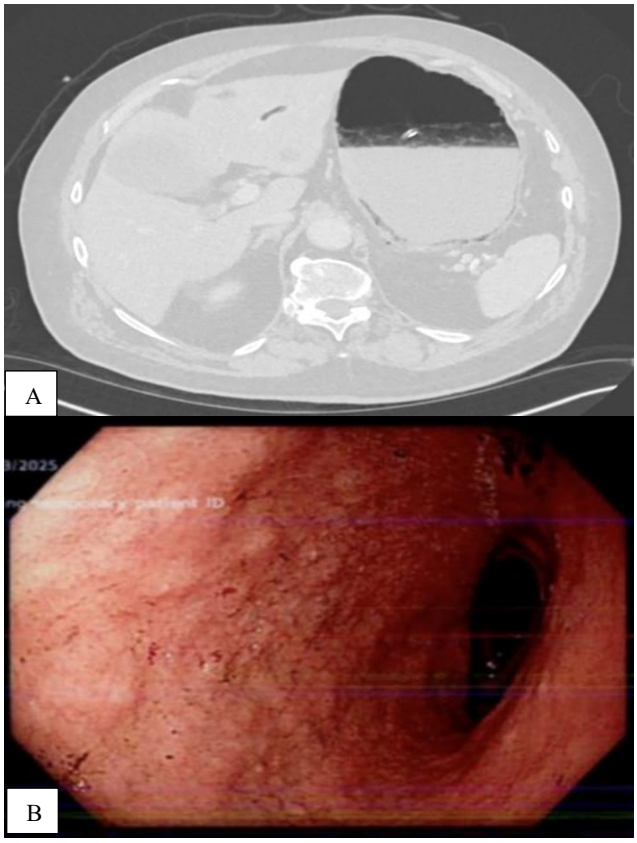

Figure 2: (A) Axial slice image (left) of the abdominal CT with portal venous contrast showing PVG in segment IV of the liver and pneumatosis in the posterior gastric wall. (B) Endoscopic image (right) near the GOJ showing inflammatory changes.

Case 3

A 55-year-old female presented to emergency department with left lower quadrant abdominal pain, nausea, and vomiting. She had no diarrhoea or rectal bleeding. CT of the abdomen and pelvis revealed hyper-enhancement of

terminal ileum to ascending colon with associated PVG, raising concern for ischemic colitis (Figure 3).

Her medical history was significant for previous ischemic small bowel secondary to superior mesenteric artery (SMA) thrombosis eight years earlier, which had required small bowel resection and SMA stenting. CT abdominal angiogram showed a patent SMA without occlusion. The patient remained hemodynamically stable. Laboratory investigations demonstrated a white cell count of 13×10^9/l, CRP of 4 mg/l, and lactate of 1.2 mmol/l. She was treated conservatively with intravenous ceftriaxone and metronidazole to cover for potential sepsis and discharged on day 3. Conservative management was chosen because, despite her pain and previous history of ischemic bowel, she remained clinically well with reassuring laboratory findings overall, including normal CRP and lactate. On phone review 6 weeks after discharge, she remained stable and at baseline.

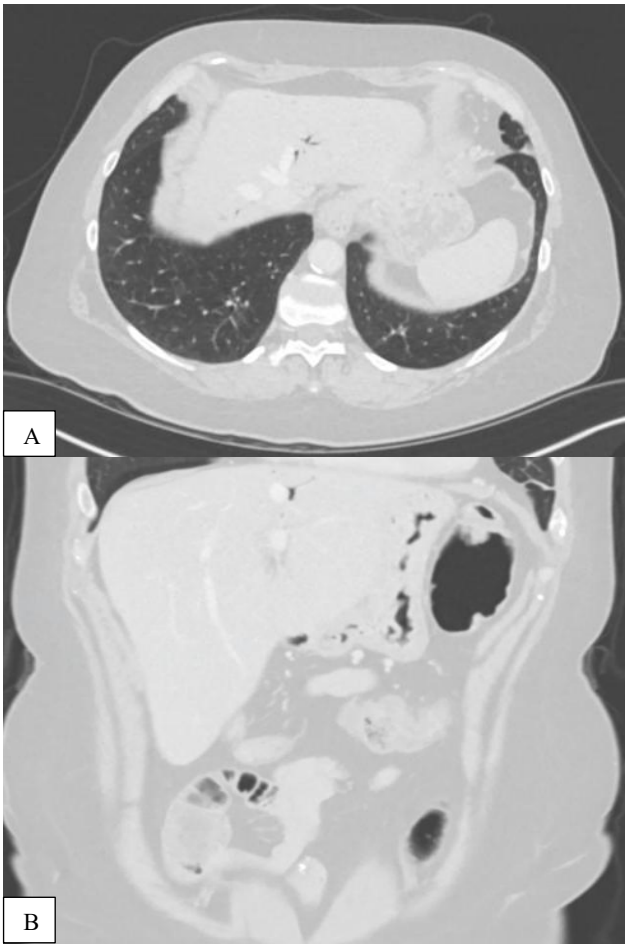

Figure 3 (A and B): Axial slice image (left) and coronal slice image (right) of the abdominal CT with portal venous contrast showing PVG in segment IIA of the liver.

DISCUSSION

PVG was first described by Wolfe et al, who correlated radiographic findings of a vascular pattern of gas in the liver on plain film with post-mortem examinations in 6 infants with necrotising enterocolitis (NEC).[1] All patients had *E. coli* in their postmortem blood cultures, and three patients demonstrated pneumatosis of the bowel wall. They concluded that PVG was caused by intraluminal enteric gas under tension, allowing it to enter the mesenteric veins and collect in the intrahepatic portal system.

In adults, PVG was described by Susman et al 5 years later in 1960, who confirmed that the branching pattern of gas over the liver on plain film originated from the portal system secondary to ischemic bowel.[3] This was novel at the time, since this branching pattern of gas would usually be found in patients with gallstone ileus instead. Since then, multiple sources of PVG have been described and can be separated into iatrogenic and non-iatrogenic sources. Common iatrogenic sources include hepatic transplantation, colonoscopy, especially with biopsy, and radiofrequency ablation of hepatic lesions. Rarer sources include contrast enemas, hepatic artery stenting, and ileus.[4] Non-iatrogenic causes are generally more clinically significant and include bowel ischemia, necrosis, and severe infection, all of which are associated with higher morbidity and mortality.

There are two main hypotheses proposed to explain PVG pathophysiology: mechanical translocation and bacterial production. Mechanical translocation occurs due to mucosal breakdown from inflammation, ischemia, or infection, disrupting the endothelial barriers and allowing gas to enter the mesenteric veins.[5] The bacterial production hypothesis suggests that translocated bacteria produce gas within the bowel wall and portal circulation, radiologically presenting as pneumatosis intestinalis and PVG respectively.[6] Wiot et al hypothesised that bacteraemia is a major contributor to PVG.[7] They were able to isolate Aerobacter aerogenes colonies in antemortem blood cultures, a known gas-forming bacterium producing carbon dioxide and hydrogen. This is also supported by their postmortem analysis of PVG, which revealed a high concentration of carbon dioxide. Given the high solubility of carbon dioxide, its detection in measurable quantities implies ongoing or persistent production. The bacterial production hypothesis likely operates in conjunction with the mechanical translocation theory in the pathogenesis of PVG.

These mechanisms are reflected in the present series. In case 1, systemic infection with a urinary source and bacteraemia may have contributed to PVG formation in the absence of bowel ischemia. In case 2, repeated vomiting and gastric distention likely increased intraluminal pressure sufficiently to cause gastric pneumatosis and secondary PVG. In case 3, despite imaging findings concerning for ischemic colitis and a previous history of ischemic bowel, preserved hemodynamic status, a patent SMA, and reassuring laboratory findings supported an initial trial of conservative management. These three cases were

managed conservatively based on combination of clinical assessment, laboratory findings, and, in case of patient 2, endoscopic investigation. Together, they demonstrate that PVG should not be interpreted in isolation, but rather in conjunction with patient's hemodynamic status, abdominal examination, biochemical profile, and adjunctive investigations where appropriate.

PVG was first identified on plain film X-ray with a radiolucent leaf-like branching pattern over the liver. Liebman et al stated that a gas pattern extending to within 2 cm of the liver periphery is indicative of PVG.[8] This is due to the direction of portal venous blood carrying gas locules towards the liver edge, which is best seen on X-ray performed in the left decubitus position. In contrast, a central presence of gas locules is likely located within the biliary system due to their opposite flow direction.

Sonography has been used to demonstrate PVG, particularly in neonatal patients with suspected NEC. Merritt et al were able to demonstrate highly echogenic particles that represent microbubbles of gas flowing within the portal vein.[9] Aggregates of red blood cells or platelets can produce a similar sonographic appearance. To prevent false positives, hepatic veins and the inferior vena cava should be used as controls for comparison.

In our facility, CT is readily available and has proven to be the most sensitive modality in the diagnosis of PVG, as well as possible underlying pathology at the same time. PVG on CT appears as one or more tubular regions of decreased attenuation consistent with gas in the portal venous system.[10] The presence of PVG in the extra-hepatic portal system may also suggest the possible location of pathology in the absence of other findings, based on crucial knowledge of portal system anatomy.

However, this remains a small case series, and management decisions were individualised to each presentation. These cases therefore support careful clinical correlation rather than suggesting that all patients with PVG can be safely managed non-operatively.

CONCLUSION

PVG is a radiological sign historically associated with bowel ischemia, sepsis, and the need for urgent surgical assessment. However, with widespread use of CT imaging, PVG is increasingly identified in a broader range of clinical contexts, including some cases that may be managed non-operatively. Early differentiation between iatrogenic and non-iatrogenic causes, together with careful assessment of hemodynamic status, abdominal examination, laboratory findings, and adjunctive investigations where appropriate, is essential in determining management. This case series supports the importance of clinical correlation and suggests that PVG, in isolation, should not be regarded as an automatic indication for surgery.

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REFERENCES

1. Wolfe JN, Evans WA. Gas in the Portal Veins of the Liver in Infants: A Roentgenographic Demonstration with Postmortem Anatomical Correlation. Am J Roentgenol Radium Therapy Nuclear Med. 1955;74(3):486-8.
2. Yoo, Soo-Kyung, et al. Clinical Outcomes in Surgical and Non-Surgical Management of Hepatic Portal Venous Gas. Kor J Hepato-Biliary-Pancreatic Surg. 2015;19(4):181-7.
3. Susman N, Senturia HR. Gas Embolization of the Portal Venous System. Am J Roentgenol Radium Therapy Nuclear Med. 1960;83:847-50.
4. Mohammed AH, Khot UP, Thomas D. Portal Venous Gas: Case Report and Review of the Literature. Anaesthesia. 2007;62(4):400-4.
5. Dibra R, Arcangelo P, Giuseppe T, Grazia L, Giovanni T, Vincenzo P, et al. Pneumatosis Intestinalis and Hepatic Portal Venous Gas: Watch and Wait or Emergency Surgery? A Case Report and Literature Review. Am J Case Rep. 2020;21:e923831.
6. Wayne E, Matthew O, Andrew W, Junlin L, Andresen KJ, Kuehn D, et al. Management Algorithm for Pneumatosis Intestinalis and Portal Venous Gas: Treatment and Outcome of 88 Consecutive Cases. J Gastrointest Surg. 2010;14(3):437-48.
7. Wiot JF, Felson B. Gas in the Portal Venous System. Am J Roentgenol Radium Therapy Nuclear Med. 1961;86:920-9.
8. Liebman PR, Patten MT, Manny J, Benfield JR, Hechtman HB. Hepatic-Portal Venous Gas in Adults: Etiology, Pathophysiology and Clinical Significance. Ann Surg. 1978;187(3):281-7.
9. Merritt CR, Goldsmith JP, Sharp MJ. Sonographic Detection of Portal Venous Gas in Infants with Necrotizing Enterocolitis. Am J Roentgenol. 1984;143(5):1059-62.
10. Sebastià C, Quiroga S, Espin E, Boyé R, Alvarez-Castells A, Armengol M, et al. Portomesenteric Vein Gas: Pathologic Mechanisms, CT Findings, and Prognosis. Radiographics: A Review Publication of the Radiological Society of North America, Inc. 2000;20(5):1213-24.

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