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Case Report

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Posted Date: September 25th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10771831/v1

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Additional Declarations: No competing interests reported.

Nonparallel Resolution of Hepatic Portal Venous Gas and Associated Bowel Abnormalities During Nonoperative Management: A Three-Case Series

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Abstract

Background: Hepatic portal venous gas (HPVG) is a radiologic sign that occurs in disorders ranging from reversible mucosal injury to irreversible intestinal ischemia. Management is determined by the underlying disease and physiologic course rather than the gas alone. Less attention has been paid to whether clearance of HPVG parallels recovery of the associated bowel abnormalities. **Case presentations:** Three patients aged 90, 74, and 66 years presented with acute gastrointestinal symptoms, HPVG, mesenteric venous gas, and bowel-wall abnormalities on computed tomography (CT). All were initially hemodynamically stable. Cases 1 and 2 had no peritoneal signs; Case 3 developed rebound tenderness and a marked inflammatory response on hospital day 2, without muscular guarding, shock, or acidemia, and serial lactate values remained below 2.0 mmol/L. In Case 1, venous gas decreased on contrast-enhanced CT on hospital day 2 while small-bowel wall edema with surrounding exudation became apparent. In Case 2, repeat CT on day 2 described absorption of the gas despite transiently increased bowel edema and abdominopelvic fluid. In Case 3, the gas had resolved by day 4 while pneumatosis and incomplete obstruction persisted. The bowel abnormalities subsequently improved, and all three patients were discharged without surgery. Follow-up CT at 1–3 months showed no recurrent HPVG. **Conclusion:** Across these cases, HPVG decreased or disappeared

before the bowel abnormalities fully recovered. Gas clearance therefore did not indicate complete bowel recovery or justify shorter observation. Continued nonoperative care requires serial abdominal examination, hemodynamic and metabolic assessment, CT review of bowel findings, and prompt surgical reassessment when warning findings appear.

Keywords: hepatic portal venous gas; pneumatosis intestinalis; nonoperative management; serial computed tomography; bowel ischemia; case series

1 Introduction

Hepatic portal venous gas (HPVG) is a radiologic finding rather than a diagnosis. It occurs in a broad range of conditions, including intestinal ischemia or necrosis, bowel obstruction, intra-abdominal inflammation or infection, and iatrogenic injury [1]. Its clinical significance therefore depends on the underlying cause and the patient's physiologic course. The immediate concern is to identify progressive or irreversible bowel ischemia without treating the presence or disappearance of HPVG as a management decision in itself [2]. Serial imaging in a 20-patient series showed that portal venous gas could disappear within several days, but the study evaluated HPVG primarily in relation to etiology, treatment, and mortality rather than as a surrogate for recovery of associated bowel abnormalities [3]. Whether pneumatosis, bowel-wall edema, fluid, and obstruction resolve on the same timescale as HPVG remains uncertain. We report three older patients with different comorbidities and risk trajectories who were managed without surgery. The series focuses on the nonparallel evolution of HPVG and bowel findings, the observations used to continue or reconsider nonoperative care, and the limits of drawing treatment indications from successful outcomes. The report was prepared with reference to the CARE reporting principles [4].

2 Case presentations

2.1 Case 1

A 90-year-old woman presented with 5 hours of predominantly epigastric pain, nausea, vomiting, diarrhea, and fresh hematochezia. Her history included hypertension, coronary artery disease, and two previous admissions for similar gastrointestinal episodes that had improved without surgery. She also reported unsupervised use of norfloxacin. On admission, her temperature was 36.1 °C, heart rate 62 beats/min, respiratory rate 18 breaths/min, and blood pressure 160/67 mmHg. She had generalized abdominal tenderness without rebound tenderness or muscular guarding. Shock and altered consciousness were absent. The white blood cell count was 11.89 $\times 10^9$/L. Arterial blood gas analysis showed pH 7.359, base excess −5.2 mmol/L, HCO_3^- 19.4 mmol/L, and lactate 1.41 mmol/L.

Admission-day unenhanced abdominal CT demonstrated mesenteric venous gas and extensive intrahepatic portal venous gas (Figure 1A). The selected admission-day bowel image showed no obvious small-bowel wall edema (Figure 1D). Contrast-enhanced CT on hospital day 2 showed less intrahepatic portal venous gas (Figure 1B), whereas small-bowel wall edema with surrounding exudation had become apparent (Figure 1E); colonic distention with gas and fluid was also present. Colorectal surgical assessment for hematochezia found no active anorectal bleeding. Treatment included bowel rest, gastrointestinal decompression, antimicrobial therapy, fluid replacement, nutritional support, and symptom-directed care. On hospital day 2, a multidisciplinary review did not support extensive bowel ischemia. Stable vital signs, improving pain and tenderness, absence of peritoneal signs, low lactate, the previous similar episodes, and the overall imaging pattern were considered together, and immediate surgery was deferred. Limited ischemic mucosal injury or ischemic colitis in the setting of infection and volume depletion was considered possible but was not confirmed.

On day 3, pH was 7.397, base excess −4.7 mmol/L, HCO_3^- 19.2 mmol/L, and lactate 1.83 mmol/L. Examination on day 4 showed continued improvement without rebound tenderness or guarding. Repeat unenhanced CT on day 5 showed resolution of the portal and mesenteric venous gas and improvement of small-bowel edema and exudation (Figures 1C and 1F). Examinations on days 8 and 9 showed continued improvement without rebound tenderness or guarding. She was discharged on hospital day 9. At 3 months, she had experienced no recurrence, and follow-up CT showed no HPVG.

2.2 Case 2

A 74-year-old man presented with 5 days of diarrhea and 6 hours of periumbilical discomfort, abdominal distention, and palpitations. His history included two pulmonary emboli, long-term rivaroxaban treatment, and hypertension. On admission, his temperature was 36.6 °C heart rate 74 beats/min, respiratory rate 20 breaths/min, and blood pressure 163/78 mmHg. The abdomen was soft without tenderness, rebound tenderness, or guarding; shock and altered consciousness were absent. The white blood cell count was 11.43 $\times 10^9$/L. Arterial blood gas analysis showed pH 7.402, base excess −1.85 mmol/L, HCO_3^- 22.8 mmol/L, and lactate 3.00 mmol/L.

Admission-day unenhanced CT showed portal and mesenteric venous gas, together with thinning and pneumatosis of some bowel segments. Extensive intrahepatic portal venous gas is shown in Figure 2A, while small-bowel and mesenteric abnormalities are represented in Figure 2D. Subsequent contrast-enhanced CT performed with mesenteric CT angiography showed newly apparent edema and thickening of other small-bowel segments and a marked reduction in intrahepatic portal venous gas; the superior and inferior mesenteric arteries and veins showed no definite abnormality. The symptoms and abdominal findings remained mild. He received bowel rest, gastrointestinal decompression, antimicrobial therapy, fluid replacement, and close observation. On hospital day 2, he passed dark-red stool, and bilateral lower-extremity deep venous thrombosis was identified. Anticoagulation was adjusted while gastrointestinal bleeding and coagulation indices were monitored.

Repeat unenhanced CT on day 2 described absorption of the portal and mesenteric venous gas (Figure 2B), although small-bowel edema and abdominopelvic fluid had initially increased (Figure 2E). A later CT on the same day showed slight improvement in bowel edema and a reduction in fluid. On day 3, pH was 7.430, base excess 1.0 mmol/L, HCO_3^- 25.1 mmol/L, and lactate had decreased to 1.42 mmol/L. Examinations on days 2 and 5 remained negative for tenderness and peritoneal signs, and the gastrointestinal symptoms resolved. By day 7, HPVG remained absent, bowel-wall thickening had improved further, and the fluid was no longer detected (Figures 2C and 2F). He was discharged on hospital day 11. At 3 months, he had no further hematochezia; lower-extremity imaging showed resolution of the deep venous thrombosis, and CT showed no HPVG.

2.3 Case 3

A 66-year-old man presented with periumbilical and right-sided abdominal pain, diarrhea, nausea, and vomiting. He had tetralogy of Fallot, severe pulmonary hypertension, severe tricuspid regurgitation, tricuspid valve prolapse, frequent atrial and ventricular premature beats, and poor cardiac function. On admission, his temperature was 36.8 °C, heart rate 90 beats/min, respiratory rate 20 breaths/min, and blood pressure 125/70 mmHg. Abdominal tenderness was present without rebound tenderness or guarding. He had neither shock nor altered consciousness. Admission-day unenhanced abdominal CT showed dilatation and pneumatosis of the ascending and transverse colon with mesenteric and portal venous gas (Figures 3A and 3D). Subsequent mesenteric arterial and venous CT angiography showed no thromboembolism. Arterial blood gas analysis showed pH 7.430, base excess −3.43 mmol/L, HCO_3^- 21.6 mmol/L, and lactate 1.11 mmol/L.

A gastrointestinal surgical consultation on hospital day 1 noted the absence of mesenteric vascular occlusion and the high operative risk associated with his cardiopulmonary disease. Antimicrobial and supportive treatment, controlled fluid administration, repeat imaging, and exploration if the condition deteriorated were recommended. On day 2, he passed one brick-red loose stool and developed diffuse tenderness and rebound tenderness without muscular guarding. The white blood cell count was 9.53 $\times 10^9$/L, procalcitonin 29.11 ng/mL, interleukin-6 708.50 pg/mL, C-reactive protein 285.16 mg/L, and N-terminal pro-B-type natriuretic peptide 3779.06 pg/mL. Transfer to intensive care was recommended, but the family declined. Antimicrobial therapy was escalated to meropenem. Arterial blood gas analysis showed pH 7.375, base excess −5.4 mmol/L, HCO_3^- 18.9 mmol/L, and lactate 1.63 mmol/L.

Rebound tenderness resolved by hospital day 3, and subsequent examinations and inflammatory markers improved. On day 4, repeat unenhanced CT showed resolution of the portal and mesenteric venous gas (Figure 3B), with residual pneumatosis and persistent colonic dilatation, multiple air-fluid levels, and findings compatible with incomplete bowel obstruction (Figure 3E). Arterial blood gas analysis showed pH 7.427, base excess −4.4 mmol/L, HCO_3^- 18.8 mmol/L, and lactate 1.83 mmol/L. Stool testing on day 5 was positive for the *Clostridioides difficile* toxin B gene, and oral vancomycin was then administered. Because the venous gas had already resolved, the HPVG could not be attributed to *C. difficile* alone. Unenhanced CT on day 9 showed

continued absence of HPVG and further improvement of the obstructive bowel findings (Figures 3C and 3F). Surgical reassessment documented passage of stool and flatus and supported continued observation. He was discharged after 2 weeks. At 1 month, he had no further hematochezia, and follow-up CT showed no HPVG.

3 Discussion

Recent case series illustrate the clinical heterogeneity of HPVG. Wei et al. reported 21 patients with an overall mortality of 28.6%; peritonitis was associated with surgical intervention [5]. In a separate 25-patient series, 18 patients underwent emergency surgery and seven received conservative treatment; 12 of the surgical patients and three of the conservatively treated patients recovered [6]. These data do not establish a general indication for either strategy. The underlying lesion, physiologic severity, and selection for surgery strongly influence the observed outcomes.

Radiologic risk assessment should consider gas distribution and accompanying bowel findings rather than the amount of intrahepatic gas alone. Among 50 patients with portal venous gas or pneumatosis, poor bowel-wall enhancement, mesenteric fat stranding, extrahepatic portal venous gas, advanced age, and renal disease were associated with ischemic disease; extrahepatic gas was the only factor independently associated with the study's high-risk category [7]. In a seven-patient HPVG series, shock, ascites, and peritonitis accompanied intestinal necrosis and poor outcome [8]. Cases 1 and 2 remained free of peritoneal signs and shock. Case 3 was less reassuring because rebound tenderness developed on day 2. Nonoperative treatment was continued initially in the setting of high operative risk, absence of mesenteric vascular occlusion, and the family's refusal of intensive care transfer. Subsequent resolution of rebound tenderness by day 3, maintained hemodynamic stability, pH of at least 7.375, serial lactate values below 2.0 mmol/L, and improvement on repeat imaging and later surgical reassessment supported continued observation. These features did not establish that the bowel lesion was benign.

The most consistent observation in this series was the difference between the rate of HPVG clearance and the rate of bowel recovery. In Case 1, venous gas decreased on contrast-enhanced CT on day 2 even though small-bowel wall edema with surrounding exudation had become apparent. In Case 2, the venous gas was described as absorbed on day 2 while bowel edema and abdominopelvic fluid had initially increased; the bowel findings began to improve on a later scan that day and had improved further by day 7. In Case 3, the gas was no longer visible on day 4, but pneumatosis and incomplete obstruction remained until later improvement. Thus, HPVG burden and the associated bowel process changed at different rates in all three patients. Clearance of the gas did not establish that intestinal injury had resolved, and the residual bowel abnormalities required continued clinical interpretation.

Available serial-imaging reports support the need to separate these trajectories. In a 2026 cohort of 16 conservatively treated patients, complete gas resolution was documented in several patients after control of the underlying disease, yet in-hospital mortality was 62.5% [9]. In a 96-year-old man, portal venous gas had completely resolved by day 3 while a small amount of gastric wall gas remained [10]. The present

series adds two distinct patterns: an early decrease or reported absorption of venous gas despite temporarily worsening bowel changes, and complete gas disappearance while pneumatosis or obstruction persisted. The repeated finding across three different clinical backgrounds supports reporting HPVG and bowel-wall evolution separately rather than describing repeat CT simply as improved or resolved.

The three cases also illustrate different degrees of risk during observation. Case 1 had improving symptoms, low lactate, and multidisciplinary review. Case 2 had an initially elevated lactate that fell rapidly and consistently benign abdominal examinations. Case 3 developed rebound tenderness and marked inflammation despite stable perfusion indices. An older patient with ischemic colitis and HPVG improved after conservative treatment when surgery was declined [11]. A surgically unfit patient with rheumatic heart disease and warfarin-induced coagulopathy also recovered with conservative care [12]. In contrast, an 82-year-old patient on long-term hemodialysis survived two episodes of portal venous gas but died during a third episode accompanied by hypotension, considerable ascites, and linear pneumatosis [13]. A separate three-case report described HPVG presenting with severe shock, with two survivors and one death [14]. These reports show why comorbidity and operative risk must be weighed without allowing them to replace serial assessment for ischemia.

The precise cause of HPVG could not be established because none of the patients underwent surgery or histopathologic assessment. In Cases 1 and 2, gastrointestinal bleeding, bowel-wall abnormalities, and the clinical course were compatible with ischemic mucosal injury, but irreversible ischemia was neither demonstrated nor pathologically excluded. In Case 3, low-flow injury, infection, altered motility, and mucosal disruption may all have contributed. The positive *C. difficile* toxin B gene result was clinically relevant, but the venous gas had resolved before targeted treatment; it therefore cannot explain the radiologic course by itself.

During nonoperative care, each reassessment should record abdominal findings, hemodynamic and mental status, lactate and acid-base measurements, vascular and bowel findings on CT, and the reason for continuing or changing management. Persistent or progressive peritoneal signs, shock, worsening lactate or acidosis, poor bowel-wall enhancement, increasing ascites, or other evidence of irreversible injury should prompt immediate surgical reassessment. This is consistent with acute mesenteric ischemia guidance, in which peritonitis is treated as evidence of likely irreversible bowel injury requiring prompt surgery [15]. Conversely, early disappearance of HPVG should not shorten observation while bowel findings or clinical abnormalities remain unresolved.

4 Limitations

This retrospective single-center series includes only three surviving patients and is subject to selection and survivor bias. The cases were heterogeneous, and management was individualized rather than standardized. No operative or histopathologic findings were available, so intestinal necrosis was not directly excluded. Follow-up lasted 1–3 months. The series cannot establish indications for conservative treatment or the safety of a particular monitoring interval.

5 Conclusion

In these three patients, HPVG decreased or disappeared before the accompanying bowel abnormalities had fully improved. Clearance of portal venous gas did not mark completion of bowel recovery. Decisions to continue nonoperative care depended on serial abdominal findings, perfusion and metabolic status, and repeat assessment of the bowel. New or progressive peritoneal signs, hemodynamic instability, worsening lactate or acidosis, or imaging evidence of irreversible intestinal injury should trigger urgent surgical reassessment.

6 Tables

Table 1 Comparative clinical features, decision points, and outcomes

Feature	Case 1	Case 2	Case 3
Age/sex	90-year-old woman	74-year-old man	66-year-old man
Key background	Coronary artery disease; two similar gastrointestinal episodes	Previous pulmonary emboli; long-term anticoagulation; inpatient DVT	Tetralogy of Fallot; severe pulmonary hypertension and tricuspid regurgitation; poor cardiac function
Abdominal course	Tenderness improved; no rebound tenderness or guarding	No tenderness, rebound tenderness, or guarding on days 1, 2, and 5	Day-2 rebound tenderness without guarding; resolved by day 3
Lactate/pH course	Day 1: 1.41 mmol/L/7.359; day 3: 1.83 mmol/L/7.397	Day 1: 3.00 mmol/L/7.402; day 3: 1.42 mmol/L/7.430	Days 1/2/4: 1.11/1.63/1.83 mmol/L and pH 7.430/7.375/7.427
Vascular/ischemia assessment	Multidisciplinary clinical and imaging review did not indicate extensive bowel ischemia	CTA: no definite mesenteric arterial or venous abnormality	CTA: no mesenteric arterial or venous thromboembolism
Early HPVG course	Day 2 contrast-enhanced CT: venous gas decreased	Day 2 CT: portal and mesenteric venous gas described as absorbed	Day 4: portal and mesenteric venous gas resolved
Bowel findings after early gas improvement	Day 2: small-bowel wall edema and surrounding exudation became evident	Day 2: bowel edema and abdominopelvic fluid increased	Day 4: pneumatosis and incomplete obstruction persisted
Later bowel recovery	Day 5: edema and exudation improved	Later on day 2: edema and fluid slightly improved; day 7: further improvement and no fluid	Day 9: pneumatosis/obstructive findings urther improved
Decision support	Day-2 multidisciplinary review; immediate surgery deferred	Serial clinical and imaging assessment	Surgical review on days 1 and 9; operative risk considered
Outcome and follow-up	Discharged day 9; at 3 months no recurrence and no HPVG	Discharged day 11; at 3 months no hematochezia, no DVT, and no HPVG	Discharged after 2 weeks; at 1 month no hematochezia and no HPVG

Abbreviations: CT, computed tomography; CTA, computed tomography angiography; DVT, deep venous thrombosis; HPVG, hepatic portal venous gas.

7 Figures

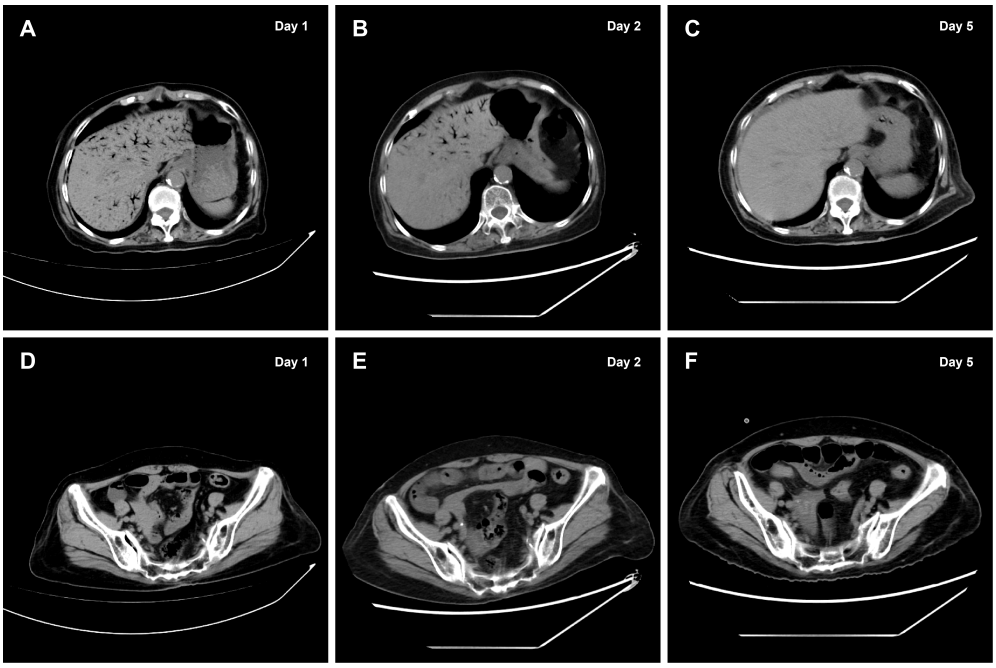


Fig. 1 Representative axial unenhanced CT images from Case 1. (A, D) Day 1 unenhanced CT: extensive HPVG (A) and no obvious small-bowel wall edema (D). (B, E) Day 2 contrast-enhanced CT: reduced HPVG (B) despite newly apparent small-bowel wall edema and mild surrounding exudation (E). (C, F) Day 5 unenhanced CT: absent HPVG (C) and improved bowel abnormalities (F).

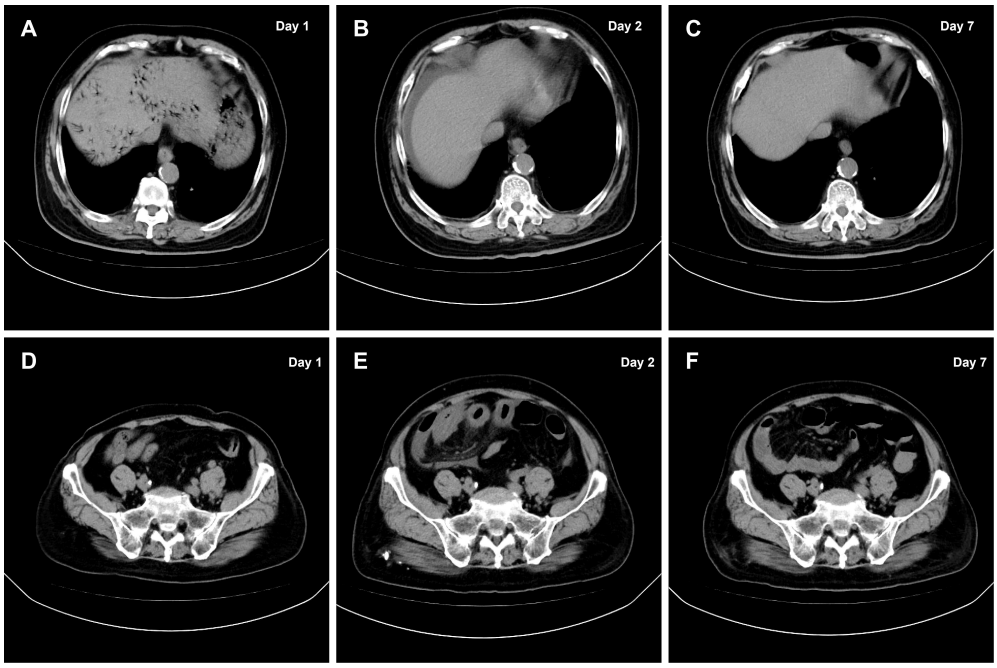


Fig. 2 Representative axial unenhanced CT images from Case 2. (A, D) Day 1: extensive HPVG (A) with small-bowel and mesenteric edema (D). (B, E) Day 2: absent HPVG (B) despite increased small-bowel wall edema and abdominopelvic fluid (E). (C, F) Day 7: continued absence of HPVG (C), improved wall thickening, and no residual fluid (F).

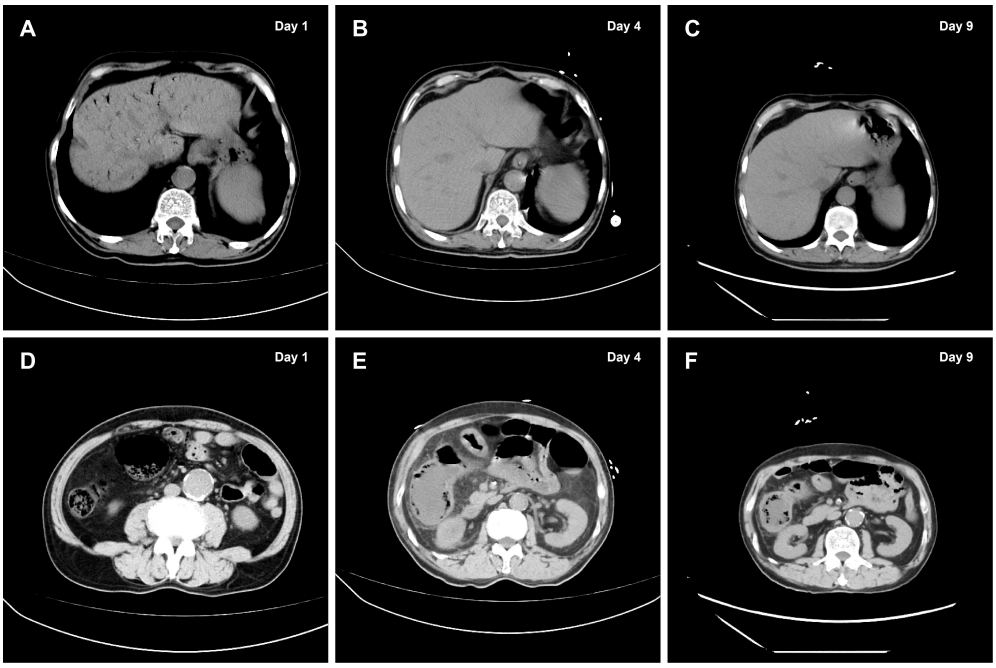


Fig. 3 Representative axial unenhanced CT images from Case 3. (A, D) Day 1: HPVG (A) with colonic dilatation (D). (B, E) Day 4: absent HPVG (B) despite persistent colonic dilatation and multiple air-fluid levels compatible with incomplete obstruction (E). (C, F) Day 9: continued absence of HPVG (C) and improvement of the obstructive bowel findings (F).

Funding. This research received no external funding.

Author contributions. XZ conceived and designed the study. JW collected the clinical data and drafted the manuscript. XYZ and XN provided research support. SC reviewed the manuscript. ZG provided guidance on CT image interpretation and critically revised the manuscript. All authors read and approved the final manuscript.

Declarations

Ethics approval and consent to participate. The study was approved by the Ethics Committee of Shijiazhuang People's Hospital (Approval No. 2026117) and was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent for the use of clinical information in this case series was obtained from all three patients or their legally authorized representatives.

Consent for publication. Written informed consent for publication of the patients' clinical details and accompanying images was obtained from all three patients or their legally authorized representatives.

Competing interests. The authors declare that they have no financial, commercial, or personal relationships that could be perceived as potential conflicts of interest related to this case series.

Data availability. The data supporting this case series are contained in the patients' clinical records and are not publicly available because of privacy restrictions. De-identified information may be available from the corresponding author on reasonable request and subject to institutional requirements.

Materials availability. Not applicable.

Code availability. Not applicable.

Acknowledgements. The authors gratefully acknowledge the medical staff of the Department of Gastroenterology, Shijiazhuang People's Hospital, for their assistance in the clinical management of the patients and preparation of the relevant case materials.

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