Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Hepatic Portal Venous Gas After Hematopoietic Stem Cell Transplantation：A Case Report

Wenyu Min
Huazhong University of Science and Technology
Han Yan
Huazhong University of Science and Technology
Wei Shi
shiwei076@hust.edu.cn
Huazhong University of Science and Technology

Case Report

Keywords: Hepatic portal venous gas, Hematopoietic stem cell transplantation, Graft-versus-host disease, Intestinal obstruction

Posted Date: November 23rd, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11532662/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Hepatic portal venous gas (HPVG) is an imaging sign. Surgical intervention was frequently recommended in previous clinical practice. HPVG secondary to hematopoietic stem-cell transplantation (HSCT) is extremely rare, with only two cases reported in the literature to date, and no standardized and unified diagnostic and therapeutic protocol has been established. This article reports an elderly male patient with acute myeloid leukemia, who developed diarrhea one month after allogeneic hematopoietic stem-cell transplantation, followed by progressively worsening nausea, abdominal pain and distension. Abdominal CT revealed hepatic portal venous gas complicated by incomplete small-bowel obstruction and colonic ileus. Markedly elevated C-reactive protein and procalcitonin indicated severe bacterial infection. Compared with previously reported cases, this patient suffered from concurrent intestinal obstruction and substantial infection. It was presumed that graft-versus-host disease injured the intestinal mucosal barrier, which triggered secondary infection due to gut microbiota translocation. Elevated intraluminal pressure caused by intestinal obstruction jointly contributed to the development of HPVG. Multidisciplinary surgical consultation ruled out intestinal ischemia and necrosis, and surgical intervention was avoided. Conservative treatment was administered. The patient's abdominal pain was relieved on the first day of treatment. Follow-up CT on day 7 showed complete disappearance of portal-venous gas, and the patient achieved a favorable prognosis. Data from the present case and previously published cases suggest that even when HPVG occurs after HSCT and is complicated by intestinal obstruction and severe infection, HPVG is not an absolute indication for surgery once high-risk factors such as intestinal ischemia and necrosis are excluded. Individualized conservative treatment can yield satisfactory clinical outcomes.

Introduction

Hepatic portal venous gas (HPVG) is an imaging sign rather than an independent disease, often complicated with digestive system diseases, and is mainly characterized by branching radiolucent areas visible on CT scans within 2 cm beneath the hepatic capsule. HPVG was first identified and described in 1955 by Wolfe and Evans in an infant with necrotizing enterocolitis[1]. A subsequent retrospective study in 1978 reported that the mortality rate of adult patients with HPVG reached 75%, and recommended surgical intervention for all adult patients with HPVG except those with stable ulcerative colitis[2]. However, a 2009 study proposed that with advances in imaging technology and diagnostic and therapeutic modalities, the early diagnosis rate of HPVG has increased significantly, while the overall mortality is not as high as previously thought but correlates with the primary disease. Such as patients with HPVG complicated with intestinal ischemia or necrosis have a mortality rate as high as 85% and require aggressive surgical treatment[3]. No dedicated international guidelines for HPVG have been established to date. This paper presents a case of HPVG in an elderly male leukemia patient after hematopoietic stem cell transplantation, whose symptoms improved significantly following conservative treatment.

Case Report

The patient was a man in his sixties admitted with a 1-week history of nausea, abdominal pain and distension. He had a prior diagnosis of acute myeloid leukemia (AML) and received allogeneic hematopoietic stem cell transplantation (allo-HSCT) one month before admission. The conditioning regimen consisted of busulfan plus fludarabine. Graft-versus-host disease (GVHD) prophylaxis included tacrolimus, mycophenolate mofetil and anti-CD25 monoclonal antibody. The donor was his son, with an HLA match of 6/10. Neutrophil engraftment occurred on day + 9 and platelet engraftment on day + 15 after stem cell infusion.

The patient developed symptoms during the post-transplant follow-up period and had diarrhea prior to admission. Differential considerations were as follows: conditioning-related gastrointestinal toxicity and engraftment syndrome typically occur within 1–2 weeks after stem cell infusion. Since symptom onset was approximately one month post-infusion with successful hematopoietic engraftment, these two entities were less likely. The patient denied a history of contaminated food intake. Outpatient tests for adenovirus, rotavirus, cytomegalovirus, Epstein–Barr virus, Clostridioides difficile, and other stool pathogens were all negative, ruling out viral diarrhea and C. difficile-associated diarrhea. Therefore, gastrointestinal acute graft-versus-host disease (aGVHD) was the leading diagnosis for his diarrhea. After outpatient symptomatic management (oral montmorillonite powder and adjustment of immunosuppressant doses), diarrhea improved; however, progressive nausea, abdominal pain and distension developed, prompting hospital admission.

At admission, his heart rate was 95 beats per minute and blood pressure was 130/79 mmHg. Physical examination revealed abdominal distension, faint bowel sounds on auscultation, and tympanic percussion note. Laboratory tests showed a white blood cell count of 24.69×10^9/L, C-reactive protein (CRP) 257 mg/L and procalcitonin (PCT) 67.92 ng/mL, indicating severe bacterial co-infection. No defecation was observed after admission, and bowel sounds were absent on physical examination. Abdominal CT demonstrated extensive arboriform gas within the liver, partially located in the main portal vein and its branches (within 2 cm beneath the hepatic capsule), consistent with the imaging features of hepatic portal venous gas. Multisegmental small bowel gaseous distension with large air-fluid levels was also noted, suggestive of partial small bowel obstruction. Colonic collapse combined with clinical signs indicated colonic paralysis(Figure A and C).The patient was definitively diagnosed with HPVG complicated by partial small-bowel obstruction and colonic paralysis.

After admission, the patient developed transient tachyarrhythmic atrial fibrillation, which resolved following temporary amiodarone administration. Surgical consultation was obtained. Although portal venous gas was present, there was no evidence of intestinal ischaemia or necrosis. Given the new-onset atrial fibrillation, surgical intervention was not recommended after comprehensive assessment, and conservative management was adopted. Management included nil per os, nasogastric decompression, parenteral nutrition, octreotide and proton pump inhibitors to suppress secretion of gastric acid, pancreatic enzymes and digestive fluids and reduce intraluminal fluid accumulation. For infection

control, meropenem, teicoplanin and other potent antibiotics were initiated because of incompletely recovered immune function. Antibiotics were de-escalated to a third-generation cephalosporin once symptoms improved and infection markers declined. After excluding mechanical obstruction, appropriate glycerine enema was administered for colonic paralysis to facilitate distal transit of small-bowel contents. Ciclosporin and corticosteroids were given for graft-versus-host disease. The patient's abdominal pain improved within one day, and bowel movement was achieved after enema. Marked relief of pain and distension was observed by day 3, with spontaneous passage of loose yellow stools and resolution of obstructive symptoms. Repeat abdominal CT on day 7 showed complete disappearance of intrahepatic gas, indicating a favourable treatment response (Figures B, D). Subsequent treatment focused on ongoing management of leukaemia and graft-versus-host disease. The clinical course is summarised in Table 1.

Table 1
The clinical course

Time point	Clinical manifestations	Auxiliary examinations	Clinical interventions	Clinical outcome
Approximately Day + 30 (pre-admission outpatient)	Diarrhea followed by progressive nausea, abdominal pain and distension	Stool and viral pathogen tests all negative	Montmorillonite powder, immunosuppressant dose adjustment	Diarrhea improved; abdominal pain and distension worsened, patient admitted to hospital
Day of admission	Abdominal distension; bowel sounds diminished to absent; tympanic percussion; transient tachyarrhythmic atrial fibrillation	Markedly elevated WBC, CRP and PCT; CT: HPVG, incomplete small-bowel obstruction, colonic ileus	Fasting, gastrointestinal decompression, parenteral nutrition; octreotide + PPI; meropenem + teicoplanin; amiodarone for cardioversion; glycerol enema; cyclosporine + glucocorticoids for anti-GVHD therapy; surgical consultation; intestinal ischemia/necrosis excluded, surgery withheld	Atrial fibrillation cardioverted; conservative strategy confirmed
Treatment day 1	Reduced abdominal pain; defecation after enema	-	Continued conservative treatment	Abdominal pain relieved, bowel movement restored
Treatment day 3	Markedly improved abdominal pain and distension; spontaneous yellow loose stools	Down-trending infection-related markers	Ongoing anti-GVHD therapy and nutritional support	Symptoms of intestinal obstruction resolved
Treatment day 7	Near-complete resolution of abdominal pain and distension	Follow-up CT: complete disappearance of portal-venous gas	Antibiotic de-escalation to third-generation cephalosporins; continued management for leukemia and GVHD	Radiological resolution of HPVG; favourable prognosis

Figure Unenhanced abdominal CT at admission (A, C) and after conservative management (B, D), all axial. (A) Extensive branching gas reaches the hepatic periphery within 2 cm of the capsule (red arrow), indicating hepatic portal venous gas; the stomach is markedly distended (asterisk). (C) Multiple small-bowel loops are dilated and gas-filled (white arrows), in keeping with incomplete small-bowel

obstruction. Follow-up CT shows near-complete resolution of the portal venous gas with a homogeneous liver (B, red arrow) and decompression of the bowel (D).

Discussion

Existing reviews categorize the etiologies of HPVG into high-risk, intermediate-risk and low-risk groups according to hazard levels. High-risk etiologies mainly include intestinal ischemia or necrosis and intra-abdominal infection; intermediate-risk etiologies comprise inflammatory bowel disease and diverticulitis; low-risk etiologies include iatrogenic endoscopy, barium meal examination, liver transplantation and other abdominal operations. Other etiologies such as chemotherapy and malignancy have been reported in relatively few case reports[3–5]. The pathogenesis of HPVG has not been fully elucidated. It is generally accepted that HPVG develops due to impaired intestinal barrier, through which intraluminal gas refluxes into the hepatic portal vein. Meanwhile, intestinal bacterial infections (e.g., Escherichia coli, Klebsiella and gas-producing bacteria) further increase intraluminal gas volume and raise intestinal pressure, facilitating gas entry into the blood circulation and thereby aggravating HPVG[3–5]. The pathogenesis in this case is consistent with the mechanism described above: intestinal mucosal injury secondary to gastrointestinal GVHD served as the initiating insult. Concomitant partial small-bowel obstruction increased intraluminal pressure with gas and fluid retention, and infection resulting from bacterial translocation further contributed to the development of HPVG.

Treatment mainly consists of conservative management and surgical intervention. Common conservative measures include therapy targeting the underlying disease, gastrointestinal decompression, nutritional support and anti-infection treatment. Individualized regimens should be formulated according to patient characteristics. The core clinical question is whether surgical intervention is required for HPVG, which depends on the risk grade of the primary disease. In a retrospective study published in 2003, Iannitti et al.[6] enrolled 26 patients with HPVG, among whom 11 received surgery. The primary diseases in the surgical group were mostly intestinal ischemia, intestinal necrosis, tumor rupture and peritonitis, with operative procedures including bowel resection, repair or ostomy. The survival rate of the surgical group was 73%. The remaining 15 patients were managed conservatively, whose primary conditions were mostly transient postoperative gas collection and mild pancreatitis, with a survival rate of 60%. The six deaths were mainly attributed to advanced malignancy or severe infection. Accordingly, they proposed that HPVG is not an absolute indication for surgery. Comprehensive evaluation combining clinical manifestations and CT findings is required to determine surgical necessity so as to avoid unnecessary operations.

Subsequently, Nelson et al.[7] further proposed the ABC stratified management strategy for HPVG: A (Aggressive): HPVG detected on plain radiography or on CT accompanied by intestinal necrosis, ischemia or peritonitis. Such patients carry a high mortality rate and require immediate laparotomy. B (Be careful): HPVG complicated with peptic ulcer, intra-abdominal abscess, inflammatory bowel disease, etc., with a mortality of approximately 20%–30%. Aggressive conservative treatment including nil per os, anti-infection therapy and dynamic monitoring should be initiated, and surgery performed when

necessary. C (Conservative): HPVG occurring after endoscopy, surgery, liver transplantation, or asymptomatic HPVG. This subgroup has low mortality and only requires symptomatic management without surgery. For instance, in one reported case[8], a 60-year-old HPVG patient had no obvious signs of intestinal necrosis or ischemia on CT, but presented with severe abdominal pain, absent bowel sounds and elevated lactate. After assessment, clinicians considered progressive intestinal ischemia or necrosis, and emergency exploratory laparotomy was performed. The patient recovered well postoperatively. In this transplant-setting case of HPVG, there were no obvious signs of intestinal necrosis or ischaemia, nor marked elevation of lactate or other deteriorating features. After surgical evaluation, high-risk conditions were excluded, and conservative treatment achieved definite therapeutic efficacy.

Two previous cases of HPVG following hematopoietic stem cell transplantation have been reported. One involved a young patient with chronic myeloid leukaemia who developed HPVG on day 22 after HSCT[5]; conservative management was adopted in the absence of intestinal necrosis or ischaemia, and recovery was achieved within two weeks. The second patient presented with steroid-refractory gastrointestinal GVHD four months after transplantation[9]; CT showed prominent HPVG with bowel wall thickening, and both clinical and radiological findings improved rapidly after conservative treatment. Compared with these two previously reported HSCT-associated HPVG cases, the present case exhibited greater complexity with respect to the timing of onset, comorbidities and infectious burden. Furthermore, we provide a more detailed description of the diagnostic and therapeutic course and propose a pathogenesis based on prior literature. A detailed comparison between these two reported cases and our case is shown in the Table 2.

It follows that HPVG after HSCT may occur not only during chronic GVHD but also at an earlier post-transplant stage complicated by obstruction and secondary infection; clinical recognition should therefore not be restricted to a fixed time window. With regard to aetiology, intestinal mucosal injury related to GVHD was identified as the triggering factor across all three cases. Compared with the prior reports, the present case highlights the additive effects of infection and obstruction in the development of HPVG. In terms of therapeutic decision-making, this case further supports that HPVG is not an absolute indication for surgery. In the setting of HSCT, even when HPVG is complicated by intestinal obstruction and severe infection, combined conservative management can yield favourable outcomes in the absence of evidence for intestinal ischaemia or necrosis, thereby avoiding unnecessary surgical intervention. Nevertheless, as a single case, it lacks generalisability, and its management strategy cannot be directly extrapolated to other comparable patients; these findings may serve only as a reference for clinicians encountering similar scenarios.

Table 2
Comparison between the present case and two previously reported cases

Items	Present case (68-yr-old male, AML, 1 mo after allo-HSCT)	A 29-year-old male patient with chronic myeloid leukemia (CML) developed HPVG 20 days after HSCT.	A 57-year-old male, 4 months after HSCT.
Clinical characteristics	Nausea, abdominal pain and distension developed one month after transplantation. Markedly elevated CRP and PCT indicated severe infection. CT demonstrated HPVG, accompanied by multi-segmental small bowel dilatation, pneumatosis intestinalis and air-fluid levels (partial small bowel obstruction), as well as colonic collapse (colonic paralysis). No signs of intestinal ischemia or necrosis were identified.	Nausea, vomiting, watery diarrhea and unexplained fever occurred on day 20 after transplantation. Mild diffuse abdominal tenderness was present without peritoneal signs. Oral mucosal changes and rashes on the trunk and upper extremities were consistent with GVHD. CT revealed typical HPVG, and sigmoid colon biopsy confirmed GVHD.	Admitted for steroid-refractory gastrointestinal GVHD. CT showed prominent HPVG with bowel wall thickening. No concomitant small bowel obstruction or levels of infection markers were reported.
Treatment	Conservative management: nil per os plus gastrointestinal decompression; octreotide combined with PPI for secretion suppression; broad-spectrum antibiotics (meropenem + teicoplanin, then switched to third-generation cephalosporins); glycerin enema to relieve colonic paralysis; cyclosporine and glucocorticoids for GVHD prophylaxis/treatment; parenteral nutritional support; multidisciplinary consultation to rule out intestinal ischemia and necrosis and avoid unnecessary surgery.	Conservative treatment was administered. Broad-spectrum antibiotics (ceftriaxone plus metronidazole) and intravenous glucocorticoids for GVHD were given after cultures were obtained; no surgery was performed.	Conservative treatment was administered with broad-spectrum antibiotics and α1-proteinase inhibitor (for progressive GVHD); no surgery was performed.
Outcome	Abdominal pain was relieved on day 1 of treatment. Repeat CT on day 7 showed complete resolution of portal venous gas, indicating a favorable prognosis. GVHD management was continued thereafter.	Rapid clinical improvement with resolution of symptoms within 7 days. Follow-up CT at 2 weeks after treatment showed complete resolution of HPVG; blood cultures were negative, and the patient was discharged in stable condition.	Rapid clinical and radiological improvement was observed, with a favorable 6-month follow-up outcome.

Items	Present case (68-yr-old male, AML, 1 mo after allo-HSCT)	A 29-year-old male patient with chronic myeloid leukemia (CML) developed HPVG 20 days after HSCT.	A 57-year-old male, 4 months after HSCT.
Presumed etiology	The primary cause was intestinal mucosal injury due to GVHD, complicated by secondary intestinal infection (bacterial translocation). Combined partial small bowel obstruction and colonic paralysis elevated intraluminal pressure, which facilitated the formation of HPVG.	The authors explicitly hypothesized that intestinal mucosal injury caused by chemotherapy and GVHD was the etiology of HPVG.	Attributed to intestinal mucosal injury and inflammation secondary to steroid-refractory GVHD; concomitant obstruction or infection was not emphasized.

Conclusion

The prognosis of hepatic portal venous gas (HPVG) largely depends on the underlying primary disease rather than the radiological sign of HPVG itself. Intestinal ischaemia and necrosis are high-risk factors associated with patient outcomes. HPVG is not an absolute indication for surgery. Clinical decisions require integrated evaluation of the primary disease, imaging features, vital signs and laboratory parameters to judge the timing of operation carefully. We report a complex case of HPVG occurring one month after allogeneic haematopoietic stem cell transplantation, complicated by partial small-bowel obstruction and colonic paralysis. After exclusion of intestinal ischaemia and necrosis via multidisciplinary consultation, individualized conservative treatment yielded a favourable outcome. On the basis of this case and previous reports, HPVG after haematopoietic stem cell transplantation may emerge at different post-transplant time points. Intestinal mucosal injury induced by gastrointestinal graft-versus-host disease constitutes the core pathogenetic basis, whereas intestinal obstruction and secondary intestinal infection synergistically facilitate the onset and progression of HPVG. Clinicians should strengthen awareness of this rare transplant-related complication and avoid unnecessary surgical intervention guided solely by the radiological detection of HPVG.

Declarations

Ethics and consent to participate This article does not involve human participants or animal experiments. No ethical approval was required for this research in accordance with local institutional guidelines.

Consent to publish: Written informed consent was obtained from patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal. All personal information and clinical data of the participants were anonymized

and de-identified to ensure patient privacy and confidentiality throughout the research and publication process.

Availability of data and materials: Not applicable—no datasets were generated or analyzed.

Competing interests: Not applicable.

Funding: This study was not supported by any specific grants from funding agencies in the public, commercial, or not-for-profit sectors. There are no financial conflicts of interest to declare.

Authors' contributions: Wenyu Min conceived and drafted the manuscript and revised it. Wei Shi revised the manuscript for important intellectual content. Han Yan provided and interpreted the imaging figures. All authors read and approved the final manuscript.

Acknowledgements: Not applicable.

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 Ther Nucl Med. 1955;74(3):486–8. PMID: 13249015.
2. 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. doi: 10.1097/00000658-197803000-00012. PMID: 637584; PMCID: PMC1396434.
3. Abboud B, El Hachem J, Yazbeck T, Doumit C. Hepatic portal venous gas: physiopathology, etiology, prognosis and treatment. World J Gastroenterol. 2009;15(29):3585–90. doi: 10.3748/wjg.15.3585. PMID: 19653334; PMCID: PMC2721230.
4. Zhou QY, Zheng ZH, Lv XL, Guo JQ, Zhang K, Xu HT. Advancement in the diagnosis and treatment of hepatic portal venous gas. World J Gastrointest Surg. 2025;17(10):109062. doi: 10.4240/wjgs.v17.i10.109062. PMID: 41178847; PMCID: PMC12576632.
5. Alqahtani S, Coffin CS, Burak K, Chen F, MacGregor J, Beck P. Hepatic portal venous gas: a report of two cases and a review of the epidemiology, pathogenesis, diagnosis and approach to management. Can J Gastroenterol. 2007;21(5):309–13. doi: 10.1155/2007/934908. PMID: 17505567; PMCID: PMC2657713.
6. Iannitti DA, Gregg SC, Mayo-Smith WW, Tomolonis RJ, Cioffi WG, Pricolo VE. Portal venous gas detected by computed tomography: is surgery imperative? Dig Surg. 2003;20(4):306–15. doi: 10.1159/000071756. Epub 2003 Jun 12. PMID: 12806196.
7. Nelson AL, Millington TM, Sahani D, Chung RT, Bauer C, Hertl M, Warshaw AL, Conrad C. Hepatic portal venous gas: the ABCs of management. Arch Surg. 2009;144(6):575 – 81; discussion 581. doi: 10.1001/archsurg.2009.88. PMID: 19528392.

8. Dibra R, Picciariello A, Trigiante G, Labellarte G, Tota G, Papagni V, Martines G, Altomare DF. 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. doi: 10.12659/AJCR.923831. PMID: 32653891; PMCID: PMC7377522.
9. Singavi AK, Shah NN. Graft-versus-host disease-associated hepatic portal venous gas. Br J Haematol. 2018;181(1):9. doi: 10.1111/bjh.15047. Epub 2018 Jan 24. PMID: 29363735.

Figures

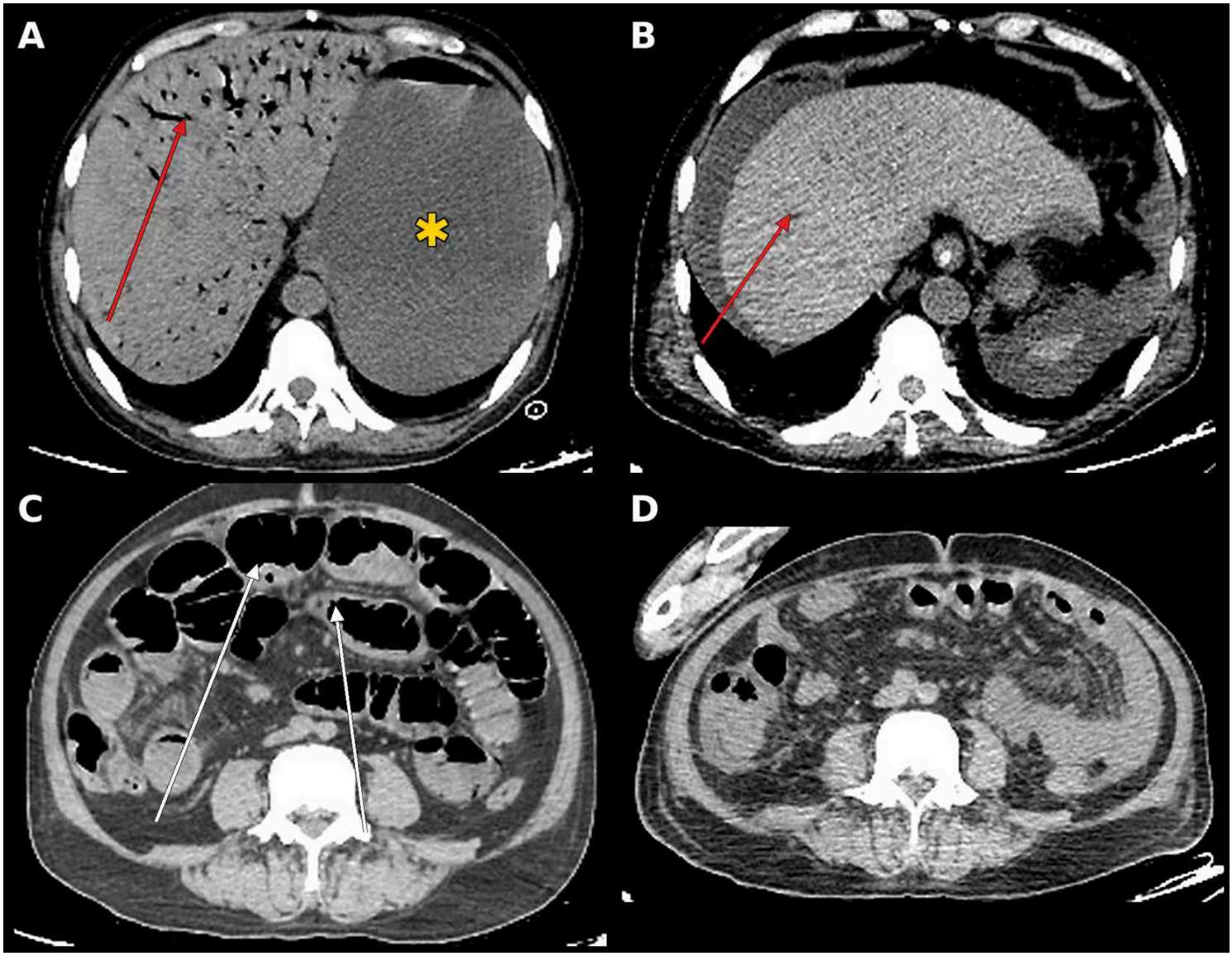


Figure 1

Unenhanced abdominal CT at admission (A, C) and after conservative management (B, D), all axial. (A) Extensive branching gas reaches the hepatic periphery within 2 cm of the capsule (red arrow), indicating hepatic portal venous gas; the stomach is markedly distended (asterisk). (C) Multiple small-bowel loops are dilated and gas-filled (white arrows), in keeping with incomplete small-bowel obstruction. Follow-up

CT shows near-complete resolution of the portal venous gas with a homogeneous liver (B, red arrow) and decompression of the bowel (D).