frontiers in Medicine

TYPE Case Report
PUBLISHED 06 November 2026
DOI 10.3389/fmed.2026.1919751

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OPEN ACCESS

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RECEIVED 25 June 2026
REVISED 08 October 2026
ACCEPTED 25 October 2026
PUBLISHED 06 November 2026

CITATION
Zhang Y, An J, Jiao C, Guo J, Li Z and Liu W (2026) Case Report: Pneumatosis cystoides intestinalis in patients with diverse comorbidities: a case series highlighting multifactorial management. Front. Med. 13:1919751. doi: 10.3389/fmed.2026.1919751

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Case Report: Pneumatosis cystoides intestinalis in patients with diverse comorbidities: a case series highlighting multifactorial management

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Background: Pneumatosis cystoides intestinalis (PCI), the cystic form of pneumatosis intestinalis, is a rare condition characterized by gas-filled cysts within the bowel wall. It is frequently misdiagnosed as acute gastrointestinal perforation because of intra-abdominal free gas, which may prompt surgery before the benign nature of the condition is recognized.
Case presentation: We report three PCI patients with distinct risk factors. The first was a 55-year-old female with PCI during acarbose exposure. She had minimal free gas and a benign clinical course, but a suspicious right-colon lesion that could not be characterized led to right hemicolectomy. The second was a 43-year-old female with autoimmune disease on immunosuppressive therapy and extensive pneumoperitoneum, who achieved full recovery with conservative treatment. The third was a 75-year-old male with combined metabolic and mechanical risk factors; 48-hour observation combined with endoscopy, which identified no overt luminal perforation, small-bowel perforation being considered unlikely on clinical grounds, avoided surgery. All patients presented evidence of radiological–clinical dissociation.
Conclusion: Our observations suggest that, in PCI, radiological severity does not reliably reflect clinical risk. We propose, as an unvalidated hypothesis rather than a clinical decision rule, that initial management be guided by recognition of the radiological–clinical dissociation rather than by imaging severity alone, provided that strict safety criteria are met. Mechanism-based strategies, including withdrawal of the offending drug when PCI is drug-associated, close observation of clinically stable patients, and surgery reserved for definite complications, may reduce morbidity. Prospective validation of this framework and derivation of evidence-based escalation thresholds are warranted.

KEYWORDS
acarbose, autoimmune disease, case series, conservative management, diabetes mellitus, pneumatosis cystoides intestinalis, pneumatosis intestinalis

1 Introduction

Pneumatosis cystoides intestinalis (PCI), the cystic variant of pneumatosis intestinalis (PI), manifests as gas-filled cysts located in the submucosal and subserosal layers of the gastrointestinal tract. Because affected individuals often remain asymptomatic, determining the true prevalence remains challenging. According to endoscopic survey data, that figure is

approximately 0.03% (1); most reported patients are adults aged 50–70 years, with no marked sex predilection (2). Clinically, the foremost challenge lies in PCI's ability to mimic acute surgical conditions, especially when free intra-abdominal gas is detected (3).

Several mechanisms contribute to PCI pathogenesis (3–6): mechanical mucosal damage secondary to elevated intraluminal pressure, bacterial translocation by gas-producing microorganisms, disturbed hydrogen metabolism, particularly in the context of alpha-glucosidase inhibitor use, and pulmonary gas tracking along mesenteric vascular planes. The relative contribution of each pathway, and whether they act independently or in combination, remain poorly characterized; our cases suggest that multiple mechanisms may coexist with variable predominance, influenced by patient-specific factors such as comorbidity burden. Drug-induced PCI, especially that triggered by alpha-glucosidase inhibitors or immunosuppressants, tends to follow a more indolent course than its ischemic or inflammatory counterparts (7). This mechanistic diversity yields a broad spectrum of clinical and radiographic presentations, making bedside risk stratification difficult.

For detecting intramural cysts, portal venous gas, and pneumoperitoneum, abdominal computed tomography (CT) offers the highest sensitivity among imaging modalities (8). High sensitivity for gas, however, does not imply specificity for perforation. Imaging features that argue for PCI include a cystic pattern of intramural gas, preserved bowel wall enhancement, and the absence of a focal wall defect (3). Endoscopy provides complementary information through direct mucosal visualization and targeted tissue sampling (9); it can confirm or exclude overt perforation of the examined segment, but because the small bowel is beyond its reach, a negative examination does not rule out small-bowel perforation. Management hinges on risk stratification guided by clinical stability and the presumed underlying mechanism; treating the primary condition takes precedence, with supplemental oxygen, antibiotics, or probiotics employed on a case-by-case basis (9). Surgical intervention is reserved for overt perforation, diffuse peritonitis, hemodynamic instability, intestinal necrosis, or refractory obstruction (10).

The central diagnostic trap in daily practice is the disconnect between radiographic severity and clinical stability; when this mismatch goes unrecognized, surgical decisions may rest on imaging alone. This diagnostic ambiguity is especially pronounced in patients with multiple comorbidities, in whom PCI can be misinterpreted as a surgical emergency. We present three cases spanning a spectrum of comorbidity burdens to illustrate this interpretive challenge: two in which integrated assessment of clinical status, imaging, and endoscopic findings averted surgery in stable patients without true perforation, and one in which the dissociation was initially overlooked, with the consequences discussed below.

2 Methods

We retrospectively identified consecutive PCI patients (ICD-10 K63.8) diagnosed between January 2021 and December 2025, cross-referenced with radiology reports. Inclusion criteria were: (1) radiological confirmation of intramural gas on CT: gas-filled cysts within the bowel wall in a cystic or linear configuration; (2) histopathological confirmation where available; (3) age ≥18 years; (4) complete clinical data spanning diagnosis through ≥4 weeks of follow-up. Exclusion criteria were: primary PCI (pneumatosis without identified predisposing condition), and incomplete follow-up (<4 weeks or lost to follow-up). All CT studies were independently reviewed by an abdominal radiologist with 20 years of experience, who was blinded to the clinical course, the final histopathological diagnosis, and the treating team's working diagnosis.

Of 7 patients screened, 1 was excluded because of age <18 years, leaving 6 eligible cases. Three were selected for detailed presentation to represent three distinct dominant risk profiles: drug-associated (Patient 1), immunosuppression-associated (Patient 2), and combined metabolic and mechanical (Patient 3) (Table 1). Selection was based on pretreatment clinical characteristics and was finalized before outcome data were reviewed. As the authors were also members of the treating team, the possibility of selection bias cannot be excluded. The remaining three patients are summarized in Supplementary Table S1, along with the reasons they were not selected for detailed presentation. Reporting the full cohort allows readers to assess the representativeness of the selected cases. No formal sample size calculation was performed, in keeping with the descriptive nature of this case series. The study was approved by the institutional ethics committee (2024-YLJS-40) and is reported in accordance with the CARE guidelines.

3 Case description

3.1 Case 1

A 55-year-old woman presented with a 6-month history of intermittent dull abdominal pain. She had a 4-year history of type 2 diabetes mellitus managed with oral acarbose. Physical examination was unremarkable. Admission laboratory findings, including peripheral white blood cell count, C-reactive protein, lactate, hemoglobin and platelet counts, were normal (Supplementary Table S2). Colonoscopy revealed multiple coalescing, raised lesions in the right colon, the largest measuring 3 cm (Figures 1A,B). Superficial biopsies showed proliferative polyps, but a submucosal malignancy could not be excluded. Abdominopelvic CT obtained 48 h after colonoscopy demonstrated cystic intramural gas confined to the ascending colon, accompanied by mild mesenteric edema and trivial pelvic effusion, with only minimal extraluminal free gas (Figure 2A). No focal bowel wall defect, bowel wall disruption, portal venous gas, or intramesenteric gas was identified, and colonic wall enhancement was preserved. These findings were initially interpreted as post-procedural changes but were retrospectively consistent with early PCI with subtle cyst rupture. Endoscopic submucosal dissection was attempted but abandoned because of submucosal fibrosis. Diagnostic laparoscopy revealed mild ascites and enlarged mesenteric lymph nodes, and intraoperative frozen-section analysis was nondiagnostic. Owing to this persistent diagnostic uncertainty, and in accordance with the patient's preference, laparoscopic right hemicolectomy was performed.

Postoperative pathology showed multiple variable-sized cystic cavities in the submucosa and serosa, lacking epithelial lining and partially covered by flattened histiocytes and multinucleated giant cells, consistent with PCI (Figures 3A,B). Acarbose was stopped and replaced with insulin-based therapy for glycemic control. She

TABLE 1 Clinical timeline and characteristics of three patients with PCI.

Feature	Patient 1	Patient 2	Patient 3
Age/Sex	55/F	43/F	75/M
Comorbidities	T2DM	RA, ASS, ILD	T2DM, HTN, CHD
Timeline			
Symptom onset	6 months prior	3 months prior	5 days prior
Prior medication exposure	Acarbose (4 years)	CTX, nintedanib, prednisone (3 months)	Acarbose (3 years)
Initial presentation	Intermittent dull abdominal pain	Polydipsia and polyuria	Nocturnal pain, flatulence
Key diagnostic event	Colonoscopy → CT (48 h post-procedure)	CT (cystic intramural gas + extensive free gas)	CT at local hospital → transfer
Critical decision point	Diagnostic uncertainty, malignancy not excludable → surgery	Hemodynamic stability → conservative	48 h observation → endoscopy
Intervention	Right hemicolectomy	Conservative (O_2, insulin, medication review)	Conservative (acarbose stop, insulin, O_2)
Definitive diagnosis	Postoperative pathology	Clinical + radiological resolution	No overt luminal perforation on endoscopy; small-bowel perforation not excluded, considered unlikely on clinical grounds

T2DM, type 2 diabetes mellitus; RA, rheumatoid arthritis; ASS, antisynthetase syndrome; ILD, interstitial lung disease; HTN, hypertension; CHD, coronary heart disease; CTX, cyclophosphamide.

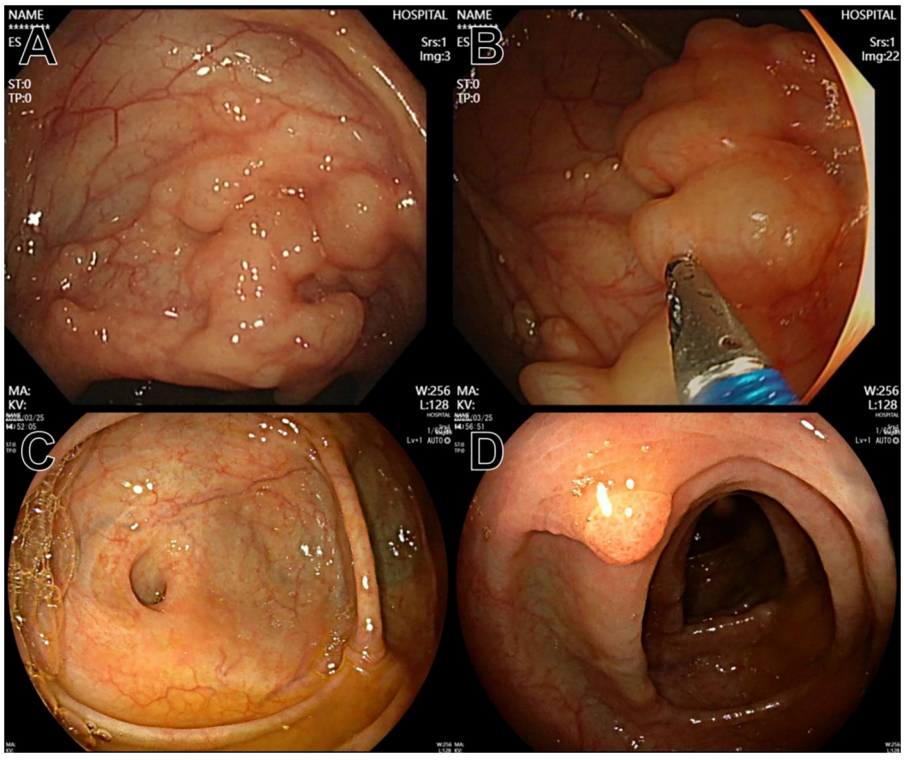


FIGURE 1
(A,B) case 1: colonoscopic examination identified multiple coalescing, raised lesions in the right colon, the largest measuring 3 cm. **(C,D)** Case 3: Colonoscopy showed non-inflamed diverticula and polypoid elevations, with no mucosal defect or perforation identified in the colon.

recovered uneventfully and was discharged on postoperative day 12. At 4-week follow-up, she expressed relief at the benign diagnosis but disappointment that the preoperative uncertainty had led to surgical resection. Repeat CT showed complete resolution.

3.2 Case 2

A 43-year-old woman presented with a 3-month history of polydipsia and polyuria, attributed to uncontrolled hyperglycemia.

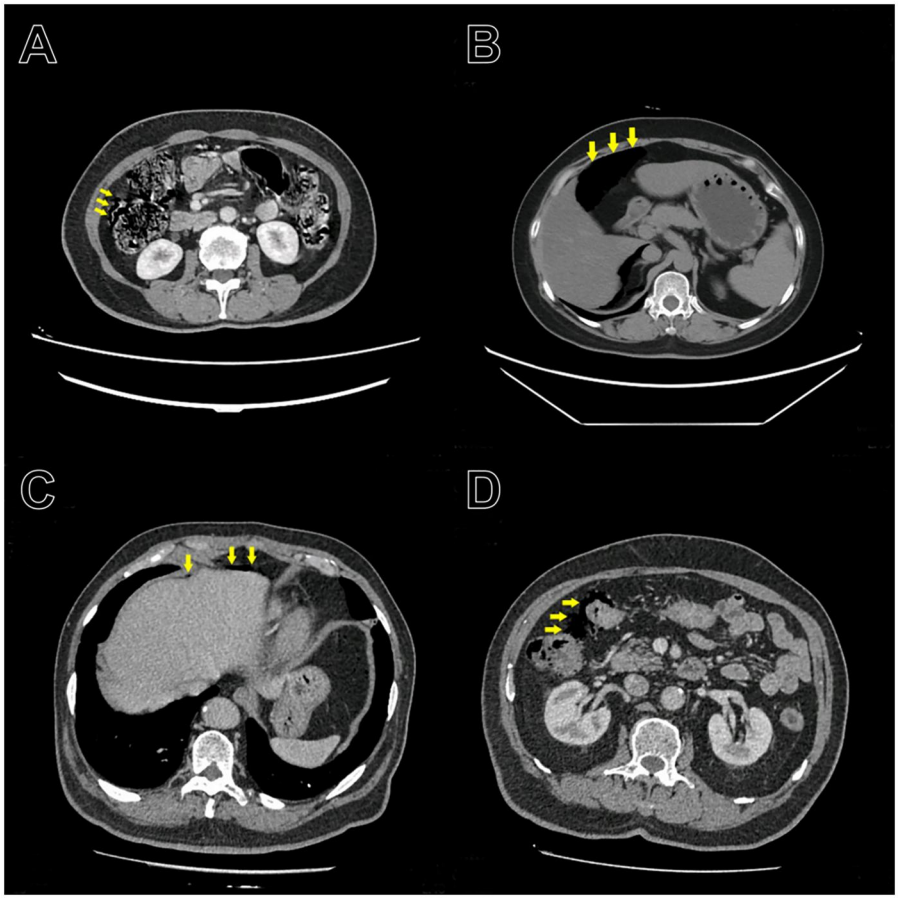


FIGURE 2
Abdominal CT findings in three patients with PCI exhibiting distinct clinical courses. **(A)** Case 1: Axial image shows cystic intramural gas collections predominantly within the ascending colon wall, with trace extraluminal gas (arrow). No portal venous gas, mesenteric fat stranding, or pneumoperitoneum is seen, consistent with uncomplicated PCI. **(B)** Case 2: Axial image demonstrates cystic intramural gas involving the right colonic wall, together with a large volume of free intraperitoneal gas adjacent to the liver surface (arrows). **(C,D)** Case 3: Axial images show irregular colonic wall contour at the hepatic flexure with intramural cystic gas and multiple pockets of free intraperitoneal gas (arrows). No overt bowel wall defect was identified on imaging.

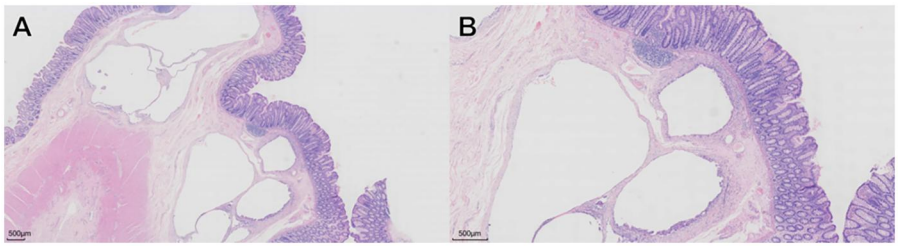


FIGURE 3
Postoperative pathology showed multiple variable-sized cystic cavities in the submucosa, lacking epithelial lining and partially covered by flattened histiocytes and multinucleated giant cells. **(A)** HE ×20, **(B)** HE ×40.

She had received cyclophosphamide, nintedanib and prednisone for 3 months prior to symptom onset for rheumatoid arthritis, antisynthetase syndrome and interstitial pneumonia. Admission laboratory investigations showed marked hyperglycemia, hypokalemia, dyslipidemia, elevated aminotransferases and and C-reactive protein (Supplementary Table S2). Chest CT on admission showed pneumomediastinum and bilateral interstitial pneumonia. Physical examination revealed hypertension (165/98 mmHg); abdominal examination was entirely unremarkable. Abdominopelvic CT on admission demonstrated cystic intramural gas localized to the right colon, accompanied by extensive free intraperitoneal gas (Figure 2B). No focal bowel wall defect, wall disruption, or abnormal bowel wall thickening was observed, and colonic mucosal enhancement was preserved. There was no portal venous gas or intramesenteric gas to suggest mesenteric ischemia or transmural perforation. Additional findings included fatty liver,

splenomegaly, tiny renal calculi and mild retroperitoneal lymphadenopathy. Given hemodynamic stability and absent peritonitis, conservative management was initiated on day 1, including high-flow oxygen, insulin-based glycemic control, and review of potentially contributing medications. The patient was managed with intensive glycemic regulation, antihypertensive, lipid-lowering and anti-inflammatory medications. Repeat CT on day 7 showed obvious reduction of intra-abdominal free gas. She was discharged on day 10 with symptomatic improvement. At 4-week follow-up, glycemic control had improved, and the patient was relieved that surgery had been avoided. Repeat CT confirmed complete resolution of the intramural gas and pneumoperitoneum.

3.3 Case 3

A 75-year-old man was evaluated for intermittent lower abdominal pain and flatulence occurring at night over a 5-day period. Abdominal CT at a local hospital on day 1 raised concern for gastrointestinal perforation. On transfer to our facility on day 2, repeat CT demonstrated cystic intramural gas involving small-bowel loops and the colon in the right upper quadrant, together with scattered free intraperitoneal gas (Figures 2C,D). No focal bowel wall defect or transmural disruption was identified; bowel wall thickness was normal with preserved mural enhancement. No portal venous gas or intramesenteric gas was present. His medical history included a 20-year duration of type 2 diabetes, hypertension for 3 years, and coronary heart disease for 20 years. Acarbose had been prescribed for 3 years. Vital signs were stable; abdominal examination was completely benign; and no peritoneal signs were elicited. Given hemodynamic stability and absent peritonitis, initial management included bowel rest, intravenous fluids, and close clinical observation. Monitoring comprised vital signs and abdominal examinations every 4 h, lactate every 12 h, and surgical consultation within 30 min. Criteria for treatment escalation were defined as the development of peritoneal signs, hemodynamic deterioration, or a lactate level exceeding 2.5 mmol/L, or CT findings suggestive of bowel ischemia. After 48 h of clinical stability, with a normal peripheral WBC count, C-reactive protein and serial lactate values consistently below 2.5 mmol/L (Supplementary Table S2), and no peritoneal signs, gastrointestinal endoscopy was performed on day 4 to look for an overt luminal perforation. Gastroscopy revealed chronic gastritis with erosion but no mucosal defect in the stomach or duodenum. Colonoscopy showed multiple chronic, non-inflamed diverticula and polypoid elevations, with no mucosal defect in the colon or rectum (Figures 1C,D). Gastroscopy and colonoscopy therefore excluded an overt perforation of the stomach, duodenum, colon or rectum. Perforation of the small bowel could not be excluded endoscopically but was considered unlikely in view of persistent clinical stability, normal serial lactate levels, and the absence of peritoneal signs throughout the observation period. Though initially reluctant to forgo surgery despite free gas on imaging, he later accepted that close observation with endoscopic exclusion of an overt luminal perforation, rather than the mere presence of free gas on imaging, justified conservative management. Conservative management was continued, including acarbose discontinuation on day 3, insulin-based glycemic control, and high-flow oxygen at 4–6 L/min via nasal cannula for 12 h daily. Abdominal pain resolved by day 7. He was discharged on day 9 with unremarkable physical examination. At 4-week follow-up, he was asymptomatic with full symptom resolution. Scheduled repeat CT at 4 weeks demonstrated complete resolution.

4 Discussion

PCI encompasses a broad clinical spectrum, ranging from incidental benign findings to life-threatening intestinal ischemia. Its diverse presentations and frequent coexistence with intra-abdominal free gas often lead to misdiagnosis as surgical emergencies, yet numerous cases resolve without any intervention. The three patients described here illustrate this heterogeneity: despite markedly different comorbidity profiles, all exhibited a common pattern of radiological–clinical dissociation, that is, a radiological abnormality not matched by the severity of the clinical picture, suggesting that clinical expression may be driven by multifactorial mechanisms rather than a single cause.

This radiological–clinical dissociation was evident across all three cases. In Patient 1, colonoscopy demonstrated coalescing right colonic lesions, and CT obtained 48 h later revealed minimal extramural free gas and mesenteric edema. Despite these concerning findings, she remained hemodynamically stable with a normal abdominal examination. Endoscopic and imaging features raised concern for an infiltrative or malignant right colonic lesion, which contrasted sharply with her benign clinical course; benign pathology was confirmed only after hemicolectomy. The diagnostic pitfall in Case 1 lies in image interpretation: PCI may mimic neoplasia endoscopically, and PCI-related extraluminal gas cannot be distinguished from perforation on CT (3, 11). Patient 2 demonstrated extensive free gas without any abdominal complaints. Patient 3 showed multiple gas shadows while experiencing only intermittent nocturnal pain. The PCI diagnoses for Patients 2 and 3 are presumptive and were not confirmed pathologically, because definitive bowel perforation could not be established. Taken together, without a high index of suspicion for PCI, these radiologic findings are readily misattributed to perforation or malignancy. In hemodynamically stable patients with free intraperitoneal gas but no peritonitis, a strategy of cautious observation with serial evaluation and targeted endoscopy may avert unnecessary surgery, even when malignancy or ischemia cannot be entirely ruled out (12).

We propose the following bedside recognition criteria, offered as hypothesis-generating observations rather than a management rule, for identifying radiological–clinical dissociation. Five distinguishing features define the clinical phenotype: (1) intraperitoneal free gas detected on CT, (2) absence of peritoneal signs, (3) hemodynamic stability, (4) serum lactate $\leq$2.5 mmol/L, and (5) no CT evidence of ischemia. When all discriminating conditions are fulfilled, a strategy of observation with close monitoring may be considered appropriate. Their components were selected for bedside practicality rather than derived from statistical modelling, and the sensitivity and specificity of the composite criteria remain unknown; any

threshold derived from this case series, including the lactate value, carries no evidential weight. Their purpose is solely to prompt a deliberate, documented second look at the patient, not to replace surgical consultation, nor to justify deferring surgical consultation.

An alternative mechanism must be considered for Case 2: the Macklin effect. Alveolar rupture may allow air to track along bronchovascular sheaths into the mediastinum; this is a known complication of interstitial lung disease, and nintedanib has been linked to pneumomediastinum. Transdiaphragmatic air dissection along fascial planes provides a competing explanation for concurrent pneumomediastinum and intraperitoneal free gas. While cystic intramural gas localized to the right colonic wall favors an intestinal source over a purely pulmonary origin, contributions from either pathway cannot be excluded.

The prevailing model posits independent, additive pathways; our case series suggests these mechanisms coexist with shifting predominance (13). Patients 1 and 3 both had acarbose exposure yet followed different clinical courses: structural lesion complexity (malignancy that could not be excluded) necessitated surgery in Patient 1, whereas incidental diverticulosis supported conservative observation in Patient 3. Both patients receiving acarbose demonstrated radiological resolution following drug discontinuation. Acarbose may predominate in susceptible individuals, and its discontinuation can interrupt the pathogenic cascade despite persistent structural factors (14). This finding is consistent with a drug-associated mechanism but does not prove causality, because other interventions were initiated concurrently, and spontaneous resolution of secondary PCI is well described. Discontinuing a potentially offending drug represents a reasonable step targeting one plausible contributor to the pathophysiology, yet structural abnormalities may still demand intervention to achieve diagnostic certainty (15); the contribution of acarbose itself cannot be quantified from these data.

A provisional classification is suggested to complement the recognition criteria: Drug-predominant (alpha-glucosidase inhibitors, immunosuppressants), Inflammatory/immunosuppression-predominant (autoimmune disease, inflammatory bowel disease), Mechanical-predominant (obstruction, diverticulosis), and Mixed. Neither the criteria nor this classification has been prospectively validated.

This approach aligns with Kuehn et al. (16), who stratify PCI by clinical stability: stable patients without peritonitis are managed with observation, whereas unstable patients require emergent intervention. Our cases illustrate both the utility and limitations of this framework. Patients 2 and 3, stable and without peritonitis, were managed conservatively. Patient 1 fell into an intermediate category: clinically stable without peritonitis but with malignancy that could not be excluded. Such structural features are not captured within Kuehn's functional framework. We propose supplementing their algorithm with an "anatomical risk" dimension to assess structural lesions mimicking malignancy; Kuehn's framework may therefore be expanded to incorporate etiology, anatomical distribution, and clinical status. Patient 2 had an identifiable nonischemic mechanism and recovered with conservative management alone despite more extensive free gas. Patient 3 required only glycemic control after endoscopy ruled out overt luminal perforation. All three patients achieved favorable outcomes, yet the morbidity seen in Case 1 underscores the risk of allowing a single alarming imaging feature, rather than the overall clinical picture, to guide management.

We acknowledge that observation carries a small risk of delayed intervention; strict monitoring (vitals every 4 h, serial lactate, surgical availability) mitigated this risk. Mortality from delayed laparotomy for ischemia ranges from 50%–70% (16), and an observation-first approach requires rigorous patient selection and frequent reassessment. Lactate is an imperfect marker in this setting: lactate levels may remain normal in early mesenteric ischemia, meaning a normal lactate cannot exclude evolving ischemia, whereas mild elevation in patients with chronic disease is often not of intestinal origin (18). Lactate serves here as one component of composite clinical judgment rather than a standalone screening test. The Case 3 protocol is proposed as a minimum standard; prospective studies should refine escalation criteria.

Endoscopy for PCI carries inherent risks limiting patient selection. Bowel insufflation increases intraluminal pressure, which may rupture pneumatosis cysts to cause perforation or worsen pneumoperitoneum by forcing air across compromised bowel wall; positive-pressure ventilation, if required during sedation, further raises transmural pressure. Endoscopy may also impair bowel-wall evaluation on subsequent cross-sectional imaging. We reserve endoscopy only for carefully selected hemodynamically stable patients without peritoneal signs, with no overt perforation on imaging and with immediate surgical backup. It is not a routine PCI investigation, and its purpose is to identify occult luminal defects requiring surgery. Endoscopy should be deferred when alternative diagnoses are suspected or imaging confirms bowel perforation. In Case 1, endoscopy alone failed to fully characterize colonic disease or exclude neoplasia; definitive tissue diagnosis required surgical resection rather than endoscopic findings alone.

A multifactorial management approach has three practical implications. First, medication reconciliation should be a priority for all patients with PCI, particularly those receiving alpha-glucosidase inhibitors or immunosuppressants. Second, reflexive prescription of oxygen and antibiotics should be balanced against individualized assessment of contributing drivers within this multifactorial framework; routine use of these agents in non-bacterial, non-ischemic PCI lacks mechanistic support, and current recommendations for oxygen therapy, antibiotics, and probiotics in PCI rest on observational data and case series rather than randomized trials (13). Third, validated biomarkers are urgently needed to distinguish multifactorial phenotypes and reduce reliance on clinical and radiological judgment alone. Fecal calprotectin or serum D-lactate may eventually allow objective differentiation between inflammatory and ischemic etiologies, though prospective validation is required (17, 18).

We acknowledge that stable patients with free gas are often managed conservatively. Our specific contribution is articulating the radiological–clinical dissociation heuristic, defining recognition criteria, and proposing a provisional PCI classification for mechanism-specific management; all of these remain hypothesis-generating.

This case series carries inherent limitations. Its small sample size and single-center design reflect the rarity of PCI; our

findings are hypothesis-generating, and a small case series such as this cannot support derivation or validation of a management algorithm. All patients had secondary PCI with defined predisposing conditions, and conclusions may not extend to primary forms of the disease. The 4-week follow-up precludes assessment of long-term recurrence. The causal relationship between acarbose cessation and resolution cannot be established, since concurrent interventions were instituted in both affected patients. Management decisions were individualized, reflecting real-world practice but introducing selection bias. Because the cohort was constructed based on radiological abnormality, irrespective of the presence or absence of radiological–clinical dissociation, this series cannot be used to estimate the prevalence of dissociation. Pooled analyses or collaborative registries may offer the most feasible path to evidence-based management in this rare condition.

5 Conclusion

PCI is a rare but clinically important condition in which radiographic appearance may bear little relation to the clinical state: radiographic abnormalities, including free intraperitoneal gas, may occur in clinically well patients. Recognition of this mismatch can avert unnecessary surgery. Conservative treatment is safe in hemodynamically stable patients without peritonitis; surgery is reserved for frank perforation, diffuse peritonitis, hemodynamic collapse, intestinal necrosis, or refractory obstruction. Clinicians caring for patients receiving acarbose or immunosuppressants who develop incidental free intraperitoneal gas should prioritize observation, medication reconciliation, and follow-up cross-sectional imaging when radiological–clinical dissociation is marked. Future work should define evidence-based escalation thresholds, validate biomarkers to distinguish multifactorial mechanistic profiles, and evaluate etiology-stratified management protocols in prospective trials.

Data availability statement

The original contributions presented in the study are included in the article/Supplementary Material, further inquiries can be directed to the corresponding author.

Ethics statement

The studies involving humans were approved by Medical Ethics Committee of Bethune International Peace Hospital. The studies were conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study. Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

Author contributions

YZ: Data curation, Methodology, Project administration, Writing – original draft. JA: Data curation, Methodology, Writing – original draft. CJ: Data curation, Investigation, Supervision, Writing – original draft. JG: Data curation, Investigation, Supervision, Writing – original draft. ZL: Data curation, Writing – original draft. WL: Data curation, Writing – review & editing.

Funding

The author(s) declared that financial support was received for this work and/or its publication. This study was supported by the Medical Science Research Project of Hebei Province in 2022 (No. 20220247). The funders did not participate in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Conflict of interest

The author(s) declared that this work was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Supplementary material

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fmed.2026.1919751/full#supplementary-material

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