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MRI Suspicion of Pneumoperitoneum Revealing Pneumatosis Cystoides Intestinalis on CT

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Abstract

Background: Pneumatosis cystoides intestinalis (PCI) is an uncommon entity defined by gas-filled cysts within the bowel wall. Although often benign, it may simulate perforation or pneumoperitoneum on imaging, particularly in patients with underlying bowel disease. Its association with Crohn's disease remains rare and diagnostically challenging.

Case presentation: A 30-year-old man with longstanding Crohn's disease, initially followed in Libya and lost to follow-up since 2021, was referred for reassessment after irregular corticosteroid use and recurrent abdominal pain. MR enterography demonstrated multifocal active small-bowel disease, dominated by a 20-cm distal jejunal segment with mural thickening up to 10 mm, heterogeneous transmural enhancement, adjacent mesenteric inflammatory change, and locoregional lymphadenopathy; additional inflammatory thickening involved the distal ileum over 12 cm. During image review, focal signal voids abutting diseased bowel loops raised the unresolved possibility of gas. As this finding could not be confidently characterized on MR images, a targeted abdominopelvic CT was obtained. CT demonstrated moderate pneumoperitoneum associated with predominantly colonic cystic intramural gas collections, establishing the diagnosis of pneumatosis cystoides intestinalis rather than frank bowel perforation. In the absence of peritoneal irritation, bowel ischemia, or hemodynamic instability, the patient was managed conservatively with surveillance alone.

Conclusion: This case highlights the diagnostic value of CT in confirming pneumatosis cystoides intestinalis when MRI findings are equivocal. In patients with Crohn's disease,PCI may mimic pneumoperitoneum or bowel perforation , making recognition of its characteristic imaging appearance essential to avoid unnecessary surgical intervention.

Introduction

Pneumatosis cystoides intestinalis is characterized by the presence of gas-filled cysts within the submucosal or subserosal layers of the bowel wall. Although uncommon, it is increasingly recognized with the widespread use of cross-sectional imaging. PCI should not be regarded as a single disease entity but rather as a radiologic-pathologic manifestation associated with a broad spectrum of underlying

conditions. In the review by Ling et al., approximately 15% of cases were classified as primary and 85% as secondary, underlining that, in adults, an underlying cause should actively be sought. The same review reported that PCI most commonly involves the colon (46%), followed by the small bowel (27%), both large and small bowel (7%), and the stomach (5%) **[1]**.

Crohn's disease is an unusual but well-documented substrate for PCI. In this setting, the presence of intramural gas may immediately raise concern for bowel perforation, ischemia, or uncontrolled severe inflammatory disease. Yet the available reports suggest that impressive radiologic findings may coexist with unexpectedly benign clinical presentations. This discrepancy between imaging alarm and clinical stability constitutes the main diagnostic challenge in Crohn-associated PCI **[2, 3, 4, 5]**.

The present case is of particular interest because MR enterography first established active multifocal Crohn involvement while also raising an equivocal suspicion of gas that could not be confidently characterized on that examination. CT subsequently demonstrated pneumatosis cystoides intestinalis associated with pneumoperitoneum rather than frank perforation.

Case presentation

A 30-year-old man with a known history of Crohn's disease was referred for reassessment of disease activity. He had been initially followed in Libya and had been lost to follow-up since 2021. During this interruption of care, he reported irregular corticosteroid use. He had no relevant surgical history and no notable family history.

He presented with diffuse abdominal pain evolving over several days, without vomiting, cessation of stool or flatus, or reported fever. On examination, his general condition was preserved. Temperature was normal, conjunctivae were well colored, and the abdomen was soft, without guarding, rigidity, palpable mass, or signs of peritoneal irritation.

MR enterography demonstrated multifocal inflammatory small-bowel involvement, dominated by a distal jejunal segment measuring approximately 20 cm in length and showing circumferential mural thickening up to 10 mm, heterogeneous transmural enhancement, and adjacent mesenteric inflammatory infiltration with locoregional lymphadenopathy. Additional inflammatory mural thickening was present in the distal ileum, measuring approximately 10 mm over 12 cm (Figure 1). Overall, the examination was consistent with active multifocal Crohn's disease.

During image interpretation, however, focal signal voids adjacent to involved bowel loops introduced a second diagnostic question, namely whether gas might also be present (Figure 2). As this finding could not be confidently characterized on MR images, a targeted abdominopelvic CT was performed as a problem-solving examination.

Abdominopelvic CT demonstrated moderate pneumoperitoneum associated with predominantly colonic cystic intramural gas collections, consistent with pneumatosis cystoides intestinalis, despite the absence of overt imaging evidence of active colonic mural involvement on MR enterography (Figure 3). CT also confirmed multifocal inflammatory bowel abnormalities, including mural thickening, mesenteric inflammatory change, and upstream small-bowel dilatation, but showed no peritoneal fluid collection and no clinico-radiological argument for bowel infarction or septic perforation.

Despite the alarming radiologic appearance, the patient remained hemodynamically stable, afebrile, and free of peritoneal signs. In this setting, and after clinico-radiologic correlation, a non-operative strategy was adopted, consisting of close clinical surveillance without immediate invasive treatment.

Discussion

Pneumatosis cystoides intestinalis refers to gas-filled cysts within the bowel wall, usually located in the submucosa or subserosa. Ling et al. emphasized that PCI is not a homogeneous disease but rather a manifestation with variable etiologies, clinical expressions, and radiologic appearances. Their review is particularly useful because it frames PCI simultaneously according to cause and topography: primary versus secondary, and colonic versus enteric or mixed forms. In their synthesis, colonic involvement was the most frequent pattern, which is important in the present case because the pneumatosis was predominantly colonic, whereas the inflammatory burden demonstrated on MR enterography was predominantly small-bowel **[1]**.

The literature also supports a useful morphologic distinction on imaging. Logue and Chaudhry noted that CT may show rounded cystic gas collections, often associated with more benign forms, or linear/crescentic mural gas, a pattern more often linked to severe pathology such as bowel ischemia, although not exclusively. This distinction matters clinically because it helps avoid the simplistic interpretation that any intramural gas necessarily reflects catastrophic bowel compromise. In practical terms, PCI should therefore be interpreted not only by its presence, but by its pattern, distribution, and clinical context **[6]**.

The pathophysiology of PCI remains multifactorial. Ling et al. and Pswarayi et al. both summarize three principal mechanisms: the mechanical theory, the bacterial theory, and the pulmonary theory. The mechanical hypothesis proposes that increased intraluminal pressure, obstruction, hyperperistalsis, or mucosal breach allows gas to enter the bowel wall. The bacterial theory suggests that gas-producing microorganisms cross the mucosal barrier and generate gas in situ. The pulmonary theory, more relevant in chronic lung disease, describes dissection of gas from ruptured alveoli through mediastinal and mesenteric planes toward the bowel wall. These mechanisms are not mutually exclusive and may overlap within the same patient **[1, 7]**.

In Crohn's disease, the association is biologically plausible. Itabashi et al., in one of the most directly relevant reports, described PCI in a patient with ileocecal Crohn's disease and suggested that mucosal ulceration, increased intraluminal pressure, and bacterial overgrowth secondary to inflammatory narrowing could all contribute to intramural gas formation. Their case is especially informative because the PCI was colonic, while the dominant Crohn lesion was ileocecal, supporting the idea that the topography of PCI does not necessarily overlap that of overt mural inflammatory activity **[5]**.

This dissociation is highly relevant here. Although MR enterography demonstrated predominantly small-bowel disease, CT showed predominantly colonic cystic pneumatosis. This pattern should not be regarded as contradictory. Ling et al. reported that the colon is the most frequent site of PCI involvement overall **[1]**. Likewise, Itabashi et al. described colonic PCI in Crohn's disease and specifically noted the absence of active lesions on the anal side of the colon and rectum, which supports the concept that PCI may involve bowel segments without obvious concomitant active mural inflammatory disease on imaging **[5]**. Vallejo et al. also discussed Crohn-associated cases in which pneumatosis did not strictly mirror the dominant inflammatory topography **[3]**.

Clinical presentation across the supplied literature is remarkably heterogeneous. Many patients are asymptomatic or only mildly symptomatic, and the diagnosis is made incidentally. When present, symptoms are generally nonspecific and include abdominal pain, distension, diarrhea, constipation, nausea, vomiting, or hematochezia. Severe manifestations such as obstruction, volvulus, ischemia, or peritonitic abdomen are reported but occur in a minority of cases. This wide spectrum is well illustrated

by the contrast between the complicated surgical case of Pswarayi et al. and the far more indolent courses reported by Santos et al., Vallejo et al., and Pendse et al. **[2, 3, 7, 8]**.

The risk factors described in the available reports further support interpretation of PCI as a secondary phenomenon in many adults. Ling et al. list Crohn's disease, intestinal stenosis, ulcerative colitis, COPD, drug exposure, and prior procedures among implicated conditions **[1]**. Pswarayi et al. similarly cite gastrointestinal surgery, COPD, connective tissue disease, malnutrition, alpha-glucosidase inhibitors, and colonoscopy **[7]**. Pendse et al. emphasize chronic steroid exposure and immunosuppressive therapy in Crohn-related pneumatosis **[2]**, while Berger et al. illustrate how connective tissue disease may coexist with Crohn's disease and complicate causal interpretation **[4]**. In the present patient, longstanding Crohn's disease, interrupted follow-up, and irregular corticosteroid use together provide a plausible substrate for PCI.

One of the most important lessons from the literature is that pneumoperitoneum in PCI does not automatically imply frank digestive perforation. Berger et al. explain that rupture of subserosal gas blebs may release air into the peritoneal cavity without true transmural perforation **[4]**. Logue and Chaudhry make a similar point from an imaging perspective, showing that benign intramural gas may coexist with pneumoperitoneum and still not represent a surgical perforation **[6]**. Santos et al. also reported free intraperitoneal air associated with PCI in the absence of intestinal necrosis or the need for emergency surgery **[8]**. This distinction is central in Crohn's disease, where the reflex interpretation of free air may too rapidly shift toward perforation.

The previously reported Crohn-related cases also support a conservative reflex when the patient is stable. Pendse et al. described successful non-operative management of extensive small-bowel pneumatosis in a patient with Crohn's disease **[2]**. Vallejo et al. reported jejunal pneumatosis on CT enterography in a non-toxic patient with presumed Crohn's disease, also without operative escalation **[3]**. Even in the case reported by Itabashi et al., surgery was ultimately driven by the Crohn stricture rather than by PCI itself, and the discussion makes it clear that PCI may be benign and should not automatically dictate resection **[5]**.

The present case illustrates the same principle through a specific imaging sequence. MR enterography fulfilled its primary role by demonstrating active multifocal inflammatory disease, but it also raised an unresolved suspicion of gas. CT then became decisive, not as a routine second examination, but as the modality capable of distinguishing mural gas from free peritoneal gas and of placing both findings into a coherent diagnostic framework. Once CT demonstrated predominantly colonic cystic intramural gas collections associated with moderate pneumoperitoneum, the key question was no longer whether air was present, but whether this air reflected catastrophic bowel injury. The absence of peritoneal signs, sepsis, hemodynamic instability, portal venous gas, or radiologic evidence of bowel infarction supported interpretation of PCI as a benign mimic rather than a surgical emergency, making surveillance the most appropriate strategy **[2, 4, 6, 8]**.

Conclusion

PCI should be recognized as a multifaceted entity that may be primary or secondary, colonic, enteric, or mixed, and cystic or linear in radiologic appearance. In Crohn's disease, it remains an unusual but plausible association driven by chronic mucosal barrier impairment, altered intraluminal pressure, bacterial factors, and possibly treatment-related vulnerability. This case highlights that predominantly colonic PCI may occur despite predominantly small-bowel inflammatory disease and that

pneumoperitoneum in this setting does not necessarily imply frank perforation. Careful clinico-radiologic correlation remains the cornerstone for avoiding unnecessary surgery.

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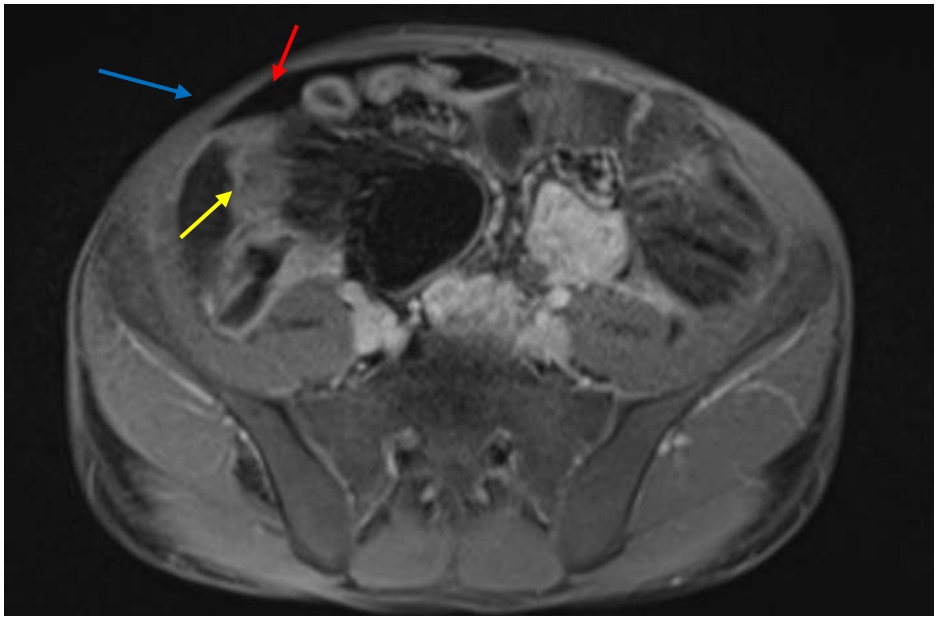
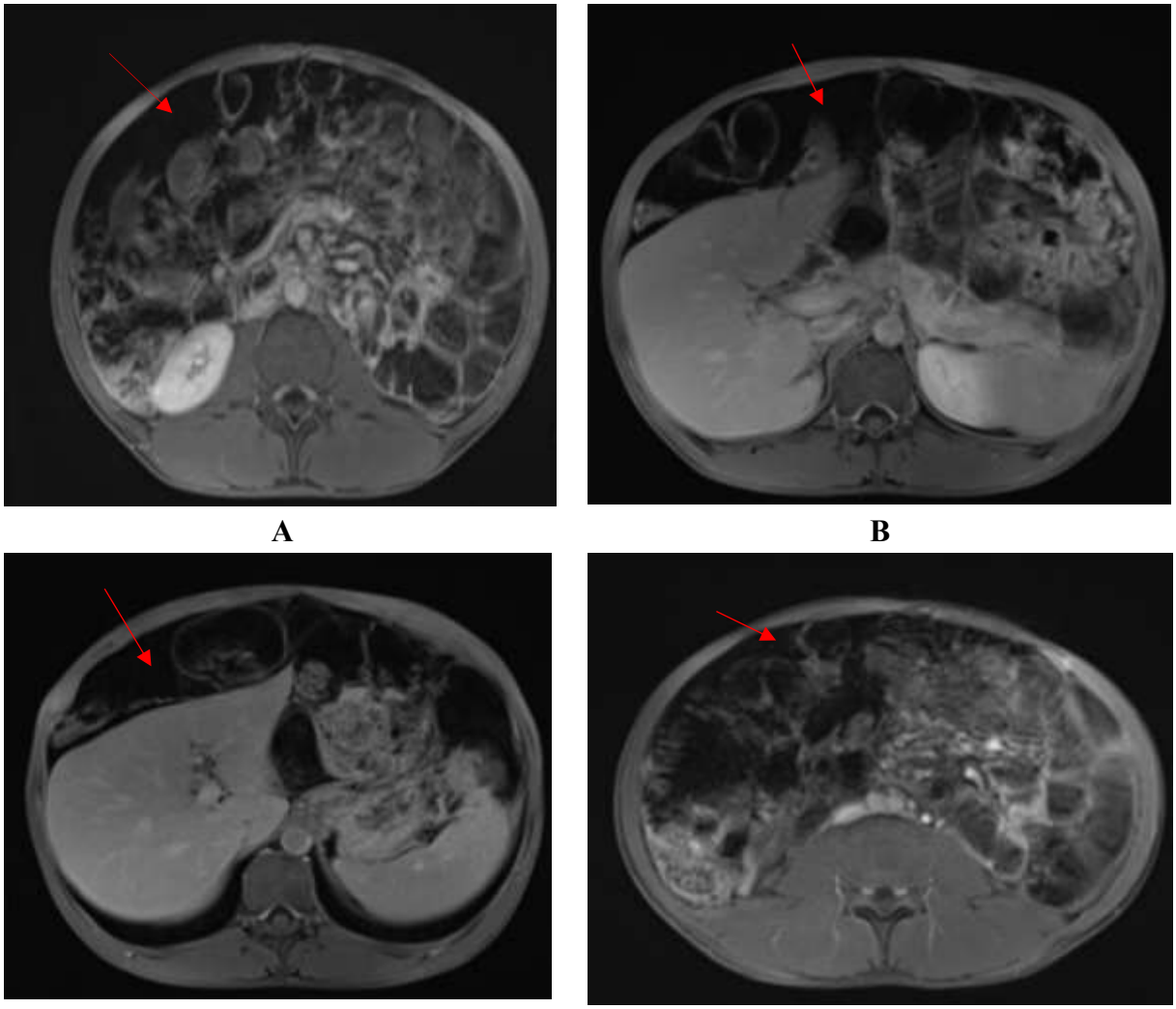


Figure 1. MR enterography findings in Crohn's disease complicated by pneumatosis cystoides intestinalis and pneumoperitoneum.

Axial MR enterography image demonstrating ileal wall thickening consistent with active Crohn's

disease (red arrow), intraperitoneal signal voids compatible with pneumoperitoneum (blue arrow), and mesenteric hyperemia reflecting active inflammatory changes (yellow arrow).

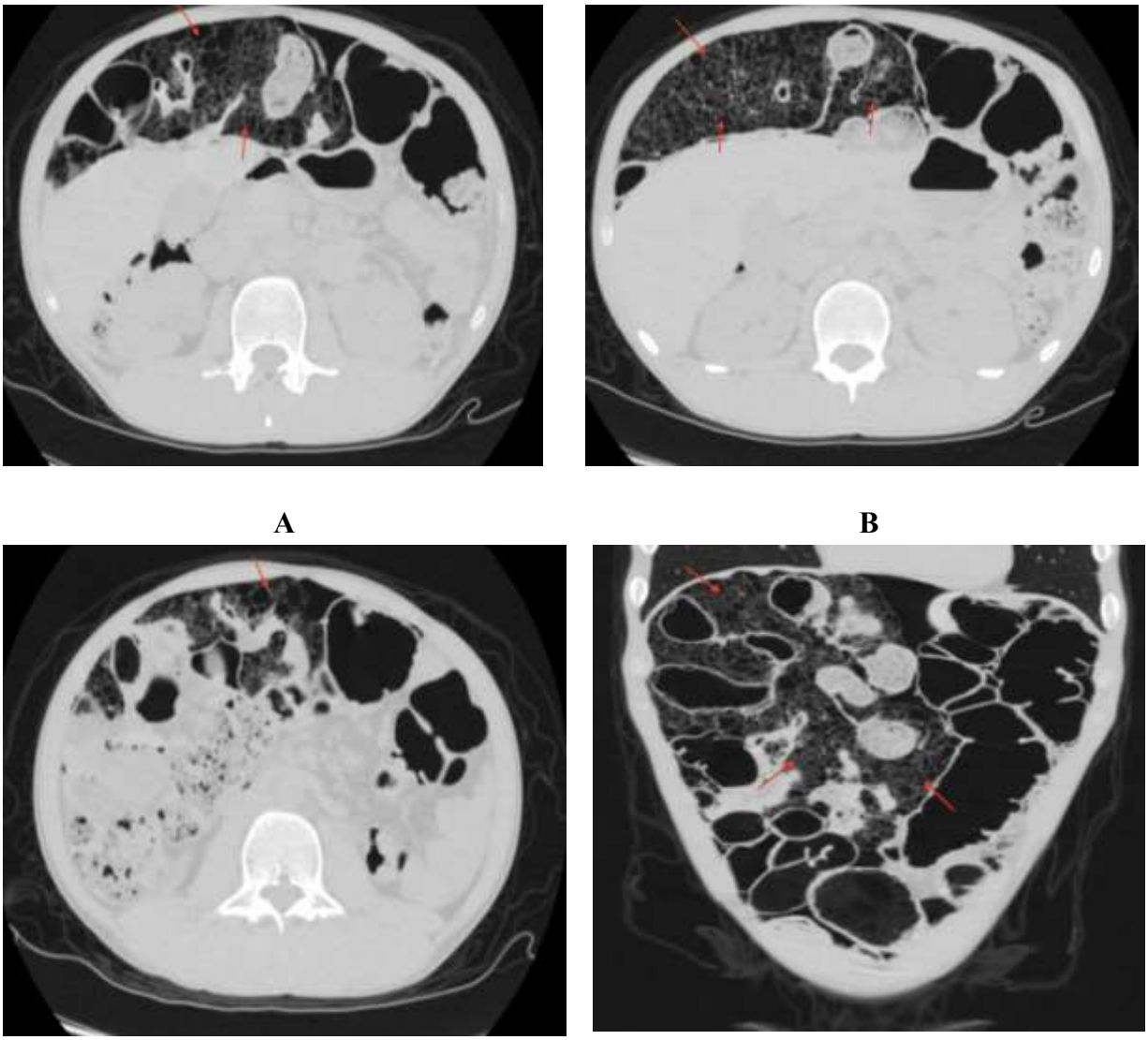


A **B**

C **D**

Figure 2 MR enterography showing multiple intraperitoneal signal voids raising suspicion of pneumoperitoneum.

(A–D) Axial MR enterography images demonstrating multiple intraperitoneal signal void foci (arrows) along the peritoneal cavity, corresponding to a flow void phenomenon that initially raised suspicion of pneumoperitoneum.



A B C D

Figure 3 : CT images demonstrating pneumatosis cystoides intestinalis.
(A–C) Axial CT images showing multiple cystic intramural gas collections within the colonic wall (arrows).
(D) Coronal reconstruction illustrating the extensive distribution of pneumatosis cystoides intestinalis.