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CASE REPORT

A Rare Case of Pneumatosis Cystoides Intestinalis in a Patient with Chronic Appendicitis

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Abstract: Chronic appendicitis is a common cause of recurrent right lower quadrant pain and is often difficult to diagnose because of its nonspecific presentation. Pneumatosis cystoides intestinalis (PCI), a rare condition characterized by gas-filled cysts in the intestinal wall, is usually associated with other gastrointestinal disorders. We report a 43-year-old woman with a one-year history of intermittent abdominal discomfort who was found to have chronic appendicitis with concurrent PCI, an unusual combination that created diagnostic uncertainty. Computed tomography revealed gas in the appendiceal region and adjacent bowel wall. Colonoscopy and laparoscopic appendectomy were subsequently performed, excluding other gastrointestinal lesions and confirming chronic appendicitis on histopathology. The patient recovered well after surgery, and follow-up imaging 6 months later showed complete resolution of PCI. This report contributes valuable insights into the complex relationship between chronic appendicitis and PCI. Awareness of this association may help improve diagnosis and management.

Keywords: chronic appendicitis, pneumatosis cystoides intestinalis, case report, gastrointestinal perforation, differential diagnosis

Introduction

Chronic appendicitis is characterized by recurrent right lower quadrant abdominal pain and poses diagnostic challenges due to its nonspecific symptoms. Although not uncommon, it is frequently underdiagnosed or misdiagnosed because of its subtle presentation.[1] Pneumatosis cystoides intestinalis (PCI) is a rare gastrointestinal disorder characterized by gas-filled cysts within the intestinal wall.[2] PCI may be primary, but most reported adult cases are secondary to other local or systemic conditions.[3–7] Secondary PCI has been reported in association with pulmonary disease, autoimmune disease, medications, organ transplantation, inflammatory bowel disease, bowel obstruction, ischemia, and other gastrointestinal disorders. Its clinical significance depends on the underlying cause and the overall clinical setting. In some patients, intramural gas represents a benign process, whereas in others it may accompany bowel ischemia, perforation, or severe intra-abdominal sepsis. For this reason, imaging findings must be interpreted together with symptoms, physical examination, and the distribution of gas on CT.

Acute exacerbation of chronic appendicitis can occasionally result in gastrointestinal perforation, which may present with imaging findings similar to PCI. Both conditions can show gas in the peritoneal cavity and around the appendix, highlighting the importance of differentiating between the two when evaluating patients with sudden worsening of symptoms.[8]

The coexistence of chronic appendicitis and PCI is unusual. We describe a patient with chronic appendicitis and secondary PCI whose imaging, endoscopic, operative, pathologic, and follow-up findings together helped clarify the diagnosis. This report highlights the importance of integrating CT findings with the patient's clinical condition and of considering PCI in the differential diagnosis of chronic appendiceal disease with unusual ileocecal gas patterns.

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Case Report

A 43-year-old unmarried female freelancer presented to the emergency department with intermittent right lower abdominal pain and discomfort that had recurred over the previous year and had worsened during the preceding day. One week before admission, she again developed bloating and intermittent pain in the right lower abdomen. She reported no upper abdominal pain, nausea, vomiting, fever, diarrhea, hematochezia, melena, or periumbilical migratory pain. She also denied shortness of breath, chest tightness, cold sweats, or other symptoms suggesting hemodynamic instability.

On admission, her heart rate was 82 beats/min, blood pressure was 123/76 mmHg, and fingertip oxygen saturation was 98%. Her abdomen was soft, without obvious tenderness, muscular guarding, palpable mass, or clear peritoneal irritation. She had a 1-year history of diabetes mellitus and was taking dapagliflozin once nightly and insulin aspartate 12 U subcutaneously once daily in the morning. She denied any history of chronic bronchitis, emphysema, or other chronic pulmonary disease. Initial laboratory testing showed a white blood cell count of 10.1 x 10^9/L, a neutrophil percentage of 82%, and a C-reactive protein level of 22 mg/L. Electrolytes and coagulation function were within normal limits.

Abdominal CT showed an elongated appendix containing intraluminal gas, extensive intramural gas within the cecal and ascending colon wall, and a small localized extraluminal gas collection adjacent to the ileocecal region. No diffuse free intraperitoneal air was identified. Because PCI is not commonly encountered in routine surgical practice, the unusual CT appearance initially raised concern during preliminary surgical image review. However, after joint reassessment with a senior radiologist and review of the relevant literature, the imaging findings, together with the patient's stable vital signs and absence of peritoneal signs, were considered more consistent with PCI associated with chronic appendicitis than with frank gastrointestinal perforation (Figure 1A and B).

After perforation had been largely excluded clinically and radiologically, colonoscopy was performed to exclude mucosal disease or another lesion in the ileocecal region. The colonoscope was advanced to the terminal ileum, where no mucosal abnormalities were observed. Notably, the mucosa of the ileocecal region appeared smooth, exhibiting clear

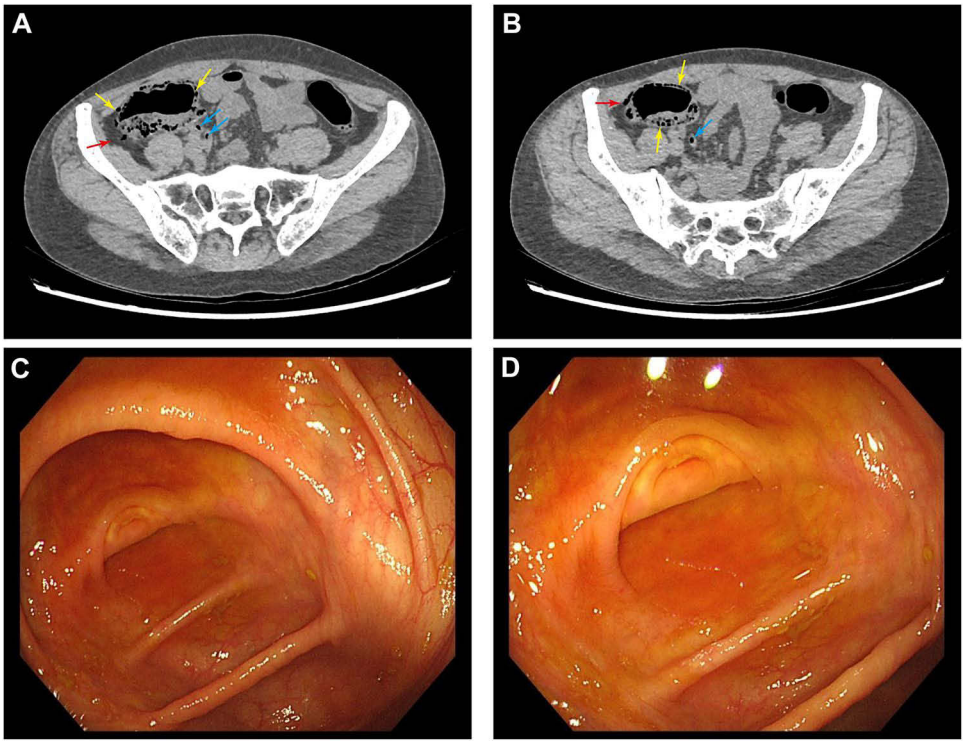

Figure 1 Abdominal imaging and colonoscopy at initial presentation. (**A**) Axial abdominal CT demonstrates extensive intramural gas within the cecal and ascending colon wall (yellow arrows), with a small localized extraluminal gas collection adjacent to the ileocecal region (red arrow); the appendix is elongated and contains intraluminal gas (blue arrow). (**B**) Axial CT section demonstrates intramural gas along the ileocecal wall (yellow arrows) without diffuse free intraperitoneal air. (**C**) Colonoscopic view of the terminal ileum and cecum reveals smooth mucosa with an intact vascular pattern and no erosions or ulcerations. (**D**) Colonoscopy shows a lip-shaped ileocecal valve and a normal appendiceal orifice without mucosal abnormality.

vascular patterns (Figure 1C). Furthermore, the ileocecal valve was lip-shaped, and no abnormalities or ulcerations were present at the appendiceal orifice (Figure 1D).

We concluded that the patient's abdominal pain over the past year was primarily due to chronic appendicitis, although the presence of PCI might have exacerbated the symptoms or resulted from chronic appendicitis. After thoroughly informing the patient and her family and obtaining their consent, we performed laparoscopic exploration and an appendectomy to alleviate the appendicitis-related symptoms. During the surgery, we observed adhesions around the appendix, likely attributable to chronic inflammation causing adhesion formation in adjacent tissues. Additionally, the appendix was tortuous with mild congestion on the serosal surface, but the ileocecal region appeared normal, lacking subserosal bubbles (Figure 2A). Because of the local chronic inflammatory adhesions, a prophylactic abdominal drain was placed at the end of the procedure. Histopathologic examination of the resected appendix confirmed chronic appendicitis. In addition to chronic inflammatory infiltration, the mural tissue showed empty-space-like changes compatible with pneumatosis-related change (Figure 2B–F).

After the surgery, we routinely administered antibiotic treatment. The patient recovered uneventfully after surgery. Her right lower abdominal pain improved on the first postoperative day, intestinal function returned, and oral intake was gradually resumed. By postoperative day 3, the white blood cell count had returned to normal, C-reactive protein had normalized, and the abdominal drain was removed. She was then discharged. At follow-up, she reported no recurrent

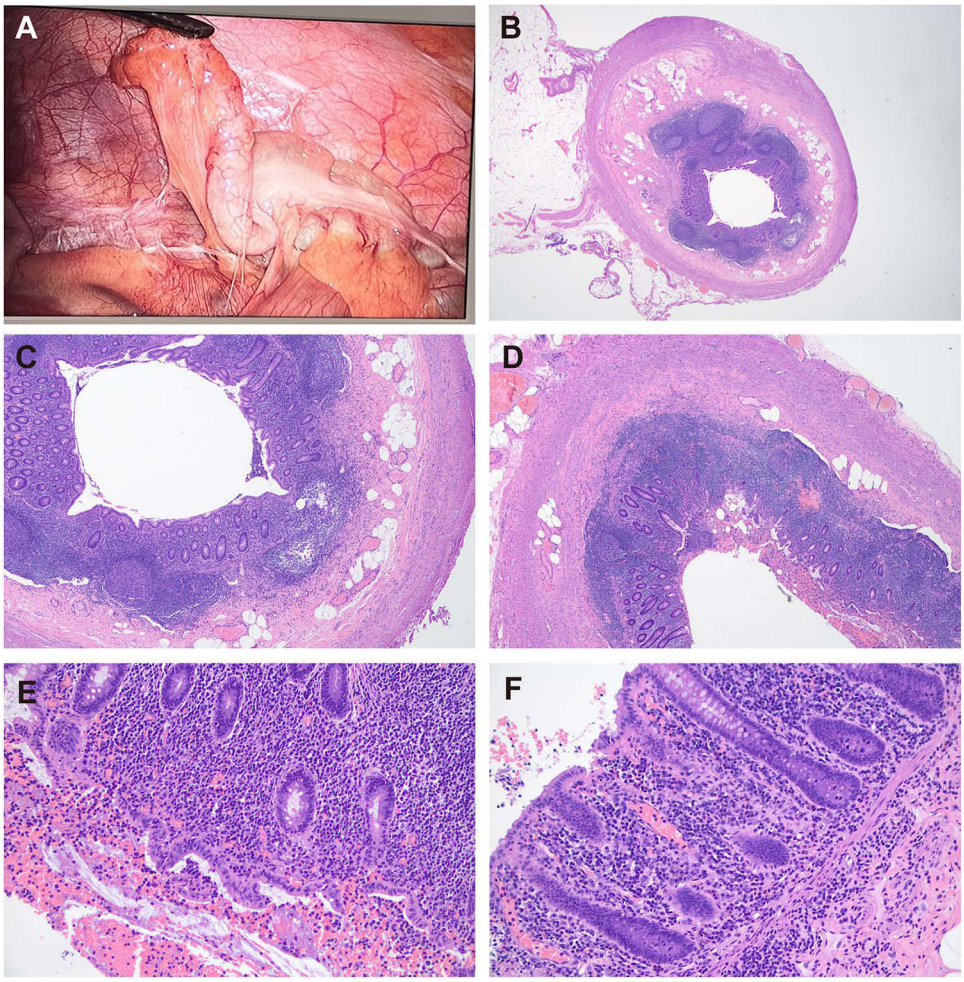

Figure 2 Intraoperative findings and histopathology of the appendix. (**A**) Laparoscopic view shows a tortuous, mildly congested appendix with pericecal adhesions; no obvious cystic lesion or subserosal gas-bubble-like structure is visible on the cecal serosa or elsewhere in the ileocecal region. (**B**) Low-power histology of the resected appendix shows wall thickening. (**C** and **D**) Hematoxylin-eosin staining at low magnification shows chronic inflammatory infiltration with several empty-space-like mural changes compatible with pneumatosis-related change. (**E** and **F**) Hematoxylin-eosin staining at higher magnification confirms chronic inflammatory infiltration and preserved glandular architecture, consistent with chronic appendicitis.

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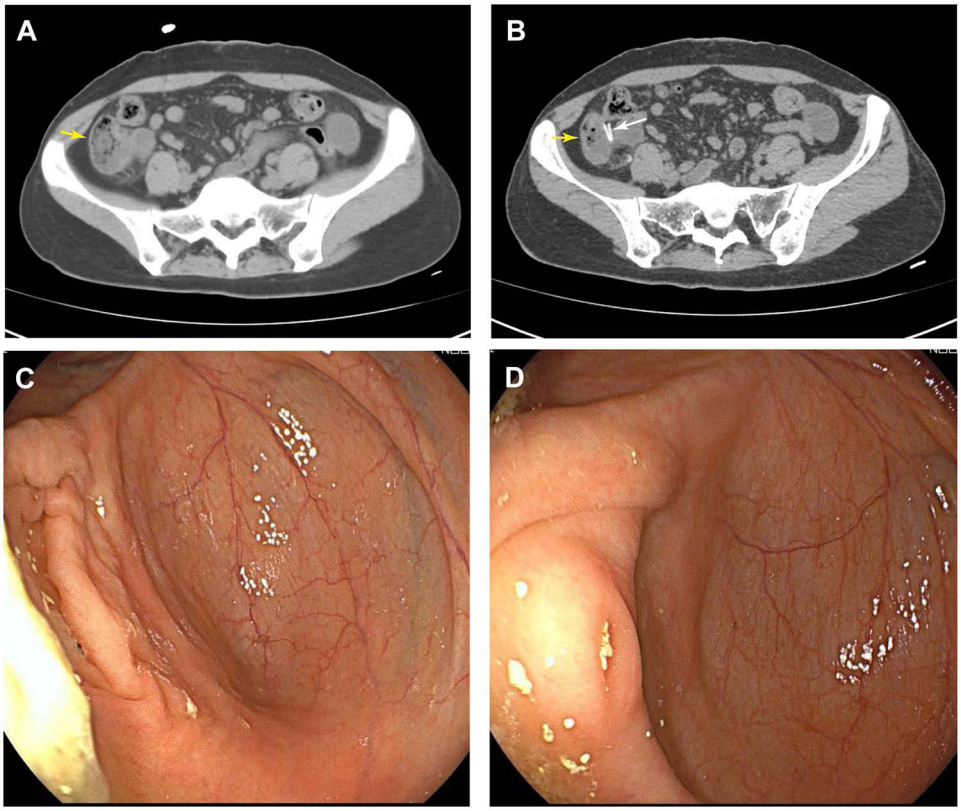

Figure 3 Six-month postoperative follow-up. (**A** and **B**) Abdominal CT obtained 6 months after surgery shows complete resolution of the previous intramural gas; the ileocecal region appears normal (yellow arrow), and a metallic ligation clip marks the appendiceal stump (white arrow). **(C** and **D)** Follow-up colonoscopy demonstrates smooth cecal and ileal mucosa with a normal-appearing appendiceal stump orifice and no residual or recurrent PCI.

abdominal discomfort and had resumed a normal diet. Six months after surgery, abdominal CT showed complete resolution of the previous intramural gas in the ileocecal region (Figure 3A and B). Colonoscopy also demonstrated a normal ileocecal mucosal surface without evidence of erosion, as well as a visible appendiceal stump orifice (Figure 3C and D).

Discussion

PCI is a rare condition characterized by gas-filled cysts within the intestinal wall. This case illustrates an unusual coexistence of chronic appendicitis and PCI. The key clinical challenge was differentiating benign intramural gas from gastrointestinal perforation. The unusual ileocecal gas pattern initially raised concern for perforation during surgical image review; however, the final working diagnosis before colonoscopy and surgery was PCI associated with chronic appendicitis. The intramural location of the gas, the absence of diffuse free intraperitoneal air, the lack of peritoneal signs, and the stable hemodynamic status all favored PCI over frank perforation.[9] Appendicitis-associated PCI is uncommon, but it has been reported in the literature, including after laparoscopic appendectomy[6,10] and in association with inflamed appendiceal disease causing bowel involvement.[2] These reports support the view that appendiceal pathology can be related to PCI in selected clinical settings.

PCI can be categorized broadly as primary or secondary.[11] Primary PCI is relatively uncommon and is usually considered benign, whereas most adult cases are secondary to another local or systemic process. Secondary PCI has been described in association with pulmonary disease, connective tissue disease, immunosuppression, gastrointestinal inflammation, obstruction, ischemia, transplantation, and drug exposure. In the present patient, the overall pattern is more consistent with secondary PCI than with primary disease because the gas distribution was localized to the ileocecal region, chronic appendiceal inflammation was present, and the intramural gas resolved completely after appendectomy. Appendicitis-related PCI has also been described in acute inflammatory settings.[12]

The possible relationship between chronic appendicitis and PCI is likely multifactorial. Among the proposed mechanisms of PCI, the bacterial and mechanical theories appear most relevant in this patient. Conservative treatment has been reported to be effective in many benign PCI cases, supporting the idea that bacterial activity and luminal gas dynamics may contribute to mural gas formation.[8,13,14] Chronic local inflammation may disrupt mucosal integrity, allowing gas-forming bacteria or bacterial products to enter the bowel wall. At the same time, repeated inflammation and altered regional motility may increase local intraluminal pressure and facilitate gas dissection into the mural layers.

Several mechanisms have been proposed for PCI, including bacterial, mechanical, and pulmonary theories.[14,15] In this case, the bacterial and mechanical explanations appear more plausible because chronic local inflammation may impair mucosal integrity and increase regional intraluminal pressure.[16] By contrast, the pulmonary mechanism is less persuasive because the patient had no chronic lung disease or respiratory symptoms. The CT findings therefore had to be interpreted cautiously in the overall clinical context rather than as direct evidence of perforation.[17,18]

PCI may be detected incidentally on imaging, but its significance depends on the underlying disorder and the clinical setting.[19] It can involve the submucosal or subserosal layer and may enter the differential diagnosis of several gastrointestinal conditions.[20] For this reason, radiologic findings should always be interpreted together with the patient's symptoms, examination findings, and laboratory data.[21]

Diabetes mellitus and antidiabetic therapy may also be relevant in this setting. Drug-associated PCI has been reported in the literature, particularly with alpha-glucosidase inhibitors, and diabetes-related metabolic or microbial factors may theoretically influence bowel gas production or mucosal vulnerability.[22] However, in this patient, a direct causal relationship with dapagliflozin or insulin cannot be established. The lesion distribution was focal rather than diffuse, the dominant pathologic process was chronic appendiceal inflammation, and the PCI resolved after surgical treatment of the appendiceal disease. Taken together, these points support chronic local inflammation as the more likely trigger.

Colonoscopy in this patient was performed only after frank gastrointestinal perforation had been largely excluded by radiologic reassessment and clinical observation. Its purpose was to exclude mucosal pathology or another lesion in the ileocecal region rather than to confirm PCI. The normal mucosal findings also supported the impression that the gas process was intramural rather than an ulcerative or perforated mucosal lesion.

The operative and pathologic findings also help interpret the case. No cystic lesion or subserosal gas-bubble-like structure was visible on the cecal serosa during laparoscopy, suggesting that the principal abnormality was not a grossly obvious serosal cystic process. Histology demonstrated chronic appendicitis and empty-space-like mural changes compatible with pneumatosis-related change. This finding should be interpreted cautiously rather than as absolute pathologic proof of PCI, but it is supportive when considered together with the CT appearance and the complete radiologic resolution after appendectomy.

Management of PCI should always be guided by the patient's clinical condition rather than by CT findings alone.[23,24] Not all intramural gas requires surgery. Conservative treatment may be sufficient in selected stable patients without signs of perforation, ischemia, or obstruction.[25] However, surgical treatment may be necessary when symptoms are driven by the associated pathology or when complications cannot be excluded.[26] In the present case, surgery was performed for symptomatic chronic appendicitis rather than because PCI itself mandated operative treatment.[3] The subsequent disappearance of the intramural gas on follow-up CT supports the interpretation that the PCI was secondary and benign in this clinical context.

Chronic appendicitis associated with PCI is rare and may mimic more urgent intra-abdominal pathology on CT. In a hemodynamically stable patient without diffuse free intraperitoneal air or peritoneal signs, intramural gas in the ileocecal region should prompt consideration of PCI as an important differential diagnosis. In this case, coordinated radiologic reassessment, cautious use of colonoscopy after perforation had been largely excluded, and definitive treatment of chronic appendicitis led to a favorable outcome and complete radiologic resolution.

Data Sharing Statement

All data generated or analysed during this study are included in this published article. Additional de-identified information can be obtained from the corresponding author (Q.Z.) upon reasonable request and with permission from the patient.

Ethics Statement

This study was conducted in accordance with the Declaration of Helsinki. This case report was reviewed and approved for publication by the Ethics Committee of Shanghai Sixth People's Hospital (Approval No. SSPH(Y)245102). Written informed consent for publication of the case details and accompanying images was obtained from the patient. All identifying information has been removed to protect patient privacy.

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Disclosure

The authors declare that they have no competing interests in this work.

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