Case Report

Extensive Pneumoperitoneum in a Hemodynamically Stable 62-Year-Old Male Managed Conservatively: A Case Report Emphasizing the Importance of Clinical Judgment Over Radiological Findings

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ABSTRACT

Pneumoperitoneum is most commonly caused by gastrointestinal perforation and classically mandates emergency surgical exploration. A distinct subgroup of spontaneous (non-surgical) pneumoperitoneum exists in which free intraperitoneal air is not accompanied by clinical peritonitis, and the source of air cannot be identified even after thorough investigation. We report a 62-year-old male who presented with diffuse abdominal pain, vomiting, and obstipation. Contrast-enhanced computed tomography (CT) of the abdomen revealed extensive pneumoperitoneum with free air under the right hemidiaphragm, multiple extraluminal air foci in the peritoneal cavity, and marked colonic distension. Despite these findings, the patient showed no signs of peritonitis and remained hemodynamically stable. After an exhaustive workup including contrast-enhanced CT, erect abdominal radiography, and sigmoidoscopy failed to identify an ongoing perforation source, a diagnosis of spontaneous pneumoperitoneum — most plausibly representing a self-sealed microscopic gastrointestinal perforation — was established as a diagnosis of exclusion. The patient was managed conservatively with nasogastric decompression, intravenous fluids, intravenous piperacillin–tazobactam for four days followed by a five-day course of oral cefuroxime after discharge, and bowel rest, with complete clinical resolution over a four-day hospitalization. Follow-up colonoscopy and upper gastrointestinal endoscopy performed fifteen days after discharge were entirely normal. This case illustrates that extensive pneumoperitoneum in a carefully selected, hemodynamically stable patient without peritonitis may be managed non-operatively when CT and endoscopy reveal no evidence of ongoing leak or contamination and close observation confirms clinical improvement. Conservative management in this setting must remain highly selective, with a low threshold for urgent laparotomy should deterioration occur.

Keywords: Spontaneous pneumoperitoneum, Conservative management, Sealed perforation, Non-surgical pneumoperitoneum, Free intraperitoneal air

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INTRODUCTION

Pneumoperitoneum, defined as the presence of free gas within the peritoneal cavity, is detected in over 90% of cases secondary to perforation of a hollow viscus, most commonly from peptic ulcer disease, diverticular perforation, or traumatic injury[1]. This condition classically presents as an acute surgical emergency requiring prompt laparotomy. However, a distinct subgroup termed spontaneous or non-surgical pneumoperitoneum encompasses various non-perforative etiologies including intrathoracic, intra-abdominal, gynecologic, and iatrogenic causes[2]. At the far end of this spectrum lies idiopathic pneumoperitoneum, the rarest form, in which no source of free air is identified despite exhaustive investigation[3].

It is increasingly recognized, however, that many cases labeled "idiopathic" or "spontaneous" pneumoperitoneum may in fact represent self-sealed microscopic gastrointestinal perforations — small mucosal or mural defects that close before the onset of generalized peritonitis or ongoing contamination [2]. Such patients may present with abdominal symptoms yet remain stable without peritonitis, precisely because the perforation has sealed. This distinction is clinically important: a sealed perforation is not

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a benign entity by definition, but rather a diagnosis that obligates rigorous exclusion of an ongoing leak and careful patient selection if surgery is to be avoided. Given the rarity of these presentations, management algorithms remain poorly defined, and many such cases historically underwent unnecessary exploratory laparotomy[4].

This case is reported in accordance with the SCARE criteria[5].

CASE PRESENTATION

A 62-year-old male presented to the emergency department with a one-day history of diffuse abdominal pain of sudden onset, associated with two to three episodes of yellowish vomiting and inability to pass stool for the preceding 48 hours. He had undergone hemorrhoidectomy four years prior but had no other significant medical or surgical history.

On examination, the patient was conscious, oriented, and hemodynamically stable. His abdomen was distended and tympanitic with diffuse tenderness. Crucially, there was no guarding, no rebound tenderness, and bowel sounds were present. Digital rectal examination revealed normal anal tone with no palpable mass. His vital signs remained stable throughout the initial assessment.

Laboratory investigations revealed a hemoglobin of 14.7 g/dL, white blood cell count of 8.7 × 10^3/µL with a differential of 82.6% neutrophils and 11.5% lymphocytes, and platelet count of 189 × 10^3/µL. Serum amylase was 69 U/L. Arterial blood gas analysis showed a pH of 7.36, partial pressure of carbon dioxide of 43.3 mmHg, partial pressure of oxygen of 40.7 mmHg, bicarbonate of 23.0 mmol/L, sodium of 148.9 mmol/L, and potassium of 3.25 mmol/L. Inflammatory markers were not significantly elevated.

An erect abdominal radiograph demonstrated a large crescent of free air beneath the right hemidiaphragm with generalized gaseous distension of bowel loops (Figure 1).

Contrast-enhanced computed tomography of the abdomen and pelvis revealed free intraperitoneal air under the right hemidiaphragm, multiple discrete foci of extraluminal air scattered throughout the peritoneal cavity predominantly in the pericolic region near the splenic flexure, and massive gaseous distension of the entire colon with a maximum caliber of 6.4 cm. The ileum was also distended to a maximum caliber of 2.5 cm, while the jejunum appeared collapsed, suggesting a functional ileus pattern rather than mechanical obstruction. Crucially, there was no extraluminal contrast leak, no identifiable bowel wall defect, no bowel wall thickening or pneumatosis to suggest ischemia, no intra-abdominal abscess, and no ascites; there was also no pneumomediastinum or subcutaneous emphysema. The liver, spleen, bilateral kidneys, and adrenal glands appeared normal (Figure 2 A,B,C).

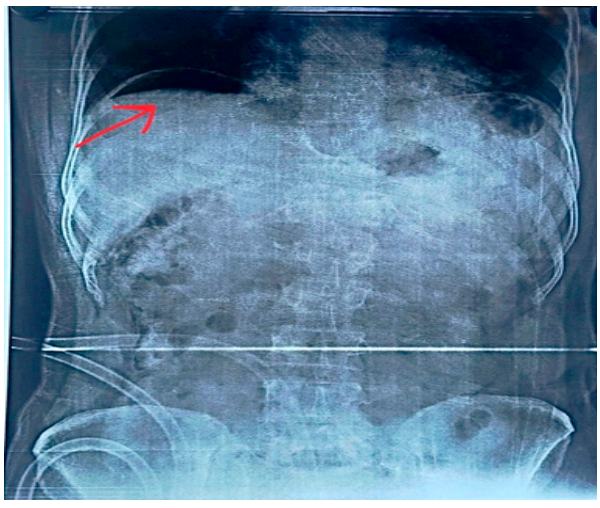


Fig. 1: Erect abdominal radiograph showing free air under the right hemidiaphragm (arrow) and gaseous distension of bowel loops

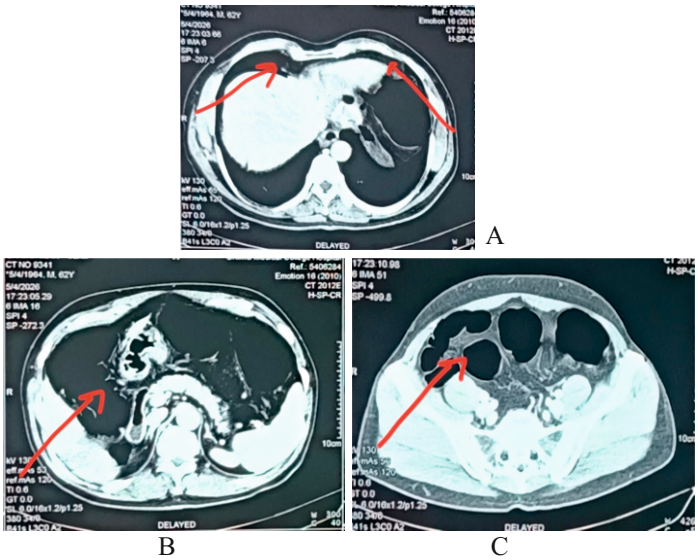


Fig. 2(A): Axial contrast-enhanced computed tomography images of the upper abdomen demonstrating free intraperitoneal air (arrows) beneath the diaphragm and around the liver surface, **(B,C)** mid and lower abdomen showing extensive pneumoperitoneum, colonic distension, and multiple extraluminal air foci in the pericolic region

Given the extensive pneumoperitoneum, an extended sigmoidoscopy was performed, which revealed normal mucosa from the anal canal to the splenic flexure. The patient was uncooperative for full colonoscopy; a complete colonic evaluation was therefore not achieved during the acute admission. No mucosal lesion, active bleeding, or perforation site was identified. Biopsy was not taken.

MANAGEMENT AND OUTCOME

Despite extensive radiological pneumoperitoneum, the patient remained hemodynamically stable with no signs of peritonitis on repeated clinical examination. After multidisciplinary discussion, a working diagnosis of spontaneous pneumoperitoneum — most plausibly a self-sealed microscopic gastrointestinal perforation — was established as a diagnosis of exclusion. The decision was made to pursue a trial of conservative management rather than emergency laparotomy, on the explicit understanding that any clinical deterioration (development of peritonitis, hemodynamic instability, rising inflammatory markers, or persistent obstruction) would prompt immediate surgical exploration.

The patient was kept nil per os with nasogastric tube decompression. Intravenous fluid resuscitation was initiated with normal saline and ringer lactate at 100 mL per hour, with potassium correction. Intravenous pantoprazole 40 mg twice daily, intravenous ondansetron 8 mg as needed for vomiting, and intravenous piperacillin–tazobactam three times daily were administered for four days. Piperacillin–tazobactam was chosen to provide broad-spectrum aerobic and anaerobic cover against translocated enteric flora in the setting of a probable sealed colonic perforation with extensive pneumoperitoneum. The patient remained afebrile throughout, with stable vital signs and no clinical deterioration.

The patient was monitored with serial abdominal examinations and vital sign assessment. Over the subsequent 48 hours, the abdominal pain gradually subsided and distension improved, with no fever, tachycardia, or worsening tenderness at any point. The patient passed flatus and tolerated oral intake without recurrence of symptoms. He was discharged on the fourth hospital day on oral cefuroxime (Ceftum) 500 mg twice daily for five days, in addition to oral analgesics, proton pump inhibitors, and stool softeners. Follow-up was arranged in the surgical outpatient department and in the urology clinic for concomitant benign prostatic hyperplasia.

The patient remained entirely asymptomatic at outpatient review. Fifteen days after discharge, colonoscopy and upper gastrointestinal endoscopy were performed; both examinations were normal, with no mucosal lesion, stricture, ulcer, or site of prior perforation identified. This completed the gastrointestinal evaluation that had been limited by poor cooperation during the acute admission and provided further reassurance that no ongoing or residual gastrointestinal source existed.

DISCUSSION

Pneumoperitoneum without peritonitis presents one of the most difficult decision-making dilemmas in acute abdominal surgery. The presence of free intraperitoneal air, particularly when extensive, reflexively triggers a recommendation for laparotomy; yet a recognized subset of patients with free air has no ongoing gastrointestinal leak and recovers without surgery[2]. The central challenge is distinguishing, at presentation, between an ongoing perforation with early or atypical presentation and a sealed or non-perforative source of air.

The justification for non-operative management rested on convergent evidence rather than any single negative finding: (1) the patient was hemodynamically stable with soft signs only — diffuse tenderness without guarding, rebound, or absent bowel sounds; (2) contrast-enhanced multidetector CT, which predicts the site of gastrointestinal perforation with high accuracy[6], showed no bowel wall defect, no extraluminal contrast extravasation, no abscess, and no ascites — findings that would be expected with an ongoing leak; (3) endoscopic evaluation to the splenic flexure revealed normal mucosa with no lesion or bleeding point; and (4) the subsequent clinical trajectory — resolution of pain and distension over 48 hours with return of bowel function — was consistent with a sealed source rather than progressive contamination. We explicitly acknowledge the principal limitation of the acute workup: colonoscopy was incomplete during the admission, and an occult proximal colonic or small bowel source could not be definitively excluded at that time. This gap was subsequently addressed by normal colonoscopy and upper gastrointestinal endoscopy fifteen days after discharge. Taken together, the evidence supports a sealed or non-

progressive source rather than an ongoing perforation; nonetheless, this case should not be interpreted as proof of a truly idiopathic process.

We agree with the view that many cases historically labeled idiopathic pneumoperitoneum represent self-sealed microscopic perforations of the gastrointestinal mucosa that close before the onset of generalized peritonitis[2]. The presenting triad in our patient — abdominal pain, vomiting, and obstipation — indicates underlying abdominal pathology and argues against a purely incidental finding. The massive colonic and ileal distension observed may have contributed to transient mural stress, allowing a minute amount of intraluminal gas to extravasate before sealing, although this mechanism remains speculative and cannot be proven retrospectively. We have therefore framed the diagnosis throughout this report as spontaneous pneumoperitoneum, most plausibly representing a sealed microscopic perforation, rather than claiming a truly idiopathic entity.

A critical learning point is the primacy of clinical judgment over radiological findings alone. While CT demonstrated extensive pneumoperitoneum that would typically mandate emergency surgery, the absence of peritonitis, hemodynamic stability, supportive CT findings (no leak, no contamination), and the negative endoscopic evaluation permitted a trial of conservative management under close observation. This aligns with the management algorithm proposed by Sakaguchi et al., which stratifies treatment based on the presence of peritonitis and the patient's general condition[1], and with the broader principle that patient physiology, not the volume of free air, should drive the decision to operate.

The literature supports selective conservatism in this setting. Tani et al. reported a similar case of pneumoperitoneum managed conservatively despite guarding and leukocytosis[7]. Tanaka et al. described a bedridden 77-year-old woman with massive pneumoperitoneum localized to the upper abdomen who recovered rapidly with conservative treatment alone [8]. At the population level, the data argue for restraint: in a retrospective study by Peker et al., spontaneous pneumoperitoneum accounted for 2.36% of 338 patients with radiologically confirmed free air[9]; Chandler et al. reported that 28% of patients with spontaneous pneumoperitoneum underwent laparotomy without identification of any perforated viscus[10]; Mularski et al. found that 23% of 196 cases of spontaneous pneumoperitoneum underwent negative surgical exploration[2]; and Morrison et al. reported an exploratory laparotomy rate of 20%[11]. Hannan et al. further reported persistent idiopathic pneumoperitoneum visible on serial imaging yet managed successfully without surgery, emphasizing that radiological resolution is not mandatory for conservative success[4]. Sidiqi et al. cautioned that asymptomatic free air remains a dangerous trap, and that contrast-enhanced CT excludes gastrointestinal perforation with only approximately 86% accuracy[12] — a figure that underlines the necessity of both careful patient selection and honest acknowledgment of residual uncertainty.

Non-operative treatment of pneumoperitoneum is appropriate only in a narrow set of circumstances: hemodynamic stability, absence of peritonitis on serial examination, CT evidence arguing against an ongoing leak (no extravasation, abscess, or significant ascites), and institutional capacity for immediate laparotomy should deterioration occur. Outside this envelope — peritonitis, instability, immunocompromise, or CT signs of ongoing contamination — surgery remains the standard of care[13]. Our case does not challenge that standard; it illustrates its careful, individualized application.

CONCLUSIONS

This case illustrates that extensive pneumoperitoneum, even when accompanied by abdominal symptoms and striking radiological findings, may be managed conservatively when a rigorous workup reveals no evidence of an ongoing perforated viscus and the patient remains hemodynamically stable without peritonitis. The subsequent normal follow-up colonoscopy and endoscopy further support the diagnosis of a sealed, self-limiting source. We favor framing such presentations as spontaneous pneumoperitoneum, frequently representing a self-sealed microscopic perforation, rather than truly idiopathic disease. Conservative management in this setting must remain highly selective and conditional — reserved for stable patients without peritonitis, supported by CT and endoscopic evidence, conducted under close serial observation, and with a low threshold for urgent laparotomy. Individualized algorithms that prioritize

patient physiology over the isolated radiological finding of free air can prevent unnecessary surgical morbidity in this rare but increasingly recognized entity.

DECLARATIONS

Ethical Statement

This case report was conducted in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed Consent Statement

Written informed consent was obtained from the patient for the 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 on reasonable request.

Conflict of Interest None

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