

Received: 2022.01.09

Accepted: 2022.03.23

Available online: 2022.03.31

Published: 2022.05.05

# Colonic Pseudolipomatosis: A Rare but Characteristic Endoscopic Condition

## Authors' Contribution:

Study Design A  
Data Collection B  
Statistical Analysis C  
Data Interpretation D  
Manuscript Preparation E  
Literature Search F  
Funds Collection G

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**Financial support:** None declared

**Conflict of interest:** None declared

**Patient:** Female, 65-year-old  
**Final Diagnosis:** Pseudolipomatosis  
**Symptoms:** Diarrhea  
**Medication:** —  
**Clinical Procedure:** —  
**Specialty:** Gastroenterology and Hepatology

**Objective:** Rare disease

**Background:** Colonic pseudolipomatosis (CP) can pose a diagnostic challenge due to its rare incidence and multiple presentations, most of them not very familiar to the endoscopist. Its etiology and pathogenesis have not been completely clarified. It can be related to mucosal iatrogenic injury caused during endoscopic examination or to chemical injury caused by residual disinfectants on the surface of the scope after cleansing. Imaging tests such as CT or MRI do not contribute to the diagnosis, but this condition has characteristic features that must be differentiated from pre-malignant lesions, like lateral-spreading tumors, in order to avoid further investigation and unnecessary treatment, such as endoscopic mucosal resection.

**Case Report:** We report a case of a 65-year-old man who underwent to a screening colonoscopy due to his strong family history of colorectal cancer. Confluent whitish laterally-spreading lesions with a round pit-pattern in white-light HD scope were identified in the cecum and ascending colon. The lesion was biopsied with a cold forceps. Histopathologic analysis revealed multiples cysts filled with gas within the mucosal layer, associated with a mild inflammatory process, mainly composed of mononuclear cells and eosinophils. No giant multinuclear cells were identified. Moreover, although there was a mild inflammatory process in the epithelium, the architectural organization and tissue maturation were preserved with no nuclear atypia, consistent with a diagnosis of colonic pseudolipomatosis.

**Conclusions:** Colonic pseudolipomatosis is a rare, benign condition that must be not mistaken for more serious conditions, as CP requires no further investigation or treatment. In this setting, proper diagnosis is key to avoid unnecessary procedures.

**Keywords:** Colonic Diseases • Colonoscopy • Lipomatosis

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/936045>



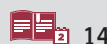
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## Background

Colonic pseudolipomatosis (CP) is a benign condition that can pose a diagnostic challenge due to its rare incidence and multiple presentations [1]. As this entity is not very familiar to the endoscopist, the diagnosis can be confused with other lesions.

There are few reports of this condition in the literature and it appears that the finding of CP is somewhat “endoscopist-specific” since there are extremely experienced endoscopists who have never seen a lesion of this type, while other endoscopists see it frequently. This would imply that some difference in technique or patient selection may play a part in the development or recognition of this lesion [2].

The etiology and pathogenesis of CP have not been completely clarified. It may be related to mucosal iatrogenic injury caused during endoscopic examination or to chemical injury caused by residual disinfectants on the surface of the scope after cleansing.

Imaging tests such as CT or MRI do not contribute to the diagnosis, but this condition has characteristic endoscopic features that must be differentiated from pre-malignant lesions, like lateral-spreading tumors (LSTs), in order to avoid further investigation and unnecessary treatment, such as endoscopic mucosal resection (EMR) [3,4].

We present a case of CP in an asymptomatic patient undergoing colorectal cancer screening videocolonoscopy, review the endoscopic and histologic features, and discuss its clinical significance.

## Case Report

We report a case of a 65-year-old married man who was born in Rio de Janeiro, with mild constipation controlled by regular fiber intake and no comorbidities. Previous lower endoscopies revealed no polyps, except for the last exam, done in 2018, in which a microvesicular hyperplastic polyp was retrieved from the sigmoid colon.

The patient was asymptomatic, and due to his strong familiar history of colorectal cancer (CRC), he underwent a screening colonoscopy in 2021. A full exam after adequate bowel preparation (Boston 9) was undertaken. Confluent whitish laterally-spreading lesions with a round pit-pattern in white-light HD scope were identified in the cecum and ascending colon. The lesion was biopsied with cold forceps (**Figure 1**). The remaining colonic segments were unremarkable.

Histological evaluation revealed accumulation of gas in the colonic mucosa. The lamina propria presented clear (air) spaces of variable size, in aggregates, isolated or confluent, with an apparently empty center, surrounded by an inflammatory infiltrate composed of mononuclear cells and eosinophils. No giant multinuclear cells were identified. The epithelial lining appeared slightly reactive and hyperplastic, maintaining regular architectural organization, without nuclear atypia. The air spaces resembled fat cells (thus the name pseudolipomatosis). The focal nature, intramucosal location, and lack of nuclei distinguish this from adipose tissue (**Figure 2**).

## Discussion

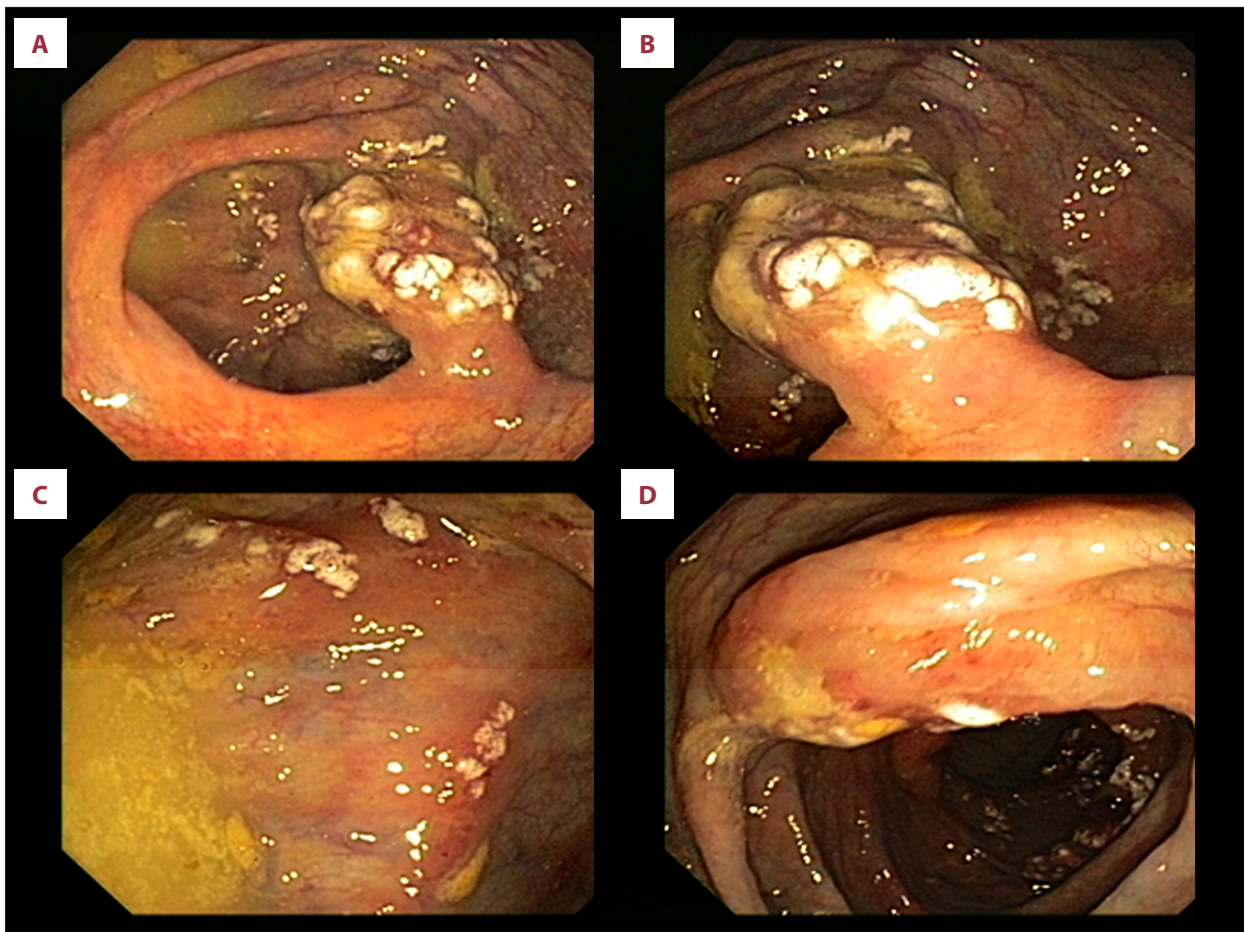
The term pseudolipomatosis was first coined by Snover et al in 1985 [2]. CP is a rare, benign condition, with underestimated prevalence [5] of about 0.02% to 0.3% in series of endoscopic exams [6]. It is most commonly found between the fifth and sixth decades of life [6], with no clear sex preference [1,5]. In general, patients are asymptomatic, but symptoms like chronic diarrhea, bloating, lower gastrointestinal tract bleeding, and positive fecal occult blood test have been associated with CP [1,5]. Mucosal lesions disappear over weeks or months without treatment [1,6,7].

CP is more often present in the left colon, while some reports [4] have described a similar incidence between right- and left-sided lesions [6,7]. Lesions have also been described in the rectum, skin, duodenum, stomach, endometrium, and oral/nasal mucosa [4].

Usually, CP manifests as yellow or whitish lesions, single or multiple, and sometimes as confluent plaques located in 1 or more colonic segments. Lesions may vary from 0.2 to 5 cm in width, most with a peripheral erythema [1]. More commonly they are visualized while withdrawing the endoscope, but sometimes large lesions are found during insertion [3].

Histopathology analysis reveals empty spaces in the lamina propria, varying from 20 to 240  $\mu\text{m}$  wide [6]. Specific staining for fat and mucin is routinely negative, and cholesterol crystals are not visualized in the exam with polarized light. No lipid deposits are identified by Sudan Black staining or by immunohistochemistry, and it is usually negative for anti-CD 31, anti-CD 34, and anti-protein S100 [1,5].

CP must be differentiated from similar conditions such as colonic lipomatosis, cystic pneumatosis, colonic lymphangioma, hyperplastic lipomatosis of ileocecal valve, colonic lipomas, malakoplakia, and pseudomembranous colitis [1,6].



**Figure 1.** Endoscopic aspect: confluent whitish lateral-spreading lesions with a round pit-pattern in white-light HD scope. (A) Cecum and ileocecal valve. (B) Cecum/ascending colon transition. (C) Cecum. (D) Ascending colon.

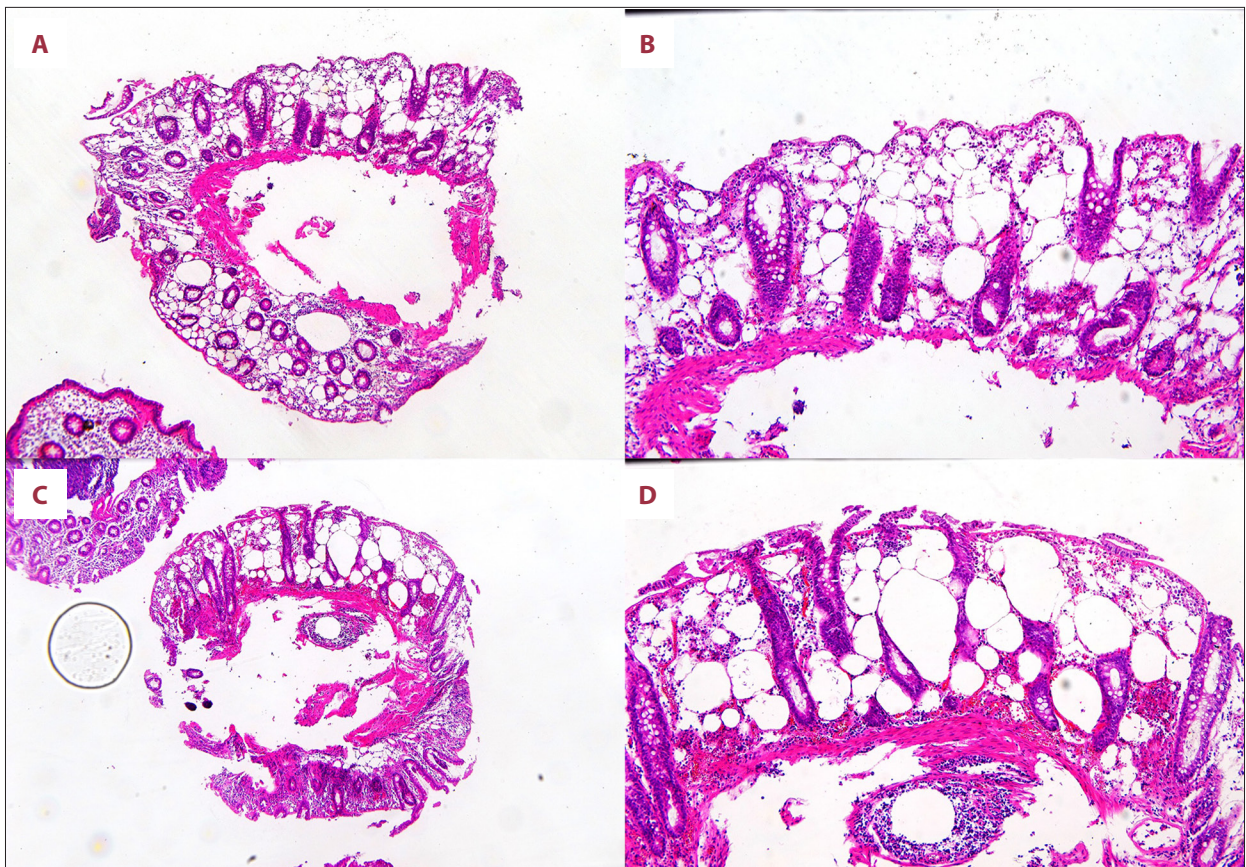
Unlike lipomatosis, pseudolipomatosis shows no adipose cells [6]. In cystic pneumatosis, colonic epithelial lining present mild local edema, with some gas bubbles bursting as the mucosal layer retracts. Histologically, it is noteworthy that there are empty cysts in the submucosa rather than in mucosal layer, surrounded by macrophages and giant cells infiltrate [8].

Colonic lymphangioma have great lymphatic vascular cavities in the colonic wall bounded by endothelial cells that are CD 31- and CD 34-positive. Hyperplastic lipomatosis of the ileocecal valve and colonic lipoma are both characterized by the presence of adipose cells. Malakoplakia of the colon reveals a chronic inflammatory process characterized by closely packed histiocytes containing calcospherites know as Michaelis-Guttman bodies [9].

CP is not considered an infectious disease, but its etiology and pathogenesis are unclear. It can be related to mucosal iatrogenic injury caused by epithelial stretch, abrasion, biopsy, and hyperinsufflation, which allow gas to infiltrate into the colonic wall. CP has also been associated with chemical injury

caused by residual disinfectants on the surface of the scope after cleansing with hydrogen peroxide [1,2,5,7]. A pulmonary source of the gas seems very unlikely, since experiments in animals and in cadavers have rarely reproduced submucosal cystic into the colonic wall, but subserosal cysts were observed [8]. Snover and Cox have suggested that CP might be operator-dependent [2].

Kim and Baek [3] described an increase in number of CP cases as glutaraldehyde was replaced with peracetic acid for scope cleansing. The authors related a series of 12 cases out of 1276 patients who received colonoscopies during a 1-year period (0.94%), and they were able to induce mucosal lesions, by comparing glutaraldehyde 2%, ethanol 70% and peracetic acid 2%. In a pig model, lesions were very similar to that observed in CP from human colonic mucosa reproduced after use of peracetic acid, in which the mucosal effect was directly related to higher concentration, while no lesion was induced after use of glutaraldehyde or ethanol [10]. Likewise, Sheehan and Brynjolfsson [11] studied the effect of peracetic acid on rat colonic mucosa, with histopathologic results very



**Figure 2.** Histopathologic analysis revealed multiples cysts filled with gas, within the mucosal layer, associated with a mild inflammatory process, mainly composed of mononuclear cells and eosinophils. Architectural organization and tissue maturation were preserved with no nuclear atypia, consistent with a diagnosis of colonic pseudolipomatosis (hematoxylin-eosin stain). (A) 4x. (B) 10x. (C) 2x. (D) 20x.

similar those reported in the Kim and Baek study, while other authors were not able to experimentally induce CP after using glutaraldehyde. Similarly, Snover et al [2] were unable to create CP by injecting air directly into colonic submucosa, and Waring et al [12] failed to induce CP after hyperinsufflation in a colon cadaver study.

Brevet et al [5] described 9 cases out of 2099 exams performed over 2 years (0.4%, prevalence). All patients were male, with a mean age of 52 years. Three cases were right-sided, 4 were found in the transverse colon, and 2 cases were left-sided. Eight cases were observed during endoscope insertion, while 1 case was observed during withdrawal, which suggests that the lesions found were probably not caused by or at least were not induced by the disinfection process, similar to the present case reported here.

Finally, Nakasono et al [13] classified CP into 2 groups by histopathologic analysis of 15 lesions from 14 patients, based on the size of the cysts. There was no difference between the groups regarded to sex, age, or other clinical conditions. Group

A, with major/minor size cyst ratio  $<3$ , presented lesions in the upper part of the muscularis mucosae layer with no submucosal involvement, smaller cysts, and no association with lymphatic follicles. Group B, with major/minor size cyst ratio  $>4$ , presented lesions in the lower part of the muscularis mucosae layer, sometimes into the submucosa, and had more variable cyst sizes and positive correlation with lymphatic follicles. Although the pathogenesis of group A was not clearly explained, the authors observed that findings of CP were closely related to the histopathologic findings observed in pneumatosis coli, suggesting that these 2 conditions had the same pathogenesis, ie, the penetration of the gas through the colonic crypts during colonoscopy [8,14].

## Conclusions

Colonic pseudolipomatosis is a rare, benign condition that must be not mistaken for more serious conditions, as CP requires no further investigation or treatment. In this setting, proper diagnosis is key to avoid unnecessary procedures.

## Institutions Where Work Was Done

Uniprocto and Gastroenterology Clinic, Brasília, and Brasiliense Laboratory, Brasília, Brazil.

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