

Colitis cystica profunda in a patient with ulcerative colitis: Case report and a brief review of the literature

Idanys Albanes³, Nelson Chang Tsang³, Luigi Colangelo², Corey Steinman², Melissa Perez⁴, Jessica Jahoda^{1,5} and Mohamed Aziz^{1,6,*}

¹ Research Writing & Publication (RWP), LLC, NY, USA.

² American University of the Caribbean, AUC, St. Maarten.

³ Ross University School of Medicine, Barbados.

⁴ St. George's University School of Medicine, Grenada.

⁵ Memorial Healthcare System, Pembroke Pines, FL, USA.

⁶ Saint Vincent's Comprehensive Cancer Center, New York City, NY.

World Journal of Biology Pharmacy and Health Sciences, 2026, 25(02), 193-200

Publication history: Received on 28 December 2025; revised on 07 February 2026; accepted on 10 February 2026

Article DOI: <https://doi.org/10.30574/wjbphs.2026.25.2.0098>

Abstract

Colitis cystica profunda (CCP) is an uncommon benign disease of the colorectum characterized by mucin-filled cystic glands in the submucosal and deeper layers of the intestinal wall. Its clinical, endoscopic, and radiological presentations are difficult to differentiate from colorectal cancer, particularly in patients suffering from inflammatory bowel disease. In this article, we report a 36-year-old man with an 8-year history of ulcerative colitis who was admitted with a history of intermittent rectal bleeding, mucoïd discharge, tenesmus, and urgency for the last 6 months. A 4-cm ulcerated sessile mass at the rectosigmoid junction, suggestive of carcinoma, was demonstrated by colonoscopy. Cross-sectional imaging revealed focal rectal wall thickening, and pelvic MRI identified a submucosal lesion with multiple cystic spaces resulting in a characteristic 'Swiss cheese' appearance. Initial superficial biopsies were nondiagnostic. The endoscopic ultrasound-guided core biopsy demonstrated colonic glands displaced into the submucosa and muscularis propria, associated with smooth muscle proliferations and fibrosis, without cytologic atypia. Immunohistochemical (IHC) studies established a benign colonic epithelial origin, supporting the diagnosis of CCP. Based on the patient's symptoms, size of the lesion, and suspicion of malignancy, the patient underwent laparoscopic low anterior resection with primary anastomosis. The final pathology confirmed the diagnosis of CCP with negative margins without dysplasia or malignancy. At 32 months of follow-up, the patient was symptom-free with no evidence of recurrence. This case emphasizes the need to consider CCP as a benign mimicker of colorectal cancer. It demonstrates the benefits of deep tissue sampling and multidisciplinary team review in achieving an accurate diagnosis and optimizing patient management.

Keywords: Colitis Cystica Profunda; Inflammatory Bowel Disease; Submucosal Lesion; Swiss Cheese Appearance

1. Introduction

Colitis cystica profunda (CCP) is a rare, benign, non-neoplastic condition characterized by the presence of mucin-filled cystic spaces within the submucosa of the colon and rectum. [1] While its exact pathogenesis remains debated, it is widely considered a reactive process secondary to chronic mucosal injury and repair, often associated with conditions such as solitary rectal ulcer syndrome, rectal prolapse, and chronic inflammatory bowel disease (IBD). [2] [3] Despite its benign nature, CCP is of significant clinical importance due to its propensity to mimic malignant processes, particularly mucinous adenocarcinoma, both endoscopically and radiographically. [1] [4] [5] The condition is

* Corresponding author: Mohamed Aziz

exceedingly rare, with an estimated incidence that remains poorly defined in the general population and even less frequently reported in the specific context of ulcerative colitis. [6]

The diagnostic journey for CCP is fraught with challenges. The most significant pitfall is the potential for misdiagnosis as a malignancy, which can lead to unnecessarily radical surgical interventions. [16] Conversely, superficial endoscopic "pinch" biopsies often fail to capture the characteristic submucosal cysts, leading to inconclusive or misleading results that may delay definitive diagnosis. [17] Furthermore, management strategies remain controversial, ranging from conservative medical therapy to surgical resection, with no established consensus on the optimal approach for symptomatic or large lesions. [6] [7] A notable gap in the current literature is the scarcity of reports detailing CCP in patients with long-standing ulcerative colitis, leaving clinicians with limited guidance on how the underlying inflammatory state influences the presentation, diagnosis, and long-term prognosis of this rare entity.

2. Case Presentation

2.1. Clinical presentation

A 36-year-old male presented to the gastrointestinal clinic with complaints of intermittent rectal bleeding and mucoid discharge for 6 months. He first noticed bright red blood, which gradually increased and occurred nearly every time he defecated. During the last 2 months, he had been suffering from intermittent crampy lower abdominal pain and tenesmus. The patient also complained of an ongoing sense of incomplete evacuation and of infrequent mucus passage. He reported no fever, weight loss, or altered bowel habit except for increased urgency. His primary care physician started topical therapy for presumed hemorrhoids without any relief. He subsequently had a colonoscopy by a gastroenterologist, which found a polypoid ulcerated mass in the rectosigmoid segment with suspicion of malignancy. The alarming appearance on endoscopy and persistence of symptoms despite conservative treatment resulted in a referral to a colorectal surgeon for further evaluation.

2.2. History, physical exam, and laboratory data

The patient was diagnosed with ulcerative colitis eight years ago and has successfully managed the condition with mesalamine since then. During this period, he suffered two mild flares, which he effectively controlled with brief corticosteroid therapy. He reported taking his medications as prescribed, and his most recent surveillance colonoscopy, performed two years ago, showed no significant findings. He denied a history of colorectal surgeries, radiation, or pelvic injury. Family history was positive for a paternal uncle with Crohn's disease but not for colorectal cancer. The patient was a non-smoker, drinking alcohol occasionally.

On physical examination, the patient was well and not in acute distress. There were no abnormal vital signs. Abdominal examination was significant for mild tenderness with palpation over the left lower quadrant, without rebound, guarding, or masses. Digital rectal examination disclosed a firm, smooth mass 6 cm from the anal verge at the anterior aspect of the rectum, which was non-fixed and movable. There was no fecal blood on the examining finger.

Initial laboratory work revealed mild anemia, hemoglobin: 11.2 g/dL and hematocrit: 34%, with normal WBC of $9.7 \times 10^3/\mu\text{L}$ and platelets at $234 \times 10^3/\mu\text{L}$. White blood cell count was normal at 7,800/ μL , and the inflammatory markers were mildly elevated (C-reactive protein: 12 mg/L and ESR: 28 mm/hour). Carcinoembryonic antigen (CEA) was normal at 2.1 ng/ml.

2.3. Radiographic studies

Colonoscopy showed a 4 cm sessile, ulcerated rectosigmoid junction lesion with surrounding erythema and friability. The overlying mucosa was inflamed, and the lesion felt firm on palpation with biopsy forceps. The remaining colon showed patchy erythema, consistent with quiescent inflammatory bowel disease.

Abdomino-pelvic contrast-enhanced CT showed focal asymmetric wall thickening of the rectum, measuring 3.8 cm in maximum diameter, with maintained fat planes between the rectum and adjacent structures. No lymphadenopathy or signs of distant metastasis were detected. There was mild stranding of the perirectal fat with no abscess formation. MRI of the pelvis demonstrated improved soft-tissue characterization, revealing a T2-hyperintense submucosal lesion with multiple small cystic spaces, giving it a conventional "Swiss cheese" appearance. The lesion showed intermediate signal on T1-weighted images and heterogeneous enhancement with gadolinium. Furthermore, the muscularis propria appeared intact, and no deep invasion was observed.

The radiologic differential diagnosis comprised: adenocarcinoma (most likely, given ulceration and patient's IBD history), colitis cystica profunda (cystic, submucosal changes), inflammatory or pseudo polyp, rectal duplication cyst, and lymphoma.

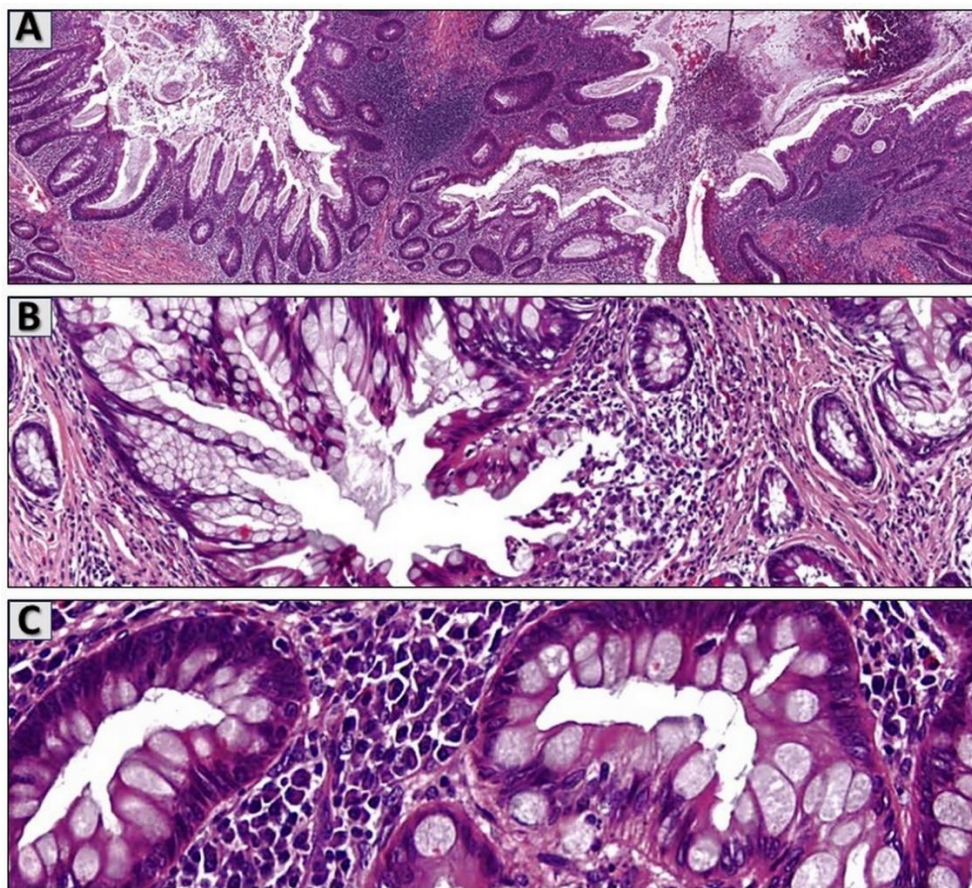
2.4. Tissue diagnosis

Initial endoscopic biopsies were performed, but superficial samples showed only ulcerated mucosa with acute and chronic inflammation, requiring deeper tissue sampling. Subsequently, endoscopic ultrasound-guided core biopsy was performed to obtain submucosal tissue for diagnostic purposes. The sampled tissue displayed a firm, nodular cut surface with multiple mucin-filled cystic spaces ranging from 2 to 15 mm in diameter within the submucosa and muscularis propria. The overlying mucosa showed ulceration and erythema.

Histopathological examination showed heterotopic benign colonic glands in the submucosa and muscularis propria, surrounded by marked smooth muscle proliferation and fibrosis. No cytological atypia was observed, and the displaced glands were lined with normal colonic epithelium containing goblet cells. Many of the glands were dilated and packed with mucin. There was chronic colitis in the overlying mucosa, with distorted crypt architecture, a chronic inflammatory infiltrate, and ulceration. There was no evidence of dysplasia or malignancy. (Figure 1A, B, C)

On IHC, the lesional glands were positive for CK20 and CDX2, compatible with colonic epithelium. The Ki-67 proliferation index was low, and the p53 reaction pattern was wild-type, excluding dysplasia. The histomorphology, together with the ancillary studies' findings, was consistent with the diagnosis of CCP.

2.5. Treatment, outcome, and follow-up



1A Low power view showing heterotopic benign colonic glands in the submucosa and muscularis propria. Many glands are dilated and filled with mucin (H&E, X20); 1B Intermediate power view showing the colonic glands surrounded by marked smooth muscle proliferation, a chronic inflammatory infiltrate, and fibrosis. (H&E X40); 1C High power view showing no evidence of dysplasia or malignancy (H&E X60)

Figure 1 Microscopic examination of the excised colitis cystica profunda

In view of the patient's continued symptomatology, the size of the lesion, the initial diagnostic dilemma at presentation, the possibility of malignancy, and the likelihood of complications such as obstruction or perforation, surgical

intervention was advised. In a multidisciplinary tumor board discussion, a laparoscopic low anterior resection with primary anastomosis was recommended. The operation was uncomplicated with low blood loss.

Postoperative recovery was uneventful. The patient started oral feeding on the 2nd postoperative day and was discharged on the 4th postoperative day. The final pathology confirmed the preoperative diagnosis of CCP with negative margins and no evidence of dysplasia or cancer.

At 6 weeks of follow-up, the patient had no rectal bleeding or mucoid discharge. His hemoglobin had improved to 14.1 g/dL. Colonoscopy at six months revealed a well-healed anastomosis with no evidence of recurrence. Because of his underlying IBD and history of CCP, the multidisciplinary team advised surveillance colonoscopy at yearly intervals for the first 2 years and then every 2 years. The patient was informed of the benign nature of CCP, but that they were to have ongoing follow-up due to its association with inflammatory bowel disease and rare reports of malignant change. At 32 months of follow-up, patients' symptoms had resolved, and they had regained their daily routine activities.

3. Discussion

3.1. Background (History, epidemiology, risk factors, and WHO classification)

Submucosal cysts present in the colon were first described by Stark in 1766 during an autopsy of two patients with chronic dysentery. [8] It was later named "colitis cystica polyposa" by Virchow in 1863 due to its multiple polypoid cystic submucosal lesions. In 1957, the condition was renamed using the term "colitis cystica profunda" by Goodall and Sinclair, along with a detailed description of the disease. [9]

Because CCP is quite rare and often presents with non-specific symptoms, epidemiological data may not be readily available, as most cases are reported as individual case reports rather than in large epidemiological cohorts. Although this condition may occur at any age, several cases have been documented in children. [10] Additionally, it is often associated with other diseases of the colon that irritate the intestinal wall, such as ulcerative colitis, Crohn's disease, Peutz-Jeghers syndrome, adenomatous polyps, dysentery/infectious colitis, spastic colitis, rectal trauma/prolapse, and spinal cord injury. However, the exact etiology remains unclear. [11] [13] [14] It occurs primarily in the rectum and sigmoid colon, usually in the anterior rectal wall. [4] [6]

CCP currently does not have an official WHO classification as it is not a neoplasm, but rather a benign, non-neoplastic entity characterized by misplaced glands and mucous-filled cysts in the submucosa. CCP can be difficult to distinguish from well-differentiated mucinous adenocarcinoma. To distinguish the CCP from other malignant dysplasias, extensive cytological and architectural assessment, along with ancillary IHC studies, is necessary. CCP should present with a bland, benign epithelium that lacks any cytological atypia. [4]

3.2. Pathogenesis, pathophysiology

Although its precise pathogenesis remains incompletely understood, CCP is widely regarded as an acquired condition arising from chronic mucosal injury and defective tissue repair mechanisms. [2] [3] Persistent inflammation, ulceration, and mechanical trauma are believed to disrupt the integrity of the muscularis mucosae, allowing surface epithelial glands to herniate into deeper layers of the bowel wall. [16] In patients with inflammatory bowel disease, particularly ulcerative colitis, repeated cycles of mucosal damage and regeneration can distort architecture and weaken structural barriers. [15] [17] Increased intraluminal pressure associated with tenesmus, chronic straining, and altered bowel motility may further facilitate glandular displacement. [3] [17]

Once embedded within the submucosa and muscularis propria, the displaced glands retain secretory activity, resulting in progressive mucin accumulation and cystic dilatation. [16] [18] This process is accompanied by a reactive stromal response characterized by smooth muscle proliferation, fibrosis, and chronic inflammatory infiltrates, which contribute to mass formation. [15] These histopathologic features explain the frequent radiologic and endoscopic resemblance of CCP to invasive malignancy. [2] [18]

From a pathophysiological perspective, the expansion of mucin-filled cysts and surrounding fibrosis leads to localized bowel wall thickening, reduced compliance, and mucosal distortion. These changes manifest clinically as rectal bleeding, mucus discharge, tenesmus, and a sensation of incomplete evacuation. [3] [15] Ulceration of the overlying mucosa further exacerbates inflammatory activity and bleeding risk. Superficial biopsies may demonstrate atypical regenerative changes, increasing the risk of misdiagnosis, such as dysplasia or carcinoma. [19] [20]

In the present case, long-standing ulcerative colitis played a significant role in the development of CCP by promoting chronic epithelial injury, impaired mucosal healing, and glandular misplacement. The absence of cytologic atypia, low Ki-67 proliferative index, and wild-type p53 expression supports the non-neoplastic nature of the lesion. It reinforces the concept of CCP as a reactive rather than premalignant process.

3.3. Comparative analysis of our case with the existing literature. (Clinical-radiology-pathology, lab, diagnosis, management, and outcome)

The 36-year-old male patient with long-standing UC aligns with the demographic most reported in CCP, particularly young individuals aged 20 to 40. [1] The case contests existing frameworks. While previous literature synonymous with CCP highlights rectal prolapse and surgical trauma, recent studies offer a considerable subset of cases linked to chronic inflammatory bowel disease (IBD). [21] [22] He had no prolapse or pelvic surgery history, placing him in the more recognized IBD-related subgroup. Rectal bleeding, mucoid discharge, and tenesmus characterize CCP and perfectly mirror the symptoms. [1] [5] [8] [22] This reveals the inadequacy of symptomatology alone for diagnosing CCP since presentation is indistinguishable from colorectal malignancy or severe proctitis.

The diagnostic workup for our case exemplifies the standard of care advocated in recent literature. The first endoscopic suspicion of malignancy, based on an ulcerated rectosigmoid mass, is almost universal. Studies show that on average, there is a 60-70% rate of preoperative misdiagnosis. [1] [5] Superficial biopsies are inaccurate and reinforce the literature's consistent warning that diagnostic tissue remains in the submucosa. [5] The pivotal role of advanced imaging is perfectly illustrated. The abdominopelvic CT, while ruling out metastasis, was non-specific. It was the pelvic MRI that provided the critical differentiating feature: the T2-hyperintense submucosal lesion with a "Swiss cheese" appearance. This sign is highly suggestive of the CCP and was instrumental in excluding adenocarcinoma from the differential diagnosis. [7] However, as Denis et al. reported, the sign is not 100% specific, particularly in IBD, where inflammation can alter tissue planes. [7] As such, the decision to proceed with EUS-guided core biopsy represents the application of the most current diagnostic algorithm, which entails using imaging to target the pathology and secure a definitive histological diagnosis while minimizing invasiveness. [5]

The histopathological findings of heterotopic benign glands, smooth muscle hyperplasia, and the absence of dysplasia are the definitive features of CCP and align with the reported histopathological descriptions. Rosano et al. cited the use of IHC (low Ki-67, CK20+, CDX2+, wild-type p53) to exclude malignancy as a best practice. This helps reduce the risk of misidentifying and misinterpreting displaced glands as invasive carcinoma. [6] The management dilemma in the current literature is whether to opt for laparoscopic low anterior resection or surveillance. There is a growing consensus in the literature for conservative monitoring of small, asymptomatic, and definitively diagnosed lesions. [23] In contrast, for cases defined by a large lesion (4 cm) and initial diagnostic uncertainty, surgical resection is recommended. [1] [8] [23] This case exemplifies the use of a significant risk/benefit equation. The potential morbidity of a resection for a benign condition is less than the potential consequences of missing a malignancy. The positive outcome, with no recurrence at 32 months, highlights the significance of surgery. [8] [21]

After complete removal, the prognosis was excellent. The patient is likely to have a very low likelihood of recurrence. [8] At 32 months post-procedure, symptom-free with no endoscopic recurrence, this best-case scenario fits. There is a nearly complete absence of documented recurrence (<5%), which is almost always attributable to incomplete excision or ongoing inciting factors. [8] The absence of early recurrence signifies a successful outcome. The surveillance strategy, including annual colonoscopy followed by biennial colonoscopy, focuses on the separate, long-term risk of UC-associated neoplasia. This addresses the dual focus recommended in recent literature on IBD-associated CCP regarding surveillance. [21] [22] IBD is a principal risk factor for CCP. [22] Jiang et al. and Chen et al. mapped the spectrum from surgical to endoscopic therapies as a function of diagnostic confidence. [5] [23] There is evidence of the effectiveness of surgery. [8]

Our case integrates all these components: an IBD-associated CCP in which diagnostic pinpointing was achieved through biopsy, yet clinical elements justified surgical intervention, resulting in a positive outcome. The parallels with literature, presentation, diagnostic hurdles, and histology affirm CCP's clinical footprint. The lesson is that, for effective CCP management, dealing with diagnostic uncertainty is paramount. It also emphasizes surgery as an ideal intervention that provides a definitive diagnosis and cure.

3.4. What have we learned from this case?

This case provides several critical diagnostic and management lessons for clinicians encountering atypical colorectal masses. First, it underscores the importance of maintaining a high index of suspicion for CCP when a patient with chronic IBD presents with a polypoid or ulcerated lesion, even when malignancy is the primary concern. A key "red flag" for clinicians is the persistence of symptoms, such as mucoid discharge and tenesmus, despite adequate control of the

underlying ulcerative colitis. From a diagnostic perspective, this case highlights the limitations of superficial biopsies and the indispensable role of advanced imaging and deep tissue sampling. The "Swiss cheese" appearance on T2-weighted MRI should be recognized as a highly suggestive radiographic marker for CCP, warranting further investigation with endoscopic ultrasound (EUS)-guided core biopsies to reach the submucosal layer.

Regarding management, this case reinforces that while CCP is benign, surgical intervention may be necessary when the lesion is large, symptomatic, or when malignancy cannot be definitively excluded. The successful laparoscopic low anterior resection in this patient demonstrates that surgical management can provide both a definitive diagnosis and symptomatic relief with favorable long-term outcomes. Furthermore, the case suggests that for patients with underlying IBD, the presence of CCP may necessitate a more vigilant surveillance strategy, not only to monitor for recurrence but also to manage the elevated baseline risk of dysplasia associated with chronic inflammation. Ultimately, this case challenges the notion that all suspicious masses in IBD patients are malignant, while reinforcing the need for a multidisciplinary approach to navigate the diagnostic uncertainty.

Abbreviations

- Colitis cystica profunda (CCP);
- Immunohistochemical (IHC);
- Inflammatory bowel disease (IBD).

4. Conclusion

We report a rare case of colitis cystica profunda presenting as a suspicious rectosigmoid mass in a 36-year-old male with a long-standing history of ulcerative colitis. This report emphasizes the diagnostic complexity of CCP, particularly its ability to mimic adenocarcinoma in patients with chronic inflammatory bowel disease. The novelty of this case lies in the specific association with ulcerative colitis and the successful use of EUS-guided core biopsy and MRI to achieve a preoperative diagnosis, thereby guiding appropriate surgical management. The primary clinical takeaway is that clinicians must consider CCP in the differential diagnosis of rectal masses in patients with IBD; a high index of suspicion combined with deep tissue sampling is essential to avoid diagnostic pitfalls and ensure optimal patient care.

Compliance with ethical standards

Acknowledgments

Special thanks to Jovia Williams, Richard Chen Rubin, and Daniel Diaz-Alejo for their assistance in reviewing the final manuscript. Additionally, we appreciate the assistance of Grammarly's language editor and the Claude Sonnet 4.5 program, which provided valuable writing support by identifying and correcting errors in grammar, spelling, punctuation, and writing style, ultimately enhancing the manuscript.

Disclosure of conflict of interest

All authors make the following declarations:

- Payment/services information: All authors have declared that they received no financial support from any organization for the submitted work.
- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

Statement of informed consent

This study was conducted as a retrospective review of archival pathology material collected during routine clinical care. All data were fully de-identified prior to analysis. No patient contact or intervention was involved, and informed consent was not required in accordance with institutional policies.

References

- [1] Ward M, Burns K, Nora I, Wiese-Rometsch W, Loewe CJ. S1869 Colitis Cystica Profunda: A Great Pretender. Official journal of the American College of Gastroenterology| ACG. 2021 Oct 1;116:S822.
- [2] Morson BC, Dawson IM. Gastrointestinal pathology. 3rd ed. Oxford: Blackwell Scientific; 1990),
- [3] Dehghani SM, Haghighat M, Imanieh MH, Geramizadeh B. Colitis cystica profunda: report of two cases and review of literature. World J Gastroenterol. 2008;14(34):5387–5390.)
- [4] Hernandez-Prera JC, Polydorides AD. Colitis cystica profunda indefinite for dysplasia in Crohn disease: A potential diagnostic pitfall. Pathol - Res Pract [Internet]. 2014 Dec [cited 2026 Jan 25];210(12):1075–8. Available from: <https://linkinghub.elsevier.com/retrieve/pii/S0344033814002684>
- [5] Alshatti Y, Alenezi O, Alenezi M, Jeragh M, Alkanderi W. Benign Yet Deceptive: A 19-Year-Old Male with A Rectal Mass Mimicking Carcinoma, Ultimately Diagnosed as Colitis Cystica Profunda. European Journal of Case Reports in Internal Medicine. 2025 Nov 24;12(12):005967.
- [6] Rosano GN, Aiello E, Pisheh SK, Deiana E, Memeo L, Colarossi C. Colitis cystica profunda associated with diverticulosis and calcification mimicking colorectal carcinoma: a case report and a brief literature review. Pathologica [Internet]. 2024 Sept [cited 2026 Jan 25];116(4):249–53. Available from: <https://www.pathologica.it/article/view/702>
- [7] Denis R, Hobbs M, Felix M, Lujan HJ, Lujan H. Colitis Cystica Profunda of the Hepatic Flexure: A Case Report. Cureus. 2024 Apr 15;16(4).
- [8] Ayantunde AA, Strauss C, Sivakkolunthu M, Malhotra A. Colitis cystica profunda of the rectum: An unexpected operative finding. World J Clin Cases [Internet]. 2016 [cited 2026 Jan 25];4(7):177. Available from: <http://www.wjgnet.com/2307-8960/full/v4/i7/177.htm>
- [9] Green GI, Ramos R, Bannayan GA, McFee AS. Colitis cystica profunda. Am J Surg [Internet]. 1974 June [cited 2026 Jan 25];127(6):749–52. Available from: <https://linkinghub.elsevier.com/retrieve/pii/0002961074903638>
- [10] Krummel TM, Bell S, Kodroff MB, Berman WF, Salzberg AM. Colitis cystica profunda: A pediatric case report. J Pediatr Surg [Internet]. 1983 June [cited 2026 Jan 25];18(3):314–5. Available from: <https://linkinghub.elsevier.com/retrieve/pii/S002234688380116X>
- [11] Epstein SE, Ascari WQ, Ablow RC, Seaman WB, Lattes R. Colitis Cystica Profunda. Am J Clin Pathol [Internet]. 1966 Feb 1 [cited 2026 Jan 25];45(2):186–201. Available from: <https://academic.oup.com/ajcp/article-lookup/doi/10.1093/ajcp/45.2.186>
- [12] Peterkin GA, Moroz K, Kondi ES. Proctitis cystica profunda in people with paraplegia: Report of three cases. This Colon Rectum [Internet]. 1992 Dec [cited 2026 Jan 25];35(12):1174–6. Available from: <https://journals.lww.com/00003453-199235120-00011>
- [13] Wang F, Frisbie JH, Klein MA. Solitary rectal ulcer syndrome (colitis cystica profunda) in spinal cord injury patients: 3 case reports. Arch Phys Med Rehabil [Internet]. 2001 Feb [cited 2026 Jan 25];82(2):260–1. Available from: <https://linkinghub.elsevier.com/retrieve/pii/S0003999301475988>
- [14] Sivokonev KP, Gens GP, Stukalova OYu, Maslov VV, Gaydamaka NV, Korobkova LI. Colitis cystica profunda: literature review and case report. Colorectal Oncol [Internet]. 2019 June 16 [cited 2026 Jan 25];9(2):62–9. Available from: <https://ok.abvpress.ru/jour/article/view/292>
- [15] Shepherd NA, Warren BF, Williams GT, Greenson JK, Lauwers GY, Novelli MR. Morson and Dawson's gastrointestinal pathology. 5th ed. Oxford: Wiley-Blackwell; 2013

- [16] Khor TS, Brown IS, Kumarasinghe MP. Colitis cystica profunda: clinicopathological features and diagnostic pitfalls. *Pathology*. 2011;43(6):530–535
- [17] Odze RD, Goldblum JR. *Surgical pathology of the GI tract, liver, biliary tract, and pancreas*. 3rd ed. Philadelphia: Elsevier; 2015
- [18] Kim JH, Kim YS, Lee JH, et al. Colitis cystica profunda presenting as rectal tumor: a case report. *J Korean Med Sci*. 2007;22(4):761–764
- [19] Yantiss RK, Antonioli DA. Colitis cystica profunda revisited. *Am J Surg Pathol*. 2001;25(5):581–588
- [20] Feakins RM. Inflammatory bowel disease biopsies: updated British Society of Gastroenterology reporting guidelines. *J Clin Pathol*. 2013;66(12):1005–1026
- [21] Suter KJ, Skinner SA. Rare presentation of colitis cystica profunda in a single rectal ulcer. *ANZ Journal of Surgery*. 2023 Apr 1;93(4).
- [22] Salem AE, Morad Z, Mujtaba BS, Pranay S, Sarah M, Barberan PC, Matthew KS, Hazem A, Mohammed A, Chaudhry MA, Khaled E. S3170 Incidental Finding of Colitis Cystica Profunda in Routine Colorectal Screening. *The American Journal of Gastroenterology*. 2024 Oct 1;119(10S):S2144.
- [23] Chen G, Jiang W, Yue M. Colitis cystica profunda of the rectum diagnosed by endoscopic submucosal dissection. *Rev Esp Enferm Dig*. 2023 Feb 1;115(2):91-2.