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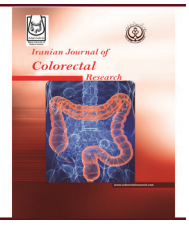

Colitis Cystica Profunda: A Benign Deceiver in the Era of Advanced Imaging

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Abstract

Colitis cystica profunda (CCP) is a rare, benign colorectal disorder characterized by mucin-filled cysts located within the submucosa. Despite its non-neoplastic nature, CCP frequently mimics mucinous adenocarcinoma clinically, endoscopically, radiologically, and histologically, posing a significant diagnostic challenge that can lead to unnecessary radical surgery. This review synthesizes evidence from searches of PubMed, Embase, and Google Scholar up to December 2025 to provide a contemporary overview of the etiopathogenesis, diagnostic challenges, differential diagnosis, and management of CCP. Accurate diagnosis relies on demonstrating the submucosal location of mucin-filled cysts and excluding invasive malignancy through a multimodal assessment. Endoscopic ultrasonography and magnetic resonance imaging assist in defining lesion depth and maintaining diagnostic confidence, while histopathological identification of a specialized lamina propria rim surrounding displaced glands helps distinguish CCP from invasive adenocarcinoma. Current management emphasizes correcting underlying mechanical factors, such as rectal prolapse and chronic straining, rather than routine radical resection. Recognizing the characteristic features of CCP is essential to avoid misdiagnosis and to support evidence-based, organ-preserving management.

Keywords: Colorectal Neoplasms; Colitis; Diagnosis, Differential; Adenocarcinoma, Mucinous; Diagnosis, Differential

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Introduction

Colitis cystica profunda (CCP) is a rare, benign disorder of the large intestine characterized by mucin-filled cystic spaces located beneath the muscularis mucosae (1, 2). Although it most commonly affects the rectum and sigmoid colon, its distribution can be localized, segmental, or diffuse throughout the colon (2, 3). Despite its benign nature, CCP's remarkable capacity to mimic more serious conditions, most notably mucinous adenocarcinoma, makes it a difficult diagnostic challenge for radiologists, pathologists, and clinicians alike (2, 4).

The historical recognition of this CCP dates back to 1766, when Stark first documented submucosal mucus-containing cysts during autopsies of two patients who had succumbed to chronic dysentery (5). In 1863, Virchow designated these lesions as "colitis cystica polyposa" to describe their often-pedunculated appearance (5). It was not until 1957 that Goodall and Sinclair introduced the modern term "colitis cystica profunda," providing the first

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comprehensive clinical and pathological description of the disease (5). Similar lesions are also observed in the stomach and small intestine, where they are known as gastritis cystica profunda and enteritis cystica profunda, respectively (6-9).

Although the fundamental pathological description of CCP has remained largely unchanged since the mid-20th century, the clinical landscape has evolved significantly. The "malignancy trap" — the misdiagnosis of mucinous adenocarcinoma —continues to pose a significant risk to patient safety despite the increased use of cross-sectional imaging and improved endoscopy, which often incidentally detect these lesions. Moreover, clinical confusion frequently occurs between CCP and its anatomical counterpart, colitis cystica superficialis (CCS), despite their distinct etiologies and treatment strategies. To promote organ-preserving, evidence-based care, this article provides a comprehensive, up-to-date (2026) review of CCP, with particular emphasis on multimodal diagnostic techniques such as endoscopic ultrasound (EUS) and radiomics, which are essential for accurately distinguishing CCP from malignant mimics and superficial variants.

Materials and Methods

Search Strategy

A comprehensive search of electronic databases, including PubMed/MEDLINE, Embase, the Cochrane Library, and Google Scholar, was conducted to identify relevant literature published from inception through December 2025. The search utilized a combination of Medical Subject Headings (MeSH) terms and free-text keywords: "Colitis cystica profunda," "Colitis cystica superficialis," "submucosal cysts," "localized colitis cystica profunda," "diffuse colitis cystica profunda," "rectal cystic lesions," and "pseudoinvasion." Boolean operators (AND, OR) were used to refine the search (e.g., "Colitis cystica profunda" AND "differential diagnosis" OR "mucinous adenocarcinoma").

Study Selection and Inclusion Criteria

The selection process was designed to capture high-quality evidence while maintaining the breadth required for a comprehensive narrative review. Studies were eligible for inclusion if they met the following criteria:

- **Study Design:** Systematic reviews, meta-analyses, original prospective and retrospective cohort studies, and key case series.
- **Focus:** Pathophysiological mechanisms, diagnostic modalities—specifically EUS, Magnetic Resonance Imaging (MRI), and Radiomics—and clinical management outcomes.
- **Language:** Articles published in English or those with official English translations available.
- **Evidence Horizon:** Special emphasis was placed on literature published between 2015 and 2025 to capture the most recent advancements in endoscopic techniques and artificial intelligence applications in gastrointestinal pathology.

Data Extraction and Synthesis

Data were extracted and qualitatively synthesized to identify key themes.

1. **Etiopathogenesis:** Mechanisms of glandular displacement and the role of the muscularis mucosae.
2. **Anatomical Differentiation:** Clinical and pathological markers distinguishing CCP (submucosal) lesions from CCS (mucosal) lesions.
3. **Diagnostic "Red Flags":** Clinical, radiological, and pathological features used to identify the "Malignancy Trap" and to exclude mucinous adenocarcinoma.
4. **Management Trends:** The evolution from radical surgical resection to conservative, organ-sparing endoscopic interventions.

Exclusion Criteria

Technical notes without clinical data, brief conference abstracts lacking peer-reviewed full texts, and duplicate publications were excluded. Small-scale case reports were included only if they presented unique diagnostic challenges, rare anatomical locations, or novel therapeutic approaches.

Pathogenesis

Although the exact cause of CCP remains unknown, it is believed to result from either acquired or congenital weakness of the mucosal muscles. Chronic mucosal damage and subsequent tissue remodeling are the primary drivers of its multi-stage development(5). This progression follows a predictable sequence, as illustrated in Figure 1.

Stage A: Chronic Inflammation and Mucosal Injury

The process begins with a primary insult to the colonic wall. While mechanical trauma and chronic low-grade inflammation from solitary rectal ulcer syndrome (SRUS) and rectal prolapse are the most common triggers facilitating the breakdown of the mucosal-submucosal barrier, extensive mucosal ulceration and architectural remodeling caused by inflammatory bowel disease (IBD), ischemia, diverticular disease, pelvic radiation, or severe infectious dysentery can also initiate this process (1, 10-15).

Surgical interventions and local trauma, including appendiceal or colonic procedures, may further contribute by altering bowel motility and local pressure dynamics. This supports the concept that CCP represents a reactive process rather than a true neoplasm (16).

Stage B: Glandular Herniation and Displacement

Following the initial injury, repetitive mechanical pulling (as seen intussusception) or the repair process itself leads to the formation of defects in the

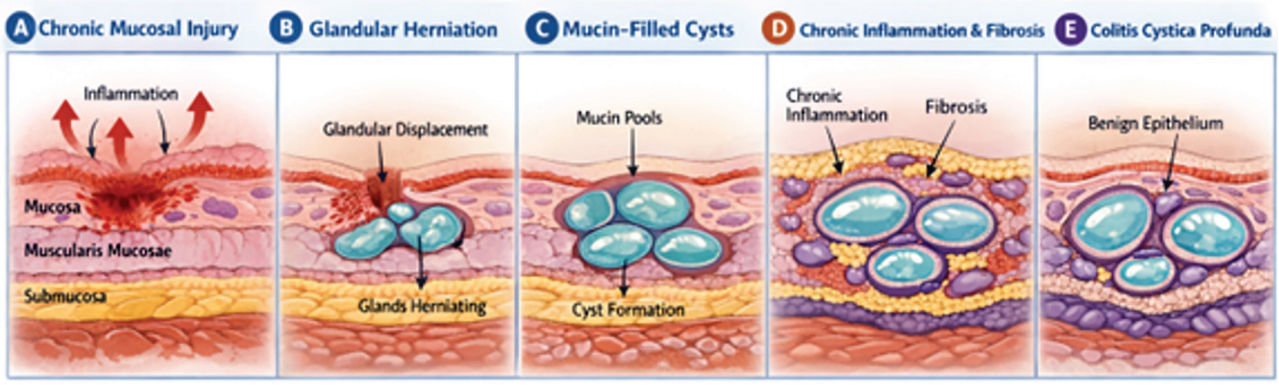

Figure 1: The Multi-Stage Pathogenesis of Colitis Cystica Profunda. (A) Initial mucosal injury and inflammation; (B) Displacement of regenerating glands through defects in the muscularis mucosae; (C) Formation of pressurized mucin pools; (D) Reactive stromal fibrosis and chronic inflammation; (E) Mature cystic lesions lined by benign epithelium.

muscularis mucosae. Regenerating epithelial glands "herniate" through these gaps into the submucosal compartment (1, 17). This "misplacement" of epithelium is the critical point of divergence toward the development of CCP.

Stage C: Formation of Mucin-Filled Cysts

Once trapped within the submucosa, the epithelial cells continue their physiological function of mucus secretion. Because these glands lack a natural exit to the intestinal lumen, the mucus accumulates under pressure. These "mucin pools" distend the glands into the characteristic macroscopic cysts that define the condition (18).

Recent case studies have reaffirmed that this accumulation causes the characteristic "lifting" of the mucosa seen endoscopically, which can be misidentified as a neoplastic process if the cystic nature is not recognized (4).

Stage D: Chronic Inflammation and Fibrosis

The displaced glands and stagnant mucin act as chronic irritants to the submucosal stroma. This triggers a reactive response characterized by chronic inflammation and progressive fibrosis. According to a large-scale analysis by Abid et al., this process often manifests as fibromuscular hyperplasia, which contributes to the mass-like appearance on imaging but remains distinct from the disordered desmoplasia seen in invasive malignancy (10).

Stage E: Mature CCP

In the final stage, the submucosa contains well-differentiated, mucin-filled cysts. Despite their deep (profunda) location, the lining consists of a single layer of benign columnar epithelium (19). A crucial "safety check" at this stage is the presence of a delicate rim of lamina propria surrounding the displaced glands, confirming their benign and displaced nature.

Clinical Presentation

The clinical manifestations of CCP are highly variable (Table 1), ranging from completely asymptomatic incidental findings detected during screening colonoscopy to debilitating bowel dysfunction (17). Because CCP is a structural consequence of chronic mucosal injury rather than a primary inflammatory disease, its symptoms often reflect those of the underlying conditions, such as SRUS, IBD or rectal prolapse.

The most common symptom is painless hematochezia (20). This is often accompanied by the passage of copious amounts of mucus (mucorrhea) or non-bloody diarrhea, which results from the rupture of superficial cysts or hypersecretion by displaced glands (19).

Cases with localized rectal involvement often present with tenesmus or a sensation of incomplete evacuation, which can lead to the "malignancy trap" when a firm, fixed mass is detected during digital rectal examination (5). In rare instances, chronic abdominal discomfort or bowel obstruction may also manifest as symptoms of CCP (21).

Diagnostic Evaluation

The diagnosis of CCP is notoriously challenging because superficial mucosal biopsies often yield results described as "normal" or showing "nonspecific inflammation," since the pathology is located beneath the muscularis mucosae. Therefore, a multimodal approach is essential to navigate the diagnostic pitfalls outlined previously (Table 2).

Endoscopy and the "Cushion Sign"

Endoscopically, CCP typically appears as a single sessile polyp, a cluster of nodules, or a submucosal bulge with intact overlying mucosa. However, it may uncommonly present as a fungating polypoid mass with surface ulceration (Figure 2).

- **The Cushion Sign:** Also known as the "Pillow Sign," this is elicited by pressing the lesion with closed biopsy forceps. A positive sign, where the lesion indents and slowly recovers (Figure 3), suggests a fluid-filled or cystic consistency rather than a solid malignancy (22).

Table 1: Clinical and Diagnostic Profile of Colitis Cystica Profunda

Feature	Clinical Presentation / Findings	Clinical Significance
Dominant Symptom	Painless hematochezia (bright red blood)	Often chronic and intermittent; may lead to iron-deficiency anemia.
Secretory Signs	Significant mucorrhea (mucus discharge)	Result of hypersecretion by displaced submucosal goblet cells.
Bowel Habits	Tenesmus, constipation, and straining	Frequently mimics or co-exists with Solitary Rectal Ulcer Syndrome (SRUS).
Digital Rectal Exam (DRE)	Firm, rubbery, or "fixed" submucosal mass	The Malignancy Trap: Often palpated as a suspicious rectal tumor during physical exam.
Anatomical Variant	Localized (Rectal) vs. Diffuse (Colonic)	Rectal forms correlate with prolapse; diffuse forms mimic inflammatory bowel disease.
Associated Conditions	Rectal prolapse or internal procidentia	Mechanical trauma is the primary driver in up to 80% of localized cases.
Rare Presentation	Asymptomatic "Incidentaloma"	Increasing frequency of detection due to screening colonoscopies for other indications.

Table 2: Multimodal Diagnostic Features of Colitis Cystica Profunda

Modality	Key Radiographic/Endoscopic Findings	Clinical "Safety Check" (Rules out Cancer)
Colonoscopy	Sessile nodules or umbilicated bulges; overlying mucosa typically intact.	**Cushion/Pillow Sign:** Lesion is compressible, suggesting fluid rather than a solid tumor.
EUS	Anechoic/hypoechoic cysts restricted to the submucosa (Layer 3).	**Layer Preservation:** Muscularis propria (Layer 4) remains intact and uninvolved.
CT Scan	Focal wall thickening; clustered cystic areas with low attenuation.	**Negative Findings:** Preservation of perirectal fat planes and absence of lymphadenopathy.
MRI (T2)	Hyperintense mucin signal.	**Diffusion Check:** Lack of restricted diffusion on DWI (unlike hypercellular malignancies).
Biopsy	Submucosal mucin pools containing benign columnar epithelium.	**Stromal Check:** A delicate rim of lamina propria or muscularis mucosae surrounds glands.

EUS: Endoscopic ultrasound; CT scan: Computed tomography scan; MRI: Magnetic resonance imaging; DWI: Diffusion-Weighted Imaging

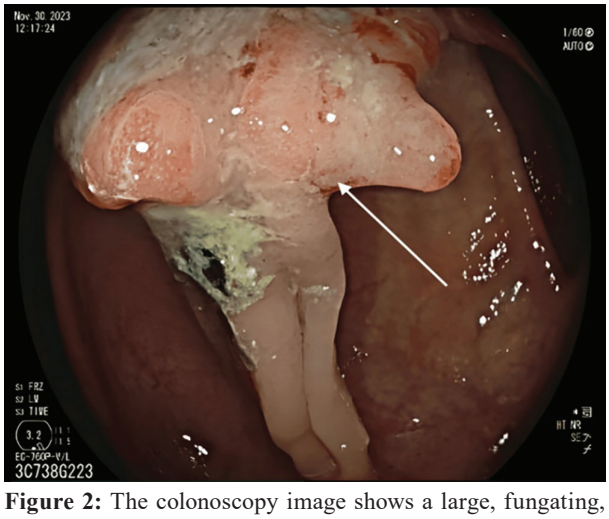

Figure 2: The colonoscopy image shows a large, fungating, ulcerated polyp at the hepatic flexure. Image source: Denis et al. (Reference 29). Reused under the terms of the Creative Commons Attribution License: http://creativecommons.org/licenses/by-nc-nd/4.0/

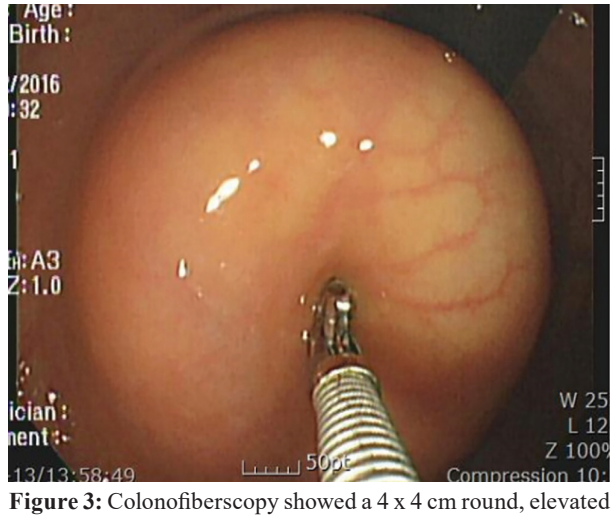

Figure 3: Colonofiberscopy showed a 4 x 4 cm round, elevated lesion covered with normal mucosa, located 5 cm from the anal verge. The cushion sign was positive. Image source: Lee SH, Lee SH (Reference 22); reused under the Creative Commons Attribution License.

• **Biopsy Strategy:** Standard "pinch" biopsies are frequently inadequate (5, 23). To reach the profunda layer, "jumbo" forceps or a "biopsy-on-biopsy" technique, sampling the same spot twice to penetrate the muscularis mucosae, is often required.

Endoscopic Ultrasound (EUS)

EUS is the most definitive tool for identifying the intramural location of CCP (Figure 4) and depicts the following:

• **Layer Localization:** EUS confirms that the cysts are located in the third layer (submucosa), effectively ruling out CCS, which is mucosal (5, 22) .

• **Morphology:** The cysts appear as well-circumscribed, anechoic (black) or hypoechoic spaces (22).

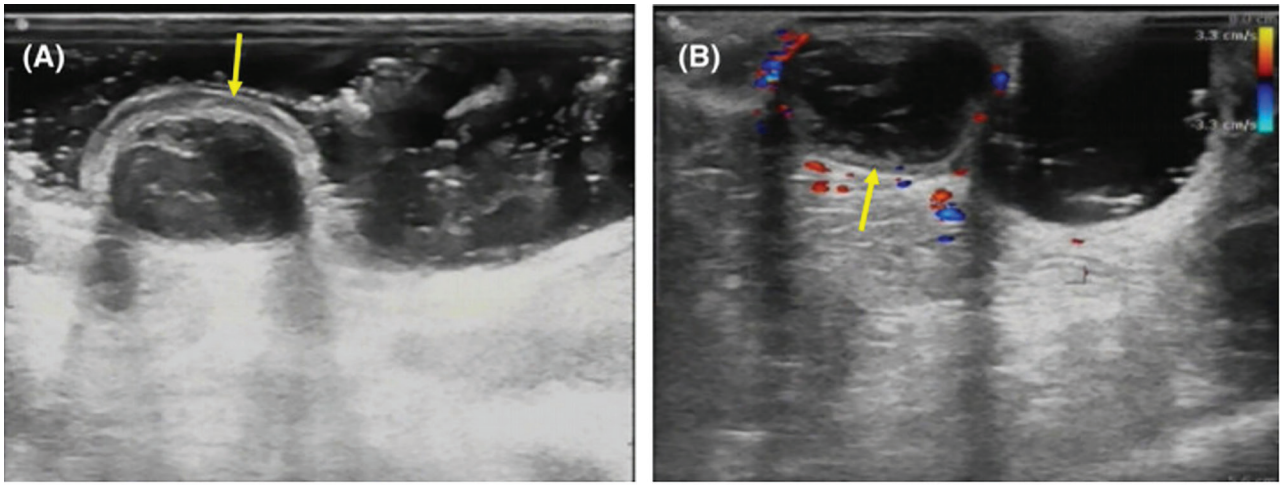

Figure 4: Endoscopic ultrasound. (A) Grayscale ultrasound shows a cystic mass in the submucosa with a clear boundary, poor internal transmission, and continuous echogenicity in the mucosal layer (yellow arrow). (B) Color Doppler ultrasound shows a small amount of blood flow signal around the cystic mass, no obvious blood flow signal inside, and continuous echogenicity in the serous layer of the intestinal wall (yellow arrow). Image source: Jiang et al. (Reference 5)); reused under the terms of the Creative Commons Attribution License http://creativecommons.org/licenses/by-nc-nd/4.0/

• **The "Safety Rim":** High-frequency EUS can sometimes visualize the preserved layers of the bowel wall, whereas malignancies typically cause disruption or infiltration of these layers.

Magnetic Resonance Imaging (MRI)

MRI is particularly useful in cases of diffuse or localized rectal CCP to evaluate the extent of the disease and its relationship to the mesorectal fascia (24-26).

• **T2-Weighted Sequences:** Mucin exhibits very high signal intensity on T2 images. CCP lesions appear as submucosal, homogeneous, variably hyperintense nodules within the thickened bowel wall, without involving the muscle layer, resembling a string of pearls (25, 26).

• **T1-Weighted Sequences:** Typical findings include thickening of the levator ani muscle, asymmetry of the rectal lumen without infiltrative signs, peripheral parietal calcifications, and absence of lymphadenopathy (26).

• **Diffusion-Weighted Imaging (DWI):** Unlike adenocarcinoma, which typically exhibits restricted diffusion on DWI due to high cellularity, the mucin pools in CCP generally do not show significant restriction, providing a key radiological "safety check."

Computed Tomography (CT) Scanning

Computed tomography of CCP typically reveals focal or segmental thickening of the rectal wall, with a predilection for the anterior segment (Figure 5). The most distinctive radiographic hallmark is the identification of well-circumscribed, low-attenuation (0–20 HU) lesions within the submucosa, representing the pathognomonic mucin-filled cystic glands (5, 27). These mucinous collections often manifest as grouped, tubular, or spherical hypodense regions embedded within the hypertrophied wall.

Following the administration of intravenous contrast, the overlying mucosa generally exhibits mild to moderate enhancement but notably lacks the signs of transmural infiltration characteristic of aggressive neoplasms. A critical "safety check" for the radiologist is the maintenance of clear perirectal fat planes and the absence of pathological lymphadenopathy; these negative findings are essential for distinguishing CCP from invasive rectal adenocarcinoma (25-27). Furthermore, CT imaging may reveal secondary morphological changes, such as rectal prolapse, internal procidentia, or features of chronic dyschezia, which provide essential clinical context supporting a benign, reactive etiology (12, 25).

Histopathology

Histopathological examination is a crucial tool for diagnosing CCP. The primary diagnostic challenge is differentiating the benign glandular displacement from the true infiltrative growth characteristic of mucinous adenocarcinoma.

Microscopic Features

The hallmark of CCP is the presence of dilated, mucin-filled cystic structures primarily located within the submucosa, beneath a muscularis mucosa that may be attenuated or disrupted (5). These cysts are typically lined by a single layer of well-differentiated, simple columnar epithelium containing abundant, mature goblet cells (Figure 6). Importantly, these specimens exhibit no glandular atypia, infiltrating morphology, stromal desmoplasia, pleomorphism, or significant mitotic activity (5).

Immunohistochemistry

Immunohistochemistry (IHC) in CCP serves both a confirmatory and exclusionary role, supporting its benign, reactive nature and aiding in its differentiation from malignant and other cystic mimics.

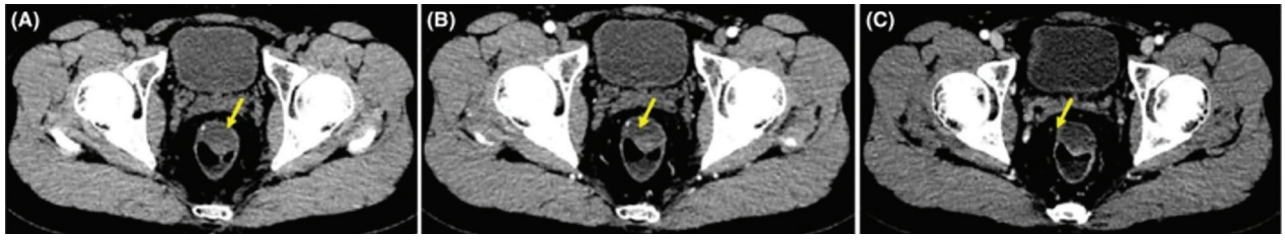

Figure 5: Contrast-enhanced CT. (A) A quasi-circular low-density focus is visible on the plain scan, with smooth rectal mucosa and an intact intestinal wall. (B, C) No enhancement is seen in the arterial and venous phases of the lesion. Image source: Jiang et al. (Reference 5); reused under the terms of the Creative Commons Attribution License http://creativecommons.org/licenses/by-nc-nd/4.0/

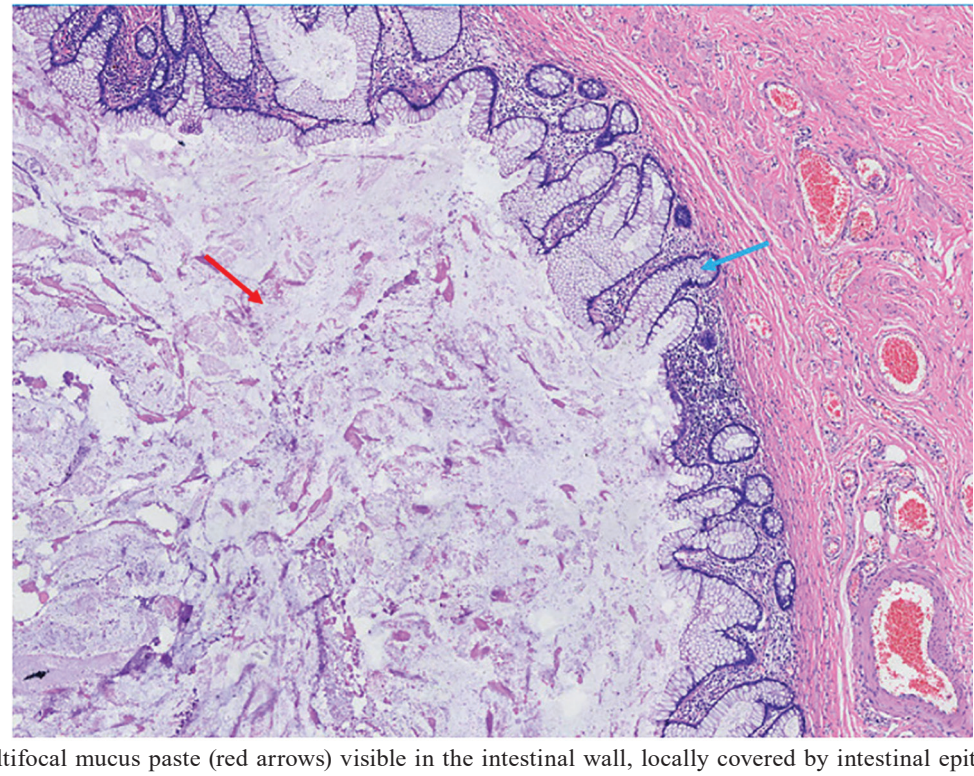

Figure 6: Multifocal mucus paste (red arrows) visible in the intestinal wall, locally covered by intestinal epithelium without heterogeneous hyperplasia (blue arrows). Image source: Jiang et al. (Reference 5); reused under the terms of the Creative Commons Attribution License http://creativecommons.org/licenses/by-nc-nd/4.0/

The entrapped submucosal glands show positivity for epithelial markers, including pan-cytokeratin (AE1/AE3), CK20, and carcinoembryonic antigen (CEA), reflecting their origin from normal colorectal epithelium. In contrast, CK7 is typically negative, consistent with colorectal differentiation (1).

The surrounding stroma often demonstrates fibrosis and smooth muscle proliferation, which can be highlighted by smooth muscle actin (SMA) and desmin. These findings support disruption and repair of the muscularis mucosae as part of the disease process. Proliferative activity, assessed by Ki-67, is low and restricted to the basal portions of the glands, and p53 overexpression is absent. These features help distinguish CCP from adenocarcinoma (1).

Differential Diagnosis

The diagnostic process for CCP is challenging due to its morphological similarities with several serious conditions (Table 3). Clinicians must carefully navigate two primary diagnostic "traps," which vary according to the lesion distribution.

Mucinous Adenocarcinoma: The most important differential diagnosis is mucinous adenocarcinoma. Both conditions present with submucosal mucin collections; however, they differ fundamentally in their stromal characteristics. While CCP features a "safety check" of benign specialized lamina propria and organized fibromuscular hyperplasia, adenocarcinoma exhibits disordered desmoplasia, necrosis, and cytological atypia (1, 4).

Polyposis Coli: In CCP involving the right colon or diffuse segments, diagnostic difficulties are more pronounced. As highlighted by Perumalsamy et al., the presence of numerous sessile elevations may lead to an erroneous suspicion of multiple polyposis syndromes, such as Familial Adenomatous Polyposis (FAP) or Peutz-Jeghers Syndrome (28). Distinguishing these disorders requires recognizing that CCP is a submucosal process, whereas true polyps are primary epithelial proliferations (29).

Colitis Cystica Superficialis: Finally, CCP must be distinguished from CCS. Although their names

Table 3: Differential Diagnosis and Distinguishing Features of Colitis Cystica Profunda

Condition	Primary Mimicry	Key Diagnostic "Separator"
Mucinous Adenocarcinoma	Malignancy Trap	Presence of cytological atypia and lack of a circumscribed lamina propria rim.
Polyposis Syndromes	Polyposis Trap	True polyps are epithelial; CCP cysts are submucosal on EUS.
Colitis Cystica Superficialis	Anatomical Mirror	Cysts are strictly mucosal; often linked to Vitamin B3 (niacin) deficiency.
Pneumatosis Cystoides Intestinalis	Radiological Mimic	Cysts contain gas and show "comet-tail" artifacts on ultrasound rather than fluid.
Endometriosis (Rectal)	Clinical Mimic	Presence of endometrial stroma and cyclic symptoms correlated with menses.
Cystic Lymphangioma	Structural Mimic	Immunohistochemistry positive for D2-40; contains serous fluid rather than thick mucin

EUS: Endoscopic ultrasound; CCP: Colitis cystica profunda

are similar, CCS involves small cysts located strictly *above* the muscularis mucosae and is typically associated with systemic nutritional deficiencies and chemotherapy, rather than the mechanical trauma associated with CCP (30, 31).

Cystic Lymphangioma of the Colon: Beyond epithelial mimics, cystic lymphangioma of the colon represents a significant structural differential diagnosis. As noted by Pham et al., these benign tumors can manifest as intramural, cystic masses that are endoscopically indistinguishable from CCP (32). The definitive distinction relies on IHC; lymphangiomas exhibit strong positivity for lymphatic endothelial markers such as D2-40 and CD31, whereas CCP cysts are lined by simple columnar epithelium that is negative for these markers.

The Coexistence Paradox: CCP and Synchronous Malignancy

Although CCP is primarily considered a benign condition that mimics colorectal carcinoma, rare cases have reported its coexistence with true colonic adenocarcinoma. Mitsunaga et al. documented instances in which a superficial adenocarcinoma directly overlaid the deep submucosal cysts characteristic of CCP (33). This finding challenges the traditional reliance on a preserved lamina propria rim as a definitive marker of benignity, as such features do not exclude the development of independent epithelial dysplasia or carcinoma at the mucosal surface. Therefore, careful endoscopic evaluation with multi-level, targeted biopsies is essential to avoid missing a superficial malignancy in the presence of benign-appearing submucosal CCP.

Management

The management of CCP is primarily conservative and individualized based on the severity of symptoms and the presence of any underlying precipitating conditions, rather than the cystic lesion itself (Figure 7). Because CCP is a benign, reactive process, aggressive surgical resection is generally avoided unless complications develop or malignancy cannot be definitively excluded (1).

Conservative Management: For most patients, particularly those with localized rectal involvement associated with SRUS or rectal prolapse, first-line therapy focuses on symptom control, medical interventions to reduce mechanical trauma, and regular follow-up (19, 34)

High-Fiber Diet and Bulking Agents: These are utilized to reduce straining and regulate bowel habits, thereby helping to mitigate the primary mechanical factors contributing to glandular displacement.

- **Biofeedback Therapy:** This treatment is recommended for patients with pelvic floor dyssynergia or internal procidentia to improve defecation mechanics.
- **Topical Treatments:** Sucralfate enemas or corticosteroid suppositories may be prescribed to reduce localized mucosal inflammation and alleviate symptoms such as tenesmus and bleeding (35).

Endoscopic Interventions: Advances in imaging techniques, such as EUS, have enhanced diagnostic confidence in excluding malignancy. Consequently, endoscopic management has become the preferred organ-sparing approach for localized lesions.

- **Endoscopic Mucosal Resection (EMR):** This technique is effective for removing smaller pedunculated or sessile lesions, providing both symptomatic relief and a definitive histopathological specimen (36).
- **Polypectomy:** Often sufficient for "polypoid" variants of CCP that are easily accessible via colonoscopy (17, 37).

Surgical Management: Surgery is reserved for specific clinical scenarios (1, 5, 34) in which conservative or endoscopic measures are insufficient.

- **Rectal Prolapse Repair:** Procedures such as rectopexy address the anatomical root cause—mechanical sliding and trauma—which often leads to the regression of CCP lesions.
- **Local Transanal Excision:** Indicated for symptomatic rectal masses causing significant obstruction or persistent hematochezia.
- **Segmental Resection:** This procedure is

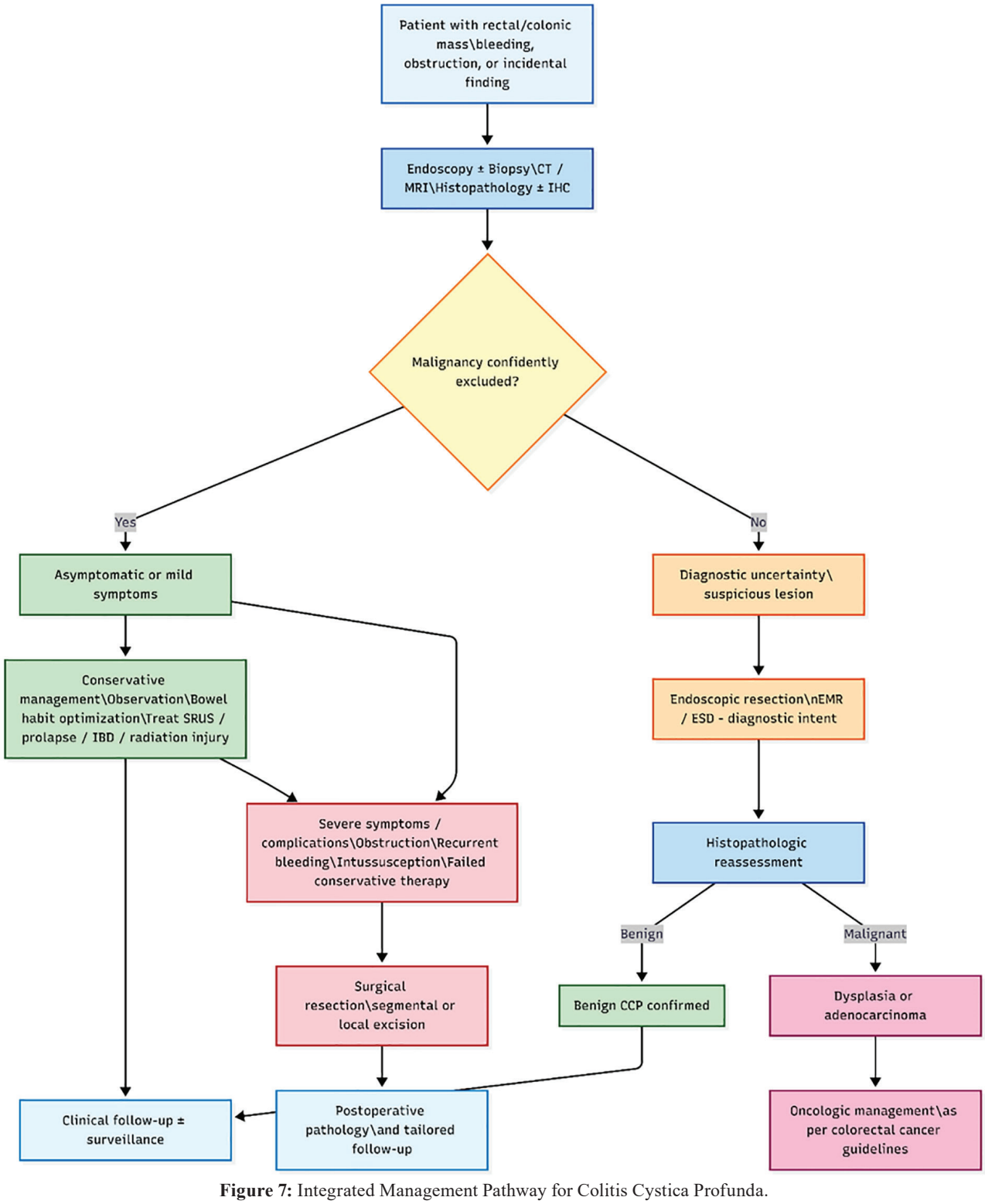

Figure 7: Integrated Management Pathway for Colitis Cystica Profunda.

considered a last resort for diffuse disease with debilitating symptoms or in cases where the "malignancy trap" cannot be resolved despite exhaustive multimodal evaluation.

In resource-constrained environments where advanced modalities such as EUS or MRI are unavailable, clinicians must rely on a combination of high-resolution transabdominal ultrasound, contrast-enhanced CT, and serum tumor markers as primary diagnostic tools. This streamlined approach, summarized in Figure 8, facilitates risk stratification by identifying "high-suspicion" features that necessitate urgent referral for definitive biopsy or specialized care. Conversely, for low-risk cases, the algorithm provides a structured pathway for serial monitoring using basic imaging, ensuring patient safety while optimizing the use of limited local resources.

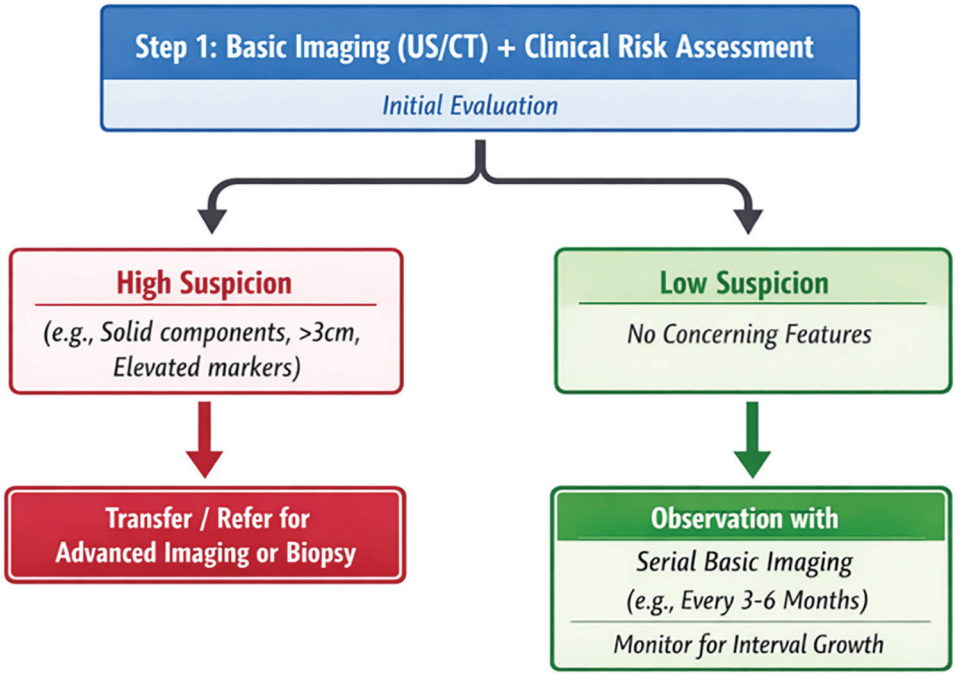

Figure 8: Minimum Diagnostic Workup and Management Algorithm for Resource-Limited Settings.

Prognosis

The long-term prognosis for CCP is generally excellent, provided that the underlying causative factors are addressed. However, clinicians must remain vigilant for recurrence. Sarzo et al. highlighted that local recurrence can occur, particularly when surgical or endoscopic excision is performed without correcting the primary mechanical dysfunction, such as rectal prolapse or chronic straining (38). This underscores the necessity of a functional approach to treatment rather than focusing solely on lesion removal.

Conclusion

CCP remains one of the most significant diagnostic challenges in colorectal medicine. Its ability to mimic mucinous adenocarcinoma—the so-called "Malignancy Trap"—demands a high degree of clinical suspicion and a coordinated, multimodal diagnostic approach. By 2026, the integration of high-resolution EUS and T2-weighted MRI has significantly improved our ability to identify the pathognomonic submucosal "string of pearls" sign, reducing the need for radical surgery.

Ultimately, the definitive "safety check" relies on histopathological confirmation of benign glandular displacement surrounded by specialized lamina propria. By recognizing CCP's diverse presentations—from its association with rectal prolapse to its rare coexistence with synchronous cancer—clinicians can avoid the pitfalls of overtreatment and provide conservative, organ-sparing management for this benign yet deceptive condition.

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Conflict of Interest: None declare.

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