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Wu YX, Tian R, Li XW, Guo JY, Tang JF, Zhou CF. Emerging non-invasive imaging biomarkers of Ki-67 in pancreatic cancer: Toward predictive precision oncology. *World J Gastrointest Oncol* 2025; 17(11): 110468 [DOI: 10.4251/wjgo.v17.i11.110468]

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The primary aim of *World Journal of Gastrointestinal Oncology* (*WJGO*, *World J Gastrointest Oncol*) is to provide scholars and readers from various fields of gastrointestinal oncology with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

*WJGO* mainly publishes articles reporting research results and findings obtained in the field of gastrointestinal oncology and covering a wide range of topics including liver cell adenoma, gastric neoplasms, appendiceal neoplasms, biliary tract neoplasms, hepatocellular carcinoma, pancreatic carcinoma, cecal neoplasms, colonic neoplasms, colorectal neoplasms, duodenal neoplasms, esophageal neoplasms, gallbladder neoplasms, *etc.*

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## Management of peritoneal metastases from colorectal cancer and small bowel adenocarcinoma in patients with inflammatory bowel disease

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### Abstract

Patients with inflammatory bowel disease (IBD) have an increased risk of developing colorectal cancer, which may ultimately result in peritoneal metastases (PM). PM in patients with IBD is by nature difficult to treat due to the chronic inflammation and immunosuppression inherent in IBD. This minireview compiled existing evidence on management approaches to PM in patients with IBD, including surgical procedures, systemic treatment, and novel therapies. A literature review was conducted by searching PubMed and Scopus through June 2025 for studies addressing PM in IBD-associated colorectal or small bowel cancer. Literature specific to PM in IBD is sparse, comprising primarily two small retrospective cohort series comparing outcomes of cytoreductive surgery with hyperthermic intraperitoneal chemotherapy (CRS/HIPEC) in patients with and without IBD. These studies indicated that in high-volume centers with careful preoperative optimization perioperative morbidity and mortality rates for patients with IBD undergoing CRS/HIPEC were similar to those without IBD. However, median overall survival (approximately 19.6-24.0 months) and disease-free survival were consistently shorter and rates of early peritoneal recurrence were higher in patients with IBD. Although CRS/HIPEC can be performed safely in selected patients with IBD and PM, long-term oncologic outcomes appear inferior compared to populations without IBD, likely reflecting later-stage presentation, distinct tumor biology, and IBD-related factors.

**Key Words:** Peritoneal metastases; Colorectal cancer; Small bowel adenocarcinoma; Inflammatory bowel disease; Cyto-reductive surgery; Hyperthermic intraperitoneal chemotherapy; Surveillance

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**Core Tip:** Peritoneal metastases in patients with inflammatory bowel disease (IBD) represent a rare but clinically significant complication of IBD-associated colorectal and small bowel cancers. This minireview highlighted the unique challenges in diagnosis, treatment, and prognosis for this population, emphasizing that while cytoreductive surgery with hyperthermic intraperitoneal chemotherapy is feasible, long-term oncological outcomes remain inferior to patients without IBD. This minireview underscored the urgent need for IBD-specific research, refined surveillance strategies, and personalized therapeutic approaches.

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## INTRODUCTION

Inflammatory bowel disease (IBD) is a chronic, recurrent inflammatory disease of the gastrointestinal tract, encompassing predominantly Crohn's disease (CD) and ulcerative colitis (UC). CD may involve any part of the gastrointestinal tract from mouth to anus and is characterized by transmural inflammation. UC involves colon and rectum only and involves continuous mucosal inflammation. Both diseases are characterized by remitting and relapsing, immune dysregulation, mucosal damage, and a significant impact on patient quality of life[1-4].

Beyond its gastrointestinal manifestations, IBD has long-term sequelae, and most importantly an increased predisposition to cancers, notably colorectal cancer (CRC). One of the complications of CRC is developing peritoneal metastases (PM)[1,5-8]. PM are defined as the dissemination of cancer cells inside the peritoneal cavity with implantation and growth of secondary tumors on the peritoneal surfaces. In CRC PM are associated with a poor prognosis and present a special diagnostic and therapeutic challenge[9,10].

Despite growing interest in aggressive management strategies for PM, such as cytoreductive surgery (CRS) with hyperthermic intraperitoneal chemotherapy (HIPEC), their application in patients with IBD remains controversial and underexplored. This minireview examined the intersection between PM and IBD with a focus on pathophysiological mechanisms, clinical aspects, and novel management algorithms. Through the integration of available evidence, we aimed to provide a general overview of this complex clinical scenario and identify areas of clinical innovation and further research.

## EPIDEMIOLOGY

Understanding how frequently PM occur among IBD-associated CRC and which patients are most at risk, underpins any strategy for early detection and prevention. In this section we summarized current data on cancer incidence in IBD and the relative rarity of peritoneal spread in this setting. The incidence of CRC is higher in patients with IBD compared with the general population. Several large population-based series and meta-analyses give an estimate of around 1.5 to 2.0 excess risk of CRC in IBD when compared with patients without IBD with standardized incidence ratios for UC and CD ranging from 1.2 to 2.5 and differing by disease duration, extent, and population[11-13]. The American Gastroenterological Association estimates that the risk of CRC in UC is about 2% at 10 years, 8% at 20 years, and 18% at 30 years of disease[14]. In a large English cohort, 1.3% of CRC cases had a history of IBD, and patients with IBD-CRC were younger at diagnosis and had a higher incidence of right-sided tumors[15].

The patients at the highest risk with IBD for the development of malignancy are patients with extensive and long-standing colonic inflammation, primary sclerosing cholangitis (PSC), family history of CRC, and a history of previous dysplasia or stricture. Patients with ulcerative pancolitis and with CD colitis have an especially increased risk. The risk rises with longer duration of disease, especially beyond 8-10 years, and more severe and widespread inflammation. The coexistence of PSC also increases the risk of CRC even at the time of PSC diagnosis and necessitates intensified surveillance[14,16,17].

Other risk factors include young age at diagnosis of IBD, post-inflammatory polyps, and family or personal history of CRC. Hereditary polypoid syndromes, such as familial adenomatous polyposis or Peutz-Jeghers syndrome, can increase the risk of malignancy. When these syndromes coexist with IBD, the cumulative risk of CRC increases significantly due to the overlapping pathways of genetic predisposition and chronic mucosal inflammation. Immunomodulator therapy,

especially when combined with biologics, further compounds the risk by predisposing patients to lymphoma and potentially other malignancies. Females with IBD are at higher risk for CRC and melanoma, whereas males exhibit a higher risk of lymphoma[17-20].

The frequency of PM as a site of dissemination in IBD-related CRC is not well quantified in the literature, but existing data suggest it is rare and no more frequent than in sporadic CRC. In a multicentric French study of surgery in patients with IBD for PM from CRC or small bowel adenocarcinoma, 14 patients with IBD were diagnosed over 21 years, indicating that PM is an uncommon event in IBD-associated malignancy[21]. In conclusion compared with patients without IBD, the cumulative risk for CRC is higher in IBD, but metastatic distribution, such as PM, does not appear different. PM are rare events in both cases.

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## PATHOPHYSIOLOGY

Before discussing PM presentation and management in IBD, it is crucial to explain how chronic inflammation causes tumor formation and peritoneal dissemination. Chronic immune activation in IBD produces reactive oxygen and nitrogen species and activates protumor cytokine signaling pathways (*e.g.*, nuclear factor kappa B, cyclooxygenase-2, interleukin-6/signal transducer and activator of transcription 3, T helper 17). These pathways cause DNA damage, mutagenesis, and abnormal epithelial repair, predisposing towards neoplastic transformation and early p53 mutations, pathways distinct from the classic adenoma-carcinoma sequence in sporadic CRC[22-26]. Nuclear factor kappa B activation promotes tumor survival, angiogenesis, and invasion. Its elevated expression in CRC with nodal or peritoneal dissemination highlights it as a potential therapeutic target although targeted inhibitors remain investigational[27-30]. Dysbiosis and deranged gut microbiota also augment procarcinogenic signaling and immune dysregulation, accelerating the inflammation-dysplasia-carcinoma sequence[31,32].

The major pathways of peritoneal spread of malignancy in IBD are direct invasion of the serosa by advanced colon or small bowel malignancies, lymphatic dissemination, and iatrogenic sources including surgical seeding at colectomy or other intra-abdominal operations. Direct extension is the most common, often in transmural CD or cancer of advanced colitis. Lymphatic spread and iatrogenic spread are less common but known entities, particularly with prior surgical intervention[21].

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## CLINICAL PRESENTATION AND DIAGNOSIS

This section outlined the key diagnostic challenges and examined available tools and strategies to improve early detection in this complex patient population. PM in patients with IBD often present with nonspecific symptoms, such as abdominal pain, bloating, and altered bowel habits, that overlap with IBD flares, frequently delaying recognition until advanced stages. This clinical ambiguity frequently contributes to delayed or missed diagnoses with significant implications for timely treatment and overall prognosis[33].

A combination of imaging modalities, minimally invasive interventions, and biomarkers is typically used to screen and evaluate PM[34,35]. CT demonstrates approximately 68% sensitivity and 88% specificity in detecting PM. In patients with IBD, however, CT accuracy may be compromised by surgical alterations, strictures, and inflammatory processes obscuring peritoneal surfaces[34,35]. Positron emission tomography (PET) with CT has shown even higher sensitivity, up to 80%, compared with CT and is a valuable adjunct when conventional imaging results are inconclusive. However, its use in IBD-specific populations must be confirmed. Diffusion-weighted magnetic resonance imaging (DW-MRI) has emerged as the most sensitive cross-sectional tool for detecting PM with meta-analyses reporting pooled sensitivity around 89%-92% and specificity of 85%-86%, outperforming CT and PET/CT in gastrointestinal malignancies. DW-MRI is increasingly favored given its superior soft-tissue contrast for small-volume disease and lack of ionizing radiation[34-36]. Where imaging is indeterminate, laparoscopy is both direct and highly sensitive as a diagnostic modality. Minilaparoscopy, a less invasive method, has been found to have outstanding diagnostic accuracy. It provides direct visualization and biopsy of peritoneal lesions and may be helpful in challenging IBD cases where noninvasive modalities are insufficient[37].

Carcinoembryonic antigen is a widely used tumor marker in gastrointestinal cancer and can help detect metastatic disease. In IBD its use is limited by the potential for false-positive elevation secondary to chronic inflammation. No studies to date have investigated the usefulness of carcinoembryonic antigen in that subset of patients with IBD and PM, and further research is needed into useful biomarkers for this population[33]. Liquid biopsy approaches, such as detection of peritoneal cell-free DNA or tumor DNA in lavage fluid, as well as serial plasma circulating tumor DNA monitoring hold promise for early detection of microscopic peritoneal disease and prognostication, often preceding radiographic findings. However, these techniques require further validation in IBD cohorts in which the inflammatory milieu may affect assay performance[38-40].

Once PM are confirmed histologically, individualized molecular profiling should be pursued to guide therapy according to general oncology practice despite the absence of IBD-specific guidelines mandating this step. Molecular characterization of PM in IBD is a developing area with a focus on assessing the actionability of mutations, prognostication, and identifying potential therapeutic targets. Subsequent studies indicated that IBD-related CRC as the predominant source of PM in these patients, frequently harbor distinct molecular alterations compared to sporadic CRC. Notably, a significant majority of patients with IBD-CRC have hypermutations and deficient mismatch repair, particularly in proximal colon tumors, that can be identified using whole-exome sequencing or immunohistochemical analysis for mismatch repair proteins. Hypermutated tumors carry a greater burden of neoepitopes and may therefore be more

sensitive to immunotherapy[41-44].

Apart from mismatch repair deficiency, molecular profiling of IBD-CRC has identified recurrently mutated genes such as *KRAS*, and *TP53*, whose possible prognostic significance and relevance to targeted therapy remain to be established. Epigenetic alterations, such as Ras association domain family protein1 isoform A suppression and perturbation of metabolic and proliferative signaling pathways (*e.g.*, receptor-interacting protein kinase 2, AMP-activated protein kinase alpha 1, yes-associated protein 1), are emerging as potential biomarkers, amenable to noninvasive testing on blood or tissue samples. Multiomics approaches integrating genomics, transcriptomics, and proteomics are under investigation to improve risk stratification and personalized management in patients with IBD at risk for PM. While these molecular approaches are yet to be included in the routine clinical evaluation in all patients with IBD, their use is expanding in specialized centers to complement histopathological and radiological evaluation, particularly in diagnostically ambiguous cases or when considering eligibility for targeted therapies[41-44].

## MANAGEMENT AND TREATMENT OPTIONS

Having established the diagnostic imperatives, management of PM in patients with IBD must balance feasibility and safety against evidence of inferior long-term outcomes. We performed a literature search on therapeutic strategies for PM in patients with IBD. We searched MEDLINE and Scopus from inception to June 2025 using combinations of terms such as “inflammatory bowel disease”, “peritoneal metastases”, “cytoreductive surgery”, “HIPEC”, “systemic therapy”, “targeted therapy”, and “immunotherapy”, supplemented by manual review of reference lists and relevant conference abstracts. Given the paucity of IBD-specific trials, we also examined broader PM literature to extract insights potentially applicable to IBD contexts, carefully considering anatomical, inflammatory, and immunosuppressive factors unique to IBD. Articles were selected based on relevance to treatment modalities, perioperative management, and adjunctive therapies. Data on intervention protocols, patient optimization, outcomes, and adverse events were collated and critically appraised in light of IBD-related challenges.

Patients with IBD and PM represent a unique clinical dilemma, illustrating the interplay of chronic inflammation, previous surgery, and tumor behavior. CRS/HIPEC offers a potential curative approach for selected patients with colorectal or small bowel PM. Yet outcomes for patients with IBD undergoing CRS/HIPEC remain poorly defined. To the best of our knowledge, only two studies, a single-center cohort and a French multicenter series, have been published on this topic, shedding light on this important clinical question[21,45].

One of the defining distinctions between patients with and without IBD lies in their clinical presentation and baseline characteristics. As seen in **Table 1**, patients with IBD differed from patients without IBD in several baseline characteristics that may influence both perioperative risk and oncological behavior. For example, the significantly lower American Society of Anesthesiologists scores in the IBD cohort ( $P = 0.005$ ) reflect younger age and fewer comorbidities at presentation. However, 30% of patients with IBD had small bowel primary tumors *vs* none in the non-IBD group in one study, underscoring a distinct tumor distribution that may affect biology and surgical planning. All patients with IBD had a history of prior abdominal surgery in contrast to the variable surgical histories in the non-IBD group. This is an important factor when anticipating adhesions and operative complexity.

**Table 1 Baseline clinical characteristics of patients with peritoneal metastases undergoing cytoreductive surgery with hyperthermic intraperitoneal chemotherapy comparing inflammatory bowel disease and non-inflammatory bowel disease cohorts across two studies**

Feature	Ben-Yaacov <i>et al</i> [45], 2024		Hammoudi <i>et al</i> [21], 2018
	IBD cohort (n = 10)	Non-IBD cohort (n = 222)	
Primary tumor site	30% small bowel primaries	100% colorectal	50% small-bowel, 50% colon/rectum
ASA score <sup>1</sup>	Lower ( $P = 0.005$ )	Higher	Not reported
Hypertension	Less frequent ( $P = 0.033$ ) <sup>3</sup>	More frequent	Not reported
Prior abdominal surgery	Universal (IBD management)	Variable	50% prior resections for IBD
Surveillance colonoscopy <sup>2</sup>	N/A		Only 2/14 had routine surveillance

<sup>1</sup>American Society of Anesthesiologists physical status classification score: Lower values indicate fewer systemic comorbidities.

<sup>2</sup>Surveillance colonoscopy indicates routine endoscopic monitoring prior to diagnosis.

<sup>3</sup> $P$  values refer to comparisons between inflammatory bowel disease and non-inflammatory bowel disease cohorts;  $P < 0.05$  considered statistically significant. When  $P$  values are non-significant yet sample sizes are small, interpret with caution due to limited statistical power.

IBD: Inflammatory bowel disease; ASA: American Society of Anesthesiologists physical status classification; N/A: Not applicable.

Although surveillance colonoscopy was reported for only 2/14 patients with IBD in Hammoudi *et al*[21], comparable surveillance data in non-IBD cohorts were not detailed, raising concern for stage-at-diagnosis bias in IBD. The peritoneal cancer index (PCI) was similar between groups, suggesting comparable disease burden at surgery, but the impact of prior surgeries on PCI assessment (*e.g.*, underestimation due to adhesions) should be acknowledged. These baseline dis-

tinctions imply that although patients with IBD may be younger and with fewer coexisting diseases, the predominance of small bowel primaries and universal history of abdominal surgery necessitate specialized preoperative planning and may influence prognosis independently of traditional factors.

Short-term outcomes after CRS/HIPEC were also equivalent between patients with and without IBD. **Table 2** shows that overall and major complication rates following CRS/HIPEC were similar between patients with and without IBD with approximately 60% experiencing any complication. Reoperation rates (approximately 27%) and zero 30-day mortality in both groups indicate that in experienced centers CRS/HIPEC can be performed safely even in patients with IBD with prior surgeries and tissue changes. Intensive care unit/hospital stay durations were reported as not significantly different. These findings support that IBD should not be an absolute contraindication for CRS/HIPEC when performed in high-volume centers with multidisciplinary expertise. Nevertheless, careful perioperative management remains critical.

However, a critical divergence emerges in oncological outcomes. As detailed in **Table 3**, patients with IBD demonstrated consistently shorter median overall survival (OS) and disease-free survival following CRS/HIPEC compared with patients without IBD. In a study from Ben-Yaacov *et al*[45], median OS was 19.6 months for IBD *vs* 53.2 months for non-IBD ( $P = 0.056$ ), a difference that narrowly misses statistical significance. Similarly, median disease-free survival of 4.9 months *vs* 9.4 months ( $P = 0.174$ ) trends toward worse outcomes in patients with IBD. Hammoudi *et al*[21] reported median OS of 24 months in IBD *vs* 44.3 months in sporadic cases (hazard ratio = 4.47) and recurrence-free survival of 8.3 months *vs* 12.9 months (hazard ratio = 2.31), indicating a significantly higher risk of early recurrence. The predominance of early peritoneal recurrences suggests more aggressive biology or delayed detection in IBD-related cancers.

The two retrospective cohort studies evaluating CRS/HIPEC in IBD-associated PM provided valuable early evidence but require cautious interpretation because of several methodologic limitations. Hammoudi *et al*[21] included only 14 IBD over two decades. The small number of IBD cases is a limitation for statistical power, especially for survival analyses, and raises concerns regarding selection bias as these patients could be those who are considered fit for aggressive surgical treatment despite presenting with advanced disease. Similarly, Ben-Yaacov *et al*[45] paired 10 patients with IBD with 222 controls without IBD who had large differences in age and comorbidity that could potentially confound results. Short-term outcomes appeared comparable, but the retrospective design precludes adjustment for residual confounders. Moreover, follow-up policy and adjuvant treatment protocols were inhomogeneous, and postoperative surveillance intervals, impacting early detection of recurrence, were inadequately described.

Clinically, these studies suggest that CRS/HIPEC can be performed safely in selected patients with IBD at high-volume centers with multidisciplinary expertise, provided that meticulous preoperative optimization ensures quiescent disease, adequate nutritional status, and judicious immunosuppression management. However, the consistently poorer long-term survival serves to reemphasize the need for finer patient selection. Importantly, clinicians should frame CRS/HIPEC in IBD-associated PM as investigational, emphasizing uncertain long-term benefit during shared decision-making and ensuring that patients understand the balance between perioperative risks and potentially attenuated survival gains. The findings reinforce the imperative for earlier detection of IBD-associated malignancies through rigorous surveillance as delayed diagnosis may underlie advanced peritoneal involvement. In conclusion management of PM in patients with IBD with CRS/HIPEC shows a dilemma: Operatively possible and safe but long-term outcomes still compromised.

## PROPOSED DIAGNOSTIC AND MANAGEMENT ALGORITHM FOR PM IN PATIENTS WITH IBD

Patients with IBD who develop PM require a nuanced, evidence-aligned approach beginning with rigorous risk stratification and vigilant surveillance. High-risk features should trigger intensified endoscopic monitoring in keeping with European Crohn's and Colitis Organization and American Gastroenterological Association guidelines that recommend initiating surveillance colonoscopy around 8-10 years after diagnosis and adjusting intervals based on risk factors[36,46-48]. In CD with small-bowel involvement, adjunctive magnetic resonance enterography is appropriate to detect neoplastic lesions beyond the reach of colonoscopy. When patients at high risk present with new or atypical symptoms, such as unexplained weight loss, persistent abdominal pain, or ascites, clinicians must promptly pursue cross-sectional imaging rather than presume an IBD flare. Contrast-enhanced CT remains the initial modality for suspected peritoneal disease. If CT is equivocal, DW-MRI offers enhanced soft-tissue contrast, and PET/CT may further delineate metabolically active implants when inflammation is controlled. Early diagnostic laparoscopy should follow inconclusive imaging in the setting of persistent suspicion as direct visualization and biopsy constitute the diagnostic gold standard. Molecular profiling of confirmed PM can be pursued in accordance with general oncology protocols[44,49-51].

Selection of patients with IBD for CRS/HIPEC must balance the demonstrated perioperative safety and acceptable short-term outcomes against the consistently shorter long-term survival. Standard CRS/HIPEC criteria, such as a PCI low enough to permit complete cytoreduction ( $PCI \leq 15-20$ ), absence of unresectable extraperitoneal disease, and adequate performance status, should be adapted by incorporating IBD-specific factors: Confirmation of disease quiescence at surgery; evaluation of residual bowel length after prior resections to anticipate postoperative absorption and nutritional issues; perioperative immunomodulation planning; and anticipation of adhesion burden requiring surgeons experienced in IBD-related complexity[21,45].

Regarding HIPEC protocols, there has been current Peritoneal Surface Oncology Group International consensus towards mitomycinC-based protocols over short-duration, high-dose oxaliplatin regimens based on concerns regarding toxicity as well as uncertain advantage of oxaliplatin over peritoneal indications[52]. Postoperative management should be in accordance with enhanced recovery principles adapted for patients with IBD with careful surveillance for anastomotic failure and regular imaging every 3-4 months during the first year to enable early recurrence detection. Clear shared decision-making is indicated, placing CRS/HIPEC in position for IBD-associated PM as experimental, given

**Table 2 Postoperative outcomes following cytoreductive surgery and hyperthermic intraperitoneal chemotherapy (cytoreductive surgery with hyperthermic intraperitoneal chemotherapy) in patients with and without inflammatory bowel disease with peritoneal metastases**

Metric	Ben-Yaacov <i>et al</i> [45], 2024		Hammoudi <i>et al</i> [21], 2018
	IBD cohort (n = 10)	Non-IBD cohort (n = 222)	
Overall complications <sup>1</sup>	60.3% (P = 0.744) <sup>5</sup>	60.3% (P = 0.444)	54.5% grade 3-4
Reoperation rate <sup>2</sup>	27%		Not separately tabulated
ICU/hospital stay <sup>3</sup>	No significant difference		Median not reported
30-day mortality <sup>4</sup>	0%		0%

<sup>1</sup>Overall complications: Any postoperative adverse event within 30 days, graded by Clavien-Dindo. Major complications: Clavien-Dindo grade III-IV events requiring surgical, endoscopic, or radiologic intervention (grade III) or life-threatening complications requiring intensive care unit management (grade IV).

<sup>2</sup>Reoperation rate: Percentage of patients requiring return to the operating room within 30 days.

<sup>3</sup>When median intensive care unit/hospital stays are not reported, indicate "not reported" and consider including these metrics in future data collection for completeness.

<sup>4</sup>30-day mortality: Perioperative death within 30 days post-surgery.

<sup>5</sup>P values (where reported) compared inflammatory bowel disease *vs* non-inflammatory bowel disease; P < 0.05 considered significant. Non-significant P values in small samples may reflect limited power.

ICU: Intensive care unit; IBD: Inflammatory bowel disease.

**Table 3 Oncological outcomes following cytoreductive surgery with hyperthermic intraperitoneal chemotherapy in patients with and without inflammatory bowel disease with peritoneal metastases**

Feature <sup>1</sup>	Ben-Yaacov <i>et al</i> [45], 2024		Hammoudi <i>et al</i> [21], 2018
	IBD cohort (n = 10)	Non-IBD cohort (n = 222)	
Median overall survival <sup>1</sup>	19.6 months (P = 0.056) <sup>2</sup>	53.2 months	24 months ( <i>vs</i> 44.3 months in sporadic; HR <sup>4</sup> = 4.47)
Median disease-free survival <sup>1</sup>	4.9 months (P = 0.174)	9.4 months	RFS 8.3 months <i>vs</i> 12.9 months (HR = 2.31)
Recurrence pattern <sup>3</sup>			10/11 relapsed; median 7.2 months; 8/10 peritoneal

<sup>1</sup>Median overall survival/disease-free survival/recurrence-free survival: Time from cytoreductive surgery with hyperthermic intraperitoneal chemotherapy to death (overall survival) or recurrence (disease-free survival/recurrence-free survival); censored at last follow-up.

<sup>2</sup>P values indicate statistical comparison. P < 0.05 considered significant but interpret cautiously in small samples due to wide confidence intervals and limited power.

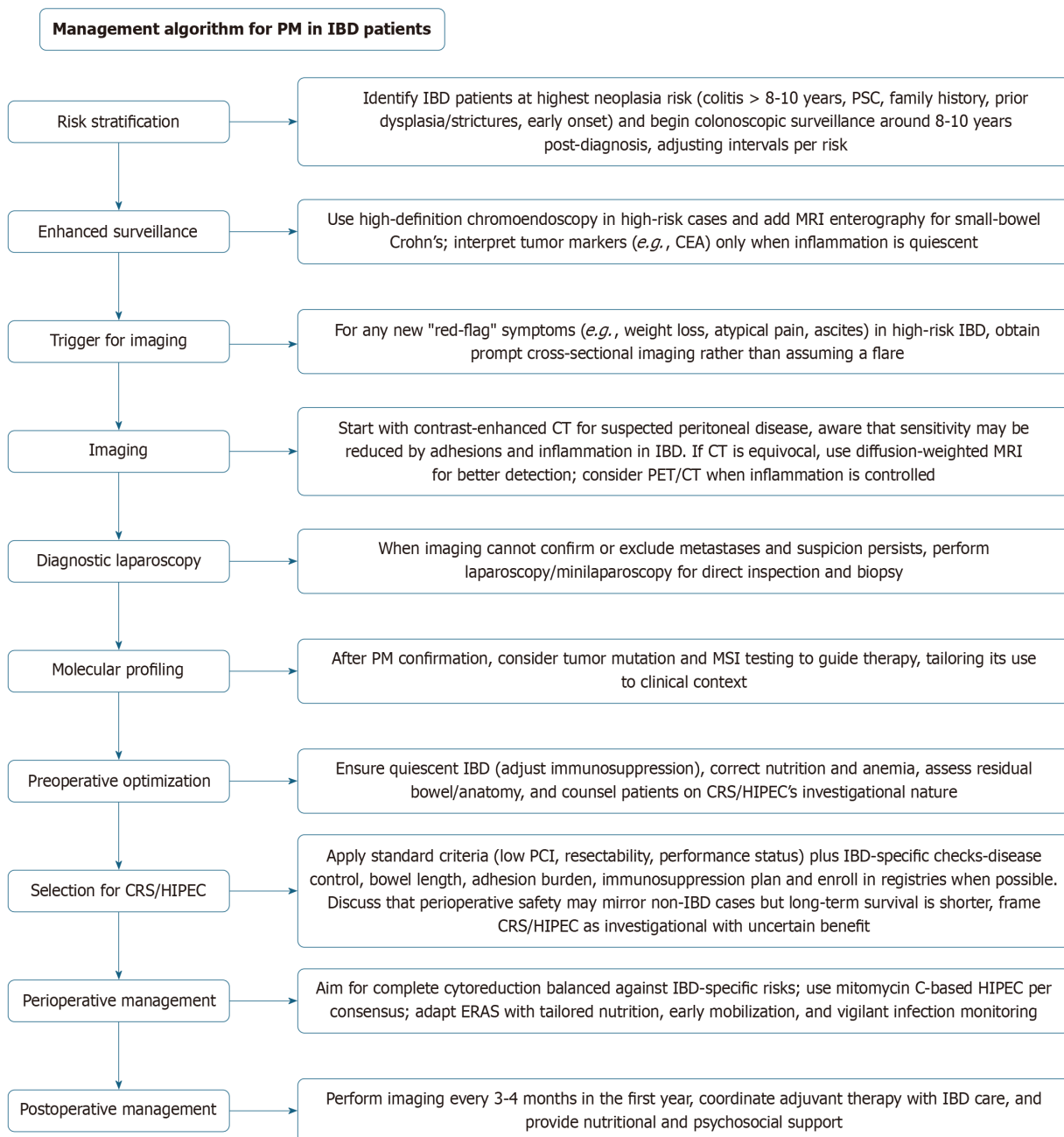
<sup>3</sup>Recurrence pattern: Specify proportion of recurrences that are peritoneal. Early recurrence within first year post-cytoreductive surgery with hyperthermic intraperitoneal chemotherapy.

<sup>4</sup>Hazard ratios compare risk of event (death or recurrence) between inflammatory bowel disease and non-inflammatory bowel disease cohorts. Include 95% confidence intervals when available.

IBD: Inflammatory bowel disease; RFS: Recurrence-free survival; HR: Hazard ratio.

limited evidence of long-term benefit[53-57].

The algorithm for patients with IBD diverges from general population PM protocols primarily through its emphasis on proactive, risk-adapted surveillance and IBD-specific perioperative considerations. Whereas most PM pathways rely on symptom-triggered imaging and standard postoperative follow-up, the IBD approach begins with early colonoscopic surveillance (8-10 years after IBD diagnosis) and adjunctive small bowel imaging in CD to detect neoplasia before peritoneal spread. In diagnosis it lowers the threshold for advanced modalities (DW-MRI, PET/CT) and early laparoscopy because adhesions and inflammatory changes reduce CT accuracy. In selection for CRS/HIPEC it overlays standard IBD-specific criteria. Perioperative and recovery protocols are tailored to address adhesion burden, altered anatomy, immunologic status, and the psychosocial toll of chronic IBD. Finally, it underscores the need for IBD-specific registries and translational research to refine selection, surveillance strategies, and adjunctive therapies, elements not present in general PM management algorithms. **Figure 1** depicts a stepwise algorithm for risk stratification, surveillance, diagnostic evaluation, patient selection, perioperative management, and postoperative follow-up in IBD-associated PM, incorporating IBD-specific considerations at each stage.



**Figure 1 Proposed diagnostic and management algorithm for peritoneal metastases in patients with inflammatory bowel disease.** IBD: Inflammatory bowel disease; PM: Peritoneal metastases; CRS/HIPEC: Cytoreductive surgery with hyperthermic intraperitoneal chemotherapy; PSC: Primary sclerosing cholangitis; MRI: Magnetic resonance imaging; CEA: Carcinoembryonic antigen; PET: Positron emission tomography; MSI: Microsatellite instability; PCI: Peritoneal cancer index; ERAS: Enhanced recovery after surgery.

## CONTROVERSIES, KNOWLEDGE GAPS AND FUTURE DIRECTIONS

Despite demonstration of perioperative feasibility in small IBD cohorts, significant uncertainties remain regarding long-term benefit of CRS/HIPEC and optimal adjunctive therapies for PM in IBD. The interaction between PM and IBD is a complex, poorly understood clinical field. While aggressive interventions such as CRS/HIPEC are increasingly considered, current treatment strategies are largely extrapolated from studies of sporadic CRC studies with limited consideration for the distinct pathophysiological and clinical features of IBD-associated malignancies. Key concerns include differences in tumor biology, challenges in surveillance, and treatment outcomes.

Current practice guidelines for the care of PM are based almost exclusively on populations without IBD, and their applicability to patients with underlying IBD, particularly CD, has not been established[21,58]. Endoscopic surveillance is highly valuable for the early detection of colorectal neoplasia in patients with IBD[59]. Techniques such as chromoendoscopy have improved the detection of dysplasia, particularly flat or multifocal lesions. However, its direct impact on preventing PM remains unclear and should be further investigated. Adherence to surveillance is suboptimal in clinical practice, and no clinical studies have conclusively demonstrated an association between endoscopic surveillance and

reduced risk of PM. A major limitation in developing evidence-based therapies is the ongoing under enrollment of patients with IBD in clinical trials assessing therapy for PM. Patients with IBD are systematically excluded from the majority of CRS/HIPEC trials due to perceptions of increased surgical risk, immunosuppressive therapy, and altered anatomy. Therefore, no robust, IBD-specific treatment regimens for PM exist. A global survey reported that only 14% of patients with IBD have participated in clinical trials due to concerns regarding placebo control arms and the intrusiveness of monitoring techniques[60].

According to the proposed diagnostic and treatment algorithm, therapeutic innovation in the management of PM in IBD should start with establishing specialized multidisciplinary tracks in tertiary centers where IBD gastroenterologists, surgical oncologists, radiologists, pathologists, nutritionists, and supportive care specialists collaborate on standard protocols. Individualized HIPEC regimens merit exploration: Modifications in agent selection, dose, or exposure time according to IBD-associated immunologic or pharmacokinetic factors might improve tolerability and efficacy, especially in the setting of altered gut function and immunologic environment of IBD. Implementation of enhanced recovery protocols adapted to patients with IBD will further optimize perioperative care. These clinical practice advances align with the Peritoneal Surface Oncology Group International guidelines to offer CRS/HIPEC in high-volume centers with the ability for complete cytoreduction and safe perioperative administration of chemotherapy with particular inclusion of IBD-related issues.

Prospective research efforts are essential to generate the evidence needed to refine selection criteria and improve outcomes. Multicenter international registries must be established to enroll patients with IBD with PM for CRS/HIPEC or alternative treatments, documenting detailed IBD history (phenotype, duration, treatments), tumor molecular profiles, perioperative variables, and longitudinal patient-reported outcomes. Registries can enable comparative effectiveness research and hypothesis generation for prognostic variables. Translational research on the IBD-associated PM tumor microenvironment examining inflammatory cytokine profiles, immune cell infiltration, and stromal interactions can elucidate therapeutic targets, including immunomodulatory agents. Biomarker development, such as circulating cell-free DNA or cytokine signatures with predictive value for early metastasis or recurrence, is necessary to further personalize patient selection and monitor minimal residual disease. Imaging studies should focus on advanced diffusion sequences or PET tracers that are less prone to inflammation for enhancing sensitivity and specificity for PM detection in IBD.

Complementary patient-reported and quality-of-life outcomes studies will define the impact of intensive interventions on this chronically ill population, informing shared decision-making. Health services research needs to address barriers to surveillance adherence in patients with IBD, developing interventions to optimize early detection. Ultimately, collaboration with professional societies such as European Crohn's and Colitis Organization and American Society of Clinical Oncology to develop consensus guidelines, education for clinicians on IBD-specific perioperative complications, and encouragement of referral networks to high-volume centers will translate these innovations and discoveries into practice, advancing care and improving outcomes for patients with IBD undergoing PM.

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## CONCLUSION

Given the limited but instructive data on CRS/HIPEC in IBD-associated PM, future efforts must shift from retrospective descriptions to proactive clinical innovation and research. Dedicated multidisciplinary care pathways should be established in high-volume centers, embedding risk-adapted surveillance to detect neoplasia before peritoneal spread. Prospective registries must capture granular IBD-specific variables, tumor molecular profiles, perioperative details, and patient-reported outcomes to refine selection criteria and perioperative optimization. Translational studies should interrogate the inflammatory tumor microenvironment and validate circulating/peritoneal biomarkers for minimal residual disease monitoring; imaging research must standardize advanced modalities and novel tracers in IBD-altered anatomy. Patients with IBD should be included in trials of systemic therapies, immunotherapies, and HIPEC regimen optimization. Finally, patient-centered research on quality-of-life and decision-making frameworks is essential to align aggressive interventions with patient values, thereby generating the evidence needed to improve long-term outcomes in this unique population.

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## FOOTNOTES

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