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Management of peritoneal metastases from ¹colorectal cancer and small bowel adenocarcinoma in patients with inflammatory bowel disease

Paramythiotis D *et al.* PM in IBD patients

¹¹ Abstract

Inflammatory bowel disease (IBD) patients have an increased risk of developing colorectal cancer, which may ultimately result in peritoneal metastases (PM). PM in IBD patients is by nature difficult to treat due to the chronic inflammation and immunosuppression inherent in IBD. This minireview will compile existing evidence on management approaches to PM in IBD patients, 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 ¹⁸outcomes of cytoreductive surgery with hyperthermic intraperitoneal chemotherapy (CRS/HIPEC) in IBD vs non-IBD patients. These studies indicate that, in high-volume centers with careful preoperative optimization, perioperative ²morbidity and mortality rates for IBD patients undergoing CRS/HIPEC are similar to those in non-IBD cohorts. However, ²median overall survival (approximately 19.6-24 months) and disease-free survival are consistently shorter in IBD patients, with higher rates of early peritoneal recurrence. Although CRS/HIPEC ⁴can be performed safely in selected IBD patients with PM, long-term oncologic outcomes appear inferior compared to non-IBD populations, likely reflecting later-stage presentation, distinct tumor biology, and IBD-related factors.

Key Words: Peritoneal metastases; ¹Colorectal cancer; Small bowel adenocarcinoma; Inflammatory bowel disease; Cytoreductive surgery; Hyperthermic intraperitoneal chemotherapy; Surveillance

Paramythiotis D, Tsavdaris D, Geropoulos G, Sacchet DA, Psarras K. Management of peritoneal metastases from colorectal cancer and small bowel adenocarcinoma in patients with inflammatory bowel disease. *World J Gastrointest Oncol* 2025; In press

Core Tip: Peritoneal metastases⁸ in patients with inflammatory bowel disease (IBD) represent a rare but clinically significant complication of IBD-associated colorectal and small bowel cancers. This minireview highlights the unique challenges in diagnosis, treatment, and prognosis for this population, emphasizing that while¹² cytoreductive surgery with hyperthermic intraperitoneal chemotherapy is feasible, long-term oncologic outcomes remain inferior to those in non-IBD patients. This minireview underscores the urgent need for IBD-specific research, refined surveillance strategies, and personalized therapeutic approaches.

¹⁰ INTRODUCTION

Inflammatory bowel disease (IBD) is a chronic, recurrent inflammatory disease of the gastrointestinal tract, encompassing predominantly³ 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 patients' quality of life[1-4].

Beyond its gastrointestinal manifestations, IBD has long-term sequelae, and most importantly an increased predisposition to cancers, notably⁸ 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, PM² 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 IBD patients remains controversial and underexplored. This minireview attempts to examine 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 aim 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 summarize current data on cancer incidence in IBD and the relative rarity of peritoneal spread in this setting. The incidence of CRC is higher in ¹⁴ 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 excess ¹⁹ risk of CRC in IBD when compared to non-IBD populations, with standardized incidence ratios for UC and CD ranging from 1.2 to 2.5 and differing by disease duration, extent, and population. The American Gastroenterological Association estimates ⁵ that the risk of CRC in UC is about 2% at 10 years, 8% at 20 years, and 18% at 30 years of disease. In a large English cohort, 1.3% of CRC cases had a history of IBD, and IBD-CRC patients were younger at diagnosis and had a higher incidence of right-sided tumors[11-15].

The highest-risk patient groups of IBD for the development of malignancy are patients with extensive and long-standing colonic inflammation, ¹⁶ 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. Women with IBD are at higher risk for CRC and melanoma, whereas men exhibit a higher risk of lymphoma[14,16-18].

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 experience of surgery in IBD patients for PM from CRC or small bowel adenocarcinoma, 14 IBD patients were diagnosed over 21 years, which indicates that PM is an uncommon event in IBD-associated malignancy[19]. In conclusion, ²⁰ compared to non-IBD patients, the cumulative risk for CRC is higher in IBD, but metastatic distribution, such as PM, does not appear different and PM are a rare event in both cases.

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 pro-tumor cytokine signaling pathways (*e.g.*, nuclear factor- κ B, ⁶ 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[20-24]. Nuclear factor- κ 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. Dysbiosis and deranged gut microbiota also augment pro-

carcinogenic signaling and immune dysregulation, accelerating the inflammation-dysplasia-carcinoma sequence.

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.

CLINICAL PRESENTATION AND DIAGNOSIS

This section outlines the key diagnostic challenges and examines available tools and strategies to improve early detection in this complex patient population. PM in IBD patients 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.

A combination of imaging modalities, minimally invasive interventions, and biomarkers is typically used to screen and evaluate PM. Computed tomography (CT) demonstrates approximately 68% sensitivity and 88% specificity in detecting PM. In IBD patients, however, CT accuracy may be compromised by surgical alterations, strictures, and inflammatory processes obscuring peritoneal surfaces. 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

avored given its superior soft-tissue contrast for small-volume disease and lack of ionizing radiation. 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 non-invasive modalities are insufficient.

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 IBD patients with PM, and further research is needed into useful biomarkers for this population. 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, where inflammatory milieu may affect assay performance.

Once PM is 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 indicates 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 IBD-CRC have hypermutation and deficient mismatch repair, particularly in proximal colon tumors, which 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.

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 ¹⁷protein kinase 2, AMP-activated protein kinase alpha 1, yes-associated protein 1), are emerging as potential biomarkers, amenable to non-invasive testing on blood or tissue samples. Multi-omics approaches integrating genomics, transcriptomics, and proteomics are under investigation to improve risk stratification and personalized management in IBD patients at risk for PM. While these molecular approaches are yet to be included in the routine clinical evaluation in all IBD patients, their use is expanding in specialized centers to complement histopathological and radiologic evaluation, particularly in diagnostically ambiguous cases or when considering eligibility for targeted therapies.

MANAGEMENT AND TREATMENT OPTIONS

Having established the diagnostic imperatives, management of PM in IBD patients must balance feasibility and safety against evidence of inferior long-term outcomes. We performed a literature search on therapeutic strategies for PM in IBD patients. 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.

IBD patients with 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 IBD patients 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, beginning to shed light on this important clinical question.

One of the defining distinctions between IBD and non-IBD patients lies in their clinical presentation and baseline characteristics. As seen in ²² Table 1, IBD patients differed from non-IBD patients in several baseline characteristics that may influence both perioperative risk and oncologic 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 IBD patients 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 IBD patients 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. Although surveillance colonoscopy was reported for only 2/14 IBD patients in Hammoudi *et al*[21], comparable surveillance data in non-IBD cohorts are 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 distinctions imply that, although IBD patients 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 IBD and non-IBD patients. Table 2 shows that overall and major complication rates following CRS/HIPEC were similar between IBD and non-IBD patients, 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 IBD patients 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 oncologic outcomes. As detailed in Table 3, IBD patients demonstrate consistently shorter median overall survival (OS) and disease-free survival following CRS/HIPEC compared to non-IBD counterparts. In 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 IBD patients. Hammoudi *et al*[21] report 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 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 provide 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 IBD patients with 222 non-IBD controls 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 protocol 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 IBD patients 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 IBD patients with CRS/HIPEC shows a dilemma: Operatively possible and safe but long-term outcomes still compromised.

PROPOSED DIAGNOSTIC AND MANAGEMENT ALGORITHM FOR PM IN IBD PATIENTS

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. In CD with small-bowel involvement, adjunctive magnetic resonance enterography is appropriate to detect neoplastic lesions beyond the reach of colonoscopy. When high-risk patients present with new or atypical ²³ 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.

Selection of IBD patients 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 extra-peritoneal 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. Regarding HIPEC protocols, there has been current Peritoneal Surface Oncology Group International consensus towards mitomycin-C-based protocols over short-duration, high-dose oxaliplatin regimens based on concerns regarding toxicity as well as uncertain advantage of oxaliplatin over peritoneal indications. Postoperative management should be in accordance with enhanced recovery principles adapted for IBD patients with careful surveillance for anastomotic failure and regular imaging, every three to four 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 limited evidence of long-term benefit.

The algorithm for IBD patients 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; and 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.

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 non-IBD populations, and their applicability to patients with underlying IBD, particularly CD, has not been established. Endoscopic surveillance is highly valuable ²⁴ for the early detection of colorectal neoplasia in IBD patients. 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 IBD patients in clinical trials assessing therapy for PM. IBD patients 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 IBD patients have participated in clinical trials, due to concerns regarding placebo control arms and the intrusiveness of monitoring techniques.

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 IBD patients will further optimize perioperative care. These clinical practice advances align with

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 IBD patients 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 IBD patients, developing interventions to optimize early detection. Ultimately, collaboration with professional societies such as ⁶European 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 IBD patients undergoing PM.

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; IBD patients should be included in trials of systemic therapies, immunotherapies, and HIPEC regimen optimization; and 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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