Section: Medicine

Systematic Review Article

IMPACT OF DISEASE DURATION, EXTENT, AND INFLAMMATORY SEVERITY ON COLORECTAL CANCER RISK IN INFLAMMATORY BOWEL DISEASE: A SYSTEMATIC REVIEW

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Received *: 07/02/2026*
Received in revised form *: 25/03/2026*
Accepted *: 11/04/2026*

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DOI: 10.70034/ijmedph.2026.2.95

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (2); 565-571

ABSTRACT

Background: Inflammatory Bowel Disease (IBD), including Ulcerative Colitis and Crohn's Disease, is associated with an increased risk of Colorectal Cancer. The risk is believed to be influenced by several disease-related factors, particularly duration of illness, anatomical extent of colonic involvement, and severity of chronic inflammation. While multiple observational studies have explored these associations, the magnitude and consistency of this risk factors remain variably reported. A comprehensive systematic review is therefore warranted to synthesize current evidence and clarify the relative contribution of these factors to colorectal cancer risk in IBD patients. **Objectives:** The objective of this systematic review is to evaluate the impact of disease duration, extent of colonic involvement, and inflammatory severity on the risk of colorectal cancer in patients with inflammatory bowel disease (IBD), and to identify high-risk patient subgroups while synthesizing available evidence to inform surveillance strategies and risk stratification.

Materials and Methods: This systematic review was conducted in accordance with the PRISMA guidelines. A comprehensive literature search was performed in electronic databases including PubMed, Scopus, and Web of Science. Observational studies (cohort and case–control studies), randomized control trials, review article, meta-analysis evaluating the association between disease duration, disease extent, inflammatory severity, and the risk of colorectal cancer in patients with inflammatory bowel disease were included. Titles, abstracts, and full texts were screened according to predefined eligibility criteria. Data extraction included the impact of disease duration, disease extent, and measures of inflammatory burden. The methodological quality of the included studies was assessed using appropriate risk-of-bias tools, and a qualitative synthesis was performed.

Results: Longer disease duration, extensive colitis, and persistent or severe inflammation were associated with an increased risk of colorectal cancer (CRC) in patients with inflammatory bowel disease. The risk increases approximately 8–10 years after diagnosis and rises with longer disease duration, particularly in pancolitis. Additional risk factors include primary sclerosing cholangitis, early disease onset, and family history of colorectal cancer, while surveillance colonoscopy improves dysplasia detection.

Conclusion: The risk of colorectal cancer (CRC) in ulcerative colitis increases with longer disease duration, extensive colitis, severe inflammation, dysplasia, and primary sclerosing cholangitis. Surveillance colonoscopy, including advanced techniques such as chromoendoscopy, improves early detection and may reduce CRC risk. Further research is needed to better understand disease mechanisms and develop improved biomarkers and preventive strategies.

Keywords: Inflammatory Bowel Disease (IBD), Ulcerative Colitis, Crohn's Disease, Colorectal Neoplasia, Chronic Inflammation, Disease Duration, Pancolitis, Surveillance Colonoscopy.

INTRODUCTION

Inflammatory Bowel Disease (IBD), encompassing Ulcerative Colitis (UC) and Crohn's Disease (CD), is a chronic relapsing inflammatory condition of the gastrointestinal tract with increasing global prevalence. One of the most serious long-term complications of IBD is the development of Colorectal Cancer (CRC), which significantly contributes to morbidity and mortality among affected individuals.

Patients with ulcerative colitis have an increased risk of developing colorectal cancer. However, the level of risk differs across studies and is influenced by factors such as disease duration, extent of colonic involvement at diagnosis, and the patient's age at diagnosis (Ekbom A et al., 1990).[1]

A study of ulcerative colitis (UC) in Stockholm County from 1955 to 1979 identified 1,274 cases. The distribution of disease extent—proctitis, left-sided colitis, and total colitis—remained stable during the study period. Incidence increased during the first two decades and later stabilized, with the highest occurrence in individuals in their third and fourth decades of life. A notable rise in incidence was observed among men over 40 years toward the end of the period. By December 1981, 109 patients had died, including 41 deaths related to UC, many occurring after surgery. Overall survival was lower than expected, particularly among patients with extensive colitis (Broström O, 1986).[2]

Patients with ulcerative colitis (UC) have a higher risk of developing colorectal cancer (CRC). Both high-grade dysplasia (HGD) and low-grade dysplasia (LGD) are considered precancerous lesions (Ünal NG et al., 2019).[3]

In inflammatory bowel disease (IBD), fecal diversion is sometimes required, resulting in a defunctionalized segment of bowel. The potential risk of colorectal cancer (CRC) in this diverted segment is often considered a reason for surgical resection. However, the incidence of neoplasia in diverted colorectal segments among patients with IBD has not been well studied (Bettner W et al., 2018).[4]

The risk of colorectal cancer (CRC) and dysplasia in patients with inflammatory bowel disease (IBD) remains debated due to varying estimates across studies. Recent Danish studies suggest that the overall CRC risk in the IBD population is comparable to that of the general population, although certain patient subgroups may have an increased risk (Aalykke C et al., 2015).[5]

Long-standing ulcerative colitis (UC) increases the risk of colorectal cancer, but surveillance is challenging because dysplasia, the main precancerous lesion, is uncommon and often appears endoscopically normal. Aberrant crypt foci (ACF), known precursor lesions in sporadic colorectal cancer, may also play a role in colitis-associated carcinogenesis (Kukitsu T et al.,2008).[6]

Despite substantial research, variability exists in reported risk estimates due to differences in study populations, follow-up duration, surveillance practices, and evolving treatment paradigms. The introduction of immunomodulators and biologic therapies may have altered the natural history of IBD-related colorectal carcinogenesis, necessitating updated evidence synthesis.

Therefore, this systematic review aims to comprehensively evaluate the impact of disease duration, extent of colonic involvement, and inflammatory severity on colorectal cancer risk in patients with inflammatory bowel disease. By synthesizing current evidence, this review seeks to inform risk stratification models, optimize surveillance recommendations, and guide long-term clinical management.

Objectives: The objective of this systematic review is to assess the impact of disease duration, extent of colonic involvement, and inflammatory severity on the risk of colorectal cancer in patients with inflammatory bowel disease (IBD), and to identify high-risk patient groups to support improved surveillance strategies and risk stratification.

MATERIALS AND METHODS

This systematic review was conducted in accordance with the PRISMA guidelines. A comprehensive literature search was performed in electronic databases including PubMed, Scopus, and Web of Science using key words Inflammatory Bowel Disease (IBD), Ulcerative Colitis, Crohn's Disease, Colorectal Neoplasia, Chronic Inflammation, Disease Duration, Pancolitis, Surveillance Colonoscopy. Observational studies (cohort and case–control studies), randomized control trials, review article, meta-analysis evaluating the association between disease duration, disease extent, inflammatory severity, and the risk of colorectal cancer in patients with inflammatory bowel disease were included. Articles without extractable data, lacked clear outcome measures or evaluable results, commentaries, conference abstracts, were excluded.

Titles, abstracts, and full texts were independently screened by two reviewers, with disagreements resolved through discussion. Data extraction and quality assessment were performed independently using standardized tools appropriate to study design. Due to heterogeneity among studies, a qualitative synthesis was conducted.

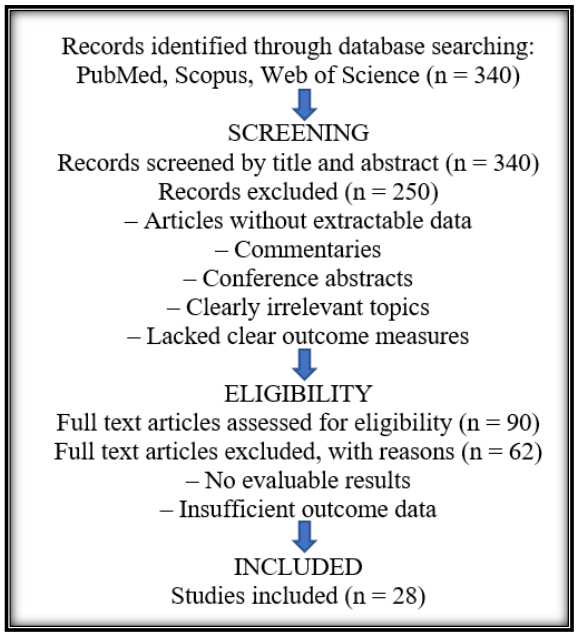

Figure 1: PRISMA flow diagram of study selection process

RESULTS

1. Disease Duration and Colorectal Cancer Risk

Patients with long-standing ulcerative colitis (UC) and Crohn's disease (CD) have a higher risk of colorectal cancer (CRC), which accounts for about 15% of deaths among individuals with inflammatory bowel disease (IBD). Unlike the typical adenoma–carcinoma sequence, CRC in IBD often develops from flat dysplasia or dysplasia-associated lesions. The risk begins to rise approximately 8–10 years after diagnosis, increasing by about 0.5–1% per year. Higher risk is associated with early disease onset, extensive colitis, severe or persistent inflammation, coexisting primary sclerosing cholangitis, and a family history of CRC (Herszenyi L et al., 2007).[7]

The pooled analysis of 116 studies estimated the overall prevalence of colorectal cancer (CRC) in ulcerative colitis (UC) patients at 3.7% (95% Confidence interval [CI]: 3.2–4.2). Based on 41 studies reporting disease duration, the overall incidence was 3 per 1000 person-years. CRC incidence increased with longer disease duration, reaching 2/1000 person-years in the first decade, 7/1000 in the second decade, and 12/1000 in the third decade, corresponding to cumulative risks of 2% at 10 years, 8% at 20 years, and 18% at 30 years (Eaden JA et al., 2001).[8]

A 30-year population-based study (1961–1990) of ulcerative colitis with onset before age 20 identified 32 patients, with an incidence of 0.7 per 100,000 per year. Most patients had extensive disease, but no cases of colorectal cancer were observed. Low-grade dysplasia occurred in some patients without progression during follow-up. Mortality and cancer risk were low, and endoscopic surveillance was preferred over prophylactic colectomy (Ahsgren L et al., 1993).[9]

During follow-up, colorectal cancer (CRC) was diagnosed in 13 patients (13/8564 person-years). The median age at diagnosis was 51 years. Longer disease duration, extensive colitis, primary sclerosing cholangitis, and dysplasia were identified as key risk factors. The cumulative CRC risk was 0.6% at 10 years, 5.4% at 20 years, and 7.5% at 30 years. Detection through surveillance colonoscopy was associated with a trend toward improved survival (Lakatos L et al., 2006).[10]

A retrospective review of the University of Chicago Inflammatory Bowel Disease Registry (1994–2004) evaluated surveillance colonoscopies in patients with ulcerative colitis (UC). Among 622 patients undergoing 1339 examinations, 46 were diagnosed with dysplasia or colorectal cancer at a median age of 48 years and a median disease duration of 20 years. Most lesions were endoscopically visible, with sensitivities of 71.8% for dysplasia and 100% for cancer (Rubin DT et al., 2007).[11]

A case–control study of patients with long-standing extensive ulcerative colitis compared 68 patients with colorectal neoplasia to 136 matched controls. Matching was performed for sex, colitis extent, age at onset, disease duration, and year of surveillance colonoscopy. No other factors reached statistical significance. Analysis showed a strong association between the severity of colonic inflammation and the risk of neoplasia. Histological inflammation remained the only significant independent predictor on multivariate analysis (Rutter M et al., 2004).[12]

Table 1: Summary of Studies Evaluating the Impact of Disease Duration on Colorectal Cancer Risk in Inflammatory Bowel Disease

Author	Key Findings
Herszenyi L et al., 2007	Patients with long-standing ulcerative colitis (UC) and Crohn's disease (CD) have a higher risk of colorectal cancer (CRC). The risk begins to rise approximately 8–10 years after diagnosis, increasing by about 0.5–1% per year.
Eaden JA et al., 2001	The pooled analysis of 116 studies estimated the overall prevalence of CRC in UC patients at 3.7%. Incidence increased with disease duration with cumulative risks of 2% at 10 years, 8% at 20 years, and 18% at 30 years.
Ahsgren L et al., 1993	A 30-year population-based study of UC with onset before age 20 identified 32 patients with no cases of colorectal cancer observed. Mortality and cancer risk were low and endoscopic surveillance was preferred.
Lakatos L et al., 2006	CRC was diagnosed in 13 patients (13/8564 person-years). Longer disease duration, extensive colitis, primary sclerosing cholangitis, and dysplasia were identified as key risk factors.
Rubin DT et al., 2007	Among 622 patients undergoing 1339 surveillance colonoscopies, 46 were diagnosed with dysplasia or colorectal cancer. Most lesions were endoscopically visible with sensitivities of 71.8% for dysplasia and 100% for cancer.
Rutter M et al., 2004	No other factors reached statistical significance. A strong association was observed between the severity of colonic inflammation and the risk of neoplasia. Histological inflammation remained the only significant independent predictor.

2. Extent of Disease and Colorectal Cancer Risk

In this cohort, 92 cases of colorectal cancer were identified in 91 patients, indicating a significantly higher incidence than expected (Standardized Incidence Ratio [SIR] 5.7; 95% CI 4.6–7.0). The risk increased with disease extent: ulcerative proctitis (Standardized Incidence Ratio [SIR] 1.7), left-sided colitis (SIR 2.8), and pancolitis (SIR 14.8). Age at diagnosis and disease extent were independent risk factors, with older age associated with a lower risk (adjusted SIR 0.51). After 35 years, the cumulative colorectal cancer risk was 30% in patients with pancolitis and 40% in those diagnosed before 15 years of age (Ekbom A et al., 1990).[1]

Colorectal cancer risk was not increased overall in ulcerative colitis (SIR 1.1), but it was higher in patients with extensive colitis (SIR 2.4) (Jess T et al., 2006).[13]

Ulcerative colitis is associated with an increased risk of colorectal cancer, estimated at 2% after 10 years, 8% after 20 years, and 18% after 30 years of disease. However, recent studies suggest this risk has declined, with an annual incidence of 0.06–0.16%. Key risk factors include longer disease duration, extensive colitis, primary sclerosing cholangitis, family history of colorectal cancer, severe inflammation, and early disease onset (Lakatos PL et al., 2008).[14]

Inflammatory bowel disease is associated with an increased risk of colorectal cancer, accounting for 10–15% of deaths in affected patients. Key risk factors include longer disease duration, extensive and severe colitis, primary sclerosing cholangitis, and a family history of colorectal cancer. Surveillance colonoscopy is recommended 8–10 years after diagnosis, with newer endoscopic techniques improving dysplasia detection (Dyson JK et al., 2012).[15]

Patients with inflammatory bowel disease have an increased risk of colorectal cancer, particularly with longer disease duration, extensive colitis, family history of colorectal cancer, primary sclerosing cholangitis, and persistent inflammation. Chronic inflammation and genetic alterations contribute to carcinogenesis (Kim ER et al., 2014).[16]

Among 532 ulcerative colitis patients, colorectal cancer developed in 0.94% and high-grade dysplasia in 0.19%. No cases occurred within the first 10 years, but risk increased thereafter, particularly in pancolitis. Disease duration beyond 10 years and extensive colitis were significant risk factors, and overall CRC risk in Indian patients appeared lower than that reported in Western populations (Venkataraman S et al., 2005).[17]

Table 2: Summary of Studies Evaluating Disease Extent and Risk of Colorectal Cancer in Inflammatory Bowel Disease

Author	Key Findings
Ekbom A et al., 1990	92 cases of colorectal cancer identified (SIR 5.7). Risk increased with disease extent: ulcerative proctitis (SIR 1.7), left-sided colitis (SIR 2.8), pancolitis (SIR 14.8); cumulative CRC risk after 35 years was 30% in pancolitis.
Jess T et al., 2006	Colorectal cancer risk was not increased overall in ulcerative colitis (SIR 1.1), but higher in patients with extensive colitis (SIR 2.4).
Lakatos PL et al., 2008	CRC risk estimated at 2% after 10 years, 8% after 20 years, and 18% after 30 years; key risk factors include longer disease duration, extensive colitis, primary sclerosing cholangitis, family history of CRC, severe inflammation, and early disease onset.
Dyson JK et al., 2012	IBD associated with increased CRC risk; key risk factors include longer disease duration, extensive and severe colitis, primary sclerosing cholangitis, and family history of CRC. Surveillance colonoscopy recommended 8–10 years after diagnosis.
Kim ER et al., 2014	Increased CRC risk particularly with longer disease duration, extensive colitis, family history of CRC, primary sclerosing cholangitis, and persistent inflammation.
Venkataraman S et al., 2005	Among 532 UC patients, CRC developed in 0.94%; no cases within first 10 years, risk increased thereafter particularly in pancolitis; disease duration beyond 10 years and extensive colitis were significant risk factors.

3. Inflammatory Severity and Colorectal Cancer Risk: Persistent and severe colonic inflammation in patients with long-standing extensive ulcerative colitis is strongly associated with an increased risk of colorectal neoplasia. Therefore, careful endoscopic and histological assessment of inflammatory activity may help improve risk stratification and guide surveillance strategies (Rutter M et al., 2004).[12]

In the retrospective case–control study by Jegadeesan R et al., 2016, evaluated patients with ulcerative colitis (UC) who underwent surveillance colonoscopy between 1998 and 2011, comparing 111 patients with colitis-associated neoplasia to 356 matched UC controls without neoplasia. Univariate analysis showed that male sex and smoking history were associated with a higher risk of neoplasia, while colonoscopic features such as scarring, strictures, inflammatory polyps, or severe inflammation were not significantly associated. In multivariate analysis, male sex remained an independent risk factor, whereas the use of 5-aminosalicylates was associated with a reduced risk.[18]

In patients with ulcerative colitis, persistent mucosal inflammation during follow-up is linked to a higher risk of colorectal neoplasia compared with those who achieve mucosal healing. This factor should be considered when determining appropriate surveillance intervals (Flores BM et al., 2017).[19]

A cohort of 418 patients with ulcerative colitis (UC) undergoing surveillance colonoscopy was analyzed to examine the association between histologic inflammation and neoplasia risk. Inflammation was graded using a standardized histologic activity index and evaluated as time-varying measures. During

follow-up, 15 patients developed advanced neoplasia and 65 developed any neoplasia. Higher levels of microscopic inflammation over time were significantly associated with progression to advanced colorectal neoplasia, identifying persistent histologic inflammation as an independent risk factor in long-standing UC (Gupta RB et al., 2007).[20]

In retrospective study by Choi CR et al., 2019, evaluated 987 patients with extensive ulcerative colitis undergoing surveillance colonoscopy between 2003 and 2012. Over a median follow-up of 13 years, 97 patients developed colorectal neoplasia. Higher cumulative inflammatory burden over time was significantly associated with increased neoplasia risk, whereas inflammation severity from a single colonoscopy was not predictive, highlighting the importance of assessing long-term inflammatory activity for risk stratification.[21]

In a 5-year registry study of 773 patients with ulcerative colitis, 55 developed colorectal neoplasia. Higher inflammatory markers and lower albumin levels were observed in affected patients. Male sex, longer disease duration, extensive colitis, primary sclerosing cholangitis, and an elevated C-reactive protein (CRP)–albumin score were independently associated with increased neoplasia risk, indicating that persistent systemic inflammation may contribute to colorectal neoplasia in UC (Koutroubakis IE et al., 2016).[22]

In the case–control study by Rubin DT et al., 2013, studied 59 patients with ulcerative colitis (UC) who developed colorectal neoplasia (CRN) were compared with 141 matched UC controls without CRN. Histologic inflammatory activity from biopsy samples was assessed using a 6-point scoring system. Higher levels of microscopic inflammation were significantly associated with an increased risk of CRN, while the use of immunomodulators was linked to a reduced risk. These findings suggest that persistent inflammation contributes to neoplasia development in UC, and immunomodulatory therapy may have protective effects.[23]

In the retrospective study by Ünal NG et al.,2019, they analyzed 801 patients with ulcerative colitis from an inflammatory bowel disease database between 1993 and 2016. Over a total follow-up of 5334 person-years, the prevalence of colorectal cancer (CRC) was 0.7% and dysplasia 0.85%. The cumulative CRC risk increased with disease duration, reaching 5.9% at 30 years. Primary sclerosing cholangitis was identified as a significant independent risk factor for CRC, highlighting the importance of risk-based surveillance strategies in UC patients.[3]

In a 20-year surveillance study of 143 patients with long-standing ulcerative colitis undergoing regular colonoscopy and biopsies, 51 developed colorectal dysplasia or cancer. Patients with primary sclerosing cholangitis had a significantly higher risk of neoplasia, with most lesions located in the proximal colon. Sulfasalazine therapy did not demonstrate a protective effect (Lindberg BU et al., 2001).[24]

This nationwide cohort study from Denmark and Sweden identified patients with Crohn's disease using national patient and pathology registers and compared them with matched population controls. Cox regression analysis was used to evaluate the risk of colorectal cancer (CRC) incidence and mortality. The study found that patients with Crohn's disease had a higher risk of CRC diagnosis and CRC-related death, with particularly increased risk among those diagnosed before age 40, with colonic involvement, or with primary sclerosing cholangitis (Olén O et al., 2020).[25]

Table 3: Summary of Studies Evaluating Inflammatory Activity and Risk of Colorectal Neoplasia in Inflammatory Bowel Disease

Author	Key Findings
Rutter M et al., 2004	Persistent and severe colonic inflammation in long-standing extensive ulcerative colitis is strongly associated with an increased risk of colorectal neoplasia.
Jegadeesan R et al., 2016	Male sex remained an independent risk factor for neoplasia, whereas the use of 5-aminosalicylates was associated with a reduced risk.
Flores BM et al., 2017	Persistent mucosal inflammation during follow-up is linked to a higher risk of colorectal neoplasia compared with patients who achieve mucosal healing.
Gupta RB et al., 2007	Higher levels of microscopic inflammation over time were significantly associated with progression to advanced colorectal neoplasia in long-standing UC.
Choi CR et al., 2019	Higher cumulative inflammatory burden over time was significantly associated with increased neoplasia risk.
Koutroubakis IE et al., 2016	Male sex, longer disease duration, extensive colitis, primary sclerosing cholangitis, and an elevated CRP–albumin score were independently associated with increased neoplasia risk.
Rubin DT et al., 2013	Higher levels of microscopic inflammation were significantly associated with an increased risk of colorectal neoplasia, while the use of immunomodulators was linked to a reduced risk.
Ünal NG et al., 2019	Prevalence of CRC was 0.7% and dysplasia 0.85%; cumulative CRC risk reached 5.9% at 30 years, with primary sclerosing cholangitis identified as a significant independent risk factor.
Lindberg BU et al., 2001	Patients with primary sclerosing cholangitis had a significantly higher risk of neoplasia, with most lesions located in the proximal colon.
Olén O et al., 2020	Patients with Crohn's disease had a higher risk of CRC diagnosis and CRC-related death, particularly increased risk among those diagnosed before age 40, with colonic involvement or primary sclerosing cholangitis.

DISCUSSION

Key risk factors of CRC included longer disease duration, extensive colitis, dysplasia, and primary sclerosing cholangitis (Lakatos L et al., 2006).[10]

Most dysplastic and cancerous lesions in UC are visible during endoscopy and can be detected during routine surveillance colonoscopy (Rubin DT et al., 2007).[11]

In patients with long-standing extensive ulcerative colitis, greater severity of colonic inflammation significantly increases the risk of colorectal neoplasia. Assessing inflammation through endoscopic and histological evaluation may help improve risk stratification in surveillance programs (Rutter M et al., 2004).[12]

Patients diagnosed with pancolitis, particularly those younger than 15 years at the time of diagnosis, may benefit from intensive surveillance, and prophylactic proctocolectomy could be considered as a preventive management option (Ekbom A et al., 1990).[1]

Chronic inflammation is considered the main driver of carcinogenesis. Improved surveillance colonoscopy and maintenance therapies such as aminosalicylates may contribute to the reduced risk, and current guidelines recommend regular endoscopic surveillance and chemopreventive therapy in ulcerative colitis patients (Lakatos PL et al., 2008).[14]

Colonoscopic surveillance, especially chromoendoscopy with targeted biopsies, is important for early detection, while anti-inflammatory therapies may have a chemopreventive role (Kim ER et al., 2014).[16]

Colonoscopic surveillance is essential for early detection, and techniques such as chromoendoscopy may improve dysplasia detection (Mattar MC et al., 2011).[26]

Recent advances have improved understanding of risk factors and management of colorectal dysplasia in inflammatory bowel disease. Identifying high-risk patients, implementing targeted surveillance, and ensuring timely treatment of dysplasia can improve outcomes in IBD-associated colorectal cancer. Continued research is needed to better understand disease mechanisms and develop new biomarkers and preventive strategies (Sato Y et al., 2023).[27]

Overall, standard white-light colonoscopic findings alone were not reliable predictors of neoplasia, suggesting a potential role for advanced imaging techniques and molecular markers to improve early detection (Jegadeesan R et al., 2016).[18]

Overall, primary sclerosing cholangitis emerged as an independent risk factor for colorectal dysplasia or cancer in patients with ulcerative colitis (Lindberg BU et al., 2001).[24]

Patients with inflammatory bowel disease, including ulcerative colitis and colonic Crohn's disease, have an increased risk of colorectal cancer. However, this risk has declined in recent years due to improved disease control, surveillance, and screening strategies. This review summarizes the epidemiology, risk factors, screening, and management of precancerous lesions and colorectal cancer in IBD (Al Sulais E et al., 2025).[28]

CONCLUSION

In conclusion, Patients with IBD, particularly ulcerative colitis, have an increased risk of colorectal cancer, mainly associated with longer disease duration, extensive colitis, severe inflammation, dysplasia, and primary sclerosing cholangitis. Chronic inflammation plays a central role in carcinogenesis, while surveillance colonoscopy—especially with techniques such as chromoendoscopy—improves early detection and risk stratification. Improved disease control, surveillance, and chemopreventive therapies may contribute to the declining CRC risk in recent years. Further studies are needed to identify reliable biomarkers and optimize surveillance and prevention strategies for colorectal cancer in inflammatory bowel disease.

Acknowledgement

The author would like to express their sincere gratitude to all researchers and scholars whose published studies contributed to the development of this systematic review. Author also acknowledges the support of colleagues who provided guidance and valuable insights during the preparation of this manuscript. The author declares no conflicts of interest, and this research received no external funding.

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