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**FRONTIER**

- 63 Peroral cholangioscopy: Update on the state-of-the-art  
*Subhash A, Buxbaum JL, Tabibian JH*

**MINIREVIEWS**

- 77 Exposed endoscopic full-thickness resection for duodenal submucosal tumors: Current status and future perspectives  
*Granata A, Martino A, Zito FP, Ligresti D, Amata M, Lombardi G, Traina M*
- 85 Endoscopic colorectal cancer surveillance in inflammatory bowel disease: Considerations that we must not forget  
*Núñez F P, Quera R, Rubin DT*

**ORIGINAL ARTICLE****Retrospective Study**

- 96 Texture and color enhancement imaging in magnifying endoscopic evaluation of colorectal adenomas  
*Toyoshima O, Nishizawa T, Yoshida S, Yamada T, Odawara N, Matsuno T, Obata M, Kurokawa K, Uekura C, Fujishiro M*

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## Endoscopic colorectal cancer surveillance in inflammatory bowel disease: Considerations that we must not forget

Paulina Núñez F, Rodrigo Quera, David T Rubin

**ORCID number:** Paulina Núñez F 0000-0003-3727-1851; Rodrigo Quera 0000-0001-5854-0526; David T Rubin 0000-0001-5647-1723.

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**Paulina Núñez F, Rodrigo Quera,** Universidad de los Andes, Digestive Disease Center, Inflammatory Bowel Disease Program, Clínica, Santiago 7620157, RM, Chile

**Paulina Núñez F,** Department of Gastroenterology, Hospital San Juan de Dios. Universidad de Chile, Santiago 7701230, RM, Chile

**David T Rubin,** Medicine Inflammatory Bowel Disease Center, University of Chicago, Chicago, IL 60637, United States

**Corresponding author:** David T Rubin, AGAF, FACG, MD, Chief Doctor, Medicine Inflammatory Bowel Disease Center, University of Chicago, 5841 S. Maryland Ave, MC4076, Room M410, Chicago, IL 60637, United States. [drubin@medicine.bsd.uchicago.edu](mailto:drubin@medicine.bsd.uchicago.edu)

### Abstract

Inflammatory bowel disease (IBD), encompassing Crohn's disease and ulcerative colitis, is a chronic immune-mediated inflammatory disease that primarily affects the gastrointestinal tract and is characterized by periods of activity and remission. The inflammatory activity of the disease involving the colon and rectum increases the risk of colorectal cancer (CRC) over the years. Although prevention strategies are evolving, regular surveillance for early detection of neoplasia as a secondary prevention strategy is paramount in the care of IBD patients. In this review article, we discuss the current evidence of the risks of developing CRC and evaluate the best available strategies for screening and surveillance, as well as future opportunities for cancer prevention.

**Key Words:** Inflammatory bowel disease; Endoscopy; Crohn's disease; Ulcerative colitis; Surveillance; Colorectal cancer

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**Core Tip:** Colorectal cancer (CRC) is one of the leading causes of death in inflammatory bowel disease (IBD) today. However, subsequent reports have shown lower rates of CRC. The expanding medical options in IBD have substantially improved our ability to control severe inflammation and likely to reduce the risk of CRC in this setting. We discuss the current evidence of the risks of developing CRC, and evaluate

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## INTRODUCTION

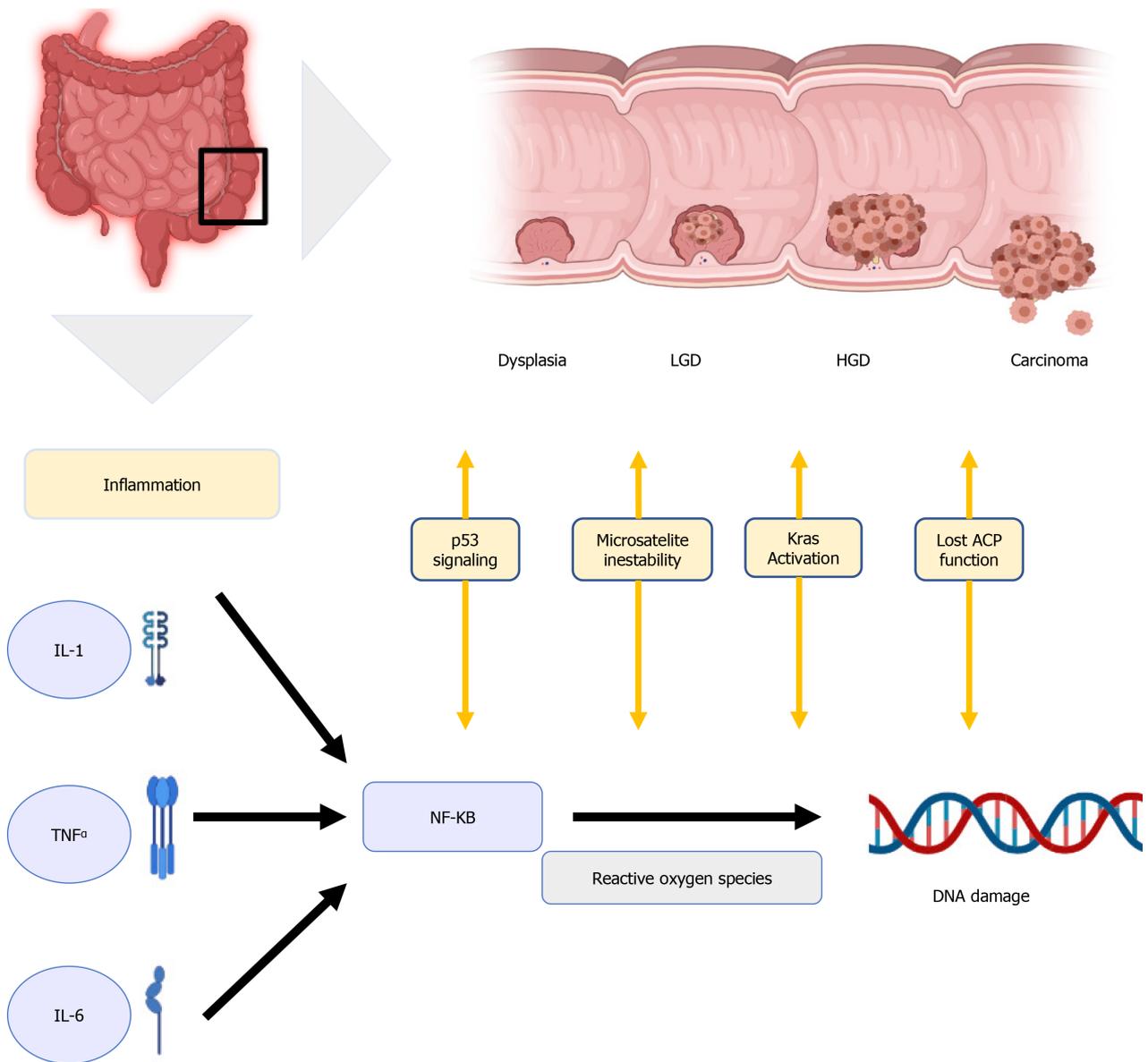
Inflammatory bowel disease (IBD) is a chronic, progressive or relapsing and remitting immune-mediated condition of the intestines[1,2]. While the pathogenesis has not been fully elucidated, it is generally considered a consequence of a dysregulated immune response to environmental triggers in genetically predisposed subjects[3,4]. CRC is a major cause of death in IBD, accounting for 10 to 15% of death in IBD[5,6]. CRC risk increases over time after IBD diagnosis. In ulcerative colitis (UC), a prior meta-analysis estimated the CRC risk to be 2%, 8%, and 18% at 10, 20, and 30 years, respectively, after disease diagnosis[7]. This risk is also higher in patients with long-standing and diffuse colonic CD [relative risk (RR) of 4.5 (95%CI: 1.3-4.9)][8]. However, later reports have shown lower rates of left-sided CRC of 2.5%, 7.6%, and 10.8% at 20, 30, and 40 years after diagnosis, respectively[9]. This lower risk may be explained due to successful CRC surveillance programs and better control of mucosal inflammation from early disease stages[10]. The more recent 40-year surveillance experience in the United Kingdom demonstrated decreasing rates of advanced CRC and interval CRC with cumulative incidences of 0.1%, 6.7%, and 10% in the first, third, and fourth decade after diagnosis, respectively[11]. The reasons for decreasing incidences are thought to reflect effective surveillance, access to surgery, and more effective therapies.

Endoscopic surveillance is the primary recommended CRC prevention strategy, with an active search of early-stage cancer or pre-cancerous (dysplastic) lesions[12]. Endoscopic surveillance has been previously suggested to start 8-10 years after IBD diagnosis based on a historical analysis by Eaden *et al* that showed a CRC risk of 2% 10 years after diagnosis[7]. However, earlier surveillance starting 8 years after diagnosis is modeled to capture an additional 6% of patients developing CRC[13], so newer guidelines embrace this earlier starting time, which may also reflect the emergence of earlier age colorectal cancers described in the population.

Historically, CRC surveillance in patients with IBD has been characterized by extensive four-quadrant non-targeted (random) biopsies to improve the detection of dysplastic mucosa. However, a newer technology that enhances digital mucosal images as high-definition white-light endoscopy (HD-WLE) and dye-assisted chromoendoscopy (CE) with magnification have improved the visualization and detection of early neoplastic lesions, and therefore have increased the diagnostic yield for dysplasia[14,15].

## CRC PATHOGENESIS IN IBD

Although the pathogenesis of IBD-related CRC is believed to be different from the pathogenesis of sporadic CRC and CRC that is associated with polyposis and non-polyposis hereditary syndromes, their molecular pathways are similar[16], involving DNA methylation, microsatellite instability, aneuploidy, activation of oncogene Kras, alteration of COX-2 enzymes, and mutation of tumour suppressor genes, with loss of p53 function[17]. One well-known molecular link between cancer and inflammation is the nuclear factor Kappa B (NF- $\kappa$ B)[18]. It can be activated by pro-inflammatory cytokines like interleukin-1 (IL-1), IL-6, and tumor necrosis factor  $\alpha$  (TNF- $\alpha$ ), ultimately producing reactive oxygen species damaging the DNA and favoring tumor development[19] in Figure 1.



**Figure 1 Physiological mechanism.** IBD: Inflammatory bowel disease; LGD: Low-grade dysplasia; HGD: High-grade dysplasia.

Inflammation plays a central role in carcinogenesis; as a consequence, the severity of flare-ups with accumulated inflammatory damage (persistence of inflammation) predisposes to the development of CCR. Choi *et al* observed that the accumulative inflammatory burden had a 2-fold increase in the risk of CCR, (95%CI: 1.5 to 2.9;  $P < 0.001$  for endoscopic and 95%CI: 1.4 to 3.0;  $P < 0.001$  for histological) for every 10 years of mild, 5 years of moderate or 3.3 years of severe activity disease[20]. The importance of this finding is that it is based not only on the most recent colonoscopy but also on several colonoscopies in a given time to assess the cumulative effect of inflammation. This persistent inflammation mechanism would explain the predominance of right-sided neoplasia that has been described in PSC patients. In a recent study, UC PSC patients who remain in clinical remission have greater endoscopic and histological activity in the right colon compared to UC patients without PSC[21].

Moreover, chronic inflammation may lead to the development of dysplastic changes in colonic mucosa. These changes can be classified as low-grade dysplasia (LGD), high-grade dysplasia (HGD), or indefinite for dysplasia[22]. LGD is characterized by hyperchromatic enlarged nuclei with preserved cell polarity, decreased mucinous differentiation, and dystrophic goblet cells[23,24]. In contrast, HGD presents as atypical cells with prominent nuclear pleomorphism, hyperchromatic stratified nuclei, and loss of cell polarity, and whenever pathologists cannot distinguish between inflammatory-associated and dysplastic changes, the sample is defined as indefinite for dysplasia[23,24]. This should be distinguished from indeterminate findings, which

are usually due to the presence of confounding amounts of histologic inflammation. Given the high inter-observer variability in grading dysplastic changes, guidelines recommend that all cases of suspected dysplasia should be evaluated by two expert pathologists[25,26].

Neoplastic progression can occur multifocally so that dysplasia can be associated with an increased risk of synchronous (simultaneous) or metachronous (six months after diagnosis) dysplasia or carcinoma[25,27].

## RISK FACTORS FOR DYSPLASIA AND CRC

Most relevant CRC risk factors in IBD include longer disease duration, greater disease extent (extensive-pancolitis) and degree of inflammation over time[28,29], family history of CRC[30], personal history of dysplasia or colonic stricture, and diagnosis of primary sclerosing cholangitis (PSC) Table 1[31,32].

Younger age at diagnosis and disease duration have been shown as risk factors for CRC in IBD patients, possibly related to more aggressive phenotypes and longer exposure to mucosal inflammation[33]. A previous meta-analysis showed that patients diagnosed before the age of 30 had a CRC standardized incidence ratio (SIR) of 8.2 (95% CI: 1.8-14.6, I2 82%) compared to patients diagnosed after 30-years-old with an SIR of 1.8 (95% CI: 0.9-2.7, I2 81%)[34]. Also, disease extension in UC has been related to a higher risk of CRC, with SIR of 6.9 (95% CI: 1.9-11.9, I2 84%) for extensive colitis and only 1.7 (CI 95% 0.6-4.5 I2 47%) for left-sided colitis; furthermore, in patients with segmental colitis in CD, there was no higher risk of CRC, with a SIR of 1.7 (95% CI: 0.9-2.6, I2 0%)[35]. There is evidence that IBD patients with a prior family history of CRC have at least a two-fold higher risk of IBD-related CRC (adjusted RR = 2.5; 95% CI: 1.4-4.4); moreover, when CRC family history is associated to first-degree relatives, diagnosed under the age of 50, the risk is even higher (RR = 9.2; 95% CI: 3.7-23)[25,35]. There are some cases of Lynch Syndrome with IBD who develop CRC at a younger age, which are more accelerated and significantly compare with patients without IBD. In this scenario, a colectomy would be necessary due to the high risk of recurrence and multiple CRC[36]. This risk has been seen in UC, and only a few cases in CD, so it does not allow conclusions to be drawn about the risk of CRC[37].

The presence of prior dysplasia or stricture is also associated with an increased risk of neoplasia in IBD[38,39]. Furthermore, colonic strictures in any setting should be considered malignant until proven otherwise.[40] Previous studies have reported variable risk of dysplasia or CRC associated with colonic strictures in UC (from 0% to 86%)[41,42] and there is insufficient data for this risk in CD[43]. Regarding the presence of inflammatory polyps, it is debated if they are related to the development of dysplasia. Historically, case-control studies have reported that patients with inflammatory polyps have 1.9-to-2.5-fold increased risk of CRC[29,44], but recent retrospective cohort studies have suggested that they do not independently predict the development of CRC, nor do they predict progression from LGD to HGD or CRC[20, 45].

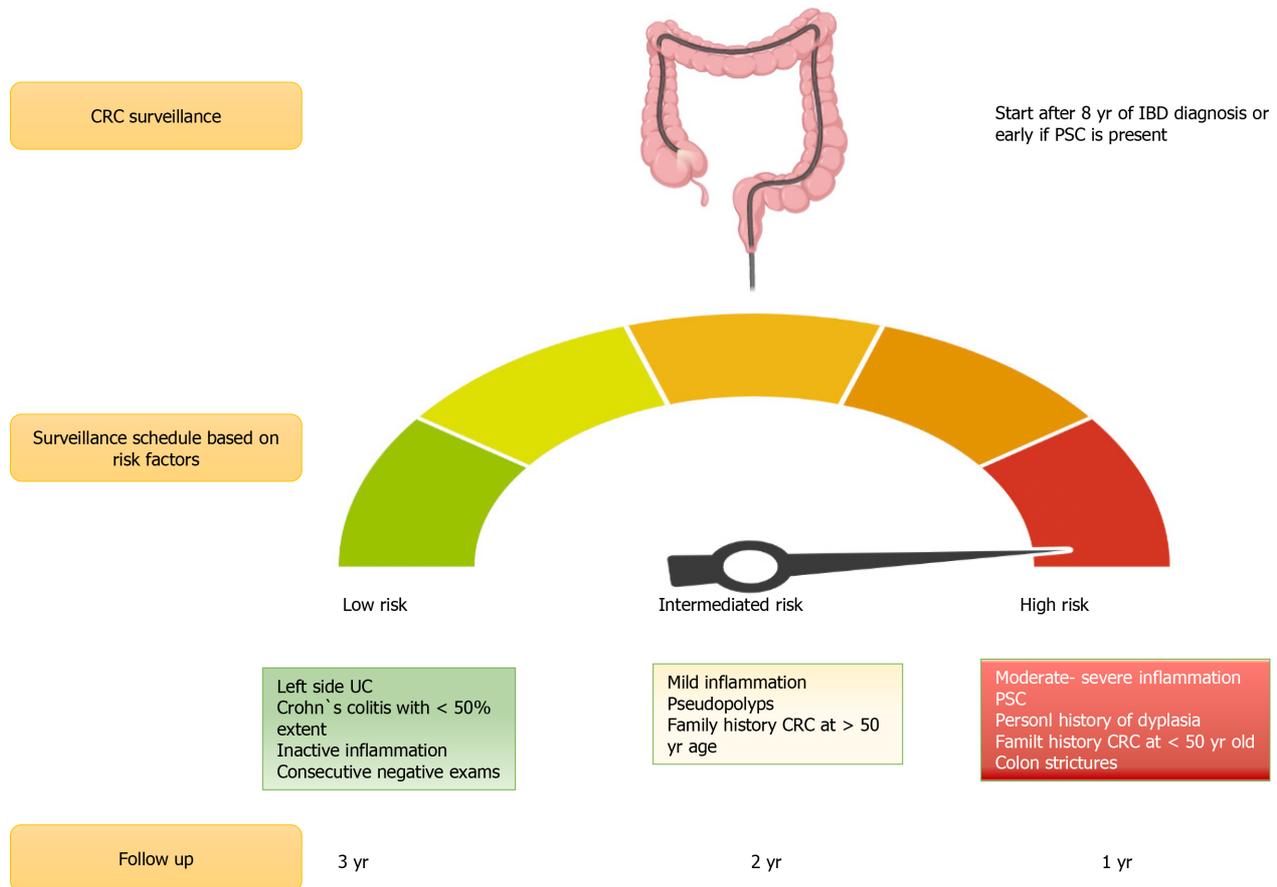
One major risk factor for CRC in IBD is the presence of concomitant PSC. A previous meta-analysis by Soetikno *et al*[46] showed that patients with PSC and UC had a higher risk for development of CRC [odds ratio (OR) of 4.09 (95% CI: 2.89-5.76)]. An observational longitudinal cohort study also reported an increased risk for CRC in patients with PSC and UC compared to patients with UC and no PSC with a SIR of 9.8 (95% CI: 1.9-96.6)[47]. Additionally, patients who are in clinical remission have a higher chance of endoscopic and histological inflammation in the right colon compared to UC patients without PSC, being the place where the CCR is most frequently found[21] in Figure 2.

## CRC SURVEILLANCE IN IBD

Recommendations for CRC surveillance in IBD vary according to the type of IBD, comorbidities, and previous family history of CRC. According to the current SCENIC consensus statements and ACG guidelines, surveillance colonoscopies should start 8 years after diagnosis in patients with left-sided or extensive UC, and in patients with a colonic CD that comprise more than 30% of the colonic surface or > 1 colonic segment [48,49]. Patients with a first-degree family history of CRC should start surveillance colonoscopies 10 years before the age their relative was diagnosed with CRC or 8 years after IBD diagnosis, whichever occurs first[50]. In patients with IBD and PSC,

Table 1 Risk factors	
Clinical risk factors	Endoscopic risk factors
Disease duration, extension, and severity	Active disease
Personal history of dysplasia	Colonic stricture
Primary sclerosing cholangitis	Pseudopolyps (post-inflammatory polyps)
Family history of CRC / dysplasia	Tubular appearance of colon

IBD: Inflammatory bowel disease; UC: Ulcerative colitis; CD: Crohn disease.



**Figure 2 Colorectal cancer risk.** CRC: Colorectal cancer; UC: Ulcerative colitis; CD: Crohn disease; PSC: Primary sclerosing cholangitis.

surveillance colonoscopies should start at diagnosis and be repeated on an annual basis[51]. Surveillance colonoscopy intervals are every 1-3 years, according to each patient risk-stratification[27,52]. Patients with isolated proctitis do not need surveillance colonoscopies[51].

## ENDOSCOPIC TECHNIQUES FOR DETECTION OF DYSPLASIA

Despite the greater surveillance efforts for early detection of CRC in IBD patients, CRC risk remains significant, and the incidence of interval cases may be due to rapid progression and unclear pathogenesis[53]. In order to perform an optimal evaluation of the colonic mucosa, optimum bowel preparation is essential[54,55].

Several advanced imaging techniques have been developed to improve visualization of mucosal defects, enhancing dysplasia and early CRC detection. High-definition white light endoscopy (HD-WLE) has demonstrated higher adenoma detection than standard definition colonoscopy in patients undergoing screening colonoscopy in non-IBD patients[56]. Chromoendoscopy uses optical or computer/bas

-ed techniques to enhance mucosal details in order to improve lesion detection and characterization[57,58]. This technique can be assisted by different dye agents applied as sprays during colonoscopy, which can be classified as contrast agents (i.e., indigo carmine)[59], absorptive agents (i.e., methylene blue), and reactive staining agents (i.e., Congo red); being the first two, the most commonly used[60]. Among dye-less chromoendoscopy, there are different optical CE techniques. Narrow-band imaging (NBI) is a type of optical CE, based in the use of blue-light technology improving characterization of detected lesions, but has shown no further benefit in primary detection of dysplasia when compared to HD-WLE[61]. Unlike NBI, other dye-less CE methods, such as flexible spectral imaging color enhancement (FICE), visualizes mucosal structures without using optical filters but capturing mucosal imaging and performing digital software-based processing of the captured images. The adequate examination requires a clean mucosa, as stools and blood can obscure interpretation of the images. DCE was more effective in identifying dysplasia compared to white light endoscopy (WLE), but without reaching significant differences compared to HD WLE [62]. Recently, a retrospective analysis also showed no differences in the detection of dysplasia with these techniques, but longer examination time using DCE (24.6 min *vs* 15.4,  $P < 0.001$ )[63].

The National Institute for Health and Care Excellence (NICE) and the European Crohn's and Colitis Organization (ECCO) have recommended the routine use of CE with targeted biopsies in IBD-CRC surveillance in their society guidelines[49]. In 2015 an international expert consensus, SCENIC (Surveillance for Colorectal Endoscopic Neoplasia Detection and Management in Inflammatory Bowel Disease Patients: International Consensus) recommended a surveillance study with high-definition colonoscopy or else the use of dye spray chromoendoscopy if a standard definition white-light exam is performed[20]. Prior to HD- WLE, the standard of care for CRC surveillance included four-quadrant non-targeted (random) biopsies every 10 cm from the cecum to the rectum, with a minimum of 32 biopsies, with the goal of detecting "invisible" dysplasia[64]. This technique intended to sample the mucosa in order to identify "invisible" lesions; we now understand that newer imaging technology, if used by experienced endoscopists, has likely made this approach unnecessary in many patients[65].

Virtual chromoendoscopy (VCE) is an optical imaging technique that uses filters to enhance the contrast of both the mucosa and the superficial vasculature, allowing a better evaluation. In a multicenter study with UC patients comparing DCE *vs* NBI, no significant difference was reported between these techniques in detecting neoplastic lesions (OR: 1.02 (95%CI: 0.44-2.35,  $P = 0.964$ )[66]. A recent randomized controlled trial comparing DCE, VCE, and HD-WLE found that both techniques were non-inferior to DCE[67]. The 2019 ACG guidelines recommend the use of DCE or NBI for the surveillance of dysplasia (conditional recommendation, low quality of evidence)[50].

Despite their low yield, random biopsies may have a role when performed in association with CE in IBD patients with a personal history of neoplasia, an appearing tubular colon, or concomitant PSC. A French multicenter study performed quadrant random biopsies every 10 cm in patients with a personal history of neoplasia, showing that 12.8% of neoplasia can be detected[68]. Saravia *et al*[69] consider that random biopsies should be performed when CE is not available or when WLE is used in the presence of inflammation or high-risk factors.

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## NEW TECHNOLOGIES IN CRC DETECTION

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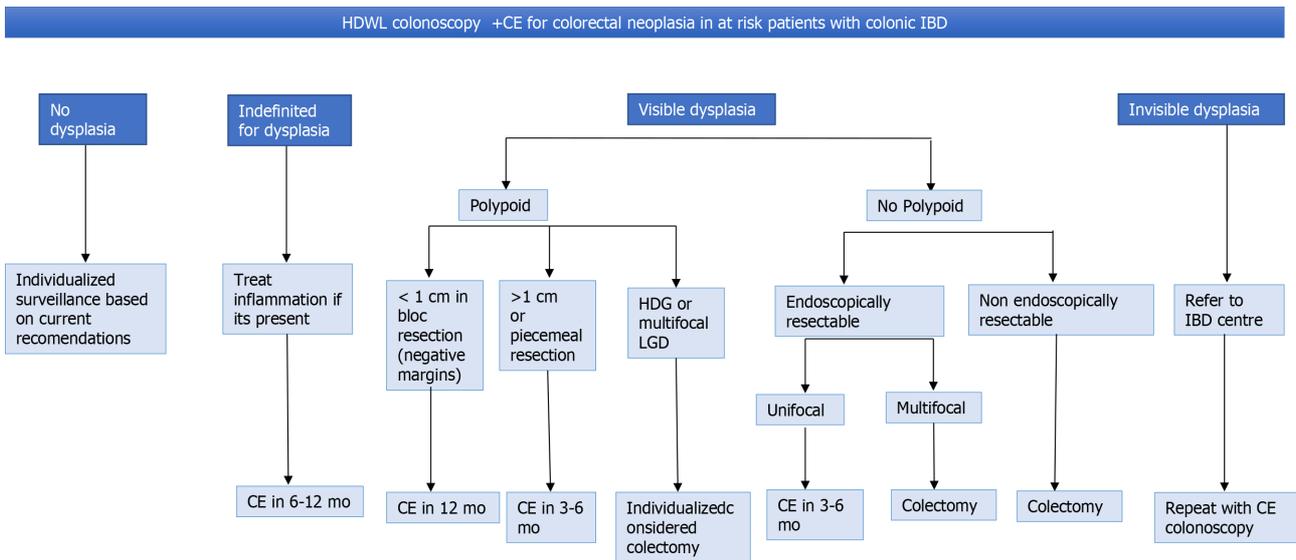
Artificial intelligence (AI) is evolving as a topic of interest in the field of gastrointestinal endoscopy. AI has been used in endoscopic polyp detection; no studies on AI in IBD surveillance have been published so far[70].

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## MANAGEMENT OF DYSPLASIA

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It is important to distinguishing polypoid from non-polypoid lesions, due to their different management, prognosis, and follow-up[71]. A meta-analysis performed by Wanders *et al* showed that patients with polypoid lesions had a lower incidence of CRC compared to patients with non-polypoid lesions, which was attributed to the complete endoscopic resection of the first type of lesions[72].



**Figure 3 Algorithm for the management of dysplasia.** Review all dysplasia with 2 experienced GI pathology. LDG: Low-grade dysplasia; HGD: High-grade dysplasia.

Less than 1 cm polypoid lesions (with negative margins) should be followed up with colonoscopy at 12 mo. For lesions greater than 1 cm or lesions that have been removed piecemeal, surveillance colonoscopy should be performed within 3-6 mo[49]. LGD had a low risk of progression to HGD or CRC from an incomplete resection if it is unifocal. In contrast, multifocal LGD carries substantial risk[73]. The rate of progression from LGD vs HGD to adenocarcinoma was significantly greater for HGD ( $P < 0.001$ )[74]. Although most dysplasias were found in the right colon, being higher in UC, the rate of progression of LGD and HGD dysplasia or adenocarcinoma was not significantly different in CD vs UC[75]. A Dutch nationwide cohort study observed that the cumulative incidence of advanced neoplasia was 21.7% after 15 years of follow-up. Male sex, older age at LDG ( $> 55$  years), and follow-up by a tertiary IBD referral center were independent risk factors for advanced neoplasia[76]. The management of HGD in a visible lesion with complete resection is controversial. The decision should be made case by case between colectomy vs shorter follow-up[77].

In cases of non-polypoid dysplasia, classically, these were sent to colectomy. However, if there is complete resection, it can be followed up instead of colectomy but, always evaluating progression factors[78].

For endoscopically invisible LGD (found only on random biopsy), it should be referred to an IBD Centre or endoscopist with experience at high-risk surveillance. Surveillance endoscopy using CE with HD-WLE is required in an attempt to identify the neoplastic lesion (or others) and to remove it endoscopically[79]. In Figure 3, the management of dysplasia/LGD and HGD is summarized.

## CONCLUSION

It is essential to know which risk factors affect the CRC risk in every IBD patient, allowing to identify the subgroups of patients who need closer surveillance and more intensive treatment. The risk of CRC is increased in IBD but not as high as previously reported. The expanding medical options in IBD have substantially improved our ability to control severe inflammation and likely to reduce the risk of CRC. The advance of new technologies allows us a better characterization of lesions and treat them on time.

Prospective studies to monitor the rate of interval cancer, the cost-effectiveness of surveillance programs are needed.

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