CME

Update on Endoscopic Dysplasia Surveillance in Inflammatory Bowel Disease

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As medical management of inflammatory bowel disease makes great advances, most patients with inflammatory bowel disease will have long life expectancies without need for total colectomy. With prolonged disease duration, however, there is increased risk of dysplasia leading to colorectal cancer. Multiple consensus and guideline documents have been published over the last decade with recommendations to optimize early detection and management of dysplastic lesions. Endoscopic technology has improved tremendously, even over the past few years. Previously invisible dysplasia has become visible in most cases with advanced imaging technologies that now allow for much clearer and more detailed mucosal inspection. New tools to facilitate endoscopic resection of visible lesions have also enabled patients to avoid colectomy, with resulting need to continue colon surveillance. There are limited or conflicting data leading to inconsistent recommendations regarding the need for random biopsies, the preferred endoscopic imaging technique, and surveillance intervals after resection of dysplasia. Similarly, there remains significant variability in the application of guidelines into daily practice and availability of and training with advanced imaging technologies. Here, we present a narrative review of which patients are at highest risk for dysplasia, the current guidelines on surveillance colonoscopy, factors affecting optimal mucosal visualization, enhanced imaging techniques, standardized reporting terminologies for surveillance colonoscopy, endoscopic management of dysplasia, indications for colectomy, and briefly on future potential technologies to assist in dysplasia detection.

KEYWORDS: inflammatory bowel disease; dysplasia; surveillance

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INTRODUCTION

Inflammatory bowel diseases (IBDs), including Crohn disease, ulcerative colitis, and IBD unclassified, are chronic inflammatory conditions known to result in an increased risk of colorectal cancer (CRC) (1–3). Older studies have shown a cumulative risk of CRC of 2% after 10 years, 8% at 20 years, and 18% at 30 years of disease duration (4). A more recent umbrella meta-analysis reported a relative risk (RR) of 1.69–2.30 for colon and rectal cancer in ulcerative colitis and Crohn disease (5). The increased risk of CRC is believed to result from active and ongoing inflammation leading to greater opportunity to genetic mutations (6).

The incidence of CRC in patients with IBD has steadily decreased over the last 2 decades (3,7–11). This is likely due to several factors. Significant advances in medical therapy of IBD have resulted in much higher rates of disease healing—both endoscopically and histologically. This results in a decreased rate of mucosal epithelial cell turnover leading to lower rates of dysplastic changes. Second, the development and implementation of guidelines recommending surveillance colonoscopy at shorter intervals than the general population has resulted in increased detection of precancerous dysplastic lesions (12). The introduction of advanced imaging techniques such as dye-based and virtual chromoendoscopy (CE) to further delineate morphological characteristics of the mucosa has been shown with strong evidence to increase dysplasia detection—by making the previously invisible lesions visible. Finally, there have been tremendous enhancements in the optical capability of colonoscopy (i.e., high definition [HD] imaging) which results in improved ability to identify and remove precancerous dysplastic lesions. All of these have resulted in increased identification of dysplastic lesions in the colon, before they can progress to CRC, resulting in decreased incidence of CRC. However, CRC in IBD continues to portend worse survival when it is detected as an interval cancer (11). This highlights the importance of a high quality surveillance colonoscopy in IBD patients at risk.

This article will aim to discuss which patients are at highest risk for dysplasia, the current guidelines on surveillance colonoscopy, factors affecting optimal mucosal visualization, enhanced imaging techniques, standardized reporting terminologies for surveillance colonoscopy, endoscopic management of dysplasia, indications for colectomy, and briefly on future potential technologies to assist in dysplasia detection.

RISK FACTORS FOR DYSPLASIA IN IBD

Male sex, young age at disease onset, disease duration more than 8–10 years, and extensive colonic disease are well-characterized risk factors for dysplasia in IBD (4,5,9,10,13,14). In a systematic review of population-based cohorts, Fumery et al (15) described a

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nearly linear increase in risk of CRC with prolonged ulcerative colitis duration. They also found an increased risk with extent of colonic inflammation—no increased risk with proctitis alone, 1.4-fold to 2.8-fold increased risk with left-sided colitis and 2.4-fold to 14.8-fold increased risk with pancolitis. Ongoing active inflammation has also been shown to be associated with future dysplasia (16–18). Similarly, scarred and shortened or tubular appearing colon after longstanding colitis has been associated with increased odds of dysplasia (19).

Primary sclerosing cholangitis (PSC), which occurs in about 2%–5% of patients with IBD, is another risk factor for CRC in IBD (20). In a population-based study, patients with PSC-IBD were shown to have a 4-fold increased risk of CRC relative to patient with IBD alone if diagnosed before age 40 (21). Multiple other studies have confirmed this increased risk, especially in patients with an early age of IBD diagnosis (22–26). As with sporadic CRC, a family history of colon cancer in a first degree relative has also been shown to increase the risk of colitis-associated CRC (27,28).

Postinflammatory or pseudopolyps were previously believed to increase CRC risk, but recent cohorts have refuted this (29,30). A large multicenter study confirmed their presence does not independently increase the risk of advanced dysplasia over a median of 4.8 years, although the colectomy rate was high (31). Colonic strictures are another area where the published literature on the risk of malignancy provides contradictory results. Most, but not all, studies report an increased risk of CRC in ulcerative colitis with strictures (12,32–36). In general, strictures should be examined closely and biopsied during surveillance.

SURVEILLANCE GUIDELINES

There are multiple guidelines and recommendations put forth on timing and performance of surveillance colonoscopy in IBD (37–43). A general theme of the guidance documents is that surveillance colonoscopy should start approximately 8 years from disease onset in patients with ulcerative colitis that extends beyond the rectosigmoid colon and Crohn disease with colonic involvement of at least one-third of the colon. In patients with concomitant PSC, surveillance should start at the time of PSC diagnosis. Most guidelines do not make separate recommendations for patients who are diagnosed beyond 50 years of age. However, some data suggest an earlier increased risk in this population, perhaps due to a longer asymptomatic period or merely the co-occurrence of sporadic CRC (44). As such, consideration could be given to earlier initiation of surveillance in patients diagnosed after age 50.

The outcome of IBD surveillance colonoscopy is affected by the quality of the examination (45). In a Dutch case-control study, delayed surveillance intervals and active inflammation were associated with advanced neoplasia, whereas colonoscopies compliant with quality indicators such as cecal intubation and adequate bowel preparation were associated with reduced risk (45). Factors that have been proposed as markers of high-quality IBD surveillance colonoscopy include bowel preparation, mucosal disease activity, endoscopist's experience, type of endoscope, and whether or not enhanced imaging was used (46). An adequate bowel preparation is most important for IBD-related dysplastic lesions because these are often flat and can be hard to discern with surrounding stool. Also, if CE is required, the presence of residual stool causes significant interference and renders the advanced imaging less effective. Both low and high volume polyethylene glycol preparations have been shown to be effective, and a recent study recommended a clear liquid diet the day before the procedure increased the success of dye-based CE (47,48).

Active mucosal inflammation can result in missing subtle dysplastic lesions, obscuring of the margins of a lesion, and increased difficulty differentiating some dysplastic features from active or regenerative inflammatory features on histopathology (49,50). Ideally, surveillance should be performed when disease is quiescent and repeated at a close interval if active and therapy is amended to achieve mucosal healing. However, surveillance should not be delayed past recommended intervals if mucosal healing is difficult to achieve, and as discussed below, segmental random biopsies may be particularly helpful in this population to detect dysplasia.

COLONOSCOPIC TECHNIQUES

Various meta-analyses have confirmed the inferiority of standard definition (SD) colonoscopy compared with HD colonoscopy in dysplasia detection in IBD (51–54). The SCENIC guidelines published in 2015 were the first to recommend HD over SD colonoscopy (40,55). Since then, multiple international guidelines have reiterated this (37,38,41,42,56). CE is the technique of either topical application of stains or pigments (dye) or utilization of light filters (virtual) to enhance visualization of mucosal surface patterns and identification of dysplasia. Figure 1. The original SCENIC guidelines recommended dye-based CE be preferred over both HD and SD white light endoscopy (WLE). Since that time, there has been published data demonstrating the lack of superiority of various virtual chromoendoscopic (VCE) techniques including narrow band imaging (Olympus, Tokyo, Japan) and iSCAN (iSCAN; Pentax, Tokyo, Japan) to detect dysplasia in IBD. A meta-analysis of 4 randomized controlled studies comparing HD-CE with HD-WLE showed similar detection rate for dysplastic lesions (RR 1.39; 95% confidence interval [CI] 0.95–2.04) (57). When comparing dye-CE with VCE, a meta-analyses by Resende showed no difference between narrow band imaging and HD-WLE in 4 randomized trials (53). A similar meta-analysis by El-Dallal also demonstrated similar rates of dysplasia detection on a per patient basis when comparing VCE with dye-based CE and HD-WLE. On a per dysplastic lesion basis, VCE was inferior to HD-WLE (RR 0.62, 95% CI 0.44–0.88) and VCE was not significantly different from DCE (RR 0.72, 95% CI 0.47–1.11) (58). Three randomized trials have demonstrated no difference in dysplasia detection between HD-WLE and i-SCAN (59–61). There has been only 1 published trial on the use of flexible imaging color enhancement (Fujinon, Tokyo, Japan) (62).

Synthesizing these results is important for putting clinical practice and various guidelines into context. Most clinicians, even those with practices heavily focused on the care of patients with IBD, have continued to use HD-WLE for dysplasia surveillance in most patients, with the availability of spot use of dye-based CE or VCE to better characterize suspicious lesions (63). This is somewhat inconsistent with many professional guidelines that continue to recommend either dye-based or virtual CE with HD colonoscopes be performed in all patients undergoing IBD surveillance, particularly with a history of prior dysplasia (37,38,41–43,64). However, most of the guidelines include an option for the use of HD-WLE, particularly by clinicians who have not developed expertise in dye-based CE (37,39). Even some of the SCENIC consensus statement authors now acknowledge that HD-WLE is an acceptable alternative to dye-based or VCE (41). One might ask what drives the resistance of clinicians to adopt dye-based CE or VCE for all patients. As previously alluded to, training is required to master dye-based CE. However, we speculate that time and reimbursement are the major barriers. Indeed, this likely explains

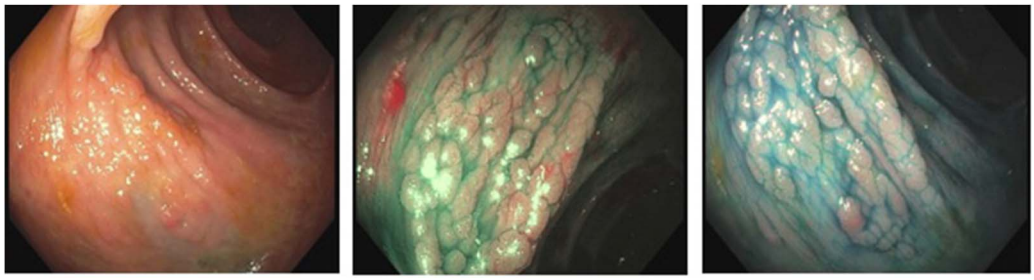

Figure 1. Paris IIa laterally spreading lesion in the ascending colon. Seen first in white light, then on narrow band imaging (after dye-spray), and dye-chromoendoscopy. Endoscopically resected, low grade dysplasia on final pathology.

why many IBD specialists use CE for some patients but not all patients with IBD. Solving the time and reimbursement challenges of CE should be a continued focus of investigation.

RANDOM VS TARGETED BIOPSIES

Before the use of CE, IBD surveillance colonoscopies routinely included random biopsies in 4 quadrants every 10 cm throughout the colon (32 in total) in addition to targeted biopsies of visible lesions. However, there has been conflicting data published on the utility and yield of this strategy primarily due to the wide range of reported dysplasia detected in the random biopsies (Table 1). Watanabe and colleagues performed a small clinical trial in Japan at 52 hospitals in 246 patients, randomly assigned to only targeted or targeted with random biopsies. There was no significant difference in neoplasias detected by each strategy, but the random arm had more biopsies taken and a significantly longer withdrawal time (71). A Canadian cohort of 454 patients examined with either of SD-WLE, HD-WLE, dye-CE, or VCE found targeted biopsies to be sufficient to detect IBD neoplasia in all modalities except SD-WLE (67). In a retrospective analysis of 1000 DCE colonoscopies in the French GETAID study data set, the yield of random biopsies was low at 1.2% per colonoscopy and 0.2% per biopsy but associated with prior neoplasia, tubular colon, and concomitant PSC (68). However, recent retrospective studies from 2 tertiary care IBD centers in the United States, using HD-WLE or dye-CE, provided stronger support for random biopsies. In 1 study, dysplasia was detected in random biopsies in 4.8% of surveillance colonoscopies over 1 year; in the second cohort, 12% of all dysplasia was found with random biopsies. Both showed PSC as the strongest risk factor (69,70).

Many professional organizations have opined on the role of random biopsies in guideline or expert opinion documents. Despite being one of the most common procedures performed for patients with IBD, there are a number of inconsistencies between the recommendations of the SCENIC group, European Crohn's and Colitis Organization, American College of Gastroenterology, British Society of Gastroenterology, and American Gastroenterological Association. These are summarized in Table 2.

SURVEILLANCE AFTER COLON RESECTION

In patients who have undergone subtotal colectomy, it is important to continue surveillance of the rectum, whether it is in continuity or diverted (e.g., Hartman pouch). Surveillance of Hartmann pouch can be more difficult than surveillance of colon in continuity with the fecal stream. Diversion colitis can make dysplasia detection more difficult. Preprocedure bowel cleansing is not possible, which generally makes dye-spray CE infeasible. Moreover, the diverted mucosa is often quite friable, with spontaneous bleeding and a potentially higher risk of complications with biopsies. Although not based on published data, when performing dysplasia surveillance of the Hartmann pouch, the authors of this review generally use HD without dye-based CE with both targeted and random biopsies.

Patients who have undergone total proctocolectomy with ileal pouch anal anastomosis (IPAA) retain a small portion of rectal mucosa where the anastomosis is formed. This mucosa is still at risk to develop CRC. In addition, there is a theoretical risk of developing small bowel cancer within the ileal pouch, although this seems to be exceedingly rare. The overall incidence of neoplasia in the ileal pouch or cuff has been shown in multiple large

Table 1. Summary of studies reporting yield of random biopsies during IBD surveillance colonoscopy

Author (year)	% Colonoscopies with dysplasia on random biopsies	% Colonoscopies with dysplasia only on random biopsies	Type of colonoscopic examination
van den Broek (2014) (65)	1.2	0.5	SD and HD scopes
Mooiweer (2015) (66)	Not reported	1.7	All SD-WLE
Gasia (2016) (67)	0.8 w/HD-WLC	0.8 (0.9 w/non-HD-WLC)	SD-WLE, HD-WLE, VCE, and dye-CE
Moussata (2018) (68)	1.9	1.2	All with SD dye-CE
Coelho-Prabhu (2021) (69)	4.8	Not reported	HD-WLE and dye-CE
Hu (2021) (70)	18	12	SD-WLE, HD-WLE, and dye-CE

CE, chromoendoscopy; HD, high definition; SD, standard definition; VCE, virtual chromoendoscopy; WLC, white light colonoscopy; WLE, white light endoscopy.

Table 2. Summary of professional organization recommendations for dysplasia surveillance in patients with IBD

	Type of examination	Random vs targeted biopsies	Surveillance interval
SCENIC Consensus (2015) (40)	High definition scope recommended If standard definition, then dye-CE recommended If high definition, then dye-CE suggested	No consensus	No recommendations
ECCO Guideline (2017) (38)	High definition recommended Based on local expertise	Random if white light Targeted only if dye-CE	1 yr if high risk (stricture or dysplasia detected within the past 5 yr, PSC, extensive colitis with severe active inflammation) 2–3 yr if intermediate risk (extensive colitis with mild or moderate active inflammation, postinflammatory polyps, or a family h/o CRC in a first degree relative diagnosed ≥ age 50 yr) 5 yr if low risk (none of the above)
ACG Clinical Guideline (2019) (43)	If standard definition, then dye-CE If high definition, then dye-CE or VCE	No recommendation	1–3 yr, based on combination of risk factors and history of dysplasia.
BSG Guideline (2019) (37)	High definition recommended If standard definition, then dye-CE recommended If high definition, then dye-CE suggested Narrow band not suggested	Targeted recommended	1 yr if high risk (as above + family h/o CRC in first degree relative aged <50 yr) 3 yr if intermediate risk (as above) 5 yr if low risk
AGA Clinical Practice update (2021) (42)	High definition scope Dye-CE should be considered in all VCE acceptable alternative if high definition	Random biopsies if white light only and all patients with PSC or h/o dysplasia Targeted biopsies if dye-CE or VCE	1 yr if high risk (as above + h/o invisible or high-risk dysplasia <5 yr, dense pseudopolyps) 2–3 yr if intermediate risk (as above + h/o invisible or high-risk dysplasia >5 yr, lower risk visible dysplasia <5 yr) 5 yr if low risk (continuous disease remission since prior scope, mucosal remission, + ≥2 consecutive examinations without dysplasia, or minimal historical disease extent)
SCENIC commentary (2022) (41)	HD-WLE, dye-CE, VCE all acceptable if endoscopist has training/expertise	Random in highest risk only (PSC, prior dysplasia, atrophic scarred colon, ongoing active inflammation)	Per ACG guideline
ECCO Guideline (2023) (39)	HD-WLE, dye-CE, or VCE	Targeted biopsies Random in high-risk (PSC or h/o dysplasia)	1 yr if high risk (family history of CRC in a first-degree relative ≤50 yr of age, colonic stricture or dysplasia, PSC, extensive colitis with severe active inflammation) 2–3 yr if intermediate risk (extensive colitis with only mild/moderate endoscopic or histologic activity, CRC in first degree family >50 yr) 5 yr if low risk

ACG, American College of Gastroenterology; AGA, American Gastroenterological Association; BSG, British Society of Gastroenterology; CE, chromoendoscopy; CRC, colorectal cancer; ECCO, European Crohn's and Colitis Organization; h/o, history of; HD-WLE, high definition white light endoscopy; NBI, narrow band imaging; PSC, primary sclerosing cholangitis; VCE, virtual chromoendoscopy.

cohorts to be less than 2% up to 15 years after surgery, although certain risk factors have been consistently identified (72–75). In patients with an IPAA, a history of colonic and especially rectal dysplasia in the resected colon, PSC and chronic or atrophic pouchitis have been shown to be risk factors for development of dysplasia in the ileal pouch (75–78). Hence, multiple consensus guidelines recommend these patients undergo annual surveillance (37,39,79,80). The utility and cost-effectiveness of continued surveillance for patients with an

Table 3. Summary of risk factors for colorectal dysplasia in inflammatory bowel disease

Risk factors with strong evidence	Risk factors with moderate evidence
Primary sclerosing cholangitis	Pseudopolyps
Young age at onset	Strictures
Ongoing active inflammation	
Extensive colitis/scarred tubular colon	
Male sex	
Family history of colon cancer	
Prior dysplasia	

IPAA without these risk factors are even less clear. Conceptually, after removal of more than 95% of the colon and rectal mucosa, the risk of CRC should be reduced by more than 95% compared with similar patients with an intact colon. However, published recommendations suggest an examination every 3–5 years, relatively similar to patients with IBD with an intact colon (37,80,81).

REPORTING TERMINOLOGY

Accurate and standardized endoscopic reporting of dysplasia is imperative to appropriate management and follow-up. This should ideally include description of the shape, size, site, surface, and surrounding area of the visible lesion (82). For the shape or morphology, SCENIC guidelines proposed the use of the Paris classification which categorizes lesions as polypoid (pedunculated Ip and sessile Is) when the lesion protrudes ≥2.5 mm into the lumen of nonpolypoid (flat elevated IIa, flat IIb, or flat depressed IIc) (40). Some flat lesions may have a mix of elevated and depressed areas. In addition, it recommends documenting whether the lesions has a distinct border and/or an ulcerated surface. However, the interobserver agreement for the IBD lesions has been shown to be poor with Kappa coefficients of 0.32–0.49 for both morphology and border assessment (83). The most discrepancy in scoring was seen for Is and IIa lesions, suggesting that the difference of 2.5 mm is hard to discern. The Kudo classification for the surface pit pattern has been validated in IBD dysplastic lesions (61,84,85). It categorizes pit patterns into 5 categories (I, II, IIIS, IIIL, IV, and V), of which I and II are benign (86). However, it was validated primarily with magnified images, and routine clinical practice in the United States does not utilize magnification and can be misinterpreted in areas of inflammation-associated regenerative mucosa (87). Another new classification called the Frankfurt Advanced Chromoendoscopic IBD LEsions has been developed with visual endoscopic components of morphology (polypoid vs nonpolypoid), irregular surface pattern and vessel architecture, and signs of inflammation in the lesion (88). It will require further validation and education before routine use. It is also important to describe the mucosa in the background of the visible lesion because background inflammation can obscure the margins of lesions. Validated scoring systems such as the Mayo Endoscopic Score, Ulcerative Colitis Colonoscopic Index of Severity, or the Ulcerative Colitis Endoscopic Index of Severity or the Crohn Disease Endoscopic Index of Severity and the Simple Endoscopic Score for Crohn Disease (89) can be used.

MANAGEMENT OF DYSPLASIA

Invisible dysplasia is dysplasia identified on random, not targeted biopsies. If invisible dysplasia is found at a white light colonoscopy, whether SD or HD, a repeat colonoscopy by an endoscopist experienced in dysplasia surveillance should be performed. Generally this will use HD-WLE with dye-CE particularly if the lesion was not detected using HD colonoscopy (37,38,40–42). Often, these lesions are nonpolypoid and likely to have been missed without enhanced imaging (38,40). This is a situation where random biopsies may be used along with dye-CE. If invisible high-grade dysplasia (HGD) persists after HD-dye-CE examination, referral for colectomy is recommended due to high risk of progression to adenocarcinoma (38,40,42,90). If unifocal low-grade dysplasia (LGD) persists after HD-dye-CE, there is an increasing trend toward following these patients with frequent dysplasia surveillance. Multiple studies have shown the rate of progression from unifocal LGD to adenocarcinoma is low, likely due to improved endoscopic and procedural quality metrics aiding in unmasking and removing what were initially invisible lesions (91–94). However, this requires a thorough risk-benefit discussion with the patient. When a histologic finding of indefinite for dysplasia is obtained on random biopsies, mucosal inflammation should be adequately treated (if possible) and the examination repeated. This is because this histologic finding can mimic reactive atypia as a result of inflammation (95). Indefinite for dysplasia findings is more concerning among patients with PSC or if aneuploidy is found as there is a higher risk of progression to LGD or HGD (33).

For visible dysplasia, there is strong evidence to support endoscopic resection if the lesion has distinct borders. Lesion demarcation can be optimized by the use of concentrated contrast application and enhanced imaging techniques including magnification and endocytoscopy when available (40,41). The goal of resection should be to achieve an en-bloc removal to facilitate histologic assessment of completeness of resection. For lesions smaller than 2 cm, this can be performed by endoscopic mucosal resection. However, for larger lesions, endoscopic submucosal dissection is preferable (96). Two meta-analyses of endoscopic mucosal resection or endoscopic submucosal dissection in IBD dysplasia reported endoscopic success of over 95% with low local and metachronous recurrence (97,98). Submucosal fibrosis is often a complicating feature of dysplastic lesions in IBD due to prior inflammation. This can render endoscopic resection quite technically challenging with reported 6.7% with major bleeding and 2.9% with perforation (99). Biopsies around the resected area were previously believed to be needed to ensure adequate resection. They have not been shown to have a high yield and hence are not recommended (100–102). After adequate endoscopic removal, progression to advanced dysplasia is very rare (103). However, patients with a history of dysplasia are at increased risk for metachronous dysplasia and warrant surveillance, as will be discussed in the next segment (93). The decision of resectability must be rendered by endoscopists with expertise in advanced resection, and this may necessitate referral to a specialty center. For unresectable dysplasia, especially multifocal, surgical resection is recommended.

SURVEILLANCE INTERVALS

A risk-based stratification to optimize surveillance intervals is now recommended by all guidelines (Table 3). Although there are

minor variations in the definitions of categories, they all recommend surveillance intervals ranging from 1 year for those at highest risk to 3–5 years for those at moderate-to-low risk of dysplasia. Although these guidelines aim to provide a framework to guide clinicians, there is wide variability in their application in clinical practice (63).

For surveillance after endoscopic resection of visible dysplasia, the American Gastroenterological Association recommends repeat colonoscopy in 3–6 months for LGD lesions >2 cm, with complex or irregular borders, that were incompletely resected after multiple attempts or for any HGD, at 12 months if the lesion was >1 cm but <2 cm with LGD, and at 24 months for <1 cm or pedunculated lesions with LGD (42). However, there are no tailored recommendations for patients with multifocal resectable dysplasia or those with metachronous resectable dysplasia. These require nuanced discussions with the patient and a multidisciplinary team to offer the optimal care to the patient. For lesions that are unresectable due to size, severe submucosal fibrosis, or cancer, a multidisciplinary discussion with referral to surgery is recommended. The 2022 European Crohn's and Colitis Organization guidelines are similar but recommend shorter intervals for lesions that are resected endoscopically.

THE FUTURE OF DYSPLASIA SURVEILLANCE

Although endoscope fidelity and enhanced imaging techniques continue to improve, alternatives such as colon capsule are also evolving. Capsule imaging is now being used to evaluate Crohn disease, although there are no publications at this time on its application to dysplasia detection in IBD (104). Colon capsule is limited by the lack of biopsy capability, and it has been well documented that histology prediction of IBD lesions from visual images is suboptimal (83,105). Artificial intelligence-based detection algorithms are also an exciting new frontier with the potential to aid endoscopists in detection of IBD dysplasia (105,106). The biggest hurdle to development of a robust system is requirement of a large number of images and videos of all morphologies of IBD dysplasia which will require a collaborative effort.

Despite the excitement about artificial intelligence in endoscopy, this technology is not expected to be widely available for dysplasia surveillance in IBD for quite some time. When and if it becomes commercially available, it may contribute to greater disparities. In the United States, many colonoscopies are still performed with SD scopes. This is likely even more common in less well-resourced countries. As such, it is important that we not forget the lessons that we learned over the course of the past several decades that contributed to a reduction in the incidence of colitis-associated CRC. Perhaps, the most important of these lessons is the need for careful inspection of the colon mucosa. Even for those who are obtaining random biopsies or using CE, careful inspection is essential.

Until the next technological breakthrough that will eliminate the increased risk of dysplasia-associated CRC, there are numerous important questions that remain and would benefit from large collaborative research efforts. Examples of these include the utility of random biopsies when performing HD-WL colonoscopy, the safety of longer surveillance intervals in low-risk populations, the optimal duration of disease before dysplasia surveillance is initiated, and the optimal timing of surveillance exams among patients with IPAA. Enormous progress has been made in the past several decades, but significant work remains to eliminate the increased risk of CRC among patients with IBD.

CONFLICT OF INTEREST STATEMENT

Guarantor of the article: Nayantara Coelho-Prabhu, MBBS, FACG.
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REFERENCES

1. Beaugerie L, Svrcek M, Seksik P, et al. Risk of colorectal high-grade dysplasia and cancer in a prospective observational cohort of patients with inflammatory bowel disease. Gastroenterology 2013;145(1):166–75.e8.
2. Olen O, Erichsen R, Sachs MC, et al. Colorectal cancer in Crohn's disease: A Scandinavian population-based cohort study. Lancet Gastroenterol Hepatol 2020;5(5):475–84.
3. Olen O, Erichsen R, Sachs MC, et al. Colorectal cancer in ulcerative colitis: A Scandinavian population-based cohort study. Lancet 2020;395(10218):123–31.
4. Eaden JA, Abrams KR, Mayberry JF. The risk of colorectal cancer in ulcerative colitis: A meta-analysis. Gut 2001;48(4):526–35.
5. Piovani D, Hassan C, Repici A, et al. Risk of cancer in inflammatory bowel diseases: Umbrella review and reanalysis of meta-analyses. Gastroenterology 2022;163(3):671–84.
6. Itzkowitz SH, Yio X. Inflammation and cancer IV. Colorectal cancer in inflammatory bowel disease: The role of inflammation. Am J Physiol Gastrointest Liver Physiol 2004;287(1):G7–17.
7. Castano-Milla C, Chaparro M, Gisbert JP. Systematic review with meta-analysis: The declining risk of colorectal cancer in ulcerative colitis. Aliment Pharmacol Ther 2014;39(7):645–59.
8. Parragi L, Fournier N, Zeitz J, et al. Colectomy rates in ulcerative colitis are low and decreasing: 10-year follow-up data from the Swiss IBD Cohort Study. J Crohns Colitis 2018;12(7):811–8.
9. Axelrad J, Olen O, Soderling J, et al. Inflammatory bowel disease and risk of colorectal polyps: A nationwide population-based cohort study from Sweden. J Crohns Colitis 2023. [Epub ahead of print]. doi: 10.1093/ecco-jcc/jjad056
10. Jess T, Simonsen J, Jorgensen KT, et al. Decreasing risk of colorectal cancer in patients with inflammatory bowel disease over 30 years. Gastroenterology 2012;143(2):375–81.e1; quiz e13–4.
11. Choi CH, Rutter MD, Askari A, et al. Forty-year analysis of colonoscopic surveillance program for neoplasia in ulcerative colitis: An updated overview. Am J Gastroenterol 2015;110(7):1022–34.
12. Wijnands AM, de Jong ME, Lutgens M, et al. Prognostic factors for advanced colorectal neoplasia in inflammatory bowel disease: Systematic review and meta-analysis. Gastroenterology 2021;160(5):1584–98.
13. von Roon AC, Reese G, Teare J, et al. The risk of cancer in patients with Crohn's disease. Dis Colon Rectum 2007;50(6):839–55.
14. Soderlund S, Granath F, Brostrom O, et al. Inflammatory bowel disease confers a lower risk of colorectal cancer to females than to males. Gastroenterology 2010;138(5):1697–703.
15. Fumery M, Singh S, Dulai PS, et al. Natural history of adult ulcerative colitis in population-based cohorts: A systematic review. Clin Gastroenterol Hepatol 2018;16(3):343–56.e3.
16. Gupta RB, Harpaz N, Itzkowitz S, et al. Histologic inflammation is a risk factor for progression to colorectal neoplasia in ulcerative colitis: A cohort study. Gastroenterology 2007;133(4):1099–105; quiz 340–1.
17. Rubin DT, Huo D, Kinnucan JA, et al. Inflammation is an independent risk factor for colonic neoplasia in patients with ulcerative colitis: A case-control study. Clin Gastroenterol Hepatol 2013;11(12):1601–8, e1–4.
18. Choi CR, Al Bakir I, Ding NJ, et al. Cumulative burden of inflammation predicts colorectal neoplasia risk in ulcerative colitis: A large single-centre study. Gut 2019;68(3):414–22.
19. Rutter MD, Saunders BP, Wilkinson KH, et al. Cancer surveillance in longstanding ulcerative colitis: Endoscopic appearances help predict cancer risk. Gut 2004;53(12):1813–6.
20. Barberio B, Massimi D, Cazzagon N, et al. Prevalence of primary sclerosing cholangitis in patients with inflammatory bowel disease: A systematic review and meta-analysis. Gastroenterology 2021;161(6):1865–77.
21. Trivedi PJ, Crothers H, Mytton J, et al. Effects of primary sclerosing cholangitis on risks of cancer and death in people with inflammatory bowel disease, based on sex, race, and age. Gastroenterology 2020;159(3):915–28.

22. Guerra I, Bujanda L, Castro J, et al. Clinical characteristics, associated malignancies and management of primary sclerosing cholangitis in inflammatory bowel disease patients: A multicentre retrospective cohort study. J Crohns Colitis 2019;13(12):1492–500.
23. Wang R, Leong RW. Primary sclerosing cholangitis as an independent risk factor for colorectal cancer in the context of inflammatory bowel disease: A review of the literature. World J Gastroenterol 2014;20(27): 8783–9.
24. Shah SC, Ten Hove JR, Castaneda D, et al. High risk of advanced colorectal neoplasia in patients with primary sclerosing cholangitis associated with inflammatory bowel disease. Clin Gastroenterol Hepatol 2018;16(7):1106–13.e3.
25. Zhang R, Lauwers GY, Choi WT. Increased risk of non-conventional and invisible dysplasias in patients with primary sclerosing cholangitis and inflammatory bowel disease. J Crohns Colitis 2022;16(12):1825–34.
26. Zheng HH, Jiang XL. Increased risk of colorectal neoplasia in patients with primary sclerosing cholangitis and inflammatory bowel disease: A meta-analysis of 16 observational studies. Eur J Gastroenterol Hepatol 2016;28(4):383–90.
27. Samadder NJ, Valentine JF, Guthery S, et al. Family history associates with increased risk of colorectal cancer in patients with inflammatory bowel diseases. Clin Gastroenterol Hepatol 2019;17(9):1807–13.e1.
28. Najafimehr H, Aghdaei HA, Pourhoseingholi MA, et al. A systematic review and meta-analysis on the association between inflammatory bowel disease family history and colorectal cancer. Gastroenterol Res Pract 2021;2021:4874459.
29. Wolf T, Lewis A, Beaugerie L, et al. Risk of colorectal neoplasia according to histologic disease activity in patients with inflammatory bowel disease and colonic post-inflammatory polyps. Aliment Pharmacol Ther 2023; 57(12):1445–52.
30. Shah SC, Itzkowitz SH. Management of inflammatory bowel disease-associated dysplasia in the modern era. Gastrointest Endosc Clin N Am 2019;29(3):531–48.
31. Mahmoud R, Shah SC, Ten Hove JR, et al. No association between pseudopolyps and colorectal neoplasia in patients with inflammatory bowel diseases. Gastroenterology 2019;156(5):1333–44.e3.
32. Cremer A, Demetter P, De Vos M, et al. Risk of development of more-advanced lesions in patients with inflammatory bowel diseases and dysplasia. Clin Gastroenterol Hepatol 2020;18(7):1528–36.e5.
33. Wan J, Wang X, Zhang Y, et al. Systematic review with meta-analysis: Incidence and factors for progression to advanced neoplasia in inflammatory bowel disease patients with indefinite and low-grade dysplasia. Aliment Pharmacol Ther 2022;55(6):632–44.
34. Laurain PA, Guillo L, D'Amico F, et al. Incidence of and risk factors for colorectal strictures in ulcerative colitis: A multicenter study. Clin Gastroenterol Hepatol 2021;19(9):1899–905.e1.
35. Fumery M, Pineton de Chambrun G, Stefanescu C, et al. Detection of dysplasia or cancer in 3.5% of patients with inflammatory bowel disease and colonic strictures. Clin Gastroenterol Hepatol 2015;13(10):1770–5.
36. Axelrad JE, Faye A, Slaughter JC, et al. Colorectal strictures in patients with inflammatory bowel disease do not independently predict colorectal neoplasia. Inflamm Bowel Dis 2022;28(6):855–61.
37. Lamb CA, Kennedy NA, Raine T, et al. British Society of Gastroenterology consensus guidelines on the management of inflammatory bowel disease in adults. Gut 2019;68(Suppl 3):S1–106.
38. Magro F, Gionchetti P, Eliakim R, et al. Third European Evidence-based Consensus on Diagnosis and Management of Ulcerative Colitis. Part 1: Definitions, diagnosis, extra-intestinal manifestations, pregnancy, cancer surveillance, surgery, and ileo-anal pouch disorders. J Crohns Colitis 2017;11(6):649–70.
39. Gordon H, Biancone L, Fiorino G, et al. ECCO guidelines on inflammatory bowel disease and malignancies. J Crohns Colitis 2022; 17(6):827–54.
40. Laine L, Kaltenbach T, Barkun A, et al. SCENIC international consensus statement on surveillance and management of dysplasia in inflammatory bowel disease. Gastroenterology 2015;148(3):639–51.e28.
41. Rabinowitz LG, Kumta NA, Marion JF. Beyond the SCENIC route: Updates in chromoendoscopy and dysplasia screening in patients with inflammatory bowel disease. Gastrointest Endosc 2022;95(1):30–7.
42. Murthy SK, Feuerstein JD, Nguyen GC, et al. AGA clinical practice update on endoscopic surveillance and management of colorectal dysplasia in inflammatory bowel diseases: Expert review. Gastroenterology 2021;161(3):1043–51.e4.
43. Rubin DT, Ananthakrishnan AN, Siegel CA, et al. ACG clinical guideline: Ulcerative colitis in adults. Am J Gastroenterol 2019;114(3): 384–413.
44. Ekbom A, Helmick C, Zack M, et al. Ulcerative colitis and colorectal cancer. A population-based study. N Engl J Med 1990;323(18):1228–33.
45. Te Groen M, Derks M, den Broeder N, et al. Quality of surveillance impacts the colitis-associated advanced neoplasia risk: A multicenter case-control study. Clin Gastroenterol Hepatol 2022. [Epub ahead of print]. doi: 10.1016/j.cgh.2022.12.010
46. Iacucci M, Cannatelli R, Tontini GE, et al. Improving the quality of surveillance colonoscopy in inflammatory bowel disease. Lancet Gastroenterol Hepatol 2019;4(12):971–83.
47. Megna B, Weiss J, Ley D, et al. Clear liquid diet before bowel preparation predicts successful chromoendoscopy in patients with inflammatory bowel disease. Gastrointest Endosc 2019;89(2):373–9.e2.
48. Restellini S, Kherad O, Bessissow T, et al. Systematic review and meta-analysis of colon cleansing preparations in patients with inflammatory bowel disease. World J Gastroenterol 2017;23(32):5994–6002.
49. Rutter MD. Importance of nonpolypoid (flat and depressed) colorectal neoplasms in screening for CRC in patients with IBD. Gastrointest Endosc Clin N Am 2014;24(3):327–35.
50. Kawachi H. Histopathological diagnosis of ulcerative colitis-associated neoplasia. Dig Endosc 2019;31(Suppl 1):31–5.
51. Feuerstein JD, Rakowsky S, Sattler L, et al. Meta-analysis of dye-based chromoendoscopy compared with standard- and high-definition white-light endoscopy in patients with inflammatory bowel disease at increased risk of colon cancer. Gastrointest Endosc 2019;90(2): 186–95.e1.
52. Gondal B, Haider H, Komaki Y, et al. Efficacy of various endoscopic modalities in detecting dysplasia in ulcerative colitis: A systematic review and network meta-analysis. World J Gastrointest Endosc 2020; 12(5):159–71.
53. Resende RH, Ribeiro IB, de Moura DTH, et al. Surveillance in inflammatory bowel disease: Is chromoendoscopy the only way to go? A systematic review and meta-analysis of randomized clinical trials. Endosc Int Open 2020;8(5):E578–90.
54. Iannone A, Ruospo M, Palmer SC, et al. Systematic review with network meta-analysis: Endoscopic techniques for dysplasia surveillance in inflammatory bowel disease. Aliment Pharmacol Ther 2019;50(8): 858–71.
55. Subramanian V, Ramappa V, Telakis E, et al. Comparison of high definition with standard white light endoscopy for detection of dysplastic lesions during surveillance colonoscopy in patients with colonic inflammatory bowel disease. Inflamm Bowel Dis 2013;19(2): 350–5.
56. Cairns SR, Scholefield JH, Steele RJ, et al. Guidelines for colorectal cancer screening and surveillance in moderate and high risk groups (update from 2002). Gut 2010;59(5):666–89.
57. Na SY, Moon W. Recent advances in surveillance colonoscopy for dysplasia in inflammatory bowel disease. Clin Endosc 2022;55(6): 726–35.
58. El-Dallal M, Chen Y, Lin Q, et al. Meta-analysis of virtual-based chromoendoscopy compared with dye-spraying chromoendoscopy standard and high-definition white light endoscopy in patients with inflammatory bowel disease at increased risk of colon cancer. Inflamm Bowel Dis 2020;26(9):1319–29.
59. Kandiah K, Subramaniam S, Thayalasekaran S, et al. Multicentre randomised controlled trial on virtual chromoendoscopy in the detection of neoplasia during colitis surveillance high-definition colonoscopy (the VIRTUOSO trial). Gut 2021;70(9):1684–90.
60. González-Bernardo O, Riestra S, Vivas S, et al. Chromoendoscopy with indigo carmine vs virtual chromoendoscopy (iSCAN 1) for neoplasia screening in patients with inflammatory bowel disease: A prospective randomized study. Inflamm Bowel Dis 2021;27(8):1256–62.
61. Iacucci M, Kaplan GG, Panaccione R, et al. A randomized trial comparing high definition colonoscopy alone with high definition dye spraying and electronic virtual chromoendoscopy for detection of colonic neoplastic lesions during IBD surveillance colonoscopy. Am J Gastroenterol 2018;113(2):225–34.
62. Gulati S, Dubois P, Carter B, et al. A randomized crossover trial of conventional vs virtual chromoendoscopy for colitis surveillance: Dysplasia detection, feasibility, and patient acceptability (CONVINCE). Inflamm Bowel Dis 2019;25(6):1096–106.

63. Lewis JD, Cross RK, Long M, et al. High degree of practice variability in colonic dysplasia surveillance for patients with inflammatory bowel disease. Inflamm Bowel Dis 2022;28(8):1289–92.
64. Pouw RE, Bisschops R, Gecse KB, et al. Endoscopic tissue sampling—Part 2: Lower gastrointestinal tract. European Society of Gastrointestinal Endoscopy (ESGE) guideline. Endoscopy 2021;53(12): 1261–73.
65. van den Broek FJ, Stokkers PC, Reitsma JB, et al. Random biopsies taken during colonoscopic surveillance of patients with longstanding ulcerative colitis: Low yield and absence of clinical consequences. Am J Gastroenterol 2014;109(5):715–22.
66. Mooiweer E, van der Meulen-de Jong AE, Ponsioen CY, et al. Chromoendoscopy for surveillance in inflammatory bowel disease does not increase neoplasia detection compared with conventional colonoscopy with random biopsies: Results from a large retrospective study. Am J Gastroenterol 2015;110(7):1014–21.
67. Gasia MF, Ghosh S, Panaccione R, et al. Targeted biopsies identify larger proportions of patients with colonic neoplasia undergoing high-definition colonoscopy, dye chromoendoscopy, or electronic virtual chromoendoscopy. Clin Gastroenterol Hepatol 2016;14(5):704–12.e4.
68. Moussata D, Allez M, Cazals-Hatem D, et al. Are random biopsies still useful for the detection of neoplasia in patients with IBD undergoing surveillance colonoscopy with chromoendoscopy? Gut 2018;67(4):616–24.
69. Coelho-Prabhu N, Bruining DH, Faubion WA, et al. A 1-year cross-sectional inflammatory bowel disease surveillance colonoscopy cohort comparing high-definition white light endoscopy and chromoendoscopy. Inflamm Bowel Dis 2021;27(5):594–602.
70. Hu AB, Burke KE, Kochar B, et al. Yield of random biopsies during colonoscopies in inflammatory bowel disease patients undergoing dysplasia surveillance. Inflamm Bowel Dis 2021;27(6):779–86.
71. Watanabe T, Ajioka Y, Mitsuyama K, et al. Comparison of targeted vs random biopsies for surveillance of ulcerative colitis-associated colorectal cancer. Gastroenterology 2016;151(6):1122–30.
72. Mark-Christensen A, Erichsen R, Brandsborg S, et al. Long-term risk of cancer following ileal pouch-anal anastomosis for ulcerative colitis. J Crohns Colitis 2018;12(1):57–62.
73. Kariv R, Remzi FH, Lian L, et al. Preoperative colorectal neoplasia increases risk for pouch neoplasia in patients with restorative proctocolectomy. Gastroenterology 2010;139(3):806–12, 12.e1–2.
74. Abdalla M, Landerholm K, Andersson P, et al. Risk of rectal cancer after colectomy for patients with ulcerative colitis: A national cohort study. Clin Gastroenterol Hepatol 2017;15(7):1055–60.e2.
75. Derikx LA, Kievit W, Drenth JP, et al. Prior colorectal neoplasia is associated with increased risk of ileoanal pouch neoplasia in patients with inflammatory bowel disease. Gastroenterology 2014;146(1): 119–28.e1.
76. Urquhart SA, Comstock BP, Jin MF, et al. The incidence of pouch neoplasia following ileal pouch-anal anastomosis in patients with inflammatory bowel disease. Inflamm Bowel Dis 2023. [Epub ahead of print]. doi: 10.1093/ibd/izad021
77. Barrett CM, Long MD, Grimm I, et al. Off the cuff markers: Surveillance and endoscopic approaches for dysplasia after ileal pouch-anal anastomosis. Dig Dis Sci 2022;67(10):4666–70.
78. Derikx L, Nissen LHC, Smits LJT, et al. Risk of neoplasia after colectomy in patients with inflammatory bowel disease: A systematic review and meta-analysis. Clin Gastroenterol Hepatol 2016;14(6):798–806.e20.
79. Shergill AK, Lightdale JR, Bruining DH, et al. The role of endoscopy in inflammatory bowel disease. Gastrointest Endosc 2015;81(5): 1101–21.e1–13.
80. Shen B, Kochhar GS, Kariv R, et al. Diagnosis and classification of ileal pouch disorders: Consensus guidelines from the International Ileal Pouch Consortium. Lancet Gastroenterol Hepatol 2021;6(10):826–49.
81. Shen B, Kochhar GS, Navaneethan U, et al. Endoscopic evaluation of surgically altered bowel in inflammatory bowel disease: A consensus guideline from the Global Interventional Inflammatory Bowel Disease Group. Lancet Gastroenterol Hepatol 2021;6(6):482–97.
82. Adamina M, Feakins R, Iacucci M, et al. ECCO topical review optimising reporting in surgery, endoscopy, and histopathology. J Crohns Colitis 2021;15(7):1089–105.
83. Guerrero Vinsard D, Bruining DH, East JE, et al. Interobserver agreement of the modified Paris classification and histology prediction of colorectal lesions in patients with inflammatory bowel disease. Gastrointest Endosc 2023;97(4):790–8.e2.
84. Carballal S, Maisterra S, López-Serrano A, et al. Real-life chromoendoscopy for neoplasia detection and characterisation in long-standing IBD. Gut 2018;67(1):70–8.
85. Bisschops R, Bessissow T, Dekker E, et al. Pit pattern analysis with high-definition chromoendoscopy and narrow-band imaging for optical diagnosis of dysplasia in patients with ulcerative colitis. Gastrointest Endosc 2017;86(6):1100–6.e1.
86. Kudo S, Hirota S, Nakajima T, et al. Colorectal tumours and pit pattern. J Clin Pathol 1994;47(10):880–5.
87. Iacucci M, Furfaro F, Matsumoto T, et al. Advanced endoscopic techniques in the assessment of inflammatory bowel disease: New technology, new era. Gut 2019;68(3):562–72.
88. Iacucci M, McQuaid K, Gui XS, et al. A multimodal (FACILE) classification for optical diagnosis of inflammatory bowel disease associated neoplasia. Endoscopy 2019;51(2):133–41.
89. Sturm A, Maaser C, Calabrese E, et al. ECCO-ESGAR Guideline for Diagnostic Assessment in IBD. Part 2: IBD scores and general principles and technical aspects. J Crohns Colitis 2019;13(3):273–84.
90. Lightner AL, Vogler S, McMichael J, et al. Dysplastic progression to adenocarcinoma is equivalent in ulcerative colitis and Crohn's disease. J Crohns Colitis 2021;15(1):24–34.
91. Fumery M, Dulai PS, Gupta S, et al. Incidence, risk factors, and outcomes of colorectal cancer in patients with ulcerative colitis with low-grade dysplasia: A systematic review and meta-analysis. Clin Gastroenterol Hepatol 2017;15(5):665–74.e5.
92. Krugliak Cleveland N, Colman RJ, Rodriquez D, et al. Surveillance of IBD using high definition colonoscopes does not miss adenocarcinoma in patients with low-grade dysplasia. Inflamm Bowel Dis 2016;22(3):631–7.
93. Choi CH, Ignjatovic-Wilson A, Askari A, et al. Low-grade dysplasia in ulcerative colitis: Risk factors for developing high-grade dysplasia or colorectal cancer. Am J Gastroenterol 2015;110(10):1461–71; quiz 72.
94. Dziegielewski C, Gupta S, McCurdy JD, et al. Pancolonic dye spray chromoendoscopy to detect and resect ill-defined neoplastic lesions in colonic inflammatory bowel disease. J Can Assoc Gastroenterol 2023;6(1):37–41.
95. Mahmoud R, Shah SC, Torres J, et al. Association between indefinite dysplasia and advanced neoplasia in patients with inflammatory bowel diseases undergoing surveillance. Clin Gastroenterol Hepatol 2020; 18(7):1518–27.e3.
96. Bak MTJ, Albéniz E, East JE, et al. Endoscopic management of patients with high-risk colorectal colitis-associated neoplasia: A Delphi study. Gastrointest Endosc 2023;97(4):767–79.e6.
97. Mohan BP, Khan SR, Chandan S, et al. Endoscopic resection of colon dysplasia in patients with inflammatory bowel disease: A systematic review and meta-analysis. Gastrointest Endosc 2021;93(1):59–67.e10.
98. Mohapatra S, Sankaramangalam K, Lopimpisuth C, et al. Advanced endoscopic resection for colorectal dysplasia in inflammatory bowel disease: A meta-analysis. Endosc Int Open 2022;10(5):E593–601.
99. Manta R, Zullo A, Telesca DA, et al. Endoscopic submucosal dissection for visible dysplasia treatment in ulcerative colitis patients: Cases series and systematic review of literature. J Crohns Colitis 2021;15(1):165–8.
100. Ten Hove JR, Mooiweer E, Dekker E, et al. Low rate of dysplasia detection in mucosa surrounding dysplastic lesions in patients undergoing surveillance for inflammatory bowel diseases. Clin Gastroenterol Hepatol 2017;15(2):222–8.e2.
101. Lahiff C, Wang LM, Travis SPL, et al. Polyp-adjacent biopsies no longer required in inflammatory bowel disease. Gastrointest Endosc 2018; 88(4):782–3.
102. Krugliak Cleveland N, Huo D, Sadiq F, et al. Assessment of peri-polyp biopsy specimens of flat mucosa in patients with inflammatory bowel disease. Gastrointest Endosc 2018;87(5):1304–9.
103. Wanders LK, Dekker E, Pullens B, et al. Cancer risk after resection of polypoid dysplasia in patients with longstanding ulcerative colitis: A meta-analysis. Clin Gastroenterol Hepatol 2014;12(5):756–64.
104. Brodersen JB, Knudsen T, Kjeldsen J, et al. Diagnostic accuracy of pan-enteric capsule endoscopy and magnetic resonance enterocolonography in suspected Crohn's disease. United European Gastroenterol J 2022; 10(9):973–82.
105. Yang DH, John S, Mitsuhiro F, et al. Endoscopic diagnosis of nonpedunculated dysplasia during surveillance of ulcerative colitis: A survey-based multinational study. Gut Liver 2020;14(5):611–8.
106. Guerrero Vinsard D, Fetzer J, Agrawal U, et al. S26. Development of an artificial intelligence tool for detection of polypoid lesions in inflammatory bowel disease (IBD-CADe). J Am Coll Gastroenterol 2022;117:S7.