Optimising inflammatory bowel disease surveillance and dysplasia management—Where do we stand?

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Abstract
Patients with longstanding extensive colitis are at an increased risk of developing colorectal cancer (CRC), and are therefore enrolled into colonoscopy screening programmes with the aim of detecting pre-cancerous dysplastic change. However, current surveillance programs face multiple limitations relating to low levels of patient enrolment, missed lesions resulting in interval cancers, and uncertainties in the management of dysplasia. Patient counselling regarding the endoscopic and surgical management options of dysplastic lesions can prove particularly challenging, due to the variable risk of progression to cancer. In this review, we discuss the histopathological diagnosis of inflammatory bowel disease (IBD)-associated dysplasia, describe the techniques to maximise dysplasia detection, and present a standardised multi-disciplinary approach to managing patients with dysplasia. The challenges presented by this patient cohort highlight the clear clinical need for further research into the development and validation of non-invasive markers of CRC risk in IBD patients undergoing surveillance.

Keywords
colon, colonoscopy, colorectal cancer, EMR, ESD, inflammation, inflammatory bowel disease, ulcerative colitis

INTRODUCTION
Patients with inflammatory bowel disease (IBD) and longstanding colonic inflammation suffer from an increased risk of colorectal cancer (CRC). For this reason, international gastroenterology & endoscopy society guidelines recommend that these patients be enrolled into colonoscopy surveillance programmes, with the aim of detecting early CRC and precursor dysplasia. CRC formation remains a key area of concern raised by IBD patient focus groups, as one of the most feared complications in patients with longstanding colitis.1

An ageing IBD population, combined with improved endoscopic techniques and increased use of colectomy-sparing medication, means that per colonoscopy dysplasia detection rates by experienced endoscopists can exceed 10%.2 Despite rising dysplasia detection rates, the risk of IBD-associated CRC appears to be decreasing, with a cumulative CRC risk of 5% in patients with more than 20 years' disease duration.3 In this review, we discuss the histopathological diagnosis of IBD-associated dysplasia, describe the techniques to maximise dysplasia detection, and present a standardised multi-disciplinary approach to managing patients with dysplasia, and conclude by addressing the ongoing research into the development of non-invasive IBD surveillance modalities.
**IBD-DYSPLASIA AS A HISTOPATHOLOGICAL ENTITY**

The diagnosis of IBD-dysplasia is by definition a histopathological one, based on assessment of structural changes at the level of the nucleus, individual epithelial cells and overall crypt architecture.\(^4,5\) These relatively subjective criteria are shown in Table 1 and exemplified in Figure 1. Dysplasia grading suffers from significant inter-observer variability, even amongst expert gastrointestinal pathologists,\(^6\) with concordance being poorest when differentiating low-grade dysplasia (LGD) from inflammation-induced regenerative epithelial change (κ 0.3–0.4). It is for this reason that all dysplasia diagnoses should be validated by a second expert gastrointestinal histopathologist.\(^7,8\) Moreover, histopathological assessment is ultimately dependent on the tissue provided to the clinical pathologist. Maximal dysplasia grading, including the presence of deep foci of invasive CRC, can therefore be missed on superficial biopsies of dysplastic lesions.\(^9\)

Endoscopic advances in the optical assessment of colorectal lesions, which utilise parameters such as lesion morphology, crypt pit pattern and vascular organisation, now offer an increasingly reliable in vivo assessment of IBD neoplastic lesions,\(^10\) allowing for more accurately targeted biopsies for histopathological confirmation.

**OPTIMISING IBD ENDOSCOPIC SURVEILLANCE**

Strategies to optimise dysplasia detection at IBD surveillance can be performed at all stages of the patient pathway; these are listed below.

**Maximising patient uptake of IBD surveillance endoscopies**

Multiple studies confirm low uptake levels of IBD surveillance amongst eligible patients. Only 54% of eligible French patients in a CESAME cohort survey had at least one surveillance colonoscopy during a 7-year study period,\(^11\) while a regional UK-based root cause analysis study of IBD-associated CRC demonstrated that nearly two-thirds of patients with IBD who developed CRC were not under surveillance, despite eligibility.\(^12\) Reasons for low surveillance uptake include the absence of centrally organised IBD surveillance programme infrastructure (akin to national breast and bowel cancer screening programmes) for robust patient enrolment and recall, increased overall endoscopy demand limiting capacity for IBD surveillance, and reduced patient concordance due to factors such as cancer risk perception, as well as poor bowel preparation & procedure tolerance. A cross-sectional questionnaire of over 350

**TABLE 1** Histopathological criteria differentiating low-grade dysplasia from high-grade dysplasia

| Criterion            | Low grade dysplasia (LGD)                                                                 | High grade dysplasia (HGD)                                                                 |
|----------------------|------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------|
| Nuclear morphology   | - Normal polarity (long axis perpendicular to basement membrane)                         | - Loss of polarity                                                                        |
|                      | - Relatively uniform in size & shape                                                      | - Markedly pleomorphic, enlarged nuclei                                                  |
|                      | - Inconspicuous nucleoli                                                                | - Prominent nucleoli                                                                     |
|                      | - Few & typical mitotic figures                                                          | - Atypical mitotic figures                                                               |
| Cellular morphology  | - Nuclear stratification confined to the basal half of the cell                         | - Nuclear stratification extending to the luminal surface                                 |
| Crypt morphology     | - Tubular, villous or serrated architecture                                              | - Increasing architecture complexity (crowding, cribriform, or papillary configurations; villiform surface) |

**FIGURE 1** Histopathological sections of UC mucosa demonstrating no neoplasia (left), low-grade dysplasia (centre) and high-grade dysplasia (right)
American patients in three tertiary-referral centres reveals timely adherence to US surveillance guidelines in only a quarter of patients due to the aforementioned reasons, with poor bowel preparation tolerance as the single commonest patient-related factor in poor concordance with IBD surveillance. In a U.K.-based study, appropriately-timed surveillance intervals in only half of enrolled patients revealed a similarly low level of adherence.

A patient-centric IBD surveillance approach recognises that CRC risk varies between patients, with well-established clinical risk factors (see Table 2) associated with greater dysplasia and CRC risk. Adhering to more intensive surveillance intervals, as defined by societal guidelines such as European Crohn’s & Colitis Organisation, is particularly vital in higher-risk cohort, where the neoplastic yield will be greatest.

**Optimising mucosal visualisation**

Achieving clear mucosal views during IBD surveillance is vital in the detection of dysplastic lesions, and includes standard endoscopic practices such as sufficient insufflation, careful washing, dynamic position changes, the use of anti-spasmodics and adequate colonoscope withdrawal times (at least 17 min according to expert consensus). Retained stools and uncontrolled inflammation are the two salient patient-related factors responsible for limited mucosal assessment. Low-volume split-dose polyethylene glycol regimens with a low-fibre diet provide the optimal bowel preparation regimen in terms of cleansing quality and patient tolerability. Optimising anti-inflammatory medical therapy before endoscopic surveillance is vital not only to improving mucosal views, but also because significant inflammation will limit histopathological assessment. In patients with refractory inflammation despite medical therapy, pre-procedure administration of a short corticosteroid course (prednisolone 20 mg once daily for 2 weeks) can significantly reduce inflammation, without incurring significant side effects. Finally, ESGE (European Society of Gastrointestinal Endoscopy) guidelines recommend that surveillance should be performed with high-definition endoscopes, using either dye spray or virtual chromoendoscopy (e.g. iSCAN and narrow-band imaging), with targeted biopsies of any suspected lesions. When adequate mucosal visualisation is achieved, random quadratic biopsies at 10 cm intervals are no longer routinely recommended, as targeted biopsies detect the vast majority of dysplastic change. The use of random quadratic biopsies should therefore be restricted to colonic segments where adequate mucosal assessment is not possible (e.g. strictures or segments with extensive inflammatory pseudopolyps).

**Improving operator performance**

Dysplasia detection is ultimately dependent on endoscopy operator experience; lesion recognition can prove challenging for less experienced endoscopists due to active inflammation, regenerative change, mucosal scarring and post-inflammatory pseudopolyps, particularly when dye spray is used. For this reason, we recommend that the provision of IBD surveillance colonoscopies is limited to experienced endoscopists with experience in this procedure and patient cohort. Limiting IBD surveillance colonoscopies to experienced endoscopists

**TABLE 2** Recommended IBD surveillance intervals as per ECCO guidelines

| Eligible cohorts | Risk level | Clinical features | Surveillance interval |
|------------------|------------|-------------------|-----------------------|
| >30% colonic involvement AND 8–10 years after IBD symptom onset OR Beginning at time of IBD diagnosis in patients with PSC | Lower | Colitis affecting <50% of the colon OR Extensive colitis with no endoscopic or histological inflammation AND No intermediate of high group risk factors | Every 5 years |
| | Intermediate | Extensive colitis with mild or moderate endoscopic or histological inflammation First degree relative diagnosed with CRC aged over 50 Inflammatory pseudo-polyposis | Every 2–3 years |
| | Higher | Extensive colitis with severe endoscopic or histological inflammation Co-diagnosis with PSC Colonic stricture in the past 5 years Dysplasia in the past 5 years First degree relative diagnosed with CRC aged under 50 | Every year |

Abbreviations: CRC, colorectal cancer; ECCO, European Crohn’s & Colitis Organisation; IBD, inflammatory bowel disease; PSC, primary sclerosing cholangitis.
may also have the added benefit of improving long-term patient compliance through improved patient comfort. To allow for adequate mucosal assessment, the highest-definition endoscope systems available should be used, and an appropriate amount of time must be allocated for each procedure (we recommend 45 min). Endoscopy units should be encouraged to use a standardised reporting format (see Table 3) to reduce inter-operator variability, and to promote data collection for service evaluation. This includes

**TABLE 3** Example of a standardised reporting format for IBD surveillance colonoscopies

| Field                                      | Sub-field                                      | Examples                                                                 |
|--------------------------------------------|-----------------------------------------------|-------------------------------------------------------------------------|
| Patient-related factors                    | Patient demographics                           | Age, gender                                                             |
|                                            | Duration of IBD diagnosis                     |                                                                         |
|                                            | Extent of IBD                                 | UC Montreal classification                                              |
|                                            | PSC status                                   |                                                                         |
|                                            | Previous dysplasia                            |                                                                         |
|                                            | First degree family history of CRC            |                                                                         |
|                                            | Bowel preparation regimen used                |                                                                         |
| Technical factors                          | Endoscopic system used                        | High-definition versus standard definition endoscope                    |
|                                            | Use of chromoendoscopy                        | Dye spray versus virtual chromoendoscopy                               |
|                                            | Endoscope withdrawal time                    | Minimum of 17 min                                                       |
|                                            | Quality of bowel preparation                  | Boston bowel preparation scale                                          |
| Description of large intestine             | Extent of inflammation                        | UCEIS score                                                             |
|                                            | Severity of inflammation                      | Mayo endoscopic score                                                   |
|                                            | Stigmata of chronic inflammation              | Mucosal scarring, pseudopolyposis, stricturing                          |
|                                            | Background biopsies taken                     | We recommend 2 × right colon, 2 × left colon and 2 × rectal biopsies to assess inflammation |
| Description of suspected dysplastic lesion(s) | Lesion site                                   | Distance of lesion from anal verge                                      |
|                                            | Lesion shape                                  | Polypoid versus non-polypoid shape                                     |
|                                            | Lesion surface architecture                   | Paris classification of polyp morphology                               |
|                                            | Lesion margins                                | Defined or ill-defined margins                                         |
|                                            | Lesion inflammation                           |                                                                          |
|                                            | Anatomical factors limiting potential endoscopic resection | Involvement of diverticulum, involvement of ICV or appendiceal orifice, proximity to dentate line |
|                                            | Biopsies taken                                | Limited to areas of diagnostic uncertainty & suspected CRC, to minimise sub-mucosal fibrosis |
|                                            | Marker tattoo                                 | Recommended for lesions >20 mm, non-polypoid lesions, and suspected CRC |
| Follow-up plan                             | Surveillance interval                         | As per relevant societal guideline recommendation for example, ECCO      |
|                                            | Adjustments needed for the next colonoscopy   | Altered bowel preparation regimen, escalation of anti-inflammatory therapy, change in operator to advanced/therapeutic endoscopist |
|                                            | Clinic follow-up                              |                                                                          |
|                                            | Need for discussion at IBD MDT meeting        |                                                                          |

Abbreviations: CRC, colorectal cancer; ECCO, European Crohn’s & Colitis Organisation; FACILE, Frankfurt advanced chromoendoscopic IBD lesions classification; IBD, inflammatory bowel disease; ICV, ileocaecal valve; MDT, multi-disciplinary team; PSC, primary sclerosing cholangitis; UCEIS, ulcerative colitis endoscopic Index of severity.
a systematic approach to the description and photo-documentation of visible dysplastic lesions using the five ‘S’: site, size, shape, surface & surrounding area.\textsuperscript{21}

The lack of structured training opportunities in endoscopic recognition of dysplastic lesions represents a significant unmet need. A recent survey of Canadian academic gastroenterologists demonstrates that chromoendoscopy uptake was <30\%, with inadequate endoscopist training identified as a major barrier.\textsuperscript{22} The ESGE Optic Diagnosis curriculum\textsuperscript{2} describes a structured approach towards gaining and maintaining competency in IBD surveillance colonoscopy, including a neoplasia detection rate of ≥10\% using targeted biopsies. The OPTIC-IBD online training platform\textsuperscript{23} is the first international, validated attempt at addressing this need: the platform developers used a standardised approach combining Frankfurt advanced chromoendoscopic IBD lesions classification,\textsuperscript{10} Kudo pit pattern assessment and inflammation scoring, using high-definition chromoendoscopy, to optically assess recordings of IBD-associated neoplastic lesions on a large endoscopy video bank. Finally, artificial intelligence software, originally developed for the detection of sporadic neoplastic colonic lesions, has shown initial promise in detecting IBD-associated dysplasia,\textsuperscript{24} with the CUDISIA trial\textsuperscript{25} representing the first prospective study to assess the impact of artificial intelligence software on IBD dysplasia detection rates.

THE MANAGEMENT OF IBD DYSPLASIA

A standardised multi-disciplinary approach to managing IBD dysplasia

IBD dysplasia cases should be discussed in a multi-disciplinary setting. For these meetings to be quorate, they should include at least one IBD physician, one advanced endoscopist and an IBD surgeon. Most visible dysplastic lesions are amenable to endoscopic resection; however, endoscopic resection of IBD dysplasia should be limited to advanced endoscopists with expertise in endoscopic mucosal resection (EMR) or endoscopic submucosal dissection (ESD)\textsuperscript{26} so as to achieve en bloc resection with clear resection margins and lower risks of recurrence.\textsuperscript{27,28} These procedures can prove technically challenging due to inflammation-induced submucosal fibrosis, and margin recognition can be subtle, particularly for flat lesions. Repeated resection attempts will only exacerbate these difficulties; for this reason, it is vital that ‘the first resection is the best resection’. For lesions larger than 20 mm, or where EMR is unlikely to be en bloc, ESD should be considered if the expertise is available.\textsuperscript{26} ESD of dysplastic lesions carries additional procedural risks, with a systematic review of 191 resections demonstrating a 6.7\% major bleeding rate and 2.9\% perforation rate; however, these complications were all managed successfully by endoscopic means at the time of the ESD.\textsuperscript{30} A meta-analysis of endoscopic resection of large dysplastic lesions (median size 23 mm) by experienced endoscopists confirms the efficacy of EMR and ESD, with local recurrence rates of under 5\%, and metachronous dysplasia risk of under 7.5\%.\textsuperscript{30} Biopsies of the flat mucosa surrounding a dysplastic lesion are of low yield, and are not routinely recommended unless there are concerns about the resection completeness, or with difficulties in assessing lesion margin.\textsuperscript{31}

If all dysplastic lesions are successfully resected endoscopically, and there is no dysplastic change in the flat mucosa surrounding the lesion(s), then these patients should undergo regular endoscopic surveillance, as the risk of CRC after endoscopic resection of dysplasia is low.\textsuperscript{32–34} The next surveillance colonoscopy can be performed after 1 year for sub-centimetre polypoid LGD lesions. The detection of a high-grade dysplasia (HGD) lesion generates clinical concern, not least due to the synchronous CRC risk approaching 15\%\textsuperscript{35}; however, endoscopic resection of HGD lesions is efficacious, with a meta-regression analysis demonstrating that HGD histology did not significantly influence future CRC.\textsuperscript{36} For higher-risk lesions that are >10 mm in diameter, have non-polypoid morphology, or contain HGD, a re-examination of the resection site for dysplastic recurrence should be undertaken after 3–6 months.\textsuperscript{7} Lesions with features of submucosal invasion, or with significant submucosal fibrosis limiting endoscopic resection, including irregular surface architecture, mucosal depression, radiating folds or failure to lift with submucosal injection, are unlikely to be resected en bloc successfully; these patients should be considered for a colectomy.\textsuperscript{31} Figure 2 summarises our current clinical approach to the management of visible colitis-associated dysplasia.

If invisible dysplasia is detected, then a repeat high-definition chromoendoscopy in an optimally-prepared patient should be performed by an experienced endoscopist, as the ‘invisible dysplasia’ may represent a missed non-polypoid lesion.\textsuperscript{7,8} Invisible HGD should prompt a referral for a colectomy due to the high associated CRC risk.\textsuperscript{8,37} If invisible LGD is detected once again despite an optimised repeat colonoscopy, then there is some equipoise as to the best management approach due to the low quality of long-term outcome data derived from small cohort studies, and the recognition that many of the ‘invisible’ lesions detected in historical studies likely would have been visible using modern endoscopic imaging.\textsuperscript{35,38} Invisible LGD is an independent predictor of long-term progression to advanced neoplasia in multivariate analyses (two to three-fold increased risk).\textsuperscript{34,38} Increasingly, clinicians and patients find it acceptable to undertake a period of intensive high-quality surveillance when unifocal invisible LGD is detected, rather than proceeding immediately to colectomy.\textsuperscript{31} Recent surveillance studies have produced varying results: a Dutch multicentre cohort surveillance study reported progression to CRC in only 3.8\% (1/26) of patients with invisible LGD over a median of 5 years follow-up.\textsuperscript{39} However, the CRC incidence for unifocal invisible LGD was 4.3 per 100 patient years (7/42) in a UK multi-centre UC cohort study.\textsuperscript{34}

The role of segmental colectomy (and in particular, rectum-sparing surgery) in patients with UC and dysplasia remains controversial due to the high rates of synchronous and metachronous neoplasia. Even in Crohn’s colitis, where the option of segmental colectomy can be considered to preserve those segments unaffected by IBD, it is interesting to note the high metachronous
CRC of up to 40% following segmental resection or subtotal colectomy. Nonetheless, a recently published retrospective case series of 17 patients with longstanding quiescent IBD and unifocal neoplasia undergoing subtotal colectomy with ileorectal anastomosis showed that the majority of these patients remain neoplasia-free after a median follow-up period of 4 years, with the 4 cases of metachronous LGD seen exclusively in patients with primary sclerosing cholangitis (PSC). This finding highlights the need for prospective studies of rectum-sparing surgery in PSC-free patients with unifocal neoplasia and quiescent IBD in the retained distal colorectum.

Risk management and communication strategies in IBD patients with dysplasia

It is imperative that patients are counselled about their continued risk of metachronous neoplasia despite successful endoscopic dysplasia resection and continued colonoscopic surveillance, and that clinicians take into consideration all lesion-specific and patient-specific risk factors when communicating risks to a patient. Dysplasia features associated with higher rates of progression to CRC include histologically-confirmed HGD, multifocality, invisibility, large (≥10 mm) lesion size, and non-polypoid morphology. Patient-specific risk factors include concomitant PSC, previous dysplasia, significant uncontrolled mucosal inflammation, limitations to adequate mucosal assessment (e.g. colonic stricturing or extensive pseudopolypsis) and a family history of CRC. The presence of these risk factors should prompt clinicians to discuss the benefits of cancer-preventative colectomy over continued long-term surveillance and endoscopic management. This is especially relevant in patients with more than one risk factor: advanced neoplasia risk increases cumulatively in the presence of multiple risk factors.

Many patients are understandably reluctant to accept life-changing surgery that results in stoma or pouch formation, particularly if they are in clinical remission. Indeed, published data indicates that patients and their clinicians tolerate significantly differing CRC risk thresholds. A multidisciplinary shared decision-making approach should therefore be used to guide management based on the patient’s informed preferences. By eliciting a patient’s values and long-term goals, a tailored discussion of the risks and benefits of surgical and endoscopic management options can be conducted effectively. Joint surgical-physician clinic appointments for such patients will facilitate these discussions, by providing a unified and streamlined clinical consultation. Patients should also be given time to deliberate and consolidate their informed preferences after discussion with specialist nurses and trained patient advocates from support groups. Any uncertainty regarding long-term outcomes should be acknowledged with patients; visual aids like UC-CaRE (www.uc-care.uk), an externally validated risk prediction webtool, can be used to help predict and communicate individualised cancer risk to patients with UC and LGD (see Figure 3).
FUTURE DIRECTIONS

There is a clear clinical need for novel validated non-invasive surveillance techniques in IBD patients, which would not only be more tolerable for patients, but that can also reduce the overall burden of colonoscopies by identifying low-risk patients who are unlikely to develop CRC. While there have been no studies assessing the role of colon capsule endoscopy in IBD surveillance, future advances in capsule image resolution and automated image analysis mean that this non-invasive technique may potentially play a role in lower-risk patients with quiescent disease (particularly those with technically challenging colonoscopies), in whom there is a lower need for concomitant histopathological assessment. Other non-endoscopic options in development include the use of blood samples as a liquid biopsy to isolate colonic epithelial cell-free DNA and circulating tumour DNA for analysis. In addition, faecal samples can be used to isolate and analyse colonic epithelial DNA. In this manner, ongoing advances in automated image analysis, next generation genomic sequencing and molecular medicine techniques have the potential to significantly improve the clinical management of this challenging patient cohort.

CONFLICT OF INTEREST

The author declares that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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REFERENCES

1. Kim AH, Roberts C, Feagan BG, Banerjee R, Bemelman W, Boger K, et al. Developing a standard set of patient-centred outcomes for inflammatory bowel disease: an international, cross-disciplinary consensus. J Crohns Colitis. 2018;12(4):408–18. https://doi.org/10.1093/ecco-jcc/jjx161
2. Dekker E, Houwen BBGL, Puig I, Bustamante-Balén M, Coron E, Dobru DE, et al. Curriculum for optical diagnosis training in Europe: European Society of Gastrointestinal Endoscopy (ESGE) position statement. Endoscopy. 2020;52(10):899–923. https://doi.org/10.1055/a-1231-5123
3. Lutgens MWMD, van Oijen MGH, van der Heijden DJMG, Vleggaar FP, Siersma PD, Oldenburg B. Declining risk of colorectal cancer in inflammatory bowel disease: an updated meta-analysis of population-based cohort studies. Inflamm Bowel Dis. 2013;19(4):789–99. https://doi.org/10.1097/mib.0b013e31828029c0
4. Harpaz N, Polydorides A. Colorectal dysplasia in chronic inflammatory bowel disease: pathology, clinical implications, and pathogenesis. Arch Pathol Lab Med. 2010;134(6):876–95. https://doi.org/10.5858/134.6.876
5. DeReo TC, Xiao SY, Liu X. Histological evaluation in ulcerative colitis. Gastroenterol Rep. 2014;2(3):178–92. https://doi.org/10.1093/gastro/gou031
6. Eaden J, Abrams K, McKay H, Denley H, Mayberry J. Inter-observer variation between general and specialist gastrointestinal pathologists when grading dysplasia in ulcerative colitis. J Pathol. 2001;194(2):152–7. https://doi.org/10.1002/path.876
7. Laine L, Kaltenbach T, Barkun A, McQuaid KR, Subramanian V, Soetikno R, et al. SCENIC international consensus statement on surveillance and management of dysplasia in inflammatory bowel disease. Gastroenterology. 2015;148(3):639–51.e28. https://doi.org/10.1053/j.gastro.2015.01.031
8. Magro P, Gionchetti P, Eliakim R, Ardizzone S, Armuzzi A, Barreiro-de Acosta M, 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. https://doi.org/10.1093/ecco-jcc/jjx008
9. Siddham RW, Higgins PDR. Colorectal cancer in inflammatory bowel disease. Clin Colon Rectal Surg. 2018;31(3):160–78. https://doi.org/10.1055/s-0037-1602237
10. Iacucci M, McQuaid K, Gui XS, Iwao Y, Lethebe BC, Lowerison M, et al. A multimodal (FACILE) classification for optical diagnosis of inflammatory bowel disease associated neoplasia. Endoscopy. 2019;51(2):133–41. https://doi.org/10.1055/a-0757-7759
11. Vienne A, Simon T, Casnes J, Baudry C, Bouhnik Y, Soulé JC, et al. Low prevalence of colonoscopic surveillance of inflammatory bowel disease patients with longstanding extensive colitis: a clinical practice survey nested in the CESAME cohort. Aliment Pharmacol Ther. 2011;34(2):188–95. https://doi.org/10.1111/j.1365-2036.2011.04711.x

12. Gordon C, Chee D, Hamilton B, Heerasing NM, Hendy P, Chanchlani N, et al. Root-cause analyses of missed opportunities for the diagnosis of colorectal cancer in patients with inflammatory bowel disease. Aliment Pharmacol Ther. 2021;53(2):291–301.

13. Friedman S, Cheifetz AS, Farraye FA, Banks PA, Makrauer FL, Burakoff R, et al. Factors that affect adherence to surveillance colonoscopy in patients with inflammatory bowel disease. Inflamm Bowel Dis. 2013;19(3):534–9. https://doi.org/10.1097/MIB.0b013e3182802a3c

14. Hussain K, Watt L, Hillemand C, Murphy S. PTH-085 are IBD patients getting appropriate surveillance colonoscopies? Gut. 2017;66(Suppl 2):A248.

15. Maaser C, Sturm A, Vavricka SR, Kucharzik T, Fiorino G, Annese V, et al. ECCO-ESGAR Guideline for Diagnostic Assessment in IBD Part 1: initial diagnosis, monitoring of known IBD, detection of complications. J Crohns Colitis. 2019;13(2):144–164K. https://doi.org/10.1093/ecco-jcc/jjy113

16. Iacucci M, Cannatelli R, Tontini GE, Panaccone R, Danese S, Fiorino G, et al. Improving the quality of surveillance colonoscopy in inflammatory bowel disease. Lancet Gastroenterol Hepatol. 2019;4(12):971–83. https://doi.org/10.1016/s2468-1253(19)30194-3

17. Restellini S, Kherad O, Bessissow T, Ménard C, Martel M, Tanjani MT, 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. https://doi.org/10.3748/wjg.v23.i32.5994

18. Baars JE, Vogelaar L, Wolfhagen FHJ, Biermann K, Kuipers EJ, van der Woude CJ. A short course of corticosteroids prior to surveillance colonoscopy to decrease mucosal inflammation in inflammatory bowel disease patients: results from a randomized controlled trial. J Crohns Colitis. 2010;4(6):661–8. https://doi.org/10.1016/j.jcch.2010.07.011

19. Bisschops R, East JE, Hassan C, Hazewinkel Y, Kaminski MF, Neumann H, et al. Advanced imaging for detection and differentiation of colorectal neoplasia: European Society of Gastrointestinal Endoscopy (ESGE) guideline - update 2019. Endoscopy. 2019;51(12):1155–79. https://doi.org/10.1055/a-1031-7657

20. Kandiah K, Subramaniam S, Thayalasekaran S, Cheddy FQ, Longcroft-Wheaton G, Fogg C, 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. https://doi.org/10.1136/gutjnl-2020-320980

21. Adamina M, Fearnls R, Iacucci M, Spinelli A, Cannatelli R, D’Hoore A, et al. ECCO topical review optimising reporting in surgery, endoscopy, and histopathology. J Crohns Colitis. 2021;15(7):1089–105. https://doi.org/10.1093/ecco-jcc/jjab011

22. Gallinger Z, Rumman A, Murthy S, Nguyen G. Perspectives on endoscopic surveillance of dysplasia in inflammatory bowel disease: a survey of academic gastroenterologists. Endosc Int Open. 2017;05(10):E974–E979. https://doi.org/10.1055/s-0043-117944

23. Iacucci M, Ingram RJM, Bazarova A, Cannatelli R, Labarile N, Nardone OM, et al. DOP14 Validation of a new OPtical diagnosis Training platform to Improve dysplasia Characterisation in Inflammatory Bowel Disease (OPTIC-IBD): a multicentre randomised controlled study. J Crohns Colitis. 2022;16(Supplement_1):i063–i064. https://doi.org/10.1093/ecco-jcc/jjab232.053

24. Maeda Y, Kudo SE, Ogata N, Misawa M, Mori Y, Mori K, et al. Can artificial intelligence help to detect dysplasia in patients with ulcerative colitis? Endoscopy. 2021;53(7):E273–E274. https://doi.org/10.1055/a-1261-2944

25. Artificial intelligence and dysplasia detection in ulcerative colitis (CUDISIA Study) - full text view - ClinicalTrials.gov [Internet]. [cited 2022 May 8]. Available from: https://clinicaltrials.gov/ct2/show/NCOT5171634

26. Soetinco R, East J, Suzuki N, Uedo N, Matsumoto T, Watanabe K, et al. Endoscopic submucosal dissection for nonpolyoid colorectal dysplasia in patients with inflammatory bowel disease: in medias res. Gastrointest Endosc. 2018;87(4):1085–94. https://doi.org/10.1016/j.gie.2018.01.013

27. Alkandari A, Thayalasekaran S, Bhandari M, Przybysz A, Bugajski M, Bassett P, et al. Endoscopic resections in inflammatory bowel disease: a multicentre European outcomes study. J Crohns Colitis. 2019;13(11):1394–400. https://doi.org/10.1093/ecco-jcc/jjy075

28. Lightner AL, Vaidya P, Allende D, Gorgun E. Endoscopic submucosal dissection is safe and feasible, allowing for ongoing surveillance and organ preservation in patients with inflammatory bowel disease. Colorectal Dis. 2021;23(6):2100–7. https://doi.org/10.1111/ceji.15746

29. Manta R, Zullo A, Telesca DA, Castellani D, Germani U, Reggiani Bonetti L, 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. https://doi.org/10.1093/ecco-jcc/jjaa158

30. Mohapatra S, Sankaramangalam K, Lopimisuth C, Moninuola O, Simons M, Nanavati J, et al. Advanced endoscopic resection for colorectal dysplasia in inflammatory bowel disease: a meta-analysis. Endosc Int Open. 2022;10(5):E593–E601. https://doi.org/10.1055/a-1784-7063

31. Murthy SK, Feuerstein JD, Nguyen GC, Velayos FS. AGA clinical practice update on endoscopic surveillance and management of colorectal dysplasia in inflammatory bowel disease: expert review. Gastroenterology. 2021;161(3):1043–51.e4. https://doi.org/10.1053/j.gastro.2021.05.063

32. Wanders LK, Dekker E, Pullens B, Bassett P, Travis SPL. East JE. Colon cancer risk after resection of polyoid dysplasia in patients with longstanding ulcerative colitis: a meta-analysis. Clin Gastroenterol Hepatol. 2014;12(5):756–64. https://doi.org/10.1016/j.cgh.2013.07.024

33. Cremer A, Demetter P, De Vos M, Rahier JF, Baert F, Moreeels T, 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. https://doi.org/10.1016/j.cgh.2019.05.062

34. Curtius K, Kabir M, Al Bakir I, Choi CHR, Hartono JL, Johnson M, et al. Multicentre derivation and validation of a colitis-associated rectal cancer risk prediction web tool. Gut. 2022;71(4):705–15. https://doi.org/10.1136/gutjnl-2020-323546

35. Kabir M, Fofaria R, Arebi N, Bassett P, Tozer PJ, Hart AL, et al. Systematic review with meta-analysis: IBD-associated colonic dysplasia prognosis in the videoendoscopic era (1990 to present). Aliment Pharmacol Ther. 2020;52(1):E974–E979. https://doi.org/10.1111/apt.15778

36. Mohan BP, Khan SR, Chandan S, Kassab LL, Ponnada S, Asokkumar R, et al. Endoscopic resection of colon dysplasia in patients with inflammatory bowel disease: a systematic review and meta-analysis. Gastrointest Endosc. 2021;93(1):67–76.e10. https://doi.org/10.1016/j.gie.2020.06.048

37. Lightner AL, Vogler S, McMichael J, Jia X, Regueiro M, Qazi T, et al. Dysplastic progression to adenocarcinoma is equivalent in ulcerative colitis and Crohn’s disease. J Crohns Colitis. 2021;15(1):24–34. https://doi.org/10.1093/ecco-jcc/jjab133

38. Fumery M, Dulai PS, Gupta S, Prokop LJ, Ramamoorthy S, Sandborn WJ, 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. https://doi.org/10.1016/j.cgh.2016.11.025

39. Ten Hove JR, Mooiweer E, Van Der Meulen De Jong AE, Dekker E, Ponsioen CY, Siersema PD, et al. Clinical implications of low grade dysplasia found during inflammatory bowel disease surveillance: a retrospective study comparing chromoendoscopy and white-light endoscopy. Endoscopy. 2017;49(2):161–8. https://doi.org/10.1055/s-0042-179394

40. Maser EA, Sachar DB, Kruse D, Harpaz N, Ullman T, Bauer JJ. High rates of metachronous colon cancer or dysplasia after segmental resection or subtotal colectomy in Crohn’s colitis. Inflamm Bowel Dis. 2013;19(9):1827–32.

41. Cleveland NK, Ollech JE, Colman RJ, Rodriguez D. Efficacy and follow-up of sub-total colectomy with ileo-rectal anastomoses in patients with colitis-associated neoplasia. Clin Gastroenterol Hepatol. 2020;17(1):205–6.

42. Yalchin M, Baker AM, Graham TA, Hart A. Predicting colorectal cancer occurrence in IBD. Cancers. 2021;13(12):2908. https://doi.org/10.3390/cancers13122908

43. Fumery M, Dulai PS, Meirick P, Farrell AM, Ramamoorthy S, Sandborn WJ, et al. Systematic review with meta-analysis: recurrence of Crohn’s disease after total colectomy with permanent ileostomy. Aliment Pharmacol Ther. 2017;45(3):381–90. https://doi.org/10.1111/apt.13886

44. Choi C-HR, Ignjatovic-Wilson A, Askari A, Lee GH, Warusavitane J, Moorghen M, 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. https://doi.org/10.1038/ajg.2015.248

45. Kabir M, Thomas-Gibson S, Hart AL, Tozer PJ, Faiz O, Warusavitane J, et al. Management of inflammatory bowel disease associated colonic dysplasia: factors predictive of patient choice and satisfaction. Colorectal Dis. 2021;23(4):882–93. https://doi.org/10.1111/codi.15460

46. Siegel CA, Schwartz LM, Woloshin S, Cole EB, Rubin DT, Vay T, et al. When should ulcerative colitis patients undergo colectomy for dysplasia? Mismatch between patient preferences and physician recommendations. Inflamm Bowel Dis. 2010;16(10):1658–62. https://doi.org/10.1002/ibd.21233

47. Siegel CA. Shared decision making in inflammatory bowel disease: helping patients understand the tradeoffs between treatment options. Gut. 2012;61(3):459–65. https://doi.org/10.1136/gutjnl-2011-300988

48. Elwyn G, Durand MA, Song J, Aarts J, Barr PJ, Berger Z, et al. A threetalk model for shared decision making: multistage consultation process. BMJ. 2017;359:j4891. https://doi.org/10.1136/bmj.j4891

49. Kisiel JB, Yab TC, Nazer Hussain FT, Taylor WR, Garrity-Park MM, Sandborn WJ, et al. Stool DNA testing for the detection of colorectal neoplasia in patients with inflammatory bowel disease. Aliment Pharmacol Ther. 2013;37(5):546–54. https://doi.org/10.1111/apt.12218

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