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REVIEW ARTICLE

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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, present a standardised multidisciplinary approach to managing patients with dysplasia, and conclude by addressing the ongoing research into the development of non-invasive IBD surveillance modalities.

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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.

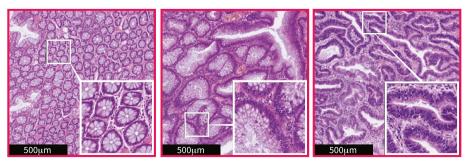
Strategies to optimise dysplasia detection at IBD surveillance can be performed at all stages of the patient pathway; these are listed helow

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 twothirds 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) Relatively uniform in size & shape Inconspicuous nucleoli Few & typical mitotic figures 	 Loss of polarity Markedly pleiomorphic, enlarged nuclei Prominent nucleoli 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)



Normal epithelium

Low grade dysplasia (LGD) High grade dysplasia (HGD)

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.¹³ Similarly, a UK-based study reveals appropriately-timed surveillance intervals in only half of enrolled patients.¹⁴

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.¹⁵ 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, in whom 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 colono-scope withdrawal times (at least 17 min according to expert consensus).¹⁶ 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.¹⁷ 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.¹⁸ 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.¹⁹ 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.²⁰ 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 pseudopolyposis).

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 pseudopolyposis, particularly when dye spray is used. For this reason, we recommend that the provision of IBD surveillance colonoscopies is limited to 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 guidelines¹⁵

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

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
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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	
	Severity of inflammation	UCEIS score
		Mayo endoscopic score
	Stigmata of chronic inflammation	Mucosal scarring, pseudopolyposis, stricturing
	Background biopsies taken	We recommend 2 \times right colon, 2 \times left colon and 2 \times rectal biopsies to assess inflammation
Description of suspected	Lesion site	Distance of lesion from anal verge
dysplastic lesion(s)	Lesion shape	Polypoid versus non-polypoid shape
		Paris classification of polyp morphology
	Lesion surface architecture	Kudo pit pattern classification
		FACILE surface & vessel classification
	Lesion margins	FACILE surface & vessel classification Defined or ill-defined margins
	Lesion margins Lesion inflammation	
	-	
	Lesion inflammation Anatomical factors limiting potential	Defined or ill-defined margins Involvement of diverticulum, involvement of ICV
	Lesion inflammation Anatomical factors limiting potential endoscopic resection	Defined or ill-defined margins Involvement of diverticulum, involvement of ICV or appendiceal orifice, proximity to dentate line Limited to areas of diagnostic uncertainty & suspected CRC, to
Follow-up plan	Lesion inflammation Anatomical factors limiting potential endoscopic resection Biopsies taken	Defined or ill-defined margins Involvement of diverticulum, involvement of ICV or appendiceal orifice, proximity to dentate line Limited to areas of diagnostic uncertainty & suspected CRC, to minimise sub-mucosal fibrosis Recommended for lesions >20 mm, non-polypoid
Follow-up plan	Lesion inflammation Anatomical factors limiting potential endoscopic resection Biopsies taken Marker tattoo	Defined or ill-defined margins Involvement of diverticulum, involvement of ICV or appendiceal orifice, proximity to dentate line Limited to areas of diagnostic uncertainty & suspected CRC, to minimise sub-mucosal fibrosis Recommended for lesions >20 mm, non-polypoid lesions, and suspected CRC As per relevant societal guideline recommendation
Follow-up plan	Lesion inflammation Anatomical factors limiting potential endoscopic resection Biopsies taken Marker tattoo Surveillance interval Adjustments needed for the next	 Defined or ill-defined margins Involvement of diverticulum, involvement of ICV or appendiceal orifice, proximity to dentate line Limited to areas of diagnostic uncertainty & suspected CRC, to minimise sub-mucosal fibrosis Recommended for lesions >20 mm, non-polypoid lesions, and suspected CRC As per relevant societal guideline recommendation for example, ECCO Altered bowel preparation regimen, escalation of anti-inflammatory therapy, change in operator

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.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.²² The ESGE Optic Diagnosis curriculum² describes a structured approach towards gaining and maintaining competency in IBD surveillance colonoscopy, including a neoplasia detection rate of \geq 10% using targeted biopsies. The OPTIC-IBD online training platform²³ is the first international, validated attempt at addressing this need: the platform developers used a standardised approach combining Frankfurt advanced chromoendoscopic IBD lesions classification.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,²⁴ with the CUDISIA trial²⁵ 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

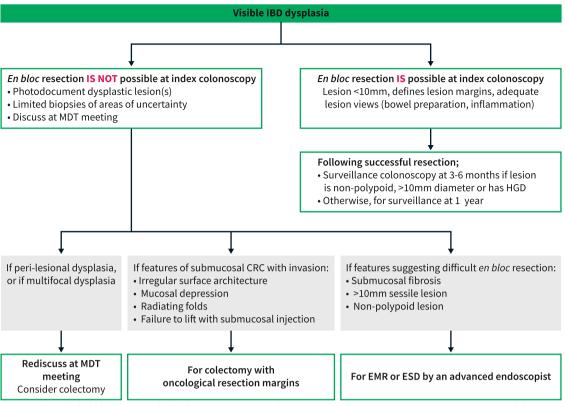
IBD dysplasia cases should be discussed in a multi-disciplinary setting. For these meetings to be quorate, they should include at least one IBD physician, one advanced endoscopist and an IBD surgeon. Most visible dysplastic lesions are amenable to endoscopic resection; however, endoscopic resection of IBD dysplasia should be limited to advanced endoscopists with expertise in endoscopic mucosal resection (EMR) or endoscopic submucosal dissection (ESD)²⁶ so as to achieve *en bloc* resection with clear resection margins and lower risks of recurrence.^{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.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.²⁹ 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%.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.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.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%³⁵; however, endoscopic resection of HGD lesions is efficacious, with a meta-regression analysis demonstrating that HGD histology did not significantly influence future CRC.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.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.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.^{7,8} Invisible HGD should prompt a referral for a colectomy due to the high associated CRC risk.^{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.^{35,38} Invisible LGD is an independent predictor of long-term progression to advanced neoplasia in multivariate analyses (two to three-fold increased risk).^{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.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.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.³⁴

The role of segmental colectomy (and in particular, rectumsparing 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



HGD = High-grade dysplasia; MDT = Multidisiplinary team; EMR = Endoscopic mucosal resection; ESD = Endoscopic submucosal dissection

FIGURE 2 Flowchart of the management of visible colitis-associated dysplastic lesions

CRC of up to 40% following segmental resection or subtotal colectomy.⁴⁰ 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).⁴¹ 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 patientspecific 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 (\geq 10 mm) lesion size, and non-polypoid morphology.^{34,42-44} Patient-specific risk factors include concomitant PSC, previous dysplasia, significant uncontrolled mucosal inflammation, limitations to adequate mucosal assessment (e.g. colonic stricturing or extensive pseudopolyposis) and a family history of CRC.⁴² 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.^{34,44}

Many patients are understandably reluctant to accept lifechanging 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.^{45,46} A multidisciplinary shared decision-making approach should therefore be used to guide management based on the patient's informed preferences.^{47,48} 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,³⁴ can be used to help predict and communicate individualised cancer risk to patients with UC and LGD (see Figure 3).

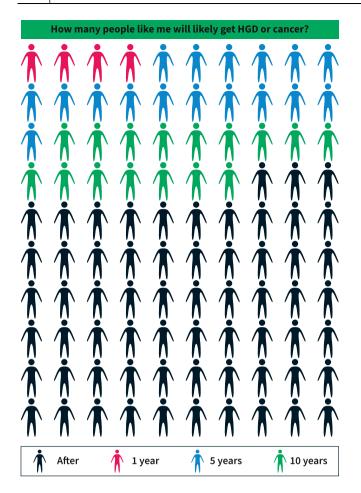


FIGURE 3 Example of a Paling chart derived from the UC-CaRE webtool (www.uc-care.uk) accessed on 27 June 2022, communicating individualised advanced neoplasia risk in a UC patient with LGD and two additional risk factors: multifocal LGD and moderate-severe active colitis. Their 1, 5 and 10-year risk of advanced neoplasia (HGD/CRC) are calculated at 4.6%, 21.1% and 37.7% respectively. CRC, colorectal cancer; HGD, high-grade dysplasia; LGD, low-grade dysplasia

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.⁴² In addition, faecal samples can be used to isolate and analyse colonic epithelial DNA.⁴⁹ 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.⁴²

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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