Endoscopy in inflammatory bowel disease when and why

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Abstract

Endoscopy plays an important role in the diagnosis and management of inflammatory bowel disease (IBD). It is useful to exclude other aetiologies, differentiate between ulcerative colitis (UC) and Crohn's disease (CD), and define the extent and activity of inflammation. Ileocolonoscopy is used for monitoring of the disease, which in turn helps to optimize the management. It plays a key role in the surveillance of UC for dysplasia or neoplasia and assessment of post operative CD. Capsule endoscopy and double balloon enteroscopy are increasingly used in patients with CD. Therapeutic applications relate to stricture dilatation and dysplasia resection. The endoscopist's role is vital in the overall management of IBD.

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INTRODUCTION

Endoscopy is a crucial tool in the management of inflammatory bowel disease (IBD). There is a spectrum of situations when an endoscopy may be of value in IBD, extending from initial diagnosis to differentiating between Crohn's disease (CD) and ulcerative colitis (UC) to long term management of both conditions.

Of the several endoscopic tools, colonoscopy remains the prime diagnostic tool. Gastroscopy, enteroscopy and endoanal ultrasound scan may be useful in the assessment of specific organ involvement in CD and to differentiate between UC and CD. Novel tools such as capsule endoscopy and double balloon enteroscopy have been playing an increasing role for small bowel Crohn's disease assessments. Both CD and UC can be complicated by primary sclerosing cholangitis (PSC): ERCP previously the gold standard to diagnose PSC has broadly been superseded by magnetic resonance cholangio pancreatography[1]. This article will focus on the role of colonoscopy in IBD as this is by far the most important tool. A brief overview of other endoscopic tools will follow.

COLONOSCOPY

Over the years, improvements in colonoscope technology have led to more comfortable procedures with better quality image definition (namely narrow band imaging, chromo endoscopy, endomicroscopy and high definition screens)[2]. Training in colonoscopy has optimised the

Table 1 Infective causes of inflammation which mimic inflammatory bowel disease

Infective cause	Endoscopic appearance
Salmonella	Friable mucosa with haemorrhages in ileum and colon
Shigella	Patchy intense erythema in ileum and colon
Campylobacter	Erythema and ulcers in colon
E.coli 0157:H7	Mild to moderately severe colitis
Yersinia	Patchy colitis with ileal aphthoid ulcers
C.difficile	Pseudo membranes and predominantly left side colitis
Klebsiella	Haemorrhagic colitis
Mycobacterium	Transverse or circumferential ulcers ileum
Neisseria	Proctitis with ulcers and peri anal disease
Chlamydia	Peri anal abscess, ulcer and fistula
Treponema	Proctitis with ulcers and peri anal disease
Schistosoma	Extensive colitis, may be segmental with polyps
Entamoeba	Acute colitis and ulcers
Herpes	Proctitis with rectal ulcers and perianal disease
Cytomegalovirus	Colitis with punched out shallow ulcers
Aspergillus	Ulcers with bleeding
Histoplasma	Predominantly right side colitis

Table 2 Non infective causes of diarrhoea

Inflammatory	Behcet's disease
Drugs	Non streroidal anti inflammatory drugs
	Gold
	Penicillamine
Iatrogenic	Radiation colitis
Vascular	Vasculitis
	Ischaemic colitis
Neoplastic	Colorectal cancer

use of this instrument for various diagnostic purposes. Colonoscopy remains the first line endoscopic investigation for suspected CD. Flexible sigmoidoscopy offers a diagnostic option for UC, with colonoscopy reserved to define the disease limit in some cases. The role of colonoscopy in the management of IBD can be summarised as follows[3,4]: (1) to establish a diagnosis; (2) to assess the disease extent and activity; (3) to monitor disease activity; (4) for surveillance of dysplasia or neoplasia; (5) to evaluate ileal pouch and ileorectal anastomosis; (6) to provide endoscopic treatment, such as stricture dilation/ stent placement.

COLONOSCOPY AS A DIAGNOSTIC TOOL

One of the pitfalls in diagnosing IBD is the failure to consider other diseases, which may give terminal ileal and colonic inflammation. By far the commonest cause of inflammation is infection. Infective causes[5] are outlined in Table 1; the typical features to assist diagnosis are also described. Other conditions that may mimic IBD[6] with colonic and terminal ileum (TI) inflammation are summarised in Table 2.

Once these conditions have been excluded there remains the challenge of differentiating between CD and UC. This activity has important implications for disease management and prognosis. Whilst most cases are straightforward, around 5% of cases particularly with colitis, final diagnosis is evasive and the disease is defined as unclassified IBD[7].

Features of UC
The endoscopic findings of active UC range from erythema, loss of the usual vascular pattern due to oedema, granularity of the mucosa and friability/spontaneous bleeding to erosions/ulceration[8] (Figures 1 and 2).

The ulceration in UC has typical features: superficial

ulcers, which may coalescence to large ulceration extending circumferentially. By virtue of the continuous inflammatory nature of UC, ulcers always surrounded by inflamed mucosa (Figure 3).

Distribution of the inflammation may be helpful in differentiating between UC and other causes of colitis particularly Crohn's colitis. Rectal involvement is invariable with continuous disease extending proximally. Recognised variations to this pattern include rectal sparing, particularly if patients have been using topical therapy, and peri-appendiceal inflammation. Small bowel involvement may occasionally be present in the form of backwash ileitis. This appearance differs from CD: diffuse continuous erythema with no ulceration compared to typical Crohn's appearance[9]. Endoscopic mucosal appearance alone might underestimate the extent when compared to the histological involvement.

Chronic UC may display quiescent disease but changes of previous activity such as post-inflammatory polyps (Figure 4) scarring (Figure 5) and a shortened tubular colon (Figure 6) may be evident. Strictures are rare in UC; its presence heralds a fivefold risk of colorectal cancer (CRC) and such patients should be followed up with care[10].

Disease extent and activity influence medical management: this is reflected in the choice of medical therapy and the route of administration as well as risk stratification of colonic cancer[11]. Hence the importance of recording these finding in endoscopic report cannot be underestimated. Disease extent is recorded as the extent of inflammation from the anal verge; mucosal involvement is not static it can progress or regress over time[12]. Disease activity is recorded as mild, moderate or severe with more than 12 disease activity scoring systems reported in the literature[13]. Commonly used endoscopic indices[14-18] are summarised in Table 3. The score used in most drug studies is the Mayo endoscopic score of activity. The Mayo score ranges from 0 to 12, with higher scores corresponding with more severe disease[19]. An "optimal" scoring instrument for UC is still to be developed and will require validation before extensive use in clinical trials can be promoted[13].

Features of Crohn's disease
Inflammation in CD can involve the entire gastrointestinal tract; 40%-55% of cases show inflammation in the terminal ileum and colon, 15%-25% colonic inflammation alone and in 25%-40% ileum is exclusively
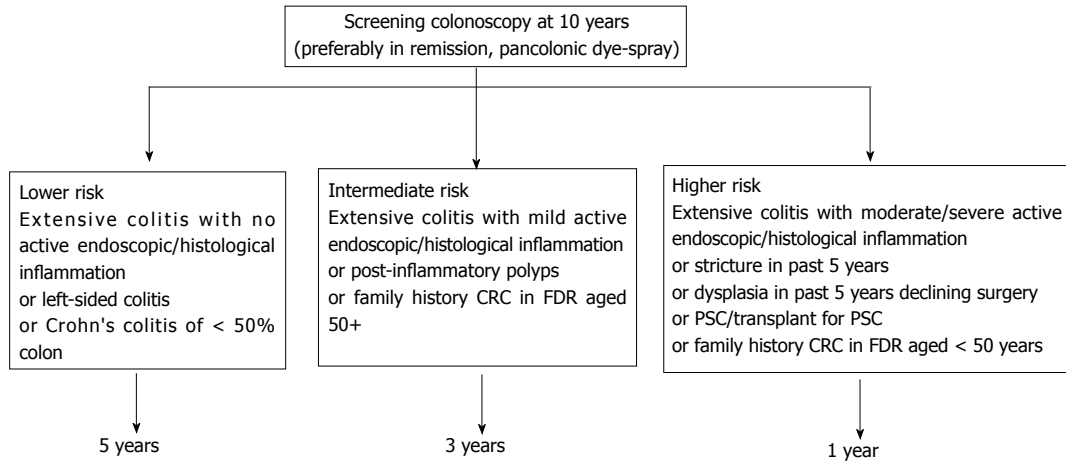
Figure 1 **British Society of Gastroenterology guidelines on surveillance of colitis.** PSC: Primary sclerosing cholangitis; CRC: Colorectal cancer.
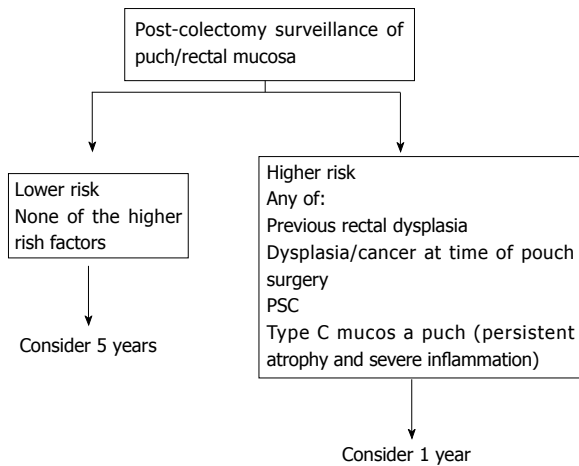

Figure 2 **British Society of Gastroenterology surveillance recommandations post colectomy.** PSC: Primary sclerosing cholangitis.
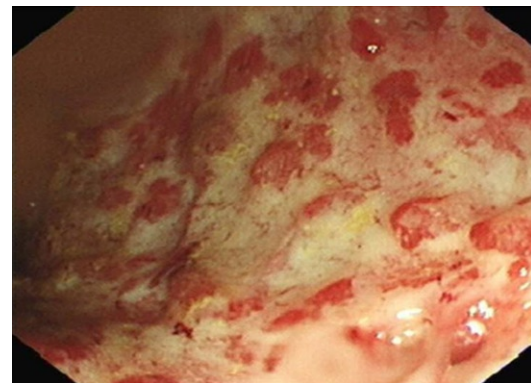

Figure 3 **Severe colitis (Sutherland score 3)**. Friable, granular mucosa with exudates overlying the surface, ulcers and sub mucosal oedema of rectum.

involved[20]. Involvement of oesophagus, stomach and proximal small bowel occurs in up to 10% of CD patients. The rectum is spared in up to 50% patients with colonic disease[6].

The endoscopic hallmark of CD is the heterogeneous patchy nature of inflammation or skip lesions (areas of inflammation interposed between normal mucosa). Ulceration in CD commonly occurs on a background of minimal inflammation[5].

CD ulcers tend to be longitudinal, polycyclic ulcers (snail track) associated with cobblestone appearance of ileum, fistulous tract and strictures either in the colonic or ileum. Circumferential inflammation is rare in CD. The ulcers are deep when compared to superficial ulcers in UC[6] (Figure 7).

The presence of small ulcerations on the ileocaecal valve or within the TI in a symptomatic individual is highly suggestive of CD (Figure 8); the possibility of tuberculosis and nonsteroidal antiinflammatory drug induced ileal ulcers should be considered[21,22]. Young people may have benign aphthous ulceration related to lymphoid hyperplasia which should not be diagnosed as CD[23].

Several activity indices for CD are in use. Most of them are complicated and time consuming. A simple scoring system suitable for clinicians is the simple endoscopic score of CD (SES-CD) which came into use recently. Table 4 summarises the features of SES-CD[24].

Biopsy specimens should be taken from ulcerated mucosa as well as from normal mucosa adjacent to inflammatory areas, in order to demonstrate the skip phenomenon. Biopsy specimens taken from the edges of ulcers and aphthous erosions maximize the yield of identifying granulomas. The practice of collecting biopsies from macroscopically normal rectal mucosa allows the differentiation between a diagnosis of UC in suspected colonic CD[21,22,25]. Table 5 summarises the prime endoscopic differences between UC and CD[26].

MONITORING DISEASE ACTIVITY

The use of colonoscopy as a diagnostic tool is non-contentious. Its value in disease monitoring is an evolving indication for the procedure. The thrust in this direction comes from the more recent focus on mucosal healing or reducing inflammatory activity in IBD. The prognostic implications of mucosal healing include reduced sur-

Table 3 Endoscopic indices used in ulcerative colitis

	0	1	2	3	4
Sutherland	Normal	Mild friability	Moderate friability	Exudates and spontaneous haemorrhages	-
Schroeder	Normal or inactive disease	Mild (erythema, decreased vascular pattern)	Moderate (marked erythema, absent vascular pattern)	Severe (spontaneous bleeding, ulceration)	-
Baron	Normal: matt mucosa, ramifying vascular pattern, no spontaneous bleeding/to light touch	Abnormal, but non-haemorrhagic: appearances between 0-2	Moderately haemorrhagic: bleeding to light touch, but no spontaneous bleeding	Severely haemorrhagic: spontaneous bleeding and bleeds to light touch	-
Feagan	Normal, smooth, glistening mucosa, with normal vascular pattern	Granular mucosa; vascular pattern not visible; not friable; hyperaemia	As 1, with a friable mucosa, but not spontaneously bleeding	As 2, but mucosa spontaneously bleeding	As 3, but clear ulceration; denuded mucosa
Powel-Tuck	Non haemorrhagic, no spontaneous bleeding or bleeding to light touch	Haemorrhagic, no spontaneous bleeding, but bleeding to light touch	Haemorrhagic, spontaneous bleeding ahead of instrument at initial inspection with bleeding to light touch	-	-
Lemann, Hanauer	Normal mucosa	Oedema, +/- loss of vascular pattern, granularity	Friability, petechiae	Spontaneous haemorrhage, visible ulcers	-

Figure 4 Post inflammatory polyp in transverse colon in a patient with ulcerative colitis.

Figure 5 Extensive scarring of sigmoid colon in a patient with long history of colitis.

gical intervention[27], prolonged remission[27], and reduced risk of colorectal cancer[10].

Patients with quiescent disease may have a relatively normal appearing mucosa with a distorted vascular pattern but without friability. Mild disease might appear oedematous and granular with distortion of the vascular markings, moderate activity is defined by the presence of a coarse granular pattern, erosions and friability of the mucosa. Severe disease displays gross ulcerations and areas bleed spontaneously[5]. The presence of severe ulceration is usually associated with refractory disease and increased frequency of complications such as perforation[5].

SURVEILLANCE FOR DYSPLASIA OR NEOPLASIA

Several studies have reported an increased risk for colorectal cancer in UC and Crohn's colitis. This risk has been examined with respect to disease duration and extent[28,29]. The cumulative risk for colorectal cancer was estimated as 1.6%, 8.3% and 18.4% after 10, 20 and 30 years of disease respectively[28]. The associated risk for extent was reported in a population based study as standardised incidence ratio of 2.8 for left sided colitis and 14.8 for

pan-colitis[30]. Risk assessment of CRC also critically relies on endoscopic appearance of the severity of disease activity: both endoscopic and histological inflammation was shown to be associated with increased risk[10,31]. Conversely, in a macroscopically normal colonoscopy the associated cancer risk was observed to be similar to age and sex-matched controls[10]. PSC is an independent risk factor for cancer with an odd ratio for developing cancer of 4.49 (95% CI: 3.58-6.41) compared to patients without PSC[32].

As a consequence of the above observations, colonoscopic surveillance for neoplasia is recommended by most gastroenterology and endoscopic societies. The purpose of surveillance colonoscopy is to identify early pre-malignant lesions indicative of an enhanced risk of CRC. The original literature focused on dysplasia-associated lesions/masse (DALM), however we now have evidence that neoplasia may be flat and subtle. The endoscopic techniques for improving dysplasia detection are discussed here in the later section.

FLEXIBLE SIGMOIDOSCOPY

One of the limitations of colonoscopy is the need for

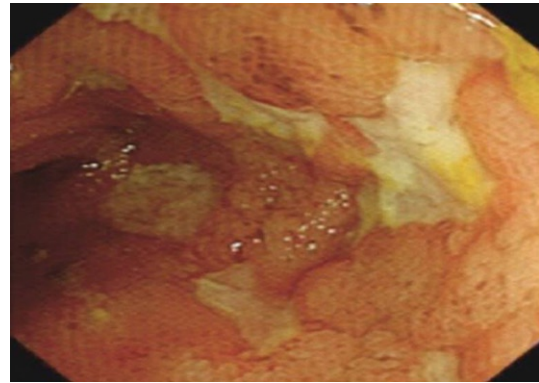**Figure 6 Shortened tubular colon in a patient with pan colitis.**

Figure 7 Deep ulcers, sub mucosal oedema and haemorrhages in the sigmoid colon in a patient with Crohn's colitis.

Figure 8 Multiple linear, deep ulcers with normal islands of intervening mucosa in the terminal ileum indicates severe Crohn's disease.
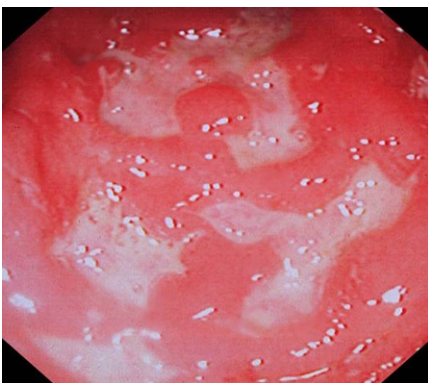
Table 4 Simple endoscopic score for Crohn's disease

Variable	Simple endoscopic score			
	0	1	2	3
Size of ulcers	None	Aphthous ulcers	Large ulcers	Very large ulcers
Ulcerated surface	None	< 10%	10%-30%	> 30%
Affected surface	Unaffected segment	< 50%	50%-75%	> 75%
Presence of narrowing	None	Single, scope passable	Multiple, scope passable	Scope impassable

Table 5 Differences in the macroscopic appearance between Crohn's disease and ulcerative colitis

Macroscopic features	UC	CD
Erythema	+++	++
Loss of vascular pattern	+++	+
Granularity of mucosa	+++	+
Cobble stone appearance	-	++
Pseudo polyps	+++	+++
Aphthous ulcers	+	+++
Deep ulcers	-	+++
Patchy inflammation	-	+++
Ileal ulcers	-	+++
Rectal involvement	++++	++

UC: Ulcerative colitis; CD: Crohn's disease.

oral bowel preparation to enhance adequate mucosal views. In some situations a limited examination of the left colon with flexible sigmoidoscopy may suffice. The procedure may be undertaken following an enema or sometimes-unprepared procedure. Sigmoidoscopy provides useful information in many situations particularly: (1) when colonoscopy is considered high risk or contraindicated e.g., acute severe colitis or fulminant colitis[13]; (2) to define the severity of the disease in established colitis; (3) to exclude superimposed infection with cytomegalovirus (CMV) and C.Difficile; and (4) to exclude other causes for symptoms when there is poor response to therapy e.g., ischaemic colitis.

OESOPHAGO-GASTRODUODENOSCOPY

Oesophagogastroduodenoscopy (OGD) in suspected IBD are recommended in paediatric population where differentiating between UC and CD can be challenging[33]. In adult IBD, there are no specific recommendations. Symptoms of dyspepsia, abdominal pain, vomiting or findings of nutritional deficiency in CD warrant an OGD. Upper gastrointestinal (GI) tract involvement occurs in up to 13% of patients with CD[34]. Moreover a minority of UC patients may also have upper GI tract inflammation, manifesting as diffuse duodenitis or gastritis, characterised by oedema, erythema, ero-

sions, and thickened mucosal folds[35]. OGD with small bowel biopsy in patients with IBD include evaluation of concomitant coeliac disease and small bowel adenocarcinoma[36]. There are therapeutic applications of OGD in patients suffering from CD; symptomatic duodenal or pyloric strictures (Figure 9) can be successfully treated with endoscopic balloon dilation[37] (Figure 10).

CAPSULE ENDOSCOPY

Small bowel capsule endoscopy (SBCE) was first introduced in 2001. Over the last decade it has evolved as a sensitive modality for the detection of small bowel

Figure 9 Linear pyloric ulcer and surrounding sub mucosal oedema-pyloric Crohn's disease.

Figure 10 Balloon dilatation of Crohn's stricture.

lesions including CD. The main advantage of SBCE is the potential to visualise the entire length of the small bowel. It is less invasive and better tolerated. When compared to radiological investigations (CT or MR enterography) it is very sensitive to detect early mucosal lesions. Recent study showed the sensitivity for diagnosis of CD of the terminal ileum 100% by SBCE, 81% MR enterography, and 76% by CT enterography, respectively[38,39].

Recent meta-analysis suggested that SBCE has the highest diagnostic yield in non-stricturing CD (69% SBCE *vs* 30% small bowel Barium follow through) and is significantly superior to the conventional endoscopy or CT/MR enterography for lesion detection. It is particularly useful in patients with established CD to detect disease recurrence[40]. There are drawbacks of SBCE. The main disadvantage is the lack of tissue sampling option. Non-diagnostic mucosal abnormalities may thereafter need to be followed by more invasive (enteroscopy) procedures for histological sampling. Additional drawbacks include obscured view due to debris, non-suitability for patients with delayed transit and the risk of capsule retention in severe stricturing disease[40].

Despite the limitations experts propose capsule endoscopy for monitoring of patients with known diagnosis of Crohn's disease and in detecting post surgical disease recurrence[41,42]. Costs and availability may however mitigate its value in repetitive testing.

ENTEROSCOPY

Double balloon enteroscopy allows a more complete evaluation of the small intestine than single balloon enteroscopy[43-45]. It complements capsule endoscopy particularly when the diagnosis of IBD is uncertain and biopsies are required and for therapeutic interventions namely dilation of small bowel strictures[43,44].

A recent study examined the value of intra-operative enteroscopy to define mucosal inflammation extent as a means of minimising resection length[46]. Intra operative small bowel endoscopy was performed on 33 occasions in 31 patients with CD to compare intraluminal to external inflammation. Endoscopic findings influenced sur-

gical decisions on 20 of the 33 occasions reducing the length of planned resection in 14 cases.

ROLE OF ENDOSCOPY IN SPECIAL SITUATIONS

### *Endoscopic surveillance*
Surveillance for CRC is indicated for patients with IBD: the risks for UC are similar to Crohn's colitis of equal colonic extent and disease duration. Endoscopic appearances are a valuable predictor of future dysplasia and CRC[2]. Rutter *et al*[10] showed that post-inflammatory polyps, strictures, shortened colons and tubular colons were associated with increased risk for future neoplasia with respective odds ratios of 2.14 (95% confidence interval 1.24-3.70), 4.22 (1.08-15.54), 10 (1.17-85.6) and 2.03 (1.00-4.08). No significant association was found with the presence of backwash ileitis, scarring, or a featureless colon.

The British Society of Gastroenterology (BSG) guidelines propose that patients with UC or Crohn's colitis should have a colonoscopy 10 years after the initial diagnosis to define the extent and activity of the disease[7]. Surveillance colonoscopy should be undertaken preferably in remission. The following risk factors dictated the risk and frequency of future surveillance procedures: disease duration and extent associated primary sclerosing cholangitis, family history of sporadic colorectal cancer, young age at diagnosis and endoscopic and histological appearance during colonoscopy[5,9,10]. Screening interval depends on the above risk factors and according to the national and international guidance. Figures 1 and 2 illustrates the summary of current BSG guidelines[7].

Several studies have shown improved detection rates for dysplasia and cancer if targeted biopsies are taken rather than random biopsies[10]. This approach may serve to mitigate the poor clinician compliance to endoscopic protocols for random biopsies every 10 cm[47]. Narrow band imaging has been shown to be no better than standard white light colonoscopy and hence cannot be recommended as an alternative to chromo endoscopy[7]. Although confocal endomicroscopy may enhance the
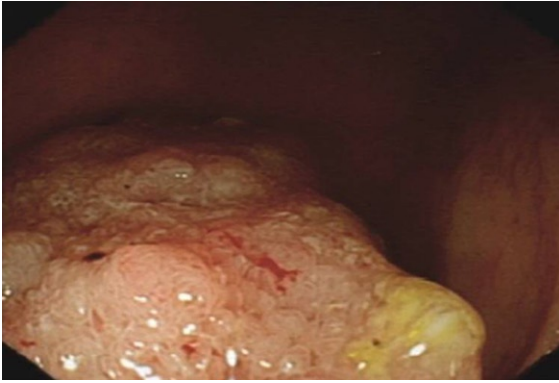
Figure 11 Dysplasia-associated lesions/masse in caecal pole in a patient with a long history of pancolitis.
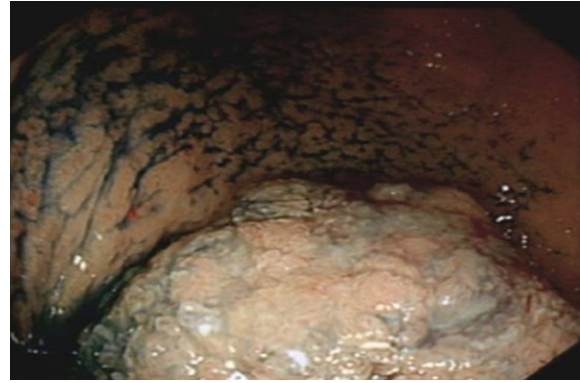
Figure 12 Dysplasia-associated lesions/masse in caecal pole after dye spray.

in vivo characterisation of lesions, it requires prior lesion detection by other means before confocal endomicroscopy can be deployed[2]. Therefore pan colonic dye spray (either with methylene blue or indigo carmine) with targeted biopsies is now recommended[7]. Intuitively such an approach may be expected to be time consuming however the colonoscopy duration was not shown to differ to standard colonoscopy[10]. A recent study by Saunders *et al*[48] described a time-saving technique using a washer pump for dye spray application: indigo carmine was successfully applied to the entire mucosal surface and reduced the procedural time by several minutes while optimising mucosal views and biopsy access.

Most cancers arise with pan colitis; there is little or no increased risk associated with proctitis and left-sided colitis carries an intermediate cancer risk[13]. There is evidence to indicate that colorectal cancer is also more likely to develop with persistent colonic inflammation even in microscopic level[2]. Hence, active inflammation noted at surveillance colonoscopy, is an indication for escalation of medical treatment.

When a dysplastic polyp is detected, it is essential to biopsy the adjacent flat mucosa at the base of the dysplastic polyp to assess the extent of disease and also to detect dysplasia in the surrounding (macroscopically normal) flat mucosa. This may help to differentiate between adenoma-like lesions (ALM) or the traditionally described DALMs[49] (Figures 11 and 12). The swathe of literature pertaining to the management of dysplastic lesions has been summarised in several review articles and lies beyond the scope of this article.

Endoscopic assessment of pouchitis
Pouchitis has been reported as a complication of restorative proctocolectomy for UC in as many as 40%-50% of patients[50]. There are no specific symptoms and signs for pouchitis, which may be similar to other pouch complications such as cuffitis, irritable pouch and CD of the pouch. Furthermore, severity of symptoms does not always correlate with the endoscopic or histological findings and the disease activity is variable with time. Therefore a cumulative assessment of clinical, endoscopic and

histological assessment is needed to make the diagnosis of pouchitis[51,52].

Pouch endoscopy (pouchoscopy) provides crucial information with respect to the severity and extent of mucosal inflammation, pre-pouch ileitis and CD of pouch and cuffitis. It also demonstrates other abnormalities such as polyps, strictures, sinuses and fistula. Supplemental information from histology may reveal granulomas, CMV inclusion bodies and dysplasia[51,53-59]. Several diagnostic criteria are available and the commonest in clinical use is the pouch disease activity index[60].

Postsurgical crohns disease
Ileal or ileocolonic CD (Montreal L1 or L3) affects 75% of the Crohn's population[6,20]. In this selected group of patients remission may be achieved by medical or surgical means with a right hemi-colectomy. The latter procedure may also be required for complications particularly strictures and penetrating disease with fistula formation.

Disease recurrence in the neo-terminal ileum is invariable. Rutgeerts' group reported endoscopic, clinical and surgical recurrence rates of 73%, 20% and 5% at 1 year respectively[12]. We reported similar rates at our centre for clinical and surgical recurrence in a retrospective series of 99 patients following surgery (28% clinical and 5% surgical recurrence at 1 year)[61]. The Rutgeerts scoring system is proposed as a means to predict post-surgical recurrence risk[62] (Table 6). The predictability of future clinical recurrence was based on neo-terminal ileal endoscopic appearances (Figure 13) at one year, with a greater risk for scores > i2[12].

Other clinical and histological risk factors for disease recurrence have been identified. The evidence for smoking is the most compelling[63]. Additional clinical factors are disease behaviour with perforating disease and previous resection for CD. Plexitis in the proximal margin of resection specimens implies more aggressive disease and greater recurrence risk[64].

Post-surgical colonoscopic examination of ileocolonic anastomosis (Figure 14) is a valuable predictor for risk of recurrence and may identify patients in need of medical therapy escalation. The optimal time interval

Figure 13 Aphthous ulcer in the neo terminal ileum in a patient who had ileorectal anastomosis(indicates recurrence of Crohn's disease). Note healthy surrounding mucosa.

Figure 14 Pin hole stricture in the neo terminal ileum (Crohn's disease).

between surgery and colonoscopy is not known. At our centre we undertake the first colonoscopy at 6 mo. We also proposed a risk stratification of patients based on their risk, with prophylactic medical therapy directed at the risk[65-70]. Figure 3 illustrates the proposed postoperative surveillance strategy.

ROLE OF THE ENDOSCOPIST

Ultimately, it is the endoscopist's interpretation of endoscopic findings that underpins clinical decisions and not endoscopic technological advances. Other than the appropriate choice of endoscopic test to answer the relevant clinical questions, there is an additional responsibility on the endoscopist to recognise and comprehensively record mucosal abnormalities. By assimilating these findings with the clinical presentation a diagnosis is often achieved and a management plan generated. Emphasis on clear, accurate and systematic reporting is paramount particularly when the endoscopist is not the treating physician. Therefore to ensure accurate communication of findings, a simple check list for reporting diagnostic or prognostic colonoscopies should include the following descriptions.

Table 6 Rutgeerts scoring system to monitor post surgery Crohn's disease activity

Score	Endoscopic features
i0	Absence of any lesions at anastomosis and in the neo terminal ileum
i1	Less than 5 aphthous ulcers (< 5 mm)
i2	More than 5 ulcers with normal intervening mucosa or large patchy lesions, or lesions confined to anastomosis (< 1 cm)
i3	Diffuse aphthous ileitis with diffuse inflammation of the ileal mucosa
i4	Diffuse ileitis with large ulcers, nodularity and stenosis.

Mucosal appearance
Appearance should be described in detail focusing on loss of vascular pattern, ulcers size, depth and extent of circumference, haemorrhages and fistula. Distribution of abnormal mucosa should include description of continuous or patchy inflammation, rectal and non-rectal involvement, peri-appendiceal involvement and TI changes.

Disease extent
Describe the extent of disease involvement for instance in UC it is expressed as inflammation distance from the anal verge and in CD length of inflamed segments.

Image labelling
Capture appropriate images of abnormal mucosa and label correctly.

Specimen collection
Collect and correctly label histology specimen. Ensure adequate number of biopsies are taken to increase the yield of histological diagnosis: current consensus is at least two biopsies from five sites including ileum and rectum[7]. Biopsies should be taken from areas of inflammation and the adjacent mucosa proximal to the area of inflammation.

When colonoscopy is undertaken for refractory or acute severe disease the following points must be considered: Alternative diagnosis (ischemia, drug induced, vasculitis, un-related infection); Complications of CD or UC (CMV or Clostridium difficile colitis or neoplasia or fistula formation).

Finally, good communication between endoscopist and histopathologist is mandatory for final decision on diagnosis. This may be achieved through regular multidisciplinary team meeting or attaching the detailed colonoscopy report to all pathology requests.

CONCLUSION

Colonoscopy is one of the most important diagnostic and prognostic tools in the diagnosis and management of IBD. Other endoscopic procedures usually supple-

ment colonoscopy for additional information or treatment of the disease. Management relies on interpretation of endoscopic findings, therefore good knowledge of the various mucosal appearances, descriptions and the implication of each finding, with careful attention to recording each finding is crucial to the optimal management of patients. Surveillance roles for colonoscopy involve optimising the procedure particularly in cancer surveillance and post-operative CD. Therapeutic applications of endoscopy are related to excision of dysplastic lesions and dilatation of strictures.

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