Reporting Colonic Biopsies in Patients With Inflammatory Bowel Disease; a Practical Approach

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Crohn's disease (CD) and ulcerative colitis (UC) are common diseases; for convenience, we lump them together as inflammatory bowel disease (IBD). Colonic biopsies are essential for establishing a diagnosis, monitoring treatment, and/or identifying complications. This article attempts to detail the histological criteria of UC and CD and also informs on a lifelong approach of reporting colonic biopsies from patients with IBD. Often the clinical information on the accompanying request form is not available to the pathologist. In these cases, the author has initiated a reproducible system of pattern-based reporting with a differential diagnosis. This type of report offers a working diagnosis for the clinician before the final diagnosis, which is recommended to be undertaken in a setting of combined clinico-pathological conference (CPC). This system carries objective parameters and standardizes reporting to significantly minimize the interobserver variations among reporting pathologists. If a CPC facility is not available, we offer an alternative evidence-based arrangement. We discourage the use the term "nonspecific colitis" as we have shown that it has no clinical value or agreed-upon and recognized histopathological features. As the paper addresses mucosal biopsies, the entity of indeterminate colitis will not be included in this article as this diagnosis is strictly based on colonic resectate.

Key Words: IBD, histopathology reporting, new system

INTRODUCTION

The continuous development of the colonoscopy technology has enabled clinicians to have a better view of the colon and provided pathologists with more material.[1] How can this material be best used? We maintain that, in addition to good biopsy specimens, there are 4 important factors that help to ensure clear communication between the pathologist and the treating clinician to reach a more accurate histopathological diagnosis. These are:

1. standardizing histopathological terminologies;
2. being aware of the range of histopathological features in colonic biopsies in inflammatory bowel disease (IBD) patients and the spectrum of "normality";
3. encouraging procurement of adequate information for the histopathologist;
4. issuing an initial pattern-based report with differential diagnosis, followed by a final diagnosis taken in a combined clinico-pathological conference (CPC) environment.

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TERMINOLOGY

Most abnormal colonic mucosal biopsies do not provide a specific etiological diagnosis but rather show a pattern of injury.

These patterns can be any 1 or a combination of the following:

Diffuse Injury

This means that the entire or most of the specimen is abnormal. In the clinical setting of IBD, this feature can be seen in both ulcerative colitis (UC) and Crohn's disease (CD).

Focal Injury

Focal injury occurs when 1 part of the specimen (or 1 fragment) is entirely normal and another part (or another fragment) is abnormal or the abnormality is less than 50% of the specimen size. In the clinical setting of IBD, this feature favors CD over UC but can be seen in ischemic colitis and radiation injury.[2, 3]

Activity of the Disease

This is reflected by the degree of acute inflammatory cell infiltrate with or without crypt abscess/cryptitis. This is usually associated with a degree of mucous depletion, which relates to the degree of disease activity reflected by the presence of inflammation, ulceration, degeneration, and regeneration of the mucosa. Care needs to be taken because histological features of chronicity in IBD may be masked in acute relapses or by superimposed infection. It is important to note that the presence of activity has no bearing on disease categorization and has no diagnostic value as it can be seen in many forms of

colitides. For example, crypt abscesses can be seen in UC, CD, or infection.

Features of Chronicity

These include crypt distortion, crypt atrophy, fibrosis of the lamina propria, significant increase in chronic inflammatory cell infiltrate, particularly situated deeper in the mucosa, ilealization of the colonic mucosa (papilliform configuration of the surface epithelium), Paneth cell metaplasia, ulcer lineage cell (pseudo pyloric) metaplasia, and dysplasia.

Extent: Continuous or Discontinuous

Specimens from different areas should be submitted separately to show the extent and topography of the disease and whether the process is continuous or discontinuous. If some biopsies are normal, it is important to document those. Discontinuous injury can be seen in ischemic colitis, CD, UC with cecal patch, and pseudo membranous colitis. It is important to biopsy endoscopically normal-looking areas. Normal endoscopy is not synonymous with normal histology. Sometimes we may identify histological abnormalities in these endoscopically normal areas, like granulomas or subtle features of chronicity.

The site of the biopsies should be marked on the colonoscopy report, labeled, and sent separately. Sending biopsies from different regions in 1 specimen jar will not help to "map out" the disease topography if 1 or more fragments are found at variance with the others. This practice is also very important in cases of dysplasia.

Presence of Granulomas

There are 2 main types of granulomas seen in different conditions. We consider sarcoid-like granulomata to be diagnostic of CD only with the appropriate clinical picture. We emphasize the point that Crohn's disease is a clinicopathological diagnosis. Crohn's granulomas are tight, neat collections of histiocytes seen more often in the submucosa than the mucosa, and that is why the deeper biopsies in CD are more informative. The other type of granulomas are the so-called "leakage" granulomas. These are in response to the leakage of mucous from the crypt to the interstitial tissue (lamina propria) when the crypt is destroyed totally or in part by acute inflammatory cells. This process of cryptolysis leads to mucous seepage to the lamina propria to provoke a granulomatous response.[4–6] Unlike Crohn's neat granulomata, leakage granulomata are often associated with neutrophilic and eosinophilic infiltrate.

On occasions, "bare" multinucleated cells are seen in the lamina propria in cases of UC; they should not be confused with microgranulomata in CD.[6]

Identification of Microorganisms

It is possible to identify in colonic biopsies microorganisms such as cytomegalo virus (CMV), mycobacteria, amoeba, and Schistosoma. CMV identification in particular is important to bear in mind in cases of flair-up of IBD symptoms. The characteristic intranuclear inclusions can be seen in the epithelium, endothelium, or in the stroma of the lamina propria. Although the picture is almost pathognomonic, immunohistochemistry is important for confirmation before starting antiviral treatment.

HISTOPATHOLOGY OF IBD

Histopathology of UC

The histopathology of UC is characterized by diffuse and continuous inflammation limited to the mucosa. Classically, the rectum is nearly always involved with a variable degree of proximal extension. The inflammation is transmucosal, with dense chronic inflammatory cell infiltrate in the lamina propria, which when the disease is active is associated with a variable degree of neutrophilic infiltrate. In more severe cases, there could be mucosal ulcerations. In chronic cases, there is a preferential basal plasmacytosis, crypt distortion in the way of branching or atrophy, and Paneth cell and ulcer lineage cell (pseudopyloric) metaplasia. The latter metaplasia is in response to frequent ulceration and the healing of such ulcers. Crypt abscesses are commonly seen, sometimes with leakage granulomas. In areas of ulceration, the inflammation extends to the underlying tissue, and this should not be confused with CD, in which the inflammation is transmural without ulceration.

Other Histological Variants of UC

Nonclassical features of UC present a potential diagnostic pitfall. Classically, UC extends continuously from the rectum and can involve a variable length of colon. However, discontinuous disease may be seen, and this finding does not exclude a diagnosis of UC.[7] The association of left-sided colitis with inflammation surrounding the appendiceal orifice, also called "cecal patch," has been described by many workers and was found to be present in up to 75% of patients with distal disease.[8] There is no clear message from various workers on whether the cecal patch entity is associated with a more severe form of UC. Mucosal healing, particularly after topical treatment with steroid enemas or oral anti-inflammatories, such as 5-ASAs or steroids, can produce apparent rectal sparing or a patchy mucosal appearance with almost complete reversion to normal, especially after the passage of time.[7–11] Apparent rectal sparing is also recognized in fulminant UC, where inflammation of the transverse colon is so severe as to make the rectum look comparatively spared. Rectal sparing is also seen in some pediatric presentations of UC.[12]

As ileal biopsies are becoming increasingly common, some have abnormalities. Backwash ileitis is thought to occur due to retrograde flow of bowel contents secondary to the ileocecal valve being rendered incompetent by inflammation, although other factors such as infection and drugs may play

a role.[13] It is present in less than 20% of all UC cases but with more than 90% of these showing pancolitis. Unlike CD ileitis, the inflammation is restricted to the first few centimeters of terminal ileum and histologically shows mild patchy neutrophil infiltration of the lamina propria, focal cryptitis/crypt abscesses, and mild villous atrophy.[7, 13]

Ulcerative colitis can rarely be associated with inflammation in the upper gastrointestinal (GI) tract, principally the stomach, in the form of focal gastritis or duodenitis. This may be the source of some confusion as until recently the presence of upper gastrointestinal involvement was regarded as a useful pointer toward a diagnosis of CD, where involvement of any of the GI tract from mouth to anus can be part of the presentation.[7, 14–16]

Other features formerly thought to be exclusive to CD are now also recognized to occur rarely in UC. Aphthous ulceration is seen in up to 17% of UC.[4, 5]

Histopathology of CD

Crohn's disease is a transmural disorder that affects the entire GI tract from mouth to anus and is associated with various extraintestinal manifestations, for example, ocular inflammation, arthritis, and biliary disease. A significant part of the diagnosis is the distribution pattern of the disease, which is classically patchy with skip lesions macroscopically and focal microscopically. The terminal ileum is the most commonly involved site, and the rectum is usually spared. Perianal disease in the form of skin tags, fistulae, abscesses, and blind sinus tracts is present at some point in 75% of cases.[5, 7, 17] The inflammation is transmural and patchy with focal crypt architectural distortion, basal plasmacytosis, and epithelioid granulomas unrelated to cryptolysis. Unlike UC, deep-seated lymphoplasmacytic infiltrate in the lamina propria is not a distinctive feature, but the presence of many lymphoid follicles with less crypt distortion favors CD. From a purely practical standpoint, there is no histological feature in UC that is not seen in CD. The reverse is not true. The degree of crypt distortion is less noticeable than in UC, but this feature is not always useful in making the distinction between UC and pseudopyloric metaplasia, which is seen more in cases of CD. As the disease is transmural, deeper biopsies are more informative. Often biopsies from perianal skin and skin tags reveal more granulomata than colonic mucosa in cases of CD.

Variants of CD

Often the distinction between CD and UC from single colonic biopsies is difficult in the best hands. It does get significantly better with multiple biopsies, particularly in CD.[18] It is estimated that CD is limited to the colon in 14% to 32% of cases. The Montreal classification recognizes that CD limited to the colon (L2) may be a distinct subtype.[19, 20] In these cases, usually only the superficial mucosa is involved in a UC-like pattern, with little or no submucosal or transmural inflammation. The presence of granulomas is variable. Diagnosis is usually made on colectomy specimens, with other CD-associated features such as fat wrapping, granulomas, skip lesions, and adhesions being variably present. Soucy et al.[19] in their study of 16 cases found that overall there were no clinical or pathological differences between colon-limited CD and classical CD, except that the mean age of onset was younger in colon-limited CD (23 years vs 35 years) and that, notably, 50% of cases of colon-limited CD had left-sided disease.

Value of Clinical Information in Assessing Colonic Biopsies

The notion that UC and CD can always be differentiated from mucosal biopsies with limited or no clinical information is answered in the following important studies: In 1 classical study,[21] single slides from confirmed IBD cases (34 UC, 24 CD) mixed with 18 normal cases circulated to 10 experienced GI pathologists, who interpreted the same cases of colonic and rectal biopsies. No clinical information was provided. They found that CD was often and consistently thought to be UC. More worryingly for normal slides, the term nonspecific colitis was often applied without any consistency.

In a separate study[22] aimed to assess the observer variability in the histopathological reporting of abnormal rectal biopsy specimens, 60 rectal biopsies from patients who presented with bloody diarrhea were circulated to 11 general consultant pathologists and were examined with no further clinical information. There were 41 patients with IBD, and the others had various other conditions. They found that clinicians' ability to distinguish CD from UC was poor; so was the case with infective colitis.

A third study to determine the effect of a single vs multiple biopsies on the accuracy of diagnosis showed that multiple biopsies significantly improved the diagnosis in cases of CD, and to a lesser extent in patients with UC.[18]

From the above studies, it is obvious that lack of clinical information may lead to inconclusive reports or even wrong diagnoses.

This in contrast to the study from McBroom et al.,[23] who factored in the value of CPC, where all information is available; they showed a major change in the clinical management in 26.5% of cases and a minor change in the clinical management in 14.7% of all cases reviewed in the CPC.

The other study that is worth mentioning concerns the justifiability of using the label of nonspecific colitis. This study was conducted between 2 separate departments.[24] The aim was to investigate the diagnostic reproducibility of the term nonspecific colitis and to assess its clinical relevance. Rectal biopsies from 35 patients who presented with acute diarrhea and were labeled histopathologically in 1 department as chronic nonspecific colitis were reviewed by 1 pathologist from a different department who was blinded to the clinical information. Full clinical follow-up was not initially available to the assessing

blinded pathologist and was subsequently set against the reclassified results. The biopsies were reclassified by the blinded pathologist as: 13 normal, 7 active inflammation with no features of chronicity, 12 with features of chronicity, 2 hyperplastic polyps, and 1 case with the typical histological features of solitary ulcer syndrome. After breaking the code, they found that none of the 13 cases reclassified as normal needed any clinical intervention and that none subsequently developed colonic disease. The authors concluded that chronic nonspecific colitis was used to cover a heterogeneous group of diseases and normal biopsies. These authors were not different from the "experts" in GI pathology, as in the Theodossi et al. study[21] mentioned earlier, and we suggest that the term nonspecific colitis is meaningless and should be dropped. Subsequently, the British Society of Gastroenterology adopted the concept and advised against using the term Non Specific Colitis.

It has become obvious from the previous studies and others that clinical information is essential to reaching a diagnosis irrespective of the expertise of the pathologists, that multiple biopsies are better than a single biopsy in differentiating UC from CD, and that the term nonspecific colitis should be abandoned.

It is evident that the colonic mucosa responds to various injuries in a limited pattern. Some diseases share 1 pattern, and sometimes a single disease assumes different patterns. Herein lie the difficulties in distinguishing 1 disease from the next based on "naked" biopsies unaccompanied by clinically relevant data.

Adequate Clinical Information

I feel that the final diagnosis of IBD is the shared responsibility of the pathologist and the treating clinician because the diagnosis of most forms of colitides, such as CD and UC, and their differentiation from other conditions, is a clinicopathological decision.[25–28] Ideally, the following information needs to be available to the pathologists before initiating the report:

1. Duration of illness: A long history of colitis may influence the pathologist to consider chronic IBD, even in the absence of histological features of chronicity. Histological features of chronicity may appear significantly after the first presentation.[26]
2. Microbiological examination of the stool: This is important not only because it may establish the cause of the disease as a case of infective colitis, but because the presence of infection may complicate and modify the picture of an already existing IBD like *Clostridium difficile*, CMV, and, less often in the Western world, amoebiasis. Indeed, it may be important to test the hypothesis, which states that an initial infection may trigger, at least in some patients, the process of chronic IBD. We feel that the first or baseline biopsy is useful to keep as a permanent record of the initial stage of the disease, which may or may not evolve into chronic IBD.
3. Endoscopic findings: This informs the pathologist of the extent and topography of the disease. In addition, some lesions have characteristic histological features that are not always matched by their endoscopic appearance, like pseudomembranous colitis.[27] The authors strongly recommend that a copy of the colonoscopy form be sent to the pathologist with the request form.
4. Radiological appearance: This complements the endoscopic features and may indicate small intestinal involvement, dilated transverse colon in a case of toxic megacolon, or features of ischemia or obstruction. Again, ideally, the radiology report should be available to the pathologist.
5. Other relevant information: It is useful to know, for example, whether the patient has traveled from an endemic area of infective colitis. Is there a history of treatment by antibiotics, chemotherapy, radiation, or heavy metals? In some cases, patients may have been operated upon, and a history of previous surgical procedure is important. For instance, diversion colitis and pouchitis are often indistinguishable from IBD.

How Should the Pathologist Report be Constructed?

The histopathology report should attempt to reach a positive histological diagnosis, but this may not be possible, and a differential diagnosis may have to be included.

Often the clinical information is not available to the clinician at the time of submitting the biopsy, or indeed, the treating clinician does not appreciate the importance of providing such clinical data and therefore the request form arrives with little or no information.

This lack of relevant clinical information may lead the pathologist to produce a report that is neither informative nor inaccurate.

In our practice, once we receive the biopsy, we issue a pattern-based report with a working differential diagnosis. The second phase is ideally conducted in a CPC setting where the outcome of the histopathological analysis will be added to the radiological features, the microbiological findings, and the clinical impression to arrive at a final diagnosis. We suggest that close clinicopathological correlation is essential to improve the management of patients with IBD. In patients where there is active diffuse or focal inflammation with no features of chronicity, it is not always possible to differentiate between IBD in the early stage of evolution and transient or infective colitis. In these circumstances, it is highly advisable to repeat the biopsy in 6 weeks' time, for in our hands, the biopsies of patients with transient and infective colitides will revert back to normal.

Examples of conclusions of the reports after analyzing each specimen separately:

1. There are good portions of colonic mucosa showing continuous diffuse, moderately active inflammation with features of chronicity manifested by crypt distortion, Paneth cell metaplasia, and villiform configuration. There is no evidence of CMV inclusions, granulomata, dysplasia, or neoplasia. In the clinical setting of IBD, the appearances are those of chronic active pancolitis with no distinguishing histological features and are compatible with UC or CD.
2. These are small and superficial portions of colonic mucosa showing focal discontinuous, moderately active inflammation with no features of chronicity. There is no evidence of CMV inclusions,

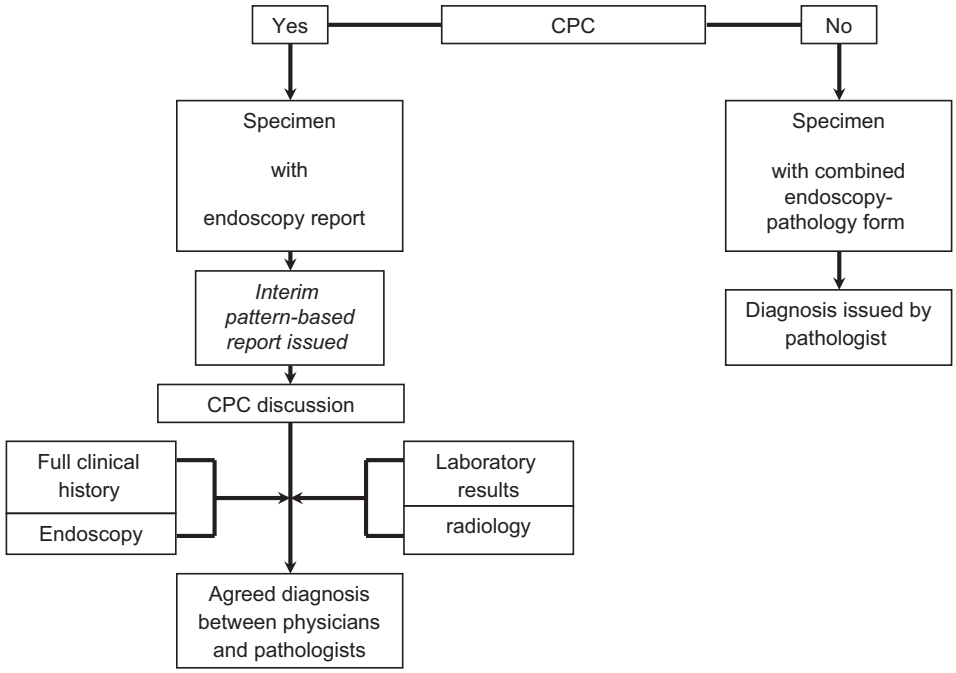

FIGURE 1. Suggested algorithm in diagnosing colonic biopsies in IBD patients (modified from Woodman et al.).[30]

granulomata, dysplasia, or neoplasia. The appearances are those of active discontinuous colitis with no distinguishing histological features and are compatible with CD in the early stage of evolution or infective or antibiotic-related colitides. A second set of biopsies in 6 weeks' time is suggested if still clinically indicated.

In practices where CPC is not available, we designed a combined endoscopy and pathology proforma rich in clinical and endoscopic information, which when implemented affected a significant increase in the definitive diagnostic rate.[29] In this prospective study, 77 cases of inflamed colons were biopsies. In the initial microscopic examination, 23 cases were classified as IBD unclassified. With the use of the combined form, the unclassified group of IBD decreased to 10 cases, and in the CPC, 2 additional cases that were previously not classifiable were reclassified as UC. We concluded that the combined form is an excellent substitute for a CPC diagnosis but still falls short of a final diagnosis in a CPC setting.

In summary, we emphasize the following points:

1. Most biopsies from patients with colitis have no pathognomonic diagnostic histological features; therefore, they have to be interpreted with the full knowledge of the clinical endoscopic, radiological, and laboratory data.
2. The histopathology report is based on describing histological patterns rather than a definitive diagnosis but offers a preliminary working differential diagnosis. The pathology report will allow the clinician, using the known clinical information, to make an appropriate management decision.[28]
3. It is highly desirable to have regular clinicopathological meetings to establish a final diagnosis.
4. In the absence of CPC, we suggest using an information-rich combined endoscopy–histopathology proforma (Fig. 1).[30]

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