Surveillance in Inflammatory Bowel Disease

Chromoendoscopy and Digital Mucosal Enhancement

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KEYWORDS

• Dysplasia • Colitis • Inflammatory bowel disease • Colonoscopy
• Chromoendoscopy • Narrow-band imaging • Autofluorescence imaging

KEY POINTS

- Patients with long-standing inflammatory bowel disease have an increased risk of developing colorectal cancer. Performing periodic dysplasia screening and surveillance may diminish this risk.
- Current surveillance practices, the mainstay of which is white-light examination with targeted and random biopsies, are imperfect, and novel approaches are needed.
- Various advanced endoscopic techniques have been studied in an effort to improve the efficacy and efficiency of dysplasia detection.
- To date, chromoendoscopy is the only technique that has consistently yielded positive results in large, well-designed dysplasia-detection trials. Most major society guidelines endorse chromoendoscopy as an adjunctive, accepted, or preferred dysplasia-detection tool.
- Narrow-band imaging, Fuji Intelligent Chromoendoscopy, i-Scan, autofluorescence imaging, and confocal laser endomicroscopy have yielded conflicting outcomes and are not ready for use in clinical practice.
- The widespread use of advanced endoscopic imaging will lead to a paradigm shift in the way gastroenterologists diagnose and treat dysplasia in inflammatory bowel disease.

INTRODUCTION

Inflammatory bowel disease (IBD), including Crohn disease (CD) and ulcerative colitis (UC), results from an inappropriate inflammatory immune response to normal intestinal microbiota in a genetically susceptible host.[1] IBD involving the colon predisposes

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patients to numerous clinical consequences, including an increased risk of developing colorectal cancer (CRC). The precise risk of cancer, which in the past may have been overestimated because of reliance on outdated evidence, remains unclear, with more recent studies estimating the relative risk of CRC in UC to be between 1 and 2.75.[2] Meta-analyses have shown that duration and extent of disease greatly affect the risk of neoplasia, with nearly one-fifth of patients developing cancer after 30 years.[3] Although most of the literature on neoplasia in IBD is based on data from studies of UC, the risk of cancer appears to be similar in Crohn colitis if at least one-third of the colonic mucosa is involved.[4]

The progression to carcinoma in IBD likely stems from chronic inflammation of the colonic mucosa. There are considerable data supporting the hypothesis that carcinogenesis in IBD typically follows a stepwise pattern from inflammation, to dysplasia, to carcinoma.[5] This pattern provides a rationale for screening and surveillance practices aimed at identifying neoplasia at an early stage. Although there have been no randomized controlled trials demonstrating a mortality benefit, there is indirect evidence justifying dysplasia screening and surveillance in IBD. Retrospective studies have demonstrated that colonoscopic surveillance decreases CRC-related mortality in patients with UC. In addition, having undergone 2 or more colonoscopies offers even more protection.[6,7] Based on such studies, periodic colonoscopic dysplasia surveillance is currently considered the standard of care for all patients with long-standing UC and Crohn colitis.

Although the practice of colonoscopic surveillance is widely accepted, its specific implementation can be controversial. At present the most common surveillance technique involves white-light endoscopic (WLE) examination of the colon with resection or biopsy of any suspicious lesions, as well as random 4-quadrant biopsies taken every 10 cm throughout the length of the colon. The rationale for obtaining nontargeted biopsies is based on the observation that dysplasia in IBD can be difficult, or impossible, to detect using standard endoscopic equipment. To achieve adequate sensitivity for dysplasia detection in flat colonic mucosa, it has been estimated that between 33 and 64 random-biopsy specimens must be obtained at colonoscopy.[8]

However, it is important to point out that most data supporting the random 4-quadrant biopsy methods predate modern endoscopic equipment, and numerous recent studies have called this technique into question for several reasons. First, obtaining the requisite 33 random biopsies still evaluates only a small fraction of the colonic mucosa. Second, practicing gastroenterologists may not strictly follow the random-biopsy protocol. A survey of more than 300 gastroenterologists in the United States found that nearly half of the respondents took less than the recommended number of biopsies,[9] which may stem from the significant time and cost associated with the protocol. Third, the random-biopsy protocol has repeatedly proved to be a very low yield technique. Van den Broek and colleagues[10] reported a dysplasia-detection rate of 0.2% for random biopsies compared with 23% for targeted biopsies. Moreover, only 1 of 475 (0.002%) patients had a change in management based on the results of a nontargeted biopsy. Fourth, in accordance with the findings of van den Broek, investigators are finding that most dysplastic lesions in IBD are in fact detectable using modern endoscopic modalities.[11] Finally, there is evidence that the rates of CRC and dysplasia may be decreasing in UC patients overall.[12] This smaller, more elusive target may further limit our ability to detect existing neoplasia using outdated techniques.

Frustration with current surveillance practices and a sense that the dysplasia target has shrunk have led investigators to seek more innovative ways of approaching this problem. Our growing understanding of the natural history of dysplasia and CRC in