Surgical Management of Nonpolypoid Colorectal Lesions and Strictures in Colonic Inflammatory Bowel Disease

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KEYWORDS

• Colorectal cancer • Inflammatory bowel disease • Dysplasia
• Nonpolypoid colorectal neoplasm • Stricture • Ulcerative colitis • Crohn's disease

KEY POINTS

- Patients with inflammatory bowel disease and dysplasia have pathologic characteristics and risks that differ from those of patients with sporadic carcinomas.
- Surgical interventions need to be more aggressive than in sporadic cases.
- An algorithm for management strategies for lesions and strictures in Crohn's disease and ulcerative colitis needs to be developed.
- A better understanding of the risks and benefits of surgical procedures for dysplasia in Crohn's disease and ulcerative colitis is required.

BACKGROUND

Colorectal cancer (CRC) arising in inflammatory bowel disease (IBD) accounts for only 1% to 2% of all general CRC cases per year. However, as CRC results in 15% of all IBD deaths, cancer screening requires special vigilance in this group. Particularly concerning is the fact that cancers in patients with ulcerative colitis and Crohn's disease often present not as mass lesions but as dysplasia, strictures, or diffuse dysplasia.

The risk of CRC in ulcerative colitis (UC) has been well studied. Most reliable risk factors associated with an increased risk of CRC in UC are related to the extent and duration of the disease. The risk for CRC development is lower before 8 to 10 years after onset of symptoms (3%); however, thereafter the risk increases by approximately

Relationships: None.
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1% per year. Various studies have shown risks of CRC in UC ranging from 5% to 20% at 20 years of the disease.[1–3] By the fourth decade of UC disease, the risk of developing CRC is as high as 56 times higher than that of the general population.[4] In 2012, a large Danish population-based study demonstrated decreasing rates of CRC in UC over the last 30 years. This decrease is due possibly to the improved medical treatment of the disease in addition to surveillance of dysplasia.[5]

The rates of CRC in Crohn's disease seem to mirror those of UC.[6,7] Crohn's patients have a 5- to 20-fold increase in risk for CRC in comparison with the general population.[7,8] The absolute cumulative frequencies of CRC after 20 years of disease in both UC and Crohn's disease are similar at 8% and 7%, respectively.[9] Because of this similarity, despite the publication of fewer data regarding CRC in Crohn's disease, guidelines and recommendations have been developed for Crohn's patients extrapolating from the body of evidence on UC.

DYSPLASIA AS A PREDICTOR

The mutation pathway to CRC in IBD is postulated to be distinct from the adenoma-carcinoma sequence seen in sporadic colon cancers. Duration and extent of disease are both associated with higher rates of dysplasia and malignancy. IBD-associated cancer often develops in younger patients, and is more likely to be diffuse, extensive, multifocal, and mucinous, compared with the population with sporadic colorectal cancer.[10–12] Cancer in Crohn's disease is more likely to be right-sided and associated with ileal/right-sided inflammation.[9]

Furthermore, IBD patients with colon cancer have historically been shown to have synchronous dysplasia at distant sites from the cancer, suggesting the potential for a field defect rather than an isolated mutation. A review from more than 2 decades ago that included 10 prospective studies with a total of 1225 UC patients demonstrated cancer in 43% of patients with biopsy-proven high-grade dysplasia (HGD). Nineteen percent of patients with low-grade dysplasia (LGD) also had a coexistent cancer.[13] Dysplasia distant to the primary carcinoma has also been shown in 23% to 70% of patients with Crohn's disease.[8] Indeed, the reported risks of synchronous lesions have been variable, as high as 71% for synchronous dysplasia and ranging from 17% to 43% for synchronous cancers.[13–19]

Interpretation of the data on synchronous cancers should, however, be made with caution, owing to the significant limitations during that era in the sensitivity of the fiberoptic technology in detecting dysplasia or cancer at index colonoscopy. Furthermore, surveillance of patients with dysplasia was not standardized (eg, performed without chromoendoscopy or image enhancement at various intervals, or in the endoscopic removal techniques). The true incidence of synchronous colorectal cancer in the setting of dysplasia, as well as the true natural history of endoscopically invisible dysplasia, is thus not known.

For high-risk patients the decision regarding whether to proceed with colectomy or local endoscopic removal with continued colonoscopic surveillance is unquestionably complex, and requires a multidisciplinary approach.

DYSPLASIA MANAGEMENT

Endoscopically Visible Dysplasia

Nowadays most IBD-related dysplasia visible, following the advancements of endoscopic imaging and techniques and a deeper understanding of its appearance, and can be removed endoscopically. Furthermore, terminology for neoplasia in IBD is now being standardized to be similar to neoplasia not related to IBD (ie, polypoid