Dysplasia and Cancer in Inflammatory Bowel Disease

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KEYWORDS

• Inflammatory bowel disease • Ulcerative colitis • Crohn disease • Dysplasia
• Colorectal cancer • Colitis • Colitis-associated cancer

KEY POINTS

- Improved medical management and endoscopic surveillance of inflammatory bowel disease have reduced the incidence of cancer and its associated mortality.
- Surveillance should begin 6 to 10 years after initial diagnosis. Most societies recommend high-definition colonoscopy with chromoendoscopy and targeted biopsies when available.
- High-grade dysplasia or cancer are indications for surgical resection. Exceptions can be considered for lesions contained in discrete adenomalike polyps that can be removed completely.
- The management of low-grade dysplasia is controversial and the choice between continued surveillance versus colectomy should be discussed with patients.
- Most patients requiring surgery should undergo total proctocolectomy with end ileostomy or reconstruction with or without ileal pouch anal anastomosis.

INTRODUCTION

Inflammatory bowel disease (IBD) is associated with an increased risk of developing dysplasia and cancer.[1–3] Dysplasia and colitis-associated cancer (CAC) develop via a different pathway than sporadic cancer and are secondary to longstanding inflammation; they are linked to the duration and extent of disease.[4] Despite improvements in medical management and endoscopic surveillance, the optimal strategies for surveillance and decision for colectomy remain under debate. Herein we review the current literature regarding the risk of dysplasia and cancer in IBD patients, the

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pathogenesis of dysplasia and cancer, current surveillance guidelines, and best practices for managing these patients.

EPIDEMIOLOGY AND CANCER RISK

Cancer risk is increased in both ulcerative colitis (UC) and Crohn disease (CD) compared with the general population. A previously published population-based study over a 35-year period demonstrated an incidence of CAC to be 95 per 100,000.[5] It is, however, believed that this risk has decreased, particularly in UC. Whether this decrease has been due to improved surveillance techniques and technology or improved medical management of disease is unclear.[5,6]

It is generally believed that the risk of disease is related to the extent and duration of disease; however, reported data vary. Eaden and colleagues[7] performed a metaanalysis of 116 studies examining the risk of CRC in UC patients demonstrated the overall prevalence of CRC to be 3.7%. They reported cumulative incidence rate of 2% at 10 years, 8% at 20 years, and 18% at 30 years. In comparison, an analysis of a colonoscopic surveillance program in patients with UC found the cumulative incidence of CRC in UC to be 2.5% at 20 years, 7.6% at 30 years, and 10.8% at 40 years.[8] Similar findings have been noted in CD, with a reported incidence of 8% at 22 years, and a median duration of disease before a diagnosis of cancer (15 years for CD and 18 years for UC).[9,10]

A population-based study over a 60-year period from Olmsted County, Minnesota, demonstrated no significant increase of CAC in UC patients overall compared with the general population (standardized incidence ratio [SIR], 1.1; 95% confidence interval [CI], 0.4–2.4). However, there did seem to be a trend toward increased risk in those with extensive colitis. This study reported a cumulative incidence of CRC in UC patients of 0% at 5 years, 0.4% at 15 years, and 2% at 25 years after diagnosis of UC.[11] In those patients with CD, there also seemed to be a trend toward an increased incidence of CAC and there was a nearly 40-fold increase in risk of small bowel cancer (SIR, 40.6; 95% CI, 8.4–118). The cumulative risk of CRC in CD was reported as 0.3% at 5 years, 1.6% at 15 years, and 2.4% at 25 years after diagnosis.[11] The CESAME (Cancers Et Surrisque Associé aux Maladies Inflammatoires Intestinales En France) Study Group published an observational study of 19,486 patients with IBD and reported an SIR of 2.2 for all IBD patients. There was no increased risk in patients with limited disease (SIR, 1.1; 95% CI, 0.6–1.8). However, those with extensive colitis (>10 years and >50% of the colon involved) had a far greater risk of CAC (SIR, 7.0; 95% CI, 4.4–10.5).[12] Finally, a Manitoba Health study of 5529 patients observed over a 14-year period demonstrated an increased risk of colon cancer in UC (SIR, 2.8; 95% CI, 1.9–4.0) and CD (SIR, 2.6; 95% CI, 1.7–4.2). A nearly 2-fold increase in risk of rectal cancer was demonstrated only in the UC population and a 17-fold increase in risk of small bowel cancer was noted in the CD population.[13]

Other non–IBD-related risk factors for development of cancer exist, primarily a concomitant diagnosis of primary sclerosing cholangitis and a family history of CRC. Numerous studies have demonstrated an increased risk of CRC in patients with IBD and primary sclerosing cholangitis.[14] A metaanalysis found that the development of carcinoma or dysplasia in patients with UC and primary sclerosing cholangitis was increased (odds ratio [OR], 4.8; 95% CI, 3.6–6.4).[15] This risk has been reported to increase after liver transplantation.[16] Much like the general population, a family history of CRC imparts an increased risk of cancer in IBD. Askling and colleagues[17] reported that IBD patients with a positive family history of CRC had an increased relative risk compared with those with no family history of CRC (SIR, 31 [95% CI, 16–52] vs SIR,