Colorectal cancer in IBD: Surgeon and oncologist's perspective

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IBD-related colorectal cancer follows a unique inflammation-dysplasia-carcinoma pathogenesis distinct from sporadic colorectal cancer, with higher rates of advanced-stage disease and interval cancers requiring individualized surgical and systemic treatment strategies.

KEY POINTS

- Colorectal cancer is the leading cause of mortality in IBD, with a 1.7-fold increased incidence risk and 1.5-fold increased mortality risk compared to the general population, though risk has decreased over time with improved disease control and surveillance.
- IBD-related colorectal cancer presents with higher T3/T4 stage and lymph node involvement than sporadic cancer, and many cases are interval cancers that are difficult to detect; incidental cancers found in inflamed colons after multiple therapies have less than 65% five-year survival, as stated by the speakers.
- The pathogenesis involves chronic inflammation with cytokines such as TNF-alpha, IL-6, and IL-23, oxidative stress, early TP53 mutations preceding dysplasia, epithelial barrier dysfunction, and gut microbiota dysbiosis, distinct from the APC-KRAS-p53 adenoma-carcinoma sequence of sporadic cancer.
- Surgical management is individualized based on disease activity, multifocality, prior dysplasia or cancer, PSC status, and patient age, with options ranging from endoscopic resection for early unifocal lesions to proctocolectomy for multifocal disease or active inflammation compromising surveillance.
- Checkpoint inhibitors for MSI-high tumors carry a relative contraindication in IBD patients with active inflammation, with up to 40% experiencing disease flare-ups; bevacizumab and other systemic therapies for microsatellite-stable disease pose challenges including perforation risk in the IBD population.

– The speaker recommended the most limited resection (segmental rather than total colectomy) for unifocal non-endoscopically resectable lesions in patients with quiet disease, noting that multidisciplinary decision-making must account for MSI status and metastatic potential when considering checkpoint inhibitor therapy.

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