Editorial

Dysplastic area in ulcerative Colitis. Endoscopic resection or total colectomy?

G. Kitis, T. Maris

The incidence of colon cancer is increased in ulcerative colitis (UC). It is estimated to occur in 1 of 333 to 1 of 400 patient-years. Approximately 18% of patients with an intact colon may develop colon cancer after 30 years of disease.[1] Factors associated with an increased risk include long duration of colitis, extensive colonic involvement, primary sclerosing cholangitis, a family history of colorectal cancer and, according to some studies, early disease onset and more severely active inflammation.[2]

One of the main objectives of physicians in patients with UC is to detect neoplasia at a surgically curative and preferably preinvasive stage, i.e., dysplasia. Gastrointestinal dysplasia is defined microscopically as replacement of the native intestinal epithelium by an unequivocally neoplastic, but as yet noninvasive, epithelium. As such, it is synonymous with the term "intraepithelial neoplasia", widely used in other organ systems.

The histological classification of dysplasia in IBD is: negative for dysplasia; indefinite for dysplasia; and positive for low-grade dysplasia (LGD) or high-grade dysplasia (HGD).

Dysplasia is classified macroscopically as elevated or flat depending respectively on whether or not it corresponds to an endoscopically visible lesion. Elevated lesions, conventionally referred to by the acronym DALM (dysplasia associated lesion or mass), span a broad spectrum that includes single and multiple polyps, bumps, plaques and velvety patches. Such lesions can easily be camouflaged among the varied gross inflammatory abnormalities commonly encountered in colons with IBD, making their endoscopic detection a daunting challenge even for experienced practitioners.[2]

Flat dysplasia is only detected microscopically in random biopsy specimens from unremarkable mucosa. Its detection therefore depends critically on adequate sampling of the mucosa by the endoscopist, that is obtaining 2-4 biopsy specimens every 10 cm of diseased bowel. Chromoendoscopy with magnifying endoscopes can enhance the detection of flat and even raised dysplasia during colonoscopy.

The critical therapeutic question is: Which is the most appropriate approach for a UC patient when dysplasia of any grade is found during surveillance colonoscopy (endoscopic resection and surveillance or total colectomy)?

Traditionally, if high grade dysplasia was found in any area (flat or DALM), the only treatment approach was total colectomy. If low grade dysplasia was found in a DALM lesion colectomy was also recommended.[3,4] The same recommendation also applied to flat multifocal low grade dysplasia, whereas for flat unifocal low grade dysplasia only surveillance was recommended.[3] However Bernstein suggests total colectomy for a definite flat low grade dysplasia of any type.[5]

It is important to emphasize that no surveillance programme rules out the risk of cancer. Only a total colectomy removes the neoplastic mucosa and the residual mucosa that is at risk for developing neoplasia. This removes both cancer risk and cancer fear.

Recent studies have suggested that, conservative endoscopic management is also a reasonable option for dysplasia, when it is found in an "adenoma-like" polyp. These polyps are endoscopically indistinguishable from sporadic sessile adenomatous polyps, i.e., discrete and

Department of Gastroenterology General Regional Hospital "G. Papanikolaou", Thessaloniki, Greece

Author for correspondence:

G.Kitis, 19, Anoixeos str., 552 36 Thessaloniki, Greece,
Tel.: +2310-350102, Fax: +2310-350050, e-mail: gpap@isth.gr

ovoid or round, completely resectable by the endoscope, and not surrounded by flat dysplasia. Such lesions have long posed a dilemma for endoscopists who were familiar with the DALM concept but reluctant to advocate colectomy for what appeared to be inocuous lesions and possibly nothing more than fortuitous adenomas. Histology has not provided a reliable means of making this distinction in individual cases, because dysplasia in the setting of colitis and in true adenomatous polyps can be virtually identical. As a result, the burden of deciding whether a polyp qualifies as DALM or true adenoma falls squarely on the shoulders of the endoscopist.

A 1999 study from The Mount Sinai Hospital reported that, in patients with chronic colitis who have no dysplasia in flat mucosa, colonoscopic resection of dysplastic polyps can be performed effectively, just as in non-colitic colons.[6] Similar conclusions were reached in a concurrent study from Brigham and Women's Hospital It was found that UC patients, who develop an adenoma-like DALM, that resembles endoscopically and histologically a sporadic adenoma, regardless of its location (either within or outside areas of documented colitis), may be treated with polypectomy and endoscopic surveillance because of its relatively benign course.[7] Likewise Goldstein et al have suggested that, dysplastic polyps found in UC patients can be removed by endoscopy safely and effectively without resorting to colectomy, as long as there is no other detectable dysplasia in flat mucosa and complete removal can be assured (by biopsy of adjacent mucosa and close "follow-up").[8] Finally Odge et al have suggested that adenoma-like DALM detected in UC patients may be treated adequately by polypectomy with complete excision and continued surveillance.[9]

In conclusion, dysplasia of any grade detected in an endoscopically nonresectable polyp or DALM or high-grade dysplasia detected in flat mucosa are both strong indications for proctocolectomy. Further evidence suggests that the same may be true even of low-grade dysplasia in flat mucosa especially if it is multifocal. However, if dysplasia of either low or high grade is detected in a discrete adenoma-like polyp, that can be readily resected endoscopically and there is no flat dysplasia immediately adjacent to the polyp or elsewhere in the colon, polypectomy is sufficient followed by a careful surveillance programme.

The clinical application of newer molecular methods to detect neoplasia (other than dysplasia), particularly gene microarrays and stool DNA testing, also deserve further study and may radically modify our approach to the management of cancer risk in ulcerative colitis patients.

REFERENCES

1. Eaden JA, Abrams KR, Mayberry JF. The risk of colorectal cancer in ulcerative colitis: a meta-analysis. Gut. 2001; 48: 526-535.
2. Itzkowitz S, Harpaz N. Diagnosis and management of dysplasia in patients with inflammatory bowel disease. Gastroenterology 2004; 126: 1634-1648.
3. Winawer S, Fletcher R, Rex D, Bond J, Burt R, Ferrucci J, Ganiats T, Levin T, Woolf S, Johnson D, Kirk L, Litin S, Simmang C. Gastrointestinal Consortium Panel. Colorectal cancer screening and surveillance: clinical guidelines and rationale—update based on new evidence. Gastroenterology 2003; 124: 544–560
4. Eaden JA, Mayberry JF. Guidelines for screening and surveillance of asymptomatic colorectal cancer in patients with inflammatory bowel disease. Gut 2002; 51(suppl V): v10–v12.
5. Bernstein C. Ulcerative Colitis with low-grade dysplasia. Gastroenterology 2004; 127:950-956.
6. Rubin PH, Friedman S, Harpaz N, Goldstein E, Weiser J, Schiller J, et al. Colonoscopic polypectomy in chronic colitis: conservative management after endoscopic resection of dysplastic polyps. Gastroenterology 1999; 117: 1295–1300.
7. Engelsgjerd M, Farraye FA, Odze RD. Polypectomy may be adequate treatment for adenoma-like dysplastic lesions in chronic ulcerative colitis. Gastroenterology 1999; 117: 1288–1294.
8. Goldstein ES, Rubin PH. Curr Endoscopic Therapy for Inflammatory Bowel Disease Treat Options Gastroenterol 2003; 6: 237-243.
9. Odze RD, Farraye FA, Hecht JL, Hornick JL. Long-term follow-up after polypectomy treatment for adenoma-like dysplastic lesions in ulcerative colitis. Clin Gastroenterol Hepatol 2004; 2: 534-541.