Large serrated polyp with KRAS mutation in inflammatory bowel disease: a “nondysplastic dysplasia-associated lesion or mass (DALM)”?

Patients with longstanding inflammatory bowel disease (IBD) have an increased risk of colorectal cancer. A causal link between chronic inflammation and cancer is well recognized. Precursor lesions include flat dysplasia (intraepithelial neoplasia) and elevated dysplasia, also known as dysplasia-associated lesion or mass (DALM) [1].

A 52-year-old woman with 20-year history of ulcerative colitis underwent surveillance colonoscopy, which disclosed a large irregular polyp in the sigmoid colon (Fig. 1). Biopsies showed a nondysplastic polyp with marked crypt dilatation within the sigmoid colon (hematoxylin and eosin, original ×100). c, d Conventional high grade dysplasia-associated lesion or mass (DALM) at the rectosigmoid junction (hematoxylin and eosin, original ×200).

This polyp revealed KRAS mutation in exon 13 (Fig. 3); tests for BRAF mutation and microsatellite instability were negative.

In 2008, Srivastava et al. [2] reported a series of three patients with longstanding IBD who developed numerous “hyperplastic/serrated” colonic polyps similar to those described in the “hyperplastic/serrated” polyposis syndrome. Two patients had synchronous colorectal cancer. KRAS mutation was detected in five of the 11 polyps. These findings suggested the possibility of a serrated pathway of carcinogenesis in IBD. In the sporadic setting, sessile serrated adenomas/polyps (SSA/P) are known precursors of mainly right-sided microsatellite instable cancers. They may also be regarded as indicator lesions, as these polyps have been associated with increased risk of synchronous and/or metachronous cancer growth, particularly of the proximal colon [3,4].

We believe our case to be the first description of a solitary serrated polyp with KRAS mutation, similar to the lesions occurring as polyposis in longstanding IBD described by Srivastava et al. [2]. These nondysplastic lesions may indicate increased risk of synchronous and/or metachronous advanced neoplasia and may be the equivalent of conventional DALMs with respect to cancer prediction (“nondysplastic DALM”).

Competing interests: None

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Fig. 3  Molecular analysis (pyrosequencing of the KRAS gene) of the serrated polyp showing a somatic missense mutation in codon 13: wildtype control (upper panel) and serrated polyp (lower panel).

References

Bibliography
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