Turk J Gastroenterol *2016; 27: 482-3*

"Are we ready to change our colorectal cancer screening practice?" Traditional serrated adenomas or serrated adenomas with dysplasia should be regarded as advanced adenomas with high grade dysplasia

Erichsen R, Baron JA, Hamilton-Dutoit SJ, et al. Increased risk of colorectal cancer development among patients with serrated polyps. Gastroenterology 2016; 150: 895-902.

Colorectal cancers (CRCs) develop mostly via adenoma-carcinoma sequence. Serrated pathway is an alternative way in one third of CRCs and is associated with serrated adenoma/ polyp (1,2). Serrated adenomas are typically three types as sessile serrated adenoma/ polyp (SSA/P), traditional serrated adenoma (TSA) and hyperplastic polyp (HP) (3,4). Some serrated adenomas used to be misdiagnosed as HPs before the mid-2000s. Therefore they received minimal intervention and inappropriate follow up after polypectomy. SSA/P and TSA are now distinguished from HP and are recognized as precursors to CRC (5,6).

Erichsen et al. (1) conducted a nationwide population-based, case-control study using the Danish databases (1977-2009). They evaluated CRC risk in patients with a history of serrated polyps. It included the patients who had received colonoscopies (n=272,342) and identified 2045 CRC cases and 8105 controls. In both groups, the first colorectal polyp that was biopsied or excised was identified and tissue blocks of HP were obtained. Four expert pathologists reviewed them with current terminology for serrated polyps. They used logistic regression to compute odds ratios (ORs) to associate the risk of CRC with the type of polyp and estimated the absolute risks by multiplying the risk in group with no polyps.

Seventy-nine cases, 142 controls had SSA/P. SSA/Ps with dysplasia were associated with high OR (4.76; 95% CI, 2.59–8.73). Women with SSAP/P had higher risk for CRC than men (OR for women, 5.05; 95% CI, 3.05–8.37 vs OR for men, 2.18; 95% CI, 1.24–3.82). SSA/P located proximal to splenic flexure had higher risk for CRC (7,8) (OR, 12.42; 95% CI, 4.88–31.58) than conventional adenomas. The OR for CRC was 4.84 with TSA (95% CI, 2.36–9.93), 2.51 with conventional adenomas (95% CI, 2.25–2.80), and 1.30 with hyperplastic polyps (95% CI, 0.96–1.77).

The 10-year risk for CRC was 4.4% for patients with SSA/Ps with cytologic dysplasia and 4.5% for TSA patients. These CRC risk ratios were higher than 2.33% for conventional adenoma and 1.21% for CRC with any previous type HPs.

Erichsen et al.'s (1) study lacked information on factors altering CRC risk including family history, smoking, aspirin, polyp size and numbers which could bias the ORs in either direction. However, it had still very important outcome. The CRC risk was particularly high for women with SSA/P, and for patients with proximal SSA/P. Evolution of SSA into invasive adeno cancer (9) can be as little as 8 months (10). Erichsen et al. (1) also underlined the importance of CRC risk as the highest risk in patients with any TSAs and SSA/Ps with dysplasia.

The current guidelines address screening and surveillance with regards to conventional adenomas and serrated adenomas. Detailed histologic types suggested in this study by Erichsen et al. (1) as SSA/Ps with dysplasia and TSAs are not incorporated into surveillance guidelines yet. This study brings attention to and emphasizes revision of current guidelines recommending complete removal of SSA/Ps with dysplasia and TSAs and close surveillance of these lesions. A recommendation would be to consider SSA/Ps with dysplasia or all TSAs as "advanced" polyps with clinical significance at least similar to high grade dysplasia in conventional adenomas. These high risk serrated adenomas deserve particularly closer follow up evaluation than conventional adenomas.

Anjana Sathyamurthy, Veysel Tahan

Division of Gastroenterology and Hepatology, University of Missouri, Columbia, Missouri, USA

Received: August 27, 2016 **Accepted:** August 27, 2016

DOI: 10.5152/tjg.2016.160006

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