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Impact of Helicobacter pylori Infection on Colorectal Adenoma-Colorectal Carcinoma Sequence

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Abstract : Background & Aims: Helicobacter pylori infection (Hp-I) has been recognized as a substantial risk agent involved in gastrointestinal (GI) tract oncogenesis by stimulating cancer stem cells (CSCs), oncogenes, immune surveillance processes, and triggering GI microbiota dysbiosis. We aimed to investigate the possible involvement of active Hp-I in the sequence: chronic inflammation–adenoma–colorectal cancer (CRC) development. Methods: Four pillars were investigated: (i) endoscopic and conventional histological examinations of patients with CRC, colorectal adenomas (CRA) versus controls to detect the presence of active Hp-I; (ii) immunohistochemical determination of the presence of Hp; expression of CD44, an indicator of CSCs and/or bone marrow-derived stem cells (BMDSCs); expressions of oncogene Ki67 and anti-apoptotic Bcl-2 protein; (iii) expression of CD45, indicator of immune surveillance locally (assessing mainly T and B lymphocytes locally); and (iv) correlation of the studied parameters with the presence or absence of Hp-I. Results: Among 50 patients with CRC, 25 with CRA, and 10 controls, a significantly higher presence of Hp-I in the CRA (68%) and CRC group (84%) were found compared with controls (30%). The presence of Hp-I with accompanying immunohistochemical expression of CD44 in biopsy specimens was revealed in a high proportion of patients with CRA associated with moderate/severe dysplasia (88%) and CRC patients with moderate/severe degree of malignancy (91%). Comparable results were also obtained for Ki67, Bcl-2, and CD45 immunohistochemical expressions. Concluding Remarks: Hp-I seems to be involved in the sequence: CRA – dysplasia – CRC, similarly to the upper GI tract oncogenesis, by several pathways such as the following: Beyond Hp-I associated insulin resistance, the major underlying mechanism responsible for the metabolic syndrome (MetS) that increase the risk of colorectal neoplasms, as implied by other Hp-I related MetS pathologies, such as non-alcoholic fatty liver disease and upper GI cancer, the disturbance of the normal GI microbiota (i.e., dysbiosis) and the formation of an irritative biofilm could contribute to a perpetual inflammatory upper GIT and colon mucosal damage, stimulating CSCs or recruiting BMDSCs and affecting oncogenes and immune surveillance processes. Further large-scale relative studies with a pathophysiological perspective are necessary to demonstrate in-depth this relationship.

Keywords : Helicobacter pylori, colorectal cancer, colorectal adenomas, gastrointestinal oncogenesis
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