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The Role of Helicobacter Pylori Infection in Gastric Carcinoma Pathogenesis

Ramakant Bhosale[1] and Dr. Nagendra Pratap Mishra[2]
Research Scholar, Department of Biochemistry[1]
Research Guide, Department of Biochemistry[2]
Sunrise University, Alwar, Rajasthan, India

Abstract: *Gastric carcinoma is one of the prevalent types of cancer globally with a notable morbidity and mortality rate. Helicobacter pylori (H. pylori) infection has been established as a significant risk factor for gastric carcinoma. This paper reviews current literature on the role of H. pylori infection in the pathogenesis of gastric carcinoma, exploring molecular mechanisms and clinical implications. Furthermore, the paper discusses potential preventive and therapeutic strategies to combat gastric carcinoma associated with H. pylori infection.*

Keywords: Prognostic Implications, Histopathology, Resection Margins

REFERENCES

[1]. Ferlay JSI, Ervik M, Dikshit R, et al. International Agency for Research on Cancer, Cancer Incidence, and Mortality Worldwide. Lyon, France: IARC CancerBase no. 11, 2013.
[2]. Bang YJ, Van Cutsem E, Feyereislova A, et al. Trastuzumab in combination with chemotherapy versus chemotherapy alone for treatment of HER2-positive advanced gastric or gastro-oesophageal junction cancer (ToGA): a phase 3, open-label, randomised controlled trial. Lancet 2010;376:687–697.
[3]. Cancer Genome Atlas Research N. Comprehensive molecular characterization of gastric adenocarcinoma. Nature 2014;513:202–209.
[4]. Correa P. Human gastric carcinogenesis: a multistep and multifactorial process—first American Cancer Society Award Lecture on cancer epidemiology and prevention. Cancer Res 1992;52:6735–6740.
[5]. Almeida R, Silva E, Santos-Silva F, et al. Expression of intestine-specific transcription factors, CDX1 and CDX2, in intestinal metaplasia and gastric carcinomas. J Pathol 2003;199:36–40.
[6]. Goldenring JR, Nomura S. Differentiation of the gastric mucosa III. Animal models of oxyntic atrophy and metaplasia. Am J Physiol Gastrointest Liver Physiol 2006;291:G999–1004.
[7]. Goldenring JR, Nam KT. Oxyntic atrophy, metaplasia, and gastric cancer. Dev Diff Dis 2010;96:117–131.
[8]. Karin M. Inflammation and cancer: the long reach of Ras. Nat Med 2005;11:20–21.
[9]. Eaton KA, Logan SM, Baker PE, et al. Helicobacter pylori with a truncated lipopolysaccharide O chain fails to induce gastritis in SCID mice injected with splenocytes from wild-type C57BL/6J mice. Infect Immun 2004; 72:3925–3931.
[10]. Lochhead P, El-Omar EM. Gastric cancer. Br Med Bull 2008;85:87–100.
[11]. Tsugane S, Sasazuki S. Diet and the risk of gastric cancer: review of epidemiological evidence. Gastric Cancer 2007;10:75–83.
[12]. Noto JM, Gaddy JA, Lee JY, et al. Iron deficiency accelerates Helicobacter pylori-induced carcinogenesis in rodents and humans. J Clin Invest 2013;123:479–492.
[13]. Iwakiri D TK. Epstein-Barr virus and gastric cancers. In: ES R, ed. Epstein-Barr virus. Norfolk, VA: Caister Academic Press, 2005:157–169.
[14]. Murphy G, Pfeiffer R, Camargo MC, et al. Meta-analysis shows that prevalence of Epstein-Barr virus-positive gastric cancer differs based on sex and anatomic location. Gastroenterology 2009;137:824–833.
[15]. Peleteiro B, Bastos A, Ferro A, et al. Prevalence of Helicobacter pylori infection worldwide: a systematic review of studies with national coverage. Dig Dis Sci 2014; 59:1698–1709.

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[16]. Odenbreit S, Puls J, Sedlmaier B, et al. Translocation of Helicobacter pylori CagA into gastric epithelial cells by type IV secretion. Science 2000;287:1497–1500.

[17]. Ohnishi N, Yuasa H, Tanaka S, et al. Transgenic expression of Helicobacter pylori CagA induces gastrointestinal and hematopoietic neoplasms in mouse. Proc Natl Acad Sci U S A 2008;105: 1003–1008.

[18]. Higashi H, Tsutsumi R, Fujita A, et al. Biological activity of the Helicobacter pylori virulence factor CagA is determined by variation in the tyrosine phosphorylation sites. Proc Natl Acad Sci U S A 2002;99:14428–14433.

[19]. Higashi H, Tsutsumi R, Muto S, et al. SHP-2 tyrosine phosphatase as an intracellular target of Helicobacter pylori CagA protein. Science 2002;295:683–686.

[20]. Amieva MR, Vogelmann R, Covacci A, et al. Disruption of the epithelial apical-junctional complex by Helicobacter pylori CagA. Science 2003;300:1430–1434.