AgNOR and rasp21 expression in gastric mucosal lesions caused by *Helicobacter pylori* infection

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Received: March 8, 1997
Revised: May 2, 1997
Accepted: October 28, 1997
Published online: December 15, 1997

Abstract

AIM: To investigate AgNOR and rasp21 expression levels in gastric mucosal lesions caused by *Helicobacter pylori* (Hp) infection in order to gain insight into the related biological processes (i.e. tumor-like behavior) and possible underlying mechanism supporting Hp pathogenesis.

METHODS: Hp infection was diagnosed in using the standard Campylobacter-like organism test along with Wathin-Starry staining. The expression of AgNOR was detected by the silver colloid staining technique. The expression of rasp21 was detected by monoclonal antibody and immunohistochemical staining using the ABC method. The study included a total of 278 patients with endoscopically- and pathologically-confirmed gastric mucosal lesions, representing chronic superficial gastritis (CSG), chronic atrophic gastritis (CAG), intestinal metaplasia, dysplasia, and gastric cancer. Among these, 146 of the

patients were Hp-positive and 132 were Hp-negative.

RESULTS: The Hp-positive group of patients showed significantly greater AgNOR in the gastric mucosal lesions than the Hp-negative group, with the exception of the CSG sub-group ($P < 0.05$ or $P < 0.01$). The positive rate of rasp21 expression in gastric mucosal lesions in the Hp-positive group was also significantly higher than that in the Hp-negative group, with the exception of the CSG and CAG sub-groups ($P < 0.05$).

CONCLUSION: Hp-positive gastric mucosal lesions show biological behaviour of tumors. Hp may act as a promoter to activate the ras gene and to stimulate cell over-proliferation.

Key words: *Helicobacter pylori*/pathogenicity; Gastric mucosal/pathology; Stomach diseases/microbiology; Stomach neoplasms/microbiology; Nucleoproteins/metabolism; Proto-oncogene protein P21/ (RAS)

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Gao HJ, Lu XZ, Zhang XY, Zhao ZQ. AgNOR and rasp21 expression in gastric mucosal lesions caused by *Helicobacter pylori* infection. *World J Gastroenterol* 1997; 3(4): 252 Available from: URL: <http://www.wjgnet.com/1007-9327/full/v3/i4/252.htm> DOI: <http://dx.doi.org/10.3748/wjg.v3.i4.252>

L- Editor: Filipodia E- Editor: Liu WX

Published by **Baishideng Publishing Group Inc**
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ISSN 1007-9327