

# Research of Helicobacter pylori infection in precancerous gastric lesions

Xiao Qiang Zhuang<sup>1</sup> and San Ren Lin<sup>2</sup>

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## INTRODUCTION

*Helicobacter pylori* (*Hp*) infection has been considered to play significant roles in pathogenesis of peptic ulcer. Additionally *Hp* is associated with the development of gastric epithelial hyperplasia and lymphoid malignancies. The International Agency for Research on Cancer has classified *Hp* as a class I carcinogen and a definite cause of gastric cancer in humans. *Hp* infection first causes chronic active gastritis and may slowly lead to infection of whole stomach. In the late stages of infection, mucosal atrophy and intestinal metaplasia (IM), and even dysplasia (DYS) occur<sup>[1]</sup>. Chronic atrophic gastritis (CAG), IM and DYS are considered markers for development of gastric cancer in high-risk individuals. In our study we analyzed *Hp* infection prevalence in 486 patients with precancerous gastric lesions.

## MATERIALS AND METHODS

The mucosal biopsy specimens were collected from 486 patients subjected to routine gastroscopy, including 163 cases of CSG, 207 cases of CAG, 71 cases of IM and 45 cases of DYS. Biopsies were taken from five sites in the stomach: one from the angle, four from the lesser and greater curvature of gastric body and gastric antrum. Each biopsy was classified according to the presence or absence of CSG, CAG, IM and DYS, and scanned by Warthin-Starry method.

Data analysis was made with *Chi*-square test. Statistical significance was defined as  $P < 0.05$ .

<sup>1</sup>Department of Gastroenterology, General Hospital, of Guangzhou Command Area, Guangzhou 510010, Guangdong Province, China

<sup>2</sup>Department of Gastroenterology, The Third Hospital of Beijing Medical University, Beijing 100083, China

Dr. Xiao Qiang Zhuang, associate professor Master of Gastroenterology, having 31 papers published.

**Correspondence to:** Xiao Qiang Zhuang, Department of Gastroenterology, General Hospital of Chinese PLA Guangzhou Command Area, Guangzhou 510010, China

Tel. 0086-20-86664097

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## RESULTS

**Table 1** Relation between precancerous gastric lesions and age and sex

Variables	n	CSG		CAG		IM		DYS	
		n	%	n	%	n	%	n	%
Sex									
Male	314	96	30.6	142	45.2	43	13.7	33	10.5
Female	172	67	38.9	65	37.8	28	16.3	12	7
Age (yrs)									
≤40	184	64	34.9	98	53.5	13	7.0 <sup>a</sup>	9	4.6 <sup>b</sup>
41-55	171	52	30.3	70	41.2	30	17.3	19	10.8
≥56	131	40	30.5	46	35.4	28	21.1 <sup>a</sup>	17	13.0 <sup>b</sup>

<sup>a</sup> $P < 0.05$ , <sup>b</sup> $P < 0.05$ .

Gastric pathology data was available for 486 cases. As shown in Table 1, there was no significant difference between the two sexes ( $P > 0.05$ ), but the prevalence rates increased with age, being significantly higher in ≥56 age group than ≤40 group for IM and DYS ( $P < 0.05$ ).

**Table 2** *Hp* infection rate in precancerous gastric lesions

Variables	n	Hp(+)		Hp(-)	
		n	%	n	%
CSG	163	39	23.9	124	76.1
CAG	207	88	42.5 <sup>a</sup>	119	57.5
IM	71	54	76.1 <sup>b</sup>	17	23.9
DYS	45	40	88.9 <sup>c</sup>	5	11.1

<sup>a</sup> $P < 0.05$ , CAG vs CSG; <sup>b</sup> $P < 0.01$ , IM vs CSG; <sup>c</sup> $P < 0.01$ , DYS vs CSG.

The prevalence of *Hp* increased steadily with increasing severity of gastric histopathology (Table 2). The detection rates of *Hp* in IM (76.1%) and DYS (88.9%) were significantly higher than that in CSG (23.9%,  $P < 0.01$ ), and in CAG than in CSG ( $P < 0.05$ ).

**Table 3** *Hp* infection distribution in precancerous gastric lesions by biopsy sites

Variables	n	ALC		AGC		A		BLC		BGC	
		n	%	n	%	n	%	n	%	n	%
CSG	39	37	94.9	36	92.3	32	82.1	18	46.2	10	25.6
CAG	88	84	95.4	80	90.9	78	88.6	53	59.9	41	46.2
IM	54	49	90.7	46	85.2	40	74.1	34	62.7 <sup>a</sup>	32	58.8 <sup>a</sup>
DYS	40	36	90.0	33	82.5	30	75.0	28	70.0 <sup>b</sup>	26	63.9 <sup>b</sup>

ALC:antral lesser curvature; AGC:antral greater curvature; A: angulus; BLC:body, lesser curvature; BGC:body, greater curvature

<sup>a</sup> $P < 0.05$ , IM vs CSG; <sup>b</sup> $P < 0.05$ , DYS vs CSG.

As shown in Table 3, the prevalence of *Hp* positivity tended to increase from BLC to BGC, *Hp* positive rates in IM and DYS were significantly higher than that in CSG in both BLC and BGC ( $P < 0.05$ ).

## DISCUSSION

The first compelling evidence linking *Hp* infection to gastric carcinoma was generated by seroepidemiologic studies<sup>[2,3]</sup>, bacterial seropositivity was significantly more common in those with gastric adenocarcinoma, with an odds ratio ranging from 2.8 to 6.0, suggesting a strong association between *Hp* and gastric malignancy.

*Hp* infection was related to both the intestinal and diffuse types of cancer as well as the precancerous lesion of IM or DYS<sup>[4]</sup>, and greater than 70% of gastric carcinomas are linked to IM, although DYS may also be seen without neoplastic disease.

Our study demonstrated that the prevalence rates of precancerous lesions varied with age, for IM and DYS, it had an upward trend with aging, while for CSG, it had a downward trend with aging, but were not different between the two sexes. The prevalence

rose steadily with increasing severity of gastric histopathology, the detection rates of *Hp* in CAG (42.5%), IM (76.1%) and DYS (88.9%) were significantly higher than that in CSG (23.9%), suggesting that *Hp* may play a role in late as well as early stages of carcinogenesis.

Our study also showed that *Hp* positive rates in IM and DYS were significantly higher than that in CSG in the lesser and greater curvature of gastric body, suggesting the more serious the gastric histopathology, the higher the *Hp* infection rate, furthermore, the higher level of *Hp* infection site. The study suggests we should take multiple site biopsy for histopathology and *Hp* examination.

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