World Journal of Gastroenterology

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DOI: 10.3748/wjg.v1.i1.41

World J Gastroenterol 1995 October 1; 1(1): 41-42
ISSN 1007-9327 (print) ISSN 2219-2840 (online)

Influence of *Helicobacter pylori* on the gastric mucosal barrier

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Author contributions: All authors contributed equally to the work.

Presented at IX World Congress of Gastroenterology, Sydney, Australia, 26-31 August 1990.

Original title: *China National Journal of New Gastroenterology* (1995-1997) renamed *World Journal of Gastroenterology* (1998-).

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Received: March 31, 1995
Revised: June 14, 1995
Accepted: August 20, 1995
Published online: October 1, 1995

Abstract

AIM: To study the influence of *Helicobacter pylori* (Hp) on the gastric mucosal barrier (GMB) by the measurement of the potential difference (PD).

METHODS: Fifty seven chronic gastritis cases were diagnosed endoscopically and confirmed by forceps mucosal biopsy. PD was measured by the Takeuchi method, and Hp was detected by both culture (modified Skirrow method) and press printing method with the Giemsa stain. Patients were divided randomly into three groups (De-Nol, WeiTong-Ling, and Placebo) for a course of 6 wk therapy.

RESULTS: PD across the mucosa of antrum was significantly lower in Hp (+) patients than in Hp (-) patients (16.44 ± 2.36 *vs* 19.58 ± 2.44, $P < 0.0001$). In Hp (+) patients, PD in the antrum increased markedly (16.88 ± 2.56 *vs* 20.03 ± 2.21, $P < 0.0001$) after Hp was cleared up by the De-Nol treatment.

CONCLUSION: Our data strongly indicated that Hp infection might cause a gastric mucosal barrier to be impaired markedly while the clearance of Hp by De-Nol recovered the integrity of the gastric mucosal barrier significantly.

Key words: *Helicobacter pylori*; Gastric transmucosal potential difference; Gastric mucosal barrier

Xu CP, Gui XY, Liu WW, Wang ZH, Pan SW. Influence of *Helicobacter pylori* on the gastric mucosal barrier. *World J Gastroenterol* 1995; 1(1): 41-42 Available from: URL: http://www.wjgnet.com/1007-9327/full/v1/i1/41.htm DOI: http://dx.doi.org/10.3748/wjg.v1.i1.41

INTRODUCTION

Helicobacter pylori (Hp) infection may play an important role in the etiology of chronic gastritis (CG) and peptic ulcer, but its mechanism remains unclear. Obvious damages to the gastric mucosal barrier (GMB) were found in various CG, but there were few reports about the influence of Hp on GMB. The purpose of this study was to explore the influence of Hp on GMB by the measurement of the potential difference (PD).

MATERIALS AND METHODS

Fifty seven CG patients were diagnosed endoscopically and confirmed by the forceps mucosal biopsies. Of the 58 CG patients, 14 had chronic superficial gastritis (CSG), and 43 had chronic atrophic gastritis (CAG). A total of 38 men and 19 women with mean age of 37.8 year were included in the study. PD was measured by the Takeuchi method[1,2] at eight sites in the gastric antrum and body. Hp was detected by both culture (modified Skirrow method) and the Giemsa stain. Fifty seven CG patients were randomly divided randomly into three groups. In the group Ⅰ, 120 mg of De-Nol (9 cases) was administrated three times a day for six weeks. The group Ⅱ, the WeiTong-Ling group, patients were administered 1.7 three times a day for six weeks. The group Ⅲ was the control group (Placebo).

RESULTS

Hp was detected in 28 of 57 patients (49.12%; CSG, 35.75%; CAG, 53.5%; $P < 0.05$). The Hp clearance rate was 77.8% in the De-Nol group and 33.3% in the WeiTong-Ling group ($P < 0.01$). In the control group, Hp was not cleared in any of the patients.

As is shown in Table 1, PD in the antrum was lower in Hp (+) patients than in Hp (-) patients ($P < 0.0001$), but no significant difference was found in PD in the gastric body. In Hp (+) patients, PD in the antrum increased markedly ($P < 0.0001$) after Hp has been cleared by the De-Nol treatment (Table 2). No significant change in PD was found in Hp (-) patients after the therapy (Table 3).

DISCUSSION

PD is a good indicator of the integrity of the gastric mucosa, and it runs parallel with the degree of mucosal damage or recovery[2]. Present data showed that Hp infection lowered the PD significantly in the antrum and exacerbated the damage of GMB. Once Hp is cleared by the medical therapies (the De-Nol treatment), the GMB improves, and the inflammatory infiltration reduces. The mechanism of damage to GMB caused by Hp is poorly understood. Goodwin *et al*[3] found that the content of neutral mucus on the Hp-infected gastric mucosa decreased markedly. Tasman-Jones *et al*[4] reported the similar results. Bode *et al*[5] found that the viscosity of mucus infected by Hp was lowered. The production of mucus in the damaged epithelial cell may be decreased[6]. Other causes weakening the GMB included the

Table 1 Potential difference in *Helicobacter pylori* positive or negative patients

Group	Antrum				Corpus			
	n	Hp (+)	*n*	Hp (-)	*n*	Hp (+)	*n*	Hp (-)
CSG	5	17.96 ± 2.70	9	20.43 ± 2.08[e]	5	32.13 ± 3.79	9	32.93 ± 5.47
CAG	23	16.44 ± 2.36	20	19.58 ± 2.44[e]	25	31.74 ± 3.63	20	31.94 ± 4.84

[e]$P < 0.001$, *vs* Hp (-). CSG: Chronic superficial gastritis; CAG: Chronic atrophic gastritis ; Hp: *Helicobacter pylori*.

Table 2 Potential difference in *Helicobacter pylori* (+) patients before and after treatment

Group	*n*	Antrum		Corpus		YPD
		before	after	before	after	
I	9	16.88 ± 2.53	20.03 ± 2.21[b]	29.58 ± 2.50	32.41 ± 2.33	3.15 ± 1.9
II	6	16.50 ± 3.12	17.80 ± 3.63	31.84 ± 5.61	33.18 ± 4.22	1.30 ± 0.79
III	6	17.93 ± 2.58	17.53 ± 3.95	31.35 ± 1.57	31.33 ± 2.70	-0.39 ± 1.7

[b]$P < 0.01$, YPD = elevated PD in antrum after treatment. Group I: The De-Nol treatment; Group II: The WeiTong-Ling treatment; Group III: Placebo; PD: Potential difference.

Table 3 Potential difference in *Helicobacter pylori* (-) patients before and after treatment

Group	*n*	Antrum		Corpus		YPD
		before	after	before	after	
I	9	18.78 ± 2.50	20.11 ± 2.46	32.73 ± 3.61	33.30 ± 3.09	1.34 ± 2.37
II	9	19.69 ± 2.71	20.86 ± 1.99	34.27 ± 5.26	34.54 ± 4.97	1.17 ± 1.34
III	4	19.46 ± 2.99	19.67 ± 2.53	37.13 ± 3.32	36.96 ± 3.42	0.21 ± 0.39

Group I: The De-Nol treatment; Group II: The WeiTong-Ling treatment; Group III: Placebo; YPD: Elevated potential difference in antrum after treatment.

loosing of the connection of epithelial cells by the Hp infection and local increment of ammonia products.

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S- Editor: Filipodia **L- Editor**: Jennifer **E- Editor**: Zhang FF

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ISSN 1007-9327