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慢性胃病患者胃蛋白酶原Ⅰ、Ⅱ水平与幽门螺旋杆菌感染的关系研究*

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摘要 目的:探讨慢性胃病患者胃蛋白酶原(PG)I、PG II 水平与幽门螺旋杆菌(HP)感染的关系。**方法:**选取 2012 年 12 月 -2016 年 12 月期间我院收治的慢性胃病患者 64 例作为研究对象,根据疾病类型分为慢性胃炎组 23 例、胃溃疡组 22 例以及胃癌组 19 例。另取同期于我院接受体检的健康志愿者 30 例作为对照组,应用免疫比浊法测定各组血清 PG I 与 PG II 水平,采用快速尿激酶法测定各组 HP 感染情况，分别对比各组研究对象 HP 感染发生情况，血清 PG I、PG II、PG I/PG II 水平,HP 感染情况与血清 PG I、PG II、PG I/PG II 水平关系。**结果:**慢性胃炎组、胃溃疡组以及胃癌组患者 HP 阳性率分别为 60.87%、63.64%、78.95%,均明显高于对照组的 13.33%(P<0.05)。慢性胃炎组、胃溃疡组以及胃癌组患者血清 PG I、PG I/PG II 水平均低于对照组,且胃癌组低于慢性胃炎组与胃溃疡组(P<0.05),慢性胃炎组和胃溃疡组血清 PG I、PG I/PG II 水平比较差异无统计学意义(P>0.05),各组血清 PG II 比较无统计学差异(P>0.05)。各组研究对象 HP 阳性血清 PG I、PG I/PG II 水平均低于 HP 阴性(P<0.05),而 PG II 水平比较无统计学差异(P>0.05),慢性胃炎组、胃溃疡组、胃癌组 HP 阳性血清 PG I 水平低于对照组,且胃癌组低于慢性胃炎组、胃溃疡组(P<0.05),胃溃疡组、胃癌组 HP 阳性血清 PG I/PG II 水平低于对照组,且胃癌组低于慢性胃炎组(P<0.05)。**结论:**慢性胃病患者 PG I、PG II 水平异常降低,HP 阳性患者 PG I、PG II 水平降低更为明显,随病变的程度增加,血清 PG I、PG I/PG II 水平也呈现出下降的趋势。

关键词:慢性胃病;胃蛋白酶原;幽门螺旋杆菌;胃癌

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The Relationship Between the Levels of Pepsinogen I, II and *Helicobacter Pylori* Infection in Patients with Chronic Gastric Disease*

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ABSTRACT Objective: To study the relationship between pepsinogen (PG) I, II level and *Helicobacter pylori* (HP) infection in patients with chronic gastric disease. **Methods:** 64 cases with chronic gastric disease who were treated in our hospital from December 2012 to December 2016 were selected as the subjects, which were divided into chronic gastritis group (23 cases), gastric ulcer group (22 cases) and gastric cancer group (19 cases) according to the type of disease. Another 30 healthy persons in our hospital during the same period were selected as the control group. The serum levels of PG I and PG II in each group were measured by immunoturbidimetry, the HP infection rate in each group was determined by rapid urokinase method, the incidence of HP infection, the serum levels of PG I, PG II, PG I/PG II in each group were compared, the relationship between HP infection and serum level of PG I, PG II and PG I/PG II were compared. **Results:** The positive rates of HP in chronic gastritis group, gastric ulcer group and gastric cancer group were 60.87%, 63.64% and 78.95%, respectively, which were significantly higher than 13.33% of the control group (P<0.05). The serum levels of PG I, PG I/PG II in chronic gastritis group, gastric ulcer group and gastric cancer group were lower than those in the control group, and the gastric cancer group were lower than that in chronic gastritis group and gastric ulcer group (P<0.05), there was no significant difference in serum PG II in each group (P>0.05). The levels of PG I and PG I/PG II in the HP positive of all the subjects were lower than that of HP negative (P<0.05), there was no significant difference in serum PG II in each group (P>0.05), serum PG I levels of HP positive in chronic gastritis, gastric ulcer group and gastric cancer group were lower than those in the control group, and the gastric cancer group were lower than those of chronic gastritis, gastric ulcer group (P<0.05), the level of PG I/PG II of HP positive serum in gastric ulcer group and gastric cancer group was lower than that of the control group, and the gastric cancer group was lower than that of the chronic gastritis group (P<0.05). **Conclusion:** The levels of PG I and PG II in patients with chronic gastric disease are decrease, the levels of PG I and PG II in HP positive patients decreased more significantly, the level of serum PG I and PG I/PG II also show a declining trend with the increase of the degree of disease.

Key words: Chronic gastric disease; Pepsinogen; *Helicobacter pylori*; Gastric cancer

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前言

慢性胃病属于临床消化系统中最为常见的疾病之一,主要包括慢性胃炎、胃溃疡以及胃癌等[1,2]。目前研究认为,慢性胃病的发病机制复杂,生物因素、遗传因素、环境因素、感染因素以及免疫因素等均是引发慢性胃病的主要因素[3,4]。其中幽门螺旋杆菌(*helicobacter pylori*, HP)感染是目前已经证实的慢性胃病发病的重要病因[5]。有研究报道指出 HP 感染患者发病初期主要表现为慢性浅表性胃炎,随着病情的不断恶化与进展,可发展成为慢性萎缩性胃炎以及非典型增生等,严重者甚至会引发胃癌[6,7]。胃蛋白酶原(pepsinogen, PG)是一种由胃粘膜分泌的蛋白酶原,随着胃黏膜病变的发生,其分泌水平会发生一定程度的变化,可有效反映机体胃黏膜生理状态[8,9]。PG 主要分为 PGⅠ与 PGⅡ两个亚型,但关于 PG I、PG II 在慢性胃病中的变化目前仍没有统一的说法。鉴于此,本文通过研究慢性胃病患者 PG I、PG II 水平与 HP 感染的关系并予以分析,旨在为临床早期有效诊断慢性胃炎以及胃癌提供参考依据,现作如下报道。

1 资料与方法

1.1 一般资料

选取 2012 年 12 月 -2016 年 12 月期间我院收治的慢性胃病患者 64 例作为研究对象,纳入标准:(1)所有患者均经胃镜检查确诊为胃炎、胃溃疡及胃癌[10];(2)入院前未接受放化疗治疗;(3)均耐受胃镜检查、血清学检查者;(4)病情较为稳定。排除标准:(1)合并心、肝、肾等脏器功能严重障碍者;(2)入院前 30 d 内接受过 HP 根治者;(3) 接受过质子泵抑制剂以及抗生素治疗者;(4)伴有精神疾病或交流沟通障碍者。根据疾病类型的不同分为慢性胃炎组 23 例、胃溃疡组 22 例以及胃癌组 19 例。另取同期于我院接受体检的健康志愿者 30 例作为对照组。其中慢性胃炎组男 14 例,女 9 例,年龄 31-77 岁,平均(53.43± 2.94) 岁。胃溃疡组男 14 例, 女 8 例, 年龄 33-74 岁, 平均(53.02± 2.85)岁。胃癌组男 13 例,女 6 例,年龄 37-77 岁,平均(53.85± 3.13)岁。对照组男性患者 19 例,女性患者 11 例,年龄 33-76 岁,平均(53.34± 3.02)岁。各组研究对象性别、年龄比较,差异无统计学意义(P>0.05),组间存在可比性。各组研究对象均签署了知情同意书,我院伦理委员会已批准同意本次研究。

1.2 研究方法

慢性胃炎、胃溃疡以及胃癌患者于入院后采集清晨空腹静脉血 5 mL,对照组于体检当日采集清晨空腹静脉血 5 mL。以 3000 r/min 的速度离心 10 min, 取上层血清保存于 -80℃冰箱中待检。采用免疫比浊法测定各组研究对象血清 PG I 与 PG II 水平。仪器为日本奥林巴斯 2700 全自动生化分析仪,严格按照试剂盒(购自北京九强生物有限公司)说明书进行操作。PG I 参考值范围为 70-210 μg/L,PG II 的为 0-15 μg/L。采用快速尿激酶法测定各组研究对象 HP 感染情况, 研究对象于检查前 4 h 禁食, 均予以胃镜检查, 并于胃窦大小弯处取一体积为 1 cm^3 样本进行测定,且留取胃黏膜组织标本。采用浓度为 4%的多聚甲醛对胃黏膜组织进行固定,行常规石蜡包埋,制作标本切片为 5 μm,予以常规伊红染色法进行染色处理。仪器为广州华友明康光电科技有限公司的 HY-IREXB 碳 13 呼气检测仪,试剂盒购自北京勃然制药有限公司,名称为尿素 13C 呼气试验诊断试剂盒,严格按照试剂盒说明书进行操作。检验标准:DOB 值 <4.0 为阴性,DOB 值≥ 4.0 为阳性。

1.3 观察指标

分别对比各组研究对象 HP 感染发生情况, 血清 PG I、PG II、PG I/PG II 水平,HP 感染情况与血清 PG I、PG II、PG I/PG II 水平的关系。

1.4 统计学方法

采用 SPSS25.0 统计学软件进行统计分析, 计数资料以率(%)表示,采用 x^2 检验,计量资料以($\bar{x}\pm s$)表示,多组数据比较应用单因素方差分析, 两组数据比较采用 t 检验,P<0.05 为差异有统计学意义。

2 结果

2.1 各组研究对象 HP 阳性率比较

慢性胃炎组、胃溃疡组以及胃癌组患者 HP 阳性率均显著高于对照组,差异有统计学意义(P<0.05);而慢性胃炎组、胃溃疡组、胃癌组患者 HP 阳性率比较差异无统计学差异(P>0.05),见表 1。

表 1 各组研究对象 HP 阳性率比较

Table 1 Comparison of the positive rates of HP between the four groups

Groups	n	HP positive (case)	HP negative (case)	HP positive rate (%)
Chronic gastritis group	23	14	9	60.87[a]
Gastric ulcer group	22	14	8	63.64[a]
Gastric cancer group	19	15	4	78.95[a]
Control group	30	4	26	13.33

Note: compared with the control group, [a]P<0.05.

2.2 各组研究对象血清 PG I、PG II 及 PG I/PG II 水平比较

慢性胃炎组、胃溃疡组以及胃癌组患者血清 PG I、PG I/PG II 水平均低于对照组, 且胃癌组低于慢性胃炎组与胃溃疡组(P<0.05),慢性胃炎组和胃溃疡组血清 PG I、PG I/PG II 水平比较差异无统计学意义(P>0.05),各组血清 PG II 比较无统计学差异(P>0.05)。见表 2。

2.3 不同 HP 感染者血清 PG I、PG II 及 PG I/PG II 水平比较

各组研究对象 HP 阳性血清 PG I、PG I/PG II 水平均低于 HP 阴性,差异有统计学意义(P<0.05),而 PG II 水平比较无统计学差异(P>0.05),慢性胃炎组、胃溃疡组、胃癌组 HP 阳性血

清 PG I 水平低于对照组，且胃癌组低于慢性胃炎组、胃溃疡组，差异有统计学意义（P<0.05），胃溃疡组、胃癌组 HP 阳性血清 PG I/PG II 水平低于对照组，且胃癌组低于慢性胃炎组，差异有统计学意义（P<0.05）。慢性胃炎组、胃溃疡组 HP 阳性血清 PG I、PG I/PG II 水平比较无统计学差异（P>0.05），见表 3。

表 2 各组研究对象血清 PG I、PG II 及 PG I/PG II 水平比较（$\bar{x}\pm s$）

Table 2 Comparison of serum levels of PG I,PG II and PG I /PG II between the four groups（$\bar{x}\pm s$）

Groups	n	PG I（μg/L）	PG II（μg/L）	PG I/PG II
Chronic gastritis group	23	86.32± 12.38[ab]	15.01± 2.85	5.75± 1.03[ab]
Gastric ulcer group	22	83.01± 11.52[ab]	15.33± 2.81	5.41± 0.73[ab]
Gastric cancer group	19	75.44± 10.01[a]	15.19± 3.01	4.93± 0.71[a]
Control group	30	99.44± 15.41	14.83± 2.59	6.71± 1.37
F	-	53.751	2.282	16.282
P	-	0.000	0.156	0.035

Note: compared with the control group, [a]P<0.05; compared with the gastric cancer group, [b]P<0.05.

表 3 不同 HP 感染者血清 PG I、PG II 及 PG I/PG II 水平比较（$\bar{x}\pm s$）

Table 3 Comparison of serum PG I, PG II and PG I/PG II in different HP infection（$\bar{x}\pm s$）

Groups		n	PG I（μg/L）	PG II（μg/L）	PG I/PG II
Chronic gastritis group	HP positive	14	76.37± 6.51[abc]	15.03± 2.91	5.08± 0.62[ac]
	HP negative	9	102.01± 10.11	14.99± 2.20	6.80± 1.02
Gastric ulcer group	HP positive	14	73.65± 7.52[abc]	15.13± 2.44	4.76± 0.69[ab]
	HP negative	8	101.25± 9.84	15.67± 2.05	6.46± 1.99
Gastric ulcer group	HP positive	15	68.34± 7.01[ab]	15.22± 2.70	4.49± 0.51[ab]
	HP negative	4	100.18± 8.65	15.13± 2.57	6.62± 2.28
Gastric ulcer group	HP positive	4	85.81± 6.01[a]	14.82± 2.32	5.58± 0.60[a]
	HP negative	26	102.03± 8.99	14.86± 2.61	6.86± 0.97

Note: compared with HP negative, [a]P<0.05; compared with the control group, [b]P<0.05; compared with gastric cancer group, [c]P<0.05.

3 讨论

近年来，随着人们生活水平的不断提高以及饮食习惯的改变，慢性胃病的发生率正呈逐年上升趋势，已成为全球范围内严重威胁人们生命健康安全的重要疾病，受到了临床广泛关注[11-13]。HP 是目前所知的唯一能在人胃中生存的微生物，属于螺旋状革兰阴性微需氧菌之一，多见于胃黏膜上皮，是临床上公认的 I 类致癌原[14,15]。HP 感染所引发的一系列炎症反应长期作用，会促使患者胃黏膜损伤、萎缩，进一步发生慢性胃炎以及胃癌前病变等，最终导致胃癌的发生。PG 主要是由胃黏膜分泌，可有效反映胃黏膜细胞和腺体的数量，从而反映胃黏膜的萎缩程度[16-18]。PG 主要包括 PG I 与 PG II 两个亚群，而在胃黏膜出现病变时，PG 分泌水平会出现一定的变化，从而促使血清中的 PG I、PG II 水平发生变化[19,20]。因此，我们可通过对血清 PG I 与 PG II 水平进行检测，从而有助于慢性胃病的鉴别诊断。

本文结果发现，慢性胃炎组、胃溃疡组以及胃癌组患者 HP 阳性率分别为 60.87%、63.64%、78.95%，均明显高于对照组的 13.33%（P<0.05），这与孙怡和 Loong TH 等的研究报道相一致[21,22]，说明了慢性胃病患者存在不同程度的 HP 感染。其中主要原因可能是在正常生理状态下，机体一旦感染 HP，免疫系统中的巨噬细胞以及白细胞均会分泌淋巴抗体对 HP 的增殖产生抑制作用。而慢性胃病患者机体免疫功能下降，从而使得免疫系统无法有效清除 HP，进一步促使 HP 大量的生长繁殖，最终导致胃黏膜病变。另有研究报道显示[23-25]，随着胃黏膜病变程度的不断恶化，HP 感染率呈逐渐上升趋势。而本文结果显示慢性胃炎组、胃溃疡组以及胃癌组患者 HP 阳性率对比均不明显，这可能在于随着胃黏膜病变逐渐恶化，促使胃黏膜组织分泌黏液性质出现变化，从而不利于 HP 的定值，进一步导致活动性 HP 量减少，最终降低了 HP 阳性率。此外，慢性胃炎组、胃溃疡组以及胃癌组患者血清 PG I、PG I/PG II 水平均低于对照组，且胃癌组低于慢性胃炎组与胃溃疡组（P<0.05），这符合 Manabe S 和 Cho JH 等人的研究报道[26,27]。说明了慢性胃病患者血清 PG I、PG I/PG II 水平存在低表达，且随着病变程度的不断增加，血清 PG I、PG I/PG II 水平下降越明显。因为 PG I 是反映胃泌酸腺细胞功能的重要指标，而 PG II 主要与胃底病变有关，PG I、PG I/PG II 可能在慢性胃病的发生、发展过程中发挥着至关重要的作用[28,29]。其中 PG 属于临床上应用于反映胃黏膜状态的可靠指标之一，且在胃病进展的不同阶段其分泌量会出现不同的改变，有利于反映胃黏膜的分泌功能。而随着胃黏膜遭受损伤，PG 的分泌会随之减少[30]。本研究结果显示，各组研究对象 HP 阳性血清 PG I、PG I/PG II 水平均低于 HP 阴性，慢性胃炎组、胃溃疡组、胃癌组 HP 阳性血清 PG I 水平低于对照组，且胃癌组低于慢性胃炎组、胃溃疡组（P<0.05），胃溃疡组、胃癌组 HP 阳性血清 PG I/PG II 水平低于对照组，且胃癌组低于慢性

胃炎组(P<0.05)。这提示了 PG Ⅰ、PG Ⅰ/PG Ⅱ水平与 HP 感染存在密切相关,且在不同胃病中关系存在一定差异。分析原因,作者认为机体在发生 HP 感染后可通过对胃泌素分泌进行刺激,胃泌酸腺细胞功能失调,主要表现为 PG Ⅰ分泌降低,而 PG Ⅱ则与 HP 感染关系不大。同时,随病变的程度增加,血清 PG Ⅰ、PG Ⅰ/PG Ⅱ水平也呈现出变化,因此临床上可以通过检测慢性胃病患者血清 PG Ⅰ、PG Ⅱ水平,为慢性胃病诊断提供辅助依据。

综上所述,慢性胃病患者 PG Ⅰ、PG Ⅱ水平与 HP 感染存在密切相关,提示了我们在临床工作中可通过检测慢性胃病患者血清 PG Ⅰ、PG Ⅱ水平,从而判断患者 HP 阳性情况,进一步有利于胃癌的早期发现,值得临床推广应用。

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