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Change and clinical significance of serum PG in patients with chronic gastritis

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ABSTRACT

Objective: To observe the change and clinical significance of serum PG in patients with chronic atrophic gastritis (CAG). **Methods:** ELISA was used to detect the peripheral blood PG level in patients confirmed with CAG, gastric polyps, and gastric cancer who were admitted in our hospital from January, 2015 to January, 2016. The normal individuals who came for physical examinations were served as the control group. The peripheral blood PG level in patients with various gastric diseases was observed. **Results:** The serum PG Ⅰ expression and PG I/PG Ⅱ in the gastritis group were significantly lower than those in the gastric polyps group and control group, but were significantly higher than those in the gastric cancer group; while PG Ⅱ expression was significantly higher than that in the gastric polyps group and control group, but was significantly lower than those in the gastric cancer group. PG Ⅰ expression and PG I/ PG Ⅱ in the gastric polyps group were significantly higher than those in the gastritis group and gastric cancer group, while PG Ⅱ expression was significantly lower than that in the gastritis group and gastric cancer group. PG Ⅰ expression and PG I/ PG Ⅱ in the gastric cancer group were significantly lower than those in the other three groups, while PG Ⅱ expression was significantly higher than that in the other three groups. The serum PG Ⅰ expression in patients with positive HP infection in the gastritis group and gastric cancer group was significantly higher than that in patients with negative HP infection, but the comparison of PG I/ PG Ⅱ was not statistically significant. The serum PG Ⅰ expression and PG I/ PG Ⅱ in patients with negative and positive HP infection in the gastritis group were significantly higher than those in patients with negative and positive HP infection in the gastric cancer group; while PG Ⅱ expression was significantly was significantly lower than that in the gastric cancer group. **Conclusions:** PG Ⅰ expression and PG I/ PG Ⅱ in patients with CAG are significantly reduced, but PG Ⅱ expression is significantly strengthened; while PG Ⅰ expression and PG I/ PG Ⅱ are significantly higher than those in the gastric cancer group, but PG Ⅱ expression is significantly lower than that in the gastric cancer group. It should pay attention to the changes of the above indicators in the clinical treatment. If PG Ⅰ expression and PG I/ PG Ⅱ are significantly reduced, but PG Ⅱ expression is significantly strengthened, it should pay attention to the probability of condition deterioration.

1. Introduction

CAG is a chronic digestive system disease, and is common in the clinic, with pathological manifestation of gastric epithelial and gland atrophy[1]. Some researches demonstrate that about 60%−90% CAG patients are caused by HP infection, and in the World Gastroenterology Society Eighth Conference (1986), HP infection is deemed to be one of the important factors for developing CAG[2,3]. Epigastric pain, belching, distention, anorexia, and anemia are mainly involved in the clinical manifestations of CAG patients, which can severely affect the patients' living qualities; moreover, CAG is also a kind of multi-pathogenic factor disease and precancerous lesion[4,5]; therefore, early accurate diagnosis and treatment of CAG is of great significance in preventing or delaying its conversion to gastric cancer. The study is aimed to observe the change and clinical significance of serum PG in patients with CAG.

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2. Materials and methods

2.1. Clinical materials

A total of 100 patients with gastric diseases who were admitted in our hospital from January, 2015 to January, 2016 were included in the study and divided into the gastritis group (n=35), gastric polyps group (n=30), and gastric cancer group (n=35). In the gastritis group, 26 were male, and 9 were female; aged from 33 to 74 years old; 20 had positive HP infection confirmed by ^{14}C-UBT test, and 15 had negative HP infection. In the gastric polyps group, 19 were male, and 11 were female; aged from 34 to 79 years old. In the gastric cancer group, 20 were male, and 15 were female; aged from 41 to 78 years old; 17 had positive HP infection confirmed by ^{14}C-UBT test, and 18 had negative HP infection. The patients in the above three groups were confirmed by the gastroscope and gastric mucosa biopsy. A total of 35 normal individuals who came for physical examinations were served as the control group, among which 18 were male, and 17 were female. Those who had vital organic diseases and taken drugs affecting the experiment results recently were excluded from the study. The comparison of gender and age among the four groups was comparable (P>0.05).

2.2. Instruments and equipment

Finnpipette, low speed centrifuge (Beijing Conson Scientific and Technological Co. Ltd), DG5033A type ELISA meter, PG-Ⅰ, and PG-Ⅱ (Beijing Nymphavn Biotechnology Co. Ltd) were purchased.

2.3. Observation indicators

The fasting venous blood was collected, centrifuged for the serum, and preserved at -80 ℃ for inspection. ELISA was used to detect the serum PG I and PG Ⅱ contents.

2.4. Statistical analysis

SPSS 12.0 software was used for the statistical analysis. The measurement data were expressed as mean ± SD, and t test was used. P<0.05 was regarded as statistically significant.

3. Results

3.1. Comparison of the serum PG expression among each group

The serum PG Ⅰ expression and PG I/ PG Ⅱ in the gastritis group were significantly lower than those in the gastric polyps group and control group (P<0.05), but were significantly higher than those in the gastric cancer group (P<0.05); while PG Ⅱ expression was significantly higher than that in the gastric polyps group and control group (P<0.05), but was significantly lower than those in the gastric cancer group (P<0.05). PG Ⅰ expression and PG I/ PG Ⅱ in the gastric polyps group were significantly higher than those in the gastritis group and gastric cancer group (P<0.05), while PG Ⅱ expression was significantly lower than that in the gastritis group and gastric cancer group (P<0.05). PG Ⅰ expression and PG I/ PG Ⅱ in the gastric cancer group were significantly lower than those in the other three groups (P<0.05), while PG Ⅱ expression was significantly higher than that in the other three groups (P<0.05) (Table 1).

3.2. Comparison of the serum PG expression in patients with negative and positive HP infection in the gastritis group and gastric cancer group

The serum PG Ⅰ expression in patients with positive HP infection in the gastritis group and gastric cancer group was significantly higher than that in patients with negative HP infection (P<0.05), but the comparison of PG I/ PG Ⅱ was not statistically significant (P>0.05). The serum PG Ⅰ expression and PG I/ PG Ⅱ in patients with negative and positive HP infection in the gastritis group were significantly higher than those in patients with negative and positive HP infection in the gastric cancer group (P<0.05); while PG Ⅱ expression was significantly was significantly lower than that in the gastric cancer group (P<0.05) (Table 2).

Table 2.

Comparison of the serum PG expression in patients with negative and positive HP infection in the gastritis group and gastric cancer group (ng/mL).

Groups	HP	n	PG Ⅰ	PG Ⅱ	PG I/ PG Ⅱ
Gastritis	Positive	20	85.22±18.61$^{*\#}$	17.62±5.22$^{*\#}$	5.22±1.87$^{\#}$
	Negative	15	64.90±19.43$^{\#}$	13.79±4.66$^{\#}$	5.05±2.06$^{\#}$
Gastric cancer	Positive	17	68.96±19.09*	21.28±6.34*	3.51±1.41
	Negative	18	53.14±23.04	16.65±4.39	3.45±1.97

$^{*}P$<0.05, when compare with the patients with negative HP infection; $^{\#}P$<0.05, when compared with the gastric cancer group.

Table 1.

Comparison of the serum PG expression among each group (ng/mL).

Groups	n	PG Ⅰ	PG Ⅱ	PG I / PG Ⅱ
Gastritis	35	78.44±20.92$^{*\Delta\blacktriangle}$	16.34±5.28 $^{*\Delta\blacktriangle}$	5.17±1.90$^{*\Delta\blacktriangle}$
Gastric polyps	35	100.71±33.35$^{\#\blacktriangle}$	11.59±2.42$^{\#\blacktriangle}$	8.81±2.68$^{*\#\blacktriangle}$
Gastric cancer	35	63.93±21.25$^{*\#\Delta}$	19.80±6.09$^{*\#\Delta}$	3.50±1.56$^{*\#\Delta}$
Control	35	161.60±34.65	13.60±3.05	12.51±3.84

*P<0.05, when compared with the control group; $^{\#}P$<0.05, when compare with the gastritis group; $^{\Delta}P$<0.05, when compared with gastric polyps group; $^{\blacktriangle}P$<0.05, when compared with the gastric cancer group.

4. Discussion

HP infection is a main factor for developing CAG. Some researches demonstrate that CAG caused by HP infection is positively correlated with gastric cancer[6-9]. HP infection can cause the long-term inflammatory damage of gastric mucosa, resulting in gastric mucosa atrophy; moreover, HP infection can also increase the secretion of gastric acid and gastrin, and destroy the balance between the mucosa defense factor and invasion factor, finally leading to the occurrence of gastric mucosa diseases[10]; therefore, early HP infection examination should be performed for patients with gastric diseases in the clinic in order to timely prevent CAG and precancerous lesion.

PG is mainly synthetized by the oxyntic gland cells, is the precursor of pepsin, is converted into pepsin through the gastric hydrochloric acid and active pepsin, and loses activity after entering the small intestines[11]. PG is mainly divided into PG I and PG Ⅱ types, whose level can reflect the pathophysiological change of gastric mucosa[12]. Some researches demonstrate that detection of PG I and PG I/ PG Ⅱ in the clinic is of great significance in the diagnosis of CAG and intestinal metaplasia (IM), while CAG is the precancerous lesion, i.e. from CAG-IM dysplasia to the gastric cancer[13,14]; therefore, early detection and treatment in the clinic play a key role in enhancing the prognosis. PG is the biological marker of gastric mucosa expression, whose serum expression level can reflect the gastric mucosa function and morphological change in different regions. PG I is an indicator to detect the oxyntic gland cell function, while PG Ⅱ is greatly correlated with the gastric mucosa lesions, whose elevation is associated with the gastric metaplasia, fundus gland duct atrophy, dysplasia, and pseudopyloric metaplasia, and PG I/ PG Ⅱcan accurately reflect the progression degree of gastric mucosa atrophy[15,16]; therefore, the combined detection of PG I and PG II can accurately estimate the pathological change of gastric gland mucosa, and provide a reliable diagnostic value. The results in the study showed that the serum PG Ⅰ expression and PG I/ PG Ⅱ in the gastritis group were significantly lower than those in the gastric polyps group and control group (P<0.05), but were significantly higher than those in the gastric cancer group (P<0.05); while PG Ⅱ expression was significantly higher than that in the gastric polyps group and control group (P<0.05), but was significantly lower than those in the gastric cancer group (P<0.05); PG Ⅰ expression and PG I/ PG Ⅱ in the gastric polyps group were significantly higher than those in the gastritis group and gastric cancer group (P<0.05), while PG Ⅱ expression was significantly lower than that in the gastritis group and gastric cancer group (P<0.05); PG Ⅰ expression and PG I/ PG Ⅱ in the gastric cancer group were significantly lower than those in the other three groups (P<0.05), while PG Ⅱ expression was significantly higher than that in the other three groups (P<0.05), indicating that in CAG patients, if PG Ⅰ expression and PG I/ PG Ⅱ are significantly reduced, but PG Ⅱ expression is significantly strengthened, it should pay attention to the probability of condition deterioration. The results in the study showed that the serum PG Ⅰ expression in patients with positive HP infection in the gastritis group and gastric cancer group was significantly higher than that in patients with negative HP infection (P<0.05), but the comparison of PG I/ PG Ⅱ was not statistically significant (P>0.05); the serum PG Ⅰ expression and PG I/ PG Ⅱ in patients with negative and positive HP infection in the gastritis group were significantly higher than those in patients with negative and positive HP infection in the gastric cancer group (P<0.05); while PG Ⅱ expression was significantly was significantly lower than that in the gastric cancer group (P<0.05), suggesting that it should pay attention to identify the change of serum PG expression in patients with CAG and gastric cancer caused by HP infection in the clinical application.

In conclusion, PG Ⅰ expression and PG I/ PG Ⅱ in patients with CAG are significantly reduced, but PG Ⅱ expression is significantly strengthened; while PG Ⅰ expression and PG I/ PG Ⅱ are significantly higher than those in the gastric cancer group, but PG Ⅱ expression is significantly lower than that in the gastric cancer group. It should pay attention to the changes of the above indicators in the clinical treatment. If PG Ⅰ expression and PG I/ PG Ⅱ are significantly reduced, but PG Ⅱ expression is significantly strengthened, it should pay attention to the probability of condition deterioration.

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