The non-invasive diagnosis of precancerous changes of stomach mucosa

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Abstract

Purpose: To detect the Helicobacter pylori (H.pylori)-induced gastric precancerous lesions leading to cancer formation, and to evaluate the possibility of non-invasive screening of dyspeptic patients to identify those having high risk of gastric cancer.

Material and methods: 178 consecutive H.pylori-positive dyspeptic patients after assessment of serum pepsinogen-1 (PG-1) and gastrin-17 (G-17) levels by enzyme immunoassay were examined with endoscopy and histology. The serologic and morphologic results were compared with estimating the sensitivity, specificity and prognostic values of the tests.

Results: There was statistically significant reverse dependence between the presence and severity of stomach mucosal atrophy (in antrum or corpus) and the proper serologic markers of stomach functional activity (G-17 or PG-1). On the other hand, the presence and the degree of intestinal metaplasia, dysplasia and gastric cancer did not correspond to the serum levels of G-17 or PG-1. The serologic method was quite sensitive in the diagnosis of non-atrophic and severe antral and corpus gastritis. Also, it was characterized by the high positive and negative prognostic values. Additionally, we have established the obvious advantage of the chromoendoscopy method in the diagnosis of intestinal metaplasia in the stomach epithelium.

Conclusions: The assays of serum G-17 and PG-1 levels can be offered as the screening tool for atrophic gastritis. The positive serologic results require further chromoendoscopic examination with mucosal biopsy to disclose the probable progression of atrophic process with development of intestinal metaplasia, dysplasia or gastric cancer.

Key words: gastric cancer, pepsinogen, gastrin, non-invasive screening, chromoendoscopy.

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Introduction

It has been precisely proven, that the cancer does not arise in healthy gastric mucosa. Various clinical and morphological studies have allowed the better understanding of pathological processes, which probably are precancerous. The malignant transformation of the normal gastric epithelial cells represents multistage process associated with progressing accumulation of genetic damages. According to the modern representations about stages of gastric carcinogenesis it is supposed that chronic Helicobacter pylori (H.pylori) infection is the trigger mechanism in 60-90% of gastric cancer cases. In a number of cases, H.pylori infection can be the only important etiological factor in development of gastric cancer. H.pylori has not been found out in a normal stomach, however it is frequently present in chronic gastritis, which progresses into atrophic gastritis, with subsequent development of intestinal metaplasia. This latter is seldom revealed in the absence of H.pylori infection. Molecular changes in H.pylori-infection, which are capable to result in the cancer development, are not established. There are no studies showing synthesis or secretion any mutagenous or carcinogenous substances by H.pylori. The current opinion is that H.pylori acts as a promotor rather than initiator of gastric carcinogenesis. H.pylori infection leads to the development of inflammatory reaction in gastric mucosa, with the production of reactive oxygen species by polymorphonuclear leucocytes and release of the cytokines by inflammatory cells, that results in the DNA damage and in stimulation of receptors triggering of cellular proliferation [1,2].

It is conventional to distinguish the precancerous (or

background) diseases and the precancerous changes of gastric mucosa. The first group represents pathological conditions, which under the appropriate circumstances can result in the development of gastric cancer (chronic gastritis, ulcer disease, polyps of a stomach, the resected stomach) i.e. associate with the increased risk of cancer development. The second group contains the morphologically proved changes of gastric mucosa reliably indicating on the development of the pathological process towards the malignant growth. These changes, under the recommendation a WHO (1978), were designated by the term "dysplasia". The possibility of progression of severe dysplasia to gastric cancer varies, according to the different data, from 8 up to 75%. The combination of precancerous conditions with precancerous changes of gastric mucosa really raises risk of cancer development. In the 1975 Correa proposed the consecutive line of events leading to gastric cancer development: normal mucosa – chronic active gastritis – chronic atrophic gastritis – intestinal metaplasia – dysplasia – carcinoma in situ. At present this cascade of changes associates with a trigger role H.pylori and is called "Correa's gastric precancerous cascade" [2,3].

The early detection of gastric precancerous changes, mainly atrophic gastritis, may be helpful in the prevention of gastric cancer or in the diagnosis of cancer at curable of stages. So, there is a critical need for valid diagnostic methods of stomach mucosal atrophy, that would be inexpensive and non-invasive, that is, suitable for screening of the large groups of the people.

Taking into account known interrelations between the morphological status and functional activity of gastric corpus and antral mucosa and the secretion of pepsinogen-1 and gastrin-17, respectively [4,5], we carried out a prospective study with the aim to detect the H.pylori-induced gastric precancerous changes, that are leading to cancer formation, and to evaluate the possibility of non-invasive screening of dyspeptic patients to identify those having high risk of gastric cancer.

Material and methods

The study was carried out according to updated Declaration of Helsinki in a group of 178 dyspeptic H.pylori-infected patients (109 female, 69 male, aged from 15 to 85 years, in average 59.8 ± 13.7 years), after informed consent for examinations. Any eventual medication with proton pomp inhibitors, H2 antagonists and NSAIDS were excluded for at least one month before examinations. Fasting serum Helicobacter pylori antibodies (Hp-Ab), serum levels of pepsinogen-1 (PG-1) and gastrin-17 (G-17) were assayed by enzyme immunoassay with Biohit GastroPanel® (Biohit Plc, Helsinki, Finland). According to the instruction of manufacturer, serum levels of PG-1 < 25 μg/l were estimated as markers of gastric corpus atrophy; serum levels of G-17 < 5 pmol/l were estimated as markers of gastric antral atrophy; serum levels of G-17 < 10 pmol/l in a combination with serum levels of PG-1 < 50 μg/l were estimated as markers of mild gastric corpus atrophy. Hp-Ab IgG titers were estimated as follows: < 32 EIU (EIU – enzyme immunoassay unit) – negative result; 32 - 44 EIU – doubtful result; > 44 EIU – positive result. The numerical meanings of assayed parameters were analyzed by the program GastroSoft® (Biohit Plc, Helsinki, Finland) enclosed to test-system Biohit GastroPanel®. On the basis of inserted data, the program composed the diagnosis in a view of the presence or absence of H.pylori-infection and mucosal atrophy, with the estimating of gastric cancer or peptic ulcer risk and with recommendations on the treatment according to Maastricht-2 consensus.

After getting the GastroSoft® diagnosis we have randomized 178 patients for the following study. The patients were undergone the upper gastrointestinal endoscopy with subsequent biopsy of the antral and corpus mucosa. For the increasing of the accuracy of endoscopic diagnosis we carried out the additional chromoendoscopy with the methylene blue staining allowing the detection of foci of intestinal metaplasia of gastric mucosa which were unrecognized by routine endoscopy. The biopsy specimens were stained with hematoxylin-eosin and PAS reaction in the combination with alcian blue at pH 2.5. The grade of stomach mucosal atrophy was estimated from 0 to 3 according to Houston visual analogous scale.

The statistical analysis was used to calculate the statistical significance of received data (Mann-Whitney criterion), the Spearman's correlation coefficient (r_s), the positive predictive value (PPV) and negative predictive value (NPV) of diagnosis by Biohit GastroPanel®.

Results

Of 178 patients, non-atrophic chronic gastritis (no antral atrophy and no corpus atrophy) was detected in 5 patients. The average meanings of serum PG-1, G-17 and anti-H.pylori IgG in this group were, respectively, 85.28 ± 35.07 μg/l, 14.44 ± 1.90 pmol/l and 85.68 ± 17.81 EIU. In all these cases there were no IM or dysplasia in stomach mucosal epithelium.

The morphological status of gastric corpus mucosa was compared to serum PG-1 levels (*Fig. 1*). The non-atrophic corpus mucosa was detected in 99 (55.62%) of 178 patients. The average meanings of serum PG-1 and anti-H.pylori IgG in this group were, respectively, 105.2 ± 5.7 μg/l and 68.76 ± 8.35 EIU. Mild atrophy of corpus mucosa was detected in 17 (9.55%) patients. The average meanings of serum PG-1 and anti-H.pylori IgG in this group were, respectively, 18.71 ± 0.95 μg/l and 77.21 ± 6.13 EIU. Moderate atrophy of corpus mucosa was detected in 36 (20.22%) patients. The average meanings of serum PG-1 and anti-H.pylori IgG in this group were, respectively, 11.91 ± 0.49 μg/l and 76.47 ± 5.04 EIU. Severe atrophy of corpus mucosa was detected in 26 (14.61%) patients. The average meanings of serum PG-1 and anti-H.pylori IgG in this group were, respectively, 6.92 ± 0.45 μg/l and 72.61 ± 4.72 EIU. The statistical analysis revealed the marked differences between the levels of PG-1 in non-atrophic and in atrophic corpus gastritis. The levels of PG-1 in mild, moderate and severe corpus atrophy were significantly lower ($p < 0.0001$) than in non-atrophic state. In turn, the levels of PG-1 in mild, moderate and severe corpus atrophy were differed significantly from each other ($p < 0.0001$).

The morphological status of gastric antral mucosa was compared to serum G-17 levels (*Fig.* 2). The non-atrophic

Figure 1. **Serum PG-1 levels compared to different degrees of corpus atrophy (dotted lines designate the mean values of PG-1, μg/l)**

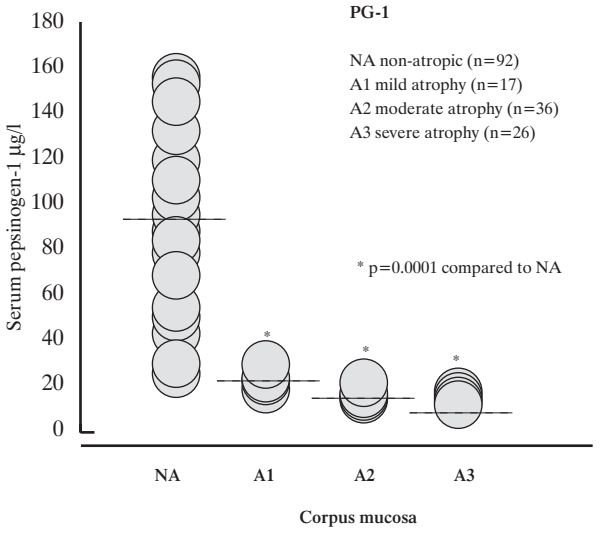

Figure 2. **Serum G-17 levels compared to different grades of antral atrophy (dotted lines designate the mean values of G-17, pmol/l)**

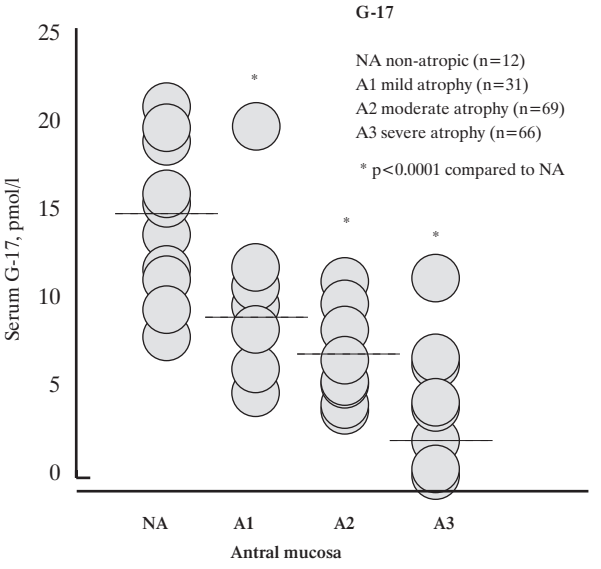

Table 1. **The comparisons of mean values of serum PG-1, G-17, Hp-Ab titers and the grade of IM**

	1 no IM (n=59)	2 mild IM (n=55)	3 moderate IM (n=41)	4 severe IM (n=23)	P_{1-2}	P_{1-3}	P_{2-3}	P_{1-4}	P_{2-4}	P_{3-4}
PG-1 (μg/l)	52.5 ± 7.74	66.07 ± 8.36	82.65 ± 10.72	53.08 ± 12.24	>0.05	<0.05	>0.05	>0.05	>0.05	>0.05
G-17 (pmol/l)	6.79 ± 0.55	5.48 ± 0.50	4.73 ± 0.67	5.11 ± 1.02	>0.05	<0.05	>0.05	>0.05	>0.05	>0.05
Hp-Ab (EIU)	81.23 ± 4.61	73.26 ± 4.81	72.09 ± 6.46	68.39 ± 7.94	>0.05	>0.05	>0.05	>0.05	>0.05	>0.05

antral mucosa was detected in 12 (6.74%) of 178 patients. The average meanings of serum G-17 and anti-H.pylori IgG in this group were, respectively, 14.57 ± 1.29 pmol/l and 62.29 ± 12.02 EIU. Mild atrophy of antral mucosa was detected in 31 (17.42%) patients. The means of serum G-17 and anti-H.pylori IgG in this group were, respectively, 8.91 ± 0.47 pmol/l and 78.25 ± 5.86 EIU. Moderate atrophy of antral mucosa was detected in 69 (38.76%) patients. The average meanings of serum G-17 and anti-H.pylori IgG in this group were, respectively, 6.40 ± 0.18 pmol/l and 74.08 ± 4.39 EIU. Severe atrophy of antral mucosa was detected in 66 (37.08%) patients. The average meanings of serum G-17 and anti-H.pylori IgG in this group were, respectively, 1.82 ± 0.26 pmol/l and 73.38 ± 5.38 EIU. The statistical analysis revealed the marked differences between the levels of G-17 in non-atrophic and in atrophic antral gastritis. The latter were significantly lower ($p<0.0001$) than in non-atrophic state. Moreover, the levels of G-17 in mild, moderate and severe antral atrophy were differed significantly from each other ($p<0.0001$).

We have detected intestinal metaplasia in 119 of 178 patients. The comparison of the grade of IM and the serum levels of PG-1, G-17 and anti-H.pylori IgG has showed the absence of the invariable, statistically significant dependence between these parameters (*Tab. 1*).

The dysplasia of stomach mucosal epithelium was detected in 113 patients. As in the cases of IM, we could not reveal the consistent and statistically significant dependence between the grade of dysplasia and the serum levels of PG-1, G-17 and anti-H.pylori IgG (*Tab. 2*).

Among 178 consecutive patients we have detected 6 cases of gastric cancer: 2 – early cancers and 4 – progressed tumors. The average meanings of serum PG-1, G-17 and anti-H.pylori IgG in these cases were, respectively, 46.73 ± 22.25 μg/l, 4.78 ± 2.086 pmol/l and 89.5 ± 11.7 EIU.

In general, our results have shown that there is a strong and statistically significant dependence between the presence and severity of stomach mucosal atrophy (antral or corpus) and the proper serologic markers (G-17 or PG-1) in H.pylori-associated chronic gastritis. On the other hand, the presence and the degree of IM, dysplasia and gastric cancer do not correspond to the serum levels of G-17 or PG-1. Thus, the serologic screening by means of Biohit GastroPanel® is useful for the selection of patients with stomach mucosal atrophy with subsequent thorough endoscopical and histological examination for the

Table 2. **The comparisons of mean values of serum PG-1, G-17, Hp-Ab titers and the grade of dysplasia**

	1 no dysplasia (n=65)	2 mild dysplasia (n=69)	3 moderate dysplasia (n=38)	4 severe dysplasia (n=6)	P_{1-2}	P_{1-3}	P_{2-3}	P_{1-4}	P_{2-4}	P_{3-4}
PG-1 (μg/l)	33.6 ± 5.41	79.98 ± 7.92	84.41 ± 10.98	71.88 ± 27.25	<0.05	<0.05	>0.05	>0.05	>0.05	>0.05
G-17 (pmol/l)	7.37 ± 0.46	5.28 ± 0.50	3.73 ± 0.66	4.53 ± 1.95	<0.05	<0.05	<0.05	>0.05	>0.05	>0.05
HpAb (EIU)	76.02 ± 4.15	77.65 ± 4.72	63.74 ± 6.32	91.27 ± 12.19	>0.05	>0.05	>0.05	>0.05	>0.05	>0.05

Table 3. **The correlations between results of different diagnostic methods**

Parameters	r_s
The detection of IM by endoscopy and by histology	0.41
The detection of IM by chromoendoscopy and by histology	0.92
The detection of antral atrophy by histology and the serum levels of G-17	- 0.75
The detection of corpus atrophy by histology and the serum levels of PG-1	- 0.71
The degree of stomach mucosal atrophy and of IM	0.21
The degree of stomach mucosal atrophy and of dysplasia	0.23
The degree of IM and of dysplasia	0.41

Table 4. **Sensitivity and specificity, PPV and NPV of Biohit GastroPanel® in diagnosis of stomach mucosal atrophy**

The degree of stomach mucosal atrophy (by histology)	Se	Sp	PPV	NPV
No antral atrophy	83%	95%	53%	99%
Mild antral atrophy	61%	84%	45%	91%
Moderate antral atrophy	67%	90%	81%	81%
Severe antral atrophy	89%	99%	98%	94%
No corpus atrophy	92%	97%	98%	91%
Mild corpus atrophy	71%	92%	48%	97%
Moderate corpus atrophy	72%	96%	81%	93%
Severe corpus atrophy	88%	97%	82%	98%

Table 5. **Sensitivity and specificity, PPV and NPV of routine endoscopy and chromoendoscopy in the diagnosis of IM**

Grade of IM	routine endoscopy				chromoendoscopy			
	Se	Sp	PPV	NPV	Se	Sp	PPV	NPV
no	98%	6%	35%	86%	94%	99%	98%	97%
mild	4%	98%	50%	68%	88%	88%	79%	94%
moderate	no data	no data	no data	no data	71%	95%	80%	92%
severe	6%	99%	33%	89%	82%	98%	82%	98%

possible development of precancerous or malignant changes in stomach mucosa.

Further we have compared the results of the serology, endoscopy and histology by means the Spearman's correlation coefficient (*Tab. 3*).

As shown in *Tab. 3*, the correlation between the results of routine endoscopy and histology was positive, but obviously weaker, than the correlation between the results of chromoendoscopy and histology. As it was expected, there was strong reverse correlation between the presence and the degree of stomach mucosal atrophy and the serum levels of the proper markers of its functional activity. The correlation between the degree of stomach mucosal atrophy and the subsequent morphological changes of mucosa – IM and dysplasia – was positive but quite weak.

The levels of sensitivity and specificity, PPV and NPV of Biohit GastroPanel® in diagnosis of stomach mucosal atrophy are presented in *Tab. 4*.

Thus, the investigated non-invasive method was quite sensitive in the diagnosis of non-atrophic and severe antral and corpus gastritis. Also, this method was characterized by the high PPV and NPV (except the cases of mild stomach mucosal atrophy).

Finally, we have carried out the comparison of sensitivity and specificity, PPV and NPV for routine endoscopy and chromoendoscopy in the diagnosis of IM (*Tab. 5*).

As a result, we have established the obvious advantage of a method of chromoendoscopy in the diagnosis of IM, that has doubtless diagnostic importance in the revealing of precancerous lesions of gastric mucosa.

Discussion

Gastric cancer remains the second biggest cause of cancer death worldwide [6]. In most cases, gastric cancer is preceded

for decades by persistent chronic active gastritis. This became clear in the 1960s and 1970s, when endoscopic biopsy sampling became possible and various excellent cohort studies on the dynamics of gastritis were performed, in particular in Scandinavia and the Baltic States. It appeared that the anatomy and function of the gastric mucosa normally remained unchanged throughout life, unless chronic active gastritis was present [7]. The most common cause of gastritis was unknown at that time, but later it appeared to be Helicobacter pylori colonization. Chronic H. pylori gastritis eventually leads in more than half of the affected subjects to gradual loss of glandular structures with its specialized cells and a collapse of the reticulin skeleton of the mucosa, a condition of atrophic gastritis [8]. As a result, the glandular layer of the mucosa becomes thinner, and glands are replaced by fibrosis and intestinal metaplasia. The major clinical importance of this condition is that it significantly increases the risk for the intestinal type of gastric cancer. This risk may be elevated up to 90 fold in subjects with severe atrophic gastritis throughout the complete stomach [9]. The annual incidence of gastric cancer among patients with atrophic gastritis varied in cohort studies between 0.3 and 1.0% [10]. This explains the interest in the diagnosis of atrophic gastritis. At present, there is a broad spectrum of questions related to the diagnosis of critical stages of gastric carcinogenesis: gastric epithelial atrophy, intestinal metaplasia and dysplasia. Therefore, it is extremely important to recognize among the dyspeptic patients those, who have very high risk of gastric malignant changes and who require dynamic surveillance, with the purpose of early diagnosis any preneoplastic changes in the stomach mucosa. Atrophic gastritis is a serious disease, which often does not receive much attention. The relationship between gastritis, atrophic gastritis and other diseases of the stomach is based on the fact that the infection and atrophy alter the physiological functions of the stomach and influence the growth and growth control of the epithelial cells in the stomach. These consequences vary, depending on whether the changes of the gastric mucosa caused by gastritis are located in the antrum or the corpus or both [11].

The most accurate diagnostic method of the gastrointestinal tract diseases is endoscopy with subsequent biopsy, which should be made in all patients with the presence of clinical symptoms. However, because of patchy character of atrophic changes in the stomach mucosa, some histological examinations could give false, negative results. Beside this, the biopsy is an expensive and labor-consuming method of research [12], so it can not be carried out in all patients in succession. In the contrary, due to invasiveness of biopsy, it is expedient to make only for monitoring of precancerous changes in the stomach mucosa. Therefore a screening method for the qualification of the patients group, recommended to biopsy is necessary. Such a method should be capable to reflect objectively the functional condition of the stomach mucosa, and hence, its morphological status.

It has been known for over two decades that the atrophic gastritis of the corpus and fundus of the stomach can be determined reliably by measuring the serum pepsinogen-I (PG-1) or the PG-1/PG-2 ratio from a blood sample [13-15]. However, it has not been possible to determine from a blood sample the types of atrophic gastritis in which the atrophic changes are located solely in the antrum. The GastroPanel® serum test also enables the determination of atrophic gastritis of this antrum-limited subtype.

Group I pepsinogens are synthesized solely in the oxyntic glands and mucous neck cells of the gastric body. On the other hand, pepsinogens of group II are uniformly synthetized in the glands of the entire stomach and to some extent also in the Brunner glands in the first part of the duodenum. The majority of the pepsinogens are secreted into the lumen of the stomach, where they are activated to the pepsin. A small proportion of the pepsinogens leaks into the blood circulation. In the case of atrophic corpus gastritis, the level of serum pepsinogen-1 decreases whereas the level of pepsinogen-2 remains stable or decreases slightly. The level of serum pepsinogen-1 or the ratio of serum pepsinogen-1 to pepsinogen-2 reflect with high reliability the number of cells and the number of oxyntic glands in the corpus area of the stomach, i.e. they reflect the degree of atrophy of the corpus mucosa. As the severity of the atrophic corpus gastritis corpus increases the level of serum pepsinogen-I or the PG-1/PG-2 ratio decreases [14-16].

Gastrin is synthesized in G-cells, which have been found in the gastric antrum. The most important stimulators of gastrin secretion into the circulation are: the vagal drive, the gastrin releasing peptide hormone (GRP; bombesin) and intraluminal factors, such as the stretching of the antrum or the protein-rich contents (diet) in the stomach. A pH-level below 2.5 lowers the secretion and release of gastrin from antral G-cell. The gastrin secreted in the antrum is over 90% of type G-17, whereas the gastrin secreted by the duodenum is primarily of type G-34. The fasting serum gastrin is primarily in the form of G-34, but the proportion of the type G-17 increases after the dietary stimulus. The secretion of gastrin-17 can be studied with a simple protein stimulation test. First, a blood sample is taken after fasting, after which the patient eats a protein-rich meal. The maximum increase in the serum level of gastrin-17 can be seen within 20 minutes. If the serum gastrin does not increase as a result of protein or other physiological stimulations it is an indication of the loss of gastrin secreting G-cells, i.e. an indication of the atrophy of the antrum mucosa. It is possible to make indirect conclusions regarding the antrum mucosa status by simultaneous assays of the serum gastrin and gastric acid output. In the cases with atrophic antral gastritis and loss of antral G-cells, serum gastrin remains low although the stomach is achlorhydric or hypochlorhydric [17].

Several research groups have renewed the interest in serology for atrophic gastritis by combining gastrin and pepsinogens with Helicobacter serology. Väänänen and colleagues present a smart algorithm for the differentiation in both antrum and corpus between atrophic and non-atrophic gastritis [18]. The algorithm was tested in a cross-sectional study correlating gastric mucosal histology with H. pylori IgG serum antibodies, serum PG-1 levels, and fasting and postprandial serum gastrin-17 levels. It appeared that in roughly 80% of the 404 cases tested, histology and serology matched a similar diagnosis. Sixty (15%) of the 404 subjects had atrophic gastritis, 6 (1%) had previously undergone antral resection, and 340 (84%) had a non-atrophic gastric mucosa either with or without inflammation. In this population with a rather low prevalence of atrophic gastritis, the negative predictive value of the serology

panel was 93-97% and the positive predictive value was 64-75%. For these calculations, the authors combined all subjects with atrophic gastritis of the antrum, the corpus, or both. The data, however, show that the serology panel performed much better in diagnosing atrophic gastritis in the corpus than in the antrum. In only 19 (50%) of the 38 patients diagnosed by serology as having antrum atrophic gastritis was this condition confirmed by histology.

This revival of interest in the serological testing of the condition of the gastric mucosa is of importance, given the fact that H. pylori eradication may cure gastritis and help to prevent further progression of gland loss. It is likely that this may also reduce the risk for gastric cancer [19], although many more data on this are needed. Screening and treatment of H. pylori infection may in theory be cost-effective for the prevention of gastric cancer [12].

Sipponen et al. [20] have recently shown, that the simultaneous detection of serum concentrations of PG-1 and G-17 and Hp-Ab titers is an effective method for non-invasive screening and diagnosis of atrophic gastritis using the blood samples of the patients.

In our present study, the use of the test-system GastroPanel® has allowed to receive statistically significant differences between the serum concentrations of PG-1 and G-17 depending on a degree of a stomach mucosal atrophy. We have obtained quite satisfactory meanings of the PPV and NPV of GastroPanel® test in the revealing the atrophic state of stomach mucosa. Moreover, this test was sufficiently sensitive and specific as it was proven by chromoendoscopy and histology.

Thus, our study has confirmed the usefulness of the test-system GastroPanel® as a "serologic biopsy" for authentic and non-invasive diagnosis of atrophic changes of a stomach mucosa in the patients with dyspepsia, associated with H. pylori infection. Beside this, we managed to show the advantage of a chromoendoscopy method prior to routine endoscopy in the diagnosis of intestinal metaplasia. Now is the time, almost a decade after the conclusion of the World Health Organization (WHO) that H. pylori is a class I carcinogen [21], to use this serology in further studies in selected and general populations. This will allow evaluation of the feasibility of screening and treatment for gastritis and prevention of gastric cancer, a cancer that is much more common than many other disorders for which screening and prevention have long been accepted in many populations.

Conclusions

The non-invasive detection of gastric mucosal atrophy by means of enzyme immunoassay with assessment of G-17 and PG-1 levels can be offered as the screening tool for gastric precancerous conditions. On the other hand, this method does not allow to diagnose the intestinal metaplasia and/or dysplasia in stomach mucosa. Therefore, the results of the serological screening indicating the stomach mucosal atrophy require carrying out the chromoendoscopy with subsequent mucosal biopsy, for revealing probable progression of atrophic process with the development of intestinal metaplasia, dysplasia or gastric cancer.

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