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ORIGINAL ARTICLE

Observational Study

Effectiveness of serological markers of gastric mucosal atrophy in the gastric precancer screening and in cancer prevention

Sergey M Kotelevets, Sergey A Chekh, Sergey Z Chukov

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Abstract

BACKGROUND

New markers are needed to improve the effectiveness of serological screening for atrophic gastritis.

AIM

To develop a cost-effective method for serological screening of atrophic gastritis with a high level of sensitivity.

METHODS

Of the 169 patients with atrophic gastritis, selected by the visual endoscopic Kimura-Takemoto method, 165 showed histological mucosal atrophy using the updated Kimura-Takemoto method. All 169 patients were examined for postprandial levels of gastrin-17 (G17) and pepsinogen-1 (PG1) using Gastro-Panel® (Biohit Plc, Helsinki, Finland).

RESULTS

We used the histological standard of five biopsies of the gastric mucosa, in accordance with the Kimura-Takemoto classification system to assess the sensitivity of G17 in detecting gastric mucosal atrophy. We also compared the morpho-functional relationships between the detected histological degree of gastric mucosal atrophy and the serological levels of G17 and PG1, as the markers of atrophic gastritis. The sensitivity of postprandial G17 was 62.2% for serological levels of G17 (range: 0-4 pmol/L) and 100% for serological G17 (range: 0-10 pmol/L) for the detection of monofocal severe atrophic gastritis. No strong

correlation was found between the levels of PG1 and degree of histological atrophy determined by the Kimura-Takemoto classification system to identify the severity of mucosal atrophy of the gastric corpus. In the presented clinical case of a 63-year-old man with multifocal atrophic gastritis, there is a pronounced positive long-term dynamics of the serological marker of atrophy - postprandial G17, after five months of rennet replacement therapy.

CONCLUSION

Serological screening of multifocal atrophic gastritis by assessment of postprandial G17 is a cost-effective method with high sensitivity. Postprandial G17 is an earlier marker of regression of atrophic gastritis than a morphological examination of a gastric biopsy in accordance with the Sydney system. Therefore, postprandial G17 is recommended for dynamic monitoring of atrophic gastritis after treatment.

Key Words: Updated Sydney system; Kimura-Takemoto classification; Prevention; Gastric cancer; Atrophic gastritis; Gastrin-17; Pepsonogen-1

Core Tip: A new assessment of serological markers of atrophic gastritis has been developed and confirmed with application of the updated Kimura-Takemoto morphological classification system. Serological markers of atrophic gastritis in this new interpretation of the classification system can more accurately detect atrophy of the gastric mucosa and complementarily increase the effectiveness of previous technologies for serological screening of precancerous changes in the stomach. Ultimately, this new model of serological screening is highly sensitive for detecting atrophic gastritis across mild, moderate and severe degrees of disease progression.

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INTRODUCTION

Atrophic gastritis is a basic precancerous disease of the stomach[1-3]. Many studies have been carried out to determine the clinical utility of detecting precancerous atrophic gastritis *via* measurement of gastric pepsinogens[4,5]. Serological markers have already demonstrated success in use for effective screening for atrophic gastritis and prevention of gastric cancer. In particular, gastrin-17 (G17) is used as a marker of atrophic gastritis in addition to gastric pepsinogens. Anti-*Helicobacter pylori* (*H. pylori*) antibody titer is another very important marker for serological screening for atrophic gastritis for predicting the risk of cancer[6,7]. Kotelevets and Chekh[8] have reported the successful use of the GastroPanel® test panel for mass serological prophylactic screenings. Zagari *et al*[9] concluded in their review of serological screening for atrophic gastritis that a cost-effectiveness analysis is needed.

Gastroendomicroscopy with narrow-band imaging technology (confocal laser endomicroscopy) aims to detect early gastric cancer and is considered as a secondary approach to prevention of gastric cancer[10-15]. Use of serological markers in cancer prevention of stomach surveillance programs may improve risk stratification for such screenings[16]. However, the relevance of primary prevention of gastric cancer using serological screening for atrophic gastritis was confirmed by Japanese authors, whose results clearly indicated that gastric cancer develops mainly by the cascade of gastritis-atrophy-metaplasia-cancer processes[17]. Scientific research, therefore, needs to be continued. The search for the best serological screening models for atrophic gastritis is paramount considering the extreme importance of primary prevention in gastric cancer management. To develop an optimal method of serological screening and determine the criteria for serological markers that support accurate assessment of atrophic gastritis. The method must also be cost-effective.

MATERIALS AND METHODS

Study subjects

This study was designed and carried out according to the updated Declaration of Helsinki, in a group of 169 dyspeptic patients (58 males, 111 females; mean age: 66.44 ± 10.22 years) with atrophic gastritis who were diagnosed by endoscopy and the Kimura-Takemoto visual endoscopic classification system. This work was performed in accordance with the Declaration of Helsinki (2000) of the World Medical Association. This study also received approval by the Ethical Committee of North Caucasian State Academy (Minutes No. 14/20 dated 29.06.2020). All patients provided informed written consent to the examination. Any patients who had been treated with proton pump inhibitors, H2 antagonists and

nonsteroidal anti-inflammatory drugs (commonly known as NSAIDs) at least one month before the study were excluded. Assessment of the type of mucosal atrophy, according to Kimura-Takemoto's grading (*i.e.*, visual endoscopic classification), was as follows: C-0, absence of atrophy; C-1, atrophy exclusively in the antrum; C-2, border of atrophy on the lesser curvature in the lower third of the gastric corpus; C-3, border of atrophy on the lesser curvature in the middle third of the gastric corpus; O-1, boundary between the lesser curvature and the anterior wall of the gastric corpus; O-2, border of atrophy within the anterior wall of the gastric corpus; and O-3, boundary between the posterior wall of the gastric corpus and large curvature. Biopsies were taken in addition to the visual assessment of atrophic gastritis and comprised six samples subsequently assessed by Kimura-Takemoto grading (C1, C2, C3, O1, O2, O3)[18,19].

Laboratory confirmation

Biopsy specimens were fixed in 10% formalin, embedded in paraffin, cut into sequential 5-μm sections, and stained with hematoxylin and eosin, PAS/alcian blue (pH = 2.5), and Giemsa stain. The grade of stomach mucosal atrophy was estimated from mild to severe, according to the Houston visual analogous scale[20]. In total, 169 persons underwent serological noninvasive screening for atrophic gastritis. The GastroPanel® (Biohit Plc, Helsinki, Finland) assay kit was used to identify parameters of postprandial G17 and pepsinogen-1 (PG1) in the fasting state. The levels of serological markers of mucosal atrophy (mild, moderate, and severe) were assessed according to levels of G17 in the antrum (mild, from 7 to 10 pmol/L; moderate, from 4 to 7 pmol/L; severe, from 0 to 4 pmol/L) and PG1 in the corpus (mild, from 15 to 25 μg/L; moderate, from 9 to 15 μg/L; severe, from 0 to 9 μg/L)[21].

Statistical analysis

In accordance with the purpose and objectives of the study, sensitivity indicators were used to perform comparative evaluations. For analyses of the various groups of mild, moderate and severe atrophy of atrophic gastritis, the percentage of each group among the total population was used. The threshold value of $P \leq 0.05$ was set as statistical significance. Given these criteria, differences were identified among patients groups. To assess the relationship between the groups, we applied the nonparametric Spearman correlation coefficient.

RESULTS

Correlation relationship between histological atrophy and PG1 levels

Stomach corpus mucosa biopsy specimens were subjected to histological analysis, with each designated as C2, C3, O1, O2, or O3. Of the 169 patients with atrophic gastritis, selected by the visual endoscopic Kimura-Takemoto method, 165 showed histological mucosal atrophy using the updated Kimura-Takemoto method. Four patients did not show histological evidence of atrophy in any biopsy specimen. One hundred and twenty-one patients had at least one of their five biopsy specimens show severe histological mucosal atrophy. The serological markers of gastric corpus mucosal atrophy have known limitations for assessment because the area of the corpus of the stomach is greater than the area of the antrum. The histological atrophy of the gastric corpus was evaluated in five biopsies (C2, C3, O1, O2, O3) of the Kimura-Takemoto classification and was considered as the reference standard for comparison with serological markers. The severity of histological atrophy in every biopsy was evaluated in four grades, namely no atrophy and mild, moderate or severe atrophy. The findings were compared with patient-correspondent PG1 serum levels. The correlation coefficient was determined between the severity of histological atrophy and the serum levels of PG1 (Table 1).

Comparative characteristics of serological markers of atrophy of PG1 and G17

No strong negative correlation has been observed between the severity of histological atrophy in biopsy specimens of the gastric corpus and serological marker PG1 level. This underlies a low eligibility of the serological marker PG1 in the assessment of the severity of gastric corpus atrophy. Thus, statistical comparison of the serological marker PG1 of atrophy in the gastric corpus using the morphological standard according to the Kimura-Takemoto classification (five biopsies from the gastric corpus) is much more correct than using the updated Sydney system (two biopsies from the gastric corpus). This can explain the low level of correlation of severity of mucosal atrophy between PG1 and the histological standard of five biopsies of the gastric corpus (C2, C3, O1, O2, O3 according to the Kimura-Takemoto classification). We, thereafter, evaluated the serological marker of atrophy, G17, to detect the severity of mucosal atrophy of the gastric corpus based on a comparison with the histological standard of five biopsies according to the Kimura-Takemoto classification. Table 2 presents the data on PG1 and G17.

PG1 detection yielded 84.3% false-negative results upon comparison with the Kimura-Takemoto histological standard. G17, in contrast, yielded only 6.6% false-negative results. In the previous part of the study, we had analyzed the comparison of serological markers with histological multifocal severe atrophic gastritis of the gastric corpus in five biopsy specimens (C2, C3, O1, O2, O3). Next, we analyzed comparisons of serological markers with histological monofocal severe atrophic gastritis of the gastric corpus. Results of that analysis are presented in Table 3.

Characteristics of the concept of monofocal atrophic gastritis

If severe histological mucosal atrophy was present in only one of five biopsy specimens (C2, C3, O1, O2, O3), then it was considered as monofocal atrophic gastritis of the gastric corpus. We have always rated such gastritis as severe corpus atrophic gastritis because it has a high risk of gastric cancer[22]. The postprandial G17 serological marker was used to detect monofocal severe atrophic gastritis of the gastric corpus. Analysis of these results is presented in Table 4. Receiver-

Table 1 Correlation coefficients between degree of histological atrophy and level of pepsinogen-1

Biopsy	Focus of a biopsy according to the Kimura-Takemoto classification	Spearman's rank correlation coefficient
C2	Border of atrophy on the lesser curvature in the lower third of the gastric corpus	-0.03 ($P > 0.05$)
C3	Border of atrophy on the lesser curvature in the middle third of the gastric corpus	-0.13 ($P > 0.05$)
O1	Boundary between the lesser curvature and the anterior wall of the gastric corpus	-0.26 ($P < 0.05$)
O2	Border of atrophy within the anterior wall of the gastric corpus	-0.44 ($P < 0.001$)
O3	Boundary between the posterior wall of the gastric corpus and large curvature	-0.46 ($P < 0.001$)

Table 2 Comparative characteristics of pepsinogen-1 and gastrin-17 markers of atrophic gastritis, *n* = 121

PG1 criteria	PG1 (0-9 μg/L): Severe	PG1 (9-15 μg/L): Moderate	PG1 (15-25 μg/L): Mild	PG1 (> 25 μg/L): False negative
Detected, *n* (%)	7 (5.8)	5 (4.1)	7 (5.8)	102 (84.3)
G17 criteria	**G17 (0-4 pmol/L): Severe**	**G17 (4-7 pmol/L): Moderate**	**G17 (7-10 pmol/L): Mild**	**G17 (> 10 pmol/L): False negative**
Detected, *n* (%)	88 (72.7)	15 (12.4)	10 (8.3)	8 (6.6)
Significance of differences	$P < 0.001$	$P > 0.05$	$P > 0.05$	$P < 0.001$

G17: Gastrin-17; PG1: Pepsinogen-1.

Table 3 Structure of monofocal severe atrophic gastritis of the gastric corpus, *n* = 37

Monofocus of severe atrophy	C2	C3	O1	O2	C3
Number of patients	20	7	5	3	2
Percent	54	19	13.5	8.1	5.4

Table 4 Use of the gastrin-17 marker for the detection of monofocal severe corpus atrophic gastritis

Serological criterion G17	Patients, *n*	Sensitivity, %	False negative, %
0-4 pmol/L	23	62.2	37.8
0-10 pmol/L	37	100	0

G17: Gastrin-17.

operating characteristic - analysis for multifocal atrophic gastritis is shown in Figure 1.

Optimal boundaries of the serological marker of atrophy postprandial G17

Postprandial G17 is the best marker of severe monofocal corpus atrophic gastritis, indicating such in the range of 0-10 pmol/L. It has a sensitivity of 100%, supported by the complete absence of false-negative results. When the range of 0-4 pmol/L was considered, however, the sensitivity dropped to 62.2%. We did not find a strong correlation between severe intestinal metaplasia and severe dysplasia using the Kimura-Takemoto histological classification (correlation coefficient = 0.53; $P > 0.05$) (Table 5).

Case presentation

History of present illness: A 63–year–old man in 2017 underwent serological screening by means of a laboratory test panel "Gastropanel" with a preventive purpose for early detection of precancerous pathology of the stomach. The patient had no complaints.

Personal and family history: The patient's father died of pancreatic cancer in 2014 at the age of 85. The patient's mother died of breast cancer in 2013 at the age of 85.

Results of serological screening in 2017: Testing for anti-*H. pylori* immunoglobulin G (IgG): Positive, G17 postprandial: 7.2 pmol/L, PG1: 90 mkg/L. The results for anti-*H. pylori* IgG: Positive and G17 postprandial: 7.2 pmol/L were considered as *H. pylori*-associated, antrum-atrophic gastritis mild according to serological criteria by Kotelevets and

Table 5 Morphological structure of intestinal metaplasia and dysplasia

IM specification	Patients, *n*	Intestinal metaplasia quota, %	
None	74	43.8	43.8
Mild	42	24.8	56.2
Moderate	35	20.7	
Severe	18	10.7	
Total	169	100	100
Dysplasia specification	**Patients, *n***	**Dysplasia quota, %**	
None	85	50.3	50.3
Mild	36	21.4	49.7
Moderate	42	24.8	
Severe	6	3.5	
Total	169	100	100

IM: Intestinal metaplasia.

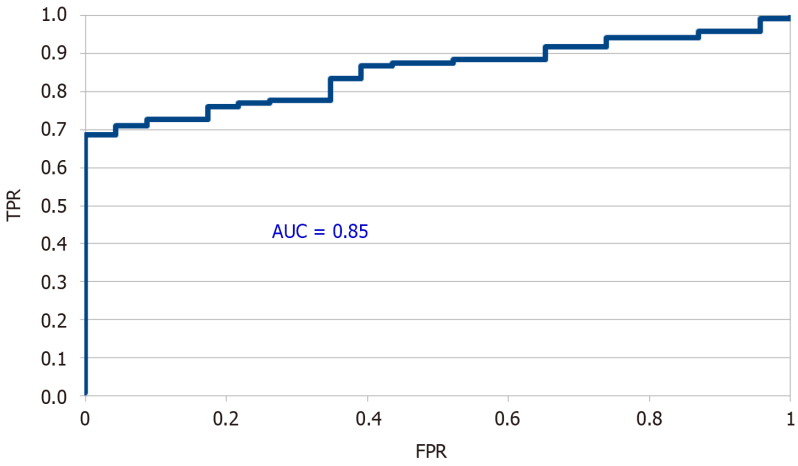

Figure 1 Receiver-operating characteristic - analysis of serological marker (gastrin-17 from 0 to 10 pmol/L) for severe multifocal atrophic gastritis. TPR: True positive rate; FPR: False positive rate; AUC: Area under the receiver-operating characteristic curve.

Chekh[23]. Additional studies were prescribed.

Physical examination: On physical examination, the vital signs were as follows: Body mass index, 22.5; body temperature, 36.3 °C; blood pressure, 120/78 mmHg; heart rate, 62 beats per min; respiratory rate, 12 breaths per min; the abdomen is soft and painless.

Laboratory examinations: Antibodies to gastric parietal cells: Negative, vitamin B12: 227 pg/mL.

Esophagogastroduodenoscopy: Endoscopic symptoms of diffuse atrophic gastritis. Biopsy and morphological examination in accordance with the Sydney system are presented in Tables 6 and 7.

Final diagnosis: *H. pylori*-associated, multifocal atrophic gastritis mild.

Treatment: 14 - daily quadruple therapy: (1) Omeprazole 20 mg twice a day; (2) Bismuth tripotassium dicitrate (de-nol) 240 mg twice a day; (3) Tetracycline 500 mg four times a day; (4) Metronidazole 500 mg twice a day; and (5) Probiotic. After the eradication treatment, the gastroprotection was appointed: De-nol 240 mg twice a day in two weeks; after de-nol sucralfate 1 g three times a day for a month; after sucralfate taking medication plantaglucid (Plantago major) 1 g three times a day for a 6 month.

Outcome and follow-up: A year after serological screening and diagnostic procedures, the following examinations were repeated: Esophagogastroduodenoscopy with biopsy in accordance with the Sydney System and histological examination

Table 6 Dynamics of multifocal atrophic gastritis of the patient

Observa-tion time	Patient age	Histological atrophy (Sydney system)					Gastrin-17 pmol/L postprand
		Biopsy 1: Antrum	Biopsy 2: Antrum	Biopsy 3: Incisura angul	Biopsy 4: Corpus	Biopsy 5: Corpus	
2017	63	Mild	Absent	Mild	Mild	Absent	7.2
2018	64	Mild	Absent	Mild	Mild	Absent	8.6
2019	65	Mild	Mild	Mild	Absent	Absent	7.4
2020	66	Mild	Absent	Absent	Absent	Mild	10.9
2021	67	Mild	Mild	Mild	Absent	Absent	17.5
2022	68	Mild	Mild	Mild	Mild	Absent	15.5
2023	69	-	-	-	-	-	14.6

Table 7 Dynamics of *Helicobacter pylori* status of the patient

Observation time	Patient age	Anti-*Helicobacter pylori* IgG	C13 urease breath test	Histological test
2017	63	Positive	-	Positive
2018	64	Negative	Negative	Negative
2019	65	Negative	Negative	Negative
2020	66	Negative	Negative	Negative
2021	67	Negative	Negative	Negative
2022	68	Negative	Negative	Negative
2023	69	Negative	Negative	-

IgG: Immunoglobulin G.

of biopsies with an assessment on a visual analog scale; G17 postprandial; three tests for *H. pylori* (anti-*H. pylori* IgG, C13 urease breath test, histological test).

Summary for 2018: As a result of anti-*H. pylori* therapy, effective eradication of *H. pylori* was obtained, which was confirmed by three tests (anti-*H. pylori* IgG, C13 urease breath test, histological test). The dynamics of multifocal atrophy of the gastric mucosa was absent after effective eradication and cytoprotective therapy. The results are presented in Tables 6 and 7.

After a dynamic examination in 2018, patient underwent gastrocytoprotective treatment with Rebagit (PRO.MED.CS Praha, a.s.) at a dose of 100 mg three times a day for four months. A year after the start of this treatment, a control annual examination was performed: Esophagogastroduodenoscopy with biopsy in accordance with the Sydney system and histological examination of biopsies with an assessment on a visual analog scale; G17 postprandial; three tests for *H. pylori* (anti-*H. pylori* IgG, C13 urease breath test, histological test). The results are presented in Tables 6 and 7.

Summary for 2019: The dynamics of multifocal atrophy of the gastric mucosa was absent after the four-month gastrocyto-protective therapy with Rebagit (PRO.MED.CS Praha, a.s.). After a dynamic examination in 2019, patient was started prolonged courses of atrophic gastritis replacement therapy with rennet received from baby calves and lambs. This therapy is similar to Abomin therapy, which the authors published in 2020[8]. The course of treatment continued for five months.

Summary for 2020-2023: Morphological dynamics of multifocal atrophy of the gastric mucosa was absent after five months of rennet replacement therapy. There is a pronounced positive long–term dynamics of the serological marker of atrophy – postprandial G17, after five months of rennet replacement therapy (Table 6). Normalization of the production of postprandial G17 indicates the restoration of the function of G-cells of the gastric antrum. Postprandial G17 is an earlier marker of regression of atrophic gastritis than morphological examination of gastric biopsy according to the Sydney system.

DISCUSSION

The correlation between the serological and histological criteria of mucosal atrophy in the antrum and in the gastric corpus has been studied using the updated Sydney system. The anatomical boundaries of serological markers of antral atrophic and corpus atrophic gastritis are established for mild, moderate, and severe atrophy. G17 is an established marker of antral atrophy, while PG1 is a marker of gastric corpus atrophy used during serological screening of gastric precancer[21,23]. Morphological-functional comparisons between histological grades determined by Kimura-Takemoto classification and the serological markers of G17 and PG1 have a fundamental difference. Accordingly, PG1 is not considered an accurate method for detecting corpus atrophic gastritis compared with the Kimura-Takemoto classification standard histological method.

The Kyoto consensus states that the detection of severity of mucosal atrophy of the stomach makes it possible to predict the development of gastric cancer[22]. Then, the question arises, how should we assess the severity of mucosal atrophy in multifocal atrophic gastritis? Many authors have characterized multifocal atrophic gastritis in accordance with the updated Sydney system[24-29]. At the same time, the updated Sydney system itself does not accurately characterize multifocal atrophic gastritis. It, unfortunately, cannot distinguish between mild, moderate and severe forms of multifocal atrophic gastritis and is only useful for the monofocal form of atrophic gastritis. Therefore, the term "multifocal atrophic gastritis" in accordance with the updated Sydney system cannot be used to predict the development of gastric cancer.

Sipponen *et al*[30] calculated the relative risk and cumulative risk of gastric cancer for different grades of atrophic gastritis of the antrum and corpus in accordance with the updated Sydney system; the calculations themselves were based on two biopsy samples from the corpus (other biopsies were from antrum). We are convinced that two biopsy specimens for the gastric corpus are not enough, since the gastric corpus is larger than the antrum. The histological Kimura-Takemoto classification more fully meets the conditions for stratification of the risk of developing gastric cancer in patients with atrophic gastritis. Moreover, for this morphological approach, five biopsy specimens should be taken from the gastric corpus. The antrum can also be subject to histological assessment according to Kimura-Takemoto classification.

The level postprandial G17 is an accurate marker of antral mucosal atrophy when using serological criteria: Mild atrophy is indicated by 7-10 pmol/L; moderate atrophy by 4-7 pmol/L; and severe atrophy by 0-4 pmol/L[21]. At the same time, a level from 0 to 10 pmol/L of postprandial G17 is a sensitive marker for the detection of severe mucosal atrophy in multifocal corpus atrophic gastritis. The results of our study allow us to recommend the use of postprandial G17 when performing serological screening of multifocal atrophic gastritis. This method of serological screening for atrophic gastritis is accurate, affordable, and low cost. The comparison of serological criteria for the postprandial G17 marker with histological criteria in accordance with the visual analogue scale of the updated Sydney system should always be correct.

If we compare severe atrophy of the antrum (level of postprandial G17 < 4 or 5 pmol/L) with histological mild, moderate and severe antral atrophy in total, then this result cannot be evaluated. This represents a methodological error, and sensitivity assessment will be very low (36.8%)[15,31,32]. Upon comparison of serological-histological antral atrophy three-quarters mild and mild, moderate and moderate, severe and severe, the sensitivity serological marker G17 increased to 64%, 59%, 96%, respectively[21]. The number of false-negative results when using such serological screening is minimal. This is very important for the detection of severe atrophy, which carries a high risk of developing gastric cancer. The high sensitivity of postprandial G17 (at 96%) for the detection of severe antral atrophy and the absolute sensitivity (at 100%) for the detection of mild, moderate and severe corpus-atrophic gastritis in total provides reason to use this marker for serological screening. The G17 marker has an advantage for identifying atrophic gastritis compared to PG1 and other atrophy markers (PG2, ratio between PG1 and 2)[33,34]. A large number of markers for identifying the risk of developing gastric cancer increases the financial costs of mass screening and makes it economically inaccessible. Some authors have suggested eliminating less effective serological markers from gastric cancer risk screening panels and adding effective prognostic markers[35-38]. The earliest precancerous changes in the stomach may indeed occur in early childhood[39]. Therefore, screening is necessary at a young age. Screening results obtained using serological markers must be confirmed by morphological methods to verify the diagnosis of atrophic gastritis[40-43]. Defining the correct marker criteria for assessing atrophic gastritis improves its sensitivity, specificity, and accuracy. The effectiveness of the marker increases significantly with a pronounced functional connection with the severity of mucosal atrophy, its localization in the stomach, and also in the absence of the influence of the inflammatory process of the gastric mucosa[44-46]. Postprandial G17 meets these requirements. We recommend that postprandial G17 be considered as the marker of choice for detecting atrophic gastritis. It is important to note that the developed fluorescent homogeneous assay (AlphaLISA) for G17 may be more suitable for large-scale screening of people at high risk of gastric cancer than traditional enzyme-linked immunosorbent assay[47]. Autoimmune gastritis, characterized with the presence of anti-parietal-cell antibodies, is an important risk factor for gastric cancer. Therefore, the use of a marker of autoimmune atrophic gastritis (anti-parietal-cell antibodies) is necessary for patients with a positive serological marker of atrophic gastritis[48]. Some authors suggest using gastroscopy to screen for atrophic gastritis, precancerous changes in the gastric mucosa, and gastric cancer. They recommend modern invasive, expensive methods for endoscopic screening: Confocal laser endomicroscopy, probe-based confocal laser endomicroscopy[49,50]. Mass population screening is not possible using invasive, expensive endoscopic technologies. These diagnostic methods are necessary at the second stage to verify the diagnosis for patients in whom a precancerous disease has been identified using serological markers of atrophic gastritis.

CONCLUSION

The new approach for serological screening of multifocal atrophic gastritis by assessment of serological levels of postprandial G17 is a cost-effective method with a high level of sensitivity. Postprandial G17 is an earlier marker of regression of atrophic gastritis than a morphological examination of a gastric biopsy in accordance with the Sydney system. Therefore, postprandial G17 is recommended for dynamic monitoring of atrophic gastritis after treatment.

FOOTNOTES

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