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Proton Pump Inhibitor Challenge to Confirm Diagnosis of Atrophic Gastritis of the Stomach: A Proposal

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ABSTRACT

Background & Aims: Chronic atrophic gastritis (CAG) is a known precancerous condition that can lead to the development of gastric cancer (GC). Low serum pepsinogen (PG) I levels have been proposed as a non-invasive marker for chronic atrophic gastritis of the stomach body, but the adequate upper cut-off for diagnosis remains controversial, as values ranging from 30 to 50 mcg/L are currently considered as a "grey zone". We aimed to identify patients with chronic atrophic gastritis (CAG) of the stomach body amongst subjects with PG-I levels ranging between 30 and 50 mcg/L by means of a proton pump inhibitor (PPI) challenge.
Methods: We selected 102 patients with baseline PG-I <60 mcg/L in whom upper gastrointestinal endoscopy with protocol biopsies staged according to OLGA system had been performed. Subsequently, all patients underwent a PPI challenge (consisting of PG-I testing after taking Esomeprazole 40 mg daily for 1 week). This population was divided into 5 groups according to PG-I levels: group A (PG-I< 30 mcg/L); group B (PG-I: 31-35 mcg/L); group C (PG-I: 36-40 mcg/L); group D (PG-I: 41-50 mcg/L); group E (PG-I: 51-60 mcg/L). By using the ROC curve, a cut-off of 30% increase from baseline PG-I was chosen.
Results: A statistically significant relationship between PG-I levels and OLGA staging was found, being 100% in the group of PG-I < 30mcg/L. Based on the value of the cut-off of 30% (calculated by ROC curve) corresponding to the delta increase between PG-I baseline value and after a one-week full dose of PPI, the positive predictive value was 95%, the negative predictive value 86%, the sensitivity 83% and the specificity 96%.
Conclusions: The use of the PPI challenge allows to identify subjects with CAG showing pepsinogen I values ranging between 30 and 50 mcg/L.

Key words: pepsinogen I – proton pump inhibitor –PPI test – atrophic gastritis – autoimmune gastritis.

Abbreviations: AIG: autoimmune gastritis; CAG: chronic atrophic gastritis; GC: gastric cancer; *H. pylori: Helicobacter pylori*; PG: pepsinogen; PPI: proton pump inhibitor; ROC: receiver operating characteristic.

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INTRODUCTION

Chronic atrophic gastritis (CAG) is a known precancerous condition that can lead to the development of gastric cancer (GC) [1-3]. *Helicobacter pylori* (*H. pylori*) infection is recognized as the most important etiological factor of chronic gastritis leading, over the years, to the development of CAG and ultimately GC [2-5]. Autoimmune gastritis is characterized by an immune-mediated process directed against parietal cells and intrinsic factor located in the upper area of gastric mucosa leading to CAG of the stomach body [6-31]. Patients with autoimmune thyroid disease are at greater risk for developing autoimmune gastritis (AIG) [9, 32].

In AIG, hypo- or achlorhydria, elevated levels of gastrin and low concentration of serum pepsinogen (PG) I are the result of damage of oxyntic glands and the ensuing atrophy of the gastric body mucosa [33-35]. Although many patients with AIG may be asymptomatic, some complain of dyspepsia, or even symptoms associated with gastroesophageal reflux [36-41].

The role of PGs as non-invasive markers for the diagnosis of atrophic gastritis, intestinal metaplasia and gastric cancer is supported by a large body of literature [42-108]. In particular, PG-I is produced by chief cells which are part of oxyntic glands [109]. Low serum PG-I levels have been proposed as non-invasive markers for severe chronic atrophic gastritis of the stomach body [9, 110] with a negative predictive value of 98%, but the diagnostic upper cut-off remains controversial

[111, 112]. Thus, since the normal range lies between 30 and 160 mcg/L, the range between 30 and 50 mcg/L could be considered a "grey zone." (Fig. 1).

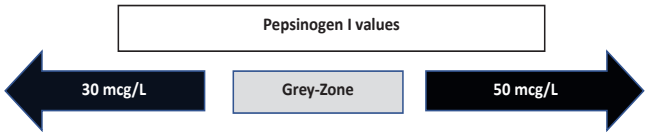

Fig. 1. Range of pepsinogen I values.

It is generally accepted that serum PG-I levels reflect gastric acid secretion [113, 114]. Moreover, it has been reported that serum PGs levels (I and II), which are predictive of the histological status of the gastric mucosa, rise during omeprazole and lansoprazole administration and rapidly return to baseline levels after discontinuing antisecretory treatment [115-119].

Previous studies show that serum PG-I is influenced by proton pump inhibitor (PPI) treatment to a greater extent than serum PG-II, and thus represents an adequate marker for monitoring the efficacy of antisecretory treatment and eventually compliance to pharmacological therapy, as well [117].

Based on the previous considerations, the execution of a non-invasive, serological test for the diagnosis of atrophic gastritis of the stomach body, as a tool to increase the appropriateness of gastroscopy, and to properly address histological sampling [2, 3, 121], represents a very interesting possibility.

In this study we aimed to identify patients with chronic atrophic gastritis (CAG) of the stomach body amongst subjects with PG-I levels ranging between 30 and 50 mcg/L (within the so-called "grey zone") by means of a PPI challenge.

METHODS

Consecutive dyspeptic patients referred from a primary care setting to our Tertiary Hospital from 01/01/2019 to 12/31/2020 were evaluated. All subjects who underwent an upper gastrointestinal endoscopy as out-patients at our Endoscopy Unit of Santorso, ULSS 7 Pedemontana, in Northeastern Italy, were considered for the study. Only patients who had been off PPI therapy for at least one month before enrollment in the study were considered. Patients with previous gastrointestinal neoplasm or chronic hepatic or renal diseases were excluded, as well as subjects with previous gastrointestinal surgery.

For each patient, we considered gender, age, indication for upper gastrointestinal endoscopy, current *H. pylori* status, previous *H. pylori* infection, and past medical history of autoimmune diseases. Information regarding the variables was recorded by the endoscopy nurse and/or the endoscopist using a reporting software (Clinical Sphere), which was also used to record other data including indication for the examination, endoscopic findings, vital parameters during the exam, final diagnosis, the amount and type of sedation used, and the presence or absence, as well as type and severity of adverse events. Internationally validated and standardized classifications and terminology were routinely used in our center's endoscopic reports. All procedures were performed using endoscopist- directed propofol-based sedation. Standard biopsies of antrum (2), incisura angularis (1), fundus and body (2) were routinely performed in all patients according to the Sydney protocol [122]. The histological evaluation of all biopsy samples was made using the OLGA classification system [123, 124].

All patients underwent two blood samples to determine PG-I levels (Biohit, Helsinki, Finland). The test was performed after overnight fasting and according to the manufacturer's instructions. The PG-I test was performed twice: at enrollment (baseline) and after one week of full-dose PPI therapy (esomeprazole 40 mg, 1 pill/day 30 minutes before breakfast). For each patient, serological results were compared against histological findings, according to both the Sydney system and OLGA staging.

Continuous variables were expressed as mean ± standard deviation (SD) for qualitative variables and as a percentage of the total for quantitative ones, using the t-test. Correlations between the degree of histologic features and serological tests were determined using Spearman's rank correlation coefficient. To determine the serum PG-I cut-off values, receiver operating characteristic (ROC) curves and Youden's index were used. Analyses of variance were used to compare the mean levels of the serological tests according to the presence of atrophic gastritis and *H. pylori* status. A p-value <0.05 was considered significant. All statistical analyses were performed with the SPSS statistical software program for Windows (version 20.1).

This study was performed following the Declaration of Helsinki; all patients gave their informed consent, and the study was approved by the local Ethics Committee (identifier: 92687).

RESULTS

During the study period, 1,458 consecutive dyspeptic patients (M:F=706:752, mean age: 49.3, range: 27-86 years) underwent upper gastrointestinal endoscopy as outpatients, and were considered for the study. According to selection criteria, the study population consisted of 102 patients (M:F=49:53; mean age: 56.7 years, range: 34-84) whose PG-I values were below 60 mcg/L. This population was divided into 5 groups as follows: group A (PG-I< 30 mcg/L), group B (PG-I: 31-35 mcg/L), group C (PG-I: 36-40 mcg/L), group D (PG-I: 41-50 mcg/L), and group E (PG-I: 51-60 mcg/L).

The distribution of 102 patients, according to PG-I levels was as follows: group A 17 patients, group B 31 patients, group C 24 patients, group D 17 patients; group E 13 patients. There were no statistically significant differences between gender or age with respect to PG-I values, all age groups and both genders being similarly distributed across PG-I groups (Table I).

After PPI intake, serum PG-I levels increased parallel to baseline PG-I values, with a greater increase observed in group E and D patients, with respect to patients in groups A, B, and C (Fig. 2). In general, lower PG-I levels were present in patients with more advanced OLGA grading; all patients with PG-I levels of 51-60 mcg/L had histologic examinations demonstrating no atrophy (as graded by OLGA 0) (Table II).

Table I. Epidemiological data (gender, mean age, range) according to the five PG-I groups, at baseline (PG-I T0)

	Number of Patients	Gender (M/F)	Mean Age (years)	Range (years)
PG-I< 30 mcg/L (Group A)	17	4/13	50.5 ± 10.9	35-75
PG-I 31-35 mcg/L (Group B)	31	16/15	55.7 ± 10.4	34-73
PG-I 36-40 mcg/L (Group C)	24	13/11	60.0 ± 10.2	37-84
PG-I 41-50 mcg/L (Group D)	17	10/7	58.5 ± 8.8	43-75
PG-I 51-60 mcg/L (Group E)	13	6/7	58.5 ± 11.2	42-74

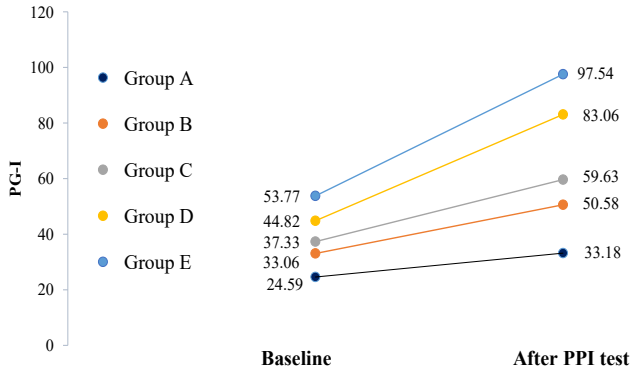

Fig. 2A. PG-I values after PPI-test in comparison to baseline levels.

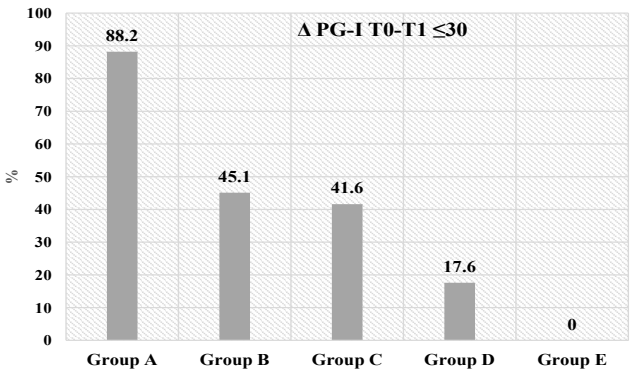

Fig. 2B. PG-I delta values (T0-T1) ≤ 30 in the different patient groups.

Table II. Distribution of histological findings (OLGA staging) for each of the 5 groups.

	Number of patients	OLGA 0	OLGA I	OLGA II	OLGA III	OLGA IV
Group A	17	0	1	6	6	4
Group B	31	12	0	7	11	1
Group C	24	14	0	5	5	0
Group D	17	14	0	1	2	0
Group E	13	13	0	0	0	0

Even more important, however, is the notion that, considering data of groups A and B together, 60% of the most severe atrophic gastritis diagnoses were recognized. The percentage rose to 90% when subjects with PG-I levels between 36 and 40 mcg/L were included. Of note, atrophic gastritis of the stomach body was absent in all group E patients.

Using the ROC curve to identify the best cut-off, with a delta increase of 30 mcg/L between baseline PG-I values and after a one-week full-dose of PPI (esomeprazole 40 mg: one tablet per day) an 88.2% of true positives for diagnosis of atrophic gastritis was obtained in the group with PG-I values ≤ 30 mcg/L (group A), 45.1% of true positive in the group B and 41.6% of true positives in the group C. Only 17.6% of true positives were identified in the group D (41-50 mcg/L). No cases of an increase of less than 30 mcg/L after a PPI challenge was found in the group E. Based on the value of the cut-off of 30% corresponding to the delta increase between PG-I baseline value and after a one-week full doze of PPI, the positive predictive value was 95.35%, the negative predictive value 86.67% (sensitivity 83.67%, specificity 96.30%) (Table III).

Table III. Statistical results of PPI challenge for diagnosis of body atrophic gastritis by using pepsinogen I levels (cut-off 30% of delta increase between baseline values and after a one-week full-dose of PPI)

Positive predictive value	Negative predictive value	Sensitivity	Specificity
95.35%	86.67%	83.67%	96.30%

DISCUSSION

The aim of this study was to establish the presence of atrophic gastritis of the stomach body at the individual level, based on gastric functional evaluation, which is the PG-I release after a PPI challenge. The basis of this lies in the fact that hydrochloric acid and pepsin are produced in the stomach body, and when atrophy is present in this region of the stomach, the compromise in function parallels the severity of gland damage.

While it has been documented that low PG-I levels correlate with a decrease in glandular integrity, the value of this parameter to establish the diagnosis of body gastritis remains uncertain, as levels fluctuate in a "grey zone" just above the cut-off lower than normal (30 mcg/L). The purpose of classifying studied subjects into 5 groups based on PG-I levels was to try to determine the optimal cut-off to confirm the diagnosis of body atrophic gastritis. For this reason, we investigated this possibility in small intervals of values beyond the cut-off lower than 30 mcg/L, from 31 to 35, from 36 to 40, from 41 to 50 and from 51 to 60 mcg/L.

The rationale to consider intervals higher than 40 mcg/L, theoretically not related to hyposecretion and, therefore to glandular atrophy, lies in the importance of the diagnosis of atrophic gastritis, a precancerous condition, with the aim of detecting as many patients as possible. The correlation between PG-I levels and the degree of severity of atrophic gastritis, according to OLGA staging, is statistically significant (p=0.0001) and shows an inverse correlation between more advanced OLGA staging and lowest PG-I values.

Through the comparison between serology and histology, only 10% of atrophic gastritis in the range between 41 and 60

mcg/L was found, with no cases when PG-I levels were ≥ 51 mcg/L. The single determination of PG-I value has an elevated positive predictive value, negative predictive value, sensitivity and specificity for diagnosing atrophy of the stomach body, as confirmed histologically. The fact that proton pump inhibitors increase PG-I levels has to be taken into consideration in order to maximize the capacity of PG-I testing to identify all cases of histologically confirmed atrophic gastritis of the stomach body. The explanation of this phenomenon lies in the fact that a reduction in total acid and pepsin secreting gland mass results in a lesser or absent response to the secretory stimulus induced by the PPI, since parietal and chief cells are located in the same structure.

These results seem to be promising, and if confirmed in other studies, suggest the validity of this simple, quick, non-invasive and economic test to select patients in whom gastroscopy will be performed with a biopsy sampling suitable to the diagnosis of body chronic atrophic gastritis, and possible investigation of the ECL endocrine component. This is based on the high specificity of the test for the diagnosis of chronic atrophic gastritis - already underlined by international consensus - and due to the very high percentage of the negative predictive value of PG-I, which when low, correlates with glandular atrophy. The proposal to use the PPI challenge for non-invasive diagnosis of body CAG, therefore, allows maximizing the ability of the PG-I to identify subjects with chronic atrophic gastritis, not only in the group of subjects with values lower than the cut-off of 30 mcg/L, but also in the "grey zone" between 31 and 40, where 43% of subjects are diagnosed with chronic atrophic gastritis.

One of the limits of the present study, however, lies in the fact that a validation cohort is lacking, and larger studies, with a control group that does not undergo PPI testing would enrich the present results and offer a more accurate view of the significance of observed changes. Moreover, although the PPI challenge is relatively non-invasive, compared to performing an endoscopic and histologic evaluation, it is noteworthy that a week of PPI treatment is required, which could be associated to post-PPI rebound phenomena, and can increase the medication burden.

Finally, the results of the study indicate that above the level of 40 mcg/L, fewer subjects with CAG are identified, but in subjects with PG-I ranging from 41 to 50, 17.6% were diagnosed with this condition. On the contrary, no subjects were found to have CAG with PG-I levels above 50 mcg/L. On the other hand, with the cut-off of 40 mcg/L, the PPI challenge allows the diagnosis of 93.87% (46 out 49 subjects) of body chronic atrophic gastritis, excluding 3 patients with a cut-off between 41-50 mcg/L.

CONCLUSIONS

Pepsinogen I values of <30 mgc/L diagnose 100% of subjects in whom histology will then confirm chronic atrophic gastritis of the stomach body, while in the presence of values ranging between 30 and 50 mcg/L (grey zone) the use of the PPI challenge can allow us to identify more subjects with CAG.

Conflicts of interest: F.D.M. declares a consulting agreement with Biohit (Helsinki, Finland). The other authors declare no conflict of interest.

Authors' contribution: F.D.M. conceived and designed the study, collected data, analysed and interpreted the results. L.F. and P.C. analysed and interpreted the data, searched the literature and wrote the manuscript. M.P.P., M.R. and G.B. collected and organized the data. M.F. and K.I.R.C., analysed the data and revised the manuscript. A.T., A.V. and A.F. criticaly revised the manuscript fro important intellectual content. F.D.M. supervised the study and the manuscript progression. All authors have read and approved the final version of the manuscript.

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