#200 - CHARACTERIZATION OF AUTOIMMUNE PROCESSES AND THE ROLE OF ANTI PARIETAL CELL ANTIBODIES IN GASTRIC PREMALIGNANT CONDITIONS AND GASTRIC CANCER

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Background. Chronic atrophic gastritis (CAG) and gastric intestinal metaplasia (IM) are considered gastric premalignant conditions. The main cause of CAG is chronic inflammatory process driven by *Helicobacter pylori* (Hp) infection. However, some autoimmune inflammatory process may be related to progression and extension of CAG/IM.

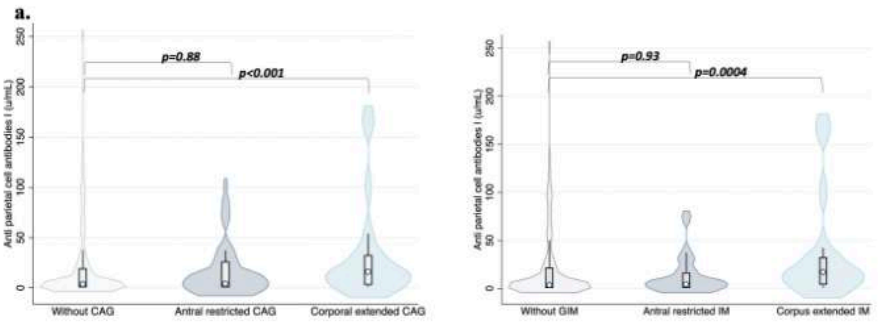
Aim. Toassess the autoimmunity process related to progression and extension of CAG/IM in patients with Hp infection.

Methods. A cross-sectional study of 220 patients attending to an esophagogastroduodenoscopy (EGD) was conducted. EGD was performed with mapping gastric biopsies collection and a blood sample was obtained. ELISA assay for anti-parietal cell antibodies (PCA) and anti-intrinsic factor antibodies (IFA) were performed. A case-control design was conducted comparing patients with normal or chronic superficial gastritis (CSG), CAG/IM and gastric cancer (GC). Titers of PCA/IFA were compared between study groups with Mann-Whitney test and logistic-regressions were performed adjusting by age and sex.

Results. 121 patients with normal histology or CSG, 74 with CAG/IM and 25 with CG were analyzed. Higher titers of PCA and IFA were observed in patients with corporal extended CAG ($p<0.001$) and IM ($p=0.0004$), but not in antral restricted CAG ($p=0.88$) and IM ($p=0.93$), in patients with *Hp* related gastritis. After excluding from the analysis patients with autoimmune gastritis, frequency of a positive PCA was higher in patients with corpus-extended CAG (58% vs. 26%; $p<0.001$), corpus-extended IM (58% vs. 26%; $p=0.013$) and OLGA III-IV patients (56% vs. 26%; $p=0.013$), compared to normal/CSG patients.

Conclusion. PCA antibodies exhibited a statistically significant association with the extension of CAG and intestinal metaplasia (IM) in non-autoimmune *Hp*-related gastritis. These results imply a potential involvement of immunity in the corpus extension of CAG and IM in the presence of *Hp* infection, further suggesting PCA antibodies' potential utility as a biomarker in this particular clinical context.

a.



b.

	PCA	p value	IFA	p value	PCA or IFA	p value
Chronic atrophic gastritis, n (%)	32 (25.6)	0.29	1 (1.9)	0.51	18 (35.3)	0.19
Without CAG	32 (25.6)	Ref.	1 (0.8)	Ref.	32 (25.6)	Ref.
Antral restricted	8 (28.6)	0.015	0 (0)	0.62	8 (28.6)	0.91
Corpus extended	26 (57.8)	<0.001	1 (4.4)	0.25	27 (60)	<0.001
OLGA staging, n (%)	32 (25.6)	Ref.	1 (0.8)	Ref.	32 (25.6)	Ref.
OLGA 0-I	25 (41.7)	0.035	2 (3.3)	0.211	25 (41.7)	0.035
OLGA II	9 (56.3)	0.013	1 (6.25)	0.087	10 (62.5)	0.003
OLGA III-IV						
Intestinal metaplasia, n (%)						
Without IM	34 (26.8)	Ref.	1 (0.8)	Ref.	34 (26.8)	Ref.
Antral restricted	7 (21.2)	0.52	1 (3)	0.30	7 (21.2)	0.52
Corpus extended	19 (57.6)	0.001	1 (3)	0.30	20 (60.1)	<0.001
Gastric adenocarcinoma, n (%)	6 (26.1)	0.86	0 (0)	0.61	6 (26.1)	0.86

APC: Anti-parietal cell antibodies; IFA: Anti-intrinsic factor antibodies; CAG: Chronic atrophic gastritis; OLGA: Operative Link for Gastritis Assessment; IM: Intestinal metaplasia.

Figure 1:

a. Anti-parietal cell antibodies titers (U/mL) according to chronic atrophic gastritis (CAG) and intestinal metaplasia (IM) extension after excluding patients with autoimmune gastritis and gastric cancer.

b. Frequency of positivity of anti-parietal cell antibodies (PCA) and anti-intrinsic factor antibodies (IFA) among patients with chronic atrophic gastritis (CAG), intestinal metaplasia (IM) and gastric cancer after excluding patients with autoimmune gastritis.