aim of the study was to evaluate the incidence of GCs development and to discover potential diagnostic markers related to GCs in patients (pts) with ACAG.

Methods: 141 pts with ACAG were enrolled between years 2006-2017 and endoscopy was performed. Pepsinogen I (PG1), Pepsinogen II (PG2) and Gastrin 17 (G17) serum levels were quantified using an enzyme-linked immune-sorbent assay kit. Serum levels of PGs and G17 were used to discriminate among pts with ACAG and pts affected by GCs in univariate and in multivariate analysis. A panel of genetic polymorphisms of PG2 gene and miRNA, that are known to modulate PG2 expression (rs9471643 C/G; rs6458238 A/G; rs8111742 A/G; rs121224 C/G; rs1002765 A/G; TATA-BOX length), was tested by real time PCR.

Results: Out of the 141 ACAG pts (26 M, 115 F; mean age 54,5), 21 (15%) (4M, 17F) presented GCs. A secondary autoimmune disorder was displayed by 98 pts (69,5%) and autoimmune thyroiditis was the most frequent (61,9%). A statistically significant difference in PG1/PG2 and G17 levels was found between ACAG pts with or without GCs (r=-0,3768 95% CI-0,5499 to -0,1726 p = 0,0005). Although it is known that PG2 levels correlate with Helicobacter Pylori (HP) infection in our series of ACAG and GCs and ACAG pts there wasn't a statistical significant difference nor in number of HP positive (+) pts nor in IgG anti HP load (HP+ GCs pts 17,6%, HP+ ACAG pts 30,2%; GCs pts IgG anti HP mean 19,42 SD: ±27,71, ACAG pts IgG anti HP mean 33,43 SD: ±41,43 p=ns). Among the 6 genetic polymorphisms, we found that rs8111742 A/G, rs121224 C/G were associated to a difference in serum PG2 levels and GCs (p = 0,0016 and p = 0,0051). No significant differences were found between pts with thyroiditis and GCs and pts without thyroiditis and with GCs (6,3% and 8,5% p = 0,07).

Conclusion: GCs are often diagnosed incidentally during endoscopy. We found a higher association between GCs type I and ACAG than data present in literature and of interest we found a statistically significant difference in PG1/PG2 and G17 levels between ACAG pts with or without GCs. The identification of a different level of PGs ratio and G17 in GCs-positive ACAG could be proposed as a potential indicative marker for a further endoscopic targeted evaluation for GCs in ACAG pts.

P – 088 Genetic polymorphisms and PG1/PG2 and G17 levels can predict gastric carcinoids in autoimmune atrophic chronic gastritis patients

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Introduction: Autoimmune Chronic Atrophic Gastritis (ACAG) is epidemiologically and biologically linked to the development of gastric carcinoids type I (GCs) and gastric adenocarcinoma. ACAG is often associated to multiple autoimmune disorders. The