DEAR COLLEAGUES,

We cordially invite you to the 9th Central European Gastroenterology Meeting (CEURGEM) which will take place in Budapest, Hungary on December 1-3, 2016.

The scientific programme will include many challenging and changing aspects regarding new endoscopic approaches, screening and surveillance of GI cancers, new biological treatment modalities in IBD, new mechanisms in functional GI disorders, gut microbiota, management of diverticular disease, treatment of Hepatitis C.

To bring you the latest findings and global perspectives a concomitant EAGEN Course will also take place with internationally renowned faculties.

The Congress and the Course is organised under the auspices of CEURGEM, EAGEN (European Society for Gastroenterology, Endoscopy and Nutrition) and the Hungarian Society of Gastroenterology.

We expect many participants from Central and Western European countries, thus we hope that this Meeting will represent a great opportunity for regional and European exchange of new scientific ideas in the field of gastroenterology.

It is a great honour to invite you to this exciting Meeting.
We hope to see you at CEURGEM-2016 in Budapest, Hungary!

László Herszényi
Meeting Chair

SCIENTIFIC ORGANISER

László Herszényi
Budapest, Hungary
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Budapest Marriott Hotel
Apáczai Csere János u. 4., H-1052 Budapest, Hungary

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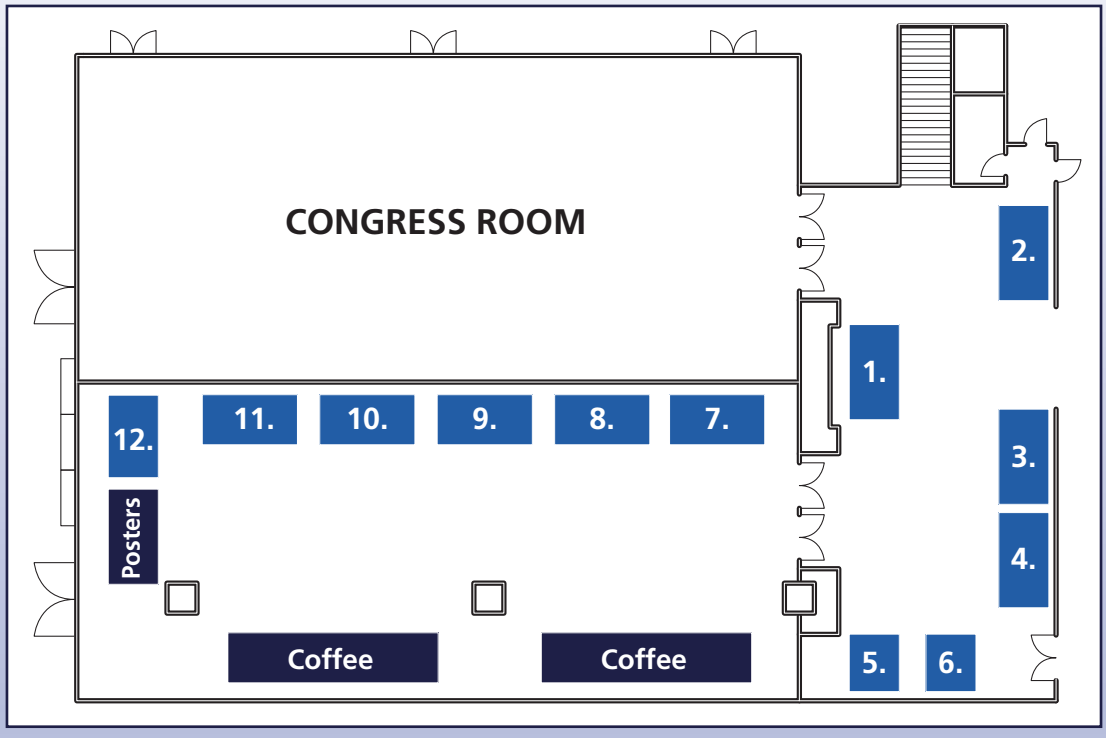

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PROGRAM

08:45–09:00	**Opening and Introduction**
	László Herszényi *Guenter J. Krejs* *Zsolt Tulassay*

09:00–11:45	**ESOPHAGUS-STOMACH**
	Chairs: *G. Krejs, Z. Tulassay, L. Herszényi*
09:00–09:15	Treatment of Upper GI bleeding – an update *István Altorjay* (Hungary)
09:15–09:30	The real impact of screening and surveillance for Barrett's esophagus *Ivan Jovanović* (Serbia)
09:30–09:45	Gastric precancerous lesions: endoscopic or serological approach? *Francesco Di Mario* (Italy)
09:45–10:00	Screening for Gastric cancer: is it justified in Europe? *Marcis Leja* (Latvia)
10:00–10:15	Discussion
10:15–10:30	**Coffee Break**
	Chairs: *F. Di Mario F. M. Leja, I. Altorjay*
10:30–10:45	Possibilities for primary and secondary gastric cancer prevention in Central Europe *Bojan Tepeš* (Slovenia)
10:45–11:00	Intestinal metaplasia of the cardia: endoscopic and clinical implications *Srdjan Djuranovic* (Serbia)
11:00–11:15	Submucosal endoscopy: from ESD to POEM and beyond *Jan Martinek* (Czech Republic)
11:15–11:30	Endoluminal Bariatric Therapy: a new paradigm of therapy for obesity? *László Bene* (Hungary)
11:30–11:45	Discussion
11:45–13:00	**Lunch**

13:00–14:30	GUT-SMALL BOWEL
	Chairs: *T. Milosavljevic, L. Kupcinskas, T. Wittmann*
13:00–13:15	Basic aspects of GI mucosal immune system *Marco Banic* (Croatia)
13:15–13:30	Mechanisms and treatment of refractory celiac disease *Márk Juhász* (Hungary)
13:30–13:45	New aspects of IBS *Dan Dumitrascu* (Romania)
13:45–14:00	Intestinal microbiota, low FODMAP diet and FGIDs: any clue? *Pietro Portincasa* (Italy)
14:00–14:15	Gut microbiota, obesity and cancer – an evidence based approach *Tomica Milosavljevic* (Serbia)
14:15–14:30	Discussion
14:30–14:45	**Coffee Break**

14:45–16:30	COLON
	Chairs: *J. Regula, H. Hammer, I. Rácz*
14:45–15:00	Diminutive colorectal polyps: are gastroenterologists willing to implement the DISCARD strategy? *István Rácz* (Hungary)
15:00–15:15	Learning from the best: how to improve the yield of screening colonoscopy? *Heinz Hammer* (Austria)
15:15–15:30	Lessons from Polish colorectal cancer screening programme *Jaroslaw Regula* (Poland)
15:30–15:45	Management of diverticular disease – current therapeutic challenges *András Taller* (Hungary)
15:45–16:00	Rectal cancer: standard surgery and new approaches *Gábor István* (Hungary)
16:00–16:15	Fecal transplantation: the beginning of the end or the end of the beginning? *Áron Vincze* (Hungary)
16:15–16:30	Discussion
17:00	**Opening Dinner** Marriott Hotel Corso Room

08:30–11:00	LIVER
	Chairs: *M. Trauner, M. Acalovschi, F. Szalay*
08:30–08:45	Hepatitis C infection: availability of new treatment modalities in Hungary and Central-Europe *Béla Hunyady* (Hungary)
08:45–09:00	Autoimmune and cholestatic liver diseases, or…NAFLD, or more specifically bile acid signaling in treatment of liver diseases and beyond *Michael Trauner* (Austria)
09:00–09:15	Cholesterol gallstones: focusing on the association with non-alcoholic fatty liver disease *Monica Acalovschi* (Romania)
09:15–09:30	Discussion
09:30–09:45	**Coffee Break**
	Chairs: *F. Farinati, I. Sporea, Z. Máthé*
09:45–10:00	Feasibility of HCC screening *Ioan Sporea* (Romania)
10:00–10:15	How is changing HCC treatment: from liver transplantation to targeted therapies *Fabio Farinati* (Italy)
10:15–10:30	New trends in liver transplantation *Zoltán Máthé* (Hungary)
10:30–10:45	Discussion
10:45–11:00	**Coffee Break**

11:00–12:30	IBD
	Chairs: *M. Banic, M. Diculescu, T. Molnár*
11:00–11:15	New and future agents on the horizon for IBD therapy *(Ferring sponsored lecture)* *Pál Miheller* (Hungary)
11:15–11:30	Availability of new biological treatment modalities in Romania *Mircea Diculescu* (Romania)
11:30–11:45	When to stop anti-TNF-α therapy in patients with IBD? *Tibor Hlavaty* (Slovakia)
11:45–12:00	Anti-TNF-refractory IBD – practical insights *Tamás Molnár* (Hungary)

12:00–12:15	Switching Between Infliximab Originator and Biosimilar in Patients with Inflammatory Bowel Disease. Preliminary Chech Observations *Milan Lukas* (Chech Republik)
12:15–12:30	Discussion
12:30–13:30	**Lunch**

13:30–16:00	**PANCREAS**
	Chairs: *P. Dité, A. Dabrowski, Á. Vincze*
13:30–13:45	Autoimmune pancreatitis – new aspects *Petr Dité* (Czech Republic)
13:45–14:00	Prevention strategies for post-ERCP pancreatitis *István Hritz* (Hungary)
14:00–14:15	Endoscopic ultrasound-guided pancreato-biliary interventions: an update on recent developments *Richárd Szmola* (Hungary)
14:15–14:30	Discussion
14:30–14:45	**Coffee Break**
	Chairs: *D. Stimac, J. Spicak, L. Bene*
14:45–15:00	Screening for pancreatic cancer – in whom and when? *Julius Spicak* (Czech Republic)
15:00–15:15	Better prognosis of pancreatic cancer: fact or fiction? *Andrzej Dabrowski* (Poland)
15:15–15:30	Palliative endoscopy in pancreatic cancer *Tibor Gyökeres* (Hungary)
15:30–14:45	Pancreatic cancer – new management strategies *Davor Stimac* (Croatia)
15:45–16:00	Discussion

16:00–16:15	**Summary and Concluding remarks**
	László Herszényi (Hungary) *Guenter J. Krejs* (Austria)

18:00	**Banquet Dinner** Lázár Equestrian Park, Domonyvölgy Departure by bus from Marriott Hotel at 17:00

Key issues and top 5 papers in the field of

- Esophagus
- Stomach
- Small bowel
- IBD
- Liver
- Colon
- Pancreas
- GI Endoscopy- New development in imaging
- Gastrointestinal bleeding
- GI Oncology
- Extraintestinal manifestations of GI disorders-Paraneoplastic syndromes
- Nutrition – Management of weight loss, loss of appetite, parenteral nutrition

08:25–08:30	**Opening, Introduction**
	Peter Malfertheiner (Germany)
08:30–09:00	**LIVER**
	Chairs: *M. Trauner, B. Hunyady*
08:30–08:50	Speaker: *Fabio Farinati* (Italy)
08:50–09:00	Discussion
09:00–09:30	**ESOPHAGUS**
	Chairs: *P. Malfertheiner, Z. Tulassay*
09:00–09:20	Speaker: *László Herszényi* (Hungary)
09:20–09:30	Discussion
09:30–10:00	**STOMACH**
	Chairs: *F. Di Mario, I. Altorjay*
09:30–09:50	Speaker: *Peter Malfertheiner* (Germany)
09:50–10:00	Discussion
10:00–10:30	**SMALL BOWEL**
	Chairs: *P. Portincasa, D. Dumitrascu*
10:00–10:20	Speaker: *Heinz Hammer* (Austria)
10:20–10:30	Discussion
10:30–11:00	**Coffee Break**
11:00–11:30	**NUTRITION – MANAGEMENT OF WEIGHT LOSS, PARENTERAL NUTRITION**
	Chairs: *T. Milosavljevic, L. Herszényi*
11:00–11:20	Speaker: *Heinz Hammer* (Austria)
11:20–11:30	Discussion

11:30–12:00	**IBD**
	Chairs: *M. Diculescu, T. Molnár*
11:30–11:50	Speaker: *Davor Stimac* (Croatia)
11:50–12:00	Discussion

12:00–12:30	**COLON**
	Chairs: *B. Tepes, D. Dumitrascu*
12:00–12:20	Speaker: *Jaroslaw Regula* (Poland)
12:20–12:30	Discussion
12:30–13:30	**Lunch**

13:30–14:00	**PANCREAS**
	Chairs: *A. Dabrowski, I. Hritz*
13:30–13:50	Speaker: *Petr Dité* (Czech Republic)
13:50–14:00	Discussion

14:00–14:30	**GI ENDOSCOPY – NEW DEVELOPMENT IN IMAGING**
	Chairs: *D. Stimac, I. Rácz*
14:00–14:50	Speaker: *Jochen Weigt* (Germany)
14:50–15:00	Discussion

14:30–15:00	**GI BLEEDING**
	Chairs: *J. Martinek, Á. Vincze*
14:30–14:50	Speaker: *István Rácz* (Hungary)
14:50–15:00	Discussion

15:00–15:30	**GI ONCOLOGY**
	Chairs: *J. Regula, J. Weigt*
15:00–15:20	Speaker: *Tomica Milosavljevic* (Serbia)
15:20–15:30	Discussion

15:30–16:00	**EXTRAINTESTINAL MANIFESTATIONS OF GI DISORDERS-PARANEOPLASTIC SYNDROMES**
	Chairs: *P. Dité, M. Banic*
15:30–15:50	Speaker: *Dan Dumitrascu* (Romania)
15:50–16:00	Discussion

16:00	**Closing, concluding remarks**
	Peter Malfertheiner (Germany)

ABSTRACTS

Dual regimens for Helicobacter pylori eradication in the 3rd millenium: systematic review and meta-analysis

György Miklós Buzás[1], Jolán Józan[2]
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[2]Semmelweis University, Department of Physiology, Budapest, Hungary

Introduction: Dual regimens for the eradication of Helicobacter pylori were proposed in the 1980's but were hindered with the advent of triple therapies in the Maastricht era.

Aim. To systematically review and meta-analyse the efficacy of improved dual regimens, introduced in the third millenium.

Materials and Methods: Studies published between 2000 and March 2016 were identified in MEDLINE. The pooled eradication rates obtained by dual and comparator therapies was calculated. Heterogeneity was assessed by I2 test.The results of comparative controlled trials were estimated by the random effect model, the effect of high dose proton pump inhibitor/antibiotics and that of CYP450 polymorphism on eradication and the geographic differences were also assessed.

Results: The pooled eradication rate of PPI-based dual therapies on an ITT basis was 70.5% (95% confidence interval 63.8-77.3%). Ranitidine-bismuth citrate regimens achieved 74.7 (65.3-84.8) (p=0.21). Per-protocol eradication rates were of 73.8 (63.6-83.7) and 83.8 (76.0-91.7), respectively (p=0.04). In comparative trials, with an I2 value of 62%, the cumulated odds ratio was of 0.64 (0.26-1.60), favouring PPI-based triple therapies. High-dose second generation vs standard dose proton pump inhibitor and amoxicillin increased the rate of eradication from 70.4% (62.6-78.4%) to 80.8% (73.6-88-1%) (p=0.0001). Second line dual regimens achieved eradication in 87.6% (77.3-97.8%) and third-line treatment in 75.3% (63.5-87.1%), both being superior to first line schedules (65.88%, 58.0-73.7%) (p= 0.05 and 0.0001, respectively). Studies performed in Asia were superior to those coming from Europe or the Americas (74.8%, vs 57.7% and 66.9%, p=0.05 and 0.07, respectively) CYPC19B slow metabolizers achieved rates of 82.1% (75.8-88.7%), while homozigotes 62.5% (45.4-77.5%) (p=0.009) and heterozigotes 73.1% (61.2-85.1%) (p=0.07).

Conclusions: Standard- and high dose proton pump inhibitor + high dose amoxicillin based therapies obtained suboptimal results both as primary and rescue treatment. Paradoxically, they were more efficient as secondary or third-line treatments. High PPI/amoxicillin dose dual therapies could not be recommended as yet, however, in individual cases they could be efficient: determination of CYPC19B status could be useful in selecting slow metabolizers, in order to obtain higher rates of eradication.

Pressure Management in Irritable Bowel Syndrome

Popa Stefan-Lucian
„Iuliu Hatieganu" University of Medicine and Pharmacology, Cluj-Napoca, Romania

Introduction: Irritable bowel syndrome (IBS) is a functional disorder which affects up to 20% of the population and is the result of interaction between genetic and environmental factors. The aim of this study is to look for the possible correlation between IBS and Pressure Management (PM), assessed by a specific questionnaire: Pressure Managment Indicator (PMI).

Materials and Methods: A total of 39 patients with IBS, according to the Rome III criteria and 37 gender and age-matched healthy controls were investigated using a self-administered questionnaire: PMI. Patients were classified into groups of IBS with diarrhea (IBS-D): 22, IBS with constipation (IBS-C): 14 and IBS with mixed symptoms (IBS-M): 3.

Results: Significant correlation between IBS and PM evidenced by organizational satisfaction, mental wellbeing, physical wellbeing, sources of pressure, type A behavior, coping was found ($p<0.001$), but there was no correlation between occupational classification, workout program, norm, health status, major disease, negative pressure in the last 3 months, smoking, alcohol consumption, work hours, number of years in the organization and IBS.

Discussion: This is the first assessment of PM in IBS, by a validated specific questionnaire. PMI scores are higher in IBS than in controls, emphasizing the role of professional stress in this condition.

Autoimmune Pancreatitis: Diagnostic and Surgical Dilemmas

Guo Yutong[1], Janina Kulka[2], Attila Szijarto[1], Ibolyka Dudas[3], Laszlo Nehez[1], Tibor Tihanyi[1], Istvan Pulay[1], Akos Szucs[1], Peter Kupcsulik[1]
[1]1st Department of Surgery, Semmelweis University, Budapest, Hungary
[2]2nd Department of Pathology, Semmelweis University, Budapest, Hungary
[3]Department of Radiology, Semmelweis University, Budapest, Hungary

Introduction: The awareness of autoimmune pancreatitis (AIP) has grown recently, yet practical strategy remains indeterminate. Localized AIP can mimic bilio-pancreatic neoplasia in both radiologic and clinical manifestations however.

Materials and Methods: 14 AIP cases from 2002 to 2016 were reviewed with special respect to diagnosis and management. CT and fine needle aspiration biopsy (FNAB) represented basic diagnostic methods in all of the cases. Two FNABs proved AIP, while in the remaining 12 cases FNABs were ambiguous and diagnoses were inconclusive. Pancreatic cancer (PC) was suspected in these cases, therefore exploratory laparotomy was performed.

Results: Out of 12 patients underwent surgery, 7 had intraoperative FNAB cytology, and the results suggested them having AIP. Choledochoduodenal anastomosis continued to be carried out without resection of pancreatic mass. Pancreatic resection happened in 5 patients based on intraoperative picture of pancreatic mass. No intraoperative cytology was performed in these cases. Posteriorly the recent resections might be regarded as unnecessary. The overall rate of unnecessary pancreatic resection is 35.7%.

Discussion: Current diagnostic criteria has been proposed to identify AIP according to one or more combinations of the following: radiological imaging, serology test for antibodies, extra pancreatic organ involvement, histopathology and response to steroid treatment. Nevertheless, atypical AIP still share many common findings with PC. IgG4 is unreliable in diagnosis since > 20% of AIP patients have normal IgG4 level and up to 10% of PC patients present increased IgG4 level. Many localized AIP are unable to be distinguished with PC on CT or MRI image. In addition, active AIP shows increased isotope uptake on PET/CT which is similar to neoplasia. Certain PC respond well to corticosteroid which makes this diagnostic method inaccurate as well.

AIP is considered as a rare disease. To avoid unnecessary pancreatic resection in merely suspected cases seems to be mandatory. Fast and accurate preoperative differential diagnosis between malignancy and AIP has become a great challenge to achieve. We suggest that the importance of intraoperative histopathology should be highlighted, especially in patients with one or more positive findings under the current AIP diagnostic criteria.

Prevalence and pathophysiology of gastro-esophageal reflux disease in patients with autoimmune gastritis

Valentina Pilotto[1], Gemma Maddalo[1], Edoardo Vincenzo Savarino[1], Costanza Orlando[1], atteo Fassan[2], Massimo Rugge[2], Daniela Basso[2], Fabio Farinati[1]
[1]Department of Surgery, Oncology and Gastroenterology, Padua University, Padua, Italy
[2]Department of Medicine, Padua University, Padua, Italy

Introduction: Autoimmune gastritis (AIG) is characterized by corpus-predominant atrophy with consequent hypo-achlorhydria. In AIG patients dyspepsia is frequent but acid reflux symptoms not uncommon, with few data available regarding gastroesophageal reflux disease (GERD) in AIG. Our study was aimed to define the prevalence of reflux symptoms in AIG patients, to evaluate the serological, histological and clinical differences in AIG patients with or without reflux symptoms and to investigate the physiopathology behind these symptoms.

Materials and Methods: One hundred and fifty AIG outpatients were evaluated and 87 were included in the study: 29 AIG patients with reflux symptoms (AIG-R) and 58 without (controls), selected with similar age and gender distribution. AIG-R underwent a pH-impedance (pH/I) and high resolution manometry (HRM). Serum biomarkers, EGDS, histology and anamnestic data were evaluated in both groups. Statistics was performed as indicated.

Results: AIG-R were 19% overall and 28% of them showed endoscopic esophageal lesions, with more frequent hiatal hernia than in controls ($p<0.02$). pH/I diagnosed acid reflux, esophageal hypersensitivity and a normal pattern in 7%, 28% and 66% patients, respectively. The number of non-acid reflux (NAR) was higher when compared with acid ones ($p<0,0001$), moreover NAR and NAR proximal extension were associated with endoscopic lesions ($p<0,03$ and $p<0,05$, respectively). HRM revealed normal pattern in 62% of patients, minor peristalsis disorders in 24%, and outflow obstruction in 14%. According to the new Rome IV criteria, 55% of patients presented "functional esophageal disorders" (Rome IV-IN). No differences were detected in serological marker and clinical presentation. AIG-R presented lower antrum gastritis ($p<0,006$) and a trend toward lower atrophy stage ($p=0,07$) when compared with controls. The two patient with acid GERD were an OLGA 0 with mild gastrin increase and an OLGA I with short segment Barrett's esophagus. Lower OLGA stages, lower corpus atrophy ($p<0,02$) and more frequent response to PPI ($p<0.05$) were associated with Roma IV-OUT status.

Discussion: AIG-R patients are not uncommon despite the hypo-achlorhydria. Acid reflux is rare in AIG, while motility and "functional" disorders are frequent. Lower corpus atrophy and OLGA stage in Rome IV-OUT patients, with an at least partly preserved secretion, is likely relevant in the pathogenesis of symptoms. Treatment should consider use of proton pump inhibitor drugs only in specific patients.

Autoimmune gastritis with previous o concurrent H. Pylori infection presents distinct functional and morphological features

Gemma Maddalo[1], Valentina Pilotto[1], Costanza Orlando[1], Matteo Fassan[2], Marco Pizzi[2], Massimo Rugge[2], Daniela Basso[2], Fabio Farinati[1]
[1]Department of Surgery, Oncology and Gastroenterology, Padua University, Padua, Italy
[2]Department of Medicine, Padua University, Padua, Italy

Introduction: Autoimmune gastritis (AIG) results in hypo/achlorhydria due to parietal cells destruction. It is characterized by lower levels of serum pespinogen (Pg) I and PgI/PgII ratio and increased levels of gastrin. Some Authors support an association between AIG and Helicobacter Pylori (HP) infection. The aim of our study was to assess epidemiologic, serologic and pathologic features of AIG patients with and without previous HP infection.

Materials and Methods: Two hundred and eleven consecutive patients with AIG, undergoing endoscopy were included. Serum gastrin, PgI, PgII and Cromogranin A levels were determined in all patients. Multiple gastric biopsies were obtained for histology, OLGA staging and HP detection. Previous or current HP infection (HP+) was confirmed in patients by anamnestic and/or pathologic and/or serologic data. Statistics was performed using non parametric tests.

Results: Present or previous HP infection was confirmed in 50/211 patients while 161 were negative (HP-). When we compared HP+ vs HP- AIG, no differences were found for age and gender distribution, antral and fundic/body atrophy, OLGA staging, PgI, PgII, PgI/PgII ratio and Cromogranin A levels. Gastrin levels and Gastrin/PgI ratio, a global marker of gastric damage we previously identified, were higher in HP- vs HP+ ($p<0.02$). Interestingly, 15% HP- presented antrum atrophy. Severity of ECL hyperplasia was higher in HP- ($p=0.02$) with a 3.5 RR of developing nodular or carcinoid lesions when compared with HP+. Serum PgI, PgII PgI/PgII and gastrin levels correlated with disease severity in HP- ($p<0.01$) but not in HP+.

Discussion: HP+ AIG have/have had a mild infection without differences in OLGA staging when compared with HP-. HP+ are characterized by lower gastrin and gastrin/PGI levels that, considered the lack of differences in OLGA staging, seem to suggest an impairment of gastrin release and could have a selective G Cell damage leading to lower levels of gastrin and Gastrin /PgI ratio. Consequently, they have lower degrees of ECL hyperplasia and a lower risk of developing carcinoids when compared with HP-.

The presence of antrum atrophy in HP- could be explained with the supposed autoimmune antrum-damage hypothesis in AIG.

In conclusion, HP+ and HP- AIG have some differences in serological phenotype and HP infection may correlate in AIG with lower ECL hyperplasia and lower risk of neuroendocrine tumor.

Autoimmune gastritis: serum biomarkers and OLGA staging

Gemma Maddalo[1], Costanza Orlando[1], Matteo Fassan[2], Massimo Rugge[2], Donato Nitti[1], Fabio Farinati[1]
[1]Department of Surgery, Oncology and Gastroenterology, Padua University, Padua, Italy
[2]Department of Medicine, Padua University, Padua, Italy

Introduction: Autoimmune gastritis (AIG) is a precancerous condition at risk for both gastric adenocarcinoma and gastric type I carcinoid. The OLGA staging (Operative Link for Gastritis Assessment) has been introduced and validated in patients with H. Pylori-related chronic gastritis (MAG), as well as in AIG. Patients with OLGA III and IV are at risk for adenocarcinoma but, with respect to AIG, gastric carcinoid can be diagnosed frequently also in OLGA II patients. The aim of this study was to assess whether AIG and its advanced stages (OLGA II-IV) can be predicted by simple serum biomarkers.

Materials and Methods: One hundred and nine consecutive patients with AAG, 157 patients with GAM and 38 controls were recruited in this study and underwent endoscopy, blood tests (ELISA) and multiple biopsies mapping for OLGA staging

Results: The distribution of the median values of PgI, PgI/PgII, gastrin and gastrin/PgI in AAG, MAG and controls was highly significantly different ($p \leq 0.0001$). Gastrin/PgI, a new parameter, was introduced considering the inverse behaviour of gastrin and PgI in AIG. In the comparison amongst controls, AIG and MAG the cut-offs identified were 30 µg/L for PgI, 3 for PgI/PgII and 6 for gastrin/PgI. With these cut-offs the determination showed high sensitivity, specificity and accuracy. The distribution of the various parameters in AIG according to the OLGA staging was significantly different (stages 0-I vs II-III/IV) for each parameter $p>0.0005$. The cut-off for gastrin/PgI had the same value between control and AIG and between AIG and MAG. In AIG, the distribution of PgI and gastrin/PgI was significantly different in the various stages of hyperplasia of ECL (linear, micronodular, nodular hyperplasia and carcinoid). Nodular hyperplasia and carcinoid aggregated in advanced stages of OLGA $p<0.0001$. A multivariate analysis identified PgI/PgII and gastrin AUC data as the best parameters to differentiate AIG, MAG and controls. A Stewise Discriminant Analysis confirmed this results, with 80% overall of the patients correctly classified by the serum parameters. Gastrin/PgI, even though not selected in the multivariate analysis is an additional promising marker

Discussion: PgI/PgII ratio and gastrin can differentiate control patients from those harbouring precancerous conditions, such as AIG and MAG, and discriminate between the two, thus confirming the role of the determination of serum markers in sorting out-patients that should undergo endoscopy.