PARTICULARITIES OF CHEMICAL GASTRITIS IN CHILDREN

Gabriela Păduraru[1], Marin Burlea[2], Valeriu V. Lupu[3], Smaranda Diaconescu[3]
[1]PhD student "Gr. T. Popa" University of Medicine and Pharmacy of Iasi, Resident Physician – Pediatrics, "St. Mary" Children's Clinical Emergency Hospital of Iasi, Romania
[2]Professor, Pediatrics, "Gr. T. Popa" University of Medicine and Pharmacy, 5th Pediatrics Clinic, "St. Mary" Children Clinical Emergency Hospital, Iaşi , Romania
[3]Assistant, Pediatrics, "Gr. T. Popa" University of Medicine and Pharmacy, 5th Pediatrics Clinic, "St. Mary" Children Clinical Emergency Hospital, Iaşi, Romania

ABSTRACT
Objectives: The authors intend to show correlations between the clinical, endoscopic and echographic aspects of the chemical gastritis and the infection with *Helicobacter Pylori*. **Materials and methods**: A lot of 298 patients hospitalized in the 5th Clinic of Paediatrics of the „Sf. Maria" Emergency Children Hospital Iași during the period January 2008 – December 2010 was studied. The patients underwent upper digestive endoscopy, abdominal echography and esogastroduodenal transit. **Results**: The symptomatology was present in the majority of patients with epigastric pain, nausea, biliary vomiting. Anatomical changes of the gall bladder were echographically detected: septate cholecyst, hypotone, thickened walls. Macroscopic lesions of the gastric mucosa, especially of the antral region and large quantities of bile in stomach were endoscopically observed. Gastric biopsy was used to detect the presence of *H. pylori*. EGD transit has shown motility changes of the superior digestive tube in most patients. The treatment consisted in administration of gastric acidity inhibitors and ursodeoxicolic acid for 21 days. A positive response to this treatment was obtained for the majority of cases. **Conclusions**: The reflux gastritis is a new clinical and therapeutic entity in the paediatric practice, frequently occurring in anatomically normal stomach. The reflux gastritis can be associated with the HP infection and aggravates it clinically. It is also frequently correlated with anatomical anomalies of the gall bladder and changes in the digestive tract motility.

Key words: reflux gastritis, upper digestive endoscopy, child

INTRODUCTION
Reactive gastropathy is the second frequent diagnosis established by gastric biopsy after *H. Pylori* gastritis. The disease is secondary to the bile reflux and was initially described after partial gastrectomy (Billroth I or II). At present, this pathologic entity is considered a nonspecific response to a variety of other gastric irritants.

MATERIALS AND METHODS
A group of 298 patients, hospitalized at the 5th Clinic of Paediatrics of "St. Mary" Emergency Clinical Hospital for Children of Iasi, was subject to studies, over the period from January 2008 to December 2010, and diagnosed with reflux gastritis, based on the endoscopic, echographic and radiological aspects (the upper gastrointestinal series).

RESULTS
During the period of the study, after the upper digestive endoscopy examination, 1170 patients were diagnosed with various forms of gastritis, of which 25.5% associated the duodenal-gastric reflux.

Being aware that the prevalence of the *H pylori* infection during the previously mentioned period was of 40.4%, we can state that the duodenal-gastric reflux is, after the

bacterial infection, the second frequent cause of chronic gastritis at the paediatric age.

The distribution on age groups shows that this aetiology generally affects older age groups, so that 78.2% of patients are aged between 11 and 18 year-old.

The increased incidence of the duodenal-gastric reflux at the 15-18 year-old age group is correlated with the increased frequency of acalculous cholecystitis and with a nutrition rich in cholecystokinetic foods.

Of the 298 patients suffering from this disease, 220 (73.8%) were female and 78 (26.2%) were male. We remark a slightly increased incidence of reflux gastritis at females. The specialty literature does not remark an increased prevalence of the disease at girls, but genetic predisposition plays an important part.

The distribution depending on the environment of origin shows that this disease appears more frequently at children originating from the urban environment, in 203 cases (68.12%), and rarely at those from the rural environment, in 95 cases (31.87%). This may be explained both by the nutrition habits of this social category, with a diet rich in cholecystokinetic foods, and by numerous stress factors, as well. Nevertheless, the specialty literature does not reveal a more increased prevalence of reflux gastritis at a particular social category.

Assessing the interdependence established between acute or chronic forms of gastritis and the duodenal gastric reflux, we have observed a statistic difference between the groups, in the sense that the duodenal-gastric reflux tends to statistically associate with chronic forms of disease ($\chi^2 = 12{,}55;\ df=1;\ p=0{,}011$).

Drawing a comparison between the group of patients with *H pylori* infection and the group with bile reflux, we have noticed that only 15.2% of those infected with *H pylori* also suffered from duodenal-gastric reflux, whereas of the group diagnosed with duodenal-gastric reflux only 24.2% had a bacterial infection, as well.

	Freq.	***%***	***Valid %***	***Cumulative %***
1-3 yrs.	5	1,7	1,7	1,7
4-6 yrs.	6	2,0	2,0	3,7
7-10 yrs.	54	18,1	18,1	21,8
11-14 yrs.	92	30,9	30,9	52,7
15-18 yrs.	141	47,3	47,3	100,0
Total	298	100,0	100,0	

Table I. Patients' distribution on age groups

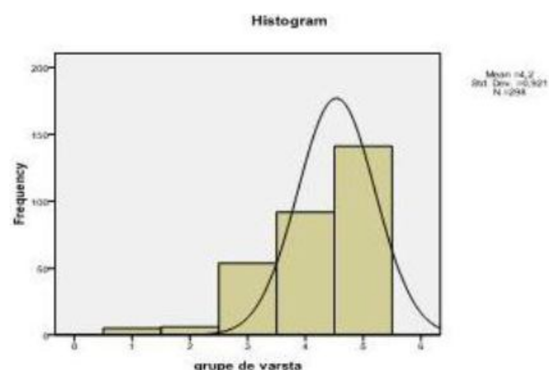

Fig. 1. Patients' distribution on age groups

We can state that patients suffering from *H Pylori* infection develop forms of gastritis which are not associated with the duodenal-gastric reflux, whereas patients with a main aetiology of bile reflux tend not to be infected with *H pylori* ($\chi^2 = 43{,}92;\ df=1;\ p<0.001$).

An increased quantity of duodenal gastric acid may precipitate and remove the bile salts normally inhibiting *H. Pylori* growth. This does not promote the appearance of foveal metaplasia, producing lesions that *H. Pylori* may colonize. Thus, a vicious circle is created, further decreasing the ability of the duodenal bulb to neutralise the acid, which overflows from the stomach, leading to the appearance of duodenal ulcerous. *H. Pylori* can survive in areas of gastric metaplasia at the duodenal level.

The symptomatology was in 70.80% of the cases (211 patients) of recurrent chronic type.

Clinic manifestations are polymorphic and non-systematized.

At present, the correlation dyspepsia-dismotility is studied on a large scale. Symptoms such as borborygmus, abdominal distension, feeling of gastric fullness, precocious satiety, nauseas, drew the attention on a series of gastrointestinal motility anomalies. Nausea is the consequence of the effect of bile salts and pancreatic ferments within the duodenal-gastric reflux. Overall gastric motility disorders acquire an important role, as well, particularly the antral-pyloric disorder. Post-alimentary epigastric pain, refractory at anti-ulcerous medication, may suggest reflux gastritis. Bile vomiting is frequently met in this disease.

In this study, the statistical analysis reveals a significant association only with digestive signs and symptoms:

- abdominal pains non-ameliorated by nutrition and anti-ulcerous drugs (χ^2 = *203,01; df=1; p<0,001*); *phi=0,417* – demonstrate that this sign is moderately correlated with chemical gastritis;
- epigastric pain (χ^2 = *155,48; df=1; p<0,001*); *phi=0,365* – show a moderate correlation established between the two variables;
- flatulence (χ^2 = *678,47; df=1; p<0,001*); *phi=0,716* – shows that this symptom is strongly correlated with reflux chemical gastritis;
- meteorism (χ^2 = *741,32; df=1; p<0,001*);
- precocious satiety (χ^2 = *769,50; df=1; p<0,001*); *phi=0,811* – shows that this symptom is highly correlated with reflux chemical gastritis;
- bile vomiting (χ^2 = *322,31; df=1; p<0,001*); *phi=0,513* – demonstrate that this sign is strongly correlated with reflux chemical gastritis;

The echographic modifications significantly associated with reflux chemical gastritis are cholecystopathies, in particular, (χ^2 = *9.33; df=1; p=0.002*) appeared at 37.58% of patients, whereas the phi indicator=0.176 for evaluating the scale of effect shows that the alteration of the bile vesicle modestly influences the appearance of a chemical gastritis.

Of all the echographic modifications of the cholecyst, the duodenal-gastric reflux statistically associated with:

- a hypotonic cholecyst (χ^2 = 18,38; df=1; p<0,001)
- a cholecyst with thick walls (χ^2 = 20,14; df=1; p<0,001)
- a cholecyst with septum (χ^2 = 15,38; df=1; p<0,001)
- a prolonged cholecyst (χ^2 = 3,62; df=1; p=0,050)

These echographic aspects are evocative of the coexistence of acalculous cholecystopathy.

Radiological modifications are nonspecific and generally indicate disorders of the gastric-duodenal motility. In this study, the following radiological aspects were significantly associated with reactive gastropathy:

- stomach with hyper-secretion liquid (χ^2 = *3,81; df=1; p=0,051*)
- oedema at the level of the stomach (χ^2 = *2,47; df=1; p=0,0 43*)
- spastic bulb with fast emptying (χ^2 = *3,11; df=1; p=0,048*)
- spastic pillory (χ^2 = *3,84; df=1; p=0,050*)

From an endoscopic viewpoint, specific lesions are described as red strips of mucous membrane with apparent bleeding. (Reactive) chemical gastritis is caused by the harming of the gastric mucous membrane through bile and pancreatic reflux or by some exogenous substances. Bile reflux causes epithelial lesions, erosions and ulcers followed by

foveal regenerative hyperplasia, mucous membrane oedema, local vascularisation lesions, bleeding and hypertrophy of the smooth muscles of the own limb. Lysolecitins and bile acids harm the gastric mucous membrane barrier, permitting the retro-diffusion of the positive ions of hydrogen and producing cell lesions. The overflow of pancreatic juices aggravates this situation. As opposed to other chronic gastritis, chemical gastritis is characterized by the presence of a minimum inflammatory infiltrate.

In this study, we discovered significant association ($\chi^2 = 35.63; df=8; p<0.001$) only in the case of an antral purpura granular form. This result stresses upon the fact that bile reflux determines important gastric lesions, especially at antral level.

Of all the 298 reflux chemical gastritis, 72 cases revealed an infection with *H pylori*, whereas the endoscopic examination shown that when a patient presents both aetiologies, the antral region is affected in 93.1% of cases (Table II). This result justifies the fact that both the bile reflux and the *H pylori* infection determine the appearance of some chronic lesions at antral level (Fig. 2 and Fig. 3).

In chemical gastropathy, alterations are more obvious in the pre-pyloric region, but can extend as far as the oxyntic mucous membrane. The histological alterations associated with the bile reflux include the oedema of the mucous membrane, congestion, fibro-muscular hyperplasia and foveal hyperplasia. Cell proliferation is associated with nuclear reactive alterations. Epithelial alterations are accompanied by a reduced inflammatory infiltrate. At patients of the studied group, it had a pathologic aspect in 42 cases. Out of these cases, 50% revealed an oedema-like pylorus, 35.71%- a punctiform pylorus and 14.28%- an eccentric pylorus (Fig. 4).

The duodenal endoscopic lesions which were significantly associated with reactive gastropathy are the granular, granular-purpura and the pseudo-polypoid congestion ($\chi^2 = 88,512; df=9; p<0.001$).

To conclude with, we can state that patients suffering from duodenal-gastric reflux are more likely to develop severe forms of duodenitis.

Therapeutic conduct in reactive gastropathy envisages three objectives: the prevention of the duodenal reflux, the neutralization of the overflow of substances and the reestablishment of the physiological properties of the structures modified by reflux.

Type of Gastritis	*Freq.*	*%*	*Valid %*	*Cumulative %*
atrophic	1	1,4	1,4	1,4
nodular antral	11	15,3	15,3	16,7
nodular-purpura antral	36	50,0	50,0	66,7
purpura-antral	20	27,8	27,8	94,4
erosive	2	2,8	2,8	97,2
hypertrophic	2	2,8	2,8	100,0
Total	72	100,0	100,0	

Table II. Distribution of gastritis with *H pylori* and the duodenal-gastric reflux

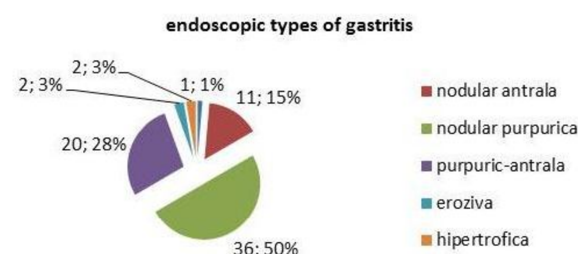

Fig. 2. Distribution of endoscopic forms of reflux gastritis

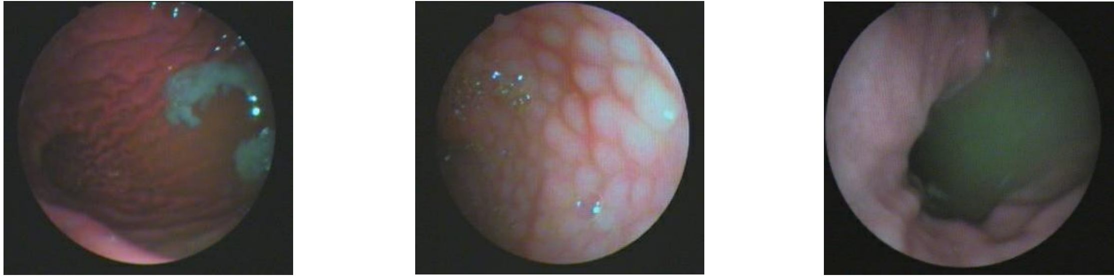

Fig. 3. Endoscopic images of hypertrophic, nodular-antral, granular gastritis

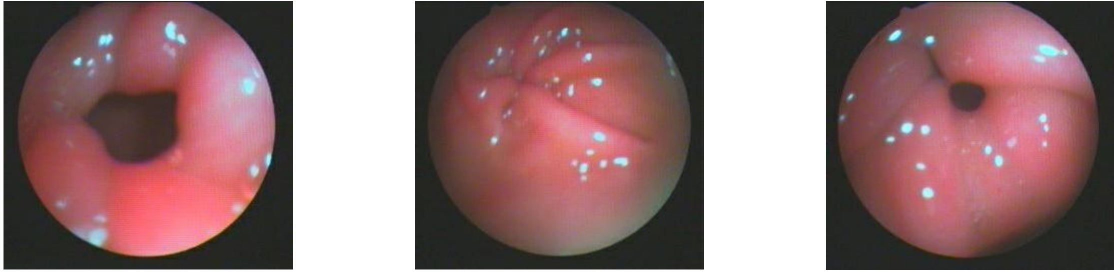

Fig. 5. Endoscopic images of pseudo-polypoid, granular, purpura duodenitis

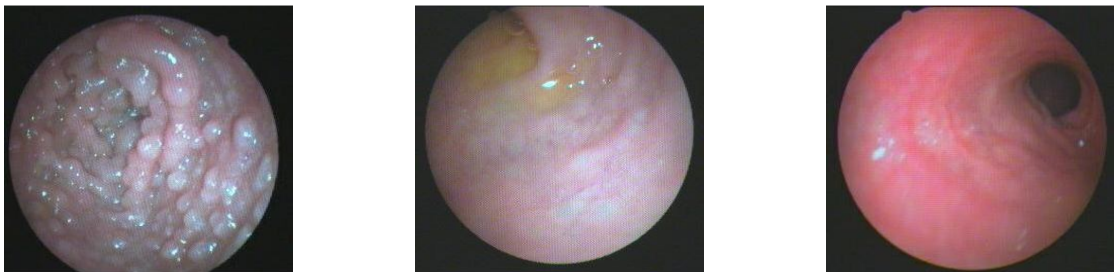

Fig. 4. Endoscopic images of the oedema-like, punctiform, spastic pylorus

The prevention of the duodenal-gastric reflux presupposes the synchronisation of the antral-pyloric / duodenal activity.

The diet consists in fractional meals (4-5 meals per day) that include all nutritional principles, in balanced proportions, ensures a hormonal profile necessary for a gastric-duodenal motility, which determines the physiological direction of gastric and intestinal chemistry.

The treatment consisted in ursodeoxycolic acid and prokinetics, which have the role to increase the rate of gastric emptying and the pyloric tonus. The evolution was favourable, with the amelioration or disappearance of the symptoms. The patients also suffering from *H. pylori* infection, were administered a treatment scheme to eradicate the bacterium (triple or four-fold therapy).

CONCLUSIONS

Reflux gastritis is a new clinical and therapeutic entity in the practise of Paediatrics, frequently appearing under the conditions of an anatomically normal stomach. It can be associated with the *H. pylori* infection, supporting and worsening it, on a daily basis.

From an endoscopic viewpoint, reflux gastritis is characterized by lesions of the antral and pyloric region, and is frequently correlated with anatomic anomalies of the bile vesicle and with alterations of the digestive tractus motility.

REFERENCES

1. Navarro J., Schmitz J.: Gastroentérologie Pédiatrique, 2e Edition, Médicine-Sciences Flammarion, Paris, 2000: 3-4, 6-7, 116-126, 167-181, 612-621
2. Meyer JH. The physiology of gastric motility and gastric emptying. In: Yamada T: Textbook of gastroenterology, Philadelphia: J.B. Lippincott 1991;137-157
3. Gao L, Weck MN, Raum E, Stegmaier C, Rothenbacher D, Brenner H. Sibship size, Helicobacter pylori infection and chronic atrophic gastritis: a population-based study among 9444 older adults from Germany. Int J Epidemiol. Jul 13 2009
4. Nelson DB, Block KP, Bosco JJ, Burdick JS, Curtis WD, Faigel DO, et al. Technology status evaluation report: ultrathin endoscopes esophagogastroduodenoscopy:March2000. Gastrointest Endosc.Jun2000;51(6):786-9.
5. Beers M, Berkow R, eds. Gastritis. In: The Merck Manual of Diagnosis and Therapy. 18th ed. 2006:Section 3, Chapter 23
6. Stein HJ, Smyrk TC, DeMeester TR, Rouse J, Hinder RA.Clinical value of endoscopy and histology in the diagnosisof duodenogastric reflux disease. Surgery 1992;112:796–803
7. RitchieW. Alkaline reflux gastritis: a critical reappraisal. Gut1984;25:975–87
8. Sobala GM, O'Connor HJ, Dewar EP, King RF, Axon AT,Dixon MF. Bile reflux and intestinal metaplasia in gastricmucosa. J Clin Pathol 1993;46:235–40
9. Niemelä S, Karttunen T, Heikkilä J, Lehtola J. Characteristicsof reflux gastritis. Scand J Gastroenterol 1987;22:349–54