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CAMPYLOBACTER PYLORI (CP) AND UPPER GI ENDOSCOPIC AND PATHOLOGICAL CHANGES IN CHILDREN

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Since Marshall report of the association between CP and gastritis or peptic ulcer there is a growing awareness of the CP presence in diseases of the stomach and duodenum.

We examined 44 children (26 Males) 5 to 17 yrs. old who had CP in antral mucosal biopsies. They had undergone gastroscopy for recurrent abdominal pain with or without vomiting or gastrointestinal bleeding. CP was identified in antral biopsies by Giemsa staining and a rapid urease test (CLO test-Delta West). At Endoscopy 17 showed a nodular pseudopolypoid pattern of the antrum and 27 a mild hyperemia and friability of the prepyloric mucosa. These antral lesions were associated with gastric ulcer in 8 cases, duodenal ulcer in 13, esophagitis in 26, duodenitis in 11. Multiple forceps biopsies were taken at sites of endoscopic damage. Histological examination of mucosal specimens showed: antral gastritis in 42 cases associated with peptic esophagitis in 13, Barrett's metaplasia in 2 and duodenitis in 11. In the same period antral gastritis was diagnosed in 65 children, CP was identified in 67% of them. No correlation was found between presence of CP and severity or type of antral inflammation. CONCLUSION: in children CP can be associated not only with antritis and ulcer, but also with esophagitis duodenitis or Barrett's esophagus.

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PEPTIC ULCER IN CHILDREN: 5 YEARS FOLLOW-UP AFTER RANITIDINE THERAPY

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H2 receptor antagonists changed the prognosis of peptic ulcer in adults, but long term follow-up studies in children after ulcer has been healed with ranitidine are lacking.

We carried a 5 yrs follow-up on 31 children (20 males) 3 to 14 yrs old with endoscopically diagnosed duodenal (23 cases) or antral ulcer (8 cases). After an 8 weeks course of ranitidine (5-10 mg/kg) 93% healed their ulcer and therapy was discontinued. Endoscopy was repeated every 6 months or when symptoms recurred. Campylobacter Pylori (CP) was searched in antral mucosa in 22 and serum Pepsinogen I (PG I) determined in 29 cases.

After 1 to 3 years 14 children (12 males) showed a recurrent ulcer crater at endoscopy, an 8 weeks course of ranitidine was repeated and maintenance never discontinued. Serum PG I was higher in relapsed children (80.5 vs. 54.9 pg/ml $P < .001$), CP was present in 12 (85%) of relapse and in 5 (29%) of non relapsed children. None relapsed on maintenance treatment.

CONCLUSION: Peptic ulcer relapse rate in children is 45%, elevated serum PG I, CP in antral mucosa and male sex have an unfavourable prognostic value

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ESOPHAGEAL pH MONITORING CRITERIA IN INFANTS WITH RECURRENT RESPIRATORY DISEASE. Yvan Vandenplas, Anne Malfroot, Isi Dab. Academic Children's Hospital Vrije Universiteit Brussel, Brussels, Belgium.

Although already reported before, the association between gastro-esophageal reflux (GER) and respiratory disease (RD) has not been generally recognized before the late 70ies, related to the development of esophageal pH monitoring (Euler; Pediatrics 1979). Silent GER has been reported to occur in as much as 61% of children with RD (Buts; Eur J Pediatr 1986). We performed a pH monit in 83 children (age 2months-15 years) with RD without digestive manifestations. pH Data were abnormal (Vandenplas; J Pediatr Gastroenterol Nutr 1987) in 38/83 infants (46%). If GER was treated efficiently, pH data normalized and RD recovered (Malfroot; Pediatr Pulm 1987). A not sufficiently emphasized difficulty is that GER parameters have been defined by gastroenterologists interested in patients with emesis, esophagitis, failure to thrive (Johnson; Am J Gastroenterol 1974). Reviewing our pH data we were struck by a typical pH pattern in 25/83 infants (30%) with RD. Although all parameters were strictly within normal ranges in all infants, pH at night (4.2, range 4.0-4.8) was 1.0-2.3 pH ($p < 0.01$) below mean pH (5.7, range 5.2-6.6) during day. Standard deviation of pH during night was smaller (0.67) versus daytime (1.43) ($p < 0.01$). Similar tracings were not observed in asymptomatic infants, or infants with digestive manifestations. 9/25 infants in whom scintiscanning was abnormal were treated for GER, and 6 recovered from their RD, suggesting GER was responsible for their RD although pH monit was considered as normal. We conclude that criteria for abnormal pH monit in infants with RD might differ from those used in digestive manifestations.

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RESPIRATORY ABNORMALITIES DURING SLEEP ASSOCIATED WITH GASTROESOPHAGEAL REFLUX IN INFANCY. Yvan Vandenplas, Liliane Sacre. Academic Children's Hospital Vrije Universiteit Brussel, Brussels, Belgium.

To determine whether gastroesophageal reflux (GER) might be a factor in the pathogenesis of (awake) apnea in some infants, we analysed the frequency of prolonged apnea ($> 15s$) and of irregularly frequently repeated short apneas (5-15s) ("respiratory dysfunction") in control infants (n:584), infants with a GER-pathology (n:60), and in infants presenting with an ALTE (Acute Life-Threatening Event) (n:62). In infants with a resp dysf (n:76), the incidence of GER (assessed by pH monitoring) was looked after. Resp function during sleep was assessed by Oxy-Cardio-Respirography (n:197) or by polysomnography (n:585). Results failed to show any causal relationship between prolonged apnea and GER. Resp dysf, although NOT indicating an increased SIDS-risk, appeared to be related to GER ($p < 0.01$). pH Data were abnormal in 8%, and resp dysf present in 5% of the control infants. In controls with a resp dysf, GER was present in 76%. According to different parts of the study GER-path was detected in 43-100%; a resp dysf was observed in 50-100%. Resp dysf was associated with abnormal pH data in 40-71%. If the GER was treated efficiently (significant decrease or normalisation of pH data), resp dysf disappeared in 95%. If GER was resistant to therapy, resp dysf was still present in 79% (15/19 infants) ($p < 0.001$). If resp dysf persisted, GER was still present in 76% (16/21 infants) ($p < 0.01$). Analysis of pH data and sleep investigations were performed double blind. A majority of ALTE-infants have significant GER, especially if ALTE occurred awake. We conclude that GER in infants is often associated to a typical breathing pattern during sleep.

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ULTRASOUND OBSERVATION OF OESO-GASTRIC JUNCTION.

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The oeso-gastric junction, the abdominal oesophagus and the gastroesophageal reflux (GER) are easily seen by ultrasound examination. We studied 91 children under 3 years, control or suffering from GER, the examination were performed by one operator using a real time echograph and a 5 or 7 mhz transducer, placed in the epigastrium, to obtain a longitudinal section of the abdominal oesophagus.

The examination is then carried out over 20 minutes by continuous scanning immediately after a liquid meal. The length and the thickness of oesophagus are measured and the number of important GER. 37 children had other examinations: baryum examination (n = 23) endoscopy (n = 17), pH metry (n = 29). In most examination in control children, the oesophageal length was more than 15 mm, thickness less than 8 mm, a few GER happened, less than 4 before 2 months, 3 before 6 months, 2 before a year and 1 beyond.

In our experience, every pathologic baryum examination or endoscopy (hiatal hernia or severe esophagitis) has been confirmed by echography, oesophagitis in the form of esophageal thickness enhancement, cardial malposition in the form of length lessening. In the same way, every pathologic pH metry was confirmed by an enhancement of the GER number. We propose oesophagus echography as the first examination of the oesogastric junction, replacing baryum examination, preceding or avoiding in case of normality, endoscopy or pH metry.

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ESOPHAGEAL ACHALASIA AND DYSAUTONOMIA IN CHILDHOOD.

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Very few cases of esophageal achalasia associated to severe dysautonomia have been reported. Among esophageal manometries performed from 1982 to 1987 (n=680), we found in 34 children (5%) a partial (insufficient relaxation of lower esophageal sphincter (LES)) or complete (increased LES pressure without relaxation) achalasia. Dysautonomia was associated in 11/34 cases (32%).

Two groups appeared considering clinical and manometric patterns. In group 1 (n=5), complete achalasia was worsening, leading to surgery in 2 cases (modified Heller's myotomy). Dysautonomia was severe (Ondine's curse, Riley-Day syndrome) or mild (alacrima with glucocorticoids deficiency, pupillar dysmotricity). In group 2 (n=6) partial, asymptomatic achalasia was found in infants hospitalized for unexplained apneic episodes ("near-miss" sudden infant death syndrome), with gastro-esophageal reflux documented by intraesophageal PH-monitoring in 4/6. Oculo-cardiac reflex studies and computerized 24-hour ambulatory electrocardiographic recordings suggested an increase in cardiac vagal tone which was the only dysautonomic manifestation. Achalasia spontaneously disappeared in 4 to 6 months.

These data seem to bring interesting physiopathological light and justify systematic extensive evaluation of autonomic nervous system when esophageal achalasia is discovered (clinical assessment of vaso-motor, sweating and nociception abnormalities; pupillar motricity, lachrymal secretion and cardiac rhythm studies).