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INDOMETHACIN AND GUT BLOOD FLOW VELOCITY IN THE PRETERM NEONATE

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The use of indomethacin (IND) in patent ductus arteriosus is said to pre-dispose to necrotising enterocolitis. We have therefore used duplex pulsed Doppler ultrasound to prospectively study, averaged over 6 cardiac cycles (CC), the peak systolic velocity (FSV), area under the peak velocity envelope (AUPVE), and the time averaged mean velocity (TAMV) of blood flow at the base of the superior mesenteric artery (SMA) and in the coeliac axis (CA). Ten studies were performed on 9 infants receiving their first of three bolus doses of indomethacin (0.2 mg/kg IV). The velocity profiles prior to indomethacin in the SMA were characterised by loss or even retrograde diastolic flow. In only 3 was there absent diastolic flow in the CA. Indomethacin led to a profound fall by 5-8 mins in the FSV, TAMV and AUPVE in the SMA.

Mean \pm SD	SMA		p
	pre-IND	post-IND*	
FSV cm/sec	73 \pm 28	36 \pm 12	<0.0003
TAMV cm/sec	21 \pm 8	9.5 \pm 4.8	<0.0009
AUPVE cm/CC	8.4 \pm 3.7	3.7 \pm 1.9	<0.0015

*velocity at maximum fall

Qualitatively similar, but attenuated, changes were seen in the CA. In no cases was this fall sustained and had usually recovered to pre-dose levels by 1-2 hours. Thus, these data provide a rational explanation for the association between IND and necrotising enterocolitis.

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INVOLVEMENT OF THE COLON IN THE PATHOGENESIS OF DIARRHEA IN JAUNDICED INFANTS IN PHOTOTHERAPY.

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Hyperbilirubinemic newborns (h.n.) in phototherapy (ph) often develop diarrhea. We have previously presented evidence that this is of secretory type (1), furthermore in an animal model we showed that bilirubin acts as a secretagogue in the proximal jejunum (2) and this can be held responsible for such effect. In order to investigate the involvement of the colon in such secretory state, we studied electrolyte and water transport, using the technique of non equilibrium dialysis of the rectum with an isotonic solution, in 10 h.n. in ph, in 10 h.n. not light treated and in 10 controls. All infants were at term and breast fed. There were no differences in birthweight, gestational age, weight and age at beginning of the study between the three groups. Bilirubin level at the study was 15 \pm 0.3 mg% in the light treated group and 13.9 \pm 0.2 mg% in the group of h.n. not in ph. Results, expressed as water and electrolyte net fluxes, are reported in the table.

Values are mean \pm SE	Water	Na	Cl	K
	μ l/min/cm ²	μ Eq/min/cm ²	μ Eq/min/cm ²	μ Eq/min/cm ²
Jaun. light-treated	2.1 \pm .04	286 \pm 8	291 \pm 11	3 \pm 3
Jaun. not light-treated	3.6 \pm .06	357 \pm 8	500 \pm 11	69 \pm 2
Not jaundiced	3.3 \pm 0.9	331 \pm 10	358 \pm 13	58 \pm 6

CONCLUSIONS: The impaired absorption of water and electrolytes seen in h.n. in ph. and not in the h.n. not light treated demonstrates: 1) the colonic involvement in the pathogenesis of secretory diarrhea and 2) that this effect is caused by the simultaneous action of hyperbilirubinemia and phototherapy.

1) M. De Curtis et al. Lancet 1,909,1982; 2) A. Fasano et al. Pediatr. Res. 18,1049,1984.

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CAMPYLOBACTER PYLORI GASTRITIS IN CHILDREN - A COMMON CAUSE OF SYMPTOMS?

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The aim of this study was to assess the incidence of Campylobacter pylori antibody in a paediatric population and relate this to symptoms, and the presence of gastritis. Three groups were studied. Group I - 51 consecutive symptomatic patients (5-16 years) undergoing upper GI endoscopy. Group II - 51 aged-matched hospital controls with no significant GI symptoms. Group III - 150 well schoolchildren. Specific serum IgG antibodies were measured using a sensitive ELISA technique. Antral biopsies were cultured from all patients in Group I and examined histologically. 20% of patients in Group I had significant titres and in all cases \geq 1:1,600. C. pylori was present on the gastric mucosa in all of this sub-group and in no other, and histological gastritis was present in all but 2 cases. In contrast only 4% of Group II and 5% of Group III had significant antibody titres and in all cases $<$ 1:1,600.

We conclude that a high specific IgG titre to C. pylori indicates active gastritis and that a significant proportion of children with upper GI symptoms have such an association. Unlike adults, few normal children appear to have been exposed to this organism.

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DIAGNOSIS OF CAMPYLOBACTER PYLORI GASTRITIS BY IgG ELISA

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11 patients age range 8-15, median 13 years, presenting with abdominal pain were investigated for Campylobacter pylori (C pylori) associated gastritis by upper GI endoscopy and serum IgG antibodies to C pylori. Control sera were obtained from 9 patients aged 9-15, median 12 years, undergoing orthopaedic procedures. Antral biopsies were examined for chronic gastritis and stained for C pylori using a modified Giemsa stain. C pylori specific IgG antibodies were assayed by an indirect ELISA technique, using a soluble bacterial antigen preparation as antigen and the results expressed as optical density measurement. 4 patients with chronic gastritis were colonised with C pylori. Serum IgG mean optical density value was 172 \pm 35 for C pylori +ve patients and 30 \pm 16 for C pylori -ve patients. Control patients had a mean optical density value of 22.9 \pm 4, similar to C pylori -ve patients.

Serum IgG assay for C pylori correctly identified those patients with C pylori associated chronic gastritis, and can be used to screen patients for the condition.

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CAMPYLOBACTER PYLORIDIS IN CHILDREN

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It was reported that Campylobacter pyloridis colonization of gastric mucosa is responsible for active chronic gastritis and is also etiologically related to peptic ulceration.

According to Marshall a very specific and of high diagnostic value is CLD-test, designed to detect the urease enzyme of Campylobacter pyloridis.

The aim of the study was to determine the incidence of the Campylobacter pyloridis infection in children with upper gastrointestinal symptoms.

Material and methods

35 children, 14 girls and 21 boys, aged 8 - 18 y., mean age 14, were included in the study. After an overnight fast endoscopy using Olympus GIF P2 instrument without any premedication was performed. Biopsies were assessed from the antral epithelium for CLD-test and histological examination.

Results

8 children had duodenal ulcer, 2 ulcer-related deformation of duodenal bulb, 9 gastritis, 16 were without pathology. Of these 16 were CLD-positive and 19 CLD-negative. Active duodenal ulcer and gastritis patients had a high incidence of Campylobacter pyloridis infection; 75 and 78% respectively. 10 CLD-positive patients (5 with ulcer and 5 with gastritis) received DE Nol (Gist Brocades) for 4 weeks, what significantly reduced the incidence of Campylobacter colonization; from 100% to 30%

Conclusions:

1. Campylobacter pyloridis infection is remarkably associated with chronic peptic disorders in children.

2. De Nol eradicates Campylobacter pyloridis from the antrum of children with duodenal ulcer and gastritis in a significant number of cases.

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CAMPYLOBACTER PYLORI IN CHILDREN. A PRELIMINARY STUDY. M. Bonamico, C. Chiesa, F. Carpino*, L. Pacifico, F. M. Magliocco*, G. Pitzalis, D. Bosco*, R. Castronuovo, M. Midulla, A. Signoretti. Departments of Paediatrics, Bio-pathology*, and Anatomy*, "La Sapienza" University of Rome, Italy.

Campylobacter pylori (CP) has been isolated from the human gastric mucosa and associated with gastritis and peptic ulcer disease. Most reports have been described in adults. We have initiated a prospective study in which we used histological stains (acridine orange, Warthin Starry silver), scanning and transmission electron microscopy, culture and urease production to identify CP in antral and duodenal biopsies obtained from children undergoing upper gastrointestinal endoscopy for chronic epigastric pain. Complete evaluation has been obtained on 12 children whose plasma gastrin and pepsinogen levels were normal. Six of them were judged to be infected with CP on the basis of histological stains, electron microscopy, and culture of antral and duodenal biopsies. In the CP positive children, gastric mucosa appeared endoscopically normal, but showed histologically moderate to severe inflammatory changes. CP was also detected in the duodenal mucosa of 3 of these patients whose endoscopic appearances revealed either micronodular aspect or mucosal erythema. The presence of CP in the duodenal bulbs did not involve areas of gastric metaplasia. Five CP positive patients were treated with amoxicillin (AMX) for 4 wks, 1 for 2 wks. Resolution of symptoms, and eradication of CP as demonstrated in biopsy specimens taken 15 days after discontinuation of therapy, occurred only in those treated for 4 wks.

CONCLUSIONS: All children with chronic epigastric pain should be evaluated for the presence of CP in the gastric mucosa in spite of normal endoscopic appearances. The organism should be also determined in the duodenal mucosa; though the association between CP and gastric metaplasia has been reported in adults, our data on children do not support it. Further studies should be done in order to clarify the role of CP in the duodenum of children. Our preliminary results suggest the efficacy of a 4-wk course with AMX in eradicating the organism.