83 LIVER DAMAGE IN JUVENILE INFLAMMATORY BOWEL DISEASE

A.Nemeth, J.Ejderhamn, B.Strandvik
Dept of Paediatrics, Karolinska Institute, Huddinge University Hospital, Stockholm, Sweden.

In order to investigate frequency and extent of liver damage in children with inflammatory bowel disease(IBD) we followed 34 patients with ulcerative colitis(UC) and 12 with Crohn's disease(CD). The children were followed prospectively from onset of disease whereby clinical and biochemical activity was compared to liver function tests(LFT). The liver damage was assessed according to LFT and biopsy was performed in all cases with LFT elevated above 3 times normal levels.
In UC the liver damage was not related to duration and/or extent:

	Duration				Extent		
LFT	at onset	<2yrs	2-5yrs	>5yrs	total	distal	proctitis
0		5	5	4	6	5	2
mild	5	3	5		9	4	1
severe	6		1		5	1	1

The signs of damage were permanent in only 1 case. No relation was found to treatment and relapses. No correlation was found between LFT and other laboratory tests. Biopsy in the 7 cases with suspected severe damage showed cirrhosis(1), cholangitis(2), chronic aggressive hepatitis(1), steatosis(1) and minor changes(2 cases). In CD 4 of the 12 patients had signs of mild liver damage, which could not be related to activity or duration of the disease or treatment.
It is concluded that there seems to be a high risk of liver damage in childhood IBD and that severe forms seem to occur at the time of onset or early in the course of the disease.

84 CRYPTOSPORIDIASIS IN CHILDHOOD DIARRHOEA

R.Tormo,D.Infante,D.Panadés,D.Yeste,T.Español, R.Bartolomé,X.Ballabriga.
Hospital Infantil "Vall d'Hebrón", Barcelona, Spain

Cryptosporidiasis usually is diagnosed in immunodeficient patients. We investigated the incidence in childhood diarrhoea. METHODS: stool coloration alcohol-acid resistent technique. We studied 20 primary and secondary immunodeficient patients with diarrhoea (group A); 43 cases attending a day care center during a gastroenteritis outbreak (group B) and 40 cases with acute diarrhoea (group C).
RESULTS: feces were positive for cryptosporidium in: group A: 2 children; the parasite was also found in jejunal mucosa; only 1 responded to Spyramicine. Group B: 17 cases; 2 had low IgA levels; the diarrhoea lasted from 1 week to 2 months and cured without treatment. Group C: 9 children; all were immunologically normal and cured without treatment.

CONCLUSIONS: because of this high incidence, cryptosporidium should be investigated in childhood diarrhoea, even in children with a normal immunological status.

85 CRYPTOSPORIDIOSIS IN IMMUNOCOMPETENT CHILDREN : EPIDEMIOLOGIC STUDY IN DIARRHEA.

C.Maurage, M.Naciri, F.Arnaud-Battandier, Hôpital de Clocheville, 37000 Tours, INRA 37380 Nouzilly, Hôpital des Enfants Malades 75015 Paris, France.

Cryptosporidium was known as a cause of severe diarrhea in immunodeficient patients. Recently it appeared to be also involved in normal population. A 12 month prospective study was performed in Tours, a town with a mainly rural population, in children hospitalized with diarrhea. 200 stools from 190 children were evaluated for the presence of Cryptosporidium by a sugar flotation method. 4 children had positive stools. Stools were watery and contained mucus, without any blood, in 2 cases. In 3 cases, the diarrhea was acute and of short duration, with dehydratation and vomiting. All 4 children were immunocompetent. Our 4 cases were negative for rotavirus and bacteria, but two had Candida albicans in the stools Control stools from the 4 children were negative for Cryptosporidium within one month, but one was found positive for Giardia.The members of the 4 cases'families (total, 11 persons) had negative stools. 2 patients were in 2 different day-care centers ; the stools of all the other children were negative (total,37 children) This 12 month study demonstrated that cryptosporidiosis can be found in immunocompetent children with diarrhea, with a frequency of 2.1 percent. Furthermore, precautions must be taken to avoid possible contamination of hospitalized immune deficient patients.

86 TRANSPORT PROPERTIES OF ISOLATED JEJUNAL EPITHELIUM FROM CHILDREN WITH CONGENITAL MICROVILLUS ATROPHY (CMA).

J.F. DESJEUX, E. GRASSET, J. NAVARRO, J. SCHMITZ.
INSERM U 290, Hôpital Saint-Lazare, 75010 Paris.

The functional defects in CMA appear to be in the mucosa itself. 10 fragments of small intestine from 3 patients with CMA were studied in vitro to determine electrical parameters (short-circuit current, Isc, and ionic conductance, G) and net ionic and glucose fluxes (Jnet). db cAMP (1mM) stimulated Isc and net Cl^- secretion ; somatostatin 14 (10^{-6}M) did not alter Isc and stimulated Na and Cl absorption ; ouabain (0.1mM) inhibited Isc ; glucose (10mM) stimulated Isc and Na absorption. The intracellular glucose accumulation was low (C/M = 2.6 + 0.3 vs 15.8 + 1.5 in control at 10mM glucose in the medium) ; it was further reduced by phloridzin and ouabain. Peroxydase permeability and G were within control range. The results indicate that the jejunal epithelium of CMA patients is not leaky ; the basic transport processes are present (Na-K ATPase, glucose-Na cotransport, Cl^- secretion). We speculate that the epithelial abnormality is an imbalance between decreased absorption and unaltered secretion.

87 CYTOCHEMICAL DETERMINATION OF IMMUNOREACTIVE PROLACTIN IN NORMAL AND ABNORMAL INTESTINAL MUCOSA.

G. Theintz, D. Duhamel, J. Cox, D. Nusslé, Cl. Le Coultre, F. Pizzolato, P.C. Sizonenko. Dept of Paediatrics & Genetics + Dept of Pathology, University Hospital, Geneva.

Prolactin-like material (PLM) has been identified in the mucosa of some coeliac patients. This finding raises questions about the nature and function(s) of this peptide, particularly as pituitary prolactin is involved in metabolism and osmoregulation in some mammals other than man. Using a peroxidase-antiperoxidase technique, we have examined the distribution of PLM in the mucosa of small and large intestines of 44 infants and children aged 1 month to 15 yrs. Material consisted in 31 jejunal biopsies [20 flat mucosae], 4 rectal biopsies [2 in aganglionic segment of Hirschprung disease], 7 proximal appendix fragments [5 acute appendicitis], 1 ileon [proximal to ileostomy] and 1 colon [Hirschprung, closure of colostomy]. Cells containing PLM were identified at all levels of the small and large intestine, principally at the bottom of the crypts. They were present in 9/20 flat and 1/11 normal jejunal mucosae. Far less PLM was found in the appendix, whether inflamed or not. Two rectal biopsies from aganglionic segment were negative for PLM whereas normal tissue proximal to colostomy was positive. These preliminary findings suggest that this gut peptide which by no means ought to be identical to pituitary prolactin could have a role in the adaptative changes of the gut mucosa.

88 The effect of serotonin on crypt cell proliferation in the human duodenal mucosa in vitro.

D. N. Challacombe, E. E. Wheeler
The Somerset Children's Research Unit,Musgrove Park Hospital, Taunton.

Cell kinetic studies on the small bowel mucosa in coeliac disease have shown an increased number of proliferating cells per crypt due to a reduction in the crypt cell cycle time. Similar findings occur in rats after the intraperitoneal injection of serotonin.In vitro studies have therefore been performed to determine whether serotonin influenced cell proliferation in the human duodenal mucosa.Normal mucosal biopsies from 18 patients were cultured for 22 hours, using an organ culture technique. Specimens from 9 patients were cultured in a basic medium and from 9 patients in the basic medium with added serotonin (2mg/ml). After 18 hours a stathmokinetic technique was used to determine the crypt cell proliferation rate (CCPR) by adding vincristine sulphate (1µg/ml) to the culture medium. Tissue samples were removed at hourly intervals for 4 hours and the mean number of metaphase arrests in 10 whole crypts was determined.The CCPR was derived by drawing a regression line between the mean values obtained on each patient. The results indicate that serotonin significantly increases the CCPR in cultured tissues ($p < 0.001$). Described abnormalities of serotonin synthesis in coeliac disease may be involved in the pathogenesis of this disorder.