83

EFFECTS F GLIADIN ON THE INTESTINAL MUCOSA IN PATIENTS WITH CELIAC DISEASE (CD)

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The influences of gliadin on the function of the intestinal mucosa were studied in children with enteral protein intolerances in a prospective study.

Group 1a* CD, n=14, gluten free diet for 1-2 months (m), histol. findings: type II-III according to SHMERLINC.
Group 1b: n=8, diet for 10-36 m, type I-II.
Group II: Cow's milk protein intolerance, n=7, cow's milk free diet for 1-2 m, type II-III.
Metho d: The netto water flux and the absorption and digestion rates of sugars were determined by perfusion of the upper jejunum immediately before and 2 up to 6 and 24 hrs after enteral infusion of gliadin.

Result : 24 hrs after gliadin infusion the digestion and absorption rates of carbohydrates decrease only in the patients with time of treatment of 1-2 m whereas there are no effects in the patients with treatment logner than 10 m. The netto water secretion increases immediately after starting the gliadin infusion independent on time of treatment.

Conclusion: the data suggests that different cell populations are influenced by gliadin in CD-patients.

84

SERUM PEPSINOGEN I (PG I) AND SEVERITY OF CAMPYLOBACTER PYLORI (CP) GASTRITIS

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Serum PG 1 levels are raised in children with CP associated gastritis and lower after eradication of the mico-organism. To assess whether PG 1 levels are related to the severity of antral inflammation we studied 28 chidren 6-15 y ears old (15 males) with superificial chronic antral gastritis: 19 were CP carriers (CP+) and 9 were CP negative (CP-). Antral biopsy specimens were obtained with a pediatric forceps at endoscopy. They were stained with H & E and Giemsa, only those containing muscolaris mucosa were considered. Density of lamina propria inflammatory cell infiltration, and numbers of intraepithelial mononuclear cells and granulocytes were evaluated and graded 0 to 3. Serum PG 1 was determined by RIA. Mean serum PG 1 (± 1SD) was 72±24 ng/ml in CP+ and 45±8 ng/ml in CP-. children ($p<.005$). Mean total inflammation score was 6 in CP+ and 2.5 in CP-. patients ($p<.01$), the most significant difference was found in the lamina propria inflammation score ($p<.001$). Serum PG 1 levels were significantly correlated with total inflammation scores (Spearman's rho = .874, $p<.01$) in CP+ but not in CP- children.

Conclusions: serum PG1 level can be considered an index of severity of antral inflammation in children with CP gastritis.

85

VEROTOXIN-PRODUCING E.COLI (VTEC) CAUSING HAEMORRHAGIC COLITIS AND HAEMOLYTIC URAEMIC SYNDROME IN THE NETHERLANDS

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There is a proven association between vero (cyto) toxin-producing Escherichia coli (VTEC) and haemorrhagic colitis (HC), and classical, diarrhoea-associated haemolytic uraemic syndrom (D+HUS), respectively. A 2 year old girl referred with barium enema-proven HC developed D+HUS within 12 hours of admission. Culture of bloody-mucoid stools on Sorbitol MacConkey agar medium (Oxoid code CM 813) grew sorbitol-negative colonies that were typed (Difco 2970-47-7) as E.coli O157:H7.

From defrozen stool specimens of 10 other children with D+HUS, admitted during 1986 and 1987, E.coli O157:H7 was isolated once more. Further, faeces extracts from 4 children, including 3 E.coli O157:H7-negatives, showed vero(cyto)toxic activity. This points to the presence of other VTEC, or other verotoxin-producing species in D+HUS. In The Netherlands, too, E.coli O157:H7 appears to be associated with HC and D+HUS. While in England and Wales E.coli O157:H7 is commonly isolated in D:HUS. This is, to our knowledge, the first report from continental Europe.

86

ANTIENDOMYSIAL ANTIBODIES IN THE MONITORING OF CONTROLLED GLUTEN CHALLENGE.

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The controlled gluten challenge /c.g.c./ although questioned recently is still the only recognized measure for the diagnosis of coeliac disease /C.D./. Therefore the sensitivity and the dynamics of antiendomysial antibodies /IgA-EmA/ were analysed in the 68 positive c.g.c. which were performed according schedule: gluten 0,5g/kg/day for the first 6 months and unlimited gluten containing diet subsequently.The c.g.c. were classified in the following categories: 41/68 c.g.c. in cases where initial diagnosis was documented histologically, 20/68 c.g.c. from rural area where initial diagnosis was based on the clinical picture only and 7/68 c.g.c. performed in teenagers in whom C.D. had been diagnosed previously to reveal cases of spontaneous recovery.
The presence of the IgA-EmA was confirmed in all 68/68 positive gluten challenges /sensitivity 100%/.In the course of c.g.c. IgA-EmA appeared earlier /range 1m-6m/ than unequivocal villous destruction has been documented /range 6m-12m/. In all 68/68 cases the IgA-EmA appeared before 12 month c.g.c. have been completed.
The IgA-EmA are the highly sensitive marker in the monitoring of c.g.c.
The c.g.c. may be limited to 12 months instead of 24 month period as recommended by now.

87

REVERSAL OF MALNUTRITION IN CHILDREN WITH CIRRHOSIS DESPITE CONTINUED IGF 1 DEFICIENCY

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Insulin-like growth factor 1 (IGF 1), the mediator of growth hormone, is manufactured in the liver but its release may be depressed by malnutrition. To date, the relative contributions of liver disease and the associated mal-nutrition to the depressed IGF 1 levels found in chidren with cirrhosis is unclear. We have therefore assessed the relative importance of nutritional status by measuring circulating IGF 1 in a group of cirrhotic children before and after reversal of severe malnutrition by intensive enteral feeding. Ten children with malnutrition and cirrhosis (biliary atresia 6; other causes 4; median age 9m range 4m-8y) were studied before and after intensive feeding over 8 weeks. Energy intake was increased by 40% and dietary nitrogen by 33%.

Nutritional status improved dramatically: pre-feed midarm circumference % of media 76.9, post 95.1 ($p<0.001$). As expected, serum IGF 1 concentrations were low before enteral nutrition (mean 2.95nmol/l, range 1-9.3; reference range for age band 6-40 nmol/l, 95% confidence limits). However, despite correction of malnutrition, IGF 1 concentrations remained depressed (mean 4.7nmol/l, range 1-12; $p<0.05$).

These data strongly suggest that the severity of the liver disease is a more important determinant of IGF1 deficiency than the associated malnutrition.

88

APOLIPOPROTEIN B (APO B) GENE IN CONGENITAL DISORDERS OF FAT ABSORPTION.

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The aim of the present study was to elucidate whether the great heterogeneity of biochemical lesions responsible for abetalipoproteinemia (ablp) and Anderson's disease (Ad) resulted from different types of genetic alterations.

4 patients with Ad (SZ, YZ, KZ and MK) and 2 with ablp (MP and SL) were studied. Earlier studies had shown that the enterocytes from the 4 Ad cases and from SL, but not from MP, contained (abnormal ?) apo B 48. DNA from these patients and from 3 normal subjects was prepared from their leucocytes. The following cDNA probes, covering the whole apo B gene, were used : A6c, ABF, AB1, RP2 and SB9, of 3,5, 6,5, 5,1, 0,3, and 6 kb respectively. DNA was treated with the following restriction enzymes : XbaI, PvuII, MspI, EcoRI, HINDIII, HinfI and TaqI and subsequently analysed on Southern blots.

Restriction fragment patterns differed greatly among the patients. SL and MP were heterozygotes for an EcoRI polymorphism at residue 4154 of mature apo B. SL, SZ, YZ, KZ were homozygotes for the 8,6 kb allele produced by XbaI while MP was heterozygote for the same allele and MK was homozygote for the 5,5 kb allele produced by the same enzyme. No major deletion or rearrangement of apo B gene could be found in any of the 6 patients.

In conclusion : 1) ablp and Ad seem not to be associated with a particular apoB allele, and 2) only a closer study of apo B gene will show the different mutations responsible for the diseases.