0031-3998/90/2803-0277$02.00/0
PEDIATRIC RESEARCH
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Vol. 28, No. 3, 199
Printed in U.S.A.

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Abstracts for the 1990 Annual Meeting, September 23–27, Vienna, Austria

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1 TRANSIENT RENAL ACIDIFICATION DEFECT DURING ACUTE INFANTILE DIARRHEA - THE ROLE OR URINARY SODIUM.

Shai Izraeli, Avinoam Rachmel, Yaacov Frishberg, Arie Erman, Bernardo Flasterstein, Menachem Nitzan, Geoffrey Boner, Depts of Pediatrics A and Nephrology, Beilinson Medical Center, Petach-Tiqva, Israel.

Excretion of acidic urine reflects the normal renal response to metabolic acidosis and is present from a very young age. We have studied urinary acidification (UA) daily during the hospital course of 16 infants with acute gastroenteritis and metabolic acidosis (MA). Urine pH on admission was higher than 5.5 in 14 (87%) patients. We hypothesized that inappropriate UA was due to Na deficiency and inadequate Na delivery to the distal nephron. Forty-one urinary samples were collected during MA. The mean pH of 24 samples with Na concentration < 10 mmol/l was significantly higher than the pH of 17 samples with Na concentration > 10 mmol/l (6.04±0.06 vs 5.19±0.1, $p<0.001$). The urine ratios of titratable acid/creatinine and total acidity/creatinine were significantly higher in Na rich urine samples ($p<0.02$), whereas ammonium/creatinine ratio was not. Following administration of furosemide or correction of the Na deficit, appropriate acidification was observed. We conclude that impaired UA is frequently found during MA in infants with acute gastroenteritis and is caused by a sodium deficit and not due to transient distal renal tubular acidosis.

2 FAMILIAL HYPERMETHIONINEMIA PARTIALLY RESPONSIVE TO DIETARY RESTRICTION.

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Three siblings, born to unrelated parents, had hypermethioninemia, failure to thrive, mental retardation, facial dysmorphy and non obstructive cardiomyopathy. Unrestricted diet or methionine load resulted in hypoglycemia, hepatic failure and prolonged increase in methioninemia. Conversely, biologic data were improved under a methionine restricted diet (40 mg/kg/day) but clinical data were only partially responsive. Hepatic S-adenosylhomocysteine hydrolase activity was decreased by 80 % in the 3 children. It was not possible to determine if the enzyme deficiency is responsible for the disease or secondary to an unknown metabolic deficit since S-adenosylhomocysteine has never been found in urine specime of either patient. This possible new inborn error in methionine metabolism is different from the hypermethioninemias previously published.

3 SIDSVICTIMS SHOW LOCAL IgM RESPONS IN TRACHEAL WALL AND IgA RESPONS IN DUODENAL MUCOSA.

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22 SIDS cases and 11 controls were examined immunohistochemically with regard to the presence of IgA-, IgM- and IgG-plasma cells in tracheal wall and duodenal mucosa. The presence of secretory component (SC) and class 2 HLA-DR determinantes were evaluated. A statistically significant increased number ($p<0.01$) of IgM plasma cells was found in SIDS tracheal wall (median 2.1, range 1.6-4.0) compared to controls (median 0.9, range 0.8-1.5). For the IgA- and IgG-isotypes, no difference was found. In duodenal biopsies, the number of IgA plasma cells (median 7.0, range 4.4-11.2) were significantly higher ($p<0.01$) than controls (median 5.4, range 3.9-7.7). SC was found primarely in tracheal glands and duodenal crypts, whereas HLA-DR was found primarely in duodenal villi- and tracheal surface-epithelium. These findings indicates that the secretory immune system is stimulated in SIDS. Maybe the immune respons act as a "trigger mechanism" which causes cerebral hypoxia either direct by release of immune mediators, or indirect by a reflectory mechanism.

4 WEIGHT GAIN OF Ca-SUPPLEMENTED VLBW INFANTS.

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VLBW infants are prone to severe postnatal bone demineralisation owing to a high requirement (3.2mMol/10g weight gain) but a low absorption rate (15-50%) of Calcium (Ca) from formulas. The absorption can be compensated for by increasing the Ca intake. There is, however, concern about fat malabsorption and decreased weight gain because of a high Ca supplement.

Methods: We therefore studied the weight gain of 70 VLBW infants (median birth weight 1020g, range 440-1485; median gestational age 28 weeks, range 24-32), who were supplemented individually by stepwise increasing an admixture of solid Ca-gluconate and Ca-glycerophosphate to the feedings until both Ca and Phosphate were excreted with urine (1-2mMol/l) (1). Infants were fed breast milk or preterm formula. The daily weight gain was calculated from 377 6-day periods and correllated to the average daily Ca intake (milk+supplement) these periods. Infants were studied for 30 days (median) (range 6-66 days). Postmenstrual age at the time of the study was 35 weeks (median) (range 29-43). **Results**: Linear regression analysis did not show any evidence that weight gain (median 20.8 g/day) was reduced by increased Ca intake within the range of 1.3 - 19 mMol/kg×day (median 6.4). Regression line was $y(g/day) = 0.00137 \times (mMol\ Ca/kg \times day) + 19.9$. No case of necrotizing enterocolitis or intestinal obstruction occurred. **Conclusion**: Individual supplementation of VLBW infants with solid Ca-gluconate and Ca-glycerophosphate does not impair weight gain, is well tolerated and effects bone mineralisation identical to in utero as has been shown earlier (1). (1) Pediatr Res 20 : 1050, 1986.