Sucrose Intolerance Tests in the Neonates

by

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Abstract

Sucrose maldigestion is difficult to diagnose especially in newborn babies.

Stool chromatography is the most useful tool to detect the type of saccharide maldigestion.

Using the clinical symptoms as parameter, there were 3 out of 28 babies suffering from sucrose maldigestion, in whom the blood glucose level did not increase more than 40 mg % after the injection of sucrose.

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Introduction

During the last few decades, it was reported that many cases of sucrase-isomaltase deficiency were found in many areas of the world (Weyzer, 1960; Antanovics et al., 1972).

Sucrose together with starch and lactose being the most source of inexpensive daily calorie intake, may account for 50% (Gray, 1971) especially in developing countries as ours. As caloric source also, the local milk formula product (SGM, Sarihusada P.T.) uses sucrose 33% and lactose 28%.

Lactose intolerance in Indonesian newborns has been reported by our colleagues, being 30% out of 70 newborns (Suryono et al., 1973). Our follow-up then revealed that twelve babies died due to marasmus and severe dehydration.

Nowadays, sucrose is also being used as oral electrolyte treatment instead of using glucose, because sucrose is cheaper and available everywhere mostly in developing countries (Saroso, 1965; Nalin, 1975; Munginah et al., 1977).

This paper is a report on twenty eight Indonesian newborn infants in which Sucrose Tolerance Tests were done and the difficulties in making conclusion will be discussed.

Material and method

Twenty-eight Indonesian newborn infants, aged 2 - 5 days, delivered in our maternity unit, Gadjah Mada University Hospital were tested on the tolerance to sucrose as mentioned below.

After fasting 4 - 6 hours early in the morning (5 to 7 p.m.) the babies were fed with 2 gm sucrose/kg of body weight diluted in water as 10 to 20% solution (Ament, 1973) at the volume of daily drinking habit.

All the babies were fullterm without any complication during pregnancy, delivery and postnatally.

Peripheral blood samples were drawn from the heel before feeding every half hour within 2 hours and put in the anticoagulant preservation (Ca oxalate).

The blood samples were then sent to the Department of Clinical Pathology as soon as possible where glucose concentration was measured by the Orthotolidine test method.

Clinical appearances such as vomiting, meteorism and diarrhea were observed during 0.5 - 4 hours thereafter.

In fifteen babies clinitest was done and in three of those with gastrointestinal symptoms, Glucose-Fructose-Tolerance-Test (GFTT) was also examined after the disappearance of the gastrointestinal symptoms.

The GFTT was managed similar to the STT mentioned above except that the solution was composed of glucose and fructose with the same amount.
ANNOUNCEMENTS

VIIth REGULAR SCIENTIFIC MEETING
OF THE COORDINATING BOARD OF THE
INDONESIAN PAEDIATRIC GASTROENTEROLOGY

and

IInd SYMPOSIUM ON BREAST FEEDING

MANADO, NORTH SULAWESI, INDONESIA, 27 — 30 AUGUST 1980

The VIIth Regular Scientific Meeting of the Coordinating Board of the
Indonesian Paediatric Gastroenterology and the IIInd Symposium on Breast
Feeding — will be held in Manado, North Sulawesi, from 27 to 30 August 1980.

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Results

Table 1 shows that mostly the blood sugar level increases 20 - 40 mg% (46% out of 28 babies) and on the other hand in 9 babies (32%) it increases more than 40 mg% while in 6 babies (21%) less than 20 mg%.

<table>
<thead>
<tr>
<th>Increase of blood sugar</th>
<th>Number of cases</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 40 mg%</td>
<td>9</td>
<td>32%</td>
</tr>
<tr>
<td>&gt; 40 mg%</td>
<td>13</td>
<td>46%</td>
</tr>
<tr>
<td>&lt; 20 mg%</td>
<td>6</td>
<td>22%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>28</strong></td>
<td><strong>100%</strong></td>
</tr>
</tbody>
</table>

TABLE 2: The results of the Clinitest in correlation with blood glucose levels

<table>
<thead>
<tr>
<th>Increase of blood sugar</th>
<th>Number of cases</th>
<th>Clinitest positive</th>
<th>Clinitest negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 40 mg%</td>
<td>5</td>
<td>33%</td>
<td>2</td>
</tr>
<tr>
<td>20 - 40 mg%</td>
<td>7</td>
<td>47%</td>
<td>1</td>
</tr>
<tr>
<td>&lt; 20 mg%</td>
<td>3</td>
<td>20%</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>15</strong></td>
<td><strong>100%</strong></td>
<td><strong>4 (26%)</strong></td>
</tr>
</tbody>
</table>

The results of the Clinitest method in correlation with the blood concentration in Table 2 revealed that the stool reducing substance exists in two babies in whom the blood sugar level increases more than 40 mg% where one was positive in the rest with blood sugar level between 20 - 40 mg% or less.

TABLE 3: The results of the clinical observation in conjunction with the increasing blood sugar levels

<table>
<thead>
<tr>
<th>The increase of the blood glucose levels</th>
<th>Number of cases</th>
<th>The clinical appearance</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 - 40 mg%</td>
<td>9</td>
<td>—</td>
<td>meteorism</td>
</tr>
<tr>
<td>&gt; 40 mg%</td>
<td>13</td>
<td>2</td>
<td>vomiting</td>
</tr>
<tr>
<td>&lt; 20 mg%</td>
<td>6</td>
<td>1</td>
<td>diarrhea</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>28</strong></td>
<td><strong>3</strong></td>
<td></td>
</tr>
</tbody>
</table>
Those babies in Table 3 (with gastrointestinal symptoms) were tested then with Gucose- Fructose- Tolerance- Test (GFTT); the results were that increasing blood sugar levels were a little bit higher than those shown in the STT but still below 40 mg/dl and without clinical symptoms.

Discussion

The dietary carbohydrate is presented to the intestinal mucosa at the duodeno-jejunal junction and the enzymes that hydrolyze these oligosaccharides are located within the membrane of the brush border surface of the columnar epithelial cells of the small intestine and are most active in the jejunum.

The mechanism of entry of glucose and fructose into the epithelial cells, as the result of sucrose hydrolyzing process, is unknown yet. It was mentioned that glucose would be actively transported with requiring energy and Na+. That is different from that of fructose where facilitated diffusion will work without requiring Na+ but allow the sugar to enter the cells and move down its own concentration gradient (Gray, 1971). The rest of unhydrolyzed disaccharides, in certain condition, takes the responsibility in the movement of electrolytes, especially NaCl and water into the lumen and will pass as reduction substances in the stool besides microbacteria and organic acids. Malabsorption will be accounted if more than 0.25% reduction substances are present in the stool and or the pH drops less than 6 (Lifshitz et al., 1971) but it normally happens in a newborn baby with breast milk (Ament, 1975).

The diagnosis of disaccharide malabsorption is usually established by considering both the result of Clinitest method and pH determination of the stool. In the case of sucrose intolerance, significant amount of reducing substance may not be found in the stool because sucrose is not a reducing factor.

Disaccharide tolerance test (such as LTT and STT) has been widely used but it may produce false positive tolerance test (“flat curve”) as high as 30%. Biopsy assay is more satisfactory, but as single biopsy can mislead the diagnosis, multiple biopsies are suggested.

The demonstration of abnormal amount of reducing substances (>0.5%) in the stool was confirmed to be a valuable screening test for sugar malabsorption and then investigation on the type of the sugar could be done by stool chromatography (Soeparto et al., 1972). Ament (1975) considered the diagnosis as sucrose maldigestion if the blood glucose concentration in STT did not increase more than 40 mg/dl during the next 2 hours (flat curve) after the injection of 2 gm sucrose/kg body weight in 20% water solution. Using this general statement Table 1 shows that maldigestion occurred in 19 (68%) out of 28 cases.

Table 2 shows that 4 (26%) out of 15 cases suffered from maldigestion un-

Of 36 very low birth weight infants (< 1,500 gm) with large patent ductus arteriosus, 24 (67%) showed satisfactory constriction or closure after indomethacin therapy (mean total dose 0.4 mg/kg). Twelve infants (33%) responded inadequately with seven infants requiring surgical ligation. Response was better in infants 8 to 14 days old compared to those more than 14 days old (89% vs 33%, P = 0.48) irrespective of birth weight or gestational age. Major complications were renal and unrelated to ductus response. Urine output fell significantly (3.65 to 1.65 ml/kg/hr, P < 0.01) and in 47% of infants serum creatinine increased ≥ 1.5 mg/dl. Creatinine was less likely to rise in infants more than 14 days old. Hyponatremia was found in 36% of infants. Serum potassium increased more frequently in infants more than 8 days old and was > 6.0 mEq/litre in 25%. Indomethacin caused a reduction in PaCO₂ (41 to 37 mm Hg, P < .01) and an increase in pH (7.32 to 7.36, P > .02) with no change in base deficit. These changes occurred even in the absence of clinical ductus closure. No other side effects of indomethacin therapy were noted. Three infants died but death was unrelated to indomethacin therapy. Overall survival was 92%, and nine infants (25%) developed mild bronchopulmonary dysplasia.


Trauma to the lower extremities is the principal cause of gait disturbance in early childhood. Three cases are presented to emphasize the relative frequency of children hospitalized for diagnostic evaluation of altered gait who have occult fractures. The cases may refresh the primary physician of the variables that serve as obstacles to accurate diagnosis.


Signs of neonatal neurologic dysfunction, recorded in approximately 40,000 infants, were evaluated prospectively for their ability to predict later motor handicap. Tenfold to 33-fold increases in risk of cerebral palsy (CP) were observed in surviving children with any one of the following characteristics: birth weight less than 2,000 gm, head circumference more than 3 SD above or below the mean, five minute Apgar score of 3 or less, diminished activity or dimin-

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less there were false positive findings. In Table 4 (Chi square test) we try to confirm it by using statistical methods. It seems that the difference in Table 3 is not significant (p > 0.01).

may be is caused by contamination of the stool by urine and as mentioned by Ament (1975) by micro organism hydrolyzation.

<table>
<thead>
<tr>
<th>Increase of blood sugar levels</th>
<th>Results of Clinistest</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Positive</td>
<td>Negative</td>
</tr>
<tr>
<td>&gt; 40 mg%</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>&lt; 40 mg%</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Total</td>
<td>4</td>
<td>11</td>
</tr>
</tbody>
</table>

$X^2 = 0.6818$  
$p > 0.01$

Those who were negative could not be considered as sucrose tolerance since sucrose is not a reducing factor. Stool pH determination is needed to detect whether or not there is any lactic acid produced by micro organisms. The most valuable data are shown in Table 3, in which the clinical symptoms were recorded. Three newborn babies out of 28 cases showed changes in the bowel activities such as meteorism, vomiting and watery stools. The result of GTTT in these three babies done on the next 2 days after the disappearance the gastrointestinal symptoms showed that the blood glucose curve was a little bit higher than that shown in STT without gastrointestinal symptoms. In this case, of course, further investigation is needed.

It was stated also that by sucrose feeding to patients with primary sucrase deficiency some increase in sucrase activity might be induced, which was proved in rats.

Acknowledgement

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REFERENCES


Infection with exposure in utero to be likely in six infants. Passive acquisition of GBS (intrapartum exposure) probably occurred in the three remaining early onset cases. This mechanism was also likely responsible for five nonbacterial infections. The four infants with late onset sepsis or meningitis were not colonized at birth or when discharged from the nursery (day 3); a possible maternal source for infection was found in only one of these infants.


A series of 1,704 infants of blood group O mothers have been studied to determine the relation between the degree of red cell sensitization and the cord hemoglobin and bilirubin concentrations. The infants with blood group A or B had significantly higher cord bilirubin and lower cord hemoglobin concentrations than the group O babies. Those infants whose red cells had the greatest evidence of sensitization had the highest bilirubin and lowest hemoglobin levels. The infants in whom no antibody was demonstrable on the red cells or in the red cell eluate also had significantly higher cord bilirubin and lower cord hemoglobin levels than the ABO compatible group; it is suggested that these infants had sufficient erythrocyte sensitization to produce mild hemolysis. ABO incompatibility represents a spectrum of hemolytic disease extending from those in which there is little laboratory evidence of erythrocyte sensitization, but evidence of hemolysis, to severe hemolytic disease in which erythrocyte sensitization is usually easily demonstrable.


The incidence of necrotizing enterocolitis encountered in neonates fed only refrigerated human milk was comparable to that in infants fed human milk and isotonic formula or isotonic formula alone. The infants fed human milk were significantly (P < 0.05) smaller, less mature, had lower Apgar scores, and were fed later than the formula-fed infants. The mean age of onset and time between first feeding and onset of NEC was similar among the three groups. These data indicate that refrigerated human milk was not effective in lowering the incidence of NEC. Possible explanations for the occurrence of NEC in neonates fed human milk include: (1) the introduction of a pathogen via contaminated milk; (2) inadequate maternal antigenic stimulation by the neonatal gastrointestinal flora; and (3) adverse effects of storage on cell number and function.