A rational approach to infant feeding in the management of sugar intolerance associated with infantile enteritis

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Introduction

SUGAR intolerance is a frequent complication of infective diarrhoea. Of the 328 cases of diarrhoea admitted to Paediatric Ward 4 of the University Hospital, Kuala Lumpur in 1973, there were 107 cases of sugar intolerance. The initial causes of diarrhoea in these 107 cases are summarised below.

For the correct management of this condition, a knowledge of the pathophysiology of sugar intolerance as well as the composition of various infant feeding formulae available locally is essential (Table 2).

Table 1:

AETIOLOGY	NUMBER OF CASES	
Salmonella	16	31
Enteropathogenic E. Coli	12	
Shigella	3	
No growth of pathogenic organism in stool culture		76
Total:		107

Table 2: Artificial Feeding Formulae with Low Lactose Content ('Lactose Free')

		Carbohydrate	Protein	Fat	Calories/fl.oz.
1	Sobee (Mead Johnson)	Sucrose	Soy-bean	Soy & coconut oils	20
2.	Isomil (Ross Div. of Abbot)	Sucrose	Soy-bean	Corn & coconut oils	20
3.	Nutramigen (Mead Johnson)	Surose	Casein-hydrolysate	Corn oil	20
4.	al_{110} (Nestle')	Glucose	Purified casein	Butterfat & corn oil	20

5.	Pregestimil (Mead Johnson)	Glucose	Casein-hydrolysate	Medium chain triglycerides	20
*6.	Sirolac (CF_1) (Nestle')	Carbohydrate-free	Coprecipitate of cow's milk protein	Butterfat	12

* *Sirolac (CF_1)* – available in fluid form from Nestle' (CSIRO), Australia. It is a milk-based formula made from a medium calcium coprecipitate which differs from ordinary cow's milk by the lack of the principal whey allergens, bovine serum albumin and beta-lactoglobulin. Butterfat is added to give a composition yielding 12 Cals./fl. oz.

Normal Absorption of Carbohydrates

Carbohydrate in the diet of an infant consists mainly of lactose, sucrose, glucose, starch and glycogen. The polysaccharides, starch and glycogen, are hydrolysed by pancreatic and intestinal enzymes into disaccharides. The disaccharides are then broken down to their monosaccharide components by disaccharide enzymes located in the small intestinal mucosa.

Starch, Glycogen $\xrightarrow{\text{amylase etc.}}$ Maltose, Isomaltose $\xrightarrow{\text{disaccharidases}}$ glucose

Sucrose $\xrightarrow{\text{sucrase}}$ glucose + fructose

Lactose $\xrightarrow{\text{lactase}}$ glucose + galactose

The absorption of glucose involves an active transport process. Fructose is probably absorbed by a process of facilitated passive diffusion.

Disaccharide Malabsorption

The intestinal mucosa is damaged to a varying extent in acute gastroenteritis, giardiasis, gastro-intestinal allergy and certain other rare chronic disorders of the small intestines. Varying degrees of villous atrophy occur and in serious cases the mucosa becomes more or less flat.

Disaccharidases are located in the external membrane of the epithelial cell. In the above disorders, the enzyme levels are reduced to a variable extent: lactase being least in amount is usually affected earliest followed by sucrase and maltase. Defective hydrolysis results in pooling of disaccharides in the gut lumen whereby bacterial fermentation occurs producing a lowering of pH to 4 or 5, gaseous distension and an osmotic diarrhoea. The continued use of the offending sugar leads to protracted diarrhoea with consequent malabsorption, marasmus and not infrequently death.

Detection of Sugar Intolerance

Sugar intolerance can be recognised clinically by a watery explosive diarrhoea associated with abdominal distension and perianal excoriation. The diagnosis is readily confirmed by stool testing.

The liquid stools are collected by lining the napkins with a plastic sheet. Acidity is estimated by using pH paper or 'Labstix'. Reducing sugars are detected by the addition of 2 volumes of water to 1 volume of liquid stool and thoroughly mixing. 15 drops of this suspension are transferred into a test-tube and a 'Clinitest' tablet added. The presence of more than ½% sugar indicate sugar intolerance. This test can be similarly performed using Benedict's solution but *not* with 'Clinistix'. It is important to realise that sucrose is a non-reducing sugar and will not be detected by this method unless hydrolysis of the sucrose is effected by the addition of 2 volumes of 0.1N hydrochloric acid to 1 volume of liquid stool, boiling for 1 minute allowing to cool and adding 'Clinitest' tablet to 15 drops of the mixture.

Clinical Application

Initial therapy of diarrhoea involves the correction of fluid and electrolyte imbalance by intravenous infusion or in mild cases with a clear fluid such

as 5% dextrose N/5 saline. If tolerated, ¼ strength cow's milk is introduced. If no diarrhoea occurs, the strength of the milk is gradually increased to full strength full-cream milk.

Should watery diarrhoea develop and reducing substance be found in the stools, a diagnosis of sugar intolerance is established. As this is most likely to be lactose intolerance, lactose must be excluded from the diet. A sucrose containing formula such as "Sobee", "Isomil" or "Sucrose-Nutramigen" is substituted (see Table 2). If diarrhoea ceases, lactose intolerance is confirmed and the child may be discharged on one of these partial 'lactose-free' feeding formulae.

Persistence of diarrhoea while on "Sobee", "Isomil" or "Nutramigen" suggests sucrose intolerance. A glucose-based formula such as "al_{110}" or "Pregestimil" is substituted. In total sugar intolerance, glucose is not absorbed and a carbohydrate-free preparation such as "Sirolac" is required. When using "Sirolac", a 10% dextrose drip is necessary to prevent hypoglycaemia and ketosis. Fructose followed by glucose are subsequently added to plain "Sirolac" in increments of about 1% daily until a concentration of 7.5% monosaccharide is reached whereby the mixture would yield 20 Cals./fl.oz. "Sirolac with 7.5% glucose is rather similar to "al_{110}" and hence the patient may be discharged with the latter and recalled for milk challenge after 1 month.

Intravenous alimentation is indicated in those patients with continuous diarrhoea despite the measures outline above. This procedure is complex and not without risk. It must be carried out in a centre where adequate facilities exist.

Milk Challenge

"Lactose-free" formulae are continued for approximately 1 month by which time the intestinal enzyme activity has usually returned to normal. The child is then readmitted for milk challenge. Three feeds of full-strength full-cream milk are offered and if tolerated the child is discharged on this milk. If diarrhoea associated with the presence of reducing substances in the stool appears, the previous formula is continued for another month and the milk challenge repeated. These principles are illustrated in the flow chart (Fig. (i)).

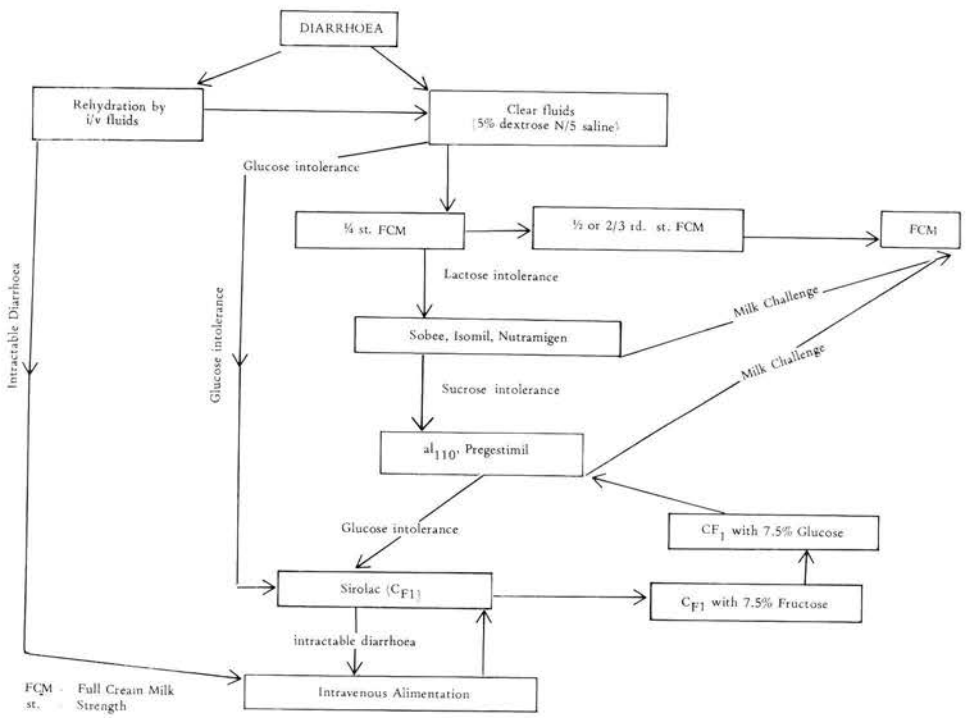

Table 3:

SUGAR INTOLERANCE IN THE FIRST MONTH OF LIFE.

Sugar Intolerance	Number of Cases
Disaccharides	13
Monosaccharides	12
Intractable diarrhoea	6
Total:	31

Results

In the 107 cases of sugar intolerance diagnosed at the University Hospital, 58 were intolerant to disaccharides (lactose and sucrose); 37 to monosaccharides (glucose and fructose) and 12 developed intractable diarrhoea (unresponsive to therapy with "Sirolac").

95% of these cases (102 patients) were below the age of one year. 31 cases occured in the first month of life (see Table 3).

The mortality rate with this regime was 13% (18 deaths). 13 of the deaths were due to profound septicaemia, 4 to intercurrent pneumonia and 1 to an Addisonian crisis. All these children were grossly debilitated on presentation to the University Hospital and most had had intractable diarrhoea for some time.

Summary

1. The incidence of sugar intolerance complicating bowel infection in 328 children seen at the University Hospital, Kuala Lumpur from January to December 1973 was 32.6%.
2. The pathophysiology of sugar intolerance is presented.
3. The diagnosis and management of sugar intolerance are discussed.

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