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Lactose Loading Test on Protein Calorie Malnutrition

by

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Introduction

Diarrhoea is a common feature of severe cases of malnutrition and usually pathogenic agents can not be isolated. Abnormal intestinal motility is not a primary feature of the diarrhoea in malnourished children (James, 1968). Sugar intolerance is considered a common cause of diarrhoea in children with kwashiorkor admitted to hospital in Kampala (Wharton et al., 1968). Dean (1957) originally suggested that the lactose of milk was the cause of severe diarrhoea encountered in cases of advanced kwashiorkor.

Lactose intolerance is almost invariably the consequence of lactase-activity, which is either strongly decreased or absent (Luyken, 1971). James (1968) suggests that the disaccharide intolerance in malnutrition is a failure in hydrolytic activity rather than a failure to absorb the monosaccharide products of hydrolysis.

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Various investigations on lactose intolerance in various races living in different parts of the world have been carried out recently. The figures show high prevalence in developing countries and among colored races (Huang and Bayless, 1967; Alzate et al., 1969; Bayless and Christopher, 1969; Luyken, 1971; Sutedjo et al., 1972; Suharjono et al., 1971; Paige et al., 1972; Erkelens et al., 1972). In the groups examined the percentage of "lactose intolerants" varies from 50% to 100%.

Lactose intolerance in kwashiorkor children was reported by several authors (Bowie et al., 1965; Chandra et al., 1969).

The purpose of the present study is to report a prevalence survey of lactose intolerance in Indonesian children with Protein Calorie Malnutrition (PCM).

Material and methods

Twenty-two children with PCM were examined for the lactose load-

ing test. The diagnosis of PCM was made on the basis of their weight being far below the local standard for normal children and other physical signs such as loss of subcutaneous fat, sparse hair with a reddish color and easily pulled out, anaemia, hepatomegaly, pigmentary skin changes and avitaminosis A in varying degrees.

Lactose loading test was performed according to the method of Hagedorn-Jensen. Lactose 2 gm/kg body weight as a 10% suspension in water was given after 7 to 9 hours of fasting. One-tenth ml of capillary blood was obtained respectively in the fasting state and 30, 60, 90 and 120 minutes after lactose loading.

The maximum rise in blood-glucose above the fasting value during the 120 minutes test period was measured; a rise of 25 mg per 100 ml or more was considered normal. If the increase was less than 25 mg per 100 ml, lactose intolerance was diagnosed.

In order to find out personal histories of milk intolerance a questionnaire was put forward with the following questions:

1. Does the child drink cow's milk or any other milk (full cream, skim, condensed milk etc) every day ?
2. If true, is it since after weaning?
3. If not, since what age?
4. (a) Did the child experience any abdominal discomfort when starting drinking milk?

   (b) If there is abdominal discomfort after drinking milk, at what age did it start?
5. When the child now occasionally drinks milk or milk containing food such as ice cream, does it get abdominal discomfort?
6. What kind of discomfort ? Nausea, vomiting, meteorism, flatulence or diarrhoea.

Results

The children ranged in age from 15 months to 4 years; their body weight varied from 5000 gm to 10 600 gm.

After lactose ingestion the children showed peak blood sugar increases ranging from 4 mg% to 47 mg% (mean 17,9 mg%) over fasting levels.

Peaks were observed at 60 minutes in 20 children (90,9%) and at 90 minutes in 2 of them (9,1%). Nineteen children (86,4%) showed flat blood glucose curves. Fig. 1 shows the flat blood glucose curve of a girl, 15 months old with a body weight of 6900 gm and a rise of only 4 mg% over fasting level.

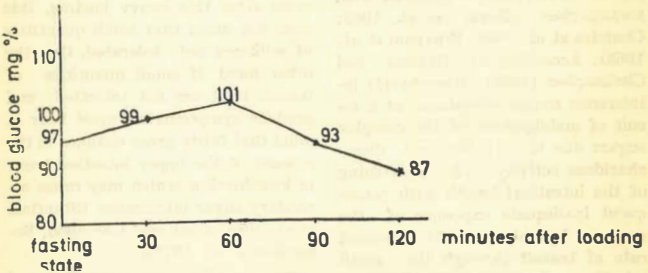

Fig. 1: *Lactose loading test on a patient with PCM; SR, female, 15 months, BW: 6900 grams. Rise of 4 mg% over fasting level at 60 minutes after ingestion of lactose.*

Almost all children came from families with low socio economic status; most of them were born at home and their parents did not know the birth weight. The answers of the questionnaire revealed that more than half of the children had personal histories of milk intolerance.

Twelve of them (54,5%) did not drink milk after weaning, whereas four consumed milk occasionally. Of the 6 children who continued drinking milk, three showed negative results and the remaining three had positive results with a blood glucose rise of 23 — 24 mg% above fasting levels.

Two of the three patients with the negative results had a rise of just 26 mg% over fasting level which is only 1 mg% above the criterion used for a positive test.

Symptoms provoked by the ingestion of the lactose load such as abdominal discomfort, flatulence and diarrhoea were noted in all patients during the test.

Discussion

Nineteen (86,4%) out of the twenty-two children tested showed flat blood sugar curves. This supports previous findings of other authors who also found high prevalence of lactose intolerance in children with kwashiorkor (Bowie et al., 1965; Chandra et al., 1969; Wharton et al., 1968). According to Bayless and Christopher (1969), disaccharide intolerance causes symptoms as a result of maldigestion of the complex sugars due to: (1) deficient disaccharidase activity; (2) shortening of the intestinal length with consequent inadequate exposure of the enzyme hydrolysis; (3) increased rate of transit through the small intestine as in some postgastrectomy situations or (4) an overwhelming of the digestive ability to the intestinal mucosa with a very large disaccharide load. One of the causes of disaccharide-splitting enzyme deficiency is damage to the intestinal mucosa due to gastroenteritis or for example coeliac disease (Bayless, 1971).

In our series a child of 10 kg for example was loaded with 20 grams of lactose which was equal to the quantity in 500 ml of milk.

Milk is still categorized as "expensive/luxury food" in developing countries such as Indonesia.

More than half of our patients (54,5%) did not consume milk after weaning. Some drink milk occasionally (once in several days) and those who continued drinking milk after weaning consumed one to two glasses a day which is about 200 to 400 ml, not in one portion but spread over the day. Therefore if complaints arise after this heavy loading, this does not mean that small quantities of milk are not tolerated. On the other hand, if small quantities of lactose/milk are not tolerated and produce symptoms we must bear in mind that fairly gross changes in the mucosa of the upper intestine occur in kwashiorkor which may cause secondary sugar intolerance (Stanfield et al., 1965; Cook and Lee, 1966; Suharjono et al., 1971).

Dean and Whitehead (1965) found that the concentration of lactose was considerably higher at all levels of the intestine than the monosaccharide glucose and galactose, and that this was associated with the flat blood sugar absorption of lactose for which a deficiency in mucosal lactase might account.

Despite rapid clinical improvement and in most cases complete biochemical recovery, the mucosal changes persist, and within the period of a follow up one year later are still notable (Stanfield et al., 1965).

Dean and Whitehead (1965) found that the disaccharide absorption was as abnormal three to for weeks after a fairly complete recovery, as it was at the start of treatment.

We were able to repeat the test on three children two months later, who at that time showed sufficient nutritional recovery and had gained weight to normal local standard, but the results were the same e.g. the blood sugar rose less than 20 mg% above fasting levels. This agrees with the findings of Chandra et al. (1968) who suggests consideration of a primary enzymic defect with consequent diarrhoea and secondary malnutrition.

Holzel et al. (1959) were probably the first authors to consider this condition as a failure to hydrolyse lactose due to an actual deficiency of lactase or to inhibition (or lack of activation) of the enzyme; the presence of this enzyme defect in two siblings of one family suggests that the condition is hereditary.

Cook and Kajubi (1966) suggest this condition to be hereditary, because in their series there were some children with a lone lactose tolerance or with a residual lactose intolerance.

Most of the kwashiorkor patients in Indonesia were brought to the doctor with the chief complaints of anorexia and chronic diarrhoea.

Sunoto et al. (1971) found a 40% incidence of lactose intolerance among Indonesian children with chronic diarrhoea.

The mechanism through which disaccharide intolerance produces diarrhoea is twofold. Unabsorbed sugars act as an osmotic-load and causes outpouring of fluid into the small intestine. Unabsorbed sugars are also acted upon by colonic bacteria resulting in the production of irritant low-molecular-weight organic acids. These two factors increase peristaltic activity and reduce transit time (Chandra et al., 1968; Bayless and Christopher, 1969). The continued presence of unsplit sugars in the gut may damage the epithelial cells further (Holzel et al., 1962), perpetuating and aggravating the enzyme deficiency (Chandra et al., 1968).

High lactose containing milk is certainly not indicated in the treatment of PCM.

At the Dr. Tjipto Mangunkusumo General Hospital in Jakarta a trial is underway, treating PCM on a low lactose diet. "Eitwit melk", a self-prepared milk is used containing only 1,4% lactose. Final results are not available yet, but the impression is that the diarrhoea stops immediately and the increase in body weight is satisfactory.

Summary

Lactose loading test was performed on twenty-two Indonesian children with Protein Calorie Malnutrition. Nineteen (86,4%) out of them showed flat blood glucose curves revealing definite lactose intolerance.

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