ALIMENTARY TRACT AND PANCREAS

Lactose malabsorption in Central Australian Aboriginal children hospitalized with acute enteritis

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Abstract The prevalence and duration of lactose malabsorption was studied in 64 Central Australian Aboriginal children requiring hospitalization for acute enteritis. Lactose malabsorption was determined by the breath-hydrogen test. Sixty-nine per cent were shown to malabsorb lactose, a figure similar to that for well nourished urban European children, hospitalized for acute enteritis. Whereas 90 per cent of the European children became lactose-tolerant 1 month later, only three of 32 aboriginal children were lactose-tolerant after 3 months. Lactose malabsorption was also associated with more frequent hospitalizations in affected children.

Coronavirus-like particles were the commonest agent isolated from Aboriginal children. The majority of the Aboriginal children were malnourished (< 80% standard weight for age) which may have been contributed to by their lactose malabsorption and explain why this failed to recover in the same way as European children. The possibility of lactose malabsorption must be taken into consideration when managing Aboriginal children with acute diarrhoeal disease and undernutrition. The use of the breath-hydrogen test allows an accurate diagnosis of lactose malabsorption to be made in Aboriginal children with diarrhoea so that appropriate dietary management can be instituted.

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INTRODUCTION

Poor growth in the toddler age group is a well recognized problem in Australian Aboriginal children.\textsuperscript{1,2} Contributing factors include an unsatisfactory diet following weaning, and recurrent infections (predominantly gastrointestinal and respiratory) with problems of housing, hygiene and sanitation being further contributing factors. Lactose malabsorption (LM) and lactose intolerance (LI) have been shown to be a common problem in malnourished children.\textsuperscript{3} LM signifies unhydrolysed lactose reaching the colon. If this results in gastrointestinal symptoms such as colic, abdominal pain, abdominal distention or diarrhoea the patient is diagnosed as having LI.

In healthy adult Aboriginals, an incidence of 84% LM and 64% LI has recently been demonstrated,\textsuperscript{4} and these appear as early as 6 years of age in 70% of healthy Aboriginal children.\textsuperscript{5} Very little information is available on the incidence of LM in Aboriginal children with acute enteritis and the duration of postenteritis LM in Aboriginal children has not been studied. With the development of the breath-hydrogen test (BHT) — a simple sensitive specific and non-invasive test for LM — it is now possible to look at this problem. It was shown previously, using the BHT, that 50% of urban European children hospitalized with acute enteritis develop LM.\textsuperscript{6} In order to determine the prevalence and duration of LM in hospitalized Aboriginal infants and children with acute enteritis, BHT was performed following an oral lactose load. These results were then compared with findings from an earlier study in well nourished urban European children.\textsuperscript{6} Accurate assessment of clinically significant LM may improve the dietary management of sick infants with diarrhoea, and hence improve their nutritional status.

METHODS

One hundred and twenty-five BHT were performed on 64 full-blood Aboriginal children, who were admitted with acute enteritis to the infectious diseases ward of the Alice Springs Hospital, Alice Springs, Northern Territory between 1 March and 8 June, 1983. Due to language limitations acute enteritis was defined as an acute illness lasting less than 10 days with diarrhoea; often fever and vomiting presented as such by the mother and for which no other cause was found. Only patients admitted between Sunday and Thursday were studied so the BHT samples could be transported to Adelaide on the day of collection.

Clinical and anthropometric data are outlined in Table 1. Thirty-four patients (53%) had a past history of gastroenteritis and 44 (69%) had a previous illness requiring hospitalization. Forty-eight (75%) of the children had been given antibiotics for their current illness, mainly for associated respiratory symptoms. In eight children (12%) a urinary tract infection was diagnosed after hospitalization, seven children (11%) had acute otitis media and 26 (41%) evidence of chronic otitis media. Seventeen children (27%) showed no clinical evidence of dehydration, 45 (70%) were 5–10% dehydrated and two (3%) had lost more than 10% of their body weight. Forty-nine patients (77%) required intravenous therapy either for rehydration or failure to tolerate oral fluids initially.

Patients were weighed using beam balance scales standardized by using 3 kg and 5 kg standard weights. Standard weight-for-age (SWFA) and weight-for-length (SWFL) were calculated using the Harvard standards. The mean age of the patients was 15 months and all were below 90% SWFA; 60% were less than 80% SWFA and 34% were below 70% SWFA. Twenty-seven patients (42%) were
Table 1  Patient history in 64 Aboriginal children

|                          | Total | LM | Lactose absorbers |
|--------------------------|-------|----|-------------------|
| Number                   | 64    | 44 | 20                |
| Sex                      |       |    |                   |
| Male                     | 36    | 27 | 9                 |
| Female                   | 28    | 17 | 11                |
| Birth weight (kg)        | 2.9 ± 0.5 | 2.82 ± 0.6 | 2.94 ± 0.5 |
| SWFA (%)                 | 78 ± 12 | 76 ± 10 | 80 ± 12 |
| SWFL (%)                 | 94 ± 15 | 92 ± 14 | 98 ± 15 |
| Number previous admissions | 3.2 ± 3.3 | 1.7 ± 1.8* |
| Number of admissions since diagnosis* | 2.1 ± 1.9 | 0.8 ± 1.1† |
| Duration of symptoms prior to admission (days) | 6.0 ± 6.8 |
| Duration hospital stay (days) | 23.7 ± 18.2 | 24.9 ± 20.9 | 21.1 ± 12.9 |

Results are given as mean and s.d. where appropriate.
*In the 12 months following completion of the study.
Significance compared with LM patients: †P < 0.05, ‡P < 0.01.

below 90% SWFL.

Stool specimens were obtained within 48 h of admission and examined by light microscopy for parasites, cysts and ova, cultured for enteric pathogens (Salmonella spp., Shigella spp., E.coli spp.) and examined by electron microscopy (EM) for viral pathogens (rotavirus, adenovirus, coronavirus-like particles and small round viruses). ELISA testing for rotavirus was also carried out. The pathogenicity of E.coli strains was determined by serotyping. Enterotoxigenic strains were not looked for.

A lactose BHT was performed on all children on the day after admission, following rehydration. The BHT was performed as previously described after oral administration of a physiologic lactose challenge (2 g/kg, maximum 15 g/100 ml water). A sustained hydrogen (H₂) rise above baseline of greater than 10 parts/10⁶ in expired air constituted a positive test. The breath sampling was carried out using air-tight syringes by GE in the Alice Springs Hospital at 0, 20, 40, 60, 90, 120, 150, 180 min and chilled samples were air freighted to the Adelaide Children's Hospital, Gastroenterology Unit and analysed within 24 h of collection. It was previously shown that less than 5% of the H₂ is lost in 24 h. If the lactose BHT was negative, lactulose (6.68 g) was used on another day to assess whether the children were capable of H₂ production, thus ruling out false negative tests. Breath samples were collected as previously described and a sustained H₂ rise of > 10 parts/10⁶ above baseline constituted a positive result, thus distinguishing between a H₂ producer and one who does not produce H₂.

Children who were not H₂ producers were not included in the study. Due to sample quality normalization, bacterial overgrowth was indicated by the presence of two distinct H₂ peaks from unabsorbed lactulose. The children were followed at monthly intervals until the lactose BHT was normal and they were tolerating a normal diet; however, this was not possible for some cases.

Statistical evaluations were made using the Student's t-test.

RESULTS

The results of the BHT for Aboriginal children are shown in Table 2. The European children included in the table were obtained from an earlier study and are included for
Table 2  Comparison of LM in hospitalized Aboriginal and European children with acute enteritis at different ages

| Age (months) | European Malabsorbers | Aboriginal Malabsorbers |
|--------------|------------------------|-------------------------|
|              | n | % | n | % |
| <6           | 28 | 6 | 21 | 8 | 5 | 60 |
| 6-11         | 31 | 16 | 52 | 26 | 17 | 65 |
| 12-24        | 32 | 21 | 66 | 26 | 19 | 73 |
| >24          | 13 | 9 | 69 | 4 | 3 | 75 |
| Total        | 104 | 52 | 50 | 64 | 44 | 69 |

comparison. 
Forty-four (69%) of the Aboriginal children showed evidence of LM, which was common at all ages studied, varying from 60% in children less than 6 months to 75% in the children older than 2 years.

The duration of LM was assessed by repeat BHT in 32 of the 44 patients on a lactose-restricted diet. Follow-up BHT were able to be carried out in this group because they were either still in hospital or in a nearby child care facility for nutritional rehabilitation. Some had been re-admitted with another illness. Eight were still malabsorbing lactose at 1 month, 9 at 2 months and 12 at 3 months. Only 10% (3) of the children were shown to become lactose-absorbers during the 3 month period of follow-up.

Three children had an early and transient rise and fall of \( H_2 \) after an oral lactose load suggestive of bacterial colonization of the small intestine, followed by a second colonic peak suggestive of LM. This was confirmed with a subsequent lactulose BHT. It was not possible to take jejunal juice for culture from these children, however, previous experience suggests that this would not have been helpful. Full symptomatic recovery occurred after addition of appropriate antibiotic therapy. No patient with clinical LI had a normal lactose BHT. Four infants (6%) were not \( H_2 \) producers.

Micro-organisms were isolated from 75% of Aboriginal patients with LM and 40% of lactose-absorbers (Table 3). The coronavirus-like particles were the commonest agent isolated. Bacterial pathogens were isolated from 40% of patients with LM compared with 15% in the lactose-tolerant group. There was good correlation between EM and ELISA for the diagnosis of rotavirus. Eight patients were shown to have rotavirus by EM and this was confirmed by ELISA. There were no EM-positive ELISA-negative results or vice versa.

DISCUSSION

This study has demonstrated that 69% of Central Australian Aboriginal children hospi-

Table 3  Comparison of results of stool cultures and stool EM in 104 European children and 64 Aboriginal children with acute enteritis

| Pathogen                     | Lactose malabsorbers | Lactose absorbers |
|------------------------------|-----------------------|-------------------|
|                              | European | Aboriginal* | European | Aboriginal |
| Rotavirus                    | 24       | 6           | 1        | 2          |
| Coronavirus-like             | —        | 19          | —        | 6          |
| Adenovirus                   | —        | 1           | 2        | —          |
| Small round viruses          | 1        | 3           | 1        | 1          |
| Salmonella spp.              | —        | 6           | 2        | 1          |
| Shigella spp.                | —        | 3           | —        | 1          |
| Campylobacter spp.           | —        | NT          | 1        | NT         |
| Enteropathogenic E.coli      | 3        | 2           | 1        | 1          |
| Parasites                    | 1        | 1           | —        | 1          |
| No pathogens isolated        | 23       | 11          | 28       | 9          |

*More than one pathogen in nine children NT not tested.
talized with acute enteritis had LM. This compared with figures of 80% LM in healthy adult Aborigines,64% of whom also had LI, and an incidence of 70% LM in healthy Aboriginal children over the age of 6 years.5 Primary acquired (adult onset, racial) lactase deficiency is the normal state in most non-Caucasian populations after weaning.11 The onset usually occurs after 3 years of age and affects the majority of individuals by 10–15 years of age.12 Secondary lactase deficiency, which may be expected to be reversible, most commonly occurs as a result of mucosal injury.

The majority of the children were suffering from malnutrition (<80% SWFA) which is almost certainly a result of inadequate intake and recurrent infections. The role of LM in this cycle is not defined, but clinical experience in Adelaide and Alice Springs has shown it could aggravate the malnutrition. In this Aboriginal population it is possible that mucosal injury secondary to infection and malnutrition has precipitated the early onset of LM. Only three of 32 infants were lactose-tolerant after 3 months which is further support for this hypothesis. It would seem likely that in a population such as this, predisposed to adult onset LM, mucosal injury and malnutrition early in life may move the time frame downwards by several years.13 In other populations and in studies using an animal model, iron deficiency, protein deficiency and parasite infestation have been shown to be important in the early genesis of lactose malabsorption.14,15 European children, however, who are not malnourished, with a similar incidence of LM following acute enteritis (Table 2), recover very quickly with 90% being lactose-tolerant 1 month after the acute illness.6

During the period of this study, there was not a rotavirus epidemic. The commonest agent isolated from Aboriginal children was the coronavirus-like particle. The pathogenicity of this agent has yet to be determined. However, its role as a causal agent of diarrhoea seems doubtful as it is seen almost as frequently in children without diarrhoea.9,10

Evidence from earlier studies suggests that Aboriginal children with acute diarrhoea and poor growth recover more quickly and gain weight faster if lactose is restricted.16 As can be seen by the mean age of the patients (15 months), LM was severe only in the age group taking transition foods and not in the fully breast-fed infant. In the past, the inability to accurately diagnose the cause of continuing diarrhoea and poor weight gain has led to multiple dietary changes and prolonged hospitalization in the hope that extended adequate nutrition would settle their diarrhoeal illness. Previous workers have shown a lack of correlation between simple tests such as the Clinitest and LM.17,18 However, using the BHT, an accurate diagnosis of LM can be made quickly and distinguished from other intercurrent illness so that appropriate dietary management can be confidently instituted both in hospital and at home. Despite the use of antibiotics prior to admission only 6% of infants were not H₂ producers which is similar to European children. LM may also be a factor in the frequent re-admissions. In the population studied, children with LM had significantly more hospitalizations both before and after the diagnosis of LM (Table 1). Five per cent of the children had evidence of bacterial overgrowth diagnosed by BHT, using previously reported criteria.8 The diagnosis would have been overlooked but for the use of the BHT. Jejunal cultures were not carried out for ethical reasons, but previous experience shows that it would probably not have helped in the diagnosis.8 All children responded to appropriate antibiotic therapy.

The management of LM in Central Australia presents many difficulties. Most of the children live in small isolated communities up to 500 km from Alice Springs, have limited water supplies, and poor sanitation and cooking facilities. On the settlements, the food for children comes mainly from the settlement store with some supplementation from traditional sources (bush tucker). In order to try and manage these children in
their own environment, a list of lactose-free or low-lactose foods and lactose-rich foods was prepared using foods commonly available in settlement shops (Table 4). The mother was instructed about the best food to use and this was reinforced through district medical officers, rural health sisters and community health workers.

The contribution of LM to diarrhoeal disease and malnutrition in Aboriginal children in Central Australia is unknown. The present study suggests it is a common and persistent problem following acute enteritis, it is associated with increased hospitalization and needs to be considered in the management of the diarrhoeal disease syndrome in these children. Further extended studies are needed to assess the importance of LM in contributing to chronic diarrhoea and malnutrition in Aboriginal children. Assessment of the ability of Aboriginal families to manage dietary modification in the home environment may be appropriate.

Table 4 List of low (good) and high lactose-containing (bad) foods

| Good foods                     | Bad foods                  |
|--------------------------------|----------------------------|
| Bush tucker                    | Cow's milk: carton         |
| Digestelact or Delact          | Sunshine, Diploma          |
| Pregestimil                    | evaporated condensed       |
| Soft meat and stews            |                            |
| Chicken                        |                            |
| Eggs                           | Soft cheese, cheese sticks |
| Damper made without cow's milk but with water or Digestelact | Milk biscuits |
| Fresh fruit                    | Sweet biscuits             |
| Vegetables: fresh, tinned, frozen | Ice cream                 |
| Bread: especially wholemeal but not milk | Rice cream               |
| Plain biscuits, like Sao       | Custard                    |
| Porridge, Weetbix, baby rice   | Milk bread                 |
| Hard cheese (e.g. Coon, Cracker Barrel) | Dried potato from store |

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