CARBOHYDRATE MALABSORPTION IN ACUTE DIARRHEA

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ABSTRACT

A group of 245 well nourished infants with acute diarrhea were screened for carbohydrate malabsorption by evaluating stool pH and reducing substances in the stools. Carbohydrate malabsorption was diagnosed in 28 cases (11%). Clinical features of carbohydrate intolerance were present in only one case. The duration of diarrhea after admission ranged from 1 to 13 days (mean 3.9 days).

An oral lactose tolerance test was consistent with lactose deficiency in 32% of all cases. Thin layer chromatography showed many carbohydrates including monosaccharides in the stools, indicating that the defect in intestinal absorption was not specific for lactose.

Key words: Acute gastroenteritis, Carbohydrate malabsorption, Oral lactose tolerance test, Chromatography.

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Received for publication: January 13, 1993; Accepted: March 9, 1994 The term malabsorption refers to a disease state characterized by defective intestinal absorption of ingested carbohydrates, while carbohydrate intolerance indicates the presence of clinical symptoms that may accompany carbohydrate malabsorption. These symptoms include abdominal distension, flatulence, abdominal cramps, and watery diarrhea(1).

Disorders of carbohydrate absorption are integrally linked with defective activity of the carbohydrate digesting enzymes of the intestinal mucosa which can be a primary disorder, as occurs in congenital deficiency states. Secondary disorders of carbohydrate malabsorption, celiac disease, iron deficiency anemia and many other conditions may be associated with secondary disorders of carbohydrate malabsorption(2). If mucosal damage is severe, more than one digesting enzyme can be affected.

Carbohydrate malabsorption (CM) has been reported to be a frequent complication of diarrheal disease in young infants, leading to a worsening and/or prolongation of the diarrheal state(3-7). In recent years, a significant decrease has been noted in the incidence and severity of CM developing after gastroenteritis. Only 1% of CM cases are reported to require a special diet(8). The aim of this study was to assess the frequency and type of CM in a group of infants presenting with acute diarrhea.

Material and Methods

The study included 245 infants between 3-12 months of age who were randomly selected from patients presenting with acute diarrhea to the hospital. Informed consent was obtained in all cases. Acute diarrhea was defined as 3 or more abundant watery stools per day, of less than 14 days duration(5,6). Initially, all these cases were screened for CM by determination of stool pH and reducing substance using Universal Indicator Paper (MERCK, Art. 9526) and clinitest tablets (AMES, Art. 2107). Kelly and Anderson's method and criteria were used in the detection of reducing substance in the stools(9). According to this method, values under 0.25% are negative, values between 0.25% and 0.50% indicate suspect cases and those over 0.50% are accepted as increased. Of 245 infants, 28 had a low fecal pH (<6) and increased (>0.5%) reducing substance in the stools and were diagnosed as having carbohydrate malabsorption(5,8,10). These cases were further investigated by oral lactose tolerance test (OLTT). In addition, thin layer chromatography (TLC) and microscopy for fat (Sudan 3), parasites (Lugol) and leukocytes (methylene blue staining) were performed in the stools. Total blood counts, leukocyte differentials, urine analysis, erthyrocyte sedimentation rate, stool cultures, and blood chemistry including urea, creatinine, sodium potassium, total protein, albumin/globulin ratios were also performed in all cases. OLTT was carried out on the day of admission by oral administration of 2 g/kg of lactose in the form of a 10% solution in water. Blood glucose was estimated on capillary samples initially and 30, 60, 90, 120 min after lactose administration. An increase of greater than 20 mg/dl in blood glucose over the initial value was considered as normal. TLC was performed on the first stools after admission and on the first stools after the administration of lactose. The method of Menzies and Seakins was used for the analysis(11).

Results

Of the 245 cases with acute diarrhea, CM was detected in 28 (11%). Only one of these infants (0.04%) showed symptoms of carbohydrate intolerance. The clinical findings in these 28 cases are shown in Table I. None of these infants had a history of chronic disease and none showed signs of protein energy malnutrition. In 26 of these cases (93%) vomiting and in 16 (57%) fever accompanied the diarrhea. Two patients (7%) were severely dehydrated. Examination of the stools did not reveal blood, mucus, fat or leukocytes in any of the cases. Blood chemistries were normal. The stools were negative for giardia and cultures were negative for Salmonella and Shigella. Rotavirus antigen could be assessed in only 9 of the 28 cases. Nine cases (32%) had an increase of less than 20 mg/dl in blood glucose following OLTT. Thin layer chromatography of the first stools after admission showed that glucose and lactose were present in 18 infants. Fructose and sucrose were present in 8 cases. Only 4 cases (14%) had one type of carbohydrate in the stools (glucose in two

TABLE I-Clinical Findings in 28 CM Cases

7.0	± 2.7 (mo)
7.45	±1.36 (kg)
3.9	± 2.9 days
9	(32%)
17	(61%)
2	(7%)
6	(21%)
17	(61%)
5	(18%)
ls (%)	
7	(25%)
21	(75%)
	7.0 7.45 3.9 9 17 2 6 17 5 ls (%) 7 21

cases, sucrose and lactose in one case each). More than one type of carbohydrate was found in the stools of the remaining cases two types in 16, three types in 7 and four different carbohydrates in 1 case). TLC examination of the stools following lactose loading revealed that stool glucose quantity had not changed from admission stools but the proportion of cases excreting lactose in the stools had increased from 64% to 92%. Similar to the results on admission, TLC demonstrated the presence of more than one type of carbohydrate in the stools in the majority of these infants.

Discussion

The frequency of CM was 11% in our series and comparable to that reported in another study on 200 children with acute gastroenteritis(7). In accordance with previous studies, our findings indicate the presence of glucose, galactose and fructose in the stools in transient disaccharide absorption disorders. In the absence of disaccharide hydrolysis defects, as also reported by others(12) OLTT results were consistent with disaccharidase deficiency only in one third of our cases indicating that CM can also occur. The pathogenesis of this finding is not clear, but may be related to a functional disturbance of the gut in acute diarrhea such as increased motility.

TLC, when done in conjunction with fecal pH determination and clinitest tablet assay methods, is suggested as a useful method in confirming and supplementing the results of these tests(11). Excretion of more than one carbohydrate in the stool has also been reported. In another study, following levulose challenge, both galactose and fructose were detected in the liquid stools of healthy volunteers receiving ampicillin(12). TLC results in our series indicate the presence of more than one carbohydrate in the majority of diarrheic infants with CM.

Rotavirus infection is known to be the most frequent condition leading to CM(7,13). In 1981, Hyams et al. reported that lactose intolerance develops in 50% of cases with rotavirus infection(14). In our study, out of the 9 CM cases who were able to be tested for rotavirus antigen, 7 were positive.

To conclude, our findings show that CM in acute diarrhea is not very rare, that it can develop within the first week of the diarrheal episode, is a self-limited condition and involves defective absorption of more than one type of carbohydrate. The clinical entity defined as carbohydrate intolerance is a much rarer condition. These results lead us to recommend that the great majority of infants with acute diarrhea can continue receiving a normal diet during the diarrheic episode provided they do not ingest high amounts, of unabsorbable carbohydrates.

REFERENCES

- Kneepkens F. Carbohydrate absorption in children. Amsterdam, Free University Press 1988, p 17.
- Shulman RJ. Enzyme and transport defects. In: Pediatrics. Philadelphia, J13 Lippincott Company, Ed Oski FA, 1990 pp 1734-1737.
- Bartrop RW, Hull D. Transient lactose in tolerance in infancy. Arch Dis Child 1986, 61:716-720.
- Gracey M, Burke V. Sugar-induced diarrhea in children. Arch Dis Child 1973, 48: 331-336.
- Kulmar V, Chandrasekaran R, Bhaskar R. Carbohydrate intolerance associated with acute gastroenteritis. Clin Pediatr 1977, 16: 1123-1127.

KARABOCUOGLU ETAL.

- Lynkaran N, Yadav M, Looi LM, et al. Effect of soy protein on the small bowel mucosa of young infants recovering from acute gastroenteritis. J Pediatr Gastroenterol Nutr 1988, 7: 68-75.
- Trounce JQ, Walker-Smith JA. Sugar in tolerance complicating acute gastroenteritis. Arch Dis Child 1985, 60: 986-990.
- 8. Editorial. What has happened to carbohydrate intolerance following gastroenteritis. Lancet 1987, 3 23-24.
- 9. Kelly KR, Anderson CM. A ward test for sugar in faeces. Lancet 1964, 2: 981-989.
- Forget P, Lombet J, Frandsfil C, et al. Lactase insufficiency revised. J Pediatr Gastroenterol Nutr 1985, 4: 868-872.

CARBOHYDRATE MALABSORPTION IN DIARRHEA

- Mensies IS, Seakins JWT. Chromatographic and electrophoretic techniques. In: Paper and Thin Layer Chromatography, 4th edn. Ed Mensies IS. Chicago, William Heinemann Medical Books, 1976, pp 183-216.
- Rao SSC, Edwards CA, Austen CJ, et al. Impaired colonic fermentation of carbohydrate after ampicillin. Gastroenterology 1988, 94: 928-932.
- Richard HJ. Viral enteritis. Pediatr Clin North Am 1988, 35: 89-103.
- Hyams JS, Krause PJ, Gleason PA. Lactose malabsorption following rotavirus infection in young children. J Pediatr 1981, 99: 916-920.