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January 1993

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Recommended Citation

Molla, A., Khurshid, M., Molla, A., Khatun, M. (1993). Malabsorption of nutrients in children with diarrhoea due to unknown aetiologies. *Journal of Pakistan Medical Association*, 43, 49.

Available at: http://ecommons.aku.edu/pakistan_fhs_mc_pathol_microbiol/585

MALABSORPTION OF NUTRIENTS IN CHILDREN WITH DIARRHOEA DUE TO UNKNOWN AETIOLOGIES

Pages with reference to book, From 49 To 51

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ABSTRACT

Intake and coefficient of absorption of nutrients were measured in 72 children during acute diarrhoea and 2 weeks after recovery. No diarrhoeal pathogens could be identified in 18 (25%) children (group I). Aetiology of diarrhoea was identified in most of the 54 children (group II). Absorption of calories, fat and carbohydrate during the recovery stage were similar in all 72 children. In group I, absorption of nutrients improved from acute stage to recovery stage as follows: fat from 62 to 85%, calories from 68 to 86% and carbohydrate from 81 to 91%. Absorption of nitrogen during the acute stage was significantly lower ($P < 0.01$) in group I (mean \pm SD: $2\% \pm 56$) than in group II (mean \pm SD: $49\% \pm 28$). The anion gap in the stool of group I children (32) was similar to those with shigellae (37) and rotavirus (38). This could partially be explained by the possible loss of anionic proteins, fatty acids and/or lactic acids in the diarrhoeal stool. Results suggest that the diarrhoea due to unknown aetiology is possibly of the invasive type. Further investigation is necessary to define the mechanism of nitrogen loss in acute diarrhoea of unknown aetiology (JPMA 43: 49, 1993).

INTRODUCTION

Nutritional consequences of diarrhoea are influenced by several factors, of which malabsorption is an important one. Quantitative absorption of nitrogen and fat in infants with diarrhoea was studied as early as 1948 by Chung and co-workers^{1,2}. He showed that absorption of nitrogen and fat increased with increase in intake. Reduced absorption of fat was obtained in a study of 10 children aged 1.5-7 months, recovering from acute infectious gastroenteritis by Jonaset al³. Ileal dysfunction and bile acid loss were thought to be the causes of impaired fat absorption. Mann et al⁴ reported that infants suffering from chronic diarrhoea due to unknown causes lose larger amounts of nitrogen (¹⁵N-yeast protein) and fat in the stool. ¹⁵N excretion was found to be related to the severity of diarrhoeal attack. Shedding of the gastrointestinal mucosa, endogenous secretions in the intestine and plasma proteins were the sources of this nitrogen loss. From 1980 to 1986, we studied the intake and absorption of nutrients during acute stage of diarrhoea in 72 children aged under five. Results obtained from these children were published previously⁵⁻⁷. The objective of the present study was to compare the intake and absorption of nutrients obtained from group I children to those of group II, in order to estimate the nutritional consequences of diarrhoea due to unknown aetiology.

PATIENTS AND METHODS

The study was carried out during 1980 to 1986 at the International Centre for Diarrhoeal Disease Research (ICDDR), B. hospital with acute diarrhoea (mean duration 23 hours) and children with moderate to severe dehydration were selected for the study. Cases with pneumonia or any other systemic illness requiring antibiotic treatment were not included. The nutritional status of the selected children varied from mild to moderate degree of malnutrition (wt/ht% median, 71-80%). The study was

approved by the Ethical Review Committee of ICDDR, B hospital. Informed consent was obtained from the parents or the guardians of the children. On admission, a thorough physical examination was done; blood was tested for complete blood count and electrolytes. Following stool tests were performed: microscopic examination, culture in SS and MacConkey's plates, ELISA (enzyme linked immunosorbent assay) for rotavirus⁸ and wet smears of fresh stools for identifying the presence of parasites. No special test was done for strongyloides, cryptosporidium, enteropathogenic E. coli (EPEC) or campylobacter. For comparison of stool electrolytes, catheter specimens of stool from group I and group II children were collected on admission. Initial dehydration was corrected within 4-6 hours of admission and thereafter maintained with i/v fluid (composition: Na⁺ 133 mmol/L; C 13 mmol/L; Cl 99 mmol/L; HCO₃ (as acetate) 48 mmol/L). First balance study was initiated after correction of initial dehydration with the feeding of marker 1 (2 charcoal tablets each weighing 5 gm), followed by a meal of known composition⁹. Marker II was fed 72 hours later and meanwhile study children were fed ad libitum six times a day. MI stool, vomitus and urine were collected starting from the appearance of the first marker until the appearance of the second marker in stool. Two weeks after discharge from the hospital, a second balance study was performed, after which children were treated for parasites or other ailments as detected by the tests. Samples from the aliquotes of food, urine, stool and vomitus collected during the balance studies were analyzed for nitrogen, fat, carbohydrates and calorie. Fat estimation was done by the Van de Kamer procedure¹⁰. Calorie estimation was done using an adiabatic bomb calorimeter. Total nitrogen content was estimated according to microkjeldahl procedure¹¹. Coefficient of nutrient absorption was calculated using the following formula: $(N_2 \text{ intake} - N_2 \text{ output in stool and vomitus}) \times 100 / N_2 \text{ intake}$. The students 't' test (two tailed) was used to examine the differences in the mean value of the outcome variables. Analysis of data was done by using SPSS packaged programme utilizing IBM computer system.

RESULTS

Data from 18 children with diarrhoea due to unidentified pathogens are the focus of this analysis.

TABLE I. Physical, biochemical and other information (mean SD) of the two groups of diarrhoeal children.

No. of patients	Group I 18	Group II 54
Age (months)	31.0 ± 17.6	34.4 ± 14.8
Range	(12-60)	(12-60)
Body wt. on admission (kg)	9.7 ± 2.1	9.5 ± 1.9
Range	(6.7-14.3)	(6.0-14.4)
Duration of diarrhoea prior to hosp. (hrs)	23.2 ± 22.0	20.7 ± 18.6
Range	(5-72)	(4-72)
1st 24 hrs. stool wt. (gn/kg/day)	80.2 ± 110.4	125.8 ± 131.0
Range	(13.2-473.1)	(1.7-605.0)
Duration of hosp. in acute stage (days)	5.4 ± 1.5	5.1 ± 1.1
Range	(3-8)	(3-10)
Diarrhoea stopped at (hr)	64.9 ± 32.4	64.6 ± 24.6
Range	(16-120)	(24-116)
Blood biochemistry on admission:		
Serum sp. gravity	1.0260 ± 0.0003	1.0267 ± 0.0002
Hct	33.7 ± 5.2	33.5 ± 3.9
Na ⁺ (mmol/L)	138.0 ± 3.6	135.9 ± 4.8
K ⁺ (mmol/L)	4.1 ± 0.7	3.6 ± 1.0
Cl ⁻ (mmol/L)	102.6 ± 8.1	103.2 ± 6.8
CO ₂ (mmol/L)	17.3 ± 2.7	15.3 ± 3.7

Table I compares the clinical and biochemical features of the patients with diarrhoea of group I and group II children. Mean body weight on admission and duration of diarrhoea before hospitalization were similar in the two groups. Stool output during the first 24 hours of admission did not differ significantly ($P > 0.05$). Blood biochemistry, duration of diarrhoea and hospital stay were also similar between the two groups. Serum bicarbonate was low in both the groups, indicating that the patients were equally acidotic, which however was corrected after rehydration. Stool electrolyte concentrations on admission were compared between different groups of children (Table II).

TABLE II. On admission electrolyte composition of stool of the study children

Aetiology	Electrolytes (mmol/L) Mean SD				
	Na ⁺	K ⁺	Cl ⁻	HCO ₃	Anion gap
Cholera (24)	91.0 ± 25	29 ± 13	83 ± 17	25.0 ± 9	13
Rotavirus (13)	46.0 ± 29	35 ± 24	36 ± 23	8 ± 7	38
ETEC (13)	65 ± 32	34 ± 22	64 ± 24	27 ± 18	7
Shigella (4)	52 ± 19	29 ± 4	32 ± 11	13 ± 10	37
Unknown aetiology (18)	64 ± 37	41 ± 18	49 ± 26	24 ± 11	32

Figures in parenthesis indicate the number of patients.

The mean anion gap in the stool of group I children was similar to those of rotavirus and shigella.

TABLE III. Intake and absorption of nitrogen in children with acute diarrhoea due to unknown (GP.I) and known aetiologies (GP. II).

	Acute		Recovery	
	Intake (gm/kg/day)	% absorption	Intake (gm/kg/day)	% absorption
Group I	0.32 ± 0.1	-2 ± 56b	0.40 ± 0.2	35.0 ± 29d
Group II	0.32 ± 0.2	49 ± 28a	0.46 ± 0.2	69 ± 19c

a vs b: P < 0.01; c vs d: P < 0.01

In Table III, intake and absorption of nitrogen were compared between group I and II. Mean absorption of nitrogen in the group I was significantly lower (P < 0.01) than that of the group II, both during the acute and recovery stages of diarrhoea. In group I, absorption of nitrogen during the acute (-2% ± 56) and recovery stages (35% ± 29) were highly variable and did not differ significantly (P > 0.05) between the two stages of diarrhoea. Intake and absorption of fat, calories and carbohydrates of group I children during the acute and recovery stages are presented in Table IV.

TABLE IV. Intake and absorption of fat, calories and carbohydrates in children with acute diarrhoea due to unknown aetiology (group I).

Nutrients	Acute		Recovery	
	Intake (gm/kg/day)	% absorption	Intake (gm/kg/day)	% absorption
Fat	1.7 ± 0.9	62.0 ± 29a	2.0 ± 0.5	85.0 ± 10.0b
Calories	64.2 ± 30.0c	68.0 ± 31.0e	91.0 ± 27d	86.0 ± 7.0f
carbohydrates	11.0 ± 4.6	81 ± 28	16.0 ± 6	91.0 ± 6.0

a vs b: P < 0.05; c vs d: P < 0.05; e vs f: P < 0.05

Absorption of fat and calories improved significantly (P < 0.05) in the recovery period compared to the acute stage of diarrhoea. However, only the intake of calories was improved significantly (P < 0.05) in the recovery period compared to the acute stage of diarrhoea. Absorption of carbohydrate in the acute stage was not impaired and did not differ significantly from the absorption at recovery stage.

DISCUSSION

Since most of the study children in group I category were on borderline nutritional status, it was important to estimate the effect of diarrhoea due to unknown aetiology on the intake and absorption of nutrients. The stool electrolyte contents of group I children were approximately similar to those with *F. coli* (Table II), but the values of anion gap in the stool samples of group I were similar to those of rotavirus and shigella groups (Table II). The anions in the stool may have been derived from anionic proteins, fatty acids or acids originating from undigested carbohydrates. In the acute stage of diarrhoea, intake of nutrients were similar in all the children with known aetiologies⁵. The intake of nitrogen in group I was not different from the intake of those with group II (Table III). However, the most striking finding was that, in the acute stage stool nitrogen loss in the group I patients was significantly high and exceeded the nitrogen intake. Thus the absorption of nitrogen was below zero (-2%) during the acute stage of diarrhoea (Table III). The source of this excess nitrogen in the stool could be endogenous,

suggesting that the diarrhoea was of invasive type. However, we have no specific data to indicate how much of the stool nitrogen originated from the ingested food or what proportion is contributed from either microbial flora or desquamated epithelium. Similar information was obtained by Mann et al⁴ where he studied protein absorption using ¹⁵N-yeast protein in a group of infants with prolonged diarrhoea due to unknown causes. It was important to note that inspite of the loss of nitrogen, group I children managed to maintain their body weight during both the study periods presumably due to the fact that adequate quantity of calories were consumed and utilized during acute period of diarrhoea and after recovery. Thus continuation of feeding is crucial to maintain the body weight during and after diarrhoeal diseases irrespective of any aetiologies.

ACKNOWLEDGEMENT

This research was supported by the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR, B). ICDDR, B is supported by countries and agencies which share its concern about the impact of diarrhoeal diseases on the developing world. Current donors giving assistance to ICDDR, B are: Aga Khan Foundation, Australia, Bangladesh, France, Japan, Saudi Arabia, Sweden, Switzerland, United Kingdom and USAID. The assistance of Biochemistry Branch, specially of Mr. Rahim and the nursing staff of ICDDR, B is acknowledged.

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