URINE AND STOOL pH VALUES IN MALNUTRITION

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Several authors have shown that doudenal enzyme activities are decreased in patients with malnutrition and kwashiorkor.[1] [2] In these conditions the walls of the small intestine are thin and show mucosal atrophy.[3] In addition to the jejunal mucosal changes in kwashiorkor,[4] Stanfield et al[5] have shown that the intestinal disaccharidases are also decreased. Similar mucosal[6] and enzyme changes have also been reported in calorie malnutrition.[7] [8] Bowie et al's studies,[9] done in kwashiorkor cases, suggest that an acquired disaccharide intolerance is in part responsible for the diarrhea. In their studies in cases of gastroenteritis, aggravation of diarrhea and the increase of lactic acid in the stool were documented, but stool pH changes were not recorded.

We thought the stool pH changes might be used as a simple test for the detection of lactose intolerance in malnourished children. Urine pH changes were also checked to find a possible relation to fermentative diarrhea occurring in calorie malnutrition.

Material and Method

The control group consisted of 16 infants, ages one month to 32 months, without malnutrition. Only two of these cases had diarrhea of one to five days' duration prior to admission. The malnutrition group contained 68 cases, which were divided into four groups using the criteria of Doğramacı and Wray.[10]

Group 1 (First Degree Malnutrition): Of ten children, aged two months to 24 months, five had diarrhea with a duration of two days to four months prior to admission. Half of this group was free of gastroenteritis.

Group 2 (Second Degree Malnutrition) : Of 22 children, aged two months to 24 months, 11 had diarrhea with a duration of three days to four months on admission. One had edema.

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Group 3 (Third Degree Malnutrition) : Of 15 children, aged 1.5 months to three years, 11 had diarrhea with a duration of four days to five months on admission. Two presented edema.

Group 4 (Fourth Degree Malnutrition): Of 21 children, aged 1.5 months to two years, 15 had diarrhea with a duration of six days to six months on admission. Edema was observed in one.

pH determinations were done in the first stool and urine samples obtained following the patients' admission by using pHydrion control paper.

Since there was no statistically significant difference between the Beckman expanded scale zeromatic pH meter and the pHydrion control paper, the measurements were thought to be accurate.[11]

Results

Stool pH determinations :		*range*
Control Group (16 cases)	6.45 ∓ 0.20	(4.7 - 7.8)
Group 1 (10 cases)	6.45 ∓ 0.26	(5.2 - 7.6)
Group 2 (22 cases)	6.63 ∓ 0.139	(5.5 - 7.8)
Group 3 (15 cases)	6.46 ∓ 0.208	(4.2 - 7.2)
Group 4 (21 cases)	6.7 ± 0.170	(4.9 - 8.5)

No difference was shown statistically between the values of the control cases and any of these groups (p values were greater than 0.05).

Urine pH determinations :		*range*
Control Group (11 cases)	5.84 ∓ 0.01	(5.2 - 7)
Group 1 (6 cases)	6.11 ∓ 0.02	(5.2 - 7)
Group 2 (19 cases)	5.78 ∓ 0.01	(4.7 - 6.4)
Group 3 (12 cases)	5.71 ∓ 0.02	(4.2 - 7.2)
Group 4 (21 cases)	5.86 ∓ 0.01	(4.2 - 7)

Although there was a statistical difference only between the controls and Group 1 ($p < 0.001$), because six samples were obtained in this group, the result should be accepted with reservations (p values between Control Group and Groups 2,3 and 4 were greater than 0.05).

Comments

Sezer determined stool and urine pH values in 65 infants, in the age range one month to 12 months, and found 7.08 (5 - 8.4) and 6.2 (4.8 - 8) respectively.[11]

With the exception of four cases with edema, our 68 malnourished children had calorie malnutrition. In these cases, as was stated previously, the intestinal disaccharidase level might be decreased about five to ten times that of normals.[7, 8] Since the stool pH values in the malnourished groups were not more acid than the controls, it might be thought that the decreased level of disaccharides was still sufficient for the splitting of lactose in the diet.

Bowie and colleagues,[9] by determining weight and lactic acid content of the stool in kwashiorkor cases, gave indirect evidence for the disaccharide deficiency; however, they could not show it in 40 per cent of their patients who did not have severe diarrhea previously. We did a lactose tolerance test (2 gm/kg) in our three cases without gasteroenteritis (one with 4th degree and two with 3rd degree malnutrition). On the next day the test was repeated by giving glucose (1 gm/kg) and galactose (1 gm/kg) together. Blood glucose curves were similar in all which indirectly might indicate absence of lactose intolerance. It has been suggested that temporary disaccharide intolerance may follow gastrointestinal infection even in previously well-nourished individuals.[12, 13] Then the disaccharidase intolerance in the cases of kwashiorkor might be most likely secondary to gastroenteritis.

Although most of the severe malnutrition cases in our study had diarrhea, it was generally of short duration and no correlation could be made between the severity and duration of the diarrhea and the stool acidity.

From this investigation we might conclude that stool pH could not be a mark of gastrointestinal disacharide intolerance or that the decrease of lactase activity in calorie malnutrition cases is not so low that lactose splitting would be disturbed.

Since fermentative diarrhea was not observed in our cases, urine pH values might indicate that the kidney in calorie malnutrition cases has not lost the capacity for producing acid urine.

Summary

Urine and stool pH values were determined in 58 and 68 calorie malnutrition cases respectively and compared with control cases. The stool and urine pH values did not seem to be related to the degree of malnutrition. Acquired lactase deficiency which might occur in malnutrition cases could not always be shown with stool pH values.

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