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D-xylose Absorption in Children with Sickle-Cell Anaemia

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Summary

Grange A. D-xylose Absorption in Children with Sickle-Cell Anaemia. *Nigerian Journal of Paediatrics* **1982; 9: 1.** Oral D-xylose absorption tests were carried out on 25 children with sickle-cell anaemia (genotype S) and on 25 age-matched non-anaemic controls with genotype A. Timed blood D-xylose values and 5 hours urinary excretion values of D-xylose were significantly lower ($P < 0.001$) in the children with sickle-cell anaemia. It is concluded that intestinal absorption of D-xylose is considerably reduced among children of the test group and the possible pathophysiologic mechanisms are discussed.

Introduction

INVESTIGATIONS have revealed several forms of gastrointestinal dysfunction in some types of anaemia. For example, Helmer and Fouts[1] and Wormsley[2] found low urinary excretion of D-xylose in the absence of steatorrhoea in patients with pernicious anaemia. Other workers[3] have shown that as D-xylose excretion improved, haematocrit increased. Diminished D-xylose absorption and minor abnormalities of the jejunal villi have also been reported in iron deficient anaemic adults,[4] and children.[5] These findings were ascribed to the iron deficiency *per se* in view of the observation that successful iron therapy led to a reversal of the abnormalities. It follows therefore, that the effects of the various types of anaemia on gastrointestinal structure and function may be explained on the basis of chronic tissue hypoxia resulting from diminution in the oxygen carrying capacity of the blood. In sickle-cell anaemia, hypoperfusion of tissues resulting from frequent thrombotic episodes may be expected to aggravate the existing hypoxic state. Thus, renal and osseous complications, as well as abdominal pains due to multiple infarcts have been well documented in sickle-cell disease.[6 7 8]

However, reports of absorption studies of the gastrointestinal tract in sickle-cell disease are scarce. The present study attempts therefore, to determine the small intestinal absorptive function in children with sickle-cell anaemia, using the D-xylose test.

Materials and Methods

The study group consisted of 14 male and 11 female children with sickle-cell anaemia (genotype S). The ages ranged from 11 months to 6 years with a mean ± SEM of 4.0 ± 1.4 years. These children were in a steady state with no evidence of haemolytic, aplastic or thrombotic crisis. The controls were 13 male and 12 female age-matched non-anaemic children with haemoglobin genotype A. The mean age ± SEM for the controls was

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4.6 ± 2.9 years with a range of 1–6 years. Informed consent was obtained from the parent or guardian of every child, prior to the study. Capillary blood was tested routinely for haemoglobin concentration, packed cell volume, sickling and genotype. Stool microscopy for ova and parasites was also routinely carried out. The urine passed immediately prior to the D-xylose test was subjected to urinalysis and subsequently discarded. Five grams of D-xylose in 100ml of water was given to the fasted patient. A further intake of 200ml of water was permitted during the test period. At exactly 1 hour and 2 hours after the D-xylose administration, capillary blood was withdrawn for D-xylose estimation. The D-xylose content of urine collected for five hours was determined by the method of Roe and Rice[9] as modified by Colombo.[10] Statistical significance was assessed by using the Student's "t" test.

Results

The mean weights and heights-for-age of both the subjects and the controls were not different (Table 1). The mean ± SEM of haemoglobin values for both groups were 7.6 ± 0.24 g/dl and 11.6 ± 0.44 g/dl respectively (Table 1.) The stool samples of all the children were negative for both the vegetative forms and ova of parasites.

The 1-hour and 2-hour D-xylose blood values (mean ± SEM) for test group were 1.07 ± 0.05 and 1.40 ± 0.09 mmol/l respectively. These were significantly less than the corresponding values of 2.26 ± 0.14 and 2.93 ± 0.07 mmol/l for the controls ($P < 0.005$). Table II compares the actual blood D-xylose values for the controls with the corrected values for the test group allowing for the relatively expanded blood volume of the latter group. Using the factor recommended by Gross and Godel,[11] the corrected blood D-xylose values for the test group were 1.43 ± 0.06 mmol/l and 1.87 ± 0.12 mmol/l at 1 hour and 2 hours, respectively. These values were significantly lower ($P < 0.001$) than the actual values obtained for the controls (Table II and Fig I). The mean ± SEM 5-hour urinary D-xylose excretion was also significantly lower ($P < 0.005$) in the test group (21 ± 1.3%) than the corresponding value of 51 ± 1.9% in controls (Table I and Fig. 2).

TABLE I

Mean Age, Weight, Haemoglobin, Urine Volume and D-xylose Blood levels in Children with Sickle-Cell Anaemia and in Controls

	Children with Sickle-Cell Anaemia (Mean ± SEM)	*Controls (Mean ± SEM)*	*P value*
No. of Subjects	25	25	
Age (yrs.)	4.0 ± 0.28	4.6 ± 0.58	NS
Weight (kg)	14.0 ± 0.64	12.7 ± 0.78	NS
Height (metres)	1.0 ± 0.04	0.93 ± 0.40	NS
Haemoglobin (g/dl)	7.6 ± 0.24	11.6 ± 0.44	0.005
5-hour Urine Volume (ml)	99 ± 37.4	115 ± 37.1	NS
5-hour Urinary D-xylose Excretion (% of dose)	21 ± 1.3	51 ± 1.9	0.005
1-hour Blood D-xylose (mmol/l)	1.07 ± 0.05	2.26 ± 0.14	0.005
2-hour Blood D-xylose (mmol/l)	1.40 ± 0.09	2.93 ± 0.07	0.005

NS = Not significant

TABLE II

Corrected D-xylose Blood Levels in 25 Children with Sickle-Cell Anaemia Compared with Actual D-xylose Blood Levels in 25 Controls

Subject	*1-Hour Mean ± SEM (mmol/l)*	*2-Hour Mean ± SEM (mmol/l)*
Sickle-Cell Anaemia (n = 25)	1.43 ± 0.06	1.87 ± 0.12
Controls (n = 25)	2.26 ± 0.14	2.93 ± 0.07
P Value	< 0.001	< 0.001

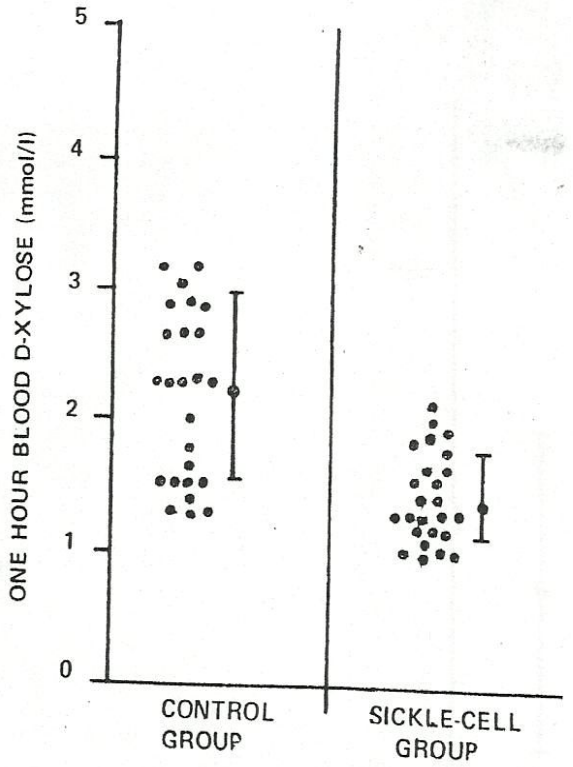

Fig. 1 One-hour blood D-xylose in 25 children with sickle-cell anaemia and in 25 controls.

Discussion

The results of the present study suggest that the absorptive jejunal capacity for D-xylose is significantly lowered in children with sickle-cell anaemia. However, other factors that could influence the rate of absorption need to be considered. It is unlikely that there is any difference in the rate of gastric emptying in sicklers when compared with controls, since the blood D-xylose values at one hour and two hours were parallel in both groups. Although the existence of an expanded fluid space consequent upon anaemia led to a lowering of the blood D-xylose values in the test group, this factor obviously had no significant effect on the relationship between corrected blood D-xylose values of the test group and those of the controls. The urinary D-xylose values of sicklers may be influenced by a poor renal concentrating ability among this group, and therefore, no valid conclusion can be based on a comparison of the urinary values alone. However, when blood values are considered along with the corresponding urinary values, they constitute a strong evidence of a poorer intestinal absorptive function among children with sickle-cell anaemia.

If the low blood values of D-xylose in sicklers signify poorer jejunal absorption, the reason for this may be twofold. It may be ascribed to prolonged tissue hypoxia caused by the anaemia *per se*. However, in sickle-cell disease, the oxygen affinity of haemoglobin is known to be decreased because of the increased level of the reduced enzyme 2, 3–DPG. Thus, the adverse effect of the anaemia *per se* on intestinal absorptive function could be partially compensated for by the ability of the patient's erythrocytes to release oxygen more readily to the tissues. On the other hand, since vascular occlusive lesions are the hallmark of thrombotic episodes in sicklers, such lesions affecting the gastrointestinal vascular bed are likely to result in some degree of hypoperfusion of the gastrointestinal tract. The cumulative effects of the anaemia and hypoperfusion may thus be sufficient to cause a degree of tissue hypoxia which could manifest as a functional abnormality. Experimental studies have shown that a temporary interruption of blood flow to the small intestine results in loss of villous cells in the rat.[12 13] There is also evidence in the human adult that impairment of the blood-supply to the intestine can lead to malabsorption.[14] The pathology in some cases was shown as partial villous atrophy[15 16] and in others, ischaemic strictures were demonstrated.[16 17] In some cases however, the jejunals morphology was found to be normal.[14] There is, therefore, a need to examine more closely, the status of small intestinal function in children with sickle-cell disease particularly in relation to the absorption of essential nutrients.

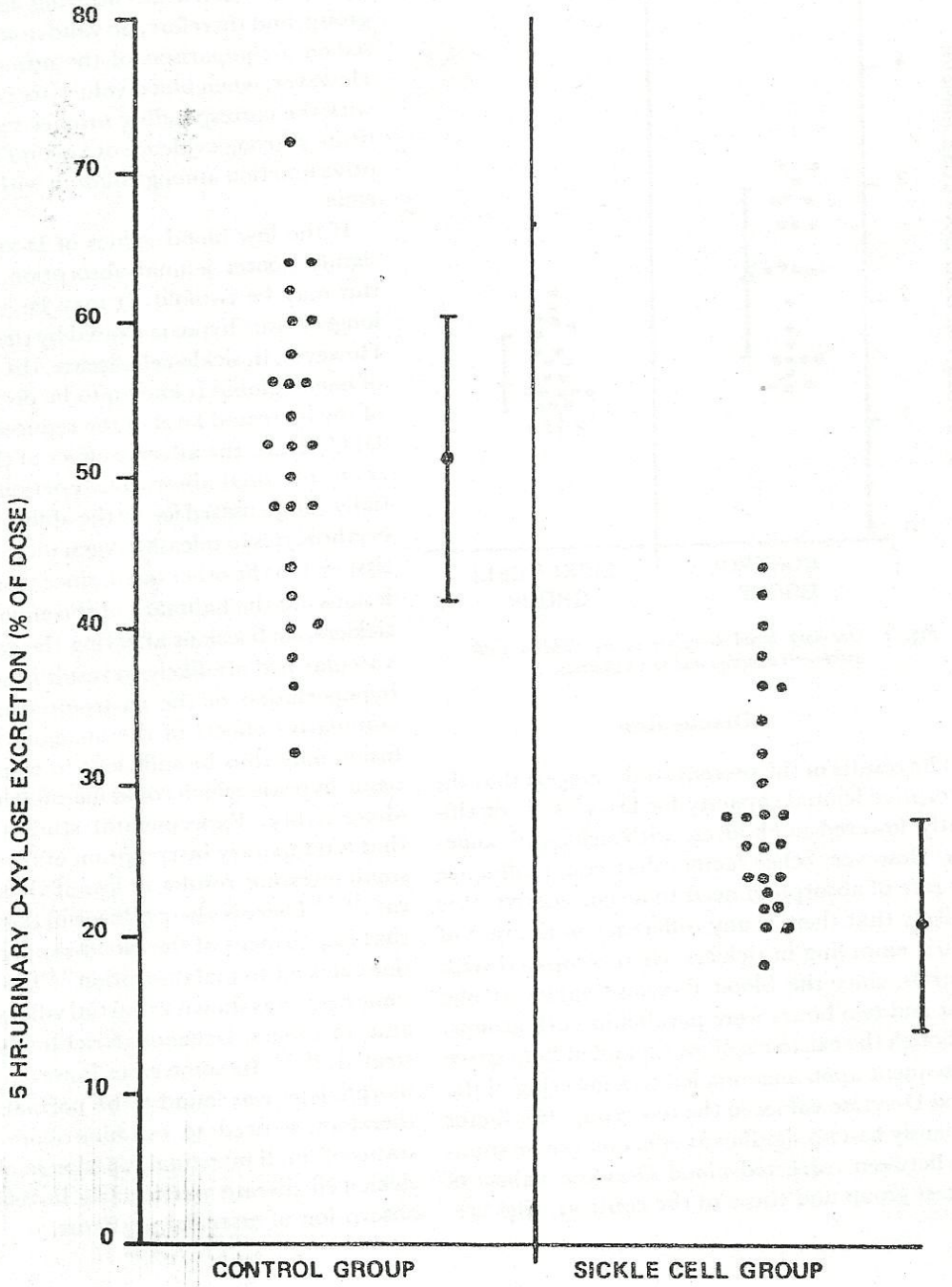

Fig. 2 5-hour urinary D-xylose excretion in 25 children with sickle-cell anaemia and in 25 controls.

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