

Short reports

Oral rehydration in acute infantile diarrhoea with a glucose-polymer electrolyte solution

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SUMMARY Seven infants with mild acute diarrhoeal dehydration were rehydrated with an oral sugar-electrolyte solution containing a glucose polymer mixture. Six of them were rehydrated successfully. The high sodium content of the solution (90 mmol/l) was based on the WHO/UNICEF recommended glucose-electrolyte solution and was implicated as the cause of increases in serum sodium in 4 infants, one of whom developed serious hypernatraemia associated with glucose-positive stools. A solution with a lower sodium and glucose-polymer content may be of nutritional benefit in the oral rehydration of acute infantile diarrhoea.

Acute infective diarrhoeal dehydration is a leading cause of infant mortality both in backward and developed countries.¹ A major advance in the management of the condition has been the introduction of oral rehydration with electrolyte solutions containing either glucose or sucrose.² Although each sugar is highly effective, sucrose supplies twice the number of calories for the same osmotic load. An electrolyte solution containing glucose-polymers could supply much greater energy at no extra osmotic cost, and thus provide much needed nutritional support during the acute episode which so often takes place in areas of endemic malnutrition.¹ We have therefore assessed a glucose-polymer electrolyte solution (G-PES) in the management of acute diarrhoeal dehydration in infants.

Patients and methods

Seven normally nourished infants (mean age 12.8 ± 0.4 months, range 3-34) suffering from acute infective diarrhoeal dehydration (rotavirus $n = 4$, enterovirus $n = 1$, no agent isolated $n = 2$) with less than 10% body weight loss were studied. The isotonic (300 mosmol/kg) G-PES used for rehydration contained Caloreen (Roussel Laboratories Ltd)

125 g/l (110 mosmol/kg and yielding 730 mmol/l free glucose on complete hydrolysis), sodium 90 mmol/l (based on WHO/UNICEF universal glucose-electrolyte solution),³ potassium 19 mmol/l, calcium 4.5 mmol/l, magnesium 5 mmol/l, chloride 40 mmol/l, and citrate 30 mmol/l. Between 100 and 150 ml/kg G-PES was given and this was supplemented by breast feeding in 2 cases and water *ad libitum* until symptoms abated. Body weight and serum electrolytes were monitored daily and blood glucose 6 hourly. Stool microbiology, sugar content, frequency, and consistency were also noted.

Results

Total fluid intake during the first 24 hours of treatment was 989.0 ± 78.0 ml (129.2 ± 18 ml/kg) of which 845.7 ± 83 ml (114 ± 20 ml/kg) was G-PES.

Diarrhoea resolved within 48 hours of admission in every infant except one (Case 4) in whom diarrhoea persisted for 5 days after admission. No sugar was detected in his stools but on changing from the G-PES to SMA feeds on day 5 the diarrhoea resolved. In the remaining infant (Case 7), diarrhoea worsened during 48 hours on G-PES. Sugar was detected in the stools and severe hypernatraemia led to a convulsion. After intravenous rehydration, the infant made a good recovery.

On admission, all infants were normonatraemic (mean 140 ± 0.4 mmol/l, range 136-143). Within 48 hours of treatment, the level of serum sodium rose to a mean of 146.2 ± 3.3 mmol/l, reflecting rises in serum sodium of >3 mmol/l in 4 infants, in one of whom the sodium rose to 162 mmol/l.

However, initial values for blood urea (6.0 ± 0.8 mmol/l) and bicarbonate (14.4 ± 1.0 mmol/l) showed significantly favourable changes after oral therapy (urea 3.2 ± 1.0 , $P < 0.05$; bicarbonate 21.8 ± 2.0 , $P < 0.02$.) However, body weights in the 5 infants successfully treated showed only moderate

Table Progress of infants treated with glucose polymer-electrolyte solution

Case	Diagnosis	Duration of diarrhoea (days)		Body weight (kg)		Urea (mmol/l)		Bicarbonate (mmol/l)		Serum sodium (mmol/l)	
		Before admission	In hospital	Before	After	Before	After	Before	After	Before	After
1	No pathogen detected	3	1	5.2	5.54	8.4	2.6	12	20	143	141
2	Rotavirus	5	2	9.1	9.43	6.9	4.0	12	21	143	146
3	No pathogen detected	7	<1	9.48	9.64	6.0	4.5	12	23	138	137
4	*Enterovirus	2	5	4.6	4.46	2.2	0.5	17	23	141	145
5	Rotavirus	7	1	16.4	16.5	4.7	1.8	18	29	136	141
6	Rotavirus	3	2	10.9	11.6	6.8	6.0	13	19	141	142
7	†Rotavirus	1	2	6.5	6.34	6.9	4.8	17	18	138	162
	Mean	4±0.9	1.9±0.5	8.9 ±1.5	9.1 ±1.6	6.0 ±0.8	3.46 ±0.7	14.4 ±1.0	21.9 ±1.4	140 ±0.4	144 ±3.1

*Diarrhoea settled on transfer to SMA feeds, glucose-negative stool. †Hypernatraemic convulsion. Intravenous rehydration required, glucose-positive stool.

increases, reflecting the mild degree of dehydration. In the remaining 2 infants body weight fell despite improvements in levels of blood urea and bicarbonate (Table), and diarrhoea persisted beyond 48 hours in one of them.

Transient increases in blood sugar above 7.5 mmol/l occurred in one infant only. With the exception of one (the oldest child aged 34 months) all the children tolerated G-PES well. Vomiting was not a limiting factor.

After the hypernatraemic convulsion, no further infants were entered into the study.

Discussion

The calorific content of G-PES (525 kcal (125 kJ)/l) exceeded that of the widely used sucrose (160 kcal (38 kJ)/l) and glucose solutions (80 kcal (19 kJ)/l) without increasing overall osmolality. Thus, the successful outcome in 6 of the 7 infants suggests that a glucose-polymer solution may have a place in the management of acute infective diarrhoeal dehydration but the tendency towards hypernatraemia, in one case severe enough to lead to a convulsion, precluded further use of the solution in that form. The changes in urea, bicarbonate, and body weight implied successful rehydration, but the rises in the concentration of serum sodium in 4 of the 7 suggest that sodium absorption in excess of water absorption was taking place.

Several factors may be implicated.¹ Firstly, the high sodium content (90 mmol/l), although based on WHO/UNICEF recommended glucose-electrolyte solution,³ might have led to excessive sodium absorption. Secondly, hypernatraemia is more likely to develop if diarrhoea sodium content is low as is the case with rotavirus infection.⁴ Persistent, low-sodium diarrhoea¹ results in greater water than sodium losses thus leading to hypernatraemia.

Thirdly, although glucose polymers are known to be powerful stimulants of sodium and water absorption in normal subjects,⁵ and in the short bowel syndrome,⁶ malabsorption of sugar may occur during acute infective diarrhoeal dehydration,^{1,7} further exacerbating water losses, such diarrhoea characteristically having a low sodium content.⁷ As sugar was detected in the stools of the most severely hypernatraemic infant, malabsorption of glucose polymers might have been implicated in this case. However, successful rehydration without hypernatraemia is possible despite evidence of sugar malabsorption.^{1,7}

Since the WHO glucose electrolyte solution may confer nutritional benefit in endemically malnourished children,¹ a glucose polymer solution as used in this study may prove to be of greater nutritional benefit. However, we believe the overall tendency for serum sodium to rise can be principally attributed to the high sodium content of our solution and we thus agree with those who advocate a more flexible approach than WHO/UNICEF towards sodium contents as low as 25 mmol/l,^{1,8} particularly in temperate climates. Thus future studies should be concerned with a formulation containing a lower sodium and glucose-polymer concentration, although such a solution could still provide greater nutritional support than the cheaper more readily available glucose and sucrose electrolyte solutions.

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Faecal excretion of alpha-1-antitrypsin in acute diarrhoea

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SUMMARY High levels of faecal α -1-antitrypsin were found in children suffering from acute rotavirus diarrhoea and in children with diarrhoea in whom no microbial pathogen could be established. The results suggest that transient protein-losing enteropathy is common in acute childhood diarrhoea and may influence the outcome of the disease.

Gastrointestinal protein loss is a common phenomenon that has been demonstrated in association with more than 70 disorders affecting the gastrointestinal tract.¹ There is a lack of information about protein loss during diarrhoea because methods have been cumbersome.¹

Crossley and Elliott,² and later Bernier *et al.*,³ have shown that measurement of faecal α -1-antitrypsin (α -1-AT) is a simple and reliable index of excessive loss of plasma proteins into the gastrointestinal tract. Recently Thomas *et al.*⁴ demonstrated its value in screening for various gastrointestinal mucosal disorders in children.

Diarrhoeal diseases are a leading cause of child death in developing countries, and the existence of a vicious circle of malnutrition and gastroenteritis is well known.⁵ Since rotavirus is a major pathogen of childhood diarrhoea in both temperate and tropical climates, it would be helpful to know if gastrointestinal protein loss occurs in acute rotavirus diarrhoea. We have now studied α -1-AT content of stools from children in hospital because of diarrhoea.

Materials and methods

During a 1-year prospective study of the aetiology of

childhood diarrhoea⁶ stool samples were randomly collected in the acute and convalescent stage of the disease for α -1-AT measurements. The faecal samples were freshly frozen at -20°C .

The patients were divided according to the aetiology of the diarrhoea into two study groups: rotavirus-associated diarrhoea with no evidence of other simultaneous enteropathogen (91 patients), and non-specific diarrhoea with no demonstrable microbial aetiology (60 patients). In the latter group no rotavirus, adenovirus, *Salmonella* sp., *Shigella* sp., *Yersinia* sp., *Campylobacter* sp., diarrhoeal *Escherichia coli* or *Giardia* sp. could be demonstrated. For comparison the α -1-AT content of stools was measured again in the same patients about 4 weeks after diarrhoea (60 patients, the control group).

Faecal α -1-AT determinations were made according to Crossley and Elliott,² using commercially available immunodiffusion plates (M-Partigen, Behringwerke, Marburg, W. Germany).

Results

Mean faecal α -1-AT concentration (\pm SE) in the rotavirus patients was 2.74 ± 0.34 mg/g dry weight (range 0-16.88). There was no statistically significant difference when compared with the non-specific diarrhoea group (3.34 ± 0.62) (range 0-28.13). Both groups of patients with diarrhoea differed significantly ($P < 0.001$) from the control group (0.44 ± 0.06) (range 0-2.80) (Fig. 1).

Faecal α -1-AT concentration was over 1.39 mg/g dry weight (mean of controls $+2$ SD) in 56%, 57%, and 3% of the children in the respective groups.