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Oral versus intravenous rehydration therapy in severe gastroenteritis

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SUMMARY A controlled, randomised trial comparing the results of oral rehydration therapy with those of intravenous fluid treatment in 470 children with severe gastroenteritis was undertaken. The oral rehydration therapy was divided into two phases—a rehydration phase that used high sodium isotonic fluid at 40 ml/kg per hour and a maintenance phase using low sodium isotonic fluid (sodium 40, potassium 30, bicarbonate 25, chloride 45, and dextrose 130 mmol/l). The results indicate that oral rehydration treatment, used according to this protocol, is successful in treating severe diarrhoea and dehydration, and has considerable advantages over intravenous fluid therapy in reducing complications associated with the treatment of hypernatraemia, in promoting rapid correction of hypokalaemia and acidosis, in decreasing the duration of diarrhoea, and in promoting a greater weight gain at hospital discharge.

Oral rehydration therapy has been widely used for some years in treating dehydrated patients.[1–3] A study in 1980 showed that oral treatment could be used successfully in severe dehydration and shock.[4] We compared the results of oral rehydration therapy, according to the protocol used in the study mentioned above,[4] with intravenous fluid treatment in 470 patients suffering severe forms of diarrhoea, vomiting, and dehydration.

Materials and methods

Patients. The study population consisted of 470 children aged 1 to 18 months, admitted to this hospital between April and September 1981 for severe dehydration, diarrhoea, or vomiting, regardless of their previous treatment and state of nutrition. All were initially examined by the hospital house officers (independently of the investigators) and the decision to admit them was based on their clinical assessment that severe dehydration, watery diarrhoea (greater than 10 ml/kg per hour), and vomiting (more than six times per 24 hours) were present (in accordance with the criteria suggested by the World Health Organisation).[3] If two or more signs in the 'severe dehydration' category (Table 1) were present, the patient was considered to have severe dehydration. A similar guide was used for moderate dehydration. Patients were randomised either to the oral treatment group (study group) or the intravenous treatment group, which was the control group. The patients' biographical data, medical history and parents' written consent were obtained for all. No patient had any medical complications other than those directly related to dehydration. Thirty six per cent of the study group and 33% of the control group were below the third centile for weight according to the standards published by the National Center for Health Statistics.[5]

The volume of fluid given to each patient, and the frequency of vomiting were recorded. Patients were weighed on hospital admission and at discharge. Follow up studies at home were randomly performed on 334 children between October and November 1981.

Laboratory studies. Laboratory studies performed on admission included venous blood sampling for serum concentrations of sodium, potassium, chloride, and bicarbonate, stool sampling (via rectal catheter) for parasites and bacteria.[6–8] Follow up laboratory studies were performed 24 hours after admission and at discharge (serum sodium, potassium, chloride, and bicarbonate). For some patients these examinations were performed more than three times.

Treatment. The oral treatment protocol consisted of

Table 1 *Clinical assessment of severity of dehydration*

Signs and symptoms	*Moderate dehydration*	*Severe dehydration*
General appearance and condition	Thirsty; restless or lethargic and drowsy but irritable to touch	Drowsy; limp, cold, sweaty, cyanotic extremities; may be comatose
Radial pulse	Rapid and weak	Rapid, feeble, sometimes impalpable
Respiration	Deep, may be rapid	Deep and rapid
Anterior fontanelle	Sunken	Very sunken
Systolic blood pressure	Normal or low	Less than 90 mmHg; may be unrecordable
Skin elasticity	Pinch retracts slowly	Pinch retracts very slowly (>2 sec)
Eyes	Sunken (detectable)	Grossly sunken
Tears	Absent	Absent
Mucous membranes	Dry	Very dry
Urine flow	Reduced amount and dark	None passed for several hours; empty bladder
% Body weight loss	6–9%	10% or more

Modified from World Health Organization guide.[3]

two phases: (a) rehydration, and (b) maintenance treatment.[4] For rehydration therapy all patients had a nasogastric tube passed and their gastric contents aspirated. Intragastric drip was started with electrolyte A solution (Table 2) at a rate of 40 ml/kg per hour (maximum=400 ml/hour). Administration of the electrolyte solution was continued at the same rate until all clinical signs of dehydration had resolved. This was achieved within a mean (SD) of 6 (2) hours. If within the first two hours of treatment the signs of dehydration had worsened or remained unchanged, oral therapy was stopped and intravenous treatment started. In this case, oral therapy was defined as a failure.

After complete rehydration, maintenance therapy with electrolyte B solution (Table 2) was given by bottle (but nasogastrically for those patients who could not take the solution orally) at a rate of about 250 ml/kg per 24 hours. Both solutions were made up by dissolving one prepared package in 250 ml of tap water. In cases of severe diarrhoea, where the stool volume was more than 10 ml/kg per hour, solution A was administered instead of solution B until the volume of stool had decreased to less than 10 ml/kg per hour.

Within 24 hours the children were fed with breast or undiluted formula milk at about 10 to 20 kcal/kg per 24 hours. This amount was gradually increased to about 100 kcal/kg per 24 hours within the next few days and simultaneously the amount of solution B was decreased as needed. No intravenous solutions or other medications were used.

Patients in the control group were treated[9] for shock (or to prevent its occurrence) with lactated Ringer solution: 20 to 30 ml/kg body weight was administered as rapidly as possible or within one hour in less severely ill patients. If clinical signs of shock persisted a second infusion of 20 to 30 ml/kg was given to restore circulation. Two thirds of the approximate losses of sodium and water were calculated and replaced during the first 24 hours of treatment, and the remaining one third during the second day. In addition to replacing losses, total fluid and electrolytes given during the first 24 hours and on subsequent days of treatment included replacement for both continuing normal losses and any abnormal losses from diarrhoea. In hyponatraemic dehydration, extra amounts of sodium needed to replace the additional losses were given over several days. In hypernatraemic dehydration, the amounts of normal maintenance fluid and sodium were reduced by 30% until hypernatraemia was corrected.

In general, the solutions used for this purpose contained: 25 to 60 mmol/l sodium, 15 to 35 mmol/l potassium, 0 to 35 mmol/l bicarbonate, and 20 to

Table 2 *Composition of electrolyte solutions A* and B† (mmol/l)*

Solution	*Sodium*	*Potassium*	*Bicarbonate*	*Chloride*	*Dextrose*	*Osmolarity (mOsm/l)*
A	80	20	35	65	70	270
B	40	30	25	45	130	270

*Made by adding the following to 250 ml of tap water: sodium chloride, 0·6575 g; sodium bicarbonate, 0·735 g; potassium chloride, 0·375 g; and anhydrous dextrose, 3·15 g.
†Made by adding the following to 250 ml of tap water: sodium chloride, 0·22 g; sodium bicarbonate, 0·525 g; potassium chloride, 0·56 g; and anhydrous dextrose, 5·85 g.

60 mmol/l chloride in 5% dextrose in water. When the frequency and volume of stools had subsided, oral feeding of half strength milk was begun. As soon as this was tolerated without exacerbation of diarrhoea, the caloric intake (volume and strength of milk) was increased gradually until the usual dietary intake was achieved. This was accomplished within a mean (SD) time of 3 (2) days.

Results

At the time of hospital admission there were no significant differences in clinical characteristics between the study and control groups. An aetiologic agent was identified in 77 children (33%) in the oral rehydration group and 62 (26%) control patients (Table 3).

A comparison of intake, output, percentage of weight gain, and duration of diarrhoea was made for the two treatment groups (Table 4). In general, children treated with oral rehydration solution took in significantly more sodium, potassium, bicarbonate, chloride, fluid, and milk than the control children on intravenous rehydration treatment.

Table 3 *Features of treatment groups on admission to hospital*

	Study group (n=236)	*Control group (n=234)*
No	236	234
Age (months), mean (SD)	7·4 (3·9)	7·9 (5)
Boy:girl (%)	63:37	57:43
Body weight at discharge (kg), mean (SD)	6·4 (1·7)	6·5 (2)
No (%) with history of vomiting	212 (90)	215 (92)
No (%) with history of severe vomiting	118 (50)	124 (53)
Days of diarrhoea before admission, mean (SD)	3·7 (2·7)	3·4 (1·9)
No (%) with history of severe diarrhoea	170 (72)	168 (72)
Rectal temperature (°C), mean (SD)	37·9 (0·8)	37·8 (0·8)
No (%) given antibiotics before admission	182 (77)	190 (81)
Estimated degree of dehydration, no (%)		
Severe with signs of shock	49 (21)	61 (26)
Moderate to severe	151 (64)	152 (65)
Pathogens identified in stools, no (%)	77 (33)	62 (26)
Enterotoxigenic *Escherichia coli*	30 (13)	27 (12)
Salmonella	22 (9)	19 (8)
Shigella	12 (5)	8 (3)
Campylobacter	1 (0·4)	0 (0)
Entamoeba histolytica	4 (2)	3 (1)
Giardia lamblia	8 (3)	5 (2)

Table 4 *Features of groups during treatment*

	Study group (n=236)	*Control group (n=234)*	
No of patients receiving intravenous fluids	1	234	
Intake (1 kg) during first 6 hours, mean (SD)			
Treatment solution (ml)	222 (14)	81 (10)	P<0·001
Sodium (mmol)	17·8 (1·1)	6·6 (0·13)	P<0·001
Potassium (mmol)	4·4 (0·3)	0·9 (0·04)	P<0·001
Bicarbonate or lactate, or both (mmol)	7·8 (0·5)	1·7 (0·28)	P<0·001
Chloride (mmol)	14·4 (0·9)	5·8 (0·3)	P<0·001
Intake (1 kg) during first 24 hours, mean (SD)			
Treatment solution (ml)	432 (54)	228 (35)	P<0·001
Sodium (mmol)	28·5 (3·1)	12·8 (2·3)	P<0·001
Potassium (mmol)	9·8 (1·2)	3·96 (0·54)	P<0·001
Bicarbonate or lactate, or both (mmol)	13·7 (1·4)	3·97 (0·65)	P<0·001
Chloride (mmol)	24·6 (3·0)	12·8 (1·9)	P<0·001
Total intake (1 kg) during illness, mean (SD)			
Treatment solution (ml)	846 (172)	680 (110)	P<0·001
Sodium (mmol)	44·5 (7·8)	31 (5·5)	P<0·001
Potassium (mmol)	22·2 (4·7)	13 (2)	P<0·001
Bicarbonate and/or lactate (mmol)	24 (4·3)	8·5 (1·5)	P<0·001
Chloride (mmol)	42·7 (8·7)	35·5 (5·7)	P<0·001
Intake other than treatment solution (ml/kg)*	242 (53)	154 (25)	P<0·001
Duration (days) of diarrhoea after hospital admission, mean (SD)	4·8 (2·1)	5·5 (2·9)	P<0·01
Percentage weight gain at discharge, mean (SD)	8·9 (3·3)	7·2 (3·2)	P<0·001

*Milk and water.

Table 5 *Concentration of serum electrolytes (mmol/l) in the two treatment groups (values Mean (SD))*

	Study group (n=236)	*Control group (n=234)*	
Sodium	*(n=178)*	*(n=205)*	
On admission	141·5 (11·7)	140·6 (9·7)	
24 hours later	138·8 (8·6)	138·5 (8·7)	
At discharge	138·9 (3·6)	138·7 (4·1)	
Hypernatraemia ($Na^+ > 150$)	*(n=34)*	*(n=24)*	
On admission	157·5 (4·8)	156 (4·6)	
24 hours later	149 (4·9)	150 (5·1)	
At discharge	139 (3·5)	139 (3·8)	
Hyponatraemia ($Na^+ < 130$)	*(n=22)*	*(n=19)*	
On admission	122 (4·4)	121 (4·3)	
24 hours later	132 (5·2)	129 (4·9)	
At discharge	138 (4·1)	138 (4·3)	
Potassium	*(n=141)*	*(n=184)*	
On admission	4·2 (0·9)	4·0 (0·9)	
24 hours later	4·8 (0·8)	4·3 (0·9)	P<0·001
At discharge	4·6 (0·4)	4·0 (0·6)	P<0·001
Hypokalaemia ($K^+ < 3·5$)	*(n=45)*	*(n=52)*	
On admission	2·5 (0·6)	2·5 (0·6)	
24 hours later	3·2 (0·5)	2·5 (0·6)	P<0·001
At discharge	4·4 (0·4)	3·4 (0·5)	P<0·001
Hyperkalaemia ($K^+ > 5·5$)	*(n=21)*	*(n=19)*	
On admission	6·3 (0·8)	6·3 (0·7)	
24 hours later	6 (0·4)	6 (0·5)	
At discharge	5·5 (0·4)	5·3 (0·5)	
Bicarbonate	*(n=166)*	*(n=165)*	
On admission	9·9 (4·8)	9·5 (5·6)	
24 hours later	15·0 (5·9)	11·7 (4·2)	P<0·001
At discharge	24·0 (2·1)	20·8 (3·7)	P<0·001
Chloride	*(n=171)*	*(n=155)*	
On admission	117·7 (12·6)	115·6 (11·6)	
24 hours later	110·3 (8·8)	114·9 (8·3)	P<0·001
At discharge	102·0 (4·1)	105·7 (2·2)	P<0·001

Serum electrolytes (Table 5). Twenty four hours after hospital admission, electrolyte abnormalities developed in 14 patients in the study group and 29 in the control group who had been normonatraemic and normokalaemic on admission: one from the study and 12 from the control group became hypokalaemic; five study group patients and three controls became hyperkalaemic; seven study group and 13 control children became hyponatraemic; and one study group child and one control became hypernatraemic. These abnormalities resolved and were not associated with symptoms.

Treatment failure. Oral rehydration treatment was considered to have failed in one patient. His clinical signs of dehydration became worse during the first two hours after admission, his stool output increased to more than 20 ml/kg per hour, and intravenous therapy was therefore instituted for six hours. Testing of stools for reducing substances was negative and no aetiologic agent was identified.

Complications. During treatment two of the 34 hypernatraemic study group patients (6%) developed generalised seizures while six of 24 hypernatraemic patients (25%) in the control group developed these seizures (P=0·05). The serum glucose and calcium concentrations were normal at this time; serum electrolyte values in both groups were sodium greater than 155, bicarbonate less than 9, chloride greater than 134, and potassium 3·4 to 4·6 mmol/l. These children were treated with diazepam and phenobarbitone, and recovered without sequelae.

Periorbital oedema developed after rehydration in eight children, four from each group. Their serum protein ranged from 4·7 to 6·1 g/dl. The periorbital oedema resolved within three to eight hours after stopping rehydration solutions.

Phlebitis developed at the injection site in five control group patients, and required antibiotic treatment.

Abdominal distention developed in four patients receiving oral rehydration but resolved after the rate of administration of electrolyte A solution was reduced.

Eight hypokalaemic patients in the control group developed paralytic ileus one to three days after admission to hospital. They recovered after being given more potassium in their intravenous fluids.

A total of 46 (19%) children in the study group and 71 (30%) in the control group vomited one to three times during the first six hours of rehydration (P<0·001).

Mortality. Two patients in the study and five in the control group died three to eight days after treatment had begun. The former two children were under the third centile for weight.[5] On admission, serum electrolytes were: sodium 136, potassium 3·5, bicarbonate 15, and chloride 110 mmol/l for one of them, and sodium 127, potassium 2·5, bicarbonate 8, and chloride 110 mmol/l for the other. Twenty four hours after admission and before death, serum electrolytes were normal and the patients were rehydrated. They died on the third and seventh day after admission.

The five control children died three to eight days after admission to hospital. Their mean serum sodium, potassium, bicarbonate, and chloride concentrations were 136, 3, 12, and 110 mmol/l on admission; 137, 3·1, 12, and 110 mmol/l 24 hours later; and 138, 3·3, 15 and 110 mmol/l before death, respectively. Two of them were below the third centile for weight.[5]

Follow up studies. Follow up studies which were performed randomly at home on 172 study group patients and 169 control children indicated that except for the six patients who had subsequently died, all the others were normal. These six children had been admitted to other hospitals due to subsequent attacks of diarrhoea two to eight weeks after discharge from this hospital. They had received intravenous treatment but had died two to 15 days after admission to hospital.

Discussion

This study shows that nearly 99% of the children with severe forms of dehydration, diarrhoea, and vomiting were treated adequately with oral rehydration therapy alone. No serious complications developed in patients in this group and nearly all of them tolerated the rapid volume (40 ml/kg per hour) of electrolyte A solution. Severe vomiting was not a limiting factor in the successful use of oral treatment in any of our patients, although 50% of them presented with a history of severe vomiting (Table 3). In addition, severe diarrhoea which was present in 72% did not prevent successful oral treatment. The protocol was also safe and effective in the correction of a wide variety of electrolyte abnormalities present on hospital admission (Table 5).

The study indicates that the frequency of vomiting during the first six hours in the orally treated group was lower than in controls ($P<0·001$); that the duration of diarrhoea was less in the study group; and that in the same group the weight gain at hospital discharge was greater ($P<0·001$).

Twenty four hours after admission, the serum potassium concentration in hypokalaemic patients was returning to normal in the study group but was unchanged in controls. This was because potassium intake per kg was 2·5 times greater in the study than the control group during this period. Similarly, hyperchloraemic metabolic acidosis was corrected more rapidly ($P<0·001$) in study group patients as their intake of bicarbonate was 3·4 times that of controls in the first 24 hours and 2·8 times during the entire period of treatment (Table 4).

Conclusion

We conclude that oral rehydration therapy according to this protocol is successful in treating severe diarrhoea, vomiting, and dehydration, and is superior to intravenous therapy in reducing the complications associated with the treatment of hypernatraemia, in decreasing the duration of diarrhoea, in promoting rapid correction of electrolyte abnormalities, and in allowing a greater weight gain at hospital discharge.

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