Current Trends in the Management of Childhood Gastroenteritis in the Community

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INTRODUCTION

Infection of the gastrointestinal tract are still amongst the most common infections of childhood. Despite improvements in the standard of living over the last fifty years, gastroenteritis still constitutes a sizeable amount of general practitioner consultations and hospital admissions. Although most infections are mild and self-limiting with the minimum of active treatment, a small proportion require more aggressive management and hospital admission. With the advent of oral rehydration solutions the management of gastroenteritis has become simpler and the complication of hypernatraemic dehydration rare.

AETIOLOGY

There are a large number of pathogens which from time to time have been incriminated as being causative organisms of gastroenteritis in childhood (Table 1). By far the most important, more so in children under two years of age, are viruses of the Rotavirus and Norwalk agent group. On their own these viruses are responsible for up to 75% of all infective diarrhoea in infancy. The prevalence of rotavirus infections depends on geographical and climatic variations as well as other minor factors as season and feeding practices.

TABLE 1

Common causative organisms of gastroenteritis in childhood.

1. VIRUSES
   - Rotavirs
   - Adenoviruses
   - Coronaviruses
   - Norwalk Agent
   - Astroviruses
   - Enteroviruses (Picorna)
2. BACTERIA
   - Diarrhoeagenic Escherichia coli species
   - Salmonella species
   - Shigella
   - Campylobacter jejunii
   - Yersinia enterocolitica
   - Aeromonas species

Bacteria causing gastroenteritis are more varied and range from the diarrhoeagenic Escherichia coli species to various Salmonella species, Shigella, Campylobacter, Yersinia and other less common bacteria. Parasites are less commonly incriminated as causing enteritis except for Giardia lamblia, frequently isolated in cases of traveller's diarrhoea.[1]

Breast feeding has been clearly shown to offer a distinct protection against enteric infections.

CLINICAL FEATURES

By definition acute gastroenteritis is the clinical syndrome of vomiting and/or diarrhoea of acute onset of infective origin, often accompanied by fever and constitutional disturbance.

Most studies have described vomiting as the first symptom, often accompanied by diarrhoea but sometimes preceding it by several hours. The diarrhoea is typically acute in onset and frequently watery in nature. Other prodromal symptoms will depend on the infecting organism. High temperatures are uncommon in viral infections but more frequently encountered in Salmonella and Shigella infections, the latter not infrequently presenting with a febrile convulsion.

Bloody diarrhoea, although more common in bacterial infections occasionally occurs in viral enteritis. Its presence, especially in the younger child should alert the clinician to the possibility of intussusception.

Anorexia, refusal to drink and irritability are common but non-specific symptoms. Direct enquiry should be made to the passage of urine, an important sign of impending dehydration being low urine output.

Estimation of the degree of dehydration is the most important task of the clinician examining a child with gastroenteritis. Further management at home or referral to hospital for intravenous therapy will depend on the state of hydration of the child (Table 2).

In general, infants with more than 5% dehydration will require referral. Children with less than 5% dehydration may be managed at home though repeated assessment may be required in borderline cases. Occasionally parental incompetence may be a valid reason for referring a child to hospital.

TABLE 2

Signs of Dehydration

2-3%	Thirst, mild oliguria.
5%	Loss of skin turgor, slightly sunken eyes. Depressed fontanelle in infants.
7-8%	Obvious loss of skin turgor, sunken eyes. Marked thirst and poor urine output. Restlessness and lethargy.
10%	As above plus poor peripheral circulation, hypotension, hyperpyrexia, peripheral cyanosis. Impending shock.

Abdominal distension and toxicity out of proportion to the degree of dehydration should make one consider the possibility of acute abdominal pathology.

With the introduction of low solute artificial milk and oral rehydration solutions there has been a dramatic fall in the incidence of complications of gastroenteritis in childhood. As a result hypernatraemic dehydration, profound metabolic acidosis hypokalaemia, and acute renal failure are now quite uncommon.[2, 3]

MANAGEMENT

The management of gastroenteritis will depend primarily on the general condition of the child and the state of hydration. Children with less than 5% dehydration who retain oral fluids may be safely managed at home.

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In infants who are still being breast fed, early introduction of breast feeding after 18 to 24 hours should be encouraged. When the number of loose stools is small and vomiting absent it is advisable to continue breast feeding throughout possibly supplemented with glucose-electrolyte solution.

In the older child and infant not on breast milk, it is current practice to start oral rehydration with a standard, commercially available glucose-electrolyte mixture for the first 24 hours.[4, 5] Further management will depend on the age of the child. Infants are regraded to quarter strength artificial milk after 24 hours and gradually to full strength feeds at 24 hourly intervals. [6, 7] If the child relapses he should be put back to the previously tolerated strength and gradually regraded once more. In the older child on evaporated cow's milk a similar regrading system may be adopted. Solids may be re-introduced as soon as milk feeds are tolerated. Rapid regrading or the early introduction of solids is a frequent cause of treatment failure. Parents should be warned that loose or soft stools may persists for up to ten days and that this is of no importance unless the stools are very watery or the child continues to lose weight. The passage of mucoid green stools, better known as "starvation stools", is frequently encountered on the third or fourth day. The mother should be reassured and told to continue regrading milk and introduce solids (Table 3).

TABLE 3

Oral Rehydration Regimen:

Day 1	ORS*
Day 2	¼ strength feed + ORS supplements
Day 3	½ strength feed + ORS supplements (**)
Day 4	¾ strength feeds + ORS supplements
Day 5	Full strength feeds

* ORS = Oral Rehydration Solution

** Solids may be introduced from day 3 if milk feeds are tolerated. Carrots, potatoes, pasta, apples and bananas are ideal solids to start with.

Infants with persisting vomiting and dehydration of more than 5% should be referred to hospital for further assessment and perhaps intravenous rehydration.

The absorption of glucose and sodium in the small intestine are closely coupled and depend primarily on their relative concentrations. Glucose-electrolyte solutions have been designed to allow ideal small bowel concentrations and thus maximise fluid, glucose and sodium absorption. The sodium content of these oral rehydration solutions is within an acceptable range and similar to sodium content of diarrhoeal fluid.[8] The use of sucrose instead of glucose is also acceptable. Oral rehydration solutions should always be reconstituted as advised by the manufacturers and never mixed with soft drinks.

The use of "mineral" or "soft" drinks in rehydration should be discouraged especially in infants. The high glucose and low sodium content of these preparations further disrupt glucose-sodium coupled absorption and tend to increase the osmotic load on the small bowel thus making the diarrhoea worse.[9] The use of anti-diarrhoeal milk substitutes is not essential in the routine management of acute gastroenteritis, but may be useful when regrading is prolonged and in the malnourished child.

The temptation to use anti-diarrhoeal agents especially in children under five years of age should be strongly resisted. Apart from their dubious effectiveness they are frequently associated with a rise in temperature and abdominal distension. One large study in children aged 3 to 11 failed to show any advantage of using kaolin, pectin or atropine derivatives over a placebo.[10]

The role of antibiotics in acute gastroenteritis is quite well defined. Most cases of enteritis are of viral origin and therefore will not benefit from antibiotic therapy. Moreover the use of antibiotics in bacterial infections has been shown to *prolong* the carrier state especially in salmonella infections.[11] Oral neomycin is contraindicated as it may cause diarrhoea and a reversible malabsorption state associated with an enteropathy.[12] The use of antibiotics should be restricted to cases which show toxicity suggesting a possible septicaemic process, and in some infants less than three months of age. The use of potentially dangerous antibiotics such as chloramphenicol, lincomycin and parenteral streptomycin should be deplored. Infections with Campylobacter jejunii giving rise to a distinct clinical syndrome of bloody diarrhoea, pyrexia, abdominal pain and arthritis respond quite well to treatment with erythromycin. Shigella infections may be treated with a variety of antibiotics such as co-trimoxazole and ampicillin.

DELAYED RECOVERY AFTER GASTROENTERITIS

Probably the most common cause for delayed recovery or relapse after gastroenteritis is rapid or improper regrading. The early introduction of full strength milk or high lactose containing foods is a challenge to the disaccharidase-depleted intestinal brush-border resulting in transient lactose intolerance. Correct regrading over a longer period is often followed by the cessation of the diarrhoea. The use of soya bean protein-based formulae, although undoubtedly helpful, is not recommended by some authorities due to the emergence of secondary soya protein intolerance. Cow's milk protein intolerance and transient gluten sensitivity are uncommon causes of delayed recovery but should be considered when diarrhoea persists and the child fails to thrive. Early referral to hospital and the use of jejunal biopsy have played an important role in the diagnosis and management of post-enteritis syndromes.

REFERENCES

1. Vesikani T, Maki M, Sukkinen HK: Rotavirus, adenovirus and non-viral entropathogens in diarrhoea. Archives of Disease in Childhood, 1980 , 56: 264-270.
2. Walker-Smith JA, Manrul PD, Placzek M: The decline in the incidence of hypernatraemic dehydration in the United Kingdom. American Jornal of Clinical Nutrition, 1981 , 34: 1975-1976.
3. Finberg L: Hypernatraemic (hypertonic) dehydration in infants. New England Journal of Medicine, 1973 , 289: 196-198.
4. Anonymous: Oral thereapy for acute diarrhoea. Lancet ii: 1981 , 615-617.
5. Anonymous: Management of acute diarrhoea. Lancet i: 1983 , 623-625.
6. Rees L, Brooke CGD: Gradual reintroduction of full strength milk after acute gastroenteritis in children. Lancet ii: 1979 , 770-771.
7. Dugdale A, Lovell S, Gibbs V, Ball D: Refeeding after acute gastroenteritis: a controlled study. Archives of Disease in Childhood, 1982 , 57: 76-78.
8. Levine MM, Hughes TP, Black RE: Variability of sodium and sucrose levels of simple sugar/salt oral rehydration solutions prepared under optimal and field condtions. Journal of Paediatrics, 1980 , 97: 324-327.
9. Head J, Hogarth M, Parsloe J, Bromkall J: Soft drinks, electrolytes and sick children. Lancet i: 1983 , 1450.
10. Diarrhoeal Disease Study Group (U.K.): Loperamide in acute diarrhoea in childhood: results of double blind, placebo controlled multicentre clinical trial. BMJ 1984 , 289: 1263-1267.
11. Anonymous: Persistent excreetion of Salmonellas. BMJ ii: 1978 , 509.
12. Lambert HP: Antimicrobial agents in diarrhoeal disease. Clinics in Gastroenterology 1979 , 8: 827-833.