THE MANAGEMENT OF ACUTE DIARRHOEA IN CHILDREN

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ABSTRACT.
Acute diarrhoea is a leading cause of less than five years old morbidity and mortality and is both preventable and treatable. This paper reviews acute diarrhoea, in children. As the second leading cause of under five years old morbidity and mortality globally, it is important to have a good understanding of its current treatment. The clinical presentation, complications and management are discussed. The prevention of acute diarrhoea is also discussed since this is an important strategy in the reduction of the burden of this disease in the community. Finally approaches to the prevention of this disease and possible solutions are suggested.

Key words: Acute, diarrhoea, management, complications, prevention

INTRODUCTION
Acute diarrhoea is one of the leading causes of under five years’ old morbidity and mortality and fortunately, it is both preventable and treatable. According to the WHO, acute diarrhoea in children is the second leading cause of under-five morbidity and mortality globally. Acute diarrhoea is defined as the passage of three or more loose stools per day or more frequent passage than is normal for the individual. Frequent passing of formed stool is not diarrhoea, nor is the passing of loose pasty stools by breastfed babies. Acute diarrhoea could be bloody or non-bloody and lasts less than 14 days.

Each year acute diarrhoea kills around 525 000 children under 5 years old. Globally there are nearly 1.7 billion cases of acute childhood diarrhoea every year. Acute diarrhoea is a leading cause of malnutrition in children 5 years old and less. It is on record that a significant proportion of acute diarrhoea can be prevented through the provision of safe drinking water and adequate sanitation and hygiene in the handling and disposal of human excreta.

AETIOLOGY
Acute diarrhoea in children is commonly caused by infections in the gut, (gastroenteritis). These infections could be caused by viral agents such as rotavirus and adenovirus, bacterial agents and other microbes.

Food poisoning, can be caused by eating food contaminated with microbes such as Escherichia coli, Salmonella species, Campylobacter species and toxins produced by bacteria such as Clostridium species. Parasitic agents of aetiological concern are cryptosporidial and giardia infections that are reported more frequently in children with immunosuppression.

Contaminated water and other drinks could be a ready source of infection as well as antibiotics induced acute diarrhoea. Non-infectious causes of acute diarrhoea are reported to be uncommon in children and examples include colitis, food intolerance and allergy.
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PATHOPHYSIOLOGY
In acute diarrhoea, there is abnormal frequency and liquidity of faecal discharges. The different causative agents have the capacities to stimulate various processes that affect enterocyte secretory and absorptive functions. In rota virus induced diarrhoea there is destruction of the microvilli tips of the enterocytes leading to impaired absorption which in the presence of osmotically active non absorbable solutes increase the osmotic load and resultant diarrhoea. In addition, some of the non-absorbable solutes can be presented to the colon where they are fermented into small chain fatty acids (SCFAs), resulting in further increase in gut osmotic load thus resulting in more diarrhoea. Bacterial and viral infection of the gastrointestinal tract can cause extensive irritation of the mucosa with enhancement of the rate of secretion. Vibrio cholera for example can initiate this process by producing a cytotoxin which cause an increase in cyclic AMP levels leading to increased secretion of sodium and water into the small bowel and resultant diarrhoea. By some unknown mechanisms, diarrhoea of increased secretion origin can be initiated and sustained through increase of ion channels. In addition to the above processes, many fold increase in the motility of the intestinal wall result in large quantities of fluid becoming available for washing the infectious agent towards the anus and at the same time, strong propulsive movements propel the fluid forwards for expulsion through the anus. Some allergic and inflammatory processes can lead to the formation of anti-enterocyte auto antibodies as in auto immune enteropathy. The presence of these auto antibodies leads to non-functioning of the regulatory T cells resulting in partial or total villus atrophy with crypt hyperplasia. The result is a disturbance in intestinal solute transport with inability to concentrate gut contents against a high osmotic gradient and interference with the absorption of fluid in the intestine and resultant diarrhoea.

CLINICAL FEATURES
The clinical features of acute diarrhoea varies from a mild stomach upset for 1 or 2 days with slight diarrhoea to severe watery diarrhoea for several days. In addition there may be abdominal cramp that may cease with stool. Other clinical features include the presence of vomiting, fever, headache, aching limbs, anorexia and acute weight loss.

When dehydration sets in, signs and symptoms of dehydration and other complications now become obvious, and are useful in classifying the degree or severity of dehydration. The signs of moderate dehydration are, passing little urine, dry mouth, dry tongue and lips, fewer tears when crying, sunken eyes . When one or more of the following signs is seen severe dehydration is said to be present:
1. Drowsiness.
2. Pale or mottled skin.
3. Cold hands and feet.
4. Very few wet nappies.
5. Fast and often shallow breathing.
6. Delayed capillary refill time.
7. Unconsciousness.
8. Unable to drink or drink poorly.
9. Skin pinch goes back very slowly over more than 2 seconds.

RISK FACTORS
There are some risk factors when present that are associated with unfavorable outcome. Some of such risk factors are:
1. Age less than 12 months.
2. LBW infants waiting to catch up their weights.
3. A child that does not drink much.

MANAGEMENT OF ACUTE DIARRHOEA
The goal of treatment is the prevention of dehydration and the treatment of dehydration whenever it occurs. The first step is taking history of the present illness which should focus on the consistency, frequency and duration of stools as well as on the presence of fever, vomiting, abdominal pain or blood in stool. Ask care giver about current or recent antibiotic use and assessment for presence of risk factors. The systems should be reviewed to seek for symptoms of complications of diarrhoea such as weight loss, decrease in urination and fluid intake. Symptoms
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of causes include urticarial rash associated with food allergy.\textsuperscript{2,6}
The past medical history seeks to evaluate for known causative disorders in the patient and family members, state of immune suppression such as HIV AIDS. This aspect of the history is meant to exclude chronic diarrhoea from HIV AIDS that is starting as an acute diarrhoea.\textsuperscript{9,10}

PHYSICAL EXAMINATION
During the physical examination the vital signs are assessed, noted and recorded for pyrexia, tachypnoea and hypotension. The weight of the child is checked for acute weight loss which can result from dehydration. A general examination is conducted for lethargy or distress. The mucous membranes are examined to assess whether they are moist or dry. When examining the extremities the focus is on the state of the skin tugor, capillary refill time and whether the hands and feet are cold and clammy as in hypovolemic shock. Because the abdominal examination may elicit discomfort it is best examined last for discomfort, distension and quality of bowel sounds such high pitch, normal or absent.

LABORATORY TESTING
Laboratory testing is unnecessary as most cases of acute diarrhoea, are self-limiting. However for non-viral diarrhoea and in the presence of complication the following laboratory tests are useful: full blood count including packed cell volume and haematocrit, total and differential white cell counts and the blood film report.
2. Stool microscopy, culture and antibiotic sensitivity.
3. Serum electrolytes, urea and creatinine.
4. Serum bilirubin and liver function test, if haemolytic uremic syndrome (HUS) complication is being considered.
5. Blood culture and antibiotic sensitivity and urine analysis, culture and antibiotic sensitivity tests as in typhoid septicaemia.

TREATMENT
In general the treatment of acute diarrhoea focuses on hydration which can usually be done orally. Intravenous hydration is rarely essential and is reserved for severe dehydration. Adjunct treatment with probiotics is suggested to have a health benefit in the form of reduction in the duration of diarrhea, especially in antibiotics associated acute diarrhoea. Probiotics are microorganisms intended to have a health benefit when consumed.\textsuperscript{5} Similarly, supplemental zinc tab administered daily for 10 to 14 days shortens diarrhoea duration and improves outcome. The dose is 10 mg daily for children less than six months of age and 20 mg for children older than six months of age.\textsuperscript{11} Loperamide an anti-diarrhoea drug is not recommended for infants and children aged 12 years and below because of undesirable side effects, which includes constipation, vomiting, abdominal pain, dizziness and more seriously fatal paralytic ileus with abdominal distension, toxic retention and megacolon.\textsuperscript{12,13}

REHYDRATION
The WHO, recommends oral rehydration solution, ORS, which should contain complex carbohydrate or 2 percent glucose and 50 to 90 mEq/L of sodium, as the fluid of choice for the treatment of dehydration. It is widely available. Sports drinks, sodas, juices and similar drinks should not be used, since they do not have sodium/glucose co-transport ability necessary for the absorption of water across the mucosal membrane during the course of acute diarrhoea. They generally have too much carbohydrate and too little sodium to take advantage of sodium glucose co-transport and the osmotic effect of excess carbohydrate result in additional fluid loss with more diarrhoea. The dose of ORS is 50 ml per kg bodyweight over 4 hours for mild dehydration and 100 ml per kg body weight, over 4 hours for moderate dehydration, for each diarrhoeal stool, an additional 10 ml per kg body weight up to a maximum of 240ml is given. The child is reassessed after 4 hours and the same volume of fluid will be repeated if signs of dehydration persist.
INDICATIONS FOR INTRAVENOUS THERAPY
The presence of one or more of the following signs is an indication for intravenous therapy:
1. Severe dehydration and hypovolemic shock.
2. Altered level of consciousness.
3. Respiratory distress and child cannot drink freely.
5. Haemodynamic instability in the setting of severe hyponatremia, < 120mEq/L or hypernatremia, > 160mEq/L.
6. Failure of ORT due to persistent vomiting.

TREATMENT OF SEVERE DEHYDRATION
The aim is to rapidly expand the plasma volume and prevent circulatory collapse thus:
1. Give 20ml/kg body weight bolus isotonic fluid intravenously as rapidly as possible. An example of such a fluid is a colloid such as albumen and a crystalloid such as normal saline or ringer lactate solution. If no intravenous site is available, begin intraosseous infusion via the marrow space of the tibia. A second bolus is given, if no response to the initial bolus.
2. Next initiate deficit replacement over the next 8 hours and maintenance and ongoing losses replacement over the next subsequent 16 hours.
3. If unable to eat over prolonged period, nutritional needs must be met through hyperalimentation or enteral tube feedings.2,3,8

DIET AND NUTRITION
The child with acute diarrhoea, should eat an age appropriate diet as soon as they have been rehydrated and not vomiting. Infants may resume breast milk or formula feeding.

COMPLICATIONS
Some complications of acute diarrhoea consist of:
1. Dehydration and electrolyte imbalance.
2. Reactive complication in other parts of the body such as cellulitis, conjunctivitis and arthritis.
3. Spread of infection to other parts of the body as seen in salmonella typhi infection.
4. Persistent diarrhoea syndromes, such as lactose intolerance and Irritable bowel syndrome.
5. Haemolytic uremic syndrome.
6. Malabsorption and malnutrition.

PREVENTION
A. Prevention of spread of infection to others:
The first step in prevention is halting the spread of infection to primary contacts. This can be achieved by the following measures, regularly clean the toilet used by the child. If a potty is used, wash with hot water and detergent and leave to dry. Wash hands of child after going to toilet. For soiled clothes, first remove stool into toilet, then wash separately. Do not allow them prepare food for others. They should stay off school, nursery etc, until at least 48 hours after the last episode of diarrhoea and or vomiting. They should not swim in swimming pools for at least 2 weeks after illness, if the cause is cryptosporidium.2,10
B. Prevention of acute diarrhoea in the general population:
For this aspect of prevention, it is recommended that there is access to safe drinking water. The use of improved sanitation approach in the disposal of human excreta while discouraging bush or open field defecation helps to reduce the incidence of diarrhoea diseases.3 It is on record that hand washing with soap and water reduces the incidence of diarrhoea by half.14,15 The practice of exclusive breastfeeding in the first six months of life reduces diarrhoea in infancy. Good personal and food hygiene and health education about how infections spreads are important in the control of diarrhoea. Vaccination with rota virus vaccine orally at 2 and 3
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months of age along with other routine vaccinations are useful in the reduction in the incidence, duration and severity of diarrhoea episodes.16

CONCLUSIONS
1. Acute diarrhoea is a common paediatric concern.
2. Infectious gastroenteritis is the most common cause.
3. Laboratory testing is rarely necessary in children with AD.
4. Dehydration is likely if diarrhoea is severe and or prolonged.
5. Oral rehydration is effective in most cases.
6. Anti diarrhoea drug such as loperamide is not recommended for infants and young children less than 12 years old.

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