Diarrhea in the tropics

Summary

- A common and major problem; a major cause of mortality in children
- Mortality due to dehydration and invasive bacteria
- Etiology: viruses, Shigella sp., Vibrio cholerae, Giardia lamblia, Entamoeba histolytica, ...
- Clinical: degree of dehydration, ± blood in the faeces, ± fever, ± acute/chronic
- Rehydration (PO or IV); nutrition, sometimes aetiological treatment necessary

General

Diarrhoea is very common in the tropics. It is often self-limiting, but its general significance cannot be overestimated. It is a major cause of malnutrition and is one of the main causes of death particularly in children.

What precisely is meant by diarrhoea varies between patients:

1. an increased number of defecations per day (e.g. more than 3)
2. a decreased consistency of the faeces or
3. an increased volume of stools (e.g. > 200 g/24h)

all are used to define the problem.

The WHO definition of diarrhoea is at least 3 evacuations every 24 hours of unformed faeces. Unformed means here that they take the shape of any container into which they are evacuated. WHO emphasises the importance of change in stool consistency rather than frequency, and the usefulness of parental insight in deciding whether children have diarrhoea or not.

Diarrhoea causes fluid loss resulting in **dehydration**. The patient also loses electrolytes, which can lead to ion imbalances, such as hypokalaemia. Acidosis develops due to the loss of bicarbonate in the stools, to reduced renal function (less acids are excreted) and to ketosis

(breakdown of body fat due to reduced food intake). Often the patient has no appetite and the nutritional status which is sometimes already poor deteriorates further. Sometimes the mother thinks she is doing good by “letting the intestines rest” and temporarily not giving food. Moderate undernourishment can then develop into severe malnutrition (marasmus and kwashiorkor). The latter is often seen if a patient has had a number of episodes of diarrhoea in quick succession.

Dysentery is a severe form of diarrhoea. Fever is common in bacillary dysentery, but rare in amoebic dysentery. Dysentery has three characteristics:

1. Abdominal pain
2. Tenesmus (pain due to cramps in the rectum) and false defecation need
3. Frequent evacuation of small quantities of faeces that are mixed with blood, mucus and/or pus

Steatorrhoea or fatty diarrhoea is characterised by large quantities of faeces with an increased fat content (the stools float on water). This occurs in certain malabsorption syndromes. The cause usually lies in disorders of the pancreas or small intestine.

Aetiology

Diarrhoea is usually caused by infections. Of the nearly 11 million deaths that occur annually among children under five years of age, diarrhoeal disease is the second leading cause (after respiratory tract infections). The most common cause of severe gastroenteritis worldwide is rotavirus which accounts for 29 to 45 percent of nearly 2 million deaths. Bacterial intestinal infections (especially dysenteria) also contribute to the high mortality.

The most common causes of diarrhoea include (non-exhaustive list):

1. **Preformed bacterial toxins**, with the bacterium itself being no longer active in the intestine. Examples include staphylococcal diarrhoea (*Staphylococcus aureus* toxin), the ingestion of *Clostridium perfringens* toxins after eating contaminated meat (pigbel) and *Bacillus cereus* toxins (contaminated rice, among other things). Incubation time very short (hours).

2. **Bacteria** which multiply in the intestines: *Salmonella, Shigella, Yersinia enterocolitica*, a whole zoo of related *Escherichia coli* strains, toxicogenic *Vibrio cholerae*, *Campylobacter jejuni*, toxicogenic *Clostridioides difficile*
3. **Protista**: *Giardia, Entamoeba histolytica, Balantidium coli*, *Dientamoeba fragilis,* microsporidia, various coccidia (*Isospora belli, Cryptosporidia, Cyclospora, Sarcocystis*). Sometimes malaria is accompanied by diarrhoea!
4. **Worms**: only in case of serious infections, e.g. *Schistosoma mansoni, Capillaria philippinensis, Strongyloides stercoralis, Trichinella spiralis*; rarely by other worms. *C. philippinensis* and *S. stercoralis* can remain several decades in the body and are able to multiply inside the human host, something that most other worms cannot achieve. They can be lethal. Since worm infections are so common in the tropics, worm eggs are often found in the stools. *However, there is not necessarily an etiological connection between the presence of helminth eggs and diarrhoea.*
5. **Viruses**: Rotavirus, Astrovirus, HIV, Noroviruses (Noroviruses cause gastro-enteritis, with important vomiting accompanying the diarrhoea).
6. **Non-infectious causes** such as laxative abuse, animal or vegetable toxins, marine biotoxins, mycotoxins, irritable bowel syndrome and inflammatory bowel diseases (Crohn's disease, ulcerative colitis) are much less common. Endocrine problems (hyperthyroidism) and related problems (vipoma, carcinoid, etc) exist but are present in a minority of persons who present with chronic diarrhoea.

It is not always important to discover the exact cause of an episode of diarrhoea: for example, it is important to distinguish between amoebic colitis and bacillary dysentery, but the difference between Rotavirus and Norwalk virus(= Norovirus) enteritis is at present not clinically relevant in the tropics.

Intestinal infections caused by protista occur everywhere but are more prevalent in tropical climates.

The climate helps protista to survive in the outside world and poor hygiene promotes their transmission. Diarrhoea is often found together with a parasitic infection, but **the causal connection must always be assessed critically**. It is important to distinguish between infection and disease. Of the many protista that are found in faeces, only a few types are potentially pathogenic. Occasionally, *Plasmodium falciparum* and *Leishmania donovani* can

cause digestive symptoms. The diarrhoea then displays no particular characteristics.

Patients with diarrhoea may be classified based on the disease duration, aspects of the stool and other clinical symptoms.

Acute diarrhoea

Acute non-bloody diarrhoea with little or no fever

If the diarrhoea is very watery and very abundant, the possibility of **cholera** must always be considered (*see separate lecture notes on cholera*).

Food poisoning by **bacterial toxins** (including staphylococci) may also causing this type of diarrhoea, resulting in explosive diarrhoea shortly after a meal. The bacteria reproduce in food and produce a thermostable toxin. The bacteria are usually killed when food is cooked or left over food is reheated.

The toxin however is not destroyed by the heat and enters the intestine, where it causes massive diarrhoea, probably by neurotoxic action on the autonomous nervous system. **Antibiotics are therefore of no value here.** Symptomatic treatment is indicated. Toxins produced by *Bacillus cereus* (often present in contaminated rice) can produce a similar picture or the “emetic syndrome”. Some milder infections, such as traveller’s diarrhoea, produce hardly any fever. In these cases bowel motion inhibitors (loperamide) can be given.

Acute non-bloody diarrhoea with fever

In children any infection of any type can be associated with diarrhoea, e.g. otitis media, tonsillitis, pneumonia, urinary infection, etc. The main pathogens are viruses, some *Escherichia coli* (ETEC is the most common pathogen in traveller diarrhoea) and mild forms of bacillary dysentery (*Salmonella*, *Shigella*, *Campylobacter* and *Yersinia*). The possibility of malaria and typhoid fever must be considered.

Acute diarrhoea with fever but without bloody stools, **generally requires no antibiotics**. The emphasis is on administering fluids and electrolytes. In small children a bacterial

infection of the intestine can rapidly give rise to bloodstream infection. Antibiotics may therefore be indicated in small children (<1 year), other vulnerable patients or in patients with persisting and/or deteriorating symptoms.

Acute bloody diarrhoea with fever

This is the picture of a **bacillary dysentery**. Pathogens are *Shigella*, *Salmonella*, *Campylobacter* and some *Escherichia coli*.

Some bacteria are very aggressive, while others give rise to milder infections.

Complications can occur:

- toxic megacolon
- rectal prolapse
- bloodstream infection
- haemolytic-uraemic syndrome. TTP-HUS, often triggered by Shiga toxin produced by *Escherichia coli* O157:H7 or other verotoxin producing bacteria (VTEC). If HUS occurs, antibiotics are contraindicated because otherwise still more toxins are released from the bacteria that have been killed, which aggravates the clinical status.
- reactive arthritis
- Reiter's syndrome [urethritis, arthritis, conjunctivitis, uveitis, hyperkeratosis of the palms of the hand (keratoderma blennorrhagicum) and painless ulcers in the mouth and on the glans (balanitis circinata)].
- After using antibiotics an overgrowth of *Clostridioides difficile* can occur in the intestine. The toxins that are produced by this bacterium cause a severe inflammation of the colon (pseudomembranous colitis) which can develop into toxic megacolon.
- A very serious complication after *Campylobacter* enteritis is the Guillain-Barré syndrome, which is characterised by ascending paralysis caused by a demyelinating process of the spinal roots.

In case of bacillary dysentery, examination of the faeces under the microscope shows numerous **white blood cells (pus) and red blood cells**. Bacillary dysentery is associated

with a marked disappearance of the normal bacterial intestinal flora. It is not possible to distinguish between the different bacteria by microscopy alone (culture is needed for this). Testing for different pathogenic *Escherichia coli* strains is difficult. E.g. testing for enterotoxicogenic E. coli requires recovery of individual bacterial clones from an agar plate inoculated with a stool sample. This should be followed by molecular evaluation for detection of specific genes. This approach is not available in most laboratories, including most labs in the West.

As always, **fluid and electrolytes** form the basis for treatment. With bacillary dysentery, **antibiotics are an important part of therapy**. The resistance of the various bacteria varies. Multi-resistant bacteria are becoming more common, especially in South and Southeast Asia. Depending on the local conditions and resistance patterns, a quinolone (e.g. ciprofloxacin) or a neo-macrolide (e.g. azithromycin) should be used. The use of diarrhoea-inhibitors (loperamide) is not recommended.

Acute bloody diarrhoea but little or no fever

The main causes are **amoebic dysentery** and to a lesser extent **mild bacillary dysentery**. Examination under the **microscope of fresh (still warm) faeces** is important in order to identify motile trophozoites.

The normal bacterial intestinal flora is maintained in amoebic dysentery. In case of severe amoebic colitis fever may be present. Amoebic dysentery is treated with medication against the trophozoites (tinidazole = Fasigyn®, metronidazole =Flagyl®) followed by medication against any remaining intestinal cysts (paromomycine = Gabbroral®, diloxanide furoate= Furamide®). Other less common causes of bloody diarrhoea without fever are acute schistosomiasis (eosinophilia, worm eggs), massive trichuriasis (microscopy), ulcerative colitis (rare in the tropics) and *Balantidium coli* (microscopy).

Ileocaecal intussusception can present with acute bloody diarrhoea followed by intestinal obstruction.

Sometimes **malignant tumours** can present with acute bloody diarrhoea.

Food poisoning with Clostridium perfringens causes necrotising enteritis. After the Second World War this became known as “darmbrand”. In the dialect of Papua New Guinea the disorder is known as “pigbel”. Pigbel has been recognised in Papua New Guinea since 1961. The disorder is seen mainly in undernourished and parasite-infested children after eating a rich meal with sweet potatoes and infected pigmeat (pig intestines are also eaten). Meals such as this are sometimes prepared on the occasion of a great feast at which the host expresses his social standing by slaughtering and serving a large number of pigs. The illness can therefore occur in epidemics.

Clostridium perfingrens

The anaerobic Gram-positive bacterium (Clostridium perfringens) is frequently present in the flora of the colon, so there must be other factors present to cause the onset of the disease. The bacterium, better known as the causative agent of gas gangrene, can produce various toxins. The bacterial strains which produce toxins can be classified into types A, B, C, D and E. All types produce alpha-toxin, which is a lecithinase (phospholipase C). Clostridium perfringens type C, responsible for pigbel, produces alpha- and beta-toxins. The toxins in the intestine are usually destroyed by proteases. In case of undernourishment there is an important deficiency in proteases such as trypsin, and as a result the toxins can remain active. If there are trypsin inhibitors present as well, such as are found in sweet potatoes, the remaining small amount of trypsin is neutralised. Adult Ascaris worms produce trypsin inhibitors. If the intestine has reduced motility, the toxin remains in contact with the wall for a prolonged period of time and causes transmural necrosis. The lesions tend to be more prominent in the jejunum, although lesions of the ileum also occur. Besides supportive therapy, treatment is based on antibiotics (chloramphenicol, benzylpenicillin or other, broad-spectrum antibiotics), type C antiserum and mebendazole. Sometimes surgery has been performed. Vaccination against type C toxin is useful.

Chronic diarrhoea

The great majority of diarrhoea episodes last less than one week, however when diarrhoea persists for 14 days or longer, it is called persistent diarrhoea. Some authors use the term “chronic” for diarrheal illnesses lasting 30 days or longer.

Chronic non-bloody diarrhoea without fever

In the tropics protista must be looked for in the first place: *Giardia, E. histolytica, Dientamoeba fragilis, Balantidium coli,* chronic intestinal capillaria, microsporidia, various coccidia (*Isospora belli, Cryptosporidia, Cyclospora, Sarcocystis*)**.** There is a long list with other diseases causing chronic watery diarrea: pellagra (niacin, vit B3 def), hyperthyroidism, irritable bowel syndrome, lactose intolerance, food allergies, coeliac disease, malnutrition, laxative abuse, neuro-endocrine tumours, intestinal lymphoma, collagenous colitis, AIDS, protein loosing enteropathy.
Campylobacter infections and some strains of *E. coli* occasionally cause persistent diarrhoea.

Chronic non-bloody diarrhoea with fever

Chronic diarrhoea, emaciation and persistent fever are important criteria for the clinical diagnosis of **AIDS**. Other clinical signs should be searched for, such as oral candidiasis, Kaposi's sarcoma lesions, chronic pruritus, severe or repetitive shingles. Serology can confirm the diagnosis. Intestinal parasites must be searched for.

Tuberculosis of the intestine is predominantly sited at the ileocecal transition. A mass can sometimes be felt there on palpation. There is sometimes ascites due to concomitant involvement of the peritoneum. Pulmonary lesions can be present, but these are certainly not a requirement for the diagnosis of intestinal TB. Intestinal tuberculosis occurs predominantly in immunocompromised individuals. It is difficult to differentiate intestinal tuberculosis from Crohn's disease because of similar clinical, pathological, radiological, and endoscopic findings. Histological interpretation of biopsies is of limited diagnostic value in the differentiation of intestinal tuberculosis from Crohn's disease, except when caseating granulomata are found. Mycobacterial culture (isolation of *Mycobacterium tuberculosis*) and PCR are helpful in making the distinction between intestinal tuberculosis and Crohn's disease.

Chronic bloody diarrhoea without fever

One has to consider persistent amoebic dysentery, severe schistosomiasis (S. mansoni, S. japonicum), inflammatory intestinal diseases, intestinal tumour and repeated intestinal

invagination. Be aware of diarrhoea due to other causes together with bleeding haemorrhoids.

Chronic fatty diarrhoea

Causes of steatorrhoea include abnormalities of the **small intestine and insufficiency of the exocrine pancreas**. Calcification of the pancreas in chronic pancreatitis can be seen in 50% of cases (X-ray of the abdomen). Concomitant diabetes mellitus should be searched for.

Non-infectious causes of intestinal abnormalities such as coeliac disease (hypersensitivity to gluten) and intestinal lymphoma are rare. Coeliac disease is associated with antibodies against gliadin (a component of gluten) and autoantibodies against tissue transglutaminase (and/or anti-endomysium antibodies). Tropical sprue is a disease of unknown origin, common in Asia but less so in Africa. The disease responds to treatment with tetracyclines and folic acid.

Some infections may result in malabsorption:

1. *Giardia lamblia*: microscopy of the faeces. These are often asymptomatic infections, so their importance should not be overestimated. Giardia can also give rise to secondary lactose-malabsorption: dairy products can no longer be tolerated.

2. *Capillaria philippinensis*: occurs mainly in the Far East but is rare. Infection is caused by eating raw fresh water fish. Like *Strongyloides*, this worm also leads to endogenous reinfection. It can therefore reproduce in the body unlike most other worms. The eggs and larvae can be found in the stools (repeated analyses are necessary). Treatment of intestinal capillariasis is with mebendazole. It is a potentially fatal infection.

3. *Strongyloides stercoralis*: serious infections cause diarrhoea, eosinophilia, pruritus and larva currens. The stools contain seldom eggs but larvae are present.

4. *Cryptosporidia* can cause malabsorption. The possibility of AIDS must be excluded in chronic cases. The parasite can be demonstrated using Ziehl stain.

5. *Cyclospora* can be compared with “large cryptosporidia” with variable acid-fastness on Ziehl stain. Treatment with cotrimoxazole is usually effective.

6. *Tropical Sprue*: the exact cause is not known, but an infectious origin seems probable. Macrocytic anaemia, glossitis, hypo-albuminemia and signs of vitamin-deficiencies (ADEK) are common. Treatment includes tetracyclines and folic acid.

Assessment of a patient with diarrhoea

The assessment of a patient with diarrhoea includes a thorough medical history on the disease duration, stool characteristics and other relevant clinical signs and symptoms as well as an estimation of the degree of dehydration. **Medical history** should focus on:

1. How long has the patient been suffering from diarrhoea? Is it acute (<14d) or chronic (>14d)?
2. Is there fever? Weigt loss? Night sweats?
3. Is there blood or pus in the faeces, or is it watery diarrhoea ?
4. Is the diarrhoea volume large (more likely small intestine) or small (more likely colon)?
5. Is there tenesmus? Suggests that the rectum has been affected by inflammation or ulceration. Diarrhoea or rectal discharge? (suggests proctitis)
6. Is there abdominal pain? Not with cholera.
7. Is the patient vomiting? Makes dehydration worse and makes therapy more difficult.
8. Are there a number of people in the area with the same symptoms? An epidemic?
9. Is the patient immunocompromised, or does he have major co-morbidity? Any (new) medication?

Acute diarrhoea is often caused by self-limiting infections (beware exceptions). Chronic diarrhoea is more often than not caused by non-infectious causes (beware exceptions, especially in immunocompromised patients). Two intestinal helminths which as a rule persist (probably for life) even in untreated immunocompetent persons are *Strongyloides stercoralis* and *Capillaria phillipinensis*.

Chronic diarrhoea can be further classified by volume, where small frequent stools are suggestive of a distal colonic disorder. Large volume watery stools are suggestive for

conditions involving the small intestine (but beware of a secreting villous colonic adenoma). Steatorrhea or fat-malabsorption suggests problems located in pancreas, bile ducts and/or small bowel.

The presence of faecal leukocytes has a sensitivity of 70% for inflammatory diarrhoea. A test for faecal lactoferrin has a higher sensitivity but is rarely available. Continuation of diarrhoea during fasting is suggestive for a secretory process. Features that suggest an organic cause as opposed to a functional cause, include a duration less than 3 months, nocturnal diarrhoea, abrupt onset, weight loss (> 5kg for an adult), stool weight more than 400 g/24h.

Clinical assessment (degree of dehydration)

The **assessment of dehydration is most important**. Dehydration is due to an insufficient intake of liquids (drinking, IV fluid) and/or excessive loss of fluid (vomiting, diarrhoea, polyuria, sweating). If loss of gastro-intestinal fluid is the cause, the patient will urinate less (oliguria) in order to minimise the loss.

If a child has lost **< 5%** of its body weight, the general condition is still quite good. The child is alert and thirsty. The mucous membranes (eyes, tongue, mouth) are moist and the turgor of the skin (elasticity) is maintained. Breathing is normal. Urine production is normal and if the child cries there are tears. The fluid deficit is < 50 ml/kg of body weight.

If 5-10% of body weight is lost the eyes are sunken, the fontanelle is hollow, the skin is no longer elastic, the lips and mouth are dry and sometimes cracked. The child is miserable, restless and cries. There are no tears. Breathing becomes more rapid (acidosis). This must be distinguished from an accompanying pulmonary infection. Urine production decreases. The fluid deficit is 50-100 ml/kg.With a fluid loss **of >10%** the child is quiet and cold. The pulse is rapid and difficult to feel (circulatory collapse), especially the radial pulse. Skin folds do not disappear, the mucous membranes are very dry, the abdomen is hollow, the eyes are deeply set and the fontanelle is deeply sunken. Usually there is no more urine. The fluid deficit is >100 ml/kg.

A rapid clinical dehydration evaluation can make use of the following items: general

appearance, skin, eyes, tongue and tears. A more detailed evaluation can determine the following items:

Table: Evaluation dehydration for children up to 36 months

Appearance:	**Normal**	**Thirsty-restless-irritable**	**Drowsy-limp**
Capillary refill	<1.5″	1.5-3″	>3″
Skin turgor	instant recoil	<2 seconds	> 2 seconds
Fontanelle	Normal	Slightly sunken	Very sunken
Eyes	Normal	Slightly sunken	Very sunken
Tongue	Moist	Sticky	Dry
Tears	Present	Decreased	Dry
Breathing (<1y)	<40/’	40-50/’	> 50/’
Breathing (1-3y)	<30/’	30-40/’	> 40/’
Heart rate (<6m)	<175/’	175-185/’	> 185/’
Heart rate (6-36m)	<150/’	150-165/’	> 165/’
Urine specific gravity	< 1.015	1.016-1.030	> 1.031

Treatment

General

Two things must always be considered:(1) the degree of **dehydration/rehydration** needs, (2) is **drug** treatment necessary? The most important thing with acute diarrhoea is to deal with dehydration and in the second place to correct protein and calorie deficiency. Etiological treatment will only be possible in a minority of cases, but should not be disregarded.

Children are very sensitive to dehydration. Fluid loss can occur very quickly with vomiting and diarrhoea: 500 ml of fluid in a child weighing 5 kg means a loss of 10% of body weight and implies a high risk of death.

IV rehydration is not always possible nor even desirable. An important development has been the discovery that many cases of dehydration of whatever origin can be counteracted by oral rehydration. This is possible because despite the diarrhoea, the mechanisms for absorbing water, sodium and glucose in the intestine are maintained. The minimum ingredients for this oral rehydration solution (ORS) are clean water, glucose and salt. While this can indeed bring about rehydration or prevent dehydration, a disadvantage is that the diarrhoea itself continues. The volume of stools is not reduced. Alternatives to glucose are ordinary sugar (sucrose; this is a glucose-fructose disaccharide) or rice powder. Rice powder is better because it reduces the volume of stools. In ideal circumstances potassium (against hypokalaemia) and bicarbonate or sodium citrate (against acidosis) can be added. Citrate is easier to store than bicarbonate. In the future there may perhaps be better formulae which also contain neutral amino-acids (glycine and alanine) and perhaps dipeptides.

There are several formulae for ORS. The WHO has developed a standard formula in which each litre of water should contain:

KCl	1.5 gram
Trisodium citrate dihydrate	2.9 gram
NaCl	2.6 gram
Glucose	13.5 gram

Treatment, in practice

- Always weigh the child and assess its general condition.
- Assess whether the weight loss is <5%, 5-10% or >10%.
- Is it dysentery or not? If yes, is it amoebic or bacillary?

With mild to moderate dehydration use ORS. The volume that should be given is 1-2 times the fluid deficit. ORS is best given by the mother and should be given over a 4 to 6 hour period. It is best if it is given with a small cup and spoon. With very small children a syringe can be used to drip the fluid into the mouth. If the child vomits a few times the treatment should be continued nevertheless. Administration using a nasogastric drip infusion is rarely necessary. The success of the treatment should be monitored by assessing the general condition of the child and its weight.

With severe dehydration (>10%) or if the treatment with ORS is not successful, IV rehydration should be used. If it is not possible to inject into a vein and a venous cut-down is not feasible and the situation is desperate, the intraosseous route can be used: the fluid enters the bone marrow of the tibia and is taken up in this way. The infusion can be rapid at first (70 to 100 ml/kg over 3 hours). If the pulse can be felt clearly again and the child has generally improved, the treatment can then be switched to oral therapy. Potassium chloride should be added in severe diarrhoea.

Newborn children with a low birth weight are very sensitive to hypernatremia. Rehydration is achieved best with 2/3 ORS and 1/3 extra salt-free water.

Food must continue to be given while the patient has diarrhoea. It used to be thought that a period of fasting (24 to 48 hours) was good for the child, but this is counterproductive. Breastfeeding should not be stopped. A balanced diet, low in residue and semi-solid is indicated. During episodes of diarrhoea, patients are catabolic (they break down their own muscle proteins for energy).

Medication

1. Antibiotics for bacillary dysentery.
2. Antiparasitic agents for amoebiasis, giardiasis, malaria, isosporiasis, Strongyloides, capillariasis, etc.
3. Zinc, folic acid and vitamin A supplements, especially in malnourished children.
4. Antimotility products loperamide (Imodium) or opiates: codeine, paregoric (= opium tincture) or laudanum reduce intestinal cramps and the frequency of bowel movements.

They are only indicated for uncomplicated diarrhoea. They do not reduce fluid loss. Anti-diarrhoeal drugs must be avoided in children because they can aggravate dysentery and can easily be given to children in too high a dose resulting in paralytic ileus and sedation interfering with oral rehydration.

5. Sometimes the main complaints is nausea. Domperidone can be used, though its use should be restricted to severe cases, especially when combined with other QTc-proloning drugs as
6. fluoroquinolones (ciprofloxacin, levofloxacin, moxifloxacin) or(neo-)macrolides (clarithromycin, azithromycin).
7. Lactobacillus and saccharomyces boulardii concentrates are probably of little benefit but more research is needed.

Prevention

Most diarrhoea is transmitted by the faecal-oral route. The prevention of these infections will therefore depend on improved general hygiene, which is determined by the general level of poverty (standard of living).
Rotavirus disease kills approximately half a million children annually in developing countries and accounts for one third of hospitalizations for diarrhoea worldwide. In 1999, the first licensed rotavirus vaccine (RotaShield) was withdrawn from the U.S. market less than a year after its introduction because it was associated with an uncommon but potentially life-threatening adverse event, intussusception, at an estimated rate of 1 incident per 10,000 vaccine recipients. The manufacture of the first licensed rotavirus vaccine was halted. In 2005, results of large clinical trials of two new vaccines, Rotateq from Merck and Rotarix from GlaxoSmithKline, were published. These are both live oral vaccines intended to be given to infants at the same time as their immunizations for diphtheria, pertussis, and tetanus, but they differ in their approaches, strains, and formulations. Rotarix is given in 2 doses with minimum 4 weeks interval. Rotateq is given in 3 doses with minimum 4 weeks interval. Both vaccines demonstrated an impressive efficacy profile and a reassuring safety profile, particularly with respect to intussusception.

A few general tips and precautionary measures for avoiding diarrhoea are recommended:

1. Food should be completely cooked/boiled.

2. Drinking water should be protected. This can be achieved in a village context (sand filters, protection of water-wells, etc). Water can be boiled and filtered, but boiling requires a lot of fuel, which is usually expensive.
3. Wash hands with soap.
4. Sanitary provisions: toilet and drinking water should be kept separate. Inexpensive, simple, build-it-yourself, ventilated, odour-free, fly-free latrines that do not require any water can be made (the Blair latrine for example).

Diarrhoea: prevention for travellers

Food: avoid raw vegetables, fruit you cannot peel yourself, unpasteurised dairy products, fish, shellfish and meat that is raw or not cooked through. (Cook it, boil it, peel it or leave it). Avoid food from street stalls. Food should be protected against flies.

Drink: drink tea, coffee or bottled water, preferably sparkling (less risk of having been tampered with). Beer can quench the thirst, but large quantities of alcoholic drinks are not recommended. Avoid bottles sealed with reused crown caps. Ice cubes are not to be trusted. Drinking water can be filtered. This can be done in a number of ways (large porcelain filters such as Berkefeld, active charcoal filters, portable Katadyne filters). Afterwards the water can be boiled or purified chemically with silver salts such as Micropur®, Drinkwell® (not active against viruses), Chloramine (250 mg per 10-50 litres) or sodium hypochlorite (Javel, Drinkwell chlorine®, Hadex®). An unpleasant taste of chlorine can be removed by adding the non-toxic sodium thiosulphate (Drinkwell-antichlorine® drops) work in for an hour. Lugol or 2% tincture of iodine (eight drops per litre) can also be used and is more active against amoebic cysts. Long-term use (more than 3 months) is not recommended. Thyroid disorders and pregnancy are contra-indications.

Chemoprophylaxis: This is normally not advised routinely, but does provide partial protection (e.g. ofloxacin). Only to be considered for short journeys where absolutely nothing should go wrong.

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