



# **COMPARATIVE STUDY BETWEEN INFANT AND NEONATE PATIENT OF DIARRHEA.**

**--- By Rabeya Islam  
ID 2005-2-70-001**



**December, 2009**



# **THESIS**

**ON**

---

---

**COMPARATIVE STUDY BETWEEN INFANT AND NEONATE  
PATIENT OF DIARRHEA**

---

---

**A research paper submitted to the department of pharmacy,  
East West University in conformity with the requirement for  
the degree of Bachelor of Pharmacy.**

**A collaboration study between Department of Pharmacy, East  
West University and Institute of Child Health and Shishu  
Sasthya Foundation.**

**Prepared by:**

**Rabeya Islam**

**ID- 2005 -2-70- 001**

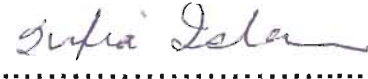
**East West University**

**Department of Pharmacy**

# CERTIFICATE


This is to certify that the thesis “comparative study between infant and neonate patient of diarrhea” submitted to the department of pharmacy, East West University, Mohakhali, Dhaka in partial fulfillment of the requirements for the degree of Bachelor of Pharmacy (B.Pharm) was carried out by Rabeya Islam (ID: 2005-2-70-001) under our guidance and supervision and that no part of the thesis has been submitted for any other degree. We further certify that all the sources of information and laboratory facilities availed of this connection is duly acknowledge.

  
..... 23.12.09  
**FARHANA RIZWAN**

  
.....  
**SUFIA ISLAM, PhD**

**Supervisor**  
**Senior lecturer**  
**Department of pharmacy**  
**East West University**  
**Mohakhali,Dhaka**

**Co- Supervisor**  
**Associate Professor**  
**Department Pharmacy**  
**East west pharmacy**  
**Mohakhali,Dhaka**

  
.....  
**Dr. CHOWDHURY FAIZ HOSSAIN**  
**Chairperson**  
**Department of Pharmacy**  
**East West University**  
**Mohakhali,Dhaka**



***This thesis paper is dedicated to my parents  
and my younger sister Lamia.***

## **Acknowledgement**

First and foremost I would like to express my sincere thanks gratitude to Mrs.Farhana Rezwan,senior Lecturer and Dr.Sufia Islam,PhD; Associate Professor Department of pharmacy,East West University,for their invaluable guidance and support throughout the entire work.

I am Greatly acknowledged to Prof Dr.A.F.M. Salim,Deputy Director(Academic) & Dr.Nobokrishna Ghosh,Assistant professor,Institute of child Health and Shishu Sasthya Foundation (ICH & SSF),for their inspiration and co-operation in my study.

I would also like to thank Dr.NH Alam,Ph.D; Scientist,Clinical Science Division,ICDDR,B;Dr.Forhad Monjur,Assistant professor,Laboratory medicine specialist and Pathologist,ICH&SSF;for their friendly assistance in the data collection during this research project.

I am especially thankful to Prof.Dr.Chowdhury Faiz Hossain,PhD;Chairman,Department of pharmacy,East West University and Prof.Dr.Muniruddin Ahmed,PhD,Pro-vice Chancellor,East west university.

Finally, I express my sincere gratitude to my caring parents for guiding me all through my life,including that for my research project.

# Content

Chapter No -----Page No

## Chapter 1

Abstract.....01

## Chapter 2

### Introduction

- Definition of diarrhea.....02
- Causes of diarrhea ..... 2-3
- Other important causes of diarrhea.....04
- Types of diarrhea.....5-7
- Histopathology of diarrhea.....07
- Causes of acute diarrhea.....8-10
- Causes of chronic diarrhea.....10-11
- Organisms that are responsible for diarrhea:
  1. *Campylobacter*..... 12
  2. *Salmonella*..... 13
  3. *E. Coli*..... 14
  4. *Ascaris lumbricoides*..... 15
  5. *Ancylostoma duodenale*..... 16
  6. *Trichuris trichiura*.....17-19
  7. *Enterobias vermicularis*.....20-24
  8. *Entamoeba coli* .....25-32

- Intestinal amoebiasis, diagnosis.....28
- Intestinal amoebiasis, differential diagnosis.....30
- Intestinal amoebiasis, treatment..... 31

**9. *Entamoeba histolytica*.....32-37**

Morphology of Trophozoites.....34

Morphology of cysts .....34

Intestinal disease.....36

Hepatic Disease .....37

Serology.....37

- Treatment of diarrhea

**Nonspecific Agents.....37**

**Oral fluids.....38**

**Antimotility .....38**

- Antibiotic Treatment.....39
- Nutritional management..... 40
- Infant diarrhea.....40-41
- Whether it is normal or infant diarrhea..... 42
- Infant diarrhea prevention and treatment.....42-43
- Neonatal diarrhea.....43
- Symptom of neonatal diarrhea.....44

- Disease caused by.....44
- Prevention of neonate diarrhea.....45
- Diarrhea treatments in general.....45
- 10 million children dying every year due to diarrhea.....46

### Chapter 3

Objective of the study.....47

### Chapter 4

Significance of the study.....48

### Chapter 5

#### Materials and method

1. Research design and place of study.....49
2. Sample size.....49
3. Research Approach.....49
4. Data collection method.....49
5. Patient information.....49
  - 5.1. Symptoms and pathological information.....50
6. Diagonosis of dirrheal patient.....50
7. Investigation of Patient.....51
8. Data analysis.....51



## Chapter 6

Case report form.....52-53

## Chapter 7:

### Result

Percent distribution of male and female patient.....54

Age distribution of the patient.....55

Number of neonates and infants are.....55

Percent distribution of infant and neonate.....56

Treatments of the patient .....57

Antibiotic treatment.....58

Percent distribution of antibiotic treatment.....59

Symptom of the patient.....60

Recovery duration..... 60

Percent of recovery time for neonate and infant.....61

## Chapter 8

Discussion.....62-63

## Chapter 9

Conclusion.....64

## Chapter 10

Reference.....65-67

# Chapter 1

## Abstract

Diarrhea diseases remain an important causes of childhood morbidity and death in developing countries. Diarrheal diseases remain an important causes of childhood morbidity and death in developing countries although diarrhea having significantly declined in re Due to successes in the implementation of oral rehydration therapy (ORT) Diarrhea disease has been reduced. More than 10 million children die each year. Most from preventable causes and almost in poor countries. 6 countries accout for 50% of worldwide deaths in children younger than 5 years and 42 countries for 90%. In 2003 an estimated 1.8 million children below 5 years died from diarrhea. 8 out of 10 of these deaths occur in the two year of life.

The objective of the study was to find out the how many infant (>1 years) and neonate (<6 year) attacked by the diarrhea admitted to Child health and Shishu Sasthya Foundation Hospital. Among the 50 patients, 37 were infants and 13 were neonate. Hospital record of children was reviewed and information was collected for this study.

Most of the patient were male (about 30) and female patient were less in number (about 20). The patient had various symptom commonly diarrhea with fever. Vomiting, cough, cold respiratory problems were also observed. In the children. 14 patients were treated with antibiotic and 36 patients did not receive any antibiotic. From 14, 10 were infants and rest of them was neonate. The common treatments were ORS, antibiotic (ampicillin, ciprofloxacin, flucloxacilin). Most of the patients received intravenous. The children also received hachure, green banana, Chira water and green coconut water, breast milk etc.

The recovery duration for most of the patient is 3 days, the symptoms disappeared in both groups patients (neonates and infants) after receiving treatment.

# Chapter 2

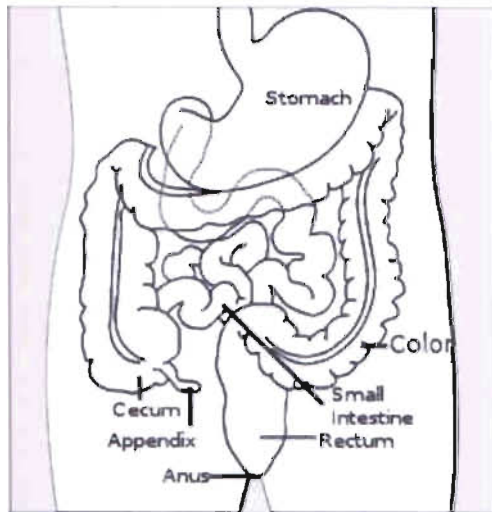
## Introduction

### **Definition of diarrhea:**

In medicine, diarrhea (from the Greek, "diarrhea" meaning "a flowing through").

- Frequent and watery bowel movements; can be a symptom of infection or food poisoning or colitis or a gastrointestinal ulcer.
- An increase in the frequency, liquidity and weight of bowl.
- A common symptom of gastrointestinal disease characterized by fluid consistency of the stools.
- Passing loose or watery stools.
- When feces are passed liquid quite frequent.
- Abnormal frequent evacuation of watery stools.
- A term that means different things to different people but is usually taken to mean a change in the bowel habit to become more loose or watery and/or an increase in bowel frequency.(Belinda Rowland,2000)

### **Causes of diarrhea:**



**Diagram: of the human gastrointestinal tract.**

Diarrhea commonly results from gastroenteritis caused by viral infections, parasites or bacterial toxins. In sanitary living conditions where there is ample food and a supply of clean water, an otherwise healthy patient usually recovers from viral infections in a few days. However, for ill or malnourished individuals diarrhea can lead to severe dehydration and can become life-threatening without treatment. (**Ruuska T ,etal 1990**).

Diarrhea can also be a symptom of more serious diseases, such as dysentery, cholera, or botulism, and can also be indicative of a chronic syndrome such as Crohn's disease or severe mushroom poisoning syndromes. Though appendicitis patients do not generally have violent diarrhea, it is a common symptom of a ruptured appendix. It is also an effect of severe radiation sickness. .(**Ruuska T ,et.al 1990**).

Symptomatic treatment for diarrhea involves the patient consuming adequate amounts of water to replace that loss, preferably mixed with electrolytes to provide essential salts and some amount of nutrients. For many people, further treatment is unnecessary. The following types of diarrhea indicate medical supervision is required:

- Diarrhea in infants
- Moderate or severe diarrhea in young children;
- Diarrhea associated with blood

- Diarrhea that continues for more than two days;
- Diarrhea that is associated with more general illness such as non-cramping abdominal pain, fever, weight loss, etc;
- Diarrhea in travelers, since they are more likely to have exotic infections such as parasites;
- Diarrhea in food handlers, because of the potential to infect others;
- Diarrhea in institutions such as hospitals, child care centers, or geriatric and convalescent homes. A severity score is used to aid diagnosis in children.(**Navaneethan U, et.al,2008**).

### **Other important causes of diarrhea:**

- Ischemic bowel disease. This usually affects older people and can be due to blocked arteries.
- Bowel cancer: Some (but not all) bowel cancers may have associated diarrhea. Cancer of the large intestine is most common.
- Hormone-secreting tumors: some hormones (e.g., serotonin) can cause diarrhea if excreted in excess (usually from a tumor).
- Bile salt diarrhea: excess bile salt entering the colon rather than being absorbed at the end of the small intestine can cause diarrhea, typically shortly after eating. Bile salt diarrhea is a bad side-effect of gallbladder removal. It is usually treated with cholestyramine, a bile acid sequestrant.
- Celiac Disease
- Intestinal protozoa such as Giardiasis.

### **Alcohol**

Chronic diarrhea can be caused by chronic ethanol ingestion.<sup>[23]</sup> Consumption of alcohol affects the body's capability to absorb water – this is often a symptom that accompanies a hangover after a binge drinking session. The alcohol itself is absorbed in the intestines

and as the intestinal cells absorb it, the toxicity causes these cells to lose their ability to absorb water. This leads to an outpouring of fluid from the intestinal lining, which is in turn poorly absorbed. The diarrhea usually lasts for several hours until the alcohol is detoxified and removed from the digestive system. Symptoms range from person to person and are influenced by the amount consumed as well as physiological differences.

(Article on diarrhea ( disambiguation ),dec 2007)

### **Types of diarrhea:**

Diarrhoea it is mean pass motion frequent time more than normal the character of stool usually it is watery in nature some time associated with blood and or mucus.

Diarrhoea is in 2 types:

- Acute- acute occur with in hours to several days.

Chronic- chronic occur within one month and more.3 grades of dehydration found:

#### **1. Mild dehydration:**

The mild one treated at home by mother increasing fluid intake by using oral re hydration solution.and increase breast feeding

#### **2. Moderate dehydration**

The moderate admitted to hospital and Chick the vital sign of child give him IV fluid till improve.



### 3. Severe dehydration:

Severe admitted to hospital evaluate the general condition of child give him iv fluid till the signs of dehydration subsided like sunken eyes; depressed anterior fontanel ;return elasticity of skin and moisture pass urine and important think reassurance of family.

### **Secretory diarrhea**

Secretory diarrhea means that there is an increase in the active secretion, or there is an inhibition of absorption. There is little to no structural damage. The most common cause of this type of diarrhea is a cholera toxin that stimulates the secretion of anions, especially chloride ions. Therefore, to maintain a charge balance in the lumen, sodium is carried with it, along with water. (Alam NH, Ashraf H (2003).

### **Osmotic diarrhea**

Osmotic diarrhea occurs when too much water is drawn into the bowels. This can be the result of maldigestion (e.g., pancreatic disease or Coeliac disease), in which the nutrients are left in the lumen to pull in water. Osmotic diarrhea can also be caused by osmotic laxatives (which work to alleviate constipation by drawing water into the bowels). In healthy individuals, too much magnesium or vitamin C or undigested lactose can produce osmotic diarrhea and distention of the bowel. A person who does not have lactose intolerance can have difficulty absorbing lactose after an extraordinarily high intake of dairy products. In persons who do not have fructose malabsorption , excess fructose intake can still cause diarrhea. High-fructose foods that also have high glucose content are more absorbable and less likely to cause diarrhea. Sugar alcohols such as sorbitol (often found in sugar-free foods) are difficult for the body to absorb and, in large amounts, may lead to osmotic diarrhea. (Alam NH, Ashraf H (2003).

### **Motility-related diarrhea**

Motility-related diarrhea is caused by the rapid movement of food through the intestines (hypermotility). If the food moves too quickly through the GI tract, there is not enough

time for sufficient nutrients and water to be absorbed. This can be due to a vagotomy or diabetic neuropathy, or a complication of menstruation. Hyperthyroidism can produce hypermotility and lead to pseudodiarrhea and occasionally real diarrhea. Diarrhea can be treated with antimotility agents (such as loperamide) **(C.Guyton,M.d,eleventh edition)**

### **Inflammatory diarrhea**

Inflammatory diarrhea occurs when there is damage to the mucosal lining or brush border, which leads to a passive loss of protein-rich fluids, and a decreased ability to absorb these lost fluids. Features of all three of the other types of diarrhea can be found in this type of diarrhea. It can be caused by bacterial infections, viral infections, parasitic infections, or autoimmune problems such as inflammatory bowel diseases. It can also be caused by tuberculosis, colon cancer, and enteritis. **(Da silva As,et al,2009)**

### **Dysentery**

Generally, if there is blood visible in the stools, it is not diarrhea, but dysentery. The blood is trace of an invasion of bowel tissue. Dysentery is caused by an excess of water by a release of antidiuretic hormone from the posterior pituitary gland. Dysentery is a symptom of, among others, *Shigella*, *Entamoeba histolytica*, and *Salmonella*. **(Alam NH, Ashraf H (2003).**

### **Histopathology of diarrhea:**

Microscopic colitis 1-4 represents an area of communication difficulty, especially between gastroenterologists and histopathologists. The authors of this commentary welcome the paper of Libbrecht et al. 5 in this issue of Histopathology which describes a form of microscopic colitis. This exemplifies the fact that the original classification of lymphocytic or collagenous colitis is a little limited for routine diagnosis and understanding of the etiology of microscopic colitis. The unhelpful term 'non-specific colitis' is best avoided, so the classification of microscopic colitis needs to evolve.

**(Essentials of Medical Pharmacology, KD.Tripathi, 5th edition)**

## Causes of acute diarrhea:

The most common causes of acute diarrhea are infectious agents (viruses, bacteria, and parasites). Other important causes include food poisoning (preformed toxins), medications, inflammatory or ischemic bowel disease, fecal impaction, pelvic inflammation (e.g. recto sigmoid abscess), and recent ingestion of poorly absorbable sugars (e.g. lactulose). **(C.Surawicz)**

Viral gastroenteritis (viral infection of the stomach and the small intestine) is the most common cause of acute diarrhea worldwide. Symptoms of viral gastroenteritis (nausea, vomiting, abdominal cramps, and diarrhea) typically last only 48-72 hrs. Unlike bacterial enterocolitis (bacterial infection of the small intestine and colon), patients with viral gastroenteritis usually do not have blood or pus in their stools and have little if any fever. **(C.Surawicz)**

Viral gastroenteritis can occur in a sporadic form (in a single individual) or in an epidemic form (among groups of individuals). Sporadic diarrhea probably is caused by several different viruses and is believed to be spread by person-to-person contact. The most common cause of epidemic diarrhea (e.g., on cruise ships) is calciviruses. The calciviruses are transmitted by food that is contaminated by sick food-handlers or by person-to-person contact. **(C.Surawicz)**

Food poisoning is a brief illness that is caused by toxins produced by bacteria. The toxins cause Abdominal Pain (cramps) and vomiting and also cause the small intestine to secrete large amounts of water that leads to diarrhea. The symptoms of Food Poisoning usually last less than 24 hours. With some bacteria, the toxins are produced in the food before it is eaten, while with other bacteria, the toxins are produced in the intestine after the food is eaten. Symptoms usually appear within several hours when Food Poisoning is caused by toxins that are formed in the food before it is eaten. It takes longer for symptoms to develop when the toxins are formed in the intestine (because it takes time for the bacteria

to produce the toxins). Therefore, in the latter case, symptoms usually appear after 7-15 hours. For more, please see the Food Poisoning article. **(Pensabene, Licia et.al)**

Staphylococcus aureus is an example of a bacterium that produces toxins in food before it is eaten. Typically, food contaminated with Staphylococcus (such as salad, meat or sandwiches with mayonnaise) is left un-refrigerated at room temperature overnight. The Staphylococcal bacteria multiply in the food and produce toxins. Clostridium perfringens is an example of a bacterium that multiplies in food (usually canned food), and produces toxins in the small intestine after the contaminated food is eaten. **(Pensabene, Licia et.al)**

There are many strains of E. coli bacteria. Most of the E. coli bacteria are normal inhabitants of the small intestine and colon and are non-pathogenic, meaning they do not cause disease in the intestines. Nevertheless, these non-pathogenic E. coli can cause diseases, however, if they spread outside of the intestines, for example, into the urinary tract (where they cause bladder or kidney infections) or into the blood stream (sepsis). Certain strains of E. coli, however, are pathogenic (meaning they can cause disease in the small intestine and colon). These pathogenic strains of E. coli cause diarrhea either by producing toxins (called enterotoxigenic E. coli or ETEC) or by invading and inflaming the lining of the small intestine and the colon and causing enterocolitis (called enteropathogenic E. coli or EPEC). Traveler's Diarrhea usually is caused by an ETEC strain of E. coli that produces a diarrhea-inducing toxin. **(Mark C.et.al, USA.)**

Disease-causing bacteria usually invade the small intestines and colon and cause enterocolitis (inflammation of the small intestine and colon). Bacterial enterocolitis is characterized by signs of inflammation (blood or pus in the stool, fever) and Abdominal Pain and diarrhea. Campylobacter jejuni is the most common bacterium that causes acute enterocolitis in the U.S. Other bacteria that cause enterocolitis include Shigella, Salmonella, and EPEC. These bacteria usually are acquired by drinking contaminated water or eating contaminated foods such as vegetables, poultry, and dairy products.

Parasitic infections are not common causes of diarrhea in the U. S. Infection with Giardia Lamblia occurs among individuals who hike in the mountains or travel abroad and is

transmitted by contaminated drinking water. Infection with *Giardia* usually is not associated with inflammation; there is no blood or pus in the stool and little fever. Infection with amoeba (amoebic dysentery) usually occurs during travel abroad to undeveloped countries and is associated with signs of inflammation--blood or pus in the stool and fever. *Cryptosporidium* is a diarrhea-producing parasite that is spread by contaminated water because it can survive chlorination. *Cyclospora* is a diarrhea-producing parasite that has been associated with contaminated raspberries from Guatemala. **(Pensabene, Licia et.al)**

Drug-induced diarrhea is very common because many drugs cause diarrhea. The clue to drug-induced diarrhea is that the diarrhea begins soon after treatment with the drug is begun. The medications that most frequently cause diarrhea are antacids and nutritional supplements that contain magnesium. Other classes of medication that cause diarrhea include nonsteroidal anti-inflammatory drugs NSAIDs, chemotherapy medications, antibiotics, medications to control irregular heartbeats (antiarrhythmics), and medications for High Blood Pressure . A few examples of specific medications that commonly cause diarrhea are misoprostol (Cytotec), quinidine (Quinaglute, Quinidex), olsalazine (Dipentum), colchicine (Colchicine), metoclopramide (Reglan), and cisapride (Propulsid). **(Pensabene, Licia et.al)**

### **Causes of chronic diarrhoea:**

Chronic diarrhea is frequently due to many of the same things that cause the shorter episodes (infections, medications, etc.); symptoms just last longer. Some infections can become chronic. This occurs mainly with parasitic infections (such as *Giardia*) or when patients have altered immunity (AIDS).

Inflammatory bowel diseases, such as ulcerative colitis and Crohn's disease, which involve the chronic inflammation of the colon, small intestine and/or other parts of the digestive tract. Crohn's Disease and ulcerative colitis, diseases causing inflammation of the small intestine and/or colon, commonly cause chronic diarrhea.

Irritable bowel syndrome is a condition that cause is not entirely understood. This creates abdominal pain, gassiness, bloating and changes in bowel habits, including diarrhea and/or constipation. The Irritable Bowel Syndrome (IBS) is a functional cause of diarrhea or Constipation. There is no inflammation. It may be caused by several different underlying problems, but it is believed that the most common cause is rapid passage of the intestinal contents through the colon. **(Thomas La Mont, MD, May2009)**

Malabsorption syndromes inhibit the intestines from absorbing various nutrients. Carbohydrate or sugar malabsorption is an inability to digest and absorb sugars. The most well -recognized malabsorption of sugar occurs with lactase deficiency (also known as lactose or milk intolerance) in which milk products containing the milk sugar, lactose, lead to diarrhea. The lactose is not broken up in the intestine because of the absence of an intestinal enzyme, lactase that normally breaks up lactose. Malabsorption of fat is the inability to digest or absorb fat. Fat malabsorption may occur because of reduced pancreatic secretions that are necessary for normal digestion of fat or by diseases of the lining of the small intestine that prevent the absorption of digested fat.

Endocrine disorders such as hyperthyroidism, diabetes mellitus and Addison's disease. Several endocrine diseases (imbalances of hormones) may cause diarrhea, for example, an over-active thyroid gland (Hyperthyroidism) and an under-active pituitary or adrenal gland (Addison's disease). **(Thomas La Mont, MD, May2009)**

Colon cancer. Colon Cancer can cause either diarrhea or constipation. If the Cancer blocks the passage of stool, it usually causes constipation. Sometimes, however, a blockage causes the secretion of water behind the blockage, and liquid stool from behind the blockage leaks around the Cancer and results in diarrhea.

Other causes include immune deficiency diseases such as acquired immune deficiency syndrome (AIDS), endocrine diseases, severe constipation, and abuse of laxatives by

individuals who want attention or to lose weight is an occasional cause of diarrhea. (J Thomas La Mont, MD, May2009)

The organisms that is responsible for different types of diarrhea

### **1.Campylobacter**

*Campylobacter* species are a group of bacteria capable of causing diarrhea in dogs, cats, humans, and other animals. They have a unique curved appearance under the microscope and are said to be “sea gull-shaped.” They are difficult to isolate as they grow in conditions of low oxygen (making them microaerophilic as opposed to being aerobic or anaerobic bacteria). With regard to pets, *Campylobacter* are generally a problem for the very young. Puppies and kittens do not have mature immune systems yet; plus, because they are small, fluid loss from diarrhea hits them much harder. Furthermore, puppies and kittens are more likely to be housed in groups where fecal cross-contamination is common so they may be more likely to become infected than adult animals. Adult animals commonly have *Campylobacter* organisms living in their intestines but they do not experience any sickness due to it. (Alam NH, Ashraf H (2003).

In humans, *Campylobacter* infection is a leading cause of gastrointestinal (GI) disease, and dogs can infect people whether they have diarrhea or not. For this reason, dogs used for therapy in assisted living communities and similar situations should be screened for *Campylobacter* by fecal culture before exposure to people with suppressed immunity. Exposure to a dog with diarrhea triples a person’s risk for developing enteritis from *Campylobacter jejune* or *Campylobacter coli*. Humans are also infected by consumption of contaminated food, water, or raw milk; only 6% of human *Campylobacter* infections are attributed to dog exposure. (Alam NH, Ashraf H (2003).

After one consumes *Campylobacter* organisms, they travel to the lower small intestine, attach, and begin to multiply. They produce a toxin that destroys the lining of the intestine with the result being a bloody, mucous diarrhea (though occasionally a more watery diarrhea is described). Sometimes a fever results, appetite becomes poor, and

vomiting can occur. Incubation is 2 to 5 days. The organism can survive as long as a month in environmental feces. (Alam NH, Ashraf H (2003).

Diagnosis is made by seeing the sea gull-shaped organisms under the microscope; however, there are so many bacterial organisms on a fecal sample that finding the culprit can be tricky. For this reason, a culture is often performed as a more accurate test. Because the organism is microaerophilic, special culture requirements must be met; the facilities of a reference laboratory are needed. (Alam NH, Ashraf H (2003

Treatment is with appropriate antibiotics, erythromycin is currently considered the drug of choice. Tetracycline is also considered effective. (Alam NH, Ashraf H (2003).

## **2. *Salmonella*:**

Most people are somewhat familiar with *Salmonella*. They know it represents a type of food poisoning, probably know it is associated with diarrhea that can be severe, and may even know that *Salmonella* species are bacteria. Most human cases of *Salmonella* infection cause fever, diarrhea, and cramping that go away on their own, but in children it can produce more severe disease. As with *Campylobacter*, the young are more susceptible to more severe illness because they are smaller and do not have mature immunity. The same is true with puppies and kittens; adult animals are almost never affected by *Salmonella* infection. . (Alam NH, Ashraf H (2003).

There is an important exception to the “*Salmonella* is rare in adult dogs” rule and that is the case of dogs fed a raw food diet. It has, unfortunately, become popular to feed raw foods to pets with the idea that a raw food diet more closely approximates the natural diet that the feline or canine body evolved to consume, and thus such a diet should be healthier than commercially prepared foods. In fact, the cooking of food is central to removing parasites, bacteria, and bacterial toxins from food. A recent study evaluating raw food diets found that 80% of food samples contained *Salmonella* bacteria and that 30% of the dogs in the study were shedding *Salmonella* bacteria in their stool. Adult dogs are often asymptomatic but any infected animal or person will shed the organism for at



least 6 weeks thus acting as a source of exposure to other animals or people. *Salmonella* organisms are very difficult to remove from the environment and easily survive 3 months in soil. Again, dogs used for therapy around the elderly or children should be cultured for the presence of *Salmonella*. **(Alam NH, Ashraf H (2003)).**

There are two syndromes associated with *Salmonella*: diarrhea and sepsis. *Salmonella* bacteria, once consumed, attach to the intestine and secrete toxins. The toxins produce diarrhea that can be severe and even life-threatening in the young. If this were not bad enough, some *Salmonella* can produce an even more serious “part two.” These bacteria are capable of invading the rest of the body through the damaged intestine.

In young animals, the syndrome resulting is similar to that of canine parvovirus, thus similar treatment is expected. **(Alam NH, Ashraf H (2003)).**

### **3.E.Coli :**

*Escherichia coli* may be the most common bacterial organism in the world. It lives in our intestines naturally and covers the world we live in. Unfortunately, some strains of *E. coli* are not so neighborly and are capable of producing diarrhea via toxin production. Like the other organisms we have discussed, this is a serious problem for the very young and more of a nuisance for adults. There are three main types of unfriendly *E. coli*:

Enterotoxigenic *E. coli*, Enterohemorrhagic *E. coli*, and Enteropathogenic *E. coli*. **(Alam NH, Ashraf H (2003)).**

Enterotoxigenic *E. coli* are a common cause of diarrhea in young animals, human infants, and are responsible for the famous traveler’s diarrhea. These bacteria produce what is called an enterotoxin in the upper small intestine. This toxin, similar to the toxin of cholera, causes the intestine cells to secrete the body’s fluid into the intestine creating spectacular watery diarrhea and what can be life-threatening dehydration for smaller living creatures. Young pigs, cattle, and other livestock are commonly lost to this kind of dehydration. Again, the younger and smaller one is, the more serious this infection is. **(Alam NH, Ashraf H (2003)).**

Enteropathogenic *E. coli* also produce diarrhea in humans and animals. Rather than a secretory diarrhea as above, they simply destroy the intestinal cells where they attach. Diarrhea still results but it creates more damage to the intestinal lining. . (Alam NH, Ashraf H (2003).

Enterohemorrhagic *E. coli* is similar to enteropathogenic *E. coli* but with more associated inflammation. This type does not seem to be a problem for small animals though they can carry it asymptotically. . (Alam NH, Ashraf H (2003).

It would seem that antibiotics would be the obvious treatment for a bacterial disease yet for *E. coli* it is surprisingly controversial. It seems that the use of antibiotics can enhance the synthesis of toxins by these bacteria plus often antibiotic use only serves to make *E. coli* more resistant in the GI tract. Antibiotics are generally reserved for those animals (or people) who seem the most sick or who have evidence of bacterial invasion in the bloodstream. Basically treatment is supportive care until the patient's immune system regains the upper hand. .(Alam NH, Ashraf H (2003).

#### ***4. Ascaris lumbricoides:***

##### **Life cycle:**

*Ascaris lumbricoides*, or "roundworm", infections in humans occur when an ingested infective egg releases a larval worm that penetrates the wall of the duodenum and enters the bloodstream. From here, it is carried to the liver and heart, and enters pulmonary circulation to break free in the alveoli, where it grows and molts. In 3 weeks, the larvae pass from the respiratory system to be coughed up, swallowed, and thus returned to the small intestine, where they mature to adult male and female worms. Fertilization can now occur and the female produces as many as 200,000 eggs per day for a year. These fertilized eggs become infectious after 2 weeks in soil; they can persist in soil for 10 years or more.

The eggs have a lipid layer, containing ascarocides and it makes them resistant to the effects of acids and alkalis as well as other unpleasant chemicals. This resilience helps to explain why this nematode is such a ubiquitous parasite.

### **Infection:**

Infections with these parasites are more common where sanitation is poor and human feces are used as fertilizer.

Prevention of this infection centers around on education, not using human feces as fertilizer, and cleanliness, especially among those who handle food.

More than 1 billion people are affected by this infection. (**Micheal j.pelezer, et.al, Microbiology Concepts and applications**)

## ***5. Ancylostoma duodenale:***

### **Life Cycle:**

Parasites are dioecious, with male and female organs in separate individuals.

Following copulation, female lays her eggs in the hosts' intestine. Eggs are passed out in the host feces.

Usual daily output of eggs for a single female hookworm is between 10,000 and 30,000 eggs.

In favorable conditions of moisture, temperature, and oxygen, eggs develop in the soil and hatch when they reach maturity. They release a rhabditiform larva which feeds for a short time and molts twice before becoming an infective filariform larva.



Filariform larvae (infective stage) of hookworm.

Larva enters the host either by being swallowed or by burrowing into the skin through hair follicles.

When it reaches the small intestine of the host, the larva molts a fourth and final time and develops to maturity.

**Damage:**

Infection by a hookworm usually results in bloody diarrhea and anemia.

Hookworm infections undermine the health of the host, causing stunting of growth and general laziness. Often accompanied by acute mental distress.

**PROPHYLAXIS:** None available

## ***6. Trichuris trichiura:***

Symptoms include:

- Light infestations are frequently asymptomatic.
- Heavy infestations may have bloody diarrhea.
- Long-standing blood loss may lead to iron-deficiency anemia.
- Rectal prolapse is possible in severe cases.

Infection occurs through accidental ingestion of eggs (which are usually found in dry goods such as beans, rice, and various grains) and is more common in warmer areas. The eggs hatch in the small intestine, and then move into the wall of the small intestine and develop. Whipworm infestation is detectable by stool examination, which can detect eggs and charcot-leyden crystals. **Mebendazole** is 90% effective in the first dose, and albendazole may also be offered as an anti-parasitic agent. Adding iron to the bloodstream helps solve the iron deficiency and rectal prolapse.

Whipworm commonly infects patients also infected with *Giardia*, *Entamoeba histolytica*, *Ascaris lumbricoides*, and hookworms. (Micheal j.pelezer,et.al,Microbiology Concepts and applications”)

Infection can be avoided by proper disposal of human feces, not eating dirt, and not eating crops fertilized with night soil.

### **Dog and cat whipworms:**



**Fig:Egg of *Trichuris vulpis***



**Fig:Egg of *Trichuris vulpis***

### **Pathophysiology:**

After 10-14 days in soil, eggs become infective. Trichuriasis is transmitted by the fecal-oral route, as with *A lumbricoides*, but in contrast to this parasite and to hookworm, no tissue migratory phase occurs. Larvae hatch in the small intestine, where they grow and molt, finally taking up residence in the large intestine. The time from ingestion of eggs to development of mature worms is approximately 3 months. Adult females lay eggs for up to 5 years.

Immunologically, cytokines such as interleukin 25 (IL-25) mediate type 2 immunity and are required for the regulation of inflammation in the gastrointestinal tract.

Children, due to a higher propensity to directly or indirectly consume soil, are more commonly and more heavily infected. Also, it is widely believed that partial protective immunity develops with age and children are not protected initially.

### **Clinical:**

### **History**

Most patients are asymptomatic. Clinical symptoms are limited to patients with heavy infection, who tend to be small children or others who eat a lot of dirt. Note that there is no pulmonary migration and, thus, no pulmonary symptoms.

- Nocturnal loose stools
- Dysentery can occur in patients with greater than 200 worms.
- Rectal prolapse
- Failure to thrive
- Symptoms of anemia (massive infection only)
- Vague abdominal discomfort
- Stunted growth

### **Physical:**

- Mild abdominal tenderness
- Signs of anemia
- Rectal prolapse
- Finger clubbing can sometimes suggest the diagnosis in infected patients.
- Direct visualization of adult worms on rectal mucosa via anoscopy or if rectum is prolapsed (adult worms only in lower colon in heavy infection)

## Causes:

Whipworm is caused by consumption of soil or food that has been fecally contaminated. (Eggs are infective or embryonated about 2-3 weeks after being deposited in the soil).

## 7. *Enterobias vermicularis*:

### Pinworm:

#### Pinworm

ICD 127.4



A pinworm (*Enterobius vermicularis*).



#### Scientific classification

Kingdom: Animalia

Phylum: Nematoda

Class: Secernentea

Subclass: Spiruria

Order: Oxyurida

Family: Oxyuridae

Genus: ***Enterobius***

#### Species

- *Enterobius vermicularis*

(Linnaeus, 1758

- *Enterobius anthropopithecii*  
(Gedoelst, 1916)
- *Enterobius gregorii* (Hugot,  
1983) (disputed)

The **pinworm** (Genus *Enterobius*), also known as **threadworm** or **seat worm**, is a common human intestinal parasite, especially in children. The medical condition associated with pinworm infestation is known as **enterobiasis**, or sometimes **oxyuriasis**.

### **Classification:**

The pinworm is a type of roundworm, and two species of pinworm have been identified with certainty. Humans are host only to *Enterobius vermicularis* (formerly *Oxyuris vermicularis*). Chimpanzees are host to *Enterobius anthropopithecii*, which is morphologically distinguishable from the human pinworm. There is also a claim for another species affecting humans, *Enterobius gregorii*, which is supposedly a sister species of *E. vermicularis*, and has a slightly smaller spicule. Its existence is controversial however, as some consider there to be insufficient evidence, and others contend that *E. gregorii* is a younger stage of *E. vermicularis*. Regardless of its status as a distinct species, *E. gregorii* is considered clinically identical to *E. vermicularis*. (Hasegawa, et.al,2005).

### **Morphology**



**Fig: Enterobius vermicularis egg under a light microscope.**



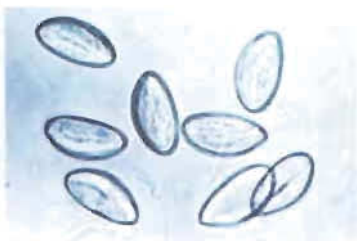
The adult pinworm male is 1–4 mm in length, while the adult female is 8–13 mm and possesses the long, pin-shaped posterior for which the worm is named. (Hasegawa, et.al,2005).

**Habitat:**

The pinworm lives in the large intestine and cecum. It is found worldwide, and causes the most common infection enterobiasis in humans. Unlike many other intestinal parasites, the pinworm does not usually enter the bloodstream or any other organs besides the intestines. Only in rare cases are pinworms found in the vagina, and even more rarely in the uterus, fallopian tubes, liver, and peritoneum, but the worms cannot survive long in these places.

The human pinworm *Enterobius vermicularis* is a ubiquitous parasite of man, it being estimated that over 200 million people are infected annually. It is more common in the temperate regions of Western Europe and North America (its existence is relatively rare in the tropics), and is found particularly in children. Samples of Caucasian children in the U.S.A. and Canada have shown incidences of infection of between 30% to 80%, with similar levels in Europe.(Hasegawa, et.al,2005).

**Reproduction:**



**Fig:Pinworm eggs are easily seen under a microscope.**

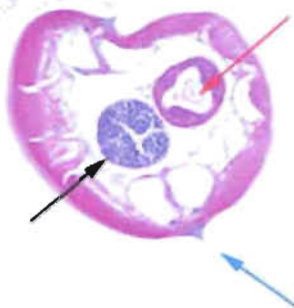
Pinworms mate by traumatic insemination. After mating, the male dies. The female migrates to the anus and emerges, usually during the night, to deposit about 10,000 to 20,000 eggs in the perianal area (around the anus). She then secretes a substance which

causes a very strong itching sensation, inciting the host to scratch the area and thus transfer some of the eggs to the fingers. Eggs can also be transferred to cloth, toys, and the bathtub. Once ingested orally, the larvae hatch in the small intestine, specifically the duodenum, and migrate back to the large intestine where they mature. Maturity is reached in 30–45 days. The eggs can survive for 2 to 3 weeks on their own outside of the human body. In some cases, the larvae will hatch in the peri-anal area and travel back inside the anus, up the rectum, and back into the intestines where they mature. (Hasegawa, et.al,2005).

### Effects:

Except for itching, pinworm infestation does not usually cause any direct damage to the body. However, the unsanitary scratching of the itch can spread germs from the fingers to other parts of the body. This scratching occurs even as the person is asleep and can easily contribute to unhealthy skin conditions or infections. Sleep disturbance may arise from the itching or crawling sensations. Some case reports suggest that severe infestation may be associated with an increased risk for appendicitis. There is also some evidence of an association between enterobiasis and diminished **zinc** levels. Which decrease the zinc level of Children. It is directly related to diarrhea. (Hasegawa, et.al,2005).

### Diagnosis:



**Fig: Male pinworm**

Pinworms are sometimes diagnosed incidentally by pathology. Micrograph of male pinworm in cross-section. Alae (blue arrow), intestine (red arrow) and testis (black arrow). H&E stain. (Hasegawa, et.al,2005).

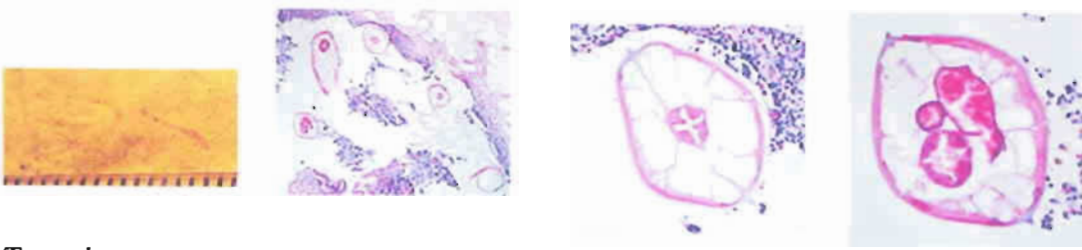
Self-diagnosis is also possible without observing worms around the anus. Crawling sensations inside the anus indicate female pinworm migration, and they may be visually detected at this time by using lubricant to insert a finger into the anus, hooking it slightly, and pulling the finger out while at the same time gently scraping the rectal wall. This may cause some of the thread-like pinworms to adhere to the lubricated finger, and they may thus be extracted from the anus. The method requires a sufficient number of repetitions, and scraping of all sides of the rectal wall. The method can also be used to provide temporary relief from intense crawling sensations caused large numbers of pinworms in the lower rectal area, simply by manually removing some of them. (Hasegawa, et.al,2005).

The diagnostic characteristics are: size 50-60  $\mu\text{m}$  by 20-32  $\mu\text{m}$ ; typical elongated shape, with one convex side and one flattened side and colorless shell. (Hasegawa, et.al,2005).

#### **Treatment:**

Anti-pinworm drugs such as **albendazole (Albenza)**, **mebendazole (Vermox, OVEX)**, **Piperazine and pyrantel pamoate (Pin-X, Reese's Pinworm Medication)** are commonly used to treat pinworms as well as ascaris lumbricoides (the roundworm). It is not a necessity to visit a doctor to get these drugs, as pyrantel pamoate (Pin-X) is available as an over-the-counter medication (albendazole and mebendazole are prescription in the US); ask a pharmacist for medicines to treat pinworms (or threadworms as they are known in the UK). These medicines kill the pinworms 95% of the time, but do not kill the eggs. (Hasegawa, et.al,2005).

**Additional images:**



Two pinworms, captured on emergence from the anus. Markings are 1 mm apart.

Pinworms are sometimes diagnosed incidentally by pathology. Micrograph of pinworms in the appendix. H&E stain.

High magnification micrograph of a pinworm in cross-section in the appendix. H&E stain.

High magnification micrograph of a pinworm in cross-section in the appendix. H&E stain.

(Hasegawa, et.al,2005).

**8. *Entamoeba coli***



*Entamoeba coli* cyst

**Scientific classification**

Domain: Eukaryota

Phylum: Amoebozoa

Class: Archamoebae

Genus: Entamoeba

Species: *E. coli*

### **Binomial name**

*Entamoeba coli*

*Entamoeba coli* is a non-pathogenic species of Entamoeba that frequently exists as a commensal parasite in the human gastrointestinal tract. Clinically, *E. coli* (not to be confused with the bacterium Escherichia coli) is important in medicine because it can be confused during microscopic examination of stained stool specimens with the pathogenic Entamoeba histolytica. (Sodeman WA ,et.al,1996).

### **Clinical significance:**

The presence of *E.coli* is not cause in and of itself to seek treatment as it is considered harmless. However it should be noted that when a person becomes infected with this benign entamoeba, other pathogenic organisms may have been consumed at the same time. (Sodeman WA ,et.al,1996).

### **Intestinal amoebiasis, clinical features:**

We can differentiate 4 different situations in intestinal amoebiasis:

- asymptomatic carriers
- amoebic colitis
- fulminant colitis
- amoeboma

### **Asymptomatic carriers**

Trophozoites can sometimes remain in the intestinal lumen for years without causing any damage: the patient is then an asymptomatic carrier. The majority (90%) of patients fall into this group. Asymptomatic carriers have by definition no symptoms of amoebiasis.

These persons can be detected by faeces analyses. This may show cysts of non-pathogenic *E. dispar* or of potentially pathogenic *E. histolytica*, which for unknown reasons is not invasive. Differentiation with cysts of *Entamoeba coli* (which are larger and have 8 nuclei), and others, is important. *Entamoeba coli* is not pathogenic. (Sodeman WA ,et.al,1996).

### Amoebic colitis

The incubation period of amoebic colitis varies greatly. When *Entamoeba histolytica* penetrates the intestinal mucosa (becomes invasive) it produces ulcerations of the colonic mucosa [Gr. histo-lytica → breaking down tissues]. The ulcerations are sharply defined and have eroded undermined edges. This is expressed clinically as abdominal pain, diarrhoea with blood in the faeces, and only moderate or no fever, with good general condition. When the rectum is affected there is tenesmus (painful cramps in the anus). Peri-anal ulcers may occur via direct spread from rectal amoebiasis. The ulcers develop rapidly and are painful. After suffering from amoebic colitis there may be persistent intestinal problems, the aetiology of which is unclear. (Sodeman WA ,et.al,1996).



Entamoeba histolytica rectitis, with spread to the perianal skin. Copyright prof Gigase, ITM

(Sodeman WA ,et.al,1996).

### Fulminant colitis

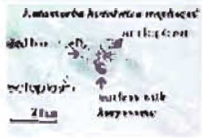
There is sometimes a fulminant course with high fever, a severely ill patient, intestinal bleeding or perforation of the colon. A slow seepage of intestinal content into the peritoneum is very likely in a severely ill patient whose condition deteriorates progressively, together with the formation of ileus (intestinal paralysis) and a distended abdomen. A fulminant course may occur if patients are treated with steroids (e.g. if amoebic colitis is wrongly thought to be Crohn's disease or haemorrhagic ulcerative colitis). (Sodeman WA ,et.al,1996).

### Amoeboma

In 1% of patients an inflammatory thickening of the intestinal wall occurs. A mass may then be palpated (amoeboma). The diagnosis may be made via biopsy. The inflammatory mass may mimic colon carcinoma. Countless trophozoites are found in the tissues (never cysts). Correct therapy produces a pronounced reduction in the volume in approximately 3 days. (Sodeman WA ,et.al,1996).

### **Intestinal amoebiasis, diagnosis:**

When amoebic dysentery is suspected, a fresh faecal sample or a swab from a rectal ulcer should be examined under a microscope. If examined quickly (a fresh stool, still warm) the colourless motile trophozoites can be seen. Motility disappears when cooled, and the parasites are then difficult to recognise. They should be differentiated from actively motile macrophages. The trophozoite (motile form) has one nucleus. When colourless this nucleus is scarcely if at all visible. Once stained the nucleus is moderately visible. Lugol staining kills the parasite almost immediately (motility disappears). Stained *Entamoeba histolytica* trophozoites have a transparent outer border (ectoplasm) and an opaque inner border (endoplasm). The border between endoplasm and ectoplasm is not distinct in *Entamoeba coli*. The trophozoite measures 20 to 40  $\mu$  m and may contain red blood cells (unlike other amoebae). The last detail is pathognomonic for pathogenic *Entamoeba histolytica*, but is not always present. Ribosomes can be arranged in characteristically shaped elongate bars with rounded ends (=chromatoid bodies).



*Entamoeba histolytica* trophozoite. Morphologically, it is only possible to differentiate *Entamoeba dispar* from *E. histolytica* if the trophozoite contains engulfed red blood cells. Only *E. histolytica* is haematophagous. Copyright ITM

The cysts have 1, 2 or 4 nuclei and measure 8 to 15-20  $\mu$  m. The nuclei are best revealed by means of an iodine stain. They have a dark circumference and a dark central point (karyosome). The karyosome of *Entamoeba coli* is not centrally located, but eccentric. Iodine staining can also detect glycogen (brown) in young cysts. Fresh cysts of *Entamoeba histolytica* also contain what are called chromatoid bodies. These are squat, oval inclusions which can easily be detected (black) with an iron-haematoxylin stain (not with iodine stain). They are not present in *Entamoeba coli* or *Endolimax nana* cysts. In active dysentery, often no cysts are found in the faeces, but if there is little **diarrhea**, the parasites have time to encysted. Since excretion of the parasites is intermittent, it is best to carry out 3 different stool analyses before deciding upon a negative result. Sometimes it is easier to reveal the parasites in a stool obtained by means of a purgative.

-Tests for tracing *Entamoeba histolytica* antigen in the feces have been developed, but need to be further evaluated. They may permit swift differentiation between *Entamoeba histolytica* and *Entamoeba dispar*. (C.E. Bennett, et.al., 2000)



## Intestinal amoebiasis, differential diagnosis:

The intestines may contain several species of harmless commensal amoeba. Differentiation with these other, non-pathogenic amoebae is important; they include:

- *Iodamoeba butschlii*: mononuclear cysts, big glycogen supply
- *Entamoeba hartmanni*: small cysts with four nuclei
- *Endolimax nana*: smaller round or oval cysts with 2-4 nuclei (measuring 6-12  $\mu$ m) and slow-moving trophozoites (L.: limax = slug)
- *Entamoeba coli*: larger cysts containing 1, 2, 4 or 8 nuclei

*Entamoeba dispar* is a special case (see above) (C.E. Bennett, et.al,2000)

In dysentery it is important to distinguish between bacillary and amoebic dysentery since their treatment is completely different. Like:

Bacillary dysentery	Amoebic dysentery
Acute onset	Gradual onset
Poor general condition	General condition normal
High fever	Little fever (adult)
Severe tenesmus	Moderate tenesmus
Dehydration frequent	Little dehydration (adult)
Feces: no trophozoites	Trophozoites present
Coproculture positive	Coproculture negative

## Intestinal amoebiasis, treatment:

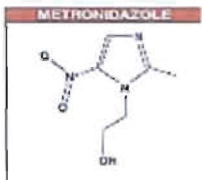
### Asymptomatic carriers

Since high percentages of the population may be cyst carriers (e.g. 10%) there is little point in treating cyst carriers found by chance in an endemic region. In any case, 90-95%

of these people are infected with the non-pathogenic *Entamoeba dispar*. If this is nevertheless desired (e.g. in people who prepare food) **diloxanide furoate** is indicated. **Iodoquinol** and **paromomycin** can be used. In regions of low endemicity it may indeed be sensible to treat the patient to prevent transmission, and also to prevent possible development of later invasive amoebiasis. (C.E. Bennett, et.al., 2000)

### Amoebic colitis

Parasites in the tissues (intestinal wall) can be treated with metronidazole or tinidazole. The dose of **metronidazole** is 500 mg q.i.d. for 5 or more consecutive days (adults). **Tinidazole** is more expensive but has fewer side effects. Alcohol is forbidden during treatment due to antabuse effect with severe nausea. These drugs are rapidly absorbed in the proximal intestine. For this reason they are insufficiently active upon the parasites in the distal intestinal lumen.



Chemical structure of metronidazole. Copyright

ITM

The latter are treated with diloxanide furoate (Furamide® = a contact amoebicide). This drug is not active, however, against parasites in the tissues. The two drugs thus complement each other. Dose: Furamide® 500 mg t.i.d. for 10 days (adults). Children: 30 mg/kg/day. Alternative contact amoebicides are iodoquinol and paromomycine (C.E. Bennett, et.al,2000)

## **9. *Entamoeba histolytica***

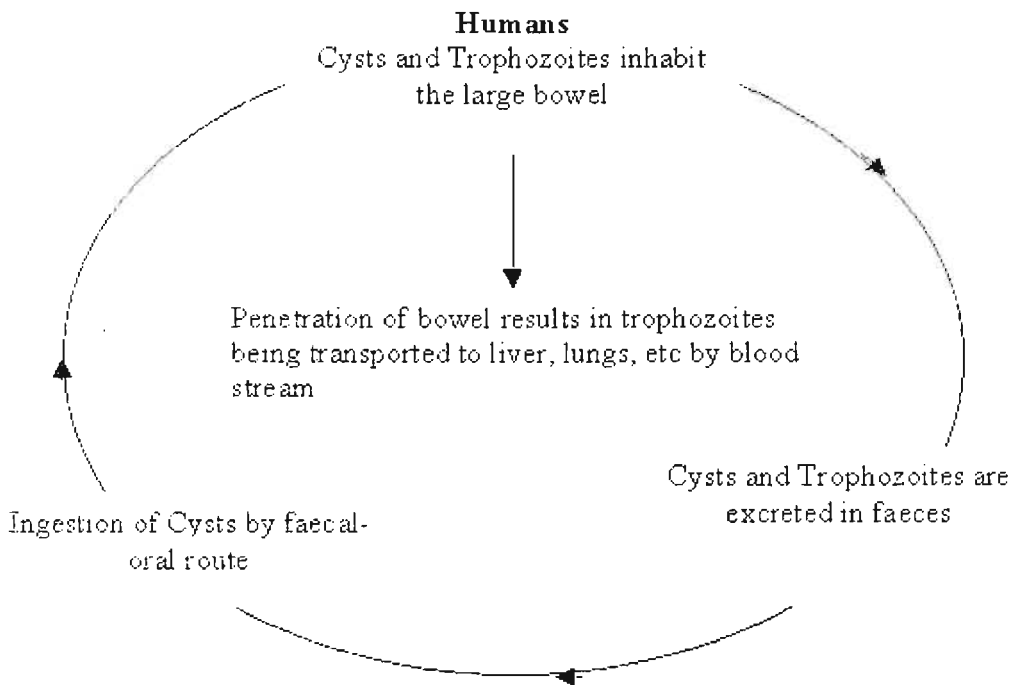
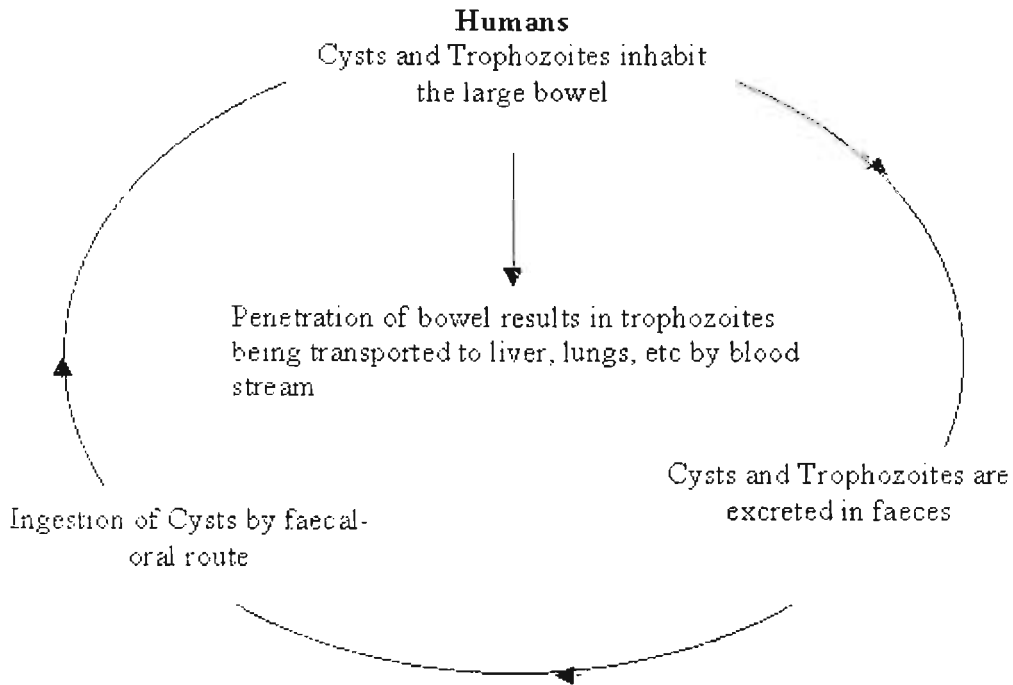
### **Introduction:**

There are a large number of species of amoebae which parasitise the human intestinal tract. Of these *Entamoeba histolytica* / *dispar* is the only species found to be associated with intestinal disease. Although many people harbour this organism world wide, only about 10% develop clinically invasive disease thus the parasite has been shown to present as two very differing clinical presentations.

1. The commensal or non-invasive luminal form where the parasite causes no signs or symptoms of disease.

2. The pathogenic or invasive form where the parasite invades the intestinal mucosa and produces dysentery or amoebomas and may give rise to extra-intestinal lesions via the blood, mainly to the liver. **(L, Polderman AM, et.al, 2003)**

*Sargeant and Williams* (1978) conclusively proved that invasive and non-invasive strains of *E. histolytica* could be differentiated by isoenzyme electrophoresis and the application of molecular biology has confirmed the presence of two distinct species with the same morphological features. The pathogenic or invasive species has retained the name *E. histolytica* and the non-pathogenic, non-invasive species has been named *E.*



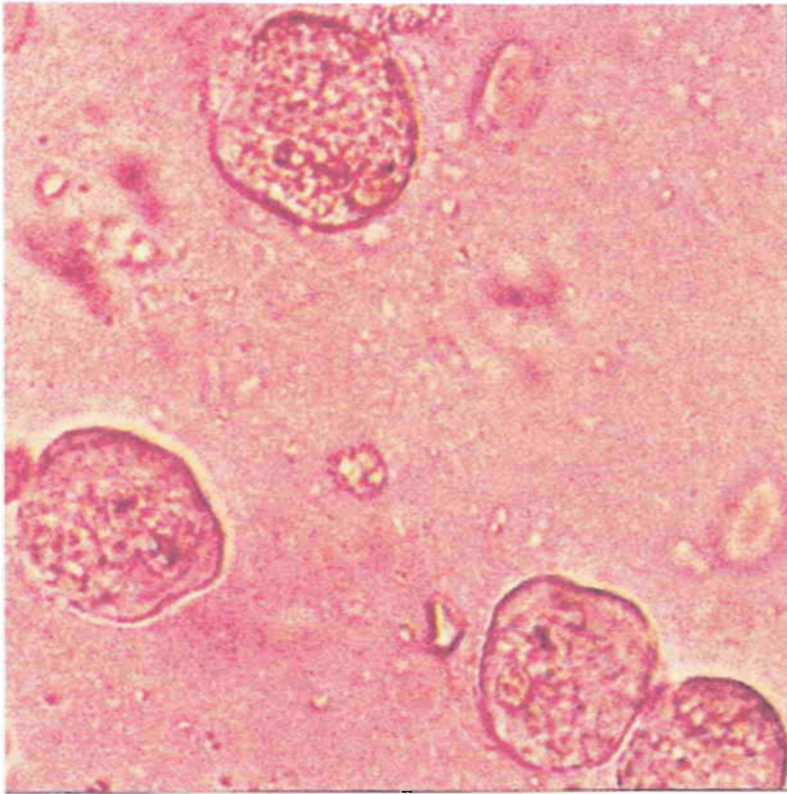
**Diagram 1:** Life Cycle of *Entamoeba histolytica*: A protozoan in which its life cycle consists of two stages; cysts and trophozoites

### **Morphology of Trophozoites:**

The trophozoites of *E. histolytica / dispar* recovered from dysenteric stools exhibit ingested red blood cells and clear pseudopodia. Those of *E. dispar* will have no ingested red blood cells. They can be up to 60µm in diameter and motility is rapid and unidirectional. On a permanently stained faecal smear e.g. Trichrome or Iron haematoxylin, the morphological features are more visible. When using Trichrome stain nuclei, chromidial bars, chromatin, red cells and bacteria stain red cytoplasm stains blue-green and background and yeasts stain green. The presence of a small centrally placed karyosome is clearly visible. With Iron haematoxylin, nuclear chromatin and the karyosome will be stained immensely black. The remainder will be varying shades of grey/black. (Fig 1)

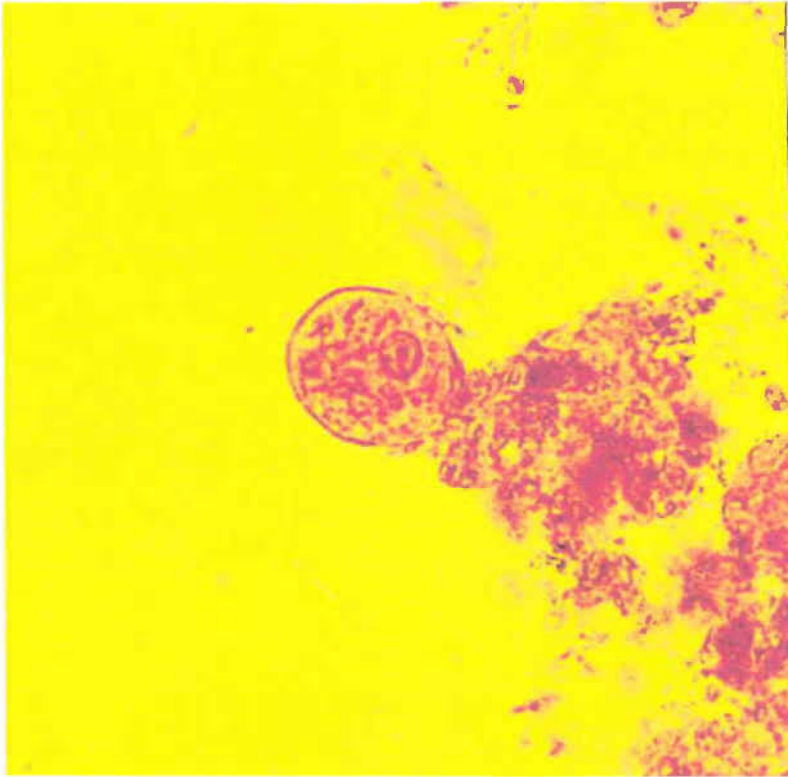
### **Morphology of cysts**

Cysts of *E. histolytica / dispar* are 10 - 15µm in diameter and contain 1 - 4 nuclei. Chromatoid bodies are usually present in young cysts as elongated bars with bluntly rounded ends. Glycogen is usually diffuse, but in young cysts it is often present as a concentrated mass, staining reddish brown with iodine.(fig-2) **(L, Polderman AM, et.al, 2003)**



**Fig 1. *Entamoeba histolytica* trophozoites in a caecum biopsy, each with pseudopods. (<30µm)**





### **Intestinal disease:**

Patients with intestinal disease may exhibit a number of symptoms including profuse diarrhea with blood and mucus, fever and dehydration. Amoebic ulcers may develop in the large colon and can also be found in the rectal area. The ulcers are usually "flask shaped" with a small opening on the mucosal surface and a larger area below the surface. Fig 3 illustrates *E. histolytica* trophozoites in the intestine, resulting in amoebiasis. (L,Polderman AM,et.al,2003)

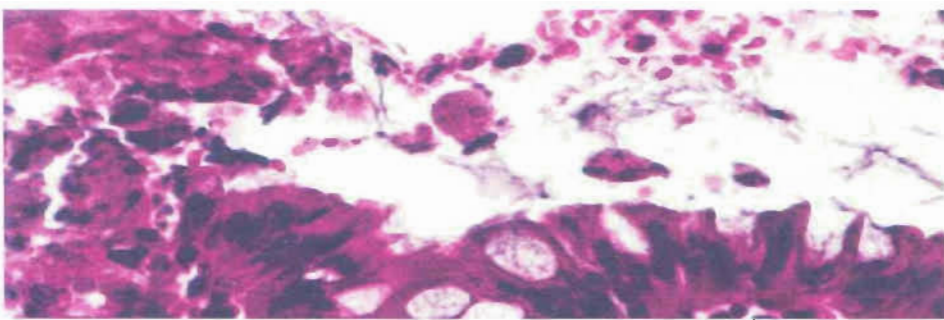


Fig 3. *Entamoeba histolytica* trophozoite present in the intestine causing Intestinal amoebiasis. (HES stain). Note in the centre of the picture trophozoites stained red. The nucleus is situated on the right in the amoeba. The central karyosome and the nucleus membrane with its chromatin are distinct.

## **Hepatic Disease:**

Trophozoites are transported from the intestine to the liver and liver disease is characterised with abdominal pain, fever, hepatomegaly and tenderness. If the abscess ruptures, there is spreading to the brain, pericardium and other sites. If left untouched the abscess will grow normally until it reaches a surface where it can discharge, e.g. the skin, the peritoneum, the pleural cavity or the pericardium. The stretching of the liver is presumably the main source of the pain. (L,Polderman AM,et.al,2003)

## **Serology:**

If visceral or hepatic amoebiasis is suspected serological tests should be done as microscopic methods do not always reveal the characteristic trophozoites. The tests of choice are indirect fluorescent antibody test (IFAT), counter immunoelectrophoresis (CIEP) and enzyme linked immunosorbent assay (ELISA). (L,Polderman AM,et.al,2003)

The search for *E. histolytica* / *dispar* is mainly carried out in Europe and North America, as there is a natural concern to ensure that patients, even in the absence of symptoms are not harbouring parasites that may lead to serious complications later on.

## **Treatment of diarrhea:**

### ***Nonspecific Agents:***

A variety of "adsorbents" have been tried in treating diarrhea. Activated charcoal has been found to be ineffective in the treatment of diarrhea. Kaolin and pectin have been widely used for diarrhea. The combination appears to give the stools more consistency but has not been shown to decrease cramps and frequency of stools nor to shorten the course of diarrhea. Lactobacillus preparations and yogurt have also been advocated; there is one study which suggests benefit. Bismuth subsalicylate preparations (1 oz of liquid or one tablet every 30 minutes for eight doses) have been shown to decrease (but not necessarily abolish) diarrhea and shorten the duration of illness in several placebo



controlled studies. There is concern about taking, without medical supervision, large amounts of bismuth and salicylate, especially in individuals who may be intolerant to aspirin or aspirin-like medicines, who have kidney disease or who take salicylates for other reason. **(The American Journal of Medicine, June 1999)**

### ***Oral fluids:***

Most cases of diarrhea are self limited and require only oral replacement of fluids and salts which have been lost in diarrheal stools. Fluid and electrolyte balance can be maintained by (safe) fruit juices, soft drinks (preferably caffeine-free and alcohol-free) and salted crackers. Iced drinks and noncarbonated bottled fluids made from water of uncertain quality should be avoided. Dairy products aggravate diarrhea in some people and should be avoided. Travelers may prepare their own fruit juice from fresh fruit. Individuals with dehydrations may require fluid and salt replacement in the form of Oral Rehydration Solution (ORS) recommended by the World Health Organization (see article in "*Health Information for International Travel*" – The Yellow Book). Each ORS packet, available at stores or pharmacies in almost all developing countries, should be added to a liter of boiled or treated water, and consumed or discarded within 12 hours if held at room temperature, or within 24 hours if held refrigerated. **(Victora CG, et.al, 2000)**

### ***Antimotility***

*Agents* which act directly on the bowel may slow diarrhea of any cause and should only be used if significant abdominal pain, significant vomiting, fever (over 100.5 degrees F) and bloody diarrhea are absent. Natural opiates (codeine and others) have long been used to control diarrhea and cramps. Synthetic agents such as loperamide (available as 2 mg pills without prescription both generically and as brand name Imodium) usually provides prompt, temporary symptomatic relief of uncomplicated TD. The usual dose is 2 tablets at onset. Loperamide can worsen bacterial dysentery and can mask worsening infection, therefore its use is discouraged. Using one dose of loperamide under extreme unusual circumstances might be reasonable, but if used, antibiotic treatment should be given for a full 5 days. Diphenoxylate (brand name Lomotil) is less effective, available by

prescription only and is not recommended. Neither diphenoxylate nor loperamide should be used in children under the age of 2 years. (Alam NH,et.al(2003).

**Antibiotic Treatment:**

Travelers who develop diarrhea (any watery stool) may benefit from antibiotic treatment. A typical three day illness can often be shortened to one day with early self treatment with an antibiotic. Those with the following should be evaluated by a local physician promptly: fever, more than mild vomiting or mild abdominal pain, blood mixed with diarrhea, or diarrhea which is severe or which does not resolve within 48 hours. Nausea and vomiting without diarrhea should not be treated with antibiotics. (Alam NH, et.al.(2003).

Options for antibiotic treatment include ciprofloxacin (Cipro), norfloxacin, TMP/SMX (Bactrim, Septra), trimethoprim and azithromycin (Zithromax); doses of two of these antibiotics are listed below. For ordinary TD, antibiotics and antimotility agents may be stopped when normal stools resume.

	ordinary (minor diarrhea)	TD	bacterial (major diarrhea)	dysentery
signs:	watery stool without other signs		fever, abdominal pain, vomiting, blood mixed with diarrhea, failure of initial antibiotic treatment or appears systemically ill	
most places	cipro 500 mg, one twice a day for 1-2 days; if diarrhea stops completely, stop cipro		if diarrhea continues (even if a bit less), continue cipro 500 mg twice daily for a total of 5 days for possible dysentery	
an option	azithromycin 250 mg, 2 on the		if diarrhea continues (even if a bit	

especially for South Asia	first day; if diarrhea stops completely, stop azithromycin	less), continue azithromycin 250 mg one daily for 4 more days (5 days total) for possible dysentery
---------------------------	--	--

Medication	dose
ciprofloxacin (Cipro) 500 mg (do not use in pregnancy)	after first liquid stool, take 1 tablet daily for 1-2 days; if diarrhea continues, take 1 tablet twice daily for a total of 5 days
azithromycin (Zithromax) 250mg	after first liquid stool, take 2 tablets at onset; if diarrhea continues, take 1 tablet daily for 4 more days

This plan should be 97% effective. It will not treat giardia or amebic dysentery. Travelers who are very ill, or in whom this plan is not effective, should be seen by a physician.

Children under the age of 2 years should have individualized management by their physician. .(Alam NH, et.al (2003).

### **Nutritional management:**

#### **Breast milk:**

It is recommended that breast-feeding should be continued, and even encouraged, during episodes of infant and neonate diarrhea. .(Alam NH, et.al (2003).

#### **Vitamins and minerals:**

All patients Diarrhea should get supplemental vitamins and minerals (retinol, folic acid, Zinc) at about twice the dose of the recommended daily allowance for 2-3 weeks to

improve mucosal repair and improve intestinal functions, as well as to replace the existing deficiency. As a guide one recommended daily allowance for a child aged 1 year is folic acid 50 µg, zinc 10mg and retinol 400 µg. Other vitamins and minerals including pyridoxine (vit B6), cyanocobalamin (vit B12), Ascorbic acid (vit C), Ergocalciferol (Vit D2), tocopherol (vit E), phytomenadione (vit K), Thiamine (vitamin B1), nicotinic acid, riboflavin (vitamin B2), calcium pantothenate, biotin, calcium, phosphorus, magnesium, iron, copper, iodine, selenium, manganese, cobalt, molybdenum at about twice the dose of the recommended daily allowance should also be supplemented in diarrhea. Locally available commercial preparation of vitamins/minerals mixture are usually suitable for use. (Alam NH, et al (2003)).

### **Infant diarrhea:**

Signs of diarrhea in infants may include such things as an increased number of bowel movements that may appear with other symptoms often caused by infections (such as acting tired, or having a fever). One of the most common causes of diarrhea among infants is viral gastroenteritis (the "stomach flu"). When it occurs in an infant, diarrhea can present special health concerns; infants with diarrhea can become dehydrated in a short period of time. Using oral rehydration solutions can help prevent or treat dehydration while the infant recovers. (MW Peterson, et al. 1976)

### **Whether normal or infant diarrhea:**

Diarrhea is defined as loose, watery, unformed stools occurring more than three times in one day. It is not the occasional loose stool or the frequent passing of formed stools.

However, it seems like every bowel movement with infants fits the definition of diarrhea. They are runny, unformed, and frequent; they have various odors and colors; and in most cases, these bowel movements are normal. So how do you tell if your infant has diarrhea?

First of all, ask yourself if your infant has experienced a change in the frequency of his or her bowel movements. Each baby has his or her own pattern of bowel movements. If,

over the last couple of days, your infant has had more bowel movements than usual, he or she may have diarrhea.

Another clue is whether or not your infant has any other symptoms. Because diarrhea can be caused by viruses or bacteria, it is not unusual for other symptoms to be present with diarrhea. This may include your infant acting sick, not wanting to feed, acting tired, having a runny nose, or fever.

Other questions that parents may ask themselves to help determine if their infant has diarrhea include the following:

- Have there been any changes in the diet (including their diet if you are breastfeeding)?
- Is their infant teething? In some infants, teething can cause diarrhea, and diarrhea can be a first sign of teething.
- Is their infant taking any medicines, such as antibiotics?

If you have answered yes to any of these questions, it is possible that your infant has diarrhea. In the rest of this article, we will discuss causes of infant diarrhea, what you should do if your child has diarrhea, and when you should see your healthcare provider.

### **Infant diarrhea prevention and treatment:**

In infants and children (especially under age 3), diarrhea is more concerning. Children can become dehydrated fairly quickly. Diarrhea in infants can cause abdominal distress and cramping, which may disrupt sleep, frequent, watery stools, and in more serious situations, pus or blood in the stools, irritability, less interest in feeding, loss of appetite, sluggishness and less activity than usual and vomiting.

Most acute diarrhea in infants and young children is due to viral gastroenteritis and is usually short-lived. Antibiotics are not routinely prescribed for viral gastroenteritis. However, fever, vomiting, and loose stools can be symptoms of other childhood infections such as Otitis Media (infection of the middle ear), Pneumonia , bladder infection, sepsis (bacterial infection in the blood) and meningitis. These illnesses may require early antibiotic treatment.

Infants with acute diarrhea also can quickly become severely dehydrated and therefore need early rehydration. For these reasons, sick infants with diarrhea should be evaluated by their pediatricians to identify and treat underlying infections as well as to provide instructions on the proper use of oral rehydration products.

Infants with moderate to severe Dehydration usually are treated with intravenous fluids in the hospital. The pediatrician may decide to treat infants who are mildly dehydrated due to viral gastroenteritis at home with ORS.

Infants that are breast-fed or formula-fed should continue to receive Breast milk during the rehydration phase of their illness if not prevented by vomiting. During, and for a short time after recovering from viral gastroenteritis, babies can be lactose intolerant due to a temporary deficiency of the enzyme, lactase (necessary to digest lactose in milk) in the small intestine. Patients with Lactose Intolerance can develop worsening diarrhea and cramps when dairy products are introduced. Therefore, after rehydration with ORS, an undiluted lactose-free formula and diluted juices are recommended. Milk products can be gradually increased as the baby improves. (MW Peterson, et al.1976)

### **Neonatal diarrhea:**

Always regard diarrhea as a SYMPTOM of a more basic illness which can take many forms. Although we need to start treating the symptom right away so things don't get out of hand, we need to be constantly trying to discover the real root of the problem. This can be an organism, an environmental factor or something as simple as too much milk. Generally it is not practical to collect feces or bodily tissue for complex laboratory testing. Instead, we have to rely on certain clues based on experience to help us figure out

what is wrong and what to do about it. Below, you will find lists. In the first, you can use the color of the feces or general descriptions to go to a page for the disease which matches those symptoms. The second list contains some of the more common causative agents of neonatal diarrhea for speedy cross reference. (MW Peterson, et al.1976)

### Symptom of neonatal diarrhea:

- Under 5 da, yellow, profuse watery, low temp
- Over 10 da, grayish, foul odor, bloody, shreds, temp
- Dark, bloody, tissues, weight loss, soiled
- Watery brown to black with mucous and blood (rare)
- White, sudden
- Big eaters, abdom pain, bloody, die with milk in belly
- Chronic runs, unthrifty, anemia, swollen belly
- Profuse watery, weakness
- Watery with mucous and fever
- Creamy w/ gas bubbles, wt loss, tenesmus (rare)

### Disease caused by:

- Campylobacteriosis
- Chlamydial
- Coccidiosis
- Cl perf C/D
- Cryptosporidiosis
- E. coli
- Malabsorption
- Nutritional scours
- Salmonella
- Viral diarrhea



### **Prevention of neonatal diarrhea:**

The following steps will help to prevent diarrhea in its many forms:

Make sure the kid is not overfed or underfed.

Vaccinate the mother with CD/T and give a Vitamin A/D shot 3 weeks before delivery.

Cleanliness is extremely important. This includes careful cleaning of birthing stalls, bedding, bottles, and udders to eliminate ingestion of organisms wherever possible.

Cut milk amounts in half at the first sign of loose bowels (for one or two feedings only).

Never let a case of diarrhea get away from you. Start treatment right away

On the other hand, loose bowels may be the result of eating too much green grass and clears up in a few hours on its own. Just keep a close watch. (MW Peterson, et al.1976)

### **Diarrhea treatments in general:**

If the kid is severely distressed (extremely runny, signs of dehydration such as unresponsive skin and/or sunken eyes), mix up a dose of "alkaline" electrolytes (or whatever kind you have). Most of the packages are designed for calves, so we will have about a half gallon of this stuff, which is much more than we need. Put about 8 ounces of this in a pop bottle and warm it to body temperature. Put a lamb nipple on the pop bottle.

### **10 million children dying every year due to diarrhea:**

More than 10 million children die each year, most from preventable causes and almost all in poor countries. Six countries account for 50% of worldwide deaths in children younger than 5 years, and 42 countries for 90%. The causes of death differ substantially from one country to another, highlighting the need to expand understanding of child health epidemiology at a country level rather than in geopolitical regions. Other key issues



include the importance of undernutrition as an underlying cause of child deaths associated with infectious diseases, the effects of multiple concurrent illnesses, and recognition that pneumonia and diarrhea remain the diseases that are most often associated with child deaths. A better understanding of child health epidemiology could contribute to more effective approaches to saving children's lives.( **B Young, et.al,University of North Carolina**)

# Chapter 3

## **Objective of the study**

Diarrheal disease remains an important cause of childhood morbidity and death in developing countries. ORT was introduced in 1979, besides this powerful interventions also implemented. More than 10 million children die each year, most from preventable causes and almost all in poor countries. 6 countries account for 50% of worldwide deaths in children younger than 5 years, and 42 countries for 90%.

Our objective of the study to know about the treatment of neonates and infants other than the ORT. For example, Kancha kola, Chira water, khichuri, and Lactose free milk. Besides this antibiotic and non antibiotic treatment, the common symptom of the neonate and infant, what percentage of neonate and infant attacked by diarrhea etc were under consideration.

We studied a retrospective analysis to compare the infants (>1 years) and neonates (<6 months) in terms of sex, symptoms, treatment profile of diarrheal disease those were admitted in Child Health and Shishu Sasthya Foundation Hospital.

The objective of the study was to describe what are the differences between the children less than 6 months and the children more than 1 year those were admitted to Hospital.

# Chapter 4

## **Significance of the study**

In 2003 an estimated 1.87 million children below 5 years died from diarrhea. On average, children below 3 years of age in developing countries experience three episodes of diarrhea each year. Diarrhea, cholera are the important cause of morbidity of adult and children.

Though diarrhea disease is a major cause of child morbidity so lots of study has already been taken to find out the problems. Our study will help to learn about the neonate and infant diarrhea in developing countries like Bangladesh. There are many factors involved here one of them is socioeconomic condition. Poor and illiterate people have no idea about the balance food, nutrition. So they are mostly attacked by diarrhea.

From Our study analysis we came to know about the treatment, common symptom, who are very much prone to diarrhea disease, the recovery time for neonate and infant, the antibiotic treatment, no of male and female patient etc.

This information gives us a clear vision about children diarrhea in a particular region of Bangladesh, which may increase the awareness about diarrhea.

# Chapter 5



## **Materials and Method**

### **1. Research design and place of study:**

It is a retrospective study between infant and neonate diarrhea. It was attempted to establish relationship between the patient of more than 1 year old and the patient of less than 6 months. January 2009 was used for this analysis. The study was conducted in Institute of Child Health of Shishu Sasthya Foundation Hospital, Mirpur, Dhaka)

### **2. Sample size:**

The study was based on 50 patients those were admitted into hospital in January 2009.

### **3. Research Approach:**

After getting the approval of the research proposal from the honorable faculty members, formal permission was obtained from the competent authorities of “Shishu Sasthya Foundation”(SSF).

### **4. Data collection method:**

The data were collected from the hospital record.

### **5. Patient information:**

Children aged 1-144 months were eligible for the present analysis. The following information was abstracted from the record of each child:

- age,
- sex,
- anthropometric measurements (weight-for-age, type of feeding, duration of diarrhea, number of stools during the last 24 hours, dehydration status, history of taking oral rehydration solution (ORS) or antimicrobial/antidiarrhoeal drugs

before admission and presence of acute lower respiratory tract infection diagnosed clinically,etc.

### **5.1. Symptoms and pathological information:**

- Loose motion
- Loose stool
- Loose motion with vomiting
- Fever
- Cough
- Convulsion
- Difficulty in Respiration
- Loss of appetite
- Feeding pattern
- H/O pain in umbilical region
- Previous clinical history

### **6. Diagnosis of diarrhea patient:**

This study was based on 50 consecutive patients of diarrhea admitted in hospital for treatment and irrespective of age, sex, and symptom. All patients possess of different types of diarrhea with various symptom. The symptoms were diagnosed on the basis of following parameters:

- Whether organism present or not.
- Causes of diarrhea
- Diet
- Sociodemographic features
- Antecedents



## **7. Investigation of Patient:**

The investigation parameters are:

- CBC
- Stool culture
- Blood culture
- Electrolytes

## **8. Data analysis:**

All the collecting data were rechecked. The data was entered into the computer with the help of SPSS windows programmed version 12.0. Then pie charts and bars were made by using these data in Microsoft Excel.



# Chapter 6

## Case report form

This data collected from ICH & SSF hospital record of January 2009.

Here,

- Age in month.
- DOA means “Date of admission”
- DOD means “Date of disperse”
- NPO means nothing per oral.
- H/o means “History of”.
- Inf means “Infusion”.
- Inj means “Injection.”

Name	Age	Sex	DOA	DOD	symptom	Treatment
Sumayra	13	Female	3/1/2009	4/01/09	no passage of urine,verfolin	NPO,FO normal saline,supp DonA Lactose free milk,Kolosal,Ace suppository
Sabbir	4	Male	16/01/09	17/01/09	loose watery stool,vomiting	NPO,FO,inj-Koloride,otosol,Don A- suppo,rice saline
Oishi	8	Female	15/01/09	17/01/09	loose watery stool,vomiting	Inf Koloride 500ml
Aushi	6	Female	2/1/2009	5/1/2009	Passage of loose stool,dehydration,vomiting	NPO,Inf Koloride 770ml
Atia	27	Female	15/01/09	17/01/09	vomiting	NPO,O2 inhaler
Thamid	0 25	Male	27/01/09	28/01/09	H/o vomiting	Inf Cholera saline.Diet normal saline
Jannatul	0 1	Female	26/01/09	28/01/09	Loose motion,vomiting,loose stool,vomiting	NPO,Inf 10% Koloride,ORS,lactose free milk,chira
Osama	7	Male	27/01/09	28/01/09	loose stool,vomiting	Diet normal inf 10% Koloride 500ml
Soad	8	Male	24/01/09	27/01/09	loose watery stool	NPO,Inf 10% Koloride,Ace suppository,ORS
Ayon	17	Male	20/01/09	22/01/09	loose watery stool,vomiting	NPO,Inf 5%
Prionto	8	Male	20/01/09	24/01/09	loose watery stool,vomiting	Koloride,CiproA,injection,Otocil,ORS Inf 10% Koloride,Diet breast feed on demand
Mahima	6	Female	21/01/09	23/01/09	loose watery stool,fever,cough,coid	Npo,Inf Koloride1000ml,inj Ciprozid IV
Anika	144	Female	21/01/09	23/01/09	loose watery stool,vomiting	NPO,Inf 10% Koloride
Essan	9	Female	20/01/09	23/01/09	vomiting after feeding,loose watery	Inj Ceftron,Inf hartsoI,Inf Lebac,Inj Fluelox,Napa,histal
Yasin	7	Male	11/1/2009	22/01/09	Loose watery,fever	Koloride,inj Aciphin 750mg,Ors,Ace suppository
Tayef	12	Male	17/01/09	22/01/09	fever high,loose motion,cough	NPO for 4hrs,inf 10% Kolosal,supp Ace
Abrar	22	Female	18/01/09	22/01/09	Fever,vomiting,passage of loose mucoid stool	100ml Kolosal,inj Taxim,Inj Aceutin,O2 inh,Sedil
Baby	0 6	Male	20/01/09	22/01/09	Passage of loose stool,relactant to food	Inj Koloride,inj Ciprozid,Tapid,syrup Otosil
Was	10	Male	18/01/09	20/01/09	Loose motion,vomiting,high fever,cold	5% Koloride 270ml,Lactose free milk
Babu	1	Female	15/01/09	28/01/09	Passage of loose watery stool	NPO,inf Dextrolac,inj Maprocin,inj Filmct,inj Butapan
Hafsa	72	Male	20/01/09	28/01/09	H/o pain in ambilical,vomiting	NPO,10% Koloride 560ml,Cap vit A
Rabbi	18	Male	23/01/09	24/01/09	loose watery stool,vomiting	Diet liq,inj10% Koloride.Syrup Ace.Sedil
Protoy	72	Male	17/01/09	23/01/09	loose stool,fever,loss of appetite	

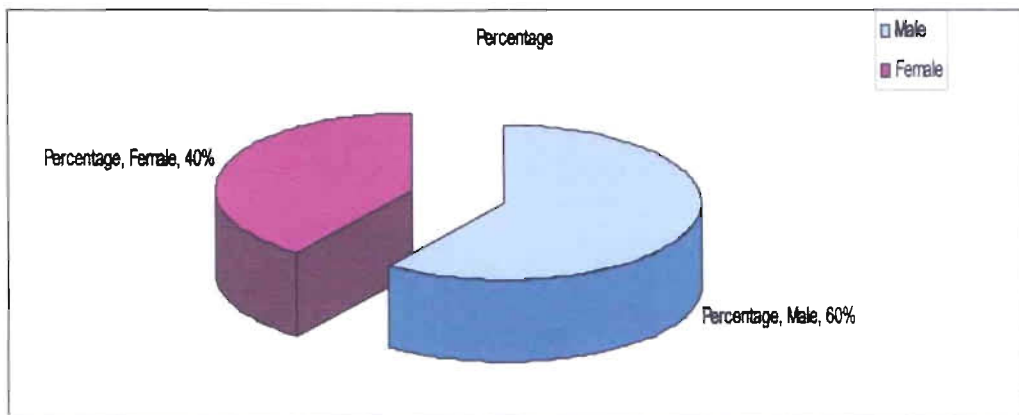
Samim	24	Male	23/01/09	23/01/09	loose motion, repeated vomiting, fever	NPO for 4hrs, inj 10% Koloride 560ml
Tahsin	17	Male	22/01/09	24/01/09	loose watery stool, vomiting, fever	NPO for 4hrs, inf Koloride 670ml
Keam	3	Male	21/01/09	24/01/09	loose watery stool	NPO, inf 5% Koloride, inj Ciprozid, inj metryl, inj Ranison
Nahida	25	Female	21/01/09	23/01/09	loose watery stool, vomiting, fever	NPO, inf 10% Koloride
Asa	15	Female	21/01/09	24/01/09	loose watery stool, vomiting	NPO, inf 5% Koloride 600ml
Sharin	7	Female	22/01/09	23/01/09	Passage of loose watery, vomiting, fever	NPO, inf 5% Koloride 650ml, Syb Ace, syp otosil
Babu	1	Male	21/01/09	23/01/09	Fever, H/o delayed crying, respiration prob	NPO, inf 10% Don A, inj Maxcef, inj Gentin
Nadia	12	Female	6/1/2009	7/1/2009	loose motion, vomiting	NPO, 5% Koloride, inj Ciprozid, syp Metryl
Nabil	12	Male	8/1/2009	10/1/2009	Passes of loose watery, vomiting	NPO, inf Koloride, ORS, Civox
Juma	14	Female	6/1/2009	8/1/2009	Respiratory distress, cough, cold, convulsion	02 inhalation, inj Ampicillin, inj Gentin, Sulbutamol
Kasfi	11	Male	7/1/2009	8/1/2009	vomiting, passes of loose stool	NPO, inf Koloride
Sabbir	12	Male	8/1/2009	9/1/2009	loose watery stool, vomiting, fever	NPO, inf Koloride, Ace
Milhi	15	Female	9/1/2009	10/1/2009	loose motion, fever, vomiting	suppository, Nebulized
Foyez	021	Male	5/1/2009	7/1/2009	loose watery stool	Diet Kacha Kala, inj Koloride 560ml
Anan	11	Male	8/1/2009	9/1/2009	Fever, vomiting, loose mucoid	Brest feeding on demand, inf 10% Koloride 250ml
Abir	5	Male	3/1/2009	8/1/2009	vomiting, loose stool	NPO, inf 10% Libotts, junior, inj Diceponin
Asia rah	20	Female	7/01/01/09	8/1/2009	Vomiting for about 6 times	NPO-UFO, inf 10% Koloride, inj Civox 35ml
Mahtab	7	Male	6/1/2009	7/1/2009	passage of loose stool, vomiting	NPO, inf 5% Koloride, ORS
Authe	15	Female	6/1/2009	8/1/2009	Cough, cold, Respiratory distress	NPO, inf Koloride 650ml, inj Ciprox, syp Otosil
Fahad	12	Male	13/01/09	15/01/09	loose motion, vomiting	Diet normal, Nebulization
Pranto	5	Male	14/01/09	15/01/09	loose watery stool	NPO, Saline 100ml
Nahian	13	Male	15/01/09	16/01/09	loose watery stool, vomiting	inf Koloride 400ml, syp: Eromyia, syp: Napa
Sayed	51	Male	13/01/09	15/01/09	Repeated vomiting	Diet normal, inj 10% Koloride, syp: otosil, DonA supp
Tusar	24	Male	2/1/2009	4/1/2009	loose and vorfolin	NPO, inj 10% baby saline, inj Libott junior
Alif	10	Male	17/01/09	20/01/09	loose motion, vomiting	NPO, inj Koloride 500ml
Jarif	8	Male	12/1/2009	15/01/09	Fever for 2 days, vomiting, loose stool	NPO, inj Koloride, inj Ciprozid, Otosil, inj Gentin, metro
Nisat	5	Female	8/1/2009	10/1/2009	loose motion for 2 days, vomiting	NPO, inf Koloride 500ml
						NPO for 4hrs, inf Koloride

# Chapter 7

## RESULT

**Table: 1**

**Percent distribution of male and female patient:**



**Fig 1: Number of male and female patient**

**There were 50 patients, where 30 were male and 20 were female. That means about 60% male and 40% female patient.**

**Table: 2**

**Age distribution of the patient:**

<b>Month</b>	<b>No of patient</b>
<b>1-10</b>	<b>27</b>
<b>10-20</b>	<b>16</b>
<b>20-30</b>	<b>3</b>

From the above table we can see that majority of the patient of 1-10 month, 16 patient were from 10-20 month, 3 patient from 20-30 month and rest of the patient were above 30 month.

**Table: 3**

**Number of neonates and infants:**

<b>Sex</b>	<b>Neonates</b>	<b>Infants</b>
<b>Male</b>	<b>10</b>	<b>20</b>
<b>Female</b>	<b>4</b>	<b>16</b>

From the above table we can see that, in male patient 10 were neonate and 20 were infant. In female patient 4 were neonate and 16 were infants.

Table: 4

Percent distribution of infant and neonate:

Age of the patient	No of patient	Percentage
Infant > 1 year	37	74
Neonate < 6 month	13	26

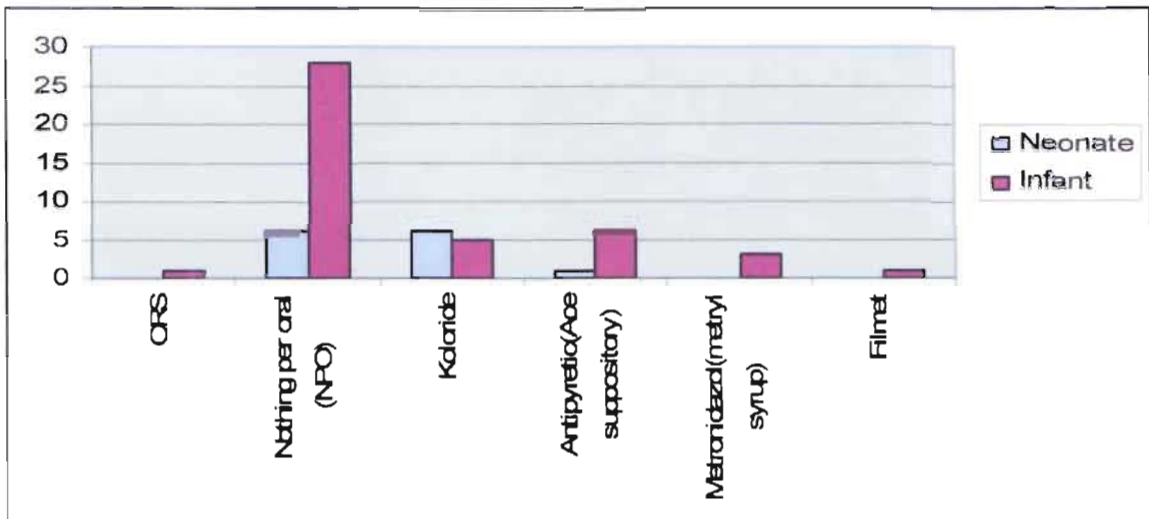
There were 50 patients, where 37 were infant (more than 1 year) and 13 were neonate (less than 6 month). 74% were infant and 26% were neonate.





**Table: 5**

**Treatments of the patient :**



**Fig 2: Treatment other than antibiotic**

This table shows that NPO was given to 32 patients, Koloride was given to 36 patients, antipyretic (Ace, Ace suppository) given to 11 patients; Metronidazol (syrup) was given to 3 patients.

**Table: 6**

**Antibiotic treatment:**

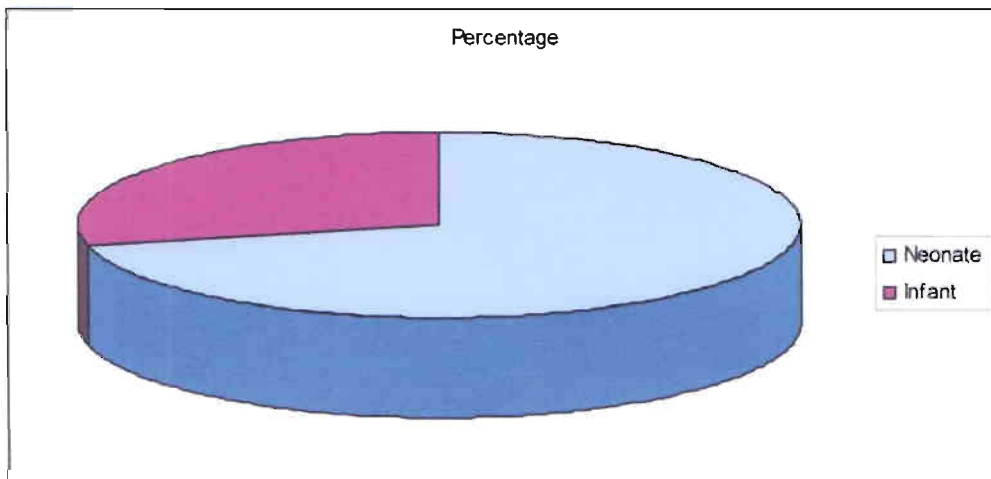
<b>Name of the antibiotic</b>	<b>No of Neonate</b>	<b>No of Infant</b>
<b>Ampicillin</b>	0	1
<b>Ciprozid</b>	1	4
<b>Ciprox</b>	0	2
<b>Civox</b>	1	1
<b>Ceftron</b>	0	1
<b>Fluclox</b>	0	1
<b>Maprocin</b>	0	1
<b>Maxcef</b>	1	0
<b>Taxim</b>	1	0

**From the above table we know that mostly infant were treated by antibiotic. Only few number of neonate treated by antibiotic. The common antibiotic is Ciprozid.**

**Table: 7**

**Percent distribution of antibiotic treatment:**

Age Distribution	Antibiotic Treatment	Percentage
Neonate	10	71.42857143
Infant	4	28.57142857



**Fig: Percent distribution of antibiotic treatment**

**From the above table we get, about 71% neonate and 28% infant were treated by antibiotic.**

**Table: 8**

**Symptom of the patient:**

The patient of “shishu sasthya foundation” was admitted mainly with dehydration, loose motion, loose watery stool, vomiting, fever etc. Loose motion with vomiting was common.

<b>Symptoms of patients</b>	<b>Neonates</b>	<b>Infants</b>
Loose watery stool	7	13
Vomiting	4	30
Loose motion	1	8
Fever	1	11

**Table: 9**

**Recovery duration:**

Recovery of the patient find out from the date of admission and date of discharge data.

<b>Duration(days)</b>	<b>No of neonates(from 13)</b>	<b>No of infants(from 37)</b>
1-3	7	25
4-6	5	9
7-9	0	1
10-12	1	2

Maximum infant were recovered by 1-3 days. Maximum neonate were also recovered by 1-3 days. Then some required 4-6 days. Very few were recovered by 10-12 days.

**Table: 10**

**Percent of recovery time for neonate and infant:**

<b>Duration</b>	<b>% of neonate</b>	<b>% of infant</b>
<b>1-3</b>	<b>53%</b>	<b>67.5%</b>
<b>4-6</b>	<b>38%</b>	<b>24.3%</b>
<b>7-9</b>	<b>0</b>	<b>2.7%</b>
<b>10-12</b>	<b>7.69%</b>	<b>5.4%</b>

**From the above table we came to know that 67% infant require 1-3 days, 53% neonate require 1-3 days. 24% infant require 4-6 days, 38% neonate require 4-6 days.**



# Chapter 8

## Discussion

Diarrhea diseases remain important causes of childhood morbidity and death in developing countries. In the early 1980s, diarrhea was the leading cause of child mortality, accounting for 4.6 million deaths annually worldwide, excluding China and Latin America. Efforts to control diarrhea over the past decades have been based on multiple, potentially powerful interventions implemented more or less simultaneously. Oral rehydration therapy (ORT). Oral rehydration therapy was introduced in 1979 and rapidly became the cornerstone of the program for the control of diarrhea disease. The annual number of deaths attributable to diarrhea among children <5 years of age has fallen from the estimated in 1980 to about 1.5 million today. **(Alam NH, Ashraf H 2003).**

Diarrhea diseases remain important causes of childhood morbidity and death in developing countries although diarrhea having significantly declined. The objective of the study was to find out the how many infants (>1 years) and neonate (<6 year) attacked by the diarrhea admitted to Child health and Shishu Sasthya Foundation Hospital. Among the 50 patients 37 were infant and 13 were neonant. Hospital record of children was reviewed and information was collected for this study.

Most of the patients were male (about 30). Female patient were less in number (about 20). The patient had various symptom commonly diarrhea with fever. Vomiting, cough, cold respiratory problem were also observed in the children. 14 patients were treated with antibiotic and 36 patients did not receive any antibiotic. From the result of the study we came to know that 74% infant was admitted and 26% neonate admitted into the hospital. 71.42% neonate and 28.57% infant were taken antibiotic.

From 14, 10 were infant and rest of them was neonate. The common treatments were ORS, antibiotic (ampicillin, ciprofloxacin, flucloxacilin). Most of the patients received intravenous. The children also received khichury, green banana, Chira water and green coconut water, breast milk etc.

From total population 60% male and 40% female patient. Here is the Table 1 that represents the number of patient admitted in Dhaka Shish hospital in January 2009. We can see that the ratio of male and female patient is not same. The number of male patient is higher than the female patient. This is a common phenomena for the third world country. Bangladesh is a country where 47% people live below the poverty line. Gender discrimination is also common here. Male patient is in highest number only for this reason. People think that only male child will take care of their parents in future not the female. Girls will get married and go to their husband's house after growing up. So it will not be a wise decision to take care about a girl. When the girl becomes sick they are not treated by the doctor. Parents think it is the wastage of money. But when a male child sick the parents become worried and take to them hospital for better treatment. Parents believe that this boy will earn in future but not the girl.

The recovery time for each patient are categorized in a range e.g. from the age group 1-3 days required for 53% neonate and 67.5% infant, 4-6 days required for 38 % neonate and 24.3% for infant, 7-9 days not required for neonate and 2.7% for infant, 10-12 days required for 7.69% neonate and 5.4% for infant.



# Chapter 9

## **Conclusion**

The management of dehydration (prevention and treatment) and maintaining the patient's usual diet during and after diarrhea remains the mainstay in the management of diarrhea disease, irrespective of etiology, while antimicrobial therapy is indicated for specific etiologic diarrheas. Antidiarrheal drugs, although commonly used, have not been seen to provide any practical benefit and most of them are contraindicated, particularly for use in children.

From the discussion and the result we came to know that infant (>1yrs) were attacked by diarrhea in large number compare to neonate (<6 month). Most of the infants required few days to recover while neonate required more days compare to infant. A large number of neonate receive antibiotic treatment compare to infant. Diarrhea with vomiting is a common symptom for infants (about 30) while loose watery stool is a common symptom for neonate (about 7).

This scenario gives a partial feature of diarrhea disease in developing country like Bangladesh. Another important thing is while 60% male patient were admitted into hospital then only 40% female patient. Here gender discrimination is predominant. It is a common features of Developing country.

For better management of diarrhea in children, every treatment center can formulate their own treatment protocol according to their needs, determined on the basis of epidemiologic knowledge of the disease and socio cultural background, although most centers in developing countries follow WHO recommended treatment guidelines.

# Chapter 10



## Reference

1. Article on diarrhea (disambiguation) ,dec 2007).
2. Alam NH, Ashraf H (2003). "Treatment of infectious diarrhea in children". *Paediatr Drugs* 5 (3): 151–65.)
3. The American Journal of Medicine ,Volume 106, Issue 6, June 1999, Pages 670-676).
4. Belinda Rowland, PhD "What about diarrhea?" American Cancer society, INC 2000(cited July,2000).
5. B Young, J Briscoe Department of Environmental Sciences and Engineering, University of North Carolina, Chapel Hill, "Journal of Epidemiology and Community Health" 1988;42:83-88.
6. C. Guyton, M.d, eleventh edition, page-822.
7. C. Surawicz ,Gastroenterology Clinics of North America, Volume 30, Issue 3, Pages 679-692S.
8. Da Silva As, Pierezan p, Wolkar P, Iosta MM, J comp pathol, Pathological findings associate with experimental infection by Trypanosoma"
9. Essentials of Medical Pharmacology, KD. Tripathi, 5<sup>th</sup> edition.
10. Hasegawa, Hideo; Yatsukaho Ikeda, Akiko Fujisaki, Liza R. Moscovice, Klara J. Petrzekova, Taranjit Kaur, and Michael A. Huffman (2005). "MORPHOLOGY OF CHIMPANZEE PINWORMS, ENTEROBIUS (ENTEROBIUS) ANTHROPOPITHECI (GEDOELST, 1916) (NEMATODA: OXYURIDAE), COLLECTED FROM CHIMPANZEES, PAN

TROGLODYTES, ON RUBONDO ISLAND, TANZANIA" (PDF). *Journal of Parasitology*, 91(6), 2005. )

11.J Thomas La Mont,MD,(Patient information:Chronic diarrhea in adults,,May 2009).

12.Mark C.(guest editor) Steinboff Symposium: Infectious disease,Jhon Hokins University,USA.*E.coli* that causes diarrhea-Myron in.levise and Pablo vial University of Maryland school of Medicine ,Boltimole,USA).

13. M Arare,A.Baxendine,and C.E. Bennett,Diagonosing Medical parasites through Caprological techniques”2000.

14.MW Peterson,RS Spendlove and RA Smart ,J Clin Microbial, March 1976 3(3);376-377.

15.Micheal j.pelezer, Jr E.C.S. Chan, Noel R.Krieg, Microbiology Concepts and applications”, First edition,page - 706)

16.Navaneethan U, Giannella RA (November 2008). "Mechanisms of infectious diarrhea". *Nature Clinical Practice. Gastroenterology & Hepatology* 5 (11): 637–47).

17.Pensabene, Licia; Zikri, Mona Abu; Dias, Jorge Amil; Casali, Luigi Gobio; Hoekstra, Hans; Kolacek, Sanja; Massar, Karin; Micetic-Turk, Dusanka; Papadopoulou, Alexandra; de Sousa, Jaime Salazar; Sandhu, Bhupinder; Szajewska, Hanna; Weizman, Zvy,Lactobacillus GG Administered in Oral Rehydration Solution to Children with Acute Diarrhea: A Multicenter European TrialGuandalini, Stefano.

18.Sodeman WA (1996). "Intestinal Protozoa: Amebas". Baron's Medical Microbiology (Baron S et al., eds.) (4th ed.). University of Texas Medical Branch.

19. Ruuska T, Peskier T (1990). "Rotavirus disease in Finnish children: use of numerical scores for clinical severity of diarrhea episodes". *Scand. J. Infect. Dis.* 22 (3): 259–67).

20. Verweij JJ, Laeijendecker D, Brien EA, Van Lieshout L, Polderman AM (2003), detection and identification of entamoeba species in stool sample by a reverse line hybridization assay, *J. clin. Microbiol* 41(11):5041-5).

21. Victora CG, Bryce J, Fontaine O, Monasch R (2000). "Reducing deaths from diarrhea through oral rehydration therapy". *Bull. World Health Organ.* 78 (10): 1246–55).

