CLINICAL PROFILE,ETIOLOGY OF DIARRHOEA >2WEEKS DURATION IN CHILDREN BETWEEN 1 MONTH-12YEARS

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CHENNAI

INSTITUTE OF CHILD HEALTH AND
HOSPITAL FOR CHILDREN
MADRAS MEDICAL COLLEGE
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CERTIFICATE

This is to certify that the dissertation titled "**CLINICAL PROFILE, ETIOLOGY OF DIARRHOEA >2WEEKS DURATION IN CHILDREN BETWEEN 1MONTH-12YEARS"** submitted by **DR.S.NANTHAKUMAR** to the faculty of paediatrics, **THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY, CHENNAI** in partial fulfillment of the requirements for the award of **M.D., DEGREE (PAEDIATRICS)** is a bonafide research work carried out by him under our direct supervision and guidance.

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DECLARATION

This dissertation entitled **"CLINICAL PROFILE, ETIOLOGY OF DIARRHOEA >2WEEKS DURATION IN CHILDREN BETWEEN 1MONTH-12YEARS"**is a bonafide work done by **DR.S.NANTHAKUMAR** at institute of child health Madras medical college Chennai during the academic year 2015-2018 under the guidance of **Prof.DR.D.NIRMALA MD.,DM.,(GASTRO)** Professor of Department of Gastroenterology, Institute of Child Health, Chennai 600008. This dissertation submitted to The Tamil Nadu Dr.M.G.R. Medical University, Chennai towards partial fulfillment of the rules and regulations for the award of M.D Degree in Paediatrics, Branch (VII)

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Abbreviations

CMPA	-	Cow’s milk protein allergy
IBD	-	Inflammatory bowel disease
IBS	-	Irritable bowel syndrome
IED	-	Intestinal epithelial dysplasia
EBF	-	Exclusive breast feeding
UGI	-	Upper Gastro intestinal
GI	-	Gastro intestinal
MAS	-	Malabsorbtion syndrome
MCT	-	Medium chain triglyceride
TB	-	Tuberculosis

CONTENTS

INTRODUCTION

Diarrhoeal disease is the second leading cause of death in children under five years old, and is responsible for killing around 525 000 children every year. Diarrhoea can last several days, and can leave the body without the water and salts that are necessary for survival. In the past, for most people, severe dehydration and fluid loss were the main causes of diarrhoea deaths. Now, other causes such as septic bacterial infections are likely to account for an increasing proportion of all diarrhoea-associated deaths. Children who are malnourished or have impaired immunity as well as people living with HIV are most at risk of life-threatening diarrhoea.

Diarrhoea is defined as the passage of three or more loose or liquid stools per day (or more frequent passage than is normal for the individual). Frequent passing of formed stools is not diarrhoea, nor is the passing of loose, "pasty" stools by breastfed babies.

Chronic diarrhoea defined as diarrhoea lasting for more than 14 days . The epidemiology has two distinct patterns; In developing countries persistence of infection either gut or extra gut,malnutrition associated diarrhoea, whereas in developed countries chronic diarrhoea is less frequent and the etiology often varies with age. The outcome of diarrhoea depends on the cause and ranges from benign, self limited to severe congenital disease, that may lead to progressive intestinal failure

The prolonged episode of diarrhoea can be classified into three types:

1. Acute onset persistent diarrhoea

In this condition diarrhoeal episode begins abrubtly, but persistent beyond 14 days. The following terminologies like 'persistent', 'protracted', 'intractable', post enteritis syndrome', 'chronic malnourishing diarrhoea' etc. have been used to describe these condition. In fact Labenthal labeled even these episodes as chronic diarrhoea[1].

i) Simple persistent diarrhoea:

WHO defines persistent diarrhoea as episodes of acute diarrhoea lasting more than 14 days duration[2].This is the most accepted field definition and it is estimated that about 3-20% of acute diarrhoeal episodes may become persistent when the definition of 'passage of 3 or more stools for 14days or more' is applied. However, field studies indicate that most of these episodes are benign in nature and not affecting general well being and nutrition of the child concerned[3].

ii) Persistent protracted diarrhoea:

Certain episodes of persistent diarrhoea, complicated by weight loss, need active intervention. These complicated episodes have been referred to as protracted diarrhoea by Shanta Krishnan *et al* [4]. This terminology was used only for those episodes of persistent diarrhoea which are associated with weight loss

2. Insidious Onset Chronic Diarrhoea

Diarrhoea of more than 14 days duration occurring in a child with some basic defects either in the GI tract, immune system or other organs. They have rather an insidious onset over several days and weeks, with an increased bulk of stools which are rarely watery. Most of these episodes occur beyond 2 years of age. The terminology “chronic” (meaning not only duration but also nature of “onset”) should be limited to define these episodes. This entity is described in western textbooks under the title of Malabsorption syndrome[5] This is probably not appropriate as most episodes of Chronic diarrhoea, at least in tropics, do not have demonstrable malabsorption and on the other hand malabsorption can exist without causing chronic diarrhoea.

Etiologically and epidemiologically, the episodes of “persistent diarrhoea” and “chronic diarrhoea” as defined above, constitute entirely separate entities and approach to their diagnosis and management is entirely different.

3. Recurrent Diarrhoea

In between these two distinct entities of “persistent” and “chronic diarrhoea”, fall many children who present with apparent history of diarrhoea of a longer duration.

Careful history shows that what these children have are distinct but frequent episodes of acute diarrhoea, occurring unusually frequently, commonly in preschool children living in poor urban slum areas. It is better to define this

condition as “recurrent” diarrhoea. More than three episodes of acute diarrhoea per year are referred as recurrent diarrhoea[6].

However many of the conditions of “chronic diarrhoea” may present as “recurrent” diarrhoeal episodes. Hence these children would also need to be investigated as cases of chronic diarrhoea.

Causes of chronic diarrhoea

- Enteric infections
  - E.Coli
  - Giardia lamblia
  - Cryptosporidium parvum
  - Cytomegalovirus
- Cow’s milk protein allergy
- Lactose intolerance
- Immunodeficiency states
- Cystic fibrosis
- Pancreatic insufficiency
- Intestinal lymphangiectasia
- Intestinal tuberculosis
- Inflammatory bowel disease
- Celiac disease

- Short bowel syndrome
- Intractable diarrhoea of infancy

    intestinal tufting enteropathy

    autoimmune enteropathy

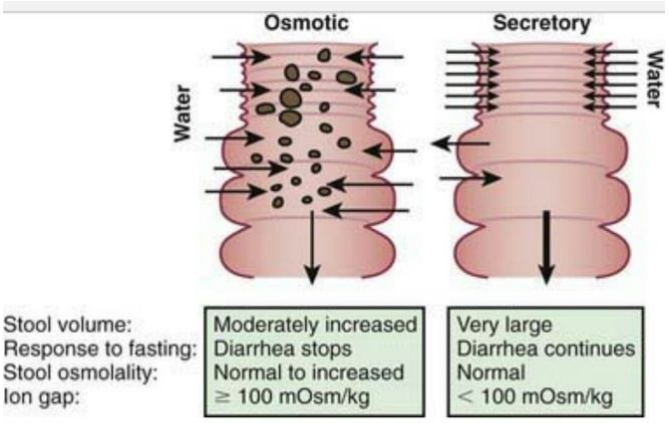
    congenital sodium/chloride diarrhoea

    congenital galactose malabsorbtion

Small bowel diarrhoea vs Large bowel diarrhoea

Features	Small bowel diarrhoea	Large bowel diarrhea
Stool volume	Large	Small
Steatorrhea	Yes	No
Blood in stools	No	Yes
Carbohydrate malabsorbtion	Yes	No
Protein malabsorbtion	Yes	No
Abdominal pain	Periumbilical	Hypogastric
Colour	Pale	Normal
Smell	Offensive	Normal
Urgency, tenesmus	No	Yes
Nutrient deficiency	Frequent	Can be

PATHOPHYSIOLOGY



Picture 1:Pathophysiology of diarrhoea

Mechanism of diarrhoea divided into osmotic, secretory, inflammatory diarrhoea and diarrhoea due to deranged motility

Osmotic Diarrhea

Absorption of water in the intestines is dependent on adequate absorption of solutes. If excessive amounts of solutes are retained in the intestinal lumen, water will not be absorbed and diarrhea will result. Osmotic diarrhoea typically results from one of two situations:

- Ingestion of a poorly absorbed substrate*:* The offending molecule is usually a carbohydrate or divalent ion. Common examples include mannitol or sorbitol, epson salt ($MgSO_4$) and some antacids ($MgOH_2$).

- Malabsorption: Inability to absorb certain carbohydrates is the most common deficit in this category of diarrhoea, but it can result virtually any type of malabsorption. A common example of malabsorption, affecting many adults humans and pets is lactose intolerance resulting from a deficiency in the brush border enzyme lactase. In such cases, a moderate quantity of lactose is consumed (usually as milk), but the intestinal epithelium is deficient in lactase, and lactose cannot be effectively hydrolyzed into glucose and galactose for absorption. The osmotically-active lactose is retained in the intestinal lumen, where it "holds" water. To add insult to injury, the unabsorbed lactose passes into the large intestine where it is fermented by colonic bacteria, resulting in production of excessive gas. A distinguishing feature of osmotic diarrhea is that it stops after the patient is fasted or stops consuming the poorly absorbed solute.

Secretory Diarrhea

Large volumes of water are normally secreted into the small intestinal lumen, but a large majority of this water is efficiently absorbed before reaching the large intestine. Diarrhea occurs when secretion of water into the intestinal lumen exceeds absorption.

Many millions of people have died of the secretory diarrhoea associated with cholera. The responsible organism, *Vibrio cholerae*, produces cholera toxin, which strongly activates adenylyl cyclase, causing a prolonged increase in

intracellular concentration of cyclic AMP within crypt enterocytes. This change results in prolonged opening of the chloride channels that are instrumental in secretion, allowing uncontrolled secretion of water. Additionally, cholera toxin affects the enteric nervous system, resulting in an independent stimulus of secretion.

Exposure to toxins from several other types of bacteria (e.g. *E. coli* heat-labile toxin) induce the same series of steps and massive secretory diarrhoea that is often lethal unless the person or animal is aggressively treated to maintain hydration.

In addition to bacterial toxins, a large number of other agents can induce secretory diarrhoea by turning on the intestinal secretory machinery, including:

- some laxatives
- hormones secreted by certain types of tumors (e.g. vasoactive intestinal peptide)
- a broad range of drugs (e.g. some types of asthma medications, antidepressants, cardiac drugs)
- certain metals, organic toxins, and plant products (e.g. arsenic, insecticides, mushroom toxins, caffeine)

In most cases, secretory diarrhoeas will not resolve during a 2-3 day fast.

Inflammatory and infectious diarrhoea

The epithelium of the digestive tube is protected from insult by a number of mechanisms constituting the gastrointestinal barrier, but like many barriers, it can be breached. Disruption of the epithelium of the intestine due to microbial or viral pathogens is a very common cause of diarrhoea in all species. Destruction of the epithelium results not only in exudation of serum and blood into the lumen but often is associated with widespread destruction of absorptive epithelium. In such cases, absorption of water occurs very inefficiently and diarrhoea results. Examples of pathogens frequently associated with infectious diarrhoea include:

- Bacteria: Salmonella, E. coli, Campylobacter
- Viruses: rotaviruses, coronaviruses, parvoviruses (canine and feline), norovirus
- Protozoa: coccidia species, Cryptosporium, Giardia

The immune response to inflammatory conditions in the bowel contributes substantively to development of diarrhoea. Activation of white blood cells leads them to secrete inflammatory mediators and cytokines which can stimulate secretion, in effect imposing a secretory component on top of an inflammatory diarrhoea. Reactive oxygen species from leukocytes can damage or kill intestinal epithelial cells, which are replaced with immature cells that typically are deficient in the brush border enyzmes and transporters necessary for absorption of nutrients

and water. In this way, components of an osmotic (malabsorption) diarrhoea are added to the problem.

Diarrhoea Associated with Deranged Motility

In order for nutrients and water to be efficiently absorbed, the intestinal contents must be adequately exposed to the mucosal epithelium and retained long enough to allow absorption. Disorders in motility then accelerate transit time could decrease absorption, resulting in diarrhoea even if the absorptive process per se was proceeding properly.

Alterations in intestinal motility (usually increased propulsion) are observed in many types of diarrhoea. What is not usually clear, and very difficult to demonstrate, is whether primary alterations in motility are actually the cause of diarrhea or simply an effect.

Lactose intolerance[32]:

Lactose is a disaccharide consisting of glucose and galactose.It is digested in the gut by enzyme lactase located in the microvilli of enterocytes. When the enzyme level is low lactose cannot be digested .This undigested lactose act as osmotic force in small intestine and enter into colon .Here it is fermented by colonic flora resulting in symptoms of lactose intolerance. Lactose intolerance was classified into primary and secondary

Primary (congenital) lactase deficiency

It is an autosomal recessive disease in which lactase activity is very low. The child develops watery diarrhoea when milk feeds introduced.

Secondary lactose deficiency

The enzyme lactase is located in villous tips, hence mucosal injury leads to secondary lactose intolerance. Symptoms of lactose intolerance are nausea, abdominal distension, abdominal pain,cramps, borborygmi, flatulence and loose stools. Diagnosed by occurrence of symptoms after ingestion of lactose and laboratory investigation.

Perianal excoriation is common due to irritation by acidic stools. The stool pH is low and there is increased reducing substance in stools. Treatment of primary lactose intolerance includes restriction of dietary lactose and use of lactase enzyme substitute. For secondary lactose intolerance depends upon its etiology

Dietary fructose intolerance.

Dietary fructose intolerance is a condition in which people have digestive symptoms—such as bloating, gas, and diarrhea—after consuming foods that contain fructose. Fructose is a sugar found in fruits, fruit juices, and honey.

Fructose is also added to many foods and soft drinks as a sweetener called high fructose corn syrup. Fructose malabsorption causes dietary fructose intolerance. The small intestine absorbs fructose, and, when a person consumes more fructose than the small intestine can absorb, fructose malabsorption results. Unabsorbed fructose passes to the colon, where bacteria break down the fructose and create fluid and gas. The amount of fructose that a child's small intestine can absorb varies. The capacity of the small intestine to absorb fructose increases with age. Some children may be able to tolerate more fructose as they get older.

Another type of fructose intolerance, hereditary fructose intolerance, is not related to fructose malabsorption. Hereditary fructose intolerance is an extremely rare inherited genetic disorder. Children with this disorder lack an enzyme needed to break down fructose. Symptoms of hereditary fructose intolerance may include abdominal pain, vomiting, and diarrhea. This disorder can also damage the liver and kidneys.

Enteric infections[21,19,16]

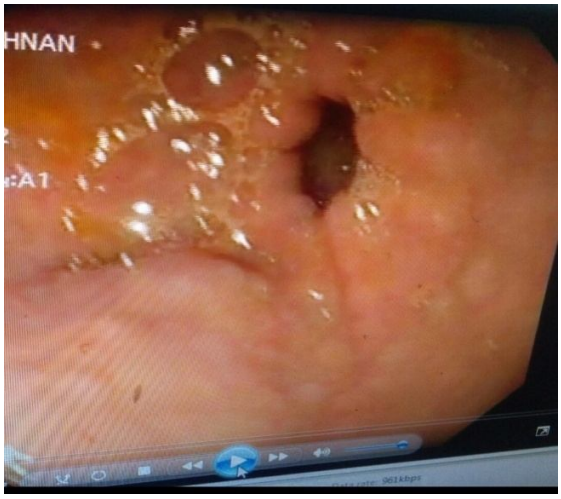
It is the most common cause for chronic diarrhoea in both developing and developed countries. In developing countries associated comorbid condition such as HIV/AIDS, Tuberculosis, malaria leads to malnutrition which impairs child′s immune response, thereby potentiating the diarrhoea or acquiring new infection

Enteoadherent Escherichia coli and Giardia lamblia are the common cause of chronic diarrhoea in developing countries whereas rotovirus and norvovirus are more common in developed countries.

Opportunistic microorganism cause diarrhoea in immunocompromised children. Clostridium difficile or cryptosporidium induce diarrhoea in cancer patients. Cryptosporidium also induce severe diarrhoea in AIDS

Cow's milk protein allergy[15,25,26]

Most commonly occurs in children below 2 years of age with diarrhoea and failure to thrive. In India it accounts for 13% of all malabsorbtion cases in children below 2 years. Family history of atopy is common in CMPA. Immediate and delayed reactions can occur with cow milk. Symptoms include vomiting, pallor, urticaria and diarrhoea with or without blood and mucus. Iron deficiency anaemia, hypoproteinemia and eosinophilia are commonly present. Cow′s milk and cow′s milk based products should be eliminated from diet.

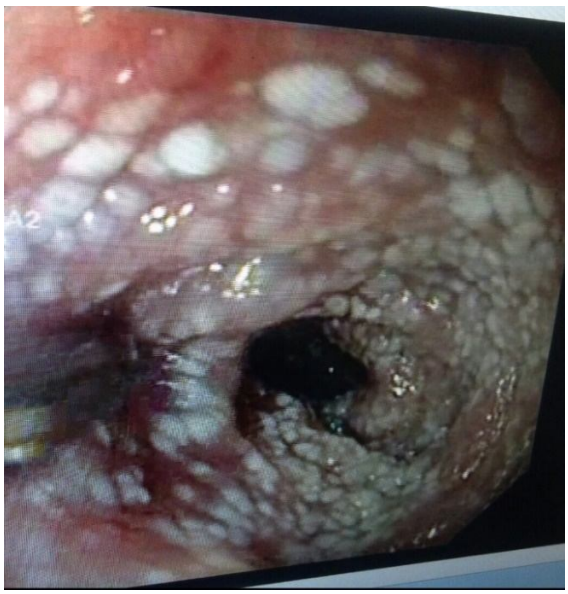


Picture 2: Colonoscopic picture showing lymphonodular appearance in a child with cow's milk protein allergy.

Intestinal lymphangiectasia[22,24]

There is ectasia of bowel lymphatic system, which leads to leakage of lymph in the bowel. It is often associated with abnormal lymphatics. Clinical features include diarrhoea, abdominal pain ,abdominal distension and pitting or non pitting peripheral edema. Abdominal and thoracic chylous effusions may be associated. Hypoalbuminemia, low immunoglobulins, hypocalcemia and lymphopenia are cardinal findings of lymphangiectasia. Barium meal followthrough shows thickening of jejunal folds and nodular lucencies. Duodenal biopsy reveals dilated lacteals in villi and lamina propria

Treated with a low fat ,high protein diet with MCT oil, calcium and fat soluable vitamins. surgery can be done if the lesion is localized to a small segment of intestine



Picture 3: Upper GI Endoscopy Picture showing smoky, fluffy mucosa in duodenum, suggestive of Intestinal lymphangiectasia.

Immunodeficiency[27]

Chronic diarrhoea can be caused by both congenital and acquired immunodeficiency. It should be suspected when there is recurrent infections at multiple sites. The common condition includes IgA deficiency, severe combined immunodeficiency (SCID), common variable immunodeficiency(CVID) and chronic granulomatous disease. Diarrhoea occurs due to enteric infection or bacterial overgrowth. Diagnosis made by measuring serum immunoglobulins, T cell counts and functions, phagocytic function(NBT test) depending upon the etiology.

Chronic diarrhoea is a common feature in children with AIDS resulting from impaired mucosal immunity, which leads to recurrent opportunistic infections. AIDS enteropathy causes chronic diarrhoea and severe weight loss without any enteric infections.

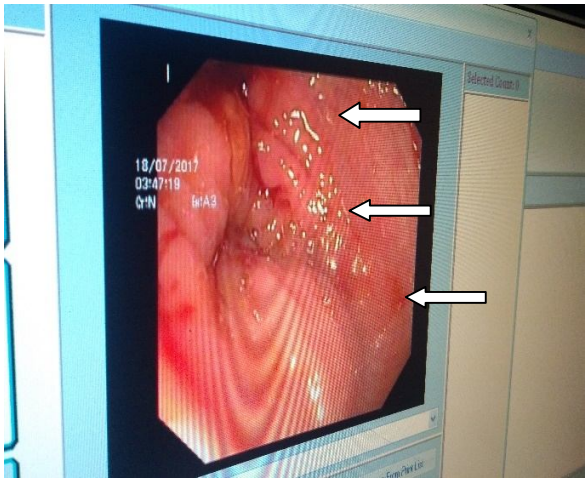
Inflammatory bowel disease (IBD)[30]

Chronic inflammatory disease of GI tract ,consist of two main types, Crohn disease and Ulcerative colitis .The incidence of IBD in children is increased worldwide .Common age of presentation in children 10-11 year .Genetic factors are important risk factors for IBD. Upto 30 % patients may have a IBD in his family

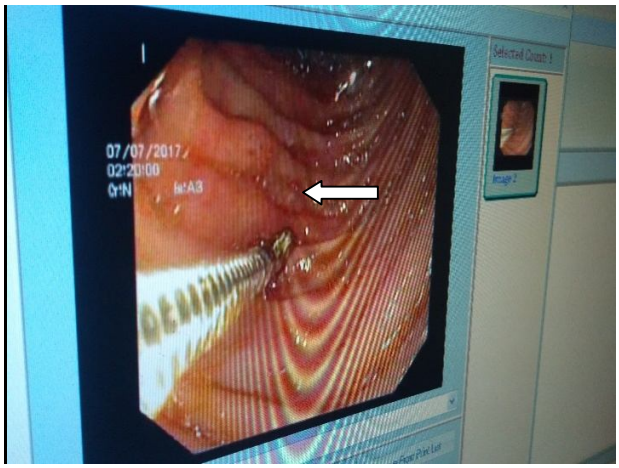
Ulcerative colitis causes diarrhoea and rectal bleeding. In crohn disease abdominal pain, diarrhoea and growth failure are the commonest features. Fever, fatigue and anorexia are present in 25-50% cases of crohn disease. 25-30% of children with IBD presented with extra intestinal manifestations like arthralgia / arthritis, Uveitis, erythema nodosum and sclerosing cholangitis

Diagnosis made by clinical history, physical examination, blood investigation, stool examination and upper GI endoscopy, colonoscopy with biopsy. Correct diagnosis is essential for management and prognosis of IBD

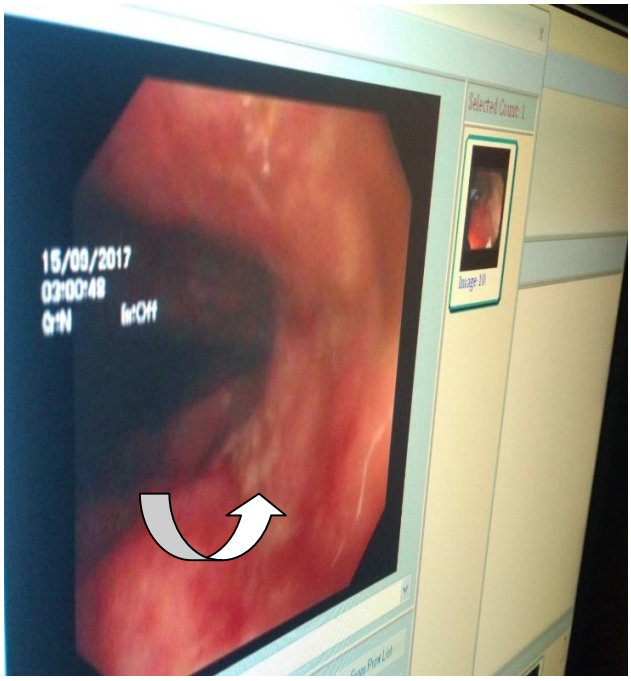
Treatment consists of steroids and immunomodulators (6-mercaptopurine, azathioprine, methotrexate and infliximab). Adequate nutrition(120% of RDA) with calcium and vitamin supplementation is necessary for children with IBD. Surgery is indicated in refractory colitis, uncontrolled hemorrhage, perforation, toxic megacolon and bowel obstruction.



Picture 4: Colonoscopic picture showing multiple aphthoid ulcers in a child with Crohn's disease



Picture5: UGI scopy picture showing multiple aphthoid ulcers in duodenum in a child with abdominal pain and diarrhoea



Picture 6: Colonoscopy picture shows edematous, erythematous mucosa with multiple ulcers in a child with blood and mucous diarrhoea

Intestinal epithelial dysplasia(IED)[28]

IED is a congenital enteropathy also known as tufting enteropathy. It is caused focal crowding of enterocytes that resembling tufts presented with early-onset severe intractable diarrhoea causing sometimes irreversible intestinal failure. The prevalence of IED higher in areas with high degree of consanguinity and in patients of Arabic origin. Infants develop watery diarrhoea within the first few days after birth. Some infants are associated with choanal, rectal or esophageal atresia. Nonspecific punctuated keratitis was presented in more than 60% of

infant. Histology reveals specific abnormalities of epithelium with disorganization of surface enterocytes with focal crowding, resembling tufts. Abnormal deposition of laminin and heparan sulfate proteoglycan (HSPG) on the basement membrane has been detected in intestinal epithelia. There is also an increased distribution of $\alpha 2\beta 1$and $\alpha 6\beta 4$ integrin adhesion molecules in tufting enteropathy.

Gastrointestinal tuberculosis[33]

Clinical features of abdominal tuberculosis depends upon thesite of disease and type of pathology.GI tract, peritoneum, lymphnode and solid organs can be involved in abdominal tuberculosis. Signs and symptoms include fever, anorexia, weight loss, chronic diarrhoea, abdominal pain, abdominal distension, ascites and ileo caecal mass.

Definitive diagnosis made by presence of acid fast bacilli(FNAC from lymphnodes, ascitic fluid, endoscopic biopsies) on Ziehl-Neelsen staining, PCR or culture. Tubercular granuloma in biopsies, enlarged lymphnode with caseation necrosis in CT abdomen also helps the diagnosis .

An exudative ascites with lymphocyte predominance and high adenosine deaminase is typical of tubercular ascites. Treated with anti tubercular drugs, surgery indicated in case of bowel perforation, obstruction or hemorrhage

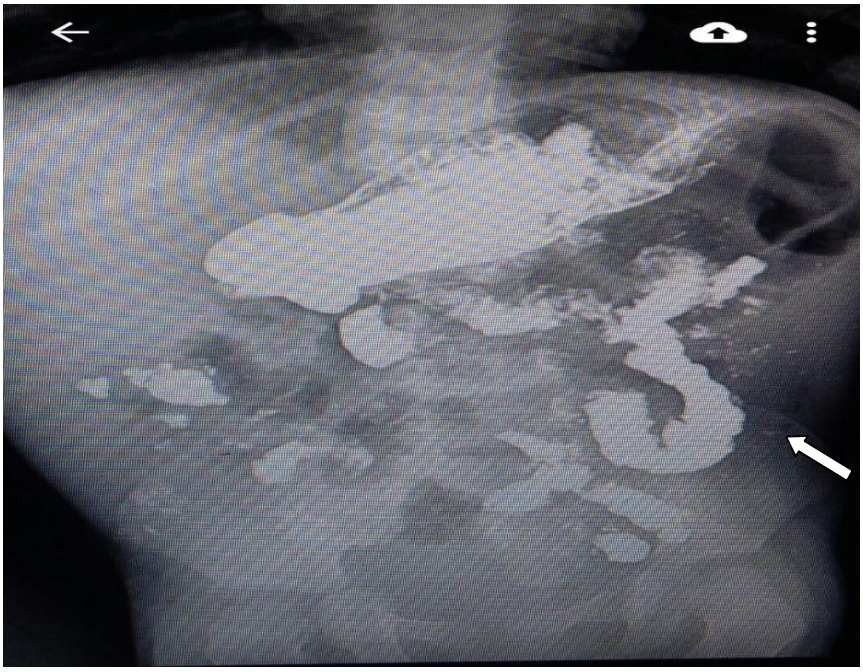
Celiac disease[31]

Celiac disease is an autoimmune disorder of small intestine that occurs in genetically susceptible individuals .It caused by a reaction to gliadin, a gluten protein found in wheat, rye and barley. Celiac disease appears to be polyfactorial, more than one genetic factor can cause the disease

Celiac disease presented with small bowel diarrhoea, growth failure, abdominal pain and anemia. Coagulation defect occurs due to vitamin K deficiency. Calcium and vitamin D malabsorbtion leads to short stature, rickets and osteopenia.

Diagnosis made by 1)serology-Anti transglutaminase antibodies to the enzyme tissue transglutaminase 2) upper GI endoscopy shows shows scalloping of duodenal folds 3) histology shows increased intraepithelial lymphocytes, partial to total villous atrophy and infiltration of plasma cells and lymphocytes in lamina propria.

Treatment consists of lifelong gluten free diet and correction of vitamin ,mineral deficiencies by supplementation. The child should be reviewed for every 3months to monitor response to treatment.



Picture 7: Barium meal –Malabsorbtion pattern

(Loss of feathery mucosal pattern)

Short bowel syndrome

Short bowel syndrome is malabsorption resulting from extensive resection of the small bowel (usually more than two thirds the length of the small intestine). Symptoms depend on the length and function of the remaining small bowel, but diarrhea can be severe, and nutritional deficiencies are common. Treatment is with small feedings, anti diarrheals and sometimes TPN or intestinal transplantation. Short bowel syndrome is a malabsorption disorder. Common reasons for extensive resection are Crohn disease, mesenteric infarction, radiation enteritis, cancer,

volvulus, and congenital anomalies. Because the jejunum is the primary digestive and absorptive site for most nutrients, jejunal resection leads to loss of absorptive area and significantly reduces nutrient absorption. In response, the ileum adapts by increasing the length and absorptive function of its villi, resulting in gradual improvement of nutrient absorption.

The ileum is the site of vitamin B_{12} and bile acid absorption. Severe diarrhoea and bile acid malabsorption result when > 100 cm of the ileum is resected. Notably, there is no compensatory adaptation of the remaining jejunum (unlike that of the ileum in jejunal resection). Consequently, malabsorption of fat, fat-soluble vitamins, and vitamin B_{12} occurs. In addition, unabsorbed bile acids in the colon result in secretory diarrhoea. Preservation of the colon can significantly reduce water and electrolyte losses. Resection of the terminal ileum and ileocecal valve can predispose to bacterial overgrowth.

Congenital zinc deficiency[29]

Congenital zinc deficiency occurs in early months of infancy after weaning from breastfeeding .Clinical features include bullous pustular dermatitis, paronychia and alopecia. Opthalmic signs include blepharitis, conjunctivitis, photophobia and corneal opacities. Neurological manifestations include emotional instability, irritability, tremors and cerebellarataxia. Growth retardation, weight loss and male hypogonadism also occurs. Acrodermatitis enteropathica(AE)

childrens have an increased susceptibility to infections due to thymic hypoplasia and absence of germinal centres in lymphnode.

These abnormalities are completely resolved by zinc supplementation. Gastrointestinal features include severe diarrhoea, steatorrhea, malabsobtion and lactose intolerance.The AE gene located in chromosome 8q24,encodes a histidine rich protein responsible for zinc transports. Mutation of this gene have been documented in AE.

Picture 8: Acrodermatitis enteropathica-Congenital Zinc deficiency

Chronic non specific diarrhoea[23]

Chronic non-specific diarrhoea is the most common benign etiology of chronic diarrhoea in children which includes functional diarrhoea (or toddler's diarrhoea) in subjects below four years of age and irritable bowel syndrome in those aged 5–18 years[7].

The clinical features are same for toddler′s diarrhoea and irritable bowel syndrome, but presentation varies with age: abdominal pain is more frequently associated with diarrhoea in older children . The hallmark of the syndrome is diarrhoea associated with normal weight gain in well-appearing subjects.

Functional Gastrointestinal Disorders

In functional GI disorders, symptoms are caused by changes in how the GI tract works. The GI tract is a series of hollow organs joined in a long, twisting tube from the mouth to the anus—the opening through which stool leaves the body. The GI tract digests, or breaks down, food and processes solid waste. Children with a functional GI disorder have frequent symptoms, yet the GI tract does not become damaged. Functional GI disorders are not diseases; they are groups of symptoms that occur together. Two functional GI disorders that cause chronic diarrhoea in children are toddler's diarrhoea and irritable bowel syndrome (IBS).

Toddler's diarrhea.

Toddler's diarrhea— also called functional diarrhea or chronic nonspecific diarrhea of childhood—is a common cause of chronic diarrhea in toddlers and preschool-age children. Children with this disorder pass three or more loose stools a day and do not have any other symptoms. They typically are growing well and gaining weight, and are healthy. Toddler's diarrhea develops between the ages of 6 months and 3 years, and it usually goes away on its own by the time children begin grade school. Researchers think a diet with too much sugar—such as the sugar found in fruit juice—relative to the amount of fat and fiber may cause toddler's diarrhea.

IBS (Irritable bowel syndrome)

The most common symptoms of IBS are abdominal pain or discomfort, often reported as cramping, along with changes in bowel habits, such as diarrhoea. The pain or discomfort of IBS typically gets better with the passage of stool or gas. IBS does not cause symptoms such as weight loss, vomiting, or blood in the stool. Possible causes include problems with nerves in the intestines, problems with nerve signals between the brain and the intestines, changes in how food moves through the intestines, and hypersensitivity to pain. Psychological problems, such as anxiety and depression, or food sensitivity may also play a role. IBS is a common cause of chronic diarrhoea in grade school-age children and

adolescents. Health care providers rarely diagnose IBS in younger children because younger children are not able to report symptoms of pain or discomfort.

Age-related Rome III criteria for functional diarrhoea.[7]

Age Criteria

Neonate and Toddlers (below 4 years):

diagnostic criteria for functional diarrhoea

1. Daily painless, recurrent passage of three or more large, unformed stools;
2. Symptoms that last more than four weeks;
3. Onset of symptoms that begins between 6 and 36 months of age;
4. Passage of stools that occurs during waking hours;
5. There is no failure-to-thrive if caloric intake is adequate

Child and Adolescent (5 to 18 years):

Diagnostic criteria for irritable bowel syndrome

1. Abdominal discomfort (an uncomfortable sensation not described as pain) or pain associated with two or more of the following at least 25% of the time:

a) Improved with defecation

b) Onset associated with a change in frequency of stool

c) Onset associated with a change in form (appearance) of stool

2. No evidence of an inflammatory, anatomic, metabolic, or neoplastic process that explains the subject's symptoms

Chronic diarrhoea may be the manifestation of maldigestion due to exocrine pancreatic disorders. In most patients with cystic fibrosis, pancreatic insufficiency results in fat and protein malabsorption. In Shwachman–Diamond syndrome, exocrine pancreatic hypoplasia may be associated with neutropenia, bone changes, and intestinal protein loss. Specific pancreatic enzyme defects result in fat and/or protein malabsorption. Familial pancreatitis associated with a mutation in the trypsinogen gene may be associated with pancreatic insufficiency and chronic diarrhoea.

Liver disorders may lead to a reduction in the bile salts resulting in fat malabsorption. Bile acid loss may be associated with terminal ileal diseases, such as Crohn's disease or following ileal resection. In primary bile acid malabsorption, neonates and young infants present with chronic diarrhoea and fat malabsorption due to mutations of ileal bile transport

Clinical approach for diagnosis

Initial evaluation : History

Duration, pattern, epidemiology

Severity, dehydration

Stool volume and frequency

Stool characteristics

Nocturnal symptoms

Fecal urgency, incontinent

Associated symptoms

(Fever ,abdominal pain, weight loss)

Extraintestinal manifestation

Relationship to meals, specific foods, fasting and stress

Medical, surgical history

Recent hospitalization and antibiotics

History of radiation

Feeding history

Immunization history

Clinical examination

General examination and nutritional status are the initial evaluation. Height, weight and mid arm circumstance measurements are required for nutritional assessment. Weight impairment occurs initially, but with duration linear growth also becomes affected.

Associated symptoms and investigations provide diagnostic clues. Fever ,blood or mucoid stools and abdominal pain may suggest inflammatory bowel disease .

Allergic disorder associated with asthma or eczema. Extraintestinal features arthritis, thrombocytopenia suggest autoimmune disease. Zinc deficiency associated with specific skin lesions (acrodermatitis enteropathica)

Investigations

Stage 1

Intestinal microscopy

Stool culture

Microscopy for parasites

Screening test for celiac disease

Level of IgA tTG

Tests for food allergy

prick/patch tests

Non invasive tests

pancreatic function(amylase, lipase, fecal elastase)

Stage 2

Evaluation of intestinal morphology

Endoscopy and histology

Electron microscopy, imaging

Stage 3

Special investigations

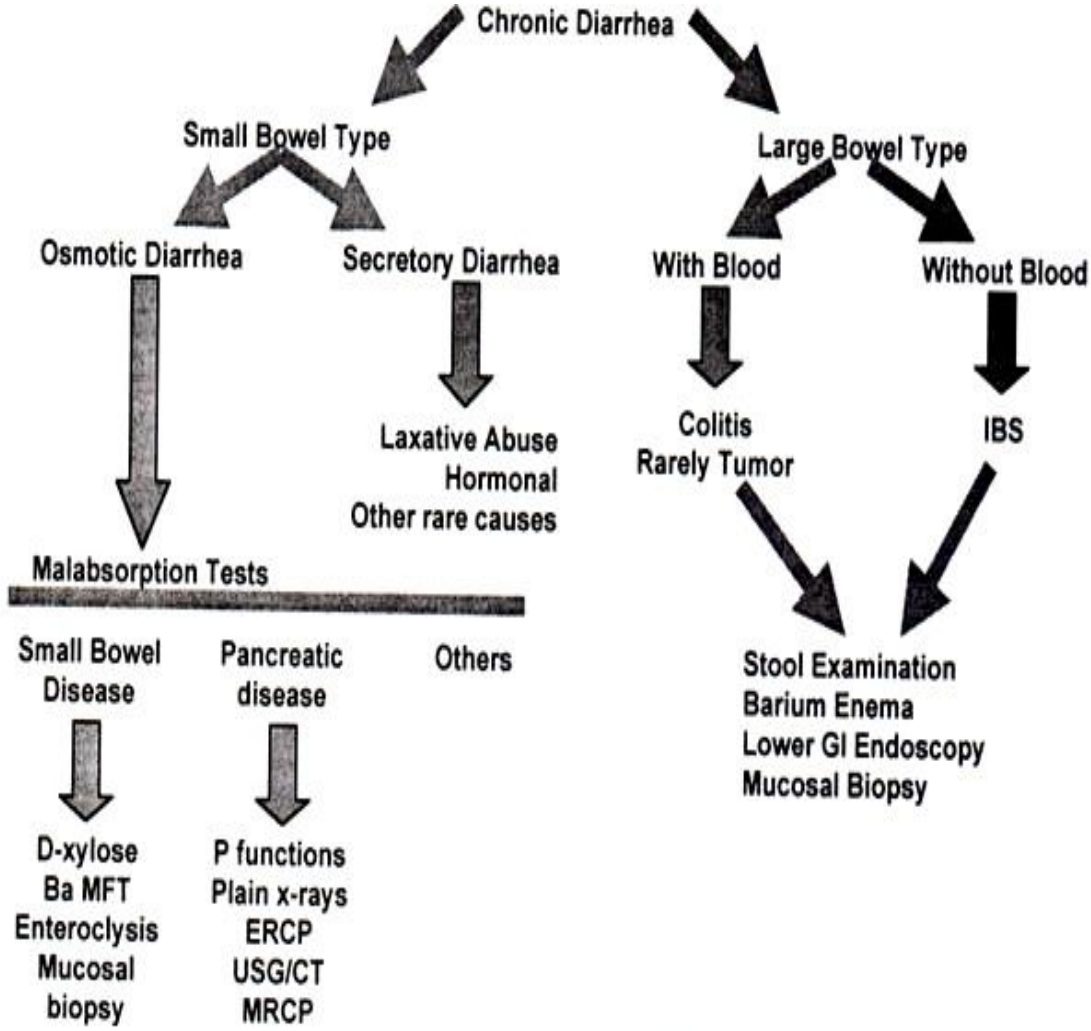
Intestinal immune histochemistry

Anti enterocyte antibodies

Auto antibodies

Motility and electrophysiologic studies

Abdominal ultrasound


Chronic Diarrhea
Small Bowel Type
Large Bowel Type
Osmotic Diarrhea
Secretory Diarrhea
With Blood
Without Blood
Laxative Abuse
Hormonal
Other rare causes
Colitis
Rarely Tumor
IBS
Malabsorption Tests
Small Bowel Disease
Pancreatic disease
Others
Stool Examination
Barium Enema
Lower GI Endoscopy
Mucosal Biopsy
D-xylose
Ba MFT
Enteroclysis
Mucosal biopsy
P functions
Plain x-rays
ERCP
USG/CT
MRCP

Treatment

- General supportive measures
- Nutritional rehabilitation
- Elimination diet
- Medications

General supportive measures

Dehydration is the main cause for death in chronic diarrhoea, so replacement of fluid and electrolyte loss are the most important early intervention.

Nutritional rehabilitation

Nutritional rehabilitation based on clinical and biochemical assessment. Harmful nutrients should be identified and eliminated. Caloric intake progressively increased to 50% or more above the recommended dietary allowance. Micronutrient and vitamin supplementation should be given . Zinc supplementation is essential for therapeutic and prevention. It stimulate ion absorbtion, restore epithelial proliferation and potentiate immune response.

Elimination diet

WHO recommends lactose free diet for all children with chronic diarrhoea. Sucrose free diet is started in sucrase –isomaltase deficiency.

Elimination sequence should begins with low to more restricted diet. In severely compromised children to start with amino acid based formula. Functional diarrhoea improved with diet based on“ 4F” principle

Reduce fructose and fluid ,increase fat and fiber

Medications

Antibiotics against bacterial agent

Immune suppression in autoimmune enteropathy

Enkephalinase inhibitor-racecadotril

Zinc supplementation

Parentral nutrition

REVIEW OF LITERATURE

Bhatnagar et al[8] conducted a study to define the etiological spectrum and outcome of chronic diarrhoea over 5 yrs duration. In this retrospective study a total of 85 children were evaluated. Mean age of onset 5.6 yrs, Boys(61.48%).Of which 6 cases had no etiology, celiac disease(36.47%),cow milk protein allergy (11.76%), Non specific diarrhea (20%),Parasitic infestation (17.64%), Intestinal TB(2.35%), Intestinal lymphangiectasia (1.17%),HIV (1.17%),CVID(1.17%),and IBD(1.17%)

Lee et al[9] in their prospective study a total of 27 children with diarrhoea more than 2 weeks duration were evaluated. The median age of onset of diarrhoea was 6 months, while the mean age was 16.4 months, the mean duration of symptoms were 66.5 days . A total of 27 cases were evaluated. Cow's milk protein intolerance was the most common cause of chronic diarrhoea in this study. CMPA(8cases),secondary lactose intolerance(4cases),gastro intestinal infections (7cases), intestinal lymphangiectasia (2cases),others (6cases). Fifteen children (56%) had a bodyweight at or below the third percentile for age.

Rastogi et al[10] Conducted a study in 47 children with diarrhoea more than 2 weeks duration. The results were tropical enteropathy (46.8%),irritable bowel syndrome(10.6%), giardiasis(14.8%),celiac disease(6.8%), non specific

diarrhoea (21.8%). Tropical enteropathy treated with anti microbial therapy, celiac disease required withdrawal of gluten from diet

Altuntas et al[11] conducted a study with 70 children of age between 1 month -15 yrs with chronic diarrhoea. Mean age of onset was 40.8 months and 52% were males. Malnutrition was detected in 80% of cases. Fever (32%), hepatomegaly and splenomegaly (20%). Etiological factors include Celiac disease(30%),tropical enteropathy (10%),CMPA(17%),Parasitic infestation (26%),IBD(10%),cystic fibrosis(10%),unknown (10%)

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Yachaa et al[12]in their study 137 children evaluated for etiology of diarrhoea >3weeks duration. Sixty two (45%) children were below 2 years of age and 75 (55%) above. Common causes of were: protracted diarrhea 45 (33%), celiac disease 35 (26%), parasitic infestations 13 (9%), milk protein intolerance 8 (6%), intestinal tuberculosis 7 (5%). In 18 (13%) patients, cause could not be determined. Protracted diarrhea (73%) and milk protein intolerance (13%) constituted the major etiology of MAS in children below 2 years of age, whereas celiac disease (43%), parasitic infestations (15%) and intestinal tuberculosis (9%) were the common causes in children above 2 years of age.

Valdovinos-Oregon et al[13] conducted a retrospective, observational, descriptive cross sectional study in children with primary intestinal lymphangiectasia in between 1992-2012.The results were 4 children found that presented with primary intestinal lymphangiectasia. Three children diagnosed before age of 3yrs. Clinical features include chronic diarrhoea, edema, lymphopenia, hypocalcemia and hypogammaglobulinemia. Upper gastro intestinal scopy and biopsy were diagnostic.

Igra mushtaq et al[14] done their study in 72 children with chronic diarrhoea All children with age less than 6 months are evaluated for etiology. Age at onset of symptoms 15 days to 6months. Results were cow′s milk protein allergy - 58(80.6%), primary intestinal lymphangiectasia-6(8.3%), cystic fibrosis-3(4.2%), SCID-2(2.8%). Remaining 1.4% consists of Abetalipoproteinemia(1), congenital chloride diarrhoea (1) and glucose-galactose malabsorbtion(1). CMPA is the most common non infectious cause of chronic diarrhoea in early infancy.

Iynkaran et al[15] in their study cow milk protein allergy is one of the important cause for persistent diarrhoea and malabsorbtion in early infancy. A combined clinical and histological criteria 1) diarrhoea while receiving cow milk protein 2) clinical recovery on a diet free of cow milk 3) normal or mildly abnormal histology of jejunal mucosa after 6-8 weeks of recovery 4) histological relapse on restart of cow milk

Sullivan et al[16]done their study in 31 children aged 6mon to 36 months with chronic diarrhoea -malnutrition syndrome to find out the prevalence of Giardiasis. 33 healthy and 11 marasmic children taken as controls. Giardiasis was diagnosed in 14 children with chronic diarrhoea-malnutrition syndrome(45%), 4 children each in healthy and marasmic controls. This study revealed the increased prevalence of giardiasis in chronic diarrhoea -malnutrition syndrome and insists the potential importance of this organism in the pathogenesis of chronic diarrhoea

Reifen et al [17]done a study in 3 children with protracted, watery diarrhoea started in early infancy in whom they found distinct histologic and ultrastructural features that they designated 'tufting enteropathy. These children presented at 1, 2, and 4 weeks of age, respectively, with protracted watery diarrhoea with volumes in excess of 1,500 ml/day and impaired growth requiring total parenteral nutrition (TPN). The diarrhoea was refractory to a variety of diets, including breast milk, as well as immunosuppressive drugs. Cessation of enteral feedings decreased the volume of diarrhoea to less than 500 ml per day in all 3 patients, 2 of 3 children achieved normal growth velocity in both height and weight within 6 months; both these children were still dependent on TPN at home at ages 8.5 and 6 years, respectively. The third children died with persisting diarrhea at 18 months of age; autopsy revealed a thin and dilated intestine with flat small bowel mucosa. Jejunal biopsies from the 3 children shows partial villous atrophy accompanied by

crypt hyperplasia and an increased number of mitotic figures in the crypts. . The most classical finding was focal epithelial 'tufts' on the surface epithelium. No inclusion bodies or secretory granules were seen on electron microscopy in the cytoplasm of villous enterocytes. Further more the epithelial surface revealed that 80 to 90% contained tufts in these 3 patients, compared to only 16% in patients with celiac disease and less than 10% in normal jejunum.

SK Mittal et al[6] in his work on Chronic Diarrhoea in Tropics, observed that up to 40% of mortality associated with diarrhoeal disease is associated with persistent diarrhoea accompanying malnutrition, 60% of persistent diarrhoea occur before6 months and 90% below 1 year of age. The etiological factors were persistent gut infection with enteroadhesive E.coli and entero-aggregative E.coli, mucosal atrophy, secondary lactose intolerance, cow's milk protein intolerance, systemic infection, parasites like Giardia lamblia and Crypto sporidium and depressed immunity particularly those with AIDS. Persistent diarrhoea with no weight loss managed with extra food and fluids. After a brief period of IV fluids normal diet started, about one third children recovered with this regimen only. About 20-30% children with persistent diarrhoea require no lactose diet. In these cases also, homemade diets like rice pulse oil diet or comminuted chicken diet had been found very useful.

Abdullah et al [18] in their study forty-eight cases of chronic diarrhea in children seen at King Khalid University Hospital over a 5 year period were analysed. The mean age at presentation was 1.8 years (range 0.08-10 years); 34 were boys and 14 girls. Forty-four patients were Saudi and four were non-Saudi Arabs. Most children presented with failure to thrive and pallor. The etiological factors identified were: the post-gastro-enteritis syndrome with or without lactose intolerance in 16 (33%); Celiac disease in ten (21%); congenital chloride diarrhoea in five (10%); glucose-galactose malabsorption and acrodermatitis enteropathica, each in three (6%); ulcerative colitis, intestinal lymphangiectasia, cow's milk protein intolerance and ataxia telangiectasia, each in two (4%); and giardiasis, immune deficiency and cystic fibrosis, each in one (2%). Five children were died

Phillips et al[19] in their study the association between Cryptosporidium, chronic diarrhoea and a proximal small intestinal mucosal enteropathy was reviewed over a six and a half year period. One hundred and twenty three children with cryptosporidiosis and no clinical evidence of immune deficiency were identified. 50% of children excreting only Cryptosporidium had chronic diarrhoea. Most cases (63%) of chronic diarrhoea occurred in the first two years of life. A mild to moderate enteropathy was present in all nine children undergoing a small intestinal biopsy and seven showed the presence of Cryptosporidium adhering to villous epithelium. All patients eventually recovered spontaneously. Cryptosporidium is a cause of chronic diarrhoea and a proximal small intestinal

mucosal enteropathy in children without immune deficiency. Screening for the parasite should be part of the investigative procedures in children with chronic diarrhoea.

Thomas AG et al[20] The value of proximal intestinal mucosal biopsy was reviewed in 381 children presenting with chronic diarrhoea over an eight year period. An enteropathy was detected in 44% of cases and was more frequently seen in those aged less than 6 months. A diagnosis was established in 91% of cases. The most common diagnosis was the post enteritis syndrome where the presence of an enteropathy indicated those requiring treatment with a cows' milk free diet. Other conditions where a biopsy facilitated diagnosis or treatment included giardiasis, enteropathogenic Escherichia coli, cryptosporidiosis, autoimmune enteropathy, and microvillous atrophy. Celiac disease was considered in 55% of children and established in 8%, clearly identifying those requiring a gluten free diet. This also emphasizes the important role of the biopsy procedure in the exclusion of specific diseases. Proximal small intestinal mucosal biopsy is an essential investigation in children with chronic diarrhoea in whom an enteropathy is suspected.

STUDY JUSTIFICATION

Chronic childhood diarrhoea had variety of etiologies , a specific diagnosis could be established in 90% of cases, by various diagnostic tests, so that appropriate therapy can be instituted to reduce morbidity and mortality.

AIM AND OBJECTIVE

Aim: The aim of my study is to determine the clinical profile, etiology of diarrhoea >2 weeks duration in children between 1mon-12yrs of age in tertiary centre in south India.

Objective: To find the common cause of chronic diarrhoea in our hospital and to find out the most useful methods for diagnosis

METHODOLOGY

Materials and methods:

- **Study design: Prospective study**
- **Study population**: Children in study age group attending gastroenterology department and admitted in medical ward, satisfying the inclusion criteria.
- **Place of study**: Department of gastroenterology and paediatric medical ward in ICH
- **Study period**: October 2016 to September 2017
- **Sample size**: 43
- **Inclusion criteria**: All children aged 1 month to 12 years presenting with diarrhoea >2 weeks duration attending the hospital
- **Exclusion criteria**: children with established diagnosis on treatment

Procedure :-

After obtaining informed consent from parent/guardian, various patient demographic characteristics, history, clinical details, anthropometry, will be entered in a pre structured Proforma. All these children will have basic investigations like CBC, peripheral smear, blood sugar, RFT,LFT, sr.Protein, sr.Zinc, sr.electrolytes urine routine, urine culture and sensitivity, NEC, Xray chest, USG,HIV, stool routine and second line investigations include upper GI endoscopy, barium contrast studies, endoscopy & colonoscopy for cases suspected

to have tuberculosis, inflammatory bowel disease. Sigmoidoscopy and biopsy incase of cow milk protein allergy.

Eligible children were enrolled in the study after obtaining a informed consent form the parents/ Guardian, explaining the details of study procedure. This study protocol was reviewed and approved by the ethical review committee. Children were subjected to complete physical examination and assessments of anthropometry. Anthropometry is a simple valuable tool and the gold standard for evaluating the nutritional status.

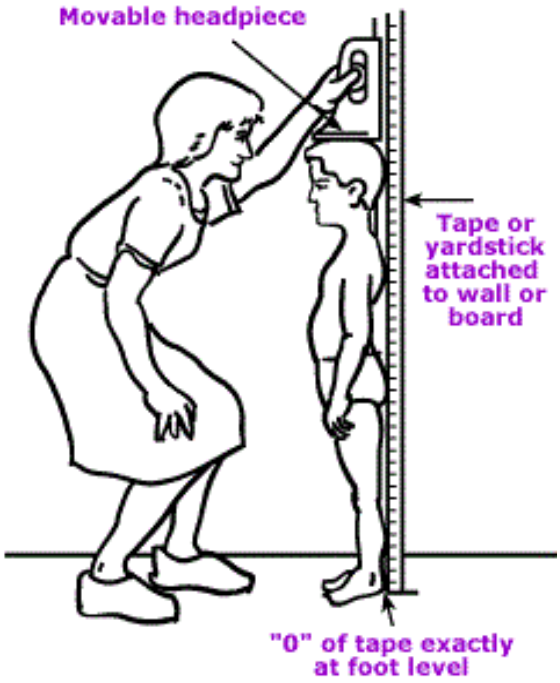
Weight

Weight was measured using a beam scale of salter type scale with pants in which the child was placed. The beam was properly balanced and moved freely when at rest and the pointer was on zero. The scale was set on a flat horizontal surface. The shoes were removed and children were weighed with as little clothing as customs permitted. The child was not in contact with any other object. Weight was either read directly or by balancing the beam depending on the type of scale. The result was read only after the beam reached the balance point or the pointer became motionless. If children were restless double weighing was done. As accuracy was less satisfactory this was used as a last resort only.

The scale was checked with standard weight and zero error was corrected to the nearest value of + 20 gms.

Height

Below the age of 2 years a horizontal measuring rod (or) infantometer was used. Height measured in lying down posture was called length. Length measurement needed two people. Shoes were removed & child was placed on a flat surface. One person preferably the mother maintained the top of child's head against the fixed vertical headboard with the child's eyes directed upwards. The other persons firmly pressed the knees together and down so that they touched the horizontal surface and then moved the mobile foot board so that if touched the heels when the feet were at right angle. Accuracy was adjusted to the nearest 0.5cm. Beyond the age of two years, a vertical measuring rod or stadiometer was used. The child was made to stand bare foot and the heels, buttocks, shoulders & occiput touching the wall and looking straight ahead. The chin was made to be straight (in Frankfurt planes).



The observer read the measurement directly after lowering the cursor orplacing a horizontally held book or wooden board in order to touch the top of head. The hair flattened and the accuracy measured to the nearest 0.5cm.Both height and weight were recorded by a single observer.

Head circumference

While measuring head circumference the maximum occipitofrontal circumference was measured by placing the flexible non stretchable tape firmly over the most prominent region of the occiput and frontal crests. The measurement

was made accurate to the nearest of0.1cm.A base line assessment including detailed physical examination was performed at the time of enrollment.

For dehydrated children Anthropometry was repeated after hydration children were treated with standard diarrhoea treatment protocol. Within 24 hour of admission 3 ml of venous blood was collected via venipuncture using plastic tubes carefully washed to make them zinc free. The blood samples were transported to lab where serum zinc concentration were measured using calorimetric Method. A Certified trace element control serum was used daily to ensure accuracy and precision. All lab analyses were blinded to diarrhoea and intervention status of the children sampled. parents/ Guardian, explaining the details of study procedure

Body Mass Index (BMI)

Body Mass Index (BMI)20 is an anthropometric index of weight and height (stature) that is defined as body weight in kilograms divided by height in meters squared. BMI is the commonly accepted index for classifying adiposity in adult sand it is recommended for use with children and adolescents.

BMI=weight (kg)/height (m)2 .

Zinc Methodology

Principle

Zinc in an alkaline medium reacts with Nitro – PAPS to form a purple coloured complex. Intensity of the complex formed is directly proportional to the amount of zinc present in the sample.

Alkaline

Zinc + Nitro ⟶ PAPS Purple Coloured complex

Medium

Normal reference values

Serum 60 – 120 µg/dl

RESULTS

TABLE 1: AGE OF ONSET

AGE	MONTHS
MEAN AGE	23.00
RANGE	1 – 120

Mean age of onset of chronic diarrhoea in our study were 23 months ,ranging from 1month to 120 months

TABLE 2: AGE WISE DISTRIBUTION

AGE	MALE	FEMALE	TOTAL
< 1YR	11	10	21(48.8%)
1-5 YR	11	6	17(39.5%)
>5-12 YR	3	2	5(11.7%)

In our study Children aged <1 year (48.8%), 1 to 5 year (39.5%) and above 5 years(11.7%).

AGE WISE DISTRIBUTION

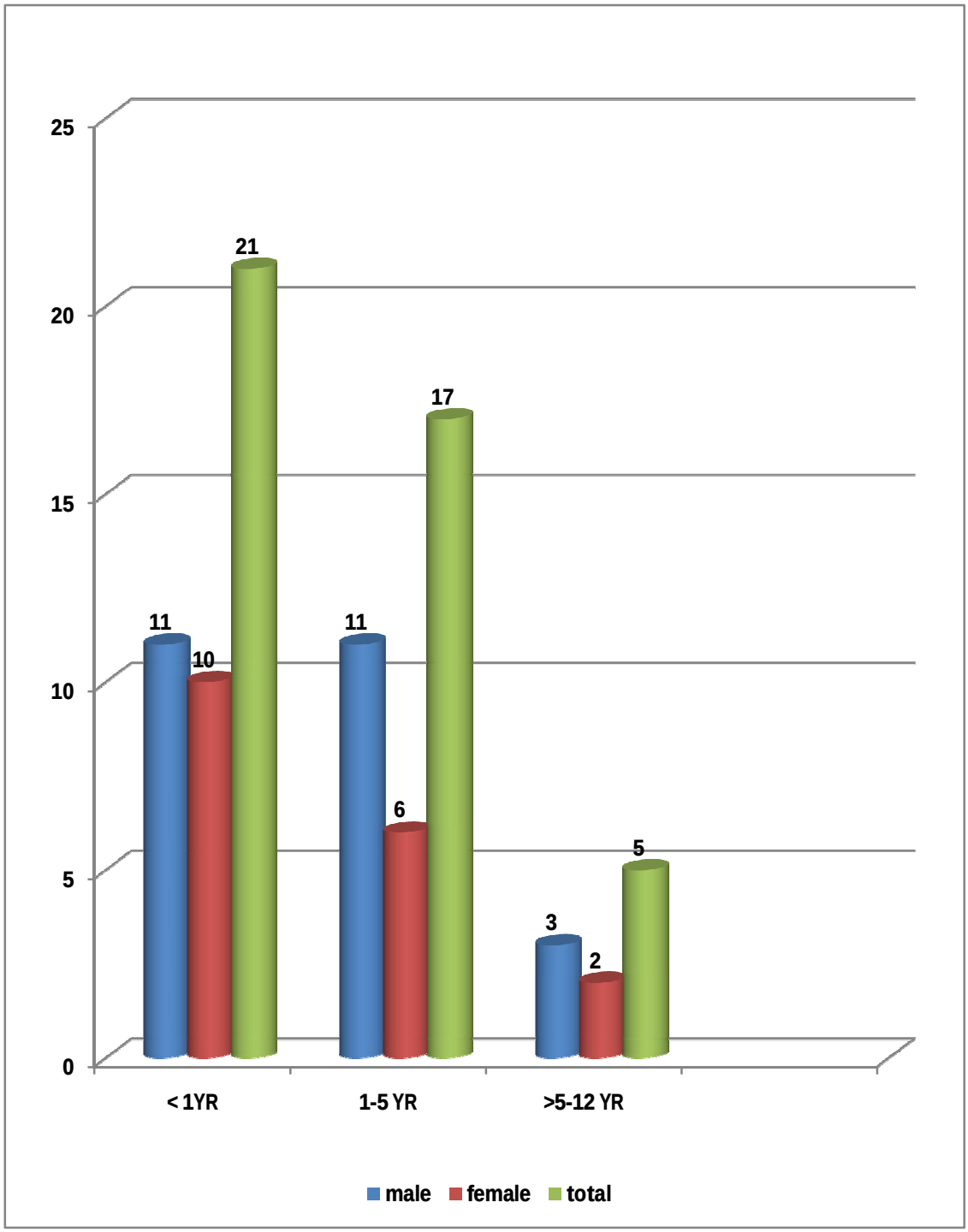


TABLE 3: SEX WISE DISTRIBUTION

SEX	NO OF BABIES	PERCENTAGE (%)
MALE	25	58.1
FEMALE	18	41.9
TOTAL	43	100.0

In our study out of 43 children 25 (58.1%) were male children and 18(41.9 %) were female children. male /female ratio was 1.4:1

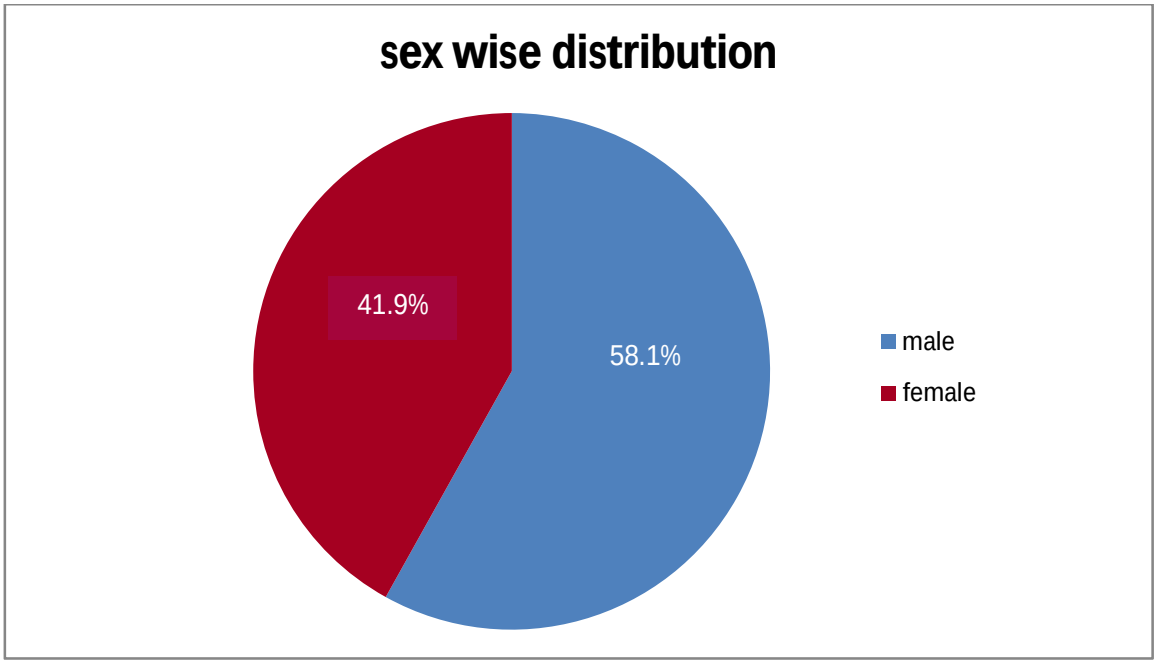


TABLE4: URBAN/ RURAL

URBAN/RURAL	NO OF BABIES	PERCENTAGE (%)
URBAN	9	20.9
RURAL	34	79.1
TOTAL	43	100.0

In this study out of 43 children, 9(20.9%) children belongs to urban area and 34 children (79.1%) belongs to rural area

TABLE 5: DIARRHOEA DURATION

DIARRHOEA	DAYS
MEAN DURATION	93.74
RANGE	18 – 420

In our study the mean duration of diarrhoea was 93.74 days, ranging from 18 - 420 days

TABLE 6: MALNUTRITION DISTRIBUTION

WT/HT	NO OF BABIES	PERCENTAGE (%)
SEVERE ACUTE MALNUTRITION	16	37.2
MODERATE ACUTE MALNUTRITION	8	18.5
SEVERE UNDERWEIGHT	3	7.0
NORMAL	16	37.3
TOTAL	43	100.0

WHO Growth chart used in children below 5yrs and IAP Growth chart used in children above 5 yrs. Malnutrition distribution of this study were 62.7%. Of which 16 children had severe acute malnutrition (37.2%), 8 children had moderate acute malnutrition (18.5%), and 3 children aged above 5 yrs with severe underweight (7%)

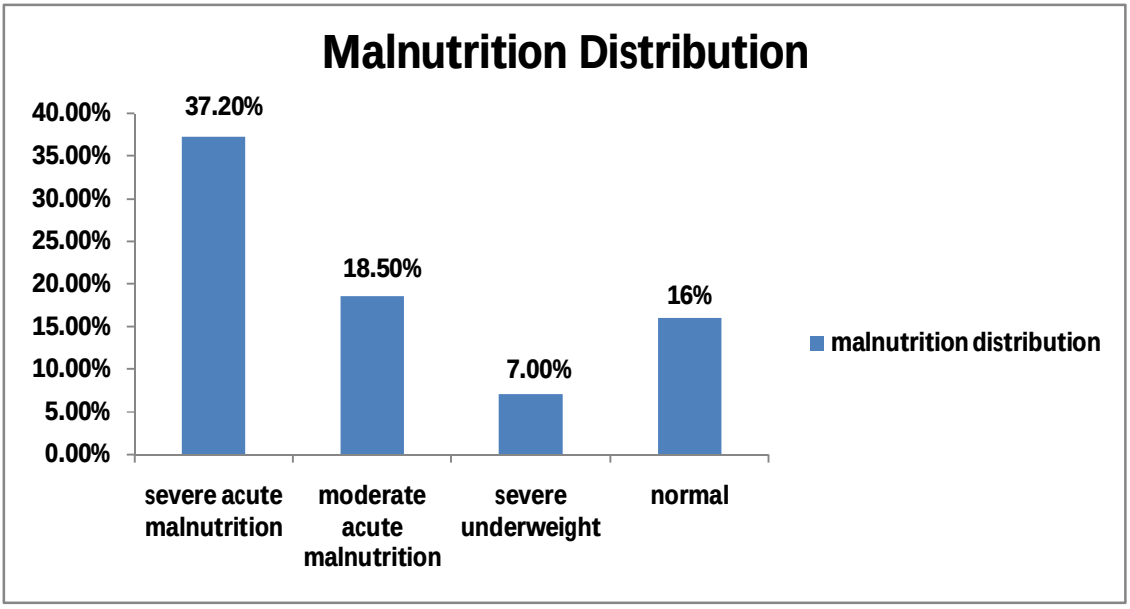


TABLE 7: TYPES OF DIARRHOEA

DIARRHOEA	MALE	FEMALE	Total
SMALL BOWEL	16	15	31(72.1%)
LARGE BOWEL	3	9	12(27.9%)
TOTAL	19	24	43(100.0%)

Small bowl diarrhoea (with watery stools), large bowel diarrhoea (small frequent stools with blood and/or mucus).

In our study out of 43 children 72.1 %(31) presented with small bowel diarrhoea and 27.9 % (12) children presented with large bowel diarrhoea . In small bowel diarrhoea out of 31 children, male (16) and female (15). In Large bowel diarrhoea out of 12 children 3 male and 9 female

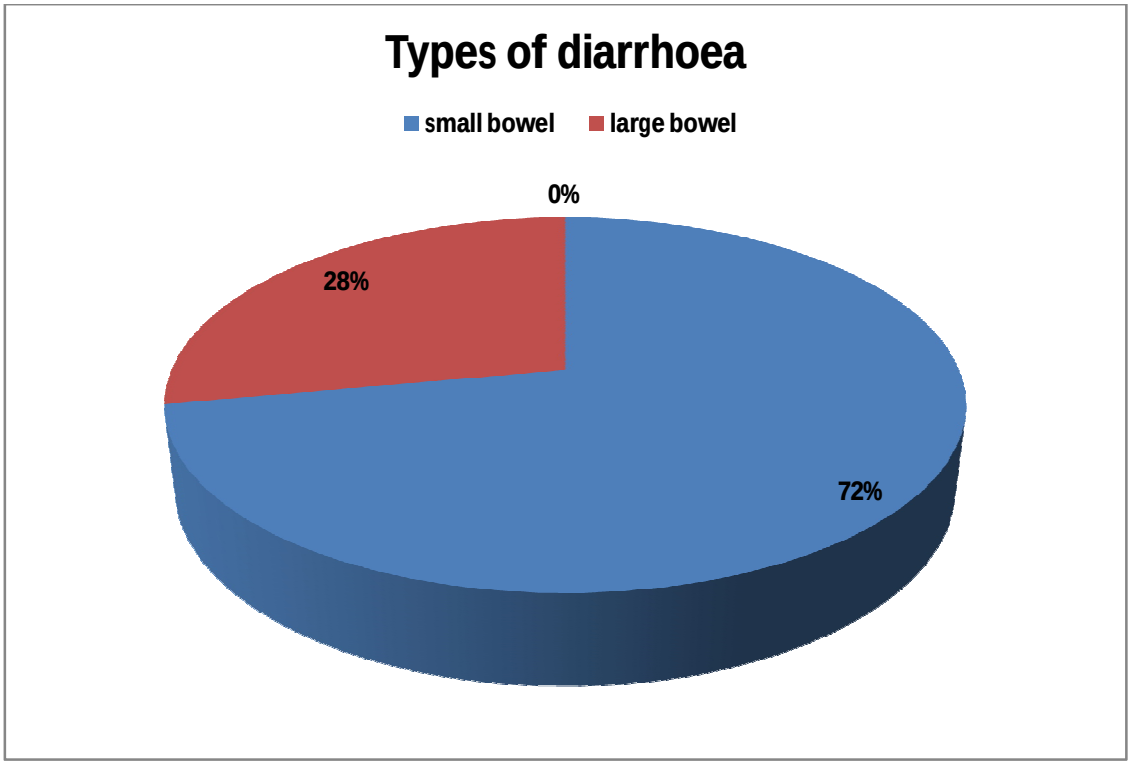


TABLE 8: EBF DURATION

DURATION	TOTAL	PERCENTAGE (%)
< 6 MONTH	17	39.5
6 MONTH	18	41.8
ON EBF	8	18.7

In our study 18 (39.5%) children had exclusive breast feeding upto 6 months, 17 children(41.8%) breast feeds less than 6 months and 8 children aged less than 6 months on EBF

TABLE 9: COW'S MILK EXPOSURE

COW'S MILK	NO OF BABIES	PERCENTAGE (%)
YES	13	30.2
NO	30	69.8
TOTAL	43	100.0

In this study out of 43 children, 13children (30.2%) had Cow's milk exposure before 1 yr of age

COW'S MILK EXPOSURE

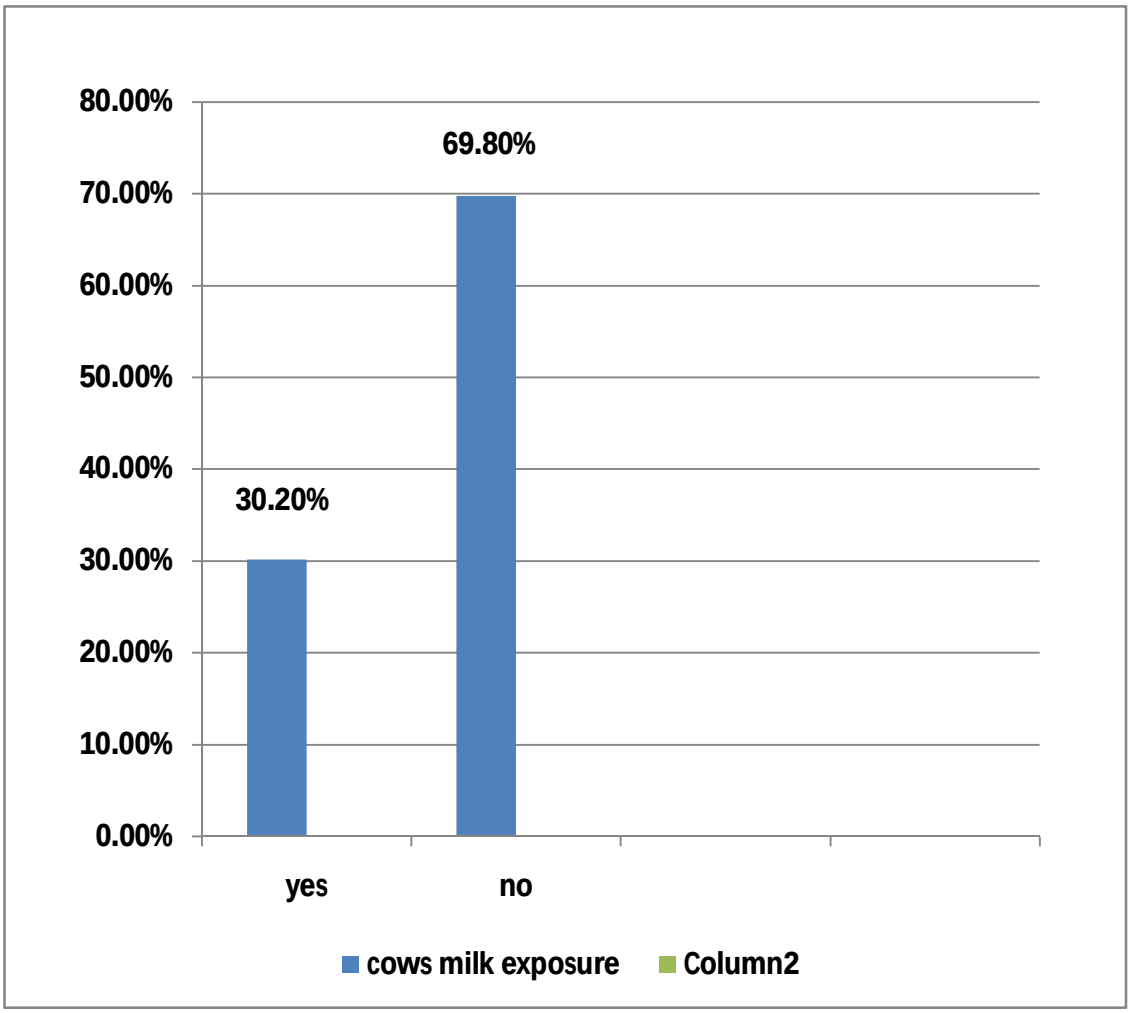


TABLE 10: GLOSSITIS

GLOSSITIS	NO OF BABIES	PERCENTAGE (%)
YES	9	20.9
NO	34	79.1
TOTAL	43	100.0

In this study out of 43 children 9 children had clinically visible glossitis (20.9%), the remaining 34 children (79.1%) were normal.

Table 11: VIT A DEFICIENCY

VIT A DEFICIENCY	NO OF BABIES	PERCENTAGE (%)
YES	4	9.3
NO	39	90.7
TOTAL	43	100.0

In this study out of 43 children, 4 children (9.3%) had Vit A deficiency, the remaining 39(90.7%) were normal. Of which 3 children had conjunctival xerosis, and 1 child presented with bitots spot

TABLE 12: EDEMA

EDEMA	NO OF BABIES	PERCENTAGE (%)
YES	**7**	**16.2**
NO	**36**	**84.8**

In this study out of 43 children ,7 children (16.2%) presented with edema, the remaining 38 children were normal

TABLE 13: HEPATOMEGALY

LIVER	NO OF BABIES	PERCENTAGE (%)
HEPATOMEGALY	**9**	**20.9**
NORMAL	**34**	**79.1**
TOTAL	**43**	**100.0**

In this study out of 43 children 9 children had hepatomegaly (20.9%), and remaining 34 (79.1%) children′s liver span were normal. Ultrasonogram were used detection.

TABLE 14: SPLENOMEGALY

SPLEEN	NO OF BABIES	PERCENTAGE (%)
SPLENOMEGALY	2	4.7
NORMAL	41	95.3
TOTAL	43	100.0

In our study 2 children had splenomegaly, the remaining were normal

15 : ANAEMIA DISTRIBUTION

ANAEMIA	NO OF BABIES	PERCENTAGE (%)
YES	30	69.8
NO	13	30.2
TOTAL	43	100.0

HB	gm/dl
MEAN HB	10.31 gm/dl
RANGE	5.2 -13.9

In this study out of 43 children, 30 children (69.8%) are anaemic. The remaining 13 children (30.2%) were normal. Mean HB 10.31 gm/dl, Ranging from 5.2-13.9 gm/dl

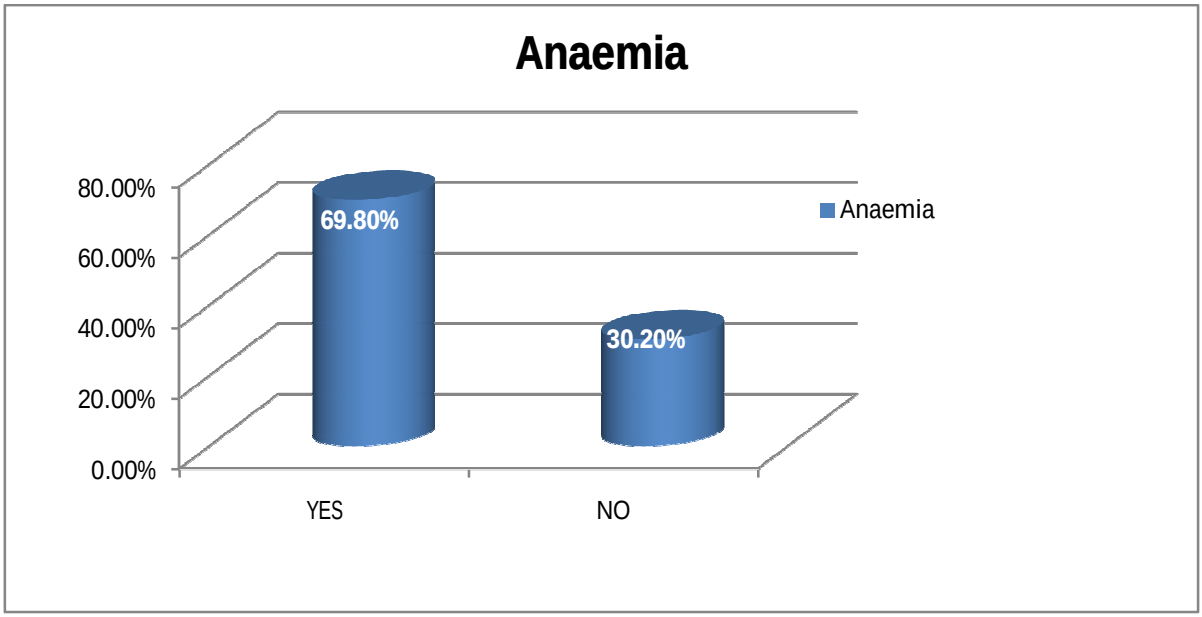


TABLE 16: HYPOPROTEINEMIA

HYPOPROTEINEMIA	NO OF BABIES	PERCENTAGE (%)
YES	11	25.6
NO	32	74.4
TOTAL	43	100.0

PROTEIN	gm/dl
MEAN PROTEIN	6.30
RANGE	3.2-8.2

In our study 11 children had hypoproteinemia (25.65%) and the remaining 32 children(74.4%) were normal. Mean protein 6.30 gm/dl,Ranging from 3.2-8.2 gm/dl

TABLE 17: CRP

CRP	NO OF BABIES	PERCENTAGE (%)
POSITIVE	17	39.5
NEGATIVE	26	60.5
TOTAL	43	100.0

In this study out of 43 children ,17 children(39.5%) were C-reactive protein positive, and 26 children were CRP negative

TABLE 18: NEC

NEC	NO OF BABIES
NG	43

Non enteric culture reports does n′t show any growth.

TABLE 19 : URINARY SEPSIS

URINE C/S	NO OF BABIES	PERCENTAGE (%)
E.COLI	3	7.0
KLEBSIELLA SP	1	2.3
NG	39	90.7
TOTAL	43	100.0

In this study Urine culture results were, 3 children shows Escherichia coli(7%), 1 children with klebsiella (2.3%) and the remaining 39 children ′s culture were normal

TABLE 20 : STOOL OVA/CYST

STOOL OVA/CYST	NO OF BABIES	PERCENTAGE (%)
CRYPTOSPORIDIUM PARVUM CYST	3	7.0
ENTAMOEBA HISTOLYTICA CYST	3	7.0
GIARDIA LAMBLIA CYST	2	4.7
NIL	35	81.4
TOTAL	43	100.0

In our study microscopic stool examination shows Cryptosporidium parvum cyst 3(7%), Entamoeba histolytica cyst 3 (7%), Giardia lamblia cyst 2(4.7%) and the remaining 35 were normal

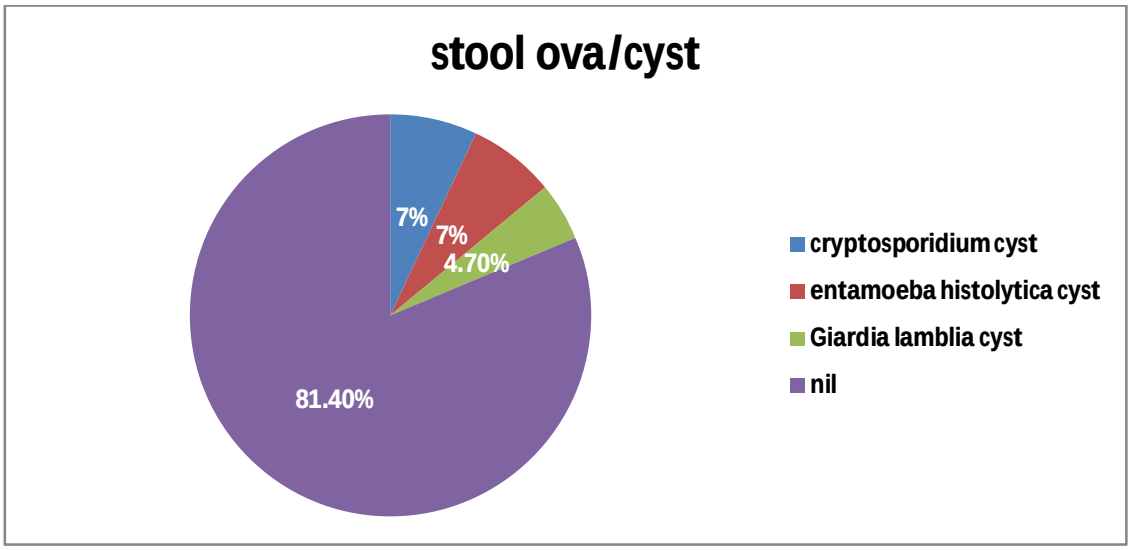


TABLE : 21 ZINC DEFICIENCY

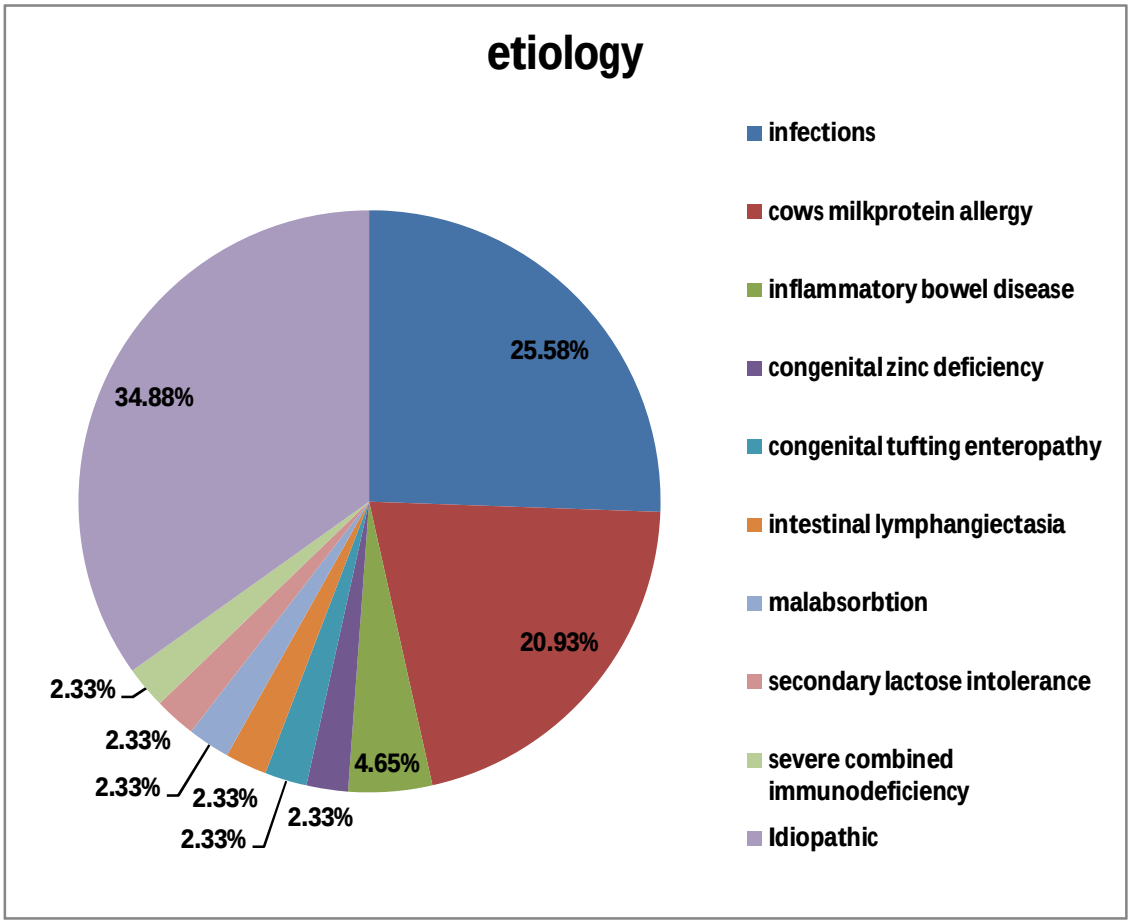
YES	**1(2.33%)**
NO	42(97.67%)

In this study zinc deficiency detected in one child.

TABLE 22: ETIOLOGY

S.NO	DIAGNOSIS	NO OF BABIES (N=43)	PERCENTAGE (%)
1	**INFECTIONS**	**11**	**25.58%**
	• SEPSIS	5	11.63%
	• CRYPTOSPORIDIUM PARVUM	3	6.97%
	• GIARDIASIS	1	2.33%
	• ENTAMOEBA HISTOLYTICA	2	4.65%
2	**COW MILK PROTEIN ALLERGY(CMPA)**	**9**	**20.93%**
3	**INFLAMMATORY BOWEL DISEASE**	**2**	**4.65%**
	• CROHN'S DISEASE	1	2.33%
	• ULCERATIVE COLITIS	1	2.33%
4	**CONGENITAL ZINC DEFICIENCY**	**1**	**2.33%**
5	**CONGENITAL TUFTING ENTEROPATHY**	**1**	**2.33%**
6	**INTESTINAL LYMPHANGIECTASIA**	**1**	**2.33%**
7	**MALABSORBTION**	**1**	**2.33%**
8	**SECONDARY LACTOSE INTOLERANCE**	**1**	**2.33%**
9	**SEVERE COMBINED IMMUNODEFICIENCY**	**1**	**2.33%**
10	**IDIOPATHIC**	**15**	**34.88%**

In this study 43 children with diarrhoea more than 2 weeks duration were examined for etiology. Results were intestinal infections(25.58%) ,Cows milk protein allergy(20.93%),Inflammatory bowel disease (4.65%),congenital zinc deficiency (2.33%), congenital tufting enteropathy (2.33%),intestinal lymphangiectasia (2.33%), malabsorbtion (2.33%),secondary lactose intolerance(2.33%), severe combined immunodeficiency (2.33%) and idiopathic (34.88%)



Small bowel etiology (72.1%)

- Idiopathic-12
- Sepsis-5
- CMPA- 4
- Cryptptosporidium parvum - 2
- Crohn´s disease-1
- Entamoeba histolytica-1
- Secondary lactose intolerance -1
- Intestinal lymphangiectasia-1
- Congenital tufting enteropathy-1
- Malabsorbtion-1
- SCID-1

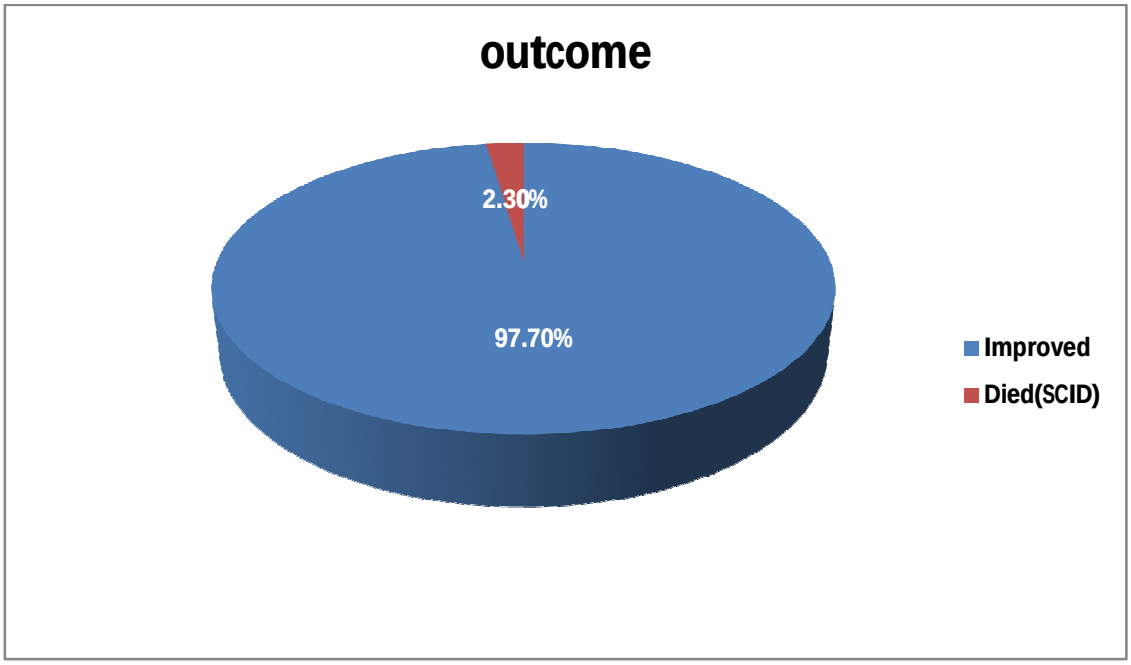
Large bowel etiology(27.9%)

- Cows milk protein allergy-5
- Congenital Zinc deficiency-1
- Idiopathic-3
- Giardiasis-1
- Entamoeba histolytica-1
- Cryptosporidium parvum -1
- Ulcerative colitis-1

TABLE23 : OUTCOME

OUTCOME	NO OF BABIES	PERCENTAGE (%)
DIED	1	2.3
IMPROVED	42	97.7
TOTAL	43	100.0

In this study out of 43 children, 42 (97.7%) children were improved with treatment and 1 (2.3%) child died



DISCUSSION

The total number of 43 children were examined during the study period. In our study out of 43 children 25 (58.1%) were male children and 18(41.9 %) were female children . male/female ratio were 1.4:1. In this study age wise distribution of chronic diarrhoea results were : 21 children (48.8%) aged < 1yr, 17 children (39.5%) aged between 1- 5 yrs and 5 children (11.7%) belongs to more than 5 yrs

Lee et al[9] in their study total number of 27 children were evaluated. Male:female ratio 1.1:1. Aluntas et al[11] done their study in 70 children of which 52% female, 48% male

Mean age of onset of diarrhoea in our study were 23 months ranging from 1 month to 120 months. Lee et al[9] in their study mean age of onset of diarrhoea were 16.4 months. Aluntas et al[11] in their study mean age of onset was 40.8 months

Mean duration of diarrhoea before admission were 93.74days in our study. Ranging from 18-420 days. In Lee et al [9] study mean duration was 66.5 days.

In our study out of 43 children, 9(20.9%) children belongs to urban area and 34 children (79.1%) belongs to rural area

Malnutrition distribution of our study were 62.7% .Of which 16 children had severe acute malnutrition (37.2%), 8 children had moderate acute malnutrition (18.5%), and 3 children aged above 5 yrs with severe underweight (7%).In Aluntas et al [11] study malnutrition was detected in 80% cases. In lee et al study 56% children had weight below 3rd percentile on admission.

In our study out of 43 children 72.1% (31) presented with small bowel diarrhoea and 27.9% (12) children presented with large bowel diarrhoea. In small bowel diarrhoea out of 31 children, male (16) and female(15). In Large bowel diarrhoea out of 12 children 3 male and 9 female. In bhatnagar et al[8] study small bowel type 103(76%), large bowel type 19(14%) and mixed 13(9.6%).

In this study out of 43 children,13children(30.2%) had Cows milk exposure before 1 yr of age

In this study out of 43 children 9 children had clinically visible glossitis (20.9%),the remaining 34 children (79.1%) were normal

In this study out of 43 children , 7 children (16.2%) presented with edema, the remaining 38 children were normal

In this study out of 43 children, 4 children (9.3%) had vit A deficiency, the remaining 39(90.7%) were normal. Of which 3 children had conjunctival xerosis, 1 child with bitot spots.

In this study out of 43 children 9 children had hepatomegaly (20.9%), and 2 children had splenomegaly(4.7%), the remaining were normal. Aluntas et al[9] in their study 20% children associated with hepatomegaly and or splenomegaly.

In this study out of 43 children , 30 children (69.8%) are anaemic. The remaining 13 children (30.2%) were normal. Mean HB was 10.31 gm/dl. Ranging from 5.2 to 13.9 gm/dl .In aluntas et al[11] study iron deficiency anemia was presented in 35 cases(44%)

In our study 11 children had hypoproteinemia (25.65%) and the remaining 32 children(74.4%) were normal. Mean protein 6.30 gm/dl. Ranging from 3.2 to 8.2 gm/dl. In aluntas et al[11]study 32% children had decreased total protein.

In this study Urine culture results were, 3 children shows Escherichia coli (7%), 1 children with klebsiella (2.3%) and the remaining 39 children ′s culture were normal

Uma Maheswari et al[21] in their study urine culture identified the following pathogen. E.coli (2 cases), Klebsiella (2 cases) and candida (1 case).

In our study microscopic stool examination shows Cryptosporidium parvum cyst 3 (7%), Entamoeba histolytica cyst 3 (7%), Giardia lamblia cyst 2(4.7%) and the remaining 35 were normal

Uma maheshwari et al[21] in their study microscopic stool examination revealed the causative organism in 13 (21.7%) cases and 6 (10%) controls. E.coli was isolated in 4 (6.6%) children while shigella was isolated in only 2 (3.3%). Giardia lamblia and Entamoeba trophozoites were documented in 2 (3.3%) and 4 (6.6%), cases respectively

In our study 43 children with diarrhoea more than 2 weeks duration were examined for etiology. Results were intestinal infections(25.58%), Cows milk protein allergy(20.93%),Inflammatory bowel disease (4.65%), congenital zinc deficiency (2.33%), congenital tufting enteropathy (2.33%), intestinal lymphangiectasia (2.33%), malabsorbtion (2.33%), secondary lactose intolerance(2.33%), severe combined immunodeficiency(2.33%) and idiopathic (34.88%)

Bhatnagar et al[8] in their study′s etiological spectrum was celiac disease(36.47%), cow milk protein allergy (11.76%), Non specific diarrhea (20%), Parasitic infestation (17.64%), Intestinal TB(2.35%), Intestinal lymphangiectasia (1.17%) ,HIV (1.17%),CVID(1.17%), and IBD(1.17%)

Aluntas et al[11]in their study Etiological factors include celiac disease(30%),tropical enteropathy (10%), CMPA (17%),Parasitic infestation (26%),IBD(10%),cystic fibrosis(10%),unknown (10%)

Yaccha et al[12] in their study, common causes of chronic diarrhoea were: protracted diarrhea 45 (33%), celiac disease 35 (26%), parasitic infestations 13 (9%), milk protein intolerance 8 (6%), intestinal tuberculosis 7 (5%). In 18 (13%) patients, cause could not be determined.

Lee et al [9] study revealed the following results CMPA (8cases), secondary lactose intolerance (4cases), gastro intestinal infections (7cases), intestinal lymphangiectasia (2cases), others (6cases)

Abdullah et al[18] in their study the etiological factors identified were: the post gastro-enteritis syndrome with or without lactose intolerance in 16 (33%); celiac disease in ten (21%); congenital chloride diarrhoea in five (10%); glucose-galactose malabsorption and acrodermatitis enteropathica, each in three (6%); ulcerative colitis, intestinal lymphangiectasia, cow's milk protein intolerance and ataxia telangiectasia, each in two (4%); and giardiasis, immune deficiency and cystic fibrosis, each in one (2%)

Rastogi et al[10] in their study results were tropical enteropathy (46.8%), irritable bowel syndrome(10.6%), giardiasis (14.8%), celiac disease(6.8%), non specific diarrhoea (21.8%).

In this study out of 43 children, 42 (97.7%) children were improved with treatment and 1(2.3%) child was died(severe combined immunodeficiency). In Lee et al[9] study out of 27 children one death occurred. In Abdullah et al study out 48 children, 5 children were died.

CONCLUSION

1. Mean age of onset of chronic diarrhoea in this study was 23 months ranging from 1month -120 months.

2. Most common age group affected in this study was < 1yr of age (48.8%).

3. Male children were more commonly affected (58.1%) than female children (41.9%). Male /female ratio were 1.4:1.

4. Rural children (79.1%) are more commonly affected than Urban (20.9%) children.

5. Mean duration of diarrhoea in this study was 93.74 days. Ranging from 18-420 days.

6. Small bowel diarrhoea (72.1%) were more common than Large bowel diarrhea (27.9%).

7. In our study Malnutrition were observed in 62.7% children

8. Vit A deficiency detected in 9.6% children

9. In this study (69.8%) of children were anaemic. Mean Hb -10.31 gm/dl, Ranging from 5.2-13.9 gm/dl.

10. Hypoproteinemia occurs in 25.65% of children, Mean protein value 6.3gm/dl .Ranging from 3.2-8.2 gm/dl

11. Urinary sepsis detected in 9.3% children. E.Coli (7%), klebsiella (2.3%)

12. Zinc deficiency was detected in 1 child

13. Commonest cause of chronic diarrhoea in our study was intestinal infections (25.58%), followed by cow's milk protein allergy(20.93%).

13. Outcome: Children improved with treatment were (97.7%). One child (2.3%) was died (SCID)

LIMITATIONS

Small number of patients

Short duration of study period (1 year)

RECOMMENDATIONS

To obtain extended etiology of chronic diarrhoea, the study period and number of patients need to be increased

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INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301A
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.S.Nanthakumar
Post Graduate in MD Paediatrics
Institute of Child Health and & Hospital for Children/
Madras Medical College
Chennai 600 003

Dear Dr.S.Nanthakumar,

The Institutional Ethics Committee has considered your request and approved your study titled **"CLINICAL PROFILE, ETIOLOGY OF DIARROHOEA > 2 WEEKS DURATION IN CHILDREN BETWEEN 1 MONTH – 12 YEARS "**
NO. 10112016.

The following members of Ethics Committee were present in the meeting hold on **01.11.2016** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.M.K.Muralidharan,MS.,M.Ch.,Dean, MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.B.Vasanthi,MD., Prof.of Pharmacology.,MMC,Ch-3	: Member
5.Prof.A.Rajendran,MS, Prof. of Surgery,MMC,Ch-3	: Member
6.Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch	: Member
7.Prof.Baby Vasumathi,MD.,Director, Inst. of O & G	: Member
8.Prof.K.Ramadevi,MD.,Director,Inst.of Bio-Che,MMC,Ch-3	: Member
9.Prof.R.Padmavathy, MD, Director,Inst.of Pathology,MMC,Ch-3	: Member
10.Prof.S.Mayilvahanan,MD,Director, Inst. of Int.Med,MMC, Ch-3	: Member
11.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
12.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
13.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

PROFORMA FOR THE STUDY

Name: Age/sex: urban/rural: hospital no:

Weight: Height/length: HC:

Detailed History of diarrhoea

Duration:

Frequency:

Consistency:

Blood/mucus:

Tenesmus:

Urgency:

Borborygmi:

Rumbling:

Fever:

Vomiting:

Abdominal pain:

Loss of appetite:

Wt loss:

Arthralgia:

Rash:

Oral ulcer:

HISTORY

Feeding history:

EBF duration:

Complementary feeding:

Solid food:

Immunization history:

Developmental history:

Clinical Examination:

General examination:

Pallor:

Glossitis:

Vit A def:

Pedal edema:

HR: RR:

Abdomen examination:

Liver:

Spleen:

Mass:

Free fluid:

Other System Examination:

CVS-

RS-

CNS-

Investigations:

- Complete Blood count
- PS
- RFT
- Sr.electrolytes
- Sr .zinc
- Sr.protein
- Blood sugar
- Urine routine:
- Stool examination -
- Ova,cyst,
- Occult blood
- Fungal elements
- Fat globules
- Reducing substance
- USG
- CXR
- Montoux:
- HIV:
- Barium meal follow through:
- Upper GI endoscopy with duodenal biopsy:
- Colonoscopy
- Anti IgA tTG

PATIENT INFORMATION SHEET

Place of study: Institute Of Child Health And Hospital for Children, Egmore, Chennai-8.

Name of Investigator: Dr. NANTHAKUMAR S

Name of Participant: **Age**: **Sex**:

Hospital No:

Study title: **Clinical profile, Etiology of diarrhoea >2 weeks duration in children between 1 month to 12 years**

We request your child to participate in the study.

Aim of the study

This study aims at determining the clinical profile and etiology of diarrhoea >2 weeks duration in children between 1 month to 12 years

Methods

In order to find out the answers to the above questions, we will be obtaining history and detailed clinical examination and draw 5 ml blood for basic investigations like CBC, peripheral smear, blood sugar, RFT, sr.electrolytes, HIV with urine routine and stool routine. your child will be subjected to imaging studies including chest Xray and USG abdomen. Second line investigations including upper GI endoscopy, barium contrast studies &colonoscopy for cases suspected to have tuberculosis, inflammatory bowel disease will be done. Sigmoidoscopy and biopsy in case of cow milk protein allergy

Can I refuse to participate in the study?

Participation in the study is purely voluntary. You may refuse to participate or withdraw from the study at any time. In both cases the treatment and care your child receives from this hospital will not be affected in any manner.

Benefits and harms of participating in the study-

Your child will not benefit directly by participating in this study. But by way of participating in this study, your child is contributing to updation of science which may benefit her/him and all other patients with this disease in future.

Drawing 5 ml blood from your child may be perceived as harm, but medically this will not compromise her/his health. Further your child will not be poked because blood for this test will be collected along with other tests advised by your doctor.

Confidentiality-

The data collected from the study will be used for the purpose of study only. The results of the study will be published. Personal information of the children participating in the study will be kept confidential. There will not be any disclosure about your child’s information without your permission.

Subject rights-

If you wish further information regarding your child's rights as a research participant, you may contact the principal investigator in the mobile number or address mentioned below.

Principal Investigator – Dr. NANTHAKUMAR S

Mobile number - 9715125434

Contact Address - II year M.D PG, Institute of Child Health and Hospital for Children, Halls road, Egmore, Chennai.

Place:

Date: Signature of Parent

INFORMED CONSENT FORM

Study place: Institute Of Child Health And Hospital For Children,
Egmore, Chennai-8.

Title of the study Clinical profile and Etiology of diarrhoea >2 weeks duration in children between 1 month to 12 years

Name of the investigator: Dr.NANTHAKUMAR.S

Name of the Participant: **Age**: **Sex**:

Hospital number:

1. I have read and understood the patient information sheet provided to me regarding the participation of my child in the study.
2. I have been explained about the nature of the study and had my questions answered to my satisfaction.
3. I have been explained about my rights and responsibilities by the investigator.
4. I will allow my child to cooperate with the investigator and undergo clinical tests subjected during the study whole heartedly.
5. I have been advised about the risks associated with my child's participation in this study.*
6. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my child's future treatment in this hospital. *
7. I hereby give permission to the investigators to release the information obtained from my child as result of participation in this study to medical journals/conference proceedings.

8. I understand that my child's identity will be kept confidential if my child's data are publicly presented/published.

9. I have decided my child can participate in the research study. I am aware that if I have any question during this study, I should contact the investigator.

10. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

Name and signature / thumb impression of the parent/guardian

Name _____ Signature_____

Date_____

Name and Signature of the investigator

Name _____ Signature_____

Date_____

Name and Signature of impartial witness 1:

Name _____ Signature_____

Date_____

Name and Signature of impartial witness 2:

Name _____ Signature_____

Date_____

ஆராய்ச்சியில் பங்கு பெறுவோர்கான தகவல் படிவம்

ஆராய்ச்சி நடத்தப்படும் இடம்	:	அரசு குழந்தைகள் நல மருத்துவமனை மற்றும் ஆராய்ச்சி நிலையம், எழும்பூர், சென்னை-8
முதன்மை ஆராய்ச்சியாளர்	:	மரு. செ,நந்தகுமார்
பங்கேற்பவர் பெயர்	:	
		வயது பாலினம்
மருத்துவமனை எண்	:	
ஆய்வின் தலைப்பு	:	இரண்டு வாரத்திற்கு மேற்பட்ட வயிற்றுப் போக்கு உள்ள ஒரு மாதம் முதல் 12 வயது வரை குழந்தைகளுக்கு வயிற்றுப் போக்கிற்கான மருத்துவ சுயவிவரம் மற்றும் நோய் காரணவியலை கண்டறியும் ஆய்வு

நாங்கள் உங்கள் குழந்தையை இந்த ஆய்வில் பங்கெடுக்குமாறு கேட்டுக்கொள்கிறோம்

ஆய்வின் நோக்கம்:

இரண்டு வாரத்திற்கு மேற்பட்ட வயிற்றுப் போக்கு உள்ள ஒரு மாதம் முதல் 12 வயது வரை குழந்தைகளுக்கு வயிற்றுப் போக்கிற்கான மருத்துவ சுயவிவரம் மற்றும் நோய் காரணவியலை கண்டறியும் ஆய்வு

செய்முறை

இந்த ஆய்விற்காக உங்கள் குழந்தையின் மருத்துவ விவரம் கேட்கப்பட்டு விரிவான மருத்துவ பரிசோதனை செய்யப்படும். மேலும் இந்த ஆய்விற்காக 5 மிலி இரத்தம் எடுத்து இரத்த அணுக்கள், சர்க்கரை அளவு, உப்பு அளவினைக் கண்டறிவோம் சிறுநீர் மற்றும் மலம் எடுத்து பரிசோதனை செய்யப்படும். எக்ஸ்ரே மற்றும் ஸ்கேன் பரிசோதனைச் செய்யப்படும். தேவைப்படும் பட்சத்தில் உணவு குழலில் நுண்குழாய் செலுத்தி குடல் பரிசோதனை செய்யப்படும்.

ஆய்வில் பங்கேற்க மறுத்தால்

இந்த ஆய்வில் பங்கேற்பது முற்றிலும் உங்களது சொந்த விருப்பமே தாங்கள் எப்பொழுது வேண்டுமானாலும் இவ்வாராய்ச்சியிலிருந்து விலகிக் கொள்ளலாம். தாங்கள் விலகி கொள்வதால் உங்கள் குழந்தைக்கு அளிக்கப்படும் சிகிச்சையில் எந்தவித மாற்றமோ, பாதிப்போ இருக்காது.

பங்கேற்பதின் இலாப நஷ்டங்கள்

இந்த ஆய்வில் இருந்து பெறப்படும் தகவல்கள் நம் நாட்டை நோயில்லாத நாடாக மாற்ற உபயோகப்படும். இவ்வாறு நாட்டின் வளர்ச்சியில் பங்கேற்ற பெருமை உங்களையும், உங்கள் குழந்தையையும் சேரும்.

இரகசியத்தன்மை

ஆய்வில் இருந்து பெறப்படும் தகவல்கள் வெளியிடப்படும்பொழுது உங்கள் மற்றும் உங்கள் குழந்தையின் அடையாளம் இரகசியமாக வைக்கப்படும்.

பங்கேற்பவர் உரிமை

இந்த ஆய்வைப் பற்றி மேலும தகவல் அறிய தொடர்பு கொள்ள வேண்டிய நபர்

முதன்மை ஆராய்ச்சியாளர்	:	செ.நந்தகுமார்
கைபேசி எண்	:	9715125434
முகவரி	:	இரண்டாம் ஆண்டு, முதுநிலை மருத்துவ மாணவர் அரசு குழந்தைகள் நல மருத்துவமனை மற்றும் ஆராய்ச்சி நிலையம், எழும்பூர், சென்னை-8.

இடம்:

தேதி: பெற்றோர் கையொப்பம்

ஒப்புதல் படிவம்

ஆராய்ச்சி நடத்தப்படும் இடம்	:	அரசு குழந்தைகள் நல மருத்துவமனை மற்றும் ஆராய்ச்சி நிலையம், எழும்பூர், சென்னை-8
முதன்மை ஆராய்ச்சியாளர்	:	மரு. செ.நந்தகுமார்
பங்கேற்பவர் பெயர்	:	
		வயது பாலினம்
மருத்துவமனை எண்	:	
ஆய்வின் தலைப்பு	:	இரண்டு வாரத்திற்கு மேற்பட்ட வயிற்றுப் போக்கு உள்ள ஒரு மாதம் முதல் 12 வயது வரை குழந்தைகளுக்கு வயிற்றுப் போக்கிற்கான மருத்துவ சுயவிவரம் மற்றும் நோய் காரணவியலை கண்டறியும் ஆய்வு

1) எனக்கு தரப்பட்ட ஆராய்ச்சியில் பங்கு பெறுவோர்க்கான தகவல் படிவத்தை முழுவதுமாக படித்து புரிந்து கொண்டேன்.
2) ஆராய்ச்சியின் தன்மை முழுவதுமாகவும் விரிவாகவும் எடுத்துரைக்கப்பட்டது. எனது கேள்விகளுக்கு விடையளிக்கப்பட்டது.
3) ஆய்வாளர் என் உரிமைகளையும், பொறுப்புகளையும் நன்கு விளக்கினார்.
4) நான் எனது குழந்தை ஆய்வாளருக்கு முழு ஒத்துழைப்பு கொடுக்கவும், பரிசோதனை செய்து கொள்ளவும் அனுமதிக்கிறேன்.
5) எனது குழந்தை ஆராய்ச்சியில் பங்கேற்பதால் ஏற்படும் சாதக பாதகங்களை விளக்கப்பட்டன.
6) நான் எப்பொழுது வேண்டுமானாலும் எனது குழந்தையை இந்த ஆராய்ச்சியிலிருந்து விலக்கிக் கொள்ளலாம் என்று எனக்கு எடுத்துரைக்கப்பப்பட்டது. அவ்வாறு விலகிக்கொள்வதால் குழந்தைக்கு அளிக்கப்படும் சிகிச்சையில் எந்த மாற்றமும் இருக்காது என அறிந்து கொண்டேன்.

7) இந்த ஆய்வில் என் குழந்தையிடமிருந்து பெறப்படும் மருத்துவ தகவலை ஆய்விதழிலிலோ, கருத்தரங்கிலோ வெளியிடுவதில் எனக்கு எந்தவித ஆட்சேபணையும் இல்லை.
8) அவ்வாறு வெளியிடப்படும்போது என் குழந்தையின் தன் அடையாளங்களை வெளியிடப்பட மாட்டாது என எனக்கு உறுதியளிக்கப்பட்டது.
9) எனக்கு இந்த ஆராய்ச்சி குறித்து எதுவும் சந்தேகம் இருந்தால் உடனே ஆராய்ச்சியாளரை கேட்டு தெளிவுப்படுத்திக் கொள்ளலாம் என தெரிவிக்கப்பட்டது.
10) இந்த ஒப்புதல் படிவத்தில் கையொப்பமிடுவதின் மூலம் இந்த படிவத்தில் உள்ளவை யாவும் எனக்கு தெளிவாக எடுத்துரைக்கப்பட்டு அதை நான் நன்கு புரிந்துக்கொண்டேன் என தெரிவித்துக்கொள்கிறேன்.

நோயாளியின் பெற்றோர் / பாதுகாவலர்

பெயர்: ....

கையொப்பம்: ....
தேதி:

ஆராய்ச்சியாளர்

பெயர்: ....

கையொப்பம்: ....
தேதி:

சாட்சி 1.

பெயர்: ....

கையொப்பம்: ....
தேதி:

சாட்சி 2:

பெயர்: ....

கையொப்பம்: ....
தேதி:

MASTER CHART

Name	Age	sex	urban/rural	ip no	wt	wt/age	ht	ht/age	Wt/Ht	HC	HC/age	diarrhoea duration	frequency	small bowel diarrhoea
Himalaya	12 mon	fch	urban	947329	10	normal	60	< -3SD	normal	43	normal	2 mon	10-12epi/day	yes
dharnesh	6mon	mch	rural	951599	7.3	normal	67	normal	normal	43	normal	45 days	5-8epi/day	yes
b/o jeevitha	1 mon	fch	rural	950654	2.48	< 3SD	48	2 to 3SD	2 to 3SD	33	normal	25 days	10epi/day	yes
vishnuwarthan	12mon	mch	rural	948086	7	-2 to -3SD	68	-2 to -3SD	normal	44	normal	18days	5-7epi/day	yes
danaraksha	12mon	fch	rural	938214	7.8	normal	76	normal	-2 to -3SD	42	normal	20 days	10 epi/day	no
yogasri	4mon	fch	rural	948864	2.7	< -3SD	55	< -3SD	< -3SD	36	< -3SD	4mon	20-25epi/day	yes
dhakshitha	14mon	fch	urban	948830	8.2	normal	75	normal	normal	42	normal	4mon	5-6epi/day	yes
Navyasri	10 mon	fch	rural	949025	5.4	-2 to -3SD	66	-2 to -3SD	< -3SD	46	normal	1 mon	7-10 epi/day	yes
sharika	4mon	fch	rural	948971	4	< -3SD	56	-2 to -3SD	-2 to -3SD	41	normal	3mon	8-10epi/day	yes
b/o birundarani	3mon	fch	rural	947631	2.9	< -3SD	54	-2 to -3SD	< -3SD	40	normal	45days	10-12epi/day	no
Md Thakir	120 mon	mch	rural	946498	15	underweight	110	stunting	severe underweight	52	normal	20 days	5epi/day	yes
dhanacheziyan	84mon	mch	rural	946022	11	underweight	100	stunting	severe underweight	54	normal	2yr	10-12 epi/day	no
Thanush	10 mon	mch	rural	946311	6.2	< -3SD	70	normal	< -3SD	43.5	normal	5 mon	5-7 epi /day	no
manikandan	8mon	mch	rural	946171	4.7	< -3SD	63	< -3SD	< -3SD	44	normal	5mon	8-10 epi/day	no
Varun	4 mon	mch	rural	946267	3.5	< -3SD	55	< -3SD	< -3SD	42	normal	1mon	8-10epi/day	yes
bargavi sudarsana	2mon	fch	rural	946004	3.4	< -3SD	56.5	normal	normal	38	normal	1mon	10-15 epi/day	yes
koushika	3mon	fch	rural	942355	4.5	normal	56	normal	normal	38	normal	45days	7epi/day	yes
kiruthika	4mon	fch	rural	939977	5.5	normal	56	< -3SD	normal	41	normal	1mon	8 epi/day	yes
b/o deepika	3mon	mch	urban	938068	4.3	-2 to -3SD	51	< -3SD	normal	36.5	<-3SD	20 days	10epi/day	yes
tharun	8mon	mch	rural	942730	7	normal	73	normal	< -3SD	44	normal	20days	10epi/day	yes
b/o kalaiyarasi	2mon	fch	urban	941477	2.8	< -3SD	53	normal	< -3SD	36	normal	45days	10epi/day	yes
pugazhnidhi	84mon	mch	rural	935384	15	underweight	108	stunting	severe underweight	50	normal	12 mon	4epi/day	no
dharshan	60 mon	mch	urban	934820	15	normal	102	normal	normal	46	<-3SD	21 days	6 epi/day	yes
Rathinilavan	24mon	mch	rural	944938	10	normal	74	< -3SD	normal	44	<-3SD	28days	10-15epi/day	yes
Madesh	60mon	mch	urban	944455	10	< -3SD	96	< -3SD	< -3SD	41	<-3SD	6 mon	3-4 epi/day	yes
dharshini	26mon	fch	urban	894591	7	< 3SD	72	< 3SD	2 to 3SD	44	normal	21days	8cpi/day	yes
chandru	40mon	mch	rural	927748	7.3	< -3SD	76	< -3SD	< -3SD	38	<-3SD	1mon	10epi/day	no
Rithish	18mon	mch	rural	929892	6	< -3SD	80	< -3SD	< -3SD	44	normal	6mon	5-6epi/day	yes
gokul	2mon	mch	rural	933207	3.28	< -3SD	53	< -3SD	normal	35	<-3SD	35days	10epi/day	yes
kavya	48mon	fch	rural	980010	12	-2 to -3SD	94	normal	normal	45	< -3SD	6mon	3epi/day	yes
sukli	36mon	fch	rural	937369	10	< -3SD	87	-2 to -3SD	normal	47	normal	20days	10epi/day	yes
yethin	10mon	mch	rural	943460	6	< -3SD	65	< -3SD	-2 to -3SD	42	normal	4mon	10-15epi/day	no
veeramani	24mon	mch	rural	939143	6	< -3SD	74	< -3SD	< -3SD	44	< -3SD	20days	4epi/day	yes
rakshitha	36mon	fch	urban	926360	4.5	< 3SD	55	< 3SD	< 3SD	37	< 3SD	25days	8cpi/day	yes
md ashil	24mon	mch	rural	943392	7.8	< -3SD	74	< -3SD	-2 to -3SD	44	< -3SD	7mon	5-10epi/day	no
vishnu	12mon	mch	rural	952781	7.7	-2 to -3SD	74.5	normal	-2 to -3SD	43.5	normal	1mon	6-7 epi/day	no
b/o jeevadharshini	3mon	fch	rural	941275	4.2	-2 to -3SD	54	-2 to -3SD	normal	38	normal	25days	5-8epi/day	yes
ashik mohamed	8mon	mch	rural	941730	6.3	-2 to -3SD	68	normal	-2 to -3SD	42	normal	1mon	4-6epi/day	yes
morshith	2mon	mch	rural	922060	3.3	< -3SD	57	normal	< -3SD	38	normal	2mon	5-7epi/day	yes
b/o gurulaksmi	2mon	mch	rural	931045	3.6	< -3SD	58	normal	< -3SD	37	normal	1mon	4-6epi/day	yes
joswa	108 mon	fch	urban	954188	25.5	normal	136	normal	normal	52	normal	18days	4-5epi/day	no
ramakanth	12 mon	mch	rural	955702	10	normal	72	normal	normal	43	normal	4mon	5-6 epi /day	no
nani	24 mon	mch	rural	958428	5.5	< -3SD	70	< -3SD	< -3SD	44	normal	14 mon	4-5 epi/day	yes

large bowel diarrhoea	tenesmus	urgency	borborgymi	rumbling	fever	vomiting	abdominal pain	appetiteloss	wt loss	arthralgia	anal excoriation	oral ulcer	EBF duration	cows milk
no	no	no	no	no	no	yes	no	no	no	no	no	no	6 mon	no
no	no	no	no	no	yes	yes	no	no	no	no	no	no	5mon	no
no	no	no	no	no	no	no	no	no	yes	no	yes	no	on EBF	no
no	no	no	no	no	no	no	no	no	yes	no	yes	no	3mon	no
yes	no	no	no	no	yes	yes	no	no	no	no	no	no	6mon	yes
no	no	no	no	no	no	no	no	no	yes	no	no	no	40days	yes
no	no	no	no	no	no	yes	no	no	no	no	no	no	6mon	yes
no	no	no	yes	no	no	no	no	no	yes	no	yes	no	6mon	no
no	no	no	no	no	yes	yes	no	no	yes	no	no	no	2mon	no
yes	no	no	no	no	no	yes	no	no	yes	no	yes	no	1mon	no
no	no	no	no	no	yes	no	no	no	yes	no	no	yes	6mon	no
yes	no	no	no	no	no	no	yes	no	yes	no	no	no	5mon	no
yes	no	no	no	no	no	no	no	no	yes	no	no	no	5mon	no
yes	yes	no	no	no	no	no	no	no	yes	no	no	no	5mon	no
no	no	no	no	no	no	yes	no	no	no	no	no	no	NIL	no
no	no	no	no	no	no	no	no	no	yes	no	no	no	1mon	no
no	no	no	no	no	no	no	no	no	no	no	no	no	on EBF	no
no	no	no	no	no	no	no	no	no	no	no	no	no	on EBF	no
no	no	no	no	no	yes	no	no	no	no	no	yes	no	on EBF	no
no	no	no	no	no	yes	yes	no	no	no	no	yes	no	3mon	yes
no	no	no	no	no	yes	yes	no	no	yes	no	no	no	on EBF	no
yes	no	no	no	no	no	no	no	no	no	no	no	no	6mon	no
no	no	yes	no	yes	yes	no	yes	no	no	yes	no	yes	6mon	no
no	no	no	no	no	no	no	no	no	yes	no	no	yes	6mon	no
no	no	no	no	no	no	no	no	no	no	no	no	no	6mon	no
no	no	no	no	no	yes	yes	no	no	yes	no	no	yes	6mon	no
yes	no	no	no	no	no	no	yes	no	no	no	no	yes	6mon	no
no	no	no	no	no	no	no	no	no	no	no	no	no	6mon	no
no	no	no	yes	no	no	yes	no	no	yes	no	no	no	4days	yes
no	no	no	no	no	no	no	no	no	no	no	no	no	6mon	no
no	no	no	no	no	no	yes	no	no	no	no	no	yes	6mon	yes
yes	no	no	no	no	no	no	no	no	no	no	no	no	4mon	yes
no	no	no	yes	no	no	yes	no	no	no	no	no	no	3mon	yes
no	no	no	no	no	no	yes	no	no	no	no	no	no	3mon	no
yes	no	no	no	no	no	yes	yes	no	no	no	no	no	6mon	no
yes	no	no	no	no	yes	yes	no	no	yes	no	no	no	6mon	yes
no	no	no	no	no	no	yes	no	no	no	no	no	no	on EBF	no
no	no	no	no	no	yes	yes	no	no	yes	no	no	no	5mon	no
no	no	no	no	no	yes	no	no	no	no	no	no	no	NIL	yes
no	no	no	no	no	yes	no	no	no	no	no	no	no	on EBF	no
yes	yes	yes	no	no	no	no	yes	no	no	no	no	no	6mon	yes
yes	no	no	no	no	yes	yes	no	no	no	no	no	yes	6mon	yes
no	no	no	no	no	no	no	no	no	yes	no	no	yes	6mon	yes

complementary feeds	solid foods	immun H/O	develoH/O	pallor	glossitis	vit A def	pedal edema	HR/min	RR/min	liver	spleen	mass	free fluid	CVS
after 6 mon	8mon	upto the age	normal	yes	no	no	yes	110	30	-	-	-	-	normal
after 6 mon	9mon	upto the age	normal	no	no	no	-	108	24	-	-	-	-	normal
-	-	upto the age	normal	no	no	no	yes	120	42	-	-	-	-	normal
4th mon	7th mon	upto the age	normal	yes	no	no	-	100	28	hepatomegaly	-	-	-	normal
after 6 mon	6th mon	upto the age	normal	no	no	no	-	100	24	-	-	-	-	normal
-	-	upto the age	normal	yes	no	no	-	130	30	-	-	-	-	normal
after 6 mon	8th mon	upto the age	normal	no	no	no	-	120	30	-	-	-	-	normal
after 6 mon	8th mon	measles not given	normal	yes	no	no	yes	120	36	-	-	-	-	normal
-	-	upto the age	normal	yes	no	no	-	120	40	-	-	-	-	normal
-	-	BCG,OPV Only	normal	yes	no	no	-	130	40	-	-	-	-	normal
after 6 mon	9th mon	upto the age	normal	yes	yes	yes	-	110	20	-	-	-	-	normal
after 6 mon	8th mon	upto the age	normal	yes	no	no	-	110	26	-	-	-	-	normal
after 6 mon	8th mon	upto the age	normal	yes	no	no	-	110	28	hepatomegaly	-	-	-	normal
after 5 mon	7th mon	upto the age	normal	yes	no	no	yes	140	60	hepatomegaly	-	-	-	normal
-	-	BCG,OPV Only	normal	yes	no	no	-	140	60	hepatomegaly	-	-	-	normal
-	-	upto the age	-	yes	no	no	-	120	30	hepatomegaly	-	-	-	normal
-	-	upto the age	normal	yes	no	no	-	120	30	-	-	-	-	normal
-	-	upto the age	normal	yes	no	no	-	130	30	-	-	-	-	normal
-	-	upto the age	normal	yes	no	no	-	120	44	hepatomegaly	splenomegaly	-	yes	normal
after 6mon	8th mon	upto the age	normal	yes	yes	yes	yes	110	40					normal
-	-	upto the age	normal	yes	no	no	-	120	36	-	-	-	-	normal
after 6mon	8th mon	upto the age	normal	yes	no	no		120	32	-	-		-	normal
after 6mon	6th mon	upto the age	normal	yes	yes	no	-	90	22	-	-	-	-	normal
after6mon	12th mon	upto the age	normal	yes	yes	no	-	106	32	-	-	-	-	normal
after6mon	12th mon	upto the age	normal	yes	no	no	yes	110	30	-	-	-	-	normal
after 6mon	9th mon	upto the age	normal	no	yes	no	-	110	30	-	-	-	-	normal
after6mon	8th mon	upto the age	yes	yes	yes	yes	-	110	30	-	-	-	-	normal
after6mon	9th mon	upto the age	normal	yes	no	no	-	110	30	-	-	-	-	normal
-	-	upto the age	-	yes	no	no	-	130	36	-	-	-	-	normal
after 6mon	6th mon	upto the age	normal	yes	no	no	-	102	20	-	-	-	-	normal
after6mon	9th mon	upto the age	normal	no	yes	no	-	110	30	hepatomegaly	-	-	-	normal
after6mon	8th mon	upto the age	normal	no	no	no	-	110	26	-	-	-	-	normal
after6mon	7th mon	upto the age	normal	yes	no	no	-	100	30	-	-	-	-	normal
after6mon	8th mon	upto the age	normal	no	no	no	-	110	30	hepatomegaly	splenomegaly	-	-	normal
after6mon	8th mon	upto the age	normal	yes	no	no	yes	104	24	-	-	-	-	normal
after6mon	8th mon	upto the age	normal	no	no	no	-	110	30	hepatomegaly	-	-	-	normal
after6mon	8th mon	upto the age	normal	yes	no	no	-	120	40	-	-	-	-	normal
after6mon	7th mon	upto the age	normal	yes	no	no	-	110	30	-	-	-	-	normal
-	-	upto the age	normal	no	no	no	-	110	30	-	-	-	-	normal
-	-	upto the age	normal	no	no	no	-	120	30	-	-	-	-	normal
after 6mon	8thmon	upto the age	normal	yes	no	no	-	110	22	-	-	-	-	normal
after 6mon	7th mon	upto the age	normal	yes	yes	yes	-	100	30	-	-	-	-	normal
after 6mon	8th mon	upto the age	normal	yes	yes	no	-	100	28	-	-	-	-	normal

RS	CNS	Anaemia	HB	TC	DC	PLT	PCV	PS	UREA	CREAT	NA	K	GLUCOSE	T.BILI	D.BILI	T.PROTEINS	Hypoproteinemia	ALBUMIN	GLOBULIN
normal	normal	no	12.4	27000	30/70	4.42	36.2	NCNC	24	0.5	138	4.5	98	<1	-	7.2	no	4.2	3
normal	normal	no	11.8	27800	31/69	4.19	34.5	NCNC	13	0.6	137	4.7	94	<1		8.2	no	5.2	3
normal	normal	no	13.2	6700	38/62	2.2	37.2	NCNC	29	0.5	139	5	92	<1	-	6.8	no	4.2	2.6
normal	normal	yes	8.2	19200	36/64	2.39	26.6	MCHC	30	0.6	146	4.6	104	<1	-	6.4	no	3.8	2.6
normal	normal	no	11.9	9500	56/42/2	2.4	34	MCHC	24	0.6	137	3.9	96	<1	-	7	no	4.3	2.7
normal	normal	yes	9.7	16400	44/43/13	6.01	29.6	MCHC	18	0.6	137	3.6	112	<1	-	5.2	yes	3.1	2.1
normal	normal	yes	10.3	9500	35/50/15	5.9	31.9	MCHC	23	0.6	134	3.8	138	<1	-	5.8	yes	4.2	1.6
normal	normal	no	11.7	11300	64/34	4.9	33.2	MCHC	24	0.6	134	4.4	92	<1	-	6.3	no	4.2	2.1
normal	normal	yes	9.6	31500	71/23/6	4.2	27.2	MCHC	15	0.5	132	4.2	125	<1	-	5.2	no	2.9	2.3
normal	normal	no	13.9	50300	80/19/1	8.2	43.2	NCNC	15	0.5	131	4.9	88	<1	-	4.5	yes	2	2.5
normal	normal	yes	10.5	14900	79/15/6	7.92	34.5	MCHC	18	0.5	135	3.3	112	<1	-	3.2	yes	0.7	2.5
normal	normal	no	12.2	12700	57/36/07	4.99	35	NCNC	20	0.6	136	3.5	71	<1	-	7.8	no	4.6	3.2
normal	normal	yes	10.4	11400	28/60/12	4.16	32	MCHC	15	0.5	135	4.6	108	<1	-	6.7	no	4.2	2.5
B/L crepts +	normal	no	12	16000	38/52/10	5.2	36.6	NCNC	31	0.6	138	5.5	89	<1	-	6.4	no	4.2	2.2
normal	normal	no	11	18500	47/40/13	5.23	32.6	NCNC	37	0.7	142	3	106	<1	-	7.4	no	3.9	3.4
normal	normal	yes	10.4	16300	33/55/12	2.96	29.8	MCHC	14	0.6	133	4.6	102	<1		6.8	no	4.2	2.6
normal	normal	yes	9.1	4800	21/77/02	4.04	28	MCHC	13	0.4	138	3.8	106	<1	-	7.4	no	4.8	3.6
normal	normal	yes	8.3	9900	19/81	3.42	25.3	MCHC	14	0.4	134	5.5	102	<1	-	6.8	no	4.4	2.4
normal	normal	yes	5.2	10490	42/55/3	28000	17.5	MCHC	14	0.4	134	4.2	118	<1	-	4.4	yes	3.1	1.3
normal	normal	yes	8.2	12200	51/34/15	6.61	28.1	MCHC	35	0.7	135	4.1	70	<1	-	6.3	no	4	2.3
normal	normal	yes	9.9	18800	61/30/9	7.4	29.9	MCHC	92	0.9	150	4.5	74	<1	-	6.6	no	4.2	2.4
normal	normal	yes	11.2	12700	50/33/17	4.88	33.5	NCNC	14	0.5	136	4	102	<1	-	7.3	no	4	3.3
normal	normal	no	12.5	12100	70/23/7	5.36	37.5	NCNC	15	0.6	134	4	128	<1	-	7.4	no	4.4	3
normal	normal	yes	9.1	14700	62/34/4	5.3	31.3	MCHC	34	0.8	126	3.3	102	<1	-	4.2	yes	2.2	2
normal	normal	no	12.5	8400	35/56/9	2.86	38.7	MCHC	27	0.6	140	4.1	102	<1	-	7.1	no	4.3	2.8
normal	normal	yes	10.5	9000	40/50/10	3.5	30	MCHC	20	0.6	135	4.5	105	<1	-	7.2	no	3.8	3.4
normal	normal	yes	11.3	14000	72/12/6	34000	37.2	NCNC	15	0.4	132	1.6	130	<1	-	7.4	no	4.4	3
normal	normal	no	11.5	10000	79/21	3.41	34	NCNC	15	0.5	133	4.5	102	<1	-	5.4	yes	3.7	1.7
normal	normal	yes	9.3	18700	39/60/1	2.81	28.2	MCHC	34	0.5	144	3.8	72	<1	-	5.7	no	3.8	1.9
normal	normal	yes	10.2	9000	47/53	5.32	30.5	MCHC	15	0.7	138	3.9	88	<1	-	6.8	no	4.2	2.6
normal	normal	no	12.1	13500	33/54/13	3.13	38	MCHC	20	0.7	140	4.1	94	<1	-	4.3	yes	2.7	1.6
normal	normal	yes	11.2	7900	80/20	2.94	31.2	NCNC	22	0.6	134	4	82	<1	-	7	no	4.7	2.3
normal	normal	yes	8.5	4600	59/41	4.2	25.2	MCHC	25	0.6	127	2.8	86	<1	-	7.7	no	4.1	3.6
normal	normal	yes	11	15800	26/74	4.77	30.9	NCNC	15	0.4	133	4.1	102	<1	-	5.1	yes	3.4	1.7
normal	normal	yes	7	8700	23/69/08	3.38	28.3	MCHC	15	0.6	139	3.9	63	<1	-	6.5	no	4.4	2.1
normal	normal	yes	10.7	21000	55/37	4.19	31.1	MCHC	14	0.4	137	3.7	71	<1	-	7.2	no	3.8	3.4
normal	normal	yes	9.2	13400	48/44/8	3.24	28.9	MCHC	15	0.6	134	4.2	96	<1	-	6.8	no	4.2	2.6
normal	normal	yes	8.8	12600	54/42/4	4.7	25.4	MCHC	13	0.8	136	3.8	102	<1	-	7.4	no	4.8	2.6
normal	normal	yes	10.1	21500	49/35/15	5.55	30.3	MCHC	19	0.6	138	4	96	<1	-	5.1	no	3.5	1.6
normal	normal	yes	11.2	32500	54/34/12	4.2	34.2	MCHC	15	0.6	134	4.3	102	<1	-	6.2	no	3.8	2.4
normal	normal	yes	9.7	12700	47/20/33	2.88	29.3	MCHC	14	0.9	136	4	124	<1	-	6.6	no	3.6	3
normal	normal	yes	8.2	5900	26/62/12	3.92	26.5	MCHC	14	0.3	135	3.7	107	<1	-	6.1	yes	3.1	3
normal	normal	yes	7.1	14410	38/54/2	2.33	27.6	MCHC	15	0.4	139	4	112	<1	-	4.2	yes	2.5	1.7

OT	PT	ZINC	URINE ALB	DEPOSIT	CRP	URINE C/S	NEC	STOOL OVA/CYST	OCCULT BLOOD	FUNGAL	FAT GLOBULES	RED. SUBSTANCE
44	17	86	NIL	NIL	negative	NG	NG	NIL	NEG	NIL	NIL	NIL
32	24	102	NIL	NIL	positive	NG	NG	NIL	NEG	NIL	NIL	NIL
21	10	96	NIL	NIL	negative	NG	NG	NIL	NEG	NIL	PRESENT	++++
52	17	127	NIL	NIL	negative	NG	NG	NIL	NEG	NIL	PRESENT	+++
92	72	65.5	NIL	NIL	negative	NG	NG	NIL	NEG	NIL	PRESENT	+
49	38	123	NIL	10-15Pus cells	positive	E.coli	NG	NIL	NEG	NIL	PRESENT	+
32	22	86	NIL	NIL	negative	NG	NG	Entamoeba hystolytica cyst	NEG	NIL	-	-
34	20	92	NIL	4-5pus cells	negative	NG	NG	NIL	NEG	NIL	NIL	+
57	25	122	NIL	NIL	positive	NG	NG	NIL	NEG	NIL	PRESENT	NIL
49	74	37	+	2-3 pus cells	negative	NG	NG	NIL	NEG	NIL	NIL	+
15	10	106	++++	2-4pus cells	negative	NG	NG	Cryptosporidium parvum cyst	NEG	NIL	PRESENT	NIL
25	15	97	NIL	NIL	positive	NG	NG	NIL	NEG	NIL	NIL	NIL
33	16	102	NIL	NIL	positive	NG	NG	NIL	positive	NIL	NIL	NIL
60	52	88	NIL	2-4 pus cells	negative	NG	NG	NIL	NEG	NIL	PRESENT	trace
26	27	96	NIL	NIL	negative	NG	NG	NIL	NEG	NIL	NIL	NIL
32	24	91.6	NIL	1-2 pus cells	negative	NG	NG	NIL	NEG	NIL	PRESENT	trace
41	42	124	NIL	2-3 pus cells	positive	KLEBSIELLA SP	NG	NIL	NEG	NIL	PRESENT	NIL
51	35	138.6	NIL	1-2 pus cells	negative	NG	NG	NIL	NEG	NIL	NIL	NIL
24	23	207.8	trace	3-5 pus cells	negative	NG	NG	NIL	NEG	NIL	NIL	NIL
34	28	126	NIL	1-2 pus cells	positive	NG	NG	NIL	NEG	NIL	PRESENT	++++
20	15	76.1	NIL	2-3 pus cells	negative	NG	NG	NIL	NEG	NIL	PRESENT	NIL
44	32	72.2	NIL	2-3pus cells	positive	NG	NG	giardia lamblia cyst	NEG	NIL	NIL	NIL
34	16	137.4	NIL	2-4pus cells	positive	NG	NG	Entamoeba histolytica cyst	NEG	NIL	NIL	NIL
54	26	89	++++	NIL	negative	NG	NG	Cryptosporidium parvum cyst	NEG	NIL	PRESENT	NIL
33	15	94	NIL	NIL	positive	E.coli	NG	NIL	NEG	NIL	NIL	NIL
54	31	127	+	4-6puscells	positive	NG	NG	NIL	NEG	NIL	PRESENT	NIL
32	14	104	NIL	NIL	positive	NG	NG	Cryptosporidium parvum cyst	NEG	NIL	NIL	NIL
52	39	50.6	NIL	1-3puscells	negative	NG	NG	NIL	positive	NIL	PRESENT	NIL
31	30	52.5	NIL	1-2puscells	positive	NG	NG	NIL	NEG	NIL	NIL	NIL
34	25	108	NIL	NIL	negative	NG	NG	NIL	NEG	NIL	PRESENT	NIL
56	25	94	NIL	NIL	negative	NG	NG	NIL	NEG	NIL	NIL	NIL
36	24	92.2	NIL	NIL	negative	NG	NG	NIL	NEG	NIL	PRESENT	NIL
42	21	79	+	4-6 pus cells	negative	NG	NG	Entamoeba hystolytica cyst	NEG	NIL	PRESENT	NIL
32	24	112.3	NIL	NIL	positive	NG	NG	NIL	NEG	NIL	PRESENT	NIL
89	49	84.9	NIL	2-3pus cells	positive	NG	NG	NIL	NEG	NIL	NIL	NIL
71	35	104.6	NIL	NIL	negative	NG	NG	NIL	NEG	NIL	NIL	NIL
34	18	147.2	NIL	NIL	negative	NG	NG	NIL	NEG	NIL	PRESENT	NIL
46	22	92.9	NIL	2-4pus cells	negative	NG	NG	NIL	NEG	NIL	PRESENT	NIL
45	50	93.6	NIL	1-2puscells	positive	NG	NG	NIL	NEG	NIL	NIL	NIL
32	24	88.4	NIL	4-6puscells	positive	E.coli	NG	NIL	NEG	NIL	PRESENT	NIL
20	15	124.3	NIL	NIL	negative	NG	NG	NIL	positive	NIL	NIL	NIL
39	16	92.4	NIL	NIL	negative	NG	NG	NIL	NEG	NIL	NIL	NIL
42	26	78.4	NIL	NIL	negative	NG	NG	giardia lamblia cyst	NEG	NIL	NIL	NIL

USG	CXR	MANTOUX	HIV	Ig Profile	BARIUM MEAL	UGI SCOPY
Spleenomegaly	normal	negative	NR		-	erythematous gastric mucosa ,whitish duodenal mucosa
Normal	normal	negative	NR		-	-
Normal	normal	negative	NR		-	duodenal mucosa-normal
Normal	normal	negative	NR		-	Lax LES,Inflammed,gastric &duodenal mucosa
Normal	normal	negative	NR			
Normal	normal	negative	NR		-	Lax LES
Normal	normal	negative	NR		-	pale duodenal mucosa
Normal	normal	negative	NR		-	
Normal	normal	negative	NR	normal		
Normal	Rt UL Pneumonia	negative	NR	normal		pale duodenal mucosa
Gr 3 RPD	normal	negative	NR		-	
Normal	normal	negative	NR	normal	-	fundal/gastritis
Normal	normal	negative	NR			
Normal	bronchopneumonia	negative	NR	normal	-	Lax LES
Normal	normal	negative	NR			
Normal	normal	negative	NR	normal	normal	
Hepatomegaly	normal	negative	NR	normal	-	pale duodenal mucosa
Normal	normal	negative	NR			
Hepatomegaly,ascities	normal	negative	NR	normal		
Normal	normal	negative	NR		-	pale duodenal mucosa
Normal	normal	-	NR	normal		pale duodenal mucosa
Normal	normal	negative	NR	normal	malabsorbtion pattern	pale duodenal mucosa
Normal	normal	negative	NR			
Gr 2 RPD	normal	negative	NR			
Normal	normal	negative	NR		normal	lax LES,antral gastritis
Normal	normal	negative	NR	Decreased		oesophageal candidiasis
Normal	normal	negative	NR			
Normal	normal	negative	NR			lax LES
Normal	normal	negative	NR			
features of malabsorbtion of small bowel	normal	negative	NR	normal	malabsorbtion pattern	pale duodenal mucosa
Hepatomegaly	normal	negative	NR	IgE Increased		Lax LES,Fundal gastritis
Normal	normal	negative	NR	normal		
hepatomegaly	residual cyst Rt upper lobe	negative	NR		normal	pale duodenal mucosa
Normal	normal	negative	NR			
Normal	normal	negative	NR			
Normal	normal	negative	NR			
Normal	normal	negative	NR	normal	-	Lax LES
Normal	normal	negative	NR	-	-	-
Normal	normal	negative	NR	normal	-	-
Normal	normal	negative	NR			
Normal	normal	negative	NR			fundal gastritis ,duodenal ulcer
Normal	normal	negative	NR			
Normal	normal	negative	NR	normal		

duodenal biopsy	colonoscopy	diagnosis	outcome
subacute inflammation	-	non specific	improved
-	-	sepsis	improved
normal	-	non specific	improved
		non specific	improved
		CMPA	improved
Chronic duodenitis with eosinophilia		CMPA	improved
villous deformity with increased lymphocytes		CMPA	improved
		secondary lactose intilerance	improved
		sepsis	improved
Chronic duodenitis		congenital zinc deficiency	improved
		cryptosporidium parvum	improved
chronic inflammation		non specific	improved
		non specific	improved
		non specific	improved
		non specific	improved
		non specific	improved
moderate inflammation		sepsis-klebsiella sp	improved
		non specific	improved
		non specific	improved
moderate inflammation with↑ intraepithelial lymphocytes		intestinal lymphangiectasia	improved
villous blunting with chronic inflammation	-	congenital tufting enteropathy	improved
no specific pathology	no specific pathology	Giardiasis	improved
		Entamoeba histolytica	improved
		cryptosporidium parvum	improved
suacute ileitis with transmural inflammation,colitis,proctitis	erythema in distal colon	Inflammatory bowel disease-crohn's	improved
villous blunting with features of giardiasis		severe combined immunodeficiency	died
		cyptosporidium parvum	improved
chronic duodenitis		non specific	improved
		CMPA	improved
moderate to focal subtotal villous atrophy		malabsorbtion	improved
Chronic duodenitis with eosinophilia	chronic nonspecific colitis	CMPA	improved
		CMPA	improved
Chronic duodenitis with eosinophilia		Entamoeba histolytica	improved
		non specific	improved
		non specific	improved
		CMPA	improved
chronic inflammation		non specific	improved
-	-	non specific	improved
-	-	sepsis	improved
		sepsis-E.Coli	improved
	severe pancolitis	Inflammatory bowel disease Ulcerative colitis	improved
		CMPA	improved
		CMPA	improved