Original Article

Acute Gastroenteritis: Its Causes, Maintenance, And Treatment

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Abstract

Diarrhoea is a huge public health issue, even in the wealthy world. For patients with severe diarrhoea, it might be difficult to determine which pathogens should be investigated before beginning antimicrobial therapy. Gastroenteritis is an infectious ailment that produces nausea, vomiting, diarrhoea, and abdominal pain. In the United States, the CDC estimates that acute gastroenteritis affects 350 million people each year, with food-borne germs accounting for 48 million of those cases. Both community-acquired diarrhoea and traveller's diarrhoea are addressed in terms of epidemiology when discussing acute gastroenteritis therapy and management. Traveller's diarrhoea affects more than half of those who travel from rich countries to developing countries. In the United States, Clostridium difficile is becoming more common in individuals of all ages. Acute hydration or sufficient fluid intake is the basic treatment for acute gastroenteritis. Since acute gastroenteritis usually resolves on its own, antibiotics are rarely needed in most cases. Antibiotic therapy may be necessary for some situations, such as those with fever and bloody diarrhoea or those with an immunocompromised state, such as those with febrile diarrhoea. Assessing and treating infants and children with acute gastroenteritis should be based on scientific data, and that is what this study aims to do. Many people die as
a result of dehydration, electrolyte imbalance, and metabolic acidosis. Dehydration and its repercussions can be minimized with proper hydration management, whether by oral or intravenous fluids. Because of the risk of side effects, antibiotics, antidiarrheal medications, and antiemetics should not be taken regularly. Rotavirus vaccines newly permitted by the Food and Drug Administration will have a substantial influence on public health since they are very effective. Inflammatory bowel disease may be exacerbated by bacterial intestinal infections (IBD). We examined the link between infectious gastroenteritis and the incidence of IBD using data from the General Practice Research Database. According to this, an infectious gastroenteritis episode may contribute to the start and/or aggravation of IBD. In this study, we attempted to outline the cause, clinical indications, epidemiology, management, and therapy of gastroenteritis. We also described the management of community-acquired and traveller's diarrhoea, as well as the importance of antibiotics and antiemetics in diarrhoea therapy. The link between gastroenteritis and IBD is also addressed in this review using a case study.

Keywords: acute gastroenteritis, community-acquired, traveller's diarrhoea, fluid replacement, IBD, antibiotic

1. INTRODUCTION

Every year, a significant number of infants and toddlers under the age of five suffer from an acute form of gastroenteritis. According to estimates provided by the CDC (Centres for Disease Control and Prevention), acute diarrhoea affects more than 1.52 million children in the United States of America each year, leading to more than 200,080 hospital admissions and around 310 fatalities [1]. It is estimated that diarrhoea claims the lives of 2.3 million children under the age of five in impoverished nations every single year. Each year, gastroenteritis accounts for between 2.4 and 3.3 million office visits and 10.3 percent of all pediatric hospital admissions in the United States. The rotavirus is responsible for one-third of all hospitalizations that are caused by diarrhoea in children under the age of five, which results in an annual cost of $350 million [2]. The condition known as gastroenteritis is shown in figure 1, which may be found here.

Even in more developed nations, diarrhoea brought on by a severe infectious agent is a major reason for concern. In Germany, adults have an annual incidence rate of 0.75 occurrences of acute gastroenteritis on average. A comparable rate was discovered in the United States [2]. When treating patients who have severe diarrhoea, two of the most difficult hurdles that clinicians must overcome are deciding whether or not to do a stool culture and whether or not to provide antibiotic treatment [3].

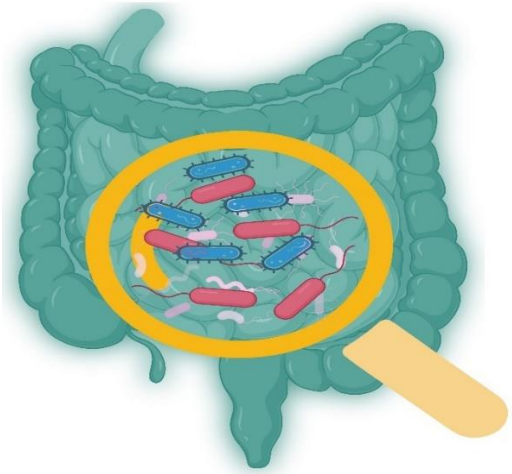

Fig 1. Representation of Acute Gastroenteritis

Diarrhoea is the condition of more frequent passage of stools or passage of three or more loose or watery stools per day. Because if children's bowel movements differ, diarrhoea should be considered abnormal. It is not diarrhoea if breastfed newborn babies pass formed faeces or "pasty" stools regularly [4].

Unformed stools of more than 250 g or more than three unformed stools per day are commonly used to indicate diarrhoea, as well as nausea, vomiting, or stomach cramps. Chronic diarrhoea (lasting 30 days or more) is distinguished from acute (lasting 14 days or less), persistent (lasting 14–29 days or more), or chronic (lasting 14–29 or more days) diarrhoea [5].

Three types of diarrhoeal illnesses may be categorized clinically:
1 Persistent distressing and debilitating diarrhoea for 4 or more days
2 Dysentry or Acute bloody diarrhoea
3 Diarrhoea persisting for at least 14 days

1.1. The Art of Clinical Presentation

Fever, nausea, vomiting, diarrhoea, and abdominal cramps are just a few of the symptoms of acute gastroenteritis. The initial symptom in youngsters may be vomiting, followed by diarrhoea, or the other way around. When faecal impaction is the only symptom, the doctor must rule out other possibilities such as metabolic problems, ingestion, diabetes, urinary tract infections, ingestion, meningitis, and gastrointestinal blockage. The frequency of emesis, strength, and color, as well as its link to feedings, are usually utilized to make a diagnosis [6].

There is always a need for a complete medical history and inspection of the body. Any associated symptoms such as fever, stomach discomfort, or urinary problems, as well as recent fluid and food consumption, should be inquired about. It's important to look at the child's recent medication and vaccine history. Look for indications of dehydration throughout the exam, such as sleepiness, sunken eyes, dry mucous membranes, and a lack of flexibility in the skin. [7].

In children all around the globe, acute gastroenteritis is caused by a viral infection most of the time. Viruses are also the most likely cause of the condition. A fever that isn't too high and diarrhoea that doesn't include blood are two of the most common signs of a viral disease. Bacterial infections can cause inflammation in the small and large intestines by penetrating the mucosal lining. The consequences of this include an elevated core temperature in addition to the detection of blood and white blood cells in the feces of young people [8].

2. EPIDEMIOLOGY

2.1. Community-Acquired diarrhoea

The most common causes of bacterial diarrhoea in Europe are Enteroaggregative E.coli, Enteropathogenic E. coli, and Campylobacter. Non-typhoidal Salmonella infections were more common in 2012 (68 per 100,000 vs. 22 per 100,000). The adoption of veterinary control programs against Salmonella species in chickens may have lowered the number of salmonella infections in the previous five years. Another important contributor to community-acquired diarrhoea in wealthy countries has been identified as the Enteroaggregative E.coli (EAEC) [9]. There has also been a rise in Clostridium difficile-related diarrhoea among people who don't fit the conventional risk profile. Community-associated infections may be linked to foodborne C. difficile transmission, although there is insufficient evidence to support or reject this hypothesis [10,11]. Vibrio species that do not cause cholera, Yersinia species, and E. coli strains that produce Shiga toxin are also rare pathogens. More than 4,000 people became infected with Shiga-Toxigenic E.coli (STEC) in 2011, and over 900 people developed hemolytic–uraemic syndrome (HUS) as a result of the outbreak [12]. Toxic sprouts were shown to be a major factor in the spread of the disease. In 2012, there were 1.5 STEC infections per 100,000 people in Europe [13].

2.2. Diarrhoea brought on by travel (Traveller's diarrhoea)

Travellers' diarrhoea affects anywhere from 20% to 50% of visitors from resource-constrained nations, depending on the locale [14]. Infected foods and drinks are the most common methods of transmission during the first 2–3 weeks of travel. People who followed the advice to 'boil it, cook it, peel it or forget it were just as likely to suffer from diarrhoea as those who experimented with their eating habits. Only 1% of tourists require hospitalization due to self-limiting illnesses [18]. 80% of cases of traveler's diarrhoea are caused by bacterial enteropathogens [15]. Enteroinvasive E. coli (EAEC), and Enterotoxigenic E. coli make up the bulk of cases, followed by Shigella, Salmonella, and Campylobacter. Acute travelers' diarrhoea is seldom caused by parasitic agents, but in situations of subacute or chronic illness, they should be explored [16].

3. MANAGEMENT

In other people, the illness is not as severe, and they can treat themselves at home without the need for medical attention. Patients who have severe diarrheal disease, such as diarrhea that is bloody, feverish, dehydrating, or profuse, need an in-depth history from the attending physician that includes both epidemiological and clinical information. All-important clinical findings include diarrhoea (fever, tenesmus, presence of mucus or blood in the stool), evidence of reduced urine output, and symptoms associated with it, such as abdominal discomfort, vomiting, or nausea [17]. Consuming hazardous food and using antibiotics are two of the most relevant epidemiological risks, along with working in a childcare center or having prior overseas travel experience.

The presence of diarrhoea in elderly patients, fever, recent antibiotic use, symptoms lasting more than one week, and signs of dryness (decreased urine output, dry mucous membranes, tachycardia, and symptoms of lethargy or hypotension of posture) prompt testing for Shigella STEC test, Salmonella, Campylobacter species, and Salmonella which should be included in the evaluation. Patients having any history should be recommended for testing for the presence of Clostridium difficile. It is important to highlight that C. difficile infections can arise in the absence of known risk factors [18].

Bacterial pathogens are usually found by testing the first or second sample, so obtaining several cultures is pointless. Stool cultures may yield a small number of bacteria (1.6–3.2 percent), but the information gathered is useful for both individual patients and the general public [19].

Enteropathogens can be identified in a single step using PCR-based diagnostic techniques. Automated multiplex systems possess multiple advantages over conventional culture which include greater sensitivity and shorter turnaround times. Pathogen nucleic acid detection does not imply that the pathogen is the source of clinical symptoms. In other cases, the patient may be an asymptomatic carrier of the virus, or the virus's nucleic acid may still be detectable even after the infection has died [20]. To detect resistance patterns in the future, PCR-based technologies and culture may be used in circumstances where good results are obtained. Remember that the patient's medical history and symptoms, not only lab findings, dictate when antibiotics should be administered to an individual patient.

People who have a fever or symptoms that point to systemic inflammatory response syndrome may need further diagnostic investigations [21]. These procedures include serum chemistry analysis, a complete blood count, and blood cultures.

3.1. Management of Body Fluid

In Children, those who are having clinical indications of water loss may benefit from biochemical testing such as blood electrolytes, urea, and creatine, as well as an evaluation of acid-base balance. Regardless of blood electrolyte values, dehydration from gastroenteritis is frequently associated with whole-body ions deficiency specifically chloride and sodium. In addition, there is often a significant loss of potassium ion concentration. Hyponatremia and hypernatremia are just measures of sodium and water losses relative to sodium and water gains. The rehydration solution should replenish both water and electrolytes lost during exercise. In many situations, the rehydration phase is followed by a fluid-maintenance phase to prevent dehydration from arising again [22].

3.2. Oral Rehydration As A Form Of Therapeutic Management

However, except for the most seriously ill patients, ORT can be utilized to aid with hydration. Clinical studies have proved the efficiency of oral rehydration therapy around 30 years ago in large clinical studies carried out in Bangladesh during cholera epidemics. Thirteenth and fourteenth researchers were able to conduct these studies after discovering that salt and glucose were co-transported into the digestive tract in an equimolar ratio in the 1960s. According to studies, salt absorption in laboratory mice was boosted by glucose [23]. As confirmed by human research, the cotransporter of Na-glucose was shown to function among patients suffering from cholera. ORT's effectiveness in the treatment of non-cholera diarrhoea was subsequently shown by controlled studies. When ORT was used to treat gastroenteritis in the United Kingdom, fatality rates dropped from 300 per year in the late 1970s to roughly 25 per year in the late 1980s. In acute gastroenteritis, dehydration due to hypernatremia is the main cause of mortality [24].

3.3. Rehydration

Several regimes in the previous years aimed to gradually rehydrate patients over 24 hours or more. However, this approach was not supported by any research. Continuing the rehydration treatment in these infants appears foolish and possibly

dangerous. Fast rehydration over three or four hours is now recommended by the majority of medical specialists. The levels of dehydration are possible to calculate the amount of fluid deficit in the body such as 50 ml/kg of replacement fluid for a 5% dehydration. To repair the shortfall within four hours, ORS can be delivered in a bottle, cup, or spoon. Fluids may be easily ingested by most children suffering from dehydration, but some sick children may require intravenous fluids [25]. Monitoring of the patient's hydration status under the supervision of a medical professional should take place before, during, and after the four-hour rehydration phase. If it is determined that the patient is not yet adequately hydrated, the remaining deficit is computed, and the operation to rehydrate them is carried out once again. Children who experience nausea or vomiting during the procedure are given an oral rehydration salt solution (ORS) right away [26]. Intravenous rehydration is the first line of treatment for children who exhibit signs of inadequate perfusion of vital organs (shock). Even though oral rehydration is a viable option in these situations, intravenous rehydration aids in the immediate increase in the volume of body fluid in these critically ill people [27].

Seizures can be reduced during rehydration by employing ORT, and this is generally agreed upon. In one study, none of the 34 neonates with hypernatraemic dehydration experienced seizures after 12 hours of rehydration using WHO-ORS. In the largest published controlled trial comparing the two methods, oral rehydration solution (ORS) or intravenous fluid (IVF) was the treatment of choice for 470 children under the age of 18 months. Intravenous therapy (IVT) was shown to be more effective in treating hypernatraemia than oral rehydration treatment (ORT). It is encouraging to see these results, even though the use of WHO-ORS (sodium 91 mmol/l) rather than the ORS presently recommended in Europe (sodium 60 mmol/l) may have had a significant role. Rehydration should be monitored frequently since rapid drops in blood sodium concentrations have been linked with an increased risk of cerebral edema and seizures [28].

4. TREATMENT

Early rehydration by intravenous fluids or employing oral electrolyte solutions is essential in the treatment of acute diarrhoeal illnesses. Antibiotics are not necessary for most people since their illness is self-limiting. Unanticipated and possibly harmful side effects might arise from every antimicrobial treatment choice. However, the use of both empirical and specialist antimicrobial treatments may nevertheless be considered in some circumstances. To reduce the frequency of treatment failures, the pattern identification of local resistance is quite important [29].

There is a correlation between the use of fluoroquinolones and the production and release of STEC toxins in experimental animals. In contrast to the findings of previous research, which suggested that using antibiotics did raise the risk of HUS, a new study found that using antibiotics did not increase the risk. Multiple investigations have been connected to an elevated risk of HUS in multiple investigations, including a large prospective analysis with 259 children [30]. In this research, antibiotic use has been linked to HUS. Even in the absence of fever, it is reasonable to believe that a person has an STEC infection when they have bloody diarrhea, stomach discomfort, or soreness. In the event of a bloody diarrhea pandemic, antibiotic treatment is not recommended for those with low or no fever who may have STEC infection [31, 32]. This is because STEC infections do not generate fever. Depending on the epidemiological conditions, acute febrile bloody diarrhoea may be caused by Campylobacter species or Shigella species. These individuals may benefit from an empirical course of antibiotic treatment [33].

4.1. Pharmacologic Treatment

4.1.1. Antimicrobial Substances

The use of antibiotics is not at all recommended and if used can leads to serious complications in simple or viral acute gastroenteritis. Prolonged carrier stage and recurrence in non-typhoid Salmonella infections are some of the effects of antibiotics usage. When antibiotics are used to treat gastroenteritis brought on by E. coli that produces the Shiga toxin, there is a possibility that the risk of hemolytic uremic syndrome will also rise. Antibiotics are only used to treat cholera, amebiasis, and giardia. They are also used to treat enteric fever and cholera, as well as acute enteritis caused by septicemia [34].

4.1.2. Antidiarrhoeal Drugs

The regular use of antidiarrhoeal drugs is not recommended at all. Nausea in babies, drowsiness and Opiate-induced ileus in babies under the age of three have been related to antimotility medications like loperamide. However, bismuth subsalicylate is ineffective in the treatment of acute gastroenteritis in young children. Children in the inpatient setting showed positive outcomes when given the enkephalinase inhibitor racecadotril, which lowers water and electrolyte output without impairing intestinal motility. However, US has still not approved racecadotril. Scientists are doing their best to determine the efficacy and safety of the drug [35].

The following are the main points:

1 Antiemetics: The need to avoid more dehydration and the need for IV therapy and subsequent hospitalization is the driving force behind the desire to stop vomiting. Patients treated in the ER with a single dose of ondansetron, a serotonergic 5HT3 receptor antagonist, were shown to have a decreased risk of hospitalization while experiencing few adverse effects [36].
2 Promethazine, a phenothiazine derivate having antihistamine and anticholinergic activity, has been proven to be less effective in suppressing emesis than newer antiemetics. There are adverse effects, such as sleepiness and

extrapyramidal effects, associated with the use of promethazine, which is only allowed by the FDA for children over the age of two. Despite being shown to be more effective than a placebo, metoclopramide has a 25% chance of causing extrapyramidal events in children when used. There are no underlying reasons for diarrhoea addressed by these drugs, and their use may divert the focus of the general practitioner away from the basic therapy of correct fluid and electrolyte replacement and early nutrition therapy [37-38].

4.1.3. A Zinc Supplement of the Tenth Order

Oxidative damage can be prevented by zinc, a vitamin. A significant amount of zinc is lost during acute or chronic diarrhoea as a result of the increased intestinal output. ORS and zinc treatment have been studied in certain impoverished nations with a high frequency of zinc insufficiency, and the results show a potential benefit. Zinc may enhance water and electrolyte absorption, even if the exact mechanism of action has not yet been established. In trials that compared zinc supplements to a placebo, it was shown that both the frequency of stools and the amount of time spent diarrheal decreased significantly. According to recommendations made by the World Health Organization and the United Nations Children's Fund for the treatment of diarrheal diseases in children, zinc should be included in ORSs. [39].

4.1.4. Foods That Serve A Specific Purpose

In fermented foods, there are live bacteria known as probiotics, which may aid the host by promoting a healthy balance of the microbiome in the digestive tract. The most commonly studied probiotic bacteria are Streptococcus thermophilus, Bifidobacterium lactis and Lactobacillus rhamnosus G. A single day can be shaved off the length of acute infectious diarrhoea by using L. rhamnosus GG in randomized controlled experiments. To better treat gastroenteritis caused by rotavirus, lactobacillus was found to be more effective, with a 2-day reduction in diarrhoea duration. In healthy patients with viral gastroenteritis, probiotics are proved to be more effective during the early days [40].

When it comes to prebiotics, the focus is on supporting the growth of beneficial bacteria in your gut rather than harmful ones. It hasn't proven to be effective in randomized controlled research, so prebiotics aren't recommended as often [41]. In experimental models, fluoroquinolones have been linked to the creation and release of STEC toxins. People with bloody diarrhoea, stomach pain, or soreness should be suspected of STEC infection in the absence of fever. Because of this, antibiotics are not recommended for those with low or no fever who may have STEC infection in the case of a bloody diarrhoea pandemic [42].

4.1.5. Antiemetics

The enterochromaffin cells in the gastrointestinal mucosa may be damaged by acute gastroenteritis, which can cause the mucosa to leak serotonin. These serotonin signals may be sent to the vomiting center through the chemoreceptor trigger zone or directly to the 5HT3 receptors in the gastrointestinal tract via the vagal afferent neurons. The vomiting reflex is triggered by the abdominal muscles, the visceral muscles of the stomach, and the esophageal muscles; the diaphragm is the organ that receives the efferent impulses from the vomiting center [43]. These symptoms include regurgitation of intestinal contents into the stomach, nausea, and the descent of the diaphragm against a closed glottis, which forces the stomach contents up into the esophagus and out through the mouth. Other symptoms include an increase in non-peristaltic contractions in the small intestine, salivation, contractions of the respiratory and abdominal muscles, a decrease in gastric tone, and an increase in non-peristaltic contractions in the large intestine [44]. Vomiting centers, peripheral receptor impulses, chemoreceptor centers, and chemoreceptor impulses are all targets of anti-emetic treatment. It aims to suppress all of these. Nearly every area thought to be involved in the genesis of vomiting contains high concentrations of the dopaminergic, histaminergic, and muscarinic receptors that are involved in nausea and vomiting. Antagonists of Dopamine blocks the D2 receptors in the chemoreceptor trigger zone, which prevents proemetic impulses from being generated. An antidote to 5-HT3 receptor activation in the small intestine and the chemoreceptor center has recently been developed to prevent nausea and vomiting. The vestibular system is targeted by antihistamines, making them useful for treating motion sickness as well as migraines [45]. The use of anticholinergic drugs like atropine and hyoscine to cure or prevent vomiting that isn't the result of motion sickness is a complete failure. These medications, like dexamethasone and trimethobenzamide, have a mysterious mode of action that is difficult to understand. Despite the lack of formal guidance, antiemetic drugs are often prescribed to children with gastroenteritis by doctors from a variety of specialties and countries [46].

4.1.5.1. Ondansetron

Since 1991, ondansetron, a carbazole derivative, has been on the market. It does not affect the dopaminergic system at all. Later, its success in the treatment of chemotherapy-induced or postoperative vomiting in children has been recognized. The use of ondansetron has shown promising results in Procedural sedation using Ketamine, Patients with migraine-induced vomiting, and acetaminophen toxicity [47]. Further studies exploring its use in vomiting caused by gastroenteritis were encouraged by these favourable results. The major outcomes of the prospective controlled trials conducted by researchers in a 2008 meta-analysis were side effects of the medication, emesis, hospitalization and return to care, intravenous fluid rehydration, cessation, and hospitalization and return to care [48].

The majority of the study requirements were satisfied by ondansetron (n = 6, participants = 745). Outpatient studies were the only ones that were not carried out in an emergency department environment [49]. Even though the great majority of research has only focused on children and young adults, this particular study included patients who were as young as 22

years old. Dehydration was a prerequisite for participation in the trials, which meant that their volunteers could not be well hydrated. In research, the scientist looked at both the effects of dehydration and the failure of oral rehydration. On the other hand, according to a different study conducted, each patient got treatment by intravenous route [50]. During the investigation, just one dosage of ondansetron was given to each one of the individuals. In three different investigations, ondansetron was given through intravenous administration. Different doses of ondansetron i.e. 0.16,0.18,0.4, 3-9 mg/ kg were used in different experiments performed by scientists. There was no evidence that it had any extrapyramidal or sedative effects. Headaches were reported to be the most prevalent side effect in other large clinical research, especially those that included pediatric patients. This was followed by weariness and then constipation, in that order. Ondansetron has a very good safety profile in terms of its tolerance. When it is completely and rapidly absorbed from the digestive tract, it is metabolized by the cytochrome P450 enzyme system in the liver, which is then followed by glucuronide or sulfate conjugation. In addition, there is a low possibility that this supplement may interact negatively with the medication that you are now taking [52].

It takes around 40 minutes for intramuscular injections to reach their peak plasma concentration while intravenous injections take about 10 minutes. Its half-life is between two and six hours. It has an antiemetic effect that lasts from 2 to 8 hours with its usual dosage. Intravenous ondansetron is injected at a dosage of 0.1 to 0.15mg/kg with a maximum of 4 mg. Among the recommended dosages for children weighing 8 to 15 kg, 2mg, 4 mg, and 8 mg are recommended for children weighing 15 to 30 kg, respectively, and up to 30 kg, three times a day. On the other hand, a single oral ondansetron dose is usually sufficient for the treatment of gastroenteritis-related vomiting. Although ondansetron has been too expensive, a generic version of the drug has just been made available, removing this barrier [53].

4.1.5.2. Promethazine

Phenothiazines, the building blocks from which Promethazine is derived, are potent antihistamines. Anticholinergic and antidopaminergic effects are also present in this substance. It has also been used to alleviate nausea and vomiting following surgery as well as motion sickness. 57,88 As a result, the drug is quite affordable and can be taken orally, via the rectum, IM, or IV in doses of 260 µg to 1ooo µg/kg body weight (up to a maximum of 25000 µg) every four to six hours as needed [54]. Clinical effects occur within 20 minutes of therapy when taken orally. Based on research data, Tibbs reported the treatment of gastroenteritis vomiting in children with promethazine and pyrilamine-pentobarbital. There was no placebo group in this experiment, and children with illnesses other than gastroenteritis were included [55-56].

According to the findings of this research, promethazine is not as efficient as pyrilamine and pentobarbital in reducing feelings of sickness and vomiting. Since its clearance in 1951, research has shown that promethazine may cause significant side effects in children, some of which can be life-threatening. These adverse responses include respiratory depression, agitation, hallucinations, seizures, and dystonic reactions [57]. According to the agency (FDA), there were 38 incidents of respiratory depression, apnea, or cardiac arrest that were reported to the FDA in 2005. Seven of the children that died were between the ages of one and two years old, and their ages ranged from one and a half months to two years. More than a half dozen of the 22 patients were given promethazine and a respiratory depressant drug at a dosage of less than 1 mg per kilogram of body weight. This was done in an attempt to treat their symptoms of an allergic reaction. There was a correlation between a broad range of dosages depending on weight and respiratory depression (0.45 to 6.4 mg per kg). Any administration of the medicine results in adverse effects such as death, irreversible disability, life-threatening episodes, and hospitalization. These adverse effects happened regardless of the method of administration (oral, rectal, or parenteral). Late in 2004, a "boxed warning" was included as a new component of the labelling for promethazine hydrochloride (Phenergan) [58]. This warning featured a contraindication for use in children less than two as well as an enhanced warning for use in children older than two. Both of these warnings relate to the same topic. It is not recommended to administer this medicine to children who are currently on other medications that have respiratory depressive effects since it has the potential to make their symptoms much worse [59].

4.1.5.3. Dimenhydrinate

It is the first generation of H1 receptor antagonists. Not only does it block H1 receptors in the nucleus tractus solitarius, but it also inhibits muscarinic-cholinergic receptors in both the vestibular apparatus and the vomiting center. Because it may be taken orally, rectally, intramuscularly, or even intravenously, using dimenhydrinate is a procedure that does not need a lot of training [60].

The recommended dosage is 1.25 milligrams per kilogram of body weight, with a maximum dose of 50 milligrams. Additionally, it has been used to treat and prevent motion sickness, radiation sickness, labyrinthine function disorders, as well as nausea and vomiting after surgical procedures. Ondansetron dimenhydrinate is available at a substantially more affordable price than its counterpart, dimenhydrinate. The sedative effect of dimenhydrinate is the most significant drawback associated with taking this medication to treat vomiting caused by acute gastroenteritis. As a result of its ability to amplify the effects of dehydration and respiratory depression, it may reduce the efficacy of oral rehydration fluid intake and put oral rehydration fluid intake at risk [60].

4.2. Therapy in

4.2.1. Community-acquired diarrhoea

There was a Swedish study that demonstrated that norfloxacin reduced the severity and duration of diarrhoeal symptoms, although it had a limited effect on those who were ill, to begin with. Salmonella clearance was delayed and Campylobacter

resistance was increased by norfloxacin treatment [61]. According to a meta-analysis, antibiotics did not shorten the duration of illness in otherwise healthy people with non-severe diarrhoea caused by non-typhoidal Salmonella. The duration of time that Salmonella remained in feces was also extended by antimicrobial therapy. As a result, in otherwise healthy people with nontyphoidal salmonellosis, antibiotic therapy is not necessary [62]. The management of community-acquired diarrhoea is represented in figure 2.

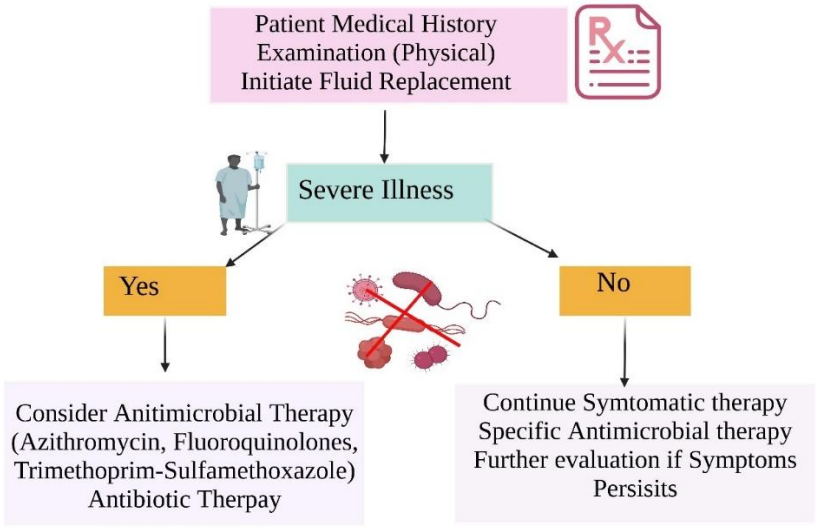

Fig 2. Management of Community-Acquired Diarrhoea

Compared to non-typhoidal Salmonella infections, Campylobacter infections in Europe were three times as prevalent in 2012. Campylobacter resistance to ciprofloxacin was shown to be as high as 44% in some EU countries, but resistance to erythromycin was just 5%. Resistant to ciprofloxacin, only 5–13% of non-typhoidal Salmonella isolates were found in Europe in 2011 [63]. If you've recently been to a foreign country, you're at risk of contracting Campylobacter, according to new research [64].

Fluoroquinolone resistance was found in 60% of Campylobacter isolates from travel-related infections, compared to 13% in cases unrelated to travel.

Although it may seem counterintuitive that fluoroquinolones are the first-line treatment therapy for community-acquired diarrhoea in European countries, studies have shown that this class of antibiotics may be effective in treating illnesses that are not associated with travel in the United States. It seems that an azithromycin is a suitable option in scenarios involving travel to other countries as well as community-acquired illnesses in both the United States and Europe [65].
If a patient has six or more stools per day, fever and bloody diarrhoea, or only a fever, symptoms that linger for more than a week or are immunocompromised, it is suggested that they get empirical antibiotic therapy. Azithromycin, fluoroquinolones, and trimethoprim-sulfamethoxazole are some of the medications available in Europe for treating bacterial infections. Pregnant women should avoid fluoroquinolones and trimethoprim-sulfamethoxazole. In some cases of severe diarrhoea due to an infectious etiology, organism-specific antibiotic therapy may be necessary [66].

4.2.2. Traveller's diarrhoea

It is essential to bear in mind that only 1 percent of people diagnosed with traveler's diarrhoea need hospitalization as a treatment option. Patients who are experiencing moderate to severe diarrhoea, on the other hand, are encouraged to take antibiotics (more than four stools per day, fever, and blood or mucus in the stool). Throughout medical history, the drugs ampicillin, trimethoprim-sulfamethoxazole, and doxycycline have been the ones that have been prescribed the most often [67]. Figure 3 displays the treatment plan for diarrhoea that might occur when traveling.

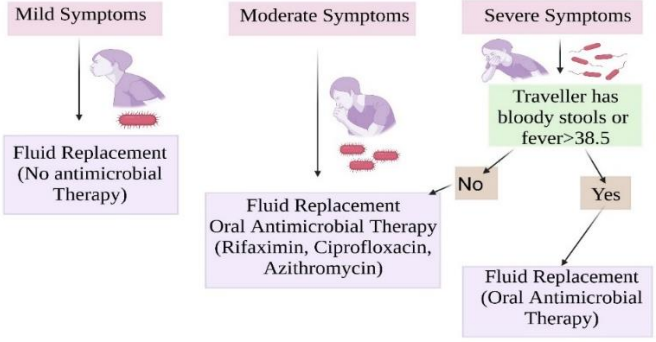

Fig 3. Management of Traveller's Diarrhoea

Because of the high rates and frequency of drug resistance, these medicines are no longer acceptable for empirical treatment [68]. Ciprofloxacin, azithromycin, and rifaximin are now recommended for self-treatment by travellers. Globally, fluoroquinolone resistance is increasing among enteropathogens such as Campylobacter, particularly in Southeast Asia but also in Europe [69].

Researchers have discovered that the non-absorbed rifamycin is effective in the treatment of diarrhoea in travelers due to non-invasive illnesses. As a prophylactic measure, it has worked well for American tourists in Mexico [70].

Staphylococci that are resistant to rifampin have been seen to develop after rifaximin dosing. Rifampin should be used with caution in individuals at risk for staphylococcal foreign-body infections since rifampin resistance is connected to treatment failure. In Thailand, Azithromax was shown to be superior to levofloxacin in the treatment of diarrhoea [71]. According to one study in Turkey, there were no significant differences between azithromycin and loperamide in terms of effectiveness [72]. Even though the single-dose regimen was more often accompanied by mild nausea upon consumption, a 1000 mg oral dosage was more efficacious than a three-day treatment of 500 mg [73].

5. ACUTE GASTROENTERITIS IN CHILDREN

It is estimated that millions of young children die each year from acute gastroenteritis, particularly in developing nations. In developed countries, it is a common reason for visits to primary care or emergency rooms, as well as hospitalization. Many people die because of dehydration, electrolyte imbalance, and metabolic acidosis [74]. The danger of dehydration and its harmful repercussions can be minimized with the use of oral or intravenous fluids. There is a danger in using antibiotics, antidiarrhoeal medications, and antiemetics daily. Rotavirus vaccines newly permitted by the Food and Drug Administration will have a substantial influence on public health because of their ability to prevent gastroenteritis [75].

Diarrhoea is a variation from the ordinary in young children's stooling habits. Acute gastroenteritis affects 3-5 billion people each year, killing nearly 2 million children under the age of five.

Gastroenteritis is the leading cause of paediatric hospitalization, outpatient visits, and deaths in the United States, affecting over 220,000 children under the age of five each year, costing the country over $1 billion (£0.5 billion; €0.8 billion) [76].

In Australia, an estimated $30 million ($12 million; €18 million; $23 million) is spent each year on approximately 10,000 hospital admissions, 2,200 emergency department visits, and 115, 000 general practitioner sessions for the same age group [77].

Among children under the age of five in the United Kingdom, 204 out of every 1000 visits to the general practitioner are for gastroenteritis, and the yearly admission rate for this age group to the hospital is roughly seven out of every 1000 [78].

Daycare children are often unwell, yet they may transmit their sickness inadvertently. Malnourished children are more susceptible to health issues. Hospitalization for diarrhoea, malnutrition, comorbidity, and electrolyte disturbance (especially hypokalemia) was more common among Aboriginal and Torres Strait Islander children in northern Australia than among their non-indigenous classmates. Gastroenteritis may have a huge impact on society, but it might be underestimated if family expenses, such as time off from work, are not taken into account [79].

5.1. Cause

Rotaviruses and noroviruses are the most common causes of vomiting and diarrhoea. Small intestinal enterocytes are damaged by viral infections, resulting in a mild temperature and watery diarrhoea that does not contain any blood. While rotavirus infections peak in late winter in temperate regions, they occur all year round in the tropics [80]. Rotavirus strains vary depending on the time of year and where in the country they are found. Infants between the ages of six months and two years are most commonly affected, with transmission occurring by fecal-oral or respiratory means [81]. The common agents causing gastroenteritis are shown in figure 4.

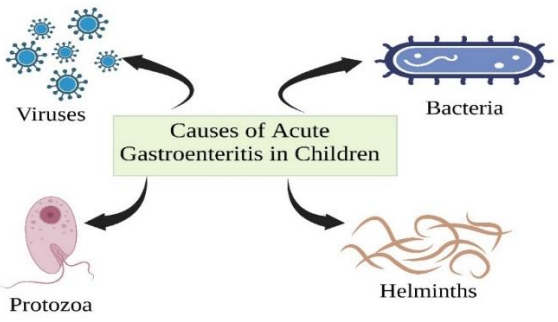

Fig 4 . Causes of Acute Gastroenteritis in Children

Several bacteria infect the intestinal linings, including Campylobacter jejuni and Salmonella species, which cause inflammation. A fever and blood or white blood cells in the stools are more common in children with bacterial gastroenteritis [82]. Infections caused by bacteria have the potential to spread throughout the body, and this is especially true in newborns. Hemorrhagic colitis (severe bloody diarrhoea) can be caused by Shiga toxin-producing Escherichia coli or Shigella dysenteriae infection, and this can be exacerbated by the hemolytic uraemic syndrome. Acute onset of microangiopathic hemolytic anemia, thrombocytopenia, severe renal impairment, and multisystem involvement characterize this disease, which affects people all over the world [83].

In young children, enteric fevers (produced by Salmonella typhi and S paratyphi) can cause severe disease, including fever, diarrhoea or constipation, leukocytosis, and possibly central nervous system involvement, including encephalopathy, an uncommon but serious side effect. No blood or white blood cells are seen within the "rice-water" vomit caused by the toxin produced by Vibrio cholerae, which promotes water and chloride expulsion from the small bowel without damaging the mucous membrane [84]. It is possible to get gastroenteritis by sharing contaminated food or drinking water with another individual ("food poisoning").

Chicken, beef, and pig meats, as well as shellfish, are among the most common sources of bacterial pathological causes (such as severe abdominal discomfort, bilious vomiting, and abdominal mass). Youngsters with diabetes mellitus and metabolic disorders are more likely to vomit than healthy children. Children with underlying diseases are more likely to experience difficulties and should be referred to a pediatrician [85].

It is not necessary or practical to collect stool samples from every kid who has gastroenteritis. As soon as an epidemic occurs, samples must be gathered to identify the virus and locate its source for the sake of public health. There should be the cultivation of bacterial pathogens and testing for viral pathogens in the samples [86]. For rotavirus and other viruses, rapid antigen detection technologies are used in most children's hospitals (such as enzyme-linked immunosorbent assay). Because nosocomial infection is common and may be used to gauge the effectiveness of contact infection avoidance, the child must be isolated as soon as possible when a rapid diagnosis is made [87]. Children with bloody diarrhoea, those who have recently been abroad, or those who are young or immunocompromised and have a high fever should also have their stools sampled. As a matter of public health policy, practitioners in many countries are compelled to notify the government about a wide range of infectious and non-infectious diseases. This is a common misunderstanding [88].

5.2. Assessment of Dehydration

Taking a hydration status reading in gastroenteritis is crucial since it dictates the urgent treatment of this ailment. It is especially dangerous for a newborn or young child who has regular vomiting and large amounts of watery diarrhoea. Dehydration is commonly misunderstood by physicians. At least 5% of a child's body weight loss is usually required before clinical signs develop. Although this information is rarely accessible, recent weight loss is a reliable indicator of dehydration. More than 5% of the population is dehydrated, characterized by prolonged capillary refill, abnormal skin turgor, and a lack of tears [89].
Dehydration may be diagnosed and treated using the World Health Organization's categorization of dehydration, which is supported by research. Before and following intravenous fluids, it is recommended that serum electrolytes be evaluated [90].

5.3. Management of diarrhoea [91]

The management of diarrhoea representing the condition and the desired treatment is shown in table 1.

Table 1. Management of diarrhoea

Condition	Treatment
Admission (Hospital administration or home care)	Dehydration, shock, high risk of dehydration, or parents who can't handle the situation at home should be monitored for mild to moderate dehydration.
Enteral or IV	There is evidence to support the use of enteral rehydration for patients with severe dehydration. However, the results were skewed by a large study that included children with severe dehydration. Weight gain, diarrhoea duration, hyponatremia, hypernatraemia, and fluid consumption are all aided by enteral and intravenous fluids. Although the IV method has a low failure rate, phlebitis can occur if it is administered intravenously.
Low or high osmolarity ORS	No difference in the rate of hyponatremia is seen while treating non-chlorea diarrhoea with reduced osmolarity solutions.
Antidiarrhoeal	Loperamide: Time spent in the hospital and weight gain were both reduced in two studies, but there was not enough evidence to say if there was a risk of side effects, and there was not enough to say if there was a risk of side effects.
Antiemetic	Ondansetron Oral replacement therapy resulted in fewer episodes of vomiting, fewer IV fluids, and a shorter stay in the hospital, but it was associated with an increased risk of diarrhoea and illness after discharge.

Complications of acute gastroenteritis [92]

1 There is a lack of water.
2 The metabolic acidosis
3 Carbohydrate intolerance and electrolyte disturbances (hypernatraemia, hyponatremia, hypokalaemia)
4 Susceptibility to re-infection
5 Food intolerance is on the rise.
6 The hemolytic uraemic condition
7 Iatrogenic-related complications
8 loss of life.

6. Relation between IBD and Acute Gastroenteritis

According to the widely accepted theory of IBD etiology, inflammation of the intestinal tissues is caused by an overactive immune system triggered by a combination of inherited and environmental factors [93].

Much of what we know about the proximal events that led to the development of IBD is unknown. Nonsteroidal anti-inflammatory medicines and cigarette usage have also been linked to IBD. But a recent assessment of the literature concluded that no such supportive literature is there in support that these pathogenic organisms are related to IBD. Enteric infections may cause an initial overshooting reaction or a lack of down-regulation of the mucosal immune response, leading to chronic inflammation, which is consistent with the inability to identify particular pathogenic agents in IBD [94].

A variety of clinical observations support this theory. Although a limited proportion of individuals develop characteristic IBD following epidemics of Shigella, Salmonella, or Yersinia. A comprehensive microbiologic study may reveal recurrent endotracheal infections were present in 22% of IBD patients at the time of diagnosis. IBD exacerbations may also be caused by bacterial intestinal infections [95]. According to earlier research (GE), an infection with infectious gastroenteritis greatly increased the likelihood of irritable bowel syndrome. Unanswered questions include whether or not acute gastrointestinal infection can be a risk factor for developing IBD; how many risk factor is there; how long the higher risk lasts after a bacterial infection; and if IBD from infectious gastroenteritis has any distinct features [96].

6.1. Methods and Equipment

6.1.1. Insights into the Population

Using data from the General Practice Research Database, researchers were able to demonstrate a connection between Crohn's disease (CD), inflammatory bowel disease (IBD), indeterminate colitis, and ulcerative colitis (UC). The database, which is maintained by the Medicines and Healthcare Products Regulatory Agency (MHPRA), is a repository for electronic medical records contributed by around 2000 general practitioners located in the United Kingdom. Patients seen by medical professionals who are members of the Overall Practice Research Database have an age and gender distribution that is comparable to that of the general population of the United Kingdom [97]. Before any medical practice's data can be considered "up to standard," that practice must first show that it is proficient at inputting data into an electronic database. Then, at regular intervals, each practice is subjected to an audit to guarantee that the data quality continues to be very high. Analyses of electronic data are performed to determine weekly consultation numbers, the extent to which data on medicinal indications is full, and information on the causes of birth and death [98]. The computerized record contains information on the patient's demographics, medications, clinical events and diagnoses, preventive therapy, hospital admissions, and the cause of the patient's death. Major diagnoses were made before the implementation of the electronic medical record system, and these diagnoses are being recorded retrospectively. Documentation of diagnoses is done with the use of codes from the Oxford Medical Indexing System and, more recently, Read codes. According to the findings of several studies, the accuracy of electronic medical records satisfies the requirements for use in clinical trials. According to prior research, a substantial number of diagnoses are documented after a visit to a specialist Ref 99 missing [100]. This is the case in the great majority of epidemiological studies, which also include studies on IBD.

6.1.2. The Research Cohorts

All patients aged 20 to 74 who had an infectious GE episode throughout the study period were included. The following criteria were used to determine eligibility: Before the GE episode, patients had to be free of cancer, alcoholism, previous GE or IBD, concomitant gastrointestinal infection, or enteritis/colitis at any point in time. Also excluded were patients with a history of diarrhoea or rectal bleeding or those who had taken specific IBD medicine in the year preceding the GE admission date [101]. Created and examined all individuals with codes that indicated they were suffering from genocide (n 47,852).

In addition to any clinical information, demographic data were also recorded. The patient profiles did not include any personal information. Stool culture for an enteric infection other than one associated with a GE episode was used to exclude patients (n 4820). A total of 6414 individuals had "documented" bacterial gastroenteritis (GE), while 36,599 patients had "bacteriologically undocumented" GE (Shigella, Campylobacter, Salmonella, or other bacteria) (clinical diagnosis of GE with negative stool culture or no mention of a stool culture recorded). Our final group of acutely infected GE patients consisted of these two groups (N 43,013). For the comparator cohort, 50,000 participants were drawn from a

similar source population as the GE cohort, but with an additional restriction that they did not have a recorded diagnosis of GE in their medical records [102-103].

6.1.3. IBD Incident Case Investigation

Every person in either of the two groups had their information recorded starting on the day they were diagnosed with GE or, in the case of the control group, on a date chosen at random.

To be considered for inclusion, patients had to have a diagnosis of UC (n 5320), Regional Enteritis (n 5600), or "IBD not otherwise characterized" (n 92). Diagnostic codes for "IBD not otherwise defined" (n 92) have been found to have reduced reliability in prior research [105]. If specific IBD medicine was administered after the diagnosis was established, particularly prednisone-containing or mesalamine medications then such individuals come under deemed cases. According to the General Practice Research Database's validity and completeness, 92 percent of the IBD diagnoses recorded were accurate [106].

Patients having one of the above IBD codes (n 335) were sorted into three groups: "definite" IBD, "probable" IBD, and noncases based on computerized patient profiles. Patients with the first-ever diagnosis of IBD, specific treatment, and/or confirmation by a letter from a consultant or release from the hospital were deemed conclusive instances. Pending cases were those patients with an IBD diagnosis who had not yet had professional confirmation (n 15). Non-cases (n = 168) were assigned to the remaining participants [107].

It was sent to general practitioners with the request that they verify the diagnosis of all patients documented with collaborating practices who had a diagnosis that was either certain or possible (n = 83). Every patient's privacy was treated with the utmost respect at all times. We were able to confirm inflammatory bowel disease in all 76 individuals whose diagnoses had already been confirmed, as well as in six out of seven cases where the diagnosis had been suspected. As a result of this, we decided to include all of the extra instances of IBD that were confirmed (n = 76), but we did not include the other possible reasons for which we were unable to consult a medical professional [108].

6.2. Analysis

To determine the incidence rate of IBD in each cohort, we divided the total number of IBD cases that occurred during the follow-up period by the total number of experiences throughout that period. During the process of calculating the prevalence of IBD, age and gender were also taken into account. A regression analysis was carried out to assess the likelihood of IBD in the GE cohort, taking into consideration variables such as gender and year of the calendar. The hazard ratios (HRs) and confidence intervals (CIs) for 95 percent were calculated for both the first year of follow-up and the entire duration of follow-up [109–112].

To take into consideration newly discovered IBD risk variables, we conducted a nested case-control study spanning both of our research cohorts [113].

This research looked at all 158 cases of inflammatory bowel disease (IBD) that occurred within the two cohorts; the date of the first diagnosis was used as the index date for this analysis. Within the time frame of the study, each participant in the two groups was given a day that was selected at random [114-116].

Using this approach, the probability of being chosen as control is directly proportional to the total length of time that an individual is exposed to risk. For the frequency analysis, 2,000 people who did not have IBD were matched up with IBD patients based on their gender, age, and the year in which they were born [117–119]. The odds ratios and 95 percent confidence intervals for inflammatory bowel disease were calculated using unconditional logistic regression, which was adjusted for age, gender, calendar year, general practitioner visits in the previous year, and other IBD risk factors. This allowed for an accurate estimation of the ORs and CIs. It was determined that these values provided appropriate measurements of the relative risk for IBD [120-125].

6.3. RESULTS

The GE cohort had forty individuals with CD, sixty-four with UC, and four with IBD type undefined. In terms of IBD distribution, there were no differences between the documented (bacterial) and undocumented GE subgroups in terms of IBD distribution in the comparator cohort that was free of GE [126]. Campylobacter was the most common microbe found in the bacterial GE cohort (4124 patients), followed by Salmonella in 1885 patients, and Shigella in 312. In the remaining 93 cases of bacterial GE, the infection was caused by another bacterium (most likely Staphylococcus, C. difficile, E.coli) [127-129].

According to the research findings, there was a 68.4 per 100,000 person-years overall incidence rate of IBD in patients following an episode of GE. Both the documented bacterial GE cohort and the bacteriologically undocumented GE cohort were included [130]. There was a 30.0 per 100,000 person-years estimate in the control group [131]. In Campylobacter and Salmonella-infected individuals, the incidence rate of IBD was 105 and 60 per 100,000 person-years, respectively. On average, in the first year following an infectious gastrointestinal episode, there was an increased risk of developing

IBD for those who were part of the GE cohort as opposed to those in the control group [132-133]. Patients with proven bacterial GE had an IBD risk of 2.7, whereas patients with bacteriologically undocumented GE had an IBD risk of 2.4. When it came to getting CD, those in the GE group were far more likely than those in the UC group, especially in the first year after the infectious episode [134].

In the nested case-control study, researchers were unable to find any correlation between UC or CD and osteoarthritis, rheumatoid arthritis, depression, anxiety, asthma, chronic pulmonary sickness, gastroesophageal reflux disease, and appendicitis, or any of the other aforementioned conditions [135]. Persons who have CD are more likely to have peptic ulcers compared to those who have UC, which suggests that some of these illnesses are associated with upper gastrointestinal CD. Peptic ulcers are more prevalent in people who have CD compared to those who have UC. Smokers and ex-smokers both had a greater likelihood of getting CD, while ex-smokers had a slightly higher risk of developing UC. This finding was consistent with what was hypothesized. Those who were given antibiotics during the first two weeks following their first episode of GE had a lower risk of developing inflammatory bowel disease (IBD) compared to patients who were not given antibiotics [136-140]. gender, the year on the calendar, the number of visits to the primary care physician in the previous year, and other IBD risk indicators were taken into consideration. It was determined that these values provided appropriate measurements of the relative risk for IBD [120-125].

7. CONCLUSION

Since acute gastroenteritis usually resolves on its own, antibiotics are rarely needed in most cases. Patients with community-acquired diarrhoea who have a fever and bloody diarrhoea or feverish diarrhoeal illness, or who are immunocompromised, should consider receiving an empirical antibiotic. Individuals with traveller's diarrhoea should be treated with antibiotics in cases of mild to severe illness. To minimize the frequency of treatment failures, it is essential to identify the patterns of local resistance. The initial therapy for acute gastroenteritis is still oral rehydration therapy. Many doctors still don't think that many antiemetic drugs are good for their management. One study shows that ondansetron decreases the number of times a person throws up, improves the effectiveness and adherence of ORT, and decreases the number of times a person needs IV treatment. However, more research is needed. As a result, there may be fewer people needing medical attention. Anecdotal evidence suggests that ondansetron will reduce health care costs in people with acute gastroenteritis, notwithstanding the lack of a thorough economic study. When compared to a placebo, ondansetron did not influence the frequency of follow-up visits. Pediatric gastroenterology and infectious disease experts in Europe produced an evidence-based guideline in 2008 on how to treat acute gastroenteritis in children in the European Union. Antiemetics should be recommended only to those patients who can benefit from them, as well as those antiemetics that have the highest therapeutic value. Excellent acute gastroenteritis therapy must always start with the following elements: Fast oral rehydrating over 3 to 4 hours, hypotonic oral rehydration solution, oral rehydration for dehydration; oral rehydration solution supplementation for ongoing losses, rapid alimentation (macro and micro nutrient intake) with normal feeding; use of the diluted formula is unjustified; use of the special formula is unjustified, Continue breastfeeding at all times; is recommended. Some people may benefit from antiemetic medications as a third pillar. Three separate sets of criteria should be evaluated: clinical conditions, host-related characteristics, and the environment. If the symptoms are minimal and it is feasible to keep an eye on them, it may be best to wait for the findings of microbiology. Some cases of traveller's diarrhoea require antibiotics. Depending on the etiology and local resistance trends, the antibiotic should be selected accordingly. While it is vital to reduce the overuse of antibiotics, they can be lifesaving when used in the right circumstances. Clinical and epidemiological considerations must be considered while using drugs. According to epidemiological research, most acute gastroenteritis episodes are self-limiting, and laboratory testing should only be conducted if the results might change the treatment and prognosis of a specific patient. A thorough history and physical examination, according to ample evidence, can be used to diagnose acute diarrhoeal illness, estimate the severity of dehydration, determine whether additional testing is required, and initiate appropriate treatment. Restoring an unrestricted diet to children in the maintenance phase of therapy is strongly recommended based on available research. Acute gastroenteritis is usually not a good candidate for pharmacological treatment, and the use of medications can impede the natural course of the illness.

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Conflict of Interest

None conflict of interest has been declared by authors.

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