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ACUTE GASTROENTERITIS

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Abstract:

Introduction: *Gastroenteritis is defined as the presence of an inflammation in the stomach, small intestines, and/or large intestine, causing the development of a combination of abdominal pain, cramping, nausea, vomiting, and/or diarrhea. Acute gastroenteritis often lasts less than fourteen days. This is considered to be the contrast of persistent gastroenteritis, that lasts between fourteen and thirty days, and chronic gastroenteritis, that lasts longer than thirty days.*

Aim of work: *In this review, we will discuss acute gastroenteritis.*

Methodology: *We conducted this review using a comprehensive search of MEDLINE, PubMed, and EMBASE, January 1985, through February 2017. The following search terms were used: Acute gastroenteritis, causes, presentation, management, emergent management of acute gastroenteritis*

Conclusions: *Acute gastroenteritis is considered to be a relatively common infectious syndrome, that is associated with the development of a combination of vomiting, nausea, diarrhea, and abdominal pain. Contact precautions, public health education, and prudent use of antibiotics are still necessary goals. Complicating these efforts are that there is increasing antibiotic resistance to C difficile and a new strain, NAP1/027/III, has been emerging since the early 2000s. This new strain has a high association with community onset and has been linked to increasing frequency and severity of illness. There is research into the possibility that the community onset may be related to animals and to retail meat, where the new strain has been detected. Preventing dehydration or providing appropriate rehydration is the primary supportive treatment of acute gastroenteritis*

Key words: *Acute gastroenteritis, causes, presentation, management.*

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INTRODUCTION:

Gastroenteritis is defined as the presence of an inflammation in the stomach, small intestines, and/or large intestine, causing the development of a combination of abdominal pain, cramping, nausea, vomiting, and/or diarrhea. Acute gastroenteritis often lasts less than fourteen days. This is considered to be the contrast of persistent gastroenteritis, that lasts between fourteen and thirty days, and chronic gastroenteritis, that lasts longer than thirty days. [1]
In this review, we will discuss the most recent evidence regarding Acute Gastroenteritis.

METHODOLOGY:

• Data Sources and Search terms

We conducted this review using a comprehensive search of MEDLINE, PubMed, and EMBASE, January 1985, through February 2017. The following search terms were used: Acute gastroenteritis, causes, presentation, management, emergent management of acute gastroenteritis

• Data Extraction

Two reviewers have independently reviewed the studies, abstracted data, and disagreements were resolved by consensus. Studies were evaluated for quality and a review protocol was followed throughout.
The study was approved by the ethical board of King Abdulaziz University Hospital

EPIDEMIOLOGY:

In the US, acute gastroenteritis is usually seen as a nuisance instead of being the life-threatening illness it could be in other low-income countries. Despite the presence of possible associated significant morbidity and mortality attributed to acute diarrheal conditions in the US, large observational studies in the US have not been as large as those done in other low-income countries. The CDC, on the other hand, estimate that there are more than 350 million cases diagnosed with acute diarrheal illnesses in the US every year.
Acute gastroenteritis is considered to compare with upper respiratory conditions as the most common infectious disease syndromes. [2] Using data obtained from the National Center for Health Statistics, the CDC recently stated that mortality due to all-cause gastroenteritis elevated from about 7000 per year to more than 17,000 per year between the years 1999 and 2007. Adults aged more than sixty-five years old were about eighty-three percent of these deaths and the organism C difficile was responsible for about two-thirds of these deaths, making a reflection that the most significant morbidity and mortality rates are present in extremes of age. [3]

Etiology of acute gastroenteritis

Acute gastroenteritis is usually caused by many organisms. Evaluating the accurate incidence and etiologies of acute gastroenteritis is generally challenging because not all patients reports their clinical manifestations or ask for medical care. additionally, stool analysis, that is used to detect bacterial causes of gastroenteritis, is only positive in about two percent of cases. [4]

Viral etiologies that can cause acute gastroenteritis are dominated by rotavirus and norovirus. In the US, it is thought that up to twenty-five million cases of viral gastroenteritis happen every year, leading to three-to-five million office visits and 200,000 hospital admissions. [5] Rotavirus can cause a specifically severe dehydrating gastroenteritis affecting younger children. The severity of the disease is even worse when accompanied by malnourishment, making rotavirus an important cause of death in children around the world, leading to about 500,000 deaths every year. [6] The introduction of the rotavirus vaccine in the US and Europe has been beneficial at decreasing rotavirus gastroenteritis.

There has been a sixty-seven decline in positive laboratory diagnosis which is attributed to the applications of vaccination. [7] Norovirus, on the other hand, is associated with the most outbreaks of nonbacterial acute gastroenteritis among all ages. It usually happens in epidemic outbreaks in schools, nursing homes, cruise ships, prisons, and other group settings. Clinical manifestations of severe vomiting are generally self-limited, lasting twelve to sixty hours. Transmission of this virus is achieved through the fecal-oral route, with viral shredding lasting on average ten to fourteen days following the onset of clinical manifestations. [8]

Rotavirus and enteric adenovirus could be detected using rapid assays for the viral antigen in stool. Norovirus is best detected using reverse transcriptase–polymerase chain reaction.

Etiology of chronic gastroenteritis

Etiologies of persistent or chronic gastroenteritis can include parasitic infections, drugs, inflammatory bowel disease (including ulcerative colitis, collagenous colitis, Crohn disease, microscopic colitis, and others), eosinophilic gastroenteritis, irritable bowel syndrome, lactose intolerance, celiac disease, colorectal cancer, malabsorption, bowel obstruction, and ischemic bowel. Immuno-compromised individuals are most susceptible to chronic gastroenteritis infections. Crytosporidium has

been found to be an etiology of chronic diarrhea in patients diagnosed with AIDS. It is also responsible for huge outbreaks in care centers and swimming pools and has contaminated water supplies. One of the several reasons for these outbreaks is that the oocytes are resistant against the application of bleach or other disinfectants, making them easily transmittable by contact with contaminated surfaces or by person-to-person contact.

Giardia is another common organism that causes chronic gastroenteritis. It is found in contaminated streams but is also present in care centers and public swimming pools. Giardia is known to cause clinical manifestations that include flatulence, bloating, and explosive, foul-smelling, pale, diarrhea.

TYPES OF ACUTE GASTROENTERITIS:

During the remainder of this article we will focus on acute bacterial gastroenteritis and review the common organisms that are associated with the development traveler's gastroenteritis, foodborne gastroenteritis, and/or antibiotics-associated gastroenteritis. For each of those diseases, we will discuss the transmission, the pathology, the incubation period, clinical manifestations, clinical manifestations duration, treatment, and prophylaxis.

Traveler's Diarrhea

Travelers to low-income countries usually present to their primary care physicians with concerns about developing traveler's diarrhea and how to prevent or treat this condition in cases it occurs; forty percent to sixty percent of travelers to low-income countries usually develop this condition. It must also be considered if diarrhea starts within ten days of their return home. For epidemiologic concerns, traveler's diarrhea is usually categorized into classic form, moderate form, and mild form.

The classic form is diagnosed when there are three or more unformed bowel movements every twenty-four hours in addition to the presence of at least one of the following criteria: vomiting, nausea, fever, abdominal pain, and blood in the stool. The Moderate form is diagnosed when there are one or two unformed bowel movements every twenty-four hours in addition to the presence of at least one of the above-mentioned clinical manifestations. On the other hand, the Mild form is usually diagnosed when there are one to two unformed bowel movements without the presence of any of the previously mentioned other clinical manifestations.

Traveler's diarrhea is generally transmitted through contaminated food or contaminated water. It could be caused by bacterial, viral, or parasitic organisms. Bacterial organisms cause most cases of traveler's diarrhea. The most common bacterial organisms are considered to be enterotoxigenic E coli, which is followed by Salmonella, Campylobacter jejuni, and Shigella. In one study that included about 322 subjects, ETEC was found to be responsible for twelve percent of bacterial traveler's diarrhea, Salmonella was found to be responsible for eight percent of bacterial traveler's diarrhea, Campylobacter jejuni was found to be responsible for six percent of bacterial traveler's diarrhea, and Shigella was found to be responsible for less than one percent of bacterial traveler's diarrhea. In one other study that included about 636 travelers, ETEC was found to be responsible for thirty percent of bacterial traveler's diarrhea and enteroaggregative E coli was found to be responsible for twenty-six percent of bacterial traveler's diarrhea. [9]

The presence of a coinfection with an additional organism was detected in about twenty percent of travelers. [10] sites of the world with the highest risk are certain countries in Asia, the African continent, and in Central America and South America.

Pathogenesis

Incubation period is generally about four to fourteen days following the arrival to an endemic country. Common clinical manifestations can include anorexia, malaise, abdominal pain and cramping, nausea, watery diarrhea, and vomiting, along with low-grade fever. When the condition is caused by Campylobacter jejuni or Shigella, clinical manifestations might progress to bloody diarrhea, colitis, and tenesmus. Duration is usually one to five days and usually self-limited, however, in eight percent to fifteen percent of cases, clinical manifestations can possibly last longer than seven days.[11] If nausea, bloating, or other gastrointestinal clinical manifestations last for longer than fourteen days, an alternative diagnosis, such as the presence of a parasitic infection should be considered.

DIAGNOSIS:

The diagnosis of the disease is usually based on clinical examination and confirmation is often not required as traveler's diarrhea is usually self-limited. Stool analysis might be beneficial in individuals who develop severe clinical manifestations, chronic disease, bloody diarrhea, and/or fever. Stool analysis, on the other hand, cannot differentiate between nonpathogenic E coli and ETEC or enteroaggregative E coli.

TREATMENT:

Fluid replacement is considered to be the most

important treatment in patients with symptomatic disease. It can be applied with or without the application of specific diet restrictions. There is limited evidence on whether a clear liquid diet versus an unrestricted diet potentially affects the duration and/or the severity of clinical manifestations as traveler's diarrhea is often self-limited, lasting three to five days. Oral rehydration is considered to be ideal, but IV hydration might be required in the setting of severe dehydration.

The administration of antibiotics might shorten the course of the condition by one to two days. Travelers usually request a prescription for antibiotics that might be taken at the onset of clinical manifestations. Ciprofloxacin is usually enough, despite that resistance to quinolones has been increasing, specifically in cases of Campylobacter jejuni. Quinolones are not approved for the use in pregnancy or for the management of traveler's diarrhea in pediatrics. Azithromycin is considered to be appropriate in these sub-groups. In adults, a single dose is enough. In pediatrics, recommended dosing is estimated to be about ten mg/kg as a single dose, not to exceed 1 g.
Antimotility drugs, like loperamide or diphenoxylate, could potentially reduce stool frequency but do not change the course of the disease. Their use must be avoided in patients who show signs of fever or rectal bleeding. Lactobacillus GG, which is a specific probiotic, has been also proven to reduce the diarrhea which is caused by the pathogens that classically cause traveler's gastroenteritis. Other Lactobacillus preparations, on the other hand, based on non-viable probiotics, have not. [12]

Prevention
In the year 2001, the Infectious Diseases Society of America released their guidelines to help travelers reduce their risk of developing traveler's diarrhea: Water should be boiled for three minutes to kill organisms. Two drops of bleach or five drops of iodine will kill pathogens in water within thirty minutes. Freezing will not kill pathogens. Avoid ice, request bottled beverages, and use a straw instead of a glass.

Fruit that is peeled is usually safe. Fruit that is not peeled or raw vegetables must be avoided. Steam table buffets are associated with a high risk of developing traveler's gastroenteritis. Communal condiments are usually contaminated and must be avoided. drugs, like H2 blockers and proton pump inhibitors, could elevate susceptibility to traveler's diarrhea. These drugs are known to lower gastric acidity and could potentially elevate the risk of developing traveler's diarrhea by allowing more organisms to survive transit to the small bowel. In addition, conditions or drugs that cause slowing of the gastric motility generally allow the number of pathogens to increase.

Foodborne Acute Gastroenteritis
The CDC estimated that forty-eight million cases of foodborne bacterial gastroenteritis are diagnosed every year in the US alone, causing about 125,000 hospital admissions, three thousand deaths, and costs that are higher than $150 billion.2 The Foodborne Disease Active Surveillance Network was established in the year 1996 by the CDC to detect foodborne gastrointestinal conditions in the US. Data released from this ten-site study that covered forty-six million patients suggested that one in five cases of gastroenteritis are due to foodborne pathogens. Data from the year 2010 showed little significant overall change in the known foodborne etiologies of acute gastroenteritis over the past four years. The data did, on the other hand, show a decrease in Shiga toxin–producing E coli and Shigella. Vibrio gastroenteritis incidence increased over this time period and Salmonella was unchanged, despite higher awareness and more efforts to reduce the incidence of these infections. generally, management of foodborne gastroenteritis could range in cost from $78 in the state of Montana to $162 in the state of New Jersey. The total cost per case, including productivity losses, could be as high as $1506, as noted in the state of Connecticut.

Clinical manifestations of mild food-borne disease include the development of a Sudden onset of nausea and vomiting following eating, which is usually due to the ingestion of a preformed toxin. Stool culture is not usually contributory, and Diagnosis is often made clinically. antibiotics treatment is not needed in these cases as it is caused by preformed enterotoxin. Supportive care and IV antiemetics can help control vomiting. Rapid spontaneous recovery in one day is typical.

Salmonella bacteria can also be transmitted following the consumption of contaminated raw eggs or undercooked eggs, raw milk, meats, peanuts, ice cream, fruits, and/or vegetables. Transmission can also occur following contact with infected animals, like turtles and pet ducklings. Salmonella can survive the acidity of the stomach, leading to colonization in the intestines. Then, it moves across the intestinal epithelium, either by direct invasion of enterocytes or through dendritic cells which are inserted into epithelial cells. Once present, the inflammation begins to release cytokines, neutrophils,

macrophages, T cells, and B cells. This inflammatory response reduces normal intestinal flora and allows the organism to proliferate. The nontyphoid Salmonella produces a more localized response and the typhi serotype (which causes typhoid fever) tends to be associated with more invasion and more usually leads to the development of bacteremia. [13]

The disease usually lasts for one to seven days. Clinical manifestations include vomiting, nausea, cramping abdominal pain, fever, and sometimes, bloody diarrhea. Treatment is not often recommended with the presence of some exceptions including the presence of a severe illness, patients in extremes of age, the presence of a valvular heart disease, the detection of uremia, or the presence of a malignancy. In these cases, a cephalosporin or a fluoroquinolone for five to seven days can be indicated to prevent the development of severe complications. The overuse of antibiotic can potentially increase the rates of carrier state. Other complications include the development of transient reactive arthritis, that could be seen in up to thirty percent of adult patients, and Reiter syndrome, that occurs in about two percent of patients. Most infected patients recover in two to five days. Sustained or intermittent bacteremia might occur in immune-compromised patients.

Campylobacter jejuni can usually develop among patients younger than five years of age. Transmission occurs by handling or eating raw or undercooked poultry or raw milk or cheeses, by contaminated drinking water, or by handling infected animals. mechanisms of the disease include the direct invasion of epithelial cells of the colon inducing inflammation. Incubation is usually about one to ten days, and the disease lasts for about five to fourteen days. Clinical manifestations often include the development of rapid onset of fever, headache, chills, and malaise which can be followed by the occurrence of abdominal pain, vomiting, nausea, and diarrhea. Diarrhea might be bloody or melanotic in up to ninety percent of patients. the use of Empiric antibiotics for treatment is not usually recommended in otherwise healthy patients. Stool analysis can be appropriate. Antibiotics are usually known to shorten the disease by about one day. Campylobacter jejuni gastroenteritis has been associated with the development of a postinfectious Guillain-Barre´ syndrome, with an incidence one per 1000. In cases of severe clinical manifestations, this might be less reversible. Most patients recover within one week following the onset of the disease. Relapses can be common but tend to be milder than the first infection. Mortality following infection is rare. [14]

CONCLUSIONS:

Acute gastroenteritis is considered to be a relatively common infectious syndrome, that is associated with the development of a combination of vomiting, nausea, diarrhea, and abdominal pain. The Centers for Disease Control and Prevention estimated that there were more than 350 million patients with acute gastroenteritis in the US every year, and forty-eight million of these patients are usually due to foodborne bacteria. Traveler's diarrhea can affect more than half of people traveling from developed countries to other low-income countries. Prevention can be summarized by extreme caution, "boil, cook, peel, or forget it." Except in cases of high fever, the presence of bloody diarrhea, immune-compromised patients, or patients with significant other comorbidities, identifying a specific organism is not indicated in patients with suspected acute bacterial gastroenteritis as the disease is usually self-limited. In both adult and pediatric patients, the prevalence of Clostridium difficile is increasing in the United States. Contact precautions, public health education, and prudent use of antibiotics are still necessary goals. Complicating these efforts are that there is increasing antibiotic resistance to C difficile and a new strain, NAP1/027/III, has been emerging since the early 2000s. This new strain has a high association with community onset and has been linked to increasing frequency and severity of illness. There is research into the possibility that the community onset may be related to animals and to retail meat, where the new strain has been detected. Preventing dehydration or providing appropriate rehydration is the primary supportive treatment of acute gastroenteritis

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