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DIARRHEA IN CHILDREN AND INFANTS CAUSED BY E.COLI: A REVIEW ARTICLE

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Abstract:

Although *Escherichia coli* is one of the most researched microbes on the planet, its properties are always evolving. the intestines of healthy animals and people, the bacteria *Escherichia coli* can be found. The vast majority of strains of these bacteria are either harmless or can cause just little diarrhea. Other strains, as E. coli O157:H7 have the ability in some people, produce severe stomach cramps, bloody diarrhea, and vomiting.

Escherichia coli established infection in their host by different way. The diagnostic methods development and these method were used for identifying and categorizing *E. coli* bacteria into different pathotypes has resulted from a better understanding of these pathogenic pathways. The goal of this paper is to give overview of many types of *Escherichia coli* that cause diarrhea.

Keywords: ***Escherichia Coli, Diarrhea, Children***

INTRODUCTION

In poor nations, diarrhea considered as a primary morbidity and mortality causes in children under age five (Huang *et al*., 2002). In some locations, *Escherichia coli*, a frequent cause of gastroenteritis, contributes for 30% of the whole type bacteria that cause diarrhea. (Alikhani *et al*., 2007).

In Asia, Africa, and America, *Escherichia coli* infection was one of the top causes of death, with 4.6-6 million people dying each year.(Rice *et al*., 2000; Velayati *et al*., 1987). At this moment, the rate of fatalities per 1,000 births is reaching 9.7. (Alikhani *et al*., 2007).

Escherichia coli is a common bacteria that comes in a wide range of serotypes, from extremely pathogenic to virulent strains. Numerous *E. coli* diarrhea causing strains have been discovered via extensive investigation, each with its own set of pathogenic properties. (Harris, 2005).

COMMON CLINICAL SIGN AND SYMPTOMS OF DIARRHEA :

Diarrhea, often known as diarrhoea, is a condition in which you have at least you should have three symptom every day loose, liquid, or watery bowel motions. It usually lasts a few days and might cause dehydration due to the loss of liquids. Dehydration is identified by a lack of normal skin stretchiness also irritable behavior. Reduced urination, skin discoloration, a fast heart rate, and a loss of attention can all occur as the illness worsens. (WHO,2020).

Mild bouts of diarrhea go away in a few days, but severe cases can lead to dehydration and nutritional issues. The most serious complication of diarrhea is dehydration, which occurs when a patient loses up to a gallon of water each day, as well as minerals (electrolytes) that are necessary for normal physiological functions. The main electrolytes are salt and potassium. (Ansaruzzaman *et al*., 2000).

Severe dehydration can put the body into shock and be lethal; infants and children are more vulnerable to dehydration than adults. (Rice *et al*., 2000).

E.COLI AS A CAUSE OF DIARRHEA:-

Investigation showed presence of numerous classes of *E.coli* that causes diarrhea, each with its unique set of pathogenic and virulence features that span epidemiology and clinical syndromes. enterotoxigenic *E. coli* (ETEC), Enteropathogenic *E. coli* (EPEC), enteroaggregative *E. coli* and enteroinvasive *E. coli* (EIEC), are examples of these bacteria (EAEC) (Gomes *et al*., 2004; Tramutal *et al*., 2008). The prevalence of each of these kinds differs by countries, and the pathogenic pathways have only recently been discovered. (Tarblulsi *et al*., 2002; Elias *et al*., 2002). Diarrheal illness incidence has decreased as a result of the adoption of a WHO-guided local diarrheal disease control program, which includes breast feeding promotion, therapy by oral rehydration, and particular health education about diarrhea disease. (Regue *et al*., 1990).

About 0.1 % of the gut microbiota is made up of *E. coli* and other facultative anaerobes.,(Eckburg *et al*.,2005) In lack or presence of oxygen, bacterium can grow. It will grow through fermentation, creating characteristic "mixed acid and gas" as end products. Within the periplasm of *E.coli* cells, there is a one layer peptidoglycan. N-acetylmuramic acid

connected to peptide comprising of L-alanine, D-glutamic acid, meso-diaminopimelic acid, and D-alanine via an amide bond in the peptidoglycan's usual subunit structure. *E.coli* motile by Peritrichous flagella in liquid. Fimbriated *E.coli* are prevalent, The most common pili were type 1, which were expressed as an on/off phase as switch fashion, resulting in piliated and nonpiliated states. (Eisenstein, 1987).

There was a lot of variety in *E.coli* isolates, due to a lot of different (O and K) somatic antigen mixtures and flagellar (H) antigens. Approximately ,there is 150 antigenically distinct O-antigens in *E.coli* cell wall (WHO, 1987). There are two or four different types of K type capsule material. (Whitfield & Roberts, 1993). The serotype (for example, O157:H7, O111:H8) is the combination of O:H.

Types of *Escherichia coli*

- Enterotoxogenic *E.coli* In both people and animals, ETEC stains are a communal cause of secretory diarrhea. They create heat-stable (STa and STb) and/or heat-labile (LT) toxins.
- *Enterohaemorragic E.coli* (EHEC) stains have been linked to food-borne infections, which are spread through the consumption of undercooked minced beef and unpasteurized milk. Shiga-like toxin1 and 2 (stx1) (stx2), and diversity yeilded from these stains. They were involved in a series of episodes of diarrhea that resulted in complication . (Brown *et al.*, 1998). EHEC, develops an adhering and effacing intestinal mucosa lesion, which requires a functional eaeA chromosomal gene. Only occasional cases of EHEC have been described in Colorado. (Wieler *et al.*, 1998).
- *Enteroinvasive E.coli (EIEC)* causes a variety of human disorders. These type of *E.coli* and *Shigella spp*. Are connected biochemically, genetically and pathogenically . EIEC causes invasive inflammatory colitis and dysentery, with bloody and mucous stools, fever, and severe cramp's as symptoms. EIEC attack epithelium layer of intestine, primarily large intestine, They rarely go to the bloodstream, but get into the lamina propria, which is just under the epithelium of the intestine in which they interact with immune cell especially macrophages which produce pro-immune peptides. (Casalino *et al.*, 2003).
- *Enteroaggregative E.coli* are type of *E.coli* strain that bind to HEP-2 cells in vitro (EAEC) (Narto &Kaper, 1998) EAEC were previously connected to outbreaks and diarrhea in patients with AIDS in the developing world. in industrialized world. these illness was recognized as watery diarrhea and cramps in stomach, while there is no fever. There was absent for the bloodstream invasiveness. (Sousa &Dubreuil, 2001).
- Diffusely Adherent E.coli (DAEC) are distinct by the existence of the diffusely adherent pattern in the HEP-2 adherence assay, can cause a watery diarrhea disorder in adults and children. Most of the DAEC stains contain a external fimbria identified as F1845, which are coded by either plasmid or a chromosome. In extremely young children, DAEC can induce diarrhea. (Scaleky *et al.*, 2002).

EPEC Clinical Symptoms of Infection:

Enterpathogenic *E.coli* considered the most communal reason of diarrhea in the infant in impoverished nations, a recent outbreak revealing a 30% mortality rate. EPEC infection is projected to kill hundreds of thousands of children each year. (Vallance & Finlay, 2002).

In babies, the EPEC infection causes severe diarrhea, fever, vomiting, and stomach pain that can last for up to 14 days. Acute watery diarrhea compine by a copious of mucus but with no blood, vomiting,nausea, headache,stomach cramps, chills and fever in adults. The disease lasted anywhere from 6 hours to 3 days, with an average of 24 hours. (Scalesky *et al.*, 2002). EPEC's disease-causing processes were not linked to heat-labile enterotoxins (LT), heat-stabile toxins (ST), Shiga-like invasiveness, or verocytotoxigenic characteristics, despite the infectious dosage being 10^5-10^{10}. They appear to eliminate the microvilli without causing any additional invasion. (Girarad *et al.*, 2005; Jafari *et al.*, 2005).

PATHOGENICITY:

Pathogenicity of *E.coli* refers to their ability to infect people and other animals with sickness. (Scaletsky *et al.*, 2002; Wieler *et al.*, 1998). *E.coli* Pathogenicity typically involves numerous separate and diverse pathways. (Kaper , 1998; Oswald *et al.*, 2000) To be a pathogenic, the *E.coli* necessity have the pathogenic component genes and are able to manufacture the gene produces in vivo. (Manjarrez-Hernandez *et al.*, 2000; Beutin *et al.*, 2004; Cleary *etal.*,2004).

Pathogen that cause enteric disease must be able to move through the intestine of the host., that was generally necessary to resistance to reduced pH effect on the bacteria and tolerance of digestive components such as bile. EPEC are known for their ability of causing human diarrhea, especially in young infants. The cause of acid tolerance and bile resistance is unknown, on the other hand one of *E.coli*'s pathogenic strategies was the ability to express a variety of adhesion mechanisms as well as they can enter to the gut and create toxins. (Chart, 2000).

As an external pathogen.,EPEC causes disease by adhering on the host cell surfaces then introducing virulence components to the cell the bacteria adhere on to it by using type III secretion system. (Lai *et al.*,1997).

Bacteria that make it through the small intestines and large intestines first enter the mucosa of the gut, colonize in the epithelium of intestinal, and then cause disease in competition with commensal bacteria. (Kaper et al., 2004). Various toxins may be expressed once in close proximity to the intestine (Beutin et al., 2004; Girarad et al., 2005). Fimberil adhesions can be found in bacteria that infiltrate the stomach of the host and create poison (Nagy & Fekete, 1999; Bradly et al., 2001).

The bacteria have the ability of to get "free iron" during pathogenesis from a host is another well-known pathogenic mechanism; however, the quantity of "free iron" in a mammalian as a host is very low and insufficient to allow E.coli stains to proliferate. (Braun &Braun 2002).

Extracellular iron is connect to high attraction iron-binding glycoproteins in serum and many other bodily fluids, transferrin in blood and lymph, and lactoferrin in external secretion and also milk. (Braun & Braun, 2002).

Due to the lipids in *E.coli* or gram negative bacteria, they are powerful complement activators. A lipopolysaccharide moiety is a group of lipopolysaccharide molecules. (Crane and Oh, 1997; Roxas et al.,2007).

EPEC strain that forms a close bond with mammalian cells causes a localized rise in intracellular calcium and increases inositol phosphate synthesis. (Crane & Oh .1997).

An rise in membrane-bound protein kinase C (PKC) action is followed by a reduction in cytosolic PKC action in EPEC-infected HeLa cells. Because the intimin mutant had no influence on activity, increasing protein kinase C activity required close bacterial adhesion. (Finlay *et al.*, 1992; Frankel et al., 1998).

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