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The Biological Characteristics of Human Rotavirus and Their Relationship to Gastroenteritis, As a Literature Review

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***Abstract*—** Rotavirus Group A (RVA) is still the leading cause of severe acute diarrhea in children all over the world. Ruth Bishop and her colleagues identified the virus in humans in 1973, and it belongs to the Reoviridae family, Genus Rotavirus (Group III – dsRNA). The virus has a segmented double-stranded RNA genome and is non-enveloped, icosahedral, and triple-layered. Each of the 11 segments of the viral genome encodes at least six structural proteins (VP1 through VP4, VP6 and VP7), as well as five or six nonstructural proteins (NSP1-6) depending on the strain. Rotavirus is known for transmitting from person to person via the feces–oral route. The RV may also be spread through feces-contaminated water in low-income communities. The RV might also be handed down from one child to the next by polluting contaminated surfaces. Rotaviruses (RVs) cause diarrhea and enterocyte death when they infect mature enterocytes on the tips of the villi of the small intestine. Symptoms of RV infection, including acute watery diarrhea, nausea, vomiting, and a low-grade fever, can last for days and cause dehydration. Rotavirus infection was detected by electron microscopy, immunochromatography, EnzymeLinked Immunosorbent Assay (ELISA), RNA PAGE, and reverse transcription polymerase chain reaction (RT-PCR). Due to the lack of a particular antiviral treatment, effective RV vaccinations are beneficial in reducing morbidity and death. Oral Rotavirus vaccinations are available worldwide (RotaTeq and Rotarix).

Keywords— Rotavirus, Discovered, Structures, Target organ, Gastroenteritis, Pathogenesis,

I. INTRODUCTION

Rotaviruses are a significant cause of severe acute gastroenteritis, killing an estimated 453,000 children each year, mostly under the age of five, with over 85 percent of these deaths happening in Asia and Africa's less developed countries. During childhood, children are exposed to numerous Rotavirus infections, which are nearly usually asymptomatic [1]. The Rotavirus Group A (RVA) is still the most common cause of severe acute diarrhea in children across the world [2]. RVA was expected to have killed more than 128,000 children under the age of five worldwide in 2016, with more than 104,000 deaths happening in Sub-Saharan Africa. The virus belongs to the family Reoviridae, Genus Rotavirus (Group III – dsRNA), and was discovered in humans by Ruth Bishop and her colleagues in 1973 [3]. The virus is non-enveloped, icosahedral, and triple-layered, having a segmented double-stranded RNA genome [4]. The viral genome is divided into 11 segments, each of which encodes at least six structural proteins (VP1 through VP4, VP6 and VP7), as well as around five or six nonstructural proteins (NSP1-6) depending on the strain [5]. The antigenic determinants in the VP6 protein, which is found in the virus's inner capsid, allow categorization into seven serogroups ranging from A to G, with group A being the most prevalent cause of infantile diarrhea [6]. The RVAs are divided into G and P genotypes based on viral proteins (VP) 7 and VP4 [7]. There are now 27 G-genotypes, 35 P-genotypes, and a minimum of 73 G/P-genotype combinations. G1–G4 and G9 are the most frequent G-genotypes seen in youngsters across the world, according to reports [8 Rotavirus infection can be asymptomatic or symptomatic, with a 1–3 day incubation period and a 5–7day sickness. Though it's most commonly linked with infants, immunocompromised individuals, and the elderly [9]. Rotavirus is recognized for its fecal–oral mode of transmission from person to person. In poor areas, the RV may also be spread by feces-contaminated water. It's also possible that the RV might be passed from one child to the next by contaminating contaminated surfaces [10]. Rotaviruses (RVs) infect mature enterocytes on the tips of the villi of the small intestine, producing diarrhea and enterocyte death [11]. Malabsorption of fluids and electrolytes caused by extensive damage to the small intestinal villous epithelium, activation of the enteric nervous system induced by neurological dysfunction and the resulting disruption of blood flow due to damaged enterocytes, and the action of RV non-structural protein, NSP4, an enterotoxin, have all been proposed as mechanisms for RV-induced diarrhea [12]. RV infection symptoms, including as acute watery diarrhea, nausea, vomiting, and a low-grade fever, can continue for many days and induce dehydration [13]. Initially, Rotavirus infection was diagnosed via electron microscopy, which involved seeing and observing the Rotavirus's wheel-like morphology [14]. Nowadays,

antigen detection is used to diagnose Rotavirus infection in the laboratory. A rapid test utilizing lateral flow immunochromatography is a good alternative to the EnzymeLinked Immunosorbent Assay (ELISA) and has high sensitivity [15]. Because of their excellent sensitivity and specificity for detecting RV in various clinical specimens, enzyme-linked immunosorbant tests are the most commonly employed [16]. For fast, precise, and sensitive genotyping of Rotaviruses, a number of molecular techniques are available, most of which are based on reverse transcription PCR [17]. Rotavirus dsRNA was isolated from feces samples and identified using RNA PAGE and RT-PCR targeting the incomplete VP7 gene [18]. Electron microscopy is labor-intensive and costly, despite its great specificity. Although RT-PCR is very sensitive and specific, it is costly, labor demanding, and requires highly skilled personnel. As a result, neither method is suited for routine diagnosis [19]. Because of no specific antiviral therapy is available, effective RV vaccines are effective to prevent morbidity and mortality [20) [21]. Internationally, oral Rotavirus vaccines available (RotaTeq and Rotarix) [22]. Rotarix (RV1 product from one strain G1P[8] which is used as a human vaccine in two doses, RotaTeq (RV5) resulting 2 from combining five strains (G1, G2, G3, G4, and G1P[8]). RotaTeq is used as a vaccine in three doses [23].

II. REVIEW

History of Rotavirus

In the second half of the twentieth century, Rotavirus was discovered in the intestinal tissue of mice and the rectum of monkeys. Similar viruses were discovered in the feces of cattle suffering from diarrhea in 1969. The first human cases were discovered in 1973 when an electron microscope was used to examine the remnants of the mucous membrane in the feces of children with diarrhea [24]. Bishop and colleagues discovered viral particles in the cytoplasm of duodenal epithelial cells of infants with non-bacterial diarrhea in 1973 [25]. Despite having no relation to influenza, Rotavirus is one of several viruses that cause diseases and is sometimes referred to as stomach flu [26]. Thomas Henry Flewett proposed the name Rotavirus in 1974. [27].

Rotavirus structure

Rotavirus is currently the most common cause of gastroenteritis in children around the world [28]. Researchers were able to display the structures of pathogenic RV particles to near-atomic precision with to advances in imaging technologies like as X-ray and electron microscope images [28, 29]. Rotavirus is a type of Reoviridae virus that has double-stranded RNA [30]. The virus has an 11-segment genome [31]. Rotavirus (RV) capsids are multi-layered icosahedral capsids that are not enveloped [32]. Rotavirus is one of the Reoviridae family and possesses double-stranded RNA [30]. The virus has genome with 11 segments [31]. Rotavirus (RV) has non-enveloped multi-layered icosahedral capsids [32]. RVs belong to the Sedoreovirinae subfamily of the Rotavirus genus, and they encode six structural proteins (VP1–VP4, VP6, and VP7) as well as six nonstructural proteins (NSP1–NSP5/NSP6). RVs are classified into ten different species (A–J). RV species A, B, C, and H (RVA, RVB, RVC, and RVH, respectively) infect both people and animals, but RV species D, E, F, G, H, I, and J (RVD, RVE, RVF, RVG, RVI, and RVJ, respectively) have only been discovered in animals [33]. Traditionally, genotypes have been categorized using a binary system based on two genes that code for outer capsid proteins, VP4 and VP7, respectively, creating G and P genotypes [34]. VP7 and VP4 make form the triple-layered particle's outermost layer. VP6 makes up the central layer of the viral particle [32]. The inner layer, also known as the inner core, is largely made up of VP2 which surrounds the 11-segment double-stranded RNA (dsRNA) genome, VP1 which is an RNA polymerase enzyme, and VP3 which is a guanylyltransferase [32, 35]

Transmission

Rotavirus is spread through the feces-to-mouth route. It attacks the cells that line the small intestine and generates an exotoxin, which causes gastroenteritis, severe diarrhea, and occasionally death due to dehydration [36]. Rotavirus is spread by contaminating one's hands with infected people's feces, which subsequently spreads to the mouth or respiratory system [26, 37]. Non-fecal routes of infection may have a role in transmission [38] despite all changes in life style like as water supply, sanitation, personal cleanliness, food quality, nutrition, and maternal education that assist to lower the overall risk of Rotavirus infections [39]. Rotavirus is a relatively stable virus that may survive for weeks or months in the environment if not cleaned [40].

Target organ of Rotavirus

Glycan bonds are found on the surface of mature intestinal lining cells, and they are thought to be a complement to the virus's appendages [33]. Detection of Rotavirus (RV) in extra intestinal regions such as the liver, kidney, and central nervous system (CNS) of children with RV gastroenteritis has been reported in other investigations, however CNS localization in RV infection appears to be unusual [41]. In animals, Rotaviruses have been found in the digestive system, glands, respiratory system, and urinary system [42]. If the mucous membrane lining the gut is severely infected, the virus may enter the bloodstream [43].

Replication

The virus invades cells by receptor-mediated endocytosis and forms an endosome, a vesicle. Proteins in the third layer (VP7 and the VP4 spike) disrupt the endosome membrane, causing a calcium concentration differential. The VP7 trimers are broken down into single protein subunits, leaving the VP2 and VP6 protein coatings surrounding the viral dsRNA, resulting in a double-layered particle (DLP) [44]. The eleven dsRNA strands are protected by the two protein shells, and the viral RNA-dependent RNA polymerase converts the double-stranded

viral genome into mRNA transcripts. The viral RNA escapes innate host immune responses, such as RNA interference, that are activated by the presence of double-stranded RNA by staying in the core [45]. Rotaviruses generate mRNA for both protein production and gene replication during infection. The viroplasm, where the RNA is copied and the DLPs are formed, accumulates the majority of Rotavirus proteins. Positive sense viral RNAs, which serve as templates for the production of viral genomic dsRNA, are protected against siRNA-induced RNase destruction in the viroplasm [46]. Viroplasm is formed around the cell nucleus as early as two hours after virus infection, and consists of viral factories thought to be made by two viral nonstructural proteins: NSP5 and NSP2. Inhibition of NSP5 by RNA interference in vitro results in a sharp decrease in Rotavirus replication. The DLPs migrate to the endoplasmic reticulum where they obtain their third, outer layer (formed by VP7 and VP4). The progeny viruses are released from the cell by lysis, as in Figure 1 [47, 48].

Immunity

Reinfections are frequent in Rotavirus illnesses. Rotavirus immunity is short-lived; recurrent symptomatic infections are prevalent in both children and adults, albeit usually with less severity. Cross protection between serotypes, on the other hand, develops after numerous infections [49]. With each infection, immunity builds, making future infections less severe; adults are rarely afflicted [27, 50]. IgM is the first immunoglobulin produced in response to an antigen. IgM is secreted as a pentamer and is composed of five units (similar to one IgG unit) and one molecule of a J chain. IgM Immunoglobulin is considered the most efficient in the interaction of antibodies with antigens, and it is important to identify viruses and bacteria because it has 10 binding sites, IgM may be useful in the diagnosis of certain infectious diseases [51]. Infection with the Rotavirus has been related to the development of autoimmune disorders in humans [43, 52]. The issue of protective immunity against Rotavirus infection is linked to the virion's complexity [53].

Epidemiology

Rotavirus illness kills 527,000 children under the age of five each year, according to the World Health Organization (WHO). Rotavirus kills 82 percent of children in the world's poorest countries [54]. According to the WHO, Rotavirus is the second leading cause of mortality in infants, behind pneumonitis, and is responsible for more than half of all instances of acute diarrhea [55]. Human Rotavirus (RV) is the most prevalent cause of severe diarrhea in children under the age of five worldwide, with the highest prevalence in poor nations [56]. the vast majority of which occur in Africa and Asia [57]. Rotavirus is the cause of over half of all acute gastroenteritis cases in all nations, and it is responsible for nearly ninety-five percent of fatalities in children under the age of five, according to World Health Organization data [58]. The harm done by Rotavirus diarrhea is not confined to humans; it may also impact poultry [59]. In low- and middle-income nations, severe diarrhea and pneumonia are among the most prevalent causes for children to be admitted to hospitals [60]. According to hospital records, the largest number of fatalities owing to Rotavirus infection occurred in Southeast Asia and India, accounting for 41% of all Rotavirus-related deaths [22]. Furthermore, the virus was found in a number of Iraqi cities, including Baghdad, Erbil, Basra, Ramadi, Babylon, and Najaf, with percentages ranging from 30% to 51%. [55]. According to reports from big hospitals in Jeddah, Rotavirus is a common cause of severe diarrhea among children in Iraq's bordering nations, including Saudi Arabia [61]. Ninety percent of human gastroenteritis cases are caused by Rotavirus A, which has been found all across the world, and children under the age of five are infected at least once in their lives [62]. The World Health Organization (WHO) has recommended that Rotavirus vaccination be used worldwide to prevent diarrhea-related mortality in children under the age of five. Since the 1990s, childhood mortality rates have fallen considerably, owing to improvements in drinking water and sanitation, as well as the prevention of nutritional deficits caused by vomiting and diarrhea [2]. Between 2000 and 2009, data from 20 countries/regions revealed that the most prevalent strains were G1P[8] (23.6 percent), G3P[8] (18.9%), G2P[4] (11.8 %), and G9P[8] (7.4%). [63]. Infections are spread through the community, although other research has found that Rotavirus is the most common cause of diarrhea in infants under the age of six months [64].

Symptoms

Compared to other types of acute gastroenteritis, Rotavirus gastroenteritis is usually accompanied with severe illness symptoms (vomiting, diarrhea, dehydration, etc.) that lead to an increase in the number of hospitalized cases [65]. Infections with the Rotavirus can cause severe dehydration, electrolyte imbalances, and acid-base imbalances [66].

Fever, nausea, and vomiting are common symptoms, followed by abdominal cramps and frequent watery diarrhea that can last 3-8 days. Children who are infected may also have a cough and a runny nose [67]. RVGE is characterized by diarrhoea and vomiting. Severe dehydration, which occurs as a result of fluid loss owing to frequent diarrhea and vomiting, can result in mortality, especially in impoverished nations [68]. Viral infections can cause problems such as heart failure, epileptic seizures, and respiratory infections, in addition to severe dehydration and electrolyte imbalance [69]. One to three days is the incubation period. Watery diarrhea is the most common symptom of RV infection in young children and animals [20]. Rotavirus infections cause a wide range of symptoms, from mild to severe. Vomiting, diarrhea, fever, and a general sense of illness, including fatigue and weakness, can all be symptoms in adult patients [70]. According to studies, the virus overlaps symptoms with other infections, making it difficult to pinpoint the source clinically [25]. Infection can cause reduced sodium, glucose, and water absorption, as well as lower levels of

intestinal lactase, alkaline phosphates, and sucrose activity, as well as isotonic diarrhea [40, 71].

Mechanisms of virus induced diarrhea

Rotavirus is resistant to digestive enzymes and stable at low pH. The viruses invade the villus of the intestine. Once within the cell, the virus replicates, resulting in cell lysis and the release of viral offspring that will infect nearby cells. This cytolysis virus causes villus atrophy, which reduces the intestine's digestion and absorption ability, and is the source of diarrhea's malabsorption component [49]. Despite the fact that most kinds of diarrhea in children are mild, acute diarrhea causes substantial volumes of liquid to be lost. Because the fluid lost at the initial sign of diarrhea is not replaced for, these droughts can result in mortality and other serious effects. Diarrhea is one of the most common symptoms of the digestive system caused by various types of pathogens such as Bacteria, Virus, and Parasites [72]. Poor absorption is one of the most common symptoms of diarrhea, as it leads to an iron deficiency when bacteria and viruses infect the intestinal villi, causing damage to the villi and sudden absorption of proteins, carbohydrates, fat, calcium, and vitamins in the ileum [73].

Diagnosis methods

The virus was first diagnosed using an electron microscope to examine its wheel-like shape as in Figure 2, but now several methods are used, including the latex agglutination technique, which identifies virus antigens in the stool and is considered a simple and quick first step, as well as the use of ELISA, which is a specialized method for its high sensitivity to detect the virus [74].The difficulty for diagnostic procedures is to come up with a quick and sensitive way to treat patients quickly. Isolation the virus is used on a small scale, but quantitative reverse transcription of the polymerase chain reaction was used for detection of the most common virus in the world [39]. There are a number of approved test kits on the market that are sensitive, specific, and detect all Rotavirus serotypes; however, some techniques rely on the interaction between antigens and antibodies that are produced after infection (immunological technique). Electron microscopy, isolation, culture methods, nucleic acid detection, and so on are some of the others. In research facilities, other techniques such as electron microscopy and polyacrylamide gel electrophoresis are utilized, and (RT-PCR) can detect and identify all species and serotypes of human Rotavirus [75]. In other studies, ELISA has been found to be more effective than electron microscopy in detecting Rotavirus antigens quickly [76]. Rotavirus identification and viral load and type determination can be done using nucleic acid amplification by Real-Time PCR or multiplex probe-based rapid RT-PCR, respectively [77]. Individual enteric viruses can now be detected using reverse transcription-polymerase chain reaction (RT-PCR) assays. These assays can detect a single virus at a time, but they can't detect infections with multiple viruses. For the detection of enteric viruses, multiplex RT-PCR (m RT-PCR) assays have been developed [59]. Multiplex RT-PCRs using a variety of primer and probe sets are commonly used to classify fecal (group A) Rotavirus strains into G- and P-types based on the genes encoding the capsid (Glycosylated) VP7 and (Protease-sensitive) VP4 proteins [53]. The virus was first diagnosed using an electron microscope to examine its wheel-like shape, but now several methods are used, including the latex agglutination technique, which identifies virus antigens in the stool and is considered a simple and quick first step, as well as the use of ELISA, which is a specialized method for its high sensitivity to detect the virus [74]. The difficulty for diagnostic procedures is to come up with a quick and sensitive way to treat patients quickly. Isolation the virus is used on a small scale, but quantitative reverse transcription of the polymerase chain reaction was used for detection of the most common virus in the world [39]. There are a number of approved test kits on the market that are sensitive, specific, and detect all Rotavirus serotypes; however, some techniques rely on the interaction between antigens and antibodies that are produced after infection (immunological technique). Electron microscopy, isolation, culture methods, nucleic acid detection, and so on are some of the others. In research facilities, other techniques such as electron microscopy and polyacrylamide gel electrophoresis are utilized, and (RT-PCR) can detect and identify all species and serotypes of human Rotavirus [74]. In other studies, ELISA has been found to be more effective than electron microscopy in detecting Rotavirus antigens quickly [76]. Rotavirus identification and viral load and type determination can be done using nucleic acid amplification by Real-Time PCR or multiplex probe-based rapid RT-PCR, respectively [77]. Individual enteric viruses can now be detected using reverse transcription-polymerase chain reaction (RT-PCR) assays. These assays can detect a single virus at a time, but they can't detect infections with multiple viruses. For the detection of enteric viruses, multiplex RT-PCR (m RT-PCR) assays have been developed [59]. Multiplex RT-PCRs using a variety of primer and probe sets are commonly used to classify fecal (group A) Rotavirus strains into G- and P-types based on the genes encoding the capsid (Glycosylated) VP7 and (Protease-sensitive) VP4 proteins [53].

Treatment

Children with Rotavirus diarrhea have been treated with oral fluids and sustained feeding. The World Health Organization (W.H.O.) and the United Nations Children's Fund (UNICEF) both recommend using zinc supplements to help lessen the intensity and length of diarrhea [78]. Effective RV vaccinations are critical to preventing morbidity and death because no particular antiviral treatment is available. The only way to treat RV infection is to replenish fluids and electrolytes [20]. Vaccines are the most effective public health intervention for Rotavirus illness prevention and control [38]. A Rotavirus gastroenteritis vaccine was initially approved in August 1998; however, it was withdrawn in 1999 due to a connection with intussusception. In 2006 and 2008, second-generation vaccinations were approved [38]. Rotashield (Wyeth-Lederle) was an oral Rotavirus

vaccination that was withdrawn from the market around the turn of the century due to the emergence of intussusception symptoms. Rotarix (GlaxoSmithKline Biologicals, Rixensart, Belgium) and RotaTeq (RotaTeq, Merck and Co., Whitehouse Station, New Jersey) are the two currently licensed oral Rotavirus vaccines [21]. RotaTeq (RV5) is a pentavalent vaccination combining five human-bovine re-assortment strains and is delivered in a three-dose schedule. Rotarix (RV1) is a monovalent human vaccine originating from a G1P [8] strain and is administered in a two-dose regimen (G1, G2, G3, G4, and P[8]). RV1 is given at two and four months of age in the United States, while RV5 is given at two, four, and six months of age [23]. Despite evidence that RVA vaccinations give substantial protection against hospitalizations, the acute gastroenteritis morbidity associated with RVA infections remains high globally, according to the World Health Organization (WHO) [79]. The Iraqi Ministry of Health put the Rotavirus vaccine in the national vaccination schedule in January 2012 in all health care centers to prevent children against the most severe causes of diarrhea [55]. By the end of 2014, more than 70 nations have included the Rotavirus vaccine in their standard childhood vaccination regimens [80]. By September 2016, 86 nations throughout the globe have established National Immunology Programs (NIPs) [81]. As of August 2018, Rotavirus vaccines were available in 96 countries [82].

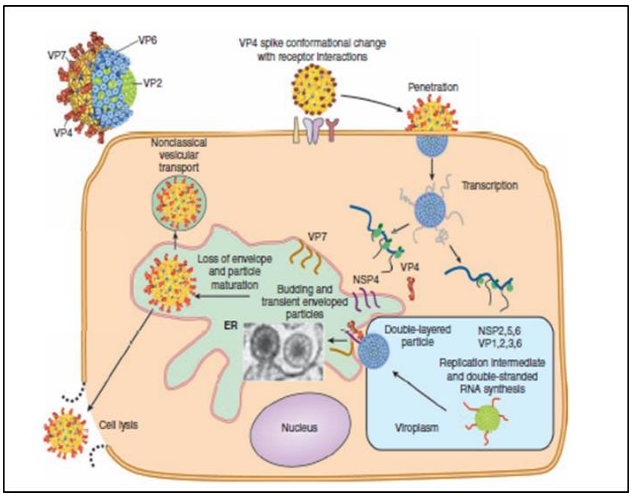

Figure.1: Overview of the Rotavirus replication cycle [51].

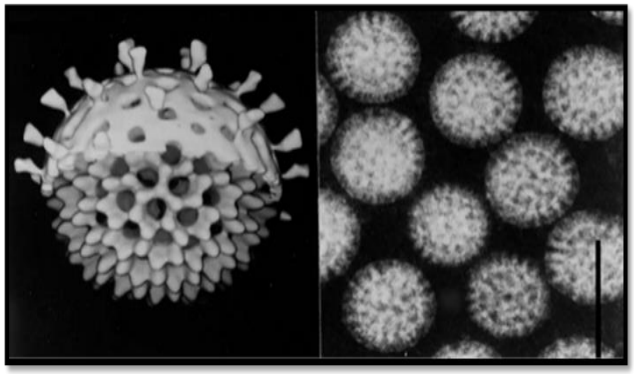

Figure.2: Rotavirus viewed by electron microscopy [83].

III. CONCLUSION AND FUTURE SCOPE

Rotavirus is one of the most important and most important viruses that cause gastroenteritis in children as well as in adults, but the maturation of the immune system in adults does not show any clinical signs, unlike children, in addition to the lack of hygiene and sterilization in the stages before and after weaning children. This virus spreads in cold seasons and during rainy times, but in tropical regions it spreads in all seasons of the year. Hand and mouth contamination is the main method of transmission between children, especially in nurseries and closed areas. The RNA of this virus is characterized by several pieces (segmented), so there is a chance for reassortment between the pieces of the RNA in animals and humans. The virus can be diagnosed with several tests. The rapid test is cheaper and faster compared to other tests, but sometimes it gives a false positive. There is no specific treatment for this virus, but doctors rely on treating clinical symptoms such as fever and diarrhea. Fortunately, there are vaccines for this virus and they are available in many countries around the world**.**

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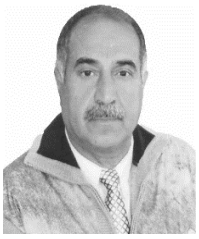

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