e-ISSN: 3025-6224

Scientific Journal of Pediatrics (SJPed)

Journal website: https://phlox.or.id/index.php/sjped

Study of Pathophysiological Aspects of Acute Diarrhea Due to Rotavirus Infection

Ardina Permatasari[1*], Muhammad Ilhamsyah[1]

[1]Stikes Suaka Insan, Banjarmasin, Indonesia

ARTICLE INFO

Keywords:
Acute diarrhea
Inflammation
Infection
Rotavirus

***Corresponding author:**
Ardina Permatasari

E-mail address:
ardina.psari@gmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.59345/sjped.v1i2.67

A B S T R A C T

Rotavirus is a highly contagious virus that enters the body through the consumption of food or water contaminated by the feces of an infected individual. After entering the body, rotavirus infects the epithelial cells lining the small intestine, which is the main site for the absorption of nutrients and water. Rotavirus infects intestinal epithelial cells by attaching to the cell surface and entering the cell. This triggers changes in the epithelial cells, including changes in the receptors present on the surface of the cells. Viruses damage epithelial cells by remodeling the cell's cytoskeleton. This causes epithelial cells to lose their ability to absorb nutrients and water properly. Over time, damage to these epithelial cells causes increased intestinal leakage, allowing fluid to enter the intestines faster than it can be absorbed. The literature search process was carried out on various databases (PubMed, Web of Sciences, EMBASE, Cochrane Libraries, and Google Scholar) regarding studies of the pathophysiology of acute diarrhea due to Rotavirus infection. This study follows the preferred reporting items for systematic reviews and meta-analysis (PRISMA) recommendations. Rotavirus causes damage to intestinal epithelial cells by remodeling the cell cytoskeleton. This results in epithelial cells losing their ability to absorb nutrients and water properly. Rotavirus infection triggers inflammation in the intestines. Infected cells release inflammatory mediators, such as cytokines, which trigger response inflammation. This inflammation causes intestinal irritation, increased bowel movements, and increased mucus production. Fluid loss through diarrhea is a major consequence of rotavirus infection. Babies and children, especially, are especially susceptible to serious dehydration. Dehydration can be life-threatening if not treated quickly and efficiently.

1. Introduction

Acute diarrhea caused by rotavirus infection is a significant global health problem, especially in children throughout the world. Rotavirus is a common cause of diarrhea in children, and this infection can have serious consequences, especially if it is not treated quickly. To fully understand the impact and causes of acute diarrhea due to rotavirus, it is necessary to elucidate the pathophysiology or mechanism of its spread. Rotavirus diarrhea is not just a common gastrointestinal symptom; it is the result of a viral infection that damages intestinal cells, disrupts the absorption of nutrients and water, and produces symptoms such as watery diarrhea, vomiting, and fever. In addition, this infection can also cause serious dehydration if not treated properly.[1-5]

Rotavirus is a highly contagious virus that enters the body through the consumption of food or water contaminated by the feces of an infected individual. After entering the body, rotavirus infects the epithelial cells lining the small intestine, which is the main site for the absorption of nutrients and water. Rotavirus infects intestinal epithelial cells by attaching to the cell surface and entering the cell. This triggers changes in the epithelial cells, including changes in the receptors present on the surface of the cells. Viruses damage

epithelial cells by remodeling the cell's cytoskeleton. This causes epithelial cells to lose their ability to absorb nutrients and water properly. Over time, damage to these epithelial cells causes increased intestinal leakage, allowing fluid to enter the intestines faster than it can be absorbed. Rotavirus infection causes inflammation in the intestines. Infected cells release inflammatory mediators, including cytokines, which trigger response inflammation. This inflammation can also result in increased bowel movements and increased mucus production, all of which lead to symptoms of diarrhea. Fluid loss through diarrhea is the primary outcome of rotavirus infection. Infants and children, in particular, are particularly susceptible to serious dehydration from rotavirus diarrhea, which can cause weakness, decreased skin turgor, sunken eyes, and, if not treated quickly, can become a life-threatening condition. Recognizing the pathophysiology of acute rotavirus diarrhea is an important step in understanding why this infection can cause serious complications.[6-10]

2. Methods

The literature search process was carried out on various databases (PubMed, Web of Sciences, EMBASE, Cochrane Libraries, and Google Scholar) regarding the study of the pathophysiology of acute diarrhea due to Rotavirus infection. The search was performed using the terms: (1) "infection" OR "rotavirus" OR "diarrhea" OR "pathophysiology" AND (2) "acute diarrhea" OR "rotavirus." The literature is limited to clinical studies and published in English. The literature selection criteria are articles published in the form of original articles about the study of the pathophysiology of acute diarrhea due to Rotavirus infection. Studies were conducted in the timeframe from 2013-2023, and the main outcome was a study of the pathophysiology of acute diarrhea due to Rotavirus infection. Meanwhile, the exclusion criteria were studies that were not related to the study of the pathophysiology of acute diarrhea due to Rotavirus infection, the absence of a control group, and duplication of publications. This study follows the preferred reporting items for systematic reviews and meta-analysis (PRISMA) recommendations.

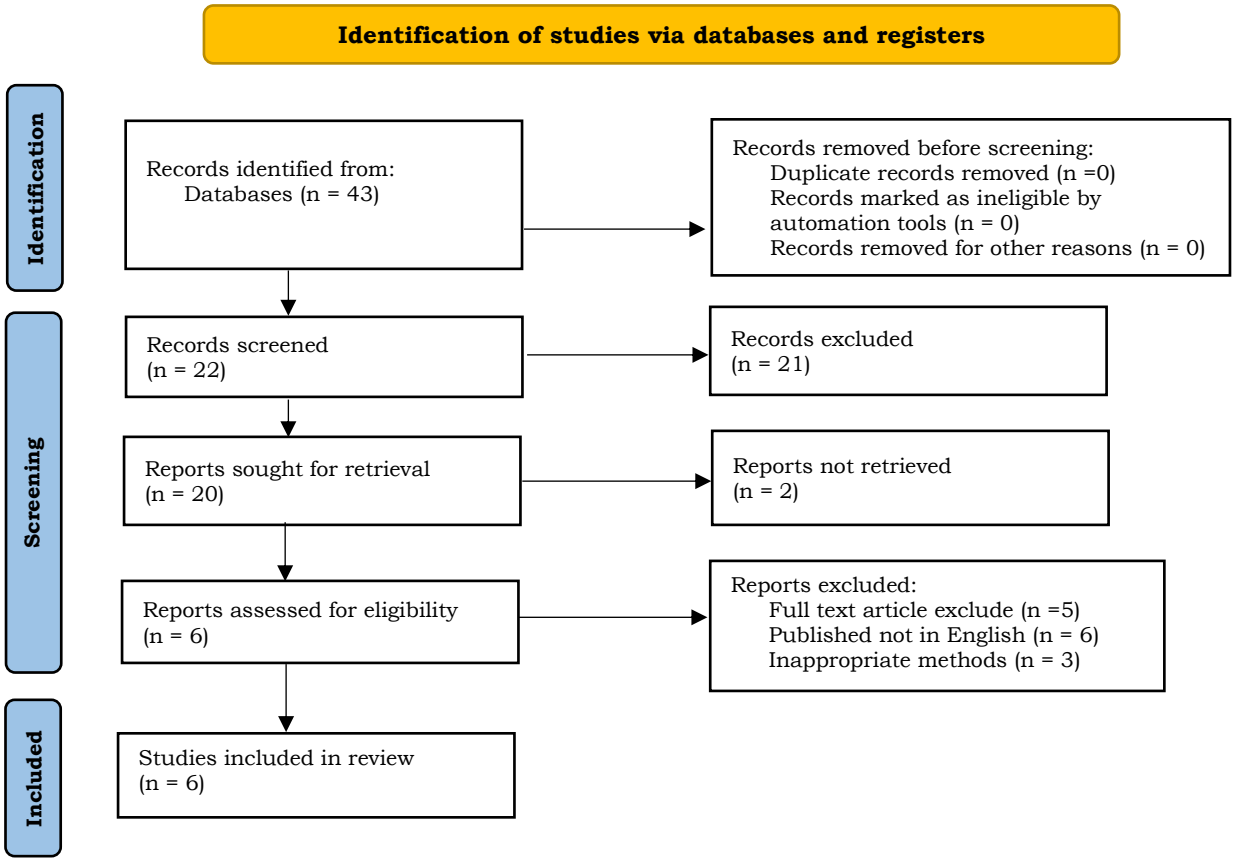

Figure 1. PRISMA flowchart.

3. Results and Discussion

Intestinal epithelial cell infection

Rotavirus infects intestinal epithelial cells by attaching to the cell surface and then entering the cell. It is the initial stage of rotavirus infection that is of great importance in the pathophysiology of this disease. When rotavirus attaches to the surface of intestinal epithelial cells, it interacts with special receptors on the surface of these cells. These interactions trigger changes in epithelial cells, including changes in receptors and internal cell processes that allow viruses to enter epithelial cells. After entering the epithelial cells, the rotavirus begins to replicate itself and damage these cells, which ultimately disrupts the absorption of nutrients and water and triggers symptoms of diarrhea. This is one of the key stages in the pathophysiology of acute diarrhea due to rotavirus, which causes the inability of the intestine to function normally, resulting in severe diarrhea and increased intestinal leakage.[11,12]

When rotavirus attaches to the surface of intestinal epithelial cells and enters those cells, it triggers a series of important changes in the epithelial cells, including changes in the receptors present on the cell surface. Rotavirus uses these receptors as an entry point into cells and to infect cells more effectively. Changes in intestinal epithelial cells caused by rotavirus are one of the key mechanisms in the pathophysiology of acute diarrhea due to rotavirus infection. This results in damage to the epithelial cells, which interferes with the normal absorption of nutrients and water and triggers symptoms of diarrhea and inflammation in the intestines. Understanding the role of these changes in epithelial cells is an important step in identifying ways to prevent and treat the impact of rotavirus infection on the human body.[13,14]

Damage to epithelial cells

When rotavirus damages epithelial cells by remodeling the cell's cytoskeleton, it disrupts the integrity of those cells so that they lose their ability to absorb nutrients and water efficiently. With a damaged cell cytoskeleton, intestinal epithelial cells cannot absorb nutrients properly. This can cause malabsorption of food, which in turn can result in nutritional deficiencies and stunted growth, especially in vulnerable children. Damage to epithelial cells also results in increased intestinal leakage. This means that fluids and electrolytes can easily pass into the intestines faster than they can be absorbed, which is one of the main causes of watery diarrhea. Significant fluid loss through rotavirus-related diarrhea can quickly lead to dehydration. Children, especially babies, are the group most vulnerable to dehydration due to diarrhea. Dehydration that is not treated quickly can be very dangerous and life-threatening. This explanation highlights how damage to intestinal epithelial cells is one of the key mechanisms causing the symptoms and serious consequences of acute rotavirus diarrhea. This pathophysiology also underscores the importance of effective management to replace fluid losses and restore electrolyte balance in patients with rotavirus infection.[15,16]

Inflammation and the immune response

Rotavirus infection triggers inflammation within the intestines, which in turn plays an important role in the development of diarrheal symptoms. Inflammation in response to the body's natural resistance to infection. In the case of rotavirus infection, infected intestinal epithelial cells release inflammatory mediators, such as cytokines. This leads to local inflammation within the gut, which is the body's attempt to fight the virus. In addition to local inflammation, the body also triggers a systemic immune response. The immune system attempts to eliminate the rotavirus virus by producing antibodies and other immune cells. Inflammation and the body's immune response result in several important consequences. Increased inflammation within the gut causes irritation and increased bowel movements, which contributes to diarrhea symptoms. In addition, mucus production in the intestines increases as part of the body's protective response. All of this together causes symptoms of diarrhea that often last for quite a long time during rotavirus infection. Although inflammation can cause uncomfortable symptoms, it is also an important part of the body's response to fighting rotavirus infection. Inflammation helps the body destroy viruses and activates the immune system

to provide long-term protection. Understanding the role of inflammation and the immune response in the pathophysiology of acute rotavirus diarrhea helps explain why symptoms such as watery diarrhea and flatulence frequently occur during this infection. Additionally, it also emphasizes the importance of inflammation management and body protection when treating patients infected with rotavirus.[17,18]

Dehydration

Dehydration is one of the serious consequences of acute rotavirus diarrhea, and can have life-threatening consequences if not treated quickly and efficiently. Symptoms of dehydration can include weakness, decreased skin turgor (skin that appears loose when pulled and released), sunken eyes, dry mouth, and decreased urine output. Children who are dehydrated often become very fussy and inactive. Severe dehydration, especially in babies and young children, can be life-threatening. Significant fluid loss can disrupt vital organ function and blood circulation. Therefore, detection and treatment of dehydration in infants and children is a top priority in treating acute diarrhea due to rotavirus. Treatment of dehydration usually involves administering intravenous or oral fluids to replace fluids lost through diarrhea. The use of an electrolyte-rich oral rehydration solution can help restore fluid and electrolyte balance quickly. Prevention of dehydration through management of rotavirus diarrhea is essential. Helping children to drink more fluids and providing foods appropriate for their age can help reduce the risk of dehydration during an infection.[19,20]

4. Conclusion

Rotavirus causes damage to intestinal epithelial cells by remodeling the cell cytoskeleton. This results in epithelial cells losing their ability to absorb nutrients and water properly. Rotavirus infection triggers inflammation in the intestines. Infected cells release inflammatory mediators, such as cytokines, which trigger response inflammation. This inflammation causes intestinal irritation, increased bowel movements, and increased mucus production. Fluid loss through diarrhea is a major consequence of rotavirus infection. Babies and children, especially, are especially susceptible to serious dehydration. Dehydration can be life-threatening if not treated quickly and efficiently.

5. References

1. Parashar UD, Burton A, Lanata C, Boschi-Pinto C, Shibuya K, Steele D, et al. Global mortality associated with rotavirus disease among children in 2004. Journal of Infectious Diseases. 2019; 200(2): S9-15.
2. Estes MK, Greenberg HB. Rotaviruses. In: Knipe DM, Howley PM, editors. Fields Virology. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2019.
3. Ramig RF. Pathogenesis of intestinal and systemic rotavirus infection. Journal of Virology. 2020; 78(18): 10213-20.
4. Gerna G, Sarasini A, Baldanti F, Marchi A, Zavattoni M, Furione M. Prospective study of the natural history of rotavirus infection in neonates and young infants. Journal of Medical Virology. 2021; 50(3): 261-7.
5. Bishop RF, Davidson GP, Holmes IH, Ruck BJ. Virus particles in epithelial cells of duodenal mucosa from children with viral gastroenteritis. The Lancet. 2020; 301(7811): 1281-3.
6. Coulson BS. Unicomb LE, Pitson GA, Bishop RF. Simple and specific enzyme immunoassay using monoclonal antibody for serotype 2 (P2A) rotavirus and its use in diagnosis of neonatal infection. Journal of Clinical Microbiology. 2019; 25(2): 205-10.
7. Kollaritsch H, Kundi M, Giaquinto C, Paulke-Korinek M. Rotavirus vaccines: a story of success. Clinical Microbiology and Infection. 2019; 21(5): 416-7.
8. Clark B, McKendrick M. A review of viral gastroenteritis. Current Opinion in Infectious Diseases. 2020; 17(5): 461-9.
9. Tate JE, Burton AH, Boschi-Pinto C, Parashar UD; World Health Organization–Coordinated Global Rotavirus Surveillance Network. Global, regional, and national estimates of rotavirus

mortality in children <5 years of age, 2000–2013. Clinical Infectious Diseases. 2019; 62(suppl_2): S96-105.
10. Nataro JP, Mai V, Johnson J, Blackwelder WC, Heimer R, Burghardt RC, et al. Diarrheagenic Escherichia coli infection in Baltimore, Maryland, and New Haven, Connecticut. Journal of Infectious Diseases. 2019; 193(2): 147-55.
11. Steele D, Steele AD, van der Linde F. Nephrotoxicity in children with acute gastroenteritis treated with furosemide. Pediatric Nephrology. 2020; 14(4): 286-90.
12. Finkbeiner SR, Zeng X, Utama B, Atmar RL, Shroyer NF, Estes MK. Stem cell-derived human intestinal organoids as an infection model for rotaviruses. mBio. 2022; 3(4): e00159-12.
13. Crawford SE, Ramani S, Tate JE, Parashar UD, Svensson L, Hagbom M, et al. Rotavirus infection. Nature Reviews Disease Primers. 2019; 3(1): 1-16.
14. Sai L, Sun J, Shao L, Chen S, Liu H, Ma L. Epidemiology and clinical features of rotavirus and norovirus infection among children in Ji'nan, China. Virology Journal. 2020; 10(1): 302.
15. Ansari SA, Springthorpe VS, Sattar SA, Rivard S, Rahman M. Potential role of hands in the spread of respiratory viral infections: studies with human parainfluenza virus 3 and rhinovirus 14. Journal of Clinical Microbiology. 2021; 29(10): 2115-9.
16. Fischer TK, Valentiner-Branth P, Steinsland H, Perch M, Scheutz F, Molbak K, et al. Protective immunity after natural rotavirus infection: a community cohort study of newborn children in Guinea-Bissau, West Africa. The Journal of Infectious Diseases. 2022; 186(2): 593-7.
17. Bines JE, Patel M, Parashar U. Assessment of postlicensure safety of rotavirus vaccines, with emphasis on intussusception. The Journal of Infectious Diseases. 2019; 200(2): S282-90.
18. Barman P, Ghosh S, Banerjee A, Chawla-Sarkar M. Rotavirus NSP1 inhibits interferon induced non-canonical NFκB activation by interacting with TNF receptor associated factor 2. Virology. 2019; 505: 193-204.
19. Shaw RD. Salivary antibodies directed against the trypsin cleavage products of rotavirus SA-11 in human volunteers. Journal of Clinical Microbiology. 2021; 25(1): 24-8.
20. Clark B, McKendrick M. A review of viral gastroenteritis. Current Opinion in Infectious Diseases. 2020; 17(5): 461-9.