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Acute Diarrhoea in Children Under Five: Unraveling the Causes, Consequences, and Clinical Management

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A B S T R A C T

Diarrhea is one of the leading causes of morbidity and mortality in children under five worldwide. According to WHO, diarrhea is defined as three or more loose or watery stools per day, and is classified as acute, persistent, or chronic based on its duration. In Indonesia, the prevalence of diarrhea in children under five reaches 12.3%, with the highest rates in the 1–4 year age group. The main causes of diarrhea are gastrointestinal infections by viruses, bacteria, and parasites. Rotavirus is the most common cause, followed by Escherichia coli, Cryptosporidium, Entamoeba histolytica, and Giardia lamblia. The pathophysiology of diarrhea is divided into two main mechanisms: secretory and osmotic. Secretory diarrhea occurs due to increased secretion of fluid and electrolytes by the intestinal mucosa, while osmotic diarrhea is caused by impaired absorption of solutes. Common complications include dehydration, electrolyte imbalances such as hyponatremia, hypokalemia, and hyperchloremia, and impaired kidney function due to excessive fluid loss. In severe dehydration, metabolic acidosis can occur due to decreased tissue perfusion and lactic acid accumulation. The management of diarrhea in children refers to WHO guidelines, which include oral or intravenous rehydration therapy according to the degree of dehydration, zinc supplementation for 10–14 days, and regulation of fluid and electrolyte intake. Understanding the etiology, pathophysiology, complications, and appropriate management is crucial in preventing diarrhea-related deaths in children.

Keywords: Acute diarrhoea, Dehydration, Electrolyte Imbalance, Kidney Function, Acid Base Balance

1. Introduction

According to WHO, diarrhea is defined as three or more loose or watery stools per day (or more frequent bowel movements than usual for an individual). Based on duration, diarrhea is divided into chronic, persistent, and acute diarrhea (Nemeth and Pfleghaar, 2022). Acute diarrhea is a type of diarrhea with a duration of less than 14 days. Chronic and persistent diarrhea are types of diarrhea with a duration of more than 14 days. In Indonesia, the term persistent diarrhea is used to refer to infectious etiology, while the term chronic diarrhea refers to non-infectious etiology (Wolayan et al., 2020). Diarrhea is a symptom of intestinal infection, which can be caused by various organisms such as bacteria, viruses, and parasites. These organisms can spread and contaminate food or drinking water (WHO, 2023). Diarrhea is the second leading cause of death in children under 5 years old worldwide. The prevalence of diarrhea is 9.8%, with the highest age group being 1-4 years old at 11.5% and infants at 9% (Yenny Apriani, Sastra Putri and Widiasari, 2022). The 2018 Basic Health Research states that the prevalence of diarrhea for all age groups is 8% and the prevalence rate for children under 5 years old is 12.3%. Meanwhile, in infants, the prevalence of diarrhea is 10.6%. In the 2018 Sample Registration System, diarrhea remained one of the main causes of death in neonates at 7% and in infants aged 28 days at 6%. Diarrhea is a cause of death in post-neonatal children at 14% based on data from Komdat Kemenkes. Based on the 2020 Indonesian Health Profile data, infectious diseases, especially diarrhea, are a cause of death in children aged 29 days - 11 months. In 2020, diarrhea was still responsible for 14.5% of deaths, with 4.55% of diarrhea deaths occurring in children under 5 years old.

2. Anatomy and Histology

The digestive system is an organ that functions to absorb substances from outside the body, which are then circulated to all body cells through blood vessels. Anatomically, the digestive system is divided into two parts: hollow organs, including the tract from the oral cavity to the anus, and accessory organs, such as the pancreas, liver, gallbladder, and salivary glands. Generally, hollow organs consist of four layers: the mucosa, submucosa, muscularis, and serosa. The digestive system comprises organs responsible for absorbing nutrients and other substances from the external environment, which are subsequently distributed to body cells through the circulatory system. Structurally, it is divided into two main components: hollow organs, forming a continuous tract from the oral cavity to the anus, and accessory organs, including the pancreas, liver, gallbladder, and salivary glands. The walls of hollow organs generally consist of four principal layers—mucosa, submucosa, muscularis externa, and serosa. The mucosal layer, which serves as a barrier between the external environment and internal tissues, is composed of epithelial tissue, the lamina propria, and the muscularis mucosae. The epithelium facilitates protection, secretion, and absorption, while the lamina propria, containing blood vessels, lymphatics, nerves, and glands, provides structural and vascular support. The muscularis mucosae, made up of smooth muscle fibers, aids in local movements and secretion distribution. Beneath it lies the submucosal layer, consisting of fibroelastic connective tissue rich in blood and lymphatic vessels, nerves, and parasympathetic ganglia, with submucosal glands present in the esophagus and duodenum. The muscularis externa, comprising two layers of smooth muscle separated by the myenteric (Auerbach's) plexus, regulates peristaltic motion and maintains gastrointestinal tone. The outermost serous layer, composed of dense irregular connective tissue, serves as a protective covering and transitions into the adventitia when fused with adjacent connective tissues (Sorenson and Brelje, 2008).

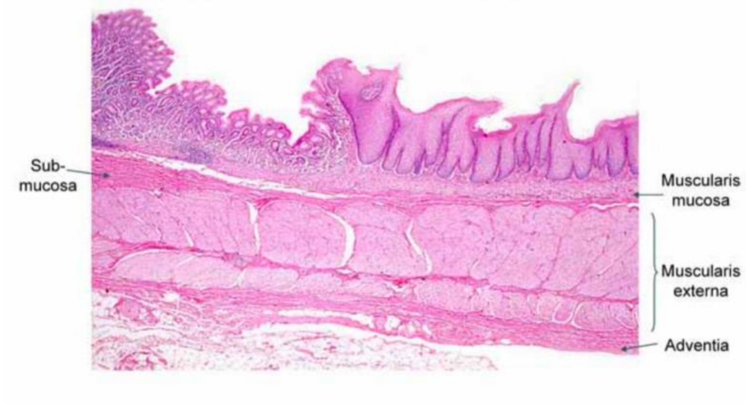

Figure 1. Histology of the mucosal, submucosal, muscularis, and serosa layers (Sorenson and Brelje, 2008)

3. Etiology

Acute diarrhea is generally an infectious gastrointestinal disorder caused by various organisms such as viruses, bacteria, and parasites. Viruses cause 70% of diarrhea cases in children in both developing and developed countries. Rotavirus is the viral pathogen responsible for 37% of diarrhea-related deaths in children under 5 years old (Prescilla, 2023). Bacterial infections cause about 10-20% of diarrhea cases (Kumari, Kumar and Mishra, 2023). Enterotoxigenic E. coli (ETEC) is the main bacterial cause of diarrhea in children in developing countries, followed by Campylobacter, Salmonella, and Shigella (Prescilla, 2023).Entamoeba histolytica and Giardia lamblia are the main parasitic causes of acute diarrhea (Worku et al., 2023). Rotavirus and E. coli are the most common pathogens among children of all age groups, while parasitic pathogens such as Cryptosporidium, Giardia, and Entamoeba spp. are prevalent in children aged 3–5 years (WHO, 2024).

4. Pathophysiology

4.1 Pathophysiology of Rotavirus acute diarrhea

Acute diarrhea due to rotavirus occurs when the NSP4 protein replicates in enterocyte cells, causing villi shortening and loss, microvilli loss, mononuclear cell infiltration, endoplasmic reticulum expansion, and mitochondrial swelling in enterocyte cells, followed by stimulation of enterochromaffin cells, leading to the release of 5-Hydroxytryptamine (5-HT) or serotonin, a neurotransmitter that regulates gastrointestinal motility and induces nausea and vomiting (Pawłuszkiewicz et al., 2025). The release of 5-HT3 activates 5-HT3 receptors in the myenteric plexus. The myenteric plexus increases intestinal motility and stimulates the release of Vasoactive Intestinal Peptide (VIP) through the submucosal plexus at nerve endings adjacent to crypt cells. VIP release increases cellular cAMP levels, resulting in the secretion of sodium chloride (NaCl) and water into the intestinal lumen. Excessive secretion of sodium chloride and water leads to hyponatremia and dehydration (Crawford et al., 2017).

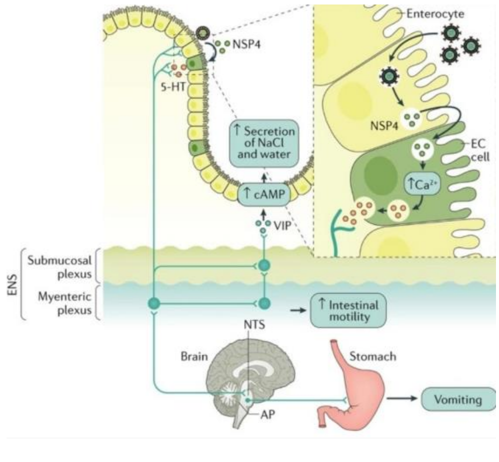

Fig. 2 Pathophysiology of Rotavirus infection diarrhea (Crawford et al., 2017)

4.2 Pathophysiology of Enterotoxin E.coli acute diarrhea

Enterotoxigenic Escherichia coli (ETEC) are Gram-negative bacilli indistinguishable from commensal E. coli in the human gut. ETEC pathogenesis involves intestinal colonization followed by enterotoxin production. Various ETEC strains isolated in clinical practice have combinations of exotoxins that contribute to their overall virulence. The most significant are two types of enterotoxins, heat-labile toxin (LT) and heat-stable toxin (ST). These toxins disrupt normal intestinal function, causing massive fluid secretion, which results in watery diarrhea. Heat-stable toxin (STa) binds to guanylate cyclase C (GC-C) receptors on enterocytes, converting GTP to cyclic GMP (cGMP). Increased cGMP activates protein kinase II (PKGII), which phosphorylates CFTR, increasing chloride secretion. Simultaneously, cGMP inhibits phosphodiesterase 3 (PDE3), increasing cAMP levels and further promoting chloride efflux. The resulting electrolyte imbalance drives fluid secretion into the intestinal lumen, causing watery diarrhea (Josune Salvador-Erro, Pastor and Gamazo, 2025)

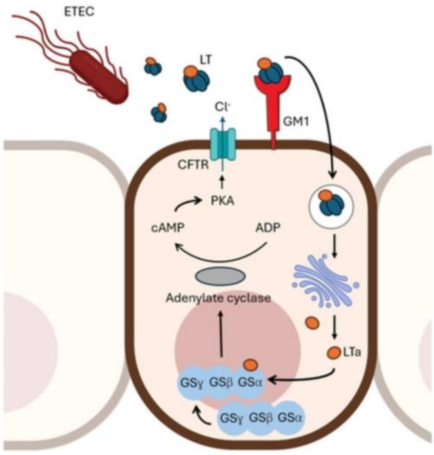

Fig. 3 Pathophysiology of Heat-stable toxin A

(Josune Salvador-Erro, Pastor and Gamazo, 2025)

After ETEC colonization and toxin release, heat-stable toxin (STb) initiates its action by binding to sulfated, acidic glycosphingolipid receptors on intestinal epithelial cells. The toxin is then internalized, activating pertussis toxin-sensitive GTP-binding protein (Gαi3), which triggers calcium influx through ligand-gated channels. Increased intracellular calcium forms a complex with calmodulin, activating calmodulin-dependent protein kinase II (CAMKII). This activation opens calcium-activated chloride channels (CaCC), inducing chloride efflux into the intestinal lumen. Simultaneously, protein kinase C (PKC) and cystic fibrosis transmembrane conductance regulator (CFTR) are stimulated, promoting the secretion of chloride, bicarbonate, water, and other electrolytes. PKC also suppresses sodium absorption through NHE3 channels, increasing luminal sodium concentration. Additionally, the calcium surge activates phospholipase A2 and C, releasing arachidonic acid, which is converted into prostaglandin E2 (PGE2) and serotonin (5-HT). These mediators further enhance intestinal fluid and electrolyte secretion through secondary pathways (Butt, Saleh and Gagnon, 2020)

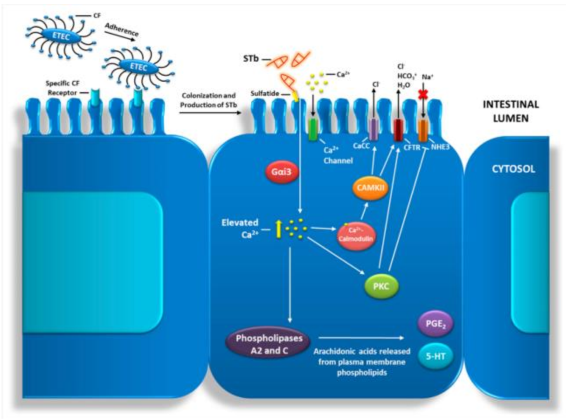

Fig. 4 Pathophysiology of Heat-stable toxin B

(Butt, Saleh and Gagnon, 2020)

Heat Labile Toxin (LT) from ETEC exerts its pathogenic effects by binding to GM1 ganglioside receptors on the apical surface of enterocytes via its B subunit, facilitating toxin internalization. Once inside the cell, the enzymatically active A subunit (LTA) is released into the cytoplasm, where it catalyzes the ADP-ribosylation of the stimulatory Gsα protein. This modification activates adenylate cyclase, leading to the accumulation of intracellular cyclic AMP (cAMP). Increased cAMP subsequently activates protein kinase A (PKA), which phosphorylates the cystic fibrosis transmembrane conductance regulator (CFTR), stimulating chloride secretion into the intestinal lumen. The resulting efflux of chloride and water disrupts osmotic balance, leading to the copious watery diarrhea characteristic of LT-mediated ETEC infections (Josune Salvador-Erro, Pastor and Gamazo, 2025)

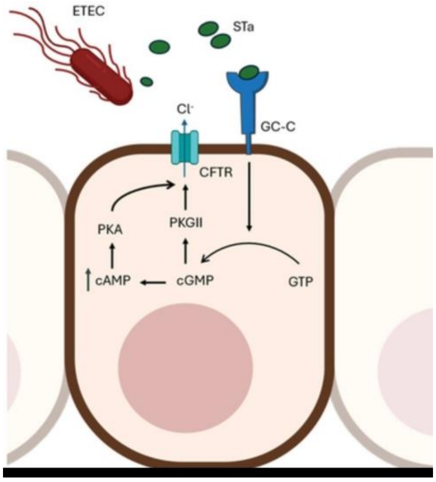

Fig. 5 Pathophysiology of Heat-labile toxin

(Josune Salvador-Erro, Pastor and Gamazo, 2025)

4.3 Pathophysiology of acute Cryptosporidium diarrhea

Cryptosporidium exists in the environment as oocysts containing four infectious sporozoites (Gerace, Presti and Biondo, 2019). Upon ingestion, oocysts reach the small intestine, where excystation releases sporozoites that invade epithelial cells and multiply asexually within parasitophorous vacuoles (Apsari, 2020). This process yields thick-walled oocysts that are shed in feces and thin-walled oocysts that cause auto-infection, the latter contributing to severe chronic disease in immunosuppressed hosts. Pathogenesis involves lamina propria inflammation, epithelial damage with villous atrophy, and malabsorption due to mucosal injury. The parasite also evades host immunity by inhibiting apoptosis of infected cells, allowing for persistent intestinal infection (Janssen and Snowden, 2020).

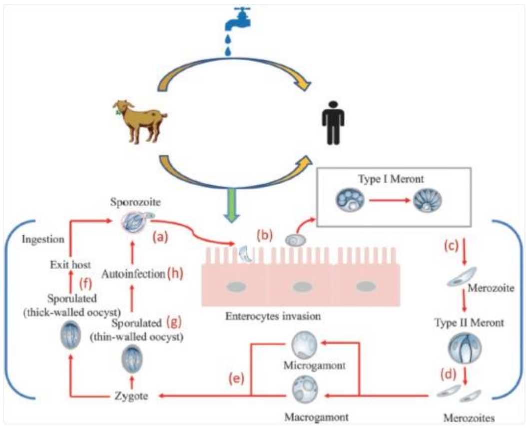

Fig. 6 Pathophysiology of Cryptosporidium spp. (Gerace, Presti and Biondo, 2019)

4.4 Pathophysiology of acute Giardia lamblia diarrhea

Giardia has a simple two-stage life cycle: trophozoites, which actively multiply in the small intestine, and cysts, the dormant form shed into the environment. Infection occurs through the consumption of food or water contaminated with cysts. In the stomach and duodenum, excystation releases trophozoites that rapidly replicate in the small intestine. As they move into the large intestine, encystation occurs, and cysts are shed in feces to restart the cycle. Pathogenesis involves trophozoite attachment to the intestinal epithelium via a ventral disc, disrupting tight junctions and brush border enzymes. The release of thiol proteinases and lectins further damages cells, increasing permeability, impairing nutrient absorption, and causing diarrhea (Dunn and Juergens, 2022).

The necessity of trophozoite adhesion in Giardia lamblia-induced epithelial damage is still under investigation, as some studies suggest that the parasite's excretory-secretory products alone can injure intestinal cells. These secreted molecules contribute to Giardia pathogenesis by inducing brush border microvilli shortening, disrupting epithelial junctional complexes, and activating signaling pathways that regulate cell cycle arrest and apoptosis. In children, such epithelial injury can lead to intestinal dysfunction, contributing to growth faltering and diarrheal disease (Gutiérrez and Bartelt, 2024).

4.5 Pathophysiology of acute Entamoeba histolytica diarrhea

Entamoeba histolytica has two forms: the environmentally resistant cyst and the active, invasive trophozoite. Infection begins when cysts are ingested and transform into trophozoites in the intestine, where they invade and destroy the mucosal epithelium. Pathogenesis primarily involves trophozoite adherence to colonic epithelial cells via a galactose–N-acetylgalactosamine lectin, leading to host cell death through cytolysis and apoptosis.This interaction triggers the release of interleukin-1α and pro–interleukin-1β, with the latter activated by amoebic cysteine proteinases. Active IL-1β stimulates NF-κB signaling in nearby cells, promoting the production of cytokines and inflammatory mediators such as COX-2, IL-1, and IL-8. These mediators recruit neutrophils and macrophages to the infection site, where trophozoites can directly damage neutrophils, amplifying epithelial injury and inflammation. Macrophage-derived factors, including TNFα, further exacerbate tissue destruction and contribute to the inflammatory pathology characteristic of amoebiasis (Chou and Austin, 2023).

4.6 Pathophysiology of acute Osmotic diarrhea

Osmotic diarrhea arises from the accumulation of poorly absorbed and osmotically active solutes in the intestinal lumen, which inhibits water and electrolyte absorption. Common causes include the use of certain laxatives, such as lactulose and magnesium citrate, or maldigestion of food substances like milk. This condition is typically identified by an increased osmotic load in the stool and is distinguished by its resolution during a fasting period (Woods, 2011). Patients with lactase deficiency commonly experience bloating, flatulence, and watery diarrhea due to the inability to hydrolyze and absorb lactose, which remains in the intestinal lumen as an osmotically active solute that draws water. Similarly, fatty diarrhea can be caused by malabsorption conditions such as celiac disease or chronic pancreatitis. In the latter case, insufficient pancreatic enzyme secretion impairs the digestion of fats, carbohydrates, and proteins, preventing their proper absorption. This leads to characteristic symptoms, including upper abdominal pain, excessive flatulence, and the passage of large, pale, foul-smelling stools associated with fat malabsorption (Nemeth and Pfleghaar, 2022). Infections that directly damage intestinal epithelial cells by rotavirus or bacterial toxins from Shigella spp. cause malabsorption and osmotic diarrhea (Drancourt, 2017).

5. Complications and Clinical Manifestation

Diarrhea causes complications ranging from volume depletion and electrolyte disturbances to kidney failure due to tubular necrosis in dehydrated children (Rehana et al., 2022). Based on a study at the Department of Pediatrics, North West General Hospital, Peshawar, the clinical manifestations of diarrhea patients included watery stools, abdominal pain, fever, vomiting, and lethargy (Arif et al., 2021). Dehydration is a condition where the body loses excessive fluids and electrolytes and cannot replace the lost fluids and electrolytes (WHO, 2023). According to WHO, the degree of dehydration is divided into three: severe dehydration, mild dehydration, and no signs of dehydration. Severe dehydration is a condition where the body fluid deficit is more than 100 ml/kg or >10%. In children under five years old, the degree of dehydration can be classified as no signs of dehydration if the fluid deficit is <5% and generally asymptomatic, mild dehydration with a deficit of 5–10% characterized by sunken eyes, lethargy, tachycardia, tachypnea, normal blood pressure, and decreased urine volume, and severe dehydration with a deficit of >10% characterized by very sunken eyes, severe lethargy, hypotension, tachypnea, tachycardia, and oliguria (<400 ml/day) (Vega & Avva, 2023).

Electrolytes are acid-base salts found in body fluids, which dissociate or ionize into charged particles or ions. The functions of electrolytes include maintaining osmotic pressure and distributing water to various fluid compartments of the body, maintaining pH in good condition, regulating cardiac and other muscle activity optimally, and playing a role in oxidation-reduction reactions of ion transfer, as well as acting as enzyme cofactors in catalytic processes (Ramdany, 2022).Electrolyte imbalance is a condition where there is an increase or decrease in electrolyte levels, leading to disruption of normal bodily functions and potentially life-threatening complications (Shrimanker and Bhattarai, 2023).

Sodium is responsible for maintaining extracellular fluid volume and regulating cell membrane potential. Sodium regulation occurs in the renal proximal tubules. Sodium is reabsorbed via sodium chloride symporters controlled by the hormone aldosterone. Hyponatremia is a condition where the sodium concentration is no more than 135 mEq/L. Symptoms experienced by hyponatremia patients include dizziness, nausea, and delirium (Shrimanker and Bhattarai, 2023). Hypernatremia is a condition where the serum sodium concentration exceeds 145 mEq/L. This is caused by water deficit from increased water loss in diarrheal stools, and also due to reduced water intake during illness. In rare cases, it can be caused by an increase in total body sodium after ingesting concentrated formula, improperly prepared highly concentrated oral electrolyte solutions, and administering high-solute beverages to children with diarrhea (Anigilaje, 2018).

Potassium is an electrolyte found in the intracellular compartment. Potassium plays a role in the electrical excitation of nerve and muscle cells, as well as acid-base regulation in blood and tissues. Loss of potassium electrolytes can result in arrhythmias, muscle weakness, fatigue, sporadic or familial paralysis, and muscle twitching (Shrimanker and Bhattarai, 2023). Hypokalemia occurs as a result of the activation of the Renin Angiotensin Aldosterone System caused by dehydration, leading to aldosterone secretion. Aldosterone stimulates the epithelial sodium channel (ENaC) for sodium and water reabsorption and potassium excretion, so this activation of the Renin Angiotensin Aldosterone System can cause hypokalemia (Fountain, Lappin and Kaur, 2023). Clinical manifestations of hypokalemia can include asymptomatic, rhabdomyolysis, arrhythmias, heart failure, or paresis (Kardalas et al., 2018). Hyperkalemia occurs when potassium concentration exceeds 5.5 mEq/liter. Hyperkalemia can be caused by acute kidney injury due to severe dehydration (Simon, Farrell and Hashmi, 2023). Acute kidney injury involves the distal nephron and extends to the collecting tubules, causing direct damage to cells responsible for K+ secretion such as acute tubular necrosis due to ischemia or toxins (Palmer and Clegg, 2017). In patients with acid-base balance disorders, especially in cases of metabolic acidosis, extracellular potassium concentration can increase (Hall, 2010). This occurs because hyperkalemia impairs NH4+ excretion through decreased NH3 synthesis in the proximal tubule and decreased NH4+ reabsorption in the thick ascending limb, leading to a decrease in medullary interstitial NH3 concentration and thus a decrease in net renal acid secretion. Consistent with the central role of hyperkalemia in the onset of acidosis, lowering serum K+ concentrations can improve associated metabolic acidosis (Thomas, 2020).

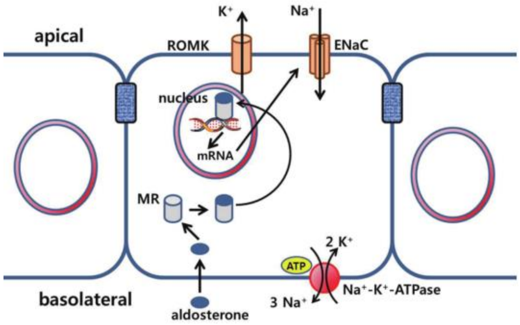

Fig. 7 Mechanism of potassium excretion in the collecting tubules (Cheong, 2013)

Chloride is an anion found in extracellular fluid. Most chloride levels are regulated by the kidneys through filtration by the glomerulus and reabsorption in the proximal and distal tubules (Shrimanker and Bhattarai, 2023). Chloride functions to maintain electron neutrality in the extracellular space along with bicarbonate. This law states that for any solution, the net charge after accounting for all cations (positive ions) and anions (negative ions) must be zero. However, at its nominal value, the subtraction between measured cations (sodium) and measured anions (chloride and bicarbonate) of blood does not yield zero, with a range of 4 to 12 mmol/L called the "normal anion gap" (Pandey and Sharma, 2019). Hyperchloremia is a condition when serum chloride in the blood is more than 106 mEq/L, while hypochloremia is a condition when serum chloride in the blood is less than 96 mEq/L (Morrison, 1990).""'Hyperchloremia occurs due to the loss of bicarbonate ions, which causes an imbalance in electroneutrality in the extracellular space, leading to an increase in chloride ions in the body as a compensatory effort for the loss of bicarbonate ions to maintain electron neutrality in the extracellular space (Sandeep Sharma and Sandeep Aggarwal, 2019). Hypochloremia occurs due to the loss of large amounts of gastric secretions through vomiting or diarrhea (Morrison, 1990).

The kidney is an organ located in the posterior abdomen, outside the peritoneal cavity, containing 800,000 to 1,000,000 nephrons. Each nephron contains a cluster of capillaries within Bowman's capsule called the glomerulus, where a large amount of fluid is filtered from the blood, and a tubule located between where the filtered fluid is converted into urine on its journey through the kidney (Hall, 2010). Creatinine is a non-protein nitrogen (NPN) waste product resulting from the breakdown of creatine and phosphocreatine and can also serve as an indicator of kidney function. Creatine is synthesized in the liver, pancreas, and kidneys from the transamination process of the amino acids arginine, glycine, and methionine. Creatine circulates throughout the body and is converted into phosphocreatine through phosphorylation in skeletal muscle and the brain. The majority of creatinine is produced in the muscles. Therefore, plasma creatinine concentration is influenced by the patient's muscle mass (Salazar, 2014). In dehydrated conditions, a decrease in creatinine excretion in the plasma through urine will lead to an increase in its levels along with an increase in the degree of dehydration (Ibrahim, Umboh and Manoppo, 2024). Blood urea Nitrogen is an NPN waste product of amino acids derived from protein breakdown undergoing deamination to produce ammonia. Ammonia is then converted into urea by liver enzymes. Therefore, urea concentration depends on protein intake, the body's capacity to catabolize protein, and adequate urea excretion by the renal system (Salazar, 2014). In severe dehydration, a decrease in total intracellular and extracellular fluid can lead to decreased tissue perfusion and circulatory volume, thereby inhibiting kidney function. This is characterized by a decrease in the glomerular filtration rate (GFR) (Ibrahim, Umboh and Manoppo, 2024).

According to Adiba et al. (2022), metabolic acidosis can be caused by reduced blood circulation in the kidneys and body tissues in diarrhea patients with severe dehydration, leading to impaired excretion of organic acids by the kidneys and accumulation of lactic acid due to tissue hypoxia. In addition to lactic acid accumulation, a decrease in bicarbonate ions can contribute to metabolic acidosis. There are three causes of decreased bicarbonate ions in the body: gastrointestinal disorders, kidney disorders, and exogenous causes. Gastrointestinal disorders are caused by the secretion of bicarbonate ions into the intestinal lumen to maintain the neutrality of the acidic food environment from gastric emptying. These bicarbonate ions will be reabsorbed and converted into bile. However, in cases of diarrhea, bicarbonate in the intestines is lost through feces due to increased intestinal motility. Further bicarbonate secretion from the pancreas and intestinal mucosa will lead to blood acidification due to bicarbonate loss (Sandeep Sharma and Sandeep Aggarwal, 2019). In cases of Rotavirus infection diarrhea, stimulation of enterochromaffin cells causes the release of serotonin, which will induce the nausea and vomiting centers in the medulla oblongata to stimulate the nausea and vomiting reflex (Crawford et al., 2017). The vomiting reflex is one of the causes of HCl loss in the stomach (Tinawi, 2021). The loss of large amounts of gastric secretions, such as hydrochloric acid, will lead to the loss of hydrogen secretion carried out by parietal cells in the gastric mucosa, thereby causing a relative increase in bicarbonate in the blood, leading to alkalosis (Brinkman and Sharma, 2020).

6. Management of Acute Diarrhoea

Based on the WHO publication "The Treatment Of Diarrhoea a Manual for physicians and other senior health workers," the management of diarrhea in children under five years old is adjusted according to the degree of dehydration experienced. In children without signs of dehydration (fluid deficit <50 ml/kg), therapy involves administering oral rehydration fluid such as oral rehydration salts (ORS) or other electrolyte-containing fluids or salt-free fluids, with a volume of 50–100 ml per diarrheal episode for children under two years old and 100–200 ml for children aged two years and above. For mild to moderate dehydration, ORS is given for the first four hours, totaling 200–1200 ml according to age group, then continued as needed. Meanwhile, for severe dehydration, rehydration is performed rapidly via intravenous infusion, and if the child can still drink, ORS can still be given orally while waiting for the infusion to be set up (WHO, 2005). The 2024 WHO guidelines strongly recommend low-osmolarity oral rehydration solution (LORS) for children up to 10 years of age with acute watery diarrhea and dehydration. Although direct trials in children aged 5–10 years are still lacking, supporting evidence from biological plausibility, clinical experience, and safety data justifies its use. LORS effectively reduces diarrhea duration, stool output, and treatment failure compared to standard ORS, while also being approximately 70% more cost-effective, widely available, and feasible (Kundu, Das and Medhagopal, 2025).

In addition to fluid therapy, zinc supplementation is an important part of diarrhea management in children. Zinc supplementation is given for 10–14 days to accelerate intestinal mucosal recovery, reduce the duration and severity of diarrhea, and lower the risk of recurrence. The recommended zinc dose is 10 mg per day for children under six months of age and 20 mg per day for children aged six months and above (WHO, 2005). However, according to Kundu, Das and Medhagopal (2025), oral zinc supplementation is recommended for 10 to 14 days, using a reduced daily dose of 5 mg to decrease the incidence of vomiting while maintaining therapeutic effectiveness. This is an update from previous protocols that suggested higher doses of 10 or 20 mg per day and expands the indication for zinc therapy to include older pediatric populations.

Routine antimicrobial use in pediatric diarrhea is not recommended, as clinical presentation rarely allows differentiation between infections responsive to such agents—like those caused by enterotoxigenic Escherichia coli—and those that are not responsive, including rotavirus or Cryptosporidium infections. Furthermore, the lack of pathogen sensitivity data, increased treatment costs, risk of adverse reactions, and contribution to antimicrobial resistance underscore the need to restrict their use (WHO, 2005). However, according to Kundu, Das and Medhagopal (2025), antibiotic therapy is strongly recommended for children up to 10 years of age with bloody diarrhea, as the potential risks of untreated infection outweigh the low to moderate certainty of evidence, with ciprofloxacin as the first line and ceftriaxone as the second-line treatment according to standard dosing guidelines. Antimicrobials are generally only indicated for cases of bloody diarrhea, while anti-protozoal, anti-diarrheal, and anti-emetic drugs offer little therapeutic value in managing acute or persistent diarrhea. The management of protozoal infections, such as amebiasis and giardiasis, centers on metronidazole therapy. The recommended pediatric dose for amebiasis is 10 mg/kg body weight three times daily for five days—which can be extended up to ten days in severe cases—while for giardiasis, 5 mg/kg three times daily for five days is recommended.with treatment reserved for confirmed persistent infections showing Giardia duodenalis cysts or trophozoites in stool or intestinal fluid (WHO, 2005). For protozoal diarrhea, nitazoxanide provides only a small reduction in symptom duration, while azithromycin has not shown consistent mortality benefits in non-dysenteric cases. Due to the indirect nature of the available evidence, uncertain clinical benefits, and cost and equity considerations, the Guideline Development Group (GDG) emphasizes caution in updating treatment recommendations (Kundu, Das and Medhagopal, 2025).

7. Conclusion

Diarrhea in children under five years old remains a major health problem caused by various pathogens such as Rotavirus, E. coli, Giardia duodenalis, Cryptosporidium spp., and Entamoeba histolytica. The primary management is rehydration to prevent dehydration and electrolyte imbalance, accompanied by zinc supplementation to accelerate recovery. Antimicrobial use is not routinely recommended because most cases are non-bacterial and carry a risk of resistance. Antimicrobial therapy is only given for bloody diarrhea or confirmed protozoal infections, with metronidazole as the primary choice. A rational management approach in accordance with WHO guidelines is important to reduce morbidity and mortality from diarrhea in children.

8. Illustrations

All figures should be numbered with Arabic numerals (1,2,3,….). Every figure should have a caption. All photographs, schemas, graphs and diagrams are to be referred to as figures. Line drawings should be good quality scans or true electronic output. Low-quality scans are not acceptable. Figures must be embedded into the text and not supplied separately. In MS word input the figures must be properly coded. Lettering and symbols should be clearly defined either in the caption or in a legend provided as part of the figure. Figures should be placed at the top or bottom of a page wherever possible, as close as possible to the first reference to them in the paper.

Acknowledgements

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