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Acute Diarrhea in Children

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SUMMARY
Acute diarrhea (AD) is the most frequent gastroenterological disorder, and the main cause of dehydration in childhood. It is manifested by a sudden occurrence of three or more watery or loose stools per day lasting for seven to 10 days, 14 days at most. It mainly occurs in children until five years of age and particularly in neonates in the second half-year and children until the age of three years. Its primary causes are gastrointestinal infections, viral and bacterial, and more rarely alimentary intoxications and other factors. As dehydration and negative nutritive balance are the main complications of AD, it is clear that the compensation of lost body fluids and adequate diet form the basis of the child's treatment. Other therapeutic measures, except antipyretics in high febrility, antiparasitic drugs for intestinal lambliasis, anti-amebiasis and probiotics are rarely necessary. This primarily regards uncritical use of antibiotics and intestinal antiseptics in the therapy of bacterial diarrhea. The use of antiemetics, antidiarrhetics and spasmolytics is unnecessary and potentially risky, so that it is not recommended for children with AD.
Keywords: acute diarrhea; etiopathogenesis; diagnostics, therapy

INTRODUCTION

Acute diarrhea is the most frequent gastrointestinal disorder and the main cause of dehydration in childhood [1, 2, 3]. It is characterized by a sudden occurrence of three or more watery or loose stools daily [1-5]. In addition, the initial phase of the disease is often accompanied by anorexia, vomiting, abdominal pain and elevated body temperature [1, 2, 3].

Acute diarrhea primarily occurs in children during the first five years after birth, and particularly in the second half-year and in small children [1, 2, 3]. Although it is present worldwide, the highest incidence is recorded in the developing countries. Except for neonatal pathological conditions and pneumonia, acute diarrhea is globally primarily due to dehydration, i.e. hypovolemia, electrolyte disbalance and acidosis, and in recurrent cases and general malnutrition, the leading cause of mortality in children until completed fifth year of life (Table 1) [1, 2, 3, 6, 7]. According to the data of the World Health Organization (WHO) from 2004, one-and-a-half million children in the world die of acute diarrhea, mainly in countries with low standard of living [7]. The same WHO document reports that over 80% of children who died of acute diarrhea are from African and South Asian countries, where most were from India (380,600), Nigeria (151,700), Democratic Republic of Congo (899,000), Afghanistan (82,100) and Ethiopia (73,700).

Table 1. Etiological factors of mortality of children under the age of five in the world [7]

Factors		%
Neonatal factors	Total	40.3
	Prematurity	14.1
	Intrapartal complications	9.4
	Sepsis and meningitis	5.2
	Other	11.6
Pneumonia		14.1
Diarrhea		9.9
Malaria		7.4
Injuries, AIDS, meningitis, morbilli and other		28.3

ETIOPATHOGENESIS

The most frequent cause of acute diarrhea are gastrointestinal infections, viral and bacterial, and rarely parasitic (Tables 2 and 3) [1-5, 8, 9, 10]. The infections are spread by fecal–oral transmission, i.e. contaminated food and water or direct or indirect contact with an infected individual [9, 10]. Particularly high contagiousness show rotavirus, norovirus and *Shigella* [10, 11]. Viral causes of acute diarrhea, in addition to the classical manner, can be spread through aerogenic transmissions [10]. Prevalence of specific intestinal pathogens is age-related but it also depends on the stage of development of the child's surroundings [3, 9]. The most frequent etiological factors of acute infective diarrhea in Europe, North America and Australia, the developed countries, particularly at the age range from six months to five years, are viruses (rotavirus, norovirus, adenovirus, calicivirus, astrovirus and others), while bacterial causes of the disease (*Campylobacter jejuni*, *Salmonella*, *Shigella* and pathogenic species of *Escherichia coli*), which primarily affect children in the first six months after birth and after five years of age, are much rarer [1, 2, 3, 8]. *Giardia lamblia*, *Entamoeba histolytica* and *Cryptospo-*

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Table 2. Causes of acute infective diarrhea [8, 10]

Viruses (~70%) [8]	Rotavirus, norovirus (norwalk-like virus), adenovirus (serotypes 40 and 41), astrovirus, enterovirus
Bacteria (10-20%) [8]	*Campylobacter jejuni*, *Salmonella* (animal/non-typhoidal species), *Shigella*, *Yersinia enterocolitica*, *Escherichia coli* (enteropathogenic and enterotoxigenic), *Yersinia pseudotuberculosis*, *Clostridium difficile*, *Salmonella typhi* and *paratyphi*, *Vibrio cholerae*
Protozoa (<10%) [8]	*Giardia lamblia*, *Cryptosporidium*, *Entamoeba histolytica*, *Dientamoeba fragilis*, *Blastocystis hominis*
Helminths	*Strongyloides stercoralis*

ridium are even more rare causes of acute diarrhea [1, 2, 3, 8]. Although this country is not considered part of the economically developed group of countries, owing to unenviable level of children's healthcare insurance, as well as to people's education level, the situation is also similar in our surroundings. In children in developing countries, especially in tropical and subtropical regions, bacterial diarrheas were significantly more frequent, including cholera, typhoid and paratyphoid fever, although there as well as in developed countries rotavirus ranks as the leading single cause of the disease [3]. In these environments parasitic diarrheas were significantly more frequent as well [3].

In addition to gastrointestinal infections, acute diarrheal disorders are caused by alimentary intoxications, wide-spectrum antibiotics, oral iron preparations, laxatives, cytostatics, gastric secretion suppressors, stress-related conditions and severe extraintestinal infections in infancy period, such as sepsis, urinary tract infection, otitis media, pneumonia and other [1]. It is necessary to point out that a prolonged usage of wide-spectrum antibiotics even in children, particularly those with chronic inflammatory intestinal diseases and malignancies, can cause most severe *Clostridium difficile* (pseudomembranous) enterocolitis [9, 12, 13].

Infectious causes of acute diarrheal disorder colonize the small bowel and/or the large bowel (Table 4) [3, 9, 14, 15]. Viral infection affects only the small bowel causing invasion and destruction of the mature epithelium, while bacteria and parasites, depending on the type, exert their pathogenic effect in both bowel segments.

From the pathogenetic point of view, infectious diarrheal disorders are classified into three basic groups, i.e. secretory, osmotic-secretory and exudative-secretory [1, 9, 15]. Secretory diarrhea is caused by *Vibrio cholerae* and toxigenic strains of *E. coli*, osmotic-secretory by viruses, and exudative-secretory by enteroinvasive bacteria (*Salmonella*, *Shigella*, *Campylobacter*) and *Entamoeba histolytica* [3, 8, 15]. Accordingly, osmotic and osmotic-secretory diarrhea is characterized by liquid stools, and exudative-secretory by aqueous-mucilaginous and often blood-stained stools [3]. Enteropathogenic *E. coli*, *Giardia lamblia* and *Cryptosporidium* adhere to mucosal surface of the proximal small bowel, thus, by compromising its function, primarily causing a malabsorptive form of diarrheal disorder [3].

Alimentary intoxications are characterized by a secretory diarrheal disorder caused by the ingestion of food contaminated by enterotoxins of *Staphylococcus aureus*, *Clostridium perfringens* and *Bacillus cereus* [3, 16, 17]. Contrary to infections, there is no bacterial colonization of bowls. *Staphylococcus aureus* excretes a thermostabile, while *Clostridium perfringens* and *Bacillus cereus* excrete a thermolabile enterotoxin.

Table 3. Categorization of most frequent causes of acute diarrhea in Europe, according to children's age [5]

Year	Virus
<1	Rotavirus
	Norovirus
	Adenovirus
	*Salmonella**
1–4	Rotavirus
	Norovirus
	Adenovirus
	*Salmonella**
	Campylobacter
	Yersinia
>5	*Campylobacter*
	*Salmonella**
	Rotavirus

* non-typhoidal species

Table 4. Localization of the causes of gastrointestinal infections [9, 14]

Cause	Localization
Salmonella	Distal ileum and colon
Campylobacter	
Enteroinvasive *Escherichia coli*	
Yersinia enterocolitica	
Vibrio cholerae	Small bowel
Enterotoxigenic *Escherichia coli*	
Viruses (rotavirus and other)	
Giardia lamblia	
Cryptosporidium	
Shigella	Colon
Entamoeba histolytica	

Diarrhea, as a component of antibiotic therapy occurs as the consequence of the disintegration of colonic bacterial flora [18, 19]. The most severe disorder of this type is *Clostridium difficile* enterocolitis [12, 13]. Erythromycin, azithromycin and other macrolides, except for antibiotic effect, also act stimulatively on the gastrointestinal motility, thus it is not rare that their application is followed by feelings of nausea, vomiting, abdominal pain and diarrhea [20].

Other medications cause a diarrheal disorder by various mechanisms – oral iron preparations by irritative (prooxidative) effect, purgatives by laxative, chemotherapeutics by cytotoxic, gastric secretion suppressors (proton pump inhibitors and H2 blockers) by prokinetics, etc [21].

Stress conditions disturb vegetative body function, including the gastrointestinal motility and secretion, which constitute the bases for diarrheal episodes in persons with irritable bowel syndrome [22].

Pathogenesis of diarrhea, as a component of extraintestinal infections, is highly complex and insufficiently clear. It occurs as a consequence of antibiotics use, but also as a consequence of other, numerous factors that disturb the gastrointestinal integrity [23].

CLINICAL FEATURES

Basic clinical characteristics of acute infective diarrhea are relatively short incubation period, sudden onset manifested by frequent watery or loose stools and a complete recovery within 14 days (Table 5) [1, 2, 3, 19, 24, 25]. Enteritis is characterized by watery and postprandial, and colitis by mucous or mucous-hemorrhagic stools [3, 26]. In most cases the initial phase of the disease is followed by increased fever (one to three days), vomiting, loss of appetite, abdominal pain, and in case of colitis a false need to pass stools, and tenesmus. Owing to the natural passive immunity acquired prenatally, in infants aged six to nine months, particularly in those who are breastfed, gastrointestinal infections in general, and in particular viral ones, are usually asymptomatic or with mild clinical symptoms [3, 24].

Table 5. Incubation period in acute infective diarrheas [10]

Cause	Incubation period (days)
Rotavirus	3 (1–3)
Norovirus	1 (1–2)
Astrovirus	1–2
Salmonella (non-typhoidal)	1 (0.3–1)
Campylobacter	3 (1–7)
Shigella	3 (1–7)
Giardia lamblia	9 (7–14)
Cryptosporidium	7 (1–14)

Table 6. Classification of dehydration

According to	Classification
Degree of body weight loss [27]	Mild (<5%)
	Moderate (5–10%)
	Severe (>10%)
Osmolality [29]	Isotonic (275–295 mOsm/kg)
	Hypotonic (<275 mOsm/kg)
	Hypertonic (>295 mOsm/kg)
Blood level of sodium [27, 29]	Isonatremic (Na^+ 130–150 mmol/L)
	Hyponatremic (Na^+ <130 mmol/L)
	Hyperosmolar (Na^+ >150 mmol/L)

Table 7. Water and electrolyte deficit in isotonic diarrheal dehydration [27, 28]

Parameter	Deficit
Loss of body weight (%)	5
	10
	15
Water (ml/kg)	50
	100
	150
Na^+ (mmol/kg)	4
	8
	12
K^+ (mmol/kg)	3
	6
	9

Contrary to infections, alimentary intoxications are characterized by a very short latent period (usually 10–12 hours, sometimes 30 minutes) and clinical course (mostly one day), as well as the absence of febrility [16, 17]. Besides watery diarrhea, the disease is almost regularly followed by an intensive feeling of nausea, vomiting and abdominal colic.

Basic complication of acute diarrheal disorder is dehydration developing due to diarrhea, vomiting and fever [1, 2, 3]. According to severity, it can be mild, moderate or severe, while according to osmolality, which is primarily defined by the level of sodium in serum, isotonic, hypotonic or hypertonic (Table 6) [8, 27, 28, 29].

Electrolyte deficit is equivalent to the degree of dehydration (Table 7) [28]. It is the highest in hyponatremic, followed by that in isonatremic, and it is the lowest in hypernatremic dehydration. Owing to compensatory mechanisms, in most children diarrheal dehydration is isonatremic (85%), and much rarer hypernatremic (5–15%) or hyponatremic (5–10%) [28, 30]. Hypernatremic dehydration occurs most often in infants, particularly six to nine months after birth, who are febrile, with osmotic diarrhea and on a diet of non-adapted cow's milk, while hyponatremic dehydration occurs in infants who are primarily inadequately rehydrated, i.e. without sufficient sodium substitution, undernourished and with prolonged diarrheal disorder [30]. In addition, more severe dehydration is followed by decompensated metabolic acidosis, hypo- or hyperkalemia and hypoglycemia, and the most severe sensorial disorder, convulsions and anuria [2, 30]. As a consequence of severe hypovolemia, i.e. prolonged hypoperfusion and renal hypoxia, tubular necrosis is also possible [31].

Second most frequent complication that occurs due to anorexia, vomiting, diarrhea and fever is negative nutritional status followed by reversible loss of body weight (BW) [1]. However, in cases with frequent and prolonged diarrheal episodes, particularly in children of the youngest age, poorly nourished or treated with an overly restrictive diet, the loss of BW can progress to severe overall malnutrition [2, 3]. In children during the first years of life, febrile (benign) convulsions are also common [32]. Rarer complications of the disease are bacteremia and consequential metastatic infections (osteomyelitis, meningitis, endocarditis, liver and spleen abscesses, etc.) that primarily develop in younger infants and immunocompromised children with *Salmonella enterocolitis*, as well as chronic post-infective diarrhea induced by overly restrictive diet and/or unnecessary antibiotic therapy, mostly present in the first and rarely in the second year of life, intestinal invagination and perforation, paralytic ileus, toxic megacolon, rectal mucosal prolapse, amebic liver abscesses and others [2, 3, 9].

Rare complications of acute infections also involve immune-mediated extra-intestinal manifestations, which usually occur after the cessation of diarrhea (Table 8) [3, 4, 9].

Table 8. Imune-mediated complications of acute infective diarrhea [9]

Complications	Causes
Erythema nodosum	*Yersinia, Campylobacter, Salmonella*
Guillain–Barré syndrome	*Campylobacter*
Hemolytic-uremic syndrome	*E. coli* O157:H7, *Campylobacter*, *Yersinia*
Hemolytic anemia	*Campylobacter*, *Yersinia*
IgA nephropathy	*Campylobacter*
Reiter syndrome	*Shigella*, *Salmonella*, *Campylobacter*, *Yersinia*
Glomerulonephritis	*Shigella*, *Campylobacter*, *Yersinia*
Reactive arthritis	*Salmonella*, *Shigella*, *Yersinia*, *Campylobacter*, *Cryptosporidium*

DIAGNOSTICS

Diagnosis of acute diarrhea is based on anamnesis, complete clinical examination and adequate laboratory analyses [1-5, 9]. Data on the frequency and appearance of stools, acceptance and tolerance of food, diuresis, as well as the presence of vomiting, fever, abdominal pain and other complaints, are obtained by parents or a custodian, or by the child itself if of older age. It is also important to acquire knowledge about the presence of identical problems in the child's surroundings (family, collective), as well as the consumption of unsafe food or water. Within physical examination, which must always be all-inclusive, special attention should be paid to the degree of dehydration, the state of consciousness, as well as other complications, either intestinal or extraintestinal. Laboratory analyses involve serum values of Na, K, Cl, acid-base status, creatinine, glucose, biochemical parameters of inflammation (C-reactive protein, leukocytosis, erythrocyte sedimentation rate), standard urine examination, and in certain cases hemoculture as well. Patients suspected of lactose intolerance, which represents a frequent manifestation of viral diarrheas, it is useful to determine the presence of reductive substances in stool [3, 4, 9]. Confirming viral particles in stool by the use of the agglutination test is a practical, highly reliable and most frequent procedure in the diagnosis of rotavirus and adenovirus gastroenteritis [33]. Similarly, the verification of antigens of *Giardia lamblia*, *Entamoeba histolytica* and *Cryptosporidium* and *Clostridium difficile* toxins A and B in stool currently present the method of choice in the diagnosis of parasitic and pseudomembranous enterocolitis [12, 13, 34]. Patients with suspected intestinal invagination or perforation require radiological and ultrasound examination of the abdomen, or other examinations depending on the type of complications.

THERAPY

In children acute diarrhea mostly withdraws spontaneously, thus the treatment basis consists of replacement of lost water and electrolytes and adequate nutrition [1, 2, 3, 5]. Probiotics and symbiotics can be useful, while the application of antibiotics is justified only in certain cases [1, 2, 3, 5]. There is evidence that Smecta (diosmectite) and racecadotril represent useful adjuvants in the therapy of this pathologic condition [5]. Antipyretics are not indicated for children with fever below 39°C, except if there is an additional reason for their administration [35]. Antipyretic of choice for children's age is paracetamol, but if a child is older than three months (BW>5 kg), then ibuprofen is the medicine of choice as well [36]. The treatment of acute diarrheal disorder, except in the case of severe dehydration or some other serious complication, does not require hospitalization [37].

Rehydration of children with acute diarrhea

Mild and moderate dehydration caused by acute diarrheal disorder are in about 95% of cases successfully corrected orally, i.e. by the use of oral rehydration solutions (ORSs), while in conditions of severe dehydration rehydration is performed by intravenous route [3, 27, 38]. Therapy with water and electrolytes does not involve only deficit correction but also coverage of the present pathologic and physiologic losses [27].

Intravenous rehydration

In the initial phase of rehydration of the patient with severe dehydration followed by shock or preschock condition, in order to restore circulating volume, it is necessary to use intravenous infusion of 0.9% NaCl or Ringer's lactate in the dosage of 10–30 ml/kg BW during one to three hours [27]. If the patient's condition does not improve, the same treatment is repeated again once or twice during the next one to three hours. To restore volemia, bolus or more rapid infusion is applied, i.e. in doses of 20 ml/kg BW of 0.9% NaCl during 10–20 minutes, which can be repeated up to twice within one hour [39]. During this procedure, in addition to the insight into the patient's condition, it is also necessary to keep assessing central venous pressure and dieresis. In cases of restored volemia and absent dieresis, acute tubulonecrosis should be kept in mind, and accordingly further treatment is to be continued [30]. If the patient's condition is stabilized, which is almost always the case, a full correction of fluid deficit in isonatremic and hyponatremic dehydration requires a period of 24–36 hours, and in hypernatremic dehydration 36–48 hours [27, 30]. In isonatremic and hyponatremic dehydration 50% of fluid restoration is achieved during the first eight hours, and the remaining 50% during the next 16–24 hours, while in hypernatremic dehydration this procedure must be more gradual [27]. Sodium deficit in hypotonic dehydration is calculated by the following formula: Na^+ (mmol) = BW (kg) × 0.6 × (135 – actual serum Na^+ in mmol/L). With the aim of preventing a relative hypernatremia, the normalization of serum sodium must be slow, i.e. not faster than 0.5 mmol/L per hour [27, 38]. This must be strictly taken into account, because a rapid correction of hyponatremia can cause myelinolysis (demyelination) at the level of the

central nervous system, particularly the pons, followed by permanent sequelae, and even lethal outcome [27]. In symptomatic hyponatremia, bolus NaCl 3% is administered at a speed rate of 1 ml/min until the rise of sodium concentration in the serum up to 120 mmol/L, i.e. until the clearly visible improvement of consciousness and cessation of seizures [38]. As 1 ml/kg of 3% NaCl increases sodium for 1 mmol/L, its intravenous application in the dosage of 4–6 ml/kg usually results in the withdrawal of symptoms [38]. Because of the danger of brain edema as well as cerebral hemorrhage and thrombosis, the speed rate of serum sodium decrease in hypernatremic dehydration also must not be higher than 10–12 mmol/L per 24 hours or 0.5 mmol/L per hour [27, 38]. So as to prevent the abovementioned complications, the correction of hyponatremia and hypernatremia requires control of serum sodium level every two to four hours [27, 38].

Decompensated metabolic acidosis with blood pH below 7.25 or HCO_3 below 10 mmol/L requires bicarbonate administered intravenously according to the following formula: $NaHCO_3$ (mmol/L) = BW (kg) × 0.3 × –BE [32]. In most cases it is sufficient to compensate one third to one half of the calculated dose.

Precondition for the compensation of potassium is patient's recovery from the state of shock, i.e. restored diuresis [27, 38]. The concentration of potassium in infusion fluid should not be higher than 40 mmol/L and is administered via a continuous intravenous infusion in the dose of 3–4 mmol/L per 24 hours. More severe forms of acute diarrhea also present hyperkalemia that is normalized after the correction of dehydration and metabolic acidosis.

Oral rehydration

Rehydration by natural (oral) route is based on the active sodium-glucose cotransport [40-43]. Intake of ORSs, composed of a determined combination of sodium, glucose, potassium and bicarbonate or citrate, begins immediately after the appearance of diarrhea and/or vomiting and is continued until a complete normalization of digestive functions [2, 3, 41]. To prevent dehydration, either initially or after oral or itravenous rehydration, an ORS is administered at a rate of 10 ml/kg BW after each watery stool or 2 ml/kg BW after each episode of vomiting, while to correct moderately severe dehydration the administered dose is 100 ml/kg BW and for mild 50 ml/kg BW over a course of three to four hours [2, 3, 40].

An ORS is administered in frequent and small sips using a small spoon, bottle or cup [2]. It can be also given through the nasogastric tube [37]. Therapy is applied under both hospital and home conditions. By adhering to the abovementioned principles, about 50% of patients achieve rehydration after 24 hours [40]. In about 5% of cases oral fluid resuscitation remains unsuccessful and replaced with the intravenous one.

ORSs produced by Galenika in this country is available on the market under the brand name Orosal 65. The composition of the preparation is adopted to this region

Table 9. ESPGHAN and World Health Organization (WHO) recommendations for composition of ORS and Orosal 65 [42, 43]

Parameter		ESPGHAN, 1992	WHO, 1994	Orosal 65** Galenika
Electrolytes (mmol/l)	Na^+	60	60–90	65
	K^+	20	15–20	20
	Cl^-	60	50–80	60
	HCO^-/citrate^{3-}	10*	25–35	25
Glucose (g/1)		13–20	≤20	20
Osmolality (mOsm/kg)		225–260	225–331	281

* Citrate (1 mmol=3 mEq); **By prescription of N. Radlović and R. Stepanović, 1992

pathogenesis of diarrheal disorder and it fully corresponds to the guidelines of the European Society for Pediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) and WHO (Table 9) [5, 43, 44].

Finally, it should be pointed out that the compensation of water and electrolyte loss in patients with acute diarrheal disorder with drinking water, sweetened tea, fruit juices and other drinks, 5% or 10% glucose, physiological solution and similar means have no physiological basis and therefore cannot produce adequate results [40].

Nutrition

After three to four hours of rehydration, either oral or intravenous, the patient is offered food [1, 2, 3, 5]. In milder forms of the disease, i.e. without manifest dehydration and with an adequate ORS intake, diet is not interrupted [3]. This also refers to breastfed infants, whose diet is in no case interrupted [3, 37]. According to current recommendations, the menu of a child should be identical to that before the onset of the disease [1, 2, 3, 5, 37]. The only exception are children with transient lactose intolerance associated with viral diarrhea who are, in regard to artificially-fed children, given lactose-free milk formula, i.e. fermented milk products (yoghurt, sour milk, cheese) if the child is older than one year [8, 37, 45].

Antimicrobial therapy

As mentioned above, acute diarrheal disorder represents a pathological condition which, with compensation of lost fluids and adequate nutrition, withdraws spontaneously within one to two weeks. Thus, antibiotic therapy for acute bacterial diarrheas, generally viewed, is most often unnecessary [5, 9]. Moreover, in some cases it is counterproductive, because, if routinely applied in *Salmonella* enterocolitis, except in favoring germ spreading, it has no other effects, while in enterohemorrhagic *E. coli* strain infection it can contribute to the development of hemolytic-uremic syndrome [5, 9]. Absolute indication for antibiotic therapy of bacterial diarrheal disorders that occur in Europe are only salmonellosis in younger infants (< 3 months old] and patients with immunodeficiency, malignancy and chronic inflammatory bowel disease and moderately severe forms of *Clostridium difficile* enterocolitis [5]. It is

Table 10. Antibiotics in therapy of bacterial diarrhea [4, 5]

Cause	Antibiotic	Daily dose of drug and mode of application	Duration of treatment (days)
Salmonella	Ampicillin	50–100 mg/kg per os or IV in 4 doses	5–7
	Ceftriaxone	50–100 mg/kg IV or im in 1 dose	5–7
	Ciprofloxacin	20–30 mg/kg per os in 2 doses	7–10
Shigella	Ampicillin	50–100 mg/kg per os or IV in 4 doses	5–7
	Ceftriaxone	50–100 mg/kg IV or im in 1 dose	5–7
	Ciprofloxacin	20–30 mg/kg per os in 2 doses	7–10
Campylobacter jejuni	Erythromycin	50 mg/kg per os in 3–4 doses	5
	Azithromycin	5–10 mg/kg per os in 1 dose	5
Yersinia enterocolitica	TMP/SMX	10/50 mg/kg per os in 2 doses	7–10
	Gentamicin	3–5 mg/kg im or IV in 1–3 doses	7
EPEC, ETEC, EIEC	Ampicillin	100 mg/kg per os or IV in 4 doses	5
	TMP/SMX	10/50 mg/kg per os in 2 doses	5
	Ciprofloxacin	20–30 mg/kg per os in 2 doses	5–10
Clostridium difficile	Metronidazole	30 mg/kg per os in 3–4 doses	5
	Vancomycin	40 mg/kg per os in 4 doses	7

EPEC – enteropathogenic *E. coli*; ETEC – enterotoxigenic *E. coli*; EIEC – enteroinvasive *E. coli*; TMP/SMX – trimethoprim-sulfamethoxazole; IV – intravenous

understood that it's indicated in patients with threatening or manifested *Salmonella* bacteremia, as well as in cases of metastatic infections [9, 46]. Also, it is fully justified in severe forms of shigellosis [5]. In other bacterial diarrheas the application of antibiotics can only contribute to some shortening of disease course and faster elimination of the cause [5]. The list of antibiotics to be used in the treatment of acute bacterial diarrhea is presented in Table 10 [4, 5]. *Salmonella* bacteremia requires antibiotic therapy of two weeks, meningitis of four weeks and osteomyelitis of four to six weeks [46]. Antimicrobial drug of choice for fighting intestinal lambliasis and amebiasis is metronidazole [4].

Additional therapeutic measures

Probiotics, racecadotril and diosmectite have a favorable effect on the clinical course of the disease [4, 5, 37, 47, 48]. Probiotics and their combination with prebiotics (symbiotics) essentially contribute to the alleviation and shortening of the disease course, while racecadotril and diosmectite decrease fecal water and electrolyte loss. Due to the high risk of adverse side effects, loperamide and other antidiarrheal drugs, as well as antiemetics (ondansetron and similar) are not recommended for children [8, 37]. Microencapsulated probiotics and prebiotics, due to their higher stability and resistance to acid peptic and biliary pancreatic activity, have advantage over standardly designed preparations of the same type [49, 50, 51].

PREVENTION

Strict adherence to basic hygienic and sanitary measures related to food and water represents the basis in the prevention of alimentary infections and intoxications, and in regard to infections, avoiding contact with the diseased is just as important [2, 3, 4]. Apart from contact with the diseased, rotavirus vaccine is practically the only efficient measure in the prevention of rotavirus gastroenteritis [1-5, 8, 9]. There is no doubt that breastfeeding is the essential component in the prevention of the development and alleviation of infective diarrhea, particularly viral [1-4]. Also, probiotics and symbiotics have a significant role in the prevention of *Clostridium difficile* enterocolitis, and partially in the prevention of rotavirus gastroenteritis [1, 18, 47].

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Акутна дијареја код деце

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КРАТАК САДРЖАЈ

Акутна дијареја је најчешћи гастроинтестинални поремећај и главни узрок дехидратације у дечјој доби. Манифестује се наглом појавом три или више течних или обилних столица дневно у трајању од седам до десет дана, најдуже 14 дана. Најчешће погађа децу у првих пет година по рођењу, а посебно одојчад у другом полугођу и децу узраста до три године. Њени примарни узроци су гастроинтестиналне инфекције (вирусне и бактеријске), а ређе алиментарне интоксикације и други фактори. Будући да су дехидратација и негативан нутритивни биланс главне компликације акутне дијареје, јасно је да ће надокнада губитка телесне течности и одговарајућа исхрана чинити основу њеног лечења. Друге терапијске мере, изузимајући антипиретике ако је дете високо фебрилно, антипаразитне лекове уколико су заступљене интестинална ламблијаза и амебијаза, и пробиотике, ретко су потребне. То се примарно односи на некритичку употребу антибиотика и цревних антисептика у лечењу бактеријских дијареја. Примена антиеметика, антидијароика и спазмолитика је беспотребна и потенцијално ризична, те се не саветује код деце с акутном дијарејом.

Кључне речи: акутна дијареја; етиопатогенеза; дијагностика; лечење

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