

DYSFUNCTIONS OF THE GASTROINTESTINAL TRACT IN YOUNG CHILDREN WHO HAVE SUFFERED PERINATAL DAMAGE TO THE NERVOUS SYSTEM

¹Saidova L.S., ²Akramova X.A., ³Omonova U.T.

^{1,2,3}Tashkent Pediatric Medical Institute

<https://doi.org/10.5281/zenodo.11409563>

Abstract. *Currently, one of the features of the structure of childhood morbidity is the significant prevalence of chronic somatic and neuropsychiatric diseases [1]. The main causes of pathological abnormalities in children, especially in infancy and early childhood, include violations of the course of the perinatal period [2-4]. 55-98% of infants with perinatal damage to the central nervous system (CNS) have various abnormalities of the gastrointestinal tract (GIT), which are clinically manifested by regurgitation, constipation, and diarrhea [5, 6].*

Keywords: *gastrointestinal tract, sympathicotonia, Pylorospasm, abdominal pain; regurgitation, vomiting.*

The mechanisms of formation of gastrointestinal pathology in young children are complex and diverse. Many domestic and foreign experts recognize the leading role of the nervous system in the development of gastrointestinal disorders. At the same time, pathology of the central nervous system of hypoxic, traumatic or infectious origin is considered the most frequent risk factor for gastrointestinal disorders in newborns and infants [7-10]. One of the most frequent gastrointestinal dysfunctions — persistent vomiting and regurgitation—can be caused by cardia chaliasia and esophageal-gastric sphincter discoordination. Persistent expansion of the cardiac sphincter leads to gastro-esophageal reflux and esophagitis. In turn, esophagitis can cause cardiovascular insufficiency, since it increases the sympathicotonia characteristic of newborns and provokes viscerovisceral reflexes that reduce coronary blood flow.

Vomiting and regurgitation, combined with perinatal damage to the central nervous system, may also be due to the lack of inhibition of gastric motility during feeding or immediately after it. Pylorospasm, and more rarely duodenospasm, can lead to the development of concussion and vomiting syndrome. These types of gastrointestinal dyskinesia are usually accompanied by stool retention [11, 12].

As a rule, with severe vegetovisceral disorders, changes in the gastrointestinal tract quickly lose their purely functional character. They are often accompanied by dysbiosis, secondary enzymatic insufficiency, malabsorption, eating disorders, and hypotrophy. Clinical symptoms of gastrointestinal disorders in vegetovisceral syndrome in newborns include:

- * increased stool frequency or spastic constipation, spasms of the intestinal walls, regurgitation, i.e. the so-called hypermotor type of dyskinesia;

- * hypomotor type of dyskinesia — intestinal paresis, atonic constipation, flatulence;

- * discoordination of the cardiac and pyloric sphincters — pylorospasm, chaliasia, achaliasia, reflux;

- * secondary enzymatic insufficiency and intestinal dysbiosis.

Changes in gastrointestinal motility (accelerated transit of intestinal contents or, on the contrary, its stagnation) create prerequisites for disruption of oral and parietal digestion. This is

often overlaid with intestinal dysbiosis, absorption processes are disrupted, which is accompanied by prolonged diarrhea in newborns and infants. Prolonged diarrhea means diarrhea lasting more than 2-3 weeks with a stool frequency of 4 times a day or more and (or) a violation of the consistency and volume of the stool (polyphalia, steatorrhea, etc.) without its increase, as well as conditions in which intestinal disorders periodically appear, referred to in practice as "unstable stool", if this condition does not tend to improve within 3-4 weeks and is accompanied by a stop in weight gain or loss. In most infants (more than 80%), prolonged diarrhea is caused by intolerance to food products or components; in children of the first months of life, this is most often due to intolerance to lactose (less often to other carbohydrates) and cow's milk proteins (BCM).

Lactase deficiency is caused by a decrease in the activity of the enzyme lactase (hypolactasia) or a complete absence of its activity (alactasia). It can be primary (congenital) or secondary (acquired). Primary alactasia is rare. Most pediatricians are faced with variants of secondary hypolactasia.

A decrease in lactase activity can be caused by many factors that damage the structure of the small intestinal mucosa in the brush border area. These include acute intestinal infections, atrophic conditions of the intestinal mucosa (allergic enteritis, BCM intolerance, celiac disease), various intoxications, including drug use, prematurity, etc.

Lactase deficiency is manifested by symptoms of "fermentation" diarrhea, including: frequent stools (8-10 times a day or more), liquid, foamy, with a large water spot and a sour smell; bloating, flatulence, colic; possible development of dehydration.

There is also a so-called "constipated" form of lactase deficiency. In this case, the child is constipated, however, after stimulation of defecation using a gas outlet tube, the stool appears — liquid, foamy, with a sour smell. An important pathogenetic link in a number of gastrointestinal disorders is BCM intolerance, which, according to various authors, ranges from 0.2 to 7.5% [13]. Sensitization to milk proteins can occur in children who are naturally fed, due to excessive consumption of milk and dairy products by the mother during pregnancy and lactation. The development of hypersensitivity to milk proteins is often facilitated by the transfer of a child to mixed or artificial feeding using various adapted and non-adapted mixtures prepared on the basis of cow's milk. Despite the fact that many of these products are close to human milk in terms of the amount of nutrients, the proteins included in their composition are antigenically foreign to the child's body.

The clinical picture of BCM intolerance includes a number of characteristic symptoms: abdominal pain; regurgitation, vomiting; inconsistent consistency of fecal masses (alternating diarrhea with constipation); the presence of blood and mucus in the feces; atopic dermatitis — from mild to severe variants, like childhood eczema (in 50% of patients); rapid development of hypotrophy, often a lag in growth. According to literature data, 30% of children who do not carry BCG develop tolerance to soy protein, which is manifested by skin reactions, diarrhea, weight loss, the development of atrophic processes in the small intestine and colitis.

An important place among functional disorders of the gastrointestinal tract is occupied by constipation, which, according to some data, affects every 5th adult patient from childhood. In children, systematic stool retention of up to 32 hours or more is classified as chronic constipation [14]. Chronic constipation is a polyetiological disease. One of its frequent causes in infants is improper feeding (alimentary constipation). However, chronic constipation can be caused by

organic causes: congenital (Hirschsprung's disease, dolichosigma) or acquired (polyps, anal fissures, etc.). There is a group of patients with iatrogenic constipation (when taking diuretics, antibiotics, anticonvulsants). Secondary persistent constipation can be formed with hypothyroidism, rickets, heart defects, damage to the central nervous system, lumbar and sacral spinal cord, and pelvic parasympathetic nerves. Chronic constipation is observed in almost all children with residual phenomena of perinatal hypoxia, especially during the period of hypertensive-hydrocephalic decompensation. Thus, the causes of chronic constipation in children are diverse and can occur in different combinations in one child [15]. The basis of correction of digestive tract dysfunctions in infants is diet therapy. Specialized nutrition should be physiological, complete, balanced in terms of a set of basic ingredients and at the same time affect the pathogenetic mechanisms of the disease. The reduced content of lactose in the composition of sugars facilitates the assimilation of the mixture in patients with partial hypolactasia, which includes most premature babies. The presence of partially hydrolyzed whey protein reduces its antigenic properties. In addition, protein hydrolysis contributes to its better assimilation, which is important for feeding small children

Dietary correction of nutrition in patients with gastrointestinal food allergy depends on the nature of feeding the child and identified causally significant food allergens. Children of the first 3 months of life, taking into account the similarity of the mechanisms of sensitization to milk protein and soy protein, are prescribed exclusively hydrolysates of milk protein with a high degree of hydrolysis as the main food. This allows you to quickly stop the symptoms of food allergies. It is necessary to focus on the absence in the anamnesis of data on allergies to soy and legumes in the next of kin; the age of the child (at least 5-6 months); carry out gradual, within 5-7 days, introduction of the mixture into the diet; completely exclude dairy products (fermented milk products, cheese, cottage cheese, butter) from the diet; take into account individual tolerance, including monitoring the condition of the skin, the absence of regurgitation, vomiting, and stool disorders. Patients with a burdened allergic history, in whose families parents or older children suffer from allergies, when recommending feeding, give preference to hypoallergenic food products.

For pediatricians, the solution of these issues is of great importance, since it is in early childhood that the most complete correction of functional disorders and prevention of the development of psychosomatic diseases of the digestive system in children are necessary.

REFERENCES

1. Вельтищев Ю.Е. Проблемы охраны здоровья детей России // Рос. вестн. перинатол. и педиатр. — 2000. — № 1. — С. 5–9.
2. Баранов А.А., Щеплягина Л.А. Фундаментальные и прикладные исследования по проблемам роста и развития детей и подростков // Рос. педиатрический журнал. — 2000. — № 5. — С. 5–12.
3. Таболин В.А., Котлунова Н.П., Симонова Л.В. и др. Актуальные проблемы перинатальной кардиологии // Педиатрия. — 2000. — № 5. — С. 13–18.
4. Яцык Г.В., Бомбардилова Е.П. Состояние здоровья детей перС вого года жизни в России // Правда ребенка. — 2004. — № 1. — С. 42–43.
5. Лежнина И.В., Гайворонская И.Л., Кайсин П.Д. Нарушения моС торной функции пищеварительного тракта у детей с отягощенным перинатальным анамнезом и их

- коррекция / Материалы VIII конС гресса педиатров России «Современные проблемы профилактической педиатрии», Москва, 18–21 февраля 2003 г. — М., 2003. — С. 198.
6. Boylan G.B., Pressler R.M., Rennie J.M. et al. Outcome of electroC clinical, electrographic, and clinical seizures in the newborn infant // *Dev. Med. Child. Neurol.* — 1999. — V. 41, № 12. — P. 819–825.
 7. Барашнев Ю.И. Гипоксическая энцефалопатия: гипотезы паС тогенеза церебральных расстройств и поиск методов лекарственной терапии // *Рос. вестн. перинатол. и педиатр.* — 2001. — № 1. — С. 6–11.
 8. Бельмер С.В., Гасилина Т.В., Зверков И.В. и др. Пилорический геликобактер и язвенная болезнь двенадцатиперстной кишки у детей раннего возраста. // *Рос. журн. гастроэнтерологии, гепатологии, колопроктологии.* — 1997. — Т. 7, № 5 (Прил. № 4). — С. 187.
 9. Мазуркевич А.К., Соколов А.Л., Ляхманова Н.Б. Заболевание желудочнокишечного тракта у детей с последствиями перинатальных повреждений нервной системы / *Детская гастроэнтерология: настоящее и будущее:* — М. — 2002. — С. 169.
 10. Яцык Г.В. Руководство по неонатологии. — М.: МИА, 1998. — С. 397.
 11. Руководство по педиатрии / Под ред. А.А. Баранова. — М.: Ди настия, 2006. — С. 464.
 12. Cucchiara S., Bortolotti M., Colombo C. et al. Abnormalities of gastrointestinal motility in children with nonulcer dyspepsia and in children with gastroesophageal reflux disease // *Dig. Dis. Sci.* — 1991. — V. 36, № 8. — P. 1066–1073.
 13. Боровик Т.Э. МедикоСбиологические основы диетотерапии при пищевой непереносимости у детей раннего возраста: АвтоС реф. дис. ... докт. мед. наук. — М., 1994. — С. 46.
 14. Алиева Э.И. Современные вопросы клиники, диагностики и лечения запоров у детей: Автореф. дис. ... канд. мед. наук. — М., 1998. — С. 24.
 15. Заболевания органов пищеварения у детей (тонкая и толстая кишка) /. А.А. Баранова, Е.В. Климанской. — М., 1999. — С. 375.
 16. Moro G., Arslanoglu S., Stahl B., Jelinek J., Wahn U., Boehm G. A mixture of prebiotic oligosaccharides reduces the incidence of atopic dermatitis during the first six months of age // *Arch. Dis. Child.* — 2006. — № 91. — P. 814–819.
 17. Akramova, Kh, D. Akhmedova, and A. Djalilov. "ASSESSMENT OF THE LEVEL OF NEUROSPECIFIC AUTOANTIBODIES IN BLOOD SERUM IN CHILDREN BORN WITH LOW BODY WEIGHT." *Science and innovation* 2.D12 (2023): 20-29.