

**PEDIATRIC ASPECTS OF ETIOLOGY AND PATHOGENESIS  
IRRITABLE BOWEL SYNDROME**

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***Abstract.** This review article presents the etiological factors and pathogenetic mechanisms underlying the development of irritable bowel syndrome in children, the most common functional gastrointestinal disorder. The authors summarize and systematize data from international researchers and modern meta-analyses exploring the pediatric aspects of irritable bowel syndrome.*

***Key words:** Functional gastrointestinal disorders, gastrointestinal tract, IBS, children, abdominal pain, bowel disorder.*

**Introduction.** Currently, irritable bowel syndrome (IBS) is a functional intestinal disorder, which is based on the interaction of two main pathogenetic mechanisms: psychosocial impact and sensorimotor dysfunction, i.e., disturbances of visceral sensitivity and motor activity [14].

IBS remains one of the most common functional disorders associated with the gastrointestinal tract (GIT) in pediatric patients today [15]. In May 2016, the global gastroenterological community became acquainted with the Rome IV criteria at the American Gastroenterology Week; the main provisions of the consensus have already been published [5].

The etiopathogenesis of IBS is very complex and remains incompletely understood. The concept of disease development clearly involves not one, but several etiological factors, triggering not one, but several pathophysiological mechanisms. The complexity of treating such patients lies in the fact that the combination of etiopathogenetic mechanisms varies individually in each case. Among these, the following are particularly relevant today: socioeconomic status, genetic predisposition, the possibility of the disease developing in a child by parents suffering from IBS, psychological aspects, visceral hypersensitivity, gastrointestinal motility disorders, changes in the neuroendocrine system (brain-gut axis), low-grade inflammation, the concept of post-infectious IBS, microbiota imbalance, and, finally, nutritional factors, a detailed description of which can be found in our latest methodological manual [13].

The leading role in the development of IBS is attributed to psychoemotional stress and the instability of children's central nervous systems to traumatic influences. It is recognized that, in modern conditions, IBS is a biopsychosocial functional pathology, as the most severe forms of IBS are observed in children who have experienced mental and social upheaval [2].

Personality characteristics play a significant role in the development of IBS: children with IBS are characterized by poor adaptation to new life situations (first visits to kindergarten and school), dependence on parents, impulsive behavior, and a tendency toward neurotic reactions and phobic disorders. Moreover, the objective state of IBS patients usually does not correspond to the severity of their complaints, but correlates with their psychological characteristics [4, 11, 17].

Changes in intestinal motility in IBS with constipation are characterized by hypersegmental hyperkinesis (hypersegmental hyperkinesis) observed in 52% of patients, leading to prolonged retention of contents in certain segments of the intestine and slowed transit. Dystonic hypo- or akinesis (dystonic hypokinesis or akinesis) is

observed in 36% of cases, caused by disturbances in intestinal wall tone and a sharp decrease in intestinal motility. Antiperistaltic hyperkinesis is found in 12% of patients [6].

The fact that IBS is often detected in children is probably associated with the immaturity of their adaptive mechanisms and the gradual development of the functions of all organs and systems, including the gastrointestinal tract [12].

From a pathophysiological perspective, the 2016 Rome Criteria formulate a paradigm for the pathogenesis of functional gastrointestinal disorders (FGIDs) as a consequence of stress exposure with disruptions in communication along the brain-gut axis, including at the level of immune regulation. The brain-gut axis is a bidirectional, extensive communication network that, through neuroimmune-endocrine mediators, monitors and integrates intestinal functions, connects emotional and cognitive centers with intestinal mechanisms, and coordinates local adaptive responses to stress factors of any kind. Visceral afferent inputs, in turn, are perceived by the brain and influence pain perception, mood, and behavior [1, 5, 16].

A hereditary predisposition to IBS has been identified in 57% of cases. In the remaining 43% of cases, the influence of external factors is solely responsible. Sensitizing factors in IBS include:

1. history of acute intestinal infections;
2. social factor (unfavorable social environment);
3. sexual harassment;
4. physical overexertion;
5. loss of loved ones (two, one parent or close relative);
6. Divorce (for children and women). Other factors (lack of fiber in the diet, physical inactivity, smoking) play a minor role in the development of IBS.

Splenic flexure syndrome is the most common complication in IBS. It is characterized by complaints of discomfort (distension, pressure, fullness) in the left

hypochondrium, occasional pain in the left chest, upper left shoulder, and left side of the neck (simulating angina). These symptoms may be accompanied by palpitations, shortness of breath, and, rarely, a feeling of suffocation and fear. Pain is usually associated with large meals, defecation, emotional factors, and physical exertion. Factors contributing to the development of splenic flexure syndrome include poor posture and wearing tight clothing. Percussion of the left hypochondrium reveals marked tympanitis, and palpation reveals moderate sensitivity [10].

Hepatic flexure syndrome. Rare. Characterized by complaints of fullness or pressure in the right upper quadrant (simulating biliary tract pathology). Pain may radiate to the epigastric region, mid-chest, and, rarely, to the right shoulder or back. Pain intensity may fluctuate over time. Cecum syndrome. Common and characterized by complaints of pain (a feeling of fullness and heaviness) in the right iliac region radiating to the right lateral abdomen (simulating appendicitis). Palpation of the cecum may cause discomfort. Loosening the waist belt and massaging the area of the cecum projection brings some relief.

In recent years, significant advances have been made in uncovering the pathogenetic mechanisms of this disease, its diagnosis, and treatment. However, a number of unresolved issues remain, and improvements to existing differentiated treatment regimens remain relevant. The pathogenesis of this functional disorder, along with impaired motility, visceral sensitivity of the intestine, and the host immune response, is underpinned by changes in the composition of the microflora, which require correction. Intestinal dysbiosis is accompanied by the development of symptoms of intestinal dyspepsia (constipation, diarrhea, flatulence, pain, discomfort, a feeling of fullness or heaviness in the abdomen), which are present in IBS. Manifestations of indigestion syndrome (steatorrhea, amylopoorrhea, creatorrhea) are common, and asthenovegetative syndrome may develop [8, 9, 19].

Visceral abdominal pain syndrome is the leading clinical symptom in children with IBS. Chronic pain is often observed, causing distress and reducing quality of life. It is believed that visceral hyperalgesia in patients with IBS is caused by peripheral stimuli (such as intestinal wall distension) overstimulating a larger number of spinal neurons than normal, resulting in pain. Characteristically, pain in IBS is not clearly localized, but tends to migrate, with variations in radiation patterns and intensity.

Pain associated with IBS is characterized by lability: burning sensations alternate with pulling, spasmodic, or stabbing pain, accompanied by sudden, repeated urges to defecate even when eating a small amount of food [3, 7].

Currently, great progress has been made both in the diagnosis and substantiation of IBS, and in the development of a fairly wide range of treatment methods aimed at all pathogenetic mechanisms of this gastrointestinal disease [18].

**Conclusion.** Thus, a cascade of neuroimmune-endocrine mediators monitors and integrates intestinal functions, coordinates local adaptive responses to stress factors, thereby causing abdominal pain and discomfort. Visceral hyperalgesia in children is caused by peripheral stimuli (such as intestinal wall distension) overstimulating a larger number of spinal neurons than normal, resulting in pain. Children with IBS are characterized by poor social adaptation and dependence on parents, impulsive behavior, and a tendency toward neurotic reactions and phobic disorders.

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