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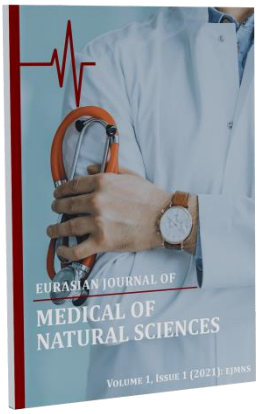

MODERN APPROACHES TO REHABILITATION OF CHILDREN WITH HIRSCHSPRUNG'S DISEASE IN THE POSTOPERATIVE PERIOD

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ABSTRACT

Hirschsprung's disease is a fairly common disease in pediatric surgery. A study of the literature has shown that if the issues of the pathogenesis of the disease are no longer considered controversial, then the introduction of new methods of examination and surgical treatment remains difficult and ambiguous. The use of certain diagnostic and treatment methods is mainly determined by the capabilities and preferences of medical institutions. At the same time, the functional results of surgical treatment leave much to be desired. The reasons for this are the lack of clearly defined deadlines for surgical correction of the defect, as well as unresolved issues of choosing a surgical treatment method suitable for each individual child.

An analysis of the literature data has shown the possibility of solving these problems by developing a rational examination program, taking into account all existing complications and determining appropriate treatment tactics.

Radical one-stage surgical treatment of Hirschsprung's disease using minimally invasive methods is becoming increasingly widespread. Modern surgical methods of treating Hirschsprung's disease in newborns and young children can reduce mortality and improve outcomes.

Introduction. Hirschsprung's disease is one of the most difficult problems in pediatric surgery. Significant advances have been made in understanding its etiology in recent decades, especially with the development of molecular genetics and new pathomorphological research. A new stage in the improvement of methods of diagnosis and treatment of Hirschsprung's disease has been developed not only due to the successes of pathophysiology and early detection of the disease, but also due to the development of surgical techniques. Surgical treatment has progressed from two- or three-stage operations to primary radical [1, 2, 3]. Recently, the radical correction of Hirschsprung's disease using minimally invasive techniques has become increasingly popular. In newborns and young children, progressive methods of treating Hirschsprung's disease contribute to reducing mortality and improving treatment outcomes. An analysis of the literature on the topic under consideration will allow us to present the current state of the issue.

Etiology

Cellular and molecular disorders during the development of the enteric nervous system and the migration of neural crest cells into the developing intestine represent the primary etiology of Hirschsprung's disease. Neuroblasts first appear in the developing esophagus after 5 weeks of fetal gestation. These cells migrate craniocaudally to the rest of the developing intestine between 5 and 12 weeks of gestation. The forms of Hirschsprung's disease are different due to the huge number of possible abnormalities during the development of the enteric nervous system and the different time when neuroblast migration stops. An early stop of migration in the developing embryo leads to a long zone of agangliosis [2].

Other factors such as damage to extracellular matrix components, abnormalities of neurotrophic factors, and molecular damage to nerve cells can also be considered contributing factors to the development of Hirschsprung's disease.

Genetic factors

The increased risk of having children with Hirschsprung's disease among relatives of patients with this disease, the predominance by gender, and the combination of other defects, syndromes, and chromosomal abnormalities with Hirschsprung's disease prove the presence of genetic factors of Hirschsprung's disease. Genetic studies have revealed a mutation in 10 different genes contributing to the development of Hirschsprung's disease [4]. The most common among them are mutations in the RET gene (7-35% of sporadic cases), the EDNRB gene (7%) and the END3 gene (less than 5%) [3, 4, 7]. More than 20 different mutations have been described in the RET protooncogene, and some changes in this gene are associated with certain forms of Hirschsprung's disease [3, 5].

And even if Hirschsprung's disease occurs as an isolated form, it is combined with congenital malformations and accompanies syndromes (such as trisomy 21, septal heart defects, congenital central hypoventilation syndrome, multiple endocrine disorders neoplasia type

2, neurofibromatosis, Waardenburg syndrome) in 5-32% of cases [4]. Trisomy 21 (Down syndrome) is combined with Hirschsprung's disease in 7% of cases. Combined malformations are most common in the gastrointestinal tract, followed by the central nervous and genitourinary systems [4, 5].

Pathophysiology

The pathogenesis of Hirschsprung's disease is based on functional obstruction of the narrow part of the colon, which makes it difficult to form peristaltic waves due to the absence of parasympathetic ganglion cells. Despite extensive research, the cause of tonic narrowing of the aganglionic intestine has not been fully elucidated. Agangliosis, cholinergic hyperinervation, impaired nerve supply by nitric oxide synthetase, and disorders of Cajal interstitial cells are collectively involved in the pathogenesis of Hirschsprung's disease, but a complete understanding of the causative factors of disorders seen in Hirschsprung's disease has not been obtained [2, 6].

Classification

The spread of agangliosis in the proximal direction from the internal anal sphincter determines the classification: form with a short zone of agangliosis — recto-sigmoid lesion (74-80%); with a long zone (subtotal) — agangliosis spreads to the splenic angle or transverse colon (12-22%); total colon agangliosis — agangliosis of the entire colon and terminal area of the ileum (4-13%). Intestinal and ultrashort forms are also distinguished. The most severe but rare form of Hirschsprung's disease is total intestinal intestinal agangliosis with the absence of ganglion cells from the duodenum to the rectum [7].

According to the clinical course of the disease, uncomplicated and complicated forms of Hirschsprung's disease are distinguished.

Clinical manifestations

The frequency of Hirschsprung's disease is approximately 1 in 5,000 births. The ratio of boys to girls in the rectosigmoid form is 4:1, and in the long zone it is 1:1-2:1. With increased alertness and improved diagnosis, the age of diagnosis of Hirschsprung's disease has significantly decreased recently, and in most cases, a newborn is diagnosed during the period. Late discharge of meconium, an increase in abdominal volume, vomiting with an admixture of bile, and enteral nutrition disorders are the most common symptoms [8].

Newborns with Hirschsprung's disease are usually full-term infants with late excretion of meconium. Almost all full-term infants lose meconium in the first 24-48 hours of life. In Hirschsprung's disease, 70-90% of newborns do not experience this during this period of life. The differential diagnosis should be carried out with the following pathology: small intestine — intestinal atresia, malrotation, inversion, meconial obstruction in cystic fibrosis; large intestine — meconium plug syndrome, anorectal malformations, small left colon syndrome; Other causes are drugs, electrolyte disturbances, hypothyroidism, sepsis, and very low birth weight. Thus, Hirschsprung's disease should be suspected in all children with late stool loss during the newborn period.

In the complicated form of Hirsch-Prung's disease, the clinical picture manifests itself in the form of low intestinal obstruction or perforation of the cecum and peritonitis in the first days of a child's life, which makes it difficult to diagnose during the newborn period. However, complications of the course of Hirsch-Prung's disease can also be observed in an older age group of patients, which is associated with the inevitable occurrence of enterocolitis in this disease.

Enterocolitis in Hirschsprung's disease

Enterocolitis is a serious complication of Hirschsprung's disease in the pre— and postoperative periods. This is a clinical condition with symptoms including diarrhea, bloating,

fever, abdominal cramps, intoxication, hypovolemia, loose stools, sometimes with an admixture of blood.

Enterocolitis can develop at any age, from the neonatal period to adulthood, regardless of medication or surgery performed. Recurrent enterocolitis can occur even in the presence of an excised stoma [7, 8]

Despite numerous studies, there is still no complete understanding of the etiology of enterocolitis. Numerous theories have been proposed to explain its occurrence, including physical enlargement of the proximal intestine, changes in intestinal contents, rotavirus, clostridia, increased prostaglandin E1 activity, immunological defect of the intestinal mucosa, etc. Some histological and immunological studies have revealed a local or systemic immunological deficiency with impaired lymphocyte function.[9].

Partial mechanical obstruction can trigger the pathogenesis of enterocolitis due to the expansion of the proximal part of the intestine, which will lead to intestinal stasis, even greater intestinal expansion, mucosal ischemia and bacterial invasion. However, this theory does not explain the occurrence of enterocolitis in the part of the intestine distal from the stoma, the occurrence of enterocolitis in the postoperative period, or the detection of enterocolitis histologically in the aganglionic intestine. The length of the aganglionic segment can be considered as a possible factor in increasing the risk of enterocolitis. Undoubtedly, the large length of the aganglionic segment causes a more pronounced proximal obstruction with high pressure, increasing bacterial stasis and proximal expansion. However, studies have shown that there is no correlation between the length of the aganglionic segment and the frequency of enterocolitis.

Mucin production is significantly reduced in patients with Hirschsprung's disease. Moreover, the ganglion segment shows similar damage to mucus production as in the aganglionic segment. This indicates an abnormal protective mucosal barrier in the intestine of patients with Hirschsprung's disease, even in a histologically normal intestine. A decrease in mucus production indicates the danger of bacterial damage to the mucous membrane and translocation of flora.

Secretory immunoglobulin A provides the main immunological barrier in the gastrointestinal tract, resistance to bacteria and prevents bacterial translocation through the intestinal wall. Undoubtedly, if the first episode of enterocolitis has occurred, it can disrupt the intestinal immunity process by producing appropriate antibodies, cause chronic changes in the intestinal mucosa and increase the risk of subsequent episodes. This may explain the real possibility of enterocolitis recurrence after stoma removal or successful surgery [7, 9].

Macrophages play an important role in inflammation in the muscle lining. An increase in the number and activity of macrophages during inflammation leads to disruption of the interstitial cells of Cajal, responsible for the rhythmic work of the intestine in areas of the intestine in which the ganglia are intact. These peristaltic disorders can worsen stasis, bacterial growth, and along with damage to normal mucus production, enhance translocation [8].

Damage at the gene level that triggers the mechanisms of enterocolitis development in Hirschsprung's disease is confirmed by the fact that, for example, children with Down's disease develop enterocolitis in almost 50% of cases, with one or more episodes [7, 8]. The presence of other concomitant abnormalities also causes an increase in the frequency of enterocolitis.

In most cases, it is possible to isolate Clostridium difficile during microbiological examination [7, 8, 9].

Diagnosis

Due to the increased vigilance of pediatricians and pediatric surgeons, the age of diagnosis of Hirschsprung's disease has decreased significantly in recent years and in many cases has become possible even in the newborn period. Hirschsprung's disease should be suspected in newborns with late discharge of meconium (within 24-48 hours), increased abdominal volume, vomiting with an admixture of bile and impaired enteral nutrition. The presence of signs of enterocolitis in a newborn child should also cause suspicion of Hirschsprung's disease. Low intestinal obstruction with preservation of the anatomical integrity of the intestine, perforation of the cecum, ascending intestine or terminal small intestine, peritonitis in the first days of a child's life can be a complicated course of Hirschsprung's disease.

In patients of the older age group, the clinical manifestations of Hirschsprung's disease are persistent constipation, which can alternate with episodes of enterocolitis; abdominal enlargement; lag in physical development.

The main diagnostic methods for Hirschsprung's disease include irrigoscopy, anal manometry, rectal full-layer biopsy, and biopsy of the rectal mucosa for histochemical or immunohistochemical examination.

X-ray examination. On an overview X-ray in a child with Hirschsprung's disease, excessive pleurisy and dilation of the colon can be found over the narrow distal section of the rectum and sigmoid colon and the transition zone between them. With severe enterocolitis, there will be signs of toxic dilatation of the colon [10, 11].

For the complicated form of Hirsch-Prung's disease, depending on the clinical stage, there will be characteristic signs of low intestinal obstruction or perforation of a hollow organ with the presence of gas in the free abdominal cavity.

In some centers, irrigoscopy is the first test in the examination of children with suspected Hirschsprung's disease with signs of intestinal obstruction. Signs of Hirschsprung's disease on irrigoscopy are pathological narrowing of the distal colon, a funnel-shaped transition zone over the affected area and manifestations of enterocolitis, as well as an abnormal rectosigmoid index, which is more pronounced in older children [12, 13]. Recent studies show that the sensitivity and specificity of irrigoscopy in older children is 70-83%, while in newborn patients this percentage is significantly lower due to the vaguely defined transition zone and the absence of a funnel-shaped expansion of the intestine above it. In addition, in the presence of concomitant enterocolitis, an irritable bowel with areas of spasm may create the appearance of pathological narrowing of the intestine. This means that up to the age of one month, the risk of both false-positive and false-negative results of irrigoscopy increases almost threefold. Also, there may be a mismatch between the X-ray zone and the zone determined during the biopsy, especially in children under 1 month old. Thus, irrigoscopy in newborns may be misinterpreted [7, 8, 14].

The use of anorectal manometry is possible in older children to diagnose Hirschsprung's disease, but anorectal manometry in newborns is quite controversial, technically difficult and unreliable in most cases [11].

Morphological examination of biopsies uses several techniques: light microscopy with hematoxylin-eosin staining of full-layer biopsies of the rectum; histochemical and immunohistochemical examination of biopsies of the rectal mucosa. Rectal full-layer biopsy is represented by the absence of ganglion cells during hematoxylin-eosin staining. Histochemical examination of biopsies of the rectal mucosa shows the presence of acetylcholine-positive hypertrophic nerve fibers confirming Hirschsprung's disease. Along with hematoxylin-eosin rectal biopsy, the use of histochemical reaction to acetylcholinesterase has made morphological diagnosis simpler and more reliable [7, 8, 11]. In a newborn child, performing a full-layer biopsy presents great technical difficulties, while a biopsy of the rectal mucosa is a technically simple manipulation. In addition, the absence of ganglion cells in the distal part of the anal canal may be a variant of the norm. Some other techniques are used to diagnose Hirschsprung's disease, such as lactate dehydrogenase, succinate dehydrogenase, NADPH-diaphorase, histochemical determination of enzymes. The histochemical reaction to acetylcholinesterase for the diagnosis of Hirschsprung's disease in newborns has a sensitivity of 91%, a specificity of 100%, and a false negative result is obtained in 8% of cases. In newborns, especially premature or functionally immature ones, the number of nerve fibers in the intestinal wall may be reduced even in the presence of Hirschsprung's disease, therefore, a decrease in the number of ACHE-positive fibers in newborns may lead to a false negative result. Despite these limitations, rectal biopsy is a more sensitive and specific method than irrigoscopy and anal manometry combined, even without the use of additional immunohistochemical techniques. Immunohistochemical examination is an important morphological method that uses specific antigen-antibody reactions.

The main immunohistochemical methods are direct and indirect immunofluorescence or direct and indirect enzymatic immunohistochemical reaction. Many immunohistochemical markers and dyes are used for research and clinical diagnosis of Hirschsprung's disease and other intestinal diseases. The most commonly used test is for calretinin, a common marker [7, 15, 16].

Calretinin is a Ca-containing protein that plays an important role in the organization and functioning of the enteric nervous system. Ca-containing proteins are involved in physiological calcium homeostasis. Ganglion cells and their derivatives secrete calretinin from the submucosal and intermuscular plexus of the intestine of the normal ganglion intestine, in contrast to the aganglionic segment in Hirschsprung's disease, which lacks calretinin. The absence of calretinin staining in the nerve processes also shows the absence of calretinin in the corresponding nerve cells, which can be used as a diagnostic test in the diagnosis of the aganglionic segment. An important aspect of immunohistochemical research is that the method allows detecting various innervation disorders, as it stains all available cells, even immature and small ones. However, other markers and methods are needed to detect nervous hypertrophy.

Treatment

Confirmation of the diagnosis of Hirsch-Prung's disease is an indication for surgical treatment.

Advances in neonatal care, anesthesia, and resuscitation over the past decades have made it possible for pediatric surgeons to perform single-stage correction of Hirschsprung's disease

with uncomplicated form and exclusion of total agangliosis. In most cases, the diagnosis is made in the neonatal period, and many centers use single-stage correction with amazing results. The main contraindications to primary transanal reduction are severe life-threatening malformations, severe enterocolitis, pronounced dilatation of the proximal intestine and general somatic disorders.

With this intervention, the aganglionic intestine is lowered transanally and resected approximately 10 cm above the level of the transition zone. A coloanal anastomosis is superimposed between the ganglion intestine and the anus [17, 18].

In cases of a long zone of agangliosis, laparoscopic mobilization of the colon and its transanal endorectal reduction can be used [19, 20].

If there is enterocolitis, intensive treatment is first performed, including correction of the water-electrolyte balance, cleansing enemas [7, 8].

In the complicated form of Hirsch-Prung's disease (intestinal obstruction, necrotizing enterocolitis with toxic dilatation of the colon, perforation of the terminal small intestine, caecum or other parts of the colon), stoma removal with biopsies at different levels of the colon and rectal biopsy. After confirming the diagnosis of Hirschsprung's disease, determining the area of intestinal agangliosis determines the type of further surgical treatment [9].

The most serious prognosis is for intestinal intestinal agangliosis. Small intestinal high stoma will inevitably cause short bowel syndrome and questionable prospects after radical surgery [20].

Results and complications

Scrupulous technique, adequate hemostasis, good blood supply to prevent ischemia, and the inadmissibility of twisting and straining of the intestine should prevent complications during transanal endocrine bowel reduction.

Enterocolitis is the main cause of increased morbidity and mortality after radical treatment of Hirschsprung's disease [7, 9]. The frequency of postoperative enterocolitis, according to various literature data, varies from 5% to 42%, depending on the definition and diagnostic methods of enterocolitis [7]. Despite the success in the treatment of children with Hirschsprung's disease, the pathogenesis of enterocolitis remains unknown. Presumably, obstructive mechanisms lead to intestinal stasis with bacterial proliferation, bacterial damage to the mucosa, followed by a local and general inflammatory response. Risk factors for enterocolitis include early age, anastomotic strictures, and eating disorders. It was assumed that a shorter muscular sleeve could reduce the incidence of enterocolitis, reducing the possibility of spasm. All these findings suggested that intestinal stasis and immature mucosal immunity due to early age may contribute to enterocolitis [17].

The formation of cicatricial stenosis after radical reduction is another serious complication. The frequency of stenosis varies from 0% to 35%, depending on the definition of the concept [18, 19]. Risk factors for stenosis include ischemia and anastomotic failure, as well as circular anastomosis. In addition, as mentioned above, stenosis is a high risk factor for postoperative enterocolitis. Most of the stenoses are treated conservatively with dilation, and only some require more aggressive surgical correction. The technique of applying an oblique anastomosis of the anus can reduce the formation of stenosis.

The failure of the colonoanal anastomosis in the form of peritonitis, perianal phlegmon, or the formation of a pararectal fistula requires removal of the stoma before the inflammatory process is stopped. Such complications are very rare, as the anastomosis is protected by the walls of the anal canal. However, in the case of ischemia in the area of the anastomosis or a hematoma with secondary infection, the formation of an inflammatory focus is possible. If the integrity of the intestine is restored and there are no deformities in the anastomosis area, the stoma is closed by applying an end-to-end anastomosis. However, if the intestine is discredited, it is necessary to re-reduce the intestine with resection of the affected area of the intestine and closure of the stoma.

The frequency of stool is usually high (5-10 times a day) immediately after surgery. Over time, six months after the operation, it reaches 1-4 times a day. Constipation occurs a few weeks or months after surgery and depends on the type of surgery, with a higher probability of occurrence after operations with the removal of the aganglionic part of the intestine (Duhamel, Ribein). Constipation occurs in about 8% of children, but their frequency can reach 20% [18, 20]. Recent publications report 37% of children with impaired stool evacuation after surgery for.

Hirschsprung's disease.Functional forms of constipation can be cured by conservative methods such as enemas or laxatives. In addition, persistent constipation as a result of achalasia of the sphincter, stricture formation, incomplete resection of the aganglionic part of the intestine or the dysganglionic intestine may require active measures such as repeated irrigoscopy, biopsy. The treatment method depends on the examination results and may include active dilation, botox, myectomy, or reninformation [6, 7, 18].

Fecal incontinence is usually detected in children over 4 years of age. A large series of studies indicates the absence of fecal incontinence after transanal reduction surgery. At the same time, some authors point to poor stool retention after reduction due to impaired stool consistency or loose stools after surgery in children with a long zone of agangliosis. 44% of children are forced to follow a diet after surgery to avoid constipation or incontinence. Other authors point out that some aspects of stool retention and consistency change over the years [17, 18].

Enuresis occurs in 5-26% of cases and is explained by iatrogenic damage to the pelvic nerves or neuropathy [7]. The use of laparoscopy or transanal reduction is designed to reduce the amount of iatrogenic damage.

Stool disorders, including constipation and incontinence, and enuresis strongly affect the postoperative quality of life of patients with Hirschsprung's disease. The presence of a short section of the colon after its resection with a long zone of agangliosis will lead to a poor quality of life, which, however, will change over the years when the patient learns to control the bowel movement and help his condition with various methods (enema, diet, etc.). The surgeon's task is to reduce these complications by developing careful surgical techniques, postoperative and long-term follow-up.

Conclusion

Great progress has been made in the diagnosis and treatment of Hirschsprung's disease. However, the functional results leave much to be desired. The reasons for this are the timing of

surgical correction of the defect, as well as the choice of a surgical treatment method suitable for each individual child.

A study of the literature has shown that if the issues of the pathogenesis of the disease are no longer considered controversial, then the introduction of new methods of examination and surgical treatment remains difficult and ambiguous.

An analysis of the literature data has shown that it is possible to solve these problems when developing an examination program, taking into account all existing complications, and determining appropriate treatment tactics.

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