American Journal of Biomedicine and Pharmacy
https://biojournals.us/index.php/AJBP
ISSN: 2997-7177

Treatment of Constipation in Children with Dolichosigma

Durdiyev Sarvarbek Hayitboy o'g'li
Urgench branch of Tashkent Medical Academy

Received: 2024 15, Oct
Accepted: 2024 21, Oct
Published: 2024 29, Nov

Annotation: One of the pressing issues in pediatric surgery and pediatric gastroenterology is chronic colonic stasis (CCS) or chronic constipation (CC) CC is defined as a persistent or recurrent condition lasting for more than three months, characterized by a reduction in the age-appropriate frequency of defecation (for children under 3 years of age, fewer than 4–6 bowel movements per week; for children older than 3 years, fewer than 3 bowel movements per week). It is accompanied by difficulty during defecation, a sensation of incomplete bowel emptying, and changes in stool consistency.

Keywords: dolichosigma in children, chronic colonic stasis associated with dolichosigma, morphological and functional, features of the sigmoid colon wall.

For millennia, humanity has suffered from chronic constipation (CC). One of the earliest references to constipation can be found in the Ebers Papyrus, an ancient Egyptian pharmacological text dating back to the 16th century BCE. Hippocrates (460–370 BCE) regarded constipation as a result of "insufficient cleansing" of the body. Avicenna, in his Canon of Medicine (980–1037), described constipation as a disease specifically affecting the large intestine. Paracelsus (1493–1541) identified constipation as one of the five invisible causes of illness, linking it to toxins, waste products, and "internal obstructions".

During the 18th and 19th centuries, physicians believed that constipation was associated with intestinal atony caused by changes in diet, reduced physical activity, and the slower pace of life brought on by urbanization. In 1929, Evans published a definition of constipation that laid the groundwork for its modern interpretation: excessive delay in the passage of food residues through the large intestine due to various causes, accompanied by irregular bowel movements, a sense of incomplete defecation, hard stools, a small quantity of stool, and discomfort. The culmination of this understanding is embodied in the Rome III criteria (2006).

Advancements in medical research have refined the understanding of the causes of CC. The term "dolichosigmoid" (referring to an elongated sigmoid colon) was first introduced by Ausburg (1870) and Lardenoix (1872) and has since become a staple in medical terminology. English surgeon W.A. Lane (1908) suggested that intestinal stasis was caused by adhesions, kinks in the intestine, or significant changes in the positioning of internal organs. To address auto-intoxication, Lane proposed a surgical treatment for CC: colectomy.

Subsequently, many researchers adhered to a mechanical interpretation of CC, attributing it to elongation and dilation of sections of the colon. Various surgical interventions were employed, including colectomies, hemicolectomies, sigmoid resections, separation of adhesions deforming the intestine, fixation of mobile sections of the colon, and the creation of interintestinal anastomoses, among others.

With advancements in understanding the pathogenesis of chronic constipation (CC) in dolichosigma, studies on the nervous apparatus of the colon, and frequent unsatisfactory outcomes of surgical interventions, surgical methods of treatment have become less common. However, there is still no differentiated approach to choosing the technique and timing of surgical treatment for CC in children with dolichosigma.

CC reduces a child's quality of life, negatively affects growth and development, leads to a wide range of psychological problems, and is often accompanied by secondary encopresis. In 60% of cases, it impacts patients' overall well-being, daily activities, and academic performance. Research indicates that 90% of children with CC experience delayed physical development, increased susceptibility to infections, and are predisposed to developing duodenogastric and gastroesophageal reflux. Chronic colonic stasis (CCS) is associated with anatomical and functional changes in pelvic organs, including the reproductive system in girls, raising concerns about the risk of pelvic adhesive-inflammatory processes.

CC is a polyetiological condition. For each patient, multiple causes of coprostasis can usually be identified. The pathogenesis of CC shares common features, including intestinal dysbiosis, impaired immune resistance, secondary disturbances in homeostasis, and morphofunctional changes in the colon wall.

The number of CC patients is steadily increasing in highly developed countries. The true prevalence of colonic transit disorders among children remains a topic of debate. Low parental reporting of constipation in children is due to insufficient public health education, while healthcare providers do not always emphasize the importance of CC, nor do they provide timely and adequate pathogenetic treatment. It is estimated that 10–25% of children suffer from CC, and it accounts for about 40% of pediatric proctological diseases. In some developed countries, up to 60% of pediatric gastroenterology patients suffer from constipation.

In 30–40% of children examined for CC, dolichosigma—the most common developmental anomaly of the colon—is detected. This condition is a borderline state between normality and pathology (elongation of the sigmoid colon with impaired fixation and topographic-anatomical positioning, as well as significant variability in its lumen), potentially predisposing to CCS. Among adults with long-standing colonic stasis, sigmoid elongation is found in 50% of cases. In neonates, the frequency of dolichosigma exceeds 30%. According to literature, dolichosigma occurs in 19–27.6% of the population, not always manifesting clinically, and only 25% of affected children seek medical attention. Boys are more prone to CC associated with dolichosigma.

Dolichosigma, as a provocateur of CC, is accompanied by recurrent abdominal pain and mimics appendicitis, cholecystitis, pancreatitis, Hirschsprung's disease, bowel obstruction, abdominal tumors, intussusception, mesenteric adenitis, and others. Without pathogenetic treatment, CC leads to changes in pelvic floor muscles within 2–3 years. CCS results in persistent disruption of normal intestinal microflora, leading to an inflammatory process in the intestinal wall. Prolonged stasis in the colon causes digestive disturbances, bowel distention, and subsequent dystrophic processes in

the colon wall, increased permeability of the intestinal barrier to bacterial toxins, and changes in cellular and humoral immunity. These factors adversely affect the growth and development of children with dolichosigma and CC. Complications such as megacolon, secondary encopresis, anal fissures, hemorrhoids, rectal prolapse, and intestinal obstruction may develop. One complication of dolichosigma is sigmoid volvulus, the third most common cause of acute intestinal obstruction in adults. Among adults with dolichosigma, 17% are diagnosed with colon cancer. A high frequency of phenotypic markers of connective tissue dysplasia has been observed in children with CC and dolichosigma. Thus, CCS in children with dolichosigma constitutes an important medical and social issue. Despite advances in pediatric coloproctology, comprehensive diagnostics and pathogenetic treatment of CCS in children with dolichosigma remain unresolved.

The sigmoid colon begins after the descending colon exits the retroperitoneal space and ends in the pelvis, where mucosal folds gradually disappear, transitioning into the rectum. The colon gets its name due to its specific shape resembling the Greek letter "sigma." The anatomy of the sigmoid colon is variable, reflected in its length (from a single loop to multiple loops) and possible changes in lumen width and topographic-anatomical position. In 70% of cases, elongation of the sigmoid colon is accompanied by increased mobility and is often associated with other visceral fixation abnormalities.

The blood supply to the sigmoid colon comes from the sigmoid arteries (3 to 5 in dolichosigma) branching from the inferior mesenteric artery; vessel diameter decreases as they move away from the mesentery. The variability in the extra-organ arterial network depends on the mesentery size and the child's body type. This fact may play a role in the development of secondary degenerative changes in the sigmoid colon wall during CC due to distension.

The size of the sigmoid colon, especially its length and additional loops, influences evacuation function disruptions in cases of impaired intestinal motility (reduced high-amplitude propulsive contractions or increased discoordinated motor activity in the distal colon). However, CCS may also result from innervation disorders in the intestinal wall during embryogenesis (primary dysganglionosis) or secondary dystrophic changes affecting nerve plexuses (secondary dysganglionosis). Primary dysganglionosis is morphologically represented by hypoganglionosis, caused by underdevelopment of the intestinal wall's nervous apparatus and a reduction in nerve cells by more than one-third, or intestinal neuronal dysplasia (underdevelopment or ectopia of ganglia).

The sigmoid colon is innervated by the inferior mesenteric and hypogastric nerve plexuses. The hypogastric plexuses include nerve branches from the ganglia of the sympathetic trunk and branches of the anterior roots of the II–IV sacral nerves, homologous to vagus nerve branches. The intra-organ nerve apparatus of the sigmoid colon consists of subserosal, muscular, and submucosal nerve plexuses containing numerous nerve elements (Meissner and Auerbach plexuses).

Intrinsic Neural Plexuses of the Large Intestine. The intrinsic neural plexuses of the large intestine are interconnected and linked to the nerve pathways that enter the intestinal wall from mesenteric nerve plexuses, forming an integral part of the peripheral nervous system.

A specific type of excitable cells has been identified in the walls of hollow tubular internal organs, exhibiting pacemaker activity. These cells, named after the prominent neurohistologist Santiago Ramón y Cajal, are known as interstitial cells of Cajal. The precursors of these cells in the human intestinal tract are cells with receptors for stem cell factor. Initially migrating into the intestine from the mesenchyme of the cranial, cervical, and thoracic regions, as well as the dorsal mesentery, they later localize within the muscular layer as individual elongated cells.

This finding opens prospects for correcting transit dysfunctions of the large intestine by targeting the pacemaker cells themselves or their receptor apparatus, potentially using cell-based technologies.

Morphological Changes in the Sigmoid Colon in Dolichosigma. Numerous studies highlight significant alterations in the neural structures of the sigmoid colon in cases of refractory decompensated chronic constipation (CC) in children with dolichosigma. Degenerative or dystrophic changes, sclerosis of the submucosal layer, and morphological disruptions in the Meissner's and Auerbach's plexuses are commonly observed. In 30% of cases, morphologically significant changes in the intramural nerve ganglia of the colon are undetectable. Morphological abnormalities manifest as mucosal atrophy, sclerosis of the submucosa and vascular walls, venous stasis, and compensatory muscle layer hyperplasia (muscle fiber hypertrophy, edema, and swelling of myocytes).

In longstanding decompensated constipation, changes extend to sclerosis of all layers of the sigmoid colon, characterized by increased reticular and collagen fiber content in the submucosal, muscular, and serosal layers and decreased smooth muscle cells in the muscular layer.

Pathophysiological Mechanisms and Microbial Dysbiosis. The mechanisms underlying impaired evacuation in chronic colostasis remain unclear. Dysbiosis, characterized by changes in the composition and quantity of gastrointestinal microbiota due to factors such as prior infections, improper nutrition, or early formula feeding, is a common trigger. The gut microbiota performs vital functions locally and systemically, with different bacterial species influencing these processes. Persistent dysbiosis can lead to facultative microbial overgrowth, homeostasis disruption, and chronic inflammation.

In cases of colonic stasis, imbalances in vascular endothelial growth factor (VEGF) and transforming growth factor-beta (TGF-β) expression impede the resolution of inflammation. Some authors suggest that specific pathogenic bacteria impair colonic motility through RNA interference, a process discovered in 2006, where small RNA molecules suppress gene expression during transcription or translation.

Structural and Functional Alterations in Dolichosigma. Structural abnormalities, including connective tissue framework and smooth muscle changes, may contribute to CC pathogenesis, especially in children with undifferentiated connective tissue dysplasia. Irritable bowel syndrome (IBS), a common functional disorder characterized by abdominal pain, discomfort, bloating, and altered bowel motility, can act as a triggering factor. IBS prevalence in children is estimated at 10–20%.

In children with IBS and connective tissue dysplasia, two patterns of mucosal changes in the sigmoid colon are observed: hyposecretory and hypersecretory, associated with abnormal synthesis of extracellular matrix components (collagen types I, III, V, and fibronectin). Superficial mucosal changes can provoke CC development.

Pathogenesis and Clinical Implications. The pathogenesis of CC in dolichosigma involves a vicious cycle. Enlarged sigmoid loops impede transit, leading to prolonged retention, stretching the intestinal wall, and promoting fermentation and putrefaction processes. This disrupts microbiota balance, increasing intestinal permeability and damaging epithelial integrity. Chronic dysbiosis exacerbates local immunity deficits and systemic complications. Persistent transit dysfunction results in progressive structural and functional deterioration of the sigmoid wall.

Future Directions. Further studies are required to clarify the relationship between structural and inflammatory changes in the sigmoid colon and various factors contributing to CC in children with dolichosigma. The observed changes indicate delayed medical intervention and underscore the need for timely diagnosis and management.

References:

1. Цветкова Л.Н. Профилактика и лечение запоров у детей. Вопросы современной педиатрии. 2022; 3 (5): 2—7.

2. Whorton J. Civilization and the colon. Constipation as "the disease of all diseases". Br. Med. J. 2020; 321 (7276): 1586—9.
3. Гиппократ. Этика и общая медицина: Перевод с древнегреческого Рудневой В.И. под ред. Трохачева С.Ю. СПб.: Азбук; 2021: 10—5.
4. Великие мыслители Востока: Пер. с англ. Н. Барановой, А. Блейз, С. Зинина, А. Коваля, Я. Никитина. М.: КРОН-ПРЕСС; Серия "Академия"; 2019: 656.
5. Гартман Ф. Жизнь Парацельса и сущность его учения. М.: Новый Акрополь; 2017: 265.
6. Комарова Е.В. Хронические запоры у детей: медицинские и социальные аспекты: автореф. дисс. д-ра мед. наук. М.; 2019.