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Causes and Consequences of the Syndrome of Excessive Bacterial Growth in the Small Intestine

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Abstract

Currently, there is a paradoxical situation where the dominant opinion in medicine recognizes the harmfulness of dysfunction of such barrier structures as the cardia, pylorus, sphincter of Oddi, heart valves, valves of the veins of the lower extremities, etc., but ignores the failure of the ileocecal obturator (bauginium damper) or its absence as a possible cause of the pathology of the digestive system, as well as various extraintestinal diseases [1].

But also I.I. Grekov (1952) expressed his position [2], which remained relevant: "Despite a number of works devoted to the proximal part of the large intestines, the pathology of this department is still insufficiently explained because the role of the Bauginium damper was completely ignored in these works.

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Introduction

Currently, there is a paradoxical situation where the dominant opinion in medicine recognizes the harmfulness of dysfunction of such barrier structures as the cardia, pylorus, sphincter of Oddi, heart valves, valves of the veins of the lower extremities, etc., but ignores the failure of the ileocecal obturator (bauginium damper) or its absence as a possible cause of the pathology of the digestive system, as well as various extraintestinal diseases [1].

But also I.I. Grekov (1952) expressed his position [2], which remained relevant: "Despite a number of works devoted to the proximal part of the large intestines, the pathology of this department is still insufficiently explained because the role of the Bauginium damper was completely ignored in these works."

We assign one of the leading roles in the digestive system to the ileocecal obturator, which delimits the functions of the small and large intestines, isolates the small intestine from reflux of the I arge intestine contents, which differs sharply in chemical composition, physical state, and bacterial spectrum [3, 4, 5]. So, according to L.G. Peretza (1955), in 1 ml of small intestinal contents there are up to 5000 microbes, and in 1 g of the contents of the colon there are about 30-40 billion [6]. As a result of NBC, billions of colon microbes are thrown into the small [4, 6], colonization of the small intestine by allochthonous (foreign) microorganisms occurs, and excessive bacterial growth syndrome (SIBR) or small intestinal dysbiosis develops [7, 8].

Vital products of microorganisms (indole, phenol, cresol, skatol, catechol, carbolic acid, hydrogen sulfide, mercaptan, ethane, methane, etc.) are absorbed into the blood and cause auto-toxicity phenomena. These metabolites cannot be sufficiently detoxified, especially for liver diseases [7, 9, 10, 11].

SIBR leads to the development of putrefactive and fermentative processes in the small intestine. At the same time, the barrier role of the intestinal wall is violated [4, 12], lymphoid tissue suffers along the gastrointestinal tract, and the result is a deficiency of immunoglobulins A and M. The body becomes less protected against microbial aggression. It was found that in 82.4% of patients with chronic colitis, in 70% of patients with chronic enterocolitis, a pronounced decrease in the immunological reactivity of the organism is observed [5].

All authors recognize NLB as one of the anatomical causes of SIBR, but they have no therapeutic effect on it [13, 14]. Currently, SIBR is recognized as a key pathogenetic mechanism in many diseases of the digestive tract and associated extra-digestive conditions [7, 10]. I. Cohn (1970) emphasizes that the ileocecal valve and ileum control the flora of the small intestine. The presence of fecal microflora in the small intestine in itself with the development of NDB is harmful, even if it does not cause clinical pathology in some patients.

M. Brotman [15] found that in the ileum, about 95% of bile acids, which again enter the liver, are normally absorbed, G. G. Nemsadze and E. P. Rybin [16, 17] were detected in a group of 192 cancer patients of the colon, a significant increase in the excretion of bile acids with the failure of the bauginium damper in comparison with its normal function. Indirect data on the possible role of NLB in increasing the excretion of bile acids was obtained by analyzing the characteristics of the metabolism of bile acids in the enterohepatic cycle [18, 19, 20].

Some authors suggest that through the formation of carcinogens from bile acids, bacteroids may contribute to an increased incidence of colon cancer [21]

When performing a right-sided hemicolectomy, the antireflux apparatus (bauginium flap) is removed and the ileocecal transition plays a major role in preventing colonic-small intestinal reflux and the development of a number of pathological conditions, including bacterial overgrowth syndrome (SIBR) in the small intestine [23-27]. The actual prevalence of SIBR is currently unknown [24]. The practitioner this syndrome is difficult to understand [23].

One of the leading links in the pathogenesis of SIBR is the premature deconjugation of primary bile acids (FA) [28, 29]. Deconjugated FAs have detergent properties, which is why they can damage the epithelial layer of the mucous membrane of the small intestine [30], up to the complete atrophy of microvilli [30], which leads to disruption of membrane digestion, creatorrhea, amylorrhea and steatorrhea, and increasing hypovitaminosis [30, 31], to exacerbate diarrheal syndrome [27, 28, 32]. In fact, this pathological



condition is poorly diagnosed due to the low specificity of its symptoms, which are often referred by clinicians to the main SIBR-producing disease [33].

One of the causes of SIBR is the retrograde colonization of the small intestine from the lower sections of the gastrointestinal tract as a result of removal of the bauginium flap during right-sided hemicolectomy, both on a planned and emergency basis [34, 35]. Currently, there is a tendency to adapt the intestinal anastomosis to the conditions of emergency surgery [36].

The elimination of NSC and the use of antireflux small-intestinal anastomosis open up the possibility of surgical prevention of precancerous diseases and colon cancer [22].

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