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COLLAGENS IN THE GASTROINTESTINAL MUCOSA: PEDIATRIC ASPECTS

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Abstract. The spectrum of collagen diseases of the digestive tract is diverse and is divided into three categories: collagenous gastritis, collagenous sprue (collagen enteritis) and collagenous colitis. According to N. Nyhlin et al. (2006), the total annual incidence of CC in the adult section is 4–6 cases per 100,000 people, being one of the causes of chronic diarrhea in elderly patients. It is known that in pediatric practice, the most common form is collagenous gastroenteritis, which is often the cause of severe iron deficiency anemia, which reacts to oral iron supplements, but may recur after drug withdrawal. The disease can be considered as a new possible cause of severe iron deficiency anemia and abdominal pain in children, therefore it is an urgent topic for randomized trials today. The literature review presents data from the analysis of scientific publications of compatriots and foreign colleagues related to collagen diseases in children, in order to increase awareness and alertness about the pathology.

Key words: *collagenous gastritis in children; chronic gastritis; collagenous colitis; microscopic colitis; collagenous sprue; severe iron deficiency anemia in children.*

КОЛЛАГЕНЫ В СЛИЗИСТОЙ ОБОЛОЧКЕ ЖЕЛУДОЧНО-КИШЕЧНОГО ТРАКТА: ПЕДИАТРИЧЕСКИЕ АСПЕКТЫ

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Резюме. Спектр коллагеновых заболеваний пищеварительного тракта разнообразен и делится на три категории: коллагеновый гастрит, коллагеновая спру (коллагеновый энтерит) и коллагеновый колит. По данным N. Nyhlin и соавт. (2006), общая годовая заболеваемость коллагеновый колитом во взрослой популяции составляет 4-6 случаев на 100 000 человек, являясь одной из причин хронической диареи у пожилых пациентов. Известно, что в педиатрической практике наиболее встречающейся формой является коллагеновый гастроэнтерит, нередко являющийся причиной тяжелой железодефицитной анемии, которая реагирует на пероральные добавки железа, однако может рецидивировать после отмены препаратов. Заболевание может рассматриваться в качестве новой возможной причины тяжелой железодефицитной анемии и болей в животе у детей, потому является актуальной в настоящее время темой для проведения рандомизированных исследований. В литературном обзоре представлены данные анализа научных публикаций

соотечественников и зарубежных коллег, связанных с коллагеновыми заболеваниями у детей, с целью большей осведомленности и настороженности по поводу данной патологии.

Ключевые слова: *коллагеновый гастрит у детей; хронический гастрит; коллагеновый колит; микроскопический колит; коллагеновая спру; тяжелая железодефицитная анемия у детей.*

The spectrum of collagen diseases of the digestive tract is diverse and is divided into three categories: collagen gastritis (CG), collagenous sprue (collagen enteritis — CE) and collagen colitis (CC) [1]. N. Nyhlin et al. (2006) report that the adult population's overall yearly incidence of collagen colitis is 4-6 instances per 100,000 individuals. This condition is one of the reasons why older patients experience chronic diarrhea [2]. Collagen gastritis is linked to the deposition of collagen in the subepithelial layer of the stomach lining in pediatric practice, as per the findings of study by Timo Käppi et al. and Changqing Ma et al. [3, 4]. Although there have been reports of kid cases with separate collagen colitis and enteritis [5, 6], they are almost invariably associated with collagen gastritis.

It is noteworthy that a 12-year study of the case of collagen gastritis in a woman described by Colletti et al. revealed progressive atrophy of the glands, intestinal metaplasia, linear neuroendocrine hyperplasia and changes in the surface epithelium, interpreted as uncertain for dysplasia [7]. The study conducted a dynamic study of stomach mucosa bioptates (within 12 years) of a girl whose symptoms (epigastric pain, anemia, hemoptysis and hematohesy) first appeared at age 14. Histologically, subepithelial collagen bands up to 75 μm thick and inflammatory infiltrates in a large number of bioptates have been identified in the stomach lining since the onset of symptoms, confirming the diagnosis of "collagen gastritis". This evidence suggests that patients with collagen gastritis may have an increased risk of developing neuroendocrine stomach tumors and adenocarcinomas, but such cases have not yet been documented in the literature.

Many researchers consider CG as a chronic disease with a benign current. According to research Timo Käppi et al. There is insufficient evidence of CG pathogenesis and anemia as a clear reason for patients seeking treatment [3]. However, the severity of the anaemia, as well as possible relapses after the cessation of iron supplementation, represent a need to increase knowledge of pathology among physicians of all specialties, especially gastroenterologists.

Collagen gastritis is a rare gastrointestinal disease, first described in 1989 by Colletti et al. a 15-year-old girl with recurring abdominal pain and bleeding from the upper gastrointestinal tract [8]. This patient and five other cases were for the first time repelled by Colletti et al. in 1998, in its work [9], which aroused the particular interest of the scientific community in this issue. Analysis of scientific publications of foreign colleagues [3] from 1989 to 2020 revealed no more than 300 cases of observation of collagen gastritis, one third of which are children. The current literature on CG with infancy consists mainly of clinical case reports, except for the description of six patient sample series (sampling criterion — detection of collagen band more than 10 μm thick in the subepithelium of the stomach lining) and three histopathological studies with limited clinical information and follow-up data. It should be noted that knowledge about the evolution of clinical, endoscopic and histological features of the disease over time is scarce, and pathogenesis remains insufficiently studied.

From the etiological point of view, the most common causes of the development of chronic gastritis (such as toxic, allergic, eosinophilic gastritis, sarcoidosis, histiocytosis, ischemic gastritis, chronic granulomatosis, etc.) have not been detected in patients with collagen gastritis. Helicobacter pylori also does not appear to be a fundamental part of the pathogenesis, and the causative barricade did not improve collagen gastritis.

Studies by Hugh J. Freeman (2005) indicate that it is currently unclear which triggers may cause collagen deposition in the mucous membrane of the digestive tract, and whether this mechanism is a by-product or cause of symptoms. Pathophysiological mechanisms of increased collagen synthesis in gastric mucosa have not been described so far in scientific publications, but there are works in which there are suspected mechanisms of collagen deposition at collagen colitis, that, given the paucity of knowledge on whether collagen diseases of the digestive tract are a single spectrum of diseases, may be applicable to collagen gastroenteritis [10].

D.A. Stampfl and L.S. Friedman, in their work on the pathophysiology of collagen colitis [11] hypothesized that collagen synthesis by the pericryptal myophyblasts could be a likely explanation for collagen. In the same paper, it is postulated that the subepithelial strip of collagen in digestive tract collagen diseases consists of type III collagen with a reduced content of type I collagen. Type III collagen is produced by subepithelial fibroblasts to restore mucosa after inflammation. The basal membrane of the normal gastrointestinal tract consists of type IV collagen, indicating the fact of increased collagen synthesis not as a primary process, but rather reparative [10]. Other researchers have found that the collagen band at CC consists mainly of tenascine (glycoprotein extracellular matrix) and collagen type VI, as well as some type III collagen [12]. Tenascin is considered a marker of proliferation and migration of mesenchymal cells; therefore, the authors imply high flow of extracellular matrix with formation of immature, loosely interstitial collagen matrix [12]. Collagen filaments of type VI help to connect cells to the extracellular matrix [12], this may explain why the collagen band can both break down and simultaneously form on other parts of the mucous membrane. However, the expression of the glycoprotein of the extracellular matrix is transient and is often limited to embryonic development. In the embryo, the expression of tenascine occurs in certain areas, such as the nerve crest, and then in areas where skeletal tissue is formed. It is re-expressed in certain adult tissues during normal and pathological tissue remodeling, such as oncogenesis or wound healing. In this case it seems most likely that tenascin is present as part of the reparative process after tissue damage and inflammation, rather than playing any etiological role in the pathogenesis of collagen diseases of the digestive tract [12].

It should be noted that at present the pathophysiology of the disease is still not fully studied. Contrary to the above hypothesis, that increasing collagen synthesis in the mucous membrane of the digestive tract is not a primary process, but rather a postinflammatory reaction, a hypothesis was later put forward in research, subepithelial deposition of collagen and protein exudate occurs due to increased vascular permeability [13, 14].

Based on previously published reports on cases of the disease, two phenotypes of CC were described: "children" and "adult" [4]. It is not clear whether they are part of the same spectrum of pathology or not. In children, inflammatory changes and collagen deposition in the mucous membrane are usually limited to the stomach [3]. In contrast, adult form is associated with diffuse collagen lesions of the gastrointestinal tract, more often collagen colitis and other autoimmune disorders such as celiac disease or diabetes mellitus [3]. Collagen deposition and inflammatory infiltration in adults can be determined throughout the gastrointestinal tract. Gastrointestinal symptoms in adults include abdominal pain, abundant diarrhea without blood impurity in the stool, and the development of malabsorption syndrome with protein loss.

In 1998, Colletti et al. also reported the first "adult" phenotype observed in an 11-year-old boy [9]. Currently there are several cases of "adult" phenotype in pediatric patients [5, 9, 15]. Data are presented showing the occurrence of collagen gastritis in children in combination with collagen colitis. For example, in the comparative study Changqing Ma et al. 31 cases of collagen gastritis (10 cases in children and 21 cases in adults) were analyzed, their clinical, endoscopic, pathological and subsequent results were described. Both children and adults had similar clinical symptoms, such as anemia (50 and 35% respectively), epigastric/abdominal pain (50 and 45%) and diarrhea (40 and 55%). Associated immune disorders were found in 2 (20%) children and 3 (14%) adults [4]. It should be noted that histologically differences between children and adults with the manifestation of collagen gastritis are also not revealed: changes in the mucous membrane in the localization of stomach lesions, the mean thickness of the collagen layer and the amount of eosinophils were found to be equivalent in two study groups. Extragastric collagen lesions have also been observed with a comparable frequency in each cohort (44% and 59%). Follow-up information was available for 22 of 31 (71%) patients. In spite of the drug treatment, 100% of children and 82% of adults have retained their clinical histology. Thus, it has been proved that there are no significant clinical pathological differences between pediatric and adult patients with collagen gastritis [4].

DIAGNOSIS

According to the research Timo Käppi et al. Six out of 15 children (45%) reported recurrent abdominal pain at targeted collection, although in most cases the pain was not described as intense or affecting daily life. However, in the only

patient with associated collagen colitis, recurrent diarrhea dominated among the presented symptoms, despite supportive therapy. Other clinical manifestations also described: heartburn and/or dysphagia, nausea, constipation, bloating, insufficient weight gain, episodes of gastrointestinal bleeding, swelling, etc. In summary, the main complaint is often anemia (e.g., complaints of fatigue and pallor) combined with gastrointestinal symptoms, including iron deficiency anemia, which in some cases triggered a diagnostic examination, it was a random find discovered when patients sought medical attention. The etiology of anemia in CG is thought to be blood loss associated with damage to the dilated capillaries captured by the sub-epithelial collagen band. Timo Käppi Wolving and others. The study notes that in some cases the clinical and/or endoscopic signs of gastric bleeding in children with CG do not correlate with the severity of anemia, which often relapses against the backdrop of supporting iron therapy. The authors suggest that iron deficiency in these patients may be the result of reduced absorption of iron due to stomach hypochlorohydria or other mechanisms, and this issue requires further study [3].

Lee Yeoun Joo and others. Clinical cases describing the endoscopic picture of collagen gastritis in children, represented by characteristic nodulity and multiple polypoid growth against the background of pale mucosa, have been demonstrated. Compared to other associated stomach diseases, in which «nodules» are homogeneous in size and mainly located in the anthral department (eg, H. pylori-associated gastritis) the nodules at KG in most cases have a more irregular form and are located in the mucous membrane of the stomach body rather than the anthral department. Such changes in the stomach lining are often observed in general endoscopic procedures and are described as nodular or nodular gastritis (NG). It is therefore necessary to distinguish between the major diseases which may manifest in a similar way when a nodular pattern of the stomach lining is found in endoscopy. Endoscopic results of CG are reported to vary according to age: from normal mucous membrane to diffuse erythema of stomach mucous membrane, erosion, gastric hemorrhages, nodules and polyp's growth [16].

Histologically, nodules in nodular gastritis are more likely to be clusters of lymphoid follicles or malignant cells, for example in lymphoma associated with the stomach lining. Meanwhile, nodules at collagen gastritis are represented rather by a patch of mucous membrane with normal architectonics, surrounded by a "depressed" reparative atrophied mucosa with subepithelial collagen deposits, thus producing a characteristic nodular appearance. Based on available scientific data, the maximum thickness of subepithelial collagen deposits at the time of diagnosis varies between 15 and 100 μm. Furthermore, histologically, most patients have cell infiltration of the gastric mucosa with a high content of eosinophils (30 eosinophils/high power field). In contrast, intraepithelial lymphocytosis (>25 surface intraepithelial lymphocytes/100 epithelial cells) is much less common [3].

TREATMENT

Currently, there are no effective and standardized treatments for patients with collagen gastritis. For example, a wide range of drugs were used in the analysis of publications, including glucocorticosteroids (Budesonide), antimetabolites (Methotrexate), H_1-histamine blocks (Ranitidine), proton pump inhibitors (Omeprazole), a synthetic analogue of prostaglandin E1 (Misoprostol), gastroprotectors (Sucralfat), preparations of 5-aminosalytic acid (Mesalazine) [17, 18]. Of the non-medicamentous treatments, note the successful treatment of collagen gastritis in a 13-year-old boy on a gluten-free diet with a decrease in clinical symptoms after a month and resolution after 6 weeks from the start of treatment [19].

It is worth emphasizing that the analysis of the publications does not provide convincing data on the persistent improvement of the endoscopic and histological picture in the form of absorption or significant reduction of subepithelial collagen deposits even against the background of the therapy. Thus, the lack of expected effect on any particular intervention, other than the clinical improvement against the backdrop of iron supplementation presented in most publications, as well as reports of spontaneous clinical resolution of symptoms without medication raise the question of further search for adequate therapy of the disease, conducting randomized clinical trials to determine a scientifically based standard of treatment for the disease. Therefore, these patients now require dynamic surveillance, symptom control, especially anemia, testing for various autoimmune and immuno-mediated diseases. It seems necessary to control the endoscopic picture of the stomach mucous membrane, as one should not forget about the potential malignant hyperplasia of endocrine cells with the development of adenocarcinoma.

ADDITIONAL INFORMATION

Author contribution. Thereby, all authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

Competing interests. The authors declare that they have no competing interests.

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