ISSN: 2053-3578 I.F. 12.34

GASTROESOPHAGEAL REFLUX DISEASE (GERD) IN CHILDREN: CLINICAL FEATURES, RESPIRATORY COMPLICATIONS, AND MANAGEMENT

Umida.Kh.Akhmadaliyeva
Andijan State Medical Institute Assistant of the
Department of Outpatient and Polyclinic Therapy
https://orcid.org/0009-0009-0991-2165
E-mail: medichka9725gmail.com, botir7077@mail.ru

Abstract: Gastroesophageal reflux disease (GERD) is a common chronic relapsing disorder of the upper gastrointestinal tract in children, characterized by both esophageal and extraesophageal manifestations. Over the past decade, the incidence of GERD has shown a notable increase in the pediatric population, while the prevalence of gastritis, gastroduodenitis, and peptic ulcers has remained relatively stable. GERD pathogenesis is multifactorial, involving dysfunction of the lower esophageal sphincter, impaired esophageal clearance, increased gastric acidity, and elevated intra-abdominal pressure. Clinical manifestations range from heartburn, regurgitation, and dysphagia to extraesophageal symptoms including bronchopulmonary, otolaryngological, cardiological, and dental disorders.

Bronchopulmonary complications, such as asthma, chronic bronchitis, and recurrent pneumonia, are particularly significant and often related to microaspiration or neural reflex mechanisms. Diagnosis requires comprehensive gastroenterological and pulmonological evaluation, including endoscopy, 24-hour pH monitoring, spirometry, and bronchoprovocation tests. Management emphasizes lifestyle and dietary modifications, pharmacotherapy with proton pump inhibitors and prokinetics, and targeted treatment of respiratory complications. Early recognition and tailored therapy are critical to improving quality of life and reducing disease burden in affected children.

Keywords: Gastroesophageal reflux disease, GERD, children, pediatric, esophagitis, bronchopulmonary complications, asthma, chronic bronchitis, proton pump inhibitors, prokinetics, esophageal pH monitoring, reflux esophagitis

Introduction

The last decade has been characterized by an increase in gastrointestinal tract (GIT) pathologies in both adults and children. Among these diseases, acid-related diseases, which

include gastroesophageal reflux disease, occupy a leading place in terms of frequency and diversity of organ and system damage. reflux disease (GERD), chronic gastritis, chronic gastroduodenitis (CGD), peptic ulcer, and chronic pancreatitis.

While the incidence of gastritis, gastroduodenitis, and peptic ulcers in the pediatric population has remained relatively stable over the past 5-7 years, GERD has shown a clear upward trend. These trends led a group of WHO experts to figuratively call GERD "the disease of the 21st century."

Gastroesophageal Reflux (GERD) is one of the most common motor disorders of the upper gastrointestinal tract. Its incidence, according to various authors, ranges from 18-25% in the pediatric population with digestive disorders.

GERD itself, as a mechanism, its clinical manifestations, and potential complications that significantly reduce the patient's quality of life, constitute a relatively new nosological entity—GERD. Among the numerous existing definitions of this disease, we believe the following is the most appropriate:

"GERD is a chronic relapsing disease characterized by certain esophageal and extraesophageal clinical manifestations and various morphological changes in the mucous membrane (MM) of the esophagus

due to retrograde reflux of gastric or gastrointestinal contents into it."

From a theoretical perspective, GERD represents a relatively rare disease model in which it is difficult to separate etiology and pathogenesis. Among the many factors that explain the onset and development of GERD, the primary one is a disruption of the cardia's "locking" mechanism , which can be absolute or relative.

In addition, the mechanism of development of this disease involves insufficient efficiency of esophageal cleansing (clearance), as well as changes in resistance Esophageal mucosa . Increased acid-forming activity of the stomach also plays a negative role, causing refluxate (components of the stomach contents) to acquire particularly aggressive properties. This is roughly how the so -called "acidic" GER operates.

At the same time, some children experience reflux of alkaline components (bile and duodenal juice) into the esophagus, which is commonly referred to as "alkaline" GER. According to the literature, true "alkaline" GER is very rare, occurring in less than 5% of children with upper gastrointestinal motility disorders.

In addition to the factors mentioned, uncompensated increase in intragastric or intra-abdominal pressure plays a significant role in the genesis of GERD.

To summarize the above, it can be said that GERD is a disease with a complex pathogenesis and the prevalence of one or more factors determines a wide range of clinical and morphological variants of this nosological form.

The clinical presentation of GERD is polymorphic and often depends on the nature of the underlying gastrointestinal pathology. However, it is quite specific and is characterized by esophageal and extraesophageal symptoms .

Esophageal symptoms include complaints such as heartburn, belching, regurgitation , bitterness in the mouth, the "wet spot" symptom, dysphagia, and odynophagia (pain behind the breastbone while eating). The last two complaints are quite rare in childhood and usually indicate

Significant motor and/or structural disorders of the esophagus. It should be noted that the severity of clinical signs of the disease in children does not always correlate with the intensity of reflux and the quality of refluxate .

The extraesophageal symptoms of GERD are also quite varied. Currently, the following extraesophageal "systemic" manifestations of this disease are distinguished: 1) bronchopulmonary (cough, shortness of breath, difficulty breathing, asthma attacks);

2) otolaryngological (hoarseness and loss of voice, recurrent pain in the throat and/or ears);

3) cardiological (heart pain, rhythm disturbances); 4) dental.

GERD-associated It's no coincidence that bronchopulmonary disorders top this list. They are the most studied, best known to practicing physicians, and have acquired a substantial evidence base in recent years.

The study of the relationship between esophageal and bronchopulmonary pathologies dates back to the late 19th century. As early as 1892, W. Osler noted the need to avoid excessive eating and was the first to describe attacks of choking associated with the aspiration of gastric contents.

GER-associated respiratory disorders are traditionally divided into two groups: "upper" (apnea, stridor , laryngitis) and "lower" (bronchial obstruction syndrome, bronchial asthma). The symptoms inherent in these conditions are characterized in the English-language literature by a special term - RARS (reflux-associated). respiratory syndrome).

ISSN: 2053-3578 I.F. 12.34

The connection between the esophagus and the bronchial tree is explained by their common origin from the primary digestive tube and the same innervation by branches of the vagus nerve.

GER can cause respiratory diseases in two ways: 1) direct, with the development of mechanical occlusion of the lumen of the tracheobronchial tree by aspiration material and 2) indirect (neural) with the development of dyscrinia , edema and bronchospasm .

The direct pathway for the development of respiratory problems in children with GERD is primarily due to macroaspiration of gastric contents, leading to mechanical bronchial obstruction and (less commonly) pneumonia. Macroaspiration of acidic material (pH < 2.5) can cause reflex closure of the airway lumen, decreased surfactant production , epithelial damage, and, in severe cases, pulmonary edema and hemorrhage.

Such dramatic developments are rare in children. Usually, a pediatrician encounters with clinical manifestations of laryngitis or bronchial obstruction of varying severity, which can often become recurrent. The leading mechanism in this case is often microaspiration , which provokes the development of laryngo- or bronchospasm by reflex with the formation of such pathologies as chronic bronchitis, recurrent pneumonia, pulmonary fibrosis, apnea.

Protection against bronchopulmonary aspiration involves coordinating the swallowing reflex and closing the glottis during swallowing. Therefore, it can be assumed that the condition of the epiglottis itself is particularly important in some cases. This latter circumstance is rarely considered by ENT specialists and endoscopists , despite the fact that it is possible to visually determine the anatomical features of the epiglottis, as well as its functional characteristics in a particular patient. Furthermore, the condition of the upper esophageal sphincter and esophageal peristalsis contribute to the development of microaspiration in GERD.

The indirect (neural) pathway for the development of GER-dependent respiratory symptoms occurs via the afferent vagal fibers , leading to the development of bronchoconstrictor reflexes and, consequently, bronchospasm . Some believe that esophagitis, which irritates afferent vagal receptors, is a necessary prerequisite for the development of bronchospasm in patients with bronchial asthma .

The mechanism described schematically looks like this: GERD - esophagitis - irritation of the afferent receptors of the vagus - increased reactivity of the tracheobronchial tree - increased lability of the bronchial muscles - bronchospasm .

ISSN: 2053-3578 I.F. 12.34

A number of authors have proposed the existence of specific receptors for esophageal mucosal damage , so-called nociceptors . It is believed that these receptors respond only to altered esophageal mucosa and are inactive in the absence of damage. This hypothesis may explain why physiological GERD does not lead to coughing or asthma attacks.

The literature contains information on the influence of certain neuropeptides on changes in bronchial conductivity, particularly in cases of esophageal mucosal injury . By influencing the tone of bronchial and vascular smooth muscle fibers and stimulating the release of histamine, leukotrienes , and other mediators, they alter the reactivity of the tracheobronchial tree.

Looking at the problem under study from the opposite perspective, it's important to note the provoking influence of respiratory pathology on the development of GER. Any respiratory disorders and symptoms can trigger GER if they alter any aspects of the " antireflux barrier."

The main mechanisms by which this pathological influence is realized can be are presented as follows: 1) an increase in the pressure gradient - an increase in negative intrathoracic and positive intra-abdominal pressure; 2) a decrease in pressure in the area of the LES; 3) an increase in acid production; 4) a violation of the evacuation of food from the stomach, etc.

To illustrate the above, we can cite some well-known examples: intra-abdominal pressure increases, particularly during forced exhalation during coughing or sneezing in asthma, cystic fibrosis (CF), bronchopulmonary dysplasia, and respiratory infections. The incidence of GERD also increases, especially in children with cardiac insufficiency or sliding esophageal hernia. Negative intrathoracic pressure increases, for example, during stridor or hiccups.

Furthermore, one should be aware of the potential side effects of theophyllines and glucocorticosteroids (GCS), widely used in the treatment of asthma. These drugs reduce LES tone, thereby causing a breakdown of the antireflux barrier. There is evidence that oral administration of theophyllines and beta-2-adrenergic agonists reduces LES tone and stimulates hydrochloric acid secretion, while inhaled systemic GCS and theophyllines do not alter LES tone. It is also known that when inhaled GCS are administered without a spacer, 80% of the inhaled dose reaches the stomach, which adversely affects both LES tone and gastric motility.

One of the most important respiratory complications of GERD is asthma. According to various authors, pathological GERD is found in 20-80% of children with asthma (according to

our data, 65%). The number of reflux episodes often correlates with the severity of respiratory symptoms, and individual episodes of GERD directly coincide with asthma attacks.

There are indications in the literature that with adequate therapy for GER, the frequency of bronchospasm symptoms decreases. The most characteristic symptom of GER-dependent broncho-obstruction is a nocturnal cough due to prolonged acidification of the esophagus in a horizontal position, decreased salivation and clearance of the esophagus, as well as the development of esophagitis.

GERD-related bronchial obstruction may be suspected in children with:

1) attacks of coughing and/or suffocation, mainly at night; after a large meal;

2) a proven combination of respiratory and “upper” dyspeptic symptoms (belching, heartburn, regurgitation , etc.);

3) positive effect of antireflux therapy

4) signs of resistance to adequate basic therapy;

5) non-atopic variants of bronchial asthma.

The system of evidence in such cases is built on the basis of a reliable diagnosis of GER using all available methods (both purely gastroenterological and pulmonological and allergological).

In our opinion, the optimal method for examining children is to use the following specialized methods:

1. FGDS with targeted biopsy, histological examination of biopsy specimens , helpil test (a type of urease test for determining Helicobacter infection) pylori), contrast X-ray of the esophagus and stomach, daily pH monitoring , acid test (Bernstein test in any modification).

2. Spirometry; bronchoprovocation tests with physical activity, saline, histamine, methacholine ; pulse oscillometry ; chest radiography (as indicated); allergy testing (general and specific: IgE , RIST and RAST).

Currently, numerous endoscopic classifications of GER in adults exist (the Savary - Miller classification, the Los Angeles classification, etc.). However, none of them are adequately adapted for children. The modified version of G. Tytgat's classification presented below takes into account the severity of both morphological and motor abnormalities associated with GER.

The system of endoscopic signs of GER in children (according to G. Tytgat as modified by V. F. Privorotsky et al ., 2002).

Grade 0. There are no signs of damage to the esophageal mucosa.

Grade 1. Moderate focal erythema and/or friability of the mucosa of the abdominal esophagus.

Grade II. Ditto + total hyperemia of the abdominal esophagus with focal fibrinous plaque and possible appearance of isolated superficial erosions, often linear, located at the apices of mucosal folds.

Grade III. Ditto, plus inflammation spreading to the thoracic esophagus. Multiple (sometimes confluent) erosions, located circularly . Increased mucosal contact fragility is possible.

Stage IV. Esophageal ulcer, Barrett's syndrome , esophageal stenosis.

A. Moderate motor impairment in the area of the lower extremity (Z-line elevation up to 1 cm), short-term provoked subtotal (one

from the walls) prolapse to a height of 1-2 cm, decreased tone of the LES.

B. Distinct endoscopic signs of esophageal reflux, total or subtotal provoked prolapse to a height of more than 3 cm with possible partial fixation in the esophagus.

B. The same + pronounced spontaneous or provoked prolapse above the crura of the diaphragm with possible partial fixation.

Histological examination of biopsies SO of the esophagus and/or stomach, as well as X-ray examinations of the upper gastrointestinal tract are carried out according to standard methods and are described in the relevant guidelines.

The "gold standard" for determining pathological GER is 24-hour pH monitoring , which not only identifies the presence of reflux but also determines its nature (physiological or pathological). The study is performed using the Gastroscan-24 or Sinectics-medical AB devices. Currently, there are no uniform standards for definitively establishing a link between GER and bronchial obstruction. Documenting this requires equipment that simultaneously assesses respiratory function and records GER. Therefore, various indices are currently being developed to assess the presence of such a link.

The principle of the acid-perfusion test (Bernstein test) is to evaluate the child’s subjective sensations under artificial Acidification of the lower third of the esophagus with a 0.1% hydrochloric acid solution (or pure lemon juice, the pH of which is known). The test is considered positive if the child experiences chest pain or heartburn within the first 3 minutes of the test.

The basic method for assessing lung function is spirometry (a test of external respiratory function). The test is performed using a computerized spirograph, which measures airflow using flow meters, and calculates volume using the integrated flow.

Bronchoprovocation tests allow us to determine the reactivity of the tracheobronchial tree. Tests with methacholine and histamine are performed only during remission. These irritants directly affect the bronchi, which contain smooth muscle fibers. This leads to contraction of the smooth muscle fibers , stimulation of cholinergic activity, and increased vascular permeability. At low concentrations, these drugs have no side effects and do not cause prolonged broncho-obstructive reactions.

Pulse Oscillometry allows us to determine the components of total respiratory resistance (frictional and reactive resistance in a certain frequency range).

The diagnostic algorithm above makes no mention of esophageal scintigraphy (to confirm reflux) or lung scintigraphy (to detect aspiration of gastric contents into the lungs) using technetium-labeled food. The limited data available in the literature indicate the low sensitivity of this method.

Treatment of gastroesophageal Treatment of reflux disease is aimed at improving the motility of the upper gastrointestinal tract, suppressing the acidity of gastric juice, and eliminating factors that lead to increased intra-abdominal pressure or decreased tone of the lower esophageal sphincter.

Much attention is paid to the lifestyle and diet of patients. Patients suffering from GERD should avoid foods that irritate the mucous membrane (hot sauces, seasonings, onions, garlic, peppers, ketchup, acidic fruit juices), reduce the tone of the lower esophageal sphincter and/or slow gastric emptying (alcohol, coffee, chocolate, cakes, pastries, fatty foods, lard, margarine), as well as foods that

Increased gas production, leading to increased intra-abdominal pressure. It is important to explain that overeating, eating in a hurry, before bed, or at night will worsen symptoms. Small meals are beneficial. Food should be chewed thoroughly, and the temperature of food should not be too hot or too cold, but should remain around 37-38°C. The last meal should be at least 2 hours before bedtime. The head of the bed is raised 15 cm using supports. This helps improve esophageal clearance by utilizing the force of gravity on the esophagus and stomach. It is important to note that raising the head alone (for example, with a high pillow) is not permitted, as this increases intra-abdominal pressure, which aggravates reflux . Avoid physical

exercise after meals, tight belts, and tight clothing, as these can increase intra-abdominal pressure and contribute to the reflux of stomach contents into the esophagus.

The main groups of drugs used in the treatment of GERD are prokinetics , acid suppressants, and antacids.

Of the prokinetics, blockers are used . Dopamine receptors— metoclopramide , domperidone , and cisapride . Cisapride is no longer used due to the risk of cardiac arrhythmia. Metoclopramide penetrates the blood-brain barrier and blocks dopaminergic receptors in the thalamus, hypothalamus, and brainstem. Its antiemetic effect is due to inhibition of the vomiting center and increased propulsive peristalsis of the stomach due to blocking peripheral dopamine receptors. Metoclopramide is not recommended for use in children due to the potential for side effects such as extrapyramidal disorders, drowsiness, fatigue, and anxiety, all associated with metoclopramide's penetration through the blood-brain barrier. Hyperprolactinemia may develop .

Another drug that blocks dopamine receptors is domperidone . It is free of the side effects of metoclopramide and is currently the drug of choice for treating children with GERD. Domperidone blocks only peripheral dopamine receptors in the stomach and duodenum, which leads to increased tone and peristalsis of the upper gastrointestinal tract. Antroduodenal coordination is improved, gastric contractility is enhanced, and thereby gastric emptying is accelerated. In addition to tablet forms, the drug is available in syrup form, which facilitates its use in pediatric practice. Domperidone is prescribed at a dose of 2.5 mg per 10 kg 3 times a day for 1-2 months. Side effects are rare (0.5-1.8% of patients). These include headache, general

Fatigue. Extrapyramidal disorders and hyperprolactinemia are extremely rare.

Proton pump inhibitors (PPIs) or H2 blockers are used to reduce gastric acidity . However, numerous studies have shown that the use of proton pump inhibitors in the treatment of gastroesophageal Reflux disease is significantly more effective. Inhibition of the proton (acid) pump is achieved by inhibiting the H+/ K +- ATPase of parietal cells. The antisecretory effect in this case is achieved not by blocking any receptors (H2-histamine, M-cholinergic) involved in the regulation of gastric secretion, but by directly influencing the synthesis of hydrochloric acid.

Proton pump inhibitors are substituted benzimidazole derivatives . Being weak bases by nature, they accumulate in the tubules of parietal cells, where pH values are lowest (1.0-0.8). In the tubules of parietal cells, the benzimidazole derivatives are converted into tetracyclic

sulfenamide . Sulfenamide covalently binds to the cysteine groups of the proton pump via disulfide bonds , which leads to inhibition of the enzyme and suppression of acid secretion. The resulting Sulfenamide penetrates membranes poorly because it is a cation. This ensures selective accumulation of the active form of the proton pump inhibitor in the secretory canaliculi of parietal cells. By acting at this stage, proton pump inhibitors cause maximum inhibition of acid formation . In children , omeprazole and rabeprazole are used at a dose of 10 to 20 mg once before dinner. The dose is selected and adjusted individually under pH monitoring .

We monitored the effectiveness of the proton pump inhibitor rabeprazole . 30 children aged 7-15 years with gastroesophageal reflux disease were examined. reflux disease. Seventeen children received rabeprazole at a dose of 20 mg once a day, 13 children received rabeprazole at a dose of 10 mg once a day. Monitoring was carried out by studying intragastric acidity using the Gastroscan 5-M and Gastroscan-24 devices (NPO Istok-sistema, Russia) . r H-metry was performed on the 7th-14th day of taking the drug and after 1-3 months.

The pronounced positive dynamics on the first day of therapy against the background of taking rabeprazole is due to the peculiarity of its metabolism.

During the 24-hour pH monitoring, the following parameters were assessed: basal acidity, food buffering action, and the presence of GER were assessed from 9:00 a.m. to 9:00 p.m. At 9:00 p.m., the patient was given the medication and the latent period was determined . and the period of action. The criterion for a patient's resistance to PPIs is the absence of an increase in gastric pH above 4 units. The following data were obtained. A plateau of 4 or higher in the corpus was achieved in four patients after the first single dose. The remaining patients experienced intermittent increases in corpus pH , but no stable plateau was achieved. The latency period averaged 5 hours. The duration of action did not exceed 5 hours.

Short-term pH-metry was performed 7-14 days after the start of taking the drug and 2-6 months after the end of taking it. The effectiveness of the drug was assessed.

Of the children receiving PPIs for 7-14 days, 8 had a body pH of 4 or higher. In 15 children, the body pH rose above 4 intermittently. In 10 children, the body pH remained below 1.5 throughout the day; the rest had normoacidity .

Follow-up after 2-6 months revealed no sustained effect in 16 patients. In 2 patients, body pH remained at 4-6 for 2 months. Eighteen children had normal acidity (body pH 1.5-2). However, only 7 patients presented any complaints.

ISSN: 2053-3578 I.F. 12.34

Thus, despite the proven effectiveness of PPIs—in relieving clinical symptoms within 1-3 days of starting treatment—there is individual patient sensitivity to PPIs. This can only be assessed by pH monitoring . Conducting a daily pH-metry allows you to select the dose and drug in each specific case.

Gel- based antacids are used as a coating agent . By coating the stomach contents, the antacid prevents injury to the mucous membrane during reflux into the esophagus, forming a protective film on its surface. Antacids for gastroesophageal reflux disease is used immediately after meals 3-4 times a day.

We observed 180 children with chronic nonspecific lung diseases (CNLD). The first group consisted of 120 children with bronchial asthma (74 women, 46 men); the second group consisted of 36 children with CF (12 women, 24 men); the third group consisted of 24 children with recurrent bronchitis (16 women, 8 men). The age of the examined children ranged from 6 to 18 years. The overwhelming majority of children in all groups (85%) showed clinical and endoscopic signs of CGD.

Among patients with bronchial asthma, 24 children (20%) were diagnosed with a severe course of the disease (in all cases, bronchial asthma was atopic , infection-dependent), 80 children (66%) had a moderate course (68 had a mixed variant, 12 had an atopic variant), and 16 children (14%) had a mild course (13 had a mixed variant, 3 had an atopic variant).

Severe course of CF was observed in 11 (30%) children (all had a mixed form), moderate course in 14 (40%) people (9 had a mixed form, 5 had a pulmonary form), mild course in 11 (30%) children (5 had a mixed form, 3 had a pulmonary form,

3 - intestinal form).

Distinguishing between different clinical patterns of recurrent bronchitis is not common in modern pulmonology. All children with recurrent bronchitis were examined during the peak of their exacerbation.

Among patients with asthma, complaints characteristic of GER were noted in 58 children (49%). Intragroup analysis revealed such complaints in 19 children (78%) with severe asthma, in 34 children (43%) with moderate asthma, and in 5 children (31%) with mild asthma.

In patients with CF, similar complaints in different combinations were found in 31 children (86%), including all children with a severe form of the disease, 12 children (86%) with a moderate course of CF, and 8 children (73%) with a mild form.

In 12 children with recurrent bronchitis (50%), complaints characteristic of GER were noted.

According to the results of the instrumental examination, GER was detected in almost 2/3 (65%) of the examined children with bronchial asthma (regardless of the severity) (the vast majority of them had an endoscopically positive variant and only 4% had an endoscopically negative variant).

Among patients with MV, GER was diagnosed in 31 children (88%), and all had an endoscopically positive variant.

In 9 children (41%) with recurrent bronchitis, an endoscopically positive variant of GER was detected.

According to the results of the intragroup analysis, GER of varying severity was detected in 68% of children with severe bronchial asthma (20% of whom had GER grades 2-3), in 30% of children with moderate bronchial asthma (17% of whom had GER grades 2-3), and in 50% of children with mild bronchial asthma (5% of whom had GER grades 2-3). According to the results of FGDS and X-ray examination, sliding hernia of the esophageal orifice of the diaphragm was detected in every sixth child with severe bronchial asthma and in every tenth child with a milder course of the disease.

Esophagitis of varying severity was detected in the majority of patients with CF (88%). Endoscopically positive GERD was detected in all patients with severe CF, in 54% of patients with moderate CF, and in 45% of children with mild CF.

Of particular interest, in our opinion, are the following data: in 90% of children with bronchial asthma and sliding hernia of the esophageal orifice of the diaphragm, pulse oscillometry data revealed significant changes in the reactance and frictional resistance parameters, which indicates non-uniform ventilation of various areas of lung tissue. In all patients with this

Based on the results of bronchoprovocation tests, the III hyperreactivity threshold was determined in the group .

Conducting a correlation analysis of spirometry data in patients in the compared groups was difficult due to objective reasons: the children were examined at different stages of their illness. However, spirometry is certainly a screening method for children with COPD and is necessary for assessing the severity of the disease and developing optimal treatment programs.

According to the data of bronchoprovocation tests (with histamine and methacholine), the III hyperreactivity threshold was detected in all children with severe bronchial asthma, in 20% of children with moderate bronchial asthma, and in 2% of children with mild bronchial asthma (the differences between the severe form and the other two are significant).

In patients with severe CF, the maximum (third) threshold of hyperreactivity was diagnosed in 66% of children.

In the group of children with recurrent bronchitis III, the hyperreactivity threshold was not diagnosed.

Based on the above, the following conclusions can be drawn:

1) GER of varying degrees of severity was detected in 65% of children with bronchial asthma, 88% of children with CF, and 50% of children with recurrent bronchitis;

2) a relationship was noted between the degree of expression of GER and the severity of COPD (in severe forms of bronchial asthma and CF, high-grade GER was detected significantly more often (^ < 0.05) than in patients with milder forms of these diseases and recurrent bronchitis);

3) in the overwhelming majority of children with COPD who have GER, an endoscopically positive variant of the latter has been proven;

4) the most unfavorable is the combination of bronchial asthma or CF (regardless of severity) with sliding hernia of the esophageal opening of the diaphragm and reflux esophagitis ; in these children, positive tests with histamine and methacholine with a high threshold of hyperreactivity are detected reliably more often (^ < 0.05) than in children with recurrent bronchitis , and serious disturbances are also determined according to pulse oscillometry data ;

5) no connection was found between Hp infection and the degree of esophageal damage in children with COPD;

6) all cases of GERD-associated bronchial asthma were identified in children with atopic and/or infection-dependent forms of this disease;

7) all children with CF should be prescribed a special instrumental examination for the early detection of esophageal damage.

Despite significant advances in recent years, many unknowns remain in the field of respiratory manifestations of GERD in children. In particular, the role of alkaline GERD and other non-acidic stimuli in the genesis of respiratory problems remains unclear.violations, not all aspects of the reflex mechanism of reflux-dependent laryngeal and bronchial obstruction .

ISSN: 2053-3578 I.F. 12.34

Conclusion:

GERD in children is a prevalent and multifactorial disease with significant esophageal and extraesophageal complications, particularly involving the respiratory system. The severity of bronchopulmonary manifestations correlates with the degree of reflux and esophagitis, emphasizing the importance of early diagnosis and comprehensive evaluation. Effective management relies on a combination of lifestyle modifications, pharmacological interventions, and careful monitoring of both gastrointestinal and respiratory symptoms. The integration of gastroenterological and pulmonological assessments is essential for optimizing treatment outcomes and preventing long-term complications in pediatric patients.

LITERATURE

1. Beytuganova I. M., Chuchalin A. G. Reflux-induced bronchial asthma // Rus . med . zhurn. — 1998. — V. 6, No. 17. — P. 1102-1107.

2. Zernov N. G., Sashenkova T. P., Ostroukhova I. P. Diseases of the esophagus in children. - M.: Medicine, 1988.

3. Privorotsky V. F., Luppova N. E. Acid-dependent diseases in children. - St. Petersburg: St. Petersburg - MAPO, 2002. - 89 p .

4. Khavkin A. I., Privorotsky V. F. Gastroesophageal Reflux disease // Acid-dependent conditions in children. - M., 1999. - P. 45-57.

5. Fedoseev G V. Mechanisms of bronchial obstruction. - St. Petersburg: MIA, 1995. - 333 p.

This situation occurs with a fourfold prevalence. Answers to these questions may become the topic of future publications.

Published with permission from the administration of the Russian Medical Journal.

6. Benjamin Stanley B., Kern Deschner . Exstraesophageal manifestations of gastroesophageal reflux // Am. J. Gastroenterol . - 1989. - Vol. 84, No. 1. - P. 1-5.

7. Ekstrom T., TibblingL . Esophageal acid perfusion airway function and symptoms in asthmatic patients with marked bronchial hyperreactivity // Chest. - 1989. - Vol. 96, No. 5. - P. 963.

8. Pediatric gastrointestinal motility disease (Ed. Wyllie R., Hyams JS). - Philadelphia, 2000. - P. 164-187.