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КЛИНИЧЕСКИЙ СЛУЧАЙ ДИАГНОСТИКИ ЭОЗИНОФИЛЬНОГО ЭЗОФАГИТА У ПАЦИЕНТА, ДЛИТЕЛЬНОЕ ВРЕМЯ СТРАДАЮЩЕГО БРОНХИАЛЬНОЙ АСТМОЙ ТЯЖЕЛОГО ТЕЧЕНИЯ И ПОЛУЧАЮЩЕГО ТЕРАПИЮ ГИБП (ДУПИЛУМАБ)

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Clinical Case of Diagnosis of Eosinophilic Esophagitis in A Patient Who Was Suffering for A Long Time with Severe Bronchial Asthma and Receiving Therapy with A Gerd (Dupilumab)

Резюме

Представлен клинический случай диагностики эозинофильного эзофагита (ЭоЭ) у пациента, длительное время страдающего бронхиальной астмой (БА) тяжелого течения и получающего терапию генно-инженерными биологическими препаратами (ГИБП): Дупилумаб. Трудности диагностики связаны с одной стороны, с необходимостью гистологической верификации диагноза, с другой стороны, с гетерогенностью проявлений заболевания. Частое сосуществование ЭоЭ и других аллергических заболеваний подчеркивает единство патогенетических путей, объединенных реакциями мукозального иммунитета. Приведенный нами клинический случай демонстрирует возможность диагностики ЭоЭ в отсутствии характерных жалоб и эндоскопической картины у пациента, имеющего поливалентную аллергию и длительный анамнез тяжелой БА. Своевременное применение эффективной терапии способствует предотвращению ремоделирования стенки пищевода с развитием стриктур, которые могут значительно ухудшать качество жизни пациента.

Ключевые слова: *Бронхиальная астма, гастроэзофагеальная рефлюксная болезнь, мукозальный иммунитет, Т2-воспаление; эозинофильный эзофагит*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

We presented a clinical case of diagnosing eosinophilic esophagitis in a patient suffering from severe asthma for a long time and receiving therapy with genetically engineered biological drugs: Dupilumab. Difficulties in diagnosis are associated, on the one hand, with the need for histological verification of the diagnosis, and on the other hand, with the heterogeneity of the manifestations of the disease. The frequent coexistence of EoE and other allergic diseases emphasizes the unity of pathogenetic pathways united by mucosal immune reactions. Our clinical case demonstrates the possibility of diagnosing EoE in the absence of characteristic complaints and endoscopic picture in a patient with polyvalent allergies and a long history of severe asthma. Timely use of effective therapy helps prevent remodeling of the esophageal wall with the development of strictures, which can significantly worsen the patient's quality of life.

Key words: *Bronchial asthma, gastroesophageal reflux disease, mucosal immunity, T2 inflammation, eosinophilic*

Conflict of interests

The authors declare no conflict of interests

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Conformity with the principles of ethics

The patient consented to the publication of laboratory and instrumental research data in the article « Clinical Case of Diagnosis of Eosinophilic Esophagitis in A Patient Who Was Suffering for A Long Time with Severe Bronchial Asthma and Receiving Therapy with A Gerd (Dupilumab)» for the journal «The Russian Archives of Internal Medicine» by signing an informed consent

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BA — bronchial asthma, EoE — eosinophilic esophagitis, GEBD — genetically engineered biologic drugs, PPI — proton pump inhibitors, SGCS — systemic glucocorticosteroids, IGCS/LABA — inhaled glucocorticosteroids / long-acting beta2-agonists, LAAC — long-acting anticholinergics, FGS — fibrogastroscopy, GERD — gastroesophageal reflux disease, SPG — spirogram, BAS — biologically active supplement

Introduction

The incidence of eosinophilic esophagitis (EoE) in bronchial asthma (BA) patients varies from 12 % to 68 % [1]; however, the pathogenetic relation between the two conditions has become obvious. The common pathogenic links include susceptibility to atopic disease, development of Th2-immune response, and effects of food and airborne allergens [2]. Being a Th2-associated disease, EoE can also be discussed as part of an atopic march [3]. A separate clinical entity of EoE has appeared relatively recently; and despite availability of a number of regulatory documents, principles of EoE diagnosis and treatment are still being actively developed and improved [2].

EoE can affect patients of any age, mostly men. The number of EoE cases grows from year to year; on the one hand, it follows the tendency towards higher incidence of allergic conditions in general; and on the other hand, it can be a result of better awareness of clinicians about this pathology. This systemic review demonstrates that asthma is more common in EoE patients vs. controls. These patients more often have polyvalent hypersusceptibility and higher IgE levels [1]. The incidence of EoE is as high as 81.7 to 118.4 cases per 100,000 people [2, 4]. It is worth noting that, among inflammatory esophagus conditions, EoE takes the second place in terms of the incidence after peptic esophagitis [4]. There are currently no epidemiological data on the incidence of EoE in Russia.

EoE is an immune-mediated oesophagus disease, which is characterised by marked eosinophilic infiltration of esophageal mucosa. The antigen can contact immune competent cells in mucosa in genetically predisposed individuals with mutated tight junction protein genes, which ensure epithelial integrity. Genetic disorders also affect the immune system factors, the increased activity of which causes polarised immune response (T2 inflammation); and the most important is hyperexpression of the gene, which is responsible for the synthesis of thymic stromal lymphopoietin (TSLP) [2]. Therefore, exposure to food allergens or airborne allergens leads to activation of dendritic or mast cells triggering T2 inflammation [5–6]. Cytokines IL-4, IL-5, and IL-13, synthesised by activated Th2 lymphocites, facilitate

eosinophil involvement in the inflammation site and their activation. Eosinophils release cytotoxic proteins and other inflammatory mediators, which cause tissue damage and, ultimately, esophageal wall remodelling. The consequence of fibroblast and smooth muscle cell involvement in the process is fibrogenesis and hyperplasia, also mediated by IL-4, IL-5, and IL-13 and impairing esophageal wall architectonics. As a result, persistent inflammation can lead to esophageal strictures, which have a great impact on the patients' quality of life and, in some cases, require surgery [2].

Challenges with EoE diagnosis are associated with the fact that eosinophilic infiltration of mucosa is not a pathognomonic sign of a desease. Eosinophilia can be observed in patients with peptic esophagitis, a number of autoimmune conditions, and gluten-sensitive enteropathy. The key role in the disgnosis is played by histologic examinations, for which reason the diagnostic criteria, called EoE-specific histologic scoring system, or EoEHSS, have been developed [7].

EoE is also known to be a heterogenic disease, both in terms of the array of clinical manifestations (from lack of symptoms to dysphagia, which significantly deteriorates the patient's quality of life) and response to therapy. Interestingly, some patients' clinical condition can improve with an elimination diet; other patients achieve remission after proton pump inhibitor (PPI) therapy; and some cases require initiation of target therapy, namely genetically engineered biologic drugs (GEBD) [2].

Here is a clinical case of eosinophilic esophagitis diagnosis in a patient, who had severe bronchial asthma (BA) for a long time and was treated with genetically engineered biologic drugs (GEBD): dupilumab.

Patient G., born in 1952, was admitted to the allergology ward on January 19, 2024, complaining of choking spells (approx. two times at night and 5 to 6 times during the day), triggered by physical exercise, strong smells, cigarette smoke, allergens; shortness of breath after low-intensity activities (climbing stairs to the second floor); attack-like cough with yellow thick discharges; heartburn; nagging pain in the left lumbar region, which gets more intense at night; occasional nasal blockage, especially in the morning and at night; and decreased sense of smell.

He did not have any signs of atopic disease as a child. The patient does not smoke and has never smoked. He is hypersensitive to captopril (in the form of Quincke's edema). Since 1996, the patient has experienced signs of rhinitis, especially in spring and summer; he occasionally took antihistamines. BA was diagnosed in 2001. Severe BA required frequent hospital admissions and 3–4 courses of systemic glucocorticosteroids (SGCS) annually. On the onsent of the condition, the patient was prescribed high doses of inhaled glucocorticosteroids + long-acting beta2-agonists (IGCS+LABA); in 2009, a third controller medication belonging to long-acting anticholinergics (LAAC) was initiated. Also, since asthma symptoms could not be controlled and the patient developed steroid resistance, a baseline therapy with prednisolone 5 mg/day was added. The patient took his medications regularly, demonstrated correct inhalation technique and proper compliance. Since the patient complained of heartburn, he underwent annual fibrogastroscopy (FGS); there were no signs of peptic esophagitis; in 2016, numerous erosions in the antral stomach were found. Because SGCS were required, the patient constantly used PPIs.

In October 2020, when the patient experienced severe BA, steroid resistance, concomitant nasal obstruction, polysensitization to domestic, epidermal and plant allergens, lack of control despite the therapy corresponding to step 5 of the Global Initiative for Asthma (GINA 2020), taking into account patient's compliance and absense of high eosinophil levels, GEBD: dupilumab was added at an initial dose of 600 mg s/c in the shoulder, with subsequent adjustment to 300 mg s/c fortnightly. After two years of therapy, the patient's condition improved: fewer choking spells during the day and at night, fewer hospital admissions, better nasal breathing and sense of smell, as well as better pulmonary function (Table 1). SGCS were discontinued during the first year of GEBD therapy.

In December 2023, the patient had community-acquired pneumonia, after which his BA aggravated for the first time since GEBD initiation, and inpatient treatment was required. The patient was admitted to the allergology ward for baseline therapy correction and develoment of a plan for further therapy with the genetically engineered biologic drug.

Table 1. *Spirogram indicators in dynamics in 2020-2021*

Indicators of respiratory function	At the time of initiation of therapy		After 12 months of therapy	
	before the salbutamol test	after the salbutamol test	before the salbutamol test	after the salbutamol test
FEV_1, %	73,7	110,5	119,4	122,8
FVC, %	88,7	110,5	114,6	118,3
FEV_1/FVC	64,75	77,88	80,07	79,43

Notes: FEV_1 — forced expiratory volume in 1 second, FVC — forced vital capacity

Table 2. Immunogram

Immunogram indicators	Actual values	Units of measurement	Reference values
Determination of total IgA	2.9	мг/мл	(0.8 — 4.0)
Determination of total IgM	0.6	мг/мл	(0.4 — 2.0)
Determination of total IgG	**5.2** <	мг/мл	(5.3 — 16.5)
Circulating immune complexes	14		(0 — 100)
T-lymphocytes (CD3+CD19-)	**56.00** <		(61.00 — 85.00)
B-lymphocytes (CD3-CD19+)	**33.20** >		(7.00 — 17.00)
T- helpers (CD3+CD4+)	48.20		(35.00 — 55.00)
T- cytotoxic (CD3+CD8+)	**7.90** <		(19.00 — 35.00)
NK-cells	8.70		(8.00 — 17.00)
T-NK cells (CD3+CD16+56+)	1.40		(0.50 — 6.00)
T- activated (CD3+HLADR+)	2.50		(0.50 — 6.00)
IRI (immunoregulatory index)	**6.10** >		(1.50 — 2.60)

Upon examination, the patient's condition is moderately severe; average height and weight: 75.0 kg, 172 cm; body mass index 25.4 kg/m2; skin is clear, moderately wet and normally coloured.

Nasal breathing is slightly obstructed on both sides; chest shape is unremarkable; percussion sound is clear and comes from the lungs, in projection of all pulmonary fields; breathing is harsh, with moderate dry rale in all fields, worsening with forced breathing; respiratory rate 19 per minute; oxygen saturation 95 %. The heart rhythm is normal, with muffled heart tones; no murmurs; heart rate: 81 bpm; pulse 81 bpm; blood pressure: 120/80 mm Hg on both arms; abdomen is not dilated and participates in breathing, soft, painless when palpated in all sections; the liver is painless along the costal margin; stool is normal. Urination is normal; no costovertebral angle tenderness on both sides; no edema.

Lab test results: white blood cells 13.96×109/L with neutrophilic shift (72.1 %, 10.07×109/L), Hb 136 g/L, platelets 211x109/L. No signs of lab activity: ESR 4 mm/h, CRP 2.4 mg/L. Total IgE 31 IU/mL (0–150). No high eosinophil levels were observed during the observation period.

The immunogram (Table 2) shows signs of B-cell sequence activation: decreased total T lymphocyte count 56 % (61–85 %), increased B lymphocyte count 33.2 % (7–17 %), T cytotoxic 7.9 % (19–35 %), immunoregulatory index (IRI) 6.1 (1.5–2.6), total IgG 5.2 g/L (5.3–16.5 g/L).

Imaging:

Comparative spirometry with salbutamol: forced expiratory volume during the first second (FEV1) 105.9–111.1 %, forced vital capacity (FVC) 113.7–114.3 %, FEV1/FVC 71.22–74.37. No signs of impaired pulmonary ventilation. Normal FVC. After inhalation of 400 μg of salbutamol, bronchodilation test was negative; FEV1 increased by 5 % (150 mL).

In order to rule out eosinophilic esophagitis, biopsy material was sampled from five sections (upper, middle and lower third of esophagus, stomach and duodenum):

No. 1. The sample contains small fragments of duodenum mucosa with moderately diffuse infiltration with lymphohistiocytic cells and moderate amount of neutrophils. Mucosa absorbs alcian blue in bottle cells.

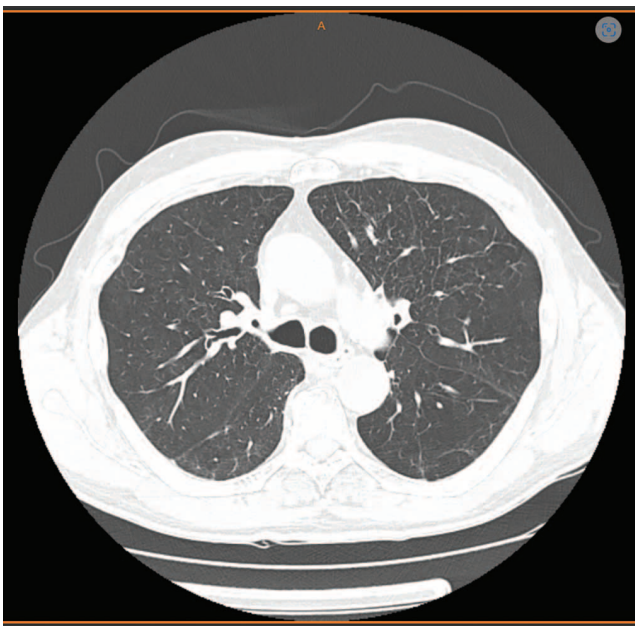
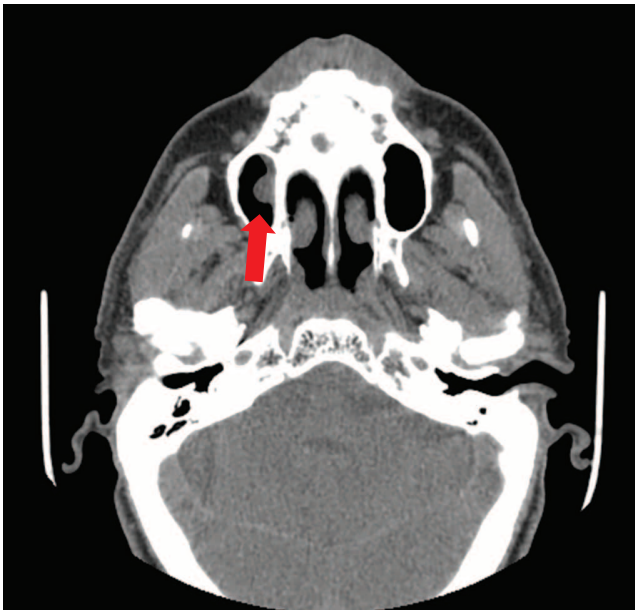


***Figure 1.** Multislice computed tomography of the chest organs (MSCT OC)*

Note. CT scan of the chest: The lungs are straightened. No infiltrative shadows. Unevenly distributed interstitial changes are present on both sides, with pulmonary emphysema, predominantly panlobular. A single discoid atelectasis is present in the right middle lobe. Large bronchi are patent. Single small mediastinal lymph nodes are present. There is no fluid in the pleural cavities.



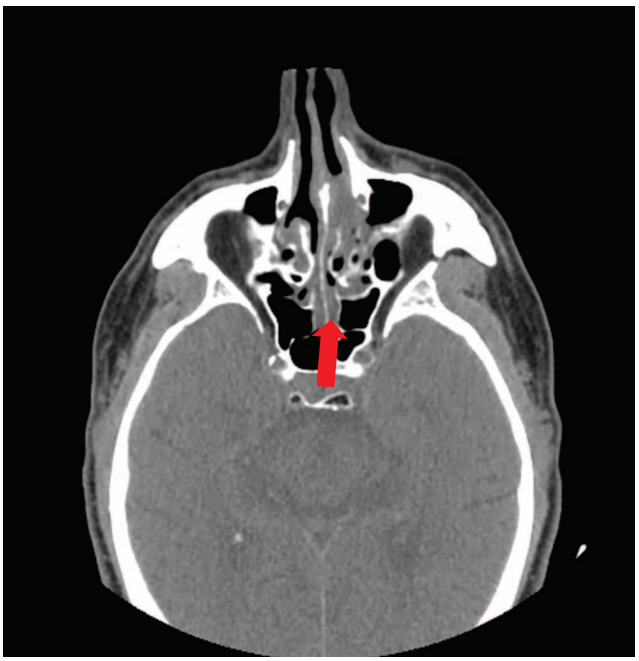


Figure 2. *Multislice computed tomography of the paranasal sinuses*

Note: The right maxillary sinus shows polypoid parietal lesions, and the ethmoidal labyrinth cells also show parietal lesions due to mucosal thickening. The frontal, left maxillary, and main sinuses are unremarkable. The nasal septum is moderately deviated in an S-shape.

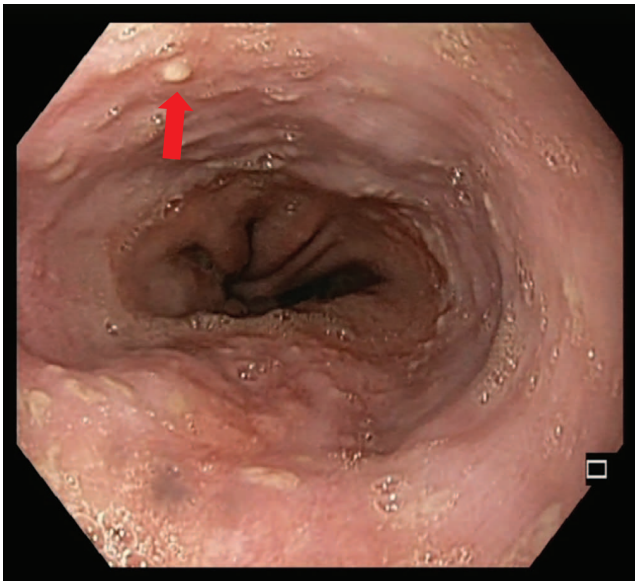


Figure 3a *— notes the presence of areas of whitish plaque in the esophagus*

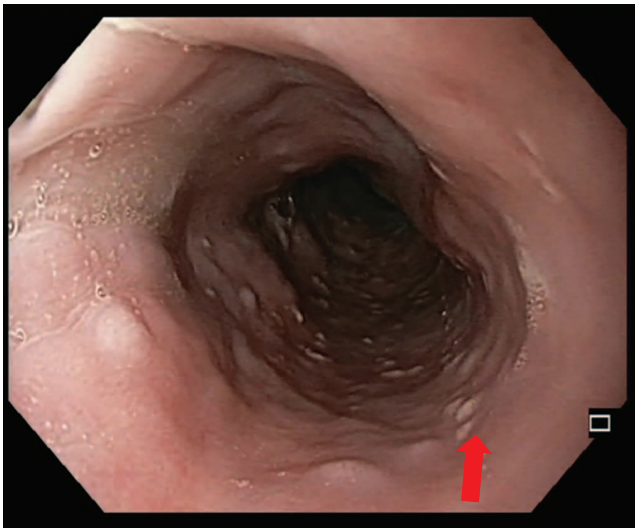


Figure 3b *— notes the presence of areas of whitish plaque in the esophagus*

Figure 3. *Esophagogastroduodenoscopy (EGD)*

No. 2. The sample contains fragments of stomach mucosa (metaplasia?). Lamina propria shows minor infiltration with neutrophils, lymphocytes, histocytes. Mucosa does not absorb alcian blue. H. pylori are not observed.

Nos. 3, 4. The samples contain layers of multilayer squamous epithelium without atypia and underlying tissue. The basal areas of epithelial layers have neutrophilic infiltration foci. Eosinophil count is 0–5 per HPF (x400 magnification). Mucosa does not absorb alcian blue.

No. 5. The samples contain layers of multilayer squamous epithelium without atypia and underlying tissue. The basal areas of epithelial layers have neutrophilic infiltration foci. Two fields of vision in basal areas have eosinophil accumulation of over 15 per HPF (x400 magnification). Mucosa does not absorb alcian blue.

Histology result: The morphological pattern may correspond to that of eosinophilic esophagitis; final diagnosis will be made taking into account clinical data, provided other pathology associated with eosinophil infiltration of esophageal mucosa has been ruled out.

Pursuant to recommendations of the Russian Gastroenterological Association [2], eosinophil infiltration and eosinophil density of ≥ 15 per high-power field (x400) at least in one biopsy sample is a criterion for diagnosing EoE. In this clinical case, a typical histology pattern was observed only in one biopsy sample from the upper third of esophagus, emphasising the importance of correct biopsy procedure (at least six different areas of mucosa).

The examination results were used to make the clinical diagnosis:

Primary disease: severe non-allergic bronchial asthma, moderate exacerbation, partially controlled. External respiration: stage 0. Respiratory insufficiency: stage 0. Firth step therapy: salmeterol/fluticasone 50/500 μg two puffs twice daily + tiotropium bromide respimat 2.5 μg two puffs in the morning + dupilumab 300 mg s/c fortnightly.

Severe persistent allergic rhinitis. Allergic intermittent conjunctivitis. Hypersensitivity to domestic, epidermal and plant allergens.

Concomitant pathology: IHD, postinfarction cardiosclerosis (2012). Exertional angina FC II. Stage III hypertensive disease, target BP value achieved, risk IV. Condition after ACVA in 2012 and 2015. Stage II discirculatory encephalopathy of mixed origin (hypertensive, atherosclerotic) with mild vestibulo-ataxic cognitive disorders. Symptomatic limb polyneuropathy, sensorimotor type.

Chronic pancreatitis, remission. Benign prostatic hypertrophy I. Chronic prostatitis.

Eosinophilic esophagitis.

Given the patient was diagnosed with EoE, a medical panel was summoned and the dose of the medication was corrected. Since 2020 the patient has been treated with GEBD because of the severe BA together with chronic rhinosinusitis polyposa; the dose was 300 mg fortnightly. In February 2024, the frequency was adjusted to once weekly as per the package insert and taking into account patient's weight. Therapy resulted in less intense esophageal symptoms; however, the patient did not come for repeated hospitalisation and no biopsy was performed over time.

Discussion

The majority of EoE cases described in Russian publications were observed in children. This is likely associated with the fact that in children the disease is often symptomatic. N. V. Bakulin at al. [8] describe a case of IgG4-associated esophagitis and EoE in a 17-year-old patient. The time from disease onset until diagnosis was 14 years; histologic samples needed to be re-examined in a specialised facility by several morphologists. The clinical presentation comprised progressing dysphagia and odynophagia; the patient had family history of atopic conditions and as a child he had atopic dermatitis. The peculiarity of this case was challenging interpretation of histology samples: initially, the morphological pattern was interpreted as low-degree intraepithelial neoplasia because of marked eosinophilic infiltration. When the samples were re-examined, typical signs of EoE were reported: more than 50 eosinophils per HPF, with clusters resembling eosinophil microabscesses. At the same time, immunohistochemistry revealed dense IgG4+ plasma cell infiltration in granulation tissue (50–70 IgG4-positive plasma cells per HPF, x400), meeting the criteria of IgG4-associated esophagus damage. A combination of the mentioned histological changes suggests common links between EoE and IgG4-associated conditions. It is assumed that IgG4 activation follows IgE-mediated response and can have a protective functions, blocking effects of IgE, including mast cell activation. There is a correlation between disease intensity and amount of intraepithelial and interepithelial eosinophils and IgG4+ plasmic cells [8].

In this clinical case, eosinophil infiltration was diagnosed only in the upper third of esophagus. It might be related to regular PPI therapy, facilitating reduction in the damaging action of hydrochloric acid on mucosa. One of the predictors of poor response to PPI therapy is the presence of significant IgG4+ infiltration in esophageal mucosa, therefore initial treatment with SGCS and GEBD should be considered these patients.

Unlike the observation in this article, EoE can be asymptomatic, with the typical endoscopic and histological pattern, as demonstrated by A. V. Paraskevova et al. [9]. In that case, EoE was diagnosed in a 52-year-old patient, who regularly took biologically active supplements (BASs) and who was diagnosed with typical signed of EoE during her preparation to surgery: circular mucosal thickening because of whitish papillary projections, longitudinal striation. Also, this case was remarkable because of the absence of a history of atopic conditions. Discontinuation of BASs and a course of PPIs resulted in complete endoscopic and histological remission; the patient did not have continuous recurrances typical of EoE; therefore, the condition was considered drug-induced (BAS) eosinophilic esophagitis. The same article describes a case of simultaneous gastroesophageal reflux disease (GERD) and EoE in one patient and analyses challenges with differential diagnosis of these conditions. It is well-known that gastroesophageal reflux disease can facilitate antigen interaction with immune competent cells and can trigger immune response, through mucosa damage, which causes EoE [10]. In a published clinical case, patient L., 68 years of age, had a long history of heartburn; he suffered from dysphagia and food regurgitation for three

years, and the patient sought medical help. In this case, peripheral blood eosinophilia was observed together with a typical endoscopic presentation (vertical sulci, mobile concentric rings, whitish effusion) and morphological signs of the disease.

Therefore, a histological examination of biopsy material taken from esophagus mucosa is essential for diagnosis of EoE. The diagnosis cannot be ruled out in the absence of typical clinical and endoscopic signs of the disease. This clinical case demonstrates possibilities of EoE diagnosis in the absence of typical complaints and endoscopic presentation in the patient with polyvalent allergy and a long history of severe BA.

EoE is treated with an elimination diet, PPIs, topical GCS and GEBD. The elimination diet means exclusion of products, which are known to cause allergic inflammation, i.e. milk, wheat, eggs, soya, nuts and fish (six-food elimination diet, SFED). SFED is a most well-studied approach, where the histological response is achieved in 67.9 % of patients vs. 13.3 % in placebo controls [11]. In a study by Frandsen L. T. et al. [12], high doses of PPIs completely eliminated symptoms in 68 % of patients and achieved histological remission in 49 % of EoE patients. A systematic review of eight double-blind placebo-controlled clinical studies of topical GCS (TGCS) therapy in 437 patients demonstrated that TGCS were associated with histological remission in 64.9 % of patients vs. 13.3 % of patients treated with placebo [11].

Given similar pathogenesis, GEBD therapy in patients with BA and EoE is justified. In their review Durrani S .R. et al. (2018) [1] conducted an analysis of GEBD efficacy in severe eosinophilic asthma when used to treat EoE: mepolizumab and reslizumab showed their efficacy for histological remission; however, their use did not affect EoE symptoms. Omalizumab did not have any beneficial effect. The most promising EoE therapy is dupilumab, since the drug has demostrated its beneficial effect both on clinical and histological remission. In May 2022, dupilumab was approved by the Food and Drug Administration (FDA) as an EoE therapy for patients over 12 years old weighing over 40 kg [13].

Dupilumab is a fully humanized anti-IL-4Rα antibody (IL-4Rα is a common receptor element for IL-4 and IL-13). IL-4 and IL-13 effects are implemented through signal transducer and activator of transcription (STAT) -6, which ensures signalling transduction to the cell nucleus. The key role in differentiation of naive Th lymphocytes into Th2 cells is played by IL-4. The shared objectives of IL-4 and IL-13 are ensuring eosinophil recruiting in mucous membranes, switching antibody synthesis to isotype IgE, dendritic cell activation, and maturation of M2 macrophages. IL-4 and IL-13 are also known to have the ability to inhibit expression of proteins, which ensure epithelial barrier integrity [14]. IL-13 is distinguished for its effects on tissue remodelling as a result of smooth muscle cell hyperplasia, collagen deposits and angiogenesis [15]. It is obvious that possible blocking of IL-4 and IL-13 effects ensures successful therapy of T2-associated conditions, including EoE. In this clinical case, the therapy resulted in positive clinical changes, improvement in pulmonary function, while dose adjustment contributed to less intense esophageal symptoms.

Conclusion

EoE is a T2-associated disease, which has been allocated a separate clinical entity quite recently and the incidence of which has been rising over the past decades. Challenges with disease diagnosis are associated with the need to verify diagnosis histologically, on the one hand, and with heterogenous disease presentaton, on the other hand. The fact that EoE is often associated with other allergic conditions underlines the existence of common pathogenetic components, unitied by mucosa-associated immunity reactions. This clinical case demonstrates possibilities of EoE diagnosis in the absence of typical complaints and endoscopic presentation in the patient with polyvalent allergy and a long history of severe BA. Timely initiation of efficient therapy facilitates prevention of esophageal wall remodelling and strictures, which can significantly deteriorate the quality of patient's life.

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