

Peculiarities of eosinophilic gastrointestinal diseases in children: combined eosinophilic lesions of the esophagus and stomach. A clinical case

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Abstract

Introduction. Eosinophilic gastrointestinal diseases are a group of chronic immune-mediated diseases of the gastrointestinal tract characterized by gastrointestinal symptoms and pathological eosinophilic infiltration of certain parts of the gastrointestinal tract in the absence of secondary causes of eosinophilia. Depending on the lesion level, eosinophilic esophagitis, eosinophilic gastritis, eosinophilic enteritis and eosinophilic colitis are distinguished. The absence of specific symptoms of eosinophilic gastrointestinal diseases complicate the diagnostic process, the key element of which is the morphological examination of biopsies of the mucous membrane of the corresponding part of the gastrointestinal tract to determine eosinophilic infiltration. To date, eosinophilic esophagitis in children is a well-defined disease with established recommendations, which facilitates the diagnosis and treatment of this pathology, which cannot be said about eosinophilic gastritis, which remains a clinical mystery with evidence based on limited individual case reports.

Presentation of the clinical case. The publication presents a clinical case of a 9-year-old girl with combined eosinophilic lesions of the esophagus and stomach. An endoscopic examination of the upper gastrointestinal tract performed on an outpatient basis, followed by a morphological assessment of biopsies of the esophageal mucosa, made it possible to diagnose eosinophilic esophagitis. A biopsy of the gastric mucosa and small intestine was not performed. The lack of stable positive dynamics against the background of prescribed treatment led to hospitalization and repeated examination, including morphological examination of the mucous membrane of the esophagus, stomach and intestines. The examination revealed eosinophilic infiltration not only of the mucous membrane of the esophagus ($>15/1\text{PH} \times 400$), but also of the antrum of the stomach (up to $67 \text{ eosinophils} \times 5\text{PH} \times 400$). As a result, eosinophilic esophagitis was diagnosed in combination with eosinophilic gastritis, for which diet therapy, proton pump inhibitors and glucocorticosteroids were prescribed, which led to an improvement in the child's condition, relief of pain and symptoms of dyspepsia, and the formation of clinical remission.

Conclusion. Despite the increasing number of studies and publications on the problem of eosinophilic gastrointestinal diseases, the criteria for diagnosis and treatment of such patients remain controversial. The presented clinical case demonstrated the difficulties that arise for a practicing physician in managing children with eosinophilic gastrointestinal diseases, in particular combined eosinophilic lesions of the esophagus and stomach, given the low symptoms and atypical clinical manifestations of this pathology, which necessitates further study of the mechanisms of development and improvement of the algorithm for the diagnosis and treatment of these diseases.

Keywords: eosinophilic gastrointestinal diseases, eosinophilic esophagitis, eosinophilic gastritis, children, teenagers

Conflict of interest

The authors declare no conflict of interest

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Особенности эозинофильных гастроинтестинальных заболеваний у детей: сочетанное эозинофильное поражение пищевода и желудка. Клинический случай

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Аннотация

Введение. Эозинофильные гастроинтестинальные заболевания — это группа хронических иммуноопосредованных заболеваний желудочно-кишечного тракта, характеризующихся гастроинтестинальными симптомами и патологической эозинофильной инфильтрацией определенных отделов желудочно-кишечного тракта при отсутствии вторичных причин эозинофилии. В зависимости от уровня поражения выделяют эозинофильный эзофагит, эозинофильный гастрит, эозинофильный энтерит и эозинофильный колит. Отсутствие специфических симптомов эозинофильных гастроинтестинальных заболеваний усложняют диагностический процесс, ключевым звеном которого является морфологическое исследование биоптатов слизистой оболочки соответствующего отдела желудочно-кишечного тракта с определением эозинофильной инфильтрации. На сегодняшний день эозинофильный эзофагит у детей является четко определенным заболеванием с установленными рекомендациями, что облегчает диагностику и терапию данной патологии, чего нельзя сказать об эозинофильном гастрите, который остается клинической загадкой с доказательствами, основанными на ограниченных отдельных отчетах о случаях.

Описание клинического случая. В публикации представлен клинический случай 9-летней девочки с сочетанным эозинофильным поражением пищевода и желудка. Проведенное в амбулаторных условиях эндоскопическое исследование верхних отделов желудочно-кишечного тракта с последующей морфологической оценкой биоптатов слизистой оболочки пищевода позволило диагностировать эозинофильный эзофагит. Биопсия слизистой оболочки желудка и тонкой кишки не проводилась. Отсутствие стабильной положительной динамики на фоне назначенного лечения привело к госпитализации и повторному обследованию, включающему морфологическое исследование слизистой оболочки пищевода, желудка и кишечника. Обследование выявило эозинофильную инфильтрацию не только слизистой оболочки пищевода ($>15/1\text{ПЗ}\times400$), но и антрального отдела желудка (до 67 эозинофилов/ $5\text{ПЗ}\times400$). В результате поставлен диагноз эозинофильного эзофагита в сочетании с эозинофильным гастритом, по поводу которых были назначены диетотерапия, ингибиторы протонной помпы и глюкокортикостероиды, что привело к улучшению состояния ребенка, купированию болевого синдрома и симптомов диспепсии, формированию клинической ремиссии.

Закключение. Несмотря на возрастание количества исследований и числа публикаций в целом — по проблеме эозинофильных гастроинтестинальных заболеваний, по-прежнему остаются дискуссионными критерии диагностики и лечение таких больных. Представленный клинический случай продемонстрировал возникающие сложности для практикующего врача в ведении детей с эозинофильными гастроинтестинальными заболеваниями, в частности сочетанного эозинофильного поражения пищевода и желудка, учитывая малосимптомность и атипичность клинических проявлений этой патологии, что диктует необходимость дальнейшего изучения механизмов развития и совершенствования алгоритма диагностики и лечения данных заболеваний.

Ключевые слова: эозинофильные гастроинтестинальные болезни, эозинофильный эзофагит, эозинофильный гастрит, дети, подростки

Конфликт интересов

Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи

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List of abbreviations:

EGI:	eosinophilic gastrointestinal diseases,
GIT:	gastrointestinal tract,
EoE:	eosinophilic esophagitis,
ЭоГ:	eosinophilic gastritis,
MM:	mucous membrane,
PPI:	proton pump inhibitors,
EGDS:	esophagogastroduodenoscopy,
EGDP₁₈:	eosinophilic gastritis transcriptome diagnostic panel,
eos/hpf:	the number of eosinophils in the field of view of a high-resolution microscope at a magnification of 400x, eos/0.27 mm ² hpf — the number of eosinophils in a high-power field, the standardized field area is 0.27 mm ² .

manifestations, namely: eosinophilic gastritis, (EoG), eosinophilic colitis, as well as eosinophilic enteritis, including eosinophilic duodenitis, eosinophilic jejunitis, and eosinophilic ileitis [2].

Eosinophilic esophagitis

Eosinophilic esophagitis is a chronic, slowly progressive immune-mediated disease of the esophagus characterized by marked eosinophilic inflammation of the esophageal mucosa (SM) and the development of submucosal fibrosis, which can clinically manifest as swallowing disorders (dysphagia, obstruction of the esophagus by food bolus, vomiting of swallowed food, etc.) [3].

The prevalence of EoE in different countries ranges from 5 to 10 people per 100,000 population [4]. Other data show that the incidence of EoE is 5.1 per 100,000 people per year, and the prevalence is about 29.5 cases per 100,000 population [5]. The results of a study of the frequency of EoE in children show 0.5–1 case per 1,000 children per year [6]. At the same time, there has been an increase in the incidence of EoE in recent years [7]. Environmental exposure in genetically predisposed individuals is a leading risk factor for the development of EoE [8]. The presence of atopic diseases, including food allergies, asthma, atopic dermatitis, and allergic rhinitis, is also important, as they are concomitant allergic conditions in 60–80% of patients with EoE [9]. The association between atopic conditions and EoE was demonstrated in a prospective study that found asthma in 48% and allergic rhinitis in 37% of patients with EoE [10].

INTRODUCTION

Eosinophilic gastrointestinal diseases (EGID) are rare inflammatory diseases of the digestive system. EGIDs represent a group of chronic immune-mediated gastrointestinal diseases characterized by gastrointestinal symptoms and pathological eosinophilic infiltration of certain areas within the gastrointestinal tract (GIT) in the absence of secondary causes of eosinophilia [1]. The nosological structure of EGID includes eosinophilic esophagitis (EoE) and a group with extraesophageal

The pathogenesis of EoE involves the formation of a Th2-mediated response followed by the release of cytokines (IL-4, IL-5, IL-13, IL-9) and the synthesis of eotaxin-3, resulting in eosinophilic infiltration of the esophageal mucosa, which causes the destructive action of proteolytic enzymes of eosinophils on the epithelium, the lamina propria of the esophageal mucosa, and the submucosal layer [3]. Although antigen stimulation leads to an inflammatory cascade of T cells, eosinophils, mast cells, and basophils, which ultimately triggers mucosal remodeling, the role of the epithelium in initiating this cascade and subsequent inflammation has only been partially studied [11].

The clinical manifestations of EoE are determined by the patient's age: in younger children, poor growth, developmental delays, vomiting, epigastric pain, refusal to eat, need for water during meals or slow eating, coughing during meals, while older children predominantly experience symptoms of dysphagia, food impaction, heartburn, chest pain, refusal to eat solid foods, and transition to eating pureed foods [9, 10].

Endoscopic examination is important for diagnosing EoE, assessing response to treatment, and long-term monitoring of disease activity [9]. Typical endoscopic features of EoE include edema (decreased vascularization), immobile esophageal rings ("trachealization" of the esophagus), white exudate, linear furrows, strictures, narrowing of the lumen, "crepe paper" changes in the mucosa (a sign of severe mucosal fragility), and mucosa that feels firm on biopsy ("pull sign" or "stretch sign") in patients with fibrosis [9, 12]. Previous studies have shown that with 6 or more biopsies, the diagnostic sensitivity approaches 100%, but from a practical standpoint, the preferred approach is to obtain at least 2–4 biopsies from 2 different areas of the esophagus (proximal and distal) while targeting areas of visible inflammation, if present [9]. It should be emphasized that esophageal biopsy sampling is of paramount importance for diagnosis and assessment of response to treatment, while endoscopic imaging of the disease relies on visual assessment and a validated endoscopic reference system (EREFS) [13]. EREFS classifies the five key signs of EoE (edema, rings, exudates, furrows, and strictures) according to severity [9]. Thus,

endoscopic assessment of activity complements histological and symptomatic data, providing a more comprehensive analysis of overall disease activity [13]. As an established diagnostic biomarker for EoE, it is important to quantitatively assess the peak eosinophil count from an esophageal biopsy. The threshold value for intraepithelial eosinophilic infiltration is considered to be 15 or more eosinophils in the field of view of a high-resolution microscope at 400x magnification (eos/hpf) or ≥ 60 eosinophils per 1 mm² [9]. Although eosinophils are traditionally considered indicator cells in EE, given that they are clearly visible with standard hematoxylin and eosin staining, they are only one of many cells that infiltrate the esophagus in EE. The role of T cells, basophils, mast cells, NK cells, and fibroblasts in EoE is currently being actively studied. [9, 14, 15].

Clinical guidelines for EoE treatment recommend the use of proton pump inhibitors (PPIs) and corticosteroids in combination with diet or as monotherapy [3]. A systematic review and meta-analysis of 33 studies, 11 of which were prospective and included 619 patients (including 188 children), reported that PPI use causes clinical response and histological remission (<15 eos/hpf) in 60.8% of adult patients and 50.5% of children, with twice-daily PPI use being more effective than once-daily use (55.9% vs. 49.7%) [17]. With regard to the use of PPIs in children with EoE, histological remission (from 44.5 to 12 eos/hpf) was achieved after administration of omeprazole 1 mg/kg twice daily for 8–12 weeks in combination with an elimination diet consisting of 4 foods (4FED; cow's milk, soy, egg, and wheat) [9]. A comparative study of the efficacy of budesonide and fluticasone administered over an 8-week treatment period showed that patients in each group had favorable histological, endoscopic, and symptomatic outcomes after treatment, with no significant differences between the groups: histological response (<15 eos/hpf) was achieved in 71% and 64% of the groups receiving budesonide and fluticasone, respectively, and post-treatment dysphagia and EREFS scores were also similar [18]. These data allow budesonide and fluticasone to be recommended as initial therapy with topical steroids for EoE [9]. For patients with EoE who do not respond to PPI therapy, biological therapy

with dupilumab (a monoclonal antibody directed against the alpha subunit of IL-4 receptors, IL-13) in the form of a subcutaneous injection at a dose of 300 mg weekly, provided that the patient is over 12 years of age and weighs more than 40 kg [19]. For patients aged 1–11 years, the dose of dupilumab is selected according to body weight, but such use is not registered in the Russian Federation [20].

In addition to drug therapy, great attention is paid to the nutrition of patients with EoE. According to current clinical guidelines, it is preferable to prescribe a 6FED elimination diet, which excludes dairy products, wheat, soy, eggs, nuts, and fish/shellfish from the diet [3]. Despite 6FED effectiveness, problems remain in the form of the restrictive nature of this diet and the need to perform a series of endoscopies to identify a specific food trigger from the six proposed for exclusion, which in turn prompted research into less restrictive elimination diets and an “escalating” diet approach [9]. Patients started with a less restrictive elimination diet, namely: dairy products and wheat were excluded (2FED); As a result, 43% of children and adults achieved remission [21]. When prescribing the elimination of milk only (1FED) compared to prescribing 6FED in adult patients with EoE, the frequency of histological responses (<15 eos/HPF) was similar between the two groups (34% vs. 40%; $p = 0.58$) [22]. Retrospective cohort studies in the pediatric population using 1FED showed a histological response rate of more than 50–60%. [23].

Eosinophilic gastritis

Eosinophilic gastritis is characterized by dense eosinophilic infiltration of the stomach wall and is the second most common EGID after EO [24].

Epidemiological data are rather scarce, published in 2016 and containing information on the incidence of EO — 6.3 per 100,000 people. [1]. Among the adult population, the incidence of EoG is 7.1 per

100,000 and is more prevalent in women — 7.9 cases per 100,000, compared to men — 5.4 cases per 100,000 [25]. In childhood, EoG is more common: the prevalence in children under 5 years of age is 17.6 per 100,000 boys and 16.7 per 100,000 girls [1]. The onset of the disease does not correlate with age, but most often occurs before the age of 14 [26]. It is known that 57% of patients with EoG had at least one atopic disease in their medical history [27]. A positive result for food products or airborne allergens in skin allergy tests was also reported in 7 out of 14 patients with EoG [1]. Genetic factors are considered an important predisposing factor for the development of EoG, namely 7 genes (TXN, PRDX2, NR3C1, GRB2, PIK3C3, AP2B1, and REPS1), which may act as biomarkers for the disease [28]. In addition, it was found that more than 90% of the genes of patients with EoG differ from the genes of patients with HP-associated gastritis and gastric cancer [1]. To expand the diagnosis of EoG, the EGDP₁₈ panel was developed, which uses gastric mRNA transcripts and circulating protein levels to distinguish patients with active EoG, helping to classify patients with intermediate eosinophil levels [29]. The study evaluated gene expression and circulating blood proteins, which allowed for the diagnosis of EoG based on gastric mucosa biopsy samples and blood samples. As a result, the developed tissue and molecular platforms allow us to reveal the probable pathogenesis of EoG, explaining the endoscopic and morphological features of this pathology [29].

The clinical symptoms of EoG depend on the location and depth of eosinophilic infiltration, i.e., which layer of the stomach is involved in the inflammatory process: the mucous membrane, muscle layer, or serous layer [30]. Most often, the gastric mucosa is affected, and the main symptoms are pain in the epigastric region, nausea, vomiting, and possibly a feeling of early satiety [5]. In children and adolescents, these symptoms may be

accompanied by growth and puberty retardation, as well as amenorrhea [31].

Given the lack of specificity of the clinical manifestations of EoG, diagnosis of the disease is usually delayed; it has been shown that diagnosis can occur up to two years after the onset of symptoms [32]. It is important to be alert to EoG in order to perform a timely endoscopic examination of the stomach with biopsy sampling for further morphological examination. It has been established that in 66% of cases, the gastric mucosa has a normal appearance [33]. The endoscopic features of EoG include nodular, erythematous gastric mucosa with linear hemorrhages, polyps, erosions, or ulcers [1]. A definitive diagnosis of EoG can only be made based on the results of morphological examination of gastric mucosa biopsies. Eosinophilic infiltration should be ≥ 30 eos/0.27 mm² hpf [1].

EoG treatment is currently empirical and begins with diet therapy, as with other EGIDs. The most effective approach is empirical avoidance of allergy-causing foods, which provides approximately 82% of clinical results [1]. Corticosteroids may also be used—budesonide suspension or enteric-coated capsules, crushed and dissolved in water or juice [34]. Short-term use of systemic corticosteroids can relieve the symptoms of EoG, but the possibility of recurrence requires long-term maintenance therapy [1]. When an elimination diet is combined with topical corticosteroids for 6 months, 75% of patients experience a reduction in clinical symptoms and a decrease in eosinophilic infiltration from 145.4 eos/hpf to 50.8 eos/hpf in the gastric mucosa [27]. The use of anti-IL-5 and montelukast has been described, but their effectiveness has not been confirmed yet [1].

An analysis of scientific literature on EGID has shown that eosinophilic esophagitis in children

is a clearly defined disease with established recommendations, which facilitates the diagnosis and treatment of this pathology, which cannot be said about eosinophilic gastritis. EoG remains a clinical mystery with evidence based primarily on the description of a series of clinical cases [36–38], even despite the consensus on EGID adopted by ESPGHAN/NASPGHAN in 2024 [1]. In this regard, the possibility of combined EoE and EoG pathology may complicate the diagnosis and treatment of these diseases. We present a case of combined eosinophilic esophagus and stomach disease, emphasizing the particular importance of pediatricians' clinical awareness in the early diagnosis and management of such patients.

CLINICAL CASE DESCRIPTION

A 9-year-old girl, A., was admitted to the pediatric somatic department of the City Clinical Hospital No. 20 in Rostov-on-Don with complaints of nausea, belching air and sourness, heartburn, abdominal pain, decreased appetite, unstable stool (constipation for up to 2 days, followed by mushy stool without mucus or blood), weakness, and headache.

Medical history. It is known that the complaints first appeared in January 2023, and the child was observed and examined on an outpatient basis. An esophagogastroduodenoscopy (EGD) performed on an outpatient basis revealed decreased vascularization of the esophagus, white exudate, linear furrows, and immobile esophageal rings, which determined the need for a biopsy. Morphological examination revealed eosinophilic infiltration of the esophageal mucosa >15 eos/hpf, characteristic of eosinophilic esophagitis. The girl took budesonide, esomeprazole, and antacid drugs (sodium alginate + sodium bicarbonate + calcium carbonate) for three months without any visible positive dynamics. In June, a follow-up EGDS

with biopsy of the esophagus was performed, which revealed persistent eosinophilic infiltration >15 eos/hpf. Based on the results, a 1FED diet (exclusion of cow's milk) was recommended, PPIs were prescribed, and an esophagoprotector (sodium hyaluronate, sodium chondroitin sulfate) for 1 month. This treatment regimen reduced dyspeptic symptoms and decreased eosinophilic infiltration (<15 eos/hpf). However, after several months, pain and dyspeptic syndromes recurred, and hormone therapy with budesonide 0.5 mg*2 times a day was resumed against the background of continued PPI (esomeprazole 20 mg*2 times a day) and antacid preparations (sodium alginate + sodium bicarbonate + calcium carbonate) in a suspension of 3 ml twice daily. In November 2024, the child was hospitalized in the pediatric department of City Clinical Hospital No. 20 due to the ineffectiveness of treatment. A review of the medical history revealed that the recommended therapy and diet excluding foods containing cow's milk protein (1FED) were not fully implemented. *Life history.* The girl was born from the second pregnancy, which was complicated by the threat of miscarriage, during the second delivery by cesarean section at 39 weeks, with a double tight cord around the neck at birth. The baby's birth weight was 3500 g, and her length was 52 cm. Anthropometric indicators corresponded to the gestational age. The child was breastfed until the age of two, during which time the mother followed a dairy-free diet, as the child was found to be allergic to cow's milk proteins. The girl is being monitored by an allergist for atopic dermatitis and seasonal pollinosis (ragweed). Her father has a history of bronchial asthma and food allergies.

Examination data. The skin is dry, without any rash. The oral mucosa is clean, with no carious teeth. The tongue has a white coating at the root. The abdomen is soft on palpation, painful in the epigastric

and pyloroduodenal areas; the liver and spleen are not enlarged. Stool is independent, unstable, without pathological impurities. Body weight is 19.5 kg, height is 112 cm, physical development is harmonious, microsomatotype with grade 2 weight deficiency.

A clinical blood test revealed eosinophilia: 6% and 492 cells/ μ L for relative and absolute eosinophil counts, respectively. Tests for helminthiasis and an ultrasound scan of the abdominal organs revealed no abnormalities. Endoscopic findings during EGD were unremarkable: the esophagus was pink, the cardia was completely closed, diffuse hyperemia was found in the stomach only in the antral section, the pylorus was rounded and closed, the bulb and postbulbar sections are normal, and the rapid urease test for *H. pylori* infection is negative.

The conducted histological examination allowed us to draw the following conclusion: chronic mild active (1+) esophagitis with the presence of a large number of intraepithelial eosinophils ($>15/1PZ*400$); chronic HP-negative mild superficial inactive fundal gastritis with desquamation of the integumentary epithelium, edema, an increased number of eosinophils in the infiltrate (up to 67 Eph/5 PZ*400). It should be noted that eosinophilic inflammation was not detected in the rectal mucosa; minimal lymphoid infiltration with a few plasma cells was detected, which allowed us to establish the diagnosis of catarrhal proctitis. The obtained results of the histological examination contradict the data of EGDS, which did not reveal any pathology in the esophagus, and in the stomach they found manifestations of minimal inflammatory activity, which once again proves the particular importance of morphological examination for the verification of eosinophilic gastrointestinal diseases in children.

A thorough examination of the child, taking into account the patient's medical history and clinical features, led to a diagnosis of combined

eosinophilic esophageal and gastric involvement: uncomplicated eosinophilic esophagitis, moderate activity (K20), and eosinophilic gastritis (K52.8). Rectosigmoidoscopy results, combined with morphological findings revealing lymphoid infiltration with a few plasma cells in the rectal mucosa, led to the diagnosis of mild chronic proctosigmoiditis as a concomitant condition not associated with eosinophilic inflammation. Given the combined pathology (EoE + EoH), the following were recommended: 1) prolonged elimination diet 6FED, 2) proton pump inhibitors (esomeprazole 20 mg *2 times a day for up to 8 weeks; in case of a positive clinical response — long-term maintenance therapy with PPIs for up to 10 months), 3) budesonide in the form of a viscous suspension of 1 mg 2 times a day for 12 weeks. To prevent oral candidiasis, rinsing the mouth after taking the hormonal drug is recommended. For the treatment of proctosigmoiditis, sodium alginate was prescribed 1/2 sv *1 time per day for 1 month. Against the background of the therapy received during the hospital stay, the child's condition improved, pain syndrome and dyspepsia symptoms were relieved. The child was discharged in satisfactory condition, with recommendations for further treatment at the place of residence. Observation of the child over the past 6 months

has shown that, despite strict adherence to dietary therapy and treatment, the girl's well-being and condition remain satisfactory.

CONCLUSION

The increasing incidence of EoE and EoG is confirmed by an analysis of scientific publications on this issue. Currently, there is an understanding of the role of certain risk factors for the development of EoE and EoG: the patient's allergic history and genetic factors. However, many gaps remain in the study of the pathogenesis and treatment of these diseases. The presented clinical observation demonstrates the non-specificity of symptoms, as well as the possibility of the absence of visible endoscopic changes in patients with EoE and EoG. Only a morphological study followed by an assessment of eosinophilic infiltration allows a practicing physician to make a definitive diagnosis and choose further patient management tactics. The main factors in achieving successful management of patients with EoE and EoG are early diagnosis of the combined pathology, as well as an integrated approach, including an adequate combination of drug treatment and dietary therapy. Particular attention should be paid to explanatory work with the child's family, which will ensure that parents understand the need for strict adherence to the prescribed treatment.

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ИСТОЧНИКИ ФИНАНСИРОВАНИЯ

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