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EVALUATION OF ANTHROPOMETRIC PARAMETERS OF PATIENTS WITH CONGENITAL BULLOUS EPIDERMOLYSIS WITH AN ALLERGIC REACTION

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Abstract

Congenital epidermolysis bullosa (CEB) is a prototype of a group of genodermatoses with skin fragility. CEB is a severe orphan disease characterized by lesions of not only the skin but also the mucous membranes, with the formation of blisters and erosions in response to minimal mechanical stress. Epidemiological data vary worldwide; the prevalence of CEB, according to various sources, ranges from 9.6 to 11.07 per 1,000,000 people. According to the international association DEBRA International one affected person is born worldwide in 50,000-100,000 people. The prevalence of CEB in the Russian Federation, according to the registry of the "Children-Butterflies. BELA" charitable foundation and individual statistical studies, is 3.64-3.77 per 1,000,000 people. The etiology of the disease is based on variations in genes encoding several proteins that are part of the structural framework of the dermis and dermal -epidermal junction, which ensures the stability and adhesion of keratinocytes. The localization of the defective protein affects the level of blister formation, and accordingly, the clinical form of the disease. Given this heterogeneity, according to the latest revision of the VBE classification (2020), four main clinical forms of the disease are defined. These include: simple epidermolysis bullosa (SEB; in which blisters affect the intraepidermal layer), junctional epidermolysis bullosa (with damage at the lamina level), and junctional epidermolysis bullosa (with damage at the lamina level). lucida of the basement membrane), dystrophic epidermolysis bullosa (DEB; in which blistering occurs below the basement

Modern American Journal of Medical and Health Sciences
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Volume 01, Issue 07, October, 2025
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membrane) and epidermolysis bullosa Kindler (CBE; when lesions can be located at various levels within the epidermis). PBE and CBE are considered to have more favorable clinical manifestations and outcomes. Forms such as CBE are quite rare. In clinical practice, patients with dystrophic and simple forms of VBE are primarily observed. As a rule, VBE is characterized by early manifestation (in the neonatal period or the first months of life), followed by a continuous course with frequent exacerbations. Symptoms and complications experienced by patients with VBE can vary greatly, since the forms of VBE are phenotypically heterogeneous. Clinical manifestations of VBE can be diverse: from localized blisters on the hands and feet to generalized blisters on the skin and mucous membranes and lesions of internal organs. Severe course of the disease is characterized by a high risk of disability and a shortened life expectancy. The course of VBE can also be complicated by the presence of various comorbid pathologies.

The management of patients with VBE requires a multidisciplinary approach and focuses on preserving tissue sensitivity, wound care, active nutritional support, and early conservative or surgical interventions to correct extracutaneous complications when possible. Extensive skin lesions, as well as those affecting the gastrointestinal (GI) mucosa, in most patients with VBE lead to a significant reduction in barrier function, creating conditions for excessive influx of antigens, including allergens, into the body's internal environment. This creates a high risk of developing transcutaneous sensitization and subsequent allergies. The contact of allergens with affected skin areas and their local interaction with antigen-presenting cells (APCs), such as dendritic cells, triggers a cascade of immune reactions. This leads to the activation of T lymphocytes, which promotes the development of a Th2 immune response and the production of specific IgE antibodies (sIgE). Chronic inflammation accompanying VBE, with constant skin contact with the allergen, contributes to increased sensitization. Epidermal barrier damage in patients with VBE is chronic due to constant mechanical stress, leading to persistent inflammation, including type 2 inflammation.

Despite the significant damage to barriers in children with VBE, the topic of food sensitization and food allergy (FA) in these patients remains virtually unstudied.

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ISSN (E): 3067-803X
Volume 01, Issue 07, October, 2025
Website: usajournals.org

Specific genodermatoses including VBE, are known to be diseases characterized by a predominance of type 2 T-helper cells and activation of the type 2 inflammatory immune response. There is also evidence of the presence of eosinophils in skin biopsies of patients with VBE, which may also be a marker of the T2 response. Some publications indicate the involvement of tissue eosinophils in the development of chronic inflammation. Elevated levels in patients with VBE have also been described. A case of elevated serum levels and pronounced tissue eosinophilia in a child with DBE without clinical signs of atopic dermatitis is described. This explains the emergence of the first studies on the successful use of genetically engineered biological therapy with omalizumab in adult patients with EBE and high levels and dupilumab in patients with DBE.

At the same time, there are no data describing the characteristics of PA in children with VBE and there are no algorithms or protocols for diagnosing and treating PA in patients with VBE. Analysis of comorbid conditions, including PA, is a pressing issue, given the frequent difficulties in selecting optimal therapy and developing an adequate diet for this category of patients.

The aim of the study was to optimize the management of patients with congenital epidermolysis bullosa and comorbid food allergy based on the study of food sensitization and food allergy characteristics in children with congenital epidermolysis bullosa.

Research objectives:

To verify the simple form of congenital epidermolysis bullosa based on the results of molecular genetic testing, compare it with the disease phenotype, and characterize sensitization to food proteins in children with this form of the disease. Based on a molecular genetic study, to verify the dystrophic form of congenital epidermolysis bullosa and to study the characteristics of sensitization to food proteins in this group of patients in comparison with children with a simple form of the disease and children in the control group. To conduct a comparative assessment of the frequency and characteristics of clinical manifestations of food allergies in children with simple and dystrophic forms of

Modern American Journal of Medical and Health Sciences
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Volume 01, Issue 07, October, 2025
Website: usajournals.org

congenital epidermolysis bullosa. To assess the impact of comorbid food allergy on the severity of the disease, the severity of itching, and the nutritional status of patients with congenital epidermolysis bullosa. To develop a personalized diet therapy strategy and determine its effectiveness in the complex treatment of children with congenital epidermolysis bullosa and food allergies.

Methodology and research methods.

An analysis of scientific data from Russian and international literature concerning the problems of immunopathogenesis in patients with congenital epidermolysis bullosa was conducted. The study was carried out using a cohort design. A prospective study with outcome monitoring and adherence to evidence-based medicine principles, using modern clinical, laboratory, instrumental, and statistical methods, was conducted. A total of 173 children were included in the study, including 119 with the dystrophic form and 54 with the simple form of congenital epidermolysis bullosa. The clinical examination included a detailed medical history, including information on the presence of allergies, dietary habits, and clinical manifestations of the disease, with an assessment of the severity of congenital epidermolysis bullosa using the EBDASI index, in accordance with existing guidelines.

Study results

For the first time in global practice, a comprehensive study of food sensitization and food allergy characteristics was conducted on a representative group of children with the rare disease congenital epidermolysis bullosa. A detailed characterization of the spectrum of sensitization to food allergens in children depending on age and disease type was provided. A relationship between the clinical manifestations of congenital epidermolysis bullosa and total IgE concentrations was described for the first time particularly clearly in children with the dystrophic form of the disease.

For the first time, data on the frequency of food allergies and food sensitization in children with simple and dystrophic forms of the disease were obtained from a representative group of patients.

Modern American Journal of Medical and Health Sciences
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Volume 01, Issue 07, October, 2025
Website: usajournals.org

For the first time, an assessment was made of the influence of food allergy on the severity of the underlying disease, the severity of itching and the nutritional status of patients with congenital epidermolysis bullosa.

For the first time, an algorithm for the diagnosis and dietary therapy of food allergies in children with congenital epidermolysis bullosa has been scientifically substantiated.

The study results allowed us to evaluate the frequency of sensitization to various food allergens in children with dystrophic and simple epidermolysis bullosa, to determine the characteristic spectrum of sensitization to food proteins and its age-related characteristics. A relationship was established between the level of total immunoglobulin E and the activity of congenital epidermolysis bullosa, which significantly contributes to the understanding of immune disorders in congenital epidermolysis bullosa. The obtained data expand existing knowledge on the role of T2 inflammation in the pathogenesis of congenital epidermolysis bullosa and can serve as a basis for further research in this area. Also, based on the study results, the frequency of food allergies in children with different forms of congenital epidermolysis bullosa was established for the first time. The clinical manifestations of food allergies were characterized, and the impact of food allergies on the clinical picture of the disease was assessed.

The results obtained during the study allowed the development of personalized algorithms aimed at identifying food allergies in children with congenital epidermolysis bullosa and optimized existing approaches to managing patients with food allergies, taking into account the characteristics of patients with congenital epidermolysis bullosa. Personalized recommendations for creating an elimination diet for children with congenital epidermolysis bullosa and food allergies optimize patient management, help reduce disease activity, and improve quality of life. These recommendations can be incorporated into comprehensive treatment programs for children with congenital epidermolysis bullosa, ensuring more effective disease management and improved outcomes. The study demonstrates the need for a multidisciplinary approach and close collaboration between dermatologists and physicians of other specialties, including allergists

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and nutritionists, to provide comprehensive medical care to patients with congenital epidermolysis bullosa.

This research study analyzed food sensitization and food allergies using current, evidence-based, and modern methods of allergy diagnostics in children. The effectiveness of the elimination diet was determined by assessing improvements in disease activity using the EBDASI scale. The collected data were analyzed using modern methods, including descriptive statistics, statistical significance assessment ($p < 0.05$), and correlation analysis. Congenital epidermolysis bullosa (CEB) is a group of orphan inherited diseases with phenotypic diversity, characterized by disruptions in intercellular communication in the epidermis, leading to the formation of blisters, erosions, and scars on the skin and mucous membranes in response to minimal mechanical stress. Epidemiological data on the incidence and prevalence of CEB worldwide are variable and have only been systematized since the creation of patient registries in various countries. In a large US study that included data from 3,271 patients, the incidence and prevalence of VBE in the country were 19.57 and 11.07 per 1 million people, respectively. In Italy, as of 2022, more than 700 patients had been registered, with an overall prevalence per 1 million newborns of 10.1 and an incidence of 20.1. In the UK, according to the registry, approximately 5,000 people suffer from various forms of VBE. According to DEBRA Brasil (an association supporting patients with VBE in Brazil), there are more than 900 patients in Brazil (unpublished data), but no definitive epidemiological studies have been conducted. Precise data on the prevalence of VBE in Russia are lacking. According to the registry of the BELA. Butterfly Children charitable foundation, the prevalence of VBE in the Russian Federation is 3.77 per 1 million people. Among children, the prevalence rate of VBE is 15.48 cases per 1 million people (as of January 1, 2024). According to separate statistics, the average prevalence of VBE in the constituent entities of the Russian Federation is 3.64 cases per 1 million people, with the highest rate recorded in the Republic of Dagestan—19.73 cases per 1 million people.

Epidermolysis bullosa (EB) encompasses a wide spectrum of disease phenotypes, ranging from mild skin fragility caused by minor molecular changes to severe cutaneous and extracutaneous lesions caused by the absence of key adhesion

Modern American Journal of Medical and Health Sciences
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Volume 01, Issue 07, October, 2025
Website: usajournals.org

proteins. According to the current classification (2020), there are four main forms of the disease: simplex, dystrophic, junctional, and epidermolysis bullosa. Kindler. Each form can also include several subtypes, of which there are more than 30. Each form differs not only in phenotypic presentation but also has its own genetic profile (molecular defect), mode of inheritance, clinical manifestations, course, and severity.

Genetic defects in skin structural proteins lead to instability of the microarchitectural connections between the dermis and epidermis, resulting in barrier disruption. More than 29 pathogenic nucleotide variants associated with the development of VBE are known. Genes associated with VBE encode intracellular, transmembrane, or extracellular proteins, primarily structural components of the cytoskeleton (keratin 5 and 14), the cellular matrix (integrin abr4, collagen type 17, laminin 332, collagen type 7, integrin subunit a3, kindlin-1), or intercellular junctions (desmoplakin , plakophilin , plakoglobin).

The classification of VBE is complex and based on the morphological analysis of a skin sample using immunohistological methods and the results of medical genetic testing. Since pathogenic nucleotide variants in the same gene can be inherited in an autosomal dominant or recessive manner, this leads to the development of different clinical phenotypes.

Depending on the level of the molecular and structural defect in the skin, clinical manifestations may include scaling, blisters, erosions, ulcerations, wounds, or scars. Thus, the phenotype of various forms of VBE correlates with the defective gene. Thus, in PBE, lesions occur at the level of the basal layer of the epidermis, in DBE, at the level of the lamina densa, in PgrBE, blister formation occurs at the level of the lamina lucida, and in CBE, blister formation occurs at various levels in the epidermis. Thus, skin lesions may be more superficial and lead to erosions, as in PBE, or they may be deeper and lead to ulcerations, as in PgrBE , DBE, and CBE.

Blisters can occur locally in a limited area or as a generalized lesion, affecting various areas of the body. Skin lesions are chronic due to constant mechanical stress. The mucous membranes of the oral cavity, esophagus, trachea, genitourinary system, and eyes are also susceptible to the formation of erosions,

Modern American Journal of Medical and Health Sciences
ISSN (E): 3067-803X
Volume 01, Issue 07, October, 2025
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ulcers, and scars. An active healing process can lead to the formation of contractures, microstomia, and esophageal strictures. Epidermolysis bullosa simplex (EBS) is the most common form of EBS. In most cases, it is inherited in an autosomal dominant manner, but rare autosomal recessive subtypes have been reported.

Conclusions

Sensitization to food allergens is detected in a significant proportion of children with congenital epidermolysis bullosa (60.1%), and more often in children with the dystrophic form of epidermolysis bullosa than in children with the simple form of the disease. Children with dystrophic epidermolysis bullosa, in contrast to children with the simple form, are characterized by polysensitization, a broadening of the spectrum, and an increase in the frequency of food sensitization with age. Children with congenital epidermolysis bullosa are characterized by a high frequency of clinically significant food allergy (15%), which manifests later in children with the dystrophic form of the disease than in children with the simple form. For children with the dystrophic form, immunoglobulin E-mediated food allergy is more typical. The main manifestations of food allergy in children in both groups are skin symptoms. The presence of comorbid food allergy in children with congenital epidermolysis bullosa is associated with more severe manifestations of the underlying disease, impaired wound healing, and changes in the phenotypic presentation of the disease. Children with dystrophic epidermolysis bullosa and comorbid food allergy have higher severity scores for epidermolysis bullosa. A significant strong association was found between total serum immunoglobulin E levels and disease activity. Children with dystrophic epidermolysis bullosa are characterized by higher mean total immunoglobulin E levels, which increase with age, averaging three times the age-appropriate norm. A properly selected elimination diet has been shown to reduce disease activity scores in children with congenital epidermolysis bullosa and comorbid food allergy.

Modern American Journal of Medical and Health Sciences
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