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O. V. Ponochevna
National medical academy of postgraduate education named after P. L. Shupik, Kyiv

Juvenile allergic pneumonitis

Key words: *pneumonitises, allergy, eosinophilia, children.*

According to modern ideas interstitial lung disease (ILD) is characterized by lesions of the respiratory part of pulmonary interstices, the development of inflammation (alveolitis) and fibrosis with the release of «honeycomb lung», which clinically manifested by progressive respiratory failure and predominantly restrictive desorders [11]. ILD conditionally divided into disease with known etiology (these include hypersensitive pneumonitis or exogenous allergic alveolitis, and drug toxic alveolitis), and diseases of unidentified etiological factor (fibrosing alveolitis, sarcoidosis, vasculites). Besides emit so-called secondary ILD that observed lesions in systemic connective tissue desease [4, 10].

American Thoracic Society in 2002 suggested the following classification of ILD in children [11]:

Diseases specific for infants:

A. Disorders in persons with normal immune system:

1. Infectious and postinfectious processes.

2. Disorders associated with exposure to environmental factors: hypersensitive pneumonitis, inhalation of toxic substances.

3. Aspiration syndrome.

4. Eosinophilic pneumonia.

B. Disorders associated with systemic diseases:

1. Immune disease.

2. Accumulation diseases.

3. Sarcoidosis.

4. Histiocytosis of Langerhans cells.

5. Malignant neoplasms.

C. The disease in immunosuppressive patients.

D. Diseases disguised ILD.

Despite the clinical and morphological polymorphism, options for most of them begins with a fairly stereotyped changes in pulmonary interstitial inflammatory infiltration in a varying degree, productive alveolitis, subsequently formed fibrosis progression rate which may be different. This is the only consistent processes pathophysiological mechanisms involving different cell types and a wide range of cellular mediators. ILD course in children is complicated by the fact that the pathophysiological processes occurring in the body that develops [14, 16]. It is available, which is a genetic predisposition to the development of diffuse lung disease due to the peculiarities of the inflammatory reaction and excessive formation pulmonary fibrosis of epithelium in response to inflammation [17]. Despite the great diversity of clinical forms of ILD, they are characterized by general clinical symptoms, the appearance of which should see a doctor suspected ILD. However, often from the onset to the final diagnosis are months or even years.

It should be emphasized that different nosological forms of ILD have their radiographic features. However, they are inherent and general features. Thus, in all forms of ILD in the early stages of the disease appears mainly enhance and deformation of lung pattern, reducing the transparency of the lung fields type «ground glass» small focal shades. As the progression of lung deformation pattern becomes more pronounced, are signs of interstitial fibrosis, cavities, formed a picture of «honeycomb lung».

The most accurate diagnosis of most ILD is possible only on the basis of evaluation of lung biopsy material. Lung biopsy is currently considered the «gold» standard in the diagnosis of ILD, which allows not only diagnosis but also to predict prognosis, and possible response to therapy [19, 23]. Despite the undeniable value of research, and possible biopsy indicated not all cases. According to major medical centers, lung biopsy is actually performed only in 11–12 % of patients with ILD [1].

Among ILD in children most clinical importance is hypersensitive pneumonitis (exogenous allergic alveolitis, EAA). In 1932, the symptoms of the disease were described by JM Campbell 5 farmers who were working with moldy bread. The term «exogenous allergic alveolitis» was proposed by J. Pepys in 1967. Most forms hypersensitive pneumonitis treated as professional pathology. However, the possible formation hypersensitive pneumonitis in children of all ages who was in contact with allergens through various features of the home microenvironment.

Formation of hypersensitive pneumonitis in children is associated with living in damp areas, close contact with pets and birds. The most important of unfavorable domestic ecology thermophilic actinomycetes considered antigens and birds. The disease is immunologically induced inflammation of the lung parenchyme, in which the process involved the walls of the alveoli and upper airway due to repeated inhalation of organic dust and various other substances [9]. Hypersensitive pneumonitis is seen as immunopathological disease, of which the leading role belongs to allergic reactions III and IV types (classification Gell and Coombs). Of particular importance in the diagnosis of hypersensitive pneumonitis provided identify specific precipitating antibodies to «guilty» antigen. Most patients with hypersensitive pneumonia determined by precipitating IgG-antibodies to significant allergens (mostly to fungal, epidermal and domestic) in various credits. Detection of even small children precipitating antibodies in combination with the characteristic data of clinical history are considered a reliable marker of the disease. It should be noted that the hypersensitive pneumonitis, especially in children, may experience related IgE-mediated immediate hypersensitive reactions, which explains the frequent combination with bronchial asthma (25 % of cases) [7].

Diagnostic criteria of EAA (hypersensitive pneumonitis) is not final. Among the necessary criteria published American Thoracic Society (1998) isolated:

• Diagnostic criteria: contact with a specific antigen, shortness of breath on exertion, wheezing for breath, lymphocytic alveolitis (if performed bronchoalveolar lavage).

• Features that suggest the diagnosis of EAA: repeated episodes of fever, infiltrative changes in the lungs according to radiography, reduced diffusion capacity of the lungs, detection in serum precipitating antibodies to a specific antigen, granulomas on biopsy lung (usually a need for it lacks) improvement after cessation of exposure to the allergen.

• Hypersensitive pneumonitis may have different variants of the course and forecast possible clinical recovery, but at the same time, the disease can lead to irreversible lung damage architectonics. It depends on many factors, including the nature and duration of exposure of antigen, the immune response of the patient, as well as timely diagnosis and adequate treatment.

The term eosinophilic pneumonia is a group of diseases characterized by eosinophilic infiltration of lung tissue and eosinophilia in peripheral blood and / or cerebrospinal fluid.

Depending on the etiology, eosinophilic pneumonia is divided into several different types. Among the obvious reasons there are drugs and chemical agents in the environment, parasitic infections and malignant tumors. Eosinophilic pneumonia may also be autoimmune in nature (Charge – Strauss syndrome). Often they observe the presence of hypersensitive reactions I, III and IV type unknown etiology. With no apparent reason eosionofilic pneumonia is called idiopathic. Depending on the clinical picture distinguish acute and chronic idiopathic eosinophilic pneumonia [13].

Idiopathic eosinophilic pneumonia includes diseases of varying severity. Leffler's syndrome is a benign form of acute eosinophilic pneumonia with typical migrated pulmonary infiltrates and minimal clinical manifestations. Chronic eosinophilic pneumonia presented severe systemic symptoms (fever, night sweats, coughing, lack of appetite and weight reduction over several weeks or months). When radiography of the chest often have peripheral infiltrates that describe how the fire eclipse, likes pulmonary edema. Some patients suffer not allergic asthma. The sudden disappearance of symptoms and changes on radiographs often occurs within 48 hours of starting treatment glucocorticosteroids.

Pathological picture of eosinophilic pneumonia is presented by infiltration of eosinophils, plasmatic cells, large and small mononuclear cells and oedema of intraalveolar membranes, vessels and mucous plugs in the bronchioles. It is assumed that the accumulation of eosinophils and release them substances may be due to an allergic reaction for unknown allergen.

Eosinophils perform phagocytic and oxidative function, like that of neutrophils. Release of cationic eosinophil granule proteins causes tissue damage. These proteins include eosinophilic peroxidase, lisofospholipase, eosinophilic neurotoxin basic protein and eosinophilic cationic protein. Eosinophils can secrete cytokines, including interleukins 1 and 5, release hemoattraktants granulocytes and complement C4, which increases vascular permeability. Eosinophils can also perform the function of antigen-presenting cells.

Systemic diseases that are classified as rare, the symptoms of which include destruction of lung tissue, vasculitis with damage to lung tissue (Goodpasture's syndrome, Wegener's granulematosis syndrome Churg-Strauss), characterized by lesions of small vessels, including capillaries and may have symptoms inherent ILD [22].

Churg-Strauss syndrome (CSS) is a systemic necrotizing vasculitis with vascular lesions of small and medium caliber, with granulematous inflammation of the pulmonary system in the presence of asthma and eosinophilia. In 1951, J. Churg and L. Strauss reported 13 cases of disseminated necrotizing vasculitis in patients with severe asthma, fever and eosinophilia, and in 1994 the Consensus Conference of the nomenclature of systemic vasculitis was determined it belongs to this group of these diseases [24]. Classic CSS starts with upper respiratory tract is allergic rhinitis, nasal poliposis or rhinosinusitis [8, 12]. At the same time or later asthma develops, and in most patients it will be the main clinical syndrome for several years. In 38–77 % of cases transient pulmonary infiltration detected. One-third of patients present with eosinophilia effusion in the pleural fluid. In the future, there are progressive weight loss, fever, fatigue, arthralgia, occasionally arthritis, myalgias, skin lesions as hemorrhagic purpura, erythema, rash, skin necrosis, livedo, subcutaneous nodules [14, 19–21].

Churg – Strauss vasculitis/eosinophilic granulematosis with angiitis has clear diagnostic criteria adopted

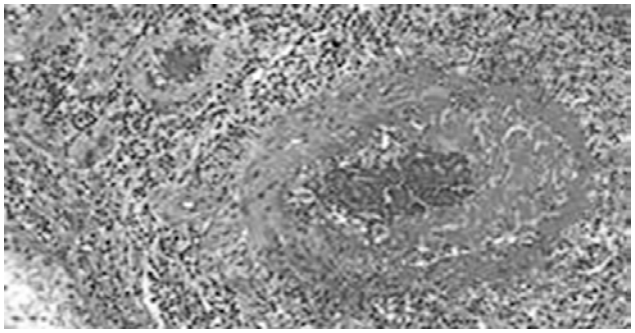

Figure. Necrotizing vasculitis pulmonary vessels

Table

Phase course CSS

Phase disease	Characteristic
I. Prodromal period	Lasts up to 10 years, characterized by various allergic manifestations: allergic rhinitis, polyposis proliferation of the nasal mucosa, recurrent after surgical removal; difficult controlled asthma
II. The second phase - eosinophilic infiltrates	Characterized by eosinophilia in peripheral blood and eosinophilic infiltration of various tissues, including the lungs and digestive tract (eosinophilic pneumonia Leffler's syndrome, eosinophilic gastroenteritis) and others
III. The third phase of the disease - the development of systemic vasculitis	The dominant features are systemic necrotizing vasculitis, which is characterized by multiple organ lesions of the lungs, heart and peripheral nerves and skin

by the American College of Rheumatology in 1990 [12]. Required 4 of 6 the following criteria to achieve a sensitivity of 85 % and specificity of 99.7 % diagnostics:

1. Asthma.
2. Eosinophilia more than 10 %.
3. Neuropathy (mono- or poly-).
4. Migrated pulmonary infiltrates.
5. Sinusitis.
6. Extravascularis eosinophilia. Eosinophils accumulation of in extravascular space (according biopsy) (Figure).

It is assumed that the pathogenesis of vasculitis is an autoimmune process as evidenced by: pronounced signs of allergy (allergic rhinitis, asthma, positive skin tests), enhanced T-cell immune response (pulmonary angiocentric granulomatosis), altered humoral immunity (hypergammaglobulinemia, especially by immunoglobulin E (IgE), high content rheumatoid factor) immunocomplex reactions (circulating immune complexes containing IgE and perinucleous direct neutrophil cytoplasmic antibodies to myeloperoxidase). Recently, much attention is paid to the pathogenetic significance of neutrophil cytoplasmic antibodies in patients with CSS. However, we must take into account that their presence is only half of the patients can not explain all cases of vasculitis.

Diagnosis of the CSS is always difficult. Despite the landmark course of the process, divide the phase of the disease is almost impossible, because they are occurring for years and have no clear clinical picture (Table). Diagnosis is reveald as result almost pathological formation process and is based on the identification of specific organ lesions and detection of eosinophilia (at any stage of the disease), but there are cases without peripheral eosinophilia with severe eosinophilic tissue infiltration. A characteristic feature is the presence of antibodies to neutrophil myeloperoxidase (MPO-ANCA) in 48–66 % of patients. Confirm the diagnosis of morphological study. Among organ lesions, according to Jeff Singh (2002) [18], the CSS dominate: asthma (100 %), pulmonary infiltrates (50–60 %), skin lesions (68–80 %), neuropathy (40–50 %), lesions of the gastrointestinal tract (35–40 %), heart (25–40 %), renal involvement (10–12 %). Fever and weight loss are found in 68–80 % of cases [21]. Defeat lung morphological except asthma symptoms include: granulemas with eosinophilic infiltration, eosinophilic abscess, necrotizing angiitis, eosinophilic pneumonia. Among prognostically significant visceral manifestations often (36–62 %) lesions of the digestive system with gastroenteritis eosinophilic infiltration or inflammation of mesenteric vessels occur. Abdominal symptoms is dominated in clinic, rarer there are nausea, vomiting, diarrhea, bleeding, ground.

Complications of vasculitis are ulceration of the stomach and bowel, bleeding, perforation, peritonitis, there are necrotic and ischemic damage of the digestive system with the development of heart attacks, vasculitis of pancreas, gallbladder, liver [20–22].The peripheral nervous system is affected in 64–75 % of patients. Often there is multiple neuritis, at least is neuropathy by type «gloves and socks». Classically, patients complain of pain, weakness and decreased sensation in the area of innervation of the affected nerve. Often 2 or more affected nerve trunk. The symptoms involvement of central nervous system occur in 3–7 % of patients and manifest as encephalopathy, stroke, subarachnoid hemorrhage, epileptiform convulsions, hyperkinesis or mental disorders [8]. Hemorrhagic or ischemic stroke is developed, because there are cerebrovasculitis and, in some cases, hypertension. The defeat of the cardiovascular system is found in 15–64 % of cases and is clinically characterized by pericarditis, myocarditis, endocarditis, coronaritis and result in myocardial infarction [12, 14, 19]. Kidney damage is seen in 15–88 % of patients. It observed proteinuria, hematuria, signs of renal failure and high blood pressure [22]. Interstitial lung diseases in children include various nosological forms and are currently one of the important problems of pulmonology of childhood. Beginning of disease is usually the same type, but the evolution of the pathological process leading to the different morphological forms of the disease and its various consequences. The most relevant are various ILD diagnosis early, still reversible stages of disease, and the search for new modern approaches to the treatment of life-threatening diseases child.

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ЮВЕНІЛЬНІ АЛЕРГІЧНІ ПНЕВМОНІТИ

О. В. Поночевна

Резюме

У статті розглядається місце алергічних пневмонітів у класифікації інтерстиційних захворювань легень. Розглянуті захворювання – гіперсенситивний пульмоніт, еозинофільна пневмонія, синдром Чарга – Стросс – належать до різних груп інтерстиційних захворювань легень, але мають однотипний початок. Надалі еволюція захворювань проходить певні патоморфологічні етапи з виходом в інтерстиційний фіброз легень. Діагностика даної патології на ранніх стадіях складна, особливо в дитячому віці, у зв'язку з тим що первинні прояви однотипні і маскуються під різну патологію, наприклад, алергічний риніт, синусит. Діагноз встановлюється через роки на етапі інтерстиційного фіброзу, коли результатом є інвалідність. Таким чином, удосконалення ранньої діагностики алергічних пневмонітів дасть змогу оптимізувати терапію та спостереження пацієнтів.

Ключові слова: *пневмоніти, алергія, еозинофілія, діти.*

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О. В. Поночевна
канд. мед. наук, доцент кафедри педіатрії № 1
Національна медична академія післядипломної освіти
ім. П. Л. Шупика, м. Київ
вул. Дорогожицька, 9, м. Київ, Україна, 04112
тел.: +38 (044) 238-77-11; +38 (066) 570-33-80
e-mail: elenaponochevna@ukr.net

ЮВЕНИЛЬНЫЕ АЛЛЕРГИЧЕСКИЕ ПНЕВМОНИТЫ

Е. В. Поночевная

Резюме

В статье рассматривается место аллергических пневмонитов в классификации интерстициальных заболеваний легких. Рассматриваемые заболевания – гиперсенситивный пневмонит, эозинофильная пневмония, синдром Чарга – Стросс – относятся к разным группам интерстициальных заболеваний легких, но имеют однотипное начало. В дальнейшем эволюция заболеваний проходит определенные патоморфологические этапы с исходом в интерстициальный фиброз легких. Диагностика данной патологии на ранних стадиях затруднена, особенно в детском возрасте, в связи с тем что первичные проявления однотипны и маскируются под различную патологию, например, аллергический ринит, синусит. Диагноз устанавливают спустя годы на этапе интерстициального фиброза, когда исходом является инвалидность. Таким образом, совершенствование ранней диагностики аллергических пневмонитов позволит оптимизировать терапию и наблюдение пациентов.

Ключевые слова: *пневмониты, аллергия, эозинофилия, дети.*

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Е. В. Поночевная
канд. мед. наук, доцент кафедры педиатрии № 1
Национальная медицинская академия последипломного образования
им. П. Л. Шупика, г. Киев
ул. Дорогожицкая, 9, г. Киев, Украина, 04112
тел.: + 38 (044) 238-77-11; + 38 (066) 570-33-80
e-mail: elenaponochevna@ukr.net