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PARVOVIRUS INFECTION UNDER THE MASK OF AN ALLERGY: A CASE REPORT

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Background The prevalence of parvovirus infection is quite high, the frequency of detection of serological markers increases with age – from 2-10 % in the group of children under 5 years old, to 40-60 % in young and middle-aged people. Widespread maculopapular rashes on the body are the most significant and often the only clinical sign of the disease among children and are frequently misdiagnosed as allergic reaction.

Objective To analyze a clinical case of parvovirus infection in a boy with allergy.

Report An 8-year-old boy appealed to the department in December on the 13th day of the disease with complaints of widespread nonpruritic lacy rash.

The boy suffered from pollinosis in spring and cross-food allergy to stone fruits every year from 2 years old. He has heredity burdened by allergy diseases.

The illness began with nonspecific prodromal symptoms, such as rhinitis, sore throat and subfebrile fever, lasted for 3 days. On day 4 erythematous rash on cheeks appeared, 2 days later non-itchy, maculopapular rash developed on the trunk and limbs. Ambulance service regarded these symptoms as a toxic-allergic reaction. The child got dexamethasone IM, chloropyramine IM without any significant effect. Then the boy was treated with oral cetirizine, but the rash spread to the upper and lower extremities. The child also followed a strict hypoallergenic diet. On physical examination the patient's condition was satisfactory. There was red rash on the face ('slapped cheek' rash) and pink nonpruritic maculopapular lacy rash on the entire body, except the feet and palms. According to the results of the blood serum anti-B19 IgM was detected. Symptoms were relief without treatment for 5 days.

Conclusion Maculopapular rashes, the lack of effect of antiallergic therapy necessitate advanced diagnostic. It is necessary to conduct differential diagnosis with infectious diseases despite an aggravated allergy history. Setting the correct diagnosis avoids the prescribing of unnecessary drugs and diets.

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BIOLOGICALS IN SEVERE ASTHMA – CASE REPORT

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Severe asthma is defined BY poor symptom control despite the use of high-dose inhaled corticosteroids (in children younger than 12 years $\geq 400\mu\text{g}$ of budesonide or equivalent and $\geq 1000\mu\text{g}$ in older children). It is estimated that 2-5% of asthmatic children have severe asthma. There is an important difference between difficult to treat asthma caused by poor compliance and/or inadequate use of medications, environmental factors and comorbidities, and severe asthma that is

resistant to medications (therapy-resistant asthma). Patients with severe asthma have persistent symptoms although they are using standard medications properly (inhaled and oral corticosteroids, long acting β_2 -agonists and antileukotriene). Therapy of the difficult-to-treat asthma includes intensive individual education with psychological support, while for the therapy-resistant asthma, biologicals (anti-IgE, anti-IL-5, anti-IL-5R, anti-IL-4R) are being used. Omalizumab is a monoclonal antibody which binds to circulating free IgE, reducing the possibility of free IgE to bind to basophil receptors and thus reducing IgE mediated immunological response. It is being used in the therapy of severe allergic asthma for the last 15 years in children and adults. Studies show the efficacy and safety of omalizumab in reducing symptoms, number of asthma exacerbations and reducing the dose of inhaled corticosteroids. In this case report we shall present a preschool girl with allergic asthma since she was two years old. In the fourth and fifth year of life she had frequent asthma exacerbations despite the use of anti-inflammatory medications. When she was six years old, she had a severe asthma exacerbation triggered by respiratory infection. Anti-inflammatory therapy with medium dose fluticasone propionate and salmeterol was started. In the next year she had frequent exacerbations which were treated in an ambulatory setting with oral corticosteroids. New combination therapy, high-dose budesonide with formoterol and leukotriene, was introduced. Despite adequate follow-up and regular use of anti-inflammatory therapy she continued to suffer exacerbations. In the seventh year of life extensive allergy, pulmonology and immunology workup was done. She had 10.6% eosinophils in peripheral blood, total IgE 4524 IU/ml, specific IgE for *D. pteronyssinus* >100 kIU/L, specific IgE to ragweed >100 kIU/L and FVC 108%, FEV1 83%, FEV1/FVC 0.66 (77%), MEF50 40%. Differential diagnostic conditions with similar and asthma worsening comorbidities were ruled out. Since she had features of severe asthma, treatment with omalizumab was initiated in the Children's hospital Srebrnjak, and further follow-up and therapy administration were continued in our institution. From the beginning of the therapy with omalizumab improvement in symptoms and pulmonary function was observed with reduction in asthma symptoms and no exacerbations needing systemic steroids.

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SERUM SICKNESS-LIKE REACTION IN A PEDIATRIC PATIENT TREATED WITH OMALIZUMAB FOR SEVERE ASTHMA

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Omalizumab is a recombinant DNA-derived humanized IgG1k monoclonal antibody that specifically binds to free human immunoglobulin E (IgE) in the blood and interstitial fluid and to membrane-bound form of IgE (mIgE) on the surface of mIgE-expressing B lymphocytes. This drug inhibits allergic responses by binding to serum IgE, thus preventing interaction with cellular IgE receptors. Omalizumab is also capable of downregulating the expression of high affinity IgE receptors on inflammatory cells, as well as the numbers of eosinophils in both blood and induced sputum. The clinical effects of omalizumab in severe asthma include improvements in respiratory symptoms and quality of life, reduction of asthma