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

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10.1136/archdischild-2021-europaediatrics.169

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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10.1136/archdischild-2021-europaediatrics.170

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. pteronyssimus* $>100\text{ kIU/L}$, specific IgE to ragweed $>100\text{ kIU/L}$ 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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10.1136/archdischild-2021-europaediatrics.171

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

exacerbations, emergency room visits, and use of systemic corticosteroids and rescue bronchodilators. Omalizumab is relatively well tolerated, and only rarely induces anaphylactic reactions. Therefore, this drug represents a valid option as add-on therapy for patients with severe persistent allergic asthma inadequately controlled with high doses of standard inhaled treatments. We report a case of serum sickness-like reaction in an 11-year old boy with a history of severe persistent allergic asthma poorly controlled in spite of high dose inhaled corticosteroid/long-acting β 2-agonist. Total serum IgE was 480 kU/L, body weight was 38 kg. Four days after his first injection of 450 mg omalizumab he developed fever over 39°C, cramping abdominal pain, fatigue, cervical lymphadenopathy, diffuse joint aches and loose stools, up to three times a day. Three days later, he developed generalized pruritic urticarial rash and feet edema. The patient has been treated first in the primary care setting for 3 days with oral methylprednisolone 1 mg/kg and antihistamine but was referred to the subspecialist on the 4th day. Laboratory tests showed leukocytosis ($15.9 \times 10^9/L$) with predominantly neutrophilia (70.9%) and increased C-reactive protein (197.2 mg/L). Abdominal ultrasound was unremarkable. Circulating immune complexes were within normal range, however, the blood sample has been taken on the 7th day after the onset of symptoms. Further evaluation for infection was negative. Oral methylprednisolone was discontinued and antihistamine was given for a total of 10 days. After 16 days, all the symptoms resolved completely. Omalizumab has been found to be generally safe, though immediate adverse reactions have been documented. However, the serum sickness-like reactions in patients treated with omalizumab are rare and non-specific in nature, so their true incidence is difficult to determine. It remains to ascertain which factors increase risk for serum sickness-like reaction to omalizumab.

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SEVERE PROTEIN LOSS DUE TO PROTEIN LOSING ENTEROPATHY OR SEVERE ATOPIC DERMATITIS IN AN EXCLUSIVELY BREASTFED INFANT – A CASE REPORT

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10.1136/archdischild-2021-europaediatrics.172

Protein loss is often the result of kidney or intestinal disease (protein losing enteropathy) and can cause a number of serious, potentially life-threatening complications such as hypotension, thrombocytosis, electrolyte imbalance and cerebellar ischemia. Recent research suggests an association between extremely severe atopic dermatitis (AD) and allergic enteropathy.

Case Report An exclusively breastfed 6-month-old infant was admitted to the Department of Gastroenterology of our hospital due to failure to thrive, electrolyte imbalance and severe AD (SCORing Atopic Dermatitis; SCORAD 40). On admission, the infant was in poor general condition, dehydrated, malnourished (body weight 4870 g, -3.98 z-score; body length 63 cm, -1.66 z-score), with exudative erythematous morphs

scattered throughout the body. Initial laboratory results showed microcytic hypochromic anemia (Hb 110 g/L), hypoalbuminemia (16 g/L), hypogammaglobulinemia (IgG1 2g/L), thrombocytosis ($1600 \times 10^9/L$), hyponatremia, high values of total immunoglobulin E (IgE) and eosinophilia. Due to the severe presentation and deeply disturbed values of laboratory parameters, extensive gastroenterological, immunological and allergological workup was performed. Polysensitization to a number of nutritional and inhalation allergens has been proven, and exclusive amino acid based formula has been introduced into the diet. During hospital course, patient developed superficial thrombophlebitis and methicillin-resistant *Staphylococcus aureus* (MRSA) bacteremia, treated with vancomycin, which was replaced with teicoplanin due to severe 'red man' syndrome. Given the initial presentation, hypersensitivity to numerous allergens, unclear primary cause of protein loss (due to enteropathy or severe AD) and burdened family history (father has Crohn's disease), esophagogastroduodenoscopy and ileocolonoscopy with small bowel biopsies were performed. In the small intestine biopsy sample, eosinophilia (> 30 eosinophils per HPF) was found without villous atrophy, and with neat calprotectin levels in stool sample. Due to severe hypogammaglobulinemia, skin infections, and bacteremia, the differential diagnosis included primary immune deficiency (STAT3 deficiency, DOCK8 deficiency, PGM3 deficiency, IPEX), but all available immunological tests were unremarkable (immunophenotyping of peripheral blood lymphocytes, lymphocyte function test, respiratory burst, thymus ultrasound, and bone marrow morphology). Exclusive amino acid based formula diet was continued in infant, with topical corticosteroids under wet-dressing therapy, and intravenous immunoglobulin replacement therapy every four weeks. With the gradual improvement of the general condition and the regression of skin symptoms, introduction of solid foods started according to the findings of allergy testing. At 17 months of age, the patient gained weight (TM 12.5 kg, 0.61 z); his skin status is improving, although frequent use of topical corticosteroids is necessary. There were no infections, no anemia or thrombocytosis, and albumin and immunoglobulin supplementations were no longer required.

Conclusion The main mechanism of protein loss in infants with extremely severe atopic dermatitis is probably due to damaged skin, and partially to the eosinophilic inflammation of the small intestine. Immunoglobulin loss, potentiated by physiological or transient hypogammaglobulinemia in infants, poses a very high risk for severe, potentially life-threatening infections.

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CARDIAC BIOMARKERS IN DIAGNOSING ACUTE MYOCARDITIS IN ADOLESCENTS

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10.1136/archdischild-2021-europaediatrics.173

Aim To present a cohort of adolescents admitted into our clinic for precordial pain, mimicking myocardial infarction. Cardiac biomarkers were crucial in diagnosing and monitoring acute myocarditis.