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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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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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.