DARIER’S DISEASE PRESENTING AS RECURRENT ECZEMA HERPETICUM IN 10-YEAR OLD BOY: CASE REPORT

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10.1136/archdischild-2021-europaediatrics.150

Eczema herpeticum (EH) is a disseminated herpes simplex infection of the skin most commonly occurring in patients with atopic dermatitis (AD), seborrhoeic dermatitis, Hailey–Hailey disease, allergic contact dermatitis, psoriasis and Darier’s disease (DD). It is considered a dermatologic emergency due to its high mortality rate if misdiagnosed or left untreated.

We present a case of a 10-year-old boy with a longstanding history of AD referred to Allergology and Immunology Department for recurrent eczema herpeticum secondary to herpes simplex 1 (HSV-1) infection and possible primary immunodeficiency.

For the past two years, the patient has received chlorpro-mazine therapy for aggressive behaviour. First episode of EH was diagnosed at the age of six following intensive topical corticosteroid therapy for AD and sun exposure, another known risk factor for HSV infection. After the third EH episode, prophylaxis with oral acyclovir was commenced. The skin changes were treated with topical steroids and oral antibiotics in flares of the disease, with poor clinical response. On presentation he was of good general condition, adipose, of unremarkable somatic status except numerous symmetrical yellowish-brown keratotic papules and plaques over the forehead, cheeks and lateral side of the neck. The nail plate had multiple red and white longitudinal streaks. The allergy tests revealed increased total immunoglobulin E (IgE) and sensitization to ragweed. Immunological work up showed normal immunoglobulins and good specific immunity but decreased number of T-cells (CD3+ 1020/μL (1320-3300), CD3+CD8 + 281/μL (390-1100) with normal T cell response after antigen stimulation. Skin biopsy confirmed Darier’s disease. Oral retinoids were started with good clinical response.

Acyclovir prophylaxis was continued. We present the case of the 10-year-old patient with DD and impaired T-cell-mediated immunity initially misdiagnosed as AD. The occurrence of EH in patients with DD is rare and possibly related to immune system dysfunction as additional risk factor along with impaired skin barrier. This case highlights the importance of early recognition of the association of DD with impaired cellular immunity and the importance of adequate treatment due to possible fatal outcomes.

CLINICAL EFFICACY OF BAC-SET® FORTE MULTISTRAIN PROBIOTICS COMPLEX IN THE PREVENTION OF ADENOTONSILLAR PATHOLOGY IN PRESCHOOL CHILDREN

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10.1136/archdischild-2021-europaediatrics.151

Goal of research: to study Bac-Set® Forte multistrain probiotic complex efficacy and safety in preschool children with adenotonsillar pathology.

Materials and Methods For the period 2016 – 2019, 346 children (3-6 years old) with a history of chronic pathology of pharyngeal and palatine tonsil and suffering from frequent recurrent respiratory infections were observed.

The average age was 4.53 ± 2.71 years. Preschool children of the treatment group (n=230) received multistrain probiotic complex Bac-Set® Forte as a prevention of exacerbations of chronic nasopharyngeal pathology on a daily basis, in addition to irrigation and elimination therapy (nasal shower with 0.9% NaCl solution); 1 capsule a day was prescribed for 30 days. The control group (n=116) was on irrigation and elimination therapy only. The analysis of the efficiency and safety of the multistrain probiotic complex was carried out before and after the preventive course.

Results a year before the start of the preventive course with Bac-Set® Forte multistrain probiotic complex, the pharyngeal tonsil hypertrophy of degree 2 and with complication by adenoiditis was observed in 76.3% of children in the treatment group and in 75.8% of children in the control group (p=0.2376). By the end of the study, only 30.4% of patients in the treatment group showed no improvement in the clinical picture (p=0.000). A year after the recovery stage, 62.7% of patients which were on the multistrain probiotic complex had recovered nasal breathing (p=0.001); the symptoms of adenoiditis were almost stopped in 51.8% of patients (p=0.000); 82.7% of patients had a decrease in the volume of the pharyngeal tonsil from degree 2 to 1 (p=0.000); 78.9% of patients had a normalization of the rhinoscopic picture (p=0.000); endoscopic control confirmed a decrease in the size of the palatine tonsils in 56.8% of patients (p=0.000).

The degree of hypertrophy of the pharyngeal and palatine tonsils in preschool children who did not receive the multistrain probiotic complex did not change and even increased in dynamics in 81.4% of patients.

Conclusions The study results have confirmed the efficiency and high tolerability of Bac-Set® Forte multistrain probiotic complex. Prospective observation of children who was on Bac-Set® as a prevention of exacerbations of adenotonsillar pathology have confirmed its efficacy in the formation of respiratory tract immunity. The obtained data allow us to recommend the addition of Bac-Set® Forte multistrain probiotic complex in programs for the prevention of exacerbations of adenotonsillar pathology in children.

ANAPHYLAXIS TO COLD TRIGGERED BY CAMPYLOBACTER JEJUNI ENTERAL INFECTION: A CASE REPORT

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10.1136/archdischild-2021-europaediatrics.152

Background Anaphylaxis in children may sometimes be difficult to recognise and needs a high degree of suspicion. The most common causes are food allergens, medications and hymenoptera venoms. Physical factors like extensive cold contact can also result in systemic reaction ranging from generalized urticaria to anaphylaxis. Case report: A 12-year old boy was getting into the cold sea and he suddenly developed general malaise, nausea, abdominal pain, diarrhea and itchy skin. When emergency medical assistance arrived his blood pressure was 70/50 mmHg and he was noted to have generalized hives
and swollen lips. Following parenteral administration of epinephrine and glucocorticoid together with intravenous bolus of fluids the boy recovered. Two hours earlier he had eaten tuna steak. His mother tasted a piece of it and reported mild tongue pruritus.

Therefore the boy was diagnosed of having scombroid poisoning. Two days later he performed some experiments at home. He realized that his hand in a container with cold water, as opposed to a hand in warm water, has swollen and blushed. When he drank a small amount of an ice tea he felt throat tightness. Barefoot walking on the cold floor caused to him itching of the soles of the feet. The ice cube test was performed at hospital. The test resulted positive and diagnosis of cold urticaria and anaphylaxis was made.

For the whole next year the boy suffered from hypersensitivity reactions to cold. Continuous oral antihistamine therapy made symptoms less severe.

However, it is interesting that the boy got over an acute enteral infection caused by Campylobacter jejuni just a week before the reported episode of anaphylaxis to cold occurred. Moreover, two days before the symptoms of the enteral infection started the boy swam in a pool with very cold water (colder than sea water) and had no reactions. Conclusion: The exact mechanisms of hypersensitivity reaction and histamine release to cold are not completely elucidated. There are many infective agents that have already been associated with cold urticaria but Campylobacter jejuni was not one of them. Future investigations are needed to reveal the pathophysiologic pathways that connect these two conditions.

Food-protein induced enterocolitis syndrome (FPIES) is a non-IgE mediated gastrointestinal hypersensitivity, categorised into two major phenotypes: acute and chronic. Acute FPIES manifests as profuse, repetitive vomiting, 1–4 hours after exposure to a triggering food, sometimes with diarrhoea 5–10 hours later, leading to dehydration and lethargy. In chronic FPIES symptoms develop over days or weeks with intermittent emesis, progressive diarrhoea, abdominal distension and failure to thrive. The pathophysiology of FPIES remains obscure, but there is evidence of profound activation of innate cells, eosinophils, and pan-T-cell activation. Most FPIES cases are induced by cow’s milk proteins and soy in infants, although other single or multiple foods can be triggers, including solid foods, where there are significant geographical differences. The incidence ranges from 0.015 to 0.5%. There is a significant lack of awareness among professionals about FPIES and significant delay of diagnosis. There are some adult patients, too. Diagnosis is purely clinical, and there are diagnostic criteria for patients presenting with possible FPIES and diagnostic criteria for the interpretation of oral food challenges (OFC), as shown here. The most important aspect of management in patients with FPIES is the avoidance of triggering food. The natural course is favourable, and there are no reports of long-term complications in children with FPIES and it is largely a self-limiting, generally benign disorder of childhood.

Patients with FPIES usually have undetectable specific IgE to triggering food, except in rare cases of atypical FPIES. In addition to classic acute FPIES symptoms, children with atypical FPIES can also have symptoms consistent with an acute IgE-mediated hypersensitivity such as urticaria, angioedema, flushing, and wheezing, and are likely to have a more protracted course. Rarely, patients with IgE-mediated food allergies develop non-IgE-mediated food allergies and vice versa. Our patient is a child who had been avoiding cow’s milk due to immediate cutaneous reaction on ingestion, with positive specific IgE and skin prick-to-prick test. After ten months of avoidance, and negative new specific IgE, she was challenged for resolution. She tolerated cow’s milk during the 2 hour observation period, but then developed delayed profuse vomiting. FPIES was confirmed with another, FPIES designed oral food challenge, and she has been advised to continue using an extensive hydrolysat formula. Due to the known possibility of developing FPIES to other proteins, their cautious introduction is advised. Further follow-up will be taken, considering OFC every 18–24 months.

**154 FOOD ALLERGY WITH IDIOPATHIC ANAPHYLAXIS AND MAST CELL ACTIVATION SYNDROME DIAGNOSTIC CHALLENGE**

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10.1136/archdischild-2021-europaediatrics.154

**Background**

Food-related anaphylaxis is common in a paediatric age group. Allergic reactions with multisystemic involvement can potentially point towards mast cell activation syndrome.

**Case Presentation**

6-year-old child initially presented with allergic reaction after eating strawberries. He developed lip swelling, breathing difficulties, itchy rash. Symptoms resolved with oral antihistamine and GP prescribed adrenaline autoinjector (AAI) with suspected anaphylaxis reaction.

At 8 years of age, he had an allergic reaction to orange fruit shoot with lip and tongue swelling, itchy rash. He responded well to oral antihistamine. Similar episode occurred few days later along with breathing difficulty and he received IM adrenaline in the hospital. He also had provocation test with orange fruit shoot and developed anaphylaxis requiring IM adrenaline, IV chlorphenamine.

Later, severe anaphylaxis reactions occurred in school, but there was no identifiable cause found. At one stage, required AAI once a week over 5 weeks period at school with no trigger. On few occasions there was no itchiness but only lip and facial swelling present. He also had chronic urticarial rash for which was prescribe oral cetirizine and was in control.

Once he developed urticarial rash after contact with latex tablecloth in school, after which plan made in place to avoid contact with latex cloth. At this point of time, he was referred to tertiary care centre for further opinion.

Over the next 2 years anaphylactic episodes resolved. Later, developed neurological symptoms of tremor and feeling of being disoriented. School noticed that, his concentration has been lacking. Eye symptoms with redness, itchiness and discomfort were developed.

**Investigations**

Skin prick test showed positive 6mm, Peanut-3mm, Walnut-4mm, Hazelnut-4mm, Almond-3mm, Strawberry-3mm, House dust mite-2mm, Birch pollen-3mm. Total serum...