**Material** We analyzed a prospective study on 87 patients diagnosed with AID. We analyzed the initial immune status, for all these patients.

For another group of 53 children, diagnosed with selective IgA immunodeficiency, we realized a six-year period survey of the level of the T suppressor lymphocytes, the T helper/T suppressor ratio, and of the presence of auto antibodies: Anti DNA, rheumatoid factor (RF).

**Results** 8 patients (8%), from the group diagnosed with AID, were also identified with selective deficit in IgA at the moment of the initial diagnosis.

In the group of 53 patients with underlying IgA immunodeficiency, 2 patients developed over the 6 years of the survey, a significative titer of anti DNA antibodies. In one patient the presence of the RF was detected, 4 children presented a decrease of the T suppressor level, with a rise of the immune ratio. None of these patients presented clinical signs suggesting an AID.

**Conclusions** The IgA immunodeficiency may be a risk factor for subsequent AID. There is a higher risk for AID in patients who develop anti DNA antibodies, RF or a persistent decrease of the T suppressor lymphocytes.

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**Introduction** Asthma is chronic disease which in recent years prevalence shows increase rate. In this study we analyze triggers that parents or child will tell as a risk factor for asthma exacerbation in children with asthma.

**Aim** Presenting cases treated in pulmonology clinic during their exacerbation and triggers that can lead to asthma exacerbation.

**Method** It is a prospective study, we include 92 children who came with symptoms of asthma exacerbation in pulmonology clinic. Asthma was classified according to GINA classification and evaluate from parent/caregiver or child about risk factors that lead to exacerbation. Factors were listed by GINA.

**Results** Asthma exacerbation symptoms at children that were examined are cough(100%), difficult breathing(90%) and wheezing(24%) and chest tightness. From all children in our study 34% had one trigger, 16% 2 triggers and the others had more than 3 triggers that lead to asthma exacerbation. Triggers of asthma exacerbation are changing weather(cold air) (36%), viral infections(48%), passive smoking (36%), pollen(16.6%) and 25% of parents don’t know risk factors that lead to asthma exacerbation. Most of the children in the study lives in town (75%).

**Conclusion** Our study shows that viral infection and cold air are very common triggers of asthma exacerbation especially in small children while in children older than 5 years passive smoking is very present (36%) as risk factor.

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**Background** PFAPA is a chronic condition including recurrent fever episodes, aphthous stomatitis, pharyngitis, adenitis. According to a previous study, even between febrile attacks, there is increasing of pro-inflammatory mediators.

**Aims**

1. To evaluate serum interleukin phenotype between febrile episode in PFAPA patients from our clinic;
2. To establish correlation between CR reactive protein (CRP) and pro-inflammatory interleukins: tumor necrosis factor-alpha (TNFα), interleukin-8 (IL-8);
3. To evaluate link between CRP and anti-inflammatory interleukins: interleukin-10 (IL-10);
4. To identify a sensitive biological marker to estimate PFAPA evolution.

**Methods** Authors analyzed 2 groups: “PFAPA group” represented by 6 patients and “control group” containing 4 no-PFAPA patients. Inclusion criteria: patients up to 10 years of age that fulfilled PFAPA diagnosis criteria; patients between febrile attacks; negative procalcitonin (PCT) blood value in order to exclude bacterial infections for study patients. Exclusion criteria: patients during febrile attacks. Both groups patients were tested for serum levels of PCT, CRP, IL-8, TNFα, IL-10. Data was statistically analyzed using independent “t” test.

**Results** Both group patients have normal serum levels for interleukins 8/10 and high TNFα values. Mean value for TNFα was 11.26 pg/ml in PFAPA group and 15.52 pg/ml in no-PFAPA group. Regarding CRP values, mean value for PFAPA patients was 19.72 (range between 2.4–95) as compare to 5.04 in no-PFAPA patients.

**Conclusions** TNFα, IL-8, IL-10 aren’t useful to appreciate PFAPA evolution pattern. CRP remains a sensitive marker for disease activity in PFAPA patients, even out of fever attacks. Our study didn’t confirm previous study data.

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**Introduction** An important characteristic of the herpes simplex virus, is their ability to persist in the tissues of their hosts for many years after initial infection as intracellular viruses. Characteristic life of virus (chronic persistent and ciclic replication) in organisms is often followed by immune dysregulation.

**Materials and methods:** Clinically manifestations in patients with herpesvirus infections were examined. We analysed: white blood cell count, hemoglobin level, serum immunoglobulins level, enzymes of cell destruction, oxidative metabolism of the peripheral blood phagocytes as ability of NBT reduction, serum level of IFN-γ, IL-4 and DHEAS, cortisol were measured by ELISA test.

**Results** Our patients had and all of them had positive ELISA test on virus-HSV. Our parameters approved low level of hemoglobin, monocytosis, lymphocytosis, virecrosis and leukopenia. Our patients had high level LDH, CPK, low ability of NBT reduction. High levels of IFN-γ followed high levels of LDH, CPK, GOT and GPT.

**Conclusion** Chronic activation of immune system is background of pathogenetic mechanisms during herpes simplex virus infection. Different level of DHEAS and cortisol are part of regulatory mechanisms of immune response across endocrine system. Increase levels of DHEAS in our patients can display chronic inflammation. Absence of increase level of cortisol may suggest that our patients had a little “acute” fase of infection opposite a lot of chronic disorders. Analyse of immunoregulatory mechanisms is essential to order level and place of damage cells, tissue and organs. It is important for therapy and prognosis of disease.

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**Introduction** Parameters of immune system disregulation during herpes simplex virus infection in childhood

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