HENOCH-SCHÖNLEIN PURPURA IN PEDIATRICS: FIVE YEARS OF EXPERIENCE AT A RESEARCH AND TRAINING HOSPITAL IN ISTANBUL

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Our aim was to describe the epidemiological, clinical, laboratory characteristics of patients with HSP and to compare them with existing studies.

The medical records of children with HSP, who met the Ankara criteria for HSP diagnosis, were retrospectively reviewed at the Haydarpasa Research and Training Hospital between the years 2010-2016. The epidemiological, etiological and clinical features, laboratory results were assessed.

In our study, the age of the patients ranged from 3 to 12 years (mean age 7 years). 93.3% of the patients were under the age of 10. (53.3%) were male and 21 (46.6%) were female. The male/female ratio was 1.14/1.

In our study, 24 cases (53.3%) were most seen in the winter season, followed by the autumn season with 12 cases (26.67%). There was significant clustering in the winter-autumn seasons.

In our study, 21 cases (46.67%) had symptoms of upper respiratory infection in the last few weeks, 9 (20%) had analgesic-antipyretic and/or antibiotics use. 6 cases (13.3%) described as acute gastroenteritis and/or diarrhea, and 9 patients (20%) had a high fever. In 10 (22.2%) patients, no etiological factors could be identified.

Clinical features examined; 44 (97.7%) of patients applied to the health institution with purpura, 22 (48.8%) joint pain and/or swelling, 10 (22.2%) abdominal pain and 5 (11.1%) with fever. Only 1 patient (2.2%) complained at the time of application that they could not only walk without rash.

Anemia was detected in 6 (13.3%) patients and leukocytosis in 30 (66.6%) patients. Trombocytosis was found in 24 (53.3%) patients. Erythocyte sedimentation rate was high in 66% of patients, while CRP was positive in 73.3% of patients. Microscopic hematuria was found in 14 (31.1%) patients, proteinuria was found in 6 (13.3%) patients. Total IgA value was high in 5 patients (11%), C3 and C4 were found to be normal in all. PT/INR values were normal in all patients.

Our results are compatible with the literature, there are no significant differences in the epidemiological and clinical profile than reported elsewhere except IgA values in the studies conducted in the literature are significantly higher than in our study.

Adalimumab (ADA) is fully humanized, monoclonal antibody against tumour necrosis factor (TNF-α), and is efficacious biological treatment option for patients with juvenile idiopathic arthritis (JIA). However, some patients who responded to the ADA therapy subsequently lose response to this treatment and present with relapse of JIA.

In a period of five years (2015-2020), a cohort of 19 patients with JIA, treated with ADA in Children’s Hospital Zagreb, was followed. Disease activity was evaluated based on counts of joints with active disease, erythrocyte sedimentation rate (ESR) and physician global assessment of disease activity. Blood samples were collected from patients who presented with JIA relapse and suspected loss of response (LOR), as well as randomly from patients who had achieved appropriate disease control with ADA therapy as a control group. Levels of adalimumab and anti-adalimumab antibodies (AAA) were measured using enzyme-linked immunosorbent assays (ELISA) (Immundiagnostik AG, Bensheim, Germany). Use of concomitant methotrexate (MTX) therapy was noted, as well as time of AAA development from the beginning of ADA therapy.

Out of 19 patients on ADA, six presented with LOR. Out of six, positive AAA and subtherapeutic ADA concentrations were found in four patients, while in two patients ADA concentrations were subtherapeutic but AAA negative. Two of the six relapsed patients did not receive MTX at the time of sampling due to adverse reactions to this drug. The median time to development of relapse of JIA and proven AAA was eleven months (2-20 months) after initiation of ADA therapy. Adalimumab has so far been replaced by tocilizumab in four patients with LOR. Of the thirteen patients in the control group, two had positive AAA and subtherapeutic ADA concentrations, and one had only subtherapeutic ADA concentrations. Four of the thirteen patients in the control group did not receive MTX at the time of sampling, all four had negative AAA, and one also had a subtherapeutic concentration of ADA.

In our patients with relapsed JIA, positive AAA and subtherapeutic ADA concentrations were associated with loss of therapeutic response. However, the occurrence of AAA and subtherapeutic concentrations of ADA in the control group also indicate the need for further research in a larger number of patients, as well as the need for serial measurement and testing the ability of AAA to neutralize ADA.