

## CORRESPONDENCE

## Samter's triad and eicosanoid imbalance in children with recurrent nasal polyps

DOI:10.1111/j.1399-3038.2012.01288.x

To the Editor,

Chronic pediatric rhinosinusitis (CRS) is a complex disease in which the significance of several predisposing factors changes with age and differs in many respects from the adult form of CRS, as per the European Position Paper on Rhinosinusitis and Nasal Polyps (EP3OS) group (1). The presence of pediatric CRS gives a specific indication for diagnostics toward allergy, immunologic deficiencies (innate or acquired), primary ciliary dyskinesia, or cystic fibrosis. CRS with nasal polyps (NP) is a very rare disease in children and adolescents, and sometimes it accompanies cystic fibrosis (1). There are reports in the literature describing single of pediatric or adolescent cases of NP with asthma and non-steroidal antiinflammatory drug (NSAID) intolerance (2–4), which is otherwise known as Samter's triad. According to the current state of knowledge, the pathogenic mechanism of AERD (aspirin-exacerbated respiratory disease) is based on eicosanoid imbalance (5). Here, we report three cases of Samter's triad in pediatric/adolescent patients (two girls and one boy). Two patients were 17 years old, and one was 13 years old. None of them had a family history of NSAID intolerance, NP, or asthma. Because of young age and a danger of anaphylactoid reaction, no oral provocation test was performed. The diagnosis for Samter's triad was made based on the presence of NP and asthma plus a clinical history of NSAID intolerance. Additionally, the *in vitro* functional eicosanoid test (FET) was performed. In all patients, NPs were diagnosed first when they were on average 11.7 years old. In one patient, asthma and NSAID intolerance were diagnosed at the same time as NP, whereas in the other two patients, the comorbid diagnoses were made 1 and 2 years later. Szczeklik reported the diagnosis of Samter's triad in adults 4 years after onset of NP (5). In our pediatric/adolescent patients, we have seen faster progression of NP into Samter's triad, which can possibly indicate more aggressive form of the disease. All patients underwent 1–3 sinus surgeries. At the time of their first sinus surgery, the patients were between 10 and

15 years of age. No angioedema or urticaria was reported. In two patients, asthma attacks were induced by aspirin and in one patient by diclofenac and ibuprofen. To examine the balance between leukotrienes and prostaglandins in our patients, we used the *in vitro* FET performed on peripheral leukocytes. To the best of our knowledge, the FET was never before used to investigate eicosanoid imbalance in children and adolescents. Analyzed eicosanoid concentration and proportions were classified as eicosanoid imbalance (AIT value) (6). An eicosanoid imbalance was already detected by FET in adults suffering from patients with Samter's triad (7). FET sensitivity is 96%, and FET specificity is 89% (8). Our patients had increased AIT values (mean AIT value 1.40), implicating mild eicosanoid imbalance (6). To date, however, standard FET values were established only for adults and not for children. Histologic examination of the NP tissue has demonstrated eosinophilia, which is similar to the adult form of Samter's triad (9). One patient was allergic to birch, as per positive prick test and specific IgE (ImmunoCAP assay class 2). Possible immune deficiencies, cystic fibrosis, and cilia dyskinesia were excluded in our patients using immunoglobulin measurements, sweat test, and evaluation of ciliary function. To our knowledge, this is the first time when Samter's triad was diagnosed in children/adolescents with the use of complete nasal endoscopy, immunologic examination, histologic examination of eosinophils, and the measurement of eicosanoids in FET. The laboratory tests performed in our adolescent patients match these of adults with Samter's triad. Our results imply potential need for diagnosis toward AERD in children and adolescents with NP.

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