month. According to dose schedule the monoclonal antibody Omalizumab apply to them, it applying for 6 month.
Results: All the patient improved their Urticaria between weeks 3 and fort of application of the drug, getting the control of the symptoms between the month 2 and 3 in the 5 patients, without requiring other drugs for their control, and remained asymptomatic for 3 and forth months discontinuity the product up to 6 months, not reactivity the Urticaria, the older case takes now 1 year without activity of his disease.
Conclusions: Omalizumab must be considered to be another therapeutic alternative in patients with idiopathic Urticaria.

ASPIRIN-EXACERBATED RESPIRATORY DISEASE

282
Successful Treatment of Severe Nonallergic Asthma by Omalizumab. An Observation

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Background: Omalizumab (Oma) is used in Europe for treatment of severe allergic asthma we describe the use of anti-IgE for severe nonallergic aspirin-induced asthma.
Methods: A non-smoking woman with negative family anamnesis, negative skin prick test (28 allergens), no specific IgE antibody and IgE of 107 kU/l developed a chronic rhinitis with anosmia, sinusitis and severe asthma at the age of 31 years. She suffers from nasal polyps (7 operations) and intolerance to aspirin and ibuprofen as well as alcohol; both leading to severe breathlessness. She was treated over years without success with oral steroids, ICS, LABA's and montelukast. The asthma was not under control and unstable. Therefore, we decided to try Oma as an additional medication. Since 25.08.2010 she is receiving 150 mg Oma/month without any change in other medication. After only 2 months she reported a remarkable reduction in their bronchial and nasal symptoms including improved smell and that she can drink small amounts of alcohol and also stands aspirin. We performed appropriate provocation tests to prove this observation.
Results: The FEV1 increased from 1.4 L on the 25.08.2010 to 2.4 L after only 2 months of treatment with Oma and was stable at this normal level for the next 12 months (till October 2011). The severity of symptoms (night and day) were dramatic reduced and the quality of life increased significantly (asthma-control-test normalized from 11 to 21 points). A double-blind placebo-controlled (DBPC) test with 125, 250, and 500 mg aspirin (cumulative dose 875 mg) was negative (all-day well-being, no changes in lung function). The DBPC-test with 3 doses of alcohol (sum of 10 g) was also negative.
Conclusions: A severe, difficult-to treat nonallergic asthma with nasal polyposis, ASS, and alcohol-intolerance (Samter's syndrom) was successfully treated with additionally given Oma. The injection of 150 mg Oma per month induced a clearly improved quality of life, "an asthma under control", improved the lung function with respect to FEV1 and leads to an unexpected tolerance against ASS and alcohol. To our knowledge this is the first reported observation on successful treatment of a Samter's syndrome using Oma.

283
Relationship between Aeroallergen Sensitization and Asthma Severity in Patients with Aspirin-Exacerbated Respiratory Disease

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Background: The pathogenesis of aspirin-exacerbated respiratory disease (AERD) is presumed to involve the aspirin/non steroidal anti-inflammatory drug (NSAID)-induced abnormal metabolism of arachidonic acid, resulting in the production of 5-lipoxygenase metabolites, particularly leukotriene C4. Aspirin intolerance occurs around the same time as asthma onset, and a few of the patients with AERD had suffered from pediatric asthma. Although atopy is not associated with the pathogenesis of AERD, some of the patients with AERD have aeroallergen sensitization. There are few studies in which the association between the pathogenesis of AERD and atopy has been clarified.
Methods: Ninety AERD patients, whose aspirin sensitivity was determined by the aspirin challenge test, and 100 aspirin-tolerant asthma (ATA) patients, whose age and sex were adjusted, participated in this study. Atopy was defined as a positive reaction in an intradermal test to one or more of 19 common aeroallergens, or a positive reaction above class one in Immuno CAP RAST. We analyzed the relationships between aeroallergen sensitization and clinical settings of AERD patients.
Results: The atopic and non atopic AERD groups showed median serum total IgE concentrations of 464 and 130 IU/l (P value = 0.004), respectively. The asthma of atopic patients with AERD was milder than that of non atopic patients with AERD. (P value = 0.05) The Lund-Mackay score of atopic patients with AERD was lower than that of non atopic patients with AERD. (P value = 0.02)
Conclusions: Two-thirds of the patients with AERD showed aeroallergen sensitization. The asthma and sinusitis in atopic patients with AERD were significantly milder than those in non atopic patients with AERD. Aeroallergen sensitization might prevent the worsening of asthma in patients with AERD.

284
Persistence of Nasal and Bronchial Symptoms in Patients with Samter's Syndrome with Treatment Medical and Surgical in a 2 Year Period

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Background: Know the causes of nasal and bronchial symptoms persistence in patients with Samter's syndrome under treatment in a period of time.
Methods: Cohort study. Inclusion criteria: Patients with asthma diagnoses, hypersensitivity to aspirin and nasal polyps. Exclusion criteria: Other kind of asthma, COPD. Twelve patients were followed from June 2009 to June 2011. Nasal and bronchial symptoms were assessed every 6 months using the Visual Analogue Scale of severity (VAS) from EPOS guidelines and spirometry from GINA. All were treated with intranasal mometasone furoate 200 mcg at day, montelukast 10 mg at day, salmeterol plus fluticasone 50/100 powder 2 inhalation every 12 hours, fluticasone spray 150 mg every 12 hours, loratadine tablet 10 mg if was necessary, with modifications of doses every 3 months.Patients diagnosed at 6 months with sinusitis and nasal polyposis were administered amoxicillin plus clavulanate 1.5 g daily for 5 weeks. The patients without response at 6 and 18 months were prescribed clarithromycin 400 mg daily for 4 weeks. All patients underwent CT of the sinuses through the Lund-Mackay system, chest CT scan, skin prick test. Evaluated by otolaryngology at the 6, 12, and 18 months.
Results: In the 98, 2% had negative skin prick tests. At 6 months, 58.3% had nasal symptoms with VAS <7. At 33.3% reported bronchial relapses with FEV1 <80. At year nasal symptoms increased, with WAS> 7 in 66.6%. The bronchial relapse decreased to 16.6%. At year and a half it increased nasal