symptoms in 75% of patients, with VAS> 7. At 41.6% had obstruction of 100% and pansinusitis. They needed antibiotic scheme. At 2 years in 83.3% had a VAS>7. At 58.3% had pansinusitis. The bronchial relapse did not increase. We determined the presence of VAS> 7 and pansinusitis (OR = 4). The bronchial relapse did not influence with increasing VAS (OR = 1).
Conclusions: Nasal symptoms persistent were secondary to the nasal polyps and pansinusitis with higher levels of VAS. It was determinated a 4-fold risk over pansinusitis with a VAS> 7 (OR = 4). It should be stressed the palliative surgical treatment in earlier stages and desensitization protocols.

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Clinics and Laboratory Characteristics of Asthmatic Patients with Aspirin Exacerbated Respiratory Disease (AERD)

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Background: Clinics and laboratory characteristics of asthmatic patients with aspirin exacerbated respiratory disease (AERD): asthma, polyposis and aspirin hypersensitivity.
Methods: Asthmatic patients with AERD history were evaluated. They were evaluated about asthma severity, rhinitis severity, history of polypectomy, and atopy. Some complementary exams were performed: total and specific IgE, serum eosinophilia, spirometry and nasal fibroscopy.
Results: Forty-seven patients concluded the study. The mean age of the patients was 53.1 years old and eighty-five percent were women. All patients had nasal polyposis and 23 patients (49%) had performed polypectomy. Thirty-nine patients (83%) had moderate/ severe persistent rhinitis and thirty-6 patients (77%) had moderate or severe persistent asthma and all of them were in inhaled corticosteroid treatment. The spirometry was classified as mild obstructive ventilatory disturbed (FEV1 ^{3} 60%) in 31 patients (66%). The mean value of total IgE was 427 IU/mL. The mean number of eosinophils was 477 cell/mm^3. The specific IgE to inhaled allergens was present in 22 patients (47%), who also had family history of atopy.
Conclusions: AERD is clinic syndrome related to chronic and severe inflammation of superior and inferior respiratory tracts, and is complicated with chronic rhino sinusitis, recurrent polyposis and asthma. In this study, thirty-six patients (77%) had history of rhino sinusitis and 50% had moderate and severe asthma. Atopy was confirmed in 47% of the patients. Polypectomy was performed as therapeutic treatment in 23 patients (49%). The prevalence of AERD in asthmatic patients is around 40%, and therefore, an early diagnosis is essential.

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Association Analysis of Member RAS Oncogene Family Gene Polymorphisms with Aspirin Intolerance in Asthmatic Patients

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Background: Member RAS oncogene family (*RAB1A*) converts small G protein to rab1 protein, a inflammation of blood eosinophils. Thus, functional alterations of the *RAB1A* gene may contribute to Aspirin exacerbated respiratory disease (AERD).
Methods: Asthmatics (n = 1277) were categorized into Aspirin exacerbated respiratory disease (AERD) and aspirin-tolerant asthma (ATA). 8 SNPs were genotyped. Messenger RNA expression of the *RAB1A* gene by peripheral blood mononuclear cell is measured by Real time PCR and reverse transcriptase polymerase chain reaction (RT-PCR). Human PBMC culture supernatant expression of *11-dehydrothromboxane B2*. Protein expression of the RAB1A gene by PBMC is measured by *RNAi* (Knock down) analysis.
Results: The logistic regression analysis showed that the rare allele frequency of +41170 C>G on intron 5 was significantly lower in the AERD group (n = 261) than in the ATA group (n = 1016) ($P = 0.002$). The linear regression analysis revealed a strong association of +41170 C>G with the aspirin challenge induced-FEV$_1$ fall ($P = 0.00008$). RT-PCR and real time PCR revealed an exon-4-deleted variants. The level of full-length *RAB1A* mRNA did not differ, but the variants was significantly higher in *+41170 G* homozygotes than in *+41170 C* homozygotes ($P = 0.002$). Intron based PCR was was used to amplify transcripts of PBMC pre-mRNA in which intron-5 had been removed (mRNA) while another set of primers was used to amplify intron 5-containing pre-transcripts (pre-mRNA). Knock down analysis of RAB1A Transcripts level in hPBMC. Thromboxane B$_2$ were increased in the siRNA treated when compared those of scramble and control. After knock down analysis, the levels of thromboxane B$_2$ were significantly decreased in PBMC culture supernatant.
Conclusions: The rare allele of *+41170 C>G* may play a protective role against aspirin hypersensitivity via a lower catalytic activity of the *RAB1A* gene attributed to the increase of a non-functioning variants of *RAB1A*.

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Nasal Nitric Oxide Levels after Lysine Aspirin Nasal Challenge in Subjects with Aspirin Induced Asthma

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Background: Changes in nasal nitric oxide (nNO) levels after nasal lysine aspirin (lys-ASA) challenge have not been determined.
Methods: Fourteen patients with aspirin induced asthma (AIA) with or without nasal polyps with aspirin were included to the study. Hypersensitivity had to be confirmed by positive result of oral aspirin challenge Ten healthy subjects served as the control group. 12 mg of lys-ASA were applied unilaterally. Nasal symptoms were assessed using visual analogue scale (VAS) and nNO and peak nasal inspiratory flow (PNIF) measurements were performed before and 1, 2, 4 and 24 hours after the challenge. The result of the challenge was considered as positive when at least 20% fall of PNIF as well as 20% increase of total VAS score were observed.
Results: Ten patients (71.4%) had clinically positive result of the challenge. We observed significant fall in nNO levels in AIA patients after 1 and 2 hours after the challenge (653. 1 ± 420. 2 at baseline versus 490. 3 ± 456. 0; $P = 0.0029$ and 439. 9 ± 556. 4 ppb; $P = 0.0076$; respectively). The decrease in nNO level was more pronounced in patients with clinically positive result of the challenge (510. 1 ± 212. 5 at baseline versus 283. 3 ± 173. 4; $P = 0.005$; 159, 6 ± 166,1; $P = 0.005$ and 331. 0 ± 312.0 ppb; $P = 0.037$ after 1, 2 and 3 hours, respectively). In 4 subjects with clinically negative result of the challenge we noticed a trend towards higher nNO concentrations after lys-ASA challenge (1010. 8 ± 625. 2 at baseline vs 1341. 3 ± 670. 5 ppb after 4 hours). No significant changes in nNO levels after the challenge were observed in healthy controls.
Conclusions: NO levels decrease after lys-ASA nasal challenge in subjects with AIA and clinically positive nasal provocation. An unexpected trend towards increase in nNO levels was observed in subjects with AIA and clinically negative provocation Potential usefulness of nNO measurement in aspirin nasal provocation needs further evaluation.

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IL1B but not IL8 Polymorphisms Are Increased in Aspirin Exacerbated Respiratory Disease Patients Versus Aspirin Tolerant Asthmatics

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