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[Abstract:0005] OP-105 [Accepted: Oral Presentation] [Asthma Allergy]

IL-33, IL-25, and ECP Levels were Decreased in Severe Persistent Allergic Asthma Patients with the Anti-IgE (Omalizumab) Treatment

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Objectives: Interleukin-33(IL-33), considered as an alarmin released upon tissue stress or damage, is a member of the IL-1 family and binds the ST2 receptor. First described as a potent initiator of type2 immune responses through the activation of T helper2(TH2) cells and mast cells, IL-33 is now also known as an effective stimulator of TH1 immune cells, natural killer (NK) cells, iNKT cells, and CD8 T lymphocytes. Moreover, IL-33 was shown to play an important role in several cancers due to its pro and anti-tumorigenic functions. IL-25, also named IL-17E, is a distinct member of the IL-17 cytokine family, which can promote and augment T helper type 2 (Th2) responses locally or systemically.

Methods: Group IA (n:32, pre-omalizumab), when they were first diagnosed and Group IB (post-omalizumab) after 12 months of treatment during the disease remission. They all underwent omalizumab (anti-IgE; Xolair®; Novartis Pharmaceuticals, Basel, Switzerland) treatment for 12 months every two weeks (375mg).The concentrations of IL25 and IL33 in the serum were measured using commercially available Enzyme-linked immunosorbent assay kits. Total and specific IgE levels were enumerated by fluoroenzyme immunoassay (ImmunoCAP) using an ImmunoCAP (Pharmacia, Uppsala, Sweden) kit. Values>100 and 0.35 kU/L for total and specific IgE levels, respectively, were considered abnormal. Serum levels of 25(OH)D were quantified by a radioimmunoassay (Cobra Quantum, Packard, MN, USA) and categorized into sufficient (‡30 ng/mL), deficient (<20 ng/mL) or insufficient (20 to<30 ng/mL) based on previous recommendations. Blood samples for IL-33,IL-25,ECP,25(OH)D, total IgE measurement were always taken in the morning. The results were reported as means of duplicate measurements. Medical history, lung function tests, measurement of FENO were performed on the same day.

Results: In our study, we found significant changes in the, IL-33, IL-25, FeNO, total IgE, ACT, FEV1, and FVC at baseline and 12 months after the onset of anti-IgE treatment.

Conclusion: The attachment of IgE in the mast cell activation typically requires the cross-linking of Fc_2RI-bound IgE by antigen or anti-IgE antibodies. In a transcriptome analysis of 8,793 genes, sensitization of the mast cells with monoclonal IgE was found to upregulate 58 genes more than two-fold, compared to the results obtained from the non-sensitized mast cells. We also believe that the circulating IL-25, IL-33, may also have a key role for the relationship between severe allergic asthma and chronic inflammation. Nonetheless, further comprehensive, large-scale studies are needed to confirm the clinical relevance of these preliminary results.

Keywords: IL-33, IL-25, omalizumab, asthma