FRI0159 LONGSTANDING ACTIVE GIANT CELL ARTERITIS (GCA) TREATED WITH INFlixIMAB: REPORT OF FOUR CASES

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Background TNF-alpha was demonstrated in up to 60% of the cells in all areas of inflamed arteries of patients with GCA, suggesting a primary role in the inflammatory process. Infliximab, a chimeric monoclonal anti-TNF-alpha, demonstrated remarkable efficacy and safety in the treatment of several rheumatic conditions associated with a chronic inflammatory response.

Objectives To date, no data are available on the therapeutic employment of infliximab in patients with GCA.

The aim of this study was to evaluate the efficacy of infliximab infusions in longstanding, still requiring corticosteroids (CS) GCA.

Methods Four patients (3F,1M; median age:75 ys.) with longstanding (median disease duration: 4 ys.), biopsy proven, active GCA, still requiring CS and whose disease course has been complicated by serious CS-related adverse events, were scheduled to receive 3 infusions of infliximab (3 mg/Kg) at weeks 0, 2 and 6. If a clinical and humoral remission was obtained, CS were withdrawn after the 2nd infusion.

Results Three patients had a complete remission of clinical signs/symptoms and normalisation of ESR and CRP after the 2nd infusion. The remission persisted after the 3rd infusion and over the follow-up (7, 6 and 6 months, respectively).

The fourth patient did not respond to therapy and withdrew from the study after the 2nd infusion. Infliximab was well tolerated without any side-effects.

Conclusion Our encouraging results suggest that TNF-alpha blockade may represent a valid alternative therapeutic approach to longstanding GCA, still requiring CS and complicated by severe adverse events. The small number of patients and the open study design do not allow conclusive considerations.

FRI0160 CHEMOKINE AND CYTOKINE PROFILE IN SYSTEMIC VASCULITIDES

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Background Systemic vasculitides include a heterogeneous group of clinical syndromes characterised by inflammation and necrosis of blood vessel walls. Any type of vessels throughout the body may be involved with protean clinical manifestations, and pathogenetic mechanisms remain mainly unknown.

Objectives Aim of our study was to test the hypothesis that chemokines, cytokines, and eosinophilic inflammation markers could be involved in systemic vasculitides pathogenetic process.

Methods We studied 19 patients with systemic vasculitides diagnosed according to ACR 1990 criteria: 7 pts. with Churg-Strauss vasculitide (CS), 7 pts. with Wegener's Granulomatosis (GW), 5 pts. with Panarteritis Nodosa (PAN) and 24 normal healthy subjects (NHS) as controls. Serum levels of the following mediators: macrophage inflammatory protein 1α (MIP1α), monocyte

chemotactic protein 1 (MCP1), RANTES, interleukin 5 (IL5), granulocyte macrophage-colony stimulating factor (GM-CSF) and eosinophil cationic protein (ECP) were tested by ELISA. For the statistical analysis U-Mann Whitney test has been used.

Results Among chemokines, serum levels of MCP1 and RANTES were significantly higher in systemic vasculitides than NHS ($p < 0.001$ and $p < 0.007$ respectively), while no differences were demonstrated in MIP1α serum levels. Regarding cytokines studied, IL5 and GM-CSF serum levels were significantly higher in systemic vasculitides than NHS ($p < 0.001$). Also ECP serum levels resulted increased in systemic vasculitides than NHS ($p < 0.04$) with a significant correlation between ECP levels and blood eosinophilia in vasculitides patients. Furthermore, a specific subgroup analysis showed that serum levels of RANTES, IL5, and ECP resulted increased mainly in CS and GW, while MCP1 and GM-CSF mainly in CS and PAN.

Conclusion Our study demonstrates the increased production of chemokines and cytokines in systemic vasculitides patients. Moreover, the finding of higher ECP serum levels in our patients induce us to hypothesise that eosinophils are actively involved in vasculitides etiopathogenesis.

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FRI0161 HUMORAL AND CELL-MEDIATED IMMUNE RESPONSE TO COW'S MILK PROTEINS IN BEHÇET'S DISEASE

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Background Behçet's disease (BD) is a multisystemic inflammatory disease characterised by a number of immunological variables. The relevance of these findings is not sufficient to support an autoimmune pathogenesis. Consumption of cow's milk proteins might be a risk factor for the development of autoimmune disease.

Objectives To investigate the humoral and cell-mediated immune response against cow's milk proteins in BD.

Methods Sera from 47 patients with BD and 38 healthy subjects were studied. Anti beta-casein (bC) and anti beta-lactoglobulin (bLG) antibodies were detected by ELISA. Peripheral blood mononuclear cells from 16 BD patients and from 8 healthy controls were cultured in the presence of PHA, beta-casein or beta-lactoglobulin and IL-4 and Interferon-gamma measured in the culture supernatants by ELISA.

Results Significant higher levels of anti-bC and anti-bLG were found in the sera of patients with active disease (anti-bC-IgA: 1.89 ± 2.2 SDU; -IgG: 3.64 ± 5.93 ; anti-bLG-IgA: 2.07 ± 2.51 ; -IgG: 1.72 ± 2.7) compared with those of inactive patients (anti-bC-IgA: 0.68 ± 0.92 ; -IgG: 0.83 ± 1.26 ; anti-bLG-IgA: 0.70 ± 1.12 ; -IgG: 0.37 ± 0.65) or controls. Higher levels of IFN-gamma but not IL-4 were found in the supernatants of active patient cultures but not in those of patients with inactive disease or controls in both bC and bLG studies. Levels were comparable to those obtained with PHA.

Conclusion The occurrence of anti-bC and anti-bLG in BD and studies on the cellular immune response with Th1 cytokine secretion might further support the putative role of cow's milk