cytokines and chemokines including IL-12B (P=0.07), IL-6 (P=0.07), IL-10 (P=0.12), IL-18 (P=0.19), and IL-4 (P=0.63).

Conclusion: This study illuminates the downstream effects of neutralizing TNFα not previously investigated. Adalimumab had a pronounced effect on downregulation of the inflammatory CXCL subfamily chemokines IL-8, CXCL5, CXCL9, and CXCL10. This helps explain findings of diminished inflammatory cell migration into joints seen in the first trials with TNFα inhibitors and could be an important mechanism of action of TNFα inhibitors.[2] Further characterization of downstream effects of the multiple DMARDs used for the treatment of immune mediated inflammatory arthritis will help guide treatment strategies for these patients.

REFERENCES


AB0070
THE RELATIONSHIP BETWEEN THE LEVEL OF NESFATIN-1 AND THE CLINICAL MANIFESTATIONS OF RHEUMATOID ARTHRITIS
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Background: Nesfatin-1 is actively studied in the pathogenesis of metabolic disorders. An excess of this factor in the brain leads to loss of appetite, a feeling of fullness, as well as a decrease in body weight. Similarly, elevated levels of nesfatin are associated with depressive disorders [1]. We studied the level of nesfatin in the serum of patients with rheumatoid arthritis (RA) and found a relationship with systemic inflammation and functional impairment [2].

Objectives: To study the relationship of serum nesfatin-1 levels with the clinical manifestations of RA.

Methods: To identify the relationship between serum nesfatin-1 and the clinical manifestations of rheumatoid arthritis, all patients were divided into 2 groups. The first group - 1 (66 patients) with elevated serum nesfatin (> 37.95 ng/ml). The second group - 2 (44 patients) - with normal values (<37.95ng/ml). In both groups, we studied the clinical manifestations of RA.

Results: A high level of nesfatin in RA patients was typical for patients with a higher degree of activity in DAS28 (y2 = 8.37; p = 0.04), seroscopy for the rheumatoid factor (RF) - (y2 = 5.53; p = 0.02), the duration of the disease is more than 10 years (y2 = 9.53; p = 0.01). At the same time, there was no significant correlation between the level of nesfatin and the extra-articular manifestations of RA (y2 = 2.09; p = 0.14) and the degree of radiological damage to the joints (y2 = 4.45; p = 0.21).

Conclusion: This study shows that serum Nefatin-1 levels are significantly higher in patients with more unfavorable RA, than in RA with minimal clinical manifestations. These data confirm the pathogenetic role of Nesfatin-1 in the development of clinical manifestations associated primarily with RA activity, but to a lesser extent with organ lesions in RA. The relationship of the level of nesfatin with the duration of the disease is of particular interest, since there is no correlation with the degree of X-ray damage to the joints and organ damage. There are literary data on the relationship of depression and late stage of RA [3].

REFERENCES

Disclosure of Interests: None declared

AB0071
SERUM S100A8/A9 (CALPROTECTIN) IN FAMILIAL MEDITERRANEAN FEVER DOES NOT CORRELATE WITH DISEASE ACTIVITY
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Background: Familial Mediterranean Fever (FMF) is caused by mutations in MEFV. The protein product pyrin is expressed in monocytes, neutrophils and eosinophils. Acute inflammatory attacks are accompanied by a dramatic hepatic acute phase response. S100A8/A9 is damage associated molecular pattern and a TLR4 ligand expressed in neutrophils, monocytes and early infiltrating macrophages. We aimed to investigate S100A8/A9 in 39 patients with FMF, 45 healthy carriers and wild type controls.

Objectives: To measure S100A8/9 in patients with FMF, carriers and healthy controls.

Methods: All patients were genotyped. Patients and healthy controls (HC) serum S100A8/A9 levels, cell surface expression on monocytes and neutrophils as well as intracellular peripheral blood mononuclear cells (PBMC) expression were measured by flow cytometry (FACS). CD14 cells were isolated and following overnight incubation with or without LPS, S100A8/A9 was measured in the supernatants by ELISA. Patients and HC monocyte apoptosis was compared.

Results: Serum levels were measured in 84 samples from 31 patients with homozygous or compound mutations [median 9061ng/ml [range 500-38470], 79 samples from 39 symptomatic patients who were MEFV heterozygotes [median 9394ng/ml [range 1744-38119], 80 samples from 45 individuals with MEFV variants but without clinical features of FMF [median 10939ng/ml [range 2447-40000]. There was no difference in calprotectin concentrations between the different mutations. All the groups described had significantly higher levels than healthy controls (n=16 median 2836ng/ml [range 1058-6175]<p=0.001). Minimal monocyte and neutrophil cell surface expression was detectable. Following LPS stimulation there was significantly more S100A8/A9 detected in the supernatants in patients than healthy control CD14. There was also a trend to an increased intracellular monocyte S100A8/A9 expression.

Conclusion: Patients with pyrin mutations both with and without clinical disease have greatly elevated serum S100A8/A9 levels without detectable cell surface expression in well-controlled disease with a trend to an increased monocyte intracellular expression. Upon monocyte stimulation with LPS, increased S100A8/A9 is secreted. The exact mechanism by which these patients, especially those with mutations but no clinical disease, demonstrate sustained elevated serum S100A8/A9 remains to be elucidated but does not appear to result in a significant clinical sequelae.

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