a trend in favor of ADA, except for Sarcoidosis subgroup in the VISUAL II trial (Figure). Overall safety for both trials has been previously reported [1,2].

Conclusions: These exploratory analyses from the VISUAL I and VISUAL II trials show significantly higher efficacy in ADA-treated patients over PBO in "idiopathic/other" diagnoses of patients with both active and inactive non-infectious uveitis. Furthermore, across different uveitis etiologies, these analyses suggest that ADA-treated patients had a prolonged time to treatment failure compared to PBO.

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FRI0619 COMPARING CANAKIMUMAB AND ANAKINRA IN YOUNG GREEK WOMEN WITH RESISTANT RECURRING PERICARDITIS

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Background: Resistant – recurring pericarditis (RRP) is often associated with autoinflammatory syndromes. Conventional therapy with glucocorticoids and colchicine is often enough unsuccessful, thus nowadays the use of anakinra in such cases is becoming common every – day practice.

Objectives: Our goal was to determine whether RRP was associated with gene mutations relevant with autoinflammatory diseases and then compare the effectiveness of canakimumab and anakinra.

Methods: We studied 18 patients with RRP (all women, from 14 to 36 years old) determing the existence of mutation from the infevers database (25 genes, from MEFV and TNFRSF1A to MVK and SLC 29A3, all related with autoinflammatory diseases). All the patients were proven positive to one of the above mutations (all heterozygous) and then treatment with anakinra (n=9, 100mg sc once daily) and canakimumab (n=9, 150mg sc q4wk) was administered.

Results: During the study (3 years), all anakinra patients stopped receiving per os glucocorticoids and were free of any new pericarditis episodes. 3 of them (33%) stopped any treatment after a year of anakinra therapy and none was presented with any skin reactions.

On the contrary, all canakimumab patients were not able to free themselves of the glucocorticoid treatment, since at least one new episode of pericarditis to each patient was recorded. Two of them were free of pericarditis the last year of the study (though being treated with 2,5mg prednisolone per os daily).

Statistically speaking, the use of IL-1R antagonist in recurrent – resistant pericarditis associated with autoinflammatory mutations was proven much more successful ($p<0,01$) than the use of the IL-1b Mab.

Conclusions: Though the sample of patients (n=18) was too small in order to set us able to reach any safe conclusions, it is quite possible that anakinra may be one effective solution that can prevent the long term administration of corticoids to patients with recurrent pericarditis Canakimumab was not recorded with similar positive results.

Disclosure of Interest: None declared

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FRI0620 MIR-204-3P INHIBITS THE PRODUCTION OF TLR4-RELATED CYTOKINES IN FAMILIAL MEDITERRANEAN FEVER BY TARGETING THE PIK3 SIGNALING PATHWAY

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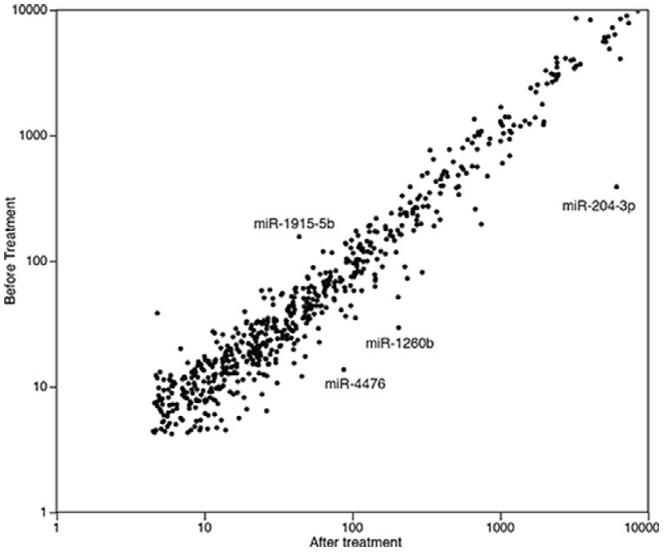
Background: MicroRNAs (miRNAs) are endogenous small RNAs and post-transcriptionally regulate gene expression by pairing with target. There has been emerging evidence showing the association of aberrantly expressed circulating miRNAs in the serum with the pathogenesis or progression of diseases including cancer and autoimmune disease. Although a number of circulating miRNAs associated with inflammation have been identified, the roles of them in patients with FMF and the underlying mechanism remain to be elucidated.

Objectives: The aim of this study was to identify a serum miRNAs profile and potential biomarkers in FMF and clarify their gene targets for understanding the pathogenesis of autoinflammatory diseases.

Methods: We performed miRNA microarray in the serum from FMF in attack and in remission. We subsequently examined the expression of miRNAs that varied before and after the attack in macrophages derived from THP-1 cells stimulated with toll-like receptor (TLR) ligands. Macrophages derived from THP-1 cells transfected with pre-miRNA and anti-miRNA were stimulated with TLR ligands for 24 hours. We collected the supernatants for the quantification of inflammatory cytokine production. To identify the target genes, we overexpressed its miRNA and performed Agilent expression microarray. Transfection with reporter construct and pre-miRNA and anti-miRNA was performed to confirm suppression of target mRNA.

Results: We found that miR-204–3p was greatly decreased in the serum from FMF patients in attack. In vitro study, the expression of miR-204–3p was suppressed by LPS stimulation in macrophages derived from THP-1 cells. Inhibition of miR-204–3p significantly induced the production of TLR4-related cytokines whereas overexpression of miR-204–3p inhibited their production. Bioinformatic analysis showed that miR-204–3p is predicted to target genes implicated in TLR pathway through regulation of PIK3 signaling. Reporter assay revealed that miR-204–3p directly suppressed the luciferase activity of 3'UTR of PIK3CG reporter construct.



Conclusions: These data suggest that serum miR-204–3p has a potential as a useful biomarker among patients with FMF and that miR-204–3p plays a critical role as a suppressor to regulate the production of TLR4 related cytokines by targeting PIK3 signaling pathway.

Disclosure of Interest: None declared

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FRI0621 PREVALENCE OF DIFFERENT ORBITAL ANATOMIC STRUCTURES AFFECTION IN IGG4-RELATED OPHTHALMIC DISEASE: SINGLE CENTER EXPERIENCE

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Background: According to 2009 Japanese nationwide survey of IgG4-related disease (IgG4-RD), orbit is the leading site of affection [1]. However diagnostic criteria and nomenclature of IgG4-RD were developed a few years later [2,3].

Objectives: To evaluate the peculiarities of clinical, laboratory, histological and immunohistochemical presentation of IgG4-related ophthalmic disease (IgG4-ROD).

Methods: During 2004 – 2016, 82 patients were diagnosed with IgG4-RD 53 of whom had IgG4-ROD (men – 17, women - 36). The diagnoses of IgG4-RD and IgG4-ROD were established using comprehensive diagnostic criteria [2,3]. In all patients full clinical, ophtalmological, dental and serological (rheumatoid factor, C-reactive protein, IgG, IgG4, IgM, IgA, ANA, anti-Ro/La, C3/C4 complement) examination was carried. In all cases diagnosis was verified pathomorphologicaly with immunohystochemical staining (anti-CD 138, CD 68, IgG, IgG4, κ-chain, λ-chain), but only in 43 patients the diagnosis of IgG4-RD was established on orbital tissues biopsy. Some patients at baseline were tested on B-cell clonality