the macrophages depended on the elasticity of bacterial cell walls and on the time of their joint cultivation.

Conclusions: LAB and bifidobacteria strains stimulate immunomodulatory cytokines and active oxygen and nitrogen oxides compounds production in macrophages. Strains with a more elastic cell wall according to AFM data demonstrated higher resistance to intracellular digestion in macrophages and higher level of their activation. AFM might be considered as a fast and accurate method to assess parameters of probiotic strains cell wall to predict their beneficial immunomodulatory properties. Further large-scale preclinical research needed for future application in rheumatic diseases treatment and prevention.

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AB0044 HYPER Ig-D SYNDROME TREATMENT WITH HYDROXYCHLOROQUINE

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Background: Hyper IgD syndrome is a minor form of mevalonate kinase deficiency caused by a its gene mutation and considered an auto-inflammatory disease inherited in an autosomal recessive manner, characterised by periodic episodes of fever, arthralgia, lymphadenopathy, skin rash, headaches, and abdominal pain. These attacks can occur spontaneously or be triggered by infections. Treatment options include courses of NSAIDs, steroids, colchicine, statins, I.V immunoglobulin, Cyclosporine, anti-IL-1 agents (anakinra or canakinumab) and anti-TNF antagonist Etanercept, with transient response. To date, there is no report with long term hydroxychloroquine treatment¹⁻³.

Objectives: To report a case of Hyper Ig-D syndrome with multiple recurrent episodes and successful treatment using Hydroxychloroquine alone.

Methods: We report a case of 36-year-old female, presented with a first manifestation at age 35 with fever and extended cellulitis of the left arm treated successfully with antibiotic, rapidly followed with urticarial rash involving the upper side of the body, face and upper limbs migratory type lasting less than 24 hours suspecting allergic reaction. She was under supportive treatment (anti-histaminic and low dose steroids) that improved her condition temporarily. She developed multiple recurrent similar episodes for 7 months with high grade fever up to 39°C, inflammatory polyarthralgia affecting small joints, recurrent left elbow erythematous plaque with few subcutaneous nodules, considered as panniculitis type lesion (Picture-1) along with urticarial rash. Series of tests were undertaken along with skin biopsy.

Results: Biopsy of the lesion showed intense dermal neutrophil infiltrates. CRP fluctuating between 50 to 95 mg/L during the episodes. Neutropenia (1.7 to 1.9), Normal findings for ANA and SSA antibodies, ACE, Lysozyme enzyme, C3 and C4. Other infectious work-up was negative including Quantiferon God test. Protein electrophoresis found hypergammaglobulinemia. The Ig-D level was 263 mg/L and on another occasion 313 mg/L (normal <153 mg/L), the samples were taken during flare up and after recovery. The patient was treated by Colchicine for more than 3 months without beneficial effect. A trial with hydrochochloroquine 400 mg daily brought a progressive improvement with lesser episodes at 3rd month and no recurrences at 6 months. After one year of therapy the patient was symptomless on a lower dose 200 mg daily with completely normalised inflammatory markers.

Conclusions: We conclude that Hyper Ig-D syndrome can be treated by hydroxy-chloroquine. This is the first report in the literature conducting this treatment option that can bring attention for further case by case trials.

Abstract AB0044 – Figure 1

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AB0045 ACTIVATED RNASE L AS A NOVEL DISEASE ACTIVITY BIOMARKER IN PSORIATIC ARTHRITIS

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Background: Almost 60% of psoriasis (PsO) patients with psoriatic arthritis (PsA) are estimated to be untreated, undertreated or/and undiagnosed.¹ Delayed diagnosis leads to permanent joint damage causing major functional decline and diminished quality of life. However, in the absence of diagnostic biomarkers, the diagnosis of psoriatic arthritis is clinical and hence difficult to establish from non-rheumatologists.² Recent studies have demonstrated the upregulation of type I interferon (IFN)–inducible genes in paired peripheral blood cells (PBC) and synovial biopsies of patient with PsA.³ Oligoadenylate synthetases (OAS) are type I IFN-stimulated family of proteins that are activators of the latent Ribonuclease L (RNase L) pathways. The OAS-RNase L system is a potent host antiviral IFN-responsive system that is completely inactive in normal conditions, but once activated mediates a broad array of pro-inflammatory cellular processes.⁴