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A longitudinal study to investigate the role of il-6 and il-17 in the pathogenesis of early undifferentiated inflammatory arthritis

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Background: Nearly half the patients presenting to early arthritis clinics with peripheral inflammatory undifferentiated arthritis (UA) will evolve into rheumatoid arthritis (RA). However the factors that govern the transition are not fullyunderstood.

Previous research indicates that IL-6 is a pro-inflammatory cytokine in RA that can drive Th17 cell development in mice and humans. Data from experimental arthritis models suggest that Th17 cells are pathogenic via production of the pro-inflammatory cytokines IL-17 and TNFα, leading to monocyte and fibroblast activation, and involvement in osteoclastogeneis and joint damage.

Objectives: The aim of this study is to investigate if interleukin-6 (IL-6) and/or interleukin-17 (IL-17) and Th17 cells are biomarkers for disease progression and severity in early UA and RA patients.

Methods: We performed a longitudinal study recruiting 20 patients with either undifferentiated inflammatory arthritis or early rheumatoid arthritis. We also recruited 20 age and sex-matched healthy controls.

The patients were assessed at baseline, 6 and 12 months for the ACR criteria, Rheumatoid factor (RF), Anti-CCP, ESR, CRP, X-Ray of the hands and feet, joint count, patient global assessment, DAS28 and Quality of Life (HAQ) measurements. Peripheral blood and serum samples were taken and PBMC isolated. Cell subset analysis (CD3, CD4/CD8, CD19, CD14, Tregs) was performed ex vivo. PBMC were also stimulated ex vivo with medium, PMA/Ionomycin or LPS and then stained intracellularly for IL-17, IL-6, TNFα and IFNγ together with T cell and monocyte markers. Cells were also cultured overnight under these conditions for collection of cell culture supernatants for cytokine analysis.

Results: Our initial analysis demonstrates that significant correlations are present at baseline between SJC and the percentage of CD3+CD4+IL6+ T cells (r=0.63, p=0.009) and % CD14+IL6+ monocytes, (r=0.51, p=0.035); and for ESR and MHAQ with %CD3+CD4+IL6+ T cells (r=0.58, p=0.01, and r=0.58, p=0.044, respectively). A significant negative correlation was found for SF36 Physical Function and SF36 role of limitation due to Physical impairment with %CD3+CD4+TNFα+ T cells (r=-0.56, p=0.023, and r=-0.62, p=0.011, respectively). We also observed a negative correlation between SF36 Pain and %CD3+CD4+IL17+ T cells (r=-0.52, p=0.038). In addition, a significant difference was found between HCs and patients (values are expressed as mean ±SD) in the percentage of CD19+ B cells (HC 6.03±1.56 vs., Patients 8.3± 4.03, p = 0.03), CD3+CD4+ T cells (HC 37.67±11.61 vs. Patients 30.19±10.61, p=0.04) and CD3+CD4+TNFα+ T cells (HC 64.41±11.64 vs. Patients 73.91±11.84, p= 0.03).

Conclusions: We are currently analysing if the clinical and/or immunological parameters at baseline, or during follow-up, correlate with disease outcome at t=12 months. We will also analyse sera and cell culture supernatants for cytokine expression via Luminex/ELISA and correlate these to clinical outcome at 12 months.

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Therapy of the osteoarthritis of the temporomandibular joint

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The treatment of osteoarthritis primarily consists of rest therapy (restricting jaw movements), the use of pharmaceuticals (analgesics, antiinflammatories), splint therapy, thermotherapy or mini-invasive therapy. Our study investigated the effectiveness of the various therapeutic options in the treatment of osteoarthritis in 6 month long term study. We compared the effectiveness of rest therapy (restricting mouth opening, analgesic therapy), splints, arthrocentesis of the upper joint space, and arthrocentesis in combination with splint therapy. We looked at 100 patients diagnosed with osteoarthritis of the temporomandibular joint. We only included patients with symptoms in one temporomandibular joint (TMJ). This study shows that arthrocentesis combined with the use of a splint is a beneficial first-stage treatment method for osteoarthritis. (80% patients with good outcome 6 months after commencement of therapy).

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