THU0512

WHAT IS HIDDEN BEHIND SYSTEMIC JUVENILE IDIOPATHIC ARTHRITIS, ADULT ONSET STILL'S DISEASE AND SECONDARY MACROPHAGIC ACTIVATION SYNDROME. THE UTILITY OF THE BIOMARKERS

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Background: Systemic Juvenile Idiopathic Arthritis (SJIA) and adult onset Still's disease are rare diseases of unknown etiology that share several clinical and laboratory features. The pathogenesis of these two diseases is complex and multifactorial, with an important role of innate immunity: activation of neutrophils and macrophages and increased levels of cytokines dependent on the activation of the inflammasome. A proportion of these patients will develop a Macrophage activation syndrome (MAS), a serious and life-threatening complication.

Objectives: To identify immunological markers that allow a differential diagnosis between patients with inactive SJIA, active SJIA or MAS. To evaluate if there are differences between biomarkers in SJIA and adult onset Still's disease

Methods: Observational, prospective and multicenter study. Inclusion criteria: patients with SJIA or adult onset Still's disease, followed in Paediatric or Adult Rheumatology Department from 5 Madrid hospitals. Group 1: Patients with active SJIA or adult onset Still's disease +/- diagnosis of secondary MAS at the first visit, have been followed-up performing a second clinical and analytical visit at 3 months. Group 2: Patients with inactive SJIA or adult onset Still's. Clinical and analytical data were collected, PBMCs were obtained before and after treatment, and several leukocyte subtypes were evaluated by flow cytometry (FACS). The intracytoplasmic expression of TNF- α , IL-1b and IL-6 in monocytes was determined by flow cytometry before and after stimulation with lipopolysaccharides (LPS) in the presence of monensin (Golgi inhibitor). In addition, IL-18 levels were determined by ELISA in all visits.

Results: A total of 25 patients were included (18 SJIA and 7 adult onset Still's diseases), 2 of them being excluded from the analysis because exclusions criteria. 30,4% patients presented MAS at the first visit (5 SJIA and 2 adult onset Still's disease) and other 2 at some time during the disease. The median of IL-18 was 296.1 (CI 108.3-346.0) in patients who have never had MAS and 899.85 (CI: 397.32-1095.6) in the other group. Patients with active MAS had a CD4/CD8 ratio close to the lower limit (1.1 +/- 0.1 SD) reflecting a decrease in the CD4 + population and an increase in CD8 +. On the other hand, the rest of the groups presented a CD4/CD8 ratio of 2.6 +/- 0.5 SD in patients without disease activity and 2.0 +/- 0.7 SD in patients with active SJIA or adult onset Still's disease but without MAS. No significant differences were found between patients with SJIA or adult onset Still's disease.

Abstract THU0512 – Figure 1

Conclusion: The results of this study show that the analysis of three determinations: %CD4, %CD8 and the CD4/CD8 ratio can help in the early detection of patients with MAS secondary to SJIA or adult onset Still's disease. The determination of IL-18 has been in our study a marker of MAS, similar to several published studies. No significant differences were found in the immunological tests between patients with SJIA and adult onset Still's disease with active or inactive disease.

REFERENCE:

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THU0513

WHOLE BLOOD CELLS FROM PATIENTS WITH SYSTEMIC JUVENILE IDIOPATHIC ARTHRITIS (SJIA) IN CLINICAL INACTIVE DISEASE DISPLAY A DYSREGULATED RESPONSE TO TLR-4 STIMULATION

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Background: Systemic juvenile idiopathic arthritis (SJIA) is a polygenic autoinflammatory disease. Innate immune mechanisms appear to play a central role in the pathogenesis of the disease. Nevertheless, a better understanding of the pathophysiology of SJIA is still needed to identify patients responsive to IL-1 or IL-6 targeted therapies.

Objectives: In this study, we evaluated the production of IL-1 β , IL-6 and TNF- α by fresh whole blood cells isolated from SJIA patients in disease remission, after stimulation with the TLR-4 ligand lipopolysaccharide (LPS), and we investigated whether SJIA patients that respond or not respond to treatment with the IL-1 receptor antagonist anakinra show a different response.

Methods: We collected fresh whole blood samples from SJIA patients during clinical inactive disease (inactive SJIA, n=19) and SJIA patients during active disease (active SJIA n=4). Active and inactive disease was defined at time of sampling. As controls, fresh whole blood samples from healthy subjects (HS, n=10) were used. Whole blood cells were left unstimulated or stimulated with 10ug/mL of LPS for 24 hours. Cytokine levels (IL-1 β , IL-6 and TNF- α) released in the supernatants were measured by ELISA. Response to anakinra was defined as achievement of clinical inactive disease off glucocorticoids at 6 months after initiation of anakinra treatment.

Results: We found that LPS-stimulated cells from inactive SJIA patients released significantly higher amounts of all the inflammatory cytokines tested, compared to HS (p<0.01). In addition, cells from inactive SJIA patients produced significantly higher levels of IL-1 β also compared to active SJIA patients (p<0.05). When we divided inactive SJIA patients in two groups (responders and non-responders), depending on the clinical response to anakinra treatment, we observed that in both groups of patients IL-1 β , IL-6 and TNF- α levels were significantly higher than those observed in HS. In addition, we found that LPS-stimulated whole blood cells from non-responder inactive SJIA patients released significantly higher levels of IL-1 β and TNF- α compared to responder inactive SJIA patients.

Conclusion: Our preliminary results show a dysregulated production of inflammatory cytokines by whole blood cells from SJIA patients in remission disease, when stimulated with a TLR-4 agonist.

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THU0514

ROLE OF THE ORAL MICROBIOME IN CHRONIC NON-BACTERIAL OSTEOMYELITIS IN CHILDREN

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Background: Chronic non-bacterial osteomyelitis (CNO) is a rare autoinflammatory disease of the bone and bone marrow. The presentation of the disease is broad, but pain and osteoedema on MRI are the hallmarks of CNO. While the activation of inflammasomes and the subsequent release of IL-1 β seems to play a central role, the exact molecular pathophysiology of CNO is largely unexplained. In respect of the potential role of the microbiome to date only experimental data on mice are available, indicating that the manipulation of the microbiome through dietary modulation might alter the course of the disease and also plays a role in the release of IL-1 β from innate immune cells¹.

Objectives: To identify the impact of the oral microbiome on CNO in children and adolescents. Dysbiosis as a state of pathological alteration of the microbiome has been discussed as an environmental factor in rheumatic diseases in adults and in children². Human data in CNO are lacking.

Methods: 20 patients (12 male and 8 female, aged 3 to 13 years) with a median of 10.3 years were examined. Two different sites of the oral cavity (center and left part of the tongue) were swabbed. In total, 143 individual samples were retrieved and subjected to 16s r-RNA amplicon sequencing: DNA was extracted using the QIAamp® DNA Microbiome Kit. The V4 region of the 16S ribosomal RNA gene was amplified by PCR with V4 specific primers. A limited cycle amplification step was performed. Libraries were normalized and pooled, and sequenced on a MiSeq system (Illumina). Raw sequence reads were demultiplexed and clustered into sub-OTUs (operational taxonomic units) via Deblur in Qiita. Qiime2 was used to calculate alpha- and beta- diversity for multiple alternative metrics. Statistical significance was calculated with non-parametric tests (Mann-Whitney and Permanova).

Results: The administration of medication in general (mostly NSAIDs) had a significant impact on the microbiome (p-value < 0.02). With limited statistical power of the small cohort, gender, age and disease activity do not significantly influence the microbiome.

There is a significant difference between the variable swab sites at the 1% level (p<0.01). Whereas swabs from the center of the tongue exhibit more *Streptococci salivarius*, the left outer part of the tongue shows more *Haemophilus parainfluenza*.

Conclusion: To the best of our knowledge, this is the first study of the oral microbiome in children and adolescents in CNO. Our data suggests that administration of medication to CNO patients alters the microbiome. It is also influenced by a change of the swab site. Limited patient numbers did not allow to show an effect on disease activity, but enrollment will continue.

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Paediatric rheumatology

THU0515 PAIN IS THE MAIN DETERMINANT OF WELL-BEING IN OLIGO- AND POLYARTICULAR JIA: EVIDENCE FROM THE PHARMACHILD REGISTRY

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Background: Juvenile idiopathic arthritis (JIA) affects patients' well-being as the result of a complex interplay of multiple factors, including disease activity, symptoms, physical and emotional quality of life, and treatment burden. Little evidence exists about the relative contribution of these elements to disease impact.

Objectives: 1) To identify direct and indirect determinants of well-being, expressed by VAS-measured patient/parent global assessment of well-being (PGW), in children with oligo- and polyarthritis. 2) To assess whether the impact of the major determinants varies with level of disease activity.

Methods: We analyzed data of 1873 patients evaluated in 4464 prospective visits from the international JIA pharmacovigilance registry Pharmachild. Patient-reported outcomes were collected through the Juvenile Arthritis Multidimensional Assessment Report (JAMAR). Evaluated predictors of PGW included VAS-measured physician global assessment, inflammatory markers, active joint count, VAS-measured pain, morning stiffness duration, Juvenile Arthritis Functional Score (JAFS), Physical (PhHS) and Psychosocial (PsHS) subscales of the Pediatric Rheumatology Quality of

Life (PRQL) scale, disease damage measured through the Juvenile Arthritis Damage Index (JADI) and adverse events (AE). We used path analysis to assess direct and indirect effects of variables on PGW. We repeated the analysis on subsets of visits stratified by disease activity, measured with JADAS10, to test differences across activity states.

Results: Pain severity proved the strongest direct determinant of PGW (b 0.521, p<0.001), followed by Psychosocial (b 0.191, p<0.001) and Physical Health (b 0.158, p<0.001). Stiffness (f2 0.047, p<0.001) and joint damage by the JADI (f2 0.025, p<0.001) were also indirectly associated with PGW, through their effects on function and PhHS. Patient-reported adverse events (AE) had a small effect on PGW (f2 0.025, p<0.001) through their impact on PsHS. Relations among variables and relative coefficients are depicted in the graph. Multi-group comparison between Remission, Low (LDA), Moderate (MDA) and High Disease Activity (HDA) groups revealed that for subjects in Remission, pain had a lower impact on function and physical health compared to those with LDA, while remaining the strongest predictor of PGW. Among the HDA group, the direct effect of pain on well-being and the impact of physical health on psychosocial health was greater than in other activity states (table). The effect of AE remained significant across all activity categories.

Abstract THU0515 – Figure 1.

	Path Coefficients (b)				Comparison			
	REM n=2262	LDA n=665	MDA n=1351	HDA n=186	REM/ LDA	HDA/ REM	HDA/ LDA	HDA/ MDA
Pain ->	0.312	0.401	0.400	0.402	p	p	p	p
JAFS					0.042	0.131	0.488	0.001
Pain ->	0.292	0.377	0.391	0.409	p	p	p	p
PhHS					0.032	0.041	0.324	0.018
Pain ->	0.367	0.420	0.467	0.614	p	p	p	p
PGW					0.208	0.003	0.016	0.033
PhH ->	0.400	0.429	0.409	0.557	p	p	p	p
PsHS					0.652	0.002	0.018	0.003

Conclusion: Pain was the main determinant of PGW in all disease activity states. The level of PGW also reflected other aspects of disease impact, particularly physical and psychosocial distress, and, to a lesser extent, treatment adverse events. The impact of pain and physical functioning on psychosocial health and well-being varies with disease activity, being greater in patients with higher disease activity.

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- [1] Moorthy et al, *Pediatric Rheumatology* 2010; 8:20

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THU0516

LONG-TERM SAFETY OF SUBCUTANEOUS TOCILIZUMAB ADMINISTRATION IN SYSTEMIC AND POLYARTICULAR JUVENILE IDIOPATHIC ARTHRITIS

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Background: Tocilizumab (TCZ) administered intravenously (IV) was effective for the treatment of polyarticular (p)JIA and systemic (s)JIA.^{1,2}

Objectives: To evaluate the long-term safety and efficacy of subcutaneous (SC) TCZ in patients (pts) with pJIA or sJIA enrolled in a long-term extension (LTE) phase of two 52-week, open-label studies.

Methods: Pts aged 1-17 years received body weight (BW)-based TCZ SC: pJIA pts who failed/could not tolerate MTX received TCZ 162 mg every 3 weeks for BW <30 kg or every 2 weeks (Q2W) for BW ≥30 kg; sJIA pts with inadequate response to NSAIDs and glucocorticoids received TCZ 162 mg Q2W for BW <30 kg or weekly for BW ≥30 kg. All pts discontinued biologic DMARDs (approximately 50% switched from TCZ IV to TCZ SC). After 52 weeks, pts continued BW-based TCZ in a separate LTE study; safety data (adverse events [AEs], serious AEs [SAEs]) in the LTE study to clinical cutoffs December 1, 2017 (pJIA), and February 28, 2018 (sJIA), are reported for all pts who received ≥1 dose of TCZ SC and had ≥1 postdose safety assessment.

Results: Most pJIA (n=44) and sJIA (n=38) pts were female (72.7% and 55.3%) and white (88.6% and 84.2%); median (range) age was 9.0 (2-18) years. AE rates (Table) were similar regardless of BW. Most AEs were grade 1 or 2; grade ≥3 AEs were reported by 10/44 (20.8%) pJIA pts and 4/38 (10.5%) sJIA pts, most commonly nasopharyngitis (pJIA, 17/44 [38.6%]; sJIA, 11/38 [28.9%]). Other AEs reported in ≥15% of pts included arthralgia, gastroenteritis, cough, vomiting, diarrhea, pyrexia, headache, and oropharyngeal pain (pJIA) and upper respiratory tract infection, cough, pyrexia, arthralgia, and rash (sJIA). No opportunistic infections developed. Neutropenia AEs were reported by 6/44 (13.6%) pJIA pts and 7/38 (18.4%) sJIA pts. SAEs occurred in 5/44 (11.4%) pJIA pts (furuncle, appendicitis, pneumonia, eye pain/headache, infectious mononucleosis) and 2/38 (5.3%) sJIA pts (pneumonia, craniocerebral injury from a fall); only pneumonia (pJIA) was considered treatment related. Neutralizing anti-TCZ antibodies developed in 2 (4.7%) pJIA pts and no sJIA patients. No deaths were reported in the LTE study.

Conclusion: In this LTE study in children with pJIA or sJIA, SC TCZ continues to have an acceptable tolerability profile with no new safety concerns.

Abstract THU0516 –Table 1.

Table 1. Safety Summary	pJIA		sJIA	
	TCZ SC Q2W (n=44) n (%)	TCZ SC Q2W (n=44) n (%)	TCZ SC Q2W or Q2W (n=44) n (%)	TCZ SC Q2W (n=44) n (%)
Adverse Events				
AEs	24 (54.5)	24 (59.1)	17 (38.6)	17 (45.0)
SAEs	10 (22.7)	4 (9.1)	5 (11.4)	5 (12.5)
Deaths	0	0	0	0
Discontinuation due to AEs	1 (2.3)	1 (2.3)	1 (2.3)	1 (2.3)
Discontinuation due to other reasons	1 (2.3)	1 (2.3)	1 (2.3)	1 (2.3)
Discontinuation due to unknown reasons	0	0	0	0
Discontinuation due to unknown reasons	0	0	0	0

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