SAT0381

NEUTROPHIL-RELATED BIOMARKERS DISCRIMINATE SEPTIC ARTHRITIS FROM OTHER INFLAMMATORY ARTHRITIDES: DATA FROM THE BOSSA TRIAL

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Background: Septic arthritis is a life threatening purulent invasion of a joint by an infectious agent which produces arthritis. If untreated, septic arthritis causes structural damage to the joint. Unfortunately no relevant biomarkers are available for the diagnosis of this disabling condition. We aimed at determining whether calprotectin (S100A8/A9) and Human neutrophil alpha-defensins (HNP1-3) could discriminate septic from other inflammatory arthritides

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Methods: Patients joint effusions for which septic arthritis was suspected were prospectively collected in Grenoble Hospital. Patients with inflammatory synovial fluid (*i.e.* with white blood cell >2000/mm³ and >80% polymorphonuclear neutrophils (PMN)) were included in this trial. Diagnosis of septic arthritis was retained if bacteria were cultured from inflammatory synovial fluid and/or blood samples. Diagnosis of pseudo gout was retained when pyrophosphate calcium crystals were observed in inflammatory synovial fluid. Diagnosis of rheumatoid arthritis was retained according to rheumatologist opinion.

C Reactive protein (CRP), both neutrophil-related proteins calprotectin and human neutrophil alpha-defensins (HNP1-3) levels were assessed in synovial fluids.

Threshold for biomarkers were determined by ROC curve analysis. Sensibility, Specificity, Positive (PPV) and Negative (NPV) Predictive Values at a pre specified threshold were calculated. Biomarkers with $p \leq 0.05$ were included into a multivariate model. Multivariate logistic regression with stepwise selection was performed to build the final combined model.

Results: A total of 74 patients were included: septic arthritis (n=26), pseudo gout (n=28) and Rheumatoid arthritis (n=20). Patients with septic arthritis group were more likely to be male (69% vs. 31%, $p=0.030$), were younger (median age (range) 65.5 (27–84) vs. 72.5 (33–91), $p=0.047$), displayed higher synovial fluid PMN count (9,600 (1,800–68,000) vs. 6560 (750–22,500, $p=0.047$), LR+12.2 and LR- 0.26.

Calprotectin was significantly increased in patients with septic arthritis (190 (0.1–247) vs. 62 (0–208) mg/L, $p<0.001$, figure 1) whereas only a trend of an HNP1-3 rise was shown in arthritis septic (6.8 (0.5–56.6) vs. 3.4 (0.1–6.4) mg/L, $p=0.050$). Optimal Thresholds for calprotectin, HNP1-3, and synovial fluid PMN were respectively 150 mg/L, 6.5 mg/L and 7000/mm³.

In the multivariate model, including calprotectin, HNP1-3, synovial fluid PMN count and gender, calprotectin was the only biomarker discriminating septic arthritis from non-septic inflammatory arthritides with 76% sensitivity, 94% specificity, 86% PPV and 88% NPV.

Conclusions: Our data show here that synovial fluid calprotectin is a relevant biomarker to discriminate septic arthritis from other inflammatory arthritides. This biomarker should be tested in an independent cohort.

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SAT0382

UTILITY OF NEUTROPHIL CD64 & SERUM TREM-1 IN DISTINGUISHING BACTERIAL INFECTION FROM DISEASE FLARE IN SLE AND ANCA ASSOCIATED VASCULITIS

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Background: Treating physician is challenged to differentiate between disease activity vs. infection as to the cause of fever in patients with SLE and ANCA associated vasculitis (AAV). Presently, there is no biomarker to reliably differentiate between them.

Objectives: We aimed to determine the usefulness of CD64 (FcγR1) expression on neutrophils and sTREM-1 (soluble triggering receptor expressed on myeloid cells) in serum in distinguishing bacterial infection from disease flare in SLE and AAV.

Methods: 20 healthy controls and patients of SLE and AAV admitted to our unit either with disease flare or bacterial infection were recruited over a period of 1 year. Neutrophil CD64 expression was measured by flow cytometry and sTREM by ELISA from blood samples collected on the day of admission.

Results: Among the 76 patients included in the study 51 (SLE-35 and AAV-16) had disease flare while 25 (SLE-21 and AAV-4) had infection. 72% had fever at presentation. The percentage of neutrophils with CD64 expression and their mean fluorescence intensity in patients with infection were significantly ($p<0.05$) higher as compared to those without infection and controls (Table-1). The sensitivity and specificity of CD64 expression on neutrophils to diagnose bacterial infection (using a cut off value of 30%) was 85% and 84%, respectively. Whereas, the sensitivity and specificity of procalcitonin was 75% and 85% respectively. There was no significant difference in soluble TREM-1 levels between the two groups. Total leucocyte count (TLC), ESR, CRP, C3, C4 levels were not significantly different between disease flare and infection group. On subgroup analysis (Table-2) patients of SLE with infection had a higher CRP, TLC and Procalcitonin compared to those with disease flare ($p<0.05$). ROC curves are depicted in figure 1.

Table-1 Comparison of various parameters between healthy controls, patients with active disease and infection

Characteristic	Active disease (n=51)	Infection (n=25)	Healthy controls (n=20)
CD64 expression on neutrophils (%)	7.7 (2.6–13)	68.8 (56.9–86.5)*	7.05 (1.4–9.5)
Mean fluorescence intensity of CD64	456 (20–986)	1037 (229–1828)*	99.5 (54.7–140.7)
sTREM-1 (pg/ml)	1184 (717–1609)**	899 (531–1284)**	255.1 (95.1–634.8)
Procalcitonin (pg/ml)	157.7 (115.1–209.3)**	301 (141.8–339.3)*	77.6 (61–101.8)
Total leucocyte count (cells/mm ³)	9700 (4600–12400)	10750 (6000–15000)	-
CRP (mg/L)	1.9 (0.86–6.05)	5.4 (1.83–8.9)	-
ESR (mm/hr)	70 (30–103)	75 (55–110)	-

* $p<0.05$ vs. active disease group and healthy controls

** $p<0.05$ vs. healthy controls

All values expressed as median (25th–75th Interquartile range)

Abstract SAT0382 – Table 2. Subgroup analysis of patients with SLE and ANCA associated vasculitis

Variable	SLE with active disease (n=35)	SLE with Infection (n=21)	AAV with active disease (n=16)	AAV with infection (n=4)
CRP (mg/dl)	1.3 (0.39–2.3)	5.54 (1.8–9.2)*	7.3 (2.64–10.5)	11.7 (2.08–20.85)
TLC (cells/mm ³)	6400(3400–9500)	11 000(5925–16650)*	12 200(10550–20400)	12 000(10,300–20100)
nCD64 (%)	9.42 (3–13.7)	69.4 (59–87.1)*	4.25 (2.27–8.5)	76 (42.5–80.9)
Procalcitonin (pg/ml)	174 (101–204)	316 (231–345)*	172 (127–257)	104 (86–356)

* $p<0.05$ vs. SLE with disease flare

All values expressed as median (25th–75th Interquartile range)

Abstract SAT0382 – Figure 1. ROC analysis of various parameters in patients with underlying SLE

Conclusions: CD64 expression on neutrophils is helpful in differentiating bacterial infection from disease flare in patients with SLE and AAV.

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SAT0383 RHEUMATOLOGICAL DISORDERS FOLLOWING CHIKUNGUNYA INFECTION – A RETROSPECTIVE STUDY OF 882 CASES FROM NEW DELHI

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Background: Chikungunya virus (CHIKV) is notoriously arthritogenic and is known to cause a wide spectrum of rheumatological disorders.

Objectives: To study the Rheumatological Manifestations of CHIKV Infection.

Methods: Record of 1641 consecutive patients who presented to the outpatient department (OPD) with history of chikungunya infection (fever, joint pains±rash) during the period of 1 st July 2016 to 31 st October 2016 were checked and recruited from private hospital, camps and clinic from urban and suburban population from 1 st February to 31 st December 2017. The study cohort was subjected to laboratory tests – serum IgM, IgG and RT-PCR for chikungunya virus. Those patients who tested positive for ≥1 lab tests were included in the study. Of the 1641 patients, 408 patients either refused the lab tests or were lost to follow up. Of the remaining 1233 patients, 882 (71.53%) patients tested positive for ≥1 lab tests and 351 (28.47%) tested negative for all the 3 lab tests. These patients were classified into rheumatological disorders as per validated criteria.

Results: We studied 882 consecutive patients of chikungunya infection with some rheumatological manifestations. Mean age of study cohort was 53.64 ±15.82 years with male:female of 398 (45.12%):484(54.88%). Mean disease duration of post CHIKV infection was 08.45±3.64 months. Serologically, isolated serum IgM CHIKV was positive in 128 (14.51%) patients, IgG CHIKV in 340 (38.54%) patients and RT-PCR CHIKV in 223 (25.28%) patients. Combination of serum IgG and IgM CHIKV both were positive in 62 (7.03%), IgM and RT-PCR CHIKV 49 (5.55%) and IgG and RT-PCR CHIKV in 40 (4.54%) patients. All the 3 serum IgM, IgG and RT-PCR CHIKV were positive in 50 (5.67%) patients.

Of these 882 patients, 143 (16.21%) patients had rheumatological disorder prior to CHIKV infection which flared up after the infection. Of the 143 patients, 69 (48.25%) had osteoarthritis (OA) knee, 29 (20.27%) had mechanical low back ache, 12 (8.39%) cervical spondylitis, 10 (6.99%) had rheumatoid arthritis (RA), 7 (4.89%) each adhesive capsulitis and ankylosing spondylitis (AS), 3 (2.1%) psoriatic arthritis (PsA), 2 (1.4%) each had SLE, dequervian tenosynovitis and granulomatous with polyangitis.

Remaining 739 patients presented with various rheumatological disorders, most common of which was post chikungunya polyarthralgias in 568 (76.86%) patients. Of the 739 patients, 40 (5.4%) patients developed new onset OA knee, 38 (5.14%) met ACR/EULAR 2010 criteria for RA, 28 (3.78%) met modified New York criteria for AS, 29 (3.92%) developed undifferentiated arthritis, 17 (2.3%) patients developed various enthesitis and tenosynovitis, 1 (0.14%) patients fulfilled ACR 1997/SLICC 2012 criteria for SLE, 20 (2.7%) patients fulfilled ACR 2010 criteria for fibromyalgia, 5 (0.67%) patients fulfilled CASPAR criteria for PsA, 3 (0.4%) patients fulfilled 2015 classification criteria for Gout.

Family history of RA was present in 4 (10.52%) patients and AS in 9 (32.1%) patients. Co-morbidities as hypothyroid were present in 132 (17.86%) patients, hypertension in 88 (11.90%) and diabetes mellitus in 168 (22.73%) patients.

Limitation of study – This study was not community based and only those patients who had persistent symptoms were recruited. Those patients whose symptoms of CHIKV infection subsided were not included.

Conclusions: CHIKV is a worldwide epidemic which precipitates chronic inflammatory rheumatological diseases.

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SAT0384 EVOLVING PATTERNS OF REACTIVE ARTHRITIS

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Background: Reactive arthritis (ReA) seen by rheumatologists may be changing in frequency (less common) and severity (less than full triad of symptoms and less chronic ReA). Epidemiologic changes in ReA could be due to less food borne illness, cleaner water, and possibly more rapid treatment of sexually transmitted diseases or for other unknown reasons.

Objectives: To understand rheumatologists' perspectives about changes in frequency, severity, and manifestations of ReA.

Methods: After obtaining ethics approval, 548 members of the Canadian Rheumatology Association (CRA) were surveyed via email with a reminder email. There were 6 groups of questions: demographic information, views from respondents regarding the prevalence of ReA (including acute, recurrent, and chronic), tests ordered to investigate suspected ReA, treatments prescribed for ReA, causes of ReA in their practices, and perspectives on changes in the incidence, severity and causes of ReA over time. Descriptive statistics were used to analyse the data. Results were by physician report and were not confirmed by chart audits.

Results: Sixty-six rheumatologists completed the survey (15.5% response rate). The results of the survey indicated that 47% of rheumatologists believed that the incidence of ReA is declining, compared to 6% who thought it was increasing; and that the common causes may be changing (39% agreed/strongly agreed with a mean 3.4/5 on the Likert scale).

Acute, chronic, and recurrent ReA were all perceived to have similar frequencies in their practices. In terms of presentation, asymmetric oligoarthritis occurred in the majority of ReA seen by those surveyed (78%). Full triad ReA (arthritis, conjunctivitis, urethritis) was thought to occur in 21% of ReA cases, and patients with conjunctivitis were very likely to exhibit the rest of the triad. Similarly, patients with recurrent ReA were more likely to exhibit the full triad (43%) compared to acute or chronic ReA (14%). Rheumatologists believed that the infectious cause of ReA was found in only 35% of cases. The data indicate that the most common cause of ReA was 'unknown' infectious organisms, followed by gastrointestinal (GI) infections and sexually transmitted infections (STIs).

Multiple tests were ordered to investigate ReA. The three most common investigations ordered by respondents included testing for chlamydia (66%), C-reactive protein (CRP) (62%), and human leukocyte antigen (HLA-B27) (50%). Imaging was ordered by 39% of respondents with sacroiliac (SI) joint imaging ordered by 21%, X-rays of the affected joints by 15%, and other imaging by 7.5%. Figure 1 shows these results.

Treatments used for ReA varied, as shown in figure 2. The most common treatments for ReA were nonsteroidal anti-inflammatory drugs (NSAIDs) (97% frequently or always used), intra-articular corticosteroid injections (65% frequently or always used), and disease-modifying antirheumatic drugs (DMARDs) (45% frequently or always used). Sixty-six percent said they used tumour necrosis factor alpha inhibitors (TNFi) at least occasionally in chronic ReA.