

IFN signature ($r=0.66$, $p<0.001$ and $r=0.46$, $p<0.001$ respectively). ROC-curve analysis revealed a better performance of galectin-9 (AUC 0.86) than CXCL10 (AUC 0.78) or traditional serological biomarkers for SLE (AUC <0.75) to detect an IFN signature. The expression of galectin-9 was increased in both pDC and mDC in SLE and APS, in particular in IFN-high patients. *In vitro*, IFN α upregulated galectin-9 expression in pDC and mDC.

Conclusion Galectin-9 is produced by dendritic cells in SLE and APS upon activation by IFN α and serves as an easily measurable biomarker that outclasses CXCL10 or traditional measures of disease activity to detect an IFN signature in patients with SLE and APS.

S5A:6 ANTI-CARBAMYLATED PROTEINS ANTIBODIES IN SLE PATIENTS WITH JOINT INVOLVEMENT: A POSSIBLE NEW BIOMARKER FOR EROSIIVE DAMAGE

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Purpose The concept of non-erosive arthritis in Systemic Lupus Erythematosus (SLE) changed during the last years, thanks to more sensitive imaging techniques, such as ultrasonography (US), allowing the identification of erosive damage in up to 47% of patients. The predictive role of Rheumatoid Arthritis (RA)-specific auto antibodies has been investigated. In particular, anti-citrullinated peptide antibodies (ACPA) have been identified in about 50% of SLE patients with x-Ray detected erosive arthritis. More recently, anti-carbamylated proteins antibodies (anti-CarP) have been demonstrated in seronegative RA, with a significant association with erosive damage. In the present cross-sectional study, we assessed the association between anti-CarP and erosive damage in a cohort of SLE patients with joint involvement.

Methods We evaluated 152 SLE patients (1997 ACR criteria; M/F 11/141, mean \pm SD age 46.4 ± 11.3 years, mean \pm SD disease duration 144.9 ± 110.5 months) with joint involvement. Clinical and laboratory data were collected in a

standardised computerised electronically filled form. All patients underwent blood draws to detect Rheumatoid Factor (RF) and ACPA, by using commercial ELISA kits, and anti-CarP by home-made ELISA (results were expressed in arbitrary units (AU)/ml and values above 340 IU/ml were considered positive). US was performed to assess the bone surfaces of metacarpophalangeal and proximal interphalangeal. At each joint, according with OMERACT definition, the presence of erosions was registered with a dichotomous value (0/1), obtaining a total score, ranging from 0 to 20.

Results The anti-CarP prevalence was 28.3%, similar to RF (27.6%) and significantly higher to ACPA (11.2%, $p=0.003$). The mean \pm SD titer of anti-CarP was 890.5 ± 794.9 IU/ml. Thirty-nine patients (25.6%) showed an US-detected erosive arthritis: all the patients referred at least one episode of clinical synovitis. Erosive arthritis was associated with anti-CarP ($p=0.004$) and ACPA ($p=0.0008$). A correlation between anti-CarP titer and US-erosive score was observed ($r=0.2$, $p=0.01$). Of note, anti-CarP were identified in 24.5% of double negative (ACPA-/RF-) patients, with erosive damage in 25% of them.

Conclusions We identified a significant association between anti-CarP and US-detected erosive damage in SLE-related arthritis, in terms of frequency and severity. Our results suggest that anti-CarP could be considered as a candidate biomarker of severity in SLE patients with joint involvement.

S5d – Supportive therapies

S5D:4 LOW VITAMIN D IS ASSOCIATED WITH THROMBOSIS IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Background/purpose Low vitamin D is common in systemic lupus erythematosus (SLE). It is also found in antiphospholipid syndrome. Vitamin D has effects on tissue factor, PAI-1, thrombomodulin and platelet aggregation that suggest it has an anti-thrombotic role. We asked whether low vitamin D was associated with thrombosis in SLE, adjusting for lupus anticoagulant.

Abstract S5D:4 Table 1 Associations of first vitamin D measurement with thrombosis

| | Positive for Thrombotic Event | | No Thrombotic Event | | P-value |
|-----------------------------------|-------------------------------|-----------|---------------------|-----------|---------|
| | Mean (SD) | N (%) | Mean (SD) | N (%) | |
| Any Thrombotic Event | | | | | |
| Vitamin D (ng/ml) (Mean/SD) | 27.6(15.1) | | 30.6(14.6) | | 0.0008 |
| Vitamin D < 40 ng/ml (N/ %) | | 299(80.4) | | 759(75.4) | 0.064 |
| Stroke | | | | | |
| Vitamin D (ng/ml) (Mean/SD) | 28.9(15.2) | | 29.9(14.7) | | 0.5408 |
| Vitamin D < 40 ng/ml (N/ %) | | 79(75.2) | | 988(76.9) | 0.7914 |
| Myocardial Infarction (MI) | | | | | |
| | Mean (SD) | N (%) | Mean (SD) | N (%) | |
| Vitamin D (ng/ml) (Mean/SD) | 30.2(16.9) | | 29.8(14.7) | | 0.883 |
| Vitamin D < 40 ng/ml (N/ %) | | 35(70) | | 1032(77) | 0.3258 |
| DVT | | | | | |
| | Mean (SD) | N (%) | Mean (SD) | N (%) | |
| Vitamin D (ng/ml) (Mean/SD) | 25.9(13.4) | | 30.4(14.9) | | <0.0001 |
| Vitamin D < 40 ng/ml (N/ %) | | 171(87.2) | | 895(75) | 0.0002 |

We next adjusted for race, age, sex and lupus anticoagulant. Low vitamin D remained associated with DVT