

and insulin resistance. Adiponectin, on the contrary, was independent of SLE activity, but had a favorable, although weak, effect on blood pressure and lipid profile.

PO.3.66 TLR9 PROTEIN LEVEL IS ASSOCIATED TO PROINFLAMMATORY CYTOKINE LEVEL OF IL10 AND INF1A IN SLE PATIENTS

¹E Grau García*, ²L Gomez-Lechón Quirós, ¹C Riesco Barcena, ¹AV Huaylla Quispe, ¹S Leal Rodríguez, ¹C Páveles Peralas, ¹L Mas Sanchez, ¹P Muñoz Martínez, ¹M De La Rubia Navarro, ¹I Cánovas Olmos, ¹J Ivorra Cortés, ¹JJ Frago Gil, ¹L González Puig, ¹I Martínez Cordellat, ¹C Nájera Herranz, ¹R Negueroles Albuixech, ¹JE Oller Rodríguez, ¹FM Ortiz-Sanjuán, ¹E Vicens Bernabéu, ¹JA Roman Ivorra. ¹Rheumatology Department. HUP La Fe. ~ Valencia ~ Spain; ²Rheumatology Department. Hospital Francesc de Borja ~ Gandia ~ Spain

10.1136/lupus-2022-elm2022.95

Purpose The aim is to investigate the association among TLR7 and TLR9 serum levels with previous viral infections, disease activity and proinflammatory cytokine levels in SLE patients.

Methods Cross-sectional observational study in SLE patients (SLICC/ACR 2012 criteria) and healthy controls (HC). Previous infection data with RNA (HCV) and DNA virus (CMV, Epstein-Barr, Herpes simplex, Parvovirus B19 or HBV), disease activity and clinical data were collected. Biological samples of SLE patients and HC from the medical visit were supplied to the TLR7, TLR9, IL10 and INF1A determination by enzyme-linked immunoassay.

Results 94 SLE patients (91.5% female) with a mean age of 51 (13) years old and 35 HC (80% female) and 42 (12) years old were recruited. Mean age at diagnosis was 33 (14) years old and mean disease evolution was 19 (10) years. Mean SLE-DAI index was 5.35 (4.58).

The 48.94% of patients reported almost one DNA virus infections, the 2.13% reported HCV infection, the 4.25% with HCV and DNA virus, and the 31.92% not reported any infection. HC had no history of acute (3 months) or lasting chronic infections with viruses or bacteria.

TLR7 and TLR9 did not correlate between them. TLR9 levels were significantly higher in SLE patients than HC ($P < 0.001$). Even though TLR7 levels did not show any difference between both groups, an association with the age of individuals was observed ($P < 0.001$).

No association among TLR7 or TLR9 levels with CRP, ESR, anti-dsDNA, ENAs or antiphospholipid antibodies was observed, and nor with disease activity, age at diagnosis and disease evolution time. In contrast, however, we reported low TLR7 levels in SLE patients and antiphospholipid syndrome in comparison to those without antiphospholipid syndrome ($P = 0.001$).

High TLR9 levels were significantly associated to increased levels of IL10 and INF1A in SLE patients ($P < 0.001$). TLR7 levels were not associated with INF1A levels but it is noticeable that there is a tendency to increase TLR7 levels in cases with increase of IL10 levels.

Conclusions TLR9 is increased in SLE patients in comparison to HC. TLR7 increases with age. No evidence of association between previous infections and TLR levels was found. Nor do we observe any difference in TLR level according to auto-antibodies presence or disease activity, probably due to the long-term SLE evolution and a good control of the disease.

There was, however, an association between high TLR9 levels and increase of IL10 and INF1A.

PO.3.67 MEASURING IFNA2 LEVELS BY A SINGLE-MOLECULE ARRAY IN CLINICAL PRACTICE OF CHILDHOOD-ONSET SLE PATIENTS DOES MATTER; RESULTS FROM A SINGLE CENTER LONGITUDINAL STUDY

Marleen Verkaaik, Wahadat M Javad*, Hongchao Qi, Cornelia G van Helden-Meeuwssen, Erika Huijser, Lotte van den Berg, Jens Göpfert, Marleen Verkaaik, Marco Schreurs, Sylvia Kamphuis, Marjan A Versnel. Dept. Immunology, Erasmus University Medical Center, Rotterdam, The Netherlands, Dept. Paediatric Rheumatology, Sophia Children's hospital, Erasmus University Medical Center, Rotterdam, The Netherlands, Department of Biostatistics, Erasmus University Medical Center, Rotterdam, The Netherlands, NMI Natural and Medical Sciences Institute at the University of Tübingen, Dep. of Applied, Biomarkers and Immunoassays, Reutlingen, Germany

10.1136/lupus-2022-elm2022.96

Introduction Type-I interferon (IFN-I) pathway activation plays a pivotal role in the pathogenesis of SLE and has been proposed as biomarker for disease activity. IFN-I pathway activation can be measured by determining the expression of IFN-I stimulated genes or a so-called IFN signature. Ultrasensitive single-molecule array (Simoa) technology enables measurement of IFN protein concentrations at subfemtomolar concentrations. Parallel use of these measuring methods in longitudinal cohorts of childhood-onset SLE (cSLE) patients in relation to disease activity could help in translating the most relevant technique for use in clinical practice.

Objective To determine the association of serum IFN α 2 levels and whole blood IFN-I stimulated gene expression with disease activity and study their potential to mark specific disease activity states in a longitudinal cohort of cSLE patients.

Methods Serum IFN α 2 levels were measured in 338 samples from 48 cSLE patients and 67 healthy controls using an IFN α 2 Simoa assay (Quanterix) on an HD-X analyser. A 5 gene IFN-I signature was measured by RT-PCR in paired whole blood samples. Disease activity was assessed by the clinical SELENA-SLEDAI (cSLEDAI) and BILAG-2004. Low disease activity was defined by the Low Lupus Disease Activity State (LLDAS) and flares were characterized by the SELENA-SLEDAI flare index. Analysis was performed using linear mixed effect models.

Results A clear positive correlation was present between serum IFN α 2 levels and the IFN-I gene signature ($r = 0.78$, $p < 0.0001$). Serum IFN α 2 levels and the IFN-I gene signature showed the same significant negative trend in the first three years after diagnosis. In this timeframe, mean baseline serum IFN α 2 levels decreased with 55.1% (delta 172 fg/mL, $p < 0.001$) to a mean value of 164 fg/mL, which was below the calculated threshold of 219.4 fg/mL. In the linear mixed model, serum IFN α 2 levels were significantly associated with both the cSLEDAI and the BILAG-2004 ($p < 0.001$ and $p < 0.01$), while the IFN-I gene signature did not show this association ($p = 0.35$ and $p = 0.23$). Moreover, 69.7% of the time points in LLDAS had a serum IFN α 2 level under the calculated threshold, while only 31.9% of the time points in LLDAS reached an IFN-I gene signature below the calculated threshold. Both techniques were equally capable of marking disease flares (79.2% above threshold vs 87.5% above threshold).

Conclusions Serum IFN α 2 levels measured by Simoa, but not the type-I IFN gene signature, are associated with disease activity scores and characterize disease activity states in cSLE patients. Hence, this technique has the potential to be implemented in clinical practice.

PO.3.68 SYSTEMIC LUPUS ERYTHEMATOSUS-RELATED INTESTINAL PSEUDO-OBSTRUCTION TREATED WITH IMMUNOSUPPRESSANTS

L Mustafayeva, I Durucan, AY Ayla, S Ugurlu*. *Istanbul University-Cerrahpasa, Cerrahpasa Medical Faculty, Division of Rheumatology, Department of Internal Medicine ~ Istanbul ~ Turkey*

10.1136/lupus-2022-elm2022.97

Purpose It is known that morbidity and mortality risks are higher in systemic lupus erythematosus-related (SLE) intestinal pseudo-obstruction (IPO) cases. Therefore, it is crucial to recognize and treat these cases promptly. Here, we present a case of SLE-related IPO in a 41-year-old female patient.

Methods Laboratory tests and radiodiagnostic methods including magnetic resonance imaging (MRI), computed tomography (CT), and plain radiography were utilized for diagnosis.

Results The patient who had polyarthritis, malar rash, and photosensitivity first presented to our clinic six years ago. She had a normal capillary pattern in nail fold capillaroscopy and no sclerodactyly. She also had ANA positivity and was diagnosed with SLE. The patient discontinued drug therapy and clinical follow-ups three years after the diagnosis. Then, she presented to our outpatient rheumatology clinic one month ago with nausea, abdominal pain, bloating, and bilateral lower extremity paresthesia. The patient was not able to walk without support, but muscle strength was normal in all extremities. There were no clinical signs of SLE activation except neurological complaints. ANA was positive at 1/640 dilution with nucleolar staining pattern. Complement levels were low. Anti-ds-DNA and anti-topoisomerase I were negative. CRP level was 25 mg/L. The rest of the lab parameters were normal. No pathological signs were detected in cranial MRI, cranial MR angiography, and spinal MRI. Cerebrospinal fluid had protein level 200 mg/dL, <math>\times 5</math> white blood cells.



Abstract PO.3.68 Figure 1

No viral pathogen was found. The patient was given pulse steroid. After three doses of 1g prednisolone, the patient stated she had constipation and was flatulent for ten days. Subsequently, air-fluid levels were seen in the erect abdominal radiography (Figure 1). Her complaints were resolved by nasogastric tube insertion. Ileus was confirmed by CT. IPO was the possible diagnosis for an SLE patient with this clinical presentation. 1g cyclophosphamide was administered after the seventh dose of corticosteroids. Extra three doses of corticosteroids were given because the patient was not improving clinically. Eventually, rituximab was given two weeks after cyclophosphamide and the patient recovered. Major complaints in SLE-related IPO cases are abdominal pain, bloating, nausea, constipation, and diarrhea. The small intestine is usually affected. Intestinal perforation is a rare but life-threatening complication.¹ Gastrointestinal symptoms are not specific to IPO, thus diagnosis may not be straightforward. The presence of abdominal symptoms at first evaluation, air-fluid levels in abdominal plain radiography, and absence of any prominent reason explaining the condition led us to diagnose IPO.

Conclusion Early diagnosis and appropriate timing to begin corticosteroid treatment play a crucial role to have better outcomes. We urge the clinicians to consider IPO in SLE patients who are admitted to the hospital with gastrointestinal symptoms.

REFERENCES

1. Wang JL, Liu G, Liu T, Wei JP. Intestinal pseudo-obstruction in systemic lupus erythematosus: a case report and review of the literature. *Medicine* (Baltimore). 2014 Dec;93(29):e248.

PO.3.69 A CASE OF LATE ONSET OF PRIMARY ANTIPHOSPHOLIPID SYNDROME

S Salvucci, L Manfredi*, L Gamba, G Montozzi, R Santangeli, G Moroncini. *Ospedali Riuniti di Ancona University Hospital ~ Ancona ~ Italy*

10.1136/lupus-2022-elm2022.98

Purpose Antiphospholipid syndrome (APS) usually affects young patients and is rarely described in the elderly. Here, we present a case of late-onset of triple-positive primary APS characterized by recurrent thrombotic events.

Methods In November 2019, an 80-year-old man presented the first deep vein thrombosis in his left leg. He received fondaparinux treatment for one year, but the thrombosis relapsed one month after stopping treatment. The search for thrombophilic causes has shown positivity for antiphospholipid antibodies (LAC, anticardiolipin, and anti-beta2 glycoprotein I IgM at high titres). In addition, the patient underwent first-level diagnostics and endoscopic examinations excluding other immunological causes, infections and malignancy. A diagnosis of primary APS was made and the warfarin therapy started with a range of INR between 2 and 3. In November 2021, the patient was brought to our attention following the recurrence of thrombosis of the deep iliac and femoral veins despite anticoagulant treatment. Laboratory tests confirmed the presence of triple high titre positivity of antiphospholipid antibodies and a total body CT scan confirmed the absence of suspected malignant lesions. Serological studies with a comprehensive autoimmunity panel for other autoimmune disorders were negative and an echocardiographic study ruled out the presence of cardiac involvement.