

**LP-162** **PROGNOSTIC FACTORS FOR THE CHRONIC THROMBOCYTOPENIA IN SYSTEMIC LUPUS ERYTHEMATOSUS**

<sup>1</sup>Soo Min Ahn\*, <sup>1,2</sup>Ji Seon Oh, <sup>1</sup>Yong-Gil Kim, <sup>1</sup>Chang-Keun Lee, <sup>1</sup>Bin Yoo, <sup>1</sup>Seokchan Hong, <sup>1</sup>Rheumatology, Asan Medical Center, University of Ulsan College of Medicine, Republic of Korea; <sup>2</sup>Information Medicine, Big Data Research Center, Asan Medical Center, Republic of Korea

10.1136/lupus-2023-KCR.243

**Background** Immune thrombocytopenia (ITP) is a common hematologic manifestation in systemic lupus erythematosus (SLE). Thrombocytopenia can persist for more than one year, which is defined as chronic thrombocytopenia. This study aimed to identify the clinical characteristics and risk factors for the chronic thrombocytopenia in SLE-ITP.

**Methods** We retrospectively reviewed patients who were diagnosed with SLE-ITP at a tertiary hospital between January 2000 and December 2021. The clinical and laboratory characteristics were analyzed according to the progression of chronic thrombocytopenia. Factors associated with chronic thrombocytopenia were evaluated by logistic regression analysis.

**Results** Of 121 SLE patients with ITP, 29 (24.0%) patients progressed to chronic thrombocytopenia lasting more than 1 year. The mean initial platelet count was lower in patients with chronic thrombocytopenia than those without (29.7 vs. 49.3 × 10<sup>9</sup>/L, P < 0.001). Multivariable analysis showed that body mass index (BMI) (adjusted odds ratio [aOR] = 1.194, 95% confidence interval [CI] = 1.014–1.406), severe thrombocytopenia (< 20 × 10<sup>9</sup>/L) (aOR = 3.974, 95% CI = 1.290–12.240), and recurrence of thrombocytopenia within 1 year (aOR=10.052, 95% CI=3.177–31.803) were significantly associated with the risk of chronic thrombocytopenia.

**Conclusions** Approximately one-quarter of the patients progressed to chronic thrombocytopenia in SLE. High BMI, severe thrombocytopenia, and recurrence of thrombocytopenia within 1 year were risk factors for the development of chronic thrombocytopenia in patients with SLE-ITP.

**LP-167** **UNCOMMON INITIAL PRESENTATIONS OF SLE – A CASE SERIES**

Madusha Jayasinghe\*, Duminda Abeysinghe. *Rheumatology, Rheumatology and Rehabilitation hospital, ragama, Sri Lanka*

10.1136/lupus-2023-KCR.244

**Description** A 29 year old patient presented with progressive dyspnea (mMRC grade 3) over 4 months. Her 2D Echocardiogram revealed moderate pulmonary hypertension with right ventricular dilatation. She had a history of inflammatory polyarthritis and constitutional symptoms. Her ANA was 1: 80 (Homogenous pattern). HRCT-Chest, CTPA, bubble contrast study and serology tests including APLS screening were all negative. A diagnosis of pulmonary arterial hypertension (PAH) was made. She also had an incidental detection of diffuse splenic calcification which is a rare association of SLE. She was treated with immunosuppressants and vasodilators for which she had a symptomatic improvement.

A 31 year old patient presented with progressive abdominal pain, distention and diarrhea for 2 weeks duration. She

underwent an exploratory laparotomy which revealed gross ascites and dilated small intestinal loops. Subsequent Abdominal CT showed small intestinal wall ischemia involving multiple arterial territories. She also reported on alopecia and gave a history of ITP during her pregnancy in 2016. With positive ANA (1:1280 titer), low complement levels, negative APLS screening and exclusion of other causes, SLE with mesenteric vasculitis was confirmed.

A 40 year old patient presented with a 2 week history of fever, nonproductive cough, inflammatory type polyarthritis and bilateral axillary lymphadenopathy. She later developed a pancytopenia and alopecia. Her ANA was 1:10000; Diagnosis of SLE was made. Axillary lymph node biopsy showed necrotizing lymphadenopathy and immunohistochemistry was strongly positive for CD68 favoring Kikuchi disease.

**Conclusions** Initial clinical presentations of SLE can be highly variable; can mimic infections and malignancies and may lead to delays in diagnosis. Therefore the clinicians should be vigilant on rare presentations of SLE such as PAH, mesenteric vasculitis and necrotizing lymphadenopathy to initiate timely treatments. Timely management in mesenteric vasculitis is crucial to prevent the possible catastrophic complications like necrotic bowel, perforation and sepsis.

**LP-168** **PREVALENCE AND ASSOCIATION OF AUTOANTIBODIES WITH LATENT TUBERCULOSIS IN FIRST-DEGREE RELATIVES OF PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS**

<sup>1</sup>Gayathri Ms\*, <sup>2</sup>Abilasha Narayanan, <sup>1</sup>Chengappa Kavadiachanda, <sup>1</sup>Molly Mary Thabah, <sup>2</sup>Sonali Sarkar, <sup>1</sup>Prakash Babu Narasimhan. <sup>1</sup>Clinical immunology, Jawaharlal Institute of Postgraduate Medical Education and Research, India; <sup>2</sup>Social and Preventive Medicine, Jawaharlal Institute of Postgraduate Medical Education and Research, India

10.1136/lupus-2023-KCR.245

**Background** Chronic and latent infections like tuberculosis (TB) are known triggers for developing autoimmunity. It is not known the consequences of latent TB infection (LTBI) in individuals who are genetically predisposed to develop autoimmune disease. Although several studies reported autoantibodies in first-degree relatives (FDR) of SLE, association between LTBI and these antibodies is not clear. This study focused on the prevalence of autoantibody levels in FDRs of SLE and assess any association with LTBI.

**Methods** This is a single center cross sectional study. FDRs of SLE who were apparently healthy and without past h/o TB were recruited (n=167). Demography, comorbidity and various autoantibodies (antibodies to beta-2 glycoprotein, cardiolipin, thyroid peroxidase, cyclic citrullinated peptide, glutamic acid decarboxylase, antinuclear antibody) were measured. LTBI was assessed using TB-IGRA (IFN-γ release assay). Based on results of TB-IGRA and seropositivity to antibodies, FDR were divided into 4 groups- Autoantibody positive and negative groups with and without LTBI. Basal IFN-γ and mycobacterium tuberculosis antigen specific IFN-γ levels were assessed in the unstimulated and stimulated tubes respectively.

**Results** In FDRs, overall prevalence of LTBI 25.7% (n=43) which is lesser than prevalence in the general population (40%). Prevalence of LTBI was numerically higher among FDRs positive for any autoantibody compared to the negative

group (32.6% vs 21.8%), but without statistical significance. Seropositivity of various autoantibodies was comparable between those with and without LTBI. Basal and stimulated IFN- $\gamma$  levels was lower in IGRA and autoantibodies positive group ( $p=0.014$ ) compared to IGRA positive antibody negative group.

**Conclusions** This study showed higher prevalence of LTBI in antibody positive FDRs of SLE. Basal and stimulated IFN- $\gamma$  levels were lower in antibody positive group, in contrast to SLE patients who have higher basal IFN- $\gamma$ . Further longitudinal studies would be required to see the effect of these autoantibodies on LTBI and risk of progression to TB.

**LP-170 PREDICTORS OF NEUROPSYCHIATRIC FLARES IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: RESULTS FROM FIVE PHASE III CLINICAL TRIALS OF BELIMUMAB**

<sup>1</sup>Leonardo Palazzo, <sup>1</sup>Julius Lindblom\*, <sup>1</sup>Nursen Cetrez, <sup>1</sup>Henri Ala, <sup>1,2</sup>Ioannis Parodis.  
<sup>1</sup>Division of Rheumatology, Department of Medicine Solna, Karolinska Institutet, Sweden;  
<sup>2</sup>Department of Rheumatology, Faculty of Medicine and Health, Örebro University, Sweden

10.1136/lupus-2023-KCR.246

**Background** Belimumab has shown ability to reduce flare rates in systemic lupus erythematosus (SLE), but little is known about its potential benefits in neuropsychiatric (NP)SLE. We investigated predictors of NP flares in SLE patients under standard therapy with or without add-on belimumab.

**Methods** Data from five clinical trials of belimumab in SLE (BLISS-52, BLISS-76, BLISS-NEA, BLISS-SC, EMBRACE) were utilised (N=3638). Flares were defined using the British Isles Lupus Assessment Group (BILAG) activity index. Predictors of flares were investigated throughout a 52-week follow-up using univariable and multivariable Cox regression. A subgroup analysis in patients with baseline NP BILAG E was performed to determine predictors of de novo NP flare. Organ damage was assessed using the SLICC/ACR Damage Index (SDI).

**Results** In total, 105 (2.9%) NP flares were documented. In multivariable analysis, male sex (HR: 2.37; 95% CI: 1.31–4.28;  $p=0.004$ ), baseline NP BILAG B-D (HR: 5.91; 95% CI: 3.86–9.06;  $p<0.001$ ), and high baseline SDI score (HR: 1.35; 95% CI: 1.21–1.50;  $p<0.001$ ) were highly associated with NP flare development. Belimumab use yielded no clear protection. In a separate analysis of SDI domains, the NP domain (HR: 3.25; 95% CI: 2.72–3.88;  $p<0.001$ ) was the strongest predictor of NP flare, with cognitive impairment (HR: 14.29; 95% CI: 9.22–22.14;  $p<0.001$ ), transverse myelitis (HR: 21.89; 95% CI: 5.40–88.72;  $p<0.001$ ), and neuropathy (HR: 8.87; 95% CI: 5.59–14.09;  $p<0.001$ ) mainly driving this association. In the subgroup analysis of the NP BILAG E population, male sex was the strongest predictor of de novo NP flare (HR: 3.26; 95% CI: 1.51–7.04;  $p=0.003$ ).

**Conclusions** Current or former NPSLE activity and established organ damage in the NP domain at baseline were the strongest predictors of NP flare. Whether belimumab treatment protects against NP events remains unclear.

**LP-171 FIBROMYALGIA AND GLUCOCORTICOIDS USE DRIVES SELF-PERCEIVED DEPRESSION IN SYSTEMIC LUPUS ERYTHEMATOSUS: INSIGHTS FROM A LARGE PROSPECTIVE AND MULTICENTER STUDY USING RELESSERPROS REGISTER'S DATABASE.**

<sup>1</sup>Iñigo Rúa-Figueroa, <sup>2</sup>Julia Martínez-Barrio, <sup>3</sup>Norman Jiménez, <sup>4</sup>María Galindo-Izquierdo, <sup>5</sup>Esther Uriarte-Isacelaya, <sup>6</sup>Antonio Fernandez-Nebro, <sup>7</sup>Jaime Calvo, <sup>6</sup>Sara Manrique-Ariza, <sup>8</sup>Jose Rosas, <sup>9</sup>Rocio Caño-Alameda, <sup>10</sup>J. Naváez, <sup>11</sup>Inmaculada Ros, <sup>12</sup>Elena Aurrecochea, <sup>13</sup>Vicenç Torrente-Segarra, <sup>14</sup>Clara Sanguesa, <sup>15</sup>Mercedes Freire-González, <sup>16</sup>Eva Tomero-Muriel, <sup>17</sup>Loreto Horcada, <sup>18</sup>Clara Moriano, <sup>19</sup>Mireia Moreno, <sup>20</sup>Carlotia Laura-Iñiguez, <sup>21</sup>Ricardo Blanco, <sup>22</sup>Ana Pérez-Gómez, <sup>23</sup>José Luis Andréu-Sánchez, <sup>24</sup>Sandra Garrote-Corral, <sup>25</sup>Santiago Muñoz-Fernández, <sup>26</sup>Gema Bonilla, <sup>27</sup>Nuria Lozano-Rivas, <sup>28</sup>Carlos A Montilla-Morales, <sup>29</sup>O Ibaranguoitia, <sup>30</sup>Lorena Expósito, <sup>31</sup>Elia Vals-Pascual, <sup>32</sup>Angela Pecondon-Español, <sup>33</sup>Sergio Machín, <sup>34</sup>Eva Salgado-Perez, <sup>1</sup>Maria Celia-Erausquin, <sup>35</sup>Tarek Carlos Salman-Monte, <sup>36</sup>Raúl Menor-Almagro, <sup>37</sup>Alejandro Muñoz-Jimenez, <sup>38</sup>Irene Altabás-González, <sup>39</sup>Jorge Juan Frago-Gil, <sup>38</sup>Jose María Pego-Reigosa\*.  
<sup>1</sup>Rheumatology, Hospital De Gran Canaria Doctor Negrin, Spain; <sup>2</sup>Rheumatology, Hospital Gregorio Marañón, Spain; <sup>3</sup>Rheumatology, Instituto De Investigación Sanitaria Galicia Sur, IRIDIS Group, Spain; <sup>4</sup>Rheumatology, University Hospital October 12, Spain; <sup>5</sup>Rheumatology, Donostia Unibertsitate Ospitalea, Spain; <sup>6</sup>Rheumatology, Hospital Regional Universitario de Málaga, Spain; <sup>7</sup>Rheumatology, Araba University Hospital, Spain; <sup>8</sup>Rheumatology, Hospital Marina Baixa, Spain; <sup>9</sup>Rheumatology, General University Hospital of Alicante, Spain; <sup>10</sup>Rheumatology, Bellvitge University Hospital, Spain; <sup>11</sup>Rheumatology, Son Llàtzer Hospital, Spain; <sup>12</sup>Rheumatology, Hospital Sierra Llana, Spain; <sup>13</sup>Rheumatology, Hospital de Sant Joan Despí Moisès Broggi, Spain; <sup>14</sup>Rheumatology, Germans Trias i Pujol Hospital, Spain; <sup>15</sup>Rheumatology, University Hospital of A Coruña, Spain; <sup>16</sup>Rheumatology, Hospital de La Princesa, Spain; <sup>17</sup>Rheumatology, Navarra Hospital Complex, Spain; <sup>18</sup>Rheumatology, University Hospital of León, Spain; <sup>19</sup>Rheumatology, Hospital Parc Taulí de Sabadell, Spain; <sup>20</sup>Rheumatology, Hospital Lugo, Spain; <sup>21</sup>Rheumatology, Marqués de Valdecilla University Hospital, Spain; <sup>22</sup>Rheumatology, Hospital Universitario Príncipe De Asturias, Spain; <sup>23</sup>Rheumatology, Puerta de Hierro Majadahonda University Hospital, Spain; <sup>24</sup>Rheumatology, Ramón y Caja Hospital, Spain; <sup>25</sup>Rheumatology, Infanta Sofia University Hospital, Spain; <sup>26</sup>Rheumatology, La Paz University Hospital, Spain; <sup>27</sup>Rheumatology, Hospital Clínico Universitario Virgen de la Arrixaca, Spain; <sup>28</sup>Rheumatology, Salamanca University Hospital, Spain; <sup>29</sup>Rheumatology, Basurto University Hospital, Spain; <sup>30</sup>Rheumatology, Hospital Universitario De Canarias, Spain; <sup>31</sup>Rheumatology, Doctor Peset University Hospital, Spain; <sup>32</sup>Rheumatology, Miguel Servet University Hospital, Spain; <sup>33</sup>Rheumatology, Hospital Materno Infantil De Gran Canaria, Spain; <sup>34</sup>Rheumatology, Complejo Hospitalario Universitario De Ourense, Spain; <sup>35</sup>Rheumatology, Hospital Del Mar, Spain; <sup>36</sup>Rheumatology, Hospital Universitario De Jerez, Spain; <sup>37</sup>Rheumatology, Virgen Del Rocío University Hospital, Spain; <sup>38</sup>Rheumatology, Complejo Hospitalario Universitario De Vigo, Spain; <sup>39</sup>Rheumatology, La Fe University and Polytechnic Hospital, Spain

10.1136/lupus-2023-KCR.247

**Background** Little is known about depression in SLE. To evaluate the prevalence of depression and associated factors in a large, multicenter SLE cohort (RELESSER-PROS).

**Methods** Prospective longitudinal study of SLE patients answering positively to the depression question of the Lupus Impact Tracker (LIT) questionnaire (question number 7, LITQ7 'I was depressed') over 5 consecutive annual visits (V1 to V5). Self-perceived depression was answered from 0 ('none of the time') to 4 ('most of time'). Covariates with potential impact in depression were considered. Friedman test and GEE models were used.

**Results** 1463 patients were included. Mean age 55 years, 90% female. Mean disease duration: 14 years. Fibromyalgia was present in 5.7%. Glucocorticoids use ranged from 49.4% to 57%, depending on the visit. SLEDAI ranged from 0 to 2 and SDI from 1 to 2. Prevalence of 'depression any time' was