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FRI0295 HIGH RISK OF IDIOPATHIC OSTEONECROSIS IN SLE PATIENTS WITH HIGH ANTIPHOSPHOLIPID SCORE AND HYPERTRIGLYCERIDEMIA

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Background: Systemic lupus erythematosus (SLE) patients are prone to develop idiopathic osteonecrosis (ION) compared to other connective tissue disease patients or healthy subjects. ION has been shown to occur as a result of ischemia, however, the involvement of antiphospholipid antibodies (aPL) in its pathophysiology remains to be elucidated. In the last years, our group introduced a quantitative marker of aPL “antiphospholipid score (aPL-S)”, which well reflected the risk of developing thrombosis [1].

Objectives: We aimed to identify the impact of aPL on the development of ION using aPL-S.

Methods: A single center retrospective study comprising 82 consecutive patients who were diagnosed SLE at the Rheumatology department of Hokkaido University Hospital and underwent magnetic resonance imaging (MRI) of hip joints from January 2000 to December 2016. Among all the enrolled patients, aPL-S, which is calculated from 0 to 86, as well as classical risk factors for ION were evaluated.

Results: All 82 patients (13 males and 69 females) were given glucocorticoids. ION of the femoral head was diagnosed by MRI scan in 37 patients. Male (ION(+): ION(-) 10/37 (27%) vs 3/45 (7%), p=0.016), malar rash (ION(+): ION(-) 22/37 (59%) vs 16/45 (36%), p=0.045), aPL positivity (ION(+): ION(-) 22/37 (59%) vs 15/45 (33%), p=0.026), high aPL-S (>30) (ION(+): ION(-) 9/37 (24%) vs 3/45 (7%), p=0.031), hypertriglyceridemia (fasting triglyceride levels >150 mg/dL) (ION(+): ION(-) 23/37 (62%) vs 12/45 (27%), p=0.002) and high dose of glucocorticoids (equivalent to 0.8mg/kg or more prednisolone) (ION(+): ION(-) 34/37 (92%) vs 21/45 (47%), p<0.001) were identified as risk factors for ION at univariate analysis. Multivariate analysis confirmed high aPL-S (OR 5.27; 95% CI 1.08 to 34.52, p=0.040), hypertriglyceridemia (OR 5.66; 95% CI 1.79 to 20.73, p=0.003) and high dose of glucocorticoids (OR 16.11; 95% CI 4.01 to 92.54, p<0.001) as independent variables. Of note, in 6 patients who had both high aPL-S and hypertriglyceridemia, 83% (5/6) developed ION (Figure 1). Conversely, systemic lupus erythematosus disease activity index and pulsed methylprednisolone therapy were not identified as risk factors for ION.

Conclusions: We newly identified high aPL-S as a risk factor for ION. Furthermore, SLE patients who have both high aPL-S and hypertriglyceridemia are at very high risk of ION. These findings suggest the involvement of microvascular occlusion in the pathophysiology of ION in SLE.

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FRI0296 SINGLE CENTER EXPERIENCE WITH 300 SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS

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Background: Systemic lupus erythematosus (SLE) is an autoimmune disease with diverse clinical manifestations.

Objectives: Here, we present 300 patients with SLE attending our clinic between 2001 and 2017.

Methods: Demographics, clinics, laboratory findings, Systemic Lupus Interna-

tional Clinics (SLICC)/American College of Rheumatology (ACR) damage index scores and treatments were analyzed. Diagnosis was confirmed with 1997 ACR (1) or 2012 SLICC (2) classifications. Descriptive statistical tests were used for analysis.

Results: Demographics and clinical characteristics are presented in Table 1. Anti-dsDNA was positive in 185 (61.6%), anti-Sm was positive in 35 (11.6%), anti-phospholipid antibodies was positive in 67 (22.3%), direct Coombs test was positive in 69 (23%) patients. Complement levels were low in 171 (57%) patients. In patients with renal disease, class IV lupus nephritis was the most common form (37 patients [12%]) followed by class II nephritis (34 patients [11%]). Forty-six patients had antiphospholipid syndrome. Treatments patients ever received are presented in Table 2. Cyclophosphamide treatment was given mostly for renal disease. Within 22 patients receiving rituximab, 2 had thrombocytopenia and 1 had hemolytic anemia unresponsive to other treatments, 1 had protein-losing enteropathy and 1 had lupus enteritis; the rest had lupus nephritis. One patient received intravenous immune globulin (IVIG) for severe neutropenia and 2 patients for severe thrombocytopenia. One patient received plasmapheresis for vasculitis. One patient received IVIG, plasmapheresis, rituximab for severe renal failure, alveolar hemorrhage requiring intensive care unit admission and 1 patient received same treatment for severe unresponsive hemolytic anemia.

Table 1. Demographics, clinical characteristics of patients

Age (years)	46±13 (min-max: 20–83; median: 45)
Duration of follow up (months)	58±52 (min-max: 1–180; median: 48)
Gender (Female/Male)	267 (89%)/33 (11%)
Muco-cutaneous (n, %)	214 (71.3%)
Arthritis (n, %)	198 (66%)
Renal disease (n, %)	121 (40.3%)
Leukopenia/lymphopenia (n, %)	200 (66.6%)
Hemolytic anemia (n, %)	19 (6.3%)
Thrombocytopenia (n, %)	58 (19.3%)
Serositis (n, %)	55 (18.3%)
Nervous system disease (n, %)	24 (8%)
Anti-phospholipid Syndrome (n, %)	46 (15.3%)

Table 2. Treatment

Steroid/pulse treatment (n, %)	294 (98%)/65 (21.6%)
Hydroxychloroquine (n, %)	300 (100%)
Azathioprine (n, %)	196 (65.3%)
Mycophenolate mofetil (n, %)	83 (27.6%)
Cyclophosphamide (iv) (n, %)	78 (26%); 10±4.5 cycles (min-max: 2–17)
Rituximab (n, %)	22 (7.3%)
Intravenous immunoglobulin (IVIG) (n, %)	7 (2.3%)
Plasmapheresis (n, %)	4 (1.3%)

Conclusions: In long term, SLICC/ACR damage index was highest in patients received pulse steroid for renal disease (min:0, max:5). Four (1.3%) patients had pulmonary hypertension, 31 (10.3%) had avascular necrosis, 24 (8%) had viral infections requiring treatment or bacterial infections requiring admission due to immunosuppression, 12 (4%) had malignancy. SLE is an autoimmune disease requiring multi-faceted approach.

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FRI0297 ROLE OF SERUM INTERLEUKIN-6 IN BLOOD BRAIN BARRIER DAMAGES IN NEUROPSYCHIATRIC SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: Neuropsychiatric manifestation in systemic lupus erythematosus (NPSLE) is one of the most serious complications of the disease. We have recently demonstrated that the breakdown of blood brain barrier (BBB) plays a crucial role in the development of diffuse psychiatric/neuropsychological manifestations (diffuse NPSLE), allowing influx of neuron-reactive autoantibodies from systemic circulation into the brain. However, the mechanism of BBB damages remains unclear. On the other hand, although CSF interleukin-6 (IL-6) has been shown to be elevated in NPSLE, there has been no report on serum IL-6 in NPSLE.

Objectives: The present study was designed in order to elucidate the roles of serum IL-6 in the pathogenesis, especially in development of BBB damages, in NPSLE.

Methods: Paired serum and cerebrospinal fluid (CSF) samples were obtained from 101 SLE patients when they presented active neuropsychiatric manifestations (69 patients with diffuse psychiatric/neuropsychological syndromes [diffuse NPSLE] and 32 patients with neurologic syndromes or peripheral nervous system involvement [focal NPSLE]) and from 22 non-SLE control patients with non-