positive correlation with ESR, dsDNA, C3, C4. Cytokines didn’t correlate with SLEDAI.

Conclusions IL6 and TNFɑ are reliable markers of disease activity, but lack significant clinical correlation.

### 235 HIERARCHICAL CLUSTER ANALYSIS OF SYSTEMIC LUPUS ERYTHEMATOSUS

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Background and aims Systemic lupus erythematosus (SLE) is a heterogeneous disorder with diverse manifestations and serologic features. The purpose is to categorise SLE patients into similar initial characteristics.

Methods Hierarchical cluster analysis approached to 389 SLE patients and 10 laboratory values. Laboratory values were transformed into Z-score for hierarchical clustering. Ward’s method as agglomeration method was a criterion applied with spearman correlation as distance metric. Clinical characteristics among clusters were examined by ANOVA with Tukey and Fisher’s exact test. To find each SLE cluster using initial laboratory, linear discriminant analysis was applied.

Results Three clusters were revealed by initial laboratory data; Cluster 1 had higher anti-dsDNA antibody, ANA titer and ESR, and low complements, lymphocyte, haemoglobin and platelet counts, Cluster 2 had lower anti-dsDNA antibody, ANA titer and ESR, and Cluster 3 had lower anti-dsDNA antibody titer, WBC and lymphocyte counts, and higher ANA titer. As a result from analysing cumulative manifestations and treatment, Cluster 1 showed more frequent malar rash, alopecia and renal disease with higher SLEDAI, and more use of cyclophosphamide and azathioprine. Also, oral ulcer was developed frequently in Cluster 2. During disease duration, total and mean corticosteroids and the number of flare were higher in Cluster 1.

Conclusions With initial laboratory values, SLE patients could be divided 3 clusters. Each Cluster showed different characteristics in clinical manifestations and treatment patterns. This predictive model considered disease severity had 84.6% of total predictability.

### 236 ANTI-NUCLEAR ANTIBODY PATTERN AND CLINICAL MANIFESTATION PRESENTATION IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Background and aims Anti-nuclear antibody (ANA) pattern analysis by immunofluorescence microscopy remains an important diagnostic tools for Systemic Lupus Erythematosus (SLE) in Indonesia. Although the utilisation of ANA pattern are used by physician to diagnosed SLE, there are very sparse study to explain on SLE clinical presentation and ANA pattern in Indonesia. This study was compiled to determined ANA pattern and clinical manifestation presentation in ANA positive patients.

Methods This study was an observational cross-sectional study in Hasan Sadikin General Hospital Bandung, 217 patients that had satisfied American College of Rheumatology (ACR) Criteria (1997) and ANA pattern were positive when diagnosed with SLE. Data was acquired from “RSHS Lupus Registry” database.

Results The study population consist of 217 patients, whom 208 patients (96%) were female and 206 patients (96%) were Sundanese (median age was 33, ranging from age 14 to 62 years), had Speckled ANA pattern (S-ANA) 98 patients (45%), Homogenous ANA Pattern (H-ANA) 63 patients (29%), Nucleolar ANA Pattern (N-ANA) 23 patients (11%), Speckled-Nucleolar ANA Pattern (SN-ANA) 18 patients (8%), and other staining patterns 15 patients (7%).The majority of clinical manifestation in S-ANA and H-ANA patients were haematological involvement with 69 patients (70%) and 42 patients (67%) respectively, and N-ANA and SN-ANA were malar rash with 20 patients (87%), and 13 patients (72%) respectively.

Conclusions The most frequent ANA pattern among SLE patients in this study is S-ANA pattern. The most common clinical presentation found is haematological involvement in S-ANA and H-ANA and malar rash in N-ANA and SN-ANA.

### 237 ISCHAEMIC STROKE IN SYSTEMIC LUPUS ERYTHEMATOSUS, DISTRIBUTION OF SUBTYPES AND A RISK GENOTYPE IN THE STAT4 GENE

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Background and aims We investigated the distribution of ischaemic stroke subtypes, classified according the Trial of Org 10 172 in Acute Stroke Treatment (TOAST) system, among patients with systemic lupus erythematosus (SLE). Genetic susceptibility in the signal transducer and activator of transcription factor 4 (STAT4) gene, defined by the single nucleotide polymorphism (SNP) rs10181656(G) were explored.

Methods We identified 69/665 SLE patients with stroke. Medical charts were retrieved and brain, cardiac and vascular imaging at the time of stroke were examined. Classification was performed according to TOAST: large-artery atherosclerosis (LAA), cardioembolism (CE), small-artery occlusion (SAO), stroke of other determined aetiology (OC) and stroke of undetermined aetiology (UE). Occurrence of the anti-phospholipid syndrome (APS) was documented. Evaluators were blinded to genotypes. General population controls (n=658) and SLE patients free from previous cerebrovascular disease (n=517) were used as comparators.
Results 56/69 patients with ischaemic stroke had charts with sufficient information for TOAST classification. Median age was 52 (17-84) years, 91% were female. All strokes classified as OC were attributed to APS. TOAST classification is presented in Table 1. Stroke of OE/APS and CE origin were associated with the STAT4 risk genotype as presented in Table 2.

Conclusions The majority of ischaemic strokes among SLE patients were of APS or CE origin. These two subtypes were associated with genetic susceptibility in the STAT4 gene. Patients with APS associated strokes were remarkably young. STAT4 genotype could, in addition to antiphospholipid antibodies and echocardiography, add information about stroke risk and help identify patients who will benefit from prophylactic anticoagulation treatment.

Background and aims In recent years hemophagocytic syndrome (HS) has been increasingly reported in patients with systemic lupus erythematosus (SLE).

Methods We reviewed the medical records of adult patients with SLE and HS for a recent 6 years period (2010–2015). The diagnosis of SLE was made using ACR criteria and of HS using Hunter criteria.

Results Among 110 consecutive patients, 13 (12 women) was identified having HS. The mean age was 37.69 +/- 11.4 years (21-68). HS revealed lupus in 3 patients. Fever, pericarditis and splenomegaly were found in 100%, 54% and 46% at presentation of HS. Bone marrow aspiration indicated hemophagocytosis in all patients. Laboratory features were bicytopenia or pancytopenia, high C-reactive protein level (mean 93 mg/L) hyperferritinemia (mean 11.082 ng/ml), hypertriglyceridemia (mean 4.2 g/L) in all patients. All patients had anti-nuclear antibodies when the HS occurred. Serum complement C3 was low in 10 patients. HS was associated with a lupus flare in 8 patients. Infections was diagnosed in 11 patients. Both conditions was considered present in 6 patients.

Corticosteroids were initially administered in all patients. Immunosuppressant therapy was used together with corticosteroids in 7 patients. Intravenous immunoglobulin was given in 3 cases. Anti-tuberculosis treatment was used also as first line treatment in 4 patients with life threatening presentation. All patients had a good outcome with a mean follow-up of 25 months.

Conclusions The occurrence of HS was most frequently associated with the SLE disease activity and bacterial infection. Profound cytopenia, high SLEDAI score are the characteristics of SLE patients with HS in our series.

Background and aims Systemic lupus erythematosus (SLE) is a systemic connective tissue disease involving multiple organ systems. There is lack of data on clinical and immunological profile in SLE patients from Indian subcontinent.

Objectives To describe clinical profile, outcome and laboratory profile of 226 SLE patients from records maintained at a tertiary care centre in India from 2008 – 2016

Methods All patients who satisfied 1997 revised criteria of the ACR or the 2012 New SLICC Classification Criteria for SLE were included in the analysis. The medical records were analysed for clinical and laboratory profile of SLE patients.

Results A total of 226 SLE patients records were analysed. The patient to male ratio was 9:1 (204 females, 22 males). The mean follow up was 4.2 years (Range 2–8 years). The overall mean age at diagnosis was 27 years (range 15–46 years).