Table 1. Description of complications

<table>
<thead>
<tr>
<th>Complications of US-guided CNB</th>
<th>11/20 (55%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients presenting complications, n/N (%)</td>
<td>11/20 (55%)</td>
</tr>
<tr>
<td>Description of transient complications</td>
<td>2</td>
</tr>
<tr>
<td>Swelling at biopsy site, n</td>
<td>1</td>
</tr>
<tr>
<td>Bleeding, n</td>
<td>1</td>
</tr>
<tr>
<td>Hematoma, n</td>
<td>0</td>
</tr>
<tr>
<td>Local Pain, n</td>
<td>0</td>
</tr>
<tr>
<td>Local infection, n</td>
<td>0</td>
</tr>
<tr>
<td>Sialocele or fistula, n</td>
<td>0</td>
</tr>
<tr>
<td>Anaesthesia/paresthesia, n</td>
<td>0</td>
</tr>
<tr>
<td>Transient facial palsy (&lt; 1 hour), n</td>
<td>1</td>
</tr>
<tr>
<td>No persistent complications reported</td>
<td>10</td>
</tr>
</tbody>
</table>

POST-BIOPSY COMPLICATIONS QUESTIONNAIRE

1. Did you have any swelling at biopsy site?  
   YES  NO  
   If yes, how long?  
   Physician control

2. Did you have any hematoma at biopsy site?  
   YES  NO  
   If yes, how long?  
   Physician control

3. Did you have any bleeding at biopsy site?  
   YES  NO  
   If yes, how long?  
   Physician control

4. Did you have pain at biopsy site?  
   YES  NO  
   If yes, choose a number from 0 to 10 to describe pain intensity  
   If yes, how long?  
   Physician control

5. Did you have any local infection?  
   YES  NO  
   Physician control

6. Did you have any anesthesia / paresthesia in the biopsy area?  
   YES  NO  
   If yes, how long?  
   Physician control

7. Did you have any sialocele or fistula in the biopsy area?  
   YES  NO  
   Physician control

Figure 1. Post-biopsy complication Questionnaire

Disclosure of Interests: Alen Zabotti Speakers bureau: UCB, Novartis, Janssen, Paid instructor for: Amgen, Consultant of: Janssen, Ivan Giovannini: None declared, Sara Zandonella Callegeri: None declared, Valeria Manfre: None declared, Michele Lorenzoni Consultant of: not relevant for this study, Enrico Pegolo: None declared, Cathryn Ann Scott: None declared, Alessandro Tel: None declared, Massimo Robiony Consultant of: not relevant for this study, Grant/research support from: not relevant for this study, Chiara Zuiani Consultant of: not relevant for this study, Grant/research support from: not relevant for this study, Salvatore De Vita Consultant of: GSK, Roche, Grant/research support from: not relevant for this study

DOI: 10.1136/annrheumdis-2021-eular.1804

POS0736 IDENTIFICATION OF MOLECULAR PHENOTYPES AND IMMUNE CELL INFILTRATION IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS ACCORDING TO LONGITUDINAL GENE EXPRESSION

S. Song1,2,3, S. X. Zhang1,2,3, J. Qian1,2,3, J. Zhao1,2,3, J. Shi1,2,3, Y. Hu1,2,3, J. Chen1, G. Y. Li1,2, P. F. He1, X. Li1,2,3, The Second Hospital of Shanxi Medical University, Department of Rheumatology, Taiyuan, China; 2Shanxi Li Xiaofeng Medical Groups, Department of Rheumatology, Taiyuan, China; 3Ministry of Education, Key Laboratory of Cellular Physiology at Shanxi Medical University, Taiyuan, China; 4Shanxi Medical University, Institute of Medical Data Sciences, Taiyuan, China

Background: Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with highly heterogeneous clinical presentation characterized by disease unpredictable flares and multi-systemic involvement1,2. This clinical heterogeneity calls for design a molecular stratification to improve clinical trial design and formulate personalized treatment therapies.

Objectives: This research was conducted to develop a reliable method to stratify SLE patients combined gene expression information and disease status.

Methods: The mRNA expression profile of GSE138458 (contained 307 patients and 23 controls) and GSE49454 (contained 111 patients and 16 controls) were downloaded from the publicly GEO databases. After background adjustment, batch correction, and other pre-processing, obtaining a big gene matrix to identify the differentially expressed genes (DEGs) in SLE compared with healthy controls, which were screened by P value < 0.01. SLE subtypes were identified by non-negative matrix factorization (NMF) based on DEGs. Acquired signature genes in different SLE subtypes were conducted to process pathway enrichment analysis in Metascape. SLEDAI score and immune cell infiltration was also performed between subtypes by software package R (version 4.0.3).

Results: Total 1202 DEGs were imputed to NMF unsupervised machine learning method. Patients with SLE were stratified into two subsets based on 184 signature genes derived from obtained DEGs(Fig.1A, 1B). GO and KEGG enrichment analysis showed that signature genes were mainly involved in negative regulation of innate immune response, toll-like receptor signaling pathway, regulation of immune effector process and so on(Fig.1C). Patients in Sub1 group had severe disease activity measures compared with those in Sub2(Fig.1D). SLEDAI scores from GSE49454 dataset were also higher in Sub1 compare with Sub2(Fig.1E). Further, immune cell infiltration results revealed an insufficient of regulatory T cell, CD8 T cells and naive CD4 T cells in Sub1 and neutrophils cells in Sub2(P<0.05)(Fig.1F).

Conclusion: Our findings indicate that patients with SLE could be stratified into 2 subtypes which had different lymphocyte status and closely related to disease activity. This phenotyping may help us understand the etiology of the disease, inform patient in the design of clinical trials and guide treatment decision.

REFERENCES:

Acknowledgements: This project was supported by National Science Foundation of China (82001740), Open Fund from the Key Laboratory of Cellular Physiology (Shanxi Medical University) (KLCP2019) and Innovation Plan for Postgraduate Education in Shanxi Province (2020B078).

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.1812

POS0737 LOW PRECONCEPTIONAL COMPLEMENT LEVEL IS RELATED WITH ADVERSE OBSTETRIC OUTCOME IN A MULTICENTRIC COHORT OF PREGNANCY IN PATIENTS WITH APS AND APL POSITIVITY

D. Lin1, C. Nalli1, L. Andreoli1, F. Crisafulli1, M. Frosi1, M. G. Lazzaroni1, V. Bittadze2, A. Calligaro3, V. Cantù4, R. Caporalì5, F. Carubbi6, C. Chighizola7, P. Coniglario8, F. Conti9, C. De Carolis10, T. Del Rossi11, M. Favaro10, M. Gerosa12, A. Iuliano13, J. Khizroeva2, A. Makatsariya2, P. L. Meroni12, M. Mosca1, P. Melissà1, R. Perricone8, P. Rovere-Querini16, G. D. Sebastiani13, C. Tani12, M. Tonello11, S. Truglia9, D. Zucchi12, F. Franceschini1, A. Tincani1.

1University and ASST Spedali Civili of Brescia, Rheumatology Unit, Brescia, Italy; 2I.M. Sechenov First Moscow State Medical University of the Ministry of Health of the Russian Federation (Sechenov University), Department of Obstetrics and Gynecology, Moscow, Russian Federation; 3University Hospital of Padua, Padua, Rheumatology Unit, Department of Medicine, Padua, Italy;