Table 1. Description of complications

Complications of US-guided CNB	
Patients presenting complications, n/N (%)	11/20 (55%)
Description of transient complications	
Swelling at biopsy site, n	2
Bleeding, n	1
Hematoma, n	1
Local Pain, n	7
Local infection, n	0
Sialoceles or fistula, n	0
Anaesthesia/paraesthesia, n	0
Transient facial palsy (< 1 hour), n	1
No persistent complications reported	

All the above specified complications were transient (< 12 weeks). No persistent complications were reported in the follow up.

POST-BIOPSY COMPLICATIONS QUESTIONNAIRE

1. Did you have any swelling at biopsy site? If yes, how long? Physician control	YES NO YES NO
2. Did you have any hematoma at biopsy site? If yes, how long? Physician control	YES NO YES NO
3. Did you have any bleeding at biopsy site? If yes, how long? Physician control	YES NO YES NO
4. Did you have pain at biopsy site? If yes, choose a number from 0 to 10 to describe pain intensity If yes, how long?	YES NO
5. Did you have any local infection? Physician control	YES NO YES NO
6. Did you have any anesthesia / paresthesia in the biopsy area? If yes, how long? Physician control	YES NO YES NO
7. Did you have any sialoceles or fistula in the biopsy area? Physician control	YES NO YES NO

Figure 1. Post-biopsy complication Questionnaire

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POS0736

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

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Background: Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with highly heterogeneous clinical presentation characterized by disease unpredictable flares and multi-systemic involvement^{1,2}. This clinical heterogeneity calls for design a molecular stratification to improve clinical trial design and formulate personalization 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.

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Fig.1: (A) NMF consensus clustering of 418 SLE patients based on 1202 differentially expressed genes. Columns and rows represent patients and signature genes, respectively. (B) Heatmap of two clusters defined by signature genes. Colors indicate relative gene expression value, with red for high expression and blue for low expression. (C) GO and KEGG pathway enrichment analysis of signature genes indicate possibly differential pathways. (D) Disease activity distribution in the two subtypes. (E) Boxplot show the difference of SLEDAI scores among two subtypes in GSE49454. (F) Violin plot show the fraction of 22 immune cell subpopulations in two subtypes based on CIBERSORT algorithm.

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POS0737

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

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