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POS0741
HISTOPATHOLOGIC PATTERNS OF LUPUS NEPHRITIS PREDICT THE RISKS OF MORTALITY - A SINGLE-CENTER RETROSPECTIVE STUDY

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Background: Lupus nephritis is a significant complication of systemic lupus erythematosus and is associated with increased risks of end-stage kidney disease and mortality.

Objectives: The retrospective observational study aims to investigate which component of the National Institutes of Health Activity and chronic indices of lupus nephritis can predict mortality.

Methods: We identified 528 SLE patients with biopsy-proven lupus nephritis between 2006 and 2019. Two patients with class VI lupus nephritis were excluded, and a total of 526 patients were analyzed. Serum creatinine, urine protein-to-creatinine ratio (UPCR), and serologic markers for SLE disease activity were measured at the time of the renal biopsy. The histopathologic findings of renal biopsies were classified by utilizing the International Society of Nephrology Renal Pathology Society (ISN/RPS) classification.

Results: Among 526 patients enrolled, 64 expired, and 44 were female (68.8%, p = 0.004). Class IV (a vs IV) comprised the most (n = 39, 60%), followed by class V (n = 18, 29.7%). Lower eGFR was observed in the death group, compared with the survival group (median: 24.7 vs. 80.5, p < 0.001). There were no significant differences in UPCR and serologic markers for SLE (dsDNA, C3, and C4). Total scores of chronicity index and the scores for each index were higher in the death group. Interestingly, although total scores of activity index in death and survival groups did not differ significantly, the scores for cellular crescents tended to be higher in the death group (1.38 ± 1.77 vs. 0.72 ± 1.24, p = 0.002).

In the univariable analysis, age, male sex, eGFR, activity index scores, cellular crescents, chronicity index scores, and all CI components (global obsolete glomeruli, tubular atrophy, interstitial fibrosis, fibrous crescents) and tubulointerstitial nephritis were significantly associated with increased risk of death. When male sex, age, and creatinine were included as confounders in the Cox regression model, only cellular crescents (HR 1.73 [95% CI: 1.10, 2.73]) but decreased with global obsolete glomeruli (HR 0.12 [95% CI: 0.02, 0.91]).

Conclusion: In this single-center observational study, fibrous crescents in females and cellular crescents in males were significantly associated with increased risks of mortality.

REFERENCES:

Disclosure of Interests: None declared
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Table 1. Logistic regression of predictors for mortality in patients with lupus nephritis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariable (Female)</th>
<th>Multivariable (Female)</th>
<th>Multivariable (Male)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR p value</td>
<td>HR p value</td>
<td>HR p value</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>1.03 (1.01-1.05)</td>
<td>0.002</td>
<td>1.02 (0.98-1.07)</td>
</tr>
<tr>
<td>Male sex</td>
<td>2.10 (1.23-3.55)</td>
<td>0.006</td>
<td></td>
</tr>
<tr>
<td>UPCR (mg/mg)</td>
<td>1.02 (0.95-1.09)</td>
<td>0.616</td>
<td></td>
</tr>
<tr>
<td>eGFR (mg/dl)</td>
<td>0.97 (0.96-0.99)</td>
<td>0.001</td>
<td>0.99 (0.96-1.00)</td>
</tr>
<tr>
<td>Activity index</td>
<td>1.06 (1.01-1.11)</td>
<td>0.027</td>
<td></td>
</tr>
<tr>
<td>Cellular crescents</td>
<td>1.29 (1.12-1.50)</td>
<td>0.001</td>
<td>1.03 (0.63-1.67)</td>
</tr>
<tr>
<td>Chronicity Index</td>
<td>1.16 (1.07-1.26)</td>
<td>0.001</td>
<td>1.03 (0.63-1.67)</td>
</tr>
<tr>
<td>global obsolete glomeruli</td>
<td>1.37 (1.08-1.76)</td>
<td>0.011</td>
<td>1.24 (0.55-2.77)</td>
</tr>
<tr>
<td>Tubular atrophy</td>
<td>1.65 (1.28-2.13)</td>
<td>0.001</td>
<td>0.41 (0.06-2.82)</td>
</tr>
<tr>
<td>Interstitial fibrosis</td>
<td>1.71 (1.52-2.23)</td>
<td>0.001</td>
<td>3.70 (0.52-26.24)</td>
</tr>
<tr>
<td>Tubulointerstitial nephritis</td>
<td>2.38 (1.40-4.03)</td>
<td>0.001</td>
<td>5.23 (1.51-18.09)</td>
</tr>
</tbody>
</table>
GO enrichment showed that these DEGs were primarily enriched in biological pathways, cell localization and molecular function and revealed that LN-related genes mainly involved in immune response. KEGG pathway annotation enrichment analysis revealed these DEGs were closely associated with Staphylococcus aureus infection, complement and coagulation cascades (Figure 1D). Fourteen hub genes (IFT3, IRF7, OAS3, GBP1, RSAD2, MX1, IFT2, IFI6, MX2, IS-F15, IFT1, OAS2, OASL, OAS1) were identified from PPI network (Figure 1E, 1F).

Results: DEGs were identified by Cytoscape. Online database to construct protein-protein interaction (PPI) network. Hub Genomes (KEGG) pathway enrichment analysis performed. We used STRING annotating DEGs for Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) pathway enrichment analysis performed. We used STRING online tool to construct protein-protein interaction (PPI) network. Hub Genomes (KEGG) pathway enrichment analysis performed.

Methods: GSE32591 dataset included 32 LN glomerular biopsy tissues and 15 donors. GSE104948 was a memory chip, which obtained from the GEO database. Glomerular tissues from living donors. The Oligo package was used to process the data to obtain the expression matrix files of all the related genes. ggplot2, heatmap packages were used to DEGs visualization. Masetcapse online database to construct protein-protein interaction (PPI) network. Hub Genomes (KEGG) pathway enrichment analysis performed. STRING online database to construct protein-protein interaction (PPI) network. Hub Genomes (KEGG) pathway enrichment analysis performed. STRING online database to construct protein-protein interaction (PPI) network. Hub Genomes (KEGG) pathway enrichment analysis performed.

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POSO043 GENE EXPRESSION MICROARRAY IN LUPUS NEPHRITIS BY BIOINFORMATIC ANALYSIS

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Background: Nephritis is one of the predominant causes of morbidity and mortality in patients with lupus. The lack of understanding regarding the molecular mechanisms of lupus nephritis (LN) hinders the development of specific targeted therapy for this progressive disease.

Objectives: In this study, we use bioinformatics method to analyze the genes involved in regulating the potential pathogenesis of LN.

Methods: The expression profile of LN (GSE104948 and GSE32591) was obtained from the GEO database. GSE104948 was a memory chip, which included 32 LN glomerular biopsy tissues and 3 glomerular tissues from living donors. GSE32591 dataset included 32 LN glomerular biopsy tissues and 15 glomerular tissues from living donors. The oligo package was used to process the data to obtain the expression matrix files of all the related genes. P<0.05 and log2(FC)<2 were setted as cut-off criteria for the DEGs. ggplot2, heatmap packages were used to DEGs visualization. Masetcapse online database was used to annotating DEGs for Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) pathway enrichment analysis performed. We used STRING online database to construct protein-protein interaction (PPI) network. Hub Genes were identified by Cytoscape.

Results: In differential expression analysis, 357 DEGs were identified, including 248 up-regulated genes and 109 down-regulated genes (Figure 1A, B). GO enrichment showed that these DEGs were primarily enriched in biological pathways, cell localization and molecular function and revealed that LN-related genes mainly involved in immune response. KEGG pathway annotation enrichment analysis revealed these DEGs were closely associated with Staphylococcus aureus infection, complement and coagulation cascades (Figure 1D). Fourteen hub genes (IFT3, IRF7, OAS3, GBP1, RSAD2, MX1, IFT2, IFI6, MX2, IS-F15, IFT1, OAS2, OASL, OAS1) were identified from PPI network (Figure 1E, 1F).

POSO044 A NEGATIVE INTERFERON BIOMARKER CD169 / SIGLEC-1 RULES OUT SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: While there have been advances in the therapy of systemic lupus erythematosus (SLE) in recent years, there have been no major new findings in SLE biomarkers [1,2]. Type I interferon (IFN) plays a pivotal role in the pathogenesis of SLE [3]. In 2008, we first described CD169 / SIGLEC-1 (sialic acid-binding immunoglobulin-like lectin-1), an interferon-induced adhesion molecule on monocytes in SLE patients [4]. For over five years SIGLEC-1 has been routinely assessed in our clinic.

Objectives: To evaluate and compare the diagnostic utility of the type I IFN induced SIGLEC-1 with established biomarkers in the initial diagnosis of the disease.

Methods: We analyzed retrospectively 232 patients who were on suspicion of SLE at Charité University Hospital Berlin between October 2015 and September 2020. Patients underwent full clinical characterization, and biomarkers were compared with the type I IFN induced SIGLEC-1. The diagnostic utility of the type I IFN induced SIGLEC-1 was evaluated in patients with established and those with suspicion of SLE.

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