

value for IL-6 of  $7.13 \pm 4.87$  pg/ml) vs. non-renal involvement ( $7.54 \pm 7.11$  pg/ml), with statistically significant value. The ROC analysis showed that IL-6 has predictive value for renal involvement in SLE patients (area under the curve = 0.701,  $p = 0.012$ , 95% confidence interval 0.551–0.852). A cut-off value of 5.26 has a sensitivity of 68.8% and a specificity of 70% for predicting renal damage in SLE patients (figure 1). No significant statistical difference was identified in IL-17A levels regarding renal involvement.

**Conclusions** Our findings indicate that serum IL-6 is a promising target for renal involvement. Circulating IL-17A showed no positive relationship with SLE activity. Further investigations are needed to determine their sensitivity and specificity as biomarkers of disease activity.

### P21 DISCOVERY OF POTENTIAL URINARY BIOMARKERS FROM KOREAN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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**Objective** This study aimed to demonstrate the potential of Activated Leukocyte Cell Adhesion Molecule (ALCAM), Hemopexin, and Peroxiredoxin (PRDX) 6 as urine biomarkers for systemic lupus erythematosus (SLE).

**Methods** We collected urine samples from 138 patients with SLE from Ajou Lupus Cohort and 39 healthy controls (HC). The concentrations of urine biomarker levels were analyzed by enzyme-linked immunosorbent assay kits specific for ALCAM, hemopexin and PRDX 6, respectively, according to the manufacturer's protocols. Receiver operating characteristic (ROC) curve analysis was performed to evaluate diagnostic utility and Pearson's correlation analysis was conducted to assess the relationships among the disease activity and urine biomarkers.

**Results** Patients with SLE showed a 5.7-fold increase in urinary ALCAM levels compared to HCs (6,760.5 pg/ml vs. 1,192.6 pg/ml,  $p < 0.001$ ). In urinary hemopexin and PRDX6, the average levels were also significantly higher in patients with SLE compared to HCs (hemopexin, 649.8 ng/ml vs. 202 ng/ml,  $p < 0.001$ ; PRDX6, 0.78 ng/ml vs. 0.17 ng/ml,  $p = 0.003$ ). ALCAM, hemopexin, and PRDX6 showed more significant diagnostic value, especially for lupus nephritis (LN), and the area under the receiver operating characteristic curve for LN was 0.850 for ALCAM (95% CI, 0.778–0.921), 0.781 for hemopexin (95% CI, 0.695–0.867), and 0.714 PRDX6 (95% CI, 0.617–0.812). In correlation analysis, all were significantly associated with anti-double stranded DNA (ALCAM,  $r = 0.350$ ,  $p < 0.001$ ; hemopexin,  $r = 0.346$ ,  $p < 0.001$ ; PRDX6,  $r = 0.191$ ,  $p = 0.026$ ) and SLEDAI (ALCAM,  $r = 0.526$ ,  $p < 0.001$ ; hemopexin,  $r = 0.479$ ,  $p < 0.001$ ; PRDX6,  $r = 0.262$ ,  $p = 0.002$ ).

**Conclusions** Urinary ALCAM, hemopexin and PRDX 6 were highly expressed patients with SLE compared to HCs. Thus, we suggest that urinary ALCAM, hemopexin and PRDX 6 can be potential biomarkers for SLE, especially valuable in the diagnosis of LN.

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### CONSISTENT BENEFITS FROM BELIMUMAB IRRESPECTIVE OF ANTIPHOSPHOLIPID AUTOANTIBODY PROFILE, YET NOT IN THE PRESENCE OF LUPUS ANTICOAGULANT

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**Objective** To assess whether presence of antiphospholipid antibodies (aPL) in serum at baseline could foreshadow treatment response after 52 weeks of belimumab therapy, based on pooled data from randomised controlled trials (RCTs) in systemic lupus erythematosus (SLE).

**Methods** Data from five RCTs of belimumab in SLE were pooled, i.e., BLISS-52, BLISS-76, BLISS-Northeast Asia, EMBRACE, and BLISS-SC. Patients were divided into six subgroups based on aPL serology at baseline: anti-cardiolipin (aCL) positive, aCL negative, lupus anticoagulant (LAC) positive, LAC negative, positive for any aPL, negative for all aPL. Binary logistic regression models were conducted across four treatment-response constructs every fourth week from baseline, up to and including week 52, i.e., SLE Responder Index (SRI)-4, British Isles Lupus Assessment Group-based Composite Lupus Assessment (BICLA), Lupus Low Disease Activity State (LLDAS), and Definitions Of Remission In SLE (DORIS) remission. The analyses did not discriminate across different antibody isotypes, and the models were adjusted for trial variance. Participants with insufficient data were excluded from analysis.

**Results** In total, 3086 patients received belimumab at the approved dose of 10 mg/kg monthly intravenously or 200 mg weekly subcutaneously (N=1869) or placebo (N=1217). Of these, 1257 patients had data on LAC at baseline, four of which lacked data on SRI-4. At the 52-week follow-up, belimumab outperformed placebo in inducing treatment response for patients who were LAC negative at baseline (odds ratio [95% confidence interval]: SRI-4: 1.58 [1.21–2.05],  $p=0.001$ ; BICLA: 1.35 [1.03–1.78],  $p=0.03$ ; LLDAS: 1.84 [1.25–2.71],  $p=0.002$ ; DORIS: 1.84 [1.09–3.13],  $p=0.023$ ) while there was no significant difference between treatment arms for patients who were LAC positive (SRI-4: 1.54 [0.88–2.72],  $p=0.132$ ; BICLA: 1.45 [0.81–2.60],  $p=0.214$ ; LLDAS: 1.51 [0.71–3.23],  $p=0.289$ ; DORIS: 2.54 [0.70–9.18],  $p=0.154$ ). Belimumab was not shown to benefit aCL positive patients towards attainment of DORIS remission ( $p=0.187$ ) or aPL negative patients towards BICLA response ( $p=0.071$ ). In all other subgroups, results in all models favoured belimumab.

**Conclusion** Consistent benefits from belimumab were observed irrespective of aCL or aPL seropositivity, with the exception of LAC positive patients in whom belimumab-induced attainability of SRI-4, BICLA, LLDAS, and DORIS remission was abated.

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