remaining patients, remission lasted for 9 and 16 months after GCs withdrawal. Each flare required intake of low prednisolone doses for 3-4 weeks.

**Conclusion:** GCs withdrawal is an achievable goal in SLE and may be attempted after a long-remission, and possibly after aggressive intensive care in the early stages of SLE.

**Disclosure of Interests:** None declared

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**AB0390**

**ASSOCIATION BETWEEN INITIAL SERUM “TUMOR NECROSIS FACTOR-LIKE WEAK INDUCER OF APOTOPSIS” (TWEAK) LEVEL AND TREATMENT RESPONSE IN SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) NEPHROPATHY**

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**Background:** SLE is a chronic inflammatory immunologic abnormalities disease, with involvement of antinuclear antibodies. The SLE renal involvement is clinically apparent in approximately 50% patients (Norby et al., 2017). It is very important to introduce the prompt treatment to prevent the permanent end stage renal disease.

**Objectives:** This study aimed to identify the serum biomarkers that correlate with pretreatment disease activity in patients with SLE nephropathy and predict the treatment outcome so that we may identify the unresponsive cases and switch to the other biologic agents like anti-TWEAK monoclonal antibody in the biologic failure scenario.

**Methods:** This was a hospital-based prospective analytical study conducted from January 2018 to November 2019 in Rheumatology Department, Yangon Specialist Hospital. 88 SLE nephropathy patients with 24-hour urinary protein above 0.5g/day planned to have 6 months course of IV cyclophosphamide were enrolled. The paired serum sample of each patient was analyzed by ELISA twice to get the mean serum TWEAK value. Pretreatment SLE disease activity was assessed by the SLEDAI 2k. After the completion of 6 months of aggressive treatment, the treatment response was assessed by measuring the 24 hour urinary protein.

**Results:** Among the 88 patients, 63 patients (71.6%) had completed total 6-months course and 25 patients (28.4%) had not completed: 11 patients (12.5%) expired and 2 patients (2.27%) had been changed to other DMARD and 12 patients (13.63%) did not attend the follow up clinic. The mean serum TWEAK level was 856 ± 77 pg/ml in 88 patients. According to the range of serum TWEAK level, most of the patients had serum TWEAK level of 601-900 pg/ml (53.4% of the study population). There was positive correlation between pre-treatment SLEDAI 2k score and pretreatment serum TWEAK level (r=0.464 and P <0.001). When the SLEDAI 2k score was grouped into mild, moderate, high and very high disease activity, the serum TWEAK level also had positive association with the different levels of disease activity (p<0.001). Among 63 treatment completed patients, 55 patients (87.3%) were the treatment responders but 8 patients (12.7%) were treatment non-responders. There was significant difference in the pretreatment SLEDAI 2k in terms of disease activity between treatment responder and treatment non-responder (p<0.001). There was significant difference in the pretreatment SLEDAI 2k in terms of reduction in 24-hours urinary protein between treatment responder and treatment non-responder (p<0.001). There was no significant difference in the level of pretreatment serum TWEAK level between treatment responders and treatment non responders (p=1.000). There was also no significant difference in the pretreatment serum TWEAK level between treatment responders and treatment non-responders in terms of reduction in 24 hours urinary protein (p=0.804).

**Conclusion:** Although the pretreatment serum TWEAK level had a positive correlation with pretreatment disease activity of SLEDAI 2k, it did not reflect the outcome of the responsiveness to the intensive therapy.

**References:**


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**AB0391**

**EFFECT OF HCQ ON LLDAS ACHIEVEMENT IN SLE PATIENTS**

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**Background:** HCQ for SLE in Japan has been administered in many cases after approval. Therefore, the effect of additional administration of HCQ on low disease activity of SLE was considered to be clearer.

**Objectives:** To clarify the effect of HCQ treatment on the control of disease activity in SLE patients.

**Methods:** All SLE patients with low disease activity (LDA) enrolled in this study started additional HCQ treatment from January 2016. All patients with LDA enrolled in this study started HCQ treatment and had been receiving oral HCQ continuously for at least 3 months without using other immunosuppressive treatments or glucocorticoids. Disease activity was evaluated by SLEDAI, CLASI, and LLDAS, and serum complement values, anti-DNA antibodies, and pro-inflammatory cytokines were analyzed as immunological biomarkers before and after HCQ treatment.

**Results:** 52 of 100 patients were enrolled in this study (M:F; 4:48, average age; 40.6±13.4), 24 lupus nephritis patients were in sustained remission. 29 patients (56%) achieved LLDAS and 3 patients (6%) achieved clinical remission (CR) before HCQ administration. Of the 20 patients (38%) who did not achieve LLDAS before HCQ administration, the LLDAS achievement rates at 3, 6, and 12 months after additional HCQ were 47%, 59%, and 81% (including 12.5% of CR achievement rates), respectively. Serum levels of MRP8, MRP14, TNF-α, IL-6, VEGF-A, IL-1ra, MIP-1α and IL-2 decreased significantly 3 months after additional HCQ treatment. In addition, serum levels of MRP8, MRP14, TNF-α, IL-6 and IL-2 also decreased significantly 3 months after additional HCQ treatment despite achieving LLDAS or CR. The expressions of IFN-α didn’t decrease significantly in 9 cases that could be detected. The magnitude of the changes in serum MRPs, MRP14, IL-8 and IL-1α levels in patients with a history of LN was significantly higher than in those without a history of LN. The magnitude of the reduction in serum MCP-1 levels in patients not achieving LLDAS with a history of LN was significantly higher than in those without a history of LN (W=0.046).

The change of CLASI activity score was correlated with the change in serum levels of MRP14 and MCP-1 with univariate analysis (MPR14: r=−0.41, P=0.017, MCP-1: r=−0.58, P=0.0006). The change of serum C3 levels had a negative correlation with MCP-1 (r=−0.33, P=0.022).

The magnitude of the change in serum levels of MRPs, TNF-α, IL-8, MCP-1, MIP-1α and IL-1α in patients achieving LLDAS were correlated with the change of CLASI activity score with univariate analysis (MPR14: r=−0.49, P=0.041, TNF-α: r=0.74, P=0.0038, IL-1α: r=0.66, P=0.038, MIP-1α: r=0.63, P=0.037, Figure 1). Moreover, the change of serum C3 and C4 levels in them had a negative correlation with the change of serum MCP-1 levels (Figure 2).

**Figure 1.** Correlation between change of CLASI activity scores and serum MCP-1 levels in SLE patients with LLDAS (IL-8: r=0.77, P<0.0001, MCP-1: r=0.80, P<0.0001).

**Figure 2.** Correlation between change of serum C3 and C4 levels and serum MCP-1 levels in SLE patients with LLDAS (C3: r=−0.40, P=0.028, C4: r=−0.37, P=0.047).

**Conclusion:** Additional administration of HCQ is useful for cytokine control even in LLDAS-achieved cases, and particularly contributes to the improvement of serum MRP8, MRP14, TNF-α and MCP-1 levels.

In addition, regulation of IL-8 and MCP-1 is important for control of renal lesions of SLE and therefore, further study of the influence of HCQ on LLDAS-achieved cases is expected.