

**Methods** Cross-sectional study of patients with SLE. Demographics, coronary risk, disease activity and inflammatory markers were studied. The diagnosis of MS was established with the NHLBI/AHA criteria. Statistical analysis was performed using SPSS 20.0 software and a P value < 0.05 was considered significant.

**Results** 126 patients with SLE, 107 women (84%) and 19 men (15%), age 41 ± 13 years old and disease duration 9 ± 7 years. The prevalence of MS was 33.3%. No association was found with age, education level, smoking or steroid use in patients with MS. In multivariate analysis only elevated erythrocyte sedimentation rate (ESR) had a statistical significance (p = 0.012). Positive association was found between higher values of ESR and hypertriglyceridemia (p = 0.0002), body mass index (p = 0.0043) and lower levels of HDL and C3 (p = 0.0152).

**Conclusions** The prevalence of MS in our population (33%) was higher than reported in the SLICC registry (15%). The association of metabolic and inflammatory characteristics increases cardiovascular risk by a proinflammatory state. The results suggest the need for early diagnosis and treatment of MS to reduce cardiovascular comorbidity in patients with SLE.

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#### VITAMIN D DEFICIENCY AS NOVEL RISK FACTOR OF ACCELERATED ATHEROSCLEROSIS CARDIOASCULAR DISEASES IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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**Background and aims** Vitamin D deficiency is common in women living near to equatorial line. Deficiency of vitamin D could lead to more severe manifestation of autoimmune disease, it may accelerate complication in atherosclerosis cardiovascular disease.

**Objective** We aimed to evaluate the impact of vitamin D deficiency in the severity of SLE, especially in the appearance of ASCVD in SLE patients.

**Methods Design:** Systematic review and meta-analysis.

**Data Sources:** Electronic databases (CENTRAL; Medline; Springerlink; Cochrane Database) were searched up to May 2015.

**Review methods:** Longitudinal study that compared level of serum vitamin D in SLE patients with and without ASCVD manifestation were included. Study selection, data extraction and risk of bias assessment (Cochrane risk of bias tool) were performed by five reviewers.

**Results** Total of 16 trials (1723 participants) were included. Meta-analysis of 8 trials (781 participants) found that serum vitamin D (25-hydroxyvitamin D) levels in severe SLE patients with ASCVD manifestation were significantly lower compared to non-ASCVD SLE patients during remission (pooled RR 0.64; 95% CI = 0.34–0.77; p = 0.005). No statistically significant difference in serum vitamin D were observed in meta-analysis of other 8 trials (pooled RR 0.96; 95% CI = 0.54–1.7; p = 0.12). Moreover, there is a significant inverse correlation between serum vitamin D levels and Systemic Lupus Erythematosus Disease Activity Index 2000 ( $r = -0.373$ , p = 0.016).

**Conclusions** Results demonstrates that vitamin D deficiency could be a novel risk factor for accelerating endothelial dysfunction and ASCVD in SLE patients. Vitamin D supplementation might modulate an immunosuppressive effects, endothelial repair mechanisms, and endothelial function in SLE patients with significant ASCVD risk.

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#### IMPACT OF DISEASE ACTIVITY ON ORGAN DAMAGE RISK OVER TIME IN SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) –THE HOPKINS LUPUS COHORT

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**Abstract 189 Table 1** Time-dependent cox proportional hazard models: effect of disease activity on the risk of new organ damage.

Variable	Model 1		Model 2		Model 3	
	HR (95% CI)	P-value	HR (95% CI)	P-value	HR (95% CI)	P-value
Age at cohort entry	1.03 (1.03-1.04)	<0.001	1.03 (1.03-1.04)	<0.001	1.03 (1.03-1.04)	<0.001
SDI score at cohort entry	1.06 (1.02-1.11)	0.002	1.07 (1.03-1.11)	<0.001	1.07 (1.03-1.11)	<0.001
Immunosuppressant use during follow-up	1.23 (1.05-1.43)	0.012	1.15 (0.99-1.35)	0.076	1.15 (0.98-1.35)	0.079
Average corticosteroid use (≥7.5 mg vs. <7.5 mg)	1.74 (1.49-2.04)	<0.001	1.61 (1.37-1.89)	<0.001	1.61 (1.37-1.89)	<0.001
SELENA-SLEDAI score during follow-up (≥6 vs. <6)	1.40 (1.17-1.67)	0.012	0.82 (0.65-1.03)	0.088	NA	
Proportion of clinic visits with SLEDAI score ≥6 during follow-up	NA		3.82 (2.71-5.39)	<0.001	3.14 (2.42-4.08)	<0.001

**Background and aims** Disease activity increases risk of irreversible organ damage in SLE.

To understand the impact of disease activity (SELENA-SLE-DAI) and proportion of time with a certain level of disease activity on risk of developing new organ damage (SLICC/SCR Damage Index (SDI) score).

**Methods** Cox Proportional hazard models were used to estimate the impact of disease activity as time-dependent variables on the risk of developing any new organ damage over time.

**Results** Patients (n=2199) were followed for an average of 6.2 years (mean age at cohort entry, 38.0 years; mean disease duration, 5.1 years). The most frequent types of organ damage occurring over time were ocular (cataract) and musculoskeletal (osteoporotic fractures). In Model 1, excluding the variable "proportion of clinic visits with SLEDAI score >6," age, and SDI score at cohort entry, SLEDAI score during follow-up and corticosteroid use during follow-up were significant predictors of risk of developing any new organ damage. When including the "proportion of clinic visits with SLEDAI score >6" (Model 2), SLEDAI score during follow-up was no longer significant. In Model 3, excluding the time-dependent variable "SLEDAI score during follow-up" from the model, the effect of "proportion of clinic visits with SLEDAI score >5 was slightly reduced but remained significant.

**Conclusions** Higher organ damage risk was observed in patients with high levels of disease activity for a greater time compared to those with high levels of disease activity for a lesser time. These findings call for active measures to control disease activity over time in SLE.

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#### CASE REPORT: ELECTROLYTE ABNORMALITY CAUSED BY SUSPECTED ACQUIRED GITELMAN SYNDROME IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENT

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**Background and aims** Gitelman syndrome is a hereditary autosomal recessive abnormality of the kidney, that had presentation as hypokalemia, metabolic alkalosis, hypomagnesemia, and hypocalciuria. It is caused by mutation of SLC 12A3 gene encoding thiazide sensitive sodium-chloride co transporter. However, acquired Gitelman Syndrome can be found in SLE

**Methods** A 37 years old woman was admitted to hospital because of 7 days fever. She also had butterfly rash, hemolytic anaemia, and leucopenia. She was diagnosed as having SLE with hematologic and skin manifestation. Her sodium and potassium level were low. Her electrolyte imbalances were response well with supportive treatment. Ten days later, she had recurrent hypokalemia. Her haemoglobin level was decreased, and she was given packed red cell transfusion in her second admission.

**Results** One month later, she had neurologic manifestation, hemolytic anaemia, discoid rash, and low complement level.

Her sodium, potassium, calcium, and magnesium levels were still low. High dose steroid and chloroquin were given. She was discharged from the hospital well, but had recurrent admission five days later due to spasm of the face and extremities, can't open mouth, dyspnea, recurrent hyponatremia, hypokalemia, hypomagnesemia, and hypocalcemia. Her blood gas analysis showed metabolic alkalosis. She was diagnosed as having electrolyte abnormality due to suspected acquired Gitelman syndrome.

**Conclusions** She was given pulse dose steroid, mycophenolate mofetil, and parenteral correction of electrolyte abnormality. She responded well, and discharged from the hospital.

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#### REASONS FOR HOSPITALISATION/HOSPITALIZATION AMONG FILIPINO PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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**Background and aims** To describe reasons and outcomes of hospitalisation among Filipino patients with systemic lupus erythematosus (SLE).

**Methods** Retrospective hospital chart review of Filipino SLE patients confined at University of Santo Tomas (UST) Hospital in Manila, Philippines from January 2011 to December 2015. Excluded were admissions for routine infusions. Final diagnoses were categorised as SLE-related or non-SLE related. Effect on SLE-relatedness of disease duration, age at SLE diagnosis and length of hospitalisation were analysed using Chi-square and Pearson's correlation coefficient.

**Results** There were 430 patients (95.58% female, 78% adults >18 years old) with 596 hospitalizations, Median number of hospitalisation per year per patient was one (range 1–3). Average age at hospitalisation was 28.98+12.95 SD years (range 5–71), average disease duration 6.51+6.30 SD years (range <1–16). Mean length of hospitalisation was 4.22+4.61 SD days (range 1–38). 479 (80%) hospitalizations for SLE-related reasons included lupus flare (357), lupus flare with concomitant infection (68), kidney biopsy (28) and renal failure requiring dialysis (26). Of 117 non-SLE related hospitalizations, infection with inactive SLE was recorded in 40 (29%). Among 16 deaths, 9 were infection-related and 7 were SLE-related. There was no significant association of age at SLE diagnosis, disease duration nor length of hospitalisation with SLE-relatedness.

**Conclusions** In this cohort of Filipino SLE patients, majority of hospitalizations were due to active SLE and/or infection, with infection having high risk for poorer outcomes. These findings strongly reinforce need to effectively control disease while minimising infection risk usually due to immunosuppressives.