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 proinflamatory state. The results suggest the need for early diagnosis and treatment of MS to reduce cardiovascular comorbidity in patients with SLE.
Background and aims Disease activity increases risk of irreversible organ damage in SLE. To understand the impact of disease activity (SELENA-SLEDAI) 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 (osteoarticular 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.

CASE REPORT: ELECTROLYTE ABNORMALITY CAUSED BY SUSPECTED ACQUIRED GITELMAN SYNDROME IN SYSTEMIC LUPUS ERYSERMATOSUS 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 anemia, 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 anemia, 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 hypotension, hypokalemia, hypomagnesemia, and hypocalcemia. Her blood gas analysis showed metabolic alkalosis. She was diagnosed as having electrolyt 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.