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 (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.

CASE REPORT: ELECTROLYTE ABNORMALITY CAUSED BY SUSpected ACQUIRED GITelman SYndrome IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENT

A Paramaiswari*, L Kurnia Sari, E tanoyo, N narulita. Sardjito General Hospital/Gajah Mada University School of Medicine, Internal Medicine, Yogyakarta, Indonesia

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 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.