is a systemic autoimmune disease affecting all organ systems. The aim was to present a cohort of lupus patients who in the course of the disease presented with Hashimoto's thyroiditis.

Methods A cohort of 10 patients, female, aged 21–42 years, suffering from SLE is presented. The patients were diagnosed with lupus and were either on treatment with hydroxychloroquine or with hydroxychloroquine and prednisone. Within this cohort a female patient aged 42 years had also antiphospholipid antibodies and had suffered a stroke at the age of 36.

Results Within this cohort 6 patients had positive both anti-thyroglobulin and thyroid peroxidase antibodies, 3 patients had positive only anti-thyroglobulin antibodies and 1 patient had positive only thyroid peroxidase antibodies. Within this group, 7 patients were euthyroid and were followed up, while 3 had hypothyroidism and were on treatment with thyroxine.

Conclusion In conclusion, SLE may be accompanied by Hashimoto's thyroiditis. In another cohort a two-fold increased risk of Hashimoto's thyroiditis was observed in lupus patients. The presence of anti-Sm antibodies was found to favor this association. In another cohort hypothyroidism, subclinical hypothyroidism and subclinical hyperthyroidism accompanied by the presence of thyroid autoantibodies was observed in a group of lupus patients. It appears that lupus patients may present with Hashimoto's thyroiditis with or without hypothyroidism and should be screened for this disorder during long-term observation.

PO.4.90 REMISSION AND CLINICAL PATTERNS OF SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) IN SOUTHERN PAKISTAN: A RETROSPECTIVE COHORT STUDY

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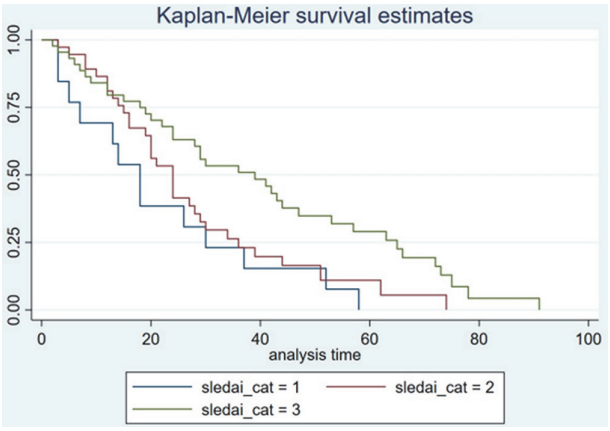
Background SLE is a difVcult to treat autoimmune disease due to clinical heterogeneity, unpredictability of disease iares and limited therapeutics. These challenges are worsened in a low-middle income country (LMIC) setting, yet clinical epidemiology from LMIC may have global beneVts.

Objectives To determine (i) the clinical pattern of SLE and (ii) the effect of SLE severity and treatment regimen on time to remission.

Methods A retrospective cohort study of 200 SLE patients' medical records (2014–20) from ImmunoCure clinic was conducted. Patients fulVlled ACR criteria 1997 for SLE classiVcation. SLEDAI-2K categories were used as outcome measure: mild (score <=6), moderate (7–10), severe (>10) to evaluate clinical pattern of SLE. Statistical analyses were performed using STATA v16.0. Kruskal-Wallis test was used for continuous measures, and Pearson's chi square test was used to compare categorical variables across SLEDAI severity. Remission status based on DORIS criteria and time to remission (>1year; n=94) was the secondary outcome.

Total doses of all drugs were calculated. Survival regression performed with Kaplan Meier curve.



Abstract PO.4.90 Figure 1

Results Most frequent antibodies are anti-dsDNA (63%), SSA (24%) and Ku (17.5%). Anti-cardiolipin (aCL) antibodies associate with severe SLE (OR = 3.6, P<0.01). Most common presentations were arthritis (85%), alopecia (53%), anemia (38%), rash (35%) and CNS disease (28%). Nephritis, CNS disease, cytopenias and oral ulcers are signiVcantly associated with severe SLE (P<0.01). ILD is in 10% of our cohort. Frequency of severe SLE was 47.5%, whereas mild disease was 16.5%. Mean duration of follow up was 41±19 months.

Every month of follow-up increased the odds of remission by 6% (P<0.05). Clinical remission on treatment (at Pred<=5mg) was successfully achieved in 62% patients. Complete remission (off all drugs & Pred) was achieved in 24 patients (14 in severe SLEDAI category) out of 200, with a mean post remission follow-up of 18 ± 15 months.. Hazard of time to remission is 61% (CI: 0.21–0.77, P=0.01) less in severe SLE as compared to mild SLE disease activity (Figure 1).

Conclusion Sustained remission is possible even in severe SLE in a LMIC setting if adequate immunosuppression is provided with persistent clinical follow-up.

PO.4.91 BANK1 AND IL-10+ B CELLS: BRINGING SOME LIGHT TO THE RELATIONSHIP BETWEEN MICROBIOTA COMPOSITION AND AUTOIMMUNITY DEVELOPMENT IN A MURINE MODEL OF LUPUS

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Background A new feature that seems to be decisive in autoimmune pathogenesis is the gut microbiota composition. However, its exact role remains to be determined. In systemic lupus erythematosus (SLE), an autoimmune disease characterized by persistent inflammation affecting multiple organs, the contribution of the gut microbiota is particularly elusive. The B cell scaffold with ankyrin repeats (Bank1) gene, which plays a role in TLR7 signalling, has been genetically associated with lupus in humans, and associated with a