For the aim of this study, we excluded from the analysis NMSC (ICD-10: C44) and diagnoses based on autopsy only or Death Certificate Only.

**Results:** 2504 patients were followed-up for a total of 18,006 person-years (median follow-up: 6.8 years). After 5 and 10 years of follow-up, the cumulative cancer incidence was 2.6% and 8.5%, respectively. The most common cancers were breast (n=34), lung (n=24), colon-rectum-anus (n=20), and non-Hodgkin lymphomas (NHL) (n=20). Overall, no excess cancer risk was noted (SIR=0.87, 95% CI: 0.75-1.00), whereas the number of observed NHL cases was more than two-fold significantly higher than expected (SIR=2.52, 95% CI: 1.30-4.39). The subgroup analysis showed a higher risk of NHL among SS patients (SIR=3.84, 95% CI: 1.92-6.87) and SLE patients (SIR=2.69, 95% CI: 0.99-5.84). Conversely, the study population showed a decreased risk for cancers of breast (SIR=0.61, 95% CI: 0.42-0.85) and corpus uteri (SIR=0.21, 95% CI: 0.03-0.77).

**Conclusion:** The incidence of NHL was higher among patients with SS and SLE. Surveillance for haematological malignancies in these patients is recommended. The lower risk of cancer for breast and corpus uteri in CTD indirectly supports cancer screening programs and highlights the role of the continuous clinical follow-up for these chronic conditions.

**REFERENCES:**


**Disclosure of Interests:** None declared

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**A MACHINE LEARNING ANALYSIS OF FACTORS PREDICTING ORGAN DAMAGE PROGRESSION IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS USING THE SPANISH SOCIETY OF RHEUMATOLOGY LUPUS REGISTRY (RELESSER)**

J. M. Pego-Reigosa1, W. Fakhouri2, S. Diaz-Cerezo3, A. Cooper4, A. M. Saunders5, G. Segall6, C. Sapin7, S. Moyano8, I. Rua-Figueroa8, M. University Hospital Complex of Vigo. IRDIS (Investigation in Rheumatology and Immunomediated Diseases)-VIGO Group, Gastro-Sur Health Research Institute (IISGS), Rheumatology Department, Vigo, Spain; 2Eli Lilly and Company Ltd, Value, Evidence, and Outcomes (VEO), Braacknell, United Kingdom; 3Eli Lilly and Company Ltd, Medical Affairs, Madrid, Spain; 4IQVIA, Real World Solutions (RWS), London, United Kingdom; 5IQVIA, Integrated Real World Evidence & Solutions (IRES), London, United Kingdom; 6Eli Lilly and Company Ltd, Statistics-Europe, Neully Sur Seine, France; 7Doctor Negrin University Hospital of Gran Canaria, Rheumatology Department, Las Palmas de Gran Canaria, Spain

**Background:** Evidence shows that around half of the patients with systemic lupus erythematosus (SLE) develop irreversible organ damage due to the disease itself or to other factors (e.g., steroid treatment). It is essential to have a comprehensive understanding of factors that predict organ damage progression to identify at-risk patients and inform clinical decision making.

**Objectives:** To develop an algorithm, using machine learning (ML) methodology, that predicts organ damage progression in SLE patients.

**Methods:** The Spanish Society of Rheumatology Lupus Registry (RELESSER) with patient records from 45 Rheumatology Units across Spain was used. RELESSER data were collected from 2011 to 2021 and captured demographic and comprehensive clinical information. In this analysis, a sample of 2,676 patients was used. The Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index (SDI) was used to measure organ damage progression between 2015 (start of the prospective data collection in RELESSER) and 2020. To predict the risk of an increase in the SDI, 102 variables were identified as potential predictors. A ML model (gradient boosting trees) was developed and validated by a simple logistic regression (LG) model. The area under the receiver operating characteristic curve (AUCROC) was used to quantify the improvement over random chance (an AUCROC of 0.5). Shapley Additive Explanatory (SHAP) values were used in the ML model to identify predictors and their contribution to damage progression.

**Results:** Of all patients, 13% experienced organ damage progression, with 2-year patient follow-up. The ML algorithm was better at identifying these patients (AUCROC 0.68) than the LG model (AUCROC 0.63) (Figure 1). ML model performance can be contextualized using a random sample of 100 SLE patients of whom 13 suffered organ damage progression, the model would successfully identify 12. However, 66 additional patients would be incorrectly identified (True Positive 90%; False Positive 79%). The top 5 predictors of damage progression, across all patients, were patient age >49 years,

**Figure 1. ROC AUC plot**

Creatinine > 0.9 mg/dl, cardiovascular-related disease complications (>3 complications), low hematocrit level recorded recently (<41.9 months ago), and triglycerides > 81.5 mg/dl. At the patient level, the 5 patients with the highest predicted risk had strong predictors of progression, with key predictors being patient age at study entry >49 years, age at diagnosis >40 years, cardiovascular-related disease complications, and increased creatinine >0.9 mg/dl. The 5 patients with the lowest predicted risk had strong predictors of no change with key predictors being not having a low hematocrit level recorded recently, triglycerides <81.5 mg/dl and patient age at study entry <49 years.

**Conclusion:** We developed a machine learning model, using an exhaustive set of variables in RELESSER which successfully predicted short-term organ damage progression in SLE patients, and outperformed a standard regression model. If the model were to be used as a clinical tool, only light-touch interventions should be carried out due to high false positive rate. Further model optimizations, including exposing the model to longer follow-up data and testing it in non-Spanish patients is needed.

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**LONG-TERM OUTCOME OF SYSTEMIC LUPUS ERYTHEMATOSUS (SLE); DATA FROM THE LARGE POPULATION-BASED SOUTHEAST SLE COHORT (NOR-SLE)**


1Oslo University Hospital, Rikshospitalet, Department of Rheumatology, Oslo, Norway; 2Department of Medicine, University of Bergen, Bergen, Norway; 3Department of Rheumatology, Ullevål University Hospital, Oslo, Norway; 4Department of Rheumatology, Rikshospitalet, Oslo University Hospital, Oslo, Norway; 5Department of Rheumatology, Kista University Hospital, Stockholm, Sweden; 6Department of Rheumatology, Haukeland University Hospital, Bergen, Norway; 7Department of Rheumatology, Aalesund University Hospital, Aalesund, Norway; 8Department of Statistics, University of Oslo, Oslo, Norway.
Background: Population-based studies on Systemic Lupus Erythematosus (SLE) patients with a verified diagnosis is considered the gold standard to find true outcomes in SLE, but few population-based SLE cohorts have follow-up over 15 years [1]. Norway is among the few countries worldwide where social and structural factors facilitate the gathering of complete population-based cohorts in rare disease like SLE due to its healthcare organization.

Methods: The study included all SLE patients who were resident in the South-east region of Norway during 1999 - 2017 and met the 1997 American College of Rheumatology classification criteria for SLE. All SLE diagnosis was confirmed by chart review. SLE patients and 15 controls for each case (matched by age, gender and ethnicity) were linked to the Norwegian Cause of Death Registry. We examined survival by means of Kaplan-Meyer estimates and determined if immediate cause of death differs between SLE patients and the general population.

Results: We identified 1298 SLE patients in the region, of whom 673 was incident cases; all captures within one year from diagnosis. Of the incident cases, 76 (11%) died during 8434 years of follow-up (Table 1). The five-, ten-, 15- and 20-year survival for incident SLE patients (controls) was respectively 98 (98), 94 (96), 87 (94) and 82 (88) % and differed significantly first after ten years of disease duration compared to controls. Figure 1 shows 20-year survival for incident SLE patients and matched controls; stratified by gender. SMR for all SLE cases was 2.3 (95 % CI 1.5 - 4.0); female SLE 2.2 (95 % CI 1.6 - 3.9) and male SLE 2.5 (95 % CI 1.5 - 4.0); female SLE 2.5 (95 % CI 1.6 – 3.9) and male SLE 2.5 (95 % CI 1.5 – 4.0). The most common immediate cause of death in SLE was coronary artery disease (CVD) except pulmonary embolism and cerebral bleeding) and of infections A00-B99, J10-18, N39, M86 or U07.

Conclusion: Mortality in SLE is substantially increased. Differences in survival compared to the general population only appear after ten years of disease duration. CVD was the most common immediate cause of death and more frequent compared to the general population only appear after ten years of disease duration.

REFERENCES:

Disclosure of Interests: None declared


Table 1. Patient demographics, follow-up time and number of deaths in the total Systemic Lupus Erythematosus (SLE) cohort and in incident SLE patients.

<table>
<thead>
<tr>
<th>Total SLE cohort</th>
<th>Incident SLE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Female</td>
</tr>
<tr>
<td>n = 1298</td>
<td>n = 577</td>
</tr>
<tr>
<td>Of European descent, n (%)</td>
<td>1140 (88)</td>
</tr>
<tr>
<td>Juvenile onset*, n (%)</td>
<td>93 (7)</td>
</tr>
<tr>
<td>LN†, n (%)</td>
<td>470 (36)</td>
</tr>
<tr>
<td>Cumulative ACR criteria, µ (SD)</td>
<td>5.4 (1.2)</td>
</tr>
<tr>
<td>Follow-up years, total</td>
<td>19252</td>
</tr>
<tr>
<td>Deaths, n (%)</td>
<td>282 (23)</td>
</tr>
<tr>
<td>Age at diagnosis, years μ (SD)</td>
<td>35.5 (15.7)</td>
</tr>
<tr>
<td>Disease duration at death, years μ (SD)</td>
<td>20.4 (12.5)</td>
</tr>
</tbody>
</table>

POS1417 INTEROSSEOUS TENDON INFLAMMATION IN THE HANDS: A NOVEL FEATURE OF DEVELOPING RHEUMATOID ARTHRITIS? RESULTS FROM A LARGE MRI STUDY IN CLINICALLY SUSPECT ARTHRALGIA

B. Van Dijk1, H. W. Van Steenbergen1, M. Reijnierse2, S. Khidir1, L. J. Wisse3, M. C. Derутер4, A. Van der Helm-van Mil5, 6, 7, Leiden University Medical Centre (LUMC), Rheumatology, Leiden, Netherlands; 7Leiden University Medical Centre (LUMC), Radiology, Leiden, Netherlands; 6Leiden University Medical Centre (LUMC), Anatomy & Embryology, Leiden, Netherlands

Background: Inflammation around the tendons of the hand interosseous muscles (interosseous tendon inflammation; ITI) on MRI was recently reported in rheumatoid arthritis (RA) patients and in ACPA-positive individuals with musculoskeletal symptoms. We therefore hypothesized that ITI is an early RA-feature that precedes clinical arthritis.

Objectives: To examine this we assessed the frequency of ITI in clinically suspected arthralgia (CSA) patients and compared this to the frequency in the general population. Additionally we investigated the relation between ITI and other locally inflamed tissues (synovitis/tenosynovitis/ostitis) in MCP-joins of CSA patients as well as the association with future arthritis development.

Methods: 867 consecutive patients presenting with CSA and 193 symptom-free controls from the general population underwent contrast-enhanced hand-MRI. MRIs were evaluated for ITI and for synovitis/tenosynovitis/ostitis, using the rheumatoid arthritis MRI scoring system (RAMRIS). CSA patients were followed for clinical arthritis development (median follow-up 25 months). Logistic and Cox-regression were used. ACPA-stratification was performed. To gain a better understanding of the anatomical relationships, 3D MRI-reconstruction of the interosseus and lumbrical muscles and tendons was performed in a patient with ITI.

Results: At presentation, 10% of CSA patients had ITI, compared to 1% of symptom-free controls (p<0.001). ITI was more frequent in ACPA-positive than ACPA-negative CSA (27% versus 7%; p<0.001). 72% of patients with ITI also had synovitis and/or tenosynovitis at the MCPs (37% synovitis; 7% tenosynovitis; 27% both synovitis and tenosynovitis). Also in multivariable analyses, adjusted for simultaneous presence of synovitis/tenosynovitis/ostitis, ITI was more likely if synovitis (OR 2.2 (95%CI 1.2-4.2) or tenosynovitis (9.7 (5.5-170)) was present at MCPs. The 3D MRI-reconstruction indicated that ITI is continuous with MCP flexor tenosynovitis (Figure 1). CSA patients with ITI developed arthritis...