75 FCGRIIA POLYMORPHISMS ARE ASSOCIATED WITH LUPUS NEPHRITIS IN MEXICAN PATIENTS

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10.1136/lupus-2019-lsm.75

Background Systemic Lupus erythematosus (SLE) is an autoimmune disease mediated by the deposit of immune complexes of autoantibodies which are potentially inflammatory since they can activate the innate immune response through binding to FcgR. The SLE prevalence is 80 per 1 00 000 inhabitants, this prevalence is similar in other population groups, however, the presence of Lupus Nephritis is particularly high in Mexicans (60% of renal disease compared to 12% in Caucasian population), this data is relevant since the diversity of susceptibility genes that have been identified in Mexican patients are from Caucasian origin, despite this the lupus phenotype in Mexican Mestizo is different to any other population. The most important thing to consider is the impact that lupus nephritis causes in health status and quality of life of patients. In this study, the aim was to investigate whether the distribution of the FcgRIIa polymorphisms determines susceptibility to lupus nephritis, the main clinical manifestation in mestizo Mexican patient.

Methods A total of 111 patients that fulfilled the American College of Rheumatology (ACR) classification criteria for SLE and 102 healthy volunteers have been included in this study.

FCGR2A genotypes were determined by polymerase chain reaction-based allotyping methods with allele-specific probes; the clinical features were obtained from patients official medical records.

Results About polymorphism, when different groups of patients were compared: SLE with and without renal activity (defined as ACR criteria) there were found statistical differences between the groups, the genotype RR-131 (p=0.04, OR=3.61) was increased in patients with renal activity or history of this clinical manifestation. Additionally, it was found that the patients classified by biopsy with proliferative membrane glomerulonephritis, were mostly the patients with the GG genotype (60%).

Conclusions The results demonstrate that FcgRIIa polymorphism is associated with susceptibility to lupus nephritis in Mexican patients. The hypothesis for this association could be related to the role of this receptor in the clearance of immune complexes, mainly by innate immune cells like neutrophils which have been identified in biopsies of these patients. Additionally, the activation of this receptor by the engage with immune complexes activate different immune lineage cells which function is affected by the presence of polymorphisms. Functional studies are necessary to determine how this polymorphism affects the effector activity of these cells and the consequences in renal tissue.

Funding Source(s): None

Abstract 75 Table 1 FcgIIa genotypes in SLE patients.

	LEG+act renal	*LEG sin act. Renal*			
	n=77	*n=34*	*pC*	*OR*	*IC95%*
Genotipos	*n (frecuencia)*	*n (frecuencia)*			
R131/R131	25 (0.32)	4 (0.12)	**0.04**	**3.61**	**1.15–11.35**
R131/H131	41 (0.53)	19 (0.56)	NS		
H131/H131	11 (0.15)	11 (0.32)	**0.05**	**0.35**	**0.13–0.91**

Comparison of FcgIIa genotypes among patients who have presented renal activity compared with patients who have not presented renal activity.

76 RISK OF HIP FRACTURE AMONG PATIENTS WITH NEWLY DIAGNOSIS OF SYSTEMIC LUPUS ERYTHEMATOSUS. A POPULATION-BASED STUDY

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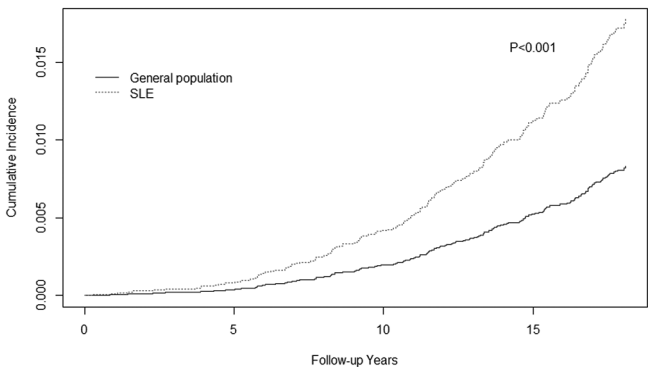
10.1136/lupus-2019-lsm.76

Background Hip fractures (HP) have serious long-term effects, including 1 year mortality rate of up to 30% and poor functional recovery. Studies on the risk of HP fracture in SLE are limited due to the use of selected samples, failure to adjust for time-varying treatments and confounders. Our objective was to assess the risk of HP in patients with newly diagnosed SLE compared to the general population.

Methods Using physician billing data and a previously validated SLE case definition, we assembled an incident cohort of all patients with SLE who received care between January 1997 and March 2015 in the province of British Columbia, Canada. The main outcome was the first ever HP during follow-up. HP (ICD-9-CM codes 820.0, 820.2; ICD-10-CM codes S72.0, S72.1, S72.2) were identified using hospitalization data. We excluded patients with previous HP, pathological fractures or Pagets disease before the index date (SLE diagnosis). Non-SLE controls were randomly selected from the general population and matched (1:5) to SLE patients on birth year, sex, and index year. First, we used Kaplan-Meier estimates and log-rank test to compare time to first hip fracture between SLE and non-SLE controls. Multivariable analyses adjusting for baseline covariates known as potential risk factors for HP were done. In addition, marginal structure Cox models were then used to estimate the impact of having SLE on the risk of hip fracture, adjusting for time-dependent covariates, including glucocorticoid use, number of outpatient, inpatient and rheumatologist visits.

Results We identified 5047 patients with a new diagnosis of SLE and 25 235 non-SLE controls (mean age 40 years; 86% females for each cohort), yielding 73 and 273 HP, respectively. The incidence rate for HP for SLE and non-SLE were 0.93



Abstract 76 Figure 1 Cumulative incidence of hip fracture