

corticosteroid use have been described as cardiovascular risk factors but there is controversy surrounding antimalarials as a protective factor. Our objective is to analyze associated factors with the presentation of cardiovascular events such as high blood pressure (HBP), acute myocardial infarction (AMI), stroke and thromboembolic disease (TED)

Methods A cross-sectional study was done with 1175 records of patients with SLE that fulfilled either ACR 1997 or SLICC 2012 classification criteria that had been in medical care between 2015 and 2017 in a rheumatology specialized institution in six cities of Colombia. We describe sociodemographic, clinical and immunoserological characteristics and a comparative analysis was done with chi2 and Mann Whitney's U with a combined outcome of cardiovascular disease obtaining an OR of crude associations that were adjusted for several variables

Results Women represented 91% of the cohort with a median age of 44 years (IQR 21) and 8 years of disease duration (IQR 11) with a mean age at diagnosis of 32 years, 5,4% were active smokers and 15% had smoked in the past. Cardiovascular events were found in 32% of the patients with HT as the most common. Other cardiovascular outcomes such as stroke, TED and AMI were infrequent with a prevalence of 3.3%, 2.9% and 2% respectively. In bivariate analysis, age >36 years and corticosteroid use were associated with a significantly higher risk, while the use of antimalarials for more than 6 years was found to protect for cardiovascular risk with no difference between chloroquine and hydroxychloroquine use (table 1)

Conclusions Our cohort is comparable with other SLE cohorts regarding the frequency of cardiovascular events. Up to 32% of the described population presented a cardiovascular event and arterial hypertension was the most frequent. Continuous use of antimalarials for more than 6 years has a protective effect against cardiovascular events such as arterial hypertension, stroke, acute myocardial infarction and thromboembolic

disease. The benefit seen only after 6 years of continuous use probably reflects the need of a long period of time before some of the potential benefits of these medications are seen

Funding Source(s): None

299

INCREASED MORTALITY AMONG PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS AFTER HYDROXYCHLOROQUINE DISCONTINUATION

¹Antonio Avina-Zubieta*, ²April Jorge, ³Mary A DeVera, ⁴Na Lu, ⁴John Esdaile, ⁵Hyon Choi. ¹Arthritis Research Canada, University of British Columbia; ²Division of Rheumatology, Allergy, and Immunology, Massachusetts General Hospital; ³University of British Columbia; ⁴Arthritis Research Canada; ⁵Massachusetts General Hospital

10.1136/lupus-2019-ism.299

Background Hydroxychloroquine (HCQ) is near-universally recommended for patients with SLE. Use of this medication has previously been associated with a substantial survival benefit among SLE patients. We aimed to determine the potential temporal association between HCQ discontinuation and all-cause and cardiovascular disease (CVD) mortality.

Methods We conducted a population-based case-control study using an administrative health database including the entire population in the province of British Columbia, Canada (>5 million individuals). We identified cases with SLE who died and each case was matched on age, sex, and SLE disease duration with living controls with SLE. We used conditional logistic regression to assess the association between current use of HCQ or recent discontinuation of HCQ and the risk of all-cause and cause-specific mortality relative to remote HCQ users. Remote users were defined by a duration greater than 365 days between the last HCQ prescription and the index date (i.e., death date). Recent users had a duration less than 365 days since the last HCQ prescription and index date. Current users had active HCQ prescriptions spanning

Abstract 299 Table 1 Risk of Death with Current Usage, Non-Usage, and Recent Discontinuation compared with Remote Usage of Hydroxychloroquine among patients with Systemic Lupus Erythematosus

All-Cause Mortality	Cases, N	Controls, N	Crude Odds Ratio (95% CI)	Adjusted Odds Ratio (95% CI)
Remote HCQ Users	72	106	1.0 (reference)	1.0 (reference)
Recent HCQ Discontinuers	65	34	3.03 (1.77-5.17)	3.78 (2.07-6.91)
Current HCQ users	32	156	0.30 (0.18-0.49)	0.35 (0.20-0.59)
HCQ Non-users	121	206	0.83 (0.55-1.24)	0.93 (0.59-1.44)
Cardiovascular Disease Mortality				
Remote HCQ Users	17	28	1.0 (reference)	1.0 (reference)
Recent HCQ Discontinuers	17	11	2.57 (0.96-6.92)	4.63 (1.31-16.42)
Current HCQ users	9	44	0.32 (0.12-0.87)	0.37 (0.11-1.27)
HCQ Non-users	40	59	1.11 (0.51-2.44)	1.15 (0.45-2.99)
Other Cause Mortality				
Remote HCQ Users	35	52	1.0 (reference)	1.0 (reference)
Recent HCQ Discontinuers	33	18	3.17 (1.45-6.93)	3.90 (1.56-9.75)
Current HCQ users	13	72	0.28 (0.14-0.58)	0.32 (0.14-0.71)
HCQ Non-users	57	95	0.89 (0.49-1.64)	1.18 (0.29-2.37)

the index date. Fully adjusted models included chronic kidney disease, Charlson comorbidity index, glucocorticoids, and cardiovascular medication use assessed at the time of SLE diagnosis.

Results We identified 290 SLE cases who died and 502 matched controls among 792 individuals with SLE. The mean age at index date was 65.6 years for cases and 64.7 years for controls. The majority were female (87.9% of cases and 91.4% of controls). The mean SLE disease duration was 5.3 years for both groups. Adjusted odd ratios (ORs) for all-cause mortality relative to the remote users were 0.35 (95% CI: 0.20, 0.59) for current users and 3.78 (95% CI: 2.07, 6.91) for subjects who recently discontinued HCQ (table 1). HCQ non-users had the same risk of death as remote users (OR 0.93 [95% CI: 0.59, 1.44]). Similar trends were seen for the risk of mortality due to CVD.

Conclusions In this study, we found a nearly four-fold increased risk of death associated with recent HCQ discontinuation and a substantially increased risk of CVD death. This could be partially explained by a direct protective effect of HCQ that is rapidly lost following discontinuation. We also demonstrated a 65% reduced risk of death among current HCQ users compared with remote users. By leveraging remote

users as the comparison group, we reduced the potential for confounding by indication.

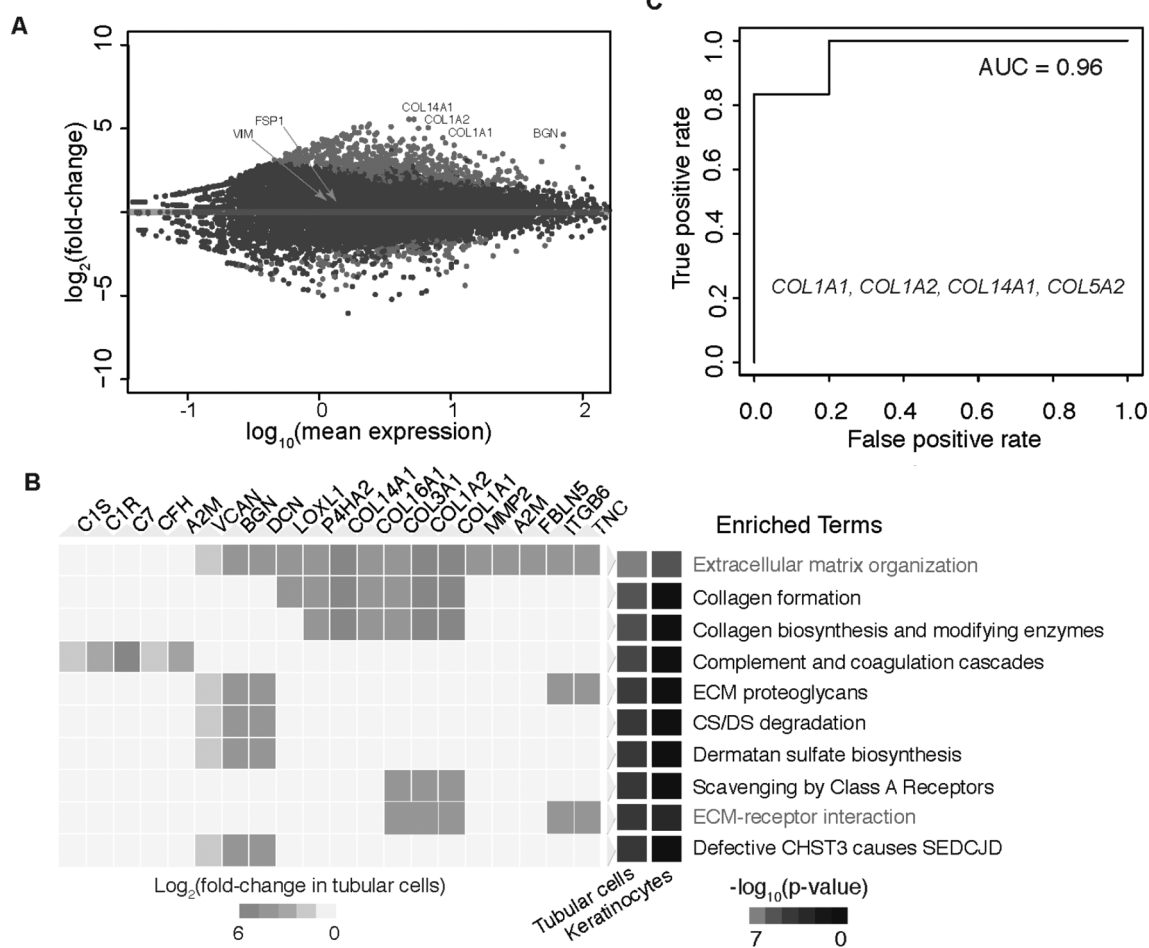
Funding Source(s): CIHR (Grants MOP 125960 and THC 135235).

300 INSIGHTS FROM SINGLE-CELL RNA SEQUENCING OF SKIN AND KIDNEY IN LUPUS NEPHRITIS

¹Evan Der*, ²Hemant Suryawanshi, ²Pavel Morozov, ²Manjunath Kustagi, ³Beatrice Gailav, ³Saritha Ranabothu, ⁴Peter Izmirly, ⁴Robert Clancy, ⁴Michael Belmont, ⁵Mordecai Koenigsberg, ¹Michele Mokrzycki, ⁵Helen Rominiecki, ⁵Jay Graham, ⁵Juan Rocca, ⁴Nicole Bornkamp, ¹Nicole Jordan, ¹Emma Schulte, ⁴Ming Wu, ³James Pullman, ⁶Kamil Slowikowski, ⁶Soumya Raychaudhuri, ⁷Joel Guthridge, ⁷Judith A James, ⁴Jill Buyon, ²Thomas Tuschl, ⁸Chaim Putterman. ¹Albert Einstein College of Medicine; ²The Rockefeller University; ³Children's Hospital at Montefiore; ⁴NYU School of Medicine; ⁵Montefiore Medical Center; ⁶Broad Institute; ⁷Oklahoma Medical Research Foundation; ⁸Albert Einstein College of Medicine and Montefiore Medical Center

10.1136/lupus-2019-lsm.300

Background Classification and treatment decisions in lupus nephritis (LN) are largely based on renal histology. Single-cell



Abstract 300 Figure 1 A fibrotic gene signature as a potential prognostic marker for patients non-responsive to treatment. A) MA plot of differential expression analysis performed between tubular cells of patients responsive (n=13) or non-responsive to treatment (n=5). Significantly differentially expressed genes are colored in red. B) Pathway enrichment analysis of genes identified as upregulated in patients non-responsive to treatment. -Log10(p-value) of each pathway is shown for both keratinocytes and tubular cells colored from least significant (black) to most significant (red). Log2 fold change in gene expression between patients non-responsive to treatment compared with patients responsive to treatment in each pathway is indicated for tubular cells from smallest (grey) to highest (orange). C) Receiver operating characteristic curve of the logistic regression equation of differentially expressed fibrotic genes, COL1A2, COL1A1, COL14A1, COL5A2, with area under the curve (AUC) indicated.