

treatment withdrawal is contemplated. Until improved blood or urine biomarkers have been developed, repeat biopsy will remain a useful tool for the clinic.

## REFERENCES

1. Malvar A, Alberton V, Lococo B, et al. Kidney biopsy-based management of maintenance immunosuppression is safe and may ameliorate flare rate in lupus nephritis. *Kidney Int.* 2019
2. De Rosa M, Azzato F, Toblli JE, et al. A prospective observational cohort study highlights kidney biopsy findings of lupus nephritis patients in remission who flare following withdrawal of maintenance therapy. *Kidney Int.* 2018;**94**:788–794.
3. Arriens C, Chen S, Karp DR, et al. Prognostic significance of repeat biopsy in lupus nephritis: Histopathologic worsening and a short time between biopsies is associated with significantly increased risk for end stage renal disease and death. *Clin Immunol.* 2017;**185**:3–9.

## Oral presentations

### 01 HYDROXYCHLOROQUINE BLOOD LEVELS AND RISK OF THROMBOTIC EVENTS IN SYSTEMIC LUPUS ERYTHEMATOSUS

Maximilian F Konig, Jessica Li, Michelle Petri. *Medicine, Rheumatology, Johns Hopkins University School of Medicine, Baltimore, USA*

10.1136/lupus-2020-eurolupus.15

**Background** Hydroxychloroquine (HCQ) has a primary role in the treatment of systemic lupus erythematosus (SLE). Beyond its pleiotropic immunomodulatory effects on TLR and type I interferon signaling, HCQ use has been found to be protective for thrombosis in SLE. Optimal dosing of HCQ in SLE is unknown. The longitudinal measurement of HCQ blood levels may provide an opportunity to individualize weight-based dosing strategies and reduce risk of toxicity. We examined the association of HCQ blood levels with thrombotic events in a longitudinal SLE cohort.

**Methods** 812 SLE patients with HCQ level measured prior to the thrombosis were included: 93% female, 43% African-American, 46% Caucasian. HCQ blood levels were quantified by liquid chromatography-tandem mass spectrometry. Mean HCQ blood levels ( $\pm$  SD) over all cohort visits prior to occurrence of thrombosis were calculated for each patient. Thromboses were defined as venous (DVT/PE or other venous) or arterial thrombosis (stroke, myocardial infarction, digital gangrene or other arterial).

**Results** Thrombosis had occurred during prospective follow up in 44 patients (5.4%), venous in 3.0% and arterial in 2.5%. Lupus anticoagulant was strongly associated with a history of any thrombosis (OR 3.25,  $P < 0.0001$ ), venous thrombosis (OR 3.53,  $P < 0.0001$ ), and arterial thrombosis (OR 3.08,  $P < 0.0001$ ). A prospective analysis shows that for any thrombosis and for venous thrombosis, the HCQ blood level was significantly lower. Higher prescribed doses of HCQ (as opposed to HCQ blood levels) were also associated with decreased odds of any thrombosis and venous thrombosis in a separate cross-sectional analysis (OR 0.88,  $P = 0.04$  and OR 0.83,  $P = 0.009$ , respectively for each 1 mg/kg increase in prescribed HCQ).

**Conclusions** HCQ blood levels are inversely associated with risk of any thrombosis and of venous thrombosis in patients with SLE in a prospective analysis. Reduction of HCQ dosing, as suggested by the American Academy of Ophthalmologists, could reduce or eliminate the benefit of hydroxychloroquine to prevent thrombosis.

**Acknowledgements** The Hopkins Lupus Cohort is supported by NIH Grant RO1 AR 69572.

### 02 EFFECT OF TREATMENT ON ANTIPHOSPHOLIPID ANTIBODIES IN SLE

<sup>1</sup>Michelle Petri, <sup>2</sup>Laurence S Magder, <sup>1</sup>Daniel W Goldman. <sup>1</sup>Medicine, Rheumatology, Johns Hopkins University School of Medicine, Baltimore; <sup>2</sup>Dept. Epidemiology, University of Maryland, School of Medicine, Baltimore, USA

10.1136/lupus-2020-eurolupus.16

**Background** Unlike primary antiphospholipid syndrome patients, most SLE patients with antiphospholipid antibodies are on one or more treatments for their SLE that might affect levels of their antiphospholipid antibodies. We examined the effect of prednisone and hydroxychloroquine on antiphospholipid antibodies in an SLE longitudinal cohort.

**Methods** 943 SLE patients, who were tested for each anticardiolipin isotype (aCL IgG, IgM and IgA; INOVA) and lupus anticoagulant (LAC; dRVVT with further confirmatory testing) for at least 10 quarterly clinic visits, were included. We compared visits positive for antiphospholipid antibodies (aCL > 20 and aCL > 40; dRVVT > 45) to visits negative for antiphospholipid antibodies, with respect to treatment, using conditional logistic regression and conditioning on the patient.

**Results** Prednisone treatment significantly reduced the levels of aCL IgG isotypes (aCL IgG > 40 and some prednisone but less than 10 mg/day, OR 0.61 95% CI 0.46–0.80  $p = 0.0004$ ), but not aCL IgM, IgA, or LAC. Hydroxychloroquine treatment significantly reduced aCL IgG (aCL IgG > 40, OR 0.35 95% CI 0.22–0.55  $p < 0.0001$ ), aCL IgM (aCL IgM > 40, OR 0.56 95% CI 0.36–0.87  $p = 0.010$ ) and LAC (OR 0.71 95% CI 0.58–0.86  $p = 0.007$ ) but not aCL IgA.

**Conclusions** Prednisone does not reduce IgM or IgA isotypes of anticardiolipin, or LAC. These results explain why prednisone does not reduce thrombosis in SLE. Hydroxychloroquine, on the other hand, significantly reduces all antiphospholipid types except for the IgA isotype of anticardiolipin. This may explain why IgA isotypes are more common in SLE. It may also explain why hydroxychloroquine leads to only a 50% reduction in thrombosis, as IgA isotypes do confer some risk of thrombosis.

**Acknowledgements** The Hopkins Lupus Cohort is supported by NIH Grant RO1 AR 69572.

### 03 CHANGES IN GUT MICROBIOTA AFTER SYMBIOTIC SUPPLEMENTATION IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: A RANDOMISED, DOUBLE-BLIND, PLACEBO-CONTROLLED TRIAL

<sup>1,2,3</sup>Alvina Widhani, <sup>1,2</sup>Samsuridjal Djauzi, <sup>4</sup>Fransiscus D Suyatna, <sup>5</sup>Beti Ernawati Dewi, <sup>4</sup>Melva Louisa, <sup>5</sup>Andi Yasmon, <sup>6</sup>Susan Rahayu. <sup>1</sup>Allergy and Clinical Immunology Division, Dept. of Internal Medicine, Faculty of Medicine, Universitas Indonesia, Jakarta; <sup>2</sup>Dr. Cipto Mangunkusumo National General Hospital, Jakarta; <sup>3</sup>Doctoral Program in Biomedical Science, Faculty of Medicine, Universitas Indonesia, Jakarta; <sup>4</sup>Dept. of Pharmacology and Therapeutic, Faculty of Medicine, Universitas Indonesia, Jakarta; <sup>5</sup>Dept. of Microbiology, Faculty of Medicine, Universitas Indonesia, Jakarta; <sup>6</sup>Molecular Biology Unit, Integrated Laboratory, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

10.1136/lupus-2020-eurolupus.17