and partial remission as more than 50% reduction in proteinuria to sub-nephrotic levels and serum creatinine within 10% from baseline.

Results: We have included 55 patients (80% women) with biopsy-proven LN (age at biopsy 38±13 years). On renal histology, one patient was class I LN, two patients were class II, 7 were class III, 5 were class III+V, 23 were class IV, two were class IV+V, 11 class VI and 4 had other features. Median baseline NLR was 3.64 (IQR: 1.85, 6.93), PLR was 167 (IQR: 116, 300) and MPV was 10.0 (IQR: 9.2, 10.8). There were no differences in any of the three parameters between different classess of LN (all $p>0.05$). Eighteen patients achieved complete remission (CR), 16 partial remission and 21 did not achieve remission. None progressed to ESRD and two patients died, both from sepsis. There no differences in NLR, PLR and MPV at the time of biopsy between group which achieved CR/PR vs. no remission (all $p>0.05$; Figure 1). We constructed a thorough multivariate regression model which showed that neither NLR, PLR or MPV are predictors of renal remission in LN.

Conclusion: NLR, PLR and MPV are not useful prognostic markers in predicting renal remission in LN.

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AB0350

MATERNAL AND FETAL OUTCOME IN MEXICAN WOMEN WITH PRIMARY ANTIPHOSPHOLIPID SYNDROME, SECONDARY ANTIPHOSPHOLIPID SYNDROME AND SYSTEMIC LUPUS ERYTHEMATOSUS WITH ANTIPHOSPHOLIPID ANTIBODIES

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Background: Primary antiphospholipid syndrome (APS) is associated with a higher frequency of maternal-fetal adverse outcomes, with increased risk when APS is associated with systemic lupus erythematosus (SLE).

Objectives: We compared maternal-fetal complications in patients with primary APS, APS associated with SLE and SLE with anti-phospholipid antibodies (aPLs).

Methods: Observational, analytical, retrospective, cross-sectional, comparative study, between 2009-2019. Three groups were compared: primary APS, APS associated with SLE and SLE with anti-phospholipid antibodies (aPLs).

Results: We include 179 pregnant women, 31.8% with primary APS, 35.8% with APS associated with SLE, and 32.4% with SLE and aPLs. A history of a higher frequency of abortions was found in APS associated with SLE (62.5%, $p<0.001$) and thrombosis (61.9%, $p<0.001$) and aCL IgG + in primary APS (80.7%, $p=0.011$). More frequent use during pregnancy of antimalarials is applied in associated APS (81.30%, $p<0.001$) and aspirin / heparin in primary APS (96.5% / 94.7%, $p<0.001$). Abortions were observed more frequently in APS associated with SLE (23.80%, $p=0.021$) and preeclampsia / HELLP in primary APS (31.6% and 15.8%, $p=0.047$) who were also less frequently the use of antimalarial (24% vs. 81.3% vs. 72.4%, $p<0.001$) compared to the other groups. Table 1

Conclusion: Patients with APS and SLE have an increased risk of miscarriage whereas those with primary APS have an increased risk for preeclampsia / HELLP. The use of antimalarials could reduce the risk of hypertensive disorders of pregnancy.

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Table 1. Comparison between groups of adverse maternal-fetal outcome.

	Secondary antiphospholipid syndrome (n=64)	Primary antiphospholipid syndrome (n=57)	Systemic lupus erythematosus with antiphospholipid antibodies (n=58)	P
Fetal complications	48 (76.20%)	43 (75.40%)	31 (56.40%)	0.034
Growth restriction	9 (14.30%)	8 (14%)	7 (12.50%)	0.955
Oligohydramnios	6 (9.50%)	5 (8.80%)	5 (8.90%)	0.989
Prematurity	23 (52.30%)	25 (56.80%)	19 (42.20%)	0.37
Abortion	15 (23.80%)	3 (5.40%)	9 (16.10%)	0.021
Stillbirth	7 (11.10%)	10 (17.90%)	5 (9.10%)	0.343
Low birth weight	20 (52.60%)	21 (48.80%)	15 (37.50%)	0.373
Malformations	5 (8.10%)	0 (0%)	0 (0%)	0.01
Maternal complications	40 (63.50%)	25 (43.90%)	21 (36.80%)	0.01
Pre-eclampsia.	15 (23.80%)	18 (31.60%)	7 (12.30%)	0.046
HELLP	4 (6.30%)	9 (15.8%)	2 (3.50%)	0.047
Premature rupture of membranes	7 (11.10%)	5 (8.8%)	4 (7.10%)	0.75
Maternal death	1 (1.60%)	0 (0%)	1 (1.80%)	0.616

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AB0351

PROGRESSION TO SEVERE LUPUS NEPHRITIS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: UPDATE FROM A COLOMBIAN COHORT

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Background: Continuous monitoring of patients with Systemic lupus erythematosus (SLE) provides relevant information

Objectives: To update the analysis of clinical and immunological characteristics associated with time to severe renal involvement in patients with Systemic Lupus Erythematosus in a Colombian cohort followed from January 2015 to October 2020

Methods: A retrospective follow-up study based on clinical records. Patients with SLE diagnosis fulfilled either 1987 American College of Rheumatology Classification Criteria for SLE or 2011 Systemic Lupus International Collaborating Clinics (SLICC) classification criteria for SLE. We included patients with the diagnosis of lupus nephritis according to Wallace and Dubois criteria. Patients who did not have at least two follow-up measurements or had a cause of nephritis other than lupus were excluded. The primary outcome was defined as the time from diagnosis to severe renal involvement defined as creatinine clearance ≤ 50 ml/min, 24-hour proteinuria ≥ 3.5 grams or end-stage renal disease. Updated age and sex-adjusted survival functions and Hazard ratios (HR) with 95% confidence intervals and p-values were estimated using parametric Weibull models for interval-censored data. P values <0.05 were considered statistically significant. Descriptive statistics were previously reported in EULAR 2020 (1)

Results: 548 patients were analyzed: 67 were left-censored as they presented renal involvement at entry, 25 were interval censored as outcome occurred between study visits (19 new events), and 456 were right-censored as involvement was not registered during follow-up. In this cohort update Age and sex-adjusted Hazard Ratios for high blood pressure were HR = 3.1 (95%CI 1.5-6.3; p -value = 0.003) and Anti-RO (per unit increase) HR = 1.003 (95%CI 1.001-1.005; p -value = 0.029). Figure 1 shows the updated age and sex-adjusted survival function

Conclusion: In this cohort update, we found similar clinical and immunological characteristics associated with time to severe renal involvement in SLE patients to those reported in (1). However, continuous follow-up allows us to deepen our understanding of the progression to severe renal involvement in SLE patients

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Vasculitis – large vessel vasculitis

AB0352 ANTICARDIOLIPIN ANTIBODIES AND ACTIVITY OF GIANT CELL ARTERITIS

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Background: Anticardiolipin antibodies (aCL) can be detected in newly diagnosed GCA as reactive antibodies to endothelial lesions. Their prognostic role, as a marker of disease activity, has not been extensively studied in GCA.

Objectives: Our aim was to determine whether aCL IgG might represent laboratory marker of active GCA.

Methods: We included patients with new clinical diagnosis of GCA supported by histology or imaging between September 2011 and July 2019, who completed at least a 48-week follow-up at our secondary/tertiary rheumatology center. Follow up visits with predetermined clinical and laboratory tests, including aCL IgG, were performed 12, 24, 48, and 96 weeks after diagnosis. GCA relapse was defined as worsening or new disease activity after initial remission. Other reasons for the disease-related symptoms, elevated inflammatory markers (C reactive protein or erythrocyte sedimentation rate) had to be excluded. aCL IgG were determined in the patients' sera samples at baseline and at follow up visits, using an in-house solid phase enzyme-linked immunosorbent assay¹. A value above the 99th percentile of the healthy control population was taken as significant.

Results: During the observation period we identified 288 newly diagnosed GCA patients. Two hundred and twelve GCA patients (66.5% females, median (IQR) age 73.9 (67.0–78.7) years) fulfilled the study inclusion criterion, among them 145 patients completed the 96-week follow up visit. At baseline, 129/212 (60.8%) GCA patients had positive aCL IgG. During in total 781 follow up visits, we recorded 77 (9.9%) episodes of active/relapsing GCA (clinical, laboratory or combined in 4 (5.2%), 48 (62.3%), 25 (32.5%) episodes, respectively). aCL IgG were present at 155/781 (19.8%) measurements (at 24/77 episodes of relapsing/active and 131/573 episodes of quiescent GCA). The correlation between active/relapsing GCA and aCL IgG positivity was only weakly positive (r coefficient=0.094; $p=0.015$).

Conclusion: The role of aCL IgG as a biomarker for GCA activity seems to be rather limited.

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AB0353 ADRENAL INSUFFICIENCY AFTER GLUCOCORTICOID TREATMENT OF GIANT CELL ARTERITIS

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Background: Adrenal insufficiency is frequently neglected and underappreciated, potentially severe complication of systemic glucocorticoid therapy.

Objectives: We aimed to evaluate the prevalence of glucocorticoid induced adrenal insufficiency in giant cell arteritis (GCA).

Methods: We analysed adrenal function data in a cohort of GCA patients diagnosed between July 2014 and July 2019, in whom discontinuation of methylprednisolone therapy was planned. Adrenal function was tested by Corticotropin stimulation test (CST). To perform the CST, methylprednisolone was substituted with hydrocortisone (20mg qd in three divided doses) for one to four weeks before the test. Adrenal insufficiency was defined as cortisol level <450 nmol/l measured 30 minutes after the corticotropin injection; additionally, the result of the CST was defined as borderline when the cortisol level 30 minutes after corticotropin injection was between 450 nmol/l and 500 nmol/l.

Results: Adrenal function was tested in 74/215 GCA patients before definite methylprednisolone withdrawal (after a median 13.5 (12.9 – 22.4) months of glucocorticoid therapy). The mean (SD) methylprednisolone dose, prior to substitution with hydrocortisone and subsequent CST, was 3.1 (1.6) mg. Adrenal insufficiency was detected in 36/74 patients (48.6%); additionally, 10/74 patients (13.5%) had a borderline CST result. Seventeen patients with either adrenal insufficiency or borderline CST result, had a repeated CST after median (IQR) 11.6 (8.9; 12.6) months. Adrenal insufficiency persisted in 11/17 (64.7%) patients, and 1/17 patients had a borderline CST. A third CST was performed in 4/12 patients with abnormal second CST after median (IQR) 8.3 (6.9; 10.6) months. Adrenal function recovered in one patient, while the adrenal insufficiency persisted in the remaining 3 patients.

Conclusion: Adrenal insufficiency is a common and potentially long-lasting glucocorticoid induced adverse event in GCA patients.

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AB0354 FDG-PET-DETECTED LARGE VESSEL VASCULITIS DOES NOT PREDICT DISEASE OUTCOME IN PATIENTS WITH GIANT CELL ARTERITIS AND POLYMYALGIA RHEUMATICA

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Background: Giant cell arteritis (GCA) and polymyalgia rheumatica (PMR) are tightly associated inflammatory conditions of the elderly [1]. Both disorders can exhibit an increased articular and vascular uptake of 18-fluorodeoxyglucose (18-FDG) at positron emission tomography (PET)/computed tomography (CT) scan [2].

Objectives: This study evaluated if large-vessel vasculitis (LVV) detected by PET/CT in patients with PMR and/or cranial GCA had a negative prognostic value.

Methods: 108 patients (35 men and 73 women) with a median age of 74 years (range 50-92 years) were prospectively enrolled in our centre over 4 years. PMR was diagnosed by Bird et al. criteria and GCA by the ACR criteria. Six patients died shortly after the first visit (V_0) and six were lost at follow-up. Of the remaining 96 patients, 77 were classified as PMR, 6 as GCA and 13 were affected by both diseases.

At V_0 , patients underwent a clinical, laboratory and PET/CT evaluation, and were stratified according to the presence or not of LVV. Follow-up visits