complex. To reduce non-specific binding buffer was added after this incubation prior to centrifugation. The sensitivity of the assay was <1.6 ng/ml. The recovery was approximately 100% for 25(OH)D3. Within and between batch precision was <12% and<11%, respectively.

Results In the cohort of SLE patients low blood levels of 25 (OH)D3 were observed. A positive relationship between 25 (OH)D3 blood levels and complement levels was observed, namely low 25(OH)D3 levels were positively correlated with low complement levels. An inverse relationship was observed between 25(OH)D3 levels and disease activity, namely low 25 (OH)D3 levels were related with high disease activity.

Conclusions Vitamin D is a hormone directly related to the regulation of the musculoskeletal system. Vitamin D also has extraskeletal actions. The immunomodulatory action of vitamin D appears to be a key action of the hormone. In the work presented herein low blood levels of vitamin D were observed in SLE patients which were positively related to complement levels and inversely related to disease activity.

Poster session 4: APS, family planning, fertility, pregnancy and neonatal care

PS4:66 PROLONGED EXPOSURE TO ANTIPHOSPHOLIPID ANTIBODIES IS ASSOCIATED WITH ENDOTHELIAL DYSFUNCTION IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

[1]C Cho, [2]Y Park, [1]I Baek, [3]W Kim. *[1]Yeouido St. Mary's Hospital, Seoul, South Korea; [2]St. Vincent Hospital, Suwon, South Korea; [3]Seoul St. Mary's Hospital, Seoul, South Korea*

10.1136/lupus-2018-abstract.112

Objective Antiphospholipid syndrome has been shown to be associated with increased cardiovascular mortality, but the role of antiphospholipid antibodies (aPL) on endothelial dysfunction remains elusive. We investigated the association between endothelial dysfunction and aPL in systemic lupus erythematosus (SLE) patients.

Methods 188 SLE patients and 62 controls were enrolled. Endothelial function was measured by flow-mediated dilatation (FMD). Cardiovascular risk factors were assessed and quarterly measurement of anti-cardiolipin (aCL) and anti-β2 glycoprotein I Ab were used to calculate time-integrated values throughout disease duration. Circulating endothelial progenitor cell (EPC), defined by CD34+/KDR +mononuclear cells, was quantified by flow cytometry.

Results Median FMD was significantly lower in SLE patient than in controls (6.9 versus 9.3%, p<0.001). In univariate analysis, older age, hypertension and persistent positive lupus anticoagulant (LAC) were associated with decreased FMD in SLE patients (p=0.034, p=0.011, p=0.020). Time-integrated aCL value (TI-aCL), but not a single value, was correlated with decreased FMD (p=0.003). Multivariate analysis showed that hypertension and TI-aCL were independent factors for decreased FMD (p=0.011, p=0.011); addition of positive LAC increased the adjusted probability of decreased FMD (p=0.003). FMD was correlated with EPC number (ρ=0.342, p=0.005) and TI-aCL was also an independent factor of reduced EPC after multiple adjustment (p=0.024). The predicted probability of endothelial dysfunction at median EPC level was higher in group with high TI-aCL than in group with low TI-aCL (p=0.004).

Conclusion Cumulative burden of aPL was closely associated with endothelial dysfunction in SLE patients, which was mediated in part by reduction of EPC.

PS4:67 LOSING ANTIPHOSPHOLIPID ANTIBODY POSITIVITY POST THROMBOSIS IN SECONDARY ANTIPHOSPHOLIPID SYNDROME

[1]M Khawaja, [2]L Magder, [1]M Petri. *[1]Johns Hopkins University School of Medicine, Dept of Rheumatology, Baltimore, USA; [2]University of Maryland School of Medicine, Dept of Epidemiology and Public Health, Baltimore, USA*

10.1136/lupus-2018-abstract.113

Objectives Loss of positivity of antiphospholipid antibodies has been observed in clinical practice post thrombosis with secondary APS. Our study aimed to define the frequency of this loss.

Methods In this prospective study, the inclusion criteria comprised of SLE patients having at least two positive aPL markers in 3 years prior to the thrombosis. Patients with at least two post-thrombosis visits were included. Positive antiphospholipid markers comprised of RVVT>45, aCL IgG ≥20, aCL IgM ≥20 and aCL IgA ≥20. Loss of aPL was defined as being negative for these markers for all visits after the thrombosis, excluding the first visit post thrombosis. Percentages of loss of antiphospholipid markers after thrombosis was calculated. Further analyses were done for different types of thromboses (arterial vs venous). There were 17 patients with arterial and 16 patients with venous thromboses.

Results The analysis included the numbers and percentages of patients with loss of aPL after thrombosis, as shown in the table below.

Abstract PS4:67 Table 1

aPL Measure	Proportion (%) with loss of aPL	
Any Thrombosis		
RVVT>45	3/26	12%
aCL IgG>=20	3/10	30%
aCL IgM>=20	3/9	33%
aCL IgA > =20	0/2	0%
Venous Thrombosis		
RVVT>45	2/16	13%
aCL IgG>=20	3/7	43%
aCL IgM>=20	2/5	40%
aCL IgA > =20	0/1	0%
Arterial Thrombosis		
RVVT>45	2/17	12%
aCL IgG>=20	1/9	11%
aCL IgM>=20	1/10	10%
aCL IgA > =20	1/3	33%

Conclusions In secondary APS due to SLE, loss of aPL positivity post thrombotic event was up to 43% for anticardiolipin IgG, IgM and IgA, but only 13% for lupus anticoagulant (measured by RVVT). Loss of lupus anticoagulant positivity appears rare. Our analyses, however, are limited due to the small numbers of prospectively followed events.

PS4:68 **CAROTID AND FEMORAL ATHEROSCLEROSIS IN ANTIPHOSPHOLIPID SYNDROME: EQUIVALENT RISK WITH DIABETES MELLITUS IN A CASE-CONTROL STUDY**

M Tektonidou, E Kravvariti, G Konstantonis, N Tentolouris, PP Sfikakis. *National and Kapodistrian University of Athens – 1st Department of Propaedeutic and Internal Medicine, Athens, Greece*

10.1136/lupus-2018-abstract.114

Background Antiphospholipid syndrome (APS) may carry a worse prognosis for vascular complications when co-existing with subclinical atherosclerosis, however, the association between the two conditions remains ambiguous.

Methods We evaluated ultrasonographic markers of subclinical atherosclerosis in carotid and femoral arteries of 86 patients with thrombotic APS (43 primary APS (PAPS), 43 systemic lupus erythematosus-associated APS [SLE/APS]), 86 patients with diabetes mellitus (DM) and 86 healthy controls, individually matched for age and gender, and investigated their associations with traditional and disease-related factors in APS.

Results Carotid plaques were found in 28% of PAPS, 23% of SLE/APS, and 30% of DM patients versus 9% of controls (p=0.006). Femoral plaques were found in 33% of PAPS, 19% of SLE/APS, 20% of DM, and 9% of controls (p=0.032). Multivariate regression-derived relative risk estimates for atherosclerotic plaques in any location were 2.72 for PAPS, 2.63 for SLE/APS, and 1.98 for DM (p=0.004, 0.009, 0.032 respectively), after adjusting for age, gender, hypertension, dyslipidemia, smoking, BMI, and family history of coronary disease. Among patients with APS, atherosclerotic plaques were associated with the number of traditional CVD risk factors in both PAPS (RR=2.75, p<0.001) and SLE/APS (RR=1.84, p<0.001), and with IgG anti-beta2-glycoprotein I antibodies in SLE/APS.

Conclusions Patients with PAPS and SLE/APS have a nearly 2.5-fold risk of atherosclerotic plaques in carotid and femoral arteries compared to healthy controls, similar to DM patients. Atherosclerotic plaques are associated with the number of traditional risk factors in both APS and SLE/APS, and with IgG anti-beta2-glycoprotein I antibodies in SLE/APS.

Abstract PS4:68 Table 1 Baseline characteristics of study participants and vascular ultrasonagraphic findings by disease group

	APS, n=86		DM	Controls
	PAPS n=43	SLE/APS n=43	n=86	n=86
Baseline characteristics				
Age (mean±SD)	46.4± 12.6	46.4 ± 10.9	46.7± 12.0	46.4± 12.1
Gender (% female)	63	84	73	73
Disease duration [median (Q1, Q3)]	7(2, 15)	11 (4, 19)	14 (6, 21)	
Hypertension (%)	32	26	37	28
Dyslipidemia (%)	23	21	37	15
Smoking (% ever)	56	58	59	42
Smoking (pack years)	14 ± 18	12 ± 19	16 ± 22	7 ± 13
BMI (kg/m2)	29.1± 4.8	26.7 ± 6.2	29.6± 8.2	26.7 ± 5.1
Family history of premature CAD (%)	7	7	14	12
Number of traditional CV risk factors except DM (%)				
- 0-1	23	49	40	53
- ≥ 2	77	51	60	46
Anti-cardiolipin IgG positivity (%)	67	79		
Anti-cardiolipin IgM positivity (%)	53	60		
Anti-β2-glycoprotein I IgG positivity (%)	56	47		
Anti-β2-glycoprotein I IgM positivity	44	44		
Lupus anticoagulant positivity	74	72		
Triple antiphospholipid antibody positivity	44	51		
Statin current (%)	21	12	30	11
Antiplatelet agents, current (%)	33	47	14	0
Systemic anticoagulation, current (%)	91	79	0	2*
Hydroxychloroquine, current (%)	21	63	-	-
Corticosteroids, current (%)	12	65	-	-
Immunosuppressives, current (%)	14	47	-	-
Vascular Ultrasound Findings				
LCCA IMT (mean±SD)	0.669±0.119	0.627±0.147	0.704±0.177	0.655±0.139
LCCA IMT > 0.9 mm (%)	5	8	31	23
Atherosclerotic plaques (%)				
- Carotid arteries	28	23	30	9
- Femoral arteries	33	19	20	9
- Any site	44	28	34	15

* Two of the control subjects were on anticoagulation for atrial fibrillation without CAD APS: Antiphospholipid syndrome; DM: Diabetes Mellitus; PAPS: Primary APS; SLE/APS: Systemic Lupus Erythematosus with APS; Q1: 1st quartile; Q3: 3rd quartile; BMI: Body mass index; CAD: Coronary artery disease; CKD: Chronic kidney disease; LCCA IMT: Left Common Carotid Artery Intima-Media Thickness

Abstract PS4:68 Table 2 Relative risk of increased atherosclerotic plaque numbers conferred by participant disease group versus controls, after adjusting for the effect of traditional CV risk factors

	Relative risk*	95% Confidence Interval	p-value
Carotid and femoral plaques			
PAPS	2.72	1.37 – 5.40	0.004
SLE/APS	2.63	1.28 – 5.41	0.009
DM	1.98	1.06 – 3.67	0.032
Carotid plaques			
PAPS	2.36	1.01 – 5.52	0.047
SLE/APS	2.86	1.20 – 6.83	0.018
DM	2.87	1.36 – 6.07	0.006
Femoral plaques			
PAPS	2.44	1.14 – 5.20	0.021
SLE/APS	1.91	0.84 – 4.38	0.124
DM	1.08	0.49 – 2.37	0.844

*Estimates derived from multiple negative binomial regression controlling for the effect of age, gender, hypertension, body mass index, pack years of smoking, family history of premature coronary artery disease
PAPS: Primary Antiphospholipid Syndrome; SLE/APS: Systemic Lupus Erythematosus with Antiphospholipid Syndrome; DM: Diabetes Mellitus