

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 extraskelatal 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

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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- β 2 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 ($\rho = 0.342$, $p = 0.005$) 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.

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LOSING ANTIPHOSPHOLIPID ANTIBODY POSITIVITY POST THROMBOSIS IN SECONDARY ANTIPHOSPHOLIPID SYNDROME

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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.