was performed, by two certified echocardiographers, in all study subjects. Distribution was evaluated with Kolmogorov-Smirnov test. Comparisons were done with χ^2 and Fisher's Exact test for qualitative variables, and Student's t test and Mann-Whitney's U test for quantitative variables. A p -value <0.05 was considered statistically significant.

Results: When comparing demographic characteristics there were no significant differences in age, gender, and comorbidities between SLE patients and controls. In the echocardiographic findings a significant difference was found in the left ventricular ejection fraction (LVEF), lower in SLE patients compared to controls (57.02% vs 61.89%, $p=0.001$), in the global longitudinal strain (GLS), reduced in SLE patients (-19.55% vs -22.00%, $p=0.001$), in the tricuspid annular plane systolic excursion (TAPSE), lower in SLE patients (22.00mm vs 24.00mm, $p=0.011$), in the left atrial volume index, higher in SLE patients (28.44ml/m² vs 22.29ml/m², $p=0.014$) and in the presence of mitral regurgitation, more prevalent in SLE patients (31.6% vs 10.5%, $p=0.024$) (Table 1).

Conclusion: Patients with SLE have a worse left ventricular function, evaluated by LVEF and GLS, a worse right ventricular systolic function, evaluated by TAPSE, an increased left atrial volume index and a higher prevalence of mitral regurgitation, which are associated with a higher risk of CV death. It is important to consider including a transthoracic echocardiogram as part of the CV evaluation in patients with SLE, which may result in an early detection of CV abnormalities and an opportune treatment.

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Table 1. Demographic characteristics and echocardiographic findings.

	SLE patients (n=38)	Controls (n=38)	p
Age years, median (p25-p75)	37.5 (24.5-43.2)	45.0 (24.0-50.0)	NS
Women, n (%)	34 (89.5)	37 (97.4)	NS
T2DM, n (%)	1 (2.6)	3 (7.9)	NS
HTN, n (%)	9 (23.7)	3 (7.9)	NS
Dyslipidemia, n (%)	3 (7.9)	3 (7.9)	NS
Obesity, n (%)	1 (2.6)	5 (13.2)	NS
Active smoking, n (%)	7 (18.4)	3 (7.9)	NS
Echocardiographic findings			
Left ventricle indexed mass g/m ² , median (p25-p75)	64.00 (50.66-87.09)	60.30 (52.97-76.74)	0.009
RWT, median (p25-p75)	0.37 (0.30-0.46)	0.38 (0.33-0.45)	NS
Left ventricular ejection fraction %, mean $\pm$ SD	57.02 $\pm$ 7.37	61.89 $\pm$ 5.23	0.001
Global longitudinal strain %, median (p25-p75)	-19.55 (-21.02 - -15.95)	-22.00 (-21.00 - -20.00)	0.001
Left atrium indexed volume ml/m ² , median (p25-p75)	28.44 (20.34-33.56)	22.29 (17.97-27.13)	0.014
TAPSE mm, median (p25-p75)	22.00 (20.00-24.00)	24.00 (21.00-25.00)	0.011
Valvular abnormalities			
Aortic regurgitation, n (%)	2 (5.3)	1 (2.6)	NS
Mitral regurgitation, n (%)	12 (31.6)	4 (10.5)	0.024
Tricuspid regurgitation, n (%)	16 (42.1)	17 (44.7)	NS

SLE, systemic lupus erythematosus; NS, not significant; T2DM, type 2 diabetes mellitus; HTN, hypertension; RWT, relative wall thickness; TAPSE, tricuspid annular plane systolic excursion.

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ANTIPHOSPHOLIPID ANTIBODIES AND ANTICOAGULANT TREATMENT: CAPILLAROSCOPIC FINDINGS

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Background: Antiphospholipid syndrome (APS) is an autoimmune condition characterized by arterial and/or venous thrombosis and/or obstetric morbidity, associated with the presence in the serum of antiphospholipid antibodies (aPL) [1]. Subjects with confirmed positivity of aPL in absence of thrombotic/obstetric manifestations are identified as aPL carriers [2]. The microangiopathy detected by nailfold videocapillaroscopy (NVC) in APS and in aPL-carrier patients is poorly investigated, as well as the possible interference of anticoagulant drugs [3].

Objectives: To compare microvascular damage in APS, aPL carriers and a group of patients (CTR) without aPL positivity and on regular warfarin therapy for cardiovascular indications

Methods: NVC investigations were performed as part of standard procedures in APS patients (18, mean age 50.0 $\pm$ 12.8 years), aPL carriers (24, mean age 46.4 $\pm$ 16.4 years) and CTR without aPL (18, mean age 74 $\pm$ 12.5 years) in therapy with oral anticoagulant (warfarin) for non-immunological vascular complications (atrial fibrillation, mechanical heart valve, deep venous thrombosis). Only patients affected by primary APS form were selected from data files (2006 Sapporo classification criteria). The following NVC parameters were available: dilated capillaries, giant capillaries, microhemorrhages (with particular attention to linear and thin hemosiderin deposits, arranged perpendicularly and parallel to the nailfold bed, "comb-like"), abnormal shape (i.e. branched "bushy" capillaries) and capillary number reduction. Those parameters were scored according to a semi-quantitative scale [4,5]. Statistical analysis was performed by non-parametric tests. Any p values equal or lower than 0.05 was considered statistically significant.

Results: APS patients showed a higher score for dilated capillaries ($p=0.001$), more frequent microhemorrhages ($p=0.03$), in particular "comb-like" microhemorrhages ($p=0.007$) than simply aPL carriers. Of note, there wasn't a statistically significant difference in the number of microhemorrhages between APS and CTR group ($p=0.23$), but again the number of "comb-like" hemorrhages, was almost absent in the CTR group ($p=0.03$). No significant correlation was found between the different aPL subtypes and the NVC parameters.

Conclusion: APS patients showed significantly higher number of non-specific NVC abnormalities than aPL carriers. Anticoagulant treatment could represent a further risk factor for the appearance of microhemorrhages in all the patients, being the NVC "comb-like" pattern mainly associated with the APS. Further investigations with larger cohorts of patients are needed for the definition of a possible APS specific NVC-pattern.

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