Table 1. Description of study population

	LN		RA		tDMARDs		Non tDMARDs	
	N	%	N	%	N	%	N	%
Age (years)								
18-34	1700	26.6	9015	8.3	1828	11.1	7187	7.8
35-44	1743	27.2	16662	15.4	2816	17.1	13846	15.1
45-54	1563	24.4	29955	27.7	4452	27.1	25503	27.8
55-64	1397	21.8	52685	48.6	7354	44.7	45331	49.3
Gender								
Female	5385	84.1	78419	72.4	12653	76.9	65766	71.6
Male	1018	15.9	29898	27.6	3797	23.1	26101	28.4

Table 2. IRs per 1,000 PY [95% CI] of SI for LN and RA patients

	LN total PY = 22065		RA total PY = 365033		tDMARDs		Non tDMARDs	
					total PY = 60999		total PY = 304034	
Pneumocystis carini pneumonia	28.2 [26.0-30.4]	8.5 [8.2-8.8]	12.6 [11.7-13.5]	7.7 [7.4-8.0]				
Septicaemia	23.3 [21.3-25.3]	12.1 [11.7-12.4]	13.3 [12.3-14.2]	11.8 [11.5-12.2]				
Cytomegalovirus infection	13.4 [11.9-14.9]	4.4 [4.2-4.6]	6.2 [5.6-6.8]	4.1 [3.8-4.3]				
Tuberculosis	7.2 [6.0-8.3]	6.7 [6.5-7.0]	18.2 [17.1-19.3]	4.4 [4.2-4.7]				
Mycosis	5.1 [4.1-6.0]	2.7 [2.6-2.9]	4.6 [4.1-5.1]	2.4 [2.2-2.5]				
Aspergillosis	4.7 [3.8-5.6]	2.7 [2.5-2.9]	2.1 [1.7-2.5]	2.8 [2.6-3.0]				
Herpes	3.3 [2.5-4.0]	2.4 [2.2-2.6]	2.9 [2.5-3.3]	2.3 [2.1-2.5]				

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AB0436

CHANGES OF LYMPHOCYTE SUBSETS AND CLINICAL INDEXES IN PERIPHERAL BLOOD OF PATIENTS WITH ANTI-PHOSPHOLIPID SYNDROME

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Background: Anti-phospholipid Syndrome (APS) is a non-inflammatory autoimmune disease, which can be divided into primary and secondary. Changes in lymphocyte numbers in APS are caused by disruption of the immune balance.

Objectives: The levels of lymphocyte subsets in peripheral blood of patients with anti-phospholipid syndrome were observed and their clinical indexes were analyzed.

Methods: 53 patients with anti-phospholipid syndrome (APS) were collected as the case group and divided into two groups of A, B according to whether primary and 50 health examiners as the healthy control group. The levels of peripheral lymphocyte subsets and laboratory data [erythrocyte sedimentation rate (ESR),

Table 1. Comparison of clinical data of two case groups and healthy control group M (p25, p75)

Groups	Case group A	Case group B	Healthy control group	H value	P value
History of thrombus (case)	4 ^a	8 ^a	-	13.709	0.001
History of adverse pregnancy (case)	3 ^a	19 ^a	-	34.596	<0.001
ESR	20.00 (12.25,111.00) ^a	35.00 (14.25,95.00) ^a	9.00 (6.00,13.00)	34.381	<0.001
CRP	15.00 (4.03,37.83) ^{ab}	7.33 (1.76,21.13) ^{ab}	2.10 (1.28,2.31)	35.263	<0.001
PLT	228.00 (189.50,573.25) ^{ab}	197.00 (66.00,260.50) ^{ab}	258.50 (228.25,272.25)	33.482	<0.001

Note: ^aComparison with healthy control group $P < 0.05$; ^bComparison with case group $P < 0.05$.

Table 2. Comparison of lymphocyte subsets in peripheral blood of two case groups and healthy control group M (p25, p75)

Groups	T (ng/ml)	CD4 ⁺ T (ng/ml)	CD8 ⁺ T (ng/ml)	CD4 ⁺ T/CD8 ⁺ T
Case group A	1140.22 (878.86,1819.56) ^{ab}	606.18 (473.50,873.45) ^b	494.55 (252.90,1022.24) ^b	1.56 (0.75,2.52) ^a
Case group B	664.89 (281.36,1360.00) ^{ab}	383.64 (142.46,678.25) ^{ab}	220.19 (114.43,464.50) ^{ab}	1.53 (0.94,3.07) ^a
Healthy control group	1250.50 (1070.00,1576.50)	639.00 (543.00,787.50)	422.00 (333.25,526.50)	1.55 (1.24,2.04)
H value	11.444	14.704	6.498	18.607
P value	0.003	0.001	0.039	<0.001
Groups	Th2	Th17 (ng/ml)	Treg (ng/ml)	Th17/Treg
Case group A	10.26 (7.36,14.72)	7.07 (3.65,12.17)	21.98 (18.00,34.25)	0.33 (0.12,0.48)
Case group B	2.78 (1.22,3.56) ^a	7.20 (3.29,10.50)	20.29 (13.03,31.31) ^a	0.31 (0.25,0.38)
Healthy control group	10.80 (5.39,15.83)	5.45 (4.06,7.56)	29.09 (21.63,42.07)	0.18 (0.13,0.33)
H value	10.884	2.452	7.516	2.590
P value	0.004	0.293	0.023	0.274

Note: ^aComparison with healthy control group $P < 0.05$; ^bComparison with case group $P < 0.05$.

C-reactive protein (CRP), platelets (PLT)]levels in the three groups were analyzed. The measurement data is not subject to normal distribution using the Median-Quartile method for statistical description; multiple sample comparisons using the Kruskal-Wallis H test; $p < 0.05$ as the difference is statistically significant.

Results: (1) The rates of thrombosis and adverse pregnancy in the two case groups were significantly increased. In the two case groups, the ESR and CRP were higher than those in the healthy control group, and the CRP in group A was higher. (2) Compared with the healthy control group, the levels of CD8⁺ T, CD4⁺ T/CD8⁺ T in case A group were increased, while the levels of total T, CD4⁺ T, CD8⁺ T, CD4⁺ T/CD8⁺ T, Th2 and Treg cells were decreased. However, there was no significant difference in Th17 cells level and Th17/Treg ratio compared with the healthy control group.

Conclusion: APS patients were more prone to have thrombosis, adverse pregnancy and thrombocytopenia. The changes of lymphocyte subsets were seen in peripheral blood, and the primary and secondary had different directions and different degrees of manifestation. However, whether the secondary factors can aggravate the clinical indicators of APS is still unclear.

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AB0437

RISK FACTORS ASSESSMENT FOR SUBCLINICAL ARTEROSCLEROSIS IN PRIMARY SJÖGREN'S SYNDROME.

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Background: Some autoimmune diseases, including rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE), are considered to be independent risk factors for vascular morbidity and mortality. These pathologies present accelerated atherosclerosis, partly because of a chronic sustained inflammation, greater prevalence of cardiovascular risk factors (CVRFs) and pharmacological therapy. However, regarding primary Sjögren's syndrome (pSS), available data are heterogeneous and proceed from small case series. For this reason, the aim of this study was to provide further information on the identification of atherosclerosis in pSS and its possible association with clinical and analytical parameters of the disease.

Objectives: To assess presence of subclinical atherosclerosis by means of carotid ultrasound in patients with pSS and to analyze clinical, analytical and CVRF along with their potential association with the presence of subclinical cardiovascular affection.

Methods: This is a cross-sectional study of 38 patients with pSS (all patients met ACR/EULAR¹ classification criteria for pSS) and 38 age and sex matched controls. Demographic variables and classical CVRFs were collected (Hypertension, Diabetes mellitus, dyslipemia, Body Mass Index and smoking habit) and the presence of subclinical atherosclerosis was assessed by carotid ultrasound with carotid intima-media thickness (CIMT) measurement and determination of the presence of atheromatous plaques², both in pSS patients and controls. Disease