for PE seem to benefit from HCQ during pregnancy. Future research may reveal mechanisms by which HCQ might lower the incidence of PE.

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Table 1. Patient characteristics and risk profile

		HCQ only (n=39)	LDA only (n=39)	HCQ + LDA or LDA (n=43)	no HCQ or LDA (n=69)
Patient characteristics	Age (years), median (IQR)	29 (25-33)	32 (30-33)	30 (28.5-33.5)	31 (28-35)
	BMI, median (IQR)	23.5 (22-25)	23 (22-24)	22 (22-24)	23 (22-24)
	Chronic Hypertension, n (%)	7 (17.9%)	9 (23.1%)	6 (14.0%)	9 (13.0%)
	Preconception counseling, n (%)	23 (59.0%)	28 (71.8%)	35 (81.4%)	41 (59.4 %)
Obstetrical history	Nulliparous, n (%)	20 (51.3%)	26 (66.7%)	33 (76.7%)	36 (52.2%)
	Previous fetal loss, n (%)	1 (2.6%)	10 (25.6%)	14 (32.6%)	10 (14.5%)
SLE characteristics and therapy	Previous PE, n (%)	2 (5.1%)	4 (10.3%)	4 (9.3%)	5 (7.2%)
	Disease duration (years), median (IQR)	7.0 (2.0-11.0)	7.5 (3.0-11.5)	6.7 (3.4-10.0)	6.0 (3.0-9.2)
	SLEDAI, median (IQR)	2.0 (1.0-4.3)	2.0 (0-2.5)	4.0 (2.0-4.0)	2.0 (0-4.0)
	Lupus nephritis, n (%)	9 (23.1%)	13 (33.3%)	18 (41.9%)	14 (20.3%)
	Anti-dsDNA antibodies, n (%)	22 (56.4%)	15 (38.5%)	36 (83.7%)	33 (47.8%)
	Prednisolone therapy, n (%)	26 (66.7%)	15 (38.5%)	25 (58.1%)	29 (42.0%)
	Prednisolone (mg/d), median (IQR)	5.0 (5.0-8.0)	5.0 (5.0-7.5)	5.0 (4.0-5.0)	5.0 (5.0-8.0)
	APS, n (%)	-	12 (30.8%)	14 (32.6%)	2 (2.9%)
aPL status	Any positive aPL, n (%)	-	20 (51.3%)	19 (44.2%)	5 (7.2%)
	LAC, n (%)	-	15 (38.5%)	13 (30.2%)	3 (4.3%)
	ACL, n (%)	-	14 (35.9%)	13 (30.2%)	4 (5.8%)
	β 2-GP1, n (%)	-	12 (30.8%)	12 (27.9%)	2 (2.9%)

LAC = Lupus anticoagulant, ACL = Anticardiolipin antibody, β 2-GP1 = β 2-Glycoprotein I antibody

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AB0342

OBSTETRIC AND THROMBOTIC ANTIPHOSPHOLIPID SYNDROME: ARE THEY DIFFERENT ENTITIES?

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Background: Several studies showed two main clinical phenotypes of antiphospholipid syndrome (APS): thrombotic (TAPS) and obstetric APS (OAPS). Although they have the same autoantibody profile, one of them developed thrombosis and other one obstetric morbidity.

Objectives: To study clinical, demographic and antibody profile in patients with TAPS and OAPS.

Methods: we retrospectively evaluated TAPS and OAPS patients who were included in Argentine Antiphospholipid antibodies registry. We studied clinical, demographic and antibody profile in both groups.

Results: 238 patients were included in the registry. 201 (84.81%) of them were female. 122 (60.69 %) of them fulfilled APS Sydney classification criteria, 47 (38.52%) TAPS and 52 (42.62%) OAPS. 23 (18.85%) patients had both thrombotic and obstetric events so they were excluded in this analysis.

Arterial Hypertension (HBP) and Hyperlipidemia were more frequent in TAPS versus OAPS. Older age was found in TAPS as well as in association with Systemic lupus erythematosus (SLE). There was no difference in antibody profile between the 2 groups, and the Global Antiphospholipid Syndrome Score (aGAPSS) was higher in TAPS than OAPS.

18 (38.3%) of TAPS patients had at least 1 pregnancy. Mean number of pregnancies of TAPS was 2.5 (1.10) and 3.84 (1.86) in OAPS. Thrombotic events were not found in TAPS during pregnancy and puerperium. HBP and gestational diabetes (GD) and other pregnancy related comorbidities were found in TAPS.

	OAPS (n=52)	TAPS (n=47)	P		OAPS (n=52)	TAPS (n=18)
SLE, n (%)	11 (21.2)	28 (59.6)	0.0002	N	% (DE)	N % (DE)
aGAPSS, mean (R/IQ)	4 (5)	8 (5)	<0.0001	Abortions (<10 weeks)	33 38.4 (36.2)	8 26.5 (35.3)
Age, mean (DE)	39.3 (6.24)	43.1 (13.5)	<0.0001	Live Birth	36 33.5 (28.1)	16 72.2 (34.7)
HBP, n (%)	5 (9.6)	15 (31.9)	0.0121	>37 weeks	21 16.4 (2.38)	16 61.1 (31.7)
Hyperlipidemia, n (%)	4 (7.7)	12 (25.5)	0.0267	Prematurity <37 >34 weeks	11 8.76 (1.97)	0 -
GD, n (%)	3 (5.8)	2 (4.3)	0.9999	Prematurity <34 weeks	9 6.37 (1.49)	1 1.39 (5.89)
Obesity, n (%)	8 (15.4)	4 (8.5)	0.2912	Pre eclampsia >34<37 weeks	1 0.490 (0.0350)	2 4.63 (0.138)
Smoking, n (%)	11 (21.1)	13 (27.6)	0.4019	Placental Hematoma	3 1.86 (8.18)	1 2.78 (11.8)
Sedentary life-style, n (%)	16 (30.8)	17 (36.2)	0.8486	Abruption Placentae	2 1.96 (9.80)	2 4.17 (12.9)
Triple Positivity	5	4	0.8323	Normal delivery	20 17.8 (27.9)	14 47.7 (33.9)
Double Positivity	9.6 1	8.5 3		Cesarean section	22 17.2 (21.6)	2 11.1 (32.3)
Simple Positivity	1.9 29	6.4 19		Urgent Cesarean section	13 9.80 (20.3)	4 12.5 (24.6)
	38.5	40.4		GD	2 1.37 (7.49)	1 2.78 (11.8)
				HBP	6 3.46 (10.3)	3 10.2 (26.3)

Conclusion: Antibody profile was similar in TAPS and OAPS. However, clinical manifestations and cardiovascular risk were different. These results should be evaluated in prospective studies.

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AB0343

THE CHARACTERISTICS OF T CELLS IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS WITH ANXIETY BASED ON MACHINE LEARNING

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Background: Systemic lupus erythematosus (SLE) is an autoimmune disease, the immune system of patients to be disordered, especially in T cell subsets¹. They were prone to mental diseases, anxiety particularly, which lead to suicide². The recent study had reported that CD4⁺ T cells in the peripheral blood played the key role in like anxiety behavior of mice³. Although there showed that the level of serum TNF- α in SLE patients with anxiety was higher than without anxiety⁴, finding the important special mediators especially in T cell subsets was still necessary for the prevention of anxiety in SLE patients.

Objectives: In total, 108 SLE patients, which met the diagnostic criteria of the American Society of rheumatology (v1997), were enrolled in this study from Affiliated Hospital of Nantong University, China. Exclusion criteria included other autoimmune diseases and active infection (including hepatitis B or C virus, Epstein-Barr virus, human immunodeficiency virus or Mycobacterium tuberculosis infection).

Methods: We surveyed the abundance of 74 immune cell subpopulations from 108 SLE patients using flow cytometry, and investigated their differences between patients with and without anxiety (24 versus 84). Moreover, machine learning including Lasso regression, Random forest (RF) and Sparsity partial least squares discriminant analysis (sPLS-DA) was employed to build models and further selected important features for the classification of SLE patients with anxiety.

Results: SLE patients with anxiety showed higher body mass index (BMI) and lower quality of life. In their peripheral blood, the proportion of internal cell subsets composition of Th cell and Treg cells changed. By machine learning, we

finally found that BMI and PD1⁺CD28⁺ Treg played important roles in developing lupus anxiety.

Conclusion: In this study, machine learning was applied to build models to select the most important T cell subset in SLE patients with anxiety. These findings suggested that BMI and imbalance of PD1⁺CD28⁺ Treg containing effector memory Treg cells and effector Treg cells mostly played important roles in the development of SLE anxiety.

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AB0344

PREVALENCE AND RISK FACTORS FOR SEROSITIS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: A CASE CONTROL STUDY

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Background: Systemic lupus erythematosus (SLE) is an autoimmune multisystemic disease with a wide variety of clinical manifestations, being one of them serositis, which includes pericarditis, pleuritis and peritonitis [1]. Incidence of serositis ranges between 11-54% [2].

Objectives: To determine the prevalence of serositis in patients (pt) with SLE attended at the outpatient Lupus Unit and identify factors that could be used as predictors of this manifestation.

Methods: Retrospective case-control study. 297 medical records of SLE pt were reviewed: 28 pt were diagnosed with serositis (cases) and were age- and sex-matched with 2 controls with SLE without serositis. Differences between cases and controls were analyzed as well as factors associated with serositis.

Results: Patient's characteristics are described in Table 1. The prevalence of serositis in our cohort was 9.42%. The difference between the prevalence of serositis in men and women was statistically higher in men, 30% vs 7.9% (p=0.001, CI 95%: 1.7%-42.4%). Serositis was diagnosed at an age of 41 ±14 years (y) and in 40.7% it was the first symptom. Time from SLE diagnosis to serositis was 4±5.3 y. 10 pt had recurrences: 6 had 2, and 4 had 3. Incidence of pericarditis and pleuritis was 78.6% and 82.1% respectively. 2 pt suffered from pericardial tamponade. Mean prednisone dose received during the serositis was 44.6±26.6mg. At the moment of serositis 100% were ANA+, 85.7% anti-dsDNA+, 80% CrithidiaDNA+, 57.9% antiRo60+, 52.6% antiRo52+, 15.9% antiLa+, 47.4% antiRNP+, 21.1% antiSm+, and 77.8% had low C3.

When looking for serositis-associated factors we only found association with anti-dsDNA measured by Crithidia (p=0.016) and different measures of glucocorticosteroids (GC), having cases needed higher doses than controls (Table 1). Those with serositis had significantly received more mycophenolic acid (p=0.021) and marginally, more belimumab (p=0.056).

Table 1. Characteristics of cases and controls. *indicates mean±SD, Bold=statistically significant difference.

VARIABLES	CASES (28)	CONTROLS (54)	p-value
Female	22 (33.3%)	44 (66.7%)	0.775
Male	6 (37.5%)	10 (62.5%)	
Caucasian	20 (32.8%)	41 (67.2%)	0.693
Hispanic American	7 (38.9%)	11 (61.1%)	
Others	1 (33.3%)	2 (66.6%)	
Arthritis	22 (38.6%)	35 (61.4%)	0.219
Leukopenia	17 (34.0%)	33 (66.0%)	1.000
Lymphopenia	25 (33.8%)	49 (66.2%)	1.000
Thrombocytopenia	5 (20.8%)	19 (79.2%)	0.128
Nephritis	8 (50.0%)	8 (50.0%)	0.152
Raynaud's phenomenon	6 (42.9%)	8 (57.1%)	0.263
Haemolytic anaemia	3 (42.9%)	4 (57.1%)	0.686
Antinuclear antibodies	28 (34.6%)	53 (65.4%)	1.000
Anti-dsDNA	23 (37.7%)	38 (62.3%)	0.295
AntiSm	9 (42.9%)	12 (57.1%)	0.425
AntiRo52	10 (41.7%)	14 (58.3%)	0.444
AntiRo60	13 (37.1%)	22 (62.9%)	0.645
AntiRNP	14 (43.8%)	18 (56.3%)	0.159
CrithidiaDNA	19 (47.5%)	21 (52.5%)	0.016
Low C3	21 (39.6%)	32 (60.4%)	0.224
Low C4	13 (38.2%)	21 (61.8%)	0.637
Low CH50	13 (39.4%)	20 (60.6%)	0.480
SLICC*	0.54 (±0.9)	0.49 (±1.1)	0.717
SLEDAI*	3.1 (±3.1)	2.98 (±3.2)	0.844
Corticosteroids			
Ever	27 (38.6%)	43 (61.4%)	0.051
Pulses ever	9 (60.0%)	6 (40.0%)	0.033
Maximum prednisone dose ever:			
<10mg	18 (85.7%)	3 (14.3%)	0.003
10-29mg	14 (87.5%)	2 (12.5%)	
30-60mg	11 (45.8%)	13 (54.2%)	
>60mg	8 (47.1%)	9 (52.9%)	

Conclusion: The prevalence of serositis was 9.42%. Serositis is significantly more frequent in SLE men than in women: almost 1/3 will develop serositis, so we need greater awareness of serositis in SLE men.

CrithidiaDNA+ was identified as an associated factor of serositis. Furthermore, pt with serositis significantly received more pulses of GC and a higher maximum dose throughout the disease, which could imply a more aggressive form of SLE than in those without serositis. No correlation was found between serositis and any other characteristic.

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AB0345

Th9 CELLS AND THEIR ASSOCIATED CYTOKINES: THE PROBABLE PLAYERS IN SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) AND TYPE 1 DIABETES MELLITUS (T1DM)

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Background: SLE is an autoimmune disease (AID) of unknown origin. Several factors can contribute to immune dysfunction in SLE.

Interleukin 9 (IL9) is a newly emerging T cell-derived factor preferentially expressed by CD4⁺T cells: T helper 9 (Th9)

IL9 targets different cell lineages, and can contribute to the development of allergic & AIDs

Whether abnormal expression and secretion of IL9 are present in SLE patients (pts) still unidentified. It is also unclear whether IL9 exerts main proinflammatory or anti-inflammatory activities in SLE. T1DM is characterized by inflammation of the pancreatic islets of Langerhans. Insulinitis progresses over time and β cells become destroyed then clinical DM is established. T1DM is regarded as a T cell-driven AID

Objectives: Evaluation of the expression of CD4⁺ IL9⁺ T cells & the level of IL9 in SLE pts compared to both healthy subjects & pts with another AID: T1DM. Also, to evaluate the correlation of these expressions with clinical features, laboratory parameters and SLE activity

Methods: The study included: **Group I** 25 SLE pts fulfilling SLICC classification criteria divided into 2 subgroups (gps) according to SLE disease activity index (SLEDAI) IA: 20 pts with mild to moderate activity (<12) IB: 5 pts with severe activity (>12) recruited from rheumatology clinic or internal medicine ward (Rheumatology unit), Main University Hospital, Alexandria. **Group II** 15 healthy individuals as a first control gp. **Group III** 15 pts with T1DM fulfilling the American Diabetes Association criteria as a second control gp. All pts were subjected to history taking, clinical examination, laboratory investigations: CBC, LFT, KFT, ESR, CRP, ANA, Anti-dsDNA, Th9 cell expression detection by flowcytometry and serum IL9 by ELISA

Results: There was no statistical difference between all gps as regards age & sex but a significant increased ESR in SLE compared to controls & T1DM p<0.001 p=0.001

Th9 expression was highly increased in SLE pts, range 0.13-4.54% & mean ±SD=1.50 ± 1.47% than both control gps. In healthy controls Th9 ranged between 0.0-1.29% with mean 0.37 ±0.52%, while in T1DM pts ranged between 0.03