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% anti-Ro60+, 52.6% anti-Ro52+, 15.9% anti-La+, 47.4% anti-RNP+, 21.1% anti-Sm+, 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
Anti-Sm	9 (42.9%)	12 (57.1%)	0.425
Anti-Ro52	10 (41.7%)	14 (58.3%)	0.444
Anti-Ro60	13 (37.1%)	22 (62.9%)	0.645
Anti-RNP	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.

REFERENCES:

- [1] Hochberg MC. Updating the American College of Rheumatology revised criteria for the classification of systemic lupus erythematosus. *Arthritis Rheum* 1997;40:1725
- [2] Ryu S, Fu W, Petri MA: Associates and predictors of pleurisy or pericarditis in SLE. *Lupus. Science*. 2017, 4(1):e000221.

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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