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.

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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

to 2.13% with mean of $0.67 \pm 0.59\%$. A high significant difference was found between SLE pts and controls $p=0.001$, an insignificant rise was seen in SLE pts compared to T1DM pts $p=0.157$. A high significant increase in Th9 was found in severe SLE: mean of $3.74 \pm 1.15\%$ than in pts with mild to moderate SLE: mean $0.94 \pm 0.88\%$ $p<0.001$

IL9 level was highly increased in SLE pts: mean of 42.83 ± 23.98 pg/ml than both control gps. In healthy controls the mean was 8.54 ± 13.27 , while in T1DM with mean of 29.17 ± 16.09 pg/ml. A high significant difference was found between SLE pts and normal controls $p<0.001$ but an insignificant rise with T1DM $p=0.294$. A high significant increase in IL9 in pts with severe ds compared to mild to moderate pts $p<0.001$. A significant direct correlation between Th9 & IL9 and SLEDAI/105 A significant direct correlation between damage index and Th9 $p=0.040$ but not IL9 $p=0.053$

In SLE no significant relation between Th9 or IL9 & clinical manifestations or disease duration. A direct correlations between Th9 & ESR $p=0.046$ and CRP $p=0.025$, a significant correlation between IL9 and CRP $p=0.033$, no correlations between Th9&IL9 level and anti-dsDNA $p=0.593$ & 0.4 Significant direct correlation between Th9 and IL9 in T1DM pts, still no correlation with glyce-mic profile. IL9 levels were significantly increased in SLE with elevated CRP $p=0.033$ & the % of Th9 cells were increased with elevated ESR and CRP $p=0.025$, 0.046

Conclusion: In SLE pts; IL9 level and Th9 cells expression were significantly elevated compared to healthy controls. IL9 levels and the percentages of Th9 directly correlated with the SLE disease activity. IL9 levels also were significantly increased in T1DM pts compared to controls, but they were less expressed than in SLE. This suggests an important role of IL9 in the pathogenesis AIDs as SLE

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AB0346 RHUPUS SYNDROME: CLINICAL ANALYSIS OF 21 PATIENTS

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Background: Rhupus syndrome is a clinical condition characterized by a combination of the clinical and immunologic features of rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) (1). However, this definition did not establish whether Rhupus syndrome is a distinct clinical entity or is an overlap between RA and SLE. To date, fewer than 200 cases of Rhupus have been reported (1).

Objectives: Our purpose was to analyze the clinical and serological characteristics of patients with Rhupus and compare them with patients with SLE.

Methods: We undertook a retrospective study covering 10 years (2009 to 2019) of experience in our University Hospital: Department of Internal Medicine and department of Rheumatology. 238 patients hospitalized were evaluated during this period. The clinical and laboratory parameters of 21 patients with Rhupus syndrome were compared with those of 217 SLE without RA patients

Results: Twenty one patients were included (all fulfilled ACR criteria for SLE as well as for RA). They were 19 female and 2 male. The mean age of patients at the disease onset was 43.5 years. Among these, 9 patients (42.8%) were diagnosed with RA at the onset of disease and then developed SLE over an average period of 6.2 years. Six patients (28.5%) had a diagnosis of SLE prior to the RA diagnosis with an interval of 8.6 years between diagnoses. Six patients (28.5%) were diagnosed with RA and SLE concomitantly. Patients with Rhupus experienced more often arthritis, joint swelling, morning stiffness and joint erosions than patients with SLE alone ($p<0.001$). Comparison of extra-articular manifestations showed that, renal involvement was less often in patients with Rhupus when compared to patients with SLE-alone. There were no significant differences in the prevalence of malar rash, serositis, neurological and hematological features between the Rhupus and SLE-alone groups. Rheumatoid factor and anticyclic citrullinated peptide (CCP) antibody were significantly more prevalent in the Rhupus group than in the control group. The incidences of increased erythrocyte sediment rate (ESR) and C-reactive protein (CRP) were also significantly higher in Rhupus patients. (Table 1)

Table 1. Comparison of clinical and biological features between Rhupus Syndrome (Rhupus Group) and SLE-alone patients (Control Group).

	Rhupus Group N=21 (%)	Control Group N=217 (%)	p
Malar rash	12 (57.1)	120 (55.3)	0.87
Serositis	6 (28.5)	47 (21.7)	0.46
Lupus nephritis	4 (19)	56 (25.8)	0.49
Hemolytic anemia	1 (4.7)	8 (3.6)	1
Leucopenia	8 (38)	90 (41.4)	0.15
Thrombopenia	4 (19)	27 (12.4)	1
Polyarthritis	21 (100)	54 (24.8)	<0.001
Erosion of joint	21 (100)	0	<0.001
Increased ESR	20 (95.2)	155 (71.4)	0.019
Increased CRP	18 (85.7)	88 (40.5)	<0.001
Anti-DNA antibodies	16 (76.1)	134 (61.7)	0.61
Anti-Sm antibodies	6 (28.5)	63 (29)	0.57
Rhumatoide Factor	12 (57.1)	52 (23.9)	0.04
Anti-CCP antibody	6 (28.5)	14 (6.5)	0.027

CRP: C-reactive protein, ESR: erythrocyte sediment rate, Anti-CCP: anticyclic citrullinated peptide antibody.

Conclusion: Patients sharing features of both RA and SLE have been observed infrequently. In fact, the incidence of Rhupus syndrome in SLE patients is estimated at 1.3% (2). In our study, this incidence was 8.8%. The findings of our study are consistent with those of most studies with respect to the frequencies arthritis, lupus related features, and serological disorders (2,3).

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AB0347 RENAL BIOPSY IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: IS IT ONLY LUPUS NEPHRITIS?

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Background: Renal biopsy is a cornerstone in the diagnosis and management of renal involvement in patients with systemic lupus erythematosus (SLE). However, non-lupus nephritis has been also observed in SLE patients with renal disease (1).

Objectives: The aim of this study was to draw attention to the causes of non-lupus nephritis in SLE patients with kidney biopsy.

Methods: This retrospective, descriptive study included 139 SLE patients who had at least one kidney biopsy between 2001 and 2020. All patients had fulfilled the SLICC or EULAR/ACR criteria for SLE. According to the pathology report results, 116 of the patients were diagnosed with lupus nephritis (LN), 18 patients had non-lupus nephritis, 2 biopsies were normal, and 3 biopsies were insufficient. Demographics, SLE disease duration, and renal biopsy diagnosis were derived from our hospital medical records.

Results: Of the 23 patients (female:18/male:5), the mean age at the SLE diagnosis was 30.5 years and the median SLE disease duration was 8.5 (11.6) years. Pathologic report findings were compatible with focal segmental glomerulosclerosis in 6 patients, membranous nephropathy with no cellular proliferation and inflammation in 4 patients, thrombotic microangiopathy in 3 patients, IgM nephropathy in 2 patients, tubulointerstitial nephritis in 2 patients, and proliferative glomerulonephritis with monoclonal IgG deposits in one patient. There were no different for SLE manifestation in both groups. LN vs other renal pathologies