

Table 1. Pregnancy outcomes

Pregnancy, n	SLE, n=16	Control, n=15	p
Birth	9 (56.3%)	13 (87%)	0,00
Abortion	2 (12.5%)	0	0,00
Unfavorable pregnancy outcome	5 (31.2%)	2 (13%)	0,00
Missed miscarriage	4 (25%)	0	0,00
Spontaneous abortion	1 (6%)	2 (13%)	ns

Analysis of gynecological history indicate that episodes of menstrual disorders were significantly more often reported in SLE pts (50% vs 20% in controls, $p=0.001$), similarly, gynecological diseases were also documented in 50% of SLE pts (chronic salpyngo-oophoritis, colpitis, endometriosis and uterine endometrioma, subserous uterine myoma, cervical dysplasia, cervical erosion), meanwhile low AMH was found only in 4 SLE pts; there was only 1 subject with gynecological condition – teratoma of the ovaries – in the control group (favorable outcome – surgical removal, preserved fertility and two births after surgery). **Conclusion:** The incidence of unfavorable pregnancy outcomes is significantly higher in patients with SLE compared to the control group of healthy women.

Disclosure of Interests: None declared

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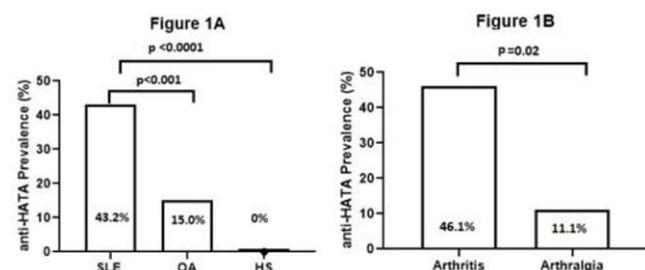
AUTOANTIBODIES DIRECTED AGAINST HOMOCYSTEINYLATED ALPHA 1 ANTITRYPSIN AS A POTENTIAL NEW BIOMARKER FOR ARTHRITIS IN PATIENTS AFFECTED BY SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: Joint involvement represents one of the most frequent features in patients affected by Systemic Lupus Erythematosus (SLE). This manifestation is characterized by a great heterogeneity in phenotype and severity: the application of more sensitive imaging techniques identified an erosive damage in about 25% of patients (1). This damage has been associated with autoantibodies, such as anti-citrullinated (ACPA) and anti-carbamylated proteins (antiCarP), previously identified in patients Rheumatoid Arthritis (RA) patients. Recently, homocysteinyllated alpha 1 antitrypsin (Hcy-1A1AT) has been identified as a new antigenic target of autoantibodies in seronegative RA patients: in detail, anti-homocysteinyllated alpha 1 antitrypsin (anti-HATA) antibodies have been identified in 75.7% of patients (2). **Objectives:** In the present study, we aimed at determining the prevalence of anti-HATA in a cohort of SLE patients.

Methods: We evaluated patients affected by SLE according to the 1997 ACR criteria. Demographic, clinical, and laboratory data were collected in a standardized computerized electronically filled form. Each subject underwent peripheral blood sample collection. Hcy-1A1AT was obtained by in vitro modification of native A1AT and used as antigens by ELISA to test the presence of anti-HATA in sera obtained from enrolled subjects. Finally, we investigated the presence of ACPA and Rheumatoid Factor (RF) commercial ELISA kits and of anti-CarP (home-made ELISA) by a home-made ELISA in SLE patients' sera. As control, we enrolled 40 patients affected by Osteoarthritis (OA) and 41 healthy subjects (HS).

Results: The present analysis included 88 SLE patients (M/F 6/82 median age 47 years (IQR 17), median disease duration 156 months (IQR 180). Joint involvement was observed in 75 SLE patients (85.2%): in detail, 65 patients referred arthritis and the remaining 10 inflammatory arthralgias. We identified the presence of anti-HATA IgG in 38 SLE patients (43.2%). This prevalence was significantly higher in comparison with OA and HS subjects [15.0% ($p<0.001$) and 0% ($p<0.0001$), respectively; Figure 1A]. Focusing on the SLE cohort, no differences were observed between patients with and without joint involvement in anti-HATA IgG prevalence (41.3% versus 34.7%, respectively; $p=0.34$). However considering SLE patients according to the presence of arthralgia and arthritis, the prevalence of anti-HATA was significantly higher in patients with arthritis in comparison with those patients with arthralgias (46.1% versus 11.1%, $p=0.02$; figure 1B). Finally, no significant association between anti-HATA and the other tested autoantibodies (RF, ACPA, anti-CarP) was found.



Conclusion: We evaluated the prevalence of anti-HATA in a cohort of SLE patients. The prevalence of these autoantibodies was significantly higher in SLE patients than in OA patients and in HS. The association with arthritis suggests a possible role for anti-HATA as biomarkers of SLE-related joint involvement.

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QUALITY OF LIFE IN PATIENTS WITH ANTIPHOSPHOLIPID ANTIBODIES DIFFERS ACCORDING TO ANTIPHOSPHOLIPID SYNDROME DAMAGE INDEX (DIAPS)

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Background: Antiphospholipid syndrome (APS) is a rare autoimmune disorder that mainly affects young individuals. Due to the potentially devastating effects of some of its clinical manifestations, such as ischemic stroke and thrombotic microangiopathy, APS can have a deep negative impact on the quality of life (QoL) of the affected patients due to damage accrual (2).

To capture the full consequences of the disease, including both thrombotic and extra-criteria APS specific features, the antiphospholipid syndrome (APS) damage index (DIAPS), a 37-item score, was conceived.

Objectives: The aim of this study was to validate the DIAPS in a cohort of antiphospholipid antibodies (aPL) positive subjects, comparing it to the risk stratification score for the development of clinical manifestations of APS (Global APS Score -GAPSS) and QoL questionnaires filled out by APS patients.

Methods: This retrospective study included a total of 84 consecutive persistent aPL positive patients who attended the San Giovanni Bosco Hospital (Turin, Italy). Patients were then divided based on their diagnosis in 3 groups: primary APS (PAPS), secondary APS (SAPS) and aPL positive patients without clinical manifestations of APS, according to Miyakis criteria (1). Demographic, clinical and laboratory characteristics, were retrieved from electronic medical records and are summarized in Table 1. DIAPS was calculated as previously reported by adding together all points corresponding to the clinical manifestations of the patients (3).

Results: A total of consecutive 84 patients were enrolled in the study: 39 primary APS patients (PAPS), 28 secondary APS patients (SAPS) and 17 aPL positive patients without APS-related clinical manifestations. APS patients had significantly higher levels of DIAPS when compared to aPL asymptomatic patients (mean DIAPS 2.6 ± 1.8 vs. 1.5 ± 1.9 , respectively; $p<0.05$). Moreover, SAPS patients had significantly higher levels of DIAPS when compared to PAPS patients (mean DIAPS 3.1 ± 1.9 vs. 2.2 ± 1.7 , respectively; $p<0.05$). When comparing GAPSS and DIAPS levels in all the patients, we found a significantly positive correlation between these two scores (mean GAPSS 11.9 ± 5.2 and mean DIAPS 2.4 ± 1.9 ; Pearson 0.241; $p<0.05$). Finally, when applying the SF-12 score to our cohort, the mean physical component score and mental component score were lower than the average population (39.3 ± 11.3 and 42.3 ± 8 , respectively) and we observed a negative correlation trend between DIAPS and both PCS and MCS (Pearson -0.133 and -0.183).

Table 1. Clinical and laboratory characteristics of the patients

	PAPS (n=39)	SAPS (n=28)	aPL asymptomatic (n=17)
Anagraphic			
Mean age (S.D.) at data collection	50.6±13.7	47.4±12.1	48.6±11.3
Sex (females), n	29 (74.4%)	29 (74.4%)	29 (74.4%)
Secondary diagnosis, n	N/A	SLE 24 (85.7%) SSJ 1 (3.6%) UCTD 1 (3.6%) MCTD 2 (7.1%)	SLE 8 (45.1%)
Clinical manifestations of APS			
Thrombosis, n	30 (76.9%)	26 (92.9%)	N/A
Arterial thrombosis, n	20 (51.3%)	14 (50%)	N/A
Venous thrombosis, n	17 (43.6%)	15 (53.6%)	N/A
Pregnancy morbidity, n	11 (28.2%)	4 (14.3%)	N/A
aPL profile at diagnosis			
LA, n	31 (79.5%)	23 (82.1%)	12 (70.6%)
aCL (IgG/M), n	22 (56.4%)	18 (64.3%)	10 (58.9%)
Anti-β2GPI (IgG/M), n	18 (46.2%)	16 (57.1%)	3 (17.6%)
aPS/PT (IgG/M), n	23 (59%)	21 (75%)	8 (47.1%)