SAT0451

DISEASE COURSE PATTERNS IN SYSTEMIC LUPUS ERYTHEMATOSUSK. Tselios, D. Gladman, Z. Touma, J. Su, N. Anderson, M. Urowitz. *Rheumatology, Toronto Western Hospital, University of Toronto, Toronto, Canada*

Background: Previous studies described three patterns of disease activity over time in systemic lupus erythematosus (SLE), namely long quiescent, relapsing remitting and persistently active. However, they enrolled prevalent patients, many of whom in the late stages of the disease. As such, the patterns of disease course since diagnosis are not known.

Objectives: The aim of the present study was to assess the prevalence and characteristics of such patterns over 10 years of follow-up in an inception cohort.

Methods: The inception patients of a large lupus cohort (enrolled within 18 months of diagnosis, n=883), with at least 10 years of follow-up and no time interval >18 months between consecutive visits, were investigated. Monophasic (M) pattern was defined as a clinical SLEDAI-2K=0 [serology (anti-dsDNA antibodies and C3/C4 levels) excluded], achieved within five years since enrollment and maintained for ≥10 years after that. Relapsing-remitting (RR) pattern was defined based on ≥2 remission periods (a remission period equals two consecutive visits with a clinical SLEDAI-2K=0), while patients with no remission were categorised as persistently active (PA). Descriptive and regression analyses were used to compare the different groups regarding cumulative damage at 10 years, mortality and flare rate beyond 10 years.

Results: Of 267 patients who fulfilled the inclusion criteria, 27 (10.1%) were monophasics, 180 (67.4%) RR and 25 (9.4%) PA. Thirty-five patients (13.1%) had only one remission period ("hybrid"). There were no significant differences regarding the demographic, clinical, immunological and therapeutic characteristics among groups at enrollment. At 10 years, PA patients had received significantly more glucocorticosteroids [39.4±24.3 g vs. 16.6±10.7 g and 27.3±18.4 g for the M and RR groups, p<0.001] and accumulated significantly more damage [SLICC/DI=2.36±1.6 vs. 0.93±1.07 and 1.22±1.33, respectively, p<0.001]. A trend for higher mortality was observed in the PA patients [24% vs. 13.3% and 11.1% for the RR and M patients respectively, p=0.22] whereas death occurred earlier [18.4±4.4 vs. 20.1±7.2 and 24±4.1 years, p=0.11]. Beyond 10 years, the annual flare rate was higher for the PA and RR patients as compared to the monophasics [0.50±0.40 vs. 0.47±0.57 vs. 0.10±0.18 respectively, p<0.001]. Multinomial regression analysis for group membership showed Black race [OR=2.78, 95% CI=1.05–7.31, p=0.039] and higher adjusted mean SLEDAI-2K over the first two years [OR=1.21, 95% CI=1.11–1.32, p<0.0001] to be associated with a more severe disease course.

Conclusions: Approximately 70% of lupus patients followed a relapsing remitting course from diagnosis onwards, while 10% displayed a monophasic and another 10% a persistently active course. Black race and more severe disease over the first two years were associated with a worse disease course.

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SAT0452

ENDOTHELIUM DYSFUNCTION IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS WITH AND WITHOUT NEPHRITISN. Mohannad¹, A.I. El Zawawy², M. Elbordeny³, D. Emara⁴, S. Ibrahim⁵. ¹Internal Medicine department, Rheumatology Unit, Alexandria University Hospitals; ²Internal Medicine department, Rheumatology Unit; ³Department of Clinical and Chemical Pathology; ⁴Department of Radiodiagnosis, Alexandria University; ⁵Internal Medicine, Ministry of Health, Alexandria, Egypt

Background: Organ-specific manifestations in systemic lupus erythematosus (SLE) are highly influenced by the inherent characteristic of the vasculature⁽¹⁾ Endothelial dysfunction might have significant roles in the pathogenesis of glomerular diseases. CD31 or platelet endothelial cell adhesion molecule-1 is known to have roles in angiogenesis, platelet function, thrombosis, and regulation of leukocyte migration through vascular walls⁽²⁾

Objectives: to assess the endothelium dysfunction in SLE patients (pts) and to determine its association with lupus nephritis (LN)

Methods: Study included 60 pts with SLE fulfilling the SLICC criteria for classification of SLE: 30 with (LN), 30 SLE without LN and 10 matched healthy controls. Routine laboratory tests, lipid profile, renal functions, titres of autoantibodies [ANA, anti ds-DNA, anticardiolipin antibodies ACL(IgM, IgG)], flowcytometry to detect PECAM-1/CD31, doppler ultrasound to detect FMD of the brachial artery and SLEDAI were done to all SLE pts. Pts with diseases known to affect endothelial function: smoking, DM, essential HTN, known CAD were excluded

Results: Correlation between FMD% and different parameters are shown in table 1. There was a statistical significant positive relation between CD31 and urea in LN (p=0.014), non LN (p=0.014), total SLE pts (p<0.001), creatinine in non LN group

(p=0.025), 24 hour protein in LN (p<0.001), non LN (p<0.001) and total SLE pts (p<0.001), cholesterol in LN (p<0.001), non LN (p=0.001) and total SLE pts (p<0.001), LDL in LN (p=0.001), non LN (p=0.009) and total SLE pts (p<0.001), TG in total pts (p=0.048), anti-dsDNA in LN (p=0.024) and total SLE pts (p=0.008), SLEDAI in LN (p=0.001) and total SLE pts (p<0.001).

	LN		FMD% Non LN		Total patients	
	r	p	r	p	r	p
Demographic data						
Age	0.033	0.862	0.275	0.141	0.057	0.666
D. duration	-0.730*	<0.001*	-0.574*	0.001*	-0.332*	0.010*
Renal function						
Urea	-0.771*	<0.001*	-0.689*	<0.001*	-0.731*	<0.001*
Creatinine	-0.084	0.657	0.196	0.300	-0.324*	0.012*
Creatinine clearance	0.084	0.660	-0.024	0.898	0.200	0.126
24 hour Urine protein	-0.543*	0.002*	-0.392*	0.032*	-0.631*	<0.001*
Lipid profile						
Cholesterol	-0.438*	0.016*	-0.534*	0.002*	-0.530*	<0.001*
LDL	-0.837*	<0.001*	-0.779*	<0.001*	-0.766*	<0.001*
Triglyceride	-0.252	0.179	0.086	0.652	-0.468*	<0.001*
Immunological profile						
Anti DNA	-0.517*	0.003*	-0.648*	<0.001*	-0.565*	<0.001*
SLEDAI	-0.792*	<0.001*	-0.853*	<0.001*	-0.676*	<0.001*

Fig 1 The performance of FMD to detect LN was better than anti-dsDNA and than CD31, where FMD was 80% sensitive and 100% specific at cut off point >189 >49%

ROC curve for anti-dsDNA, CD31 % and FMD % to detect LN in SLE pts

Abstract SAT0452 – Figure 1. ROC curve for anti-dsDNA, CD31% and FMD% to detect LN in SLE pts

Conclusions: Endothelial dysfunction is the first step in the atherogenic process. FMD and CECs may be used as markers for endothelial dysfunction in SLE. FMD correlated with proteinuria and may be used as an early marker for LN. CECs may play a role in atherosclerosis, vasculitis and LN

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