Figure 2. (a) SLE+AS mice had significantly higher serum PCSK9 concentrations than mice in AS group and C57BL/6 mice; (b)-(c) Expression of PCSK9 was interfered via injection of lentiviruse-PCSK9 small hairpin RNA targeting the mouse PCSK9 gene into the tail vein. Compared with SLE+AS group(b), SLE+AS+PCSK9i group(c) exhibits bright green fluorescence in atherosclerotic plaque; (d)-(f) Immunohistochemistry showed that PCSK9 overexpression was observed in SLE+AS mice than those in AS group and SLE+AS+PCSK9i group. Mice in SLE+AS+PCSK9i group showed alleviated inflammatory cell infiltration and atherosclerotic lesion than those in SLE+AS group and AS group(d-f); Immunohistochemistry of PCSK9, $\times 200$; g-i: Oil red O stain, $\times 40$; (j)-(l) Mice in SLE+AS+PCSK9i group exhibited lower serum TNF- α and IL-1 β levels and attenuated expression of TLR4 and NF- κ B p65 in atherosclerotic plaque than those in SLE+AS group; (m)-(o) PCSK9 silencing had no significant effect on lipids parameters(TCHO, LDL-C, TG and HDL-C) in SLE+AS mice($p>0.05$).

Conclusion: PCSK9 participates in atherogenic inflammation in SLE patients and murine model of SLE with AS. This process is associated with TLR4-NF- κ B pathway, probably independent of lipids parameters alteration. HCQ can effectively reduce PCSK9 levels in SLE patients.

REFERENCES:

[1] Sabatine MS, et al. N Engl J Med. 2017, 376(18):1713-1722.
[2] Ni JQ, et al. Clin Dev Immunol. 2013;2013:476856.

Disclosure of Interests: None declared
DOI: 10.1136/annrheumdis-2019-eular.2943

OP0299

ASSESSMENT OF ROLE OF URINARY HEPARANASE IN LUPUS NEPHRITIS PATIENTS AND ITS CORRELATION WITH DISEASE ACTIVITY

Mustafa Abou-Alfa^{1,2}, Rasha Abdel Noor¹, Hala Nagy³, Nesreen Kotb¹. ¹Tanta University, Internal Medicine, Tanta, Egypt; ²GOTHI, Internal Medicine Nephrology unit, Cairo, Egypt; ³Tanta University, Clinical Pathology Department, Tanta, Egypt

Background: Lupus nephritis (LN) is one of the most common and serious complication of Systemic Lupus Erythematosus (SLE) and assessment of its activity is crucial. Heparanase has been proposed to be important in the pathogenesis of proteinuria in various forms of glomerulonephritis.

Objectives: To assess the ability of urinary heparanase to identify SLE with nephritis and the relation of this marker to lupus activity.

Methods: This cross sectional study was carried out on 90 subjects; 70 patients with SLE and 20 healthy volunteers as a control. All patients and controls were subjected to full history taking and complete clinical examination, routine investigations. Immunological assay and assessment of disease activity by systemic lupus erythematosus disease activity index (SLEDAI) score, renal SLEDAI (r-SLEDAI) were done for lupus nephritis group. The lupus patients were divided according to SLEDAI score into 4 groups; 20 with active lupus nephritis, 17 with non-active LN, 18 with active lupus without renal involvement, 15 with non-active lupus without renal involvement. Urinary Heparanase levels were measured by using ELISA for all groups.

Results: The level of urinary heparanase was significantly higher in LN groups than non-LN groups and control. It was also significantly higher in active LN than non-active LN patients. There was a significant positive correlation between urinary heparanase and 24 hours urinary proteins, total SLEDAI, and r-SLEDAI, and significant negative correlation between urinary heparanase and C3 & C4. ROC curve analysis revealed that urinary heparanase predicted presence of lupus nephritis activity with a sensitivity of 80%, and a specificity of 91.43%.

Conclusion: Urinary heparanase levels are increased in patients with active LN and correlate with disease activity markers, indicating that it can serve as a new useful biomarker for lupus nephritis activity.

REFERENCES:

[1] Kim K-J, Kim J-Y, Park S-J, Baek I-W, Yoon C-H, Kim W-U, et al. SAT0227 Urinary heparanase activity is elevated in patients with lupus

nephritis and correlate with protein excretion. Annals of the Rheumatic Diseases. 2013;71(Suppl 3):548-9.
[2] Cho C, Kim K, In-Woon B. 206 Increased urinary heparanase levels are associated with active lupus nephritis. Archives of Disease in childhood; 2017.

Table (1). : Comparison between the different studied groups regarding HPSE (ng/ml)

	Group I Active LN (n=20)	Group II Non active LN (n=17)	Group III Active lupus Without nephritis (n=18)	Group IV Non-active lupus Without nephritis (n=15)	Group V Control (n=20)	F	P
HPSE (ng/ ml)	2.36 $\pm$ 0.62	1.44 $\pm$ 0.11	1.23 $\pm$ 0.07	1.31 $\pm$ 0.07	0.29 $\pm$ 0.13	115.716 [*]	<0.001 [*]
p ₁	<0.001 [*]	<0.001 [*]	<0.001 [*]	<0.001 [*]			
Sig. bet.	p ₂ <0.001 [*] , p ₃ =0.914, p ₄ <0.001 [*] , p ₅ =0.719, p ₆ <0.001 [*] , p ₇ =0.230						
Grps							

F: F for ANOVA test, Pairwise comparison bet. each 2 groups was done using Post Hoc Test (Tukey)

- p: p value for comparing between the different groups
- p₁: p value for comparing between group V and each other group
- p₂: p value for comparing between group I and group II
- p₃: p value for comparing between group III and group IV
- p₄: p value for comparing between group I and group III
- p₅: p value for comparing between group II and group IV
- p₆: p value for comparing between group I and group IV
- p₇: p value for comparing between group II and group III

Table 2. Agreement (sensitivity, specificity) for HPSE (ng/ml) to diagnose active lupus nephritis

	Cut off	Sensitivity	Specificity	PPV	NPV
HPSE (ng/ ml)	>1.48	80.0	91.43	72.7	94.1
HPSE (ng/ml)					
AUC				0.877	
p				<0.001 [*]	
95% C.I				0.771 – 0.982

Disclosure of Interests: None declared
DOI: 10.1136/annrheumdis-2019-eular.1616

OP0300

A NEUTROPHIL SIGNATURE IS STRONGLY ASSOCIATED WITH CARDIOVASCULAR RISK IN GOUT

Daisy Vedder¹, Martijn Gerritsen¹, Michael Nurmohamed¹, Ronald van Vollenhoven², Christian Lood³. ¹Amsterdam Rheumatology and Immunology center, location Reade, Amsterdam, Netherlands; ²Amsterdam Rheumatology and Immunology center, Amsterdam UMC, Amsterdam, Netherlands; ³University of Washington, Department of Medicine, Division of Rheumatology, Seattle, United States of America

Background: Similar to other rheumatic diseases, patients with gout have an increased cardiovascular morbidity and mortality, not fully explained by traditional cardiovascular risk factors. Instead, gout-specific factors, including xanthine oxidase induced oxidative stress, increased lipid oxidation and chronic low-grade inflammation, have been suggested as important contributors.(1) Neutrophils, through formation of neutrophil extracellular traps (NETs), partake in pro-