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ELEVATED CYSTEINE-RICH PROTEIN 61 IN SYSTEMIC LUPUS ERYTHEMATOSUS-ASSOCIATED PULMONARY ARTERIAL HYPERTENSION

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Background and aims Previous study has demonstrated Cysteine-rich protein 61(Cyr61) was highly expressed in systemic lupus erythematosus(SLE) patients. However, the role of Cyr61 in pulmonary arterial hypertension (PAH) remains unknown. This study aimed to explore the value of Cyr61 for PAH in SLE patients by comparing the plasma Cyr61 levels in SLE patients with/without PAH.

Methods Plasma samples from two tertiary medical centers were obtained from 54 patients with definite SLE-PAH, 52 age, gender and SLEDAI matched SLE patients without PAH, and 54 age and gender matched healthy controls. Plasma Cyr61 concentration was measured by enzyme-linked immunosorbent assay.

Results Plasma Cyr61 concentration in SLE-PAH patients was significantly higher than the matched SLE patients and healthy controls (median(IQR): 172.5 (143.8, 218.2), 124.9 (104.1, 154.7), 58.17 (28.9, 80.4) respectively, $P < 0.001$) (Figure 1). The sensitivity and specificity of Cyr61 in predicting the presence of PAH in entire SLE patients were 79.6% and 67.3%. Receiver operating characteristic curve analysis showed the area under the curve was 0.757 (95% CI: 0.662~0.852), with 140.6 pg/ml as the cutoff concentration (Figure 2). Further multivariate logistic regression analyses revealed high Cyr61 level (>140.6) is an independent risk factor for SLE patients to develop PAH (OR: 7.822 (95% CI: 2.224~41.138)) (Table 1). Additionally, weak to moderate positive correlations were observed between Cyr61 concentration and serositis,

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haematological involvement, red blood cell distribution width, right ventricular systolic pressure and right ventricular diameter measured by echocardiography in entire SLE population.

Conclusions Plasma Cyr61 level was significantly higher in SLE-PAH patients than SLE patients without PAH. Cyr61 may be used as a biomarker for PAH complication in SLE patients.

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ANTIBODIES TOWARDS ATP-BINDING CASSETTE TRANSPORTER ABCA1: A NEW MECHANISM FOR ATHEROSCLEROSIS IN SLE?

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Background and aims Systemic Lupus Erythematosus (SLE) is considered an independent risk factor for cardiovascular disease. ATP-binding cassette transporter ABCA1, also known as the cholesterol efflux regulator protein, is a ubiquitous cholesterol transporter that is highly expressed in macrophages. Its main function is to donate cholesterol to apolipoprotein A-I in lipid-poor HDL particles. As such, ABCA1 closely influences HDL levels and its role in atherosclerosis has been increasingly studied.

This study was undertaken to determine if antibodies against ABCA1 can be detected in patients with SLE.

Methods Serum from 48 patients were compared with an age and sex-matched control group. Patients were divided in groups A (13 patients) and B (35 patients), respectively with up to 3 and at least 4 SLICC classification criteria (2012). IgG anti-ABCA1 and anti-HDL antibodies were assessed by home-made ELISAs. Plasma lipid profile was determined by standard enzymatic techniques.

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