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PLASMA THROMBOSPONDIN-1 LEVELS ARE ASSOCIATED WITH ARTERIAL AND VENOUS THROMBOTIC EVENTS IN SYSTEMIC LUPUS ERYTHEMATOSUS

¹Marina Barguil Macedo, ²Iva Gunnarsson, ²Agneta Zickert, ²Elisabet Svenungsson, ¹Christian Lood. ¹Division of Rheumatology, University of Washington, Seattle, USA; ²Division of Rheumatology, Karolinska Institutet, Stockholm, Sweden

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Background Thrombospondin-1 (TSP-1) is the most abundant protein inside platelet alpha-granules, being secreted upon their activation. The role of TSP-1 in thrombosis and hemostasis remains dubious, with conflicting data showcasing both protective and harmful mechanisms. As young patients with systemic lupus erythematosus (SLE) present a markedly heightened risk of MI, we aimed to assess plasma (p) levels of TSP-1 in order to discern their relevance as a marker for atherothrombosis. We further evaluated the correlation with venous thrombosis (VT), and specifically with pulmonary embolism (PE).

Methods TSP-1 levels were measured in plasma samples (SLE = 308, population controls (PC) = 308) from the Karolinska Biobank by a commercial ELISA kit (R&D Cat# DY3074), according to the manufacturer's instructions. The 95th percentile of PC was used as a cut-off for high levels. Mann-Whitney U-test and Fisher's exact test were used, with a significance level of $p < 0.05$.

Results Levels of pTSP-1 were higher in SLE samples (mean of 12.3 ug/mL) as opposed to PC (mean of 10.1 ug/mL). When adopting the 95th percentile of healthy controls (19.23 ug/mL), a clear differentiation was evidenced ($p < 0.001$), with 19% of SLE samples above the upper percentile. Absolute values of pTSP-1 were significantly higher in SLE patients with a history of MI ($p = 0.017$) or PE ($p = 0.028$), as compared to patients without those manifestations. Further, high levels of pTSP-1 were associated with past history of VT ($p = 0.038$) in SLE. Our results are in contrast to previous works, which showed either reduction or no difference between levels in SLE versus in HC.

Conclusions In a large cohort of well-characterized SLE patients, pTSP-1 levels were found to be elevated, and associated with a history of MI or PE. The strength of our work relies on our robust sample size, well-characterized patient

cohort, as well as inclusion of population cohort (instead of healthy controls). Future work aims to determine whether levels of pTSP-1 can predict development of thrombotic event in SLE.

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TIMING OF THROMBOEMBOLIC EVENTS IN SYSTEMIC LUPUS ERYTHEMATOSUS ASSOCIATED TO ANTIPHOSPHOLIPID SYNDROME; RESULTS FROM A POPULATION-BASED STUDY SET IN NORWAY

^{1,2}Sigrid R Moe, ^{1,2,3}Hilde Haukeland, ¹Torild Garen, ⁴Antonela Botea, ⁵Anniken Orre, ⁶Heidi Øvreås, ⁷Thea Bøe, ⁸Gro Å Wivestad, ⁹Nenad Damjanic, ¹⁰Cathrine Brunborg, ^{11,12}Sella A Provan, ^{1,2}Øyvind Molberg, ¹Karoline Lerang. ¹Dept. of Rheumatology, Oslo University Hospital, Oslo, Norway; ²Institute of Clinical Medicine, University of Oslo, Oslo, Norway; ³Dept. of Rheumatology, Martina Hansens Hospital, Gjøttum, Norway; ⁴Dept. of Rheumatology, Betanien Hospital, Skien, Norway; ⁵Dept. of Rheumatology, Vestre Viken Hospital Trust, Drammen, Norway; ⁶Dept. of Rheumatology, Lillehammer Hospital for Rheumatic Diseases, Lillehammer, Norway; ⁷Dept. of Internal Medicine, Vestfold Hospital Trust, Tonsberg, Norway; ⁸Division of Rheumatology, Dept. of Medicine, Hospital of Southern Norway Trust, Kristiansand, Norway; ⁹Dept. of Rheumatology, Ostfold Hospital Trust, Graalum, Norway; ¹⁰Oslo Centre for Biostatistics and Epidemiology, Research Support Services, Oslo University Hospital, Oslo, Norway; ¹¹Center for treatment of Rheumatic and Musculoskeletal Diseases (REMEDY), Diakonhjemmet Hospital, Oslo, Norway; ¹²Section for Public Health, Innland Norway, University of Applied Sciences, Hamar, Norway

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Objective To estimate occurrence of thromboembolic event in a population-based setting and compare characteristics of new-onset Systemic Lupus Erythematosus (SLE) with and without Antiphospholipid Syndrome (APS).

Methods We included all new-onset SLE cases residing in the Southeast Norway area 1999–2017. Follow-up ended 31 December 2017. All cases had diagnosis confirmed by chart-review, fulfilled the 2019 EULAR/ACR classification criteria and were captured within one year of diagnosis.

All APS fulfilled the 2006 Sapporo classification criteria. Arterial thromboembolic events included ischemic stroke, transient ischemic attack, myocardial infarction or angina pectoris identified either by individual-level chart-review or by ICD-code (G45–46, I63 excluding I63.6, I20–I25, R96) in The National Cause of Death Register. Venous thromboembolic events included syndromes caused by occlusion of major venous vessel identified by chart-review. We estimated incidence rates per 100 person-years at risk and 95% confidence intervals (CI) using Poisson distribution.

Results Of the 749 cases with new-onset SLE 1999–2017, 84 (11%) had coexisting APS. APS cases were more prone to develop thrombocytopenia, neuropsychiatric disease and anti-dsDNA positivity during the disease course than cases without APS (table 1).

By the end of follow-up, 174 (23%) cases had ever experienced at least one arterial or venous event (TE). APS cases were younger at their first TE than those without APS (mean age 37 versus 59, p -value < 0.001). TE tended to coincide with SLE diagnosis in APS cases (figure 1).

In the 675 cases without previous TE at SLE diagnosis, 38 and 69 TE occurred within one year and five years disease duration. Overall 5-year incidence rate for TE was 2.6 (95% CI 2.0–3.3). APS cases exhibited a high incidence of TE the first year after SLE diagnosis (51.1, 95% CI 33.4–74.8), that decreased to 8.6 (95% CI 4.7–14.7) one to five year after SLE diagnosis. Cases without APS had a considerable lower first-year incidence of TE (2.3, 95% CI 1.4–3.8) and the