INTRODUCTION Clinical activity of SLE may wax and wane, but persistent, active systemic inflammation leads to organ damage and rises morbidity and mortality. Early damage is mostly related to disease activity, whereas later damage, in particular atherosclerosis, infections and malignancies are usual complications of long-standing disease and treatment with immunosuppressive agents. One of the major late causes of death in SLE is thrombosis, in particular stroke and myocardial infarction due to CAD. In these patients, the increased cardiovascular morbidity is not fully explained by traditional risk factors and this may lead to under-recognition and under-treatment. Petri, et al. proposed an equation for cardiovascular disease risk in SLE, which combines classical parameters and disease activity markers. Other scores such as the GAPSS (Global AntiPhospholipid Syndrome Score) have been recently evaluated. The importance of aPL in thrombosis in general is well defined, as they constitute the culprit of the so-called anti-phospholipid syndrome (APS). Their role in sustaining the high risk of cardiovascular complications of SLE patients is under-debated.

OBJECTIVE To study the role of the anti-phosphatidylserine/prothrombin (aPS/PT) antibodies, included in the GAPSS score, in contributing to the thrombotic risk of SLE patients.

METHODS We enrolled 172 patients from Ospedale San Raffaele. 132 patients with SLE (111/132, 84% without secondary APS, SAPS, and 21/132, 16% with SAPS), 19 with primary APS (PAPS) and 21 healthy controls. Each recruited patient was tested for aPS/PT IgG and IgM through ELISA by INOVA Diagnostic, Inc. San Diego, CA USA.

RESULTS 36/111 (32.4%) SLE without APS, 15/21 (71.4%) SAPS, 13/19 (68.4%) PAPS and 3/21 (14.3%) healthy donors were aPS/PT+. aPS/PT+SLE patients had a higher cardiovascular risk according to the Petri’s score, when compared to aPS/PT-patients, irrespectively of a positive or negative history of overt APS (Mean ±SD Petri’s score=20.8±18.1, 14.0±12.8 and 23.8±22.5, 11.6±9.3 respectively, p<0.05). Accordingly, the GAPSS score was significantly higher in APS patients than in APS negative patients with SLE (12.1±5.7, 11.5±4.6 and 4.9±4.9 respectively, p<0.001). Patients with a GAPSS score >10 had also higher prevalence of pregnancy complications.

CONCLUSION aPS/PT antibodies are associated with a high risk of thrombosis and CAD in SLE. aPS/PT assays should be routinely introduced in the management of these patients.