with the presence of a cardiovascular risk factor (p=0.04) while CD was associated with anxiety and depression in at risk individuals (p=0.047). A relationship between CD and level of education, gender and current work was also observed.

Conclusions In this exploratory study we identified an association between conventional cardiovascular risk factors and cognitive dysfunction. However there was no association between any of the immune parameters and MoCA score. Prevention of cognitive dysfunction in SLE should focus on early identification and treatment of cardiovascular risk.

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REFERENCES

Background TWEAK, MCP-1 and NGAL, mediators in pathogenesis of systemic lupus erythematosus (SLE), are proinflammatory cytokines/chemokines that are thought as potential biomarkers reflecting disease activity. In this study, we aimed to investigate the association of serum (s) and urine (u) levels of TWEAK, MCP-1 and NGAL with disease activity in both renal and non-renal SLE.

Methods Thirty active patients with SLE (15 renal and 15 non-renal) were recruited. Thirty-one inactive patients with SLE (16 renal and 15 non-renal), 14 patients with ANCA-associated vasculitis (AAV) all of whom had active renal involvement and 20 healthy volunteers were selected as control groups. Serum and urine levels of TWEAK, MCP-1 and NGAL were tested using ELISA.

Results Sixty-one SLE patients, 51 (83.6%) of whom were female, with a median disease duration of 83 (23.5–135) months and a median age of 35 (27–47.5) were included in the study. Serum and urine levels of TWEAK and NGAL were significantly higher in the active SLE group compared with the inactive SLE (n=31) group (sTWEAK: p=0.005; uTWEAK: p=0.026; sNGAL: p<0.001; uNGAL: p=0.002); whilst no significant differences regarding serum and urine MCP-1 levels were observed (p=0.189 and p=0.106). sTWEAK (p=0.237), sMCP-1 (p=0.141), uMCP-1 (p=0.206), sNGAL (p=0.419) and uNGAL (p=0.443) levels did not differ between patients with active LN and non-renal active SLE; yet levels of sTWEAK were higher in patients with active LN (p=0.006). There were no differences between active LN and renal active AAV. Levels of all biomarkers were correlated with SLEDAI (sTWEAK: p=0.001; uTWEAK: p=0.006; sMCP-1: p=0.049; uMCP-1: p=0.014; sNGAL: p<0.001; uNGAL: p=0.002).

Conclusions sTWEAK, uTWEAK, sNGAL and uNGAL are significant biomarkers showing disease activity in SLE. However, our results implicate that these biomarkers may not be specific for SLE, and can be elevated in patients with active renal involvement of AAV. sTWEAK may be of use for discriminating active nephritis from non-renal active disease in SLE. Further studies are awaited to confirm these results.

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Abstracts