Cerebral small vessel disease (SVD), including white matter hyperintensities (WMH) and lacunes of presumed vascular origin, affects gait by disrupting important white matter tracks. However, little is known about the relationship between the structural network connectivity and gait in subjects with cerebral SVD, and the interaction with cognitive function. We assessed gait characteristics (stride length, cadence and stride width, as well as Tinetti and Timed-Up-and-Go test) of 423 subjects with cerebral SVD. The structural network was constructed using diffusion tensor imaging and tractography. We applied graph-theory to calculate network efficiency from the undirected weighted network. Associations between network measures, conventional MRI markers for SVD, gait performances and cognitive index as global cognitive function were tested. Network measures were associated with conventional MRI markers for SVD (WMH and lacunes) and with gait performances. Stride length, Tinetti and Timed-Up-and-Go test were associated with network measures, independent of WMH or lacunes. However, after adjusting for cognitive index, these associations diminished and were not significant anymore. In the mediation analyses, cognitive index mediated the association between gait performance and network measures, independent of WMH or lacunes. Regional analysis showed widespread involvement of cortical regions in gait performance, including frontal motor, cingulate and visuospatial regions, which diminished after adjusting for cognitive index. These results suggest that measures for network disruption were associated with gait performance, possibly indirectly by disrupting brain network responsible for cognitive function and hereby impairing gait performance.

**ESOC-0630**

**23. Small Vessel Disease**

Decreased kidney function is associated with progression of cerebral microbleeds in lacunar stroke patients

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**Introduction:** Cross-sectional studies found impaired kidney function to be associated with the presence of cerebral microbleeds in stroke patients. It is hypothesized that both impaired kidney function and cerebral microbleeds represent small vessel disease in different organs. Our aim was to determine whether kidney function is related to progression of cerebral microbleeds to further confirm the association in a longitudinal study design.

**Methods:** In 117 lacunar stroke patients, in whom baseline brain MRI (including gradient echo images) and estimated glomerular filtration rate (eGFR) were available, we obtained a follow-up brain MRI after 2 years. eGFR was calculated using the Cockcroft-Gault equation. Presence of cerebral microbleeds on baseline and follow-up MRI was scored visually and progression of microbleeds was defined as the presence of any new microbleed on follow-up MRI. The relationship between progression of cerebral microbleeds (dependent variable) and eGFR (independent variable) was assessed by logistic regression analysis adjusting for age, sex and 24-hour ambulatory mean arterial pressure (MAP).

**Results:** Progression of cerebral microbleeds was present in 21 patients (17.9%). In binary logistic regression analysis lower eGFR was associated with progression of cerebral microbleeds (OR 1.37 per 10 ml/min/1.73 m², 95% CI 1.03–1.82, with correction for age, sex and MAP).

**Conclusion:** We found an association between lower eGFR and progression of cerebral microbleeds. Cerebral microbleeds and chronic kidney disease are both seen as manifestations of microvascular organ damage and our findings support the assumption that small vessel disease should be considered as a multi-system disorder.