Banerjee and Werring published a single centre recruitment experience for a small biomarker pilot study, which aimed to recruit 10 patients with CAA.

The BOCAA (Biomarkers and Outcomes in Cerebral Amyloid Angiopathy) study recruited 10 CAA patients over 18 months. All patients were recruited from a prospective CAA database (n = 186); the majority of patients (n = 146, 78.5%) were ineligible for the BOCAA study. The most common reasons for exclusion were co-existent cognitive impairment or dementia (n = 42), failure to meet the imaging (modified Boston) criteria (n = 41), and anticoagulant or dual antiplatelet use (n = 18).

Recruitment of CAA patients to a small pilot study is feasible from a single specialist centre; however, centralised multicentre research databases will allow for more effective and co-ordinated recruitment to larger studies. Any future trial will need to consider how best to define mild disease, factors that influence group heterogeneity, and the impact of comorbidities that could limit participation in multimodal testing - but be mindful that more stringent entry criteria will limit recruitment capabilities [1].

Banerjee et al., describe in detail the clinical and neuroimaging findings in three patients with early-onset symptomatic amyloid-β cerebral amyloid angiopathy following childhood exposure to cadaveric dura (by neurosurgical grafting in two patients, and tumour embolization in a third). The observations provide further in vivo evidence that cerebral amyloid angiopathy might be caused by transmission of amyloid-β seeds (prions) present in cadaveric dura, and has diagnostic relevance for younger patients presenting with suspected cerebral amyloid angiopathy [2].

Thirty-three patients with probable CAA underwent multimodal brain magnetic resonance imaging at 2 time points (mean follow-up time: 1.3±0.4 years). Brain networks of the hemisphere free of intracerebral hemorrhages were reconstructed using fiber tractography and graph theory. The global efficiency of the network and mean fractional anisotropies of posterior-posterior, frontal-frontal, and posterior-frontal network connections were calculated. Patients with moderate versus severe CAA were defined based on microbleed count, dichotomized at the median (median=35).

Global efficiency of the intracerebral hemorrhage-free hemispheric network declined from baseline to follow-up (-0.008±0.003; P=0.029). The decline in global efficiency was most pronounced for patients with severe CAA (group×time interaction P=0.03). The decline in global network efficiency was associated with worse executive functioning (β=0.46; P=0.03). Examination of subgroups of network connections revealed a decline in fractional anisotropies of posterior-posterior connections at both levels of CAA severity (-0.006±0.002; P=0.017; group×time interaction P=0.16). The fractional anisotropies of posterior-frontal and frontal-frontal connections declined in patients with severe but not moderate CAA (group×time interaction P=0.007 and P=0.005). Associations were independent of change in white matter hyperintensity volume.

Brain network impairment in patients with CAA worsens measurably over just 1.3-year follow-up and seem to progress from posterior to frontal connections with increasing disease severity [3].

1)

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2)

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[3)]

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