High field in vivo Magnetic Resonance Imaging of lenticulostriate arteries in CADASIL

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Background Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is a hereditary form of small vessel disease. Although postmortem studies have demonstrated wall thickening in leptomeningeal and lenticulostriate perforating arteries,[1] it is unclear whether wall thickening also leads to luminal narrowing in these vessels.[2] High-field MRI scanners enable high resolution imaging of the lenticulostriate arteries.[3,4] The aim of this study is to examine the luminal diameters of lenticulostriate arteries in living CADASIL patients and to investigate whether luminal narrowing of these arteries is associated with the number of lacunar infarcts in the basal ganglia.
Methods 22 NOTCH3 mutation carriers (MCs) and 11 healthy controls were examined. MRI was performed on a whole body human 7T MR system (Philips Healthcare, Best, the Netherlands), equipped with a quadrature birdcage transmit and 16-channel receive array head coil. 3D time-of-flight magnetic resonance angiography (TOF MRA) of the circle of Willis and lenticostriate arteries was performed using the following protocol: 3D RF-spoiled gradient echo images with a scan duration of 10:41 minutes per scan; repetition time (TR)/ echo time (TE)/ flip angle (FA) = 16 ms / 4.3 ms / 30°, 161 slices, 180 x 170 mm field of view, 784/737 matrix size - resulting in a nominal resolution of 0.23 x 0.23 x 0.23 mm.. The total number, length and total cross-sectional area of lenticulostriate arteries were measured and compared between mutation carriers and controls using Mann-Whitney U tests. These measurements were correlated with age, disease duration and number of lacunar infarcts in the basal ganglia.
Results Figure 1 shows coronal MIPs from a CADASIL patient and control. Table 1 lists the properties of the lenticostriate arteries. No differences between MCs and controls were found in length, total number or total cross-sectional area of lenticostriate artery lumina ($p > 0.05$). Measurements of lenticostriate artery lumina were not associated with lacunar infarct load in the basal ganglia area ($p > 0.05$).

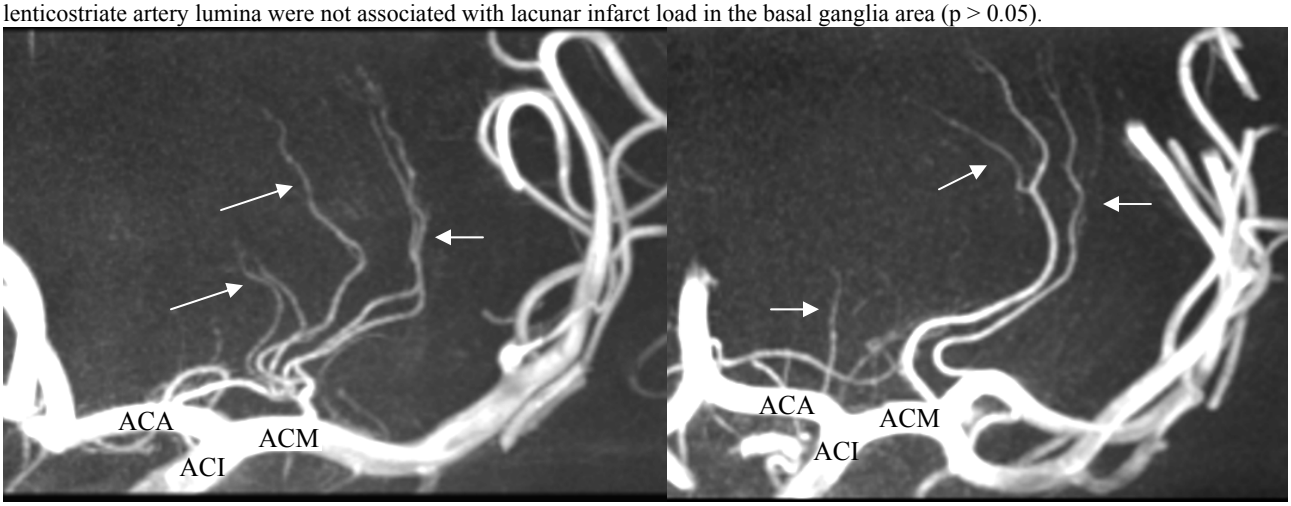

Figure 1. Coronal MIP of a 3D-TOF MRA in a CADASIL patient (left) and a control subject (right) of similar age, showing the lenticulostriate arteries (arrows). ACM - medial cerebral artery, ACI – internal carotid artery, ACA – anterior cerebral artery.

Table 1. Lenticostriate arteries in NOTCH3 MCs and controls

	CADASIL (n = 22)	Control (n = 11)	p-value
Total number of end branches (mean, SD)	14.6 (5.8)	12.8 (5.4)	0.31
Length of lenticostriate system (mm)(mean, SD)	21.9 (4.1)	22.4 (4.2)	0.71
Number of arteries at 5 mm (mean, SD)	10.2 (4.2)	9.2 (2.8)	0.52
Number of arteries at 10 mm (mean, SD)	8.0 (3.2)	7.6 (2.6)	0.74
Number of arteries at 15 mm (mean, SD)	6.0 (3.0)	6.4 (2.4)	0.69
Number of arteries at 20 mm (mean, SD)	3.0 (2.0)	3.4 (2.6)	0.63
Number of arteries at 25 mm (mean, SD)	0.6 (1.0)	1.0 (2.0)	0.32
Total cross-sectional area of arteries at 10 mm (mm^2)(mean, SD)	1.26 (0.50)	1.08 (0.42)	0.31

Conclusions High-resolution 3D TOF MRA on 7-Tesla shows that CADASIL patients have normal luminal diameters of lenticostriate arteries, suggesting that the mechanism leading to lacunar infarcts in the basal ganglia is more likely based on hemodynamic disturbances than on luminal narrowing.

References:
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