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Involvement of the recurrent artery of Heubner with contralateral middle cerebral artery infarction

An 86-year-old Chinese woman presented with left-sided weakness for 6 h. Her medical history included hypertension, ischaemic heart disease, atrial fibrillation and hypercholesterolaemia. She was conscious and communicative, with blood pressure 166/102 mm Hg and irregular pulse (102 beats/min). Examination showed right-gaze deviation

and left-hemiparesis (Medical Research Council power grade 0 and 2 in the upper and the lower extremity, respectively, National Institute of Health Stroke Scale 11 points).

Computed tomography of the brain showed hyper-dense right middle cerebral artery (MCA) and early ischaemic changes in its territory (fig 1A,B).

Transcranial Doppler sonography showed right MCA occlusion with flow diversion to the ipsilateral anterior cerebral artery (ACA). Low flow velocity in the contralateral A1-ACA suggested its hypoplastic nature.

After 2 days, she developed increased weakness in the left-lower extremity (Medical Research Council power grade 0), transient choreiform movements of the right upper extremity, followed by right hemiparesis (power grade 3, National Institute of Health Stroke Scale 19 points) and new speech difficulties (non-fluent aphasia, syntax errors, repetition impairment and word-finding difficulty). Repeated brain computed tomography showed an established right MCA infarction, brain swelling with mild midline shift and subfalcine herniation (fig 1C). New infarcts were noted in the right ACA territory, left caudate head and internal capsule.

In subfalcine herniation, the expanding hemisphere forces the ipsilateral cingulate gyrus under the falx-cerebri, compressing the ipsilateral ACA. Recurrent artery of Heubner is an inconstant branch of ACA, usually arising just beyond the anterior communicating artery. It supplies blood to the anteromedial part of the head of the caudate nucleus and the anteroinferior internal capsule. In patients with hypoplastic A1-ACA, both A2 segments and their branches are perfused by one patent ACA. In our patient, subfalcine herniation caused compression of the right ACA and compromised the left recurrent artery of Heubner, with resultant infarctions in the corresponding territories.

Figure 1 Temporal evolution in right middle cerebral artery (MCA) infarct. Non-enhanced computed tomography scan of the brain performed 6 h after symptom onset, showing a dense right MCA (A) together with early ischaemic changes in its territory (B). Brain computed tomography scan repeated after 3 days (C) shows well-defined hypodensity in the right MCA. A mass-effect and subfalcine herniation (curved arrow) are seen with resultant infarctions in the territories of the ipsilateral anterior cerebral artery (ACA) and contralateral recurrent artery of Heubner. A schematic diagram (D) shows the absent left A1-ACA; both recurrent arteries of Heubner receive perfusion from the right A1-ACA.

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