Diffusion-Weighted Imaging in Stroke Attributable to Internal Carotid Artery Dissection

The Significance of Vessel Patency

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Background and Purpose—In stroke attributable to spontaneous dissection of the internal carotid artery (sICAD), arterial patency may influence the pattern and extent of cerebral ischemia.

Methods—In 40 consecutive patients with stroke caused by sICAD, we compared the number, size and pattern of cerebral diffusion-weighted imaging lesions between patients with stenotic sICAD (n=15) and occlusive sICAD (n=25).

Results—Patients with stenotic sICAD had more ischemic lesions (median 5, interquartile range 1 to 10) than patients with occlusive sICAD (2, 1 to 3; P=0.014). Lesion diameters were larger in occlusive sICAD (62, 50 to 99 mm) than in stenotic sICAD (25, 10 to 50 mm; P=0.007). Border-zone infarction occurred only in stenotic sICAD (7/15, 47%). Most patients with occlusive sICAD had territorial infarcts (22/25, 88%).

Conclusions—In stroke attributable to sICAD, diffusion-weighted imaging characteristics may be influenced by the patency of the carotid artery. Differences in the pathogenesis of cerebral ischemia might exist between patients with stenotic and those with occlusive sICAD. (Stroke. 2008;39:483-485.)

Key Words: DWI ■ internal carotid artery dissection ■ occlusion ■ stenosis ■ stroke

Spontaneous dissection of the cervical internal carotid artery (sICAD) is an important cause of stroke in young adults.1 The intramural hematoma in sICAD may result in stenosis or complete occlusion of the ICA.2 The pattern and extent of cerebral infarction in sICAD may vary depending on arterial patency.

The aim of this exploratory study was to test whether characteristics of brain infarction assessed by diffusion-weighted imaging (DWI) differ between ICA-stenosis and ICA-occlusion caused by sICAD.

Subjects and Methods

Study Population and Diagnosis of sICAD

Fourty-four consecutive patients from 2 Swiss University hospitals (inclusion periods 84 and 93 months) were selected with: (1) First acute ischemic stroke attributable to sICAD, (2) sICAD diagnosed based on ≥1 of the following criteria: (i) intimal flap visible on carotid ultrasound (present in 11/43 patients); (ii) mural hematoma visible on MRI (33/43) or CT (3/5); (iii) a nonatherosclerotic, tapered, flame-shaped ICA-occlusion or a string-like ICA-stenosis. (3) DWI. (4) Assessment of ICA patency on carotid ultrasound (present in 11/43 patients); (ii) mural hematoma visible on MR angiography (MRA); n=43, CT angiography n=5, digital subtraction angiography (DSA) n=2.

ICA patency was dichotomized into stenotic sICAD and occlusive sICAD according to ultrasound and angiography (n ultrasound+angiography = 42, n ultrasound+angiography = 1, nDSA+MRA=1) results. In 4 patients (9%) ultrasound and angiography findings were inconsistent. They were excluded from further analyses.

Diffusion-Weighted Imaging

DWI was performed using previously published protocols.4,5 We assessed number, size (largest axial diameter), and vascular territories of all hyperintense DWI-lesions with hypo-/isointense appearance on apparent diffusion coefficient maps. The following 4 lesion patterns were distinguished, using a published classification system with mild modifications: pattern 1 (territorial infarct involving cortical and subcortical structures); pattern 2 (subcortical infarct in the territory of deep perforating branches); pattern 3 (multiple, mainly cortical lesions in the distal territory of the MCA); pattern 4 (border-zone infarction with lesions predominantly involving the junction of cerebral vascular territories).

Statistical Analysis

Number and diameter of DWI lesions were compared between groups with Mann-Whitney tests. Contingency between DWI-lesion patterns and patient groups was tested with Fisher exact test. P<0.05 was considered statistically significant. Data are expressed as median and interquartile range (IQR).

Results

Fifteen patients with stenotic sICAD and 25 patients with occlusive sICAD were included. Clinical findings are provided in the Table.
DWI was performed 5 (IQR, 2–7) days after stroke onset in patients with stenotic sICAD and after 2 (IQR, 0–4) days in occlusive sICAD (P = 0.024). The number of DWI-lesions was higher in patients with stenotic sICAD (5 [IQR, 1–10]) than in those with occlusive sICAD (2 [IQR, 1–3]; P = 0.014). Lesion diameters were larger in occlusive sICAD (62 mm [IQR, 50–99]) than in stenotic sICAD (25 mm [IQR, 10–50]; P = 0.007). The distribution of DWI-lesion patterns differed between the 2 groups (P < 0.001). Territorial infarcts (pattern 1) were present in 22/25 patients (88%) with occlusive sICAD, whereas most patients with occlusive sICAD had territorial infarcts. Border-zone infarction (pattern 4) exclusively occurred in stenotic sICAD (7/15, 47%; Table, and Figures 1 and 2).

**Discussion**

This exploratory comparison of DWI-lesion characteristics between stenotic and occlusive sICAD yielded the following results: (1) patients with stenotic sICAD had more DWI lesions than patients with occlusive sICAD; (2) DWI-lesions were larger in occlusive sICAD; and (3) border-zone infarction only occurred in stenotic sICAD, whereas most patients with occlusive sICAD had territorial infarcts.

Multiple acute DWI-lesions—a marker of embolism—were found in two thirds of our study population, supporting previous reports in sICAD.8,10 Our results therefore substantiate the concept of arterial thromboembolism in the pathogenesis of stroke in sICAD. As a novel observation, number and size of DWI lesions were influenced by ICA patency. Furthermore, border-zone infarction, which had been reported frequently in sICAD,8 was only seen in stenotic sICAD in our population. In turn, most patients with occlusive sICAD had territorial infarcts. The different distribution of DWI-lesion patterns between ICA stenosis and ICA occlusion resembles that of atherosclerotic ICA disease but has not been reported in sICAD.

Our findings might reflect differences in the pathogenesis of cerebral ischemia between stenotic and occlusive sICAD. Ischemia due to stenotic sICAD seems to be characterized by multiple small emboli, often located in border-zone territories where the clearance of emboli may be reduced.11 In contrast, the larger territorial infarcts in occlusive sICAD suggest single, large emboli. Future research should investigate whether the suggested pathogenetic importance of arterial patency in sICAD may translate into a potential role in treatment decisions. With this goal in mind, arterial patency should be assessed in ongoing and future therapeutic trials in sICAD.

The most important limitation of our study is the small sample size. Thus, our results should be considered as tentative only. Confirmation in a larger dataset is required. Moreover, the sample size disallowed analyses of associa-
tions between different degrees of luminal narrowing and DWI-lesion characteristics. Furthermore, the retrospective analysis of a databank population lead to heterogeneity in usage and timing of diagnostic tools. In addition, the distinction between severe ICA stenosis and ICA occlusion is challenging in sICAD. In order to minimize the risk of misclassification, all patients with inconsistent findings on ultrasound and angiography \( n = 4, 9\% \) were excluded.

In conclusion, our study suggested that characteristics of cerebral infarction in sICAD may be influenced by the patency of the carotid artery.

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**Disclosures**

None.

**References**

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