Stroke Occurrence and Patterns Are Not Influenced by the Degree of Stenosis in Cervical Artery Dissection

Olivier Naggara, MD, PhD; Audrey Morel, MD; Emmanuel Touzé; Jean Raymond, MD; Jean-Louis Mas, MD, PhD; Jean-Francois Meder, MD, PhD; Catherine Oppenheim, MD, PhD

Background and Purpose—In stroke attributable to spontaneous dissection of the cervical artery, it is unclear whether the occurrence and pattern of stroke depend on the degree of stenosis.

Methods—In 147 consecutive dissection of the cervical artery patients with (n=88) and without stroke (n=59), we compared the number, volume, and patterns of cerebral diffusion-weighted imaging stroke lesions among patients with <70% stenosis (Group 1), ≥70% stenosis (Group 2), and occlusion (Group 3).

Results—The presence (26 of 45 in Group 1, 32 of 59 in Group 2, 30 of 43 in Group 3; P=0.27) and the number of diffusion-weighted imaging lesions (mean±SD [interquartile range], 3.5±3.9 [1–4] in Group 1; 4.2±4.1 [1–5] in Group 2; 3.3±4.0 [1–3] in Group 3; P=0.85) were independent of lumen patency, whereas volume of diffusion-weighted imaging lesions was larger in occlusive dissection of the cervical artery (82±90 mm [17–91] versus 34±54 [2–48]; P=0.03). There were no differences in the breakdown of diffusion-weighted imaging lesion patterns according to degree of stenosis.

Conclusions—The occurrence and diffusion-weighted imaging lesion patterns in dissection of the cervical artery patients may not be influenced by the degree of stenosis of the dissected artery. Occlusive dissection of the cervical artery was associated with larger infarcts. (Stroke. 2012;43:1150-1152.)

Key Words: acute stroke ■ brain infarction ■ dissection ■ imaging ■ ischemia ■ MR angiography ■ stroke in young adults ■ stroke in young

Extracranial cervical artery dissection (CAD) accounts for nearly 20% of ischemic strokes in young adults.1 The mural hematoma in CAD may result in stenosis or occlusion of vertebral or carotid arteries. The aim of this study was to test whether occurrence and pattern of stroke on diffusion-weighted imaging (DWI) differ according to severity of arterial lesions.

Patients and Methods
We selected 172 acute (<7 days) CAD patients from a cohort of 5895 consecutive patients referred for suspected acute stroke or for primary stroke prevention between 2002 and 2010. CAD diagnosis was based on the presence of a mural hematoma on MRI. Patients who had DWI at admission, ultrasound examination, and contrast-enhanced MR angiography (n=169) or catheter angiography (n=3) were selected. Only the symptomatic dissection was considered. In case of multiple dissections without ischemic symptoms, we considered only the CAD with local symptoms. Lumen patency was divided into <70% stenosis (Group 1), ≥70% stenosis (Group 2), or occlusion (Group 3). Whenever ultrasound and angiography findings were inconsistent (n=25), patients were excluded from further analyses. DWI was performed using previously published protocols.2 Two observers independently assessed the presence, number, volume and pattern of acute DWI ischemic lesions. Based on published classifications and templates,3–5 we identified: (1) strokes in pial artery territories (Pattern 1, ie, complete or incomplete territorial stroke involving the cortex and subcortical structures; (2) strokes in perforating artery territories (Pattern 2, ie, stroke in the territory of deep perforating branches, sparing the cortex junctional area); and (3) junctional strokes (Pattern 3) when located at the boundaries of 2 arterial territories. Multiple DWI lesions were defined as multiple noncontiguous infarcts and multiple pattern as DWI lesions belonging to distinct pattern. Disagreements between readers were solved by consensus.

Statistical Analysis
Kappa coefficients and their 95% CIs were used to assess interobserver agreement for stroke patterns and lumen patency. Chi-square and Mann-Whitney U tests were used to test contingency among occurrence of DWI lesions, stroke pattern, and patient groups (<70% stenosis: Group 1, ≥70% stenosis: Group 2, and occlusion: Group 3), and to compare number and volume of DWI lesions between groups.

Results
The study population included 147 patients (100 carotid and 47 vertebral dissections; Table 1). Eighty-eight (60%) patients had DWI lesion and 59 (40%) did not. Agreement was...
high for pattern identification ($\kappa=0.87$; 95% CI, 0.77–0.96) and group definition ($\kappa=0.90$; 0.83–0.96). Overall, 297 DWI lesions were found with multiple lesions in 49 of 88 patients (56%) and multiple patterns in 44 of 88 patients (50%) with DWI lesions. Occurrence of DWI lesion (Group 1: 26 of 45, Group 2: 32 of 59, Group 3: 30 of 43; $P=0.27$) and the number of DWI lesions (Group 1: 3.5±3.9, [1–4] Group 2: 4.2±4.1 [1–5], Group 3: 3.3±4.0, [1–3]; $P=0.85$) were similar regardless of group assignment. Volume of DWI lesions was larger in Group 3 (82±90 mm$^3$ [17–91]) than in Group 1 or Group 2 (34±54 mm$^3$ [2–48]; $P=0.03$).

There was no difference in the breakdown of DWI lesions patterns according to degree of stenosis (Table 2). Similar results were found when analyses were restricted to carotid dissection or vertebral dissection and when lumen patency was dichotomized into stenotic or occlusive CAD (data not shown).

**Discussion**

The present study yielded the following results: (1) occurrence of stroke and the number of DWI lesions were not influenced by the degree of stenosis in CAD patients; (2) stroke volume was larger in occlusive CAD; and (3) stroke patterns were similar regardless of the degree of stenosis of the dissected artery.

In line with others,6–8 in case of brain infarction, we found multiple acute DWI lesions in more than half of patients, a result that substantiated the concept of arterial embolism in the pathogenesis of stroke in CAD.7 As a novel observation, the occurrence of stroke in CAD patients seems to be independent of the degree of stenosis and hemodynamic compromise. Statistical analysis did not demonstrate that the degree of obstruction may favor certain stroke patterns.

The lack of association between the degree of stenosis and the occurrence of stroke and predefined stroke patterns challenged the hypothesis of a preferred mechanism in the different stenosis subgroups in CAD and the idea that mechanism of stroke in CAD resembles that of atherosclerotic disease.9 Bonati et al showed different DWI lesion patterns in 15 stenotic and 25 occlusive CAD.6 However, the number of patients was too limited to perform analyses of associations between different degrees of luminal narrowing and DWI lesion presence and patterns. Future therapeutic trials should investigate whether the assessment of arterial patency may translate into a potential role in treatment decisions in CAD patients.

An exploration of the likely mechanism of the primary DWI lesion, no matter how reliable, may not apply to secondary events. This represents, in addition to selection bias of patients addressed to the referral center and the variability in the timing of imaging studies, the most important limitation of our study, because the present study only explored the link between degree of stenosis and primary DWI lesion and assumed that subsequent events will follow the same mechanism. We did not use catheter angiography, the standard of reference for cervical artery stenosis measurement, given that catheter angiography is rarely used in CAD patients. We cannot exclude the possibility that, because of the imprecision of contrast-enhanced MR angiography in measuring degree stenosis, group errors might have occurred. Such misclassifications would have particularly affected vertebral artery dissection, in which standard measurement criteria have not yet been established. However, vertebral artery dissections were equally distributed among degree of stenosis groups. Finally, the population was fairly small and may be underpowered, especially for the occurrence of brain infarction.

In conclusion, our study suggests that the occurrence and pattern of stroke may not be influenced by the degree of...
stenosis of the dissected artery, an additional argument in favor of embolism mechanism of stroke in CAD.

Disclosures
None.

References
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