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Stroke Patterns of Internal Carotid Artery Dissection in 40 Patients

Christian Lucas, MD; Thierry Moulin, MD; Dominique Deplanque, MD; Laurent Tatu, MD; Didier Chavot, MD; and the DONALD Investigators*

Background and Purpose—Internal carotid artery dissection (ICAD) is a frequent cause of ischemic stroke in young patients. Whether cerebral ischemia is of embolic or hemodynamic origin remains to be determined. Heparin is often administered in ICAD; however, a drug trial can hardly be conducted because of the low recurrence rate after the acute stage. Therefore, the best therapeutic approach should be determined on the basis of the presumed mechanism of cerebral ischemia. One way to approach the mechanism of stroke in ICAD is to determine stroke patterns. We postulated that most cortical and large subcortical infarcts (≥ 15 mm) are of embolic origin and that small subcortical infarcts (< 15 mm) and junctional infarcts are not. The aim of our study was to determine the stroke patterns in 40 consecutive patients with ICAD.

Methods—The patients (26 women and 14 men; mean age, 42.8 years) had a total of 65 ICADs. Seventeen patients were free of any vascular risk factor. CT scans, MRI scans, and angiographic features were analyzed by observers who were blinded to the clinical findings.

Results—We found 34 cortical infarcts, 25 large subcortical infarcts, 1 small subcortical infarct, and 5 junctional infarcts.

Conclusions—Most infarcts related to ICAD are cortical infarcts or large subcortical infarcts; small subcortical infarcts and junctional infarcts are infrequent. Therefore, these findings suggest that most infarcts occurring in carotid artery dissection (CAD) are probably embolic rather than hemodynamic in origin. According to this presumed mechanism, anticoagulation seems a logical treatment at the early stage of CAD. (*Stroke*. 1998;29:2646-2648.)

Key Words: cerebrovascular disorders ■ dissection ■ internal carotid artery ■ stroke ■ stroke classification

Internal carotid artery dissection (ICAD) is an increasingly recognized cause of stroke that accounts for up to 20% of ischemic strokes in young adults.^{1,2} According to a common view, ICAD leads to embolic rather than to hemodynamic cerebral infarction.³ However, systematic investigations of the underlying mechanism of cerebral ischemia have rarely been performed.^{4,5} The presumed mechanism of stroke may be approached by determining the stroke pattern in patients with ICAD. Two previous studies^{4,5} found conflicting results: one suggested a prominent hemodynamic compromise in ICAD,⁴ and the other suggested a prominent embolic mechanism.⁵ Determining whether most ICADs lead to cerebral ischemia because of artery-to-artery embolism or because of hemodynamic failure is valuable, because it may influence the therapeutic approach: heparin may be more appropriate in the first mechanism but is not likely to be effective in the second, and it might even lead to an increase of the mural hematoma. Because of the low rate of recurrence of cerebral ischemia in patients with carotid artery dissection (CAD), a

drug trial remains difficult.⁶ Therefore, the therapeutic approach should be based on the presumed mechanism of cerebral ischemia in patients with CAD. One possible approach is to determine the stroke patterns in CAD. The aim of this study was to determine stroke patterns in consecutive patients with ICAD. It was beyond the scope of this study to use other approaches, such as ultrasonography.

Subjects and Methods

The study was conducted in a retrospective way in 40 consecutive patients admitted to 2 stroke units for a proved ICAD responsible for ischemic stroke. CAD was defined as angiographic evidence of a string sign, double lumen, or intimal flaps, or as an occlusion associated with mural hematoma on cervical MRI. The following data were prospectively collected: age (in years), sex, and presence of arterial hypertension (defined as systolic blood pressure > 150 mm Hg or diastolic blood pressure > 90 mm Hg or current treatment with antihypertensive drugs), diabetes mellitus (defined as repeated fasting serum glucose level > 1.4 g/L or current use of antidiabetic drugs), dyslipemia (defined as fasting serum level of triglycerides > 1.5 g/L or fasting cholesterol serum level > 2.5 g/L or

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TABLE 1. Epidemiological Data in the Study Population of 40 Patients With Internal Carotid Artery Dissection

Epidemiological Data	N=40 (%)
Men/women	14/26
Arterial hypertension	11 (27.5)
Diabetes mellitus	1 (2.5)
Dyslipemia	9 (22.5)
Cigarette smoking	14 (35)
Migraine	7 (17.5)
Migraine with ophthalmic aura	3 (7.5)
No vascular risk factors	17 (42.5)
Recent cervical trauma	12 (30)

current therapy), current cigarette smoking, migraine according to International Headache Society criteria,⁷ recent trivial or obvious cervical trauma, and initial symptomatology. These 40 patients underwent either 2 CT scans or 1 CT scan (at admission) and 1 MRI scan showing the presence of mural hematoma (n=22) at the acute stage (during the first week); angiography (n=40) was performed at onset during the first week and again 3 months later (n=33). This was the accepted way to manage patients with CAD in our centers before availability of MR angiography as a routine procedure. Both carotid and vertebral arteries were studied during each angiographic procedure. CT scans, MRI scans, and angiographies were analyzed separately by 2 observers. In case of a discrepancy between the observers, a consensus reading was used. Observers were blinded to clinical data. They determined whether the following lesions were present: cortical infarcts (any infarct involving the cortex or the cerebellar surface); subcortical infarcts (any infarct involving basal ganglia, thalamus, internal capsule, or centrum ovale and sparing the cortical surface), divided into 2 subgroups (those ≥ 15 mm and those < 15 mm); and junctional and watershed infarcts (any infarct located between 2 arterial territories). This classification was made with the use of maps of vascular territories⁸ and in accordance with etiologic concepts.^{9,10} Cortical and subcortical infarcts in the territory of multiple lenticulostriate arteries were considered embolic, whereas infarcts between the middle cerebral artery (MCA) territory and anterior cerebral artery territory or between MCA territory and posterior cerebral artery territory were considered hemodynamic infarcts. Angiographic findings were defined as occlusion, stenosis, double lumen, and pseudoaneurysm, according to previously reported criteria.¹¹ Dysplasia was defined by tortuous aspect and redundancies of >1 cervical artery on cerebral angiography.

Results

The study population consisted of 26 women and 14 men, aged 18 to 61 years (mean age, 42.8 years; 95% CI, 39.2 to 46.3). Patients had a total of 45 ICA dissections (26 right, 19 left). Five patients had bilateral ICA dissections; of these 5 patients, 2 had a bilateral ICA dissection with unilateral vertebral artery dissection, and 1 had a dissection of both the ICA and the vertebral artery.

Seventeen patients were free of any vascular risk factor (Table 1). Seven patients had a history of preexisting migraine according to International Headache Society criteria,⁷ including 3 with ophthalmic aura. A recent cervical trauma (either trivial, such as from carrying a heavy burden, dancing, or painting a ceiling, or obvious, such as chiropractic maneuvers or a car accident) was found in 12 patients. ICAD was associated with headache in 8 patients, cervical pain in 3, and both headache and cervical pain in 6 patients. Eight patients had a Horner's syndrome (2 isolated,

TABLE 2. Stroke Patterns of 65 Infarcts in 40 Internal Carotid Artery Dissections With Presumed Mechanisms of Cerebral Ischemia According to Etiological Concepts

	65 Infarcts (%)	Occlusion (n=21)	Stenosis (n=24)	Presumed Mechanism
Cortical (MCA territory)	34 (52.3)	21	13	Embolitic
Large striatocapsular	25 (38.4)	11	14	Embolitic
Subcortical (< 15 mm outside internal junctional zone)	1 (1.5)	0	1	Embolitic
Internal junctional/watershed	5 (7.7)	3	2	Hemodynamic

3 with cervical pain, and 3 with headache). We found 24 stenoses (12 right, 12 left), with 20 string signs and 21 occlusions (14 right, 7 left) at the acute phase. Eleven patients had dysplasia, and 9 had a pseudoaneurysm on the ICA (6 on the right ICA and 3 on the left). A second angiography performed 3 months later in 33 patients showed persistence of the same abnormality in 9, partial reopening in 2, and complete angiographic recovery in 22.

The patients had a total of 65 infarcts (Table 2). All were recent infarcts, except in 1 patient, who had a recent infarct in the left hemisphere due to left occluded carotid artery dissection and an old infarct on the right side, without evidence of old carotid artery dissection or dysplasia. All infarcts were located in the territory of the dissected carotid artery except for the previous patient. We found 34 cortical hemispheric infarcts, 25 subcortical infarcts > 15 mm, 1 subcortical infarct < 15 mm, and 5 junctional or watershed infarcts. The 5 junctional or watershed infarcts were isolated, and 3 of the 5 were associated with a homolateral occluded carotid artery. There were no differences in the breakdown of stroke patterns according to the stenotic or occlusive angiographic feature (32 large cortical or subcortical infarcts in patients with occlusion and 28 in those with stenosis; 3 junctional or watershed infarcts in patients with occlusion and 2 in those with stenosis), except for multiple infarcts. We found 13 patients with multiple infarcts. Patients with multiple infarcts had mainly occlusions (9 patients) rather than stenoses (4 patients). Therefore, cortical infarcts and subcortical infarcts accounted for 92.2% of all infarcts, whereas junctional infarcts accounted for only 7.7%.

Discussion

Our study showed that most infarcts related to ICAD are cortical or large subcortical infarcts. Epidemiological and clinical data are similar to these of the literature^{6,11} except for the sex ratio. We found a strong predominance of women (65%). This result also could explain why we found 11 dysplasia aspects (27.5%) on initial angiography. According to the current conception of the relationship between the mechanism of infarction and stroke patterns, cortical or subcortical infarcts are more likely to be of embolic origin,^{10,12,13} whereas junctional or watershed infarcts are more likely to be of hemodynamic origin.⁹ Using this simplification, we found that only 7.7% of patients with CAD had a

presumed hemodynamic cerebral ischemia, whereas 92.2% had a presumed embolic infarct. However, even if this simplification provides an easy approach of the presumed mechanisms of cerebral ischemia in most patients, one should bear in mind that at the individual level, junctional infarcts are sometimes associated with a possible embolic mechanism.¹⁴ Therefore, we have probably overestimated the rate of hemodynamic infarcts in our study. Despite this overestimation, the number of patients with presumed hemodynamic infarcts is extremely low (7.7%). Our study therefore supports the hypothesis that most infarcts in ICAD are of embolic origin. On the other hand, the 2 pathophysiological mechanisms are probably linked. Indeed, we can easily imagine that in the first step of an ICAD, the hemodynamic mechanism is prominent during the constitution of the wall hematoma, whereas the embolic mechanism is prominent after this phase. We found that patients with occlusion primarily had multiple infarcts, also suggesting an embolic mechanism. However, it is difficult to reach a conclusion concerning this finding, because carotid artery dissection is a dynamic process at the acute phase and some occlusions can return to stenosis very quickly. In the study of Weiller et al,⁴ the authors reported a similar frequency of embolic and hemodynamic infarcts, but their study population consisted of only 11 patients. We reached the same conclusion as Steinke et al,⁵ who showed that 37 of 67 patients (55%) with ICA dissection had brain infarcts, of which territorial artery MCA infarcts of variable size accounted for 60%; large laterostriate infarcts were present in 11%, and a presumed hemodynamic mechanism was found in only 16% of infarcts. A thrombus formation in the dissected artery with secondary distal embolism is the most likely mechanism of cerebral infarction in CAD. Evidence of distal emboli is rare because of the widespread use of heparin at the acute phase of ischemic stroke, especially in young patients, and because of the limitations of angiography. In the study of Steinke et al,⁵ the autopsy of a patient who died of an MCA infarct provided

arguments for an embolic mechanism: all large MCA branches were occluded by a recent thrombus originating from an intimal tear 4 cm distal to the right carotid bifurcation. This study therefore suggests a prominent embolic origin in patients with ICAD and ischemic stroke.

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