Mechanism of Ischemic Infarct in Spontaneous Cervical Artery Dissection

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

Background and Purpose—It is unclear whether strokes in patients with spontaneous cervical artery dissection (CAD) are due to secondary thromboembolism or to a reduction in cerebral blood flow from the primary cervical lesion. The aim of this study was to identify the most likely mechanism of stroke using cervical and cerebral imaging parameters in patients with CAD.

Methods—The study was approved by the local Ethics Committee. Informed consent was waived. We retrospectively evaluated the cerebrovascular ultrasound, cervical MR angiography, and stroke brain MRI in consecutive patients with CAD. An embolic mechanism was considered in the case of direct visualization of an intracranial embolism as a susceptibility vessel sign on T2* or in the case of pial artery territory infarction on diffusion-weighted imaging. A hemodynamic mechanism was considered in the case of watershed infarction and in the case of an association of watershed infarction and pial artery territory infarction when ≥2 of the following were present: severe stenotic or occlusive CAD, reduced intracranial velocity on cerebrovascular ultrasound or signal on MR angiography, or hyperintense vessel sign on fluid-attenuated inversion recovery. The remaining patients were considered to have a mixed mechanism.

Results—Of 172 consecutive patients with CAD, 100 (58%) had acute stroke on diffusion-weighted imaging. Stroke was attributed to a thromboembolic mechanism in 85 of 100 patients, a hemodynamic mechanism in 12 of 100 patients, and a mixed mechanism in 3 of 100 patients.

Conclusions—Stroke in patients with CAD is most frequently associated with both direct and indirect signs of artery-to-artery embolization on imaging, a finding that should help design future therapeutic trials. (Stroke. 2012;43:1354-1361.)

Key Words: acute stroke □ diffusion-weighted imaging □ dissection □ embolic stroke □ MRI □ stroke in young adults

Extracranial cervical artery dissection (CAD) accounts for nearly 20% of cases of ischemic stroke in young adults. Some authors have suggested that artery-to-artery embolism is the main mechanism of stroke in CAD, whereas others assume that reduced flow from the primary cervical lesion plays a crucial role, yet determining whether most CADs leads to cerebral ischemia because of embolism or because of hemodynamic failure may influence the therapeutic approach. For several authors, reduced flow from the primary cervical lesion could be approached through endovascular revascularization, whereas anticoagulation or antiplatelet regimens would be more appropriate to prevent secondary embolic events.

Imaging can be used as an end point in distinguishing hemodynamic from thromboembolic infarct. Acute thromboembolism may be demonstrated directly on brain MRI using a T2* sequence, because intraluminal acute thrombus appears as a signal loss along the course of occluded symptomatic cerebral arteries. A presumed embolic mechanism may also be evoked indirectly on diffusion-weighted imaging (DWI) in the case of a pial or perforating territory stroke pattern, whereas junctional or watershed infarcts are more likely to be of hemodynamic origin. The aim of this study was to identify the most likely mechanism of stroke using cervical and cerebral imaging parameters in consecutive patients with CAD.

Patients and Methods

Patients

The study was approved by the Ethics Committee of Ile de France III. Informed consent was waived. The article was prepared in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement. The population was nested within a longitudinal cohort of consecutive patients referred to our institution for suspected acute stroke, transient ischemic attack, or prevention of stroke between January 2002 and January 2010 (n = 5895). This prospectively maintained database was available at the Stroke website.
retrospectively queried to identify all patients with demonstration of a CAD based on ≥2 of the following criteria: (1) intimal flap or mural hematoma visible together with a normal internal carotid artery bulb on cerebrovascular ultrasound; (2) mural hematoma visible on cervical axial fat-suppressed T1-weighted imaging; and (3) intimal flap or a nonatherosclerotic, tapered, flame-shaped artery occlusion or a string-like stenosis with normal internal carotid artery bulb on cervical contrast-enhanced MR angiography (CE-MRA) or conventional digital subtracted angiography (DSA). We included patients with CAD who met the following inclusion criteria: (1) first acute stroke attributable to CAD; (2) stroke brain MRI performed within the first 4 weeks after onset of the first (neurological or local) symptoms; and (3) assessment of dissected artery patency and intracranial arteries on cerebrovascular ultrasound and CE-MRA or DSA.

Imaging Acquisition

**Magnetic Resonance Imaging**

Brain MRI was performed on a 1.5-T Sigma MR Unit (General Electric Healthcare, Milwaukee, WI) using previously published protocols. Briefly, the standardized stroke protocol included 6-mm-thick bicommissural axial fluid-attenuated inversion recovery (FLAIR), gradient recalled echo T2 or T2*, DWI using spin-echo echoplanar imaging, and noncontrast 3-dimensional time-of-flight MR angiography of the circle of Willis. The neck imaging protocol consisted of at least fast spin-echo T1-weighted fat-suppressed axial sequence followed by CE-MRA of brain-supplying arteries (intraaxial meglumine gadoterate; Dotarem, Guerbet, France; 0.1 mmol/kg body weight) using an 8-channel phased-array coil for head and neck imaging.

**Cerebrovascular Ultrasonography**

In all patients, cervical and intracranial arteries were investigated by 4-MHz Doppler sonography and color-coded duplex sonography (linear 7.5–12 MHz, sectoral 5–7.5 MHz; Philips-ATL HDI 5000 U; Philips Medical Systems, Bothell, WA), transcranial Doppler (2-MHz probe), and color-coded sonography (2–2.5 MHz; Philips-ATL HDI 5000 U) using transtemporal and suboccipital approaches.

**Imaging Analysis**

Imaging analysis was performed independently by 2 observers, a senior neuroradiologist (with 7 years’ experience) and a junior radiologist (with less than 6 months’ experience).

**Direct Signs of Secondary Intracranial Embolic Lesions**

Observers had to judge the presence or absence of a susceptibility vessel sign (SVS) on T2*, defined as a hypointense signal within a vessel sign due to blood product. Patients with an association of stroke patterns1 and/or stroke pattern2 on DWI but without stroke pattern3 and without signs of hemodynamic compromise were considered embolic stroke in anterior circulation (see subsequently).

**Evaluation of the Primary Cervical Lesion**

Using CE-MRA and ultrasound examinations, we categorized cervical-dissected artery patency as normal, occluded, or stenotic (≥70% stenosis); the degree of stenosis was measured by comparing the diameter of the lumen at the site of stenosis (D stenosis) with the normal diameter of the lumen distal to the stenosis (D distal) using the following formula: % stenosis = (1 − [D stenosis/D distal]) × 100%. On FLAIR, we searched for hyperintense vessel sign, defined as focal or tubular hyperintensities in the subarachnoid space in corresponding symmetrical nonoccluded carotid or vertebral artery territories.

**MR-Defined Stroke Patterns**

FLAIR sequences were analyzed to search for signs of chronic infarct and DWI sequences were analyzed to assess the number and pattern of acute brain ischemic lesions. 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 the consequence of embolism in pial or perforating arteries, whereas junctional or watershed infarcts are more likely to be of hemodynamic origin. The vascular territory of brain infarcts was determined using published templates and the topographical distribution of ischemic lesions was divided into 3 patterns. We identified: (1) strokes in pial artery territories (pattern1), that is, complete or incomplete territorial infarct involving the cortex, the cerebellar surface and subcortical structures, unique or multiple noncontiguous infarcts belonging to distinct pial territories; (2) strokes in perforating artery territories (pattern2), that is, subcortical infarct, with (a) involvement in the territory of deep perforating branches: basia ganglia, internal capsule, thalamus, centrum semiovale, midbrain; (b) sparing of the cortex; and (c) no morphological or topographical distribution of junctional infarcts; and (3) junctional strokes (watershed, border zones; pattern3), defined as any infarct located between 2 arterial territories (Figure 1).

**Statistical Analysis**

MedCalc statistical software (Version 9.4.2.0; Mariakerke, Belgium) was used for the analysis. Kappa coefficients and their 95% CIs were used to assess interobserver agreement for stroke pattern definition, SVS on T2*, asymmetry of intracranial vessel on 3-dimensional time-of-flight MR angiography, hyperintense vessel sign on FLAIR, and dissected artery patency. The Mann-Whitney test was used to compare the number of DWI lesions and volume between patients with nonocclusive and occlusive dissected artery. The Fisher exact test was used to compare the proportion of embolic stroke in anterior versus posterior circulation. A 2-sided probability value of <0.05 was considered statistically significant.

**Results**

We identified 172 consecutive patients with CAD (198 dissected arteries, 131 carotid CADs, and 67 vertebral CADs) including 100 patients (100 of 172 [58%]) with acute stroke on DWI (117 dissected arteries: 80 carotid CADs and 37 vertebral CADs). The remaining 72 patients had no DWI visible on cervical contrast-enhanced MR angiography (CE-MRA) or conventional digital subtracted angiography (DSA). We assigned to group1 all patients with SVS on T2* and/or stroke pattern1 and/or stroke pattern2 on DWI but without stroke pattern3 and with signs of hemodynamic compromise (see subsequently).

**Thromboembolic Stroke Group (Group1)**

We assigned to group2 all patients with isolated stroke pattern3 and/or stroke pattern1 and/or stroke pattern2 on DWI but without stroke pattern3 and without signs of hemodynamic compromise (see subsequently).

**Hemodynamic Stroke Group (Group2)**

We assigned to group3 all patients with isolated stroke pattern3 and/or stroke pattern1 and/or stroke pattern2 on DWI but without stroke pattern3 and with signs of hemodynamic compromise (see subsequently).

**Mixed Mechanism (Group3)**

We assigned to group4 all patients with isolated stroke pattern3 and/or stroke pattern1 and/or stroke pattern2 on DWI but without stroke pattern3 and with signs of hemodynamic compromise (see subsequently).

The vascular territory of brain infarcts was determined using published templates and the topographical distribution of ischemic lesions was divided into 3 patterns. We identified: (1) strokes in pial artery territories (pattern1), that is, complete or incomplete territorial infarct involving the cortex, the cerebellar surface and subcortical structures, unique or multiple noncontiguous infarcts belonging to distinct pial territories; (2) strokes in perforating artery territories (pattern2), that is, subcortical infarct, with (a) involvement in the territory of deep perforating branches: basia ganglia, internal capsule, thalamus, centrum semiovale, midbrain; (b) sparing of the cortex; and (c) no morphological or topographical distribution of junctional infarcts; and (3) junctional strokes (watershed, border zones; pattern3), defined as any infarct located between 2 arterial territories (Figure 1).

**Mixed Mechanism (Group3)**

We assigned to group3 all patients with isolated stroke pattern3 and/or stroke pattern1 and/or stroke pattern2 on DWI but without stroke pattern3 and with signs of hemodynamic compromise (see subsequently).
the population. Median delay from onset to brain MRI was 3 days (interquartile range, 1–7; mean, 5.1; range, 0–26 days). None of the patients had an old stroke. Among the 100 patients with stroke, 50 patients had multiple infarct patterns. The total number of noncontiguous DWI lesions was 338 with multiple lesions in 56 of 100 patients and lesions in multiple territory in 13 of 100 patients. Interobserver agreements were high for SVS on T2* ($\kappa=0.91; 95\%$ CI, 0.82–1) for asymmetry of signal or size in intracranial vessel on 3-dimensional time-of-flight MR angiography ($\kappa=0.88; 95\%$ CI, 0.80–0.96), for hyperintense vessel sign on FLAIR ($\kappa=0.86; 95\%$ CI, 0.77–0.95), for stroke pattern definition on DWI ($\kappa=0.84; 95\%$ CI, 0.76–0.92), and for dissected artery patency on CE-MRA ($\kappa=0.90; 95\%$ CI, 0.83–0.96). Stroke was attributed to a thromboembolic mechanism in 85 of 100 patients with direct visualization of SVS in the symptomatic intracranial artery in 57 of 100 patients and stroke pattern1 and/or pattern2 in 85 of 100 patients. Stroke was attributed to a hemodynamic mechanism in 12 of 100 patients, including 4 patients with isolated watershed infarction and 8 patients with stroke pattern1 and/or pattern2 associated with pattern3 and a hemodynamic score $\geq 2$. Stroke was attributed to a mixed mechanism in the remaining 3 patients (3%). Overall, stroke involved pial artery territories (pattern1, 83 of 100) or perforating artery territories (pattern2, 17 of 100) in all except 4 patients (96%). When an SVS was present, we did not observe isolated watershed infarction. In 4 patients with SVS, a pattern3 was associated with a pattern1 and in 1 case with...
both pattern1 and pattern2. The proportion of embolic and hemodynamic stroke did not differ between anterior and posterior circulation stroke (embolic stroke: 52 of 70 and 23 of 30, respectively, \(P\) = 1; hemodynamic stroke: 9 of 70 and 3 of 30, respectively, \(P\) = 0.51).

**Discussion**

This exploratory study of cervical and brain MRI in 100 carotid and patients with vertebral CAD stroke yielded the following results: (1) intracranial thrombus was seen in 57 of 100 patients; (2) pial and perforating artery territory stroke was present in 96 of 100 patients; (3) multiple DWI lesions were seen in 56 of 100 patients; and (4) isolated watershed infarction was seen in 4 of 100 patients. These results suggest that stroke in cervical artery dissection was most frequently associated with artery-to-artery embolic events both in anterior and posterior circulation.

Determining whether most dissections lead to cerebral ischemia because of artery-to-artery embolism or because of hemodynamic failure is important, because it may influence the therapeutic approach. Current therapeutic options in CAD include antiplatelet (eg, aspirin), anticoagulation, endovascular treatment with stent deployment, and often a combination when medical treatment fails to prevent ischemic stroke.8–10 The natural history of CAD indicates a risk of recurrent stroke, mainly within the first weeks or months.40,41 By analogy to cardioembolic stroke (eg, atrial fibrillation), in which anticoagulation is superior to antiplatelets for secondary stroke prevention,42 many physicians use anticoagulants on the assumption that this prevents embolism from thrombus at the dissection site more effectively than antiplatelet agents.15,43 However, a large study44 and a systematic review of nonrandomized studies showed no evidence of a therapeutic benefit favoring either antiplatelet or anticoagulant treat-
ment in preventing stroke, transient ischemic attack, or death in CAD. The presumed benefit of the deployment of a stent all along the dissection includes dissected artery flow restoration. Although many patients who have failed conservative medical therapy are referred for endovascular treatment with angioplasty and stent placement, there have been no well-designed studies to support this practice. Although valid therapeutic trials should address these questions, a trial remains difficult. In the absence of comparative studies, the presumed mechanism of cerebral infarction is unlikely to impact on clinical decisions but may be useful to define selection criteria for randomized controlled trials.

The literature provides support for several concepts regarding the primary cervical lesion and the secondary intracranial lesion in CAD. An autopsy case has provided the only direct demonstration of secondary intracranial emboli. Three studies described indirect signs of emboli on transcranial Doppler monitoring studies, but the sample sizes of these studies limit the significance of these results. According to current concepts relating mechanism and stroke pattern, strokes in pial or perforating artery territories are more likely to be embolic, whereas border-zone infarcts are more likely to be hemodynamic. This scheme has been used, in DWI- and CT-based studies, to shed light on the stroke mechanism in patients with CAD with conflicting results. The apparent discrepancy between DWI- and CT-based study results may be due to the better identification of acute ischemic pattern when using DWI as compared with CT scan. Thus, CT might underestimate multiple punctuate lesions and perforating artery stroke. Like others, we observed hemodynamic infarct patterns in 10% of patients. Our results challenge the findings of Lanczik et al who demonstrated border-zone infarcts in 7 of 24 and 9 of 14 patients, respectively. In addition to the small number of patients of these studies, these results were most likely overestimated because of variability in the vascular anatomy and the misclassification of branch artery occlusion as watershed infarcts. Benninger et al, based on stroke pattern on CT scan and/or MRI, suggested that embolism is the essential stroke mechanism in CAD. As a novel observation, we directly demonstrated the occlusive intracranial thrombus in vivo with a T2* sequence in 57% of patients. The T2* SVS has been used in patients with acute stroke for 10 years. The susceptibility effect has been ascribed to local ferromagnetic field distortion associated with deoxyhemoglobin components. Most successive studies reported detection rates of approximately 50%, in cohorts of patients with hyperacute stroke, with a 100% specificity.
We considered as an in vivo gold standard for direct visualization of the thrombus. In addition, an embolism may have lysed or migrated at the time of brain MRI. However, such misclassifications would have weakened the link between stroke and the embolic mechanism. The SVS has never been compared with DSA in patients with hyperacute stroke to determine its value in predicting the arterial occlusion site and extent. Consequently, one can argue that SVS may correspond not to an embolus but to a prolongation of the thrombus from the dissected cervical artery into the intracranial arteries. This third theoretical mechanism might have occurred in occlusive CAD. However, the autopsy of a patient who died from a middle cerebral artery stroke did not provide evidence for this mechanism. The luminal thrombus extended from 4 cm distal to the right carotid artery to the supraclinoid portion of the carotid artery, but thrombotic material in branches of the middle cerebral artery was independent of the supraclinoid thrombus. In line with our results, the authors concluded that there was a secondary embolism from the primary cervical lesion. A second limitation is the interindividual variability of the arterial territories. However, even supposing that some additional border-zone infarcts were missed on DWI, we did not use the stroke pattern alone to determine the stroke mechanism. Third, we did not systematically use DSA, the standard of reference for cervical artery stenosis measurement, given that DSA is rarely used in patients with CAD. We cannot exclude the possibility that, because of the imprecision of CE-MRA and Doppler ultrasound in measuring the degree of 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. Finally, the present article can only explore mechanisms of the primary clinical event and assume that, for each patient, subsequent events will follow the same mechanism. We should bear in mind that an exploration of the likely mechanism of the primary clinical event, no matter how reliable, may not apply to secondary clinical events.

In conclusion, our study suggests that thromboembolism, rather than hemodynamic infarction, is the most frequent cause of stroke in CAD. This implies that prevention of artery-to-artery embolism could play a crucial role in the management of these patients. Our findings may also help the design of a randomized controlled trial to obtain scientific evidence of the role of heparin, antiplatelet therapy, and stenting in CAD.

### Disclosures

None.

### References


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12. Mourand I, Brunel H, Vendrell JF, Thouvenot E, Bonafe A. Endovascular
18. Lutsep HL, Albers GW, DeCrespigny A, Kamat GN, Marks MP, Moseley
20. von Elm E, Altman DG, Egger M, Pocock SJ, Gotzsche PC, Vandenb-
8. Kadkhodayan Y, Jeff DT, Moran CJ, Derdeyn CP, Cross DT III. Angio-
7. Pham MH, Rahme RJ, Arnaout O, Hurley MC, Bernstein RA, Batjer HH,
6. Weiller C, Mullges W, Ringelstein EB, Buell U, Reiche W. Patterns of
1. Lucas C, Moulin T, Deplanque D, Tatu L, Chavot D. Stroke patterns of
3. Bladin CF, Chambers BR. Clinical features, pathogenesis, and computed
2. Benninger DH, Georgiadis D, Kremer C, Studer A, Nedeltchev K, Baum-
4. Benninger DH, Georgiadis D, Kremer C, Studer A, Nedeltchev K, Baum-
gartner RW. Mechanism of ischemic infarct in spontaneous carotid dis-
1125–1135.
39. Damasio H. A computed tomographic guide to the identification of
1699–1708.
20. von Elm E, Altman DG, Egger M, Pocock SJ, Gotzsche PC, Vandenb-
9. Yong SW, Bang OY, Lee PH, Li WY. Internal and cortical border-zone
35. Bogousslavsky J, Van Melle G, Regli F. Middle cerebral artery pial
32. Tatu L, Moulin T, Bogousslavsky J, Duvernoy H. Arterial territories of
115:91–100.
19. Yong SW, Bang OY, Lee PH, Li WY. Internal and cortical border-zone
10. Weiller C, Mullges W, Ringelstein EB, Buell U, Reiche W. Patterns of
1125–1135.
40. von Elm E, Altman DG, Egger M, Pocock SJ, Gotzsche PC, Vandenb-


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Mecanismo del infarto isquémico en la disección arterial cervical espontánea

El mecanismo que subyace en la isquemia cerebral al producirse una disección de la arteria carótida puede incluir la formación de un trombo intumescido y un estrechamiento mecánico del diámetro luminal que disminuye la circulación y aumen
ta la hipotensión o ambas cosas. En consecuencia, el tratamiento optimo debe dirigirse a la etiología "verdadera" en cada paciente. Morel y colaboradores intentaron identificar el mecanismo más probable del ictus con el empleo de parámetros de técnicas de imagen cerebrales y cervicales en pacientes consecutivos con disecciones de la arteria carótida. Estos autores realizaron retrospectivamente a 100 pacientes (media de edad: 45 años) de una base de datos prospectiva que presentaron una disección de la arteria carótida y que sufrieron un ictus demostrado mediante RM. atribuible a la disección de la arteria carótida y para los que se dispuso de exploraciones de imagen para valorar el vaso de la disección y las arterias intracranales. El acierto interobservadores para la interpretación de las diversas técnicas de imagen fue elevado para cada modalidad de imagen utilizada (Kaplan Y. 0.84-0.91). Según la etiología probable del ictus, se asignó a los pacientes a 3 grupos: tromboembólico, hemodinámico o mixto. Se observó un trombo intracranal en 57 pacientes, hubo un ictus del territorio de arterias de la pia y perforantes en 86 pacientes; se observaron lesiones múltiples en imágenes con ponderación de difusión en 55 pacientes; y se identificaron infartos isquémicos en el límite de las zonas de isquemia en 4 pacientes. Los ictus se clasificaron como tromboembólicos en 83 de 100 pacientes, hemodinámicos en 12 de 100 pacientes, y mixtos en 3 de 100 pacientes. Esto sugiere que el ictus tras una disección de la arteria carótida se debe habitualmente a una embolia intracranal. Sería interesante saber qué tipo de tratamiento se eligió así como el momento de aplicación en relación con la RM. Según lo indicado por esos resultados, cabría suponer que la endartectomía sería la modalidad de tratamiento presente. Sin embargo, tal como señalan los autores, en malas noticias no se ha establecido una superioridad clara de este tratamiento. Así pues, el presente estudio es importante porque aporta un fundamento para futuros estudios encaminados a evaluar la eficacia de diferentes pasos de tratamiento en relación con la etiología más probable del ictus. (Comentamos al artículo Mechanism of Ischemic Infarct in Spontaneous Cervical Artery Dissection. Audrey Morel, Olivier Nogaro, Emmanuel Touze, Jean Raymond, Jean-Louis Mas, Jean-Francois Meder, and Catherine Oppenheim. Stroke. 2012;43:1364-1361.)
特発性頸部動脈解離における脳梗塞の機序

Mechanism of Ischemic Infarct in Spontaneous Cervical Artery Dissection

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Abstract

背景および目的: 特発性頸部動脈解離 (CAD) 患者における脳卒中が、二次的な血栓塞栓症に起因するのか、または頸部の原発病変からの脳血流量の減少に起因するのかは不明である。本研究の目的は、CAD 患者を対象に、頸部および脳の撮像パラメータを用いて、脳卒中の最も可能性の高い機序を同定することであった。

方法: 本研究は、当院の倫理委員会によって承認された。インフォームド・コンセントは免除された。我々は、連続した CAD 患者について、脳血管超音波画像、頸部 MR 血管造影画像および脳卒中の脳の MRI 画像を後向きに評価した。頭蓋内の塞栓が T2* 強調画像上で磁化率血管サイン (susceptibility vessel sign) として直接可視化された症例、または拡散強調画像上で脳軟膜動脈領域の梗塞が認められた場合を、塞栓性機序とした。分水嶺梗塞の症例、または分水嶺梗塞と脳軟膜領域の梗塞を併発した症例においては、以下の所見が2項目以上認められる場合に、血行力学的機序とした：重度の狭窄または閉塞性 CAD、脳血管超音波検査での頭蓋内流速の低下または MR 血管造影画像上のシグナル減少、もしくは FLAIR 像上の高信号血管サイン (hyperintense vessel sign)。それ以外の患者は、複合的な機序をもととみなした。

結果: 連続した172例の CAD 患者のうち100例（58%）で、拡散強調画像上に急性脳卒中が認められた。脳卒中は、100例中85例では血栓栓塞性の機序に起因し、12例では血行力学的機序に起因し、3例では複合的な機序に起因していた。

結論: CAD 患者の脳卒中は、画像上は直接および間接的な血管原性 (artery-to-artery) 閉塞に関連している頻度が最も高く、この結果は今後の治療試験をデザインする上で有用である。