Causes and Mechanisms of Territorial and Nonterritorial Cerebellar Infarcts in 115 Consecutive Patients

Pierre Amarenco, MD; Claude Lévy, MD; Ariel Cohen, MD; Pierre-Jean Touboul, MD; Etienne Roullet, MD; Marie-Germaine Bousser, MD

Background and Purpose  Territorial cerebellar infarcts have mainly a thromboembolic mechanism. Cerebellar infarcts less than 2 cm in diameter have recently been reported as nonterritorial infaracts, but it is not clear whether they are low-flow or embolic infarcts. The aim of the present study was to compare the characteristics and causes of territorial and nonterritorial infarcts in a prospective series of 115 patients.

Methods  We collected data from 115 consecutive patients with cerebellar infarcts (79 territorial and 36 nonterritorial [ie, less than 2 cm]), using magnetic resonance imaging (88 patients) and computed tomography.

Results  Patients with territorial infarcts and those with nonterritorial infarcts had similar vascular risk factors and clinical presentations and an equal frequency of cardiac source of embolism (32% versus 42%; P=NS) and of large artery occlusive disease (23% versus 19%; P=NS). Occlusive lesions of large arteries at angiography occurred at the level of one cerebellar artery (5% versus 0%; P=NS) and proximal to the ostia of the cerebellar arteries (18% versus 19%; P=NS).

Infarcts distal to occlusive lesions were subdivided into unilateral vertebral artery occlusive disease (presumed artery-to-artery embolic mechanism; 18% versus 5%; P=NS) and low-flow state distal to bilateral vertebral or basilar artery occlusion (presumed hemodynamic mechanism; 0% versus 14%; P=.004). Patients with nonterritorial infarcts had more frequent hypercoagulable state (17% versus 1.25%; odds ratio, 15.6 [95% confidence interval, 1.8 to 135]). For the remaining patients, the mechanism of the infarct was unknown (34% versus 22%; P=NS).

Conclusions  Cerebellar infarcts less than 2 cm in diameter (ie, nonterritorial) have the same high rate of embolic mechanism as territorial infarcts (47% versus 49%; P=NS), have more frequent hypercoagulable state, and sometimes have a hemodynamic mechanism. (Stroke. 1994;25:105-112.)

Key Words  • cerebellar infarction • epidemiology • embolism • magnetic resonance imaging

With magnetic resonance imaging, it has become possible to diagnose cerebellar infarcts and delineate their topography with high sensitivity. Infarcts can be recognized in the territory (ie, territorial infarcts) of the posterior inferior (PICA), anterior inferior (AICA), superior (SCA), cerebellar arteries, and their branches (eg, the medial branch and the lateral branch of the PICA and SCA). More recently, very small cerebellar infarcts less than 2 cm in diameter have been recognized located at the boundary zone between various territories (ie, nonterritorial infarcts). The precise analysis of the locations of cerebellar infarcts may help clinicians predict the mechanism of ischemia: SCA infarcts are mainly embolic, PICA infarcts are equally divided in embolic and atherosclerotic occlusion of the intracranial vertebral artery, and AICA infarcts are mainly due to basilar artery or in situ atherosclerotic occlusion of a basilar artery branch. Although these territorial infarcts are mainly thromboembolic, it is not clear whether nonterritorial infarcts have a different pathogenesis and are due to a hemodynamic rather than a thromboembolic mechanism. This distinction could have important implications for the management of patients with cerebellar infarctions less than 2 cm in diameter.

The aim of the present study was to compare the characteristics and causes of territorial and infarcts less than 2 cm in diameter (ie, nonterritorial) in a prospective study of 115 consecutive patients with cerebellar infarcts and to examine the possibility of different mechanisms within very small nonterritorial cerebellar infarcts.

Subjects and Methods  Patients were selected in the following manner. From January 1987 through May 1993 we prospectively collected data from patients with cerebellar infarcts referred to us at the Hôpital Saint-Antoine in Paris, France; the patients had been diagnosed by computed tomography (CT), magnetic resonance imaging (MRI), or both. They represented 115 patients, which is 10.45% of patients with brain ischemia seen during the same period of time. Eighty-eight were diagnosed during the last 3 years. Twenty-seven were diagnosed with CT only (52% in the first 3 years) and 88 with MRI (86% in the last 3 years).

All patients had electrocardiography, 96 had transthoracic echocardiography, and 72 transesophageal echocardiography. Four-vessel angiography was performed in 52 patients; all others had complete neck ultrasound and transcranial Doppler examinations. Angiography was usually not performed in
Classification of Infarcts and Causes

Infarcts were classified into territorial infarcts (ie, occupying the territory of the PICA, medial branch of PICA [mPICA], lateral branch of PICA [IPICA], SCA, medial branch of SCA [mSCA], lateral branch of SCA [lSCA], or AICA) and non-territorial infarcts, either cortical or deep, that could not be located in a defined territory according to previously published templates (Figure). These nonterritorial infarcts were all very small (less than 2 cm in diameter).

Causes were classified as follows:

**Presumed cardiac source of embolism.** This classification included acute myocardial infarction (less than 3 weeks), known atrial fibrillation with or without mural thrombus, acute bacterial endocarditis, dilated cardiomyopathy, mitral stenosis or prosthetic heart valve, and atrial septal aneurysm (excursion greater than 15 mm) with normal extracranial and intracranial arteries (isolated patent foramen ovale, isolated wall hypokinesia, and isolated left atrial enlargement were not considered likely sources of embolism).

**Large-artery disease.** Angiography or autopsy evidence of (1) vertebral or basilar artery occlusion with otherwise documented atherosclerotic disease, (2) atherosclerotic plaque of vertebral or basilar artery with tight stenosis, and (3) extracranial vertebral artery dissection with occlusion or stenosis was included in this category. These patients were further subdivided into those with (1) occlusion at the level of one cerebellar artery or (2) proximal lesions with either unilateral vertebral artery occlusive disease (presumed artery-to-artery embolism) or bilateral vertebral artery or basilar artery occlusive disease and a low-flow state at angiography (presumed hemodynamic mechanism).

**Presumed branch disease (in situ atherosclerotic branch occlusion).** This category included infarcts in the territory of one branch of a cerebellar artery (eg, short circumferential artery rising from AICA or mPICA) in the absence of large-artery occlusive disease and of cardiac source of embolism and in the presence of either diabetes or angiographically documented branch occlusion.

**Others.** Patients with hypercoagulable state, hematologic disorders (eg, thrombocytopenia), documented cholesterol emboli, or previously known small-artery disease (eg, multiple intracranial lacunas with no source of embolism or large artery disease) were included in this category.

**Unknown.** This category included patients with none of the conditions defined above or in whom no angiography was performed; these patients could have one or more risk factors, isolated patent foramen ovale or mitral valve prolapse, atherosclerotic plaques in the anterior circulation, or vertebral or basilar artery occlusion of unknown nature.

Ninety-six patients could be easily classified in the five etiopathological groups defined above because of the presence of cardiac source of embolism or hypercoagulable state (angiography was not routinely performed in these patients) or because cerebral angiography or complete autopsy was performed. Nineteen additional patients with no cardiac source of embolism and no angiography performed were classified as of undetermined etiopathogenesis. This group of 19 patients was analyzed together with the unknown cause group. All 19 patients had complete neck ultrasound examination and transcranial Doppler performed.

Statistical Analysis

We used the $\chi^2$ test to compare the territorial versus nonterritorial infarct groups. Results reached statistical significance at $P < .05$.

Results

Distribution

The patients were separable into two groups: 79 with territorial and 36 with nonterritorial infarcts. Nonterritorial infarcts were all less than 2 cm in diameter. Territorial infarcts involved the PICA territory (35 patients), including 19 mPICA and 4 IPICA territory infarcts; SCA territory (30 patients), including 16 mSCA and 6 lSCA territory infarcts; both PICA and SCA territories (4 patients); AICA territory (9 patients); and PICA, SCA, and AICA territories (1 patient). Thirty-six patients had nonterritorial infarcts located at the boundary zone of the PICA, SCA, AICA, and of their branches, either deep (in 3 patients), cortical (in 31), or both (in 2). Six of these were associated with a posterior cerebral artery territory infarct. Eleven patients with territorial infarcts also had contralateral nonterritorial infarcts. These included mainly patients with SCA territory infaracts (7 patients) and 1 patient with both PICA and PCA territory infarcts; the others had PICA or AICA infarcts.

Clinical Features

Vertigo was the most frequent symptom in both large territorial and nonterritorial infarcts. Vomiting, unsteadiness of gait, and dysarthria were significantly more frequent in patients with territorial infarcts, whereas transient loss of consciousness was more frequent in
patients with nonterritorial infarcts (Table 1). At neurological examination, ipsilateral limb dysmetria, ipsilateral axial lateropulsion, gait ataxia, and dysarthria were the four most frequent signs in both types of infarcts, but ipsilateral limb dysmetria was significantly more frequent in patients with territorial infarcts. A variety of other signs (Horner’s syndrome, facial palsy, nystagmus) were occasionally found, mainly in patients with territorial infarcts. Neurological examination was normal in 37% of patients with nonterritorial infarcts but in only 4% of those with territorial infarcts (P=.0001).

Baseline Characteristics

No statistical difference was found between territorial and nonterritorial infarcts in regard to risk factors and underlying causal conditions (Table 2).

Causes and Mechanisms

Causes and mechanisms are indicated in Tables 3 and 4. No difference was found between the two groups in regard to cardioembolism (32% versus 42%), presence of large-artery occlusive disease (23% versus 19%), and unknown causes (34% versus 22%). In contrast, the two groups differed in the frequency of atherosclerotic branch disease (10% versus 0%) and in that of hypercoagulable state (1.25% versus 17%; odds ratio [OR], 15.6 [95% confidence interval (CI), 1.80 to 135]; P=.005).

Patients with nonterritorial infarcts had more frequent hypercoagulable state. These patients had thrombocythemia, polycythemia, disseminated intravascular coagulation, hyperesinophilia, and systemic lupus with antiphospholipid antibodies. One patient had cholesterol emboli, and one presumably had lipohyalnosis.
TABLE 2. Baseline Characteristics in 115 Consecutive Patients With Cerebellar Infarct

<table>
<thead>
<tr>
<th></th>
<th>Territorial Infarcts* (n=79)</th>
<th>Nonterritorial Infarct† (n=36)</th>
<th>Odds Ratio (95% Cl)</th>
<th>P (χ² Test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, y</td>
<td>59.53</td>
<td>59.83</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Male</td>
<td>54 (68%)</td>
<td>21 (58%)</td>
<td>0.65 (0.29-1.46)</td>
<td>.295</td>
</tr>
<tr>
<td>Hypertension</td>
<td>40 (51%)</td>
<td>23 (64%)</td>
<td>1.73 (0.77-3.88)</td>
<td>.185</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>25 (32%)</td>
<td>15 (42%)</td>
<td>1.54 (0.68-3.48)</td>
<td>.295</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>17 (22%)</td>
<td>8 (22%)</td>
<td>1.04 (0.40-2.70)</td>
<td>.932</td>
</tr>
<tr>
<td>High serum cholesterol</td>
<td>15 (19%)</td>
<td>7 (19%)</td>
<td>1.03 (0.38-2.80)</td>
<td>.953</td>
</tr>
<tr>
<td>Obesity</td>
<td>13 (16%)</td>
<td>10 (28%)</td>
<td>1.95 (0.76-5.00)</td>
<td>.159</td>
</tr>
<tr>
<td>Patent foramen ovale</td>
<td>13 (16%)</td>
<td>3 (8%)</td>
<td>0.46 (0.12-1.73)</td>
<td>.243</td>
</tr>
<tr>
<td>Atrial septal aneurysm</td>
<td>8 (10%)</td>
<td>5 (14%)</td>
<td>1.43 (0.43-4.73)</td>
<td>.555</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>8 (10%)</td>
<td>2 (6%)</td>
<td>0.52 (0.11-2.59)</td>
<td>.420</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>6 (8%)</td>
<td>2 (6%)</td>
<td>0.72 (0.14-3.73)</td>
<td>.690</td>
</tr>
<tr>
<td>Mural thrombus</td>
<td>1 (1%)</td>
<td>4 (11%)</td>
<td>9.75 (1.05-90.64)</td>
<td>.056*</td>
</tr>
<tr>
<td>Peripheral artery disease</td>
<td>3</td>
<td>2</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>0</td>
<td>2</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Mitral valve prolapse</td>
<td>2</td>
<td>1</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Cancer</td>
<td>3</td>
<td>0</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Antiphospholipid antibody syndrome</td>
<td>0</td>
<td>2</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>2</td>
<td>0</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Lupus erythematosus</td>
<td>0</td>
<td>2</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Valvulopathy</td>
<td>0</td>
<td>1</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Hypereosinophilia</td>
<td>0</td>
<td>1</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Thrombocythemia</td>
<td>0</td>
<td>1</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Polycythemia</td>
<td>0</td>
<td>1</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

Cl indicates confidence interval.

*Involving the territory of the posterior inferior cerebellar artery (PICA), medial branch of PICA, lateral branch of PICA, superior cerebellar artery (SCA), medial branch of SCA, lateral branch of SCA, or anterior inferior cerebellar artery in isolation or in combination.

†Infarcts <2 cm (located not in a defined territory, mainly at the boundary zones of the cerebellar artery territories or of their branches).

because of the presence of multiple supratentorial lacunes. The patient with hypereosinophilia had multiple infarcts in watershed territories of both the cerebellum and cerebrum; he died 7 months later. At necropsy no arteritis was found, but multiple end arteries were occluded by fibrous thrombi. Examination of the heart showed severe and thick subintimal fibrosis and a very large organized fibrin thrombus filling 40% of the left ventricular cavity.

Although there was no difference between territorial and nonterritorial infarcts in regard to the presence of large-artery occlusive disease, some differences were found regarding the mechanism of the infarcts. We distinguished between occlusions at the level of the ostium of one cerebellar artery and occlusive diseases proximal to the ostium. Occlusions at the level of the ostium of one cerebellar artery were found in 4 patients, all of whom had territorial infarcts. In unilateral occlusive disease of large arteries proximal to the ostium of cerebellar arteries, an arterial source of embolism was present in the vertebral artery (mainly atheroma of the origin of the vertebral artery) in 14 (18%) patients with territorial infarcts and 2 (6%) with nonterritorial infarcts. This difference is not statistically significant (OR, 0.27 [95% CI, 0.06 to 1.27]). The likely mechanism was therefore artery-to-artery embolism.

Bilateral vertebral artery or basilar artery occlusive disease proximal to the ostium of the cerebellar arteries with a low-flow state at angiography (presumed hemodynamic mechanism) was found in 5 (14%) patients with nonterritorial infarcts but in none with territorial infarcts (P=.004). The first patient had a proximal basilar artery occlusion, with filling of the distal basilar artery and of the posterior cerebral arteries by the posterior communicating artery when the carotid artery was injected, and filling of the SCA branches via the cortical anastomoses with the PICA when the vertebral artery was injected. The second patient had an occlusion at the origin of the right vertebral artery and a tight (greater than 95%) atherosclerotic stenosis of the left vertebral artery with bilateral distal refilling via the deep cervical arteries. He had two ipsilateral infarcts less than 2 cm in diameter between SCA and PICA and between AICA and PICA, probably "border zone" infarcts. The third patient had an occlusion of the right vertebral artery and a dissecting aneurysm of the left
vertebral artery in its extracranial course. Bilateral infarcts less than 2 cm in diameter were present between PICAs and SCAs and were probably border zone. The fourth patient had bilateral tight stenosis of the vertebral artery origin with an infarct less than 2 cm in diameter between both mPICAs on the vermis, which was probably border zone. Finally, the fifth patient had a tight stenosis of the basilar artery and a border zone infarct between AICA and PICA.

Overall, a presumed embolic mechanism (cardiac plus artery-to-artery embolisms) was equally frequent, with a particularly high rate in both territorial and nonterritorial infarcts (49% versus 47%). In contrast, a hemodynamic mechanism was found only in patients with nonterritorial infarcts (14%). Likewise, hypercoagulable states were rare and mostly found in patients with nonterritorial infarcts (17% versus 1.25%).

Among 35 patients with infarcts of unknown cause, 16 patients had complete investigation of the heart and cerebral arteries (15 had transesophageal echocardiography plus angiography and 1 autopsy), of which 7 had risk factors for atherosclerosis, 2 had a patent foramen ovale, 2 had both risk factors and patent foramen ovale, and 5 had no risk factors. The remaining 19 patients had no cardiac source of embolism and no angiography performed (all had neck ultrasound examination and transcranial Doppler): 15 had risk factors including 5 patients with normal ultrasound examination of cerebral arteries, 8 with internal carotid artery stenosis, and 3 with ultrasound suggesting severe vertebrobasilar artery occlusive disease (1 with stenosis of intracranial vertebral artery, 1 with basilar artery narrowing, and 1 with bilateral intracranial vertebral artery occlusion); finally, 4 patients had no risk factors for atherosclerosis (including 2 with left atrial enlargement and 1 with patent foramen ovale). Further redistribution of these 19 patients in the five etiopathological groups did not modify statistical results.

**Discussion**

In a previous study of cerebellar infarcts, we determined that very small (less than 2 cm) infarcts were located at the boundary zones of the territories of the PICA, SCA, AICA, or of their branches. The mechanism was variable. Twenty-three percent of patients had postural symptoms, but an association with systemic hypotension was rare (4%). Focal hypoperfusion was far more frequent and due to large-artery occlusive disease (57% of patients, divided into 34% with atherosclerotic occlusive disease and 23% with cardiac source of embolism) and to end-artery disease (19% of patients). We concluded that the mechanism of these small nonterritorial cerebellar infarcts could be either hemodynamic or embolic. Therefore, to address this point, in the present study we compared the characteristics and causes of 36 nonterritorial (ie, less than 2 cm) cerebellar infarcts with 79 territorial infarcts in 115 consecutive patients, prospectively collected in one center. Symptoms and signs were qualitatively similar in both territorial and nonterritorial infarcts, but some quantitative differences were observed (Table 1). Most symptoms (mainly vertigo, vomiting, unsteadiness of gait,
<table>
<thead>
<tr>
<th>Causes</th>
<th>Total Cases (n=115)</th>
<th>All PICA (n=35)</th>
<th>Full PICA (n=12)</th>
<th>mPICA (n=19)</th>
<th>IPICA (n=4)</th>
<th>All SCA (n=30)</th>
<th>Full SCA (n=8)</th>
<th>mSCA (n=6)</th>
<th>ISCA (n=16)</th>
<th>PICA+SCA (n=4)</th>
<th>PICA+SCA+AICA (n=1)</th>
<th>AICA (n=9)</th>
<th>Nonterritorial Infarcts (Infarcts &lt;2 cm) (n=36)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac source of emboli</td>
<td>40 (35%)</td>
<td>12 (34%)</td>
<td>7 (37%)</td>
<td>12 (40%)</td>
<td>3</td>
<td>8 (50%)</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>Unknown†</td>
<td>35 (30%)</td>
<td>14 (40%)</td>
<td>3 (9%)</td>
<td>9 (47%)</td>
<td>0</td>
<td>12 (40%)</td>
<td>3</td>
<td>2</td>
<td>7 (44%)</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Atherosclerotic occlusive disease of the VA or BA</td>
<td>17 (15%)</td>
<td>3 (9%)</td>
<td>2 (9%)</td>
<td>2 (9%)</td>
<td>1</td>
<td>5 (17%)</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>2†</td>
</tr>
<tr>
<td>BA</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2†</td>
</tr>
<tr>
<td>Intracranial VA</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>VA origin</td>
<td>11 (10%)</td>
<td>3 (9%)</td>
<td>2 (18%)</td>
<td>1 (10%)</td>
<td>5 (17%)</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2†+1</td>
</tr>
<tr>
<td>Branch disease</td>
<td>8 (7%)</td>
<td>2 (25%)</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7 (%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>End-artery disease</td>
<td>7 (6%)</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>Dissection of the VA§</td>
<td>8 (7%)</td>
<td>4 (50%)</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1+1†</td>
</tr>
</tbody>
</table>

All 115 patients had cardiac workup; 52 had cerebral angiography and 3 had complete autopsy. PICA indicates posterior inferior cerebellar artery; mPICA, medial branch of PICA; IPICA, lateral branch of PICA; SCA, superior cerebellar artery; mSCA, medial branch of SCA; ISCA, lateral branch of SCA; AICA, anterior inferior cerebellar artery territory; VA, vertebral artery; and BA, basilar artery. *Diagnosis only based on angiography (15), autopsy (1), or clinical evidence (1) of BA occlusion (locked-in syndrome with massive pontine infarction and bilateral cerebellar and occipital infarcts). †Fifteen had angiography performed and 1 had complete autopsy including extracranial and intracranial vessels examination. ‡Arteritis, thrombocythemia, polycythemia, hypereosinophilia, disseminated intravascular coagulation, cholesterol emboli, multilacunes. §Diagnosis based on angiography. ||Including 7 with patent foramen ovale (2 mPICA and 5 SCA), 9 internal carotid artery plaques, 8 with only risk factors for atherosclerosis (hypertension 8, diabetes 2, smoking 2, peripheral artery disease 1), and 5 with no risk factors. ††Bilateral VA occlusive disease.
headache, and dysarthria) were more frequent in patients with territorial infarcts, and this reached statistical significance for vomiting, unsteadiness of gait, and dysarthria. The only symptom that was significantly more frequent in those with nonterritorial infarcts was loss of consciousness, present in 6 patients and in only 1 with territorial infarcts. Among these 6 patients were 3 of the 5 with a presumed hemodynamic border zone infarct (OR, 40 [95% CI, 5 to 308]). At examination, again all signs (mainly limb dysmetria, ipsilateral axial lateropulsion, gait ataxia, and dysarthria) were more frequent in patients with territorial infarcts than in those with nonterritorial infarcts, and this was statistically significant for ipsilateral dysmetria (61% versus 17%). More than a third of patients with nonterritorial infarcts but only 4% of those with large territorial infarcts had no neurological signs. It thus seems that in a given patient, the clinical presentation does not allow differentiation between territorial and nonterritorial infarcts except for the rare occurrence of transient loss of consciousness, which points to a very small nonterritorial infarct and might suggest a hemodynamic mechanism (OR, 40 [95% CI, 5 to 308]).

In regard to vascular risk factors and causes, again territorial and nonterritorial infarcts were very similar (Table 3). One third of patients in both groups had a cardiac source of embolism, one fourth had large-artery occlusive disease, and in one fourth no precise cause could be identified. In the small group of remaining patients, small-branch atheroma prevailed in territorial infarcts and hypercoagulable states in nonterritorial infarcts.

The main identified mechanism in both types of infarcts was embolism (either cardiac or artery-to-artery embolism). This is already well established in territorial infarcts but also suggests that infarcts less than 2 cm in diameter are in fact very small territorial infarcts due to the involvement of small distal arteries. The higher frequency of hypercoagulable states in patients with very small nonterritorial infarcts also points to involvement of small distal arteries. Thus, nonterritorial infarcts appear to correspond much more frequently to “end zone” infarcts, since 64% of these patients had an embolic mechanism or a local disease likely explaining the occlusion of a very distal artery, rather than to border zone infarcts, since in only 5 patients (14%) was a low-flow state demonstrable distal to occlusion. Thus, hemodynamic failure was the likely mechanism in only 5 patients (with border zone infarct), but in those patients the coexistence of a small distal embolus may also have occurred. These two mechanisms (ie, low flow and embolism) are not mutually exclusive. It is likely that both conditions might be needed in some patients for emboli to be symptomatic.

This issue of whether infarcts located at the watershed areas of two arterial territories are due to very small emboli in very distal branches (end zone infarcts) or to hemodynamic failure in the very distal field of the arterial territory (border zone infarct) is still unclarified. There is no way to definitely determine this on an individual basis either intravital or at autopsy. Positron emission tomography studies did not show evidence of misery perfusion in watershed areas in patients with watershed infarcts, although this remains controversial. 

On one hand, a small embolus may detach from a proximal occlusive thrombus and migrate distally in a very small branch supplying part of the watershed area. On the other hand, cortical anastomoses, in the cerebellum as well as in the cerebral hemispheres, protect the brain tissue against ischemia when an ipsilateral artery is totally occluded. Therefore, small parts of brain tissue may be damaged in the very distal field of a proximally occluded artery if some anastomoses are lacking. This is particularly true for the cerebellar arterial blood supply, which respects the rule that the extrinsic arterial anatomy is variable but that the intrinsic arterial (ie, penetrating arteries that are terminals with no collateral) disposition is fixed.

In the present series we found features in common in both territorial and nonterritorial (ie, less than 2 cm) infarcts in regard to vascular risk factors, clinical presentation, causes, and mechanisms. This suggests that these two types of infarction are essentially the same and that their extent and site simply more likely depend on the size of the embolus causing the infarct. The smaller the embolus is, the smaller the recipient artery, and therefore the more distal is the (end zone) infarct. Therefore, there should be no difference in the evaluation and treatment of patients with territorial and those with nonterritorial cerebellar infarcts, except in the few cases of nonterritorial infarcts with a low-flow state.

Acknowledgment

We wish to thank Louis R. Caplan for his helpful criticism of this article and Michael G. Hennerci and his staff for helpful discussion.

References


Causes and mechanisms of territorial and nonterritorial cerebellar infarcts in 115 consecutive patients.
P Amarenco, C Lévy, A Cohen, P J Touboul, E Roullet and M G Bousser

Stroke. 1994;25:105-112
doi: 10.1161/01.STR.25.1.105

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/25/1/105

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/