Posterior Cerebral Artery Infarction From Middle Cerebral Artery Infarction

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Background: While it is known that posterior cerebral artery (PCA) infarction may simulate middle cerebral artery (MCA) infarction, the frequency and localization of this occurrence are unknown.

Objective: To determine the frequency of PCA infarction mimicking MCA infarction and the territory of the PCA most commonly involved in this simulation.

Design: We studied 202 patients with isolated infarction in the PCA admitted to our stroke center to determine the frequency of PCA infarction simulating MCA infarction, the involved PCA territory, and the patterns of clinical presentation.

Results: We found 36 patients (17.8%) with PCA ischemic stroke who had clinical features suggesting MCA stroke. The PCA territory most commonly involved was the superficial PCA territory (66.7%), followed by the proximal PCA territory (16.7%) and both the proximal and the superficial PCA territories (16.7%). The principal stroke mechanism was cardioembolic (54.1%) in the superficial PCA territory, lacunar (46.2%) in the proximal PCA territory, and undetermined (40.2%) in both the proximal and the superficial territories. Among the 36 patients, the most common clinical associations were aphasia (13 patients), visuospatial neglect (13 patients), and severe hemiparesis (7 patients).

Conclusions: Posterior cerebral artery infarction simulating MCA infarction is more common than previously thought. Early recognition of the different stroke subtypes in these 2 arteries may allow specific management.

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Methods

We studied all patients with a first, isolated infarction restricted to the PCA who were admitted to our stroke center between January 1, 1983, and December 31, 2003. The patients' data were encoded prospectively into the computerized Lausanne Stroke Registry. We included patients in whom brain images showed infarctions involving only the PCA. We excluded all patients with uncertain neuroradiological confirmation of PCA ischemia, patients with an associated stroke in the anterior circulation or brainstem (except the midbrain), patients with degenerative diseases, and patients with extensive white matter disease, which could confound the neurological examination. Also, we did not include patients in whom the stroke diagnosis was uncertain. All patients underwent a standard neurological and neuropsychological examination.
tion and systematic investigations, including brain computed tomography, Doppler ultrasonography, electrocardiography, and routine blood tests. Magnetic resonance imaging, magnetic resonance angiography, arteriography, transesophageal echocardiography, and 24-hour electrocardiography Holter monitoring were performed on selected patients.

The demographic characteristics, cerebrovascular risk factors, clinical features, and probable etiology of stroke were assessed using the TOAST (Trial of ORG 10172 in Acute Stroke Treatment) classification. Strokes were divided into 3 groups according to the infarction location as seen on neuroimaging: (1) proximal or deep (PPCA), (2) superficial or cortical (SPCA), or (3) both (P+SPCA). Proximal PCA infarctions mimic MCA stroke differently from SPCA infarctions. A PPCA localization was defined as involving the midbrain, thalamus, and posterior limb of the internal capsule. An SPCA localization was defined as involving the territories of the anterior and posterior temporal, calcarine, and parietooccipital arteries. We defined a PCA infarction simulating an MCA infarction as a PCA stroke with the signs and symptoms reported as typical of an MCA stroke, combined with the absence of classic findings of PCA infarction. The following signs and symptoms of PCA infarction were considered classic:<sup>8-10</sup>: sensory deficits, movement disorders, third nerve palsy, visual field defects, and visual hallucination, perseveration, and agnosia for SPCA lesions; and discrete motor abnormalities, headache, visual field defects, and sensory deficits for P+SPCA lesions. Aphasia and visuospatial neglect were considered typical of PCA stroke, combined with the absence of classic findings of PCA infarction. The following signs and symptoms of PCA infarction were considered classic:<sup>8-10</sup>: sensory deficits, movement disorders, third nerve palsy, and vertical gaze abnormalities for PPCA lesions; headache, visual field defects and visual hallucination, perseveration, and agnosia for SPCA lesions; and discrete motor abnormalities, headache, visual field defects, and sensory deficits for P+SPCA lesions. Aphasia and visuospatial neglect were considered typical of PCA stroke only in PPCA infarctions, because the former may be found in up to 50% of left thalamic lesions<sup>11</sup> and the latter in up to 62% of right thalamic lesions.<sup>12</sup> Moreover, the frequencies of aphasia and visuospatial neglect are 0% to 8%,<sup>13-15</sup> and 7% to 12%,<sup>8,15</sup> respectively, in patients with SPCA stroke. Severe motor deficit,<sup>16-19</sup> alien hand syndrome,<sup>19</sup> grasping,<sup>20</sup> and asterixis<sup>21</sup> are atypical but have been reported in PCA stroke.

Categorical data are presented as percentages and were analyzed using χ² test and estimated 95% confidence intervals. Statistical analysis was performed using SPSS version 11.0 (SPSS Inc, Chicago, Ill). P<.05 was considered statistically significant.

RESULTS

We initially identified 391 patients with a first symptomatic event in the PCA; after applying the exclusion criteria, 202 patients remained for analysis. The distribution of clinical features and risk factors is summarized in Table 1. Of the 202 patients, 122 (60.4%) were men, and the mean age at onset for all PCA strokes was 61 years (PPCA group, 60 years; SPCA group, 62 years; and P+SPCA group, 58 years). The lesions were located in the left hemisphere in 111 patients (55.0%), the right in 79 (39.1%), and bilaterally in 12 (5.9%).

The presumed etiologies are presented in Figure 1. The main cause of all PCA strokes was cardioembolism (39.4%), followed by undetermined (26.9%), lacunar (19.4%), and atherothrombotic (13.5%) etiologies. When we analyzed each PCA territory separately, the most common etiologies for PPCA infarctions were lacunar (46.2%) and cardioembolic (30.2%) (P<.01), while cardioembolic (54.1%), undetermined (23.3%), and atherothrombotic (19.9%) etiologies (P<.002) were the most frequent for SPCA infarctions. The presumed causes of P+SPCA stroke were undetermined (40.2%), cardioembolic (34.2%), and atherothrombotic (19.9%) (P<.001).

The clinical features are summarized in Table 2. The most common clinical findings were motor weakness (114 patients [56.4%]), visual field abnormalities (106 patients [52.5%]), and sensory deficits (91 patients [45.0%]).

Posterior cerebral artery infarctions simulating MCA infarctions occurred in 36 patients (17.8%) (Figure 2). Of these, 24 (66.7%) involved the SPCA, 6 (16.7%) the PPCA, and 6 (16.7%) the P+SPCA territories. The most common etiology for all PCA infarctions combined was cardioembolic (38.8%) (P<.04).

For all 36 PCA strokes simulating MCA infarctions, the most common clinical signs were aphasia (13 patients [36.1%]), visuospatial neglect (13 patients [36.1%]),
and severe hemiparesis (7 patients [19.4%]), but these findings were not statistically significant (P ≥ .3) (Table 3). The main clinical findings associated with the simulation were the following: in the PPCA territory, severe motor deficits (50.0%) and asterixis (33.3%); in the SPCA territory, aphasia (54.1%) and visuospatial neglect (34.1%); and in the P + SPCA territories, severe motor deficits (50.0%) and asterixis (50.0%).

To our knowledge, this is the first large clinical study that has focused on the frequency of PCA infarctions mimicking MCA infarctions. Chambers et al described 12 patients with PPCA occlusion in whom the initial clinical diagnosis was an MCA infarction. That study did not estimate the frequency of such misdiagnosis, and infarctions of the SPCA territory were not considered. Chavot et al identified mimicry of MCA infarctions in 18.3% of PCA infarctions but did not distinguish between PPCA and SPCA strokes and did not specify the criteria for diagnosing the simulation.

The most common abnormality of hemispherical PCA infarctions is contralateral visual field defects due to infarction of the striate cortex or the optic radiations. The high frequency is probably due to the inclusion of patients with thalamic infarctions. Furthermore, Georgiadis et al suggest that the presence of sensory symptoms or signs in patients with PCA occlusive disease indicates lateral thalamic ischemia. Our series confirms this observation, as only 9.7% of our patients with hemispherical PCA stroke had sensory abnormalities. Moreover, sensory deficits were found in 60.0% of patients with PPCA territory infarctions and in 72.0% of patients with P + SPCA territory infarctions. In studies of series of patients with SPCA infarctions, motor deficits were reported in 28% of patients. In our study, motor deficits were seen in 36.1% of patients with SPCA infarction vs 56.4% of patients in the entire cohort. In addition, there are a few recorded cases of hemiplegia in patients with PCA infarctions, mainly as a result of lesions of the cerebral peduncle or the anterior segment of the posterior limb of the internal capsule, and this is the main clinical feature distinguishing PPCA infarctions from other types of PCA strokes. Aphasia can occur in dominant hemisphere infarctions involving the ventral lateral thalamic nucleus. Although rare, anomic and transcortical sensory aphasia has been documented in patients with large infarctions involving the left posterior temporal artery. In our se-

**Table 2. Clinical Findings**

<table>
<thead>
<tr>
<th>Finding</th>
<th>All Patients (N = 202)</th>
<th>PPCA Group (n = 80)</th>
<th>SPCA Group (n = 72)</th>
<th>P + SPCA Group (n = 50)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor deficit</td>
<td>114 (56.4)</td>
<td>50 (62.5)</td>
<td>26 (36.1)</td>
<td>38 (76.0)</td>
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<tr>
<td>Visual field defect</td>
<td>106 (52.5)</td>
<td>1 (1.3)</td>
<td>64 (88.9)</td>
<td>41 (82.0)</td>
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<tr>
<td>Sensory deficit</td>
<td>91 (45.0)</td>
<td>48 (60.0)</td>
<td>7 (9.7)</td>
<td>36 (72.0)</td>
</tr>
<tr>
<td>Headache</td>
<td>59 (29.2)</td>
<td>12 (15.0)</td>
<td>20 (36.1)</td>
<td>21 (42.0)</td>
</tr>
<tr>
<td>Abnormal movement</td>
<td>12 (5.9)</td>
<td>6 (7.5)</td>
<td>2 (2.8)</td>
<td>4 (8.0)</td>
</tr>
<tr>
<td>Neuropsychological deficit</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>28 (13.9)</td>
<td>13 (16.3)</td>
<td>1 (1.4)</td>
<td>14 (28.0)</td>
</tr>
<tr>
<td>Visuospatial neglect</td>
<td>52 (25.7)</td>
<td>19 (23.8)</td>
<td>11 (15.3)</td>
<td>22 (44.0)</td>
</tr>
<tr>
<td>Memory</td>
<td>66 (32.7)</td>
<td>24 (30.0)</td>
<td>15 (20.8)</td>
<td>27 (54.0)</td>
</tr>
</tbody>
</table>

**Table 3. Patients With Posterior Cerebral Artery Stroke Simulating Middle Cerebral Artery Stroke**

<table>
<thead>
<tr>
<th>Localization</th>
<th>PPCA Group (n = 6)</th>
<th>SPCA Group (n = 24)</th>
<th>P + SPCA Group (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe motor deficit</td>
<td>3</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Asterixis</td>
<td>2</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Palmmontal reflex</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>plus grasping</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visuospatial neglect</td>
<td>0</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Aphasia</td>
<td>0</td>
<td>9</td>
<td>0</td>
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<tr>
<td>Visuospatial neglect</td>
<td>0</td>
<td>4</td>
<td>0</td>
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<tr>
<td>plus aphasia</td>
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<td></td>
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<tr>
<td>Palmmontal reflex plus</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>visuospatial neglect</td>
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</tr>
</tbody>
</table>

**Table 2. Clinical Findings**

Abbreviations: PPCA, proximal posterior cerebral artery; P + SPCA, proximal and superficial posterior cerebral artery; SPCA, superficial posterior cerebral artery.

*Data are given as number (percentage).
ties, aphasia was present in only 1.4% of patients with SPCA lesions but in 28.0% of patients with P+SPCA lesions. This high frequency of aphasia in thalamic strokes confirms the role of this area in language disturbances. Visuospatial neglect is uncommon in SPCA infarctions. However, in PPCA strokes, this clinical feature may be found in 17.5% to 60% of patients due to paramedian and tuberothalamic infarctions. In the present series, frequencies of visuospatial neglect in SPCA, PPCA, and P+SPCA strokes were 15.3%, 23.8%, and 44.0%, respectively.

Lacunar infarction was the most common stroke subtype (46.2%) in patients with PPCA infarctions. The main presumed cause of isolated SPCA infarctions in our study (54.1%) was cardioembolism, which is in agreement with the 57% to 77% reported previously. Cardioembolism was the principal etiology implicated in PPCA stroke simulation of MCA stroke. Nevertheless, because most of the patients had only a computed tomographic scan as the diagnostic examination, we cannot exclude the possibility that some patients could have had undetected microembolizations in the MCA concomitant with PCA infarctions.

Mimicry of MCA stroke was found in 36 patients (17.8%) with PCA stroke. The territory of the PCA most commonly responsible for simulation was the SPCA (24 patients), with the PPCA and the P+SPCA each being responsible in 6 patients, which is in disagreement with previous findings that the most common mimitors of MCA strokes are PPCA lesions. This discrepancy can be explained by the definition of clinical mimicry used in our study, in which thalamic aphasia and visuospatial neglect were not considered uncommon findings in association with PPCA lesions. In fact, after thalamic injuries, these clinical features may occur in 37.5% to 62% of patients. Furthermore, cognitive abnormalities are rare in SPCA territory infarctions. Based on the differences in cognitive symptoms between the PPCA and SPCA territories, we believe that it would not be correct to combine these territories for analyzing MCA stroke simulation.

In conclusion, PCA stroke simulating MCA stroke is more common than previously thought, accounting for 17.8% of all PCA strokes, and the SPCA is the principal simulator. Different pathophysiologic mechanisms may be implicated in the PCA and the MCA. Consequently, distinguishing between MCA and PCA infarctions is important, with potential implications for treatment, outcome, and future investigation. Our data highlight the heterogeneity and complexity of stroke syndromes.

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