Posterior Cerebral Artery Territory Infarcts in the New England Medical Center Posterior Circulation Registry

Yasumasa Yamamoto, MD; Alexandros L. Georgiadis, MD; Hui-Meng Chang, MD; Louis R. Caplan, MD

Background: Infarcts in the territory of the posterior cerebral arteries (PCAs) are common. Although associated clinical symptoms and signs are known, the mechanisms of stroke and the anatomical distribution of PCA territory lesions caused by the various stroke mechanisms are less well defined. Published reports have selected only special subgroups of patients.

Patients and Methods: We studied stroke mechanisms, infarct distribution, and clinical findings among 79 patients in the New England Medical Center Posterior Circulation Registry in whom brain imaging scans showed infarcts that involved 1 or more cortical territories of the PCA.

Results: Forty-eight patients (61%) had infarcts limited to the PCA territory (pure PCA), while 31 (39%) also had infarcts in other territories (PCA+). Infarcts were in the cortical territory of the PCA in 47 patients (59%) and were cortical and deep in 32 (41%). Infarcts that were cortical and deep were more common in PCA+ lesions. Stroke mechanisms were embolism of cardiac origin (32 [41%]), proximal arterial disease (25 [32%]), cryptogenic embolism (8 [10%]), intrinsic PCA disease (7 [9%]), vasoconstriction (4 [5%]), and coagulopathy (3 [4%]). Patients with cardiogenic embolism and intrinsic PCA disease often had pure PCA territory infarcts, while patients with proximal arterial disease more often had PCA+ infarcts. Visual abnormalities were present in 66 patients (84%). Motor weakness, cognitive and behavioral abnormalities, and ataxia were found in 20 patients (25%); only 12 (15%) had sensory signs.

Conclusions: The great majority of pure PCA and PCA+ territory infarcts are caused by cardiac or intra-arterial embolism. Intrinsic PCA disease, vasoconstriction, and coagulopathy are less common causes of infarction.

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Infarcts in the territory of the posterior cerebral arteries (PCAs) are common and their clinical symptoms and signs are well known.1 However, the mechanisms of stroke and the anatomical distribution of PCA territory lesions caused by the various stroke mechanisms have been less well defined. Some published reports have selected only special groups, such as calcarine infarcts on computed tomographic (CT) scan,2 PCA stenosis,3 fatal infarcts studied at necropsy,4 and thalamic infaracts.5,6

Introduction of magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), and improved ultrasound techniques, including echocardiography, now makes it possible to better define the brain and vascular lesions in patients with PCA territory infarcts. We report the stroke mechanisms, location, and clinical features of a large series of consecutive patients with PCA territory infarcts.

RESULTS

PATIENT DEMOGRAPHICS AND RISK FACTORS

Among the 79 patients studied, 46 were men and 33 were women (mean age, 61.5 years; range, 25-90 years). Stroke risk factors included hypertension (n = 48), diabetes mellitus (n = 19), coronary artery disease (n = 34), hypercholesterolemia (n = 17), and cigarette smoking (n = 23).

DISTRIBUTION OF INFARCTS

Pure PCA vs PCA+ Infarcts

There were 48 patients (61%) with pure PCA and 31 (39%) with PCA+ infarcts. The great majority of patients with a cardioembolic source (26 [81%]), intrinsic PCA disease (6 [86%]), and vasoconstriction (3 [75%]) had pure PCA infarcts. In con-
PATIENTS AND METHODS

PATIENT SELECTION

We reviewed the New England Medical Center Posterior Circulation Stroke Registry during the 10-year period from January 1986 through December 1995 for all patients with PCA territory infarcts. This registry is a prospective collection of all patients with posterior circulation strokes seen at one medical center. For our review, we included all patients in whom brain imaging scans showed infarcts that involved 1 or more cortical territories of the PCA (ie, occipital, parieto-occipital, and medial-inferior temporal lobes). Isolated infarcts that involved only the thalamus or midbrain were excluded, as these might be caused by penetrating artery disease. Among the 415 patients in the registry, 110 had symptoms and signs indicating PCA territory ischemia. Thirty-one patients did not meet our criteria; 4 because they had only transient ischemic attacks (TIAs), 21 because either the clinical information was inadequate or neuroimages had been destroyed, 5 because they did not fulfill the neuroimaging criteria, and 1 because the patient had a subarachnoid hemorrhage complicated by an infarct. Seventy-nine patients remained for analysis.

INVESTIGATIONS

Of these 79 patients, all had either CT (n = 62), MRI (n = 57), or both. The extracranial and intracranial vessels were evaluated with extracranial neck ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), transcranial Doppler ultrasonography (n = 51), and cerebral angiography (n = 28). Thirty-seven patients had transthoracic echocardiography. In selected patients, further evaluations with transthoracic echocardiography (n = 22), cardiac rhythm monitoring (n = 14), cardiac nuclear imaging, and special studies for coagulation abnormalities were performed. Risk factors, transient ischemic attacks, and clinical features were recorded.

DISTRIBUTION OF INFARCTS

Patients were divided into 2 groups according to the distribution of infaracts on neuroimaging scans. The first group included patients with infarcts limited to PCA territory (pure PCA). The second group included patients with infarcts involving the PCA and other posterior circulation territories (PCA+). The PCA territory infarcts were characterized as cortical only or cortical and deep (thalamus or midbrain or both). The cortical distribution was defined according to arterial territories (calcarine, parieto-occipital, or temporal artery, the latter consisting of the anterior and posterior temporal arteries). The size of thalamic infarcts was noted. In the PCA+ group, we noted the location of posterior circulation brain lesions outside the PCA territory in the brainstem and cerebellum. Infarcts in the anterior circulation and watershed regions were also noted.

CAUSES OF STROKE

The patients were divided by cause into the following etiologic groups. (1) The intrinsic PCA disease group included patients with PCA-occlusive disease involving the main trunk or cortical branches that correlated with infarct topography in the absence of any significant proximal arterial or cardiac disease. (2) The proximal arterial disease group included patients with 1 or more significant lesions in the aortic arch, subclavian artery, vertebral artery, or basilar artery; previously described criteria were used. (3) The cardiac source of embolism group included patients with defined cardiac sources of emboli and no intrinsic severe posterior circulation occlusive vascular lesions. The accepted cardiac sources of stroke for this group included atrial fibrillation, severely reduced left ventricular function (or regional wall akinesia), intracardiac clots, right-to-left shunts, wall aneurysms, and stroke related to cardiac procedures. (4) The cryptogenic embolism group included patients who had history and findings consistent with an embolic infarct. In this group, either no source was identified, or 2 or more sources (ie, cardiac, aortic, or vascular) were found, and the actual source was indeterminate. (5) The vasoconstriction group included patients with migraine and a consistent clinical history who were identified as having a stroke in the absence of the above causes. (6) The coagulopathy group included patients with diseases predisposing them to hypercoagulable states or blood test results showing hypercoagulability and in whom the other causes were not present.

In 47 patients (59%), infarcts were limited to the cortical territory, while 32 patients (41%) had cortical and deep infarcts. Among patients in the pure PCA group, cortical infarcts only were present in 34 (43%), while 14 (11%) had cortical and deep infarcts. In patients with PCA+ lesions, cortical infarcts were present in 13 (16%) and cortical and deep infarcts in 18 (23%).

Deep territory infarcts were those located in the thalamic and midbrain regions. Thalamic involvement occurred in 30 patients (38%), 15 (12%) with pure PCA infarcts and 15 (12%) with PCA+ infarcts. These infarcts were limited predominantly to the lateral thalamus in the territory of the thalamogeniculate and posterior choroidal artery branches of the PCAs. Of the patients with thalamic involvement, 26 had an embolic source (cardiac, n = 12; proximal arterial disease, n = 10; cryptogenic embolic, n = 4); 2 had vasoconstriction; 1 had intrinsic PCA disease; and 1 had coagulopathy. Midbrain involvement (7 [9%]) was found only in PCA+ infarcts. Embolism was the cause of stroke in 6 of the 7 patients with midbrain infarcts (cardioembolic, n = 3; proximal arterial disease, n = 2; cryptogenic embolism, n = 1; coagulopathy, n = 1). Bilateral midbrain infarcts were present in 3 patients.

Distribution of Cortical and Cortical and Deep Infarcts

In 47 patients (59%), infarcts were limited to the cortical territory, while 32 patients (41%) had cortical and deep infarcts. Among patients in the pure PCA group, cortical infarcts only were present in 34 (43%), while 14 (11%) had cortical and deep infarcts. In patients with PCA+ lesions, cortical infarcts were present in 13 (16%) and cortical and deep infarcts in 18 (23%).

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The calcarine artery territory, the most common cortical PCA territory involved in infarcts, was affected in 73 patients (92%), including 43 (59%) with pure PCA infarcts and 30 (41%) with PCA+ infarcts. Nine patients had bilateral calcarine artery territory infarcts (cardioembolism, n = 4; proximal arterial disease, n = 2; cryptogenic embolism, n = 2; coagulopathy, n = 1). The temporal artery territory, the second most common territory involved in infarcts, was affected in 31 patients (39%), including 19 (61%) with pure PCA infarcts and 12 (39%) with PCA+ infarcts. Temporal artery without calcarine artery involvement was present in only 5 patients (6%).

Distribution of Infarcts Outside of PCA Territory

The most common territory involved in infarcts outside the PCA territory was the cerebellum. Fifteen patients had posterior-inferior cerebellar artery (PICA) territory infarcts, 14 had superior cerebellar artery (SCA) territory infarcts, 5 had anterior-inferior cerebellar artery (AICA) territory infarcts, and 2 had small infarcts difficult to localize within the cerebellum. Within the posterior circulation, 2 patients had medullary infarcts; 8 infarcts were located in the pons (Table 2).

Seventeen patients (22%) also had anterior circulation territory infarcts. Thirteen of the anterior circulation infarcts involved partial or full cortical territories. Eight of these were in patients with cardioembolism. The remaining 4 infarcts involved the territory of small penetrating arteries (ie, internal capsule, putamen, head of caudate nucleus, and corona radiata). Two patients (3%) had posterior watershed infarcts between the middle cerebral artery and PCA, and both had cardioembolism. The patients with large anterior circulation infarcts had a history of strokes compatible with the lesions found. In some patients with smaller old anterior circulation infarcts, there was no history of an old stroke.

MECHANISMS AND ETIOLOGY OF VASCULAR AND CARDIAC LESIONS

Thirty-two patients (41%) had cardiac sources of embolism, 25 (32%) had proximal arterial disease, 8 (10%) had cryptogenic embolism, 7 (9%) had intrinsic PCA disease, 4 (5%) had vasoconstriction, and 3 (4%) had coagulopathy.

Cardiac Source of Embolism

Most of the 32 patients with cardiac sources of embolism had more than 1 cardiac abnormality. Those with cardiac lesions included 14 with myocardial wall abnormalities in the hypokinetic or akinetic regions (2 patients had dilated cardiomyopathy), 12 with atrial fibrillation, 5 with patent foramen ovale, 2 with bacterial

Table 1. Distribution of Infarct Type by Stroke Mechanism*

<table>
<thead>
<tr>
<th>Infarct Type</th>
<th>No. (%) of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intrinsic PCA Disease (n = 7)</td>
</tr>
<tr>
<td>Pure PCA</td>
<td>6 (86)</td>
</tr>
<tr>
<td>Cortical</td>
<td>5 (71)</td>
</tr>
<tr>
<td>Cortical and deep</td>
<td>1 (14)</td>
</tr>
<tr>
<td>PCA+</td>
<td>1 (14)</td>
</tr>
<tr>
<td>Cortical</td>
<td>1 (14)</td>
</tr>
<tr>
<td>Cortical and deep</td>
<td>0</td>
</tr>
</tbody>
</table>

*PCA indicates posterior cerebral artery; pure PCA, infarcts limited to the PCA territory; and PCA+, infarcts involving the PCA and other posterior circulation territories.

Table 2. Distribution of Brainstem and Cerebellum Territories in PCA+ Infarcts by Stroke Mechanism†

<table>
<thead>
<tr>
<th>Site</th>
<th>No. of Infarcts†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intrinsic PCA Disease (n = 1)</td>
</tr>
<tr>
<td>Medulla (n = 2)</td>
<td>0 1 1 0 0</td>
</tr>
<tr>
<td>Posterior-inferior cerebellar artery (n = 15)</td>
<td>0 10 3 0 2</td>
</tr>
<tr>
<td>Pons (n = 9)</td>
<td>1 4 2 0 1</td>
</tr>
<tr>
<td>Anterior-inferior cerebellar artery (n = 5)</td>
<td>1 4 0 0 0</td>
</tr>
<tr>
<td>Superior cerebellar artery (n = 14)</td>
<td>0 7 3 3 1</td>
</tr>
<tr>
<td>Nonlocalizable (n = 2)</td>
<td>0 1 0 1 0</td>
</tr>
<tr>
<td>Total Infarcts</td>
<td>2 27 9 4 4</td>
</tr>
</tbody>
</table>

†Number in parentheses are numbers of patients.

†PCA+ indicates infarcts involving the posterior cerebral artery (PCA) territory and other posterior circulation territories.
endocarditis with mitral valve vegetations, 1 with atrial septal aneurysm, 2 with intracardiac thrombus, and 8 with strokes after recent cardiac procedures (aortic valve replacements, n = 2; coronary bypasses, n = 3; coronary angiographies, n = 3). Stroke was detected within the first 3 days after surgical procedures in all patients (on average, 1.25 days after the procedure). Pure PCA infarcts were most common (26 [81%]). Six patients in this group (19%) had PCA+ infarcts.

### Proximal Arterial Disease

Of the 25 patients with proximal arterial disease, vascular lesions involved the aorta in 2, extracranial vertebral arteries (ECVAs) in 13, intracranial vertebral arteries (ICVAs) in 5, basilar artery (BA) in 4, and internal carotid artery with fetal posterior communicating artery in 1. The aortic lesions consisted of protruding atheromatous plaques. Since transesophageal echocardiography was not uniformly performed (n = 22 [28%]), other patients with cryptogenic or cardiogenic embolism may also have had undetected aortic lesions. Among patients with ECVAs lesions, 4 had bilateral disease and 9 unilateral, for a total of 17 lesions. Among these, 9 arteries were occluded, 7 were stenotic, and 1 had extrinsic compression of the ECVAs by a spur. Eleven of these lesions involved the ECVAs origins; 2 were bilateral. Two patients had bilateral ECVAs disease caused by dissections; the remaining patients had atherosclerotic disease. One patient with ECVAs disease also had unilateral ICVA disease, and another had unilateral ICVA and BA disease. Among patients with ICVA disease, there were 7 vascular lesions (2 had bilateral disease), 4 with stenosis, and 3 with occlusions. Two patients also had BA disease. Among patients with BA disease, there were 3 with stenosis and 1 with occlusion. The infarcts in these patients were distal to the occlusion and probably represented intra-arterial embolism from the BA occlusive lesion. The remaining patient with proximal artery disease had severe carotid artery stenosis with fetal posterior communicating artery, while the rest of the posterior circulation vessels were normal. This patient had a parieto-occipital infarct, presumably caused by the carotid artery lesion. Most infarcts were PCA+ (Table 3).

### Cryptogenic Embolism

This group consisted of 2 subgroups. In the first subgroup of 5 patients, no obvious source of embolism was identified. The results of cardiac and vascular studies were normal. Four patients had pure PCA infarcts and 1 had PCA+ infarct. In the second subgroup (n = 3), both a cardiac and another potential source were identified. Determination of the primary cause of infarction was difficult. One patient had atrial fibrillation and polycythemia rubra vera with a platelet count of 750 × 10^9/L. Another patient had both ischemic cardiomyopathy and a thoracic aortic aneurysm. The remaining patient had a stroke on the first day after coronary artery bypass surgery and also had aortic atheromas. These 3 patients all had PCA+ infarcts.

### Intrinsic PCA Disease

Of the 7 patients with intrinsic PCA disease, 6 had proximal PCA stenosis and 1 had proximal PCA occlusion shown on vascular imaging. Two patients also had concomitant mild disease in the contralateral PCAs, and 1 had distal BA stenosis as well. Of these patients, 6 had pure PCA infarcts and 1 had PCA+ infarct.

### Vasoconstriction

These 4 patients all had a history of migraine. One patient also had hypercalcemia, with a calcium level of 3.38 mmol/L (13.5 mg/dL), caused by a parathyroid adenoma. Results of cardiac evaluations were normal. In the patient with hypercalcemia, the initial angiogram showed BA occlusion that had recanalized on later testing. There were 3 pure PCA infarcts and 1 PCA+ infarct.

### Coagulopathy

There were 3 patients in this group. One patient had disseminated intravascular coagulopathy after complicated coronary artery bypass surgery. Another had adenocarcinoma of unknown origin with multiple arterial and venous occlusions. These 2 patients had PCA+ infarcts. The remaining patient had antiphospholipid syndrome and a pure PCA infarct.

### CLINICAL FEATURES BY STROKE MECHANISM

Nineteen patients (24%) had preceding TIAs or long, fluctuating ischemic prodromes. Four of these 19 patients had migraine with neurologic deficits during their attacks, of whom 3 had similar attacks just preceding their stroke. Most of the patients with prodromes or TIAs had arterial disease rather than embolism of cardiac origin.

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### Table 3. Distribution of PCA Territory Infarcts in Patients With Proximal Arterial Disease by Site

<table>
<thead>
<tr>
<th>Infarct Type</th>
<th>Aortic Atheromas (n = 2)</th>
<th>Extracranial Vertebral Artery (n = 13)</th>
<th>Intracranial Vertebral Artery (n = 5)</th>
<th>Basilar Artery (n = 4)</th>
<th>Internal Carotid Artery and Posterior Cerebral Artery (n = 1)</th>
<th>Total (n = 25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pure PCA</td>
<td>1</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>PCA+</td>
<td>1</td>
<td>9</td>
<td>4</td>
<td>3</td>
<td>0</td>
<td>17</td>
</tr>
</tbody>
</table>

*PCA indicates posterior cerebral artery; pure PCA, infarcts limited to the PCA territory; and PCA+, infarcts involving the PCA and other posterior circulation territories.*
including intrinsic PCA disease (n = 6), proximal arterial disease (n = 9), and vasoconstriction (n = 3). Visual TIA s were most common (n = 13), followed by sensory (n = 7) and motor (n = 5) symptoms. The most common clinical sign was visual field loss. It was found in 66 patients (84%), was absent in 8 patients (10%), and in 5 patients (6%) could not be ascertained for various reasons (eg, coma, somnolence, or bilateral cataracts). Motor weakness was present in 23 patients (29%) (3 with bilateral limb weakness) but was usually of minor severity. Sensory loss was detected in 12 patients (15%). Twenty-one patients (27%) had ataxia, usually slight and affecting one limb. Twenty patients (25%) had some cognitive or behavioral abnormality. Memory loss, agitation, and confusion were more commonly found than alexia and anomia. There were only 2 deaths (3%).

**Intrinsic PCA Disease**

There were 4 men and 3 women with intrinsic PCA disease (mean age, 57 years; range, 49-67 years). All had hypertension, 4 had diabetes, 2 had coronary artery disease, and 2 had hypercholesterolemia. Three patients had prior strokes and 1 patient had a previous central retinal artery occlusion.

Transient ischemic attacks and/or long prodromes with visual involvement were present in 6 patients. In 4 patients, discreet TIA s were present from 2 weeks to 12 months before strokes. These symptoms were visual in 2 patients, sensory in 2, and motor in 1. In 4 patients, stroke was preceded by a fluctuating prodrome lasting 2 days to 2½ weeks. Only 1 patient had sudden-onset signs with no preceding TIA s. Visual symptoms were present in all patients with TIA s and included hemifield blurring, visual dimming, seeing lines or stars, visual hallucinations, and macropsia. All patients had objective visual field loss; calcarine artery territory infarcts were present in all. One patient had hemisensory loss with bilateral infarcts. Motor signs were slight (2 patients had hemiparesis). One patient was agitated and confused. There were no deaths in this group.

**Proximal Arterial Disease**

There were 15 men and 10 women with proximal arterial disease (mean age, 61 years; range, 28-84 years). Seventeen of these patients had hypertension, 6 had diabetes, 8 had coronary artery disease, and 7 had hypercholesterolemia. Six patients had previous strokes. A history of TIA s was elicited in 9 patients, ranging from 1 day to 6 months earlier. Visual symptoms were present in 6 patients, sensory symptoms in 3, and motor symptoms in 1. Three patients had disease of ECA origin (bilateral, n = 1; concomitant ICVA disease, n = 2); 3 had ICVA disease (bilateral, n = 1; BA disease, n = 1); and 3 had BA disease. The onset was sudden in 22 patients. In the remaining 3 patients, stroke was noted on awakening. In 10 patients, there was further deterioration over 1 to 7 days. Eighteen patients had visual symptoms, ranging from blurring to double vision. Objective visual field loss was detected in 21 patients. One patient had a severely depressed level of consciousness and could not cooperate with visual field assessment; 3 had no visual loss. Calcarine artery territory infarcts were present in 23 patients and temporal artery territory infarcts in 9 patients (isolated, n = 2). In the 21 patients with visual field loss, 19 had calcarine artery territory infarcts, and 2 had only temporal artery territory infarcts. Sensory involvement was present in 5, and all had thalamic infarcts. Another 5 patients with thalamic infarcts had no sensory symptoms or signs. Motor weakness was usually slight, present unilaterally in 5 patients and bilaterally in 2. Four patients were confused and agitated; 2 had poor memory. There were no deaths in this group.

**Cardiac Source of Embolism**

There were 22 men and 10 women with cardiac source of embolism (mean age, 64 years; range, 30-90 years). Seventeen had hypertension, 8 had diabetes, 21 had coronary artery disease, and 5 had hypercholesterolemia. Three patients had a history of previous strokes. Only 1 patient in this group had a TIA (1 day prior to stroke). That patient noted left hand numbness and weakness for 5 minutes; the next day, on awakening, he noticed a visual field defect and paresthesia of his left hand. Onset was sudden in 19 patients; 2 strokes occurred after surgery and 9 were discovered on awakening. The other 2 patients had unclear onset; 1 was ill in intensive care for a long time and the other was confused and schizophrenic. Visual field loss was documented in 24 patients (all had calcarine artery territory infarcts). Five patients had no visual field defects (2 had temporal artery territory infarcts only and 2 had calcarine artery territory infarcts), and visual field loss could not be ascertained in 3 patients because of poor visual acuity or poor neurologic state (all had calcarine artery territory infarcts). Thirty patients had calcarine artery territory infarcts, 18 had temporal artery infarcts, 4 had parieto-occipital artery infarcts, 13 had thalamic infarcts, and 3 had midbrain infarcts. Four patients had hemisensory loss, 3 of whom had thalamic infarcts. Thirteen patients had motor weakness, which was most often slight, except for 1 patient who had hemiplegia and eventually died. Cognitive and behavioral abnormalities were common in this group, probably because of the larger size of the infarcts: 9 patients had agitation with confusion, 2 had poor memory, 3 had alexia without agraphia, and 1 had anomia. There was 1 death, a 90-year-old woman with atrial fibrillation and a pure PCA infarct.

**Cryptogenic Embolism**

There were 3 men and 5 women with cryptogenic embolism (mean age, 63 years; range, 30-87 years). Four of these patients had hypertension, 2 had heart disease, 3 were smokers, and 1 had 4 previous miscarriages. There were no preceding TIA s. Onset was sudden in 4 patients, occurred on awakening in 2, and 2 were unable to provide details (1 was unconscious and the other had dementia). Visual field loss was found in 7 patients (6 with calcarine artery territory infarcts and 1 with temporal artery territory infarcts). The eighth patient was comatose and visual field loss could not be ascertained.
Hemisensory loss was noted in 1 patient who had a thalamic infarct. One patient had slight hemiparesis. One patient had poor memory. Altogether there were 7 calcarine artery infarcts, 1 temporal artery infarct, 1 parieto-occipital artery territory infarct, 3 thalamic infarcts, and 1 midbrain infarct. There was 1 death. The patient who died presented comatose, with signs of raised intracranial pressure and bilateral calcarine artery, midbrain, and superior cerebellar artery territory infarcts as part of a "top of the basilar" syndrome.

Vasoconstriction

There were 3 men and 1 woman with vasoconstriction (mean age, 53.5 years; range, 25-78 years). Four had migraine (3 known, 1 diagnosed on admission) and 1 patient had hypertension. One patient also had hypercalcemia (3.38 mmol/L [13.5 mg/dL]) caused by underlying hyperparathyroidism. Visual symptoms were present during migraine attacks in all 4 patients, consisting of colored lines in 1 and visual field loss in 3. In all 4 patients, symptoms were noticed on awakening, there was no neurologic progression, and there was good clinical recovery. Visual field loss was present in all. One patient also had slight hemiparesis. None had sensory loss, although 2 patients had sensory symptoms, 1 of whom had a thalamic infarct. None of these patients had a cognitive or behavioral abnormality. There were 3 calcarine artery, 2 temporal artery, and 2 thalamic infarcts in this group.

Coagulopathy

Underlying diseases determined the clinical features of the 3 patients with coagulopathy. One 71-year-old woman with hypertension and diabetes developed disseminated intravascular coagulation after coronary artery bypass surgery that was complicated by cardiac tamponade, bleeding, and hypotension. On the second postoperative day, she was found to have neurologic deficits. A 68-year-old man had recurrent arterial and venous thrombosis. He first had an infarct in 1 occipital lobe, followed later by infarction in the contralateral occipital lobe, infarction in the middle cerebral artery territory, bilateral deep venous thrombosis, and a myocardial infarct, all within 1 year. He was eventually diagnosed as having metastatic adenocarcinoma of unknown origin. A 43-year-old woman presented with sudden hemianopia and an occipital lobe infarct. She later had recurrent hemiparesis. Results of cardiac testing and vascular imaging were normal. She had the antiphospholipid syndrome. All 3 patients had hemianopia, 2 had hemiparesis, and none had sensory loss. No important cognitive or behavioral abnormalities were found. In this group, there were 3 calcarine artery territory infarcts, 1 thalamic infarct, and 1 midbrain infarct.

This large, consecutive series of patients with PCA territory infarcts provides information about infarct distribution, stroke mechanisms, and clinical features. The distribution of infarction within and outside PCA territory yields clues as to the causative mechanism of stroke.

INFARCT DISTRIBUTION

Previous studies of patients with PCA territory infarcts did not separate pure PCA territory and PCA+ infarcts.7,10,11 In our series, we found that pure PCA territory infarcts (48 [61%]) were more common than PCA+ infarcts (31 [39%]). In pure PCA infarcts, involvement of cortical territory alone was more common than cortical and deep territory involvement (34 [71%] vs 14 [29%]). This discrepancy was not found in PCA+ infarcts, for which cortical and deep territory involvement was slightly more common (18 [58%] vs 13 [42%]). For the whole series, cortical involvement was slightly more common than cortical and deep involvement (47 [59%] vs 32 [41%]). Castillo et al.12 studied 13 patients with deep and cortical involvement. They found such involvement uncommon, accounting for 5% of their pure PCA infarcts. In their series, cardioembolism was the most common mechanism (7 of 13 cases). Others have reported cortical and deep involvement in 11 (29%) of 38,10 29 (23%) of 127,11 and 2 (4%) of 56 PCA infarcts in their series. In our study, the cortical territory most often involved in infarcts was the calcarine artery territory (n = 73 [92%]). The anatomical divisions between the anterior and posterior temporal arteries are not always clear; therefore, we did not attempt to differentiate the 2. In our study, the temporal artery territory was involved in 31 patients (39%) and the parieto-occipital artery territory in only 6 (8%). In 1 CT study of 54 patients, 45 (83%) had infarcts in the temporal artery territory, 43 (80%) in the calcarine artery territory, and 16 (30%) in the parieto-occipital artery territory.7 In a study of 38 patients in which infarcts were classified according to lobes rather than arterial territories, 29 (76%) had infarcts in the occipital lobe, 28 (74%) in the posterior temporal lobe, and 8 (21%) in the posterior parietal lobe.10 These studies were CT-based and most patients also had angiography.

The thalamus was involved in 30 (38%) of our patients with PCA territory infarcts. Thalamic involvement was equally distributed between pure PCA and PCA+ infarcts. In 3 large, CT-based series, the thalamus was involved in 42 (19%) of 221 patients.7,10,11 Magnetic resonance imaging is a more sensitive imaging tool, and this probably accounts for the higher frequency of thalamic infarcts detected in our study. Embolism was the stroke mechanism in 26 patients (87%) with thalamic infarcts. In 1 study, the thalamus was involved almost 4 times as often as the midbrain.12 Bilateral thalamic involvement was uncommon in our series; only 4 patients (5%) had bilateral thalamic infarcts. Seven of our patients (9%) had midbrain infarcts, and these occurred only in PCA+ infarcts. Bilateral midbrain lesions were present in half of these patients. Almost all patients with midbrain infarcts had an embolic source of stroke—cardiac, proximal artery, or cryptogenic. Involvement of the midbrain probably requires a very proximal PCA occlusion, more common with embolism than with atherosclerotic disease.1,13-15
had cancer-related coagulopathy. Midbrain infarcts are not easily seen on CT scans.

The literature on midbrain infarcts in patients with confirmed PCA disease consists mainly of reports on small series or single case reports.5,16-18 Bogousslavsky et al19 studied a consecutive series of 281 patients with posterior circulation infarcts, 22 of whom (8%) had pure midbrain infarcts. The causes of stroke for these 22 patients were cardioembolic in 5 (23%), large vessel disease in 6 (27%), small vessel disease in 5 (23%), undetermined in 5 (23%), and 1 patient had polycythemia vera as the cause. In our study, we excluded pure midbrain infarcts in an attempt to eliminate patients who had penetrating artery disease rather than PCA disease. Of territories outside the supply of the PCAs, the cerebellum was most often involved. The PICA and SCA territories of the cerebellum were equally affected. Anterior-inferior cerebellar artery territory cerebellar infarcts occurred at one third the frequency and pontine infarcts at one half the frequency of PICA and SCA territory cerebellar infarcts, while medullary infarcts were rare. In patients with proximal artery disease, PCA+ infarcts were more common than pure PCA infarcts. Among the 27 patients with PCA+ infarcts who had proximal artery disease, the PICA cerebellum was most often involved (n = 10), followed by the SCA (n = 7), the AICA (n = 4), and the pons (n = 4) (Table 2). Anterior circulation and watershed infarcts were more frequent in patients with cardioembolic strokes (10 of 17).

**STROKE MECHANISMS**

The vast majority of PCA territory infarcts are embolic; in our series, 65 (82%) were embolic. Previous studies have shown that embolism is the most common mechanism of PCA territory infarction (55%–77%).2,11,14,20-23 In our series, cardiac disease was the most common source of embolism (32 [41%]). Patients with a cardiac source of embolism usually had pure PCA infarcts (25 [81%] of 32 patients). In other studies, a cardiac source of embolism ranged from 17% to 47%.2,11,14,20-23 The next most common source of embolism in our series was proximal arterial disease (25 [32%]). Proximal arterial disease was often found in the ECVAs, especially at their origins from the subclavian arteries (11 of 17 lesions). This emphasizes the point made by previous authors that ECVA disease is not as benign as was formerly believed.13,24 Bilateral ECVA disease was common in our series (8 of 17 lesions were bilateral). One patient had presumed ECVA compression during surgery caused by neck posturing, as described previously.25,26 Two patients with ECVA lesions had dissections; the remainder had atherosclerotic disease. In our experience, emboli that arise from the ECVAs often produce infarction in the PICA cerebellum as well as in branches of the distal BA, including the SCA territory of the cerebellum and the PCAs.4 The next most common arterial sources of embolism in our series were the ICVAs, followed by the BA. In a study of 75 patients with ICVA disease, 28 (37%) had distal posterior circulation territory infarcts that included the PCA territory.27 One patient (1%) had an infarct from severe carotid artery stenosis in the presence of a fetal posterior communicating artery; this patient has been described elsewhere in detail.9

In our series, 17 patients with proximal arterial disease (68%) had PCA+ infarcts. Three patients (4%) had a cardiac source as well as other existing conditions that could have caused the infarcts. Five patients (6%) had no obvious source of embolism even after extensive evaluations. In previous studies that had high frequencies of unknown source of embolism, the cardioembolic rates were lower (Table 4). These patients probably had plaque disease in the aorta, proximal arterial lesions, or cardiac sources not detectable by technology then available. Migraine-related strokes are well described. Stroke may occur with the first migraine attack and usually affects the posterior circulation, especially the occipital lobes.1,2,13,28 In addition, 1 patient in our study also had hypercalcemia, a condition known to cause vasoconstriction, mental state abnormalities, seizures, and strokes.29-31 Reversible vasoconstriction has been shown in some patients with hypercalcemia, and vasospasm was reversed after the serum calcium level normalized. Our patient with hypercalcemia had BA occlusion that had recanalized on later angiography. Table 4 compares the mechanisms of stroke in our series with those in other reports.

**CLINICAL FEATURES**

One fourth of our patients had prior TIAs. Visual symptoms were most common (54 [68%]), followed by somato-
In 1992, Caplan et al described 10 patients with occlusive PCA disease and found that preceding TIAs. In that series, no patient had intrinsic PCA disease. In a separate report, Pessin et al described 6 patients with PCA territory infarcts, of whom 3 (9%) had preceding TIAs. Servan et al noted the predominance of visual symptoms and sensory symptoms (29 [37%]), consistent with previous studies. Transient ischemic attacks were common in patients with intrinsic PCA disease (6 [86%]), proximal arterial disease (9 [36%]), and vasoconstriction (3 [75%]). In 1987, Pessin et al described 35 patients with PCA territory infarcts, of whom 3 (9%) had preceding TIAs. In that series, no patient had intrinsic PCA disease. In a separate report, Pessin et al described 6 patients with intrinsic PCA disease and found that preceding multiple TIAs were common (5 [83%]) and were characterized predominantly by visual and sensory symptoms. In 1992, Caplan et al described 10 patients with occlusive disease of ECVA origin who had embolic strokes, among whom only 1 patient had a TIA. Thirty patients (40%) in the New England Medical Center Posterior Circulation Registry with ICVA disease had TIAs. Similarly, in our patients with proximal arterial disease, TIAs were more common in patients who had ICVA and BA disease than in those with PCA disease.

The onset and early course of stroke were quite variable. The onset was sudden in 48 patients; the deficit was discovered on awakening in 18 patients. In 6 patients, the clinical course was gradual and progressive. Two patients had their neurologic signs detected after they awakened from anesthesia, and the onset and early clinical course were unclear in 5 patients. Sudden onset usually occurred in patients with proximal artery disease (70 [88%]), cardioembolism (52 [66%]), or cryptogenic embolism (40 [50%]), and was consistent with the embolic mechanism of stroke. In 10 patients (8%) with proximal artery disease, there was further deterioration after onset. Death was rare; there were only 2 fatalities (3%). Visual field abnormalities were the most common clinical sign. Previous studies also noted the predominance of visual symptoms and signs. The visual field defect frequently was a hemianopia or quadrantanopia, or, less frequently, scotomas. Despite the fact that 30 patients (38%) had thalamic infarcts, sensory loss was detected in fewer than half (12 [15%]). Many of the thalamic infarcts did not involve the somatosensory nuclei in the ventrolateral portion of the thalamus. Twenty-three patients (29%) had motor weakness. The weakness was usually slight (3 patients had hemiplegia; 1 each with midbrain, pontine, and thalamic infarcts). Hemiplegia in patients with PCA territory infarcts is usually caused by involvement of the cerebral peduncles. Sometimes motor abnormalities may be caused by severe sensory loss or incoordination. Ataxia of the limbs and/or gait was found in 21 of our patients (27%). Since testing for cognitive and behavioral abnormalities varied, we did not emphasize the presence of neuropsychological abnormalities in our series. One fourth of our patients who could be adequately tested had some behavioral or cognitive abnormality. Agitation with confusion and memory loss were the most common abnormalities. Alexia, agraphia, and anomia were less common. No patient had visual agnosia. The cognitive and behavioral abnormalities that accompany PCA territory infarction have been discussed and analyzed in detail elsewhere.

Table 5 shows the frequency of clinical findings in various series of patients with PCA territory infarcts.

<table>
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<tr>
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<tbody>
<tr>
<td>Visual field defect</td>
<td>64 (84)</td>
<td>26 (76)</td>
<td>118 (93)</td>
<td>35 (100)</td>
<td>66 (84)</td>
</tr>
<tr>
<td>Sensory</td>
<td>24 (32)</td>
<td>NM</td>
<td>37 (29)</td>
<td>7 (20)</td>
<td>12 (15)</td>
</tr>
<tr>
<td>Motor</td>
<td>NM</td>
<td>16 (47)</td>
<td>36 (28)</td>
<td>3 (9)</td>
<td>23 (29)</td>
</tr>
<tr>
<td>Cognitive and behavioral‡</td>
<td>31 (41)</td>
<td>11 (32)</td>
<td>41 (32)</td>
<td>7 (20)</td>
<td>20 (25)</td>
</tr>
<tr>
<td>Ataxia</td>
<td>NM</td>
<td>NM</td>
<td>NM</td>
<td>1 (3)</td>
<td>21 (27)</td>
</tr>
</tbody>
</table>

*NM indicates that these data were not mentioned in the report.
†Only patients with occipital infarcts and hemianopic visual field defects were included.
‡Most often alexia, anomia and other language abnormalities, memory defects, confusion, visual neglect, and agnosias.

CONCLUSIONS

Our series confirms that PCA territory infarcts are usually caused by embolism. Embolism of cardiac origin is the most common cause. Proximal arterial disease, especially that involving the ECVAs at their origins, is also a common source of embolism to the PCAs. Infarcts caused by embolism of cardiac origin and intrinsic PCA disease usually produce pure PCA infarcts, while artery-to-artery embolism tends to produce PCA+ infarcts. The PICA and SCA cerebellum are the most commonly involved structures outside the PCA territory in patients with PCA territory infarcts. Cortical PCA territory involvement is more common than involvement of both the cortical and deep territories. The occipital lobe supplied by the calcarine arteries is most often involved, followed closely by involvement of the posterior temporal lobe. Thalamic involvement occurs in one third and midbrain infarction in one tenth of patients with PCA territory infarcts. These deep territory infarcts are usually caused by embolism. Mortality in patients with PCA territory infarcts is low. Understanding the clinical and neuroimaging features and mechanisms of PCA territory infarcts will facilitate evaluation and treatment of these patients.

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REFERENCES


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