Anatomy of Sensory Findings in Patients With Posterior Cerebral Artery Territory Infarction

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Background: Posterior cerebral arteries (PCAs) supply the ventrolateral thalamic sensory nuclei and white matter sensory tracts to the somatosensory parietal cortex. Patients with PCA territory strokes often have visual, memory, cognitive, and sensory signs. Clinicoanatomic correlation of visual, cognitive, and memory functions are well defined, but, to our knowledge, no systematic study has analyzed the anatomy of sensory abnormalities.

Objective: To assess the frequency and anatomic correlation of sensory symptoms and signs in patients with PCA territory infarction.

Patients and Methods: Sixty patients with hemispheric and hemispheric and deep PCA territory infarcts apparent on computed tomographic and magnetic resonance imaging scans were studied for the presence of sensory findings and location of infarcts.

Results: Sensory symptoms or signs were present in 15 (25%) of 60 patients. Among patients with sensory findings, 11 of 15 had infarcts in the ventrolateral thalamus in the territory of the thalamogeniculate or lateral posterior choroidal arteries. The other 4 patients had no ventrolateral thalamic or white matter infarction but had severe proximal vascular occlusive lesions that could have caused temporary thalamic ischemia. One of these 4 patients had a medial thalamic infarct and transient hemisensory symptoms. Twelve patients had thalamic infarcts and no recorded sensory findings. Seven patients with thalamic infarcts (6 medial and 1 ventrolateral) had no sensory findings, and sensory findings could not be accurately assessed in 4 patients with ventrolateral and 1 patient with medial thalamic infarcts.

Conclusions: All patients with PCA territory infarcts and sensory findings either had thalamic infarcts in thalamogeniculate or lateral posterior choroidal artery territory or had thalamic ischemia. Sensory findings in PCA territory infarction indicate ventrolateral thalamic ischemia.

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HE 3 MAIN categories of symptoms in patients with posterior cerebral artery (PCA) territory infarction are neurobehavioral, visual, and somatosensory. Although the anatomic substrates of the visual and neurobehavioral abnormalities have been extensively analyzed, few studies have addressed the clinicoanatomic correlations of the somatosensory symptoms and signs. Sensory abnormalities in patients with PCA territory ischemia could be due to thalamic ischemia, which would indicate occlusion of the precommunal or proximal postcommunal PCA; to ischemia of white matter (thalamoparietal) tracts caused by occlusion of the more distal PCA or its parieto-occipital branches; or to brainstem ischemia due to vascular disease in the more proximal vertebrobasilar system.

We studied patients with PCA territory infarction to assess the frequency and anatomic correlation of sensory symptoms and signs.

RESULTS

Data on 98 patients were initially retrieved from the New England Medical Center Stroke Data Bank and Posterior Circulation Stroke Registry. Four patients had only TIs and no lesions on computed tomographic or magnetic resonance imaging scans; 12 patients had only deep infarcts, which could have been caused by branch artery disease; 6 had conditions other than ischemia (including hemorrhage); and 12 had watershed infarcts or combined anterior and posterior circulation disease. The 4 patients for whom insufficient clinical detail was available were excluded, and findings in the 60 remaining patients...
PATIENTS AND METHODS

All patients with PCA territory infarcts were retrospectively retrieved from the New England Medical Center Stroke Data Bank and Posterior Circulation Stroke Registry. The dates of stroke ranged from 1986 to 1995. Available neuroimaging studies and clinical information were reevaluated. The patients were included in the study if they fulfilled the following neuroimaging criteria: (1) PCA territory infarcts on computed tomographic and/or magnetic resonance imaging scans, which had to be available for review, and (2) infarction not limited to deep structures, but involving PCA-supplied cortical territory (medial/inferior temporal, occipital/parieto-occipital lobes). Patients with cortical infarcts in the middle cerebral artery–PCA watershed distribution were excluded. Patients also had to fulfill the following clinical criteria: (1) sufficient notes about neurologic status, particularly regarding sensory symptoms and signs; (2) no coexisting symptomatic carotid artery disease or serious medical condition potentially contributing to the acute neurologic symptoms; and (3) no residual sensory deficit from prior strokes.

All reported sensory symptoms and signs were recorded, including those that could have occurred before the stroke during premonitory transient ischemic attacks (TIAs). Because no prospective protocol demanded a complete sensory examination, and testing of all sensory modalities was not performed in detail, we decided to group sensory symptoms and signs together and not attempt to separate tactile, pain and temperature, and proprioceptive abnormalities. Available outpatient charts were reviewed for improvement or persistence of sensory abnormalities.

All imaging studies were independently reviewed by a neuroradiologist (E.S.K.) who was blinded to the clinical information and who determined which, if any, thalamic vascular territories were infarcted, using the templates reported by Pullicino. We divided the patients into 4 groups depending on the presence and location of thalamic infarcts: (1) no thalamic infarct; (2) infarction restricted to the medial thalamus (supplied by the medial posterior choroidal and thalamic-subthalamic or thalamoperforating arteries); (3) infarction that included ventrolateral thalamic territory (supplied by the thalamogeniculate and lateral posterior choroidal arteries); and (4) anterior thalamic infarction (territory of the tuberothalamic [polar] artery).

In each patient, the imaging findings were correlated with the presence or absence of sensory abnormalities.

INCIDENCE AND PATTERN OF THALAMIC INFARCTION

Among the 60 patients enrolled in the study, 24 (40%) showed infarction of the thalamus, whereas 36 (60%) had no thalamic infarct (group 1). Eight patients had infarcts restricted to the medial thalamus (group 2), 7 had infarcts restricted to the lateral thalamus (group 3), and 9 had combined medial and lateral thalamic infarcts (group 3). No patients had anterior thalamic infarction. Figure 1 shows the distribution of thalamic infarcts among the 60 patients.

SENSORY FINDINGS

Forty patients had no sensory findings before or at stroke onset, while 15 had sensory symptoms and/or signs before (3) or at (12) stroke onset. In 5 patients, the presence of sensory abnormalities could not be adequately assessed, even though a detailed neurologic examination was available, because they had reduced consciousness or confusion (Figure 2).

CORRELATION OF SENSORY ABNORMALITIES WITH SITE OF THALAMIC INFARCTION

Group 1

Among the 36 patients with no thalamic infarct, 33 (92%) had no sensory symptoms or signs. Two patients (6%) had sensory symptoms during TIAs before their stroke, and 1 (3%) had a slight hemisensory loss on hospital admission. All 3 patients had occlusive vascular lesions (distal basilar artery or proximal PCA) that would reduce flow to the thalamus (Figure 3). We posit that these patients likely had thalamic ischemia without infarction.

Group 2

Eight patients had infarcts restricted to the medial thalamus, 6 of whom (75%) had no sensory abnormalities; 1
had a feeling of numbness over his left side on admission, which cleared by the time of discharge. This patient had both occlusion of the rostral basilar artery and reduced blood flow to the lateral thalamus. The sensory status of 1 patient could not be adequately assessed. The Table shows data regarding the 4 patients in groups 1 and 2 who had sensory symptoms and signs but not ventrolateral thalamic infarction on neuroimaging scans.

Group 3

Sixteen patients had infarcts that included the lateral thalamus, 11 (69%) had sensory signs or symptoms, 10 had a sensory deficit on admission, and 1 had a TIA with numbness of the left arm and leg 1 day before admission. One patient had no reported sensory symptoms or signs (6%); in 4 patients (25%), sensory findings could not be adequately assessed.

Five of the 16 patients had infarcts restricted to territory of the lateral posterior choroidal artery, 6 had infarcts restricted to territory of the thalamogeniculate arteries, and 5 had infarcts that involved thalamogeniculate and lateral posterior choroidal artery territory. All patients with infarction of the lateral posterior choroidal artery territory had a sensory deficit, as did 4 of 5 patients with combined thalamogeniculate and lateral posterior choroidal artery territory infarcts (1 could not be assessed). The presence of sensory symptoms and signs in 3 of 6 patients with thalamogeniculate artery infarcts could not be assessed. Two (67%) of the remaining 3 patients had sensory abnormalities.

Follow-up information was available for 6 of the 10 patients who had sensory findings on admission. In all 6 patients, the deficit persisted during follow-up of 19 to 93 months.

The role of the thalamus, especially the ventrolateral portions, in sensation was first suggested by Luys in 1865 and confirmed in 1906 by the report of Dejerine and Roussy on the thalamic syndrome. Later reports concerning patients with isolated small thalamic infarcts, presumed to be caused by penetrating artery disease of thalamogeniculate artery branches, provided detailed information on the pattern of sensory symptoms and signs that result from ventrolateral thalamic lesions. Foix and Masson first related thalamic infarction to occlusive disease of the posterior cerebral artery. They illustrated the typical PCA territory infarct as involving the lateral thalamus and temporal lobe. None of the number of series of patients with PCA territory ischemia have systematically attempted to correlate the presence of sensory findings with thalamic or white matter infarction. Most series included patients with isolated thalamic infarcts without cortical PCA territory ischemia. In this study, we excluded all patients with isolated thalamic infarcts. Twenty percent of our patients had sensory findings at stroke onset, and another 5% had had sensory symptoms during TIAs. In the 2 largest series of PCA territory infarcts, the authors reported an incidence of sensory involvement of 40% and 46.2%, respectively. This high incidence is probably because these studies included patients with isolated thalamic infarcts. Our results agree with the incidence reported by Pessin and colleagues (6 [17%] of 35 patients), who included only patients with superficial (occipital) or superficial and deep infarcts.

Thalamic infarcts were found in 24 (40%) of 60 patients in this series, including 8 with medial thalamic infarcts, 7 with lateral thalamic infarcts, and 9 with combined medial and lateral thalamic infarcts. No anterior...
thalamie lesions were found, presumably because the anterior thalamus is fed by the tuberothalamic arteries, which arise from posterior communicating arteries and are not branches of the PCAs. Sensory symptoms were present in 3 (8%) of 36 patients with no neuroimaging evidence of thalamic infarction, 1 (14%) of 7 with medial infarcts, and 11 (92%) of 12 with either pure lateral or combined medial and lateral thalamic infarcts. Sensory symptoms in 2 of 3 patients with no thalamic infarcts and in 1 patient with infarction, limited to the medial thalamus, were transient. All 4 patients with sensory symptoms but no medial thalamic infarction (Table) had occlusive vascular lesions that limited thalamic perfusion. We posit that these patients likely had thalamic ischemia without infarction.

Brandt et al and Milandre et al reported a 63% and 87% correlation, respectively, between sensory signs and posterolateral thalamic infarction, but the underlying lesions in the remaining patients with sensory signs were not commented on. Both studies included patients with branch territory thalamic infarcts and cortical PCA territory infarcts.

Among the 12 patients with infarcts that involved the lateral thalamus whose sensory status could be assessed, 5 had lateral posterior choroidal artery territory infarcts, 3 had thalamogeniculate artery infarcts, and 4 had combined lateral posterior choroidal and thalamogeniculate artery infarcts. The patients whose infarcts included the thalamogeniculate artery territory often had accompanying minor motor abnormalities. These included limb ataxia or dystonia on the side of the sensory symptoms and a minor degree of gait ataxia. The frequency of sensory findings in these 3 subgroups was 100%, 67%, and 100%, respectively, which clearly indicates the importance of lateral posterior choroidal artery territory lesions in the pathogenesis of sensory deficits. Bogousslavsky et al described 40 patients with computed tomography–proven thalamic lacunar infarction. Only 3 patients had infarcts in territory of the posterior choroidal arteries, and 2 of 3 patients had no sensory changes.

Our findings suggest that the presence of sensory symptoms or signs in patients with PCA occlusive disease indicates lateral thalamic ischemia. The coexistence of hemisensory symptoms with hemianopia suggests a PCA occlusive lesion, proximal to the blood supply of the lateral thalamus. Nearly always, the mechanism of proximal PCA occlusion is embolism.

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REFERENCES