Lateral Thalamic Infarcts

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A patient with occlusion of the proximal posterior cerebral artery (PCA), a lateral thalamic infarct, and hemisensory loss later developed hemianopia and hemiparesis and had extensive PCA territory infarction in the midbrain, the lateral portion of the thalamus, and the occipital lobe noted at necropsy. Two other patients had lateral thalamic infarcts on computed tomography, normal angiographic findings, and presumed thalamogeniculate artery branch occlusion. There are three clinical syndromes associated with lateral thalamic infarction: (1) hemisensory loss, hemiataxia, and involuntary movements; (2) pure sensory stroke; and (3) sensory-motor stroke. Ataxia, adventitious movements, and sensory loss are due to infarction of the lateral, posterolateral, and posteromedial ventral nuclei caused by occlusion of the PCA proximal to the thalamogeniculate artery branches or by occlusion of large thalamogeniculate arteries. Pure sensory and sensorymotor strokes are due to smaller infarcts in the posterolateral-posteromedial ventral complex and adjacent internal capsule caused by occlusion of penetrating artery branches of the thalamogeniculate arteries.

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Infarcts in the basal ganglia and cerebral white matter cause clinical syndromes that differ from cortical infarcts of comparable size.¹⁻³ Computed tomography (CT) and magnetic resonance imaging now accurately define these deep hemispheral infarcts. Within the anterior circulation, angiography and necropsy showed two patterns of vascular occlusion in patients with infarcts in the internal capsule, pallidum, and striatum: (1) occlusion of penetrating lenticulostriate⁴⁶ or anterior choroidal artery⁷ branches and (2) obstruction of the parent supraclinoid internal carotid⁸ or main-stem middle cerebral arteries^{1,8,9} before the origin of the penetrating arteries.

The posterior circulation lesion that is analogous to deep hemispheral ischemia is lateral thalamic infarction. The clinical features were described in the beginning of the 20th century.¹⁰⁻¹⁴ The anatomy of thalamic blood supply has been clarified by injection and microangiography techniques,15-20 and CT can define the distribution of thalamic infarcts,²¹⁻²⁴ but the vascular pathology causing lateral thalamic infarcts has rarely been studied. We now report clinical, CT, angiographic, and necropsy data obtained from patients with lateral thalamic infarcts to clarify the clinical features and vascular causes.

REPORT OF CASES

CASE 1.—A 69-year-old white man awakened with dizziness; shortly thereafter, he required support to walk. He had dietcontrolled diabetes mellitus, hypertension, surgical repair of a traumatic tear of the aorta, and a surgical bypass for occlusive disease of the right leg. Examination showed severe sensory loss of all modalities including pin, temperature, touch, position sense, and vibration in the left side of the face and trunk and the left limbs. The right pupil was smaller (2 mm vs 3 mm on the left) and reacted poorly to light. There was slight weakness in the left side of the face only. The left plantar

response was equivocal; deep tendon reflexes were symmetrical. The initial findings of CT were normal. Two days later, CT showed an area of low attenuation in the right lateral portion of the thalamus. Three days after admission, the patient's eves became conjugately deviated to the right at rest, but moved conjugately to the left to follow visual objects. There was a left homonymous hemianopia. The patient read only the right side of the printed page. The left hemisensory loss was unchanged, and there was no change in weakness. Repeated CT showed the previously described right thalamic infarct, with new areas of low attenuation in the medial part of the temporo-occipital lobes as well as in the lateral geniculate nucleus, compatible with new infarcts (Fig 1). Heparin therapy was begun. Angiography showed occlusion of the perimesencephalic portion of the right posterior cerebral artery (PCA). There was also subcritical stenosis of the distal left vertebral and basilar arteries. There was suboptimal visualization of the left PCA possibly secondary to stenosis of the P-1 segment. The right PCA did not originate from the right internal carotid artery, but collateral vessels from the right middle cerebral artery filled the right PCA in a retrograde direction.

Cardiac studies revealed a recent myocardial infarct and frequent premature ventricular contractions. On the 23rd hospital day, the patient had a massive gastrointestinal hemorrhage. Laparotomy revealed severe erosive gastritis, and a gastrectomy was performed. Postoperatively, the myocardial infarct was extended, and the patient had pulmonary edema, pneumonia, and intermittent hypotension. A month after admission, he developed a complete left hemiplegia. His hemianopia and hemisensory loss were unchanged. He had renal failure and hypotension, and died six weeks after his initial stroke.

Necropsy revealed occlusion of the proximal 2 cm of the right PCA by a graybrown granular thrombus. There was an atherosclerotic plaque at the origin of the artery that had severely narrowed the lumen. There was a $1 \times 2 \times 3$ -cm irregular

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Fig 1.—Case 1, Computed tomographic scan shows infarct in lateral portion of thalamus (single arrow) and occipital lobe posterior to lateral ventricle (double arrow).

zone of softening in the right lateral portion of the thalamus, extending from the level of the massa intermedia to the splenium of the corpus callosum (Fig 2). The right cerebral peduncle and substantia nigra were also recently softened (Fig 3), and there was a recent infarct in the right medial portion of the occipital lobe. Histologically, the thalamic softening was composed of several confluent infarcts, some of which were subacute and others that were recent. A microscopic old infarct was present in the left side of the thalamus.

CASE 2.—A 64-year-old white man noted numb, tingly feelings in his left arm while driving a car. The numbress spread to his left cheek and leg, and walking was clumsy. The patient had experienced two prior transient spells of numbness, tingling, and clumsiness of the left arm two and three months before, respectively. He had a history of diabetes mellitus, hypertension, and two myocardial infarcts. Examination showed slight weakness in the left side of the face and slight clumsiness and ataxia in the left arm and leg. Sensitivity to pin and light touch was reduced on the left. The patient's gait was slightly unsteady. His reflexes were symmetrical, and his plantar reflexes were flexor. Computed tomography showed a small right lateral thalamic infarct. Angiographic findings were normal except for a slight nonstenosing plaque in the distal extracranial portion of the right vertebral artery. The right PCA and its branches were normal. The patient's left-sided clumsiness gradually improved, but subjective sensory symptoms remained in the left limbs.

CASE 3.—A 60-year-old Chinese man noted prickling paresthesia of his left arm, hand, and leg. Later, these spread to include the left side of his face, the inside of his mouth, and his left thumb. His left arm and leg were slightly uncoordinated. He had no important past illnesses. On examination there was slight subjective decrease in pin and touch sensation in the left limbs. Position sense was less accurate in the left hand and foot. There was slight ataxia and uncoordination of the left limbs on finger-to-nose testing, and the patient's



Fig 2.—Case 1, Thalamic infarct (arrow) at necropsy. Right hemisphere on right side of illustration.



Fig 3.—Case 1, Right cerebral peduncle and substantia nigra are soft and necrotic. These structures are on left side of illustration.

gait was slightly ataxic. Strength, reflexes, and plantar responses were normal. He had slight dysarthria. His blood pressure was elevated throughout his hospitalization, and he had glycosuria and an elevated serum glucose level that ranged from 12.1 to 21.3 mmol/L. Computed tomography showed a small lacune in the right putamen and an infarct in the right ventrolateral portion of the thalamus (Fig 4). Angiography showed a normal right PCA and no important stenosis of intracranial or extracranial arteries. The patient was treated with antihypertensive and oral antihyperglycemic agents. Walking gradually improved. He was readmitted to the hospital for left-sided pure motor hemiplegia, and CT showed no new lesions.

COMMENT

We have described two different vascular lesions in patients with lateral thalamic infarcts. In one patient, occlusion of the PCA proximal to the thalamogeniculate artery branches caused a large lateral thalamic infarct that was later complicated by progressive symptoms and ischemia in the cerebral peduncle and occipital lobe. Two other patients, each with hypertension and diabetes, had more restricted lateral thalamic infarcts and normal opacification of the PCA. These infarcts were gradually progressive or preceded by transient is-



Fig 4.—Case 3, Computed tomographic scan shows hypodensity in right portion of lateral thalamus (arrow).

chemic attacks, and there were no recognized proximal sources of emboli. The background diathesis for small-vessel disease, absence of documented large-vessel occlusive disease, and lack of a known source for emboli make it very probable that the patients had intrinsic occlusive disease of the thalamogeniculate arteries, but necropsy verification was not available. These vascular lesions and their resultant infarcts are diagramed in Fig 5 and are compared with the arterial lesions causing striatocapsular infarction. A review of the relevant vascular and brain anatomy, historical observations, clinical findings, and imaging abnormalities explains the various syndromes and their vascular causes in patients with lateral thalamic infarction.

Relevant Vascular and Brain Anatomy

The "thalamogeniculate pedicle" of arteries arises from the ambient segment of the PCA and penetrates into the thalamus between the geniculate bodies. The pedicle consists of six to ten arteries that vary widely in diameter and originate together. These vessels are the posterior circulation counterpart of the lenticulostriate middle cerebral artery branches that supply the striatum and internal capsule. All levels of the brain stem are irrigated by midline large, penetrating, and long and short circumferential arteries. The thalamogeniculate pedicles have short circumferential branches that supply the ventrolateral portion of the thalamus including the posterolateral and posteromedial ventral nuclei, the inferior and posterior portions of the ventralis lateralis, the lateral portion of the centrome-



Fig 5.—Diagram illustrating mechanisms of deep infarction. Top left, Basal ganglionic capsular infarction due to middle cerebral artery occlusion (arrow). Top right, Smaller deep capsular infarct due to lenticulostriate artery occlusion (arrow). Bottom left, Lateral thalamic infarct due to posterior cerebral artery occlusion (arrow). Bottom right, Lateral thalamic infarct due to thalamogeniculate artery occlusion (arrow).

dian nucleus, and the rostrolateral portion of the pulvinar. The polar and thalamoperforating arteries are midline penetrators, and the cortical branches of the PCA are analogous to the large circumferential arteries. The posterolateral choroidal arteries arise from the PCA, just distal to the thalamogeniculate arteries.

Historical Observations

Dejerine and Roussy¹¹ described and explained the findings in patients with infarcts in the ventroposterolateral portion of the thalamus, which they designated le syndrome thalamique. Clinical findings came from two cases reported previously by Dejerine and Egger¹⁰ and from three reported by their colleagues Dide and Durocher¹² and Chinny.¹⁴ The microscopic pathologic findings of three of these cases were reviewed for the first time (Fig 6, top left, center, and right).¹¹ All signs were contralateral to the thalamic infarct and included (1) a rapidly regressing slight hemiparesis; (2) a more prominent and persistent sensory abnormality affecting both "superficial" (touch, pin, and temperature) and "deep" (articular and proprioceptive) modalities (The patients also showed delayed and distorted perception, inability to localize sensation, and astereognosis. Sensory abnormalities affected the face, limbs, and trunk.); (3) hemiataxia (The contralateral limbs were hypotonic and uncoordinated, a finding the authors related to the sensory loss.); (4) abnormal spontaneous movements (hemichorea and hemiathetosis with spontaneous movements, usually small, of the hand and digits, occasionally with an undulating, rhythmic character at times resembling tremor [hemitremblement]); and (5) pain and dysesthesia that developed later on the hemianesthetic side. Deep tendon reflexes were normal or only slightly exaggerated, and there was no Babinski's sign. Facial weakness was common. Some patients had dysphagia, sphincter abnormalities, and "vasomotor" abnormalities such as a cvanotic, cool hand on the affected side. Dejerine and Roussy11 did not comment on the location or nature of the vascular lesion.

Foix and Hillemand¹³ reviewed the anatomy and vascular supply of the thalamus and elaborated on what



Fig 6.—Top left, center, and right, Drawings represent three cases. Anatomy was not marked. Ependymal lining of third ventricle is on left and insular cortex on right. Black represents infarcts all cutting across posterior limb of internal capsule (from Dejerine and Roussy¹¹). Bottom, Drawing illustrates frequent coexistence of lateral thalamic and temporo-occipital lobe infarction (in black) (from Foix and Hillemand¹³).



Fig 7.-Typical restricted lateral thalamic infarct at necropsy (arrowhead).

they called the syndrome of the thalamogeniculate pedicle. In addition to the signs noted by Dejerine and Roussy,¹¹ they added a description of the "thalamic hand," which is flexed and pronated, with the thumb trapped within the other digits. Sensory symptoms could be limited to the face, an arm, or a leg and could even mimic a radicular distribution.^{16,25} Cerebellar signs were caused by interruption of the thalamic termination of superior cerebellar peduncle fibers originating in the cerebellum. Foix and Hillemand¹³ and Foix and Masson²⁶ noted the frequent association of the thalamic syndrome with a hemianopia, dubbing this combination the syndrome of the PCA. Figure 6, bottom, is a copy of the diagram Foix and Hillemand¹³ used to illustrate this point. Necropsy studies have since documented the frequent coexistence of lateral thalamic and cortical PCA territory infarcts.²⁷⁻³¹ Figure 7 shows a restricted infarct localized to thalamogeniculate territory at necropsy.

Vascular Etiology and Symptoms of PCA Occlusion

When the location and cause of occlusive lesions within the posterior circulation were analyzed at necropsy, Castaigne et al²⁸ noted that most occlusions of the PCA affected cortical branches and were caused by cardiogenic or intra-arterial embolism. An angiographic study of 23 patients who had both hemianopia and occipital infarction seen on CT showed a high incidence of embolism to the PCA or its branches,32 and PCA embolism was common in the Michael Reese Stroke Registry.33 In situ stenosis and occlusion of the PCA are more common in blacks than in whites.³⁴ Atherostenosis of the PCA most often affects the proximal segments of the artery.35

The most frequent symptoms of PCA occlusive disease are caused by infarction within the temporo-occipital lobes and include visual field, reading, and memory abnormalities.^{30,36,37} Early authors noted signs of dysfunction of the rostral portion of the midbrain and thalamus in the territories of the thalamoperforating and thalmogeniculate arteries due to PCA occlusion.13,26 Benson and Tomlinson38 reported hemiplegia caused by proximal PCA occlusion affecting the cerebral peduncle, a finding that developed later in the course of our patient 1. Another patient had occlusion of the PCA just before the ambient segment on angiography and had contralateral paresis and sensory loss without hemianopia.³⁹ Fisher⁴⁰ briefly described three patients with severe stenosis of the proximal PCA among a large group of patients with transient spells of unilateral numbness; in one patient, the spells were followed by a major PCA territory infarct. Symptoms of ischemia of the midbrain and thalamus occur only when the PCA is occluded proximally before the origin of the thalamoperforating, thalamogeniculate, and choroidal artery branches.

Brain Imaging

Computed tomography and MRI can now accurately localize thalamic infarcts. In patients with PCA disease, infarcts seen on CT scans are most often localized in the territories

of cortical branches.^{32,41} In one reported case, a low-density CT lesion in the lateral portion of the thalamus in a patient with the thalamic syndrome probably represented an old, small hematoma.²¹ A study of CT localization of 25 thalamic infarcts described 11 patients with clinically diagnosed lateral thalamic infarcts, nine of whom had CT lesions in the thalamogeniculate territory: six of whom had visual field defects; and four of whom had additional PCA territory cortical infarcts.23 Angiography, however, was not performed in every case. Takahashi et al²⁴ analyzed the CT findings in 22 patients with thalamic infarcts. The thalamogeniculate territory was involved in nine of the patients, and all had severe stenosis or occlusion of the PCA, which was embolic in at least one patient. Thalamogeniculate artery lesions were limited to the posterolateral and posteromedial ventral nuclei, the lateral portion of the centromedian nucleus, and rostrolateral pulvinar. Two patients had CT lesions in the thalamogeniculate and cortical PCA territories, and four patients had lesions in the thalamogeniculate, posterolateral choroidal, and cortical PCA territories. The posterolateral choroidal artery arises adjacent to the thalamogeniculate pedicle, and supplies the dorsolateral and posterior pulvinar territory adjacent to the thalamogeniculate supply. In one series of 38 patients with PCA occlusion seen on angiography, four patients had thalamogeniculate infarcts on CT, and three of these four had additional cortical infarction.22 Among 50 PCA territory infarcts in another series, 15 were deep (thalamic), 24 were cortical, and 11 were mixed.³⁶ While proximal posterior cerebral occlusion can cause infarction limited to the thalamogeniculate territory, more often there are accompanying lateral posterior choroidal and cortical infarcts. The clinical findings in the reported cases of CTdocumented thalamogeniculate territory infarction were similar to those in our cases: slight transient hemiparesis, hemiataxia with involuntary small hand movements, and a prominent hemisensory loss. One patient, similar to our patient 1, was a 67year-old woman with left hemihypesthesia and hemiataxia who reported dizziness, gait ataxia, and hallucinations.³¹ The thalamic infarct was large and included the thalamogeniculate territory, with some additional medial thalamic extension, and the causative vascular lesion was stenosis of the ambient portion of the right PCA.

Prior Reports of Syndromes of Lacunar Infarcts in the Lateral Portion of the Thalamus

Occlusion of small, deep, penetrating artery branches is also a recognized cause of restricted lateral thalamic infarction. Fisher^{42,43} described serial section analyses in two patients with lacunar infarcts limited to the ventral posterior somatosensory nuclei of the thalamus. The cause of the vascular lesion was severe lipohvalinosis with disorganization of the arterial wall and obstruction of the lumens of arteries 2 to 400 μ m in diameter. The proximal PCA was normal. Clinical findings consisted of transient spells or strokes in which the symptoms were limited to numbness. dysesthesia, and paresthesia, most often in the contralateral portion of the face, limbs, and trunk. Objective threshold sensory loss was usually slight.^{6,40,42,43} Necropsy performed in another patient with right hemiparesis and hemisensory loss showed a lacune in the ventroposterior nuclei, with pallor of the adjacent internal capsule caused by penetrating artery disease.44

In two other necropsies, sensory motor stroke was associated with infarction of the ventroposterior portion of the thalamus and adjacent internal capsule.6 The original report of Dejerine and Roussy¹¹ included drawings of three infarcts that caused the thalamic syndrome (Fig 6, top left, center, and right). All were larger than lacunes and involved the ventrolateral portion of the thalamus, and all had small tonguelike extensions into the posterior limb of the internal capsule and posterior putamen. The authors did not analyze the vascular lesion, and we could not find a report documenting the vascular pathology in a patient with a ventrolateral thalamic infarct of comparable size and location.

Clinical Separation of Syndromes

Several clinical clues help separate PCA disease from branch disease of the thalamogeniculate artery. These include (1) the lesions observed on CT are somewhat larger; (2) CT lesions are not restricted to thalamogeniculate territory but may also include the pulvinar, the more medial portion of the thalamus, or portions of the inferior temporal or occipital lobes; and (3) the clinical syndrome may include elements not usually a part of the thalamic syndrome. Patient 1 showed neglect of the left side of space, conjugate deviation of the eyes to the ipsilateral side, and hemianopia, findings that, to our knowledge, have not been described in thalamogeniculate territory infarction. These signs developed only when infarction was noted on CT in the temporal and occipital lobes. We have seen one other patient with conjugate eye deviation in a combined thalamic-superficial PCA territory infarct; conjugate eye deviation does not usually accompany hemianopia in patients with restricted temporooccipital infarcts. Neglect has been described in unilateral rostral brainstem lesions, usually involving the medial midbrain tegmentum and the medial portion of the thalamus.7,45-48 Progression of signs, especially if it is stepwise and occurs during a period of weeks, is unusual in branch disease.

The findings in thalamogeniculate branch occlusion infarction also differ from pure sensory stroke. Patients have abnormal motor signs of transient hemiparesis and incoordination, tremor, and abnormal spontaneous movements ("chorea or tremor") on the side of the sensory loss. The motor abnormalities are probably caused by interruption of extrapyramidal pathways from the ansa lenticularis and cerebellifugal fibers from the superior cerebellar peduncle and red nucleus that synapse in the ventrolateral nucleus of the thalamus and by involvement of the posterior limb of the internal capsule adjacent to the thalamus. We have also noted a slight but definite gait ataxia that is often more severe than the ataxia of the contralateral limbs. When infarction is limited to the posterior portion of the ventral nuclei (pure sensory stroke) patients report numbress and paresthesia, but there rarely is objective decrease in somatosensory thresholds. These smaller infarcts are probably due to disease of smalldiameter thalamogeniculate artery branches. A slightly larger branch probably irrigates both the lateral portion of the thalamus and the posterior portion of the internal capsule and putamen; occlusion causes a sensory-motor stroke. When infarcts are larger and affect fibers passing from the medial and lateral portions of the thalamus to the cortical sensory zones, sensory loss is more severe and "thalamic pain" often develops later. Unfortunately, there have not been enough morphological studies performed that characterize the underlying vascular pathology within the thalamogeniculate pedicle. Normal angiographic findings and the frequency of coexistent lacunes, hypertension, and diabetes (risk factors for penetrating artery disease) suggest that the lesion is similar in most cases to other basilar branch infarcts.^{49,50} In

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some patients, occlusion of the PCA can cause an infarct limited to thalamogeniculate territory. How many of these patients will progress, as our patient 1 did, to having a more extensive infarct in the PCA territory is unknown.

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