

Unilateral Amnesic Stroke

Six New Cases and a Review of the Literature

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Background and Purpose: Although persistent amnesia due to bilateral limbic system infarction is well described, reports of amnesic syndromes due to unilateral stroke have appeared infrequently and unsystematically. We report six new cases and review previously published reports to expand and consolidate knowledge regarding amnesic stroke.

Case Descriptions: Six patients developed acute amnesia associated with unilateral stroke. Brain computed tomography or magnetic resonance imaging revealed lesions in limbic structures, which accounted for the memory deficit. Based on these as well as similar cases in the literature, we delineate three distinct syndromes of unilateral amnesic stroke involving the territories of the posterior cerebral, anterior choroidal, and thalamic penetrating arteries. Eighty-five percent of reported cases have involved the left hemisphere.

Conclusions: Persistent as well as transient amnesia may be the sole or primary manifestation of unilateral hemispheric stroke. The vascular mechanisms of amnesic stroke are diverse. Patients presenting with acute amnesia possibly related to cerebral ischemia should be classified by documented or presumed lesion site as well as the involved vascular territory. Left amygdalohippocampal or diencephalic dysfunction may produce a particular vulnerability to global amnesia. (*Stroke* 1993;24:1033-1042)

KEY WORDS • amnesia • thalamus • hippocampus

In 1900, Bechterew¹ described a patient who experienced amnesia related to stroke. Postmortem study revealed bilateral softening involving the uncus and Ammon's horn. Later experience with patients who underwent bilateral excisions of the medial temporal lobes for treatment of seizures revealed a severe and lasting amnesic syndrome,² which confirmed the importance of bilateral hippocampal integrity for normal memory function.

In 1961, a detailed postmortem study by Victor and colleagues³ of a patient with bilateral infarctions in the posterior cerebral artery (PCA) territories established stroke as an etiology of acute and persistent amnesia. Several similar case reports followed; however, only four postmortem cases have documented amnesia in relation to unilateral PCA strokes affecting hippocampus and thalamus.⁴⁻⁷ All four patients had other deficits that were prominent such as right homonymous hemianopia and anomia for colors, and in no patient was the memory deficit documented as lasting longer than 6 months. Contralateral lesions in the thalamus,⁵ parietal lobe,⁶ and pons⁷ were not considered contributory to amnesia in these patients.

In 1973, the term "amnesic stroke" was used by Benson and colleagues⁸ to describe cases of acute amnesia associated with visual field deficit. Based on their series of 10 patients studied with radionuclide

brain scans and a review of the literature, they concluded that a case of isolated persistent amnesia secondary to stroke had never been reported.⁹ Six of their patients had apparent bilateral lesions; four were unilateral, and all were left sided. An earlier case of a patient with amnesia and left homonymous hemianopia related to right occipital stroke on brain scan was reported by Halsey.¹⁰ In 1974, Mathew and Meyer¹¹ reported two additional cases of amnesia, visual agnosia, and hemianopia related to left occipital lobe lesions.

It is possible that contralateral, small lesions may have been missed in earlier reports of amnesic stroke that relied on radionuclide scans and angiography for lesion demonstration. Computed tomography (CT) and magnetic resonance imaging (MRI) allow more reliable in vivo anatomic analysis, and the application of these techniques to patients presenting with memory dysfunction has consolidated and expanded previous conceptions of unilateral amnesic stroke.

We present the radiographic and clinical features of six patients whom we have seen recently and in whom acute amnesia was the sole or primary manifestation of stroke. In this series, patients were regarded as having amnesia if they reported memory difficulties temporally related to the onset of stroke and if conventional tests of mental status revealed impairment of delayed recall of verbal or visually presented material in the absence of attention deficit or aphasia sufficient to impair registration. The cases are summarized in Table 1. We also review all such cases in the English- and French-language medical literature that were supported by CT or MRI studies to define further the clinical spectrum of unilateral amnesic stroke.

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TABLE 1. Clinical Characteristics of Patients With Amnesic Stroke

Patient	Side	Location	Lesion	Radiologic study	Mental status	Other signs	Amnesia duration
1	Left	Occipital lobe, posterior hippocampus	Primary hemorrhage	MRI scan	Amnesia, constructional apraxia	Right inferior quadrantanopia	>52 Days
2	Left	Occipital lobe, posterior hippocampus	Hemorrhagic infarct	CT scan	Amnesia; later anomia, apraxia, alexia	Transient diplopia; later right homonymous hemianopia	>17 Days
3	Left	Temporal, occipital lobes	Infarct	MRI scan	Amnesia, alexia without agraphia	Right superior quadrantanopia	>16 Days
4	Left	Amygdala, anterior hippocampus	Infarct	CT scan	Amnesia	None	>2 Years
5	Left	Anterior thalamus	Infarct	MRI scan	Amnesia, anomia	None	>5 Months
6	Left	Occipital lobe, midthalamus, posterior hippocampus	Infarcts	MRI scan	Amnesia	Right superior quadrantanopia	10 Hours

MRI, magnetic resonance imaging; CT, computed tomography.

Case Reports

PCA Stroke

Case 1. A 67-year-old right-handed retired dermatologist was seen 1 month after the onset of memory loss. His forgetfulness was observed by his wife to affect recent memories of activities of daily living. During the week before evaluation, the patient's memory became worse, and he was aware of difficulty in remembering names. Several days later, he noted visual hallucinations of a moving black veil and an isolated hallucination of a chair in his right visual field.

The neurological examination revealed a right inferior temporal quadrantanopia, mild left ptosis, and miosis. He was alert and fully oriented. Serial-seven recitation, calculations, naming, reading, and writing abilities were normal. Verbal memory recall of three objects was three of three immediately and none of three at 5 minutes. He could not recall recent political events. There was mild constructional apraxia on clock drawing.

CT revealed a left intracerebral hemorrhage extending from the parahippocampal gyrus lateral to the apex of the tentorium superiorly into the occipital lobe. On the following day, MRI revealed a large hematoma with edema and mass effect in the same region of the occipital lobe with extension into the area of the hippocampus. Sagittal and transaxial T1- and T2-weighted images showed no lesions in the contralateral hemisphere. Transaxial MRI views as well as corresponding templates, using the method of Damasio and Damasio,¹² are presented in Fig 1. Repeat CT performed 17 days later showed near-complete resolution of the hemorrhage within a small area of low attenuation in the occipital lobe without mass effect.

At 52 days after his first examination, the patient's memory was said still to be moderately impaired, although improved. His visual field defect had resolved.

Case 2. An 87-year-old woman with a history of hypertension and paroxysmal atrial fibrillation was admitted to the hospital for confusion and visual impairment. Three days earlier, she had a 12-hour episode of palpitations, after which she became aware of memory impairment. One day before admission, the patient presented to the emergency department with com-

plaints of memory impairment, right-sided headache, and a fluctuating right visual field defect. Diplopia on right lateral gaze was noted by the examiner and resolved within 6 hours. She had a mild resting tremor of the left hand and mouth, depressed ankle reflexes, and stocking distribution hypesthesia. Visual fields were normal, and there was no extinction of double simultaneous stimuli. The patient was alert, fully oriented, and aware of her amnesia; verbal immediate memory recall of three objects was three of three, and delayed memory recall was none of three. Naming, repetition, reading, speech fluency, and calculations were normal. CT without contrast showed no abnormalities. She was discharged and treated with daily aspirin.

The patient was admitted the next day with increasing memory complaints, word-finding difficulty, and right visual field loss. Neurological examination revealed a right superior temporal quadrantanopia. She was completely oriented. Naming, comprehension, repetition, speech fluency, and praxis for three-step motor commands were normal. Serial-sevens and spelling of "world" in reverse were normal. Registration of three objects was three of three immediately and none of three for recall at 5 minutes. She knew the current president, but none before him. The patient did not recall events of the previous day.

Heparin was administered for presumed cardioembolic stroke. Carotid ultrasound revealed 30% to 50% left internal carotid stenosis and 50% to 79% right internal carotid stenosis. Transcranial ultrasound study, including the posterior circulation, was normal. On the third hospital day, the patient's memory and visual function was not improved. That evening, she experienced 30 to 90 seconds of complete blindness. This was thought to be the result of a second embolus to the basilar artery, and heparin therapy was continued. On the fifth day, she became disoriented to place and time. Mental status examination revealed anomia with alexia, agraphia, and apraxia for three-step commands. Repetition and speech fluency were normal. Delayed object recall was none of three at 5 minutes. Visual field testing showed a right homonymous hemianopia. CT showed a left occipital hemorrhagic infarction. The right hemisphere was normal

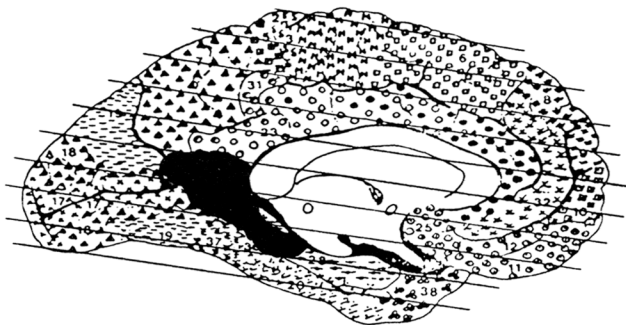
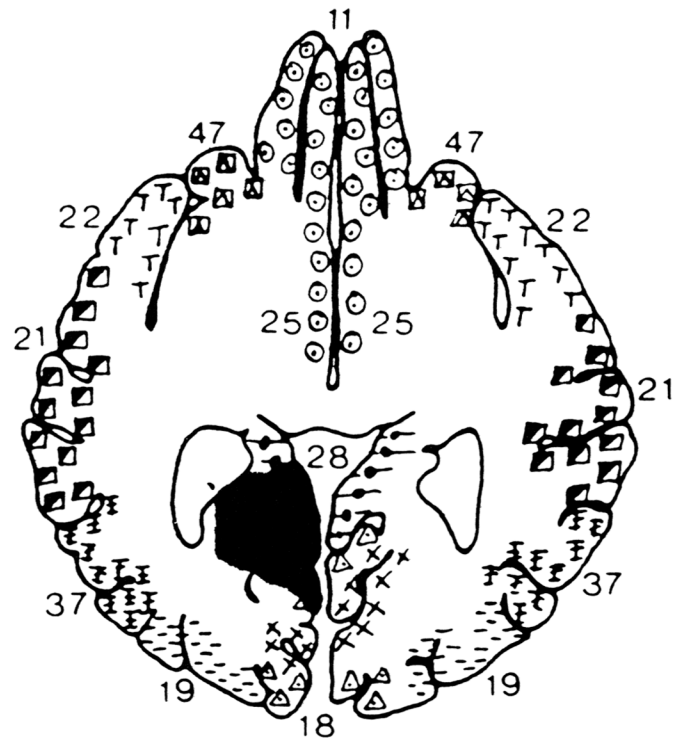
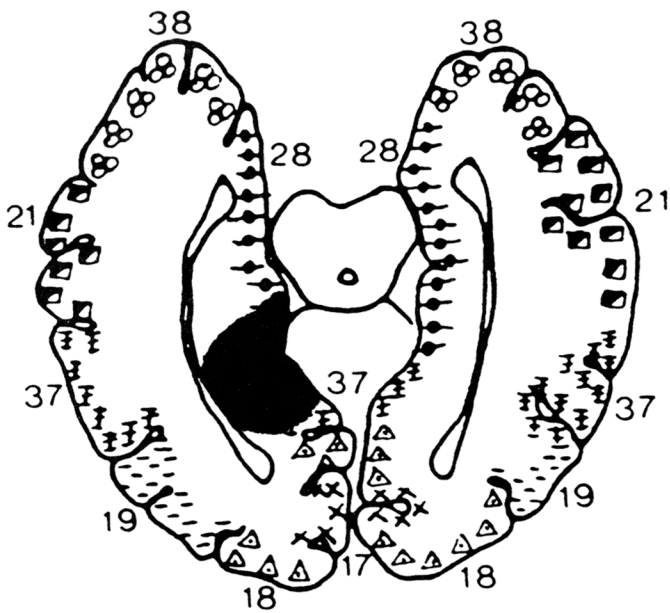
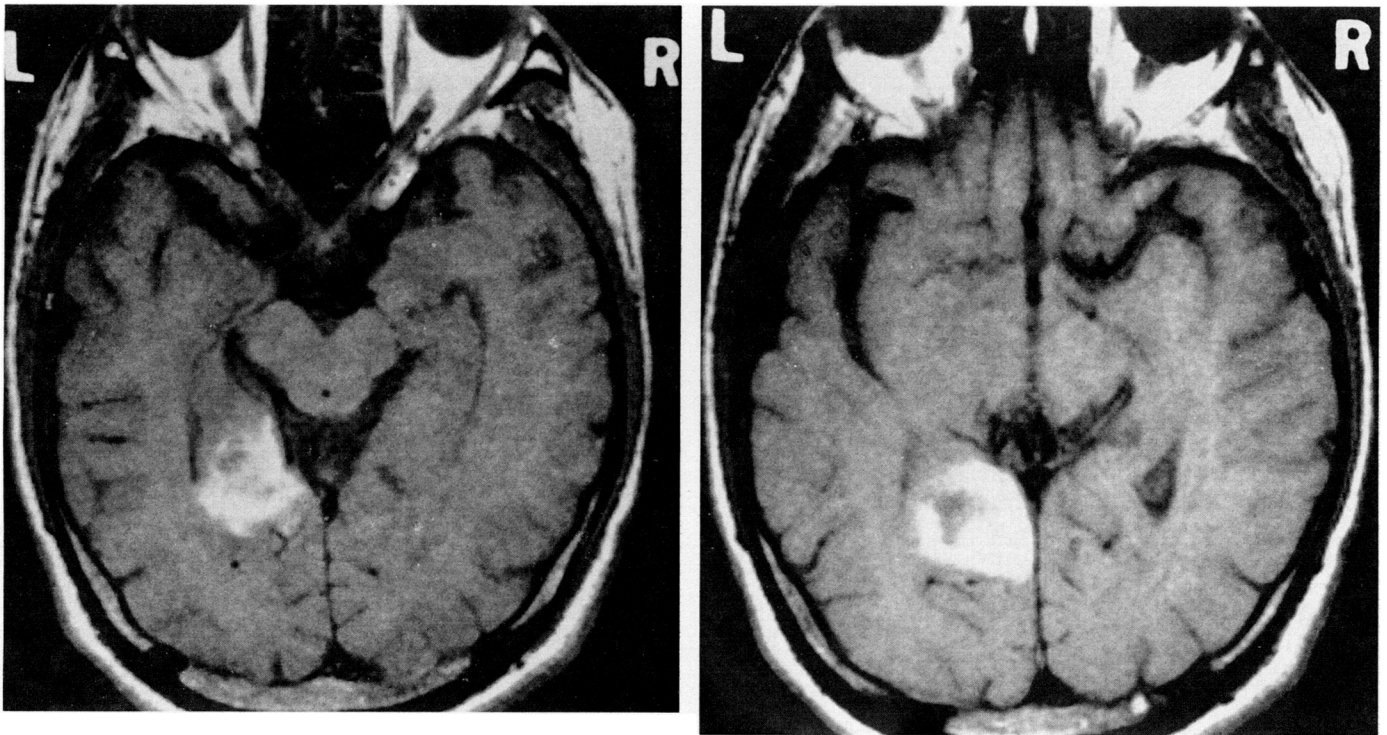


FIG 1. T1-weighted magnetic resonance imaging axial views (TR, 600 ms; TE, 20 ms) (top panels) and corresponding templates (middle and bottom panels) of patient 1. Region of hemorrhage, ischemia, and edema involves middle and posterior portions of left hippocampus and parahippocampal gyrus.

in appearance. When discharged on day 17, the patient was well oriented but had persistent impairments in naming and verbal memory.

Case 3. A 70-year-old woman with a history of diabetes and hypertension presented with a 1-week history of forgetfulness and headaches. Seven days

earlier, while bending over cleaning, the patient suddenly developed dull occipital headache and transient lightheadedness. Since that time, she and her family reported that she had memory difficulty, including forgetting what she read and money she received. Neurological examination 10 days after onset disclosed a right homonymous superior temporal quadrantanopia and absent ankle jerks. On mental status testing, she was alert, oriented, and attentive. Naming, repetition, calculations, constructions, and praxis were normal. There was no evidence of color anomia, finger agnosia, or right-left confusion. Memory recall was three of three immediately and none of three at 5 minutes. She could not recall recent meals but knew some other details of her hospitalization. She exhibited alexia without agraphia, with residual reading accomplished by laboriously spelling out words letter by letter.

Carotid ultrasound demonstrated 30% to 50% stenosis of the right carotid. Transcranial Doppler showed normal intracranial vertebral and basilar signals; bone thickening precluded insonation of the PCAs.

CT done 8 days after onset showed irregular low density in the left posterior mesial temporal lobe. MRI on day 15 demonstrated multiple high signal abnormalities in the medial left temporal and occipital lobes without mass effect. No lesions of the contralateral hemisphere were seen on this study, which included postgadolinium T1- and T2-weighted coronal images. A small amount of edema was present, but there was no enhancement of the infarct. At discharge 16 days after onset, verbal memory remained impaired but was thought to be improving.

Comment

These three patients presented with prominent memory disturbances lasting at least 16 to 52 days in association with new unilateral vascular lesions involving the posteromesial temporal lobe within the territory of the PCA. Visual field deficits, most often a superior quadrantanopia, commonly accompanied the memory disturbance but were not obligatory as evidenced by the initially normal fields in patient 2. Accompanying cognitive deficits were variable. Patient 3 exhibited alexia without agraphia, but patient 1 demonstrated only mild constructional apraxia, and patient 2 initially presented with an isolated memory disturbance. Lesions affected the left hemisphere in all three patients.

Infarction or hemorrhage in the territory of the PCA is a common anatomic underpinning of unilateral amnesic stroke. In the neurological literature, 48 previous cases of patients with memory deficits associated with unilateral PCA infarction have been reported, although in many, amnesia, while present on formal testing after the acute period, was not clearly a primary feature of the initial presentation.^{4-7,13-18} Forty-four of these 48 patients exhibited left-sided lesions.

Evidence from series of consecutive patients with PCA stroke suggests that PCA infarctions produce memory disturbance only when lesions extend to posteromesial temporal lobe structures critical for normal memory function.¹⁶ Mesial temporal involvement was present in our 3 patients and in all 32 with sublocalization data collected from the literature.

The PCA gives rise to three main groups of branches: central branches supplying the brain stem, ventricular

branches supplying the choroid plexus, and cortical branches supplying the cerebrum. Nomenclature for the cortical branches varies. Zeal and Rhoton¹⁹ recognize among the cortical branches a variable inferior temporal group of as many as five branches that supply the inferior temporal lobe.

Accompanying signs in patients with amnesia due to unilateral PCA stroke reflect the degree of involvement of the different branches of the PCA territory. Hemi-anopic or quadrantanopic visual field defects suggest lesions involving the occipitotemporal or calcarine branches supplying the optic radiations or calcarine cortex²⁰; the superior visual field is affected more often than the inferior because temporal infarctions are more likely to be accompanied by inferior than by superior occipital ischemia. Alexia without agraphia will appear if the paraventricular white matter of the left occipital lobe has been damaged, disconnecting both interhemispheric and intrahemispheric visual pathways to the angular gyrus.^{13,14} Color anomia may be seen with involvement of the mesial occipitotemporal junction of the left hemisphere.¹³ If these more posterior regions are spared, a relatively isolated memory deficit may be encountered.

Patients with amnesia produced by posterior circulation ischemia may demonstrate both transient and persistent deficits in brain stem as well as bilateral PCA territories when basilar artery thrombosis or embolism occurs. This is exemplified by patient 2, in whom transient blindness and transient diplopia occurred. Thus, the possibility of bilateral hippocampal ischemia not seen on radiologic images may be difficult to exclude in the pathogenesis of amnesic stroke.

Anterior Choroidal Artery Stroke

Case 4. A 65-year-old right-handed woman was admitted to the hospital for severe memory loss. She had noticed increasing forgetfulness in activities of daily living for several days before admission, abruptly worsening on the day of admission. She became disoriented to time and could not recall her daughter's telephone number. She was aware of her deficit and called the paramedics.

On admission, the patient's blood pressure was 240/130 mm Hg. Neurological examination was normal except for mild stocking distribution hypesthesia. Mental status testing revealed that she was alert and attentive but disoriented to the month and year. Serial-sevens were performed to 86. She could spell "world" in reverse. Calculations were normal. Verbal memory recall of three objects was three of three immediately and none of three at 5 minutes. She could not name the president. Speech was fluent. Naming, repetitions, comprehension, reading, and writing were normal. There was minimal difficulty with labeling the hands of a clock and drawing intersecting pentagons.

Her past medical history was positive for diabetes and hypertension. On admission, her blood glucose level was 415. A CT on admission showed a chronic lacunar infarct in the right anterior centrum semiovale. Carotid ultrasound showed mild turbulence of the left internal carotid but no significant stenosis. Echocardiogram showed mild mitral annulus calcification. EEG was normal. On day 3, a repeat CT showed a small area of new infarction in the medial left temporal lobe in the

region of the anterior hippocampus and amygdala. By day 5, there was no change in mental status. Warfarin therapy was initiated, and she was discharged from the hospital. At 3½ months, a repeat CT showed no change (Fig 2).

The patient's amnesia persisted. Mental status testing at 2 years revealed that she was alert but disoriented to month and year. She was oriented to place and knew the day of the week. She counted backward from 20, recited the months in reverse, and spelled "world" in reverse without error. Digit span was six forward and five in reverse. Thirty words beginning with the letters F, A, and S were generated over 3 minutes. Naming was correct for four of five fingers, five of five body parts, five of five object parts, and five of five colors. Repetition, reading, writing, and comprehension were normal. Praxis for three-step commands and calculations were normal. Verbal memory recall of a name and address was five of five immediately and four of five at 5 minutes. Recall of three unrelated words was three of three immediately, one of three at 3 minutes, and none of three at 5 minutes. Construction of a clock to command showed intact number placement and a mild error in hand placement. The patient was able to copy three abstract figures but had absent immediate recall. When she was individually presented with five different abstract figures, two of five were reproduced after a delay of 5 seconds. Visual recall of three objects hidden in a room was three of three immediately and two of three at 5 minutes.

Comment

This patient presented with memory impairment as her sole neurological complaint. There have been two reported cases of persistent amnesia following unilateral infarction in which there were no other neurological deficits; both involved infarcts in the left thalamus.^{21,22} However, our patient exhibited infarction in the left anteromesial temporal lobe, most likely in the territory of the anterior choroidal artery (AChA).

Three previous cases of amnesia related to probable unilateral AChA infarction have been reported. Two patients exhibited the more common manifestations of AChA stroke, with hemiplegia, hemisensory loss, and hemianopia in addition to an anterograde memory deficit. CT showed an infarction of the left posterior limb of the internal capsule, lateral geniculate body, and medial temporal lobe in one patient²³ and infarction of the posterior limb and genu of the left internal capsule in the other patient.²⁴ The third patient experienced right hemiparesis and dysarthria as well as verbal amnesia. CT showed an infarction of the posterior limb and genu of the left internal capsule.²⁵

The AChA originates from the internal carotid artery shortly after the takeoff of the posterior communicating artery.^{26,27} Inferolateral branches of the AChA supply the posteromedial half of the amygdala, the anterior hippocampus, and the fascia dentata.²⁶ There are rich anastomotic connections with the PCA in this area, and in the absence of a PCA hippocampal branch, the AChA may also supply the midportion and tail of the hippocampus.²⁷ Other branches of the AChA supply the optic tract, medial globus pallidus, uncus, piriform cortex, cerebral peduncle, lateral geniculate body, posterior limb of the internal capsule, tail of the caudate

nucleus, and choroid plexus of the lateral ventricle. A combined lesion of the amygdala and the anterior hippocampus is the likely substrate for the memory loss seen in our patient.²⁸

Hemiparesis, hemisensory disturbance, hemiataxia, and variable visual field deficits, alone or in combination, are the most common manifestations of AChA territory infarction. Symptomatic memory disturbances appear to be uncommon. No cases of amnesia were reported among 68 patients from several recent large series of consecutive AChA territory infarctions. In part, this is due to the fact that the mesial temporal lobe is involved in only a minority of AChA strokes, representing 38% in one series.²⁹ Underrecognition of patients with mild memory disturbance also is likely.

Our case clearly demonstrates that amnesia can occur with left unilateral AChA infarction. When the infarct is confined to the mesial temporal lobe, amnesia may arise in relative isolation; when the ischemic lesion includes the posterior limb of the internal capsule and the lateral geniculate body, accompanying hemimotor, hemisensory, and hemivisual deficits may appear.

Thalamic Penetrating Artery Stroke

Case 5. A 68-year-old right-handed woman was evaluated for persistent memory loss. She had a past history of anemia, hypertension, and squamous cell carcinoma of the base of the tongue and larynx, and she had been treated 2 years earlier with radiation and *cis*-platinum chemotherapy. She underwent modified radical neck dissection 1 year later for recurrent tumor. Bilateral carotid angiography was normal at that time.

The patient had no problems with cognition until she experienced the abrupt onset of memory loss. She could not remember where she had placed objects in the house, recent points of conversation, and events of the day. She could not balance her checkbook and had difficulty with word finding while writing but not in speech. MRI of the head and neck, including coronal images and gadolinium enhancement, was performed to evaluate her symptoms. An enhancing soft tissue mass thought to be a tumor was present in the left pharynx and extended into the jugular foramen and displaced the carotid artery. A 1-cm area of low intensity on T1-weighted images and high intensity on T2-weighted images was seen in the region of the ventral anterior nucleus of the left thalamus (Fig 3).

Neurological examination at 3 months revealed ptosis of the left eyelid and postsurgical deformities of the tongue, palate, and neck on the left side. There were no cervical bruits. The patient was alert and oriented to the month, year, and approximate date. She did not know the day of the week. Reverse counting from 20 to 1 and spelling "world" in reverse were correct. Two errors were made in reciting the months in reverse. Registration of a name and address was intact for five of five items; recall at 3 minutes was none of five. Verbal memory recall of three objects was three of three immediately and none of three at 3 minutes. Reading, writing, comprehension, repetition, and naming of high-frequency objects were normal. Naming of low-frequency objects was mildly impaired. Clock construction, praxis, and calculations were normal. She was aware of her memory impairment.

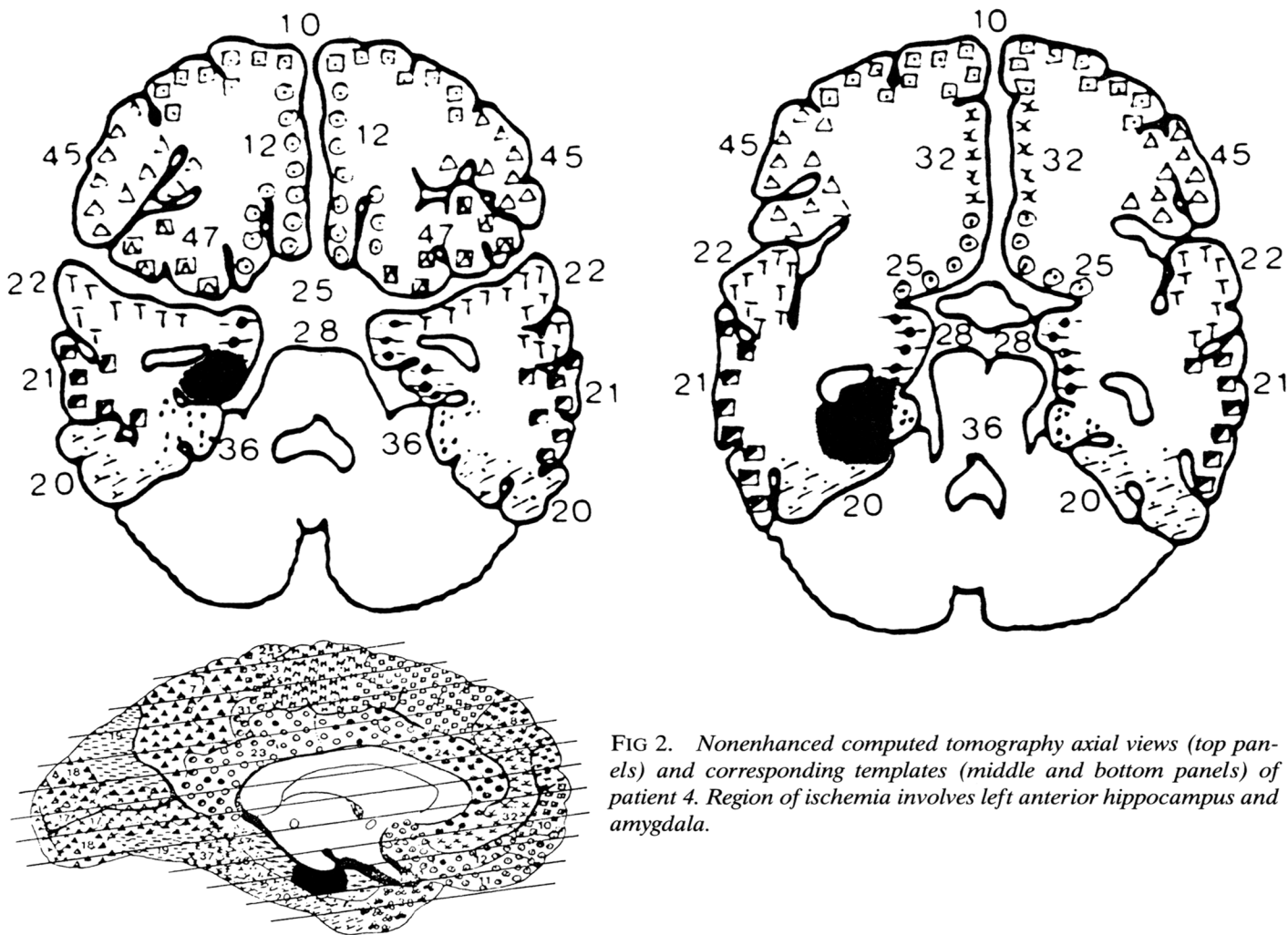
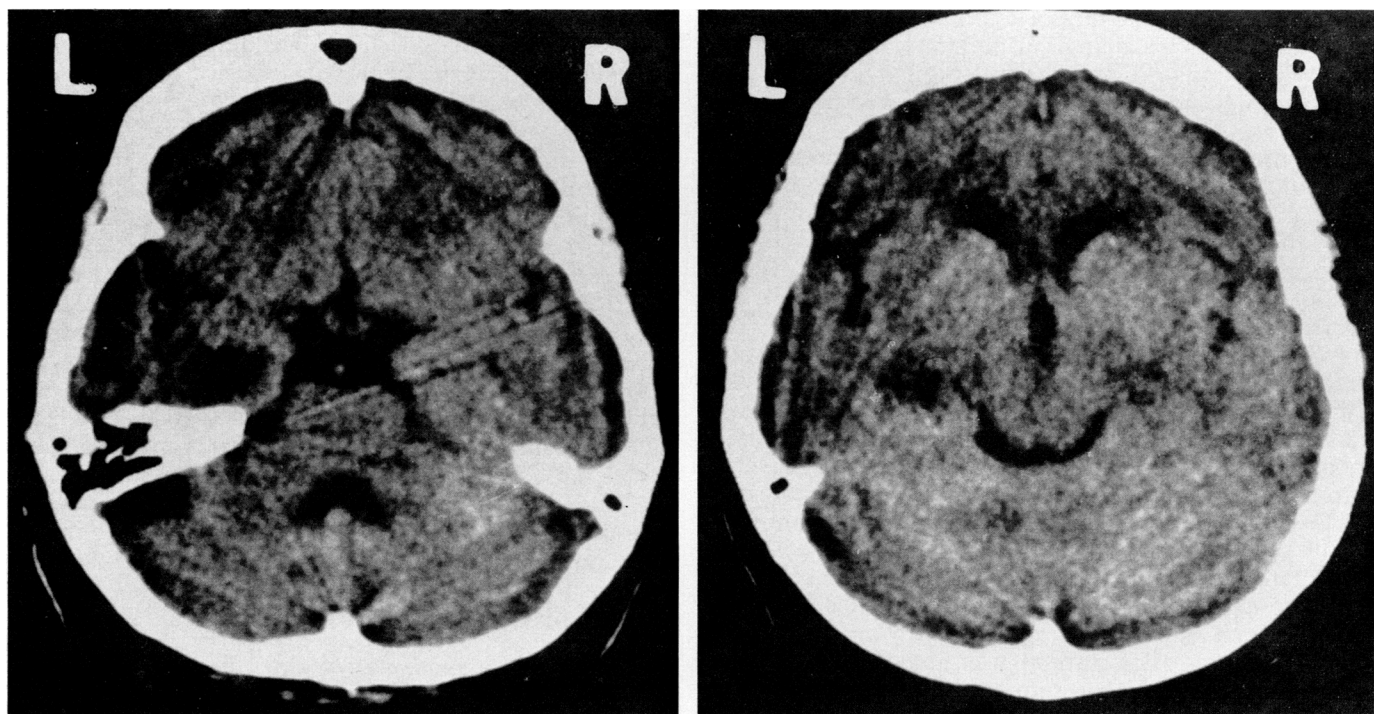


FIG 2. Nonenhanced computed tomography axial views (top panels) and corresponding templates (middle and bottom panels) of patient 4. Region of ischemia involves left anterior hippocampus and amygdala.

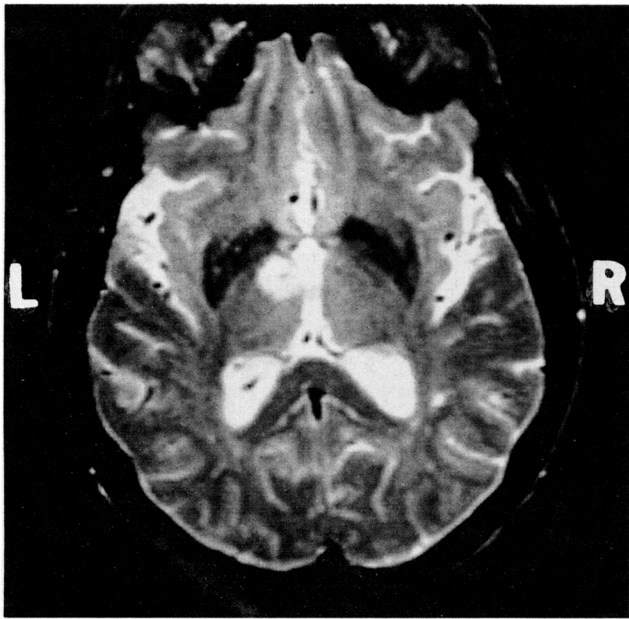


FIG 3. T2-weighted magnetic resonance imaging axial view (TR, 2500 ms; TE, 80 ms) of patient 5. Region of ischemia involves ventral anterior nucleus of left thalamus.

At 5 months, the amnesia persisted. Delayed recall of a name and address was three of five. Visual memory was slightly impaired. She was able to recall four of five abstract figures after a 10- to 15-second delay and recall three of three hidden objects in a room after a 5-minute delay. A repeat MRI, including coronal views, performed 10 days after this examination showed volume loss and low signal on T2-weighted images in the previously abnormal area of the anterior thalamus; this probably was related to residual hemosiderin from prior hemorrhagic infarction of the area. A new lesion in the left caudate nucleus showed volume loss and a mixture of high- and medium-intensity signals thought to represent infarction of 6 to 8 weeks' duration. There was no enhancement of either lesion with gadolinium. No lesions were seen in the contralateral hemisphere. The patient died unexpectedly at home in the sixth month; postmortem examination was not obtained.

Comment

This case demonstrates another common site of unilateral amnesic stroke—the thalamus. Eleven cases of unilateral thalamic hemorrhage and 41 other cases of unilateral thalamic infarction producing memory impairment have been reported in the literature.^{15,21,22,30-52} Eight of the 11 hemorrhages were left sided, but rupture into the ventricular system occurred frequently and may have produced deep bihemispheric dysfunction. The left side was affected in 32 of 41 infarctions.

Among cases of ischemic infarction, memory dysfunction primarily has been reported with lesions in two of the four main arterial territories of the thalamus: the tuberothalamic artery, as in patient 5, and the paramedian artery. The tuberothalamic artery arises from the posterior communicating artery and supplies the anterolateral thalamus, including the ventral anterior nucleus and part of the ventrolateral nucleus. The paramedian artery originates from the P1 segment of the

PCA and irrigates the medial upper midbrain and medial thalamus, including most of the dorsomedial nucleus and the intralaminar nuclear group.^{45,53} The tuberothalamic and paramedian arteries overlap in supplying hippocampal- and amygdala-related components of the thalamus that have been suggested to be critical for memory function, including the mamillothalamic tract, the ventroamygdalofugal pathway, and the dorsomedial nucleus.^{22,38,54}

Features accompanying amnesia in thalamic infarction vary with lesion site. In tuberothalamic infarctions, cognitive deficits, including aphasia with left-sided lesions and neglect and visuospatial processing impairments with right-sided lesions, often are prominent presenting features, along with transitory motor and sensory signs and a facial paresis for emotional movements.^{36,45} Paramedian infarctions typically begin with somnolence and supranuclear vertical gaze paresis. Neuropsychological deficits, including amnesia, appear as the patient improves. Hemiparesis, hemiataxia, and delayed abnormal movements also may occur.^{36,45}

Because of the common blood supply through the posterior circulation, amnesic strokes involving the thalamus may be accompanied by infarction of the medial temporal lobe, as seen in patient 6.

Transient Amnesia and Unilateral Stroke

Case 6. A 29-year-old pregnant software engineer was admitted to the hospital with acute onset of mild right-sided weakness, facial numbness, and memory loss. She could not remember where she lived. On arrival at the hospital, the patient's motor and sensory symptoms had resolved. Neurological examination revealed a right superior temporal quadrantanopia. Mental status testing was normal. She was fully oriented. Memory recall of three objects was three of three at 5 minutes.

CT was normal. She was treated with heparin. On day 2, coagulation studies, transthoracic and transesophageal echocardiogram, and vertebral and left carotid arteriogram were normal.

On day 3, the patient developed acute global amnesia lasting 20 minutes. Amnesia recurred and progressively worsened 2 hours later and lasted 10 hours. She was alert and attentive, but delayed verbal item recall (none of four) was severely impaired during the peak of this event. There was retrograde amnesia for events occurring more than 2 days before onset of the first episode. Later in the evening, as memory function was returning, the patient was presented with three unrelated words and three abstract figures to remember. Immediate recall was three of three for the words and the figures on the first trial. Five-minute recall was two of three for the words and two of three for the figures. When presented with multiple choices for the forgotten items, she was able to correctly identify the forgotten word and figure. She was aware of her deficit and felt as if she had lost her "RAM." On resolution of the amnesia, she asked how many days she had lost. Recall of the events during the time of amnesia was very poor. EEG at the time of amnesia showed bitemporal slowing attributable to drowsiness and no epileptiform activity. Sleep spindles were seen, although they were less frequent in the right hemisphere.

TABLE 2. Syndromes of Unilateral Amnesic Stroke

Vascular territory	Memory-related anatomic structures involved	Common associated symptoms	Reported cases	Laterality
Posterior cerebral artery	Hippocampus, parahippocampal gyrus, collateral isthmus	Hemianopia or quadrantanopia, pure alexia, color anomia	51	47 Left, 4 right
Thalamus: paramedian artery, tuberothalamic artery	Mamillothalamic tract, ventroamygdalofugal pathway, dorsomedial nucleus	Somnolence, vertical gaze paresis, hemiparesis, hemiataxia (paramedian artery); visuospatial deficits, emotional facial paresis, aphasia (tuberothalamic artery)	42 Infarcts 11 Hemorrhages	33 Left, 9 right 8 Left, 3 right
Anterior choroidal artery	Amygdala, anterior hippocampus, genu, posterior internal capsule	Hemiparesis, hemiataxia, hemisensory and hemivisual deficit	4	4 Left

On day 4, CT showed a large, wedge-shaped ischemic infarct in the left occipital lobe. MRI that day showed extension of this infarct into the parahippocampal gyrus. Sagittal, transaxial, and coronal T1- and T2-weighted images showed no lesions in the contralateral hemisphere. A small area of infarction also was seen in the left thalamus. The patient was transferred to another hospital.

Comment

Patient 6 exhibited transient amnesia in association with a new left occipitotemporal and thalamic infarction. This case illustrates that transient amnesia lasting less than 24 hours may be associated with unilateral stroke.

Radiologic series and case reports of the syndrome of transient global amnesia (TGA) have reported CT evidence of stroke involving left and right hemispheric structures in a minority of patients. From 0% to 10% of patients will have evidence of new infarcts, as in patient 6. From 0% to 15% of patients will exhibit older infarcts probably antedating the amnesic episode. We identified in the literature 22 patients similar to patient 6 in whom probable new unilateral infarcts were associated with transient amnesia of less than 24 hours' duration.⁵⁵⁻⁶⁴ Temporal lobe lesions were evident in nine and thalamic lesions were evident in eight, whereas lesions in areas generally unassociated with memory function, such as the basal ganglia and internal capsule, were noted in five. Sixty-eight percent of the lesions were left sided. Although many of these patients exhibited neurological deficits in addition to transient memory difficulty and therefore would not meet most criteria for "pure" TGA,⁶⁵ several patients have been reported with a clinical syndrome of TGA *sensu stricto* and new unilateral infarcts.⁶⁰

The term "transient global amnesia" should be reserved for cases in which amnesia of less than 24 hours' duration is the only symptom; only exceptionally will these cases be associated with underlying stroke. Because amnesia is a sign and symptom of disease, cases with persistent amnesia due to a documented or presumed cerebrovascular etiology most rationally are classified according to lesion site and vascular territory. As

demonstrated by our cases, a variety of stroke mechanisms, including cardioembolism and intracerebral hemorrhage, can produce amnesic stroke.

Conclusions

The term "amnesic stroke" originally was applied to cases of amnesia due to stroke in which PCA ischemia produced additional lesions of visual pathways.⁹ A review of the literature and our experience suggest that this remains a common presentation; however, transient as well as persistent amnesia may be the sole manifestation of strokes that are limited to the hippocampus or thalamus. This is an important point for the clinician to keep in mind during the initial evaluation of the patient with acute amnesia.

The two most common syndromes of unilateral amnesic stroke involve the vascular territories of the PCA with infarction of the hippocampus and of the thalamic perforating vessels with infarction of the thalamus. Involvement of the anterior hippocampus and amygdala via infarction in the anterior choroidal territory has been rarely reported as a cause of amnesia. A summary of reported cases, including the present series, is given in Table 2.

Future reports may define other unilateral anatomic lesions that produce amnesic stroke. Bilateral infarction of the basal forebrain secondary to anterior communicating artery rupture or repair is well known to produce amnesia.⁶⁶ Patients with unilateral anterior communicating artery territory basal forebrain lesions visualized on CT and prominent amnesia have been reported^{67,68}; however, skull base artifact, surgical clip artifact, and small lesion size hamper CT imaging of these infarcts. Convincing evidence of memory impairment related to unilateral basal forebrain ischemia awaits reports with MRI and, especially, postmortem documentation. A case has been reported of a patient with amnesia in association with hemorrhage in the retrosplenial cortex and cingulate bundle⁶⁹; however, involvement of adjacent contralateral structures from midline hemorrhage is difficult to exclude. Possibly, a more common cause of unilateral amnesic stroke is infarction of the genu of the internal capsule and its neighboring structures, as reported recently by Tatemichi and colleagues.²⁴ Three of

their patients developed confusion and memory loss following solitary infarcts involving the inferior capsular genu; in one, the lesion was unambiguously confined solely to the genu. Thalamocortical disconnection was the postulated mechanism of amnesia. The inferior capsular genu is supplied variably by penetrating branches of the internal carotid, anterior cerebral, and, less frequently, anterior choroidal arteries.

Reports in the literature as well as our cases reveal that amnesia more commonly is recognized in relation to left-sided stroke if the stroke is unilateral, representing 85% of the cases we reviewed. Several selection biases influence this laterality predominance. First, physicians generally rely on tests of verbal memory, which is less likely to be impaired in amnesia related to right hemisphere lesions. Second, patients with right hemisphere lesions may have anosognosia for their memory deficit contributing to underrecognition of amnesia. Third, patients with right hemisphere lesions may compensate for visual memory deficits through the use of verbal mnemonic cues. Last, some amnesic patients have been identified in the course of studies of thalamic aphasia or PCA-related pure alexia, favoring detection of left-sided lesions.

In several reported cases of both thalamic and mesial temporal unilateral infarction, material-specific effects are evident, with left-sided lesions having a greater impact on verbal memory^{13,21,31,39,44,46} and right-sided lesions having a greater impact on visual memory.^{34,36,40,45,51} However, the frequency with which left-sided lesions have been reported to produce deficits simultaneously affecting verbal, nonverbal, and autobiographical memory suggests that left amygdalohippocampal or diencephalic dysfunction may produce a particular vulnerability to “global” amnesia.^{17,32,35,36,38,48-50} Consonant with these observations in patients with observable infarctions is the finding that in the postacute epoch, patients with “pure” TGA without identifiable lesions exhibit a mild residual verbal, but not nonverbal, memory deficit.⁶⁴

The reason for variation in degree of recovery from amnesia after stroke is unknown. In cases of patients who experienced persistent amnesia after unilateral temporal lobectomy, it has been suggested that amnesia results from the presence of subclinical disease such as mesial temporal sclerosis in the contralateral hippocampus. We cannot rule out a similar mechanism for persistent amnesia in our cases even though contralateral lesions were not seen on high-resolution MRI images in three patients. The prognosis for recovery of memory function in amnesic stroke usually is good. The size of the lesion does not appear to predict the degree of recovery.

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