Acute Caudate Vascular Lesions
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Background and Purpose—We sought to evaluate demographic features, risk factors, clinical profiles, and behavioral abnormalities in patients with caudate lesion, either with infarct or with hemorrhage involving the caudate nucleus.

Methods—We studied all patients with acute caudate stroke confirmed by CT or MRI who were admitted to our stroke unit over a 5-year period. A database containing risk factors, clinical features, type and mechanism of stroke, and caudate vascular territories was analyzed.

Results—Thirty-one patients had acute caudate stroke (24 men and 7 women; mean age, 62.3 years). Caudate infarct was present in 25 patients and caudate hemorrhage in 6. The main risk factors for caudate infarct were hypertension (64%), hypercholesterolemia (32%), diabetes mellitus (28%), and previous myocardial infarct (20%). Hypertension was present in 4 patients (67%) with caudate hemorrhage, and arteriovenous malformation was present in 1 patient (17%). Small-artery disease was diagnosed in 14 patients (59%), cardiac embolism in 5 patients (20%), and large-artery disease in 2 patients (8%), and 2 patients (8%) had mixed etiology. The most frequent neurological abnormalities were abulia and psychic akinesia (48%), frontal system abnormalities (26%), speech deficits in patients with left-sided lesions (23%), and neglect syndromes in those with right-sided lesions (10%). Fifteen patients with caudate infarct (60%) and 3 patients with hemorrhage (50%) were able to return to normal daily life. Patients with infarct in the territory of the lateral lenticulostriate arteries extending to neighboring structures showed more frequent motor and neuropsychological deficits than those with infarct in the territory of the anterior lenticulostriate arteries.

Conclusions—The clinical presentation of patients with caudate hemorrhage mimicked subarachnoid hemorrhage with or without motor and neuropsychological signs. Caudate vascular lesions with concomitant neighboring structure involvement represent a specific stroke syndrome, usually caused by small-artery disease and in one fifth of the patients caused by cardiac embolism. The behavioral abnormalities were mostly due to medial, lateral, and ventral caudate subnuclei damage and coexisting lesion of the anterior limb of the internal capsule. (Stroke. 1999;30:100-108.)

Key Words: amnesia ■ aphasia ■ caudate nucleus ■ hemorrhagic stroke ■ stroke, ischemic

It is documented that the caudate nucleus is involved in degenerative diseases of the central nervous system such as Parkinson disease and Huntington disease. Brain imaging has led many investigators to study clinical, cognitive, and behavioral abnormalities of caudate vascular lesions. Earlier studies that included caudate lesions involving neighboring anatomic structures, such as the putamen, internal capsule, and white matter, did not elucidate the clinical functions of the caudate nucleus head.1–13 Studies on vascular lesions (either infarct or hemorrhage) of the caudate nucleus are few. We studied a series of patients with caudate infarcts or hemorrhages involving the head of the caudate nucleus (confirmed by CT and MRI), stroke etiology, clinical profiles, and behavioral abnormalities.

Subjects and Methods
We evaluated all patients with a diagnosis of caudate stroke admitted to Ege University Hospital Stroke Unit, Izmir, Turkey, over a 5-year period. Patients with caudate stroke were 1% of our registry, which included 3050 patients (total, 2450 ischemic stroke and 600 hemorrhagic stroke). CT and MRI were performed in all patients with caudate ischemic stroke and read by investigators blinded to neurological and neuropsychological findings in each patient. In all patients with caudate hemorrhage, CT was performed (1 to 3 examinations) with or without contrast; in those with caudate infarct, MRI with or without gadolinium contrast was also performed to delineate lesion contour. The topography of the head of caudate subnuclei included the lateral caudate nucleus (LCN), medial caudate nucleus (MCN), ventral caudate nucleus (VCN), and caudate fundus region, which were assessed by radiological investigations following previously published templates14 (Figure 1). Three major vascular areas that supply the head of the caudate nucleus were included, according to the templates of Ghika et al15: (1) Heubner's artery, a direct penetrating artery originating from the anterior cerebral artery and supplying the inferior part of the head of the caudate nucleus and the anterior limb of the internal capsule; (2) anterior lenticulostriate arteries originating from the proximal part of the anterior cerebral artery and supplying the anterior area of the head of the caudate nucleus; and (3) lateral lenticulostriate arteries originating from the middle cerebral artery and supplying a major part of the head of the caudate nucleus, anterior internal capsule, and putamen. A large caudate infarct was one >1.5 cm on >2 slices on CT and MRI.
In addition to CT and MRI, other examinations performed were complete blood cell count and urinalysis, transcranial Doppler, duplex sonography of the carotid and vertebral arteries, 12-lead ECG, and, in some cases, transesophageal echocardiography and catheter angiography. We recorded risk factors for caudate stroke such as hypertension (blood pressure $160/90$ mm Hg at least twice before stroke), diabetes mellitus (fasting blood glucose concentrations $>6.0$ mmol/L known before stroke), regular smoking, hypercholesterolemia (fasting blood cholesterol $>6.5$ mmol/L), venous hematocrit at admission, history of migraine, and heart disease (eg, old myocardial infarct, left ventricular aneurysm, hypokinesia or akinesia, chronic nonvalvular atrial fibrillation [NVAF], mitral stenosis).

We considered the following as potential causes of caudate ischemic stroke: (1) large-artery disease was presumed in patients who had a stenosis of $\geq 50\%$ of the lumen diameter in the appropriate large artery as shown on duplex, transcranial Doppler, or MR angiography; (2) small-artery disease in patients with longstanding hypertension or diabetes mellitus and a small ($\leq 15$ mm) infarct limited to the territory of deep perforators on CT scan or MRI in the absence of other etiologies; (3) potential cardiac sources of embolism including NVAF, left ventricular dyskinetic segment, intracardiac thrombus or tumor, mitral stenosis, and other less common sources; (4) mixed etiology in cases of coexistence of large-artery disease and potential cardiac sources of embolism; (5) other and undetermined etiologies.

The neuropsychological findings were evaluated by measuring cognitive, linguistic, spatial, and mnemonic functions in our neuropsychology laboratory within the first week of the stroke. The instruments included the Mini-Mental State Examination (Turkish version) (MMSE), with a total maximum score of 50 points; a score of $\leq 28$ indicates significant cognitive impairment. Language function was assessed by the Turkish Aphasia Test, measuring speech fluency, repetition, comprehension, writing, and reading functions. Sensory extinction was tested by bilateral stimulation in the tactile, visual, and auditory modalities. Visual perception, combining perceptual-sensory and motor aspects, was evaluated by the line cancellation test (maximum score of 40 points) and by the line bisection test, with a set of 10 lines of the same size but arranged randomly. Motor-exploratory neglect was assessed by blindfolded manual exploration described by Weintraub and Mesulam, which measures the exploration of ipsilesional and contralesional space by the nonparetic right hand. Verbal amnesia was assessed by the Rey Auditory Verbal Learning Test (trial 1, with a maximum score of 15 points; a score of $\leq 9$ indicates verbal amnesia). Visual amnesia was considered present when there was a deficit in recognizing figures presented visually as evaluated by F form of the Benton Visual Retention Test (total maximum score of 15 points; a score of $\leq 9$ indicates visual amnesia). Frontal lobe functions were assessed by the Stroop test and Luria’s conflicting tasks. Major depression was defined with the use of the symptom criteria of the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV). Among behavioral abnormalities, abulia was defined in patients with decreased spontaneous activity, prolonged latency in responding to questions, fatigue, and an aversion to any activity. Psychic akinesia (or loss of psychic self-activation) was defined in patients who have severe impairment of mental and psychic activity and loss of affective and motor response to external stimuli. The clinical course of all the patients was recorded in the acute phase (between first week and second week of onset) and chronic phase (between 6 months and 1 year).

We considered the following as functional outcome measures for patients: (1) independent patients with minor neurological deficit such as dysarthria and minimal hand or leg weakness that does not prevent performance of normal activities; (2) mildly dependent patients with motor deficit that limits previous activities and who need some help for walking; (3) dependent patients with major neurological deficits such as being bedridden and incontinent and who require constant nursing and attention and could not return to any previous activities; and (4) death.

Statistical data on risk factors and stroke mechanism were analyzed with $\chi^2$ and Fisher’s exact tests. Because of the small number of cases involved, the data on clinical features according to the nuclei topography were analyzed with descriptive statistics.

**Results**

There were 31 patients with caudate stroke (24 men and 7 women; mean age, 62±10 years; range, 41 to 78 years). Caudate infarct occurred in 25 patients (80%) and caudate hemorrhage in 6 patients (20%) (3 patients with left and 3 with right caudate hemorrhage). The mean age of patients with caudate infarct was 62±9 years (range, 42 to 78 years), and that for those with caudate hemorrhage was 61±13 years (range, 41 to 78 years). Eleven patients (44%) had left and right caudate infarct each, and bilateral caudate infarcts were present in 3 patients (12%). Eight patients (33%) had large infarct of the caudate nucleus involving neighboring structure (Figure 2).

**Risk Factors**

In 25 patients with caudate infarct, risk factors were hypertension (64%), hypercholesterolemia (32%), diabetes mellitus...
(28%), previous myocardial infarct (20%), cigarette smoking (16%), and family history of ischemic stroke in (8%) (Table 1). Three patients (12%) had NVAF, and cardiac mural dyskinesia was present in 5 patients (20%), including 1 with a cardiac aneurysm and 1 with a mural thrombus detected by transesophageal echocardiography. Additionally, 1 of the 25 patients with ischemic caudate stroke (patient 5) had syphilis proved by serological test, and another patient (patient 4) had a history of Hodgkin’s lymphoma. There was a history of transient ischemic attack in 13 patients (56%) with anterior circulation symptoms. The caudate ischemic stroke had a nonprogressive onset of symptoms (stabilized, 1 hour) in 20 patients (80%) and was progressive (over 2 to 24 hours) in 5 patients (20%). The symptoms of caudate hemorrhagic stroke stabilized in 1 hour in all cases. Two patients (8%) with left caudate infarct had generalized convulsion at stroke onset.

Presumed Cause of Infarct
Small-artery disease was diagnosed in 14 patients (59%), 10 of whom were hypertensive (3 had only hypertension; in the other 7, it was associated with other risk factors), 4 of whom had diabetes mellitus (3 had diabetes mellitus alone). Five patients (20%) had potential cardiac sources of embolism (5 patients had cardiac dyskinesia and 1 had mural thrombosis proved by echocardiography). Ipsilateral internal carotid artery disease (>50% stenosis or occlusion, large-artery disease) was the only potential cause of infarct in 2 patients (8%), while 2 patients had mixed etiology (1 had large-artery disease and a cardiac source of embolism [cardiac hypokinesia], and the other had large-artery disease and NVAF).

Topography of Infarcts
We identified 3 principal clinicotopographic patterns of caudate infarct: (1) left caudate infarcts; (2) right caudate infarcts; and (3) bilateral caudate infarcts (Figure 2).

Left Caudate Infarcts
Left caudate infarcts were present in 11 patients (44%). Four patients had isolated caudate involvement (patients 1, 4, 7,
and 11) with no motor and sensory deficit. Two patients had facial weakness (patients 1 and 4) during the first day of stroke onset. Isolated left caudate infarcts were associated with cognitive dysfunction and minimal motor speech disorder. In patients with extension of the lesion to the putamen (patients 2, 6, and 10), behavioral abnormalities such as abulia, confusion, and motor abnormalities occurred. Patients with large caudate involvement, which showed deep extension (patients 3, 5, 8, and 9), had prominent motor and various neuropsychological abnormalities. Nonfluent aphasia with repetition abnormality (patients 3 and 9), transcortical motor aphasia (patient 8), and global aphasia (patient 5) occurred in patients with large caudate and deep infarct. Verbal amnesia (patients 2 and 3), visual and verbal amnesia (patient 10), anomia (patients 6, 8, and 9), and ideomotor and buccolingual apraxia (patient 5) were other characteristics of left caudate infarcts (Table 2).

Six patients had infarct in the territory of the lateral lenticulostriate arteries, 4 patients had infarct in the territory of the anterior lenticulostriate arteries, and 1 had Heubner’s artery branch occlusion. Patients with infarct in the territory of the anterior lenticulostriate arteries had mild motor and neuropsychological deficits such as confusion and disorientation. Those with infarct in the territory of the lateral lenticulostriate arteries had prominent motor and neuropsychological findings such as linguistic abnormalities, frontal system dysfunction, and amnestic deficits.

**Right Caudate Infarcts**

Right caudate infarcts were present in 11 patients (44%). Three patients with right caudate infarct (patients 16, 19, and 22) had acute confusion, and 2 (patients 14 and 17) had only abulia at stroke onset. Patients with involvement of the anterior limb of the internal capsule (patients 12, 13, 19, and 20) had predominantly faciobrachial paresis, and 1 patient (patient 13) had decreased spontaneity and speech. Four patients with large infarct of the caudate nucleus (patients 14, 15, 18, and 21) had prominent faciobrachiocrural paresis, predominantly in the upper limbs in 3 patients, and behavioral abnormalities. One (patient 21) had severe impairment of motor and mental activity with flat affect and could not move unless asked to eat or to stand up.

Six patients had infarct in the territory of the lateral lenticulostriate arteries (Figure 3 top), and 5 had infarct in the territory of the anterior lenticulostriate arteries (Figure 3 middle). Patients with infarct in the territory of the right anterior lenticulostriate arteries had only neuropsychological symptoms such as confusion and abulia. Those with infarct in the territory of the lateral lenticulostriate arteries had prominent motor deficits, frontal system dysfunction, neglect, and visual amnesia.

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**TABLE 1. Demographic Data, Risk Factors, and Cause of Infarct in Patients With Caudate Infarct**

<table>
<thead>
<tr>
<th>Unilateral Caudate Infarcts*</th>
<th>Bilateral Caudate Infarcts</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
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</tr>
<tr>
<td>Age (mean±SD), y</td>
<td>58±9/66±6</td>
</tr>
<tr>
<td>Sex (M/F)</td>
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</tr>
<tr>
<td>Risk factors</td>
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</tr>
<tr>
<td>Hypertension</td>
<td>6/8</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>5/2</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>3/4</td>
</tr>
<tr>
<td>Smoking</td>
<td>2/1</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>1/2</td>
</tr>
<tr>
<td>Myocardial infarct</td>
<td>3/0</td>
</tr>
<tr>
<td>Familial history of stroke</td>
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</tr>
<tr>
<td>Transient ischemic attack</td>
<td>7/5</td>
</tr>
<tr>
<td>Presumed cause</td>
<td></td>
</tr>
<tr>
<td>Large-artery disease</td>
<td>1/1</td>
</tr>
<tr>
<td>Small-artery disease</td>
<td>6/8</td>
</tr>
<tr>
<td>Cardioembolism</td>
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</tr>
<tr>
<td>Mixed†</td>
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</tr>
<tr>
<td>Syphilis</td>
<td>1/0</td>
</tr>
<tr>
<td>Unknown</td>
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</tbody>
</table>

*Values (unless indicated otherwise) are left/right. †Large-artery disease plus cardioembolism.

**TABLE 2. Clinical and Behavioral Findings and Prognosis of Patients With Caudate Infarct**

<table>
<thead>
<tr>
<th>Unilateral Caudate Infarcts*</th>
<th>Bilateral Caudate Infarcts</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>11/11</td>
</tr>
<tr>
<td>Dysarthria</td>
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<tr>
<td>Convulsion</td>
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<tr>
<td>Motor</td>
<td></td>
</tr>
<tr>
<td>Face</td>
<td>2/0</td>
</tr>
<tr>
<td>Face, upper limb</td>
<td>2/3</td>
</tr>
<tr>
<td>Face, upper limb&gt;lower limb</td>
<td>5/4</td>
</tr>
<tr>
<td>Chorea</td>
<td>0/1</td>
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<tr>
<td>Behavioral findings</td>
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<tr>
<td>Confusion-disorientation</td>
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</tr>
<tr>
<td>Abulia</td>
<td>4/5</td>
</tr>
<tr>
<td>Psychic akinesia</td>
<td>0/1</td>
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<tr>
<td>Restlessness</td>
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<tr>
<td>Nonfluent aphasia</td>
<td>3/0</td>
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<tr>
<td>Transcortical motor aphasia</td>
<td>1/0</td>
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<tr>
<td>Global aphasia</td>
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</tr>
<tr>
<td>Motor neglect</td>
<td>0/3</td>
</tr>
<tr>
<td>Visuospatial neglect</td>
<td>0/2</td>
</tr>
<tr>
<td>Impaired conflictual tasks</td>
<td>3/2</td>
</tr>
<tr>
<td>Verbal amnesia</td>
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<tr>
<td>Visual amnesia</td>
<td>0/4</td>
</tr>
<tr>
<td>Visual-verbal amnesia</td>
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</tr>
<tr>
<td>MMSE ≥28</td>
<td>4/3</td>
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<tr>
<td>Depression</td>
<td>2/1</td>
</tr>
<tr>
<td>Prognosis</td>
<td></td>
</tr>
<tr>
<td>Independent</td>
<td>6/8</td>
</tr>
<tr>
<td>Mildly dependent</td>
<td>3/1</td>
</tr>
<tr>
<td>Dependent</td>
<td>1/2</td>
</tr>
<tr>
<td>Death</td>
<td>1/0</td>
</tr>
</tbody>
</table>

*Values are left/right.
Bilateral Caudate Infarcts

Bilateral caudate infarcts were present in 3 patients (12%). Three patients with bilateral involvement of the caudate head had acute confusion and disorientation at stroke onset. Two patients (patients 24 and 25) had unilateral faciobrachiocural paresis, predominantly in the upper limb on the side contralateral to a lesion that involved the anterior limb of the internal capsule, and psychic akinesia.

Two patients had bilateral infarcts in the territory of the anterior lenticulostriate arteries, and 1 had infarcts in the territory of the anterior and lateral lenticulostriate arteries.

Caudate Hemorrhages

Of the 6 patients with caudate hemorrhage (Table 3), hypertension was present in 4 patients (67%) and arteriovenous malformation in 1 patient (17%), while no cause of hemorrhage was determined in 1 patient (17%). Three of the patients (50%) (patients 27, 28, and 31) presented with an altered level of consciousness (confusion-disorientation). In the days after the stroke, 3 patients (patients 27, 29, and 30) had abulia (Figure 3 bottom). Headache occurred in all patients with caudate hemorrhage, and nuchal rigidity occurred in 67%. Motor deficit occurred in 3 patients (50%) along with conjugated eye deviation in 34% (patients 26 and 28), dysarthria (patient 28), and disconjugated eye deviation in 17% (patient 30).

Neuropsychological Abnormalities and Caudate Nuclei

Behavioral Findings

There were 12 abulic patients, 5 patients with right-sided caudate infarct, 3 with left-sided infarct, and 1 with bilateral caudate infarct; 3 patients with isolated caudate hemorrhage developed abulia (2 with left-sided, 1 with right-sided lesion). Three patients with caudate infarct had psychic akinesia (1 with right-sided lesion and 2 with bilateral lesion). Four patients had restlessness (3 with right-sided lesion, 1 with left-sided lesion) in the acute phase of stroke, and their lesions were confined to the head of the caudate nucleus.

Four abulic patients had isolated caudate lesion (patients 10, 17, 27, and 29), 4 had an extension of lesion to the capsule (patients 6, 13, 23, and 30), and 4 had a lesion that spread to the anterior putamen or to other neighboring structures in a deeper direction (patients 2, 14, 15, and 18). Three patients with psychic akinesia had right-sided damage of the internal capsule with MCN or LCN territory infarct (patients 21, 24, and 25), and 2 patients had additional involvement of the MCN, LCN, and caudate fundus region on the left side (patients 24 and 25).

On follow-up 3 months later, 5 patients (2 with right-sided, 2 with left-sided, 1 with bilateral lesions) developed major depression according to the criteria of DSM-IV (patients 8, 9, 21, 24, and 26). These patients had large lesions extending to deeper structures such as the nucleus accumbens and laterally to the anterior limb of the capsule.

Dysarthria

Thirteen patients had dysarthria; 10 patients had unilateral caudate infarct (5/11, right side; 5/11, left side), 2 patients had
bilateral caudate infarct (2/3), and 1 patient had left caudate hemorrhage (1/6). Among dysarthric patients, infarct was limited to the caudate nucleus in 2 (patients 11 and 17), 8 patients had involvement of the anterior limb of the internal capsule, and 2 had anterior putamen involvement. In all cases, MCN and LCN were affected, and in 5 patients only the VCN was involved.

**Language Findings**

Five patients with left-sided caudate infarcts had speech abnormalities. Among them, 3 patients developed a nonfluent type of aphasia characterized by nonfluent speech with syntax errors, repetition impairment, stuttering, word-finding difficulty, and preserved comprehension (patients 3, 6, and 9). These patients had large lesions extending to the anterior limb of the capsule. Patient 8 had only mild speech fluency deficit, agaphia with perseveration, but good repetition and comprehension (transcortical motor type of aphasia). Another patient (patient 5) with left caudate infarct developed global aphasia with impairment of speech, repetition, and comprehension. Single-photon emission CT imaging revealed hyperperfusion in the left frontal and temporoparietal lobes. One patient with bilateral caudate infarcts (patient 24) and 1 patient with left caudate hemorrhage (patient 28) presented a nonfluent type of aphasia. Aphasia resolved in 2 weeks in all patients except 1 with global aphasia.

**Neglect**

Three patients had motor-exploratory neglect (patients 18, 20, and 21), with significantly slower right hand movement during exploration of the left hemispace when compared with the right hemispace. Line cancellation and line bisection tests, which determined visuospatial function, were abnormal in 3 patients (patients 18, 21, and 26). None of the patients had extinction in tactile, visual, and auditory modalities. All patients with hemineglect had involvement of MCN, LCN, VCN, and neighboring structures.

**Other**

Twelve patients had a score of ≤28 on the MMSE (7 patients with left-sided, 3 with right-sided, and 2 with bilateral lesions). One patient with right caudate infarct had motor dysprosody, motor impersistence in eye-closing, and held-up hands for 20 seconds (patient 18), and another patient had inadequate laughing at stroke onset (patient 13). Eight patients had frontal test impairment demonstrated by the Stroop test and Luria’s conflicting tasks (patients 5, 8, 9, 18, 21, 24, 25, and 29).

**Follow-Up**

Nineteen patients received only antiaggregant therapy, and 6 patients underwent anticoagulation with warfarin therapy. Only 1 patient with caudate infarct worsened clinically after 2 months and died of congestive heart failure. Another patient with caudate hemorrhage died of generalized cerebral vasospasm shown by transcranial Doppler. This patient had no abnormality on cerebral angiography. Fifteen patients (60%) with caudate infarct and 3 patients with hemorrhage (50%) were independent. Four patients with unilateral caudate infarct (16%) (3 in the territory of lateral lenticulostriate arteries; 1 in the territory of anterior lenticulostriate arteries) were mildly dependent. Two patients with bilateral caudate infarcts, 3 patients with unilateral large caudate infarct in the territory of the lateral lenticulostriate arteries (20%), and 2 patients with caudate hemorrhage (33%) were dependent 1 year after stroke.

**Discussion**

Study of caudate strokes is useful for understanding the functions of the caudate nucleus and neighboring structures. Therefore, we investigated the clinical and behavioral aspects...
of the patients with caudate lesions due to infarct or hemorrhage. Risk factors, age, sex, and patient profiles of caudate ischemic and hemorrhagic stroke were similar to those of patients with small-artery disease and primary intracerebral hemorrhage from our registry. The rate of the localization of lesions was equal on both sides, and the majority of caudate infarcts were restricted to the head of the caudate nucleus without involving neighboring structures.

Our findings suggest that small-artery disease and a cardiac source of embolism are the main causes of caudate ischemic stroke; large-artery disease was present in only one sixth of the patients. In previous studies, small-artery disease occurred in two thirds, while a cardiac source of embolism was present in one fifth of the patients with unilateral caudate infarct. In our series, acute bilateral caudate infarcts developed primarily because of a cardiac source of embolism.

The most commonly involved arterial territories in caudate ischemic stroke were the territory of the lateral lenticulostriate arteries from the middle cerebral artery and the territory of the anterior lenticulostriate arteries from the anterior cerebral artery. In previous studies, there is no mention of arterial territories involved in patients with caudate infarcts. Actually, there is considerable overlap between the 3 arteries supplying the head of the caudate nucleus: the lateral lenticulostriate, anterior lenticulostriate, and Heubner’s recurrent artery. In previous studies, there is no mention of arterial territories involved in patients with caudate infarcts. In our series, acute bilateral caudate infarcts developed primarily because of a cardiac source of embolism.

Motor abnormalities occurred in two thirds of the patients, primarily in patients with lesion extending to the anterior limb of the capsule. The characteristics of motor deficit were facial and upper extremity weakness with increased deep tendon reflexes, clumsiness, and extensor plantar response in half of the patients. This type of motor deficit was termed “nonpyramidal hemimotor syndrome” by Caplan et al to distinguish it from pyramidal pathway lesions that originated in the precentral area. Hemichorea without motor weakness occurred in only 1 patient with caudate ischemic stroke extending to the anterior putamen. In the previous case reports, hemichorea has also been reported in ischemic and hemorrhagic caudate strokes in which the lesions extended into the anterior limb and putamen. Moreover, hemibalismus and compulsive movement disorder due to caudate and concomitant striatal lesions were described. In our series, the most prominent clinical features of caudate vascular lesions were behavioral and cognitive abnormalities, as in previous studies. Behavioral changes may have occurred as a result of loss of function in cortical zones, caused by loss of striatal efferent projections from the caudate nucleus. The caudate nucleus is the principal crossing area of basal ganglia–thalamocortical loops. As defined by different authors, the caudate nucleus connects associative cortex, including frontal, parietal, and temporal lobes, with deeper anatomic structures by cortico-pallido-nigra-thalamocortical loops. These loops are multiple, discrete, but partially overlapping and are integrated through their passage in pallidum and substantia nigra to the circumscribed nuclei of the thalamus, and from there they are projected back to their original lobar areas.

One half of the patients had abulia, characterized by decreased spontaneous activity and speech and prolonged latency in responding to questions and other stimuli. Three patients had psychic akinesia, characterized by severe mental and affective stagnation and lack of initiative for action and speech. Trillet et al observed psychic akinesia in 3 patients with apathy, flattened affect, lack of initiative for usual daily activities, stereotyped behaviors, and prolonged akinetiform attacks. Moreover, these features were previously reported in patients with bilateral globus pallidus or putaminal lesions. The abulic patients described by Fisher had lesions in the frontal lobes and underlying structures or in the thalamus and upper brain stem. Caplan et al described 10 abulic patients with left-sided preponderance of caudate lesions. Among them, 4 patients had isolated caudate lesions, others had involvement of the anterior limb, and only 1 had spread to the putamen. The mechanism of abulia was explained by interruption of the limbofrontal connection. The quantitative and temporal features of behavior were more difficult to measure, and a variety of descriptive terms have been used such as bradykinesia, abulia, psychic akinesia, and akinetic mutism. These terms describe a continuum from minor to major absence of observable behavior, and despite the advanced behavioral stage, some intellectual and cognitive functions could be retained.

Restlessness, disinhibition, and confusion occurred in 4 patients with lesions circumscribed to the caudate nucleus. Agitation, anxiety, and talkativeness were common signs in other series of patients with caudate lesions. It is well known that behavioral hyperactivity with restlessness, excitement, agitation, and shouting may occur because of lesions of the middle or posteroinferior temporal lobe and parieto-occipital lobes infarctions. Mendez et al reported that dorsolateral caudate involvement may cause decreased spontaneous verbal and motor activities, and small ventromedial lesions may result in disinhibited, inappropriate, and impulsive behaviors. In our series, patients with abulia had larger lesions than those with restlessness; it is difficult to conclude which caudate nuclei are responsible from increased or decreased activity.

Dysarthria was a common sign in patients with caudate vascular lesions, as described previously. We found dysarthria without a side predominance in one third of our patients with caudate stroke. Dysarthria can be explained by interruption of the corticolinguinal pathways to the tongue or corticostriotocerebellar loops, which play crucial roles in uniform speech patterns.

In the present study approximately one half of the patients with a left caudate lesion had minor and transient linguistic
deficits; only 1 patient with syphilitic vasculitis had global-type aphasia 6 months after stroke onset. Global-type aphasia in this patient may be explained by intrahemispheric disconnection. It is well known that different types of aphasia, such as transcortical, nonfluent aphasia, characterized by semantic and verbal paraphasias and perseverations without comprehension impairment, occur in patients with left caudate vascular lesions.1–13,35,36 In the clinical literature, global aphasia was reported in only 1 patient with hemorrhage limited to the left caudate nucleus.12 Alexander et al3 observed that patients with subcortical lesions involving the caudate nucleus, anterior limb of the internal capsule, and putamen had word-finding difficulty or hesitancy without severe aphasias. It is probable that acute disconnection of linguistic pathways between anterior and posterior speech areas, which are connected with the left caudate nucleus, and anterior limb of the internal capsule may yield a different type of aphasia.

Motor and visuospatial neglect were present in one fourth of the patients with right caudate vascular lesion, especially in those with a large lesion involving the internal capsule. Hemineglect was occasionally reported in patients with right caudate lesions and has not been systematically studied in either infarct or hemorrhage. In the previous series, contralateral hemineglect was reported in 3 cases with right caudate lesion involving the anterior limb of the internal capsule.6 Moreover, the putamen and adjacent white matter lesions are also involved in the development of hemineglect, but in the previous series, the lesions were all large and involved neighboring structures.37 In our study we did not find pure perceptual-sensory neglect, which was characterized only by tactile, visual, and auditory extinction. Motor-exploratory hemineglect may be explained by the disruption of the circuitry that relates the frontal lobe and its associative cortical regions to striatum and substantia nigra.

Another result of caudate vascular lesion was depression in one fourth of the patients, which suggests that the caudate nucleus may play a role in the regulation of human mood. In a previous study, depression was observed in one third of the caudate lesion patients without cognitive deterioration.38 Moreover, we observed that one third of the patients with left caudate lesion had verbal amnesia, while patients with right caudate lesion had visual amnesia, suggesting the role of the caudate nucleus in the integration of visual and verbal memories. In a previous study, verbal comprehension and verbal memory deficits of the patient with caudate hemorrhage have been explained by dysfunction of the corticocaudate connections.12 Mendez et al5 showed that bilateral caudate lesions might yield global dementia, while unilateral lesions might cause impairment of frontal functions and decreased free recall of episodic and semantic items. In our series, one fourth of the patients, especially those with a large caudate lesion, had deficits in tasks requiring planning and sequencing. This may be due to the disconnection of the caudate nucleus from the frontal lobe.

The clinical presentation of 1 patient with hemorrhage localized in the caudate nucleus mimicked subarachnoid hemorrhage, while 2 other patients developed only neuropsychological deficits, abulia, and frontal dysfunction. The other 3 patients with involvement of the internal capsule also had motor and neuropsychological deficits. In recent studies, these clinical patterns of caudate hemorrhage, including subarachnoid hemorrhage without focal sign, subarachnoid hemorrhage with hemiparesis or neurological deficit, and isolated neuropsychological deficits, were described clearly by different authors.7–13 Two thirds of the patients with caudate hemorrhage had hypertension, while arteriovenous malformation was present in only 1 patient. This result was similar to the findings of other series, in which aneurysms, moyamoya and moyamoya-like atherosclerotic abnormalities, and occlusive intracranial carotid artery disease were reported to cause caudate hemorrhage.39–44

Finally, we attempted to delineate the clinical presentation of the caudate nucleus during the acute phase of stroke in this study, although most of the patients with neuropsychological signs had involvement of multiple subnuclei and neighboring structures. Further studies are required to explain the different neuropsychological findings according to caudate subnuclei. Our study suggests that most of the behavioral abnormalities and dysarthria may be explained by involvement of the medial, lateral, and ventral caudate subnuclei and the anterior limb of the internal capsule.

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References

37. Damasio AR, Damasio H, Chui HC. Neglect following damage to frontal lobe or basal ganglia. Neuropsychologia. 1980;18:123–132.
Acute Caudate Vascular Lesions
Emre Kumral, Dilek Evyapan and Kaan Balkir

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