Acute Infarction Limited to the Lenticular Nucleus

Clinical, Etiologic, and Topographic Features

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Background: Chronic diseases involving the putamen and globus pallidus induce parkinsonism and other movement disorders. Sensory and motor dysfunction from deep middle cerebral artery infarction is usually due to an involvement of the internal capsule. The clinical picture associated with isolated infarction of the lenticular nucleus is less well established.

Objective: To analyze clinical features, topographic correlations, and cause of purely lenticular ischemic infarction.

Patients and Methods: We reviewed 820 consecutive patients with deep hemispheric infarct included in the Lausanne Stroke Registry between 1986 and 1998 and selected those with isolated lenticular involvement on computed tomography or magnetic resonance imaging.

Results: Thirteen patients had pure lenticular infarction. All had faciobrachiocrural hemisyndrome, while none showed acute or delayed parkinsonism or abnormal movement. Nine patients had a lesion restricted to the putamen. Two of them had ataxic motor hemisyndrome and 7 had sensorimotor hemisyndrome (with ataxia in 4, left hemineglect in 1, and deep pain in the arm and leg in 1). Four patients had a lesion of putamen and globus pallidus externus. Three of them had motor hemisyndrome (with nonfluent aphasia in 2 and ataxia in 1) and 1 had ataxic sensorimotor hemisyndrome. All infarcts were in the territory of the medial perforating branches of the medial cerebral artery. Presumed cause of stroke was small-artery disease in 5, artery-to-artery embolism in 4, cardioembolism in 3 and undetermined in 1.

Conclusions: Acute lenticular infarction induces mainly hemiparesis but no movement disorder. Associated sensory deficits, aphasia, and hemineglect underline clinically the function of the lenticular nucleus in connection with the prefrontal, temporal, and parietal cortices.

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DEEP MIDDLE cerebral artery infarction is common and its neurologic picture is dominated by the consequences of internal capsule involvement. Although pure internal capsule infarction has been well documented, 1-4 infarction limited to the putamen–globus pallidus is poorly recognized.

Two clinical syndromes associated with lesions limited to the lenticular nucleus have been defined by Giroud et al: 1) behavioral and cognitive disorders associated with infarcts within the globus pallidus and 2) motor disorders (dystonia) and cognitive disorders associated with disorders in the putamen. Bhatia and Marsden 6 reviewed the behavioral and motor consequences of focal lesions of the basal ganglia in 240 patients described in the literature with lesions of the lenticular and caudate nucleus and found that dystonia was the most frequent motor disorder and abulia, the most frequent behavioral disorder.

Similarly, lesions involving the lentiform nucleus, particularly the putamen, caudate nucleus, thalamus, and parietal cortex, often in combination, have been associated with acute and subacute posthemiplegic focal dystonia or hemidystonia. However, there was extension of the lesion into adjacent structures, including the internal capsule, in most of the cases.

Other motor disorders, such as unilateral chorea, 11 hemichorea-hemiballism, asterixis, acute stereotypies, 12 acute focal dystonia, 13 and subacute parkinsonism, 14 have also been reported after unilateral lesions of the lentiform nucleus but often also involve the caudate nucleus or the internal capsule.

Lesions involving the globus pallidus may cause behavioral and speech dis-
orders, as well as motor disorders including delayed contralateral hemidystonia or subacute choreoathetosis. In most cases, there is a delay of months to years between the onset of the actual lesion and the development of the motor disorders.

To investigate acute, purely lenticular ischemic infarction, we selected and reviewed all cases of patients with such lesions in the Lausanne Stroke Registry.

METHODS

PATIENTS AND SELECTION

Patients were selected from a collective of 3901 consecutive patients prospectively included in the Lausanne Stroke Registry between 1986 and 1998. Of 820 consecutive patients with a first-ever symptomatic subcortical hemispheric stroke, 135 had a lesion in the basal ganglia confirmed by computed tomography (CT) (93 [68.9%]) or magnetic resonance (MR) imaging (42 [31.1%]).

We excluded all patients with hemorrhagic infarcts (5 [3.7%]), involvement of the internal capsule (9 [6.7%]), or involvement of the adjacent structures (108 [80.0%]). Thirteen patients presented with infarcts involving the lenticular nucleus only; 12 of them had CT scans and 1 had MR imaging. These 13 patients form the basis for this study. In patients with CT scans, a recent appropriate lesion was diagnosed in 1 patient. Contrast CT scans showed a poorly defined lesion or no lesion, which subsequently evolved toward visible infarction consistent with clinical features on follow-up CT after 2 days.

All infarcts were in the territory of the perforating branches of the middle cerebral artery.

INVESTIGATIONS

The detailed investigations for patients included in the Lausanne Stroke Registry have been described elsewhere. In brief, all patients included in this study underwent a neurologic examination by at least 2 senior neurologists; high-resolution brain CT scanning (5-mm sections) or MR imaging with conventional axial T1- and T2-weighted spin-echo images (3-mm sections) within 24 hours after admission, and at least 1 other CT image during the patient’s hospital stay (infarct size defined as small for lesions <15 mm and large for lesions ≥15 mm); a detailed duplex neurosonographic examination; electrocardiography at admission; and 24-hour cardiac monitoring and heart echography. Catheter angiography was performed in selected cases. Blood tests consisted of blood cell count, hepatic and renal function tests, glucose level, total cholesterol level, sedimentation rate, VDRL, and tests of coagulation.

Patients were divided into 2 groups according to the distribution of infarcts on neuroimaging scans. One group consisted of patients with infarcts limited to the putamen, while the second group consisted of patients with infarcts extending into the globus pallidus externus.

The criteria for large-artery disease as the source of embolism were a stenosis of more than 50% in the appropriate large artery and ulcerated plaques on duplex examination or angiography. Diagnosis of middle cerebral artery stenosis or occlusion depended on acceleration of flow velocity and Doppler spectra.

The criteria for cardiac sources of embolism included atrial fibrillation, sick-sinus syndrome, or a recognized source on echocardiography (mural thrombus, wall akinesia or hypokinesia, endocarditis, aneurysm, prosthetic valves, or recent myocardial infarction). Small-artery disease was defined by hypertension (at least 2 blood pressure values >165/95 mm Hg before the stroke); diabetes mellitus (≥2 values of fasting blood glucose >108 mg/dL [>6 mmol/L]); absence of large-artery disease, cardioembolism, or another cause; and largest diameter of lacunar infarct less than 15 mm.

Hypertension, diabetes mellitus, regular cigarette use, and hypercholesterolemia (>2 blood values >251 mg/dL [>6.5 mmol/L]) were considered risk factors.

RESULTS

Of the 13 patients (8 men and 5 women between 30 and 86 years of age) with ischemic stroke limited to the lenticular nucleus, 9 patients had a lesion limited to the putamen and 4 patients had a lesion also involving the globus pallidus externus (Table). Six of the 13 patients had diabetes mellitus as a risk factor, 8 had arterial hypertension, 4 had hypercholesterolemia, 6 were smokers, and 3 had atrial fibrillation.

Ten patients had diabetes mellitus or hypertension or both; 5 of them had no other source of infarction and had a lacunar stroke due to presumed small-artery disease. Three patients had a cardioembolic source as the presumed cause of stroke and 4 had large-artery disease (>75% internal carotid artery stenosis in 1; ulcerated <50% internal carotid artery stenosis in 3). No patient had signs of middle cerebral artery stenosis or occlusion on transcranial Doppler ultrasonography, MR angiography, or conventional angiography. A young woman, who was a smoker and used oral contraception, had no evidence of a definite cause.

The ischemic lesion was 15 mm or larger in 5 patients with potential embolic sources (large-artery disease in 3, cardioembolic sources in 2). The lesions were smaller than 15 mm in 8 patients, of whom had small-artery disease as the presumed cause of infarct.

All patients had initial faciobrachiocephalocrural motor hemisyndrome, but none showed parkinsonism or any abnormal movement during hospital stay or follow-up.

Of the 9 patients with a lesion limited to the putamen, 2 had moderate proportional ataxic faciobrachiocephalocrural motor hemisyndrome, predominantly crural in 1. There were 3 patients with proportional faciobrachiocephalocrural motor deficit (moderate in 2 and severe in 1), with ataxia in the patient with a severe deficit and in 1 of those with a moderate motor hemisyndrome. All 3 patients had spinothalamic hemihypesthesia; it involved predominantly the upper limb in 1, associated with hemineglect, and the upper and lower limb in 2, associated with deep pain in the arm in 1 of the 2. Predominantly faciobrachial mild sensorimotor hemisyndrome with ataxia was present in 1 patient. Three patients had moderate ataxic faciobrachiocephalocrural motor hemisyndrome with ataxia predominating in the lower limbs, with spinothalamic sensory loss of the face in 2 and of the leg in another 1, associated with loss of postural sense and pallesthesia.

Four patients had a left-sided lesion involving the putamen and globus pallidus externus. Two right-handed patients had severe proportional faciobrachiocephalocrural motor hemisyndrome with nonfluent aphasia, and...
1 had moderate faciobrachialcrural motor hemisindrome with ataxia. One patient had a severe proportional faciobrachialcrural ataxic hemisindrome with unilateral spinothalamic sensory loss in arm and leg.

Follow-up with the help of the corresponding general practitioner was possible in 12 patients between 2 and 7 years (average ± SD, 4.08 ± 1.61 years) after the stroke. Ten patients recovered motor function well during the hospital stay, with complete recovery in 2 and persistence of discrete motor hemisindrome in 8 (with ataxia in 2). All 10 patients were independent at home for their daily activities after hospitalization. Two patients remained disabled by a predominantly brachial hemiparesis. Word-finding difficulties persisted in both patients with initial nonfluent aphasia due to left putaminal-pallidal infarction. Hemisensory loss, hemineglect, and deep pain were always transient disorders, regressing during hospital stay.

All 13 patients had an ischemic lesion in the perforating branches of the middle cerebral artery. The infarcts limited to the putamen were small (<15 mm in 8 patients), while the infarcts involving putamen and globus pallidus externus were larger (templates in the Figure).

**Comment**

We found that acute unilateral infarction limited to the lenticular nucleus is rare (1.6% of deep cerebral infarcts). Its clinical presentation mainly includes faciobrachialcrural hemiparesis, which may be associated with ataxia, a sensory deficit, nonfluent aphasia, and deep pain. No movement disorders were seen in these patients.

These results contrast with previous reports in which the most prominent clinical feature due to lenticular lesions was dystonia, which was not seen as an acute or subacute or delayed movement disorder in any of our patients during a follow-up of up to 3 years. However, we must emphasize that in previous studies movement disorders were a selection criterion, whereas in our study case selection depended on the localization of the lenticular nucleus without involvement of other structures. Dystonic movements and postures have been observed with isolated lesions in the caudate, thalamus, or lenticular nucleus or with combined lesions, and have been reported to be present in 63% of putaminal lesions and 36% of globus pallidus lesions. These lenticular lesions are often placed laterally and involve the putamen more than the globus pallidus.

Most putaminal lesions described in the literature resulted from head trauma and/or bilateral necrosis (due to poisoning, hypoxia, Leigh disease, or infantile necrosis) and in most cases were not limited to the putamen. Occurrence of pure ischemic stroke limited to the putamen has been rarely reported, with putaminal infarctions involving usually adjacent structures.

The basal ganglia are particularly involved even in mild or moderate traumatic injury, which might explain the development of acute or subacute dystonia. On the other hand, for delayed dystonia, aberrant neuronal sprouting, ephaptic transmission after injury, remyelination, and late inflammatory changes are suggested pathophysiologic mechanisms.

All of our patients presented with hemiparesis. With the use of only CT scanning, invisible damage to the internal capsule as a result of a putaminal lesion cannot be excluded, but most of the lesions were quite remote from this structure (Figure).

However, changes in cerebral blood flow and metabolism, which have been shown by positron emission tomography and single-photon emission computed tomographic studies, are the basis for 2 phenomena not visible on CT or MR imaging that could possibly ex-
plain hemiparesis. First, ischemic penumbra may be present, with critical hypoperfusion and preserved reversibility of function and structure, ie, ischemically affected but still viable tissue. Second, involvement of functional networks close to or remote from the structural lesion (metabolic changes or diaschisis) is another potential mechanism.

Seven of our patients presented with hemisensory loss in addition to hemiparesis. Sensory deficits are common in acute stroke, having been reported in nearly half of the patients, usually in combination with other neurologic deficits. Pure sensory loss has been described formerly with thalamic, parietal, brainstem, capsular, and lenticulocapsular lesions. Hemisensory loss associated with involvement of the lenticular nucleus but sparing of the internal capsule may be due to interruption of projections from the somatosensory cortex (Brodmann areas 1, 2, and 3) and from the somatosensory association cortex (areas 5 and 7) passing the putamen to reach the globus pallidus internus and the thalamus. Since hemisensory loss was not dissociated in our 6 patients, it might be postulated that lemniscal and spinothalamic projections through the putamen run close together.

Cognitive dysfunction was not common in our patients, with nonfluent aphasia occurring in 2 right-handed patients with left-sided lesions and in another patient with a right-sided putaminal lesion and left hemineglect. These cognitive deficits might be due to the interruption of the dorsolateral prefrontal circuit projecting from Brodmann areas 9 and 10 (responsible for aphasia) and Walker area 46 (responsible for neglect) to the dorsolateral head of the caudate nucleus, with interconnections with the posterior parietal cortex (Brodmann area 7).

Pure lenticular infarction may induce hemiparesis rather than movement disorder. Commonly associated sensory deficits, aphasia, and hemineglect underline dysfunction of connections between the lenticular nucleus...
and the prefrontal, temporal, and parietal cortices. Taking into account the limitations of our study in regard to the availability of CT scans in most patients, involvement of the internal capsule responsible for motor symptoms cannot be excluded completely.

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