Acute Infarction Limited to the Lenticular Nucleus

Clinical, Etiologic, and Topographic Features

Heike Russmann, MD; François Vingerhoets, MD; Joseph Ghika, MD; Philippe Maeder, MD; Julien Bogousslavsky, MD

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 hemispheral 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.

Arch Neurol. 2003;60:351-355

From the Departments of Neurology (Drs Russmann, Vingerhoets, Ghika, and Bogousslavsky) and Radiology (Dr Maeder), Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland.

©2003 American Medical Association. All rights reserved.

Downloaded From:  by a Non-Human Traffic (NHT) User  on 11/07/2018
orders, as well as motor disorders including delayed contralateral hemidystonia or subacute choreathetosis. 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 when emergency (<12 hours after onset) high-resolution and 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.

### 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, 6 of whom had small-artery disease as the presumed cause of infarct.

All patients had initial faciobrachialocrural motor hemisindrome, 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 faciobrachialocrural motor hemisindrome, predominantly crural in 1. There were 3 patients with proportional faciobrachialocrural 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 hemisindrome. All 3 patients had spinothalamic hemihyposthesia; 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 hemisindrome with ataxia was present in 1 patient. Three patients had moderate ataxic faciobrachialocrural motor hemisindrome 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 faciobrachialocrural motor hemisindrome with nonfluent aphasia, and...
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.

Accepted for publication September 11, 2002.

Author contributions: Study concept and design (Dr Russmann and Bogousslavsky); acquisition of data (Dr Russmann); analysis and interpretation of data (Drs Russmann, Vingerhoets, Ghika, Maeder, and Bogousslavsky); drafting of the manuscript (Dr Russmann); critical revision of the manuscript for important intellectual content (Drs Russmann, Vingerhoets, Ghika, Maeder, and Bogousslavsky); statistical expertise (Dr Russmann); obtained funding (Dr Russmann); administrative, technical, or material support (Dr Russmann); study supervision (Drs Vingerhoets, Ghika, Maeder, and Bogousslavsky).

Corresponding author: Heike Russmann, MD, Department of Neurology, Centre Hospitalier Universitaire Vaudois, CH-1011 Lausanne, Switzerland (e-mail: heike.russmann@chu.vaudswp.ch).

REFERENCES

17. Sander US, Arleha J, Obeso JA. Clinical and CT scan findings in a case of cya