Cerebral Infarction of the Basal Ganglia Due to Embolism from the Heart

JOAN SANTAMARIA, M.D., FRANCESC GRAUS, M.D., FRANCISCO RUBIO, M.D., Txomin Arbizu, M.D., and Jaume Peres, M.D.

SUMMARY We studied 8 patients with cerebral infarction in the deep territory of the middle cerebral artery (MCA). All patients had a definite cardiac source of emboli and no known factors for thrombosis. Mixed sensory and motor deficit was found in all but one patient and CT scan showed larger lesions than usually reported in lacunar infarcts. Contrast enhancement was seen in all cases in which CT scan was performed in the second or third week. It is concluded that embolic infarcts in deep cerebral territory of MCA from a cardiac source are more frequent than previously reported. This diagnosis has to be considered when CT scan demonstrates a deep cerebral infarct.

Deep Cerebral Infarcts located in the basal ganglia and internal capsule are usually caused by thrombosis of the small penetrating arteries affected by lipohyalinosis or microscopic atherosclerosis.¹ The most frequent clinical presentation is a pure motor deficit² ³ but it is sometimes accompanied by dysarthria, sensory impairment⁴ or cerebellar deficit.⁵ The size of the infarct is usually small, with a diameter of 6 to 10 mm,² but larger infarcts have been described in computed tomography (CT).³ ⁶ ⁷ and pathologic studies.²

Although a source of emboli in patients with deep cerebral infarcts is almost never considered, a few experimental⁸ and pathologic¹ studies suggest that some may be embolic. Recently, Pullicino et al⁹ found a possible cardiac source of emboli in 12% of 42 patients with small deep infarcts diagnosed by CT.

This report analyzes the clinical and CT findings of eight patients with a definite cardiac source of emboli and deep cerebral infarcts diagnosed by CT.

Patients and Methods

We reviewed records of all patients with deep cerebral infarcts confirmed by CT seen during the period 1979–1981. Patients with hypertension (blood pressure equal or more than 160/95 mm Hg in several determinations) diabetes, blood hematocrit greater than 45%, heavy cigarette smoking or vasculitis were excluded. This review yielded 8 patients without known risk factors for a thrombotic disease and with a definite heart disease with high probability of cerebral embolization. Six patients had rheumatic heart disease and mitral valve stenosis, five of them with atrial fibrillation. The disease was diagnosed by clinical history and auscultation. In three cases it was confirmed at surgery. One patient had an aortic prosthetic valve (Starr-Edwards) and anticoagulation had been stopped one year before the stroke because of gastrointestinal bleeding. Another patient had a subacute bacterial endocarditis with blood cultures positive for Streptococcus salivarius and infected vegetations were confirmed at surgery.

Results

Clinical and CT findings are summarized in table 1. The clinical deficit had an abrupt onset, reaching its peak in a few minutes in half of the patients. In two patients, the deficit progressed during the next 3 hours; and in the remaining 2, a fluctuating course over several hours was seen before stabilization occurred. Neurological examination showed a complete (5 cases) or incomplete (3 cases) hemiplegia, accompanied by hypesthesia in 6 patients. A pure motor hemiplegia was only seen in one case. In three of them, a transitory anosognosia was clearly observed. In a further patient (case 8), the motor hemiplegia was accompanied by an expressive dysphasia. Her language disorder was characterized by nonfluent conversational speech, naming and reading difficulties, dysgraphia and a normal auditory comprehension. She was almost normal one year after the stroke. Recovery of the motor and sensory deficit was complete in two patients, moderate in four, and poor in two.

EEG was performed on 7 patients. In all but one case, EEG showed a discrete focal slowing. The most common CT abnormality, seen in four cases, was a well-defined low density area placed in caudate, putamen and anterior limb of internal capsule (fig. 1). In a further 3 cases, CT showed a posterior extension (fig. 2). The patient with aphasia had a small lesion circumscribed to putamen and anterior limb of the internal capsule. In six cases, several CT scans were performed during the first two weeks, giving the evidence that the infarct was recent. Contrast enhancement was observed in all cases in which CT were performed during the second or third week. In one case, the infarct was hemorrhagic.

Discussion

Although the clinical diagnostic criteria for cerebral embolism are not universally agreed upon, in our cases the absence of risk factors for thrombosis and the presence of a definite cardiac source of emboli suggest embolism as the cause of the cerebral infarction. The mean age and the female preponderance are similar to those reported in studies of cerebral embolism secondary to rheumatic heart disease.⁹ ¹⁰
All but one patient manifested clinical patterns different from those associated with lacunar infarcts and the size of the lesions found by CT scan was usually larger than those described pathologically by Fisher et al. The deficit fluctuated or had a gradual progression in four patients, this temporal profile more suggestive of a thrombotic stroke has been described in cerebral embolism as well. In addition the Harvard Cooperative Stroke Registry reported this profile in 21% of the patients diagnosed of cerebral embolism.

The presence of anosognosia (a lack of recognition of their hemiplegia or hemianesthesia) and aphasia in four of our patients could suggest a concomitant lesion in cortical areas. In the absence of pathological verification this assumption cannot be ruled out definitively.

**TABLE 1 Clinical and CT Findings**

<table>
<thead>
<tr>
<th>Patient/age/sex</th>
<th>Type of heart disease*</th>
<th>Clinical presentation</th>
<th>Neurologic findings</th>
<th>Clinical course</th>
<th>CT findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/49/F</td>
<td>RHD with MS AF</td>
<td>abrupt onset maximum deficit immediately</td>
<td>hemiplegia (L) hyposthesia anosognosia pure motor hemiplegia (L)</td>
<td>moderate recovery anosognosia disappeared in 2 days moderate recovery</td>
<td>head of caudate AL of IC and putamen</td>
</tr>
<tr>
<td>2/37/F</td>
<td>RHD with MS and AR, AF</td>
<td>abrupt onset and gradual progression during 3 hours</td>
<td>hemiplegia (L) hyposthesia anosognosia dysarthria</td>
<td>poor recovery</td>
<td>hemorrhagic capsulo-lenticular and caudate</td>
</tr>
<tr>
<td>3/53/F</td>
<td>IHSS and SBE</td>
<td>abrupt onset</td>
<td>hemiplegia (L) hyposthesia</td>
<td>total recovery</td>
<td>caudate, putamen and AL of IC</td>
</tr>
<tr>
<td>4/59/F</td>
<td>RHD with MS</td>
<td>abrupt onset and fluctuation during 5 hours</td>
<td>hemiplegia (L) hyposthesia</td>
<td>total recovery</td>
<td>caudate, putamen and AL of IC</td>
</tr>
<tr>
<td>5/34/M</td>
<td>aortic prosthesis valve (Starr-Edwards)</td>
<td>abrupt onset, maximum deficit immediately</td>
<td>hemiplegia (L) hyposthesia</td>
<td>moderate recovery</td>
<td>head of caudate AL of IC and putamen</td>
</tr>
<tr>
<td>6/65/F</td>
<td>RHD with MS AF</td>
<td>abrupt onset and gradual progression during 3 hours</td>
<td>hemiparesia (L) hyposthesia anosognosia</td>
<td>moderate recovery anosognosia disappeared in few days</td>
<td>head of caudate AL of IC and putamen</td>
</tr>
<tr>
<td>7/49/F</td>
<td>RHD with MS AF</td>
<td>abrupt onset maximum deficit immediately</td>
<td>hemiplegia (L) hyposthesia anosognosia</td>
<td>poor recovery</td>
<td>head of caudate AL of IC and putamen</td>
</tr>
<tr>
<td>8/37/F</td>
<td>RHD with MS</td>
<td>abrupt onset and fluctuation during 6 hours</td>
<td>hemiparesia (R) aphasia</td>
<td>total recovery</td>
<td>AL of IC</td>
</tr>
<tr>
<td>*Confirmed at surgery in cases 1, 2, 3 and 8.</td>
<td></td>
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</tbody>
</table>

RHD = rheumatic heart disease; MS = mitral stenosis; AF = atrial fibrillation; AR = aortic regurgitation; IHSS = idiopathic hypertrophic subaortic stenosis; SBE = subacute bacterial endocarditis; L = left; R = right; AL = anterior limb; IC = internal capsule.
but the serial CT scans performed in all patients did not show a cortical lesion in any case. On the other hand these symptoms have been described in patients with deep cerebral infarctions or hemorrhages. Damasio et al reported atypical aphasia syndromes in six patients with infarctions that involved the left anterior limb of the internal capsule, the head of caudate and putamen. Anosognosia has usually been reported in lesions involving the right thalamus or in basal ganglia hemorrhages. Recently Healton et al described a patient with anosognosia and a pathologically verified right hemorrhagic infarct restricted to putamen, caudate and genu and posterior limb of the internal capsule.

When an embolism involves the deep cerebral areas it is usually in the context of a large infarct that extends to the cortical territory. Deep cerebral infarcts are usually caused by thrombosis of the lenticulostriate arteries or internal carotid artery and the possibility of an embolic origin has been hardly ever considered in the literature.

Fisher, in a detailed pathologic study, suggested an embolic mechanism in two cases with lacunar infarcts.
and no occlusion in the appropriate lenticulostriatated artery, although neither of them had a clear source of emboli. The possibility of an embolic occlusion of a lenticulostriatated artery would be supported by the demonstration of small emboli in the retinal arteries of patients with amaurosis fugax and ulcerated carotid atheromas. Recently Pullicino et al reported the presence of a source of emboli in 33% of their patients with small deep infarcts diagnosed by CT scan, and the same was found by Rascoc et al in 13% of their cases of pure motor hemiplegia.

Another way in which an embolic infarction can be restricted to a deep cerebral territory is when a large embolus is arrested in the trunk of the middle cerebral artery (MCA) occluding the mouths of the lenticulostriatated arteries. A deep infarction is produced whereas the superficial territory is spared by the collateral circulation. In this way two of the deep infarcts associated with an embolic cardiac source described by Blackwood et al, although not specially commented by these authors, had a pathologically demonstrated occlusion of the trunk of the MCA. Deep cerebral infarcts have also been produced in experimental studies, Crowell et al reported deep infarcts in 17 of 27 monkeys subjected to temporary MCA occlusion. The infarcts involved the anterior limb of the internal capsule, the caudate nucleus and the anterior portion of putamen. This high incidence may be due to the variations of meningocortical anastomoses between man and animals. It is our impression, however, that cerebral embolism in the deep territory of the MCA is more frequent than is reported and its better prognosis has prevented the possibility of more pathological studies. With the use of CT scan this diagnosis will probably increase.

It is worth noting that our cases are not the same as Fisher’s lacunar stroke patients. The patients described here have much larger lesions than Fisher described pathologically — the location is similar but they are not usually restricted to the territory of a single lenticulostriatated artery — and the clinical presentation indicates also a much bigger lesion. The differentiation between these two types of deep cerebral infarcts is useful in order to establish an appropriate therapy.

References

Cerebral infarction of the basal ganglia due to embolism from the heart.
J Santamaria, F Graus, F Rubio, T Arbizu and J Peres

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