Correlation of Angiographic and Sequential CT Findings in Patients with Evolving Cerebral Infarction

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The usefulness of CT and angiography for predicting the final ischemic brain damage resulting from supratentorial ischemic stroke was evaluated in 36 patients. CT was performed within 4 hr and angiography within 6 hr after the onset of symptoms. CT was used to assess the site and size of parenchymal brain damage and angiography was used to evaluate the cerebral circulation. A 3-month follow-up CT study was used to determine the site and size of final ischemic damage. Angiography was normal in six patients and showed complete occlusion in 30. Angiographic findings in patients with arterial occlusion were classified as either internal carotid artery occlusion or middle cerebral artery (MCA) occlusion. MCA occlusions were subdivided into occlusion before the origin of internal lenticulostriate arteries (type 1), occlusion beyond the origin of these branches (type 2), occlusion at the bifurcation of the main trunk (type 3), and occlusion of the peripheral branches (type 4). Collateral blood supply was also studied. Early CT findings were positive in 25 of 36 patients; the lentiform nucleus alone, the lentiform nucleus and the cortex, or only the cortex were involved. In all patients with positive early CT findings, angiography showed an arterial occlusion, often located in the main trunk of the MCA. Involvement of the lentiform nucleus on early CT was always seen in patients with internal carotid artery or type 1 MCA occlusion. Involvement of the lentiform nucleus was also observed in some, but not all, patients with types 2 and 3 MCA occlusion, depending on the site of the external lenticulostriate arteries. A lesion in the lentiform nucleus was evident on 3-month follow-up scans in all patients who had injury of the lentiform nucleus or lentiform nucleus and cortex on early CT scans. The condition of the cerebral cortex was found to be related to the development of collateral blood supply. In patients with a normal early CT scan, angiography was normal or indicated occlusion of two or three peripheral branches, and the final CT damage was always limited in extent.

When CT was performed during a very early phase of supratentorial ischemic stroke, the site and size of arterial occlusion could be documented and final brain damage could be predicted.

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The value of CT in diagnosing ischemic or hemorragic stroke is well documented [1–3]. In ischemic brain damage, CT findings become positive during the first 2–3 days after the acute event, but positive CT findings have been demonstrated as early as 6 [4, 5] and 24 [6] hr after injury. The importance of a hypodensity on an early CT study has not been evaluated thoroughly relative to the final CT-determined brain damage and the angiographic picture. We evaluated the significance of early CT hypodensities in a series of patients in whom CT and angiography were performed within 4 and 6 hr, respectively, after the onset of symptoms.

Subjects and Methods

All patients with acute focal supratentorial neurologic deficits admitted to our department over an 8-month period underwent a general clinical and neurologic examination and had a

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plain CT scan of the brain within 4 hr after the onset of symptoms. Only patients who did not exhibit severe alteration of consciousness or life-threatening disease were included in our study. Patients in whom deficits of a nonischemic origin (e.g., hemorrhage or tumor) were documented by plain CT were also excluded.

Following these criteria, 44 patients (25 men and 19 women; average age, 64.7 ± 9.2 years) were included in the study. Three patients died during the first few days. The vascular territory involved was defined in 41 patients by CT studies performed within 5–7 days. In five patients the vascular territory involved was found to be different from the middle cerebral artery (MCA). These patients were excluded from the study. The 36 patients with brain damage localized in the MCA vascular territory were considered.

CT was performed in all patients on a Siemens-Somatom CR high-resolution scanner within 4 hr after the onset of the symptoms (average time, 177 min); CT was repeated after 5–7 days and again after 3 months.

The early CT scans were analyzed for (1) a slight hypodensity in the lentiform nucleus; (2) involvement of the lentiform nucleus plus a cortical hypodensity; and (3) a slight hypodensity only in the cortex and/or sulcal effacement.

The final extent of parenchymal brain damage was evaluated by CT 3 months after the acute event. The CT findings were classified according to the anatomic MCA vascular territories involved [7]: involvement of the structures supplied by the lenticulostriate arteries (deep MCA); involvement of the areas supplied by the MCA cortical branches (superficial MCA); partial involvement of both the structures supplied by the lenticulostriate arteries and by the MCA cortical branches (partial MCA); complete involvement of both the areas supplied by the lenticulostriate arteries and by the MCA cortical branches (complete MCA); internal watershed or deep territories of superficial MCA branches (internal border), located in the white matter of the corona radiata [8]; and lacunar infarcts in the territory of lenticulostriate arteries or in the deep territories of the superficial MCA branches [9].

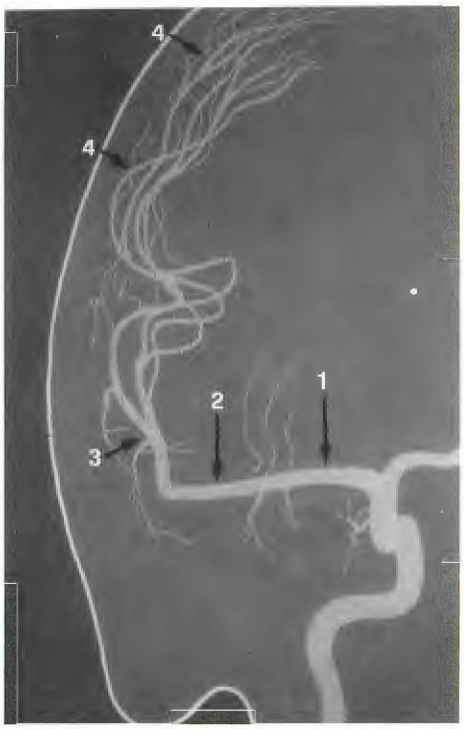

Fig. 1.—Schematic drawing of middle cerebral artery (MCA) shows different points of arterial occlusion.

***1*, Type 1: Occlusion of stem or trunk of MCA proximal to internal lenticulostriate arteries.**

***2*, Type 2: Occlusion of stem or trunk of MCA distal to internal lenticulostriate arteries.**

***3*, Type 3: Occlusion at bifurcation.**

***4*, Type 4: Occlusion of peripheral branches of MCA.**

Intraarterial digital subtraction angiography was performed in all 36 patients by direct carotid injection or retrograde brachial catheterization. Nonionic water-soluble contrast medium diluted with a saline solution to a concentration of iodine equal to 200 mg/ml was injected in all these patients within 6 hr (average, 237 min) [10]. Retrograde brachial catheterization was performed in the patients with right hemisphere involvement. The angiographic study was generally restricted to the carotid territory responsible for the clinical symptoms. In three of the four patients with internal carotid artery (ICA) occlusion, angiography was performed contralaterally as well. Angiography of the vertebrobasilar territory was not performed routinely because it was considered too invasive for acute injury; therefore, a complete assessment of the total collateral circulation was not available in all patients.

Angiographic findings in patients with complete arterial occlusion were classified as extracranial ICA occlusion at the bifurcation, extracranial ICA occlusion in the cervical segment, intracranial ICA occlusion at the siphon, and MCA occlusion.

MCA occlusions were divided into four types: type 1—occlusion of the stem or trunk of the MCA proximal to the internal lenticulostriate arteries, type 2—occlusion of the stem or trunk of the MCA distal to the internal lenticulostriate arteries, type 3—occlusion of the MCA at the bifurcation (in our patients the occlusion always involved only the upper branch), and type 4—occlusion of the peripheral branch of the MCA seen through cortical anastomoses (Fig. 1).

The anastomotic collateral blood supply was evaluated according to criteria that considered the rapidity of filling and the number of branches visualized through cortical anastomoses or via the anterior communicating artery or the external carotid artery [11, 12]. The collateral flow was timed on serial digital angiograms. The collateral blood supply was considered good when all potentially occluded vessels showed complete retrograde flow through cortical anastomoses within 5 sec after the end of the carotid injection [13].

Results

The CT and angiographic findings in our patients are summarized in Table 1. In the 36 patients with brain damage localized in the MCA vascular territory, early CT was normal in 11 and abnormal in 25. In these 25 patients, a slight hypodensity was seen in the lentiform nucleus in 14, involvement of the lentiform nucleus plus a cortical hypodensity was found in five, and a slight hypodensity in the cortex only and/or sulcal effacement were seen in six.

In the group of 14 patients in whom early CT showed a slight hypodensity in the lentiform nucleus, angiography showed a type 1 MCA occlusion in six, a type 2 in one, a type 3 in four, and an ICA occlusion in the other three (one bifurcation, one cervical segment, and one siphon). Follow-up CT was available in 12 of these 14 patients.

In four patients, final brain damage was localized in the deep MCA vascular territory involving the lentiform nucleus and part of the head of the caudate nucleus in three (Fig. 2D);

TABLE 1: Early CT and Angiographic Findings and Follow-up CT Findings in 36 Patients with Acute Supratentorial Ischemic Stroke

Early CT Finding/ Case No.	Type of Arterial Occlusion on Angiography	Collateral Blood Supply[a]	Follow-up CT (No. of Involved Branches)
Hypodensity of lentiform nucleus			
1	MCA type 1	Good	Not available
2	ICA at bifurcation	Good	Lacunar infarct
3	MCA type 1	Absent	Complete MCA
4	MCA type 1	Good	Deep MCA
5	MCA type 1	Good	Partial MCA (3)
6	MCA type 1	Absent	Complete MCA
7	MCA type 1	Absent	Partial MCA (5)
8	ICA at cervical segment	Good	Deep MCA + AChA
9	ICA at siphon	Good	Deep MCA
10	MCA type 2	Good	Deep MCA + internal border zone
11	MCA type 3	Good	Deep MCA + internal border zone
12	MCA type 3	Good	Not available
13	MCA type 3	Good	Partial MCA (3)
14	MCA type 3	Good	Deep MCA + internal border zone
Hypodensity of lentiform nucleus + cortical hypodensity			
15	MCA type 2	Absent	Partial MCA (9)
16	MCA type 1	Absent	Complete MCA
17	MCA type 1	Absent	Complete MCA
18	MCA type 1	Absent	Complete MCA
19	ICA at siphon	Absent[b]	Not available
Cortical hypodensity and/or sulcal effacement			
20	MCA type 4	Good	Superficial MCA (3)
21	MCA type 2	Absent	Superficial MCA (9)
22	MCA type 4	Absent	Not available
23	MCA type 3	Absent	Not available
24	MCA type 3	Absent	Superficial MCA (5) + internal border zone
25	MCA type 3	Good	Superficial MCA (4)
Normal			
26	Normal	–	Not available
27	Normal	–	Internal border zone
28	Normal	–	Internal border zone
29	Normal	–	Not available
30	Normal	–	Superficial MCA (1)
31	Normal	–	Lacunar infarct
32	MCA type 3	Good	Superficial MCA (3) + internal border zone
33	MCA type 4	Absent	Not available
34	MCA type 4	Good	Superficial MCA (2)
35	MCA type 4	Good	Superficial MCA (2)
36	MCA type 4	Good	Superficial MCA (3) + internal border zone

Note.—Occlusions of the middle cerebral artery (MCA) were located proximal (type 1) or distal (type 2) to the origin of the lenticulostriate arteries, at the bifurcation of the main trunk (type 3), or in the peripheral branches (type 4). ICA = internal carotid artery; AChA = anterior choroidal artery.

[a] Through cortical anastomoses.

[b] Angiography was not performed on the contralateral side.

a small lacunar infarct was found in one. Angiography showed a type 1 MCA occlusion in one patient and an ICA occlusion in the other three (one siphon, one bifurcation, and one cervical segment). A good collateral blood supply through ipsi- or contralateral anterior cerebral artery cortical anastomoses (Figs. 2A and 2B) or through the anterior communicating artery (one patient) was visualized in all four patients. Of the two patients with ICA occlusion and collaterals through the contralateral anterior cerebral artery, damage in the posterior limb of the internal capsule (anterior choroidal artery territory) was found on follow-up CT in one patient (Fig. 2D).

In six patients, final brain damage was localized in the deep and superficial MCA vascular territories; the deep lesion was localized in the whole lentiform nucleus in two (Fig. 3B) and in its lateral segment in the other four (Fig. 4B). A lesion localized in a superficial MCA territory was found in three

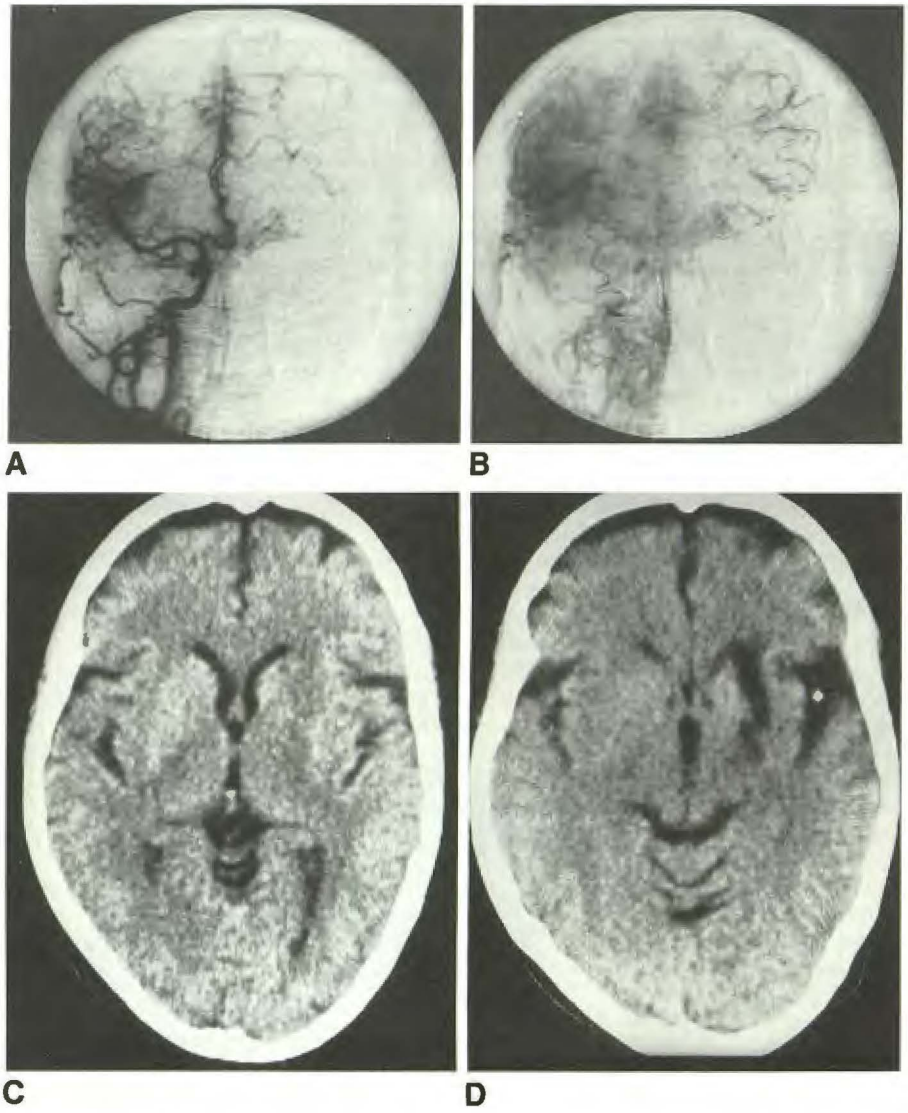

Fig. 2.—Left internal carotid artery occlusion in cervical segment.

***A* and *B*, Angiography on contralateral side shows good collateral blood supply through cortical anastomoses via anterior cerebral arteries.**

***C*, Early CT scan shows hypodensity involving left putamen nucleus.**

***D*, Follow-up CT scan shows marked hypodensity involving lentiform nucleus, posterior limb of internal capsule, and part of head of caudate nucleus.**

patients (three, five, and three branches) and involvement of the deep territory of the MCA superficial branches (internal border zone) was found in the remaining three. Angiography showed an MCA occlusion in all six (type 1, two; type 2, one; and type 3, three). In the two patients with type 1 MCA occlusion, the deep damage was localized in the whole lentiform nucleus; the damage involved only the lateral segment of lentiform nucleus in the others. In five patients good collateral flow was visualized (Figs. 3C, 3D, 4C, and 4D); it was not evident only in one patient with type 1 MCA occlusion who later developed more extensive cortical damage (five branches).

In two patients, final brain damage was localized in the entire vascular territory of the MCA; type 1 MCA occlusion without collateral circulation was present in both.

Early Hypodensity of Lentiform Nucleus plus Cortical Hypodensity

In the group of five patients with type 2 early CT findings, angiography showed an ICA occlusion at the siphon in one, a type 1 MCA occlusion in three, and a type 2 occlusion in the other. Follow-up CT was available in four patients.

The final CT brain damage was localized in the deep (lateral segment of lentiform nucleus) and superficial MCA territories (nine branches) in one patient with type 2 MCA occlusion and in the entire MCA in the three patients with type 1 MCA occlusion (Figs. 5C and 5D); anastomotic collateral flow was absent in all patients.

Cortical Hypodensity and/or Sulcal Effacement

In the group of six patients with type 3 early CT findings, angiography showed a type 2 MCA occlusion in one, a type 3 in three, and a type 4 in two. Follow-up CT was available in four patients.

Final CT brain damage was localized in the superficial MCA territory in three patients (three, four, and nine branches). A superficial MCA territory (five superficial branches) in association with internal border-zone infarction was found in the remaining patient. Angiography showed a type 2 MCA occlusion without evidence of anastomotic collateral circulation in

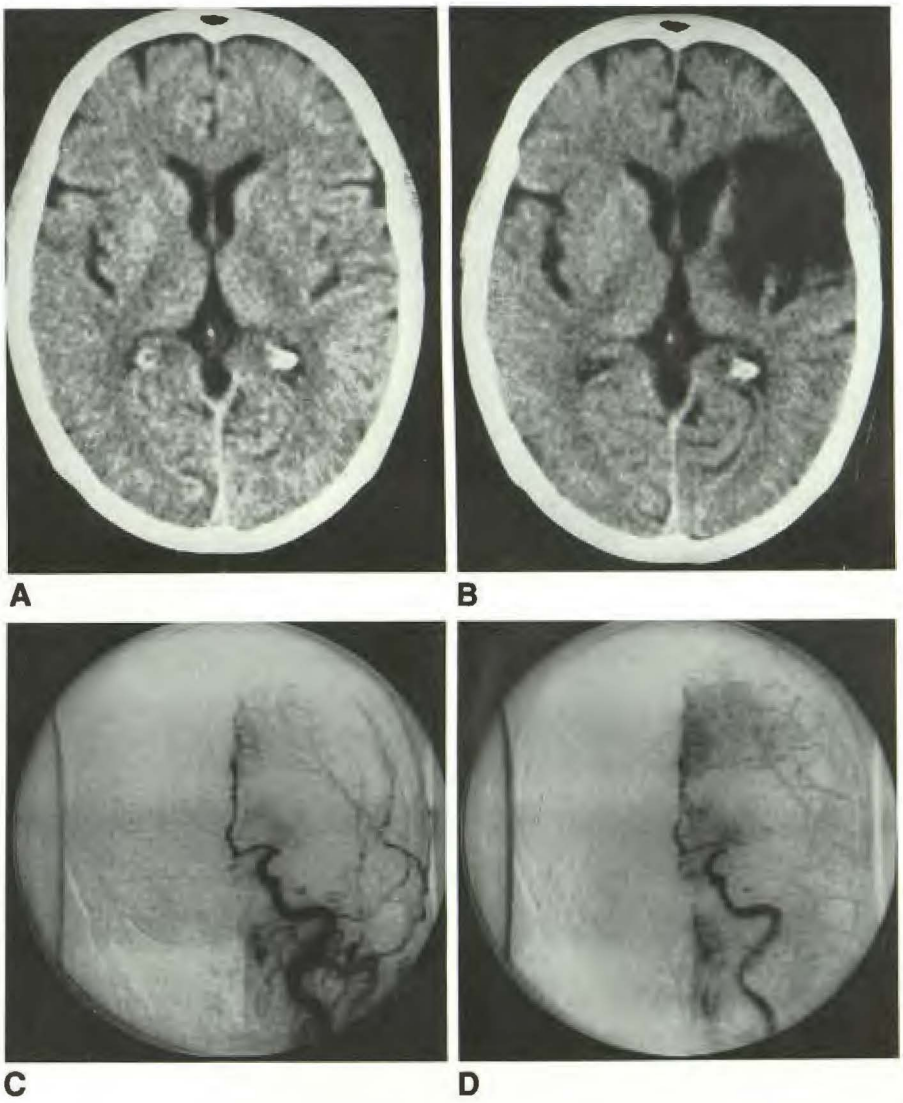

Fig. 3.—*A*, Early CT scan shows slight hypodensity of lentiform nucleus on left.

***B*, Follow-up CT scan shows marked hypodensity involving whole lentiform nucleus and cortical/subcortical temporal region (deep and superficial vascular territory of middle cerebral artery [MCA]).**

C and *D*, Occlusion of stem of MCA proximal to origin of internal lenticulostriate arteries (type 1 MCA occlusion), with good collateral blood supply through cortical anastomoses via anterior cerebral artery.

one patient, a type 3 in two (one patient presented with collateral flow) and a type 4 in one.

The patient with type 2 MCA occlusion did not develop brain damage involving the lentiform nucleus. In this patient, angiography showed a horizontal MCA segment longer than normal (2.5 cm) (Fig. 6C) [14].

Normal Early CT Scan

In the group of 11 patients with normal early CT scans, angiography was normal in six; a type 3 MCA occlusion was present in one and a type 4 in four. Follow-up CT was available in eight patients. Final brain damage was localized in the superficial MCA (one, two, and two branches) in three patients, in the superficial MCA (three branches) and internal border zone in two patients, and in the internal border-zone territory only in two; in the remaining case, a lacunar infarct was found in the basal ganglia.

Angiography did not show any occlusion in four patients, but it showed a type 3 MCA occlusion with good collateral blood supply in one case and a type 4 occlusion in the remaining three.

Correlation Among Categories of Early CT Findings

In 19 patients, early CT, which documented a slight hypodensity of the lentiform nucleus in isolation (type 1) or associated with a cortical hypodensity (type 2), was correlated with an arterial occlusion localized in either the ICA (four patients) or the MCA (type 1 in nine and type 2 or 3 in six). Anastomotic collateral flow was seen in three of the four patients with ICA occlusion; the flow was via the contralateral anterior cerebral artery through cortical anastomoses in two and through the anterior communicating artery in one patient who developed lacunar infarcts. Final CT, available in 15 of 19 patients, showed a lesion localized in the lentiform nucleus in all 15; it involved the entire lentiform nucleus in 10 and the lateral segment in five.

Early CT showed a cortical hypodensity in four of the patients with type 2 or 3 MCA occlusion; final CT, available

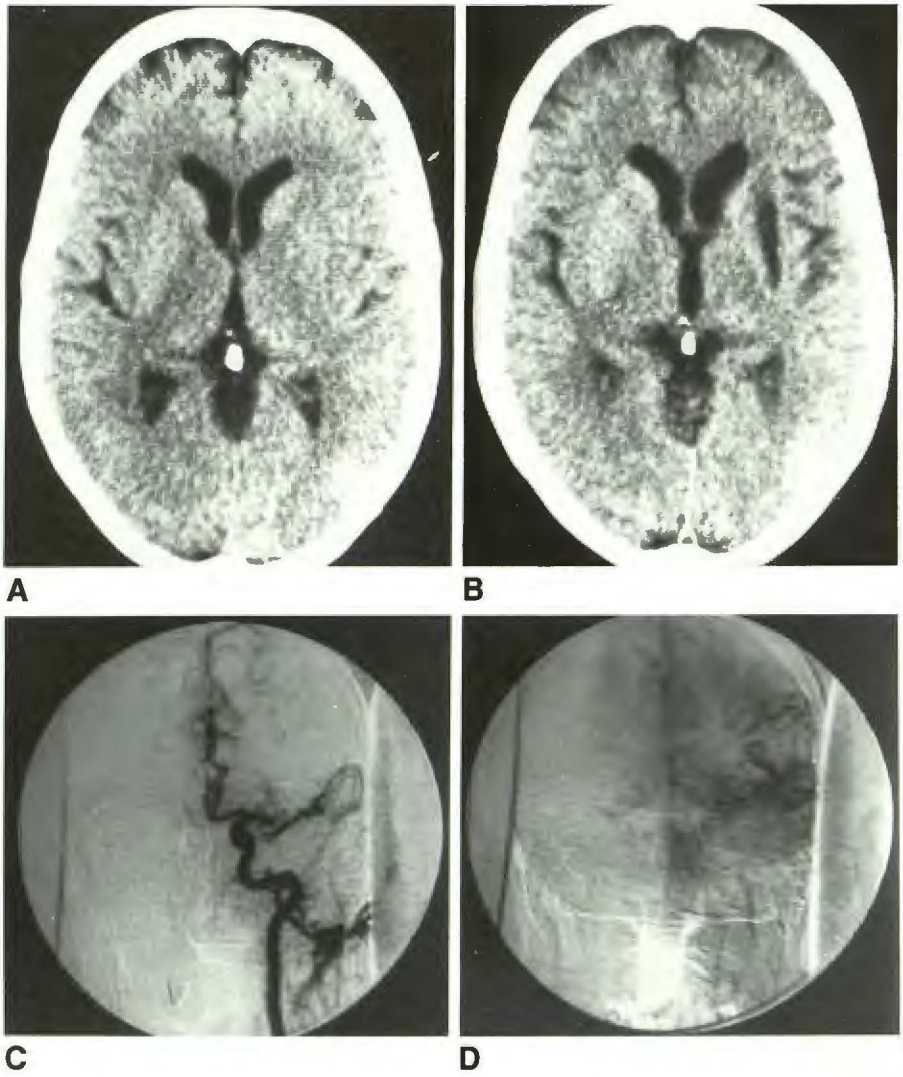

Fig. 4.—*A*, Early CT scan shows slight hypodensity of left putamen.
***B*, Follow-up scan shows marked hypodensity localized to lateral segment of lentiform nucleus (deep middle cerebral artery [MCA] vascular territory).**
***C*, MCA occlusion at bifurcation (type 3 MCA occlusion) involving upper branch.**
***D*, Good collateral blood supply through cortical anastomoses via anterior cerebral arteries.**

in three patients, did not document a lesion in the lentiform nucleus (Fig. 6B).

Discussion

Internal lenticulostriate arteries are deep end branches of the MCA; they supply the lentiform nucleus generally in the medial part [14]. There is no possibility of collateral flow in this brain territory from cortical anastomoses when a proximal stem or trunk MCA occlusion occurs [15]. Furthermore, the lentiform nucleus is a high-metabolism structure easily damaged by experimental ischemia [16]. Thus, a slight hypodensity in the lentiform nucleus already evident on early CT (4 hr) may be easily understood in patients with MCA occlusion proximal to the internal lenticulostriate origin, or in patients with ICA occlusion without collateral circulation through the anterior communicating artery. Our data agree with those of Tomura et al. [5].

A hypodensity in the lentiform nucleus on an early CT scan was also observed in patients with type 2 or 3 MCA occlusion documented by angiography within 6 hr. This finding may be explained by the fact that the lateral zone of lentiform nucleus is vascularized by external lenticulostriate arteries. These branches have different anatomic origins, arising either from the end of the MCA stem or, when the stem is short, from the MCA sylvian branch in the intracisternal segment [14]. This could explain why we also observed lesions involving the lentiform nucleus in some patients with type 2 or 3 MCA occlusion, while in other patients only cortical involvement was detected on both early and follow-up CT (Fig. 6).

Three-month follow-up CT studies in some patients with types 2 and 3 MCA occlusion showed final brain damage localized in the lateral part of the lentiform nucleus, confirming the role of external lenticulostriate branches in the vascularization of the lentiform nucleus and explaining why a lentiform nucleus hypodensity may be found during the early phase of ischemic stroke in patients with type 2 or 3 MCA occlusion. Furthermore, detection of a hypodensity in the lentiform nucleus on early CT always predicts late damage in this structure (Figs. 2–5).

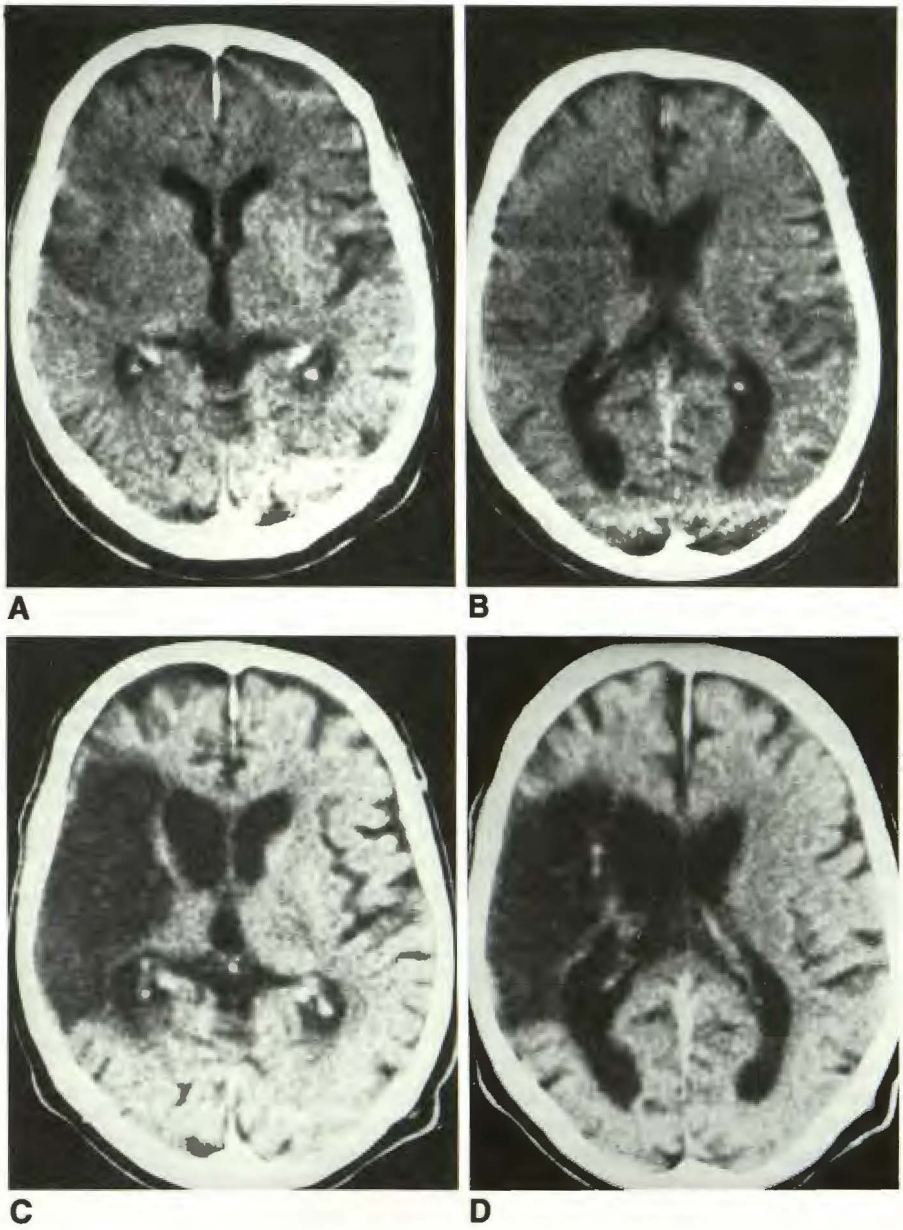

Fig. 5.—Occlusion of middle cerebral artery (MCA) stem before origin of internal lenticulostriate arteries (type 1 MCA occlusion) without collateral blood supply.

***A* and *B*, Early CT scans show slight hypodensity involving lentiform nucleus and cortical subcortical region and sulcal effacement on right.**

***C* and *D*, Follow-up CT scans show marked hypodensity involving whole lentiform nucleus and cortical/subcortical region (complete MCA vascular territory); there is enlargement (ex vacuo) of right lateral ventricle.**

Late damage localized in the lateral part of the lentiform nucleus is demonstrated in patients with type 2 or 3 MCA occlusion. Yet early CT findings in the lentiform nucleus may be due to ICA occlusion or to type 1, 2, or 3 MCA occlusion. Thus, such findings cannot help in identifying the exact site of arterial occlusion, although occlusion of the ICA or of a type 1 MCA is more likely. Involvement of the anterior choroidal artery territory on an early CT scan in patients with ICA occlusion was not seen in our study. The lack of damage in this territory on follow-up CT in one of the two patients with ICA occlusion and collateral flow throughout contralateral anterior cerebral artery anastomoses may have been due to the origin of the anterior choroidal artery from the posterior communicating artery [17].

Patients with lentiform nucleus and cortical hypodensities on early CT had an ICA occlusion or a type 1 or 2 MCA occlusion, as documented by angiographic examination; they developed late brain damage localized in the lentiform nucleus, and, owing to the absence of anastomotic collateral flow, in a large part of the cortex (Fig. 5).

An isolated cortical hypodensity on early CT might be due to a failure in the development of collateral circulation. In the case of a single branch occlusion, it could result from more distal migration of an embolus, which, by first occluding the MCA at a proximal level (types 2 and 3), produces more extensive cortical brain damage [18]. Anatomic differences in the origin of the external lenticulostriate arteries [19] could explain why we could not find damage localized in the lentiform nucleus on follow-up CT (Fig. 6). In our patients, a normal early CT study was correlated either with normal angiography or with occlusion of a peripheral branch of the MCA; final brain damage on CT was shown to be limited in

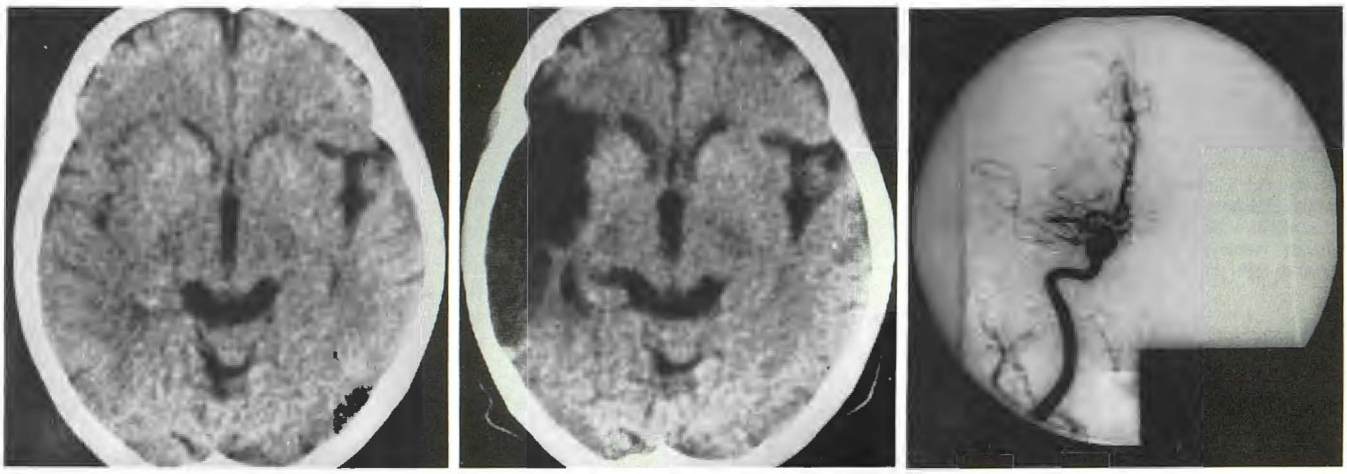

Fig. 6.—Occlusion of middle cerebral artery (MCA) stem beyond origin of internal lenticulostriate arteries (type 2 MCA occlusion) without collateral blood supply through cortical anastomoses.

***A*, Early CT scan shows diffuse hypodensity of right cerebral cortex and sulcal effacement.**

***B*, Follow-up CT scan shows marked hypodensity involving cerebral cortex (superficial MCA territory, nine peripheral branches).**

***C*, Occlusion of MCA stem beyond origin of lenticulostriate arteries; a long horizontal segment of MCA stem is evident.**

extent and localized only in cortical or subcortical brain regions.

In summary, our data indicate a high prevalence of positive early CT findings in patients with supratentorial ischemic stroke examined within 4 hr, in agreement with the findings of Tomura et al. [5]. In all patients with early CT findings, angiography performed within 6 hr documented arterial occlusive pathology, mainly intracranial. A slight hypodensity in the lentiform nucleus on an early CT scan suggests an ICA or MCA occlusion, mainly, but not exclusively, located proximal to the internal lenticulostriate arteries. A slight hypodensity of the lentiform nucleus associated with a cortical hypodensity on early CT is due either to a type 1, 2, or 3 MCA occlusion or to an ICA occlusion. A normal early CT study means either that there is no arterial occlusion or, if an occlusion is present, it is localized in a few peripheral branches of the MCA. These data predict final brain damage limited in extent and exclusively localized in cortical or subcortical territories. Finally, CT findings in a very early phase of supratentorial ischemic stroke allow us to know if an arterial occlusion is present and its probable location, and to predict the development of final brain damage.

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