CT-Defined Large Subcortical Infarcts: Correlation of Location with Site of Cerebrovascular Occlusive Disease

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PURPOSE: To correlate the location of large subcortical infarcts with the site of cerebrovascular occlusive disease. **METHODS:** We examined CT and angiographic findings of 38 patients with major arterial occlusive disease and newly developed large subcortical infarcts of 2.0 cm or more, which were classified into three types: striatocapsular infarcts in the basal ganglia, terminal supply area infarcts in the corona radiata, and terminal supply area infarcts in the centrum semiovale. **RESULTS:** Two or three of the types of infarct were sometimes combined; the combination of the striatocapsular and corona radiata infarcts was the most frequent (14 [36.8%] of 38). Thirty-four (89.5%) had atherosclerotic major arterial occlusive diseases; 22 (57.9%) had occlusive diseases of the internal carotid artery, and 12 (31.6%) had diseases of the middle cerebral artery. The other 4 (10.5%) had embolic transient middle cerebral artery occlusion. Middle cerebral artery occlusive diseases frequently produced striatocapsular (13 [81.3%] of 16) and corona radiata (13 [81.3%] of 16) infarcts but never induced the centrum semiovale lesions. On the other hand, in patients with internal carotid artery occlusive disease, the centrum semiovale (16 [72.7%] of 22) was more susceptible to ischemia than the striatocapsular region (11 [50%] of 22) or the corona radiata (9 [40.9%] of 22). **CONCLUSIONS:** Middle cerebral artery occlusive diseases frequently produced striatocapsular and/or corona radiata infarcts but never induced the centrum semiovale lesions, which were usually associated with internal carotid artery occlusive diseases.

Index terms: Arteries, stenosis and occlusion; Brain, computed tomography; Brain, infarction

It is generally accepted that lacunar infarcts resulting from occlusion of a single penetrating artery are less than 2.0 cm in maximal diameter, usually smaller (1–3). When the maximum diameter of the subcortical infarct exceeds 2.0 cm, atherothrombotic or cardioembolic stroke should be considered rather than lacunar infarcts (4, 5). Large subcortical infarcts have been classified into two pathogenetically different types: striatocapsular infarcts and terminal supply area infarcts (6–8). Affected structures of large striatocapsular infarcts include the cau-

date head, anterior limb of the internal capsule, and the putamen corresponding to the territories of the lenticulostriate arteries (9–11). On the other hand, terminal supply area infarcts are not located in the basal ganglia themselves but in the more rostral periventricular and supraventricular white matter of the corona radiata and the centrum semiovale (7). White matter infarcts in these two regions, resulting from relatively reduced perfusion in two or three neighboring arterial supply areas, have been called together "terminal supply area infarcts." However, they occur simultaneously in some patients and separately in others. The corona radiata is the watershed zone between the deep and superficial territories of the middle cerebral artery (MCA), and the centrum semiovale is the watershed zone between the superficial territories of the MCA and anterior cerebral artery (ACA) (6, 8, 12). Thus, we consider that white matter infarcts in these two regions should be discriminated. In this study we examined the
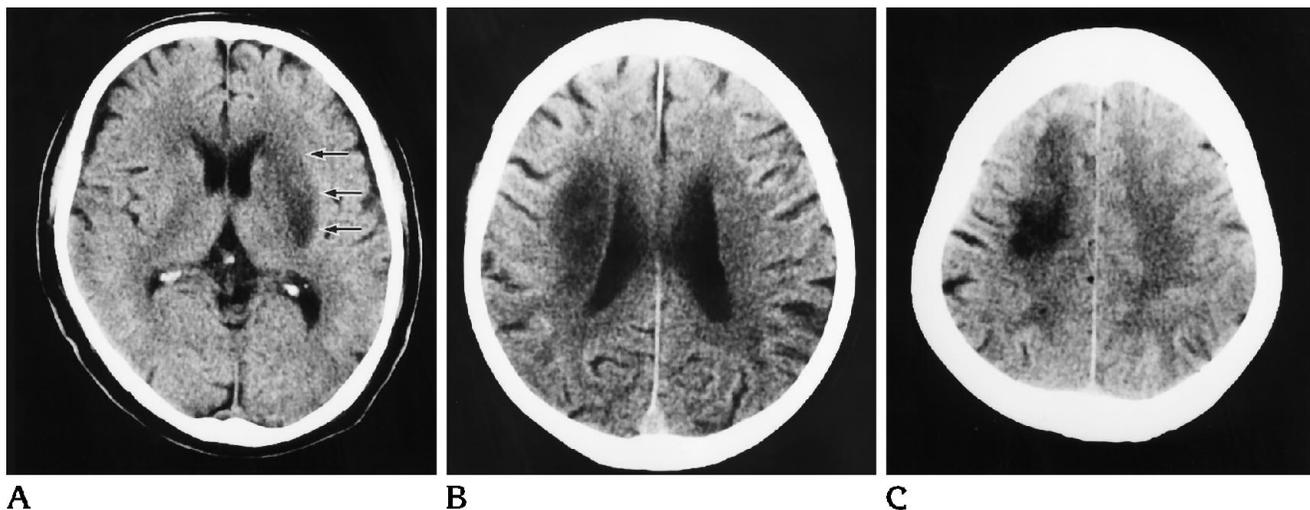
Fig 1. *A*, Representative CT findings of a typical comma-shaped large striatocapsular infarct. It occupies the left caudate head, putamen, and anterior limb of the internal capsule (*arrow*).

B, Representative CT findings of a typical terminal supply area infarct in the periventricular white matter of the right corona radiata.

C, Representative CT findings of a typical terminal supply area infarct in the supraventricular white matter of the right centrum semiovale.

relationship of the location and extent of large subcortical infarcts with the site of arterial occlusive disease.

Subjects and Methods

Between April 1991 and March 1994, 38 patients with major arterial occlusive disease and newly developed large subcortical infarcts of 2.0 cm or more were retrospectively examined. Patients were selected according to the following computed tomography (CT) and angiographic criteria: (*a*) the infarcts were restricted to the basal ganglia and/or white matter on CT, and the overlying cerebral cortex appeared normal; (*b*) the maximum diameter of the lesion exceeded 2.0 cm; and (*c*) atherosclerotic occlusive diseases or cardioembolic occlusions of the major arteries were confirmed by angiography as factors contributory to the subcortical infarcts. All patients were examined with CT and angiography within 24 hours after the onset. In all patients, newly developed infarcts were confirmed with follow-up CT performed within 3 days after the onset.

Based on the location of the lesion on CT, three types of large subcortical infarcts were classified: (*a*) the striatocapsular infarct in the basal ganglia (Fig 1A); (*b*) the terminal supply area infarct in the corona radiata (Fig 1B); and (*c*) the terminal supply area infarct in the centrum semiovale (Fig 1C). In all patients the size and the location of the lesions were evaluated with CT.

As to the size of infarcts, statistical analyses of group comparison between internal carotid artery (ICA) and MCA occlusive diseases were done with Mann-Whitney U test, and a *P* value of <.05 was considered significant.

Results

There were 31 men and 7 women, with a mean age of 64 years (range, 30 to 78 years). Thirty-four (89.5%) of them had atherosclerotic major arterial occlusive diseases; 22 (57.9%) had atherosclerotic occlusive diseases of ICA (14 occlusions, 7 tight stenoses more than 90%, and 1 mild [20%] stenosis with intraluminal thrombus), and 12 (31.6%) had atherosclerotic MCA occlusive diseases (10 occlusions and 2 stenoses more than 70%). In all patients with ICA occlusion, the ipsilateral M1 segment was opacified by collateral flow via anterior and/or posterior communicating arteries. In all patients with tight ICA stenosis, the wall of the lumen in the stenotic portion was smooth and ipsilateral M1 segment was opacified by normograde flow. Only one patient with mild ICA stenosis and intraluminal thrombus had transient MCA occlusion attributable to artery-to-artery embolism. This patient was treated with local thrombolytic therapy, and complete recanalization was achieved. In all patients with atherosclerotic MCA occlusion, angiography revealed rich leptomeningeal anastomoses via anterior and/or posterior cerebral arteries. The other 4 (10.5%) had embolic transient MCA occlusion.

The maximum diameter of the lesions ranged from 2.0 to 6.0 cm. The mean size of the lesions in 22 patients with ICA occlusive disease was 3.8 ± 1.3 cm (SD), whereas mean size in 16

Location and extent of large subcortical infarcts

Vascular Lesion	ICA Occlusive Disease	MCA Occlusive Disease	Total
Striatocapsular (SC)	2	3	5
Corona radiata (CR)	0	3	3
Centrum semiovale (CS)	9	0	9
SC + CR	4	10	14
SC + CS	2	0	2
CR + CS	2	0	2
SC + CR + CS	3	0	3
Total	22	16	38

Note.—SC indicates striatocapsular infarct; CR, terminal supply area infarct in the corona radiata; and CS, terminal supply area infarct in the centrum semiovale.

patients with MCA occlusive disease was 3.3 ± 1.3 cm. There was no significant difference in size between these two groups.

The frequency of the different location patterns of large subcortical infarcts is summarized in the Table. The combination of striatocapsular and corona radiata infarcts was the most frequent pattern (14 [36.8%] of 38), which usually was associated with MCA occlusive diseases occluding the orifices of all lenticulostriate arteries (Fig 2). Ten (62.5%) of 16 patients with MCA occlusive disease produced this combination pattern of infarct. On the contrary, it was not frequent for ICA occlusive diseases to produce this combination pattern of infarct (4 [18.2%] of 22).

On the other hand, MCA occlusive diseases never produced centrum semiovale infarct, which was usually associated with ICA occlusive diseases. Nine (40.9%) of 22 patients with ICA occlusive disease had only centrum semiovale infarcts (Fig 3). In patients with ICA occlusive disease, the centrum semiovale (16 of 22, 72.7%) was more susceptible to ischemia than the striatocapsular region (11 of 22, 50.0%) or the corona radiata (9 of 22, 40.9%).

In this study, none of 22 patients with ICA occlusive disease was associated with the lesion of the posterior limb of the internal capsule, indicating that the territory of the anterior choroidal artery is hardly impaired even in patients with ICA occlusive disease. The posterior limb of the internal capsule was impaired in only 1 patient with transient MCA occlusion.

Discussion

As described by Fisher (2) and Mohr (3), so-called lacunae resulting from occlusion of a single penetrating artery are usually less than 2.0 cm in maximal diameter. On the other hand, Bladin and Berkovic (10) defined "striatocapsular infarctions" as large infarcts at least 3.0 cm in the lenticulostriate arterial territory resulting from occlusive lesions of ICA. However, there are some patients with small striatocapsular infarctions less than 3.0 cm that, by the preceding criteria, should be lacunae but that are associ-
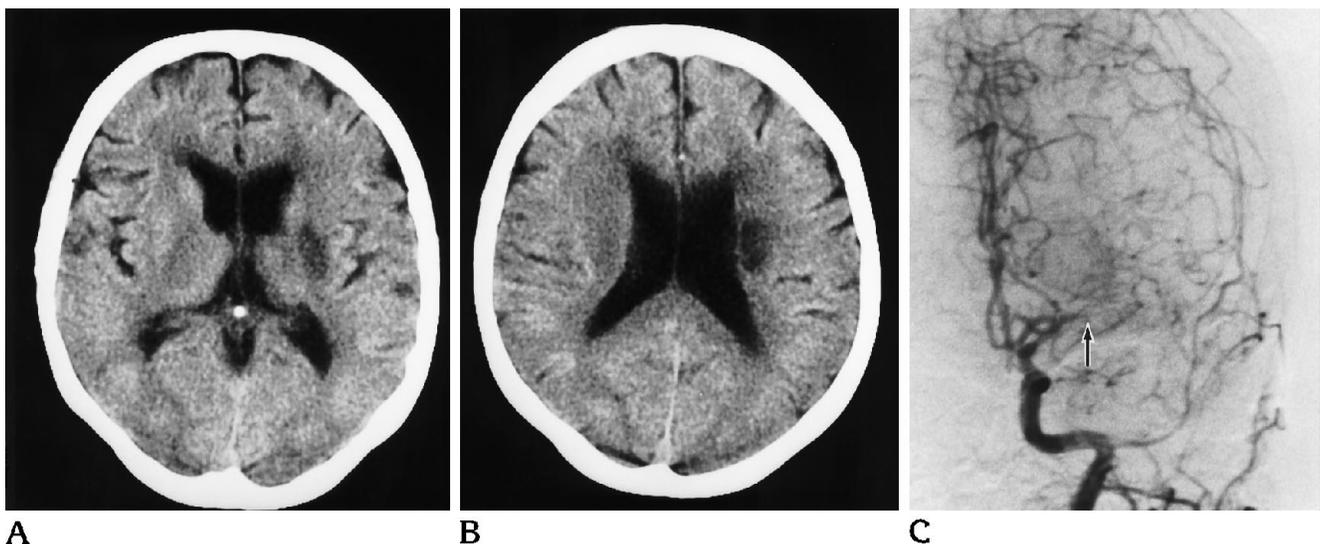

A   B   C

Fig 2. Large subcortical infarct 2.0 cm in diameter in a patient with left MCA occlusion. Blockage of the lenticulostriate arteries (*C, arrow*) caused not only the striatocapsular infarct (*A*) but also the terminal supply area infarct in the corona radiata (*B*). Because of the developed leptomeningeal anastomotic network between the superficial territories of the ACA and MCA (*C*), supraventricular white matter of the centrum semiovale remained intact.
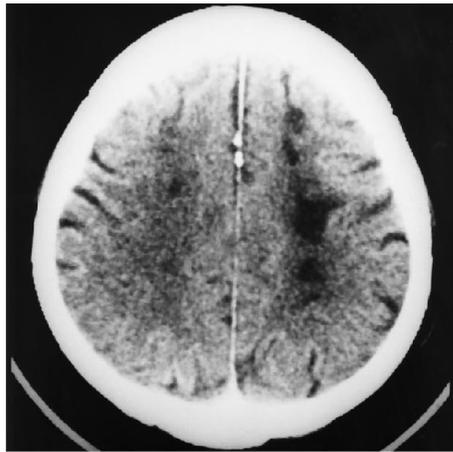
A

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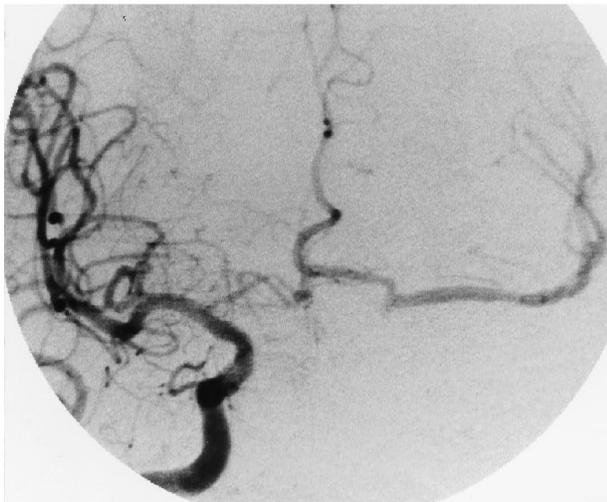
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ated with underlying major arterial occlusive disease (11). In our study, there were seven subcortical infarcts 2.0 cm in size with underlying occlusive disease in the ICA or MCA. Thus, a smaller variant of large subcortical infarct cannot clearly be distinguished from a solitary lacuna by size alone.

The typical shape of large striatocapsular infarct is commalike, lenslike, or triangular (4, 5, 7, 10, 11). The affected areas of large striatocapsular infarcts correspond to the territories of the lenticulostriate arteries and sometimes extend to the territories of the Heubner and/or anterior choroidal arteries. The posterior limb of the internal capsule is uncommonly impaired even in patients with ICA occlusive disease (13). Our present study also demonstrated that none of 22 patients with ICA occlusive disease had a lesion of the posterior limb of the internal capsule. According to the report by Leys et al (14), cardioembolism is one of the major cause of anterior choroidal artery territory infarcts. Probably because of the low incidence of embolic stroke in our present series, there was only one patient with striatocapsular infarct extending to the territory of the anterior choroidal artery who had embolic transient MCA occlusion. On the other hand, in a series of patients described by Weiller et al (11), 11 (37.9%) of 29 striatocapsular infarcts extended to the territory of the anterior choroidal artery. This may result from the fact that most patients in their series had cardiac embolization or embolization from an intraluminal ICA thrombus. Striatocapsular infarction usually occurs when several lenticulostriate arteries are simultaneously exposed to ischemia, and there is sufficient cortical blood flow attributable to rich leptomeningeal collaterals (11, 15). Occlusion of the orifices of the lenticulostriate arteries at the level of the M1 segment of MCA, either atherothrombotic permanent occlusion or embolic transient occlusion, has been supposed to be the major cause of striatocapsular infarction. As described by Levine et al (5), hemodynamic low perfusion in association with either MCA stenosis or ICA occlusive disease also may cause striatocapsular

Fig 3. Terminal supply area infarct only in the centrum semiovale (*A*) in a patient with left ICA occlusion (*B*). Because of the sufficient cross-flow via anterior communicating artery (*C*), a terminal supply area infarct occurred only in the supraventricular white matter of the centrum semiovale. Not only the lentiform nucleus but also the corona radiata remained completely intact.

infarction. In our present series, striatocapsular infarcts were seen in 13 (81.3%) of 16 patients with MCA occlusive disease and in 11 (50%) of 22 patients with ICA occlusive disease. The relatively low incidence of striatocapsular infarction in patients with ICA occlusive disease may result from the low incidence of artery-to-artery embolism and sufficient normograde blood flow at the level of the M1 segment of the MCA via anterior and/or posterior communicating arteries. Unlike in the report by Ringelstein et al (7), in our study, only 1 of 22 patients with ICA occlusive disease was shown to have artery-to-artery embolism that occurred from intraluminal ICA thrombus.

Zülch's work (12) has shown that periventricular white matter of the corona radiata is the watershed zone between the deep and superficial territories of the MCA and that supraventricular white matter of the centrum semiovale is the watershed zone between the superficial territories of the ACA and MCA. White matter infarcts in these two regions were usually induced in patients with ICA or MCA occlusive disease and together called "terminal supply area infarcts" or "subcortical watershed infarcts" (6–8). From the pathogenetic and clinical point of view, however, these two infarcts should be discriminated. In patients with MCA occlusion, the centrum semiovale in our series never was impaired, probably because of the development of rich leptomeningeal anastomotic network between the superficial territories of the ACA and MCA. On the other hand, the centrum semiovale is the farthest area for the normograde collateral blood flow to reach and it may be very susceptible to ischemia in patients with ICA occlusive disease. In our study, the centrum semiovale frequently was impaired (16 [72.7%] of 22) in patients with ICA occlusive disease, whereas none of 16 patients with MCA occlusive disease had subcortical infarcts in the centrum semiovale, suggesting that large subcortical infarcts in the centrum semiovale should prompt consideration of underlying ICA occlusive disease rather than MCA disease.

On the other hand, the corona radiata is the terminal supply area of the MCA, and infarction occurs in this region with high incidence in patients with MCA occlusive disease. In our study, all 10 patients with MCA occlusion had not only striatocapsular infarcts but also corona radiata lesions. The corona radiata was involved in 13 (81.3%) of 16 patients with MCA occlusive disease. Contrarily, corona radiata lesions were seen in only 9 (40.9%) of 22 patients with ICA occlusive disease, probably resulting from sufficient collateral flow in the lenticulostriate arteries.

In conclusion, MCA occlusive disease frequently produces striatocapsular and/or corona radiata infarcts but in our series was never associated with centrum semiovale lesions, which usually were associated with ICA occlusive disease. The centrum semiovale is more susceptible to ischemia in patients with ICA occlusive disease than the striatocapsular regions and the corona radiata.

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