Early Collateral Blood Supply and Late Parenchymal Brain Damage in Patients With Middle Cerebral Artery Occlusion

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We angiographically studied 80 patients within 6 hours after the onset of ischemic supratentorial infarction. From this group we selected 36 patients with middle cerebral artery occlusion who survived. In these 36 patients, we compared the presence of a collateral blood supply during the early phase with the extent of final parenchymal brain damage obtained by computed tomography 3 months after the event. The presence of a collateral circulation during the first few hours after the stroke reduced the size of the final parenchymal brain damage in patients with middle cerebral artery stem-trunk occlusion. The collateral blood supply was more efficient in patients who had no significant stenosing lesions of the extracranial internal carotid artery.

Our data confirm that the lenticulostriate arteries are end arteries not supplied by collateral blood vessels and suggest that lesions formerly thought to be caused by hemodynamic mechanisms (watershed infarcts) or arteriolar lesions (lacunar infarcts) may be due to middle cerebral artery occlusions. (Stroke 1989;20:735-740)

The occurrence and magnitude of infarctions in the territory of an occluded artery can depend on the prompt development of an efficient collateral circulation.¹ The location and extent of ischemic brain damage are well detected by computed tomography (CT) and magnetic resonance imaging performed several days to weeks after the ictus.²⁻⁴ However, angiographic visualization of collateral vessels during this late phase of stroke may not reflect brain perfusion during the critical early phase. To evaluate the role of the collateral blood supply, we studied the relation between parenchymal brain damage assessed by CT 3 months after the acute event and angiographic findings obtained within 6 hours after the onset of symptoms in 36 patients with cerebral hemispheric infarctions.

Subjects and Methods

All patients with acute focal supratentorial neurologic deficits admitted to our department during 14 months were evaluated by general clinical and neurologic examination and early CT scanning of the brain. We included only patients observed within 4 hours after the onset of symptoms who did not show severe alterations of consciousness or life-threatening medical disease. We excluded patients with a nonischemic etiology (hemorrhages, tumors) as documented by plain CT. Using these criteria, we included 80 patients (42 men and 38 women) with a mean±SEM age of 65.2±9.6 years.⁵

Intra-arterial digital subtraction angiography was done in all 80 patients within 6 hours after the onset of their stroke. Only the presumably symptomatic carotid artery circulation was studied, employing nonionic water-soluble contrast medium diluted with saline to an iodine concentration of 200 mg/ml. In 12 patients with occlusion of the symptomatic internal carotid artery (ICA), the contralateral carotid artery circulation was studied as well. Only one patient had transient deterioration after angiography.

We considered only occlusions of the middle cerebral artery (MCA) or of its branches, divided into four types: Type I, MCA stem-trunk occlusion proximal to the lenticulostriate arteries; Type II, MCA stem-trunk occlusion distal to the lenticulostriate arteries; Type III, MCA occlusion distal to the origin of the temporal branches; and Type IV, peripheral MCA branch occlusion.

We evaluated the collateral blood supply from the anterior cerebral arteries according to criteria that took into account the rapidity of filling and the
number of branches visualized through cortical anastomoses. Collateral vessels from the posterior cerebral artery were not evaluated. We considered the collateral blood supply to be good when all potentially occluded vessels were visualized through cortical anastomoses within 5 seconds after the end of intracarotid injection. The use of digital subtraction angiography allowed us to exactly evaluate the timing of the anastomotic collateral blood flow.

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

Extracranial associated steno-occlusive pathology was also taken into account. A carotid artery was considered to be stenosed when the lumen was reduced by >70%.

CT and angiography were first evaluated independently; CT:Angio (ratio of the final CT brain damage and the early angiographic findings) was then determined. We considered CT:Angio to be negative when the 3-month brain damage by CT was less extensive than the number of occluded vessels, to be equal when the 3-month CT brain damage matched the number of the occluded vessels, and to be positive when the 3-month brain damage was greater than that expected from the angiogram.

Results

We observed an occlusion of the MCA or its branches in 47 of the 80 patients (59%). (In the 33 remaining patients, angiography showed an occlusion of the siphon in six, a nonstenosing plaque of the extracranial ICA in 11, an extracranial ICA occlusion in eight, and a normal-appearing symptomatic artery in eight.) Eleven of the 47 patients died before they could have a 3-month follow-up CT scan. Therefore, we included only 36 patients in our study (Table 1). We observed tandem (extracranial and intracranial) lesions in 23 of the 36 patients (ICA nonstenosing plaques in 11, ICA stenosing plaques in four, and ICA occlusions in eight).

Of the 36 patients, 17 had a Type I or II MCA stem-trunk occlusion (Table 1). Eight of these 17 had a good collateral blood supply; their CT lesions were deep MCA in two, partial MCA in three, superficial MCA in one, complete MCA in one, and internal border in one. In the other nine patients with Type I or II MCA stem-trunk occlusion, there was no evidence of collateral blood flow; their CT lesions were partial MCA in two, superficial MCA in one, and complete MCA in six. Of the eight patients with Type I or II MCA stem-trunk occlusion and a good collateral blood supply, CT: Angio was negative in seven (Figures 1 and 2). In the eighth patient, the presence of a good collateral blood supply, although demonstrated by angiography 175 minutes after the onset, did not limit the extension of brain damage; CT: Angio was therefore equal. Of the nine patients with Type I or II MCA stem-trunk occlusion and no collateral blood supply, CT: Angio was equal in seven (Figures 3 and 4); in the other two, CT: Angio was negative. Analysis suggested a functional efficacy of the collateral blood supply in these 17 patients with Type I or II MCA stem-trunk occlusion (p<0.05). Extracranial associated steno-occlusive pathology of ICA was observed in three of the eight patients with and in eight of the nine patients without collateral blood flow.

Table 1 also shows the data for the 19 patients with Type III or IV MCA branch occlusion; a good collateral blood supply was present in 17. In seven of these 17, CT: Angio was negative (Figures 5 and 6). In the other 10, it was either equal (in five) or positive (in five) (Figure 7). In the two remaining patients with Type III or IV MCA occlusion but without evidence of collateral blood supply, CT: Angio was equal in one and positive in the other. Extracranial associated steno-occlusive pathology of ICA was observed in one of the 17 patients with good anastomotic collateral circulation and in one of the two patients without a collateral blood supply.

Discussion

Angiography performed during the first few hours after the onset of an ischemic stroke can provide reliable information on the pathogenesis of ischemic brain damage and on the extent of occlusive pathology of the vessels. Intracranial arterial occlusion may recanalize during the first few hours or days after the acute event so that delayed angiography can be inconclusive. In addition, the presence of a collateral blood supply visualized by angiography during the late phase does not necessarily reflect its efficiency during the acute phase.

To evaluate the efficacy of the collateral blood supply and to predict expected brain damage, we compared the extent of CT lesions after 3 months with the findings of early angiography. The efficacy of the anastomotic collateral circulation in our patients with MCA stem-trunk occlusion was demonstrated by final CT brain damage that was less extensive than the occluded vascular territories (Figures 1 and 2). In only one patient with MCA stem-trunk occlusion did the collateral circulation not limit the extent of brain damage, and it is possible that in this patient collateral blood flow developed later after the occlusion than in the other
patients. Only the very early development of a collateral blood supply (and not the time of visualization of the occluded vessels of the MCA through the anterior cerebral anastomoses, conduction time) seems to be important for functional efficiency. In this patient, a decrease in perfusion cannot be considered since steno-occlusive pathology in the ICA was not present. In the two patients with Type I or II MCA stem-trunk occlusion and without a collateral blood supply in whom the final CT lesion was less extensive than expected, an anastomotic collateral circulation from the posterior cerebral artery may be postulated.

The discrepancy between the final damage demonstrated on CT and the early angiographic findings in patients with Type III or IV MCA distal occlusion and a good collateral blood supply (Figure 7) might be explained by a preexisting stem-trunk
FIGURE 1. Digital subtraction angiograms of (a) right middle cerebral artery stem-trunk occlusion proximal to lenticulostriate arteries origin (Type I) with (b) good collateral blood supply. Final computed tomographic (c) brain damage is confined to lenticulostriate arteries territory.

FIGURE 2. Digital subtraction angiograms of (a) right middle cerebral artery stem-trunk occlusion distal to lenticulostriate arteries origin (Type II) with (b) good collateral circulation. Final computed tomographic (c) brain damage consists of internal watershed lesion.

FIGURE 3. Digital subtraction angiograms of (a) right middle cerebral artery stem-trunk occlusion proximal to lenticulostriate arteries origin (Type I) without (b) anastomotic collateral blood flow. Final computed tomographic (c, d) brain damage extends to entire middle cerebral artery territory.

FIGURE 4. Digital subtraction angiograms of (a) right middle cerebral artery stem-trunk occlusion distal to lenticulostriate arteries origin (Type II) without (b) collateral blood supply. Final computed tomographic (c, d) brain damage involves entire superficial territory of middle cerebral artery.
occlusion determining a more extensive tissue damage, followed by subsequent migration of the embolus into the more distal MCA branches. All patients with Type I MCA occlusions developed final brain damage that involved the deep territories, even in the presence of an adequate cortical collateral blood flow (Figure 1). This confirms that the lenticulostriate vessels are end arteries with no possibility of anastomotic collateral blood supply. Similar data have been presented by Caplan et al and by Saito et al who reported the development of giant lacunes in the basal ganglia of patients with occlusion of the MCA proximal to the lenticulostriate branches and yet good collateral circulation.

Seven patients developed internal border brain CT damage, and all seven had an MCA occlusion of Types II, III, or IV (Figures 2 and 5). Such lesions might therefore be due to an occlusion of intracranial vessels rather than to extracranial carotid pathology on a hemodynamic basis, as previously thought. It is possible that in these patients, the anastomotic collateral circulation had revascularized the gray and not the white matter, which is known to be in a terminal territory of the cortical branches. A similar observation applies to the two patients with lacunar infarcts (Figure 6) localized in the white matter of the corona radiata, which resulted from peripheral MCA branch occlusion.

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References


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