Middle Cerebral Artery Occlusion: Correlation of Computed Tomography and Angiography With Clinical Outcome

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The clinical outcome of 40 cases with middle cerebral artery (MCA) occlusion was examined in relation to the site of occlusion and the findings on computed tomography (CT). Patients were treated conservatively without surgery. A few had decompressive craniotomy when necessary. Outcome in 7 (18%) was good, in 6 (15%) moderate, and in 15 (38%) severe; 12 (30%) died by the follow-up at 3 months. In cases with occlusion at the origin of the MCA, hypodensity on CT scan was usually localized to the basal ganglia, presumably because of collateral circulation through the anterior cerebral arteries; the outcome in these patients was not always favorable. Cases with occlusion of the trunk or branch vessels always showed marked CT hypodensity, and clinical outcome was poor. To assess quantitatively the extent of collateral circulation, the conduction time of contrast medium from the intracranial siphon (IC) to the insular portion of the MCA (M2) through the anterior cerebral arteries was calculated on serial carotid angiograms obtained within 24 hours after stroke onset. An IC–M2 time of 5 seconds was a critical indicator of whether extensive CT hypodensity would develop (the rule of 5 seconds). Furthermore, this method predicted the appearance and extent of infarction before CT revealed hypodensity. The significance of acute reconstructive surgery is also discussed. (Stroke 1987;18:863-868)

There have been surprisingly few reports of the prognosis of patients with major cerebral arterial branch occlusions, particularly of the middle cerebral artery (MCA).1-6 The prognosis of cases with MCA occlusion varies widely in the literature, with mortality ranging from 5 to 40%, and seems to depend to a great extent on the site of occlusion. However, only Lascelles and Burrows7 and Moulin et al8 examined the relation between the site of occlusion of the MCA and the patient's prognosis. Recent advances in computed tomography (CT) technology have been helpful in clarifying the exact location and extent of the cerebral infarct. We found, however, no report on the prognosis of patients with MCA occlusion in relation to the findings on CT.

Reports on reconstructive vascular surgery, such as embolectomy, are increasing.7-12 The results of such surgery have been varied, and the indications for surgery are not always clear. Positron emission tomography (PET) can demonstrate misery perfusion in the acute stage of cerebral ischemic disease.13 Only Baron et al14 report the results of operations in patients in whom misery perfusion was identified preoperatively on PET scan.

Here we correlate the site of MCA occlusion, the location and extent of infarct on CT, and the clinical disability of patients studied within 1 week after onset. A method to measure quantitatively the collateral circulation on serial carotid angiograms is discussed. This method predicts the extent of infarction before the CT becomes positive in some patients with acute MCA occlusion.

Subjects and Methods

During the last 5½ years, 413 patients with cerebral ischemic disease were admitted to our clinic within 1 week after the onset of symptoms. Of 309 cases with angiographic examination, 46 (14.9%) were diagnosed as MCA occlusion and 27 (8.7%) as extracranial carotid occlusion. Of the 46 cases of MCA occlusion, the last 6 received reconstructive vascular surgery in the acute stage; the remaining 40 were treated conservatively (though some underwent decompressive surgery) and are surveyed in this study. Twenty-four were men and 16 women; their ages ranged from 38 to 86 with an average of 66.5 years (Figure 1). Of these 40 cases, 26 (65%) were hypertensive, 23 (58%) smoked, 3 (7.5%) showed signs of diabetes mellitus, and 3 (7.5%) had hypercholesterolemia. On angiograms, 3 (7.5%) demonstrated a slightly irregular narrowing of the cervical internal carotid artery (ICA) ipsilateral to the occluded MCA. Electrocardiogram (ECG) showed atrial fibrillation in 13 cases (33%) and myocardial ischemic changes in 9 (23%). Of these 40 cases, 19 were admitted on the day of onset (Day 0), 9 on Day 1, 9 on Day 2, and the remaining 3 on Day 5 or 6. The involved artery was on the left in 25 cases and on the right in 15.

CT and serial carotid angiography were performed immediately after admission. CT was repeated every day or every other day for 1 week. When edema and...
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mass effect due to cerebral infarct increased and patients showed any signs of uncal herniation, decompressive craniotomy was performed. Seven patients underwent this operation.

Cerebral angiography was carried out by direct puncture, and angiograms were obtained at 0.5-second intervals to the fourth second and at 1-second intervals until the tenth second after rapid injection of 10 ml of 60% Conray (Daichi Pharmaceutical Co., Ltd., Tokyo, Japan). In 21 cases with an occlusion of the MCA trunk or major branches who were admitted within 24 hours after the onset, the time for conduction of the contrast medium from the intracranial carotid siphon (IC) to the insular portion of the MCA (M2) through the anterior cerebral artery was calculated on angiograms, and IC-M2 time was determined (Figure 2).

In each case the CT showing the maximum extent of hypodensity due to infarct was selected and classified as Type I, basal ganglia type, in which hypodensity was localized in the basal ganglia, putamen, and/or centrum semiovale; Type II, extensive subcortical type, in which hypodensity involved the cortex and subcortical white matter of the frontal, temporal, and parietal lobes; Type III, hemispheric type, in which hypodensity extended to 1 hemisphere excluding the thalamus and occipital lobe; Type IV, branch type, in which hypodensity was localized in the territory of 1 superficial branch of the MCA; or Type V, normal type, in which hypodensity was not identified on CT. See Figure 3.

The findings on angiograms were categorized as Pattern 1, occlusion at the origin of the MCA; Pattern 2, occlusion at the MCA trunk distal to the lenticulo-striate arteries; Pattern 3, occlusion at the branches of the MCA involving > 2 branches in which only 1 M2 branch was demonstrated on angiograms; or Pattern 4, occlusion of 1 branch of M2. See Figure 4.

The outcome of the patients was categorized at 3 months after the onset according to their level of independence: Good, patient is independent with no or minimal neurologic deficit; Moderate, patient has neurologic impairment but is independent; Severe, patient shows severe neurologic symptoms and is dependent in daily life or is vegetative; or Dead.

Results

Site of Occlusion, Findings on CT, and Outcome

The outcome of patients is demonstrated in relation to the site of MCA occlusion and the findings on CT in Figure 5.
Of 17 cases with Pattern 1 occlusion, 12 (71%) showed Type I hypodensity and 5 showed Types II or III. In the 12 Type I cases, the pattern of occlusion showed relatively good collateral circulation through the anterior cerebral artery as described below. Hypodensity of the cortical and subcortical tissues did not occur, and only the territories of the lenticulostriate arteries most remote from the collateral system became infarcted. However, outcome in only 5 of the 12 Type I cases was Good or Moderate at 3 months after onset. Outcome in the remaining 7 Type I cases and all 5 Type II or III cases was Severe or Dead at 3 months after the onset.

MCA occlusion of the trunk (Pattern 2) or branches (Pattern 3) usually produced a huge infarct, and outcome in all cases except 2 was Severe or Dead (Figure 5). In cases with Pattern 2 or 3 occlusions, collateral circulation was usually poor, and the cortical and subcortical tissues were also infarcted.

In cases with occlusion of 1 branch of the MCA, hypodensity was localized and the outcome was Good or Moderate at follow-up.

**IC-M2 Time and Infarction**

The correlation between IC-M2 time and the type of hypodensity on CT was examined in cases with MCA occlusion of the trunk or branches. Of 21 cases in which angiography was performed within 24 hours after the onset, all 9 with IC-M2 time of <5 seconds showed Type I hypodensity. In contrast, 12 patients whose IC-M2 time was >5 seconds always demonstrated Type II or III hypodensity on CT (Figure 6). It seems, therefore, that an IC-M2 time of 5 seconds indicated whether an extensive hypodensity would develop. When IC-M2 time was examined in relation to the site of occlusion, cases with occlusion at the origin of the trunk usually showed good collateral circulation. However, in cases with occlusion of the trunk or branches, the IC-M2 time was usually >5 seconds and a large hypodensity developed.
Outcome

Of 40 cases with MCA occlusion, outcome was rated as Good in 7 (18%), Moderate in 6 (15%), and Severe in 15 (38%), while 12 (30%) were dead at follow-up 3 months after the onset. Of 7 cases undergoing decompression surgery, 4 were rated Severe and 3 died, mainly of complications (heart failure or pneumonia). There were no deaths due to cerebral herniation following a large infarction and edema. Of 12 deaths, 3 occurred within 2 weeks of onset, 4 within 1 month, 4 within 2 months, and 1 within 3 months.

Discussion

The prognosis of patients with cerebral occlusive disease depends on age, the site and nature of the occlusion, and collateral circulation.

Age of Patients and Timing of Admission

In this series, 90% of patients were >60 years of age and 50% were >70. It can be assumed that in elderly patients, complications of the cardiopulmonary system occurred while they were bedridden and, in fact, 30% of these patients died of complications within 3 months of onset (Figure 1).

In the series of Kaste and Waltimo,3 of 78 cases with MCA occlusion, only 5% died and 68% recovered to a fully independent state. These authors attributed their good results to the fact that their patients were young (mean age was 40), reflecting on their method of selecting patients; elderly and seriously ill patients were not referred to their clinic, and elderly patients often were not examined angiographically.

It is surprising that there have been no reports in which timing of the patients' admission from the onset was detailed. Since our clinic is one of only a few hospitals for cerebrovascular diseases in this district of Japan, most patients in this series were admitted to our hospital immediately after the onset without prior selection. Angiography was performed on as many patients as possible to determine the site of occlusion.

Site of Occlusion

The site of occlusion is a crucial factor in determining the prognosis of patients. Lascelles and Burrows4 analyzed this relation and classified the site into 3 categories: 1) origin of the trunk, 2) along the trunk, and 3) involving >2 branches. They concluded that the site of occlusion did not influence prognosis. Their Category 2 was equivalent to our Pattern 2 and their Category 3 corresponded to our Pattern 3. Considering the facts that our patients with Pattern 2 or 3 occlusion showed almost the same prognosis as described above and that there were few cases with Category 1 (our Pattern 1) occlusion (only 5 of 42 cases), their conclusion seems to be arguable.

Collateral Circulation

It seems probable that the size of the infarct depends mainly on the collateral circulation to the arteries distal to the occlusion. In most previous reports the collateral
circulation has been described qualitatively as "good," "poor," or "absent" according to findings on angiograms. In this study, a quantitative measurement of collateral circulation was tried; the posterior circulation was not assessed, and it is therefore impossible to give complete data on collateral supply. However, in cases with MCA occlusion, the main collateral pathways to the territory of the MCA are through the anterior cerebral artery, and our method of measuring IC-M2 time seems to be very informative for predicting the extent of collateral circulation. When IC-M2 time was <5 seconds, collateral circulation was good, cortical and subcortical infarcts did not develop, and the infarction was localized in the basal ganglia. On the contrary, when IC-M2 time was >5 seconds, collateral circulation was poor and a large infarct involving the cerebral hemisphere developed (the rule of 5 seconds). It should be emphasized that this rule predicts the extent of infarction before CT scan reveals hypodensity. It should also be stressed that good collateral circulation and a localized small infarct in the basal ganglia did not always mean a good outcome, particularly in the elderly patients in our series. Of our 17 cases with Pattern 1 occlusion, 12 did not have a large infarct, yet only 5 showed a good outcome; the remaining 7 were severely disabled or died. These results seem to be due mainly to the fact that infarcts in the basal ganglia, even if small, often produce severe neurologic deficits, and elderly patients with such severe symptoms sometimes die of cardiopulmonary complications during bed confinement. This relation between collateral circulation, size of infarct, and prognosis can explain the conclusion by Lascelles and Burrows that angiographic evidence of collateral circulation does not influence prognosis.

Nature of Occlusion (Embolic or Thrombotic)

Lhermitte et al. reported only 2 cases of arteriosclerotic thrombosis in an autopsy series of 31 cases with MCA occlusion. Spontaneous recanalizations have been observed in 24–50% of cases with MCA occlusion on repeated angiography, and in the great majority the nature of the MCA occlusion seemed embolic.

In our series of 14 cases in which repeated angiograms were obtained, 8 showed recanalization, and all were cases with occlusion distal to the lenticulostriate arteries. In cases with MCA occlusion at the origin, IC-M2 time was often <5 seconds and collateral circulation was good. Intracranial, particularly MCA, occlusion is more frequent in Japan than extracranial ICA occlusive diseases. We also often observe stenotic change at the origin of the MCA. Furthermore, only 5 of 17 cases (29%) with MCA occlusion at the origin showed atrial fibrillation or ischemic changes on ECG at admission, although 50% of the cases with MCA occlusion along the trunk or at the branches had ECG abnormalities. From these observations, we conclude that MCA occlusion at the origin was due to sclerotic thrombosis, that good collateral circulation developed during the course of the disease, and that hypodensity on CT was usually localized in the basal ganglia. On the other hand, MCA occlusion along the trunk or at the branches was of embolic origin and resulted in a large hypodensity on CT due to poor collateral circulation.

Rule of 5 Seconds and Acute Reconstructive Surgery

Recently there have been a number of reports of reconstructive vascular surgery, such as embolectomy...
of intracranial cerebral arteries, in the acute stage of ischemic diseases. In this regard, the most important and controversial problems have been with indications for such operations in patients with impending stroke. Theoretically, patients who show an ischemic penumbra are good candidates for such surgery, while it is considered harmful to operate on patients with already-infarcted brains. An ischemic penumbra has been diagnosed more often early after stroke onset, and such time-consuming diagnostic methods as PET are not so useful in this period. There has been only 1 report in which the penumbra was documented by PET scan preoperatively in a case with transient ischemic attacks. On the other hand, CT scan can disclose hypodensity following ischemia no sooner than 8 hours following onset and, as such, is also not useful for early diagnosis.

Measuring IC-M2 time and applying the rule of 5 seconds may be very informative immediately after onset in predicting the extent of infarction in cases with MCA occlusion. In our cases with MCA occlusion in which IC-M2 time is <5 seconds, large subcortical infaracts did not develop even with conservative treatment. In patients showing good collateral circulation on angiography, reconstructive surgery often improved their neurologic symptoms. Meyer et al reported that embolectomy of MCA occlusion was effective in cases with good collateral circulation.

Illustrative Case History

A 66-year-old man was admitted 3 hours after the onset of left hemiparesis and dysarthria. CT demonstrated no hypodensity. An angiogram disclosed MCA occlusion at the origin (Figure 7), and IC-M2 time was 6.5 seconds. Embolectomy was begun 7 hours after onset, and circulation in the MCA was obtained 2 hours later. He soon recovered to his preoperative state. However, on the third postoperative day, he aspirated at breakfast, and severe pneumonia followed. He died of heart failure on the nineteenth postoperative day. Angiograms on the first postoperative day showed patency of the MCA. Hypodensity was localized in the basal ganglia on CT.

IC-M2 time can predict whether extensive hypodensity will develop, even immediately after the onset and before CT demonstrates hypodensity. Therefore, this rule of 5 seconds may be informative in judging an indication for surgery or in estimating the significance of acute reconstructive surgery.

References

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