CT and Arteriographic Comparison of Patients with Transient Ischemic Attacks — Correlation with Small Infarction of Basal Ganglia

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SUMMARY Fifty patients presenting clinically with TIAs were examined angiographically. Twenty one patients (42%) had no abnormality. Twenty patients (40%) had stenosis or occlusion in the MCA, ACA or intracranial carotid, whereas 11 (22%) had involvement of their extracranial internal carotid artery. Seven of the 28 CTs performed showed basal ganglia infarcts. This suggests that the cause for the TIA was an infarct in the vascular territory of a lenticulostriate artery.

TRANSIENT ISCHEMIC ATTACKS (TIAs) are reversible episodes of neurological deficits resolving completely within 24 hours caused by focal neurological ischemia. TIAs are usually attributed to microemboli dislodged from ulcerated atheromatous plaques. Neuropathologic studies show less atherosclerotic change of the cervical portion of the internal carotid artery in the Japanese than in Americans.

We evaluated 50 patients with TIAs who had been examined by cerebral angiography and computed tomography (CT). Few angiographic abnormalities were seen in the cervical portion of the internal carotid artery. Seven of the 28 TIA patients who had CT scans had small areas of low density in the basal ganglia. We suggest that the above findings represent the characteristic nature of TIAs in the Japanese.

Materials and Methods

Fifty patients (42 males, 8 females) with transient cerebral ischemic attacks in the carotid arterial system were evaluated clinically and angiographically at the Mihara Memorial Hospital between April 1974 and August 1978. CT scans were performed in 28. The mean age was 56.6 years.

The angiograms were analyzed for the presence of atherosclerotic plaques in the internal and external cranial arteries. CT scans were obtained with a Hitachi CT-H 250 (matrix 256 × 256) within one month of the cerebral ischemic attacks in the carotid arterial system. CT scans were obtained with a Hitachi CT-H 250 (matrix 256 × 256) within one month of the cerebral ischemic attacks in the carotid arterial system. CT scans were obtained with a Hitachi CT-H 250 (matrix 256 × 256) within one month of the cerebral ischemic attacks in the carotid arterial system. CT scans were obtained with a Hitachi CT-H 250 (matrix 256 × 256) within one month of the cerebral ischemic attacks in the carotid arterial system. CT scans were obtained with a Hitachi CT-H 250 (matrix 256 × 256) within one month of the cerebral ischemic attacks in the carotid arterial system. CT scans were obtained with a Hitachi CT-H 250 (matrix 256 × 256) within one month of the cerebral ischemic attacks in the carotid arterial system. CT scans were obtained with a Hitachi CT-H 250 (matrix 256 × 256) within one month of the cerebral ischemic attacks in the carotid arterial system. CT scans were obtained with a Hitachi CT-H 250 (matrix 256 × 256) within one month of the cerebral ischemic attacks in the carotid arterial system. CT scans were obtained with a Hitachi CT-H 250 (matrix 256 × 256) within one month of the cerebral ischemic attacks in the carotid arterial system.

Results

Clinical Picture

Motor disturbances were observed in 44 patients (88%). Motor symptoms were accompanied by sensory disturbances in 7 patients (14%) and by dysarthria in 5 patients (10%). Thirty-five (70%) patients had a hemiparesis and 9 (18%) had a monoparesis (table 1). An isolated sensory disturbance or aphasia was found in 4 (8%) and 2 patients (4%), respectively. Amaurosis fugax was not a presenting symptom in this group.

Although the frequency of TIAs was variable, it occurred fewer than four times in 68% of the patients (fig. 1). In most patients successive TIAs occurred within 7 days and typically occurred within the same day. The TIAs lasted less than 30 minutes in most of the patients, with 50% lasting from 1 to 3 minutes. The same clinical pattern occurred in the initial and subsequent TIAs in 26 patients (76.5%); somewhat different patterns in 2 patients (5.8%) and completely different patterns in 6 patients (17.7%). Hypertension was found in 44% with 20 patients having systolic readings at or above 160 mm Hg and the remainder had diastolic readings in the hypertensive range (above 95 mm Hg).

Angiographic Findings (table 2)

Twenty-nine of the 50 patients (58%) revealed stenotic or occlusive changes in the intracranial or extracranial arteries. The remaining 21 patients had normal angiograms. Only 9 patients had extracranial lesions as compared to 18 patients with intracranial changes. Table 3 gives a comparison of the grade of stenotic changes in the extra- and intracranial lesions. The most frequent site of severe stenosis was in the middle cerebral artery.

Computed Tomography

Table 4 shows the correlation between angiographic and CT results. Twenty of the 28 patients had normal CTs of whom 13 were examined by CT scan within one month. Seven patients had the CT appearance of basal ganglia infarcts and 2 of these patients showed positive contrast enhancement within 7 days after onset (table 5). Five of these patients had stenotic middle cerebral arteries demonstrated angiographically. Of the 20 patients with a normal CT, 10 patients had changes in the intracranial internal carotid or middle cerebral artery and 4 in the extracranial ICA. Although the numbers are small, MCA stenosis was more common in those patients with infarcts demonstrated on
TABLE 1  Symptoms of TIAs

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Number of cases (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor disturbance</td>
<td>32 (64%)</td>
</tr>
<tr>
<td>Motor disturbance + Speech</td>
<td>5 (10%)</td>
</tr>
<tr>
<td>disturbance + Sensory disturbance</td>
<td>7 (14%)</td>
</tr>
<tr>
<td>Sensory disturbance</td>
<td>4 (8%)</td>
</tr>
<tr>
<td>Aphasia</td>
<td>2 (4%)</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
</tr>
</tbody>
</table>

CT. Angiograms and CT findings for 2 patients with middle cerebral artery stenosis and contrast enhancement of basal ganglia infarcts are shown in figures 2A, 2B, 3A and 3B. It appears that a small infarction in the territory of the lenticulostriate artery may be the cause of the TIAs.

Six of the 7 patients with infarcts had less than 4 TIAs. A constant clinical pattern (e.g., contralateral hemiparesis) and a short interval between TIAs seemed characteristic of this group.

The basal ganglia infarcts spared the internal capsule. They were located in the caudate nucleus, putamen or thalamus as shown in figure 4 and 5, which compare the lesions in patients with hemiplegia and cerebral infarction.

Conclusion

TIAs are usually attributed to arterial stenosis or occlusion,¹ or to microemboli dislodged from ulcerat-

FIGURE 1. Frequency of TIAs.

TABLE 2  Angiographical Findings of TIAs

<table>
<thead>
<tr>
<th>Type</th>
<th>Number of cases (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>21 (2) 42%</td>
</tr>
<tr>
<td>Stenosis or Occlusion</td>
<td>29 (1) 58%</td>
</tr>
<tr>
<td>Extracranial arteries</td>
<td>9</td>
</tr>
<tr>
<td>Intracranial arteries</td>
<td>18 (1)</td>
</tr>
<tr>
<td>Extra- + Intracranial arteries</td>
<td>2</td>
</tr>
<tr>
<td>( ) Aneurysm ( + )</td>
<td></td>
</tr>
</tbody>
</table>
Table 3  Sites of Angiographical Lesions in 29 TIA Cases

<table>
<thead>
<tr>
<th>Site of lesion</th>
<th>Extra-cranial</th>
<th>Intracranial</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade of stenosis</td>
<td>ICA</td>
<td>MCA</td>
<td>M2</td>
</tr>
<tr>
<td>Mild stenosis &lt;50%</td>
<td>10</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>Severe stenosis &gt;50% or Occlusion</td>
<td>(1)</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>11</td>
<td>4</td>
<td>13</td>
</tr>
</tbody>
</table>

ICA  internal carotid artery
MCA  middle cerebral artery
M1  horizontal portion of the middle cerebral artery
M2  branch of the middle cerebral artery
ACA  anterior cerebral artery

Figure 2a. Lt: Plain CT. Small low density areas were found on Rt basal ganglia after 3 days from last episode (case No. 1) Rt: Positive contrast enhancement 17 days after last episode.

Figure 3a. Plain CT. Low density area around the Rt anterior horn was found 1 day after last TIA. (case No. 2) Rt: Positive contrast enhancement 17 days after last TIA.

Figure 2b. Angiograms of case No. 1 Lt: Stenosis of MCA (M1) Rt: Normal findings of ICA.

Figure 3b. Lt: Angiogram reveals stenosis of Rt MCA (M1) Rt: Normal findings of Lt ICA.
ed atherosclerotic plaques.\textsuperscript{2,3} However, according to our results involvement of the middle cerebral artery (either stenosis or occlusion) was a more common finding than for the cervical portion of the internal carotid artery; this appears to be characteristic of the Japanese.\textsuperscript{4-7} We looked for the cause of the TIAs occurring in the territory of the lenticulostriate artery.\textsuperscript{8} Among our 50 patients, 21 revealed no angiographical abnormality, 18 displayed intracranial lesions, 9 extracranial cervical lesions and 2 both intra- and extracranial lesions. In a prospective study of 160 patients with TIAs due to atherosclerosis described by Toole,\textsuperscript{9} 141 (88\%) had arteriographically visible disease of the extracranial cervical arteries. One hundred and ten of these patients had constriction of the lumen by 50\% or more, and 13 had ulcerated plaques. Thus, patients,
without angiographic abnormalities and with intracranial lesions were more frequent in our study than in this previous report. On the other hand, Perrone had reported a relatively high percentage (34%) of permanent morphological small lesions in patients with TIA.

Patients with small ischemic lesions appeared to have fewer TIAS separated by shorter intervals than those with normal CT scans. Recurrent attacks of the same pattern of TIA was characteristic for the patients with small ischemic lesions. Therefore, these small infarctions in the basal ganglia may represent the cause of the TIA.

### References

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