Focal Parenchymal Lesions in Transient Ischemic Attacks: Correlation of Computed Tomography and Magnetic Resonance Imaging

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SUMMARY  Twenty-two patients with the clinical diagnosis of transient ischemic attacks were prospectively evaluated by computed tomography (CT) and proton magnetic resonance imaging (MRI). Nineteen patients also underwent cerebral angiography. The MRI studies were performed with a prototype superconductive magnet using a 0.6 Tesla or a 1.5 Tesla magnetic field. Two pulse sequence techniques were used resulting in T1 and T2 weighted images. All studies were interpreted descriptively by a single neuroradiologist in a blinded fashion, with special attention to focal parenchymal abnormalities. Patients with previously documented clinical strokes or reversible ischemic neurologic deficits lasting more than 24 hours were excluded. The CT scans revealed focal areas of abnormalities in 7 of 22 patients (32%), while the MRI scans showed focal changes in 17 patients (77%). All the CT lesions were clearly visualized on MRI. The MRI changes were better seen on T2 weighted images as areas of increased signal intensity. There was a marked preponderance of deep hemispheric lesions on both CT and MRI studies. Focal parenchymal abnormalities were not limited to the symptomatic vascular territory. We conclude that MRI reveals focal parenchymal changes in the majority of patients with transient ischemic attacks and is more sensitive than late generation CT scans. However, specificity appears to be poor, and may limit clinical usefulness. While the significance of the MRI "lesions" remains speculative, they may represent markers of chronic cerebrovascular disease in these patients.

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TRANSIENT ISCHEMIC ATTACKS (TIA's) are episodes of focal neurologic dysfunction referable to a specific arterial distribution, and resolving within 24 hours. The classical clinical concept initially implied a truly reversible process without permanent parenchymal damage. More recently, it has been recognized that some patients presenting with TIA's have actually sustained cerebral infarction with timely resolution of signs and symptoms. Some authors argue for a separate classification of TIA patients with parenchymal lesions, and suggest a different therapeutic approach to these patients. The advent of late generation computed tomographic (CT) scanning and magnetic resonance imaging (MRI) has added a new dimension to the problem. These more sensitive imaging techniques are identifying an increasing number of focal parenchymal abnormalities in TIA patients and are raising questions about the frequency, nature, and clinical significance of these lesions. In this study, we prospectively examine the CT and MRI findings in 22 TIA patients, and correlate these findings with the clinical presentation and angiographic findings.

Patients and Methods

Twenty-two patients with the clinical diagnosis of TIA's were prospectively evaluated by proton MRI and CT scanning as part of their initial evaluation at the Cleveland Clinic Hospital. These represented a group of consecutive cases evaluated by the cerebrovascular surgery and neurology services (I.A. and J.R.L.) over a six month period, and which fulfilled the study criteria outlined below. Spells of transient monocular or cerebral hemispheric ischemia were designated as carotid TIA's. Spells combining at least two of the symptoms of vertebrobasilar insufficiency (bilateral motor or sensory symptoms, objective vertigo, diplopia, dysarthria, homonymous hemianopsia) were designated as vertebrobasilar TIA's.

The CT scans were performed without contrast agents using a Picker 1200 scanner with a 512 X 512 sampling matrix and a 256 X 256 display matrix. Slice thickness was 10 mm, with each section obtained in 3.4 seconds at 80 mA and 130 KVP. The MRI studies were performed using a 0.6 Tesla or a 1.5 Tesla superconductive magnetic resonance prototype system manufactured by the Technicare Corporation. A contiguous multi-slice single echo technique was employed, with a section thickness of 10 mm. Four averages were obtained on the 0.6 Tesla system, and two averages on the 1.5 Tesla system. Two pulse sequence protocols were used. The first protocol provided T1 weighted images using 30 millisecond TE and 0.5 second TR with an examination time of 4.4 and 2.2 minutes, respectively. The second pulse sequence protocol provided T2 weighted images using 120 millisecond TE and 2 second TR, with an examination time of 18 and 9 minutes, respectively. Informed consent was obtained according to institutional guidelines.

The CT scans and T1 and T2 weighted images were randomized and reviewed descriptively by a single neuroradiologist who was blinded to the clinical and angiographic data. Special attention was directed to focal parenchymal areas of increased signal intensity. Supratentorial lesions involving cerebral cortex or subcortical white matter were arbitrarily defined as "superficial". Lesions involving the thalamus, basal gan-
logic deficits lasting more than 24 hours at any time in the past or with previously documented clinical strokes were excluded from this study. While the duration and frequency of TIA's varied widely, all the investigations were completed within one month of the most recent spell. The CT and MRI scans were performed on the same day or on two consecutive days. Patients with reversible ischemic neurologic deficits lasting more than 24 hours at any time in the past or with previously documented clinical strokes were excluded from this study.

**Results**

There were 15 males and 7 females. The mean age was 64 years. Fifteen patients had unilateral carotid TIA's, two patients had bilateral carotid TIA’s, and five patients had vertebrobasilar TIA’s. Table 1 summarizes the clinical, angiographic, CT scan and MRI findings in the 22 patients.

The CT scans showed focal areas of decreased attenuation in 7 patients (32%). Five patients had single deep lesions; the abnormality was in the symptomatic cerebral hemisphere in 4 of these patients (fig. 1), and in the contralateral hemisphere in 1 patient. One patient with vertebrobasilar TIA’s had a clinically silent superficial lesion in one occipital lobe. Another patient with vertebrobasilar TIA’s had multiple deep lesions in both cerebral and cerebellar hemispheres.

The MRI scans revealed focal parenchymal abnormalities in 17 patients (77%). The MRI abnormalities were best seen on the T2 weighted images as areas of increased signal intensity. The most prominent lesions were also visualized more faintly on the T1 weighted images as areas of decreased signal intensity (fig. 1). Five patients had single deep parenchymal lesions; these were within the vascular territory of transient ischemia in 3 of these patients. One patient with vertebrobasilar TIA’s had a focal superficial area of increased signal intensity in one occipital lobe. Eleven patients demonstrated multiple bilateral deep areas of increased signal intensity.

**Correlation of CT and MRI Findings**

All the abnormalities described on CT were clearly visualized on MRI (fig. 1 and 2). All patients with normal MRI had a normal CT, and all but two patients with single focal lesions on MRI had a normal CT (fig. 3). Six of 11 patients with multiple deep lesions on MRI had a CT scan interpreted as normal (fig. 4), while the rest demonstrated less delineated deep lesions on CT (fig. 2).

**Correlation of Ischemic Symptoms with CT and MRI Lesions**

Fifteen patients presented with unilateral carotid TIA’s. Five of these patients had focal abnormalities on CT scan; the focal abnormality was within the vascular territory of transient ischemia in 4 cases, and outside that territory in 1 case. Twelve of the 15 patients had focal abnormalities on MRI. The latter were confined to the symptomatic vascular territory in 4 cases, were outside the vascular territory of transient ischemia in only 1 case, and were bilateral in 7 cases. Therefore, of 15 patients with unilateral carotid TIA’s, 11 patients had focal parenchymal abnormalities in their symptomatic vascular territory. Two patients had bilateral carotid TIA’s. Neither patient had any focal abnormality on CT, while one showed bilateral focal abnormalities on MRI. Of 5 patients with vertebrobasilar TIA’s, 2 had focal abnormalities on CT scan. The CT lesions involved the occipital lobes in both cases. Four patients had focal abnormalities on MRI. These involved the occipital white matter or cerebellum in all cases.

**Angiographic Correlation of CT and MRI Findings**

Fifteen of the 17 patients with carotid TIA’s had bilateral carotid angiography. The angiographic findings were correlated with the presence of parenchymal lesions in the 30 cerebral hemispheres ipsilateral to the angiogrammed carotid arteries (table 2). While the numbers are too small for statistical analysis, 3 of the 4 CT abnormalities were ipsilateral to more severely diseased arteries, and all 4 abnormalities were in the territory of symptomatic lesions. No such trends were observed for MRI lesions. While MRI lesions were prevalent in the symptomatic vascular territory, they were often accompanied by lesions in the contralateral hemisphere as well.

**Figure 1.** Case 7 presented with multiple right carotid TIA’s. The angiogram showed bilateral high grade tandem stenoses of both internal carotid arteries. The CT scan revealed an area of decreased attenuation near the right caudate nucleus. The MRI images more clearly outlined this same lesion. (Right side is on reader's left).
TABLE 1  Clinical, Angiographic, CT and MRI Findings in 22 Patients with Transient Ischemic Attacks

<table>
<thead>
<tr>
<th>Age and sex</th>
<th>Presenting symptoms</th>
<th>Angiography</th>
<th>CT*</th>
<th>MRI*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 74 M</td>
<td>Left carotid TIA</td>
<td>Bilateral severe siphon stenosis</td>
<td>No focal lesions</td>
<td>Bilateral multiple deep hemispheric lesions</td>
</tr>
<tr>
<td>2 79 F</td>
<td>Left carotid TIA</td>
<td>Severe left ICA origin stenosis; mild right ICA stenosis</td>
<td>Single deep lesion in left frontal lobe</td>
<td>Bilateral multiple deep hemispheric lesions</td>
</tr>
<tr>
<td>3 76 F</td>
<td>Bilateral carotid TIA's</td>
<td>—</td>
<td>No focal lesions</td>
<td>No focal lesions</td>
</tr>
<tr>
<td>4 66 M</td>
<td>Left carotid TIA (amaurosis fugax only)</td>
<td>Bilateral moderate tandem ICA stenoses (origin and siphon)</td>
<td>No focal lesions</td>
<td>No focal lesions</td>
</tr>
<tr>
<td>5 64 M</td>
<td>Vertebralbasilar TIA</td>
<td>—</td>
<td>No focal lesions</td>
<td>No focal lesions</td>
</tr>
<tr>
<td>6 68 M</td>
<td>Vertebralbasilar TIA</td>
<td>Severe stenosis at left vertebralbasilar junction. Absent right vertebral artery. Mild carotid disease</td>
<td>Superficial lesion in left occipital lobe</td>
<td>Superficial lesion in left occipital lobe</td>
</tr>
<tr>
<td>7 58 M</td>
<td>Right carotid TIA</td>
<td>Bilateral severe tandem ICA stenoses (origin and siphon)</td>
<td>Small deep lesion near the head of the right caudate nucleus</td>
<td>Deep lesion near the head of the right caudate nucleus</td>
</tr>
<tr>
<td>8 59 F</td>
<td>Bilateral carotid TIA's</td>
<td>Bilateral mild ICA origin stenosis</td>
<td>No focal lesions</td>
<td>Bilateral multiple deep hemispheric lesions</td>
</tr>
<tr>
<td>9 41 M</td>
<td>Vertebralbasilar TIA</td>
<td>Severe stenosis of distal left vertebral artery. Occluded right vertebral artery no carotid disease</td>
<td>No focal lesions</td>
<td>Multiple deep lesions in both cerebral and cerebellar hemispheres</td>
</tr>
<tr>
<td>10 60 F</td>
<td>Left carotid TIA</td>
<td>Moderate left ICA origin stenosis</td>
<td>No focal lesions</td>
<td>Single deep lesion in left frontal lobe</td>
</tr>
<tr>
<td>11 55 M</td>
<td>Right carotid TIA</td>
<td>Bilateral moderate ICA origin stenosis</td>
<td>No focal lesions</td>
<td>Bilateral multiple deep hemispheric lesions</td>
</tr>
<tr>
<td>12 73 M</td>
<td>Right carotid TIA</td>
<td>Right ICA occlusion at origin — moderate left ICA origin stenosis</td>
<td>No focal lesions</td>
<td>Single deep lesion in the head of the left caudate nucleus</td>
</tr>
<tr>
<td>13 68 M</td>
<td>Left carotid TIA</td>
<td>Normal</td>
<td>Small lesion near genu of left internal capsule</td>
<td>Bilateral multiple deep hemispheric lesions</td>
</tr>
<tr>
<td>14 62 F</td>
<td>Vertebralbasilar TIA</td>
<td>Severe left vertebral artery stenosis proximal to PICA. Right vertebral artery occlusion. Mild carotid disease</td>
<td>Small lesions in anterior limb of right internal capsule and deep left occipital lobe</td>
<td>Multiple deep lesions in both cerebral and cerebellar hemispheres</td>
</tr>
<tr>
<td>15 68 F</td>
<td>Left carotid TIA</td>
<td>Bilateral moderate ICA origin stenoses</td>
<td>No focal lesions</td>
<td>Single lesion in posterior limb of left internal capsule</td>
</tr>
<tr>
<td>16 58 F</td>
<td>Right carotid TIA</td>
<td>Right middle cerebral artery aneurysm. No significant stenosis</td>
<td>No focal lesions</td>
<td>No focal lesions</td>
</tr>
<tr>
<td>17 49 M</td>
<td>Right carotid TIA</td>
<td>Right ICA occlusion at the origin</td>
<td>No focal lesions</td>
<td>Single small lesion in posterior limb of left internal capsule</td>
</tr>
<tr>
<td>18 60 M</td>
<td>Right carotid TIA</td>
<td>Normal</td>
<td>No focal lesions</td>
<td>Bilateral multiple deep hemispheric lesions</td>
</tr>
<tr>
<td>19 77 M</td>
<td>Left carotid TIA</td>
<td>—</td>
<td>Small lesion in right globus pallidus</td>
<td>Bilateral multiple deep hemispheric lesions</td>
</tr>
<tr>
<td>20 57 M</td>
<td>Left carotid TIA</td>
<td>Near occlusion at origin of left ICA</td>
<td>Single deep lesion in left frontal lobe</td>
<td>Prominent deep lesion in left frontal lobe, and bilateral multiple small deep hemispheric lesions</td>
</tr>
<tr>
<td>21 58 M</td>
<td>Vertebralbasilar TIA</td>
<td>Moderate distal basilar artery stenosis</td>
<td>No focal lesions</td>
<td>Bilateral multiple deep hemispheric lesions</td>
</tr>
<tr>
<td>22 57 M</td>
<td>Right carotid TIA</td>
<td>No significant carotid or vertebral artery stenosis</td>
<td>No focal lesions</td>
<td>No focal lesions</td>
</tr>
</tbody>
</table>

*A "lesion" is defined as a parenchymal region of decreased attenuation on CT scan, or of increased signal density on T2 weighed MRI scan.
Discussion

The pathogenesis of TIA's includes a wide variety of mechanisms including hemodynamic disturbances, microembolism, vasospasm, and platelet hyperaggregability. However, little is known about the neuropathologic correlates of TIA's. An unknown number of patients sustain infarctions with resolution of signs and symptoms within the 24 hour time frame of a clinical TIA. Neuropathologic studies are difficult to interpret since the cerebrovascular disease often progresses to more advanced and symptomatic stages, and can be complicated by terminal events prior to postmortem examination. Because of the extent of the accompanying vascular pathology in many patients, and the transitory nature of the symptoms, one can seldom differentiate clinically a new cerebral infarction with transient signs from an old silent infarction with transient symptomatic ischemia in nearby brain.

Furthermore, clinicians are appropriately questioning whether parenchymal alterations of similar pathophysiology and morphology should be classified and treated differently when, because of location, they present with different symptomatologies. In addition to accurate delineation of the vascular pathology, great emphasis is being placed on defining the extent of parenchymal damage in the individual patient prior to selecting appropriate therapy. New and rapidly changing neuroradiologic techniques are now available to the practitioner, yet the sensitivity and specificity of these techniques is not well understood, and the clinical significance of the lesions they may delineate remains unclear.

In this study, we correlated the findings on late generation CT scans and proton MRI scans with the angiographic and clinical characteristics in 22 TIA patients. Patients with clinically recognized completed strokes or reversible ischemic neurologic deficits lasting more than 24 hours at any time in the past were excluded. All patients were admitted to the study prospectively based on their clinical diagnosis, and prior to the imaging studies. The CT and MRI protocols were those adopted for routine clinical examination at our institution. While the CT and MRI scans were mixed and reviewed blindly by a single investigator, their reading was still subject to the uncertainties of "overinterpretation" or "underinterpretation" observed in daily clinical practice. Since the interval between the most recent spell and the CT scan varied widely, the data from contrast enhanced CT scans was impossible to interpret, and was excluded. Similarly, the small number of patients precluded significant correlations with the frequency and duration of the spells, especially since several patients had multiple events of varying duration (each resolving within 24 hours).

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retrospective studies. These lesions may represent ischemic parenchymal alterations presenting as TIA’s, or may reflect clinically silent morphological changes incidentally preceding or accompanying the spells. The CT lesions were more frequent in (but not limited to) the symptomatic regions of the brain, and the territory of more severely diseased arteries.

The MRI showed focal parenchymal changes in 77% of the patients. Other formal studies on TIA patients are still lacking. Because of the high sensitivity of MRI in experimental and clinical infarction, some authors have predicted a similarly high sensitivity in TIA patients. Proton MRI has detected parenchymal changes as early as 30 minutes after experimental artery occlusion in animals, presumably prior to histologically detectable infarction. The MRI lesions may represent focal flow disturbances, subtle changes in water content or distribution, cellular reactions, or frank tissue necrosis. Identical lesions have been described in degenerative diseases, demyelinating lesions, and are not necessarily related to the symptomatic presentation.

The lack of specificity, and the high frequency of bilateral changes will obviously limit the clinical usefulness of MRI. Yet, CT and MRI may provide complementary information. Lesions observed only faintly on CT are more clearly and extensively delineated on MRI. Also, many of the MRI lesions can be detected on retrospective examination of the CT scan, when they were missed on the initial CT interpretation. Periventricular leukomalacia, often a vague and subtle finding on CT, can be clearly demonstrated as multiple discrete bright focal areas on T2 weighted MRI (fig. 4). In this regard, the use of MRI has already added a useful dimension to the evaluation of TIA patients. The predominance of deep lesions on both CT and MRI studies deserves comment. The lesions may simply reflect a higher sensitivity of the imaging techniques in deeper areas of the brain. Such differential sensitivity has yet to be proven. On the other hand, the lesions may represent ischemic changes in deeper areas of the brain as a result of chronic cerebrovascular disease. Atherosclerosis and other chronic vascular changes such as lipohyalinosis can alter the hemodynamic patterns and the autoregulatory reserves, making certain areas of the watershed zone more vulnerable to transient ischemia. This can occur with or without clinically overt symptoms. Such changes in deep areas of brain should be carefully characterized in experimental models of transient ischemia and should be carefully sought in neuropathologic studies. Their temporal relationship to the ischemic spell, potential reversibility, and impact on prognosis should be addressed in future research. We propose that these lesions may represent markers of chronic cerebrovascular disease. A large scale correlation of these MRI changes with age and risk factors for cerebrovascular disease in the general population is currently underway at our institution. Until then, we shall humorously refer to deep focal MRI lesions as “U.B.O.’s” (Unidentified Bright Objects), and interpret them with great caution.

References


Figure 4. Case 1 presented with left carotid TIA’s. Angiography revealed bilateral high grade carotid siphon stenosis. The CT scan was read as normal, while the T2 weighted MRI images showed bilateral deep areas of increased signal intensity.
Focal parenchymal lesions in transient ischemic attacks: correlation of computed tomography and magnetic resonance imaging.
I Awad, M Modic, J R Little, A J Furlan and M Weinstein

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