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 superconducting 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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logic deficits lasting more than 24 hours at any time in
diologist via retrograde femoral artery catheterization.
raphy was usually performed as the initial procedure.
patients demonstrated multiple bilateral deep areas of
creased signal intensity in one occipital lobe. Eleven
cases were within the vascular territory of transient
ischemia in 3 of these patients. One patient with verte-
brasilar TIA's had multiple deep lesions in
hemisphere as well.
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 sum-
arizes the clinical, angiographic, CT scan and MRI
findings in the 22 patients.
The CT scans showed focal areas of decreased at-
tenuation 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 pa-
tient 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 abnor-
malities 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 verte-
brasilar TIA’s had a focal superficial area of in-
creased 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 le-
sions 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 vas-
cular territory of transient ischemia in 4 cases, and
outside that territory in 1 case. Twelve of the 15 pa-
tients 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 vertebrobas-
lar 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
Lesions
Fifteen of the 17 patients with carotid TIA’s had
bilateral carotid angiography. The angiographic find-
ings 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 dis-
cased arteries, and all 4 abnormalities were in the terri-
ory of symptomatic lesions. No such trends were ob-
served for MRI lesions. While MRI lesions were
prevailent 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>
<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>Vertebrobasilar TIA</td>
<td>—</td>
<td>No focal lesions</td>
<td>No focal lesions</td>
</tr>
<tr>
<td>6 68 M</td>
<td>Vertebrobasilar TIA</td>
<td>Severe stenosis at left verteobasilar 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>Vertebrobasilar 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>Vertebrobasilar 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>Vertebrobasilar 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).

The CT scans were abnormal in 32% of the patients, a figure well within the 5%-50% suggested by recent

<table>
<thead>
<tr>
<th>Carotid Arteries in 15 Patients with Carotid Transient Ischemic Attacks</th>
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</thead>
<tbody>
<tr>
<td>Cases with one or more focal parenchymal lesions in ipsilateral cerebral hemisphere on CT scan</td>
</tr>
<tr>
<td>Normal or mild (&lt;50%) carotid stenosis (n = 14)</td>
</tr>
<tr>
<td>Moderate or severe (&gt;50%) carotid stenosis (n = 14)</td>
</tr>
<tr>
<td>Carotid occlusion (n = 2)</td>
</tr>
<tr>
<td>Total (n = 30)</td>
</tr>
</tbody>
</table>

The numbers between brackets () represent those cases in which the focal lesions were within the symptomatic hemisphere.
patterns and the autoregulatory reserves, making certain 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. These 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 reservoirs, 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
Focal parenchymal lesions in transient ischemic attacks: correlation of computed tomography and magnetic resonance imaging.
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