Correlation of Clinical and Computed Tomographic Findings in Stroke Patients

K.A. Sotaniemi, MD, J. Pyhtinen, MD, and V.V. Myllylä, MD

We evaluated the correlation between clinical features and computed tomographic findings in a prospective study of 1,191 consecutive patients with acute cerebrovascular disease seen during 1 year. In the 386 patients in whom symptoms and signs initially suggested a cerebrovascular disorder, computed tomography revealed a relevant lesion in 154 (hemorrhagic in 52 [33.8%], ischemic in 102 [66.2%]) and a significant nonstroke abnormality in 14 (3.1%). Among the remaining 805 patients with symptoms and signs suggesting some central nervous system disorder other than stroke, computed tomography revealed a cerebrovascular lesion in 38 (4.7%); 35 of these lesions were ischemic. The computed tomographic finding was compatible with the final clinical diagnosis in 192 (84.2%) of the 228 patients with lesions. In the entire sample of 1,191 patients, a cerebrovascular disorder would have been missed in 38 (3.2%) without computed tomography. On the other hand, computed tomography failed to visualize a cerebrovascular lesion in 40 patients in whom such a lesion was clinically obvious. Our results emphasize that both careful neurologic assessment and a policy of early computed tomography are of crucial importance in the diagnosis of stroke and for therapeutic considerations. (Stroke 1990;21:1562-1566)

The introduction of computed tomography (CT) to clinical practice has had a great impact on our knowledge of cerebrovascular disorders, and cerebral CT has become the most commonly used primary radiologic investigation for stroke. Cerebral CT has shown that the prognosis of intracerebral hemorrhage (ICH) is not as poor as was supposed when small hemorrhages were often undiagnosed or misdiagnosed as ischemic events, and it has changed the order of diagnostic procedures for stroke. Furthermore, in differentiating ischemic infarcts from hemorrhagic lesions, cerebral CT has proved to be of crucial importance for therapeutic considerations, particularly anticoagulant treatment.

Despite a vast number of CT studies devoted to cerebrovascular disorders, the available knowledge on clinical and CT correlations in stroke is based on highly selected patient samples and little is known of the findings in routine neurologic patients. To elucidate these correlations, we prospectively studied all 1,191 neurologic patients referred for cerebral CT during 1 year in a neurological department. The relation between clinical and CT findings was assessed at two phases, first, after the neurologic evaluation made on admission and following the first CT investigation and, second, after the final diagnosis was made based on all available information.

Subjects and Methods

During the year under survey, 1,191 neurologic patients were referred for cerebral CT. This hospital-based patient sample comprised 572 men and 619 women aged 15–87 (mean 46) years. Cerebrovascular etiology of central nervous system (CNS) symptoms and signs was suggested clinically in 386 patients (32.4%). The patient sample included all those in whom ICH, subarachnoid hemorrhage (SAH), subdural hematoma, or cerebral infarction were suspected and virtually all those with suggested transient ischemic attack (TIA). Generally accepted diagnostic criteria were used.

The CT study was ordered only after recommendation by a neurologist who defined and registered the clinical data and suspected localization of the lesion. The radiologist determined the CT diagnosis. The final diagnosis was based on all the clinical, radiologic, electroencephalographic, neurosurgical, neuropathologic, and ancillary information accumulated. In addition to the 386 patients in whom CT was performed because of a suspected cerebrovascular disorder, 805 patients in whom cerebrovascular disease had not been initially suspected but in whom CT and/or the ancillary investigations revealed cerebrovascular pathology were also analyzed.

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The CT examinations were performed using either Somatom 2 or Toshiba TCT 80 A (Tokyo, Japan) equipment. All examinations included axial projections, with coronal projections when needed. The slice thickness was 4–5 mm in the posterior fossa and 8–10 mm supratentorially. Contrast enhancement was not used in patients with contraindications or in those with findings definable with certainty without contrast (e.g., ICH).

There were 178 (46.1%) emergency CT studies among the 386 in patients with cerebrovascular symptoms (four). Among the 805 patients in whom some initial clinical hypothesis other than stroke was made, CT revealed cerebrovascular lesions in 38 (4.7%) with cerebral infarcts, one with ICH, one with SAH, and one with subdural hematoma (Table 1). Thus, of the 192 patients with cerebrovascular lesions visualized by CT, the finding was clinically unexpected in 38 (19.9%).

Of the 137 cerebral infarcts, 23 (16.8%) were small deep infarcts (lacunes) and nine (6.6%) were multiple infarcts. The prevalence of lacunar infarcts was 20.0% (17 of 85) among the patients with and 11.5% (six of 52) among the patients without an initial hypothesis of cerebral infarct verified by CT. The clinical picture did not agree with the CT localization of an infarct in 16 (11.7%, 11 lacunar infarcts and five infarcts of other types) of the 137 patients with the CT finding of cerebral infarct. Considering only the 85 patients in whom the initial clinical hypothesis was cerebral infarct, the clinical picture and CT localization were compatible in 80 (94.1%).

The final diagnosis (Table 2) of ICH in 29 patients or subdural hematoma in five was based on the CT findings in all except two patients in whom the first CT scan had suggested ICH but in whom repeat CT scans proved the lesions to be hemorrhagic infarcts. The final diagnosis of SAH in 39 patients was verified by CT in 17 and by lumbar puncture in the remaining 22. Of the 137 patients with a CT finding of cerebral infarct the initial finding was hemorrhagic infarct in four (2.9%). Hemorrhagic infarct was differentiated from ICH based on the former condition's typical localization in the hemispherical convexities, its less intense density, and its nonhomogeneous structure. Furthermore, all four patients had a follow-up exam-

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**TABLE 1. Initial Clinical Hypothesis and CT Diagnosis of Patients Investigated by Cerebral CT**

<table>
<thead>
<tr>
<th>Clinical hypothesis</th>
<th>CT diagnosis</th>
<th>Normal No.</th>
<th>Normal %</th>
<th>ICH No.</th>
<th>ICH %</th>
<th>SAH No.</th>
<th>SAH %</th>
<th>Infarct No.</th>
<th>Infarct %</th>
<th>Subdural hematoma No.</th>
<th>Subdural hematoma %</th>
<th>Other No.</th>
<th>Other %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrovascular disorder</td>
<td></td>
<td>57</td>
<td>22</td>
<td>14</td>
<td>26</td>
<td>38.6</td>
<td>1</td>
<td>1.5</td>
<td>22</td>
<td>38.6</td>
<td>6</td>
<td>10.5</td>
<td>1</td>
</tr>
<tr>
<td>ICH</td>
<td></td>
<td>46</td>
<td>3</td>
<td>22</td>
<td>47.8</td>
<td>3</td>
<td>6.5</td>
<td>15</td>
<td>32.6</td>
<td>3</td>
<td>6.5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>SAH</td>
<td></td>
<td>197</td>
<td>5</td>
<td>54</td>
<td>27.4</td>
<td>5</td>
<td>2.5</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>5</td>
<td>43.1</td>
<td>1</td>
</tr>
<tr>
<td>Infarct</td>
<td></td>
<td>31</td>
<td>0</td>
<td>33</td>
<td>64.7</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>14.3</td>
<td>3</td>
</tr>
<tr>
<td>TIA</td>
<td></td>
<td>35</td>
<td>0</td>
<td>12</td>
<td>34.3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>14.3</td>
<td>13‡</td>
<td>37.1</td>
</tr>
<tr>
<td>Subdural hematoma</td>
<td></td>
<td>386</td>
<td>102</td>
<td>135</td>
<td>35.0</td>
<td>30</td>
<td>7.8</td>
<td>16</td>
<td>4.1</td>
<td>102</td>
<td>26.4</td>
<td>6</td>
<td>1.6</td>
</tr>
<tr>
<td>All</td>
<td></td>
<td>805</td>
<td>25</td>
<td>455</td>
<td>56.5</td>
<td>1</td>
<td>0.1</td>
<td>1</td>
<td>0.1</td>
<td>35</td>
<td>4.3</td>
<td>1</td>
<td>0.1</td>
</tr>
<tr>
<td>Other disorder</td>
<td></td>
<td>1,191</td>
<td>97</td>
<td>590</td>
<td>49.5</td>
<td>31</td>
<td>2.6</td>
<td>17</td>
<td>1.4</td>
<td>137</td>
<td>11.5</td>
<td>7</td>
<td>0.6</td>
</tr>
</tbody>
</table>

*Includes two with encephalitis and two with contusion hematoma.
†Includes four with tumor, one with encephalitis, and one with contusion hematoma.
‡Includes two with tumor.

CT, computed tomography; ICH, intracerebral hemorrhage; SAH, subarachnoid hemorrhage; TIA, transient ischemic attack; Other, miscellaneous minor abnormalities (mostly local or generalized atrophic changes of various severity) and most commonly without clinical relevance.

The initial clinical hypothesis and the CT finding for the 386 patients with cerebrovascular symptoms and signs are shown in Table 1. In 154 patients (39.9%) CT showed cerebrovascular lesions, of which 30 (19.5%) were ICHs, 16 (10.4%) were SAHs, 102 (66.2%) were infarcts, and six (3.9%) were subdural hematomas. Although subdural hematoma is not a primary cerebrovascular disease, we included it in our study because its phenomenology is often relative to cerebral hemorrhage. Cerebrovascular pathology of some kind other than that which was initially hypothesized was found in 27 (7.0%) of the 386 patients (ICH in eight, SAH in one, cerebral infarct in 17, and subdural hematoma in one). In 97 (25.1%) additional patients, CT revealed an abnormality other than a cerebrovascular disorder. Among these were six with cerebral tumors that presented clinically with features suggesting subdural hematoma (two) or infarct (four) and 12 with miscellaneous abnormalities, comprising encephalitis (three), hydrocephalus (two, both requiring shunting), contusion hematoma (three), and vascular malformation (four).
Contrast enhancement was used only occasionally when the CT finding was ICH or SAH or when a normal CT finding was seen with an initial clinical hypothesis of TIA. Enhancement was used in 51 of the 155 patients with a final diagnosis of cerebral infarct; in 15 of these patients enhancement provided more information than plain CT, mainly ruling out other types of lesions causing cerebral hypodensities.

The prevalence of a final diagnosis of cerebrovascular disorder (TIA excluded) was 49.0% (189 of 386) among patients with and 4.8% (39 of 805) among patients without an initial clinical hypothesis of cerebrovascular disorder; CT showed a cerebrovascular lesion in 39.9% (154) and 4.7% (38) of these patients, respectively. Thus, the CT finding was compatible with the final diagnosis in 192 (84.2%) of the 228 patients with lesions. In 38 (3.2%) of the 1,191 patients the cerebrovascular disorder would have been missed without CT. On the other hand, CT failed to visualize lesions in 40 patients (18 with cerebral infarcts and 22 with SAHs).

**Discussion**

During the year under survey, 386 (32.4%) of the 1,191 neurologic patients referred for cerebral CT were examined for the evaluation or exclusion of cerebrovascular disorders. This percentage is in accordance with the proportion of 34% cerebrovascular patients seen in our department, which alone is responsible for neurologic and CT services for an adult population of approximately 280,000. The load caused by such patients is also reflected in the proportion of emergency investigations (178 of 386, 46.1%). Our patient series is hospital-based and therefore does not represent an epidemiologic sample. Evidently, patients with serious symptomatology are overrepresented in our sample, as are patients with an atypical course of their disease.14 Our study differs from previous ones in its prospective approach and its two-phase analysis of the correlates (considering both the initial clinical hypothesis before CT and the final diagnosis). Moreover, we also surveyed the findings in patients initially suspected to have other CNS disorders.

Among 228 patients with lesions, cerebral CT confirmed the clinically suspected lesion (TIA patients excluded) in 192 (84.2%), which is almost as high a percentage as that reported in previous studies of selected patient series (87–98%).13,14,17 Our percentage is higher than those of <70% reported in comparable studies8,10 that were either retrospective or had highly selected patient series, investigated mainly in stroke units. Despite differences in the selection of patients, the proportion of ischemic CT lesions is uniform. In our sample, 137 (71.4%) of the 192 CT lesions were ischemic; previously reported values range from 75% to 78%.13,14,17 In contrast, our proportion of hemorrhagic lesions (55, or 28.6%) is considerably higher than the reported values of 6–14%.14,15,17 Our policy of investigating cerebrovascular patients early using CT18 clearly improves our detection of small hemorrhagic lesions, which often mimic ischemic infarcts.1–3

Apart from the 386 patients in whom a cerebrovascular disorder was initially hypothesized, CT revealed cerebrovascular lesions in 38 (4.7%) of the remaining 805 patients (Table 2). In three patients, the initial hypothesis of hemorrhagic infarct was also the final diagnosis, while 19% (26 of 137) of the CT-verified cerebral infarcts were considered to be unrelated to the present symptoms and signs of CNS disorder.

The differentiation between hemorrhagic and ischemic lesions is important because of the therapeutic implications.3,11,17,19 During the early phase of ICH, CT is reported to visualize intracerebral bleeding in 72–83% of cases.20 Clinical differentiation between hemorrhagic and ischemic lesions has proved problematic because small hematomas may cause symptoms and signs that are identical to those caused by infarcts,2,7,8,20 and although predictive measures have been developed,20 cases remain in which important therapeutic implications would be missed. In our study, the clinical differentiation of hemorrhagic
from ischemic lesions was successful 81.8% of the time (45 of 55 hemorrhagic lesions were classified correctly); of 31 CT-verified ICHs, five had been initially hypothesized to be infarcts. On the other hand, all four lesions classified as hemorrhagic infarcts on the initial CT scans were confirmed on later CT scans. However, patients with an infarct on their first CT scan were not systematically followed up with further CT investigations, and therefore the incidence of hemorrhagic transformation cannot be evaluated in our series.

The proportion of nonstroke lesions on CT has been reported to vary from 0.4% to 18%,21,22 and more recently from 1% to 4%,9,11,14,17 which is in agreement with our result of 3.2% (12 of 386). The reported proportion of cerebral tumors visualized by CT has ranged from 0.5% to 1.8%,10,11,14,17 while that of subdural hematomas has been 0.6%-10,15; our values are 1.6% (six of 386) and 0.6% (seven of 1,191), respectively. Thus, careful clinical analysis should (optimally) allow differentiation of a vascular lesion because otherwise the (minimum) acceptable threshold for using CT to confirm the diagnosis would not be reached.

Most effective in visualizing ICH and subdural hematoma, CT confirmed the presence of SAH in less than half our patients. This detection rate is far less than expected; rates as high as 87-97% <24 hours and 72-75% <5 days after the onset of symptoms have been claimed for detecting intracranial bleeding.23-27 The difference may be due first, to the vertebrobasilar origin of SAH in CT-negative cases24-26 and second, to the fact that because of local geography patients with nonfulminant symptomatology are not referred as urgently as those with a clear-cut clinical picture of SAH. Considering only the 20 SAH patients examined <24 hours after the onset of symptoms, the CT finding was positive in 75% (15). Our results emphasize the need for lumbar puncture when CT is negative, particularly in patients with mild symptomatology and those in whom several days have elapsed since the suggested bleeding.

The visualization of an ischemic infarct on CT scans depends greatly on the timing of the investigation.28-30 Unenhanced CT performed approximately 3 days after cerebral infarction has been reported to be sensitive since about one third of lesions visible at that time will not be detected later31 due to the fogging effect.28 From 5% to 10% of cerebral infarcts remain isodense and are visualized only by enhancement.2,31 In our patient series, >90% of the CT scans were performed 1-4 days after the onset of symptoms. The presence of an infarct was confirmed by CT in 88% (137) of the 155 patients with a final diagnosis of cerebral infarct, which compares favorably with previously reported detection rates of 60-70%.28,30 On the other hand, in almost 12% (18) of these 155 patients, the CT scan was negative or not compatible with the symptomatology. In two thirds of these 18 cases the final diagnosis was vertebrobasilar infarct, the remaining being diagnosed as lacunar infarcts. The prevalence of CT-negative cerebral infarcts varies widely, from 5% to 50%.5,28,32 Our result being of the same magnitude as those in comparable studies (8-13%)10,17. Magnetic resonance imaging (MRI) has been shown to be more sensitive than CT for detecting acute cerebral infarcts, specifically lacunar infarcts.33-35 MRI offers more sophisticated methods than CT for characterizing cerebrovascular lesions.36 However, MRI has its pitfalls37 (e.g., in visualizing bleeding) and is not as widely available as CT.

Our results demonstrate that CT combined with a careful clinical investigation, and lumbar puncture when necessary, is useful in diagnosing cerebrovascular lesions, particularly hemorrhagic lesions with specific therapeutic implications, with high certainty. It has been argued that the wide use of CT during the acute phase of stroke is not rewarding and only increases the costs of health care.14,37,38 The development of specific therapies in both hemorrhagic and ischemic disorders, however, now makes it essential to define accurately the cerebrovascular disorder in order to achieve optimal therapeutic benefits. A policy of active investigation of stroke patients during the acute phase of their illness is necessary for up-to-date treatment and to prevent the cerebral and peripheral complications threatening these patients.

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KEY WORDS • cerebrovascular disorders • tomography, x-ray computed
Correlation of clinical and computed tomographic findings in stroke patients.
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Stroke. 1990;21:1562-1566
doi: 10.1161/01.STR.21.11.1562
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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