Residual Lesions on Computed Tomography After Intracerebral Hemorrhage

C.L. Franke, MD; J.C. van Swieten, MD; and J. van Gijn, MD

Background and Purpose: We investigated the residual abnormalities on computed tomography in a series of patients with proven intracerebral hemorrhage to determine whether the type of lesion is related to the site and size of the initial hematoma.

Methods: In a partially prospective follow-up study, we studied computed tomographic scans of 42 patients with spontaneous intracerebral hemorrhage after 2–24 (median 9) months.

Results: Lobar hemorrhages had occurred in 20 patients; the other 22 hemorrhages were in the basal ganglia or thalamus. No residual lesions were found on seven scans (17%), despite residual handicap in three of these seven patients (slight, moderate, and moderately severe). In five cases, the scan showed only focal atrophy, and in two there were only focal calcifications. There were six patients with slit-like lesions (only after deep hemorrhages), 12 with rounded and isolated hypodense areas, and 10 with rounded hypodense areas connected to the ventricular system. The connection between the residual lesion and the ventricular system depended to a large extent on the size of the initial hematoma, but very little on whether it had initially ruptured into the ventricular system.

Conclusions: A retrospective diagnosis of cerebral hemorrhage on radiological grounds can be made with confidence only in a small group of patients with slit-like lesions in the basal ganglia. This diagnosis is impossible in approximately one third of cerebral hemorrhages because the abnormalities are aspecific or have completely disappeared. (Stroke 1991;22:1530–1533)
TABLE 1. Characteristics of Residual Lesions on Computed Tomography in 42 Patients With Spontaneous Intracerebral Hemorrhage

<table>
<thead>
<tr>
<th>Residual lesion categories</th>
<th>Site of initial hematoma</th>
<th>Basal ganglia</th>
<th>Lobar</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No residual lesion</td>
<td></td>
<td>4</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>Ventricular enlargement only</td>
<td></td>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Calcification only</td>
<td></td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Hypodense lesions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slit-like lesion</td>
<td></td>
<td>6</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>Round lesion surrounded by brain tissue</td>
<td></td>
<td>2</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>Round lesion connected to ventricular system</td>
<td></td>
<td>6</td>
<td>4</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>22</td>
<td>20</td>
<td>42</td>
</tr>
</tbody>
</table>

The follow-up CT scan was made between 2 and 24 (median 9) months after the event. We first studied the sites of the residual lesions. These were identified as no residual lesion, hypodense areas, or calcifications (Table 1). The hypodense lesions were further subdivided into those that extended to the ventricular system and those that did not (Table 1). We also recorded focal tissue loss, in the form of unilateral ventricular enlargement or local enlargement of cerebral sulci. The volume of the residual lesion was estimated by measuring the areas of the lesion in all slices by means of an IBM Personal Computer with graphic tablet and by subsequent multiplication with the slice thickness. Statistics were done with the \( \chi^2 \) test; a value of \( p<0.05 \) was considered significant.

### Results

The sites and sizes of the original ICHs are given in Table 2. Rupture into the ventricular system occurred in 11 patients. The results of the follow-up study are listed in Table 1. Although in seven of the 42 patients the follow-up CT scan showed no residual lesion at all (Table 3), this did not mean that these patients were without residual disability. Two patients had no symptoms; two had symptoms but no

signs and the other three patients had slight, moderate, and moderately severe handicap, corresponding to grades 2, 3, and 4, respectively, on the modified Rankin scale. The mean volume of the original hematoma in these patients was 8.7 cm\(^3\) (range 2–20 cm\(^3\)). This was significantly smaller than the mean hematoma volume (23.8 cm\(^3\)) in patients with residual lesions (\( p=0.007 \)). Focal atrophy (enlargement of cerebral sulci or part of the ventricular system) was found in 11 of the 28 patients with hypodense lesions. In five other patients, ventricular enlargement was the only finding. In two patients, calcification at the site of the previous hematoma was the only residual lesion; volumes of the original hematomas were 10 cm\(^3\) and 29 cm\(^3\).

Figure 1 illustrates slit-like lesion. Table 1 shows the locations and characteristics of the residual lesions in all 42 patients as determined by CT. Only 6 of 22 hematomas in the basal ganglia and none from lobar hematoma produced slit-like lesions in this series. Round lesions resulted in eight of 22 basal ganglia hematomas and 14 of 20 lobar hematomas. The round lesions that had no connection with the ventricular system (Figure 2) resulted mostly from lobar hematomas (10 of 12 cases). Half of these round lesions involved only the white matter, with the overlying cortex left intact.

Rupture of the ICH into the ventricular system did not signify that the residual lesion would be contiguous with the ventricular system. Only four of 11 patients with rupture of the original hematoma into the ventricular system (Table 2) later showed approximation of the residual lesion to the ventricular system on CT. Six patients without original intraventricular hemorrhage had round lesions that extended to the ventricular surface. The presence of a residual lesion extending to the ventricular system resulted from lobar hemorrhage as often as from hemorrhage in the basal ganglia. In eight of 17 patients (47%) with a hematoma volume >20 cm\(^3\), the lesion extended to the ventricle, whereas two of the 25 patients (8%) with a volume <20 cm\(^3\) did so \( (p<0.025) \). In the case of residual lesions not touching the ventricles, there was no relationship between the size of the hematoma and subsequent development of either a slit-like or a rounded lesion.

TABLE 2. Sites and Sizes of Original Intracerebral Hematomas in 42 Patients

<table>
<thead>
<tr>
<th>Location</th>
<th>No.</th>
<th>Mean volume ((\text{cm}^3))*</th>
<th>Range ((\text{cm}^3))</th>
<th>Rupture into ventricular system</th>
</tr>
</thead>
<tbody>
<tr>
<td>Putamen only</td>
<td>10</td>
<td>17</td>
<td>6–39</td>
<td>1</td>
</tr>
<tr>
<td>Thalamus only</td>
<td>6</td>
<td>6</td>
<td>0.8–10</td>
<td>3</td>
</tr>
<tr>
<td>Caudate nucleus only</td>
<td>1</td>
<td>10</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>Entire area of basal ganglia</td>
<td>5</td>
<td>50</td>
<td>22–88</td>
<td>2</td>
</tr>
<tr>
<td>Lobar</td>
<td>20</td>
<td>22</td>
<td>6–64</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>42</td>
<td>21.3</td>
<td>0.8–88</td>
<td>11</td>
</tr>
</tbody>
</table>

*Median volume 12 cm\(^3\).
A strong correlation was found between the volume of the original hematoma and the volume of any residual lesion (correlation coefficient 0.63, p<0.01). On average, the mean volume of the residual lesions was about half the volume of the original hematoma (mean 10.1 cm³, SD 15.5, and 21.3 cm³, SD 19.6, respectively). In six of the 42 patients, the volume of the residual lesion was about the same as that of the initial hematoma.

Discussion

Our study shows that the finding of a slit-like lesion on CT after ICH represents the end stage of hemorrhage in the deeper structures of the brain, but not of lobar hemorrhages. In two other CT studies after spontaneous ICH, the authors did not mention the sites of the residual lesions.6-10 The appearance of slit-like lesions after a hemorrhage in the basal ganglia, as shown in a study on experimental ICH,11 probably results more from the splitting of nerve fibers by the hemorrhage than from destruction of nerve fibers.12 Perhaps this also explains the absence of any type of residual lesion in seven of our 42 patients (17%), a proportion that agrees with two previous studies.6,10 In one of these, it was reported that patients with a normal CT scan had a normal neurological examination at follow-up,6 but three patients in our study with a normal CT scan at follow-up were still more or less restricted in their lifestyles. Probably the loss of tissue in these patients was too small for the spatial resolution of the CT scanner.

Although it seems logical to assume that rupture into the ventricular system with ICH predisposes to a permanent connection with the ventricular system, we did not find such a relationship. There must be factors other than intraventricular hemorrhage to explain such a connection. Increase of the original hematoma after the first CT scan might lead to delayed rupture into the ventricular system. An alternative explanation is progressive enlargement of hypodense areas as a result of transference of fluid from the ventricles, but we did not find clinical evidence for a temporary expansion of the lesion. We also failed to find a relationship between the presence of a connection of the residual lesion with the ventricular system and the site of the hemorrhage. Large hematomas (>20 cm³) often resulted in a
connection with the lateral ventricle, in keeping with one previous study. In two of our 42 patients, calcification was the only lesion on the follow-up CT scan. As the original hemorrhages in these cases were 10 cm³ and 29 cm³ in size, and the median volume in our series was 12 cm³, this does not support the notion that calcifications on CT after ICH correspond to smaller hematomas. Sometimes it is difficult to distinguish posthemorrhagic calcifications from those in a tumor, which explains why one of these two patients, prompted by headache, underwent a stereotactic brain biopsy 2 years after ICH. The biopsy showed only remnants of the former ICH.

With CT scanning, it is often impossible to distinguish between the residual lesion of a former ICH and an old cerebral infarct. The characteristic slit-like scars in the basal ganglia of our series occurred in only one of seven. Lesions resembling the typical image of a lacunar infarct were not found in our study but have been reported by others. Round areas of hypodensity can result from a former hematoma as well as from an old infarct, and focal enlargement of cerebral sulci or of a lateral ventricle is even more aspecific. In contrast to large-vessel infarction, the cortical area is frequently, but not always, spared in ICH, and the hemorrhage is usually not restricted to a single vascular territory. On histological examination, it is easy to detect old hemorrhagic lesions with a staining reaction for iron. The pathological–anatomic features of such residual lesions after ICH may consist of "slits" (cleft-shaped collapsed cavities) or cysts, which are often filled with fluid and are sometimes connected to the ventricular system. A large and isolated hypodense area in a cerebral lobe might resemble a low-grade astrocytoma. Magnetic resonance imaging can identify a former hemorrhage as an irregular area of signal loss on T2-weighted images, owing to the effects of hemosiderin, whereas old infarcts usually are well-defined areas of increased signal intensity, similar to cerebrospinal fluid, on T1- and T2-weighted images. However, no studies are available in which a large series of patients with ICH had magnetic resonance imaging months after the event. We conclude that, with CT, a retrospective diagnosis of intracerebral hemorrhage can be made with confidence only in patients with slit-like lesions in the basal ganglia.

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References


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