The value of computerized tomography in aneurysmal subarachnoid hemorrhage

The concept of the CT score

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Of 256 patients with aneurysmal subarachnoid hemorrhage, 131 underwent computerized tomographic (CT) scanning within 7 days of the ictus. These scans were analyzed in order to assess the quantity of blood in the main subarachnoid cisterns and cerebral fissures. The method of quantification used recognized the horizontal and vertical components of the largest clot visible on the CT scan and expressed this as the "CT score." Angiographic vasospasm was assessed and graded, based on reduction in the caliber of the major cerebral vessels. The CT score was then compared to 1) the incidence of angiographic vasospasm, 2) the clinical course, and 3) the eventual outcome. Of the patients who showed no blood on the initial CT scan, 87% were admitted in good clinical grades, whereas among patients with higher CT scores the number admitted in poor clinical grades increased. The degree of angiographic vasospasm did not relate as closely as the CT score to the clinical grade on admission or to the subsequent clinical course.

The final outcome was assessed on follow-up review, and those acquiring neurological deficits from ischemic neurological dysfunction (IND) were identified. Ninety percent of patients with no blood on the CT scan (CT score 0) had a good outcome, while 5% sustained the effects of IND. The incidence of IND gradually increased with a rise in the CT score until, with scores of 8 and above, 90% of patients suffered the ill effects of IND. The CT score proved to be a simple yet accurate prognostic indicator of the outcome of IND.

Key Words □9 subarachnoid hemorrhage □9 subarachnoid cistern □9 vasospasm □9 computerized tomography score □9 ischemic neurological dysfunction

Computerized tomography (CT) helps in the initial management of aneurysmal subarachnoid hemorrhage (SAH) by identifying the responsible lesion,\(^6,12\) a compressing hematoma,\(^8,16\) hydrocephalus,\(^3,20\) and impending or established areas of ischemic complications.\(^18\) Recent work\(^4,5\) has also suggested a correlation between the quantity of blood in the subarachnoid cisterns, as visualized on the CT scan, and the likelihood of supervening ischemic neurological dysfunction (IND). The etiology of this complication remains uncertain, but it alters the outcome of the disease in terms of mortality and morbidity.\(^16,21\) The ability to identify, especially in the initial stages, those patients at risk of developing subsequent ischemia would enable the use of available prophylactic measures in the susceptible patients, in the hope of improving their prognosis.

The purpose of this study was to establish prognostic correlations in patients with aneurysmal SAH, and to develop a method of quantifying the amount of blood in the subarachnoid cisterns and cerebral fissures as visualized on the CT scan.

Clinical Material and Methods

Patient Population

The total group included 256 patients with confirmed SAH admitted to the Department of Neurosurgery of the Atkinson Morley's Hospital during a 2-year period. Of these, 185 had CT scans within 7 days of the ictus, Day 1 being defined as the day after hemorrhage. Fifty-four patients either had negative angiographic studies or suffered severe associated medical problems which precluded further investigations and were therefore excluded from the study. The final analysis concerned the remaining 131 patients in whom an angiographically proven aneurysm was the lesion responsible for the SAH.

A detailed analysis of the CT scan appearances was
made by two independent observers. These data were then compared with the angiographic demonstration of vasospasm as well as the clinical course of the patient.

**CT Score**

The scans were performed on an EMI 1010 CT scanner with a 180 × 180 matrix using slices 8 mm thick at an angle of 15° to the orbitomeatal line. The reproduced CT images were in transparencies 11 × 8 cm in size. The CT analysis was based entirely on the visible amount of localized blood clot in the subarachnoid cisterns and cerebral fissures, as identified by accepted terminology. Diffuse subarachnoid blood visible over the cerebral convexities, and intraventricular clots or casts were disregarded in the system of quantification used in the scan analysis, but intracerebral hematomas were excluded. When blood was visible throughout the basal cisterns and cerebral fissures, the area in which it appeared most localized was accepted as the best site for clot quantification. Thus, quantification of blood clot in the subarachnoid cisterns and cerebral fissures was made by assessment of the horizontal and vertical components of the largest visible localized collection of blood seen on the initial CT scan (Fig. 1).

The horizontal measurement was its greatest transverse diameter in millimeters, as seen on the CT transparency. The minimum width of clot measurable was 1 mm, which represented an actual width of 3.3 mm (see Discussion). In the assessment of parafalcine collections, 1 mm was allowed to account for the normal falcine density. The vertical component of the subarachnoid clot was quantified simply by the number of consecutive slices on the CT transparencies in which the localized collection of blood was visualized. When using scan slices 8 mm thick and allowing for partial volume effects, each transparency with visualized blood represented a clot with a vertical height varying between 4 and 8 mm. Each additional slice of visualized blood indicated that the height of the clot was greater by a further 8 mm from the previous minimum and maximum values. The actual height of the clot, however, was not used in the CT scoring system.

Each CT scan studied was then allocated a score derived by combining the numerical values of the horizontal and vertical components of the clot. Thus, a clot with a maximum width of 5 mm, combined with visualized blood in four CT slices, was given a score of 9 (5 + 4) (Fig. 1). When there was no visible clot, the score was zero. This CT score was used in all subsequent analyses in order to assess its value as a prognostic indicator in the syndrome of IND.

**Angiographic Analysis and Grading**

An attempt was made to assess the degree of alteration in caliber of the major intracranial vessels as demonstrated by routine cerebral angiography. This investigation was performed under conventional general anesthesia when the clinical state of the patient was stable after the SAH. The majority of these studies were carried out within 48 hours of the CT scan, 95% of them being between Day 1 and Day 8. The average period between ictus and angiogram was 5 days, the longest delay being 23 days.

The vessels studied for the purpose of assessing arterial spasm were the supraclinoid internal carotid artery, the proximal middle cerebral artery, the proximal anterior cerebral artery, the middle cerebral vessels distal to the bifurcation, the distal anterior cerebral arteries, the posterior cerebral artery, the vertebral artery, and the basilar artery. The straight anteroposterior projections of the carotid and vertebral vessels were used in the actual measurements. The degree of arterial spasm was assessed by the relative narrowing of the internal diameter of the affected vessel compared with its contralateral counterpart or with a normal ipsilateral vessel.

![Fig. 1. Computerized tomography (CT) scans showing a “typical CT score 9.” Consecutive slices of CT scan, performed on Day 1 following rupture of an anterior communicating artery aneurysm, showing a blood clot in the chiasmatic cistern (A) and in the anterior interhemispheric fissure (B, C, and D). A: Horizontal dimension, measured as the greatest transverse diameter of the subarachnoid hematoma (between arrows), = 5 mm. B, C, and D: Vertical dimension, measured as the number of consecutive slices in which the clot was visualized, = 4 slices. Therefore, the CT score = 5 (horizontal) + 4 (vertical) = 9.](image-url)
CT scan score in subarachnoid hemorrhage

The nondominant proximal anterior cerebral vessel was often hypoplastic and was regarded as being unaffected by vasospasm. The angiographic grades (AG's) based on the above criteria were represented as follows:

Grade 0 (AGO) = no vasospasm detected
Grade 1 (AG1) = vessel diameter reduced by 50% or less
Grade 2 (AG2) = vessel diameter reduced by 50% to 75%
Grade 3 (AG3) = vessel diameter reduced by more than 75%.

Clinical Grading and Vasospasm

The CT scores were categorized into four separate groups (Table 1). This table also shows uniformity in the age and sex distribution of the 131 patients in the studied group, as well as the average time interval between the ictus and the initial CT scan. There was a higher proportion of posterior communicating artery aneurysms in patients with a CT score of 0 and of middle cerebral artery aneurysms in those with CT scores of 8 and above (Table 2). This particular observation could not be explained on any specific basis.

The CT score was correlated with the clinical grade on admission (Table 3), allowing the following observations to be made. When no blood was visible on the initial CT scan (that is, the CT score was 0), 87% of patients were in clinical Grades I and II, and 79% in clinical Grade I. In the higher CT scores (4 and above), the proportion of patients in poor clinical grades (Grades III, IV, and V) increased. This trend was most obvious in patients with CT scores of 8 and above, of whom 61% were in clinical Grades III, IV, and V, and 81% were in Grades II through V.

Next, the angiographically demonstrated vasospasm (reduction in arterial caliber) was similarly compared with the clinical grade on admission (Table 4), with the following observations. Of the patients without angiographic spasm, 81% had been admitted in clinical Grades I and II, and 69% were in clinical Grade I. Of the patients with spasm of any degree (that is, AG1, AG2, and AG3), 44% were admitted in clinical Grade I, and 56% in Grades II, III, IV, and V combined; 62%

<table>
<thead>
<tr>
<th>CT Scores</th>
<th>Total Cases</th>
<th>Average Age (yrs)</th>
<th>Sex</th>
<th>Interval SAH to CT (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>43</td>
<td>44.6 ± 12.3</td>
<td>20</td>
<td>23</td>
</tr>
<tr>
<td>2, 3</td>
<td>22</td>
<td>46.5 ± 11.6</td>
<td>9</td>
<td>13</td>
</tr>
<tr>
<td>4, 5, 6, 7</td>
<td>35</td>
<td>48.1 ± 12.7</td>
<td>17</td>
<td>18</td>
</tr>
<tr>
<td>8, 9, 9+</td>
<td>31</td>
<td>50.0 ± 11.7</td>
<td>14</td>
<td>17</td>
</tr>
<tr>
<td>total cases</td>
<td>131</td>
<td>47.1 ± 13.8</td>
<td>60</td>
<td>71</td>
</tr>
</tbody>
</table>

*SAH = subarachnoid hemorrhage; CT = computerized tomography. For definition of CT score see text.

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TABLE 2
Aneurysm population of each CT group*

<table>
<thead>
<tr>
<th>CT Scores</th>
<th>ACAC</th>
<th>MCA</th>
<th>PCoA</th>
<th>TCA</th>
<th>BA</th>
<th>PICA</th>
<th>Multiple†</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>7+ (3)</td>
<td>6+ (1)</td>
<td>10+ (6)</td>
<td>2</td>
<td>0</td>
<td>3+ (1)</td>
<td>4</td>
</tr>
<tr>
<td>2, 3</td>
<td>9+ (2)</td>
<td>1+ (3)</td>
<td>4+ (1)</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>4, 5, 6, 7</td>
<td>10+ (5)</td>
<td>5+ (1)</td>
<td>7+ (1)</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>8, 9, 9+</td>
<td>13</td>
<td>14+ (1)</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>total cases</td>
<td>39+ (10)</td>
<td>26+ (6)</td>
<td>23+ (8)</td>
<td>5</td>
<td>2</td>
<td>4+ (1)</td>
<td>7</td>
</tr>
</tbody>
</table>

* Aneurysms responsible for subarachnoid hemorrhage from within the multiple-aneurysm group are listed in parentheses. CT = computerized tomography; ACAC = anterior cerebral artery complex; MCA = middle cerebral artery; PCoA = posterior communicating artery; TCA = terminal carotid artery; BA = basilar artery; PICA = posterior inferior cerebellar artery. For definition of CT score see text.
† Responsible aneurysms were not identified with certainty when multiple aneurysms were present.

TABLE 3
Comparison of CT score to clinical grade on admission*

<table>
<thead>
<tr>
<th>CT Scores</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
<th>Total Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>34</td>
<td>3</td>
<td>6</td>
<td>0</td>
<td>0</td>
<td>43</td>
</tr>
<tr>
<td>2, 3</td>
<td>18</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>22</td>
</tr>
<tr>
<td>4, 5, 6, 7</td>
<td>18</td>
<td>6</td>
<td>11</td>
<td>0</td>
<td>0</td>
<td>35</td>
</tr>
<tr>
<td>8, 9, 9+</td>
<td>5</td>
<td>7</td>
<td>14</td>
<td>5</td>
<td>0</td>
<td>31</td>
</tr>
</tbody>
</table>

* For definitions of computerized tomography (CT) score and clinical grading see text.

TABLE 4
Comparison of incidence of angiographic spasm to clinical grade on admission*

<table>
<thead>
<tr>
<th>Angiographic Grade</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
<th>Total Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>AG0</td>
<td>47</td>
<td>8</td>
<td>9</td>
<td>4</td>
<td>0</td>
<td>68</td>
</tr>
<tr>
<td>AG1</td>
<td>20</td>
<td>4</td>
<td>16</td>
<td>1</td>
<td>0</td>
<td>41</td>
</tr>
<tr>
<td>AG2</td>
<td>7</td>
<td>4</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>AG3</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>7</td>
</tr>
</tbody>
</table>

* For definitions of angiographic grading (AG) and clinical grading see text.

of these patients were in clinical Grades I and II and 38% in Grades III, IV, and V, combined.

The number of patients admitted in clinical Grade I decreased as the degree of spasm increased; thus, 69% were in AG0, 49% in AG1, 47% in AG2, and 14% in AG3. This trend was not consistently observed when clinical Grades I and II were considered together, with the discrepancy occurring in the group with moderate spasm (AG2). It may be attributed to the significant reduction in the total number of patients in categories AG2 and AG3. The delay between admission and angiography must be accepted as being relevant in the interpretation of the above findings. However, only two patients deteriorated clinically prior to angiography. Their CT scores and angiographic grades were 8 and AG3, and 9 and AG1, respectively. Thus, although a trend was observed, the presence of arterial spasm was not invariably related to a poor clinical grade on admission.

With these reservations, the CT score was compared with angiographic grade, revealing a correlation between the size of the subarachnoid hematoma and the occurrence of angiographic vasospasm (Table 5). Of 43 patients with no detectable blood on CT, 37% later demonstrated angiographic vasospasm. In the presence of a subarachnoid clot of any size, 55% of patients later demonstrated angiographic vasospasm. Of patients with CT scores of 8 and above, the incidence of vasospasm was 65%.

Outcome Analysis

The all-important question was the final outcome of the patient in relation to the CT score at the time of admission (Table 6). This included the outcome of definitive surgery in 115 (88%) patients. Each group was carefully analyzed in relation to the clinical course pre- and postoperatively as well as the "final clinical outcome" (Tables 6 and 8).

CT Score 0. Of the 43 patients with a CT score of 0, 38 (90%) had a good final outcome. All of them (except one who had a carotid ligation) underwent definitive surgery. Four patients entered a poor clinical grade transiently, two following angiography and two following surgery, but all of them made a complete recovery.

Two patients died of recurrent SAH during their hospital stay. Three patients had a poor final outcome which was directly attributable to IND in two (5%); the
CT scan score in subarachnoid hemorrhage

### TABLE 6
Comparison between CT score on admission and outcome at 3 months*

<table>
<thead>
<tr>
<th>Outcome†</th>
<th>0</th>
<th>2, 3</th>
<th>4, 5, 6, 7</th>
<th>8, 9, 9+</th>
</tr>
</thead>
<tbody>
<tr>
<td>good outcome</td>
<td>38 (90%)</td>
<td>18 (82%)</td>
<td>24 (69%)</td>
<td>3 (10%)</td>
</tr>
<tr>
<td>definitive surgery</td>
<td>38</td>
<td>18</td>
<td>24</td>
<td>3</td>
</tr>
<tr>
<td>adverse clinical grade from IND: full recovery</td>
<td>8 (4 + 4)</td>
<td>3</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>poor outcome due to IND</td>
<td>2 (5%)</td>
<td>4 (18%)</td>
<td>9 (26%)</td>
<td>28 (90%)</td>
</tr>
<tr>
<td>FND at 3 months</td>
<td>1</td>
<td>4</td>
<td>9</td>
<td>25</td>
</tr>
<tr>
<td>death from IND</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>definitive surgery</td>
<td>0</td>
<td>2‡</td>
<td>8</td>
<td>23</td>
</tr>
<tr>
<td>death from recurrent SAH</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>operative morbidity/mortality</td>
<td>1</td>
<td>1</td>
<td>7</td>
<td>10</td>
</tr>
<tr>
<td>due to IND</td>
<td>0</td>
<td>1</td>
<td>6</td>
<td>9 (3 + 6)</td>
</tr>
<tr>
<td>not due to IND</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>total cases</td>
<td>43</td>
<td>22</td>
<td>35</td>
<td>31</td>
</tr>
</tbody>
</table>

* For definition of computerized tomography (CT) score see text.
† Good outcome: no fixed neurological deficit (FND); poor outcome: FND present or death due to ischemic neurological dysfunction (IND).
‡ Both patients had a ventriculoperitoneal shunt placed.

other suffered a postoperative hematoma which required surgical evacuation. Neither of the patients who had a poor outcome due to IND underwent definitive surgery. One of them was admitted in clinical Grade I, and had vasospasm of angiographic grade AG1. This patient soon deteriorated clinically with no evidence of rebleeding, and eventually died from ischemic infarction. The other was admitted in clinical Grade III, had AG3 vasospasm, and survived with an FND. Of the other five patients admitted in clinical Grade III in this group, four recovered without an FND, and one died from a recurrent hemorrhage.

Thus, 11 patients in this group suffered the effects of IND, but eight of them recovered and one died of a rebleed. Only two (5%) sustained the long-term effects of IND.

CT Scores 2 and 3. Of 22 patients with a CT score on admission of 2 or 3, 18 (82%), all of whom underwent definitive surgery, had a good final outcome. Of these, three patients (all with AG0 vasospasm) had transient postoperative deterioration due to IND but made a full recovery.

Four patients (18%) had a poor outcome, of whom only one (with AG1 vasospasm) underwent definitive surgery. One patient in this group was admitted in clinical Grade III and deteriorated further following angiography, at which time only a mild degree (AG1) of vasospasm was shown. The cause of the poor outcome in two patients remained uncertain, but was perhaps related to IND. One was admitted in clinical Grade II but deteriorated to Grade III soon after angiography (AG2); at the same time, this patient developed hydrocephalus and sustained a FND in spite of a shunt procedure. The other was a 30-year-old hypertensive woman with no vasospasm (Grade AG0), who was delivered of her first child by Caesarean section and subsequently had two prolonged episodes of hypoten-

sion due to over-enthusiastic control of her blood pressure. The other two patients who were in clinical Grade II on admission improved and made a full recovery.

Thus, the long-term effects of IND were sustained by 18% of patients in this group.

CT Scores 4, 5, 6, and 7. The 35 patients with CT scores of 4 through 7 proved to be the most difficult to evaluate. Thirty-two (91%) patients underwent definitive surgery. Twenty-four (69%) had a good final outcome at the 3-month analysis. Nine of these, however, still showed the effects of IND during their clinical course by reaching clinical Grade III but recovering subsequently without a FND. A further nine patients (26%) had a poor final outcome, of whom eight had undergone definitive surgery. In this category, therefore, 18 patients (51%) reached an adverse clinical grade due to IND, with recovery occurring in half of them.

Of the nine patients who sustained an FND, three were admitted in a poor grade and remained so, but six deteriorated postoperatively from IND. The degree of angiographic vasospasm did not relate in a uniform pattern to the incidence of clinical deterioration from IND or to the final outcome of the patient.

A detailed analysis of the behavior of this group is shown in Table 7. The striking feature is the relatively small number of patients with CT scores of 5, 6 and 7, which precludes a true statistical analysis of the significance of these scores (see Discussion). The long-term effects of IND were sustained by 26% of patients in this category.

CT Scores 8, 9, and 9+. The 31 patients with CT scores of 8, 9, and 9+ displayed the best correlation between the CT score and the sequelae of IND. Only three patients (10%) had a good final outcome, and all of them underwent definitive surgery. One of them had a smooth clinical course (CT score 8, AG1 vasospasm)
TABLE 7

<table>
<thead>
<tr>
<th>CT Scores</th>
<th>Total Cases</th>
<th>Adverse Clinical State from IND</th>
<th>Recovery (no FND)</th>
<th>Poor Outcome (with FND)</th>
<th>Angiographic Vasospasm</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>19</td>
<td>No. 7</td>
<td>39</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>5</td>
<td>6</td>
<td>4</td>
<td>65</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>8</td>
<td>6</td>
<td>75</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>7</td>
<td>2</td>
<td>1</td>
<td>50</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>total cases</td>
<td>35</td>
<td>18</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* For definition of computerized tomography (CT) score see text. IND = ischemic neurological dysfunction; FND = fixed neurological deficit.

and remained well throughout. Another patient, with a CT score of 8 and no vasospasm (AG0), showed a transient postoperative deterioration to clinical Grade III, but made a full recovery. The third, with a CT score of 9 and AG2 vasospasm, was admitted in clinical Grade II, recovered, and had an uneventful clinical course.

Twenty-eight patients (90%) had a poor final outcome with death, or an FND as a consequence of IND. Eighteen (72%) of these had evidence of angiographic vasospasm. All but five of these underwent definitive surgery; four had conservative regimens and the other suffered a myocardial infarction during induction of anesthesia, and was therefore not submitted to surgery.

Four patients were admitted in clinical Grade I. Three had a poor final outcome, although only one had angiographic evidence of vasospasm. Seven patients were admitted in clinical Grade II and all but one had a poor final outcome due to IND. Four of these seven patients had some evidence of angiographic vasospasm, but the one with the best final outcome had a marked degree of spasm (AG2). All patients admitted in clinical Grades III and IV had a poor final outcome.

Thus, 90% of patients in this group sustained the long-term sequelae of IND.

Clinical Deterioration

The final correlation considered was the pattern of clinical deterioration in relation to cerebral angiography and definitive surgery. Events such as epilepsy, postoperative hematoma, and rebleeding were excluded, and we analyzed deterioration from one clinical grade to another purely from IND. The results are depicted in Table 8.

Discussion

The main aim of this study has been to establish a relationship of the quantity of localized blood clot in the subarachnoid cisterns and cerebral fissures, to the occurrence and outcome of ischemic complications following aneurysmal SAH. We have devised a simple scoring system to quantify the localized collections of blood, as seen on the initial CT scan, and used it as a means of predicting the incidence of the IND syndrome.

Quantitative estimation of the blood clot in the subarachnoid cisterns and/or cerebral fissures presented many difficulties. Progressive loss of CT density of clot occurred with time, and early CT scanning was therefore important for a better assessment of the shape, size, and distribution of the subarachnoid clot or clots.

The study was therefore limited to patients scanned within 7 days of the ictus. Although the larger subarachnoid cisterns and cerebral fissures were often in differing relationships to the scanning angle, they were mainly in the horizontal and vertical planes. This variable was overcome, however, by the consistency of the scanning technique in the studied group.

The concept of the CT score began when a preliminary survey in a small group of patients indicated that an assessment of the horizontal and vertical components of the subarachnoid clot provided a more accurate correlation to the eventual outcome than one based on the horizontal measurement alone. In this survey, the CT computer printouts were used to measure accurately the horizontal dimension of the clot. This enabled the magnification factor (10/3) from CT transparency size (1 mm) to actual clot size (1 x 10/3 = 3.3 mm) to be calculated. Although the actual clot size was never used in the study itself, its derivation explains the universal adaptability of the CT score. This factor also shows its dramatic increase in actual clot size with each 1-mm increase of width of clot as seen on the CT scan. In the vertical dimension, this increase can be as much as 8 mm, with a unit increase of the vertical component of the CT score.

The CT score is undoubtedly only an approximate method of clot quantification. Its simplicity, however, enables easy clinical application and is also adaptable to any size of CT transparency. Provided the initial CT was performed within the 1st week after the ictus, the absence of subarachnoid blood clot indicated an excellent prognosis, with a low incidence (10%) of the sequelae of the syndrome of IND, despite a significant incidence (37%) of angiographic vasospasm.

The phenomenon of arterial caliber reduction following SAH was initially postulated as a protective mechanism. Its biphasic time course has been disputed. Angiographic studies have demonstrated its occurrence as early as Day 1, being maximal at Days 6 to 8.
and persisting for even a month after the ictus. The term "vasospasm" with its implied meaning of "vasoconstriction" became synonymous with the clinical state of IND caused by cerebral ischemia following SAH. Recent work has shown, however, that "spasm" is the result of a vascular reaction to injury, consisting of intimal and medial swelling rather than a true muscular vasoconstriction. The variation in clinical presentation in patients with demonstrable arterial spasm may reflect varying degrees of cerebral blood flow (CBF). It has been shown that marked reduction in CBF occurred only with arterial vasoconstriction exceeding 50% of control caliber.

In our study, the incidence of angiographic vasospasm did not correlate dramatically with the CT score. When no blood was visualized on the CT scans the incidence of vasospasm was 37%, whereas an overall incidence of 55% was observed with the demonstration of subarachnoid blood clot of any size. A poor clinical outcome did not invariably follow angiographic demonstration of vasospasm, and neither was such vasospasm constantly observed in those who developed an FND and cerebral infarction. Only rarely was angiography repeated, and therefore the state of the vessels was known only on one occasion. The discrepancies between angiographic vasospasm and its relation to the clinical course may be related to the timing of the angiographic examination and/or the extent to which CBF was altered by such changes. The CT score thus proved to be a better predictive index of the vulnerability to IND than did the grade of angiographic spasm.

In a few patients in this study, clinical deterioration attributable to IND occurred without correlation either to the CT score or to angiographic grade. Moreover, there was a further group with a good final outcome despite a high CT score and angiographic vasospasm. Although such patients were few in number, they illustrated the clinical impression that other factors interrelate in the ultimate prognosis of the patient with aneurysmal SAH. A further study is being carried out at present in order to identify these additional clinical factors that may determine the ultimate outcome.

Patients with a CT score of 2 or 3 have more or less the same course as those with a CT score of 0. Despite an angiographic vasospasm incidence of 55%, 82% of these patients had a good final outcome. The prognosis of patients with a CT score of 4 to 7 was somewhat poorer, with 46% showing angiographic spasm but 69% achieving a good outcome. Another feature shown in Table 6 is the number of patients in the good outcome group who at some time in their clinical course reach an adverse clinical grade (Grade III, IV, or V). When these numbers are added to those who obtain a poor outcome within the same score, the percentage of those who recover from the dangers of IND are seen to diminish gradually as the CT score increases, and become markedly less with CT scores of 8 and above. This fact illustrates the further usefulness of the CT score in predicting the immediate clinical course as well as the ultimate prognosis.

The incidence of pre- and postoperative deterioration of the clinical state (Table 8) appeared to be evenly distributed except in CT scores 4 to 7, where the greater number appeared in the postoperative period. There was no immediate explanation available for this observation. As stated earlier, this group proved somewhat difficult to evaluate and, although the trends observed in other groups persisted, the expected and the observed frequency of patients with a good outcome were the same in this score category (Table 9). It is possible that they form a group "susceptible" to the effects of IND and are triggered into it by factors such as manipulation at surgery, hydration, or blood pressure alterations. These patients appear to be the best group in which to study the additional prognostic factors that may determine the outcome of the syndrome of IND. The dramatic increase in poor final outcome (90%) in those patients with CT scores of 8 and above illustrates best the significance of the present analysis.

This study has attempted to identify, on the basis of the initial CT scan, those patients in whom an unfavorable outcome may be expected after SAH. Such a method, to be of use in clinical management, should be simple as well as reproducible. The CT scoring system we have adopted appears to serve this purpose.
References


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