Computed Tomographic Diagnosis of Intraventricular Hemorrhage
Etiology and Prognosis

Sixty-eight patients with intraventricular hemorrhage (IVH) diagnosed by computed tomography (CT) were reviewed retrospectively to determine the etiology and prognosis, relationship to delayed hydrocephalus, and effect on neurological outcome. The most common causes were a ruptured aneurysm, trauma, and hypertensive hemorrhage. Ruptured aneurysms of the anterior communicating artery can often be predicted from the non-enhanced CT scan. The total mortality rate was 50%; however, 21% of patients returned to normal or had only mild disability. Patients in whom no cause was identified had a better prognosis. Delayed hydrocephalus was related to the effects of subarachnoid hemorrhage rather than obstruction of the ventricular system by blood. IVH per se is seldom a major factor in the neurological outcome.

Index terms: Aneurysm, cerebral, I[7]730 • Brain, hemorrhage • Brain, injuries • Head, computed tomography, I[0]1211 • Hydrocephalus, I[6]4372 • Subarachnoid space, hemorrhage

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Before computed tomography (CT), the clinical diagnosis of intraventricular hemorrhage (IVH) was usually suggested by sudden coma followed by severe and often fatal brainstem dysfunction (1-3). This so-called "classical" presentation was generally associated with massive intraventricular hemorrhage. However, CT frequently demonstrates IVH even when the "classical" clinical picture is absent. Fortunately, although it usually occurs in association with severe brain trauma, even relatively massive IVH may have a good outcome (4) (Fig. 1).

While several reports have documented the ability of CT to show IVH (4-7), to our knowledge only one sizable series has been analyzed, namely that of Little et al. (8) who observed 54 patients of whom only 9 survived. Our experience suggests that the prognosis is somewhat better than these statistics indicate. We have reviewed our own cases to determine the etiology and prognosis and evaluate the role of IVH in both delayed hydrocephalus and the neurological outcome.

MATERIALS AND METHODS

Of 77 cases of CT-diagnosed IVH available for study, 9 were discarded because of inadequate follow-up, poor scan quality, or because the IVH was postoperative, leaving 68 cases for review. Premature infants were not included.

All patients had non-contrast scans on an EMI 1010S; contrast-enhanced scans were also obtained in 11 cases. In patients with a ruptured aneurysm, we attempted to locate it on the non-contrast scan, based on the site of hematoma formation. In 29 patients scanned more than once, ventricular size was followed up in order to determine the relationship between subarachnoid and/or intraventricular bleeding and delayed hydrocephalus, defined as an increase in ventricular size developing at least one week after the initial hemorrhage. Any increase in size was also correlated with the presence of blood in the third and fourth ventricles and subarachnoid space. IVH was graded according to the amount of blood within each ventricle (TABLE I), with a maximum score of 12: 1-4 was called mild, 5-8 moderate, and 9-12 severe.

Outcome was assessed using the Glasgow scale (9), in which patients are assigned to one of five categories: normal or mild disability (I), moderate disability (II), major disability (III), vegetative state (IV), or death (V). Each patient's clinical record was also reviewed to determine the cause of death, or the nature of any residual neurological deficit in the case of survivors.

RESULTS

Age and sex distributions are shown in Figure 2. Males and females

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Severe IVH and right frontal hematoma caused by a ruptured right anterior cerebral artery aneurysm. The patient was normal on discharge except for mild paresis of the left leg secondary to spasm of the vessel.

**TABLE I:** System for Grading Severity of IVH

<table>
<thead>
<tr>
<th>Lateral Ventricles</th>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>trace of blood or mild bleeding</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>less than half of the ventricle filled with blood</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>more than half of the ventricle filled with blood</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>ventricle filled with blood and expanded</td>
<td></td>
</tr>
</tbody>
</table>

(Each lateral ventricle is scored separately)

<table>
<thead>
<tr>
<th>Third and Fourth Ventricles</th>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>blood present, ventricle size normal</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>ventricle filled with blood and expanded</td>
<td></td>
</tr>
</tbody>
</table>

Total Score (maximum = 12)

**TABLE II:** Accuracy of CT Diagnosis of Aneurysms at Different Sites

<table>
<thead>
<tr>
<th>Site of Aneurysm</th>
<th>No. of Proved Cases</th>
<th>Correct CT Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior communicating artery</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Anterior cerebral artery</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Azygous artery</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Pericallosal artery</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Callosomarginal artery</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Internal carotid artery</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Middle cerebral artery</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Posterior communicating artery</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Posterior inferior cerebellar artery</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

were affected in equal numbers, with a peak incidence in the sixth and seventh decades which was composed mainly of patients with ruptured aneurysm or hypertensive hemorrhage. Patients with trauma showed a biphase distribution, with peaks at age 10-30 and 60-80. The most frequent causes of IVH were ruptured aneurysm, trauma, and hypertensive hemorrhage (Fig. 3).

Aneurysms of the anterior cerebral...
artery and its branches, including the anterior communicating artery (ACA), were the most common, constituting 11 out of 17 cases (65%) (Table II). Only ruptures involving the ACA could be predicted, being seen as localized bleeding in the anterior interhemispheric fissure, just anterior to the lamina terminalis, in 4 out of 6 cases (Fig. 4).

The diagnosis of trauma was evident from the history and physical findings and was confirmed by autopsy in 11 out of 17 patients. Swelling or hemorrhage in the extracranial soft tissue was present in 13 patients (76%). Parenchymal hematomas were found in 12 (71%). Additional findings included ventricular enlargement, midline shift, subarachnoid hemorrhage, acute subdural hematoma, cerebral contusion, skull fracture, and intracranial air (Fig. 5).

Hypertensive hemorrhage, seen in 13 cases, was established by hypertension ≥180/100 (mean, 220/120) and a deep parenchymal hematoma on the CT scan. In 3 patients, angiography showed no aneurysm; 2 patients had hypertensive hemorrhage at autopsy. In 7 cases (54%), there was bleeding into the thalamus or basal ganglia (Fig. 6). Three had bleeding into the temporal lobe, 1 into the frontal lobe, and 1 into the pons; another patient had multiple hemorrhages throughout both hemispheres and the cerebellum. Only 4 had CT evidence of subarachnoid bleeding. Other findings included ventricular enlargement and midline shift.

Arteriovenous malformation (AVM) was diagnosed by angiography in all 5 cases. Intracerebral hematomas were associated with midline shift in 3 and ventricular enlargement in 2.

Three patients had a hemorrhagic diathesis established by coagulation studies. Two were overmedicated with oral anticoagulants and 1 had profound thrombocytopenia due to chemotherapy for acute promyelocytic leukemia. No characteristic CT features were found.

In 1 patient (Fig. 7), IVH was presumably caused by metastases from renal-cell carcinoma (which had also spread to the lungs and bone), although we have no pathological proof.

In 12 of the 68 patients, the etiology was undetermined. Angiography in 8 failed to show the bleeding site, as did autopsy in 1 case. Two patients died
Outcome of IVH in four main etiologic categories. The number of cases is indicated at the top of each column.

without undergoing angiography, and autopsy was not performed. Two elderly patients recovered satisfactorily and were not investigated because of their age. There were no CT features which distinguished these 12 patients, nor was there any significant difference in the severity of IVH between this group of 12 patients and the 56 in whom the etiology was undetermined.

In most of the 29 patients with repeat scans, follow-up lasted one to four weeks (Fig. 8). While the association of delayed hydrocephalus with blood in the third or fourth ventricle was not statistically significant \( (P > 0.3) \), its association with subarachnoid hemorrhage was \( (P = 0.004) \) (TABLE III).

Outcome is shown in Figure 9. There was only 1 patient in category IV; however, as a permanent vegetative state is as serious as death for the patient and family, categories IV and V are grouped together here. The outcomes in the four categories are shown in Figure 10. Compared to the mortality rates in trauma (71%), hypertensive hemorrhage (62%), and ruptured aneurysm (53%), patients with bleeding of undetermined cause had a considerably better prognosis: the mortality rate was only 25%, while 50% survived with no deficit or only mild disability. If the 4 patients who were not investi-
gated and hence may have a discoverable cause of IVH are excluded, the mortality rate was only 1 in 8 (12.5%). The more severe the IVH, the worse the prognosis; indeed, 90% of patients with severe IVH died (Fig. 11).

IVH is seldom an isolated event. In 87% of our cases, additional abnormalities were seen on CT (TABLE IV). Patients with a poor outcome also had a higher incidence of associated abnormalities (Fig. 12). Although only 9 of our patients (13%) had no additional abnormalities, 7 had a good or excellent outcome (category I or II). In all cases, factors other than IVH were sufficient to explain the neurological outcome. In 63 cases the cause was a parenchymal hematoma, midline shift, angiographically demonstrated spasm, or injury to other organs. Of the 5 remaining patients, 3 suffered massive re-bleeding and died before a repeat CT scan could be obtained, while 2 died of postoperative neurological complications.

**DISCUSSION**

Although aneurysms of the anterior cerebral artery and its branches made up 65% of our series, they account for only 36% of all ruptured aneurysms with subarachnoid bleeding (10). Other authors have noted the increased likelihood of intraventricular rupture from aneurysms of these vessels, particularly the ACA (8, 11). Aneurysms of the ACA may readily rupture into the lateral ventricles, as only the relatively thin olfactory and paraterminal gyri separate the aneurysms from the frontal horns.

Our experience supports Yock’s report of the CT appearance of a ruptured ACA aneurysm (12). The most characteristic feature is a hematoma localized to the anterior interhemispheric fissure, just anterior to the lamina terminalis. Although previously described as an uncommon event (11), CT suggests that it is actually a relatively frequent finding which can be used to predict the site of the aneurysm based on the CT scan, something which was not possible with ruptures at other sites.

Although subarachnoid bleeding from cerebral metastases (particularly malignant melanoma) is known to occur (11, 13), intraventricular bleeding from metastases has not been reported previously to our knowledge. While the CT appearance was not specific for metastases, the angiographic demonstration of tumor vessels supports this diagnosis (Fig. 7).

The overall mortality rate of 50% in our series (Fig. 9) is considerably lower than the 83% reported by Little et al. (8), which is only partly explained by the greater percentage of cases of undetermined cause in our series (18% vs. 4%). Fourteen patients (21%) returned to normal or had only mild disability, while 10 (15%) survived with moderate disability.

As demonstrated with subarachnoid bleeding of undetermined etiology (14, 15), patients with IVH of unknown cause have a significantly better prognosis. Just as microscopic vascular
malformations have been postulated as a cause of subarachnoid bleeding of undetermined etiology (16), similar malformations adjacent to the ependymal lining of the ventricles may be responsible for some cases of IVH in which no cause is found.

Approximately 50% of our patients had mild enlargement of the lateral ventricles on the initial CT scan. As no previous scans were available and many patients were at an age when atrophy could be expected, this finding was difficult to interpret; however, any increase in ventricular size on subsequent scans was considered significant.

Allcock (17) has stated that development of hydrocephalus after IVH may be due to obstruction of the flow of cerebrospinal fluid (CSF) at the foramen of Monro or aqueduct of Sylvius; although this is likely to be true in acute cases, our study shows that hydrocephalus which develops at least one week after IVH can be correlated with subarachnoid hemorrhage on the initial CT scan rather than with IVH in the third or fourth ventricle. In such cases, the hydrocephalus is more likely to be communicating (due to oblitative arachnoiditis in the posterior fossa or impaired resorption of CSF in the arachnoid granulations) than obstructive (18, 19).

The effect of IVH on outcome could perhaps be assessed most accurately in a prospective fashion by comparison with a control group without IVH; however, obtaining an unbiased control group presents a problem in that the ultimate severity of brain damage is difficult if not impossible to evaluate prospectively. Our approach was to correlate the clinical record and the CT scan with outcome in each case. In our series, outcome was determined by associated brain injury rather than by IVH itself. Factors such as parenchymal hematoma, midline shift, cerebral edema, and vasospasm had predictable effects which in each case were sufficient to explain the neurological outcome. Patients with IVH and no additional CT abnormalities had a much better outcome, and those in whom parenchymal damage was confined to "silent" areas of the brain (such as the frontal lobes) also did well. Our findings indicate that IVH per se is seldom a major contributor to the neurological outcome in these patients.

As CT is now capable of disclosing IVH in many cases where it could not be diagnosed clinically, this finding by itself should not be overemphasized. In the great majority of cases, the eventual outcome will be determined by the presence or absence of associated brain abnormalities. Even severe IVH need not be cause for a gloomy prognosis in all cases.

SUMMARY

Sixty-eight patients with CT-diagnosed IVH were reviewed retrospectively. The most frequent causes were a ruptured aneurysm, trauma, and hypertensive hemorrhage; less frequent causes included bleeding from an arteriovenous malformation, a hemorrhagic diathesis, or probable metastases. In 12 patients the cause was undetermined. Ruptured aneurysms of the anterior cerebral artery and its branches (including the ACA) caused IVH more often than other aneurysms and could frequently be predicted by their appearance on non-enhanced CT scans.

The overall mortality rate was 50%. However, 21% of patients returned to normal or had only mild disability, while 15% had only moderate disability. Patients with IVH of undetermined cause had a much better prognosis. IVH due to cerebral trauma carried the highest mortality rate (71%) and would appear to be an ominous finding. Hydrocephalus which develops one week or more after IVH can be correlated with subarachnoid bleeding on the CT scan rather than with blood in the third or fourth ventricle and is therefore more likely to be communicating than obstructive. IVH per se seldom has a major effect on the neurological outcome.

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References