Long-Term Prognosis of Hypertensive Intracerebral Hemorrhage

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SUMMARY The diagnosis of intracerebral hemorrhage (ICH) has become precise with the advent of computerized tomography (CT). Little, however, is known concerning the long-term prognosis.

Seventy consecutive patients with primary intracerebral hemorrhage (all known etiologies except hypertension excluded) proven by CT scan were studied. Follow up, averaging 2½ years, was successful in all cases. The status of alertness, EKG, and clinical impression on admission were significant prognostic factors. As expected, mortality increased with size of the hematoma and ventricular rupture. Acute in-hospital mortality was 40%. Another 17% died during the long-term follow up, but none of them from cerebrovascular disease. Ninety-two percent of the survivors were ambulatory at follow up. Hypertensive intracerebral hemorrhages, unlike aneurysms, rarely, if ever, rebleed. Patients are not likely to have a second bleed in another location. Hypertensive intracerebral hemorrhage is more common in blacks, especially young adult males with severe hypertension, but overall mortality is lower than thought prior to the CT scan. Most survivors can achieve independence and deserve aggressive rehabilitation efforts.

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Table 1  Size of Bleed Versus Outcome

<table>
<thead>
<tr>
<th>Size of bleed</th>
<th>Died in hospital (ventricular extension)</th>
<th>Dead at follow-up (ventricular extension)</th>
<th>Alive at follow-up (ventricular extension)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small (&lt; 2 cm)</td>
<td>1 (0)</td>
<td>3</td>
<td>8 (2)</td>
</tr>
<tr>
<td>Medium (2-5 cm)</td>
<td>9 (3)</td>
<td>3</td>
<td>15 (7)</td>
</tr>
<tr>
<td>Large (&gt; 5 cm)</td>
<td>18 (14)</td>
<td>1</td>
<td>12 (2)</td>
</tr>
<tr>
<td>Total</td>
<td>28 (17)</td>
<td>7 (0)</td>
<td>35 (11)</td>
</tr>
</tbody>
</table>

Table 2 shows that all patients presenting in coma died during the initial hospitalization, and that the less impaired the state of alertness, the better was the prognosis. A normal admission EKG and lack of signs of intracranial hemorrhage (in general, signs of subarachnoid blood or mass effect) also correlated with a good prognosis.

Table 3 illustrates that 39% of patients with thalamic-ganglionic hemorrhages died during their initial hospitalization and 52% were alive at the end of follow up. Thirty-one percent of patients with lobar hemorrhages died during initial hospitalization and 50% were alive at the end of follow up.

Overall, there seemed to be a disproportionate number of black patients in this series (57 or 81%) compared to our hospital's total racial distribution (65% blacks). Unexpectedly, the youngest age group had the highest early mortality (table 4). Almost all of these were young blacks. However, regardless of race or sex, the overall mortality trends were similar (table 5).

Seven patients died during the follow up period. The causes of death were: myocardial infarction (2), other medical complications (renal failure, broken hip, and pulmonary embolus on day of discharge) and one suicide. No episodes of recurrent intracerebral hemorrhage occurred.

Of the 35 surviving follow up patients, 19 were independently ambulatory, 13 ambulatory with assistance, 3 wheelchair bound and none bedfast. Four remained aphasic. Five had gone back to work. Twenty-three had the same degree of function at follow up as at discharge, 17 had improved and 5 had worsened (all from independently ambulatory to a cane or walker).

Table 6 shows the events discovered during the follow up period.

Seven patients had their hematomas surgically treated; 3 of these died during hospitalization. Three of these 7 surgically treated patients had cerebellar hemorrhages; 2 died during hospitalization.

No seasonal periodicity was observed in the incidence of these ICH’s although May was the most common month (13) and July the least (2), while the other months varied (4-7).

All but 6 of the follow up patients were still on antihypertensive medication at the time of the last follow up.

Discussion

These data regarding the initial hospitalization and survival are similar to recent studies except in regard to lobar hemorrhages. Twenty percent of the ICH’s in the present series were lobar in location and hypertensive in origin, so hypertension can be a relatively common cause of spontaneous lobar hemorrhage. This se-
ries of lobar ICH's has different characteristics from
table 2). Prior to CT scanning, many such
these patients were initially thought to have had an
they represent patients with less catastrophic
lobar ICH's. They may also account for the lack of
detailed definition. The present series is still too small
and Davis may account for the less favorable prognosis of
hemorrhage (table 2). Prior to CT scanning, many such
obviously, they represent patients with less catastrophic
of early hospital deaths. There was a better survival and functional ability at follow up than did those with obvious signs of hemorrhage (table 2). Prior to CT scanning, many such patients would not have been diagnosed as ICH. Obviously, they represent patients with less catastrophic initial CNS involvement than the others.

Because the large majority of these patients were primary care patients from the University of Mississippi Medical Center (the V.A. patients consisted of less than 20% of the total), this population of ICH cases was not greatly biased toward less severe hemorrhages as might occur at a tertiary referral center. Of the differences in location and size between our patients and those of Ropper and Davis may account for the less favorable prognosis of our lobar ICH's. They may also account for the lack of difference in outcome between our lobar ICH patients and those with hemorrhages in other locations (table 3).

The specific clinical syndromes associated with ICH that relate to a good long-term prognosis deserve more detailed definition. The present series is still too small to permit confident conclusions when further subdivisions of the clinical categories are attempted.

It is noteworthy that a significant number (28) of these patients were initially thought to have had an infarct. They had a better survival and functional ability at follow up than did those with obvious signs of hemorrhage (table 2). Prior to CT scanning, many such patients would not have been diagnosed as ICH. Obviously, they represent patients with less catastrophic initial CNS involvement than the others.

Because the large majority of these patients were primary care patients from the University of Mississippi Medical Center (the V.A. patients consisted of less than 20% of the total), this population of ICH cases was not greatly biased toward less severe hemorrhages as might occur at a tertiary referral center. Of course, some patients with catastrophic ICH will not survive to reach even a primary medical treatment facility.

Not all patients had arteriograms. It is unlikely that

any aneurysms were missed since their high rate of recurrent hemorrhage was not found at follow up. The utility of the CT scan in identifying ICH is widely recognized and the diagnosis is extremely accurate. It was felt ethically unacceptable to perform arteriography in cases where the diagnosis was clear and no surgically treatable lesion was present or the patient was moribund on admission.

Too few patients in the present series were treated surgically for any conclusion to be drawn in regard to its efficacy. It did not seem to affect the outcome in this group.

Perhaps because of the lack of wide seasonal fluctuations in daylight and temperature in our location, or because of our inclusion of all cases of primary intracerebral hemorrhage (as opposed to a mostly pathologically studied series), we could not demonstrate a periodicity of incidence.

Our data provide some specifics relating to race. The worst prognosis was not in the older age groups, as in prior studies, but in the 30–40 year old age range. These were almost all severely hypertensive blacks. There appears to be nothing unique to ICH in blacks except frequent and severe high blood pressure. The malignant type of hypertension present in this type of patient seems to lead to devastating hemorrhage with mortality despite otherwise good health.

Very few studies have been done regarding the long-term survival and residual function of survivors of hypertensive intracerebral hemorrhage. McKissock et al9 in a short six month follow up found 2% of survivors back at work and 16% with "partial disability." However, this study was done before the advent of the CT scan and was primarily concerned with survival after surgical versus conservative therapy. There was no follow up after six months and no comment was made regarding rebleeding. Paillas and Alliez11 followed a decreasing number of surgically treated ICH patients over a variable number of years and listed 11 recurrent hemorrhages in 161 patients alive one month after operation. This study was also done in the pre-CT scan era and other etiologies besides hypertension were included. All of the patients underwent surgery for their hematomas. Although a few patients were followed for over ten years, it is difficult to draw any conclusions regarding long term function because so many patients were lost to follow up. The authors concluded that if patients survived the operation and the following several years, the majority remained stable.

Autopsy studies have suggested that up to 25% of patients who died from ICHs had evidence of earlier hemorrhages.11,12 These studies were only inferential, without clinical correlation, and other causes of these pathologic lesions could not be excluded.

In the present study, there were no episodes of rebleeding nor bleeding at other sites intracranially. Forty-five percent of the survivors of initial hospitalization were fully ambulatory and another 31% ambulatory with assistance after almost 3 years. Seventeen percent died during the follow up period and a small number were back at work. Most patients' func-

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**Table 6 Events During Follow-up Period (35 Patients)**

<table>
<thead>
<tr>
<th>Event</th>
<th>Died at follow-up</th>
<th>Ambulatory with assistance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seizures</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Pregnancy</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Myocardial infarct</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Renal failure</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Hip fracture</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Thalamic pain</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Hip surgery</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Suicide</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Rebleed</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>
tion remained the same at follow up as it had been at discharge from the initial hospitalization.

In conclusion, patients who have survived a primary (hypertensive) ICH have a relatively good prognosis for recovery of function and are not likely to have a second ICH. They deserve aggressive medical care and rehabilitation efforts.

References

Cerebral Hemorrhage in Neonates with Coarctation of the Aorta

RICHARD S. K. YOUNG, M.D., * RICHARD R. LIBERTHSON, M.D., † AND EDWIN L. ZALNERAITIS, M.D. †

SUMMARY Coarctation of the aorta is an uncommon cause of cerebral hemorrhage in the full- or near-term infant. The clinical, radiologic, and neurologic findings of four infants with aortic coarctation and cerebral hemorrhage are presented. In all four infants, cerebral hemorrhage was associated with only moderate elevation of systolic blood pressure (90–110 mmHg).

CEREBRAL HEMORRHAGE is a recognized complication of coarctation of the aorta and may occur in as many as 10% of older patients with this disorder.1 However, cerebral hemorrhage in infants with coarctation is rare, and a review of the literature yielded only one such patient.2 In this report, we present the clinical history and management of four infants who had documented coarctation of the aorta and an associated cerebral hemorrhage.

Patient 1

This 3.2 kg boy was born at 36 weeks of gestation after a normal pregnancy, labor, and delivery. On the second day of life, he became lethargic, tachypneic, and tachycardic. Physical examination revealed a grade III/VI systolic ejection murmur, and blood pressures of 66/50 mmHg in the upper extremities. His lower extremity pulses were absent.

The clinical course was marked by oliguria, metabolic acidosis (pH 7.08; pO2 117 and pCO2 25 mmHg)

and coagulopathy (prothrombin time, 21/12.5 seconds; partial thromboplastin time, 61 seconds). Cardiac catheterization showed severe periductal (adult type) coarctation of the aorta and a patent ductus arteriosus. On the fourth day of life, he underwent subclavian patch angioplasty repair of the coarctation and ligation of the ductus arteriosus. Following the operation, he was placed on dobutamine. The blood pressure was 90/60 in his upper extremities and 65/50 in the umbilical artery. Several hours later, he developed tonic seizures which were controlled with anticonvulsants. The cerebrospinal fluid was bloody. A computerized tomographic (CT) brain scan showed intraventricular and subarachnoid hemorrhage, and scattered areas of low density in the white matter (fig. 1).

Both the neurologic and cardiac status gradually improved during the early post operative weeks. However, at nine months of age he had microcephaly, spastic diplegia, and extensor plantar responses.

Patient 2

This infant male was one of twins born at 36 weeks of gestation to a healthy multigravida whose pregnancy, labor, and delivery were normal. The infant initially appeared well, but by the seventh day of life was mottled and tachypneic. Examination revealed retractions, hepatomegaly, and a grade II/VI apical systolic murmur. The neurologic examination was normal. On

References

Long-term prognosis of hypertensive intracerebral hemorrhage.
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