Intraventricular hemorrhage in severe head injury

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A series of 30 patients suffering posttraumatic intraventricular hemorrhage (IVH) after closed head injury is reviewed. Clotted blood and a mixture of blood and cerebrospinal fluid could be distinguished by computerized tomography (CT). Posttraumatic IVH was associated with diffuse brain lesions in most cases; intracerebral lesions with contusion, and subdural hematomas coexisted with posttraumatic IVH in eight and four instances, respectively. In two more cases, no CT abnormality other than IVH was noted. All patients in this series were in deep coma at the time of CT examination, and only seven survived. The early clinical findings, the site of ventricular hematoma, and the final outcome are analyzed.

KEY WORDS • computerized tomography • head injury • ventricle • intraventricular hemorrhage

Intraventricular hemorrhage (IVH), either posttraumatic or spontaneous,7,11-13 was found at surgery or pathological examination before the advent of computerized tomography (CT). The CT scan, which is a rapid, noninvasive, and reliable method for identifying blood in the ventricular compartment,1-3,7-11,13,16,19 now reveals that posttraumatic IVH is not a rarity. However, the actual incidence and prognostic implication of posttraumatic IVH have not been established at the present time. We have designed this study to assess the occurrence of IVH in our series of head-injured patients, and to correlate this finding with the clinical status, the presence of associated lesions, and the final outcome of these patients.

Clinical Material and Methods

This is a retrospective analysis of 30 patients presenting with IVH after suffering a closed head injury, mostly in traffic accidents, between November, 1977, and December, 1980. There were 19 men and 11 women, with ages ranging from 2 to 78 years (average 34.6 years). These patients represented 2.1% of all head-injured patients undergoing CT scanning and 7.1% of our total series of severe head injuries during that period. Patients were considered to have severe head injury when they were unable to open their eyes to painful stimuli, to utter words, and to obey commands for at least 6 hours after the trauma or subsequent deterioration. All severely head-injured patients were studied with CT scanning, and had continuous intracranial pressure (ICP) monitoring during the acute phase. Clinical evaluation of patients was made according to the Glasgow Coma Scale (GCS),14 and their outcome was graded using the five-point scale of Jennett and Bond.5

Patients were managed with relaxant therapy and artificial ventilation on a volume respirator (end-tidal capacity 13 to 15 ml/kg body weight; rate 10 to 13 breaths/min) to maintain arterial pCO2 at 25 to 30 mm Hg and arterial pO2 over 75 mm Hg. Dexamethasone, 8 mg every 6 hours, was administered in all cases. Intracranial hypertension was vigorously treated with mannitol, and since early 1979 with high-dose barbiturate therapy (four cases). External ventricular drainage was instituted in only one patient who had developed acute hydrocephalus.

Computerized tomography studies consisted of four-level unenhanced scans performed with an EMI brain CT unit (160 x 160 matrix). Diagnosis of posttraumatic IVH was established on the admission CT scan before either surgery or ICP monitoring was undertaken. Thus, three patients showing IVH who had been operated on before having the initial CT scan were excluded from this series, as were two other patients in whom there was some doubt about the
traumatic origin of the intraventricular bleeding. Another patient with concomitant aortic rupture was also excluded. Twenty patients underwent scanning within 6 hours after trauma, four within 12 hours, five within 2 days, and one 15 days after injury. Thirteen patients had sequential CT examinations. Charts and CT scans were read by both neuroradiologists and neurosurgeons.

Results

Computerized Tomography Findings

Blood was revealed in the ventricular system with CT density values of +45 to +80 Hounsfield units, and blood mixed with cerebrospinal fluid (CSF) with densities ranging from +25 to +45 units. There were 14 patients with intraventricular blood clots, eight with a mixture of CSF and blood, and eight more with both of these findings (Table 1). Distribution of bleeding within the ventricular cavities is shown in Table 2. The most frequent location was in the lateral ventricles, particularly in the occipital horns, this being related to the supine position of patients undergoing routine CT scanning. In no patient did clots fill the entire ventricular system, and bleeding was restricted to the four ventricles in only two patients.

Posttraumatic IVH was an isolated abnormal finding in only two patients (Fig. 1). In the remainder of cases, IVH coexisted with other brain lesions (Table 3), most frequently with diffuse brain injury (Fig. 2). Gross foci of contusions occupying both cerebral hemispheres were observed in six cases. Subdural hematomas and focal cerebral contusion coexisted with intraventricular bleeding in four and two instances, respectively. Direct spread of blood from intracerebral hemorrhage was recorded in 11 cases.

Evidence of brain-stem injury, either direct or indirect, as described by Tsai, et al.,17 was found in 12 cases (Fig. 3). Obliteration of the pontine, perimesencephalic, and cerebellopontine cisterns was discovered in four patients, three of whom underwent CT scanning within the first 6 hours after injury. Brain-stem hemorrhagic contusion was present in eight patients, six of whom were scanned within 6 hours after injury; the other two patients were examined within 12 hours of trauma. In six more patients, the CT scan was not conclusive for signs of brain-stem damage. Corpus callosum lesions were observed in eight cases in the form of a hyperdense mass. Bleeding in the posterior interhemispheric region was found in 24 cases.

Changes in the appearance of intraventricular blood were seen in the sequential CT scans. Blood persisted for more than 2 weeks after injury in two patients, one of whom had IVH as the only finding (Fig. 1). In four other patients, blood disappeared 7 days after injury. Acute or subacute dilatation of the ventricular system was not observed except in one of the two patients in whom IVH was the only finding.

Clinical Findings and Outcome

All the patients in this series were comatose during the first 24 hours after admission (Table 4). During this period, 25 patients had abnormal motor responses and 28 showed abnormal pupillary changes. Six patients underwent surgery, four in order to evacuate a subdural hematoma, and two to manage an expanding cerebral contusion.

Final outcome in this series is shown in Table 2. As may be seen, only seven patients (23.3%) survived. The mean age for the survivors was 19.8 years, compared with 40.7 years for those who died. Fourteen of

<table>
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<tr>
<th>Location of Hemorrhage</th>
<th>Good Recovery</th>
<th>Moderate Disability</th>
<th>Severe Disability</th>
<th>Persistent Vegetative Status</th>
<th>Dead</th>
<th>Total Cases</th>
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<td>one lateral &amp; fourth ventricles</td>
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<td>4</td>
<td>1</td>
<td>1</td>
<td>23</td>
<td>30</td>
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* Abbreviation: IVH = intraventricular hemorrhage.
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Fig. 1. Unenhanced computerized tomography scans of a patient with posttraumatic intraventricular hemorrhage as the only finding. A: Image obtained at admission showing intraventricular bleeding in the left lateral ventricle. B: Control scan performed 6 days later showing blood in the occipital horns. C: Control scan 3 weeks after admission still showing evidence of blood in the occipital horns.

Table 3
Associated lesions in 30 patients with posttraumatic IVH related to final outcome*

<table>
<thead>
<tr>
<th>Associated Lesions</th>
<th>Good Recovery</th>
<th>Moderate Disability</th>
<th>Severe Disability</th>
<th>Persistent Vegetative Status</th>
<th>Dead</th>
<th>Total Cases</th>
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</tr>
<tr>
<td>diffuse brain injury</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>12</td>
<td>16</td>
<td>53.3</td>
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<tr>
<td>global contusion</td>
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<tr>
<td>focal contusion</td>
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<tr>
<td>subdural hematoma</td>
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<tr>
<td>none</td>
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<td>1</td>
<td>6.6</td>
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</tbody>
</table>

* Abbreviation: IVH = intraventricular hemorrhage.

Fig. 2. Unenhanced computerized tomography scans of patients with posttraumatic intraventricular hemorrhage showing associated intracranial lesions. A: Scan from a patient with diffuse brain damage. A small parenchymatous hemorrhage may be seen near the left caudate nucleus. Blood is also seen in both atria. B: Scan from a patient with multiple contusive lesions and blood filling the right ventricular body. C: Scan from a patient with acute subdural hematoma and blood in the left temporal horn.
the 23 deaths occurred within 24 hours after injury. The average best GCS score during the first 24 hours was 6.8 for the survivors and 5.2 for those who died; this score was 5.5 for the whole series. Six of the survivors had intraventricular clots, and the seventh had a mixture of CSF and blood. The prognosis was apparently not related to the location and volume of the bleeding; the only patient in this series who made a good recovery had the biggest intraventricular clot.

Intellectual disturbances were the main cause of disability among the survivors.

**Discussion**

Posttraumatic IVH is an uncommon finding discovered in 1.5% to 5.7% of all head-injured patients studied with CT scanning. The incidence of posttraumatic IVH among cases of IVH of all etiologies is difficult to estimate. Steudel, et al., recorded 21 cases of posttraumatic IVH in a series of 60 patients with ventricular bleeding from different causes, and Scott, et al., found one among 23. To ascertain the true incidence of posttraumatic IVH as a primary event, head-injured patients who have undergone any intracranial surgical procedure before the first CT scan after admission should be excluded, because such a procedure may result in bloody CSF. Bloody ventricular fluid was found in nearly 30% of our head-injured patients who underwent intraventricular pressure measurement. The CT scan is able to delineate the location and volume of the clot in patients with posttraumatic IVH as soon as a few minutes after the impact. New and Aronow have
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documented a linear relationship between the attenuation values of the extravasated blood and its hematocrit level. They stated that the attenuation of whole blood with a hematocrit of 45% is near 28 EMI units, but that clot formation results in higher attenuation values.

Disruption of the ventricular wall by an adjacent intracerebral hematoma is the best known cause of posttraumatic IVH. Zuccarello, et al., suggested that the rupture of the subependymal veins deformed by a negative pressure following sudden dilatation of the ventricular wall at the time of the impact may explain the occurrence of posttraumatic IVH as a unique finding. Some authors have raised the possibility that the spontaneous rupture of an unsuspected peripendymal vascular malformation may have caused IVH before the impact. In the present series, 11 patients were seen to have blood spreading into the ventricles from adjacent periventricular hemorrhagic lesions, and we believe that with the aid of higher-resolution CT units this pathogenic mechanism will be shown to be responsible for IVH in most instances.

Pia distinguished three types of spontaneous IVH: 1) total hematocephaly with a clot filling the entire ventricular system; 2) partial hematocephaly with blood isolated in one ventricular region; and 3) an IVH composed of mixed blood and CSF. In our series, all except one of the survivors had partial hematocephaly. The volume of the clot did not correlate with the final outcome: the two patients with the largest ventricular clots survived. In none of our patients did a clot fill the entire ventricular system. Tsai, et al., found contrast enhancement along the lateral ventricular margins in one-third of their cases of posttraumatic IVH, and stated that this finding correlated with IVH in most instances.

More than 50% of our patients with posttraumatic IVH had diffuse brain damage as the main cerebral lesion. Zimmerman, et al., demonstrated the correlation between the CT image and the gross autopsy findings in the so-called "diffuse shearing injury of the white matter." These authors defined this lesion as the absence of a specific focal mass and the presence of bilateral cerebral swelling, eccentric hemorrhages in the corpus callosum, and subarachnoid bleeding. They found two patients with posttraumatic IVH among eight with diffuse shearing injury, emphasizing the need for repeated CT scans with thin cuts in these cases.

In our series, six patients with associated diffuse brain damage exhibited corpus callosum lesions on CT scanning, and all the 16 patients with diffuse brain damage showed the characteristic small hemorrhages in the white matter of the cerebral hemispheres or bleeding in the interhemispheric subarachnoid spaces. Brain contusion and acute subdural hematomas were observed in eight and four patients, respectively, and IVH was the sole finding in only two cases (6.6%).

The high incidence of bilateral cerebral hemispheric damage (73.3%) was very noticeable in the present series. Tsai, et al., reported 11 cases of posttraumatic IVH associated with cerebral lesions, and six with IVH as the only finding. French and Dublin reported associated cerebral lesions in all their nine patients with posttraumatic IVH, and Zuccarello, et al., found only one nonautopsied case of posttraumatic IVH as the only finding among 10 patients, four of whom had postmortem examination. These last authors observed brain-stem damage at autopsy in three patients; in one, evidence of this lesion had been seen on CT scanning. In our series, 12 patients had direct or indirect evidence of brain-stem damage on the CT scan, and we believe that both brain-stem lesions and corpus callosum hemorrhages would have been shown in most cases if autopsy had been performed.

The clinical course was unfavorable in our patients with posttraumatic IVH. More than 80% exhibited abnormal motor responses or pupillary disturbances at some time in the evolution of their disease, and 40% were decerebrate until they died. All but one of the 21 patients with posttraumatic IVH reported by Steudel, et al., were comatose on admission, as were all the 10 patients studied by Zuccarello, et al. We have not seen posttraumatic IVH in patients with mild head injury, but this sign has been reported in such patients.

In our series, 76% of our patients died and only one made a good recovery. The overall mortality rate for severe head injury in our department is approximately 40% (unpublished data), and almost all deaths are related to the three types of intracranial lesion which were associated with posttraumatic IVH in the present series, namely, diffuse brain lesion, brain contusion, and acute subdural hematoma (Table 3). Since these lesions carry a poor prognosis by themselves, even unassociated with posttraumatic IVH, it is difficult to ascertain the influence of this last pathological finding on the final outcome. However, we believe that the presence of intraventricular blood aggravates the prognosis of patients with these posttraumatic lesions. Deep coma at admission, early ventricular dilatation, and high attenuation values of the extravasated blood have been reported to correlate with poor prognosis in patients with posttraumatic IVH. Age of patients and early level of consciousness were clearly correlated with the final outcome in our series. Nevertheless, no correlation was found between prognosis and the attenuation values of the ventricular blood, and the only patient with acute hydrocephalus died despite external drainage.

In our experience, posttraumatic IVH is an uncommon sign found only in severe head injuries, and is often associated with diffuse brain damage. This phenomenon carries a very poor prognosis.

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References


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