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Acute and Massive Intraparenchymal Hemorrhages of the Posterior Fossa†

Dennis E. Wilkins, MD *

Acute, massive cerebellar and pontine hemorrhages are discussed in the context of the routine availability of cranial computerized tomography. The diagnosis of these disorders is emphasized. An updated clinical review of the etiologic, pathogenetic, and therapeutic aspects is also presented, with particular attention to the importance of hypertensive cerebrovascular disease as the most common underlying disorder.

Cerebellar and pontine hemorrhages can now be more readily diagnosed with computerized tomographic scanning (CT scan) than before it became available on a routine basis (1-5). In addition, for cerebellar hemorrhages, the CT scan has helped to document various prognostic and therapeutic categories (6-8). To use and interpret these advances, this paper provides an updated clinical review, emphasizing massive hemorrhages in the cerebellum of at least 3.0 cm in diameter and in the pons of 1.5 cm or more (5).

Etiology

Although recent evidence suggests that cerebrovascular accidents may be declining, a consistent decrease for intracerebral hemorrhage could not be documented (9). Earlier studies indicated that cerebellar hemorrhage was found in 0.4% of 3,881 consecutive autopsies. It represented 10% of all intraparenchymal, central nervous system hemorrhages, whereas pontine hematomas accounted for 7% (4-10). Significantly, the cerebellum comprises 10% of the brain's weight; the lower percentage for pontine hematomas also corresponds to its approximate size (4-5). The mean age of incidence is 64 years for the cerebellum and 54 years for the pons, a distribution essentially determined by underlying hypertensive cerebrovascular disease (5). Table I lists representative etiologies of posterior fossa hemorrhages and illustrates the marked predominance of hypertensive cerebrovascular disease.

Coagulopathy encompasses many conditions that alter the hemostatic efficiency of platelets and/or plasma clotting factors. In a report of 58 intracranial hemorrhages associated with coagulopathies, 51 were related to thrombocytopenia that occurred most frequently as a complication of leukemia, four were in hemophiliacs, and three could be attributed to the prothrombin deficiency of anticoagulant drugs or liver disease (11). Fifty-six patients had evidence of bleeding elsewhere, and four presented initially with intracranial hemorrhage. Of the 58 hemorrhages, one was pontine and three were cerebellar. A study of the complications of chronic anticoagulant therapy determined a 2% risk of major intracranial bleedings in patients treated for an average of 18 months (12). The risk of hemorrhages could not be closely correlated with prothrombin activity.

Most posterior fossa hemorrhages due to vascular malformations occur between the ages of 10 and 40, and most malformations are less than 2 cm in diameter (13) (Table II). Although all types produced lethal hemorrhages, a directly proportional relationship seems to exist between size and propensity for hemorrhage. In the series of McCormick, et al (13), 42 of 51 hemorrhages occurred with the larger arteriovenous malformations, whereas none of 38 small telangiectases were recognized as having bled. Erenberg, et al (14) examined 23 additional cases and confirmed McCormick's earlier observations. It must be kept in mind,
however, that if smaller vascular malformations are destroyed by the hemorrhage itself, no evidence of their existence will be found at autopsy.

It should be stressed that hypertensive cerebrovascular disease is of unsurpassed importance in causing posterior fossa hemorrhages. In 1959, Feigin and Prose (15) described a fibrinoid arteriolitis involving cerebral vessels 0.2 to 0.8 mm in diameter in brains sustaining a gross, hypertensive hemorrhagic insult. Microscopic aneurysmal dilatation of some arterioles and frequent extravasation of red cells into the perivascular space were also noted. As early as 1868 these dilatations, or microaneurysms, were recognized as critical lesions by Charcot and Bouchard (16), but it remained for Cole and Yates (17) in 1967 to establish a firm pathological-clinical correlation between these vascular alterations and massive hypertensive hemorrhages (Tables III and IV). The microaneurysms measured 0.5 to 2.0 mm in diameter. In the cerebellum they typically were found at branching points of penetrating vessels from the superior cerebellar artery or from the posterior inferior cerebellar artery near the dentate nuclei. Microaneurysms within the pons were frequent and most conspicuous in the paramedian arterioles of the tegmentobasilar junction.

### Pathology

A hypertensive cerebellar hemorrhage in the region of the dentate nucleus often expands to 4 to 5 cm in diameter. Slightly more than half of these hematomas rupture into the fourth ventricle, and 40% extend into the vermis (1). Only a few hemorrhages remain totally confined to the hemisphere of origin. The mass of blood displaces rather than disrupts tissue, and most clinical signs can be attributed to pressure upon the underlying brainstem. This concept is best exemplified by vermal hematomas that represent 5% of hypertensive cerebellar hemorrhages. Their course is more fulminant due to an earlier and more direct compression of the subjacent brainstem (5). Hemorrhages due to vascular malformations or coagulation disorders, though somewhat variable, are often situated lateral to the dentate nucleus and uncommonly rupture outside the cerebellum.

Massive hypertensive pontine hemorrhages, characteristically within the paramedian tegmentobasilar junction, tend to dissect symmetrically in all directions to a widely varying extent; total hematoma volume may range from 20 to 40 ml (18). About 80% rupture into the fourth ventricle, 70% extend into the midbrain, and 10% reach the thalamus (5,18). While reportedly as many as 10% of the hemorrhages enter the medulla (4), this caudal dissection is not universally recognized (5,18).

A second form of acute pontine hemorrhage that has recently been distinguished represents a significant minority of hemorrhagic lesions in this area (18). Characteristically 10 ml or less in volume, it is contained unilaterally within the pontine tegmentum except for an occasional rupture into the fourth ventricle. The etiology of this disorder remains speculative at this time.

At autopsy, additional gross vascular lesions of the central nervous system accompany 80% of the cerebellar and 60% of the pontine hemorrhages; the lacunar state was obvious in 10% (5).

### Clinical Features

As a distinctive clinical syndrome, acute cerebellar hemorrhage was first described by Fisher, et al in 1965 (1). These basic clinical features were applied by Ott's group (2) to correctly diagnose two thirds of all acute hemorrhages on
Cerebellar and Pontine Hemorrhages

TABLE III
Relationship of Microaneurysms, Hemorrhages and Hypertensive Cerebrovascular Disease (15)

<table>
<thead>
<tr>
<th></th>
<th>100 Hypertensive Patients</th>
<th>100 Normal Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microaneurysms</td>
<td>46</td>
<td>7</td>
</tr>
<tr>
<td>Massive hemorrhages</td>
<td>20</td>
<td>1</td>
</tr>
<tr>
<td>Both</td>
<td>18</td>
<td>0</td>
</tr>
</tbody>
</table>

TABLE IV
Distribution of Microaneurysms (15)

<table>
<thead>
<tr>
<th>Microaneurysm Location</th>
<th>100 Hypertensive Patients</th>
<th>100 Normal Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrum</td>
<td>31</td>
<td>7</td>
</tr>
<tr>
<td>Cerebrum and cerebellum</td>
<td>11</td>
<td>0</td>
</tr>
<tr>
<td>Cerebrum and cerebellum and pons</td>
<td>4</td>
<td>0</td>
</tr>
</tbody>
</table>

TABLE V
Surgical Results in Acute Massive Cerebellar Hemorrhage

<table>
<thead>
<tr>
<th>Author</th>
<th>Survived</th>
<th>Died</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ott, et al (2)</td>
<td>14</td>
<td>14</td>
<td>28</td>
</tr>
<tr>
<td>Brennan and Bergland (3)</td>
<td>6</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>Little, et al (17)</td>
<td>4</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Plum and Posner (20)</td>
<td>6</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td>21</td>
<td>51</td>
</tr>
</tbody>
</table>

can all too easily be dismissed as functional. Other signs present in more than half the cases include diastolic hypertension greater than 110 mmHg; dysarthria, a nearly constant finding in alert patients and occasionally the most prominent disturbance; an ipsilateral peripheral facial palsy; and small reactive pupils. When present, an abnormality of the ipsilateral abducens nucleus has substantial localizing and diagnostic value. The dysfunction may range from a simple lateral rectus paresis to a complete conjugate gaze palsy with contralateral ocular deviation.

A few cerebellar hemorrhages, most typically those of vermian origin, are characterized by a sudden lapse into coma. The presence of coma is the clinical link between cerebellar and pontine hemorrhages, as it is a cardinal feature of the latter. Indeed, pontine hemorrhage is manifested by the apoplectic onset of coma in 75% of patients, and more than 90% become comatose within 12 hours (4,5). In addition, a dramatic motor abnormality is invariably present. According to Silverstein (4), quadriplegia or generalized flaccidity is evident in 66% of patients with pontine hemorrhage, fixed hemiplegia in 20%, alternating hemiplegia in 10%, and decerebrate rigidity in 4%. As with cerebellar hemorrhage, severe hypertension is common.

Disordered ventilatory patterns occur frequently and early in pontine hemorrhage. Miotic, possibly anisocoric, pupils often show no macroscopic reaction to light for the first 24 hours. In more than half of the patients a dramatic hyperthermia ranging from 39.5°C to more than 45°C usually heralds an unrelenting and fatal deterioration. Abnormalities of the oculocephalic and oculovestibular reflexes frequently exist and aid in localization. An intriguing aspect to the clinical presentation of pontine hemorrhages noted by Silverstein (4) in 14% of his patients was the occurrence of a generalized convulsion as the initial event. Such a possibility is a significant although presently unexplained phenomenon, since seizures are not usually associated with primary pontine disorders.

The natural history of cerebellar hemorrhage indicates that 20% of patients will lapse into coma acutely, 60% will become comatose “subacutely” within one to two days, while the remaining “chronic” 20%, though occasionally deteriorating after several days, are likely to recover (20). Ultimately, 50% of patients die within the first 24 hours and 75% within the first week (2). Only one spontaneous recovery from coma has been formally documented (2). This correlation of an alert mental state with a favorable natural history has led to the suggestion that awake patients who manifest a stable course should be followed by serial clinical and computerized tomographic evaluations rather than undergo immediate surgery (6-8). Autopsy studies confirm the existence of a “benign” form of cerebellar hematoma, Fisher, et al (4) reporting four nonfatal hemor-
rhages from a series of 11 patients, and four other nonfatal events were noted within a series of 19,093 consecutive autopsies (5). These statistics, however, emphasize rather than temper the overall grim outlook.

Major reviews of acute, massive pontine hemorrhage indicate that most patients die within the first 24 hours; no survivors are noted in these reports (4,5). In rare cases, a patient with a unilateral hemorrhage will recover (21,22); the author has cared for such a patient.

**Laboratory diagnosis**

As soon as the clinical diagnosis of an acute, intraparenchymal posterior fossa hemorrhage has been made, definitive elucidation by computerized tomography is advisable (Figs. 1 and 2). Muller, et al (21) reported that they had no false negative tomographic evaluations in over 2,000 intracranial hematomas greater than 1.0 cm in diameter. Scott, et al (23) have confirmed this accuracy and emphasize the high quality achieved by the CT scan in demonstrating the full extent of the hemorrhage, the contribution of edema, the total mass effect, and the presence of extraparenchymal blood or hydrocephalus. The CT scan greatly aids in eliminating potential confusion between these hemorrhages and cerebellar or brainstem infarcts or with supratentorial processes. In addition, cerebral angiography may eventually be necessary to substantiate underlying conditions. Among routine laboratory determinations, coagulation parameters assume paramount importance and must be rigorously watched.

**Treatment**

Ballance in 1906 (24) was the first to evacuate a cerebellar hematoma. In 1960, McKissock, et al (25) demonstrated that routine operations could be performed with an acceptable mortality rate. Then in 1965, when Fisher, et al (1) determined that acute, massive hemorrhage warranted acute surgery, a new analysis of the benefit-risk ratio of surgery became mandatory (Table V). The 60% surgical survival rate compares favorably with the 20% survival implicit in the natural history. The same favorable outcome is mirrored in the report of Erenberg, et al (14) on 10 children with bleeding vascular malformations. Though quality of survival is not systematically analyzed in all series, some authors report complete functional recovery in a significant number of their patients after surgery (Table V).

But should all patients be operated upon? Since only a single patient who was comatose due to acute, massive cerebellar hemorrhage has been documented as surviving with conservative care alone (2), the argument against craniotomy has little merit in such a setting. There should be no hesitation even if a patient is near death, since more than one author has commented upon the successful reversal of brainstem failure. Secondly, according to Fisher, et al (1), no patient with bilateral extensor plantar responses has survived without surgery. Thirdly, patients with significant extraparenchymal hemorrhage or hydrocephalus, as evidenced by CT scan, have a poor prognosis without surgical intervention (17,18). Nevertheless, there remains a group of alert or slightly drowsy patients for whom none of the aforementioned criteria apply. Advocates for surgery would...
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point out that rapid deterioration may occur at any time and that surgical survival is directly correlated to preoperative mental status. On the other hand, ample evidence has accumulated for the existence of a definable minority of patients who will recover without surgery. Heinman, et al (6) have proposed that alert patients who have a stable clinical course and who lack progression by serial tomography should be followed without surgery. Theodore, et al (8), who stress that a stable course is the crucial foundation for conservative management, even question the necessity of surgery in every case characterized by lethargy or hydrocephalus or bilateral extensor plantar responses. Once a patient has survived for one week with medical therapy alone the survival rate for the natural history of cerebellar hemorrhage then meets or exceeds the surgical survival rate. Therefore, after this time, operation should be undertaken reluctantly (2,10).

In 1932, Dandy (26) demonstrated that a hematoma could be evacuated from the brainstem, even though the reported lesion was four years old. Today, the treatment of pontine hematomas remains at the level of anecdotal reports. The successful removal of massive (i.e. greater than 1.5 cm in diameter) pontine hematomas has been recorded on three occasions (27-29). These cases are remarkable for an average patient age of 7.5 years, a tegmental location, the absence of intraventricular rupture, and a conspicuous lack of hypertensive cerebrovascular disease. One case was related to an angioma, and the other two were of unknown etiology.

Hypertensive pontine hemorrhage poses formidable therapeutic difficulties. About 90% of patients are comatose within 12 hours, which is an unfavorable preoperative state, and a fulminant process within the compact brainstem develops rapidly even as surgery is contemplated. Finally, the tegmentobasilar junctional location of the hypertensive bleeding frequently involves the reticular activating system. Although these negative features argue strongly and persuasively against surgery, hypertensive pontine hemorrhage is uniformly fatal, except for rare unilateral cases which may or may not have a hypertensive basis. Consequently, an attempt at surgical evacuation may be justified; certainly the natural history cannot be made worse.

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References


