Acute Extradural Hematoma of the Posterior Fossa

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Extradural hematoma in the posterior fossa is rare and often is not considered in cases of head injury.

In 1938 McKenzie recorded a case of a child who, 30 hours after a fall from a chair, became stuporous and died 14 hours later. Autopsy revealed an extradural hematoma of the posterior fossa. In his comment he stated: "I had never heard of extradural hemorrhage over the cerebellum; the diagnosis was not even suspected." He regarded the case as a pathological curiosity, reported "in the hope that some day an acute clinician will diagnose such a condition." This hope was realized when Coleman and Thomson published a similar case with successful treatment; since then there have been other such reports. The literature on this subject has been reviewed by Beller and Peyser, by Campbell et al., and, more recently, by Reigh and O'Connell who were able to collect 80 cases. Others can be found elsewhere.

Most of the reported cases followed a subacute course, with the symptoms appearing several days after an injury to the occiput. This subacute form has been adequately described. Emphasis has been laid on the slow development of this type of hematoma, since it usually results from slow bleeding from a dural sinus, in contrast to the strong rapid bleeding from the middle meningeal artery in supratentorial hematomas.

Occasionally this type of hematoma does occur with strikingly acute symptoms leading to death in a few hours. Even in these cases prompt diagnosis and surgical intervention may save the patient. We have recently seen and successfully treated a case of this sort.

Case Report

A 17-year-old boy was found unconscious at the foot of the steps of his school and was admitted to the hospital 2½ hours later. It was related that, at the age of 10, he had an illness regarded as encephalitis.

Physical Examination. He was in deep coma with manifestations of decerebrate rigidity; both pupils were equally dilated and unreactive. His blood pressure was 120/80, his pulse 72 per min., the breathing stertorous at 50 per min., and the fingernails were cyanotic. He responded to painful stimulation by movements of pronation and hyper-extension, although the left limbs showed less movement. There was generalized hyperreflexia, with patellar and ankle clonus and bilateral extensor plantar responses. There was persistent eye deviation to the left and marked neck stiffness.

Radiological Examination. A curvilinear fracture extended close to the midline of the occiput, crossing the sulcus of the transverse sinus (Fig. 1). Right carotid angiography showed deviation of the pericallosal arteries in the antero-posterior projection, probably the result of rotation of the head and not an indication of sub-falcine herniation. A mild degree of dilatation of the lateral ventricle was suspected in the lateral projection (Fig. 2); the lateral displacement of the thalamo-striate vein in the antero-posterior projection was considered further evidence (Fig. 3). Slowed cerebral circulation was suggested by the fact that the early venous phase was only reached in the

Fig. 1. Curvilinear occipital fracture.
slowly returned. After 3 weeks he was out of bed, but showed signs of spasticity, ataxia, and dysarthria plus dysmetria of the left upper limb; he vomited occasionally.

Within 6 months there was considerable improvement. He had returned to his studies and was fully independent, but complained of headache, giddiness and difficulty in walking. The legs were still spastic; there were hyperreflexia and bilateral Babinski responses, dysdiadochokinesis and slurred speech.

**Comment.** This patient had been admitted in extremely poor condition with decerebrate rigidity and fixed dilated pupils. The left hemiparesis at first suggested a supratentorial lesion but the demonstration of an occipital fracture and an angiographic suggestion of hydrocephalus pointed to the correct diagnosis and effective surgical therapy.

**Review of Cases**

We have found 12 other cases in which coma actually developed within 12 hours (Table 1)\(^5,12,13,17,22\) and which we arbitrarily termed “acute”. Table 1 also shows that 7 cases suffered immediate unconsciousness followed by a period of recovery. In 3 cases, the original injury was so mild as not to cause

![Fig. 2. Right carotid angiography, lateral projection showing wide sweep of pericallosal arteries.](image)

3rd film; by this time, in our standard technique, we expect the dural venous sinuses to be visualized. During the angiographic examination the blood pressure rose to 180 systolic, the pulse to 120 per min., and the right pupil became more dilated.

The findings taken together suggested a state of mild hydrocephalus associated with acute intracranial hypertension. In the presence of an occipital fracture, a posterior fossa hematoma was considered a strong possibility.

**Operation.** Bilateral suboccipital burr-holes revealed a large fresh extradural hematoma about 3 cm. thick. Through a suboccipital craniectomy some 60 cc. of blood, partly clotted and partly liquid, were removed. The bleeding was found to be coming from a tear of the torcular Herophili and from torn small dural vessels. Hemostasis was achieved with gelfoam and suturing of the dura to the epicranium at the torcular. The dura over the cerebellum was incised but this did not reveal any further abnormality. A Penrose drain was incorporated in the wound closure and tracheostomy performed.

**Postoperative Course.** Immediately after operation, the pupils returned to normal size and reacted to light; breathing became quieter and slower. Twelve hours later the signs of decerebration and the left hemiparesis had disappeared. The patient’s condition was critical for several days but, over the next 2 weeks, consciousness

![Fig. 3. Same, venous phase, antero-posterior. The widened arc of thalamo-striate vein indicates ventricular dilatation, without shift. Early venous filling in the third film of the series indicates slowed circulation.](image)
Acute Extradural Hematoma of the Posterior Fossa

TABLE 1
Cases of acute extradural hematoma of the posterior fossa

<table>
<thead>
<tr>
<th>No.</th>
<th>Author</th>
<th>Year of Publication</th>
<th>Age</th>
<th>Immediate loss of Consciousness</th>
<th>Lucid Interval</th>
<th>Hours elapsed from Injury to Coma</th>
<th>Occip. Fracture</th>
<th>Origin of Bleeding</th>
<th>Expl. of Post. Fossa</th>
<th>Survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>McKenzie⁹</td>
<td>1938</td>
<td>?</td>
<td>+</td>
<td>+</td>
<td>2</td>
<td>?</td>
<td>Extension from middle fossa</td>
<td>-</td>
<td>no</td>
</tr>
<tr>
<td>2</td>
<td>Gordy¹⁰</td>
<td>1948</td>
<td>20</td>
<td>+</td>
<td>+</td>
<td>12</td>
<td>+</td>
<td>Sin. trans.</td>
<td>-</td>
<td>no</td>
</tr>
<tr>
<td>3</td>
<td>Grant &amp; Austin¹¹</td>
<td>1949</td>
<td>26</td>
<td>+</td>
<td>+</td>
<td>11-13½</td>
<td>+</td>
<td>Torcular</td>
<td>+</td>
<td>yes</td>
</tr>
<tr>
<td>4</td>
<td>Herren &amp; Zeller¹²</td>
<td>1950</td>
<td>60</td>
<td>-</td>
<td>+</td>
<td>8½</td>
<td>+</td>
<td>Sin. trans.</td>
<td>-</td>
<td>no</td>
</tr>
<tr>
<td>5</td>
<td>Herren &amp; Zeller¹²</td>
<td>1950</td>
<td>49</td>
<td>-</td>
<td>+</td>
<td>4</td>
<td>+</td>
<td>Sin. trans.</td>
<td>-</td>
<td>no</td>
</tr>
<tr>
<td>6</td>
<td>Munslow²²</td>
<td>1951</td>
<td>25</td>
<td>-</td>
<td>+</td>
<td>3</td>
<td>+</td>
<td>Sin. trans.</td>
<td>+</td>
<td>no</td>
</tr>
<tr>
<td>7</td>
<td>Beller &amp; Peyser¹</td>
<td>1952</td>
<td>18</td>
<td>+</td>
<td>-</td>
<td>0</td>
<td>+</td>
<td>Sin. trans.</td>
<td>-</td>
<td>no</td>
</tr>
<tr>
<td>8</td>
<td>Campbell et al.²</td>
<td>1953</td>
<td>64</td>
<td>+</td>
<td>+</td>
<td>2½</td>
<td>+</td>
<td>Not determined</td>
<td>-</td>
<td>no</td>
</tr>
<tr>
<td>9</td>
<td>Hooper¹⁴</td>
<td>1954</td>
<td>20</td>
<td>+</td>
<td>(+)</td>
<td>2</td>
<td>+</td>
<td>Sigm. sin.</td>
<td>+</td>
<td>yes</td>
</tr>
<tr>
<td>10</td>
<td>Hooper¹⁴</td>
<td>1954</td>
<td>49</td>
<td>+</td>
<td>+</td>
<td>4</td>
<td>+</td>
<td>Not determined</td>
<td>+</td>
<td>no</td>
</tr>
<tr>
<td>11</td>
<td>Salecy et al.²⁵</td>
<td>1954</td>
<td>30</td>
<td>+</td>
<td>+</td>
<td>2</td>
<td>+</td>
<td>Dura vessels</td>
<td>+</td>
<td>yes</td>
</tr>
<tr>
<td>12</td>
<td>Petit-Dutaillis et al.²³</td>
<td>1956</td>
<td>51</td>
<td>+</td>
<td>+</td>
<td>2</td>
<td>-</td>
<td>Not determined</td>
<td>-</td>
<td>no</td>
</tr>
<tr>
<td>13</td>
<td>Kosary et al.</td>
<td>1966</td>
<td>17</td>
<td>+</td>
<td>-</td>
<td>0</td>
<td>+</td>
<td>Torcular</td>
<td>+</td>
<td>yes</td>
</tr>
</tbody>
</table>

Initial loss of consciousness; 2 cases were in deep coma from the onset and 1 was stuporous.

During the lucid interval most patients complained of severe occipital headache and were dizzy and vomited. At this stage neurological signs were usually absent, apart from occasional neck-stiffness. Blood pressure was normal or elevated, the pulse usually fast and the pupils equal and reactive. Within a few hours drowsiness accompanied by restlessness deteriorated into deep coma. Ten cases were in this stage within 4 hours of the injury. In 3 cases coma was sudden, accompanied by respiratory arrest with cyanosis, while 3 other cases developed respiratory arrest a short time after becoming comatose.

The following neurological signs were usually detected after the patients were comatose (Table 2): 6 showed hyperreflexia and bilateral Babinski responses, 2 of them being in a state of decerebrate rigidity; 5 other cases showed hypotonia and areflexia. Campbell’s case switched from areflexia to decerebration at the moment that he became comatose. Confusing unilateral signs were found in 7 cases; 4 showed mydriasis, and 4 hemiparesis. In our case there was also lateral deviation of the eyes.

The radiological contribution to the diagnosis, in the cases reviewed, consisted only in detection of the occipital fracture, which was found in 7 cases. Two others did not show it on the roentgenogram while in 2 more no films were obtained. In the case of Petit-Dutaillis et al.²³ the verified fracture was temporal.

In 4 of the 13 cases there was no surgical treatment. In 3 cases supratentorial burr-holes only were made. The posterior fossa was explored in 6 cases and the hematoma evacuated in 2 of these after supratentorial exploration. Only 4 of these 6 cases survived. In the 2 fatal cases the hematoma was not evacuated until after the patient had ceased to breathe and had been maintained on the
respirator for from 1 to 3 hours. Only 1 of Hooper’s cases\textsuperscript{14} had a successful outcome. All of his cases had burr holes made with the patient in bed and without aseptic precautions.

In most of the cases there was no significant supratentorial lesion. In McKenzie’s\textsuperscript{19} a large extradural clot was evacuated from the middle fossa, but the patient died from pressure on the medulla by extension of the clot into the posterior fossa. In Hooper’s case No. 1\textsuperscript{14} an acute subdural hematoma was removed 5 days after the patient had recovered from the first operation.

The principal source of bleeding was considered to be the transverse sinus in 5 cases, the torcular Herophili in 2 cases, the sigmoid sinus in 1, dural vessels in 1, extension of the clot from the middle fossa in 1 case, and, in 3 cases, the source was not discovered.

The mortality in these acute cases was 9 out of 13 (70 per cent). This figure should be higher, because those cases without adequate details have not been included. For instance, Hooper’s review\textsuperscript{15} included 9 cases of extradural hematoma in the posterior fossa, 4 of which were both acute and fatal. Similarly, we do not know if there were acute cases among the fatalities in the series of McKissock \textit{et al.}\textsuperscript{20} and Brodin.\textsuperscript{2}

\textbf{Discussion}

\textit{Incidence.} Extradural hematoma in the posterior fossa has been said to constitute 0.1 to 0.3 per cent of cranio-cerebral injuries, and 7 per cent of extradural hematomas in general.\textsuperscript{2,3,14,21} Meredith\textsuperscript{22} found only 2 surgically verified cases among several thousand patients with cranial injury. Campbell and Cohen\textsuperscript{3} did not find one case among 1,186 consecutive cases. It is difficult, therefore, to assess the true frequency of this lesion which depends so much upon the observer’s awareness. Schneider and Tytus\textsuperscript{23} exemplify this by their comment that in the 23 years up to 1951 not a single case was found at their hospital, whereas, after they had become alerted to the possibility, 3 cases were detected in 18 months.

\textit{Pathogenesis.} In the majority of patients the injury resulted in a fracture crossing the transverse sinus or the torcula, resulting in a tear of the sinus. However, there are cases in which no fracture was found even at autopsy, and other cases where no tear of a sinus could be found. It also remains unexplained how bleeding from a venous sinus in which the pressure is 4 to 6 mm. Hg or less, can produce a large and fatal hematoma within a few hours. In those cases where a tear of the sinus was not found even at autopsy, it may be assumed that the hematoma had been created by bleeding from the diploic vessels or small dural vessels. It is probable that this type of bleeding is important even in cases where the sinus is injured. The

\begin{table}
\centering
\caption{Signs in 13 cases}
\begin{tabular}{|l|c|c|c|c|c|c|c|c|c|c|c|c|}
\hline
Symptoms & Total & 1 & 2 & 3 & 4 & 5 & 6 & 7 & 8 & 9 & 10 & 11 & 12 & 13 \\
\hline
Immediate loss of consciousness and lucid interval & 7 & + & + & + & + & + & + & + \\
No lucid interval & 3 & + & + & + & + & + & + & + \\
Bilateral hyperreflexia and Babinski & 6 & + & + & + & + & + & + & + & + & + & + & + \\
Hypotonicity, Areflexia & 5 & + & + & + & + & + & + & + & + \\
Mydriasis (unilateral) & 4 & + & + & + & + & + & + & + \\
Hemiparesis & 4 & + & + & + & + & + & + & + & + & + & + & + & + \\
Respiratory arrest with cyanosis & 6 & + & + & + & + & + & + & + & + & + & + & + & + \\
Occipital fracture on X-ray & 7 & + & + & + & + & + & + & + & + & + & + & + & + \\
\hline
\end{tabular}
\end{table}
expanding hematoma itself may tear bridging veins and emissary vessels, thus adding to the source of bleeding.\textsuperscript{35}

Lemmen and Schneider\textsuperscript{17} emphasized the importance of otorrhagia resulting in automatic decompression and, therefore, delay in appearance of symptoms until a sizable hematoma had accumulated. The case of Campbell \textit{et al.}\textsuperscript{3} in which coma supervened almost immediately upon cessation of bleeding from the ear, supports this conception. The free bleeding from nose and ear in the case of Beller and Peyser\textsuperscript{1} may thus explain why respiration was not impeded in spite of 48 hours of coma.

\textit{Misleading Signs.} Unilateral signs can be misleading. Unilateral mydriasis is frequent and possibly explained by upward herniation of the cerebellum through the tentorial hiatus deforming and compressing the oculomotor nerve. Associated lesions are another possible factor. Among the 80 cases reviewed by Reigh and O'Connell,\textsuperscript{21} 17 had associated supratentorial hematoma. Posterior fossa hematoma may extend upwards over the occipital lobe or itself be an extension from an occipital hematoma. Numerous examples have been described in which the relatively insignificant supratentorial hematoma was evacuated, overlooking the life-threatening posterior fossa hematoma.

\textit{Diagnosis.} The subacute cases should not usually constitute a problem. They are characterized by a lucid interval with subsequent cerebellar and bulbar signs associated with an occipital fracture and thus constitute an unmistakable clinical syndrome. The acute cases are not so obvious; the bilateral pyramidal signs and decerebrate rigidity often obscure the posterior fossa manifestations. If there are unilateral signs, attention is even more likely to be directed away from the posterior fossa. The clinical signs may be so varied or so few as to provide no help in diagnosis.

Whenever stupor follows upon an occipital injury, even when fracture cannot be demonstrated, and especially when supratentorial burr-holes prove negative, posterior fossa hematoma should be suspected. The suspicion should lead to exploration of the posterior fossa, or at least radiological contrast examinations.

In many cases a diagnostic lumbar puncture was done, but the risk involved is exemplified by the cases of Beller and Peyser,\textsuperscript{1} Fisher \textit{et al.}\textsuperscript{3} and Herren and Zeller;\textsuperscript{19} these patients died within a few minutes of the puncture.

\textit{Radiological Investigation.} The routine inclusion of the half-axial antero-posterior view of the occipital bone (Towne's projection) should ensure detection of the fracture line or suture diastasis involving a vascular channel.

Ventriculography has been used in subacute and chronic cases and this examination has been advised whenever suspicion of posterior fossa hematoma arises.\textsuperscript{20} Although the risks of lumbar puncture, with or without air injection, are well recognized, the hazards of releasing pressure from above are not adequately appreciated. Lindgren's admonition\textsuperscript{18} seems especially relevant: "Expanding lesions in the posterior fossa may give rise to ascending tentorial herniations. A puncture of the lateral ventricle, performed to decrease the intracranial pressure, may result in an increase in the herniation.''

The wisdom of emergency carotid angiography in trauma is becoming more generally accepted.\textsuperscript{16} Discussions of angiography in extradural hematoma,\textsuperscript{4,8,27,28} however, do not record any experience with acute extradural hematomas in the posterior fossa. The information to be derived from carotid angiography in these cases may be indirect or direct. Indirect information, as in our case, consists in the detection of ventricular enlargement, which could be judged acute by a slowing of cerebro-vascular circulation indicative of raised pressure. Direct demonstration of venous sinus displacement from the calvaria was achieved by carotid angiography in Petit-Dutaillis \textit{et al.}'s chronic case.\textsuperscript{29} Both these cases show the importance of serial angiography, with good venous case, in the evaluation of traumatic, no less than of neoplastic cases.

Vertebral angiography is rarely performed in emergencies. The inconsistency of vessel patterns in the posterior fossa makes it difficult to recognize displacements, other than tentorial or foramen magnum herniation. Unlike the tangential view of the cerebral cortex obtained in the antero-posterior carotid angiogram, hematomas over the cerebellum are usually posterior and so a gap
between the cortex and calvarium would not be demonstrated even in the lateral projection, unless it extended over both hemispheres. The transverse sinuses can be more easily visualized by carotid injection.

**Differential diagnosis.** Hematoma or hygroma in any part of the posterior fossa, whether intracerebellar, subdural or extradural, may have the same characteristics. Combined lesions of this type have been described. Recently Ciernbrookiewicz reviewed 20 cases of subdural hematoma in the posterior fossa, 8 of which had an acute course. Clinically, it is impossible to distinguish these from extradural hematoma, and the diagnosis can only be made by operation. Fisher et al. reporting on 98 cases of occipital fracture, described 32 serious complications including 8 with extradural hematomas, 4 subdural hematomas, 7 subdural hygromas and 3 intracerebellar hematomas.

**Management.** Recovery depends on operation, the prognosis being especially poor if breathing has already been arrested. In all cases, ample exposure is required to obtain adequate drainage of the hematoma. Incision of the dura should be made to exclude deeper lesions.

**Summary**

We have reported the successful surgical treatment of an acute extradural hematoma in the posterior fossa, and have reviewed comparable cases reported by others.

We have described the symptomatology and diagnostic measures and emphasized that the rapid development of this hematoma will quickly lead to death unless the diagnosis is suspected early and surgical treatment undertaken promptly.

**References**


