EXTRADURAL CEREBELLAR HEMORRHAGE
REVIEW OF THE SUBJECT AND REPORT OF A CASE*

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(Received for publication August 11, 1948)

EXTRADURAL hemorrhage confined primarily to the posterior fossa of the skull is an unusual lesion of considerable clinical interest. Hemorrhage of this variety arises from a tear in some portion of the lateral sinus precipitated by head injury, but trauma need not be sufficiently severe to cause immediate loss of consciousness, and fracture of the skull may be absent. As blood accumulates in the extradural space overlying the cerebellum, signs of obstruction in cerebrospinal fluid outflow appear and evidences of cerebellar dysfunction become manifest. It is the purpose of this paper to review the clinical features of this syndrome and to present the case of a patient with such a hemorrhage who recovered following operation.

SURVEY OF LITERATURE

McKenzie5 has recorded 20 cases of extradural hemorrhage, one of which was confined to the posterior fossa overlying the right cerebellar hemisphere. In another case the clot covered the left occipital lobe as well as the left cerebellum, and in a third patient clot was found to cover the right cerebellar hemisphere and extend upward over the posterior two-thirds of the right cerebrum. In the first case the patient died unoperated upon; in the second, only the posterior fossa clot was removed and the patient did not survive; in the third, the supratentorial hemorrhage was drained but posterior fossa clot was not detected, and this patient also succumbed.

Coleman and Thomson1 reported 1 case of posterior fossa extradural hemorrhage in which the diagnosis was strongly suspected before operation. Three days after injury, cerebellar exploration was performed disclosing an extradural clot over the right cerebellum reaching to the left of the midline. The clot was evacuated and the patient recovered completely. Coleman1 mentions a case seen by Mayfield in which posterior fossa hemorrhage was thought likely, but the patient died before operation could be performed. Extradural clot over the cerebellum was found at autopsy.

Turnbull8 reviewed the subject of extradural posterior fossa hemorrhage and recorded a case of a woman who had sustained minor head injury with subsequent headache, then 9 months later a second injury, the latter precipitating drowsiness, blurring of vision, and unsteadiness of gait. Suboccipital craniotomy disclosed an extradural clot over the right cerebellar lobe. The clot was removed and the patient recovered except for mild visual impairment.

* Presented before the Neuropsychiatric Section, California Medical Association Annual Session, San Francisco, April 11–14, 1948.
Scoville,\textsuperscript{7} Kessel,\textsuperscript{3} and Watts\textsuperscript{10} each described 1 case of such hemorrhage, and in these 3 instances operation was carried out, all the patients recovering. Watt's case is unusual in that the cerebellar operation was performed immediately after bilateral subtemporal exploration failed to disclose extradural hemorrhage over the cerebrum.

In a survey of 34 extradural hemorrhages Gurdjian and Webster\textsuperscript{2} reported 1 case wherein the patient sustained a perforating wound of the head just above the lateral sinus, with formation of extradural clot over the cerebellum and occipital lobes. Evacuation of the clot resulted in prompt recovery. Munro and Maltby\textsuperscript{6} appraised their 44 cases of extradural hemorrhage and recorded 1 instance of posterior fossa clot discovered at autopsy.

Autopsy material comprising 504 cranial injuries was presented by LeCount and Apfelbach,\textsuperscript{4} who found 104 extradural hemorrhages of sufficient size to produce symptoms. Eight of these were associated with fractures of the posterior fossa, but it is not made clear whether the clot in these instances lay primarily over the cerebellum or over the occipital lobes. Vance\textsuperscript{9} made an extensive appraisal of 512 skull fractures at autopsy and found sizable extradural hemorrhage in 61 cases. In 4 the clot was found over one or both occipital lobes as well as the cerebellum, and bleeding was found to have originated from a lateral sinus tear. In neither of the above autopsy studies is there record of clot confined to the posterior fossa.

**REPORT OF CASE**

J. F., a white girl 5\1/2 years old, was admitted to Children's Hospital April 7, 1947. Twelve days previously she had fallen and struck the back of her head on a hard surface without losing consciousness. In the ensuing 4 days the child became lethargic and vomited frequently without nausea but did not complain of headache. During the following week she grew excitable, cried easily and tore her clothing, and at this stage the parents observed her eyes to be crossed.

**Neurologic Examination.** The patient was irritable, but lucid and fairly cooperative. Suboccipital tenderness was notable, more marked on the right side, and there was mild resistance of the neck to flexion. Bilateral papilledema, large pupils reacting poorly to light and convergence, and bilateral 6th nerve pareses were observed but nystagmus was not present. Although gag reflexes were normal the voice had a nasal quality and the soft palate was depressed, more so on the left than right. All extremities were moderately hypotonic and ataxic, the right arm and leg appearing slightly more unsteady than the left, and as the child walked she deviated fairly consistently to the right, holding her head inclined to this side. The deep reflexes were symmetrical and of small volume throughout; a Babinski sign was present on the left.

X-rays of the skull showed questionable suture separation but no fracture. Spinal puncture was not done. EEG tracings demonstrated phase reversal in the left occiput and were interpreted as suggestive of a deep-seated lesion below the left occipital lobe. On clinical grounds, intracerebellar hemorrhage and posterior fossa subdural hematoma were considered strongly in the diagnostic possibilities, but right cerebellar neoplasm was felt to be more probable than either of these.

April 10, ventriculography was performed through posterior parietal burr holes, 40 cc. of CSF being removed and replaced with an equal amount of air. This study showed moderate internal hydrocephalus, and forward dislocation of the 4th ventricle with narrowing in its anteroposterior dimension (Fig. 1).
EXTRADURAL CEREBELLAR HEMORRHAGE

Fig. 1. Ventriculogram (retouched) after instillation of 40 cc. of air, some of which has escaped into the subarachnoid pathways. Moderate dilatation of the lateral ventricle is apparent, and there is distinct dislocation forward of the cerebral aqueduct and 4th ventricle with anteroposterior narrowing of the latter structure.

Operation. Suboccipital exploration under endotracheal ether anesthesia was carried out immediately, using a curved transverse incision along the superior nuchal line. When the left occipital plate was perforated, dark liquid and clotted blood spurted through the opening under moderate pressure. The bony opening was enlarged and the posterior rim of the foramen magnum removed, disclosing the clot to be extradural, about 6 cm. in diameter and 1.5 cm. in greatest thickness, reaching from the midline to the left sigmoid sinus (Fig. 2, insert). There was no bleeding after evacuation of the clot, which came away readily from the dura. A small opening was made in the dura to exclude subdural clot and the left cerebellar hemisphere was needled once to be certain no intracerebellar hemorrhage was present. The cisterna magna was opened and the cerebellar tonsils were elevated gently from their position of impaction in the foramen magnum. No fracture of the skull was visualized and no other pathology was in evidence. The wound was closed in layers without drainage.

Fig. 2. Diagram illustrating relation of an extradural clot to structures in the posterior fossa. Distortion of the cerebellum, forward dislocation of the brain stem with consequent displacement of 4th ventricle and aqueduct, and compression of the 4th ventricle are indicated. Insert shows approximate size and location of hemorrhage in the case presented.
Course. The patient recovered rapidly. After 5 days the left abducens palsy was less marked, and a week later this defect had almost cleared, though paresis of the right abducens nerve was still prominent. At this time ataxia of the extremities was becoming less pronounced. Patient was discharged April 28, 1947. When seen in the outpatient department 2 months later, she was entirely free of symptoms, the only finding being small excursion nystagmus in left lateral gaze. The optic fundi were negative, the gait was normal and all extremities were used actively, without ataxia. Examination 5 months after operation showed almost imperceptible nystagmus in left lateral gaze, but no other findings. The child ran and turned quickly without impairment of any sort. She has remained well to the date of writing (1 year).

DISCUSSION OF CLINICAL FEATURES

Extradural posterior fossa hemorrhage is undoubtedly an unusual lesion, yet if one includes cases in which the clot reaches upward over one or both occipital lobes of the cerebrum, the incidence is not extraordinarily low. In 3 of McKenzie's 20 cases, hemorrhage extended into the posterior fossa and, in 1 of these, it was confined entirely to the latter area. In Vance's material the incidence of such clots is more than 6 per cent of the total extradural hemorrhages (4 out of 61), and LeCount reported 8 clots involving the posterior fossa as well as the posterior portion of the supratentorial space, an incidence of nearly 8 per cent. At the Los Angeles County Hospital, no example of posterior fossa extradural hemorrhage has been noted in the past 14 years, although approximately 2,000 head injuries are treated each year and an average of 6 cases of epidural hemorrhage over the cerebrum are operated upon per year. No instance of extracerebellar hemorrhage has been recorded by the Los Angeles County Coroner in the past 12 years but precise records are not available and it is possible that posterior fossa clot has been overlooked in certain of these autopsies.

The case here presented is the only one of this variety recorded at Children's Hospital, but extradural hemorrhage in children and infants is notoriously rare and at this hospital only 2 extradural clots of consequential size have been found at surgery or autopsy in the last 15 years.

The clinical picture produced by extradural hemorrhage over the cerebellum has been well described by Coleman and Thomson and the reported cases in which complete data are given show many of the criteria set forth by these writers. Their patient was a 9-year-old child who sustained injury to the back of the head but did not lose consciousness; 24 hours later he complained of headache, vomited twice, and became drowsy, although no significant neurologic findings were evident. Symptoms persisted, but not until almost 3 days after injury were objective neurologic abnormalities detected, consisting of stiffness of the neck, marked hypotonia, and a few hours later, loss of the deep reflexes; neither papilledema nor nystagmus was present. The child lay on his left side most of the time and resented being turned. X-Ray of the skull was normal except for linear fracture of the occipital bone, entering the foramen magnum. Operation on the 3rd day disclosed an extradural hemorrhage covering the entire posterior surface of the
right cerebellum as well as a portion of the left cerebellar hemisphere. The clot was removed and the patient recovered uneventfully.

Two of McKenzie's patients presented a clinical picture similar to that given above and McKenzie makes careful note of the fact that flaccidity of the extremities was a particularly striking finding in his cases.

The case reported by Turnbull is unique in that minor head trauma was followed for 9 months by intermittent headache, a symptom which suggests that posterior fossa clot may have formed after this injury. A second injury (9 months after the first) precipitated rather abrupt symptoms of drowsiness, increasing headache, slight unsteadiness of gait, and blurring of vision in the left eye. Two weeks later the patient had stiffness of the neck, bilateral choked discs, slight incoordination of the right extremities with diminished reflexes in the right lower extremity, and a tendency to stagger to the left. Cerebellar tumor was suspected and ventriculography was done, followed by suboccipital craniotomy 3 weeks after the second injury. Extradural clot was found over the right cerebellar hemisphere, but no comment is made regarding encapsulation or direct evidence that the clot was of several months' duration. Removal of the hemorrhage was carried out and the patient recovered fully except for mild diminution in visual acuity due to secondary optic atrophy.

The following data regarding symptomatology and findings in the presence of posterior fossa extradural hemorrhage are a composite of those brought out by previous authors, principally Coleman and Thomson, and by appraisal of the case herein reported.

The patient is struck on the back of the head and may or may not lose consciousness; headache ensues, often accompanied by vomiting, and a lethargic, irritable state follows. After a period varying from a few hours to many days the lethargy deepens into coma. The patient may prefer to lie on one side (presumably the side of the lesion) and will resume this position if turned. Neurologic findings are often meagre, but neck rigidity, papilledema, nystagmus, ataxia, hypotonia, reduction or loss of deep reflexes, and tonic convulsions have been described at some time during the course of various cases, if the patient's cooperation has allowed adequate examination. Some of these signs are therefore to be expected, particularly the finding of flaccidity of the extremities. Because the hemorrhage is venous, arising from a tear in the lateral sinus, the clot often accumulates slowly and allows the "lucid interval" after injury to extend for several days (or months?), during which time the patient may complain little and exhibit no abnormalities. More commonly, symptoms or signs of rising intracranial pressure have been noted in 24 hours or less, and it is clear that when coma supervenes in the untreated case there is rapid downhill progression with cardiac and respiratory irregularities, and death.

X-Rays of the skull may show fracture line in the occipital bone crossing the lateral sinus. In the presence of supporting clinical signs such a fracture is suggestive evidence in favor of extradural hemorrhage, inasmuch as tear-
ing of the lateral sinus may occur when the skull is fractured. Skull fracture need not be present, however. Spinal puncture has not given significant information and is probably best avoided if the diagnosis is reasonably certain.

Treatment of extradural posterior fossa hemorrhage consists in suboccipital craniotomy with removal of the clot and institution of appropriate repair measures if bleeding from the sinus is encountered. Drainage for 24 hours is advisable unless absolute hemostasis has been obtained. As in the presence of extradural hemorrhage over the cerebrum, operation is indicated early, before the advent of excessive intracranial pressure with accompanying coma and medullary decompensation. In most of the reported cases there was rather slow progression of symptoms enabling the examiners to observe and recheck the odd clinical problem presented and then to proceed as with cerebellar tumor. In the presence of more rapid hemorrhage, fulminating signs of cerebellar and brain stem compression will not permit extended observation, and in these patients prompt diagnosis and operation are imperative. It seems probable that the acute type of posterior fossa hemorrhage is, on occasion, solely responsible for death following head injury, and such deaths might by greater cognizance of this lesion be prevented.

SUMMARY

The subject of traumatic extradural hemorrhage over the cerebellum is reviewed and a case of this syndrome presented in which operation was performed, with recovery of the patient.

Hemorrhage in this location may cause acute compression of the cerebellum and 4th ventricle with abrupt rise in intracranial pressure, tonic convulsions, flaccidity of the extremities and rapidly deepening stupor. However, in several reported cases there were no remarkable symptoms for 24 hours or longer, when headache, lethargy, and “cerebellar” signs appeared and gradually increased in prominence.

As soon as hemorrhage of this variety is suspected, suboccipital craniotomy and evacuation of the clot should be performed.

REFERENCES