CEREBELLAR HEMATOMA CAUSED BY ANGiomATOUS MALFormATIONS

REPORT OF FOUR CASES*

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T he occurrence of nontraumatic hemorrhage into the cerebellum is rare. A review of the literature reveals that the incidence of cerebellar hemorrhage in proportion to bleeding into other areas of the brain varies from 1 to 15 per cent.7,8,11 Conditions that usually are cited as the etiologic background include hypertensive vascular disease, blood dyscrasia, aneurysm, angiomatous malformation, acute infectious processes, diabetes, and syphilis.1,7 In many instances of cerebellar hemorrhage, the cause is not apparent. The majority of reported cases have resulted from hypertensive arteriosclerotic apoplexy while a much smaller number have occurred because of bleeding from an angiomatous malformation. Hyland and Levy3 analyzed 32 cases of cerebellar hemorrhage and, in 4 patients, an angiomatous malformation was shown to be responsible for the hematoma. McKissock et al.5 found a similar lesion to be the cause of cerebellar hemorrhage in 6 of 34 patients in their series. Single cases of cerebellar hematoma secondary to an angiomatous malformation have been reported by Müller,9 Le Beau and Feld,4 and Guillaume et al.2

Because of its rarity, a report of the clinical and pathological features of 4 cases of cerebellar hematoma caused by angiomatous malformations observed over an 11-year period at Duke Hospital seems worthy.

CASE REPORTS

Case 1. 12-year-old girl. Sudden onset of headache and temporary loss of consciousness. Ventriculogram showed lesion in posterior fossa. Suboccipital craniectomy performed. Cerebellar hematoma, secondary to microscopic angiomatous malformation, evacuated.

History. A 12-year-old white girl was admitted on Jan. 1, 1960. She had sudden onset of headache associated with vomiting 24 hours previously. The headache became progressively more severe and 15 minutes after its onset, she lost consciousness. She was unresponsive for several minutes, following which her level of consciousness gradually improved. Lumbar puncture revealed grossly bloody spinal fluid.

A review of her past medical history was unremarkable.

Examination. Blood pressure was 110/50. Temperature was 37.5°C, respiratory rate 16, and pulse rate 88. General physical findings were unremarkable. Positive neurological findings included lethargy, nuchal rigidity, and moderate right hemiparesis. There was no papilledema or intracranial bruit.

Routine laboratory studies, which included estimation of hemoglobin, count of white blood cells, and urinalysis, revealed no abnormalities. Lumbar puncture done shortly after admission yielded grossly bloody spinal fluid with xanthochromic supernatant fluid. The pressure was 380
mm. of cerebrospinal fluid. Roentgenograms of skull and chest showed no abnormalities.

Initially, an intracerebral hematoma on the left side was suspected.

Left carotid arteriography on the day of admission suggested dilated lateral ventricles (anterior cerebral artery was "stretched" on the lateral roentgenogram). Ventriculography was performed through posterior trephines on her 3rd hospital day. There was symmetrical dilatation of the lateral ventricles, as well as dilatation of the 3rd ventricle. The aqueduct of Sylvius was filled in its upper portion only and was shifted forward and to the left.

Operation. Suboccipital craniectomy was performed following ventriculography and a hematoma, measuring 4×3 cm. in diameter, was evacuated from the right cerebellar hemisphere. There appeared to be abnormal vessels in one small area in the inferior wall of the cavity of the hematoma.

Microscopic sections of this area demonstrated an angiomatous malformation which was confined to the sulci (Fig. 1). The lesion was composed of numerous variously sized vessels of venous structure. There was hyalinization of the walls of some of the vessels. The vascular anomaly was classified as a venous angioma.

Postoperative course was uneventful. Three months following operation she was asymptomatic except for hyperactive deep tendon responses on the right side.

Case 2. 15-year-old boy. Three episodes of subarachnoid hemorrhage over 2½-year period. Collapsed while hunting. Dead on arrival at hospital. Autopsy revealed cerebellar hematoma caused by angiomatous malformation.

History. A 15-year-old boy was pronounced dead on arrival at Duke Hospital on Nov. 24, 1949.

He had been in good health until April 18, 1947, when there was sudden onset of right frontal and occipital headache. Shortly after onset, according to his mother, he lost consciousness for 2 days. On regaining consciousness, he had nausea and vomiting as well as a stiff neck. He was treated with rest in bed and recovered without residual.

He remained asymptomatic until April 2, 1948. At this time, he was heard to cry out during sleep. Attempts to arouse him were unsuccessful. He was unconscious for 2 days.

Physical and neurological findings, at another hospital, were normal except for nuchal rigidity. Lumbar puncture revealed grossly bloody spinal fluid. He remained in the hospital for 12 days during which time his symptoms, which consisted of headache, stiff neck, and diplopia, gradually subsided.

He was first admitted to Duke Hospital on May 13, 1948, for investigation of subarachnoid hemorrhage. At this time, he was asymptomatic and findings on examination were unremarkable. Right carotid arteriography showed no abnormalities.

He was next admitted to Duke Hospital on Oct. 2, 1949. Two hours prior to admission, he experienced sudden onset of severe occipital head-
ache following mild physical exertion. He became nauseated and vomited. Although he became drowsy, there was no loss of consciousness.

His blood pressure was 115/70. Temperature was 38°C, pulse rate 96, and respiratory rate was 16. He was lethargic but oriented as to time, place and person. General physical findings were normal. Positive neurological findings were nuchal rigidity and questionable nystagmus on lateral gaze in either direction.

Routine laboratory determinations, which included estimation of hemoglobin, count of white blood cells, and urinalysis, were within normal limits. Lumbar puncture revealed grossly bloody spinal fluid under a pressure of 300 mm. cerebrospinal fluid.

There was gradual improvement in his complaints. At the time of discharge on Oct. 20, 1949 he was asymptomatic.

He went hunting alone on Nov. 24, 1949. When he did not return home as expected, his family began searching and found him in a moribund condition. He was pronounced dead on arrival at Duke Hospital.

Autopsy. Grossly, there was a massive hemorrhage into the left cerebellar hemisphere which arose from a vascular malformation. The lesion consisted of a worm-like mass of dilated vessels with thin walls. It was situated on the surface of the left cerebellar hemisphere just above the lateral foramen of the 4th ventricle. At one point, the vascular malformation communicated with a tortuous and dilated superior cerebellar vein which drained into the straight sinus. At another point, the vessels were in intimate relationship to the anterior inferior cerebellar artery and could be injected through this vessel.

Microscopic sections revealed that the vascular lesion was situated in the parenchyma (Fig. 2) as well as on the surface of the cerebellum. The vessels, predominantly venous in structure, varied considerably in size and in thickness of the walls. The lesion was classified as a venous angioma.

Case 3. 34-year-old woman. Recurrent headache, nausea and vomiting over 9-month period. Paralysis of left 11th and 12th cranial nerves and hypalgesia over 2nd and 3rd dermatomes on left side. Hematoma in left cerebellar hemisphere, extending down along medulla. Microscopic sections revealed angiomatous malformation.

History. A 34-year-old white woman was admitted on March 13, 1954, with complaints of intermittent headache, nausea, and vomiting.

She was well until June 1953, when she had onset of left-sided headache which was constant, but varied in intensity. The headache gradually subsided over a period of 5 weeks. A similar episode occurred on Oct. 19, 1953 and lasted 1 month. At this time, she noticed narrowing of the left palpebral fissure. She had a recurrence of headache in January 1954. This persisted and, 2 weeks prior to admission, she noted an increase in the intensity of the headache and experienced nausea and vomiting.

Examination. Blood pressure was 114/74. Pulse
rate was 94, respiratory rate 16, and temperature was 37.4°C. She appeared chronically ill and complained frequently of headache. Positive neurological findings were paralysis of the left 11th and 12th cranial nerves and hypalgesia over dermatomes C2 and C3 on the left side.

Routine laboratory determinations, which included estimation of hemoglobin, count of white blood cells, and urinalysis, were within normal limits. Roentgenograms of skull and cervical spine were unremarkable. Lumbar puncture yielded spinal fluid which contained 34,000 red blood cells and 340 white blood cells per c.mm. The majority of the white blood cells were polymorphonuclear leucocytes. The supernatant fluid was xanthochromic and the pressure was 260 mm. cerebrospinal fluid.

It was thought that the patient had a lesion, possibly a tumor, lying alongside the medulla on the left side, with extension into the upper spinal canal.

Operation. An upper cervical laminectomy and suboccipital craniectomy were performed on March 22, 1954. A reddish-purple mass was found in the lower portion of the left cerebellar hemisphere. The lesion extended down along the left side of the medulla and consisted of a currant-jelly clot surrounded by a thin fibrous capsule. It was resected together with a small portion of the adjacent cerebellar tissue.

Microscopic sections revealed an arteriovenous malformation situated within the sulci. The majority of vessels were arterial in structure as shown by the presence of internal elastic lamellae (Fig. 3). There was considerable connective-tissue reaction throughout the sulci as well as gliosis of the adjacent cerebellar folia in some regions. Evidence of previous bleeding was shown by the presence of granules of hemosiderin in the macrophages.

Postoperative course was uneventful and she was discharged on the 26th hospital day.


History. A 37-year-old white male had a right upper molar tooth extracted 10 days prior to admission, on March 21, 1949. Twenty-four hours later he suffered dizziness, nausea, staggering gait, and diplopia. These complaints persisted and, 3 days after admission, he awoke with a severe occipital headache.

Examination. Temperature was 37°C., pulse rate 80, and respiratory rate was 20. Blood pressure was 124/76. The patient appeared acutely ill. Positive neurological findings were lethargy, nuchal rigidity, bilateral dysmetria, worse on the right side, and ataxic gait. There was no papiledema.

Routine laboratory determinations, which included estimations of hemoglobin and hematocrit,

![Fig. 3. Case 3. Arteriovenous malformation situated in sulci. The two larger vessels are arterial in structure as shown by the internal elastic lamellae. Verhoeff-van Gieson, X82.](image-url)
counts of white and red blood cells, and urinalysis, were within normal limits. Roentgenograms of skull and chest showed no abnormalities.

Lumbar puncture, shortly after admission, showed an opening pressure of 270 mm. cerebrospinal fluid. There were 2,000 white blood cells per c.mm. (predominantly polymorphonuclear leukocytes) and 2,500 red blood cells per c.mm. Bacteriologic cultures of the fluid yielded no growth.

The initial clinical impression was cerebellar abscess with meningitis.

Course. Lumbar puncture was done daily and 10,000 units of penicillin were instilled on each occasion. Over the course of 8 days, there was improvement in his condition and the cellular pattern of the spinal fluid returned to normal.

Ventriculography was performed through posterior trephines on March 29. There was symmetric dilatation of both lateral ventricles and the 3rd ventricle. The aqueduct and upper portion of the 4th ventricle were dilated, kinked and displaced forward.

Operation. Suboccipital craniectomy was carried out following ventriculography and a cerebellar hematoma was evacuated. The hematoma was located in the medial portion of the left cerebellar hemisphere and also in the vermis. A portion of the wall of the hematoma as well as the clots were submitted for histologic study.

Microscopic sections of one of the clots adjacent to the wall of the hematoma revealed a lesion consisting of numerous thin-walled vessels of varying size (Fig. 4). Granules of hemosiderin were present in macrophages about the vessels. The lesion was classified as a telangiectasis.

Postoperative course was unremarkable. Initially, the patient was restless and complained of headache. He was discharged on April 12 at which time he showed considerable improvement in the degree of dysmetria and ataxic gait as compared with his preoperative status.

COMMENT

The pathologic type of angiomatous malformations responsible for the cerebellar hematoma in the present series consisted of a telangiectasis in 1 patient; a venous angioma in 2 patients; and an arteriovenous angioma in 1. The vascular lesion was obvious in the gross specimen in 1 patient only (Case 2) whereas in the remaining 3 patients histologic verification was necessary. The angiomatous malformations were mainly venous in type in all 4 cases described by Hyland and Levy. The vascular lesions were large and consisted principally of thin-walled veins of varying sizes in 3 of their cases; while in 1 the angioma was minute and required histologic demonstration. The cerebellar hematoma in Müller's case was caused by rupture of a small racemose arteriovenous malformation.
There was calcification in the arteriovenous malformation reported by Le Beau and Feld. McKissock et al. did not describe the pathologic types of angiomatos malformations in their cases.

The instances of cerebellar hemorrhage secondary to angiomatous malformations occur in the younger individuals, whereas those caused by hypertensive vascular disease generally are seen in the older age group. The age range of our 4 patients was 12 to 37 years, which is similar to that noted by others. The age range in the 4 cases of cerebellar hematoma caused by angiomatous malformation in Hyland and Levy's series was 12 to 34 years and in McKissock and associates' cases, 15 to 40 years.

A localizing diagnosis is not always possible in patients with hemorrhage into the cerebellum. McKissock et al. found that the symptoms and signs suggested a cerebellar site for the lesion in only one-fourth of the cases. Although this is true in cases of patients who were seen shortly after onset, the present study suggests that in those who survive for a longer period, a localizing diagnosis may be suspected in a higher per cent of cases. A lesion in the posterior fossa was suspected on the basis of history and examination in 3 of the 4 cases in the present series. The diagnosis was confirmed by ventriculography in 2 patients (Cases 1 and 4), whereas in a third patient (Case 3), operation was performed on the basis of the signs and symptoms. McKissock et al. performed vertebral angiography in 2 patients with cerebellar hematoma caused by angiomatous malformations, and in both instances the study was interpreted as negative. The size of the angiomatous malformations was not mentioned, but presumably both were small. Although this number of cases is too small to have statistical value, it does indicate that the presence of a cerebellar hematoma cannot be excluded by vertebral arteriography.

The small size of many of the angiomatous malformations, as well as the fact that the resultant hemorrhage may destroy the smaller angioma, probably account for the failure to demonstrate an etiologic cause in many cases of cerebellar hematoma. These points have been stressed in instances of intracerebral hematoma and hematomyelia. It is not possible to exclude a vascular malformation in those cases studied solely under operative conditions. A careful microscopic study of all clots evacuated and any suspicious areas in the wall of the cavities of the hematomas removed in instances of unexplained hemorrhage into the cerebellum, should be carried out.

SUMMARY

The clinical and pathological features of 4 cases of cerebellar hematoma caused by an angiomatous malformation are presented.

The necessity for careful microscopic study of all clots as well as suspicious areas in the walls of the cavities of hematomas in instances of unexplained hemorrhage into the cerebellum, especially in younger individuals without vascular disease, is emphasized.

REFERENCES