CEREBELLAR HEMATOMA CAUSED BY ANGIOMATOUS MALFORMATIONS
REPORT OF FOUR CASES*

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The 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 angiomatos malformation. Hyland and Levy3 analyzed 32 cases of cerebellar hemorrhage and, in 4 patients, an angiomatos 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 angiomatos 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 angiomatos 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 angiomatos 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

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† Dr. Tindall is a special clinical trainee (BT-738) of the National Institute of Neurological Diseases and Blindness.
‡ An important reference on the subject of cerebellar hematoma was not discovered until after this article had gone to press: Crawford, J. V., and Russell, D. S. Cryptic arteriovenous and venous hamartomas of the brain. J. Neurol. Neurosurg. Psychiat., 1956, n.s. 19: 1-11. The authors described 6 cases of bleeding from vascular anomalies of the cerebellum. Three patients were relieved by operative treatment; while the other 3 expired rapidly. Microscopically the lesion was composed of both abnormal arteries and veins in 3 examples and of veins alone in 1. In the remaining 2 cases the pathological type of vascular lesion was uncertain.
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 and the neurological findings were negative 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. 12, 1949. Two hours prior to admission, he experienced sudden onset of severe occipital head-

Fig. 1. Case 1. Numerous variously sized vessels of venous structure situated in sulci. Hematoxylin-eosin, ×137.