Intracranial Aneurysm as a Cause of Subdural Hematoma of the Posterior Fossa

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The occurrence of subdural hematoma in the posterior fossa is uncommon. Such lesions might be divided into two large groups: traumatic and spontaneous. The former is believed to be largely the result of rupture of the dural sinus. The first report of a traumatic subdural hematoma in the posterior fossa was that by Picken in 1928. In 1940, Coblentz reported the first successfully treated case of a hematoma of the posterior fossa in a newborn. In 1961, Estridge and Smith reported the case of a 3-year-old child in whom a post-traumatic subdural hematoma of the posterior fossa was treated surgically and successfully. In their review of the literature 15 cases were presented, 5 diagnosed at autopsy and 4 associated with hematoma of the cerebral hemispheres. In only 2 was there no definite history of occipital trauma. The source of bleeding was attributed to rupture of the lateral sinuses.

Spontaneous subdural hematomas of the posterior fossa have been reported less frequently. In 1952, Clarke and Walton reported 11 cases of subdural hematoma complicating intracranial aneurysms and angiomata. In 1 case, rupture of the posterior inferior cerebellar artery resulted in a subdural hematoma of the posterior fossa combined with a subdural extension of hemorrhage over the cerebral hemispheres. From a study of the literature it is apparent that this is the only pre-existing report of an aneurysm of the posterior fossa responsible for a subdural hematoma. The case report which follows to our knowledge represents the second such case recorded in the medical literature.

Case Report

A 34-year-old housewife and waitress entered Baltimore City Hospitals in January 1962 with severe headache. Two weeks before, following a severe attack of coughing, she suffered an occipital headache which persisted until the day of admission. On that day she experienced a "heavy" feeling in her head, vomiting, weakness, and momentary loss of vision with intense persistent headache radiating into both shoulders.

In 1957 she received electroshock therapy for a "manic depressive" reaction. There had been no known recurrence of this condition.

Examination. The patient was a stocky female with nuchal rigidity. Blood pressure was 170/110. Pulse rate was 90 per min. The right pupil was dilated and did not react to light. Subhyaloid and perivascular hemorrhages were noted in the right optic fundus. There was a mild facial weakness and sluggish deep tendon reflexes were present on the right. Plantar responses were extensor. Urinalysis, hemogram and roentgenograms of the skull were normal.

Course. Her clinical course was characterized by severe headache and mild right hemiparesis. She showed improvement initially and by the 3rd day her pupillary signs had disappeared. Bilateral carotid arteriograms were normal. Bilateral subconjunctival hemorrhages developed subsequently. On the 10th day she suddenly became unresponsive, irregular respiration developed and she was apparently unable to swallow. Her head was turned to the left and the pupils were fixed. Consciousness returned on tracheal suction, after which she complained bitterly of headache and pain in the neck. The right pupil became dilated and nuchal rigidity increased progressively. The blood pressure was now 230/120. There were no new neurological signs. Two days later saphenous cerebrospinal fluid was withdrawn; it contained many red blood cells, and levels of protein and sugar were normal. The spinal-fluid pressure was 390 mm. of cerebrospinal fluid.

On the following day hyperthermia with temperatures up to 106°F developed, coincident with hypotension and tachycardia. She was placed in a cooling blanket in order to reduce her fever, but she remained confused and drowsy. Because of her critical condition, vertebral arteriography was not attempted although the clinical diagnosis of an aneurysm of the posterior fossa was considered seriously.

On the 32nd hospital day she became markedly hypotensive, tachypneic, apneic, and died.

Autopsy Findings. The major findings were confined to the central nervous system. On removing the brain, the prosector noted 30 ml. and 50 ml. of fresh blood in the middle and posterior cranial fossae respectively. These subdural collections of blood were observed before any major veins or venous sinuses were entered. The brain weighed 1290 gm. A large ruptured aneurysm of the right vertebral artery was located 1½ cm. proximal to the origin of the basilar artery at the take-off of the posterior inferior cerebellar artery (Fig. 1). It measured 1×0.6×0.5 cm.; the site of rupture was surrounded by laminated blood clot. Fresh subarachnoidal blood in the region of the pons and brain stem was minimal although gross yellow staining of the arachnoid overlying these areas revealed iron and hematoidin pigment, indicating previous episodes of subarachnoidal hemorrhage (Fig. 2). The arachnoid was adherent to the rim of the aneurysmal defect so that the rupture was in direct communication with the subdural space (Figs. 3 and 4). Histologically the aneurysm was of the so-called "congenital" type (Fig. 5). The expanded aneurysmal wall was composed of fibrous tissue containing occasional fragments of elastica, and a recent site of rupture was identified (Fig. 6). The lateral and 3rd ventricles were slightly dilated and the central aqueduct was patent.

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Fig. 1. Inferior view of brain and cerebellum showing ruptured "congenital" aneurysm of right vertebral artery. Note the minimal amount of recent subarachnoidal bleeding around the basal meninges.

No staining by blood or pigment was evident in the ventricular system.

These findings suggested that a chronic and intermittent subarachnoidal leak had occurred from the aneurysm of the vertebral artery. As a consequence of this the arachnoid had become adherent to the aneurysmal wall so that at the time of final massive rupture most of the blood entered the subdural instead of the subarachnoidal space, resulting in death.

Discussion

This case represents a congenital aneurysm of the right vertebral artery with proven history of past leakage, a recent subarachnoidal hemorrhage, and a subdural hemorrhage of the posterior fossa which resulted in the patient's sudden demise. Examination of the brain showed very little terminal hemorrhage in the subarachnoidal space and none into the ventricles.

Spontaneous hemorrhage of the posterior fossa represents about 10 per cent of all spontaneous intracranial hemorrhages. Most of these hemorrhages occur within the cerebellum in association with hypertension with or without associated vascular malformation, neoplasms, and blood dyscrasias. Subdural hematoma in the posterior fossa probably occurs more frequently than the literature would suggest. Most of the cases on record have occurred after trauma, with an occa-

Fig. 2. Photomicrograph of subarachnoidal space at base of the brain. Note the moderate amount of old blood and blood pigment attesting to previous episodes of subarachnoidal hemorrhage. Note too the singular lack of recent massive subarachnoidal hemorrhage.

Fig. 3. Diagrammatic cross section of head showing falx cerebri in diagonal pattern, cerebrospinal fluid in stippled pattern and location of subdural hematoma in posterior fossa in solid black area.
sional case following an intracerebellar hematoma, and in at least 1 case the hemorrhage was the result of rupture of an aneurysm.

The mechanism of subdural hematoma with an aneurysm of the cerebral artery probably involves basal or parietal arachnoid adhering to the aneurysm with blood from the rupture entering the subdural space directly rather than into nervous tissue or into the subarachnoidal space. The importance of recognizing the existence of the basilar subdural space of the anterior, middle and posterior fossae, and the possibility of death being related to a massive subdural hematoma following aneurysmal rupture is obvious and needs continuing emphasis. The complication of subdural rupture of an aneurysm is infrequent. Indeed, there is no reliable syndrome recorded as yet which would allow the clinician to make the diagnosis. A high index of suspicion is therefore necessary.

The paucity of information on subdural hematoma of the posterior fossa is related possibly to a failure to recognize the condition. The hematoma is easily overlooked at the time of autopsy, for indeed the pathologist has only a fleeting opportunity to recognize it. This occurs at the moment the cerebellar hemispheres are retracted and before any of the dural sinuses are incised. If blood is observed between the arachnoid and dura mater at this time, it is almost certainly ante-mortem. However, as soon as veins or sinuses have been entered blood will flow into the posterior fossa and differentiation from a fresh hemorrhage is impossible. It is obvious that professional inspection of the posterior fossa is necessary and that this important duty should not be entrusted to an untrained assistant. It is obvious that such a diagnosis cannot be made on the brain alone. Chronic subdural hematomas of the posterior fossa probably do not exist as the occurrence of the lesion is usually associated with sudden death.

Summary

A case of spontaneous subdural hematoma of the posterior fossa resulting from the rupture of a congenital aneurysm of the right vertebral artery is reported and the literature is reviewed. The majority of hematomas of the posterior fossa are probably traumatic in origin. Only a few reported cases are unassociated with trauma. The present case is only the second reported in association with rupture of a congenital aneurysm. The probable reason for infrequent reports in the literature is discussed. The importance of the pathologist's careful inspection of the posterior fossa at the time the brain is removed is emphasized.

Fig. 4. Diagrammatic representation of how direct subdural hemorrhage from a ruptured aneurysm occurs when the margins of the aneurysm are adherent to the pia arachnoid from previous minor "leaks."

Fig. 5. Photomicrograph of ostium of "congenital" aneurysm of right vertebral artery showing sudden loss of architecture of normal vascular wall at site of aneurysmal dilatation.
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Fig. 6. Photomicrograph of aneurysmal wall with preserved internal elastic lamina of the wall of the intact vessel and dilated fibrous wall of aneurysm. Organized blood clot forms a portion of the aneurysmal wall in the upper right of picture near the site of final complete rupture.

References


