Hemorrhage in bilateral choroid plexus hemangiomas demonstrated by computed tomography

Case report

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The authors present a case in which bilateral posttraumatic hemorrhages in choroid plexus hemangiomas were demonstrated by computed tomography and histopathological study.

KEY WORDS • choroid plexus hemangioma • vascular malformation • computed tomography • choroid plexus hematoma • intraventricular hemorrhage

HEMANGIOMAS or vascular malformations of the choroid plexus are uncommon causes of intraventricular hemorrhage. They have rarely resulted in large hematomas of the choroid plexus. In this report, we present an elderly patient with bilateral hemangiomas of the choroid plexus, intraventricular hemorrhage, and bilateral choroid plexus hematomas.

Case Report

This 74-year-old woman was involved in an automobile accident; she sustained rib fractures and a laceration of the forehead. She was alert after the accident, but did not remember it. On the third day after the accident her mental status deteriorated and she developed a waxing-and-waning level of consciousness. At that time she was transferred to the hospital of the University of Pennsylvania.

Examination. On admission her pulse rate was 88, blood pressure 160/80, and temperature 100°F; she had multiple craniofacial ecchymoses, and rib fractures. She was lethargic and disoriented as to time and space. The cranial nerves were normal. Motor examination showed decreased arm strength bilaterally, more severe on the right. There were appropriate responses to pain, and her reflexes were normal except for bilateral extensor plantar responses. Routine hematological, urine, and blood biochemistry tests, skull and spine x-ray films were normal. A right carotid arteriogram revealed grossly dilated lateral ventricles without a shift of midline structures. A computed axial tomogram (CAT) showed masses
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of the density of blood in the region of the body and occipital horns of the lateral ventricles. These masses extended to the level of the foramina of Monro (Fig. 1 left).

Hospital Course. A Scott cannula was placed into the anterior horn of the right lateral ventricle to measure intracranial pressure and to remove free blood. Intracranial pressure was found to be 2 to 6 mm Hg and bloody fluid was drained from the ventricles. Ventricular fluid gradually cleared and the patient’s mental status improved. Left carotid and vertebral arteriograms showed no evidence of aneurysm, vascular malformation, or other source of hemorrhage. A Conray* ventriculogram revealed no obvious obstruction to the outflow of cerebrospinal fluid (CSF) from the ventricular system. An air ventriculogram demonstrated a large lobulated mass in the floor of the lateral ventricles. This was consistent with an enlarged choroid plexus. The ventricle was drained through the Scott cannula for 4 days; during this period the pressure was 2 to 6 mm Hg. The cannula was removed 4 days later. Subsequently the patient’s level of consciousness fluctuated and she became progressively more disoriented and febrile. Eleven days later a ventriculoperitoneal shunt was placed in an attempt to relieve the hydrocephalus. The patient continued to have fever periodically and became unresponsive about 5 weeks after admission. At this time a repeat CAT scan showed a change in the hematomas consistent with partial resolution. They had undergone a marked decrease in density and had also become less homogeneous. Again the anatomic sites of density were consistent with the choroid plexuses but occupied a much larger portion of the cavities of the lateral ventricles (Fig. 1 right) than the normal choroid plexus. The patient died suddenly 6 weeks after admission.

Postmortem Examination. Autopsy revealed the immediate cause of death to be multiple pulmonary emboli originating from blood clots in the right atrium near the infected tip of a catheter used for monitoring the central venous pressure. The heart was otherwise normal. The brain showed moderate dilation of the lateral and third ventricles. The antra of the lateral ventricles, and the occipital and temporal horns were filled with hematomas (Fig. 2). The hematomas extended anteriorly 0.5 to 0.8 cm posterior to

*Conray obtained from Mallinckrodt Pharmaceuticals, St. Louis, Missouri.
FIG. 2. Cross sections from cerebral hemispheres. Lateral ventricles contain localized hematomas.

FIG. 3. Photomicrograph showing a part of hematoma. In addition to hemorrhages (right and bottom), it contains abnormal aggregates of small vessels in the choroid plexus. Choroid plexus epithelium (at top) surrounds the hematoma. H & E, ×100.

FIG. 4. Computed tomogram in a patient with massive intraventricular hemorrhage reveals markedly dilated, blood-filled, right lateral ventricle with the choroid plexus presenting as a filling defect. Left lateral ventricle is compressed and not seen.

the foramina of Monro. The largest cross-sectional dimension of each hematoma was $3 \times 4$ cm at the level of the antra of the lateral ventricles. On microscopic examination the hematomas proved to be localized mainly in the glomus of the choroid plexuses, resulting in marked enlargement of the latter. An 0.2 to 0.4 cm thick layer of organizing blood clot surrounded the hemorrhagic choroid plexuses. Both plexuses had a similar appearance. They were mainly replaced by extravasated, lysed red blood cells, and hemosiderin-laden macrophages. In most areas there was a dense connective tissue reaction around the hematomas. These findings indicated repeated hemorrhages in the choroid plexuses with estimated ages varying from a few days to several weeks. The choroid plexuses also showed numerous blood vessels of various sizes occurring in clusters (Fig. 3). Many of the large vessels had thin walls and some had endothelium only. Some vessels were identified as arteries. Occasional vessels were thrombosed and had thick fibrotic walls.
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On the basis of these findings, the diagnosis of bilateral choroid plexus hemangiomas with repeated hemorrhages was made. There was also evidence of intraventricular and subarachnoid hemorrhages. No other lesions were found in the brain or spinal cord.

Discussion

Hemangiomas of the choroid plexus are uncommon. Only 28 cases including the present one have been reported. The ages of patients varied from 2 days to 74 years; our patient is the oldest reported. Seventeen cases (61%) occurred in the first two decades of life and females had a slightly higher incidence (59%) than males. In all cases except two, choroid plexus hemangiomas produced symptoms and were associated with intraventricular hemorrhage. In two cases, choroid plexus hemangiomas were asymptomatic and were discovered as an incidental finding at autopsy. Choroid plexus hemangiomas were bilateral in four instances (14%) including our case. Two of these had multiple associated malformations in other organs, one had a hemangioma of the elbow, and the fourth, our case, did not have any associated vascular or other malformations.

In all reported cases of choroid plexus hemangiomas that bled, intraventricular hemorrhage had occurred spontaneously. However, in our case the trauma of the automobile accident might have played a role in the rupture of vessels in the hemangioma resulting in intraventricular and intrachoroid plexus hemorrhages. Histopathologically, the age of the organizing hematoma is consistent with the clinical history (death occurred 6 weeks after trauma). However, the possibility that the hemorrhage occurred prior to trauma cannot be excluded since the presence of unusually thick fibrous connective tissue within the choroid plexuses is consistent with hemorrhages occurring prior to the accident. The fluctuation in the level of consciousness of our patient might have been due to repeated hemorrhages in the plexuses and lateral ventricles, intermittent obstruction of the foramina of Monro due to enlarged plexus, or intraventricular pressure waves independent of ventricular obstruction. In four cases, including ours, of choroid plexus hemangiomas with intraventricular hemorrhage, there were also associated large choroid plexus hematomas.

The diagnosis of bilateral choroid plexus hematomas in our case was strongly suggested by the CAT scans. Computed axial tomography is a sensitive method of detecting minimal changes in brain density and has proved to be highly accurate in detecting the presence of extravascular blood. The x-ray photon absorption capacity of extravascular blood is greater than that of the normal brain, which in turn is greater than that of CSF. Fresh blood within the ventricular system tends to be dependent because it is more dense than CSF; blood therefore may form a CSF-blood interface within the ventricle.

The choroid plexus normally presents as a soft-tissue density within the less dense CSF-filled ventricle. In the case of a large intraventricular hemorrhage, the choroid plexus presents as a filling defect within the denser blood. In that case there was no differentiation between the choroid plexus and the blood. This fact suggested that the choroid plexus was the site of hematoma formation, although a normal choroid plexus might be hidden within a large hematoma.

A second CAT scan performed about a month later revealed shrinkage of the hematomas. These also appeared less dense, indicating that they had undergone partial resorption.

These findings point out the accuracy of CAT in the diagnosis of intraventricular hemorrhage and indicate the usefulness of this technique in studying the resolution of these lesions.

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