

# Spontaneous Resolution of an Intraventricular Hematoma

## Report of a Case with Recovery

ROBERT G. OJEMANN, M.D., AND PAUL F. J. NEW, M.D.

*Neurosurgical and Radiological Services, Massachusetts General Hospital, Boston, Massachusetts*

The ominous prognosis following hemorrhage into the ventricles of the brain has been well documented<sup>5-7,9,12,13</sup> since the detailed review of this entity by Sanders in 1881.<sup>11</sup> Most intraventricular hemorrhages are reported to be characterized by sudden onset of coma, evidence of severe brainstem function and death.<sup>6,7,12</sup> This type of intracranial hemorrhage also has been shown to be a common cause of death in premature infants.<sup>9</sup> In contrast to the more common catastrophic picture, intraventricular hematomas may present a less acute course and at times simulate intraventricular tumors.<sup>1,7,9</sup> In the following case report intraventricular hemorrhage resulted from an arteriovenous malformation deep in the left occipital lobe.

### Case Report

A 30-year-old law student was admitted following the sudden onset of coma. At birth there was some difficulty with respirations and on the 3rd day of life he became cyanotic for an unknown period of time. Cerebral palsy had been diagnosed early in life. Development of motor skills was retarded. His gait was somewhat clumsy and there was a tremor of the upper extremities with activity. Speech was slightly thick but general intelligence and mental functions were good. Four years before admission he had psychotic symptoms characterized by violence and suspicion which were treated with electroshock therapy.

On the day of admission, after climbing a flight of stairs, he had the sudden onset of headache beginning in the suboccipital region and rapidly radiating to the frontal area. Within 10 min. he was comatose, and began to have difficulty with respirations and then seizures characterized by tonic movements of all extremities.

*Examination.* Initially he was completely unresponsive. There were repeated episodes characterized by extension of both lower extremities, flexion of both upper extremities, arching of his neck and back, chewing movements, increased respirations and sweating. Pupils were equal with fair reaction to light. Numerous hemorrhages were noted in both fundi. All deep tendon reflexes were hyperactive. There was bilateral clonus and both plantar responses were extensor. Lumbar puncture showed an initial pressure of 340 mm. with grossly bloody spinal fluid.

*Course.* Dilantin, phenobarbital, and paraldehyde were required to control his seizures. Vital signs remained stable. There was no evidence of localized weakness. Bilateral carotid angiography demonstrated very slight displacement of the pericallosal arteries to the

right of the midline and findings consistent with minor lateral ventricular enlargement. Four days following onset, evidence of improvement in consciousness was noted.

Two weeks after admission lumbar puncture showed initial pressure of 380 mm. with slightly pink, xanthochromic fluid containing 3,260 red cells and 63 white cells (47 lymphocytes and 16 polymorphonuclear cells). Three weeks following onset he was saying an occasional word. Lumbar-puncture pressure was 160 and fluid was mildly xanthochromic, with 200 red cells and 2 white cells. Four weeks after the hemorrhage he was speaking short phrases and following some commands.

Vertebral angiography by direct injection of the left vertebral artery (Fig. 1) demonstrated no abnormality of the vertebrobasilar system up to the level of the posterior cerebral arteries. The right posterior cerebral artery and its branches appeared normal. The left posterior cerebral trunk appeared normal, but there was evidence of minor medial displacement of the medial and lateral branches. A small arteriovenous malformation was demonstrated, lying 1.5 cm. to the left of the midline and approximately the same distance superior to the medial divisions of posterior cerebral arteries. The malformation was supplied by small branches arising from the medial division of the left posterior cerebral artery and drained by a small vein apparently into the vein of Galen. The central portion of the malformation measured approximately 6 mm. in diameter. There was less than normal capillary filling in the area lateral to the malformation. This, together with the medial displacement of the branches of the posterior cerebral artery, was interpreted as indicating an intracerebral hematoma caused by bleeding from the malformation.

Seven weeks after admission, a pneumoencephalogram (Fig. 2a and b) demonstrated moderately severe generalized enlargement of the lateral ventricles, the left being slightly larger than the right. The right foramen of Monro appeared normally patent, while the left appeared partly obstructed. The left lateral ventricle contained a large irregular mass extending from the frontal horn to the occipital horn. The mass showed numerous lobulated surfaces and a number of band-shaped extensions from the main bulk of the mass to the walls of the ventricle. The mass was most bulky in the medial portion of the lateral ventricle, with a base lying against the septum lucidum. There was a shallow but nodular filling defect projecting from the area of the septum lucidum into the posterior portion of the body of the right lateral ventricle, and the upper portion of the area of the septum lucidum was thickened considerably. The 3rd ventricle was enlarged moderately. The aqueduct and 4th ventricle were within normal limits of size and position. No definite abnormality of the subarachnoid cisterns was recognized. The findings were interpreted as indicating a large hematoma in the left lateral ventricle, with

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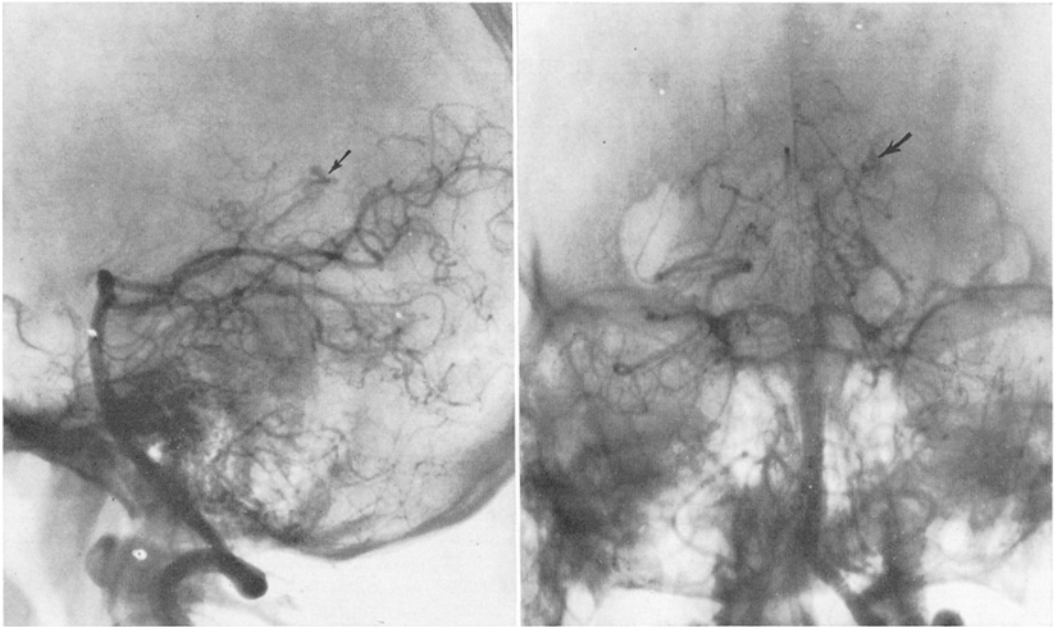


FIG. 1. Left vertebral arteriogram, first arterial phase. (*Left*) Lateral projection (*Right*) Anterior-posterior projection. The arteriovenous malformation is indicated by the arrow.

characteristic irregularity of the clot and with evidence of adherence of the clot to the ventricular walls. Contraction of the clot was indicated by the "festooned" configuration. Evidently the hematoma had ruptured through the septum lucidum to produce a minor filling defect in the medial portion of the right lateral ventricle. Spinal fluid at this time was still moderately xanthochromic. Eight weeks after admission he was discharged, having almost recovered to his prehemorrhage status.

Readmission 3 months following onset of symptoms revealed only those neurologic deficits that had been present prior to the hemorrhage. Lumbar puncture showed a pressure of 150 mm.; the fluid contained no red cells, 5 lymphocytes and 143 mg. per cent of protein. A

pneumoencephalogram at this time (Fig. 3), some 6 weeks after the first, demonstrated a decrease in size of the anterior portion of the left lateral ventricle, but an increase in size of the remainder of the left lateral ventricle and of the right lateral ventricle. The configuration of the septum lucidum appeared slightly irregular but there was no longer evidence of an intraventricular filling defect. The 3rd ventricle was still dilated. The aqueduct and 4th ventricle appeared a little enlarged but showed no displacement. The basal cisterns were well filled and appeared clear. The findings were interpreted as indicating absorption of the previously demonstrated hematoma.

There has been no further evidence of neurologic symptoms or signs. Nine months after the hemorrhage he had resumed his previous activities.

#### Discussion

Documentation of spontaneous absorption of a massive intraventricular hematoma with full recovery to our knowledge has not been recorded previously. The fate of blood in the ventricular system following spontaneous hemorrhage was first summarized by Sanders in 1881.<sup>11</sup> He reported that:

"The blood being extravasated into the ventricles may disappear by absorption completely; its fluid portion may be absorbed, while its solid parts remain undergoing calcareous or other changes; it may, perhaps, induce chronic internal hydrocephalus; it may give rise to simple serous cysts; it may cause acute ventricular meningitis; or it may remain for a long time almost unchanged and unacted upon."

Presumably the formation of a hematoma in the ventricle is related to the degree of hemorrhage,

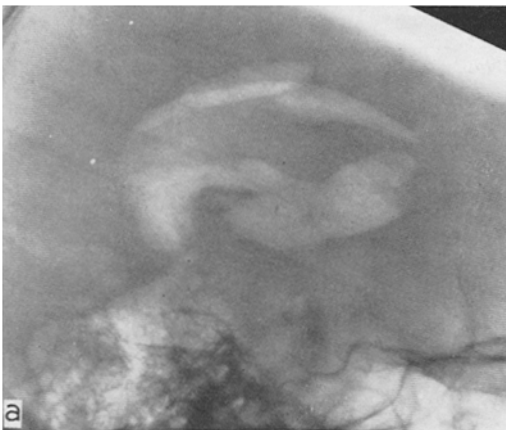


FIG. 2. First pneumoencephalogram. The intraventricular hematoma fills most of the left lateral ventricle. (a) Erect lateral projection. (See also Fig. 2b and c.)