Infantile Hydrocephalus and Hematoma in the Posterior Fossa

Presentation of Case

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The genesis of congenital communicating hydrocephalus has been discussed for many years. Two of us, from previous studies, hold the opinion that the most probable cause is an arachnoiditis depending on intracranial hemorrhage in the perinatal period. Only rarely has a hydrocephalus-producing hematoma been verified through early operation. In these cases the hematoma, because of its size and location in the posterior fossa, caused mechanical obstruction of the cerebrospinal-fluid pathways. In most cases the signs of intracranial hemorrhage are less pronounced, however, and surgical intervention consequently is not considered to be indicated. But even these moderate hemorrhages may cause a posthemorrhagic arachnoiditis resulting in communicating hydrocephalus.

We wish to present another case of interest, because these cases are rare. This report further elucidates and supports our opinion concerning the etiology of so-called congenital communicating hydrocephalus.

Case Report

C.J. was a full-term female child. Delivery was uncomplicated except for the fact that the amniotic fluid had a greenish discoloration. Circumference of the head at birth was 33.5 cm. (13¼ in.). Immediately after birth the skin was somewhat grey, there was a whining cry, and breathing was irregular with apnoeic spells. There were increased tonus of the extremities, increased irritability, and frequent tonic and clonic seizures during the first few days of life. During the first 3 weeks the infant vomited frequently.

A rapid improvement of the condition occurred during the first 3 days, followed by a slower, gradual improvement so that the patient appeared well at the age of 8 weeks, aside from the fact that the circumference of the head had increased to 40 cm. (15¾ in.). The fontanel was bulging. A lumbar pneumoencephalogram and a ventriculogram were done.

Roentgen Findings. Fractional lumbar encephalography was done according to Robertson's and Lindgren's principles. Only a small amount of gas entered the ventricular system (insufficient for detailed study of the ventricles). The cisterna magna and the cisterns dorsal to the cerebellum were well filled by gas and were wider than usual. The medullary and the pontine cisterns were of normal width. No further cisterns were filled with gas and no gas was found over the cerebral convexities. The tonsils were not depressed. Incomplete filling of cisterns and a lack of subarachnoidal gas over the convexities were both indicative of an obstruction of the extracerebral cerebrospinal-fluid pathways. The obstruction began between the pontine cistern and the interpedunculate cistern.

The thickness of the brain could be estimated roughly from the small amount of gas that entered the lateral ventricles and was found to be about 15 mm. over the frontal pole of the right lateral ventricle.

Ventriculography demonstrated a marked kink of the aqueduct causing considerable obstruction at the level of the quadrigeminal plate. The caudal portion of the aqueduct and the 4th ventricle were only partially filled with gas. No gas had passed from the ventricles into the basal cisterns. In addition to a pronounced hydrocephalus there was local atrophy of the posterior and temporal horns on the left side.

Summarizing the roentgen findings: 1) space-occupying lesion in the posterior fossa, 2) changes indicating arachnoiditis, 3) pronounced hydrocephalus, 4) local atrophy of left occipital region (probably as a result of the perinatal brain damage).

Operation. At the age of 5 weeks a bilateral suboccipital craniectomy was performed under general anesthesia. In the subdural space of the posterior fossa an encapsulated, organized hematoma was found and removed.

Course. Postoperatively the head continued to increase in size. At the age of 11 weeks the circumference was 45 cm. (17¾ in.) A second lumbar pneumoencephalogram then was done. On this examination the gas entered the ventricular system rapidly. The abnormal kink of the aqueduct now had disappeared. The dilatation of the ventricles had progressed further and the cerebrum was only 5–10 mm. thick, measured in the same place as above. As in the previous study changes indicative of arachnoiditis were found. At this time, however, it was not possible to obtain filling of the cisterns dorsal to the cerebellum.

A ventriculo-atrial shunt according to Spitz-Holter was performed and the further course has been uneventful for 20 months, aside from the fact that the child outgrew the jugular catheter, which had to be changed.

Discussion

As was true of 1 case presented previously, the genesis of hydrocephalus in our case was twofold: when the obstructing mass had been removed the hydrocephalus continued to progress because of a posthemorrhagic arachnoiditis. We know that intracranial hemorrhage, and especially subarachnoid hemorrhage, can cause hydrocephalus in adults. Undoubtedly this is also true in infants. If the subdural hematoma had not been diagnosed and treated early, as in our
case, it probably would have been more or less absorbed. The passage of gas through the sylvian aqueduct then would have been normalized and the condition probably would have been described by most investigators as a case of so-called congenital communicating hydrocephalus.

**Summary**

We have presented a case of hydrocephalus in an infant, the basic cause being intracranial hemorrhage in the perinatal period. Surgical removal of a hematoma in the posterior fossa did not give permanent relief of the raised intracranial pressure. A lumbar air study at this stage disclosed information that the passage through the intracerebral cerebrospinal-fluid pathways was normalized whereas the extracerebral ones still were occluded by arachnoiditis. The condition necessitated a ventriculo-atrial shunt.

**References**


