CASE REPORTS

MEMBRANOUS OBSTRUCTION OF AQUEDUCT OF SYLVIIUS (INTERNAL HYDROCEPHALUS) PRODUCING SYNDROME OF MIDLINE CEREBELLAR TUMOR*

A CASE REPORT

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The purpose of this case report is to call attention to an uncommon cause—congenital membranous obstruction of the aqueduct of Sylvius—for the production of the signs and symptoms of a midline cerebellar tumor, and to the unusual length of time—23 years—it took for this membrane to completely obstruct the aqueduct of Sylvius.

CASE REPORT

C.M., (G.H. Case #176376), female, aged 23 years. Vagie complaints for 15 years culminating in a 6-month period of evidences of increased intracranial pressure and cerebellar signs. Congenital membranous obstruction aqueduct of Sylvius found at operation. Improvement.

History. This right-handed white female was admitted to the Neurosurgical Service of the Graduate Hospital on 26 March 1947 with the chief complaints of vertigo and difficulty in walking.

She was born spontaneously at full-term and was considered a normal infant. Early physical and mental development was normal, and she completed a 4-year high school course at the age of 18. Up to the onset of her present illness, she was a mill worker.

At age of 5 years, she was struck by an automobile, not rendered unconscious but sustained rather severe lacerations of the left forehead which resulted in several small scars. She had no other major injuries, infectious diseases, or operations.

At age of 12, a “roaring” noise “in the head” developed. This noise was fairly constant, “hissing” in character and aggravated by stooping or any exertion. The patient stated emphatically that the noise was not “in the ears.” This symptom lasted up until the present admission to the hospital.

At age of 17, concomitant with onset of her menses, attacks of momentary unconsciousness developed. In the beginning these attacks occurred as often as 4 or 5 times a day. They were not influenced or related to her menstrual cycle, intake of food or any other known factor, but occurred diurnally. A few years before admission she was placed on dilantin (gr. 1.5), 3 times a day, and under this medication the attacks had been reduced to one in 2 months.

At age of 21, 1½ years prior to admission, she was seen by a neurologist, Dr. Joseph C. Yaskin, at which time she complained of “fainting spells” and a “roaring noise” in the head. His positive neurological findings were: Head slightly larger than normal; left eyeball more prominent than the right; normal eyegrounds; and compression of either carotid artery obliterated the noise in the head. It was Dr. Yaskin’s opinion at that time that the patient had some congenital cerebral lesion which might require surgical intervention.

Subsequent to this examination and 1 year prior to admission, the patient began to complain of vertigo and noticed difficulty in walking. These symptoms gradually became more severe until, 3 weeks before admission, she became so atactic and weak that she was confined to bed. There was no history of headaches, tinnitus, deafness, loss of vision or diplopia.

Neurological Examination. The patient was an apathetic, sallow-complexioned, well-developed girl of 23. General physical examination was essentially normal. The head was

slightly larger than normal. There was bilateral papilledema of 1 to 2 D., without hemorrhages or exudates. A fine rotatory nystagmus was present on extremes of lateral gaze. All tendon reflexes were equally increased and a bilateral Hoffmann and Babinski reflex were obtained.

![Roentgenogram of skull](image1)

**FIG. 1.** Roentgenogram of skull, showing enlargement of sella turcica with thinning of the dorsum, and prominent digital markings of the vault.

![Ventriculograms](image2)

**FIGS. 2 and 3.** Ventriculograms. Lateral view shows marked dilatation of lateral ventricles and partial filling of dilated 3rd ventricle. Anteroposterior view shows symmetrically dilated lateral and 3rd ventricles.

There was dyssynergia of the cerebellar type in all four extremities, with a well-marked ataxic gait and a positive Romberg test. The remainder of the neurological examination was normal.

Laboratory studies of urine, blood count, blood Wassermann and blood examinations of