Abducens Palsy Following Shunting for Hydrocephalus

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ABSTRACT: Over a period of 12 years, 80 patients underwent ventricular shunting for normal pressure hydrocephalus. Three developed sixth cranial nerve palsy in the first two weeks after surgery. This uncommon complication is usually transitory following the same pattern of abducens palsy after lumbar puncture or spinal anesthesia. Traction on the nerve with local ischemia has been involved as the responsible mechanism in both instances.

RÉSUMÉ: Paralysie du droit externe de l'oeil à la suite d'une dérivation pour hydrocéphalie. Sur une période de 12 ans, 80 patients ont subi une dérivation ventriculaire pour une hydrocéphalie normotensive. Trois ont développé une paralysie du sixième nerf crânien dans les deux premières semaines après la chirurgie. Cette complication peu fréquente est habituellement transitoire et évolue comme la paralysie du droit externe survenant après une ponction lombaire ou une anesthésie spinale. Une traction du nerf accompagnée d'ischémie locale a été invoquée comme étant le mechanisme responsable dans les deux cas.

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The causes of abducens palsy are multiple and have been described before in the medical literature.[1,2] Nevertheless, its occurrence after ventricular shunting for normal pressure hydrocephalus (NPH) is so unusual that it deserves special attention.

Case Reports

Patient 1

A 69-year-old male presented with a 4-year history of unsteady gait, progressive memory impairment and recent onset of urinary incontinence. On examination there was difficulty recalling current events, gait apraxia and hyperreflexia of the lower extremities. Computerized tomography (CT) of the brain demonstrated ventricular dilatation. The results of an isotope cisternogram and lumbar subarachnoid infusion test suggested a diagnosis of NPH. A ventriculoatrial shunt was placed and the opening pressure was 18 cm of CSF. A Pudenz valve with a closing pressure of 5 cm of H_2O was used. Postoperative CT scan showed marked decrease in the ventricular size and brain atrophy.

Nine days after surgery the patient complained of nausea and headache followed by double vision when looking towards the right side. The right eye had limited abduction consistent with a sixth nerve palsy. Two days later the headache and nausea disappeared; however the right abducens palsy persisted for nine weeks. His gait, urinary incontinence, and memory improved gradually.

Patient 2

A 69-year-old priest with a five-year history characterized by difficulty walking and progressive memory loss was admitted to our institution. Physical examination revealed minimal impairment of recent memory and an abnormal gait characterized by a wide base with short, unsteady steps. The tone was increased in the lower extremities with associated hyperreflexia. CT scan of the brain showed ventricular dilatation. Magnetic resonance imaging (MRI) disclosed a smooth, hyperintense border around the ventricles and marked signal loss in the aqueduct. MRI cine was compatible with NPH. A lumbar puncture was not done as part of his investigations. A ventriculoperitoneal shunt was placed. The ventricular pressure was 15 cm of CSF and a Pudenz valve with a closing pressure of 5 cm of H_2O was used.

On the seventh postoperative day, the patient complained of headache and double vision on either right or left gaze elicited initially only at distant fixation. Two days later it was clear he had developed bilateral abducens palsies. The headache was short-lived. The esotropia improved gradually for six months and then reached a plateau of 16 dioptres on primary, right and left gaze. This required surgical correction 11 months after shunting. His gait improved and one year after surgery the patient returned to his previous occupation.

Patient 3

A 67-year-old male, retired engineer, was admitted with a history of four years characterized by progressive gait unsteadiness, tendency to drop objects, decreased sex drive and recent inability to concentrate in his daily tasks. Physical examination revealed recent memory impairment, increased tone in lower limbs and bilateral up-going toes. His gait was characterized by short, shuffling steps that improved temporarily after a lumbar puncture. CT scan of the brain showed an enlarged ventricular system with minimal signs of atrophy. MRI findings were suggestive of NPH. He underwent ventriculoperitoneal shunting with no obvious complication during the procedure. The ventricular pressure measured 15 cm of CSF and a low pressure Pudenz valve with a closing pressure of 4 cm of H_2O was used.

Fourteen days after surgery he developed a short episode of nausea, vomiting and severe headache that was followed by diplopia on either right or left gaze. Marked limitation of abduction was seen in both eyes, consistent with bilateral sixth cranial nerve palsies. There were 40 dioptres of esotropia in the primary position. Three weeks later the abducens palsies started to improve and cleared completely 11 weeks after surgery.

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Discussion

Abducens nerve palsy as a complication of ventricular shunting has been reported previously in only two patients.[3] In those cases and in ours, diplopia developed within two weeks of shunting and was preceded by a short period of headache and nausea. In four of the five patients, the condition cleared within three months. One patient developed permanent bilateral abducens palsies that required extraocular muscle surgery.

The same clinical pattern of sixth nerve palsies may follow lumbar puncture, myelography or spinal anesthesia.[4-10] It has been proposed that in these cases the mechanism is related to traction on the nerve, resulting from displacement of the brain after loss of CSF support in the basal cisterns.[11] The abducens nerve may be susceptible to traction because it bends abruptly at the petrous ridge to pass forward under the petrosphenoid ligament.[11-13] Two of our patients had a lumbar puncture as part of the pre-operative investigations; however, these were done well before surgery and could not account for the abducens palsies.

The mechanism outlined above could explain the association of sixth nerve palsies and ventricular shunting. Other mechanical factors like prominent petrous ridges, large branches of the basilar artery crossing over the abducens, and variant courses of the sixth cranial nerve rendering it more susceptible to changes in intracranial pressure have been invoked trying to explain this condition.[10] These factors, if present unilaterally, could justify the cases of unilateral abducens palsy following ventricular shunting.

The delayed onset of sixth cranial nerve palsy experienced by our patients, associated with headache and nausea, suggest overdrainage of CSF with consequent traction on the nerve (Figure 1). It is not known if this complication could have been prevented by using a valve with a higher closing pressure; however, this is suggested by the fact that all our patients, as well as those reported by Black,[3] had valves with closing pressures lower than 100 mm of CSF.

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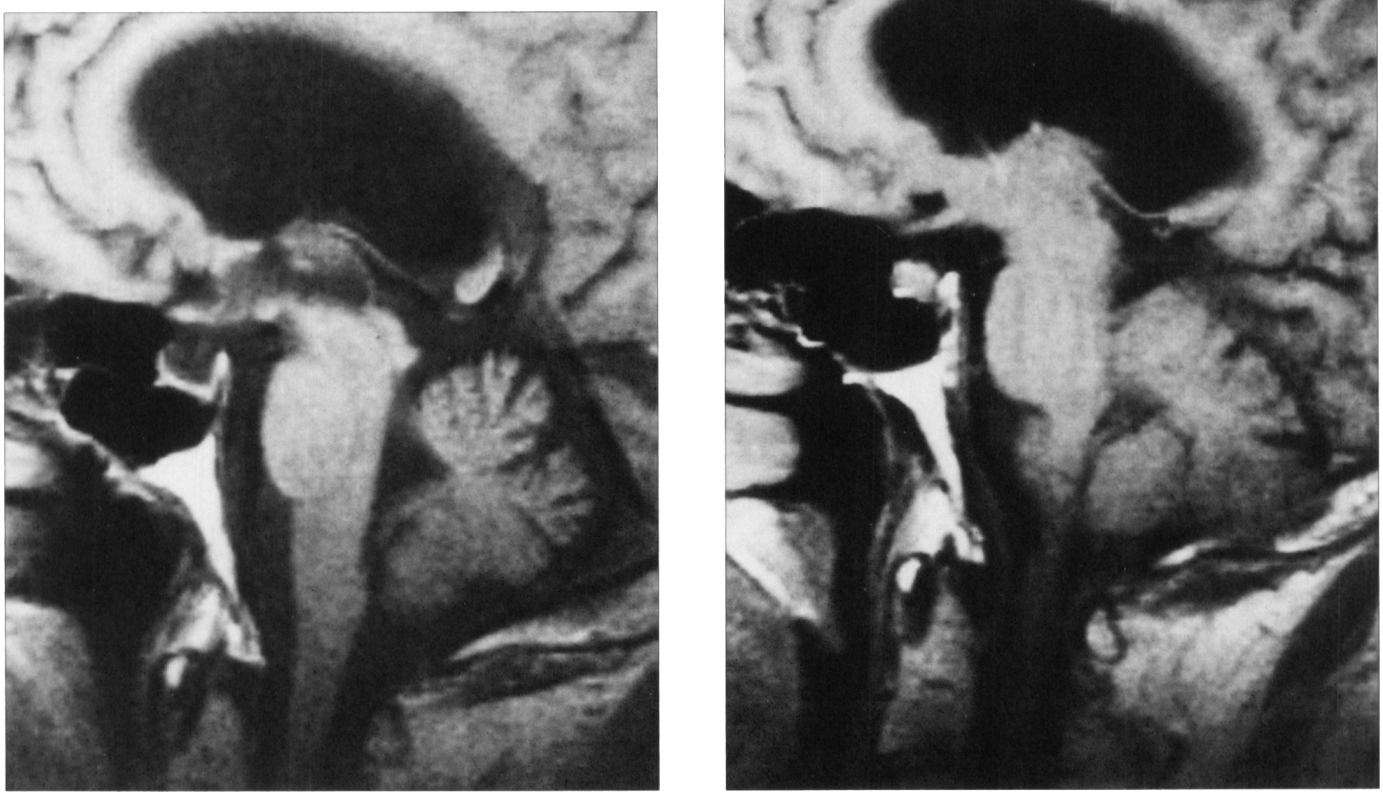

A B

*Figure 1 — Pre (**A**) and postoperative (**B**) MRI's from patient 2 showing a decrease in the amount of CSF at the pontine cistern after ventriculoperitoneal shunting. This study was obtained seven days after the onset of diplopia.*

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