“High Pressure” Cerebrospinal Fluid Rhinorrhea due to Parieto-Occipital Meningioma, a Case Report

Sang Kun Park, Kyu Chang Lee and Hun Jae Lee

Department of Neurosurgery, Yonsei University College of Medicine, Seoul, Korea

A case of non-traumatic “high pressure” cerebrospinal fluid rhinorrhea is reported. There was a huge fibroblastic meningioma in the left parieto-occipital region along with hydrocephalus.

Initially a ventriculoperitoneal shunt was done and then 12 days later the tumor mass was removed totally. After the operations the patient became free of leakage and a direct approach for the cerebrospinal fluid rhinorrhea was not needed.

Cerebrospinal fluid rhinorrhea resulting from trauma or following cranial surgery is common and well documented (Cairns, 1937; Lewin, 1954; Morley and Hetheington, 1957) and the term, spontaneous cerebrospinal fluid rhinorrhea, has been used as a clinical entity since 1899 (Thomson, 1899).

Other authors subdivided this condition into ‘primary spontaneous’ or ‘idiopathic’ rhinorrhea when a precipitating cause could not be found and ‘secondary spontaneous’ rhinorrhea when a cause, usually a tumor, was discovered (O’Connell, 1953). Quite apart from the nosological adequacy of such a subdivision, the term, spontaneous, cerebrospinal fluid rhinorrhea itself would appear to be inexact, bearing no relationship to either the pathogenesis or natural history of the disease. Ommaya et al. (1968) suggested that a careful study of the natural history of the patients suffering such rhinorrhea revealed that no actual case could fit such a definition—that is, in being truly spontaneous. So he suggested that it would be more accurate to describe all such cases, hitherto labelled spontaneous, by the more general term of non-traumatic cerebrospinal fluid rhinorrhea.

The non-traumatic cerebrospinal fluid rhinorrhea was subdivided into, high pressure and, normal pressure categories. In the high pressure category the leakage of cerebrospinal fluid is usually acting as a real safety valve, closure of which will invariably worsen the patient’s condition if the causative lesion is not treated (Ommaya, 1964).

In our case there was a huge fibroblastic meningioma in the left parieto-occipital region along with hydrocephalus. The tumor mass was removed totally 12 days after a ventriculoperitoneal shunt operation. After the shunt surgery and tumor operation the patient became free of cerebrospinal fluid rhinorrhea.

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CASE SUMMARY

A 32 year old woman was admitted with watery rhinorrhea and headache for one year. Persistent watery rhinorrhea which was severe in the morning developed about one year prior to admission following blunt trauma to her face. She had also had two generalized seizures, one 6 years previously and one 4 days before admission. Her mental state was alert. She had bilateral anosmia, papilledema, right hemihypesthesia and a suspicious right hemiparesis.

Analysis of the watery nasal discharge revealed that protein was 86 mg/dl, sugar 95 mg/dl, chloride 128 mEq/L and white blood cells 5/mm^3. Plain skull X-ray showed hyperostosis of the left occipital bone with a prominent vascular groove, ballooning of the sella turcica and enlargement of left foramen spinosum (Fig. 1). Laminogram of the sella turcica showed disappearance of the floor in its posterior portion (Fig. 2). Brain scanning with In-113 showed increased radioactivity in the left occipital region, and was suggestive of a highly vascular tumor. 4 vessel angiogram revealed a huge mass in the left parieto-occipital region (Fig. 3). The arterial supply to it were the posterior branch of the middle meningeal artery, the angular branches of the left middle cerebral artery, and the calcarine and parieto-occipital branches of the internal occipital artery. The tumor mass was stained homogeneously in the capillary phase. Several large veins drained into the superior sagittal sinus and into the straight sinus from this mass. On the 4th hospital day a ventriculoperitoneal shunt operation was performed. Subsequently cerebrospinal fluid leakage became reduced. And 12 days after the ventriculoperitoneal shunt a left parieto-occipital osteoplastic craniotomy was done after ligation of the external carotid artery, and then the tumor mass was removed totally.

The tumor mass was measured 9×6×5 cm. The surface was nodular. The cut surface showed grayish fibrous tissue and some fibrous whorls. Pathological diagnosis was fibroblastic meningioma. There was no more cerebrospinal fluid rhinorrhea after the third postoperative day. And the patient was discharged on the 9th postoperative day with neurological improvement and without cerebrospinal fluid rhinorrhea.

DISCUSSION

Cerebrospinal fluid rhinorrhea results most frequently from trauma. About 2% all head injured patients may suffer from such a problem (Lewin, 1964). Ray (1969) analyzed 41 cases of cerebrospinal fluid rhinorrhea. Among these fistulas 27% resulted from skull fracture; 10% were thought to be congenital; 37% were associated with intracranial tumors; 47% occurred following neurological or otolaryngological surgery, most often after manipulation of tumors which had encroached on nearby sinuses. In 24% of the patients there was a history of associated meningitis. In 7% the presence of increased intracranial pressure necessitated a shunting procedure before the leak could be closed. In one series five patients (12%) developed fistulas followed intracranial hypophysectomy. In 39% of the patients the fistulas were in the area of the cribriform plate and cells of the ethmoid sinus. In 15% the fistulas extended into the frontal sinus, and in another 15% they extended from the sellar turcica to the sphenoid sinus. In 22%, the site of the leak was never found.

Various factors have been considered as
contributing to the etiology of non-traumatic cerebrospinal fluid rhinorrhea. O’Connel (1953) thought that the normal rhythmic variation in the pressure of cerebrospinal fluid could pave the way for a leak, especially if an anatomical defect made the cribiform plate particularly vulnerable to these tidal movements.

Jauregg (1936) claimed that any act which increased intracranial pressure suddenly, such as sneezing or coughing, could force open an anatomical defect which hitherto was not apparent.

In certain cases of delayed traumatic cerebrospinal fluid leakage the actual precipitating cause for the rhinorrhea may be analogous to mechanisms producing non-traumatic leaks. This would explain case reports of patients with delayed traumatic cerebrospinal fluid rhinorrhea in whom associated congenital anomalies related to the fistula are found such as a persistence of the basi-occipital canal (Voena, 1959). Similarly the onset of cerebrospinal fluid rhinorrhea in certain cases of pituitary tumor treated with X-ray alone suggests a similarity between post-radiation atrophy and the post-traumatic atrophy of cerebral tissues which may be responsible for the onset of delayed traumatic cerebrospinal fluid rhinorrha (Ommaya, et al., 1968).

The category of cerebrospinal fluid rhinorrhea caused by tumors is subdivided into “direct” and “indirect.” This indicates the two ways in which the fistula may be created by the tumors, that is, either directly by erosoin of the meninges and bone or indirectly via pressure erosion of anatomically fragile areas of the skull base (Ommaya, et al., 1968). It is significant that the most fragile area is the cribiform plate.

Teng and Papaetheodorous (1965) have emphasized that pituitary tumors are the most common lesion producing cerebrospinal fluid rhinorrhea. Hydrocephalus is a rare cause of cerebrospinal fluid rhinorrhea. Occasionally hydrocephalus is associated with a congenital anomaly as the cause of a high pressure cerebrospinal fluid leak and such an association has been reported with Crouzon’s disease and Alberg-Schönberg disease (Ommaya, et al., 1968).

Focal atrophy is also a causative factor of non-traumatic cerebrospinal fluid rhinorrhea. It is considered that the contents of the cribiform plate or sella turcica atrophy may be due to ischemia. The empty space thus created is filled with a cerebrospinal fluid laden arachnoid pouch which is an enlargement of the normal arachnoid space extensions. This pouch is thought to exert a focal and continual erosive effect analogous to the creation of cranial vault excavations by the arachnoid granulations. The majority of case reports of “primary spontaneous” rhinorrhea are explicable by these categories (O’Connel, 1964). Osteomyelitis is another causative category. But this is very rare cause.

The diagnostic approach for this category was well established. Limiting himself to non-traumatic cerebrospinal fluid rhinorrhea, Ommaya, et al. (1968) suggested that a combination of accurate radiographic studies and isotope-cisternography gave the best results.

This is primarily a surgical disease. The intracranial, intradural approach is recommended for all non-traumatic cerebrospinal fistulae with careful “patching” of the fistula site, preferably using the patient’s own tissues as free grafts. Sealing of subjacent bony defects is required only when the hole is of significant size-for example, greater than 2 mm in diameter. In high pressure leaks, removal of the tumor or bypass of the obstruction should always precede repair of the fistula, which should be carried out only where it is positively established that a “safety-valve” function is no
longer required. In normal pressure leaks, obliteration of the arachnoid hernia should accompany careful intradural repair of the fistula (Dandy, 1944; Ommaya, 1968).

Rovit et al. (1969) reported a case of cerebrospinal fluid rhinorrhea with left parieto-occipital meningioma. He removed the tumor mass totally, but the cerebrospinal fluid rhinorrhea didn't stop. So a direct approach for cerebrospinal fluid rhinorrhea was done. Our case had left parieto-occipital meningioma with hydrocephalus. Blunt head trauma provoked the leakage. Shunt operation was done first to change the intracranial pressure dynamics. Following that procedure the cerebrospinal fluid rhinorrhea became reduced and the tumor was removed completely. After total removal of the tumor mass, cerebrospinal fluid leaked no more, and a direct approach for it was not necessary.

**SUMMARY**

A case of "high-pressure" cerebrospinal fluid rhinorrhea is described. A large fibroblastic meningioma with ventricular dilation was found. A ventriculoperitoneal shunt operation was performed first. Twelve days later the tumor mass was removed totally. The patient improved and cerebrospinal fluid leakage stopped.

**REFERENCES**


Jaureg W: Vom niese. Wien Med Wschr 86:9-12, 1936


O'Connell JEA: The cerebrospinal fluid pressure as an aetiological factor in the development of lesions affecting the central nervous system. Brain 76:279-298, 1953


Thomson St C: The cerebrospinal fluid: Its spontaneous escape from the nose. Cited from Cassell: London 1899

Fig. 1. Plain skull lateral and basal views.
(a) Lateral view showing prominent vascular groove with hyperostosis of posterior parietal bone and ballooning of sella turcica.
(b) Basal view showing enlargement of left foramen spinosum.

Fig. 2. Plain skull tomograms showing disappearance of the floor of sella turcica.
Fig. 3. 4 vessel angiogram showing space occupying lesion with ventricular dilatation.
(a) Carotid angiogram frontal view.
(b) Carotid angiogram lateral view.
(c) Vertebral angiogram A-P view.
(d) Vertebral angiogram lateral view.