Cerebrospinal Fluid Rhinorrhea Secondary to Small Tumor Invasion into the Superior Sagittal Sinus
—Case Report—

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Abstract

A 53-year-old male presented with non-traumatic cerebrospinal fluid rhinorrhea due to obstructive extension of a small parasagittal meningioma. Radical excision of the tumor was curative. The rhinorrhea apparently developed due to increased intracranial pressure associated with primary empty sella.

Key words: empty sella, increased intracranial pressure, parasagittal meningioma, papilledema, rhinorrhea, superior sagittal sinus

Introduction

Non-traumatic cerebrospinal fluid (CSF) rhinorrhea is usually caused by sellar pathology. For example, necrosis of a pituitary adenoma allows the passage of CSF from the subarachnoid space to the nasal cavity, resulting in rhinorrhea in the presence of a defect in the sellar dura and bone.1 Fluctuations or elevation of CSF pressure associated with benign intracranial pressure (ICP) syndrome may contribute to the development of rhinorrhea.1 However, obstruction of the superior sagittal sinus (SSS) by benign parasagittal tumor is seldom associated with rhinorrhea. Slow tumor growth is usually accompanied by well-developed compensatory mechanisms against increased ICP, and thus does not result in rhinorrhea.

We describe an unusual case of CSF rhinorrhea associated with a small parasagittal meningioma.

Case Report

A 53-year-old male was admitted with CSF rhinorrhea, and visual field impairment and decreased acuity. Neurological examination revealed bilateral papilledema. Postcontrast computed tomography (CT) demonstrated a homogeneously enhanced parasagittal mass which could not be identified by the precontrast CT (Fig. 1). Magnetic resonance (MR) imaging of the brain demonstrated a small enhanced mass predominantly located in the posterior part of the left parasagittal region and an enlarged sella filled with CSF (Fig. 2). Three-dimensional CT with contrast medium demonstrated a filling defect in the SSS corresponding to the location of the tumor (Fig. 3 upper). MR angiography demonstrat-
ed an occluded SSS without well-developed collateral venous pathways (Fig. 3 lower).

Lumboperitoneal CSF shunt placement was selected instead of radical excision of the tumor, which might have compromised collateral venous pathways. Rhinorrhea and papilledema temporarily resolved after the shunt placement. However, rhinorrhea recurred three times due to shunt malfunction within the 17-month period following this operation.

Repeated MR images 9 months after the first studies showed the enlargement of the tumor (Fig. 4). We finally performed radical excision of the tumor following repeated shunt malfunction. In contrast to preoperative expectations, complete occlusion of the SSS was not found, because venous bleeding was recognized through a small tear of the lateral wall of the SSS. The wall of the sinus attached to the tumor was grossly intact. The histological diagnosis was meningotheliomatous meningioma.

He has been free from rhinorrhea for 4 months following excision of the tumor.

Discussion

The pathophysiology of CSF rhinorrhea secondary to a small parasagittal tumor presumably involves persistent increased ICP due to obstruction of the posterior SSS by tumor growth, and coexisting primary empty sella. Obstruction of the SSS will result in variable increases in ICP depending on the effectiveness of compensating mechanisms. Obstruction of the SSS by a parasagittal meningioma without devastating clinical manifestations usually means that the tumor is in the advanced stage and there has been sufficient time for development of compensation mechanisms for increased ICP, principally extracranial and intracranial collateral venous networks. Parasagittal meningiomas tend to infiltrate into the SSS, and cavernous sinus meningiomas into the internal carotid artery. In our case, the relatively small tumor obstructed the SSS. The
tumor growth toward the SSS must have been too rapid to allow development of compensating mechanisms.

Empty sella syndrome results from extension of the subarachnoid space into the intrasellar position with subsequent remodeling of the sella turcica and flattening of the pituitary gland.4,6 There are primary and secondary types based on pituitary gland pathophysiology.5 Primary empty sella occurs in patients who have not received treatment of the pituitary gland such as radiation or surgery. The causes of primary empty sella are thought to include pituitary cyst, arachnoidal cyst, and increased ICP associated with various diseases including hydrocephalus, brain tumor, and Arnold-Chiari malformation.2 Increased CSF pressure transmitted through a defective diaphragm sellae is thought to be the most common reason for development of empty sella.2,9 Most affected patients are middle aged, obese, and generally female.2,9 Secondary empty sella occurs when the patient has received treatment for pituitary gland pathology. Our patient had no history of pituitary treatment, so the diagnosis was primary empty sella. Increased CSF pressure directly related to the presence of empty sella might be responsible for the high incidence of CSF rhinorrhea in affected patients.9 Presumably the CSF leakage in our patient was caused by a combination of persistently elevated CSF pressure subsequent to sinus obstruction and CSF pulsation into the intrasellar arachnoid space through the defective diaphragm sellae. The disappearance of the rhinorrhea following tumor removal supports the assumption that increased ICP due to obstruction of the SSS was important in the development of rhinorrhea and that empty sella probably contributed to this development. Three-dimensional enhanced CT and MR angiography were very useful for diagnosing SSS obstruction, and are less invasive than conventional cerebral angiography.

References

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