Herniation of the Third Ventricle into Empty Sella Caused by Surgery for Pituitary Apoplexy

—Case Report—

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Abstract

A 46-year-old male presented with acute visual loss in the right eye, high fever, nausea, and vomiting. This was caused by herniation of the third ventricle into empty sella at 15 months of surgery for pituitary apoplexy. The sellar-suprasellar tumor was totally removed *via* a transcranial approach. Histological examination showed chromophobe adenoma with necrotic tissue, indicating pituitary apoplexy. His visual field defect worsened 15 months after the operation, and magnetic resonance imaging revealed moderate hydrocephalus and protrusion of the dilated anterior inferior portion of the third ventricle into the sella. The optic nerve, optic chiasm, and pituitary gland were compressed onto the sellar floor. Ventriculoperitoneal shunt relieved the visual impairment. A decompressive procedure such as ventriculoperitoneal shunts is a reasonable treatment for such a marked herniation of the third ventricle.

Key words: empty sella, pituitary apoplexy, third ventricle, intrasellar herniation, ventriculoperitoneal shunt

Introduction

Herniation of the anteroinferior third ventricle into an empty sella can occur in the presence of either primary or secondary empty sella.^{4,7,8)} Diagnosis was based on ventriculography before magnetic resonance (MR) imaging was introduced, which now allows easy and accurate diagnosis. The true incidence of a herniated third ventricle into an empty sella remains unknown, but may be more common than previous diagnostic techniques could demonstrate.⁴⁾

We describe a case of herniation of the third ventricle into empty sella caused by surgery for pituitary apoplexy, and discuss the etiology and treatment of this type of third ventricle herniation.

Case Report

A 46-year-old male was admitted with acute visual loss in the right eye, high fever, nausea, and vomit-

ing. He had suffered from appetite loss and general fatigue for the past several months. Neurological examination revealed nuchal rigidity. Visual acuity in the right eye consisted of only light perception, and a perimetric test of the left eye revealed temporal hemianopsia. Endocrinological examinations showed mild suppression of pituitary function. Lumbar puncture performed in the lateral recumbent position revealed an opening pressure of 250 mmH₂O. Examination of cerebrospinal fluid revealed increased cell count (559/mm³) including 80% neutrophils, and increased protein content (145 mg/dl). Plain skull roentgenography showed a round enlargement of the sella turcica. Precontrast computed tomography (CT) revealed an isodense mass in the sellar-suprasellar region (Fig. 1 *left*). Postcontrast CT showed that the wall of the mass was enhanced (Fig. 1 *right*). Angiography showed opening of the bilateral carotid siphon and an avascular area corresponding to the mass.

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Fig. 1 *left*: Precontrast CT scan on admission revealing an isodensity mass in the sellar-suprasellar region. *right*: Postcontrast CT scan revealing enhancement of the wall of the mass.

The sellar-suprasellar tumor was completely removed through a right frontotemporal craniotomy. Histological examination of the tumor specimen found chromophobe adenoma with necrotic tissue, indicating pituitary apoplexy. The postoperative course was uneventful except for transient diabetes insipidus. A postoperative perimetric test showed left upper quadrantic hemianopsia in the left eye (Fig. 2 *left*) and only light perception in the right eye.

He reported increased visual impairment in the left eye 15 months after the operation. A perimetric test revealed worsening of his visual field defect (Fig. 2 *center*). MR imaging revealed moderate hydrocephalus and protrusion of the dilated anterior inferior portion of the third ventricle into the sella (Fig. 3 *left*). The optic nerve, optic chiasm, and pituitary gland

Fig. 3 *left*: T₁-weighted sagittal MR images 15 months after the operation for pituitary apoplexy showing the dilated anterior inferior portion of the third ventricle protruding into the sella, the distended optic and infundibular recesses, and the indistinguishable optic nerve, optic chiasm, and pituitary gland compressed onto the sellar floor. *right*: T₁-weighted axial MR image showing the distended optic and infundibular recesses located in the sella.

were compressed onto the sellar floor, and were not individually distinguishable. The optic and infundibular recesses were distended and located in the sella (Fig. 3 *right*).

A ventriculoperitoneal shunt (medium pressure) was implanted to decompress the optic nerve and chiasm. Postoperative MR imaging showed that herniation of the third ventricle was relieved, and normal anatomical conditions were observed. A postoperative perimetric test showed improvement of his visual field defect (Fig. 2 *right*).

Fig. 2 *left*: Perimetric test of the left eye after the operation for pituitary apoplexy showing left upper quadrantic hemianopsia. *center*: Perimetric test of the left eye 15 months later showing worsening of the visual field defect. *right*: Perimetric test after the ventriculoperitoneal shunting showing improvement of the visual field defect.

Discussion

The precipitating causes in the 15 previously reported cases of the third ventricle herniation were surgery and/or radiation of pituitary adenoma in 12 cases,^{3,4,6-8,11)} pituitary apoplexy in two,^{1,4)} medical treatment (bromocriptine therapy) of pituitary adenoma in two,⁴⁾ and postpartum necrosis in one case.⁴⁾ The precipitating cause in our patient was surgery for pituitary apoplexy.

A few cases have indicated that the intrasellar third ventricle herniation occurred primarily without any precipitating cause in the presence of either primary empty sella or normal sella.^{4,7)} Presumably the etiological mechanism of the primary third ventricle herniation is different from that of secondary herniation. Kaufmann *et al.*⁴⁾ pointed out that the infundibular recess was dilated on sagittal MR images in patients with primary third ventricle herniation. This indicates that increased cerebrospinal fluid pressure causing dilation of the infundibular recess may be responsible for the primary third ventricle herniation.

Persisting recessus infundibuli is an intrasellar ventricular diverticulum, but has been confused with third ventricle herniation. Persisting recessus infundibuli is considered to be a developmental malformation of the infundibular recess due to inhibition of its obliteration, but only four cases are known.^{2,5,9,10)} This rare malformation is considered to be different from third ventricle herniation etiologically.

The pathogenesis of third ventricle herniation into secondary empty sella remains unknown. Surgery and/or radiation may cause arachnoidal adhesions involving the sella and the chiasm, and these adhesions cause traction downward of the third ventricle accompanied by intrasellar cisternal herniation. Our case was characterized by the presence of hydrocephalus and the marked herniation of the third ventricle. In the presence of hydrocephalus, pulsations within the third ventricle cause thinning of the optic chiasm and the third ventricular floor, and this downward pressure may result in distension of the anterior inferior part of the third ventricle. These mechanisms would explain the marked herniation of the third ventricle in our patient.

Visual impairment associated with intrasellar herniation of the third ventricle occurred in seven of the 15 patients.^{4,7,8,11)} Presumably compression of optic nerve and chiasm caused the visual impairment. Kaufmann *et al.*⁴⁾ stressed the absence of correlation between the severity of visual symptoms and the degree of herniation, so the indications for surgical treatment must be evaluated with care. Visual impair-

ment associated with intrasellar third ventricle herniation has been treated with ventriculostomy,⁷⁾ chiasmectomy,¹¹⁾ and direct relaxation of stretched optic nerves by lysis of adhesions.⁸⁾ In our patient, the dilated anterior inferior portion of the third ventricle had compressed the optic nerve, optic chiasm, and pituitary gland onto the sellar floor. These structures are difficult to dissect without collateral injury, and chiasmectomy may cause further damage to the already compressed optic nerve, optic chiasm, and third ventricular floor (hypothalamus). Therefore, ventriculoperitoneal shunting was performed to relieve compression rather than direct surgical correction of the empty sella. Postoperative MR imaging revealed that herniation of the third ventricle was relieved, and normal anatomical conditions were observed. The postoperative perimetric test showed improvement of the visual field defect. A decompressive procedure is a reasonable choice for the treatment of such a marked herniation of the third ventricle.

References

- 1) Asai J, Fujimoto T, Fukushima Y: Empty sella as an intrasellar herniation of the third ventricle secondary to spontaneous degeneration of the prolactinoma. *No Shinkei Geka* 22: 241-246, 1994 (in Japanese)
- 2) Cabanes J: Asymptomatic persistence of infundibular recessus. *J Neurosurg* 49: 769-772, 1978
- 3) Caplan RH, Dobben GD: Endocrine studies in patients with the "empty sella syndrome." *Arch Intern Med* 123: 611-619, 1969
- 4) Kaufmann B, Tomsak RL, Kaufmann BA, Arafah B, Bellon EM, Selmon WR, Modic MT: Herniation of the suprasellar visual system and third ventricle into empty sellae: Morphologic and clinical considerations. *AJR Am J Roentgenol* 152: 597-608, 1989
- 5) Kühne D, Schwartz RB: Persisting intrapituitary recessus infundibuli. *Neuroradiology* 10: 177-178, 1975
- 6) McFadzean RM: The empty sella syndrome: A review of 14 cases. *Trans Ophthalmol Soc UK* 103: 537-542, 1983
- 7) Mortara R, Norace H: Consequences of a deficient sellar diaphragm. *J Neurosurg* 32: 565-573, 1970
- 8) Olson DR, Guiot G, Derome P: The symptomatic empty sella. *J Neurosurg* 37: 533-537, 1972
- 9) Schumacher M, Gilsbach J: A new variety of "empty sella" with cystic intrasellar dilatation of the recessus infundibuli. *Br J Radiol* 52: 862-864, 1979
- 10) Vallee B, Besson G, Person H, Mimassi N: Persisting recessus infundibuli and empty sella. *J Neurosurg* 57: 410-412, 1982
- 11) Welch K, Stears JC: Chiasmectomy for the correction of traction on the optic nerves and chiasm associated

with their descent into an empty sella turcica. *J Neurosurg* 35: 760–764, 1971

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