dopa test. The patient was suspected pure akinesia and progressive supranuclear palsy. First, high-frequency rTMS of primary motor cortex was examined, and showed the dramatic improvement. Next, chronic subdural electrodes were implanted over the motor cortex bilaterally. One year after surgery, the Unified Parkinson's Disease Rating Scale had improved remarkably, and she could walk four times faster than before. The $H_2{}^{15}O$ PET study showed a significant increase of rCBF in the left SMA and right dorsolateral prefrontal cortex after bilateral MCS. MCS may be an alternative treatment for patients with akinesia, including those with PD, and particularly for levodopa-resistant patients, who respond well to rTMS. © 2007 Movement Disorder Society

Key words: motor cortex stimulation; akinesia; repetitive transcranial magnetic stimulation.

HISTORY

A 67-year-old woman was admitted to our hospital with a 6-year history of gait disturbance. She could not stand up or walk alone because of frozen gait and impairment of postural reflexes. The patient also exhibited stuttering and dysphagia. Characteristic findings for Parkinson's disease (PD), such as tremor and rigidity, were absent. The patient had been treated for years with levodopa (L-dopa), with little effect, and her symptom was not changed by L-dopa withdrawal and load test (L-dopa: 200 mg). Psychogenic movement disorders were excluded by psychiatrist. On admission, she was on a regimen of 450 mg of L-dopa, 48.6 mg of carbidopa, 100 mg of amantadine hydrochloride, and 1 mg of pergolide mesylate.

EXAMINATION AND INITIAL TREATMENT

On admission, the patient was scored as IV on the Hoehn and Yahr Scale,[1] and 70 on the Unified Parkinson's Disease Rating Scale (UPDRS) (Table 1). Findings of magnetic resonance imaging and static study of [^{11}C] raclopride positron emission tomography (PET) showed normal pattern.

TABLE 1. *UPDRS, walking time, and step size*

	On admission	One year after MCS	After cessation of MCS	Twenty-four hr after restart of MCS
UPDRS				
Part 1	3	3	0	0
Part 2	32	22	28	18
Part 3	32	15	38	25
Part 4	3	1	3	3
UPDRS total	70	41	69	46
Walking time (s)	84.7	20	43.1	15.1
Step size (mm)	119	317	111	247

UPDRS, the Unified Parkinson's Disease Rating Scale; MCS, motor cortex stimulation; Walking time; the time to walk distance of 7 m.

Repetitive transcranial magnetic stimulation (rTMS) was applied via a figure-eight coil (MC B-70; Medtronic Functional Diagnosis A/S, Skovlunde, Denmark). The study was approved by the Ethics Committee of Osaka University Hospital. The coil was connected to a MagPro magnetic stimulator (Medtronic Functional Diagnosis A/S). An intensity of 90% resting motor threshold was used for treatment. A Brainsight frameless navigation system (Rogue Research, Montreal, Canada) was used to monitor the position and direction of the coil, and the position of the patient's head.[2]

First, 5-Hz rTMS (total 500 times; right 250, left 250 in serial order) was applied to bilateral M1 corresponding to the lower limb and to SMA with an interval of 48 hours. Her symptoms were assessed before and after rTMS according to the time-to-walk distance of 7 m, step size, and a self-assessment test (Appendix) that we created based on UPDRS parts 2 and 3; the total possible score was 52. Every assessment was performed by other physicians according to the video record without any information. After rTMS of M1, her walking speed and her step size increased to 174 and 164% compared with those before rTMS, whereas after that of SMA, her walking speed and step size were 101 and 116%. The improvement rates of self assessment score were more than 25% after rTMS of M1 and less than 5% after that of SMA. The improvement after rTMS continued for 3 hours.

Next, 10-Hz and 1-Hz rTMS of bilateral M1 were examined. A total of 500 of each of the rTMS were applied, with an interval of 48 hours between each new series. Her walking speed and step size increased to 477 and 199% after 10-Hz rTMS, and 187 and 169% after 1-Hz rTMS. Ten-hertz rTMS of M1 showed the best improvement in this series (Fig. 1).

There has been no report of electrical stimulation of M1 motor cortex stimulation (MCS) surgical treatment for pure akinesia (PA) and progressive supranuclear palsy (PSP). However, the patient showed little response to L-dopa. The patient then wished to undergo MCS.

SURGERY AND POSTOPERATIVE COURSE

Implantation of subdural electrodes was performed under general anesthesia. Bilateral craniotomy was performed over superior sagittal sinus. After the opening of dura mater, the location of central sulcus was confirmed by phase reverse of the N20 component upon stimulation of the median nerve using a subdural 20-grid electrode. A subdural quadripolar electrode (Resume II, model 3587A; Medtronic, Minneapolis, MN) was then placed bilaterally on M1 adjacent to the superior sagittal sinus.

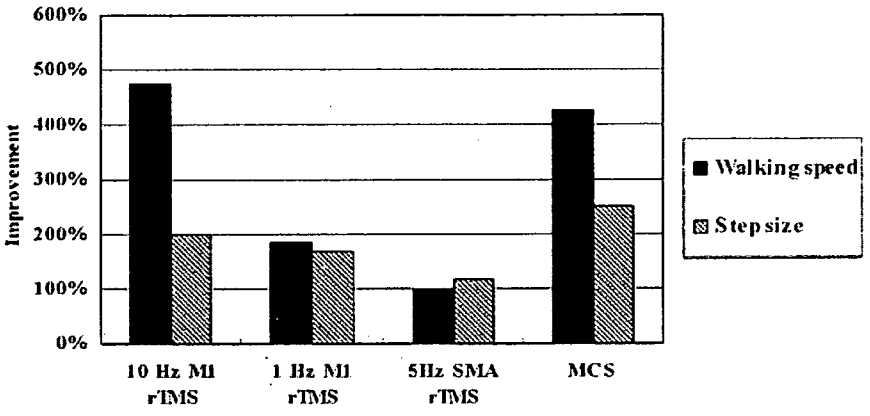

FIG. 1. Improvement of walking speed and step size (after rTMS/before rTMS). rTMS: repetitive transcranial magnetic stimulation. MCS: one year after motor cortex stimulation. Areas of significantly increased rCBF in the bilateral post-MCS phase compared to rCBF before MCS, rendered in the normalized images, indicate the left supplementary motor cortex (BA 6) and right dorsolateral prefrontal cortex (BA 9) (threshold, $P < 0.05$).

After 2 weeks of stimulation test, the best stimulation parameters were found to be 100 Hz, with a 1.8-V monophasic square-wave pulses lasting 0.21 milliseconds with a setting of array number 1 (+) and array number 2 (−) (Resume II has four arrays, numbered 0 to 3, medial to lateral) bilaterally. The two Resume II electrodes were connected to stimulation generators (I-Trel III; Medtronic) that were then implanted in the bilateral anterior chests. Stimulation was set on cycle mode (30 minutes on, and 120 minutes off).

One year after surgery, UPDRS part 3 had improved remarkably (Table 1), and she could walk four times faster than before, and her dysphagia improved too (Fig. 1).

The PET study with $H_2{}^{15}O$ was performed 6 months after surgery. The patient had not used cortical stimulation for 12 hours before the PET study. The PET study condition was as described before.[3,4] Briefly, six PET scans with $H_2{}^{15}O$ were performed before MCS as a prestimulation control and after 30 minutes of MCS. These acquisition data were analyzed with statistical parametric mapping software (SPM2; Wellcome Department of Cognitive Nerurology, London, UK).[5] Significance was accepted if a cluster showed a corrected threshold of $P < 0.05$. Significant increased rCBF after MCS revealed in the left SMA (Broadmann's area: BA 6) and right dorsolateral prefrontal cortex (BA 9) after 30 minutes of bilateral MCS.

To exclude placebo effects, we also performed a placebo-controlled single case crossover study with MCS on and off. The symptoms began to deteriorate 48 hours after cessation of stimulation, and another 24 hours later, her walking speed and step size decreased to less than half. Then, 24 hours after the stimulators were reactivated, the walking speed and step size were recovered to the precessation level (Table 1).

DISCUSSION

L-dopa-resistant akinesia, such as PSP or PA, is generally intractable and is not indicated for deep brain stimulation. Patients are treated with antiparkinsonian drugs received physical rehabilitation, and the condition worsens. The present patient showed L-dopa-resistant akinesia, but neither tremor nor rigidity was revealed. Neurologist suspected PA and PSP, and there still remained the diagnostic uncertainty. The clinical feature, a slow progress of motor symptoms without eye movement disorder in spite of a long duration, is not compatible with the diagnosis of PSP. A dopamine transporter imaging study would be useful for supporting the diagnosis; however, it has not been available in Japan. The patient's akinesia was remarkably improved with high-frequency rTMS of M1 and MCS.

rTMS of M1 has been reported for the treatment of PD. For example, 5-Hz rTMS in PD was first reported to improve akinesia.[6] Lefaucheur et al.[7] reported that 10-Hz rTMS improved bradykinesia and rigidity subscores of the upper limb contralateral to stimulation, and 0.5-Hz rTMS improved bilateral upper limb rigidity as well as gait. Because of noninvasiveness of rTMS, we selected it as an initial therapy and obtained good results, but the effect was temporary.

In the present case, rTMS of M1 improved akinesia, but that of SMA did not, and high-frequency rTMS was more effective than low-frequency rTMS. This is consistent with reports of rTMS in PD. The lack of effect of rTMS of SMA indicates that the effectiveness of rTMS of M1 is not due to a placebo effect. MCS has also been reported to improve tremor, rigidity, and akinesia in 16 cases of advanced PD.[8] And in cases of intractable pain, rTMS predicted the efficacy of MCS.[9] In our patient, we placed a subdural electrode on M1.

We had applied the cycle mode for this patient for the reasons given later. First, the beneficial effect after rTMS lasted for 3 hours. Second, for the MCS for intractable pain, the intermittent mode has been applied usually. Third, the risk of epilepsy in continuous stimulation has been worried. Consumption of battery is slower in cycle mode. In the present case, cycle mode is suited to improve the symptoms.

It has been reported that MCS activates both dopaminergic and nondopaminergic mechanisms to improve akinesia in PD.[7-10] Because L-dopa was not effective in our patient, nondopaminergic mechanisms may be involved. In the present case, $H_2{}^{15}O$-PET showed that SMA and prefrontal cortex were activated in response to MCS. It is known that SMA is involved in movement preparation and initiation.[11] In PD patients, inactivation of SMA during the execution of a motor task has been observed by rCBF,[12] and this can be reversed by treatment with dopamine analogs[12] and deep brain stimulation.[13] In cases of intractable pain, MCS increases rCBF in the thalamus, insular cortex, orbitofrontal cortex, anterior cingulate cortex, brainstem, and dorsolateral prefrontal cortex.[3,4,14] The distribution of increased rCBF in the present case differed from those in cases of intractable pain.

In a primate PD model, Drouot et al.[15] reported increased metabolic activity in the ipsilateral SMA, normalization of the mean firing rate in the globus pallidus and subthalamic nucleus, and no significant change in striatal uptake of ^{18}F-dopa in response to high-frequency MCS. Our results are almost consistent with theirs. Stimulation may relieve akinesia by improving the function of SMA via a corticocortical loop or via the basal ganglia.

rTMS is a noninvasive and effective treatment, but the effect is temporary. MCS may be an alternative treatment for patients with akinesia, including those with PD, and particularly for L-dopa-resistant patients, who respond well to rTMS.

APPENDIX A

Self Assessment Score

Movement of right leg 0. No complaints. 1. Occasional fumbling with ordinal activities but no practical disability. 2. Sometimes fumbling causing difficulty with daily life. 3. Always fumbling causing difficulty with daily life. 4. Essentially useless.

Movement of left leg 0. No complaints. 1. Occasional fumbling with ordinal activities but no practical disability. 2. Sometimes fumbling causing difficulty with daily life. 3. Always fumbling causing difficulty with daily life. 4. Essentially useless.

Movement of right hand 0. No complaints. 1. Occasional fumbling with ordinal activities but no practical disability. 2. Sometimes fumbling causing difficulty with daily life. 3. Always fumbling causing difficulty with daily life. 4. Essentially useless.

Movement of left hand 0. No complaints. 1. Occasional fumbling with ordinal activities but no practical disability. 2. Sometimes fumbling causing difficulty with daily life. 3. Always fumbling causing difficulty with daily life. 4. Essentially useless.

Speech 0. Normal. 1. Slightly affected. No difficulty being understood. 2. Sometimes asked to repeat statements. 3. Frequently asked to repeat statements. 4. Unintelligible most of the time.

Salivation 0. Normal. 1. Slight but definite excess of saliva in mouth. 2. Moderately excessive saliva; may have minimal drooling. 3. Marked excess of saliva with some drooling. 4. Marked drooling, requires constant tissue or handkerchief.

Swallowing 0. Normal. 1. Rare choking. 2. Occasional choking. 3. Requires soft food. 4. Cannot swallow.

Handwriting 0. Normal. 1. Slightly slow or small. 2. Moderately slow or small; all words are legible. 3. Severely affected; not all words are legible. 4. The majority of words are not legible.

Dressing 0. Normal. 1. Somewhat slow, but no help needed. 2. Occasional assistance with buttoning, getting arms in sleeves. 3. Considerable help required, but can do some things alone. 4. Helpless.

Walking 0. Normal. 1. Walk slowly without assistance. 2. Require stick. 3. Severe disturbance of walking, requiring assistance. 4. Cannot walk at all, even with assistance.

Tremor of right hand 0. Absent. 1. Slight and infrequently present. 2. Sometimes bothersome. 3. Interferes with many activities. 4. Interferes with most activities.

Tremor of left hand 0. Absent. 1. Slight and infrequently present. 2. Sometimes bothersome 3. Interferes with many activities. 4. Interferes with most activities.

Sensory complaints 0. None. 1. Occasionally has numbness, tingling, or mild aching. 2. Frequently has numbness, tingling or aching; not distressing. 3. Frequent painful sensations. 4. Excruciating pain.

LEGENDS TO THE VIDEO

rTMS, repetitive transcranial magnetic stimulation; MCS, motor cortex stimulation. On admission, the patient could not stand up and walk by herself. She presented freezing, short steps, and unbalanced gait. After 10-Hz rTMS, she could stand

up and walk by herself. One year after MCS, she presented remarkable improvement. She came to walk much faster with wide steps without freezing and falling.

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An Open Trial of Levetiracetam for Segmental and Generalized Dystonia

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Abstract: **Local botulinum toxin injections represent the treatment of choice for most patients with focal dystonia. However, patients with segmental or generalized forms require additional pharmacologic treatment which is often ineffective or limited by intolerable side-effects. An animal study and three case reports suggested antidystonic effects of levetiracetam, a pyrrolidone derivate, whereas a recent open-label study found no improvement in 10 patients with primary idiopathic cervical dystonia. We studied the efficacy of levetiracetam in a daily dose of 3000 mg in 10 consecutive patients with otherwise therapy refractory segmental or generalized dystonia. At 4-week follow-up, none of the patients showed improvement of dystonia, mild side-effects were observed in 3 patients.** © 2007 Movement Disorder Society

Key words: dystonia; levetiracetam; botulinum toxin

Dystonia is one of the most prevalent movement disorders with a minimum prevalence of about 100/100,000.[1] Local injections with botulinum toxin (BTX) are the treatment of choice for the majority of patients with dystonia. However, the use of BTX may be limited by the development of neutralizing antibodies against the toxin. In addition, patients with more widespread symptoms usually require additional oral medication to alleviate their symptoms. Drugs currently available to treat dystonia include anticholinergics, dopamine antagonists, benzodiazepines, baclofen, riluzole, or clozapine.[2,3] Nevertheless, in the majority of patients the above-mentioned drugs are only partially effective or their use is limited by intolerable side-effects.

Levetiracetam (LEV, Keppra), an S-enantiomer pyrrolidone derivate used in the treatment of epilepsy, has shown antidystonic effects in an animal model of paroxysmal dystonia.[4] In addition, isolated case reports have suggested beneficial effects of LEV in patients with

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Giant Intrasellar Arachnoid Cyst Manifesting as Adrenal Insufficiency Due to Hypothalamic Dysfunction

—Case Report—

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Abstract

A 67-year-old man first noticed loss of pubic and axillary hair in 1992 and then a visual field defect in 2001. He experienced loss of consciousness attributed to hyponatremia in April 2002. Magnetic resonance imaging showed a giant intrasellar cystic mass, 40 mm in diameter, that had compressed the optic chiasm. The patient complained of chronic headache, and neurological examination revealed bitemporal hemianopsia. Preoperative endocrinological examination indicated adrenal insufficiency, and hypothyroidism due to hypothalamic dysfunction. The patient underwent endonasal transsphenoidal surgery. The cyst membrane was opened and serous fluid was drained. Histological examination identified the excised cyst membrane as arachnoid membrane. The patient's headaches resolved postoperatively, but the bitemporal hemianopsia and endocrinological function were unchanged. This arachnoid cyst associated with hypothalamic dysfunction might have been caused by an inflammatory episode in the suprasellar region.

Key words: arachnoid cyst, sella, adrenal insufficiency

Introduction

Arachnoid cysts commonly occur in the middle fossa, around the cisterna magna, or in the cerebellopontine angle.[6,7] Intrasellar arachnoid cyst is rare,[2-6,8,10,11,13,17,19,20] and it is important, although difficult, to differentiate this type of cyst from other cystic lesions such as Rathke's cleft cyst, pituitary cyst (parenchymal or adenomatous), craniopharyngioma, pars intermedia cyst, and other miscellaneous cysts (epidermoid cyst, cysticercosis cyst) when considering the prognosis.[9,14] Hypothalamic dysfunction due to an intrasellar arachnoid cyst is extremely rare. Here we report on a case of a giant intrasellar arachnoid cyst presenting with hypopituitarism due to hypothalamic disorder and discuss the diagnostic and endocrinological features along with aspects of management.

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Case Report

A 67-year-old man had noticed loss of pubic and axillary hair for about 10 years beginning in 1992. A visual field defect developed in early 2001. He experienced transient loss of consciousness a few times after catching a cold in December 2001. He lost consciousness again and was admitted to emergency hospital on April 6, 2002. Blood tests showed hyponatremia (116 mEq/l), and his level of consciousness improved immediately after sodium replenishment.

Endocrinological study including evaluation of free triiodothyronine, free thyroxine, thyroid-stimulating hormone, prolactin, luteinizing hormone, follicle-stimulating hormone, growth hormone (GH), adrenocorticotrophic hormone (ACTH), and cortisol was performed, and the diagnosis was adrenal insufficiency, hypothyroidism, and hypogonadism (Table 1). He was treated with 5 mg of prednisolone and 25 μg of levothyroxine sodium per day. The serum sodium level was normalized within several days. Neurological examination revealed bitemporal hemianopsia.

Table 1 Preoperative endocrinological tests

Test	Hormone (unit)	Basal (normal range)	Maximum	Reaction
TRH	TSH (μU/ml)	1.27 (0.4-3.8)	15.95	normal
GRH	GH (ng/ml)	0.2 (0.0-6.0)	5.1	poor
CRH	ACTH (pg/ml)	22 (0-60)	147	normal
	Cortisol (μg/dl)	3.2 (4.5-24.5)	3.6	poor
LH-RH	LH (mIU/ml)	0.2 (1.8-5.2)	0.8	poor
	FSH (mIU/ml)	1.2 (2.9-8.2)	2.3	poor
	Testosterone (ng/ml)	0.0 (2.7-10.7)	unknown	unknown

Intravenous doses of TRH (500 μg), GRH (100 μg), CRH (100 μg), or LH-RH (100 μg) were administered. ACTH: adrenocorticotrophic hormone, CRH: corticotropin-releasing hormone, FSH: follicle-stimulating hormone, GH: growth hormone, GRH: growth hormone-releasing hormone, LH: luteinizing hormone, LH-RH: luteinizing hormone-releasing hormone, TRH: thyrotropin-releasing hormone, TSH: thyroid-stimulating hormone.

Table 2 Preoperative rapid adrenocorticotrophic hormone stress test results

	Time (min)					
	0	15	30	60	90	120
Cortisol (μg/dl)	1.2	2.6	3.7	5.8	6.3	6.6

ACTH secretion in response to corticotropin-releasing hormone (CRH) was within normal limits, but serum cortisol was reduced. A rapid ACTH stress test (Table 2) and consecutive ACTH test (Table 3) were performed to determine whether the adrenal insufficiency was primary or caused by reduced ACTH secretion. The adrenal response to ACTH was reduced but to consecutive ACTH was recovered. An insulin tolerance test showed the cortisol, ACTH, and GH responses were reduced. These endocrinological examinations suggested that the patient's adrenal insufficiency was due to hypothalamic disorder, which might have affected the functions of both the adrenal gland and the thyroid gland.

Preoperative skull radiography showed a markedly expanded sella turcica with thinning of the cortical bone of the dorsum sella but without abnormal calcification. Computed tomography (CT) showed a low-density intrasellar mass that was not enhanced with contrast medium. CT cisternography showed no influx into the intrasellar mass at 3, 24, and 48 hours after injection of 8 ml of iotrolan (240 mg/ml) into the lumbar subarachnoid space (Fig. 1). Preoperative T_1-weighted magnetic resonance (MR) imaging with gadolinium showed a large dumbbell-shaped intrasellar cystic mass (Fig. 2A). The signal intensity was slightly higher than that of the cerebrospinal fluid (CSF) and there was no enhancement of the cyst wall or contents. T_2-weighted MR imaging showed a homogeneous high-intensity mass, and upward displacement of the optic chiasm. The pituitary gland was not visualized clearly. MR angiography showed lateral shifting of the bilateral internal carotid arteries and elevation of the A_1 portions of the bilateral anterior cerebral arteries.

Endonasal transsphenoidal surgery was performed on July 12, 2002, to evacuate and evaluate the fluid in the cystic mass and to excise the cystic membrane. During the approach to the cystic mass, a firm membrane was seen just after drilling out the thinned floor of the sella turcica, without clear confirmation of the dura mater. Serous fluid like CSF was drained after partial excision of the membrane. CSF leakage was not noted. The floor of the sella turcica was reconstructed with cartilage and fibrin glue after autologous fatty tissue was packed into the cavity. Histological examination identified the excised membrane as arachnoid membrane without neoplastic features. The membrane contained no cylindrical epithelium or goblet cells. The patient's visual field showed slight improvement, and the results of endocrinological examination were nearly the same as before surgery, although the patient's headache disappeared. Neither diabetes insipidus nor any electrolytic abnormality was observed.

Postoperative MR imaging depicted the optic nerve in a part of the optic canal and the posterior optic tract but not around the optic chiasm because of adherence of the cyst membrane to the floor of the third ventricle (Fig. 2B). The patient was discharged on August 4, 2002. MR imaging performed 6 months later showed that the arachnoid membrane had descended into the sella turcica (Fig. 2C). The patient's visual defect persisted.

Discussion

In the present case, the diagnosis of giant intrasellar arachnoid cyst was based on adrenal insufficiency

Table 3 Preoperative consecutive adrenocorticotrophic hormone (ACTH) stress test

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
Urinary 17-OHCS (mg/day)	unknown	1.91	0.817	1.97	11	14.8
Serum cortisol (μg/dl)	unknown	unknown	<1.0	13	18	15.6
Urinary cortisol (μg/day)	unknown	unknown	unknown	24	131	190
Serum sodium (mEq/l)	140	131	131	136	144	143
Urinary volume (ml/day)	1007	1167	1644	3660	2465	1786
Urinary creatinine (g/day)	0.65	0.96	0.94	0.9	0.84	0.96

Intramuscular dose of ACTH-Z (1 mg) was administered on Day 3, Day 4, and Day 5. 17-OHCS: 17-hydroxycorticosteroids.

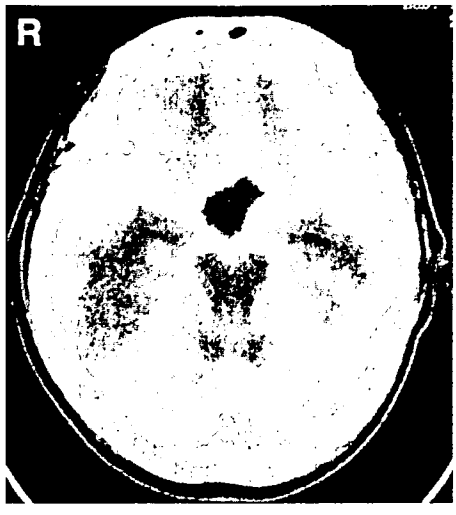

Fig. 1 Computed tomography cisternogram showing no influx into the intrasellar mass at 24 hours after injection of 8 ml of iotrolan (240 mg/ml) into the lumbar subarachnoid space.

and visual disturbance. Endocrinological examination showed that adrenal insufficiency was caused by hypothalamic dysfunction. Even after endonasal transsphenoidal resection of arachnoid membrane, the symptom did not change.

The clinical features of sellar cystic lesions such as craniopharyngioma, Rathke's cleft cyst, and intrasellar arachnoid cyst have been described.[16] Three of five patients with intrasellar arachnoid cyst had hypocortisolism, and one had hypogonadism. None required hormone replacement therapy preoperatively. Patients with intrasellar arachnoid cyst were more likely to present with neurological and ophthalmological deficits than with endocrinological dysfunction.[16] Thus, it is not surprising that only a few cases of associated endocrinological disorders have been reported.[12] However, a girl presented with suprasellar arachnoid cyst manifesting as hypothalamic pituitary dysfunction.[15] The arachnoid cyst was probably associated with a far more complex spectrum of endocrinological disorders than previously suspected.[12]

In our case, adrenal insufficiency, hypothyroidism, and hypogonadism were noted in preoperative endocrinological tests and the patient needed hormonal replacement. These abnormalities, especially adrenal insufficiency, were thought to be hypothalamic in origin, because the response of the adrenal gland to consecutive ACTH recovered, and the responses of the pituitary gland to CRH and thyroid hormone-releasing hormone were within normal limits. Unfortunately, the endocrinological abnormalities and visual defect in our patient did not improve after 1 month of post-surgical follow up.

Suprasellar arachnoid cyst might cause dysfunction of the ventromedial nucleus of the hypothalamus[1] and hypothalamic dysfunction is sometimes resolved by decompression of arachnoid cyst.[18] We consider that the lack of improvement in our patient's symptoms was possibly caused by some kind of inflammatory reaction resulting in the adherence of the cyst wall to the floor of the third ventricle, as seen during postoperative MR imaging. The detailed etiology of the inflammation was unknown. A number of patients with intrasellar arachnoid cyst have suffered pituitary gland dysfunction,[4,6,7,15,19,20] but few endocrinological details of the patients are available in most cases. Possibly the endocrinological abnormalities were of hypothalamic origin in some of these patients in whom pituitary function did not recover postoperatively. More detailed endocrinological challenge tests would have disclosed this factor. We recommend that detailed endocrinological tests should be carried out prior to surgery in all patients with sellar and suprasellar lesions.

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Fig. 2 **A: Preoperative T_1-weighted magnetic resonance (MR) image with gadolinium showing a large dumbbell-shaped intrasellar cystic mass. The signal intensity is slightly higher than that of the cerebrospinal fluid and there was no enhancement of the cyst wall or contents. B: Postoperative T_1-weighted MR image with gadolinium showing the optic nerve in a part of the optic canal and the posterior optic tract but not around the optic chiasm because of adherence of the cyst membrane to the floor of the third ventricle. C: T_1-weighted MR image with gadolinium performed 6 months after the surgery showing that the arachnoid membrane had descended into the sella turcica.**

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