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Metastatic Tumors in the Sellar and Parasellar Regions
: Clinical Review of Four Cases

Metastatic tumors in the sellar and parasellar regions are uncommon and rarely detected in clinical practice. We present four cases of sellar and parasellar metastatic tumors, which metastasized from distant organ in one case and extended directly from adjacent structures in three. Common presenting symptoms were cranial neuropathies, headache and facial pain. Invasion into the cavernous sinus was noted in all cases. We report rare cases of sellar and parasellar metastases. Also, we should consider the possibility of metastasis in these regions for patients who showed the above clinical presentations in systemic cancer patients. In extensive diseases, transient symptomatic relief could be obtained by direct surgical management, even in restricted degree.

Key Words: *Sellar Turcica; Neoplasm Metastasis; Brain Neoplasms; Cavernous Sinus*

Hyeong Joong Yi, Choong Hyun Kim, Koang Hum Bak, Jae Min Kim, Yong Ko, Suck Jun Oh

Department of Neurosurgery, School of Medicine, Hanyang University, Seoul, Korea

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Address for correspondence
Choong Hyun Kim, M.D.
Department of Neurosurgery, Hanyang University Kuri Hospital, 249-1 Gyomun-dong, Kuri 471-701, Korea
Tel: +82.346-560-2322, Fax: +82.346-560-2327
E-Mail: kch5024@email.hanyang.ac.kr

INTRODUCTION

Metastatic tumors in the sellar and parasellar regions are uncommon. They consist of less than 7% of all mass lesions in these areas (1). They are found incidentally in the autopsy or hypophysectomy to control intractable cancer pain due to rare clinical presentations. Although it is hard to find symptomatic lesions in the sellar and parasellar regions among patients with full blown systemic malignancies (2), some peculiar features, such as pituitary dysfunction and cranial neuropathies are comparable to other intracranial metastatic tumors (3). We report four cases of metastatic tumors in the sellar and parasellar regions with the results of therapeutic outcome, clinical and radiological findings.

MATERIALS AND METHODS

We reviewed four cases of metastatic tumors to the sellar and parasellar regions retrospectively. These cases were selected in thirty one patients with metastatic tumors among 433 patients with brain tumors who were operated in our medical center from January 1994 to December 1998. These sellar and parasellar metastatic tumors comprise 0.9% of all brain tumors.

Clinical and radiological assessment was made on the basis of medical records and neuroimaging studies.

CASE REPORTS

Case 1

A 33-year-old man was referred to our department for management of diplopia and dysesthetic facial pain which persisted for ten months. He had been diagnosed with nasopharyngeal carcinoma at otolaryngology department fifteen months ago. He had undertaken two times of radical neck explorations and received postoperative radiation therapy for six months. At referral, he showed external ophthalmoplegia and abnormal painful facial sensation on physical exmination.

On radiological examination, brain magnetic resonance imaging (MRI) showed partially-enhanced heterogeneous mass which occupied nasopharynx and extended to middle and posterior cranial fossae. Lesion also showed extensive bony destruction. Surgical debulking was performed in two-stage operations each toward sphenocavernous and cerebellopontine angle region, respectively. Following operations, he had received chemotherapy with 1-(4-amino-2-methyl-5-pyrimidinyl)-methyl-3-(2-chloroethyl)-3-nitrosourea (ACNU). About ten weeks afterward, he admitted to emergency room (ER) due to hemoptysis. Brain

MRI showed internal carotid artery (ICA) infiltration of carcinoma. Soon after, he died from uncontrollable bleeding.

Case 2

A 45-year-old woman presented with severe headache and tinnitus. She had been received postoperative adjuvant radiation therapy to adenocarcinoma of hard palate for nine months. On admission, she showed diplopia, lateral gaze paresis and facial weakness. Brain MRI showed relatively well-enhanced heterogeneous mass in middle and posterior cranial fossae. Operation was done for relieving mass effects in cavernous region. Surgical specimen was the same as that of hard palate. Because she refused to postoperative adjuvant radiotherapy, she discharged against medical advice. After about two years and one month, she came to ER for mental deterioration. She was stuporous for three days. She had been received gamma knife radiosurgery at another medical center to the remained mass. Subsequent brain computed tomogram (CT) showed ventricular enlargement with periventricular low density areas. Three days later, she died of generalized weakness and refusal of further neurosurgical intervention.

Case 3

A 61-year-old woman came to our department with complaints of blindness and ptosis of left eye. She had just completed a postoperative radiation therapy for adenocarcinoma of hard palate. Physical examination revealed a visual acuity of 10/200 for left eye, ptosis and sensory change on left side of face. Brain MRI showed well-enhanced mass which located in middle and posterior fossae (Fig. 1). Surgical decompression using orbitozygomatic and suboccipital approaches were performed on cavernous and cerebellopontine angle regions, respectively. Surgical specimen was confirmed as an adenocarcinoma (Fig. 2). Postoperative external beam radiation therapy with 7,000 rads was followed. Her vision recovered up to 40/200 for left eye, but ptosis did not. After one and a half year following diagnosis, she readmitted for generalized poor condition and total blindness of both eyes. Brain MRI and single photon computed tomogram (SPECT) showed extensive radiation necrosis. Pituitary deficits were remarkable, especially in thyroid and adrenocortical hormones. Hormonal replacement therapy and other medical support were done, but he died one week thereafter.

Case 4

A 54-year-old woman presented with complaints of sudden visual loss in both eyes for 4 days. Her vision deteriorated to the point that she could not count fingers from 1 meter away. Neurologic examination showed a bilateral optic atrophy with visual acuity of 10/400 for the right eye and 10/200 for the left eye. Light perception was not observed in the right eye. Four years ago, she had undertaken a radical mastectomy for adenocarcinoma of the right breast and received postoperative

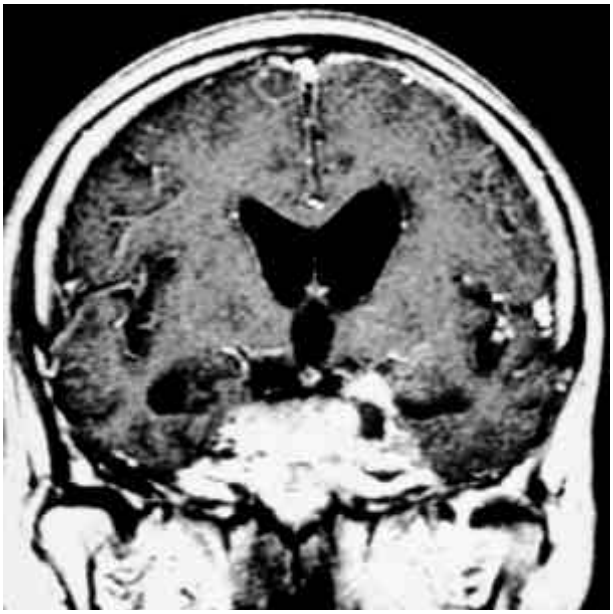

Fig. 1. Enhanced coronal MR image shows a heterogeneously enhanced mass which invades cavernous sinus on left side (Case 3).

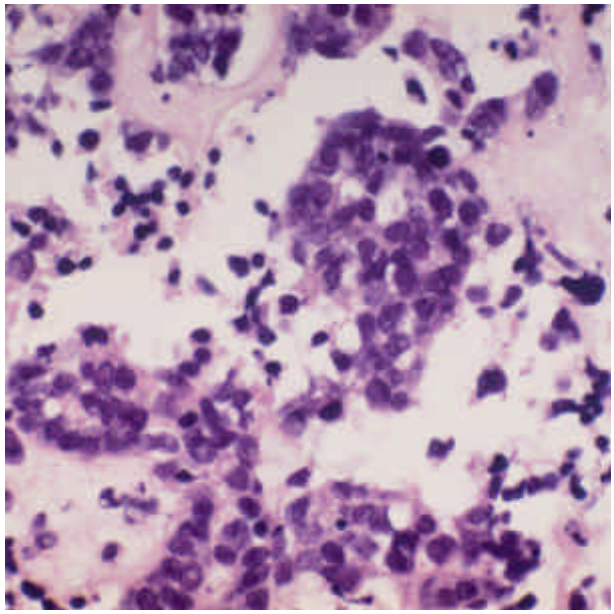

Fig. 2. Photomicrographs of surgical specimens. Glandular structures of ductal architecture admixed with necrotic foci in cystic background are noted (H&E, ×400) (Case 3).

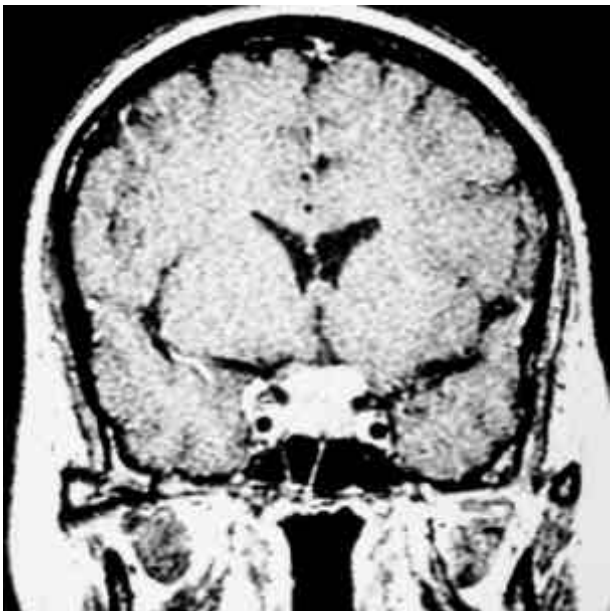

Fig. 3. Enhanced coronal MR image reveals a heterogeneously enhanced mass which compresses the optic chiasm (Case 4).

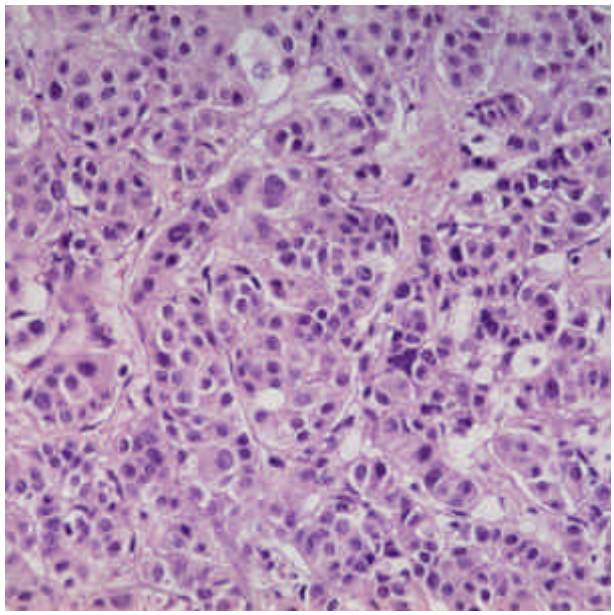

Fig. 4. Photomicrographs of surgical specimens. Glandular structures of ductal architecture and scattered mitoses with atypical cells are noted (H&E, ×400) (Case 4).

chemotherapy with radiation therapy for one and a half year. Before admission, she complained of intermittent generalized pain, including lower back pain, neck pain and headache along with generalized malaise. In radiologic studies, multiple osteolytic lesions were noted in whole spine. Brain MR imaging revealed a well-enhanced mass in the sellar and parasellar regions, encasing a right ICA and compressing an optic chiasm superiorly (Fig. 3). The patient underwent a surgical debulking through a transsphenoidal approach (TSA) with extension into the tuberculumn sellae. Pathological examination of the tumor specimen was consistent with ductal typed adenocarcinoma (Fig. 4). Because of her poor systemic condition, no additional adjuvant therapy was carried out. Transient diabetes insipidus (DI) and slight improvement of vision were seen in the immediate postoperative period, but after five weeks, she died due to systemic dissemination of tumor.

Postoperative improvement of vision was seen in case 3 and 4 and improvement of cranial neuropathy in case 1 and 3. Immediate postoperative pituitary deficits were seen in case 4 and delayed deterioration in case 3. Table 1 details some of the clinical data describing the 4 patients included in this study of the sellar and parasellar metastatic tumors.

DISCUSSION

The incidence of metastatic tumors around the sella

Table 1. Clinical summary in 4 patients with sellar and parasellar metastasis

Patient No.	Age (yr) /Sex	Presenting symptom	Duration of symptom	Primary lesion	Past history	Location	Operation (Approach)	Adjuvant treatment	Complications	Outcome
1	33/M	Diplopia Facial dysesthesia	10 mo	Nasopharyngeal Ca. (undifferentiated Ca.)	Operation postop. RTx	Sphenocavernous C-P angle	Subtemporal Suboccipital	CTx (ACNU)	Hemoptysis (ICA infiltration)	Death (10 wk after Dx)
2	45/F	Headache Diplopia Tinnitus	7 wk	Palatal Ca. (AdenoCa.)	Operation postop. RTx	Cavernous sinus C-P angle	Subtemporal	RTx (GKS)	Radiation necrosis Hydrocephalus	Death (121 wk after Dx)
3	61/F	Ptosis Visual disturbance (blindness, left) Facial dysesthesia	1 mo	Palatal Ca. (AdenoCa.)	Operation	Cavernous sinus C-P angle	Orbitozygomatic Suboccipital	RTx (7,000 rads)	Radiation necrosis	Death (78 wk after Dx)
4	54/F	Visual disturbance (blindness, both) Generalized pain	4 days	Breast Ca. (Ductal AdenoCa.)	Radical mastectomy postop. CTx & RTx	Suprasellar Cavernous sinus	Transsphenoidal	None	Transient D.I.	Death (5 wk after Dx)

M, male; F, female; mo, month; wk, week; Ca., carcinoma; RTx, radiation therapy; CTx, chemotherapy; C-P angle, cerebellopontine angle; GKS, gamma knife surgery; ICA, internal carotid artery; D.I., diabetes insipidus; Dx, diagnosis

turcica ranges from 1% to 26% of all mass lesions in these areas, including autopsy series or hypophysectomy series for pain control and an overall incidence of symptomatic tumors is 1-7% (1, 3-5). These are relatively frequent in autopsy series, but rare in neurosurgical practices as seen in our cases. The incidence is 0.9% of all brain tumors in our cases.

About 10% of patients with breast cancer had sellar metastases and 50% of metastatic tumors in the sellar region arose from primary tumors of the breast. The remaining tumors originated from lung (20%) mainly in male, and the other sources of metastases were gastrointestinal tract, prostate, melanoma, pancreas and so forth (4, 6, 7). They comprise about 1% of sellar mass lesions in a treated group by TSA (8). Our report predominantly shows a contiguous spread of neighboring malignancies rather than a distant metastasis, which contrasts a preexisting report (9). In a strict sense, virtual metastasis from a distant organ is short of evaluation. This means such a metastasis-prone malignancy work-ups are frequently overlooked.

Common signs and symptoms of sellar and parasellar metastatic diseases include headache, ocular symptoms, diabetes insipidus (DI) and anterior hypopituitarism. In a large series, the initial signs are summarized as follows: DI (70%), visual loss (20%), hypopituitarism (18%), and oculomotor palsies (12%) (3, 7). However, if the anterior pituitary dysfunction is severe, DI may be concealed by reduced mineralocorticoid function (8). The emergence of DI represents the preferential location of metastatic deposits within the posterior pituitary and infundibulum, indicating a direct blood supply from the systemic circulation to this lobe (3, 4, 6). So, cranial nerve palsies and DI are important findings that may differentiate an adenoma from a metastatic pituitary lesion. In recent studies, the most common presenting symptoms were headache (60-70%) and anterior hypopituitarism (50-60%), followed by visual field defect (50%), extraocular palsy (40%), and DI (30%) (1, 7). In our series, cranial neuropathies were also common symptoms. Compromise of visual acuity is by far the most common symptom in our cases along with other cranial nerve palsies. This reflects tumor growth patterns in our cases of contiguous spread, which laterally extends to the cavernous sinus. Interestingly, no preoperative endocrine dysfunction found in our cases was thought to be due to external compression rather than infiltration of pituitary parenchyme, as proved by radiological imaging studies.

Historical information that could suspect metastatic sellar and parasellar lesions rather than pituitary adenoma included rapid onset and progression of symptoms, such as DI and painful ophthalmoplegia, unsuccessful treatment with bromocriptine, increased age and a history of cancer (7). The adenomas frequently occur below 40 years of age, but the metastatic tumors occur over the age of 50 years (2). History alone, however, was not sufficient to recognize a metastatic tumors in these regions in our cases. Apoplectic manifestation, such as a sudden visual disturbance was easily detected, but slowly progressed symptoms, such as headache, diplopia and facial dysesthesia were neglected despite preexisting known malignancy. Therefore, clinical attention elapsed 10 months from symptom onset in one case. Occasionally, metastasis in the sellar region and within the pituitary may be the first presenting lesion in a patient not previously known to have a malignancy, even this may be the only central nervous system site of cancer (4, 5, 8, 9). Despite the above clues, sellar metastasis are rarely clinically detected except for DI for the following reasons: The majority of metastatic deposits causes no detectable enlargement of the gland, adenohypophyseal destruction must be nearly complete to produce clinical insufficiency, the malignancy widely disseminates in a patient with carcinomatosis and the overwhelming systemic complications of malignancy may mask symptoms of hypopituitarism. Therefore, survival is short (8, 10).

The hematogenous spread of distant malignancies most likely explains the majority of metastatic lesion, but as seen in our cases, nasopharyngeal tumors, a possible pathway of extension to the skull base, infiltrates the large cranial nerve foramina, which has direct access to the subarachnoid space and cavernous sinus (10).

According to the literature, there were no significant differences in survival times between the surgical and nonsurgical groups, and the extent of surgical resection or adjuvant therapy did not affect the survival times, but they did alter local tumor control and, subsequently resulted in an improved symptomatic outcome. Therefore, the role of direct treatment of sellar metastases should be focused on symptomatic relief and surgery may be considered only to preserve the visual function in particular cases even with extensive disease (5, 8). As is seen in our cases, such as pituitary apoplexy, aggressive treatment by transsphenoidal decompression of hemorrhagic or necrotic tissues could lead to a significant improvement in visual deficits, though it lasted transiently (11). Because of the abundant vascularity of the lesion and the high incidence of cavernous sinus involvement, total removal of the metastasis is unlikely. The mean survival is reported to be less than 1 year after diagnosis (4, 5), which is comparable to our results. The mean survival of our patients with metastatic tumors in the sellar and parasellar regions is 53.5 weeks, ranging from 5-121 weeks (12.5 months on average). This is comparable, or even superior to 9.4 months of surgically resected single brain metastasis series (9). This might be due to charac-

teristics of primary tumors in our cases.

According to our small experiences of sellar and parasellar metastases, these lesions had some common clinical manifestations, such as cranial neuropathies especially compromise of visual acuity, and facial dysesthesia, although there was no pituitary dysfunction in pretreatment period. Therefore, attention should be paid to emergence of such symptoms in known cancer patients and consider the possibility of metastasis in sellar and parasellar mass lesions for patients who showed the above clinical presentations. There were no differences in clinical manifestations, however, between one case of distant metastasis and three cases of direct spread in our report. This might render clinical decision making further perplexing and delaying. Furthermore, in extensive diseases, at least transient symptomatic relief could be obtained by direct surgical management.

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