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# Pituitary Metastasis: Lung Cancer Presenting as Bitemporal Hemianopsia with Diabetes Insipidus and Anterior Pituitary Deficiency

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# Pituitary Metastasis: Lung Cancer Presenting as Bitemporal Hemianopsia with Diabetes Insipidus and Anterior Pituitary Deficiency

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*Symptoms and signs of pituitary disease are rarely the first manifestations of malignancy originating in another organ. We present a case which exemplifies the key points that suggest a diagnosis of metastatic pituitary disease. Our patient's diagnosis was adenocarcinoma of the lung with a metastasis to the intrasellar and suprasellar regions, which caused diabetes insipidus, anterior pituitary deficiency, and visual field defects. The metastasis had a dumbbell appearance and extended from the sella turcica into the suprasellar region. Diabetes insipidus was the initial clinical manifestation of lung cancer in this patient. A metastasis to the pituitary should be suspected if diabetes insipidus is the initial manifestation of an intrasellar mass. (Henry Ford Hosp Med J 1987;35:185-7)*

Metastatic pituitary disease is rarely recognized clinically. Even when clinical suspicion is high, as in patients known to have cancer, tumor metastatic to the pituitary is usually recognized only at autopsy (1,2). Nevertheless, symptoms or signs of pituitary disease can be the first manifestation of malignancy originating in another organ (3-5). Such a case is presented, which exemplifies the key points that suggest a diagnosis of metastatic disease in the pituitary.

## Case Report

A 62-year-old man presented with a chief complaint of peripheral vision loss occurring over several days. He had had several weeks of mild, intermittent diplopia, but he did not seek medical care until the loss of peripheral vision began to interfere with his golf game.

On questioning, he revealed that six months ago his usual nocturia had increased to six to eight times per night with daytime polyuria and polydipsia of about two gallons of liquids per day. He had lost 20 pounds over the last six months, and for four months he had experienced fatigue, constipation, intolerance of the cold, dryness of the skin and hair, altered sense of taste, anosmia, and impaired mentation. Left-sided occipital headaches enduring several hours had been present mornings for the last two months. A two-year history of decreased libido had recently progressed to complete loss of libido with hot flushes and impotence.

His history was remarkable for a 35 pack/year habit of cigarette smoking. He had stopped smoking five years ago when chronic obstructive pulmonary disease with bronchospasm was diagnosed. One year ago, daily prednisone treatment was added to the other treatments for this disease.

Physical examination showed him to be well developed, well nourished, and in no acute distress. His vital signs were normal. His hair was fine, dry, and normally distributed. There was bilateral papilledema and diplopia on upward gaze. Goldmann visual field testing showed bilateral temporal field defects (right greater than left). His lung fields were clear with a prolonged expiratory phase, and no abnormal cardiac or abdominal signs were present. Lymphadenopathy was not present. The relaxation phase was delayed in the deep tendon reflexes, and the testicles were soft.

His complete blood count and automated chemistry profile were normal, except for a serum sodium of 145 mmol/L. The urine specific gravity was 1.004, and the urine osmolality was 220 mosm/kg. The serum thyroxine was 4.9 μg/dL, thyroid-stimulating hormone 1.2 μIU/mL, testosterone < 20 ng/dL, luteinizing hormone < 2.5 μIU/mL, and prolactin 19 ng/mL. Cortisol was not measured because of the chronic treatment with prednisone. Chest roentgenogram revealed a 4.0 cm opacity in the superior segment of the lingula, enlargement of the root of the left lung, and thickening of the soft tissues in the right paratracheal region. These findings suggested the presence of lung cancer in the lingula with metastases to the lung root and the right paratracheal area. Cranial computed tomography showed an enlarged sella turcica containing a dumbbell-shaped, enhancing mass extending into the suprasellar region to the foramen of Monro and compressing the optic chiasm (Figure).

A presumptive diagnosis was made of primary lung cancer with metastasis to the sellar and suprasellar regions causing diabetes insipidus, hypothalamic dysfunction, anterior pituitary deficiency, and visual field defects. He was treated with replacement therapy of DDAVP, thyroxine, and testosterone. Prednisone was continued. Bronchoscopic biopsy was positive for well-differentiated adenocarcinoma of the lung. He underwent frontal craniotomy to relieve the visual field defects, and histological examination of the pituitary mass revealed ade-

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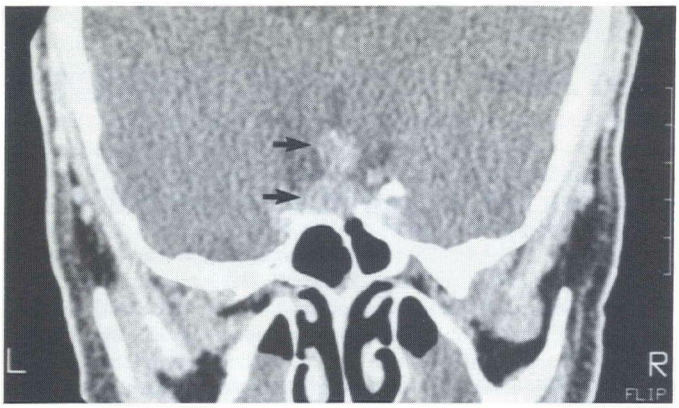


*Figure—Cranial computed tomography showing a dumbbell-shaped, enhancing mass (arrows) in the sella extending into the suprasellar region.*

nocarcinoma, similar to the lung neoplasm. Postoperatively, Goldmann visual fields showed resolution of the temporal field defects. He was subsequently treated with chemotherapy and whole brain radiation.

## Discussion

Interest in this case stems from the presentation of a symptomatic sellar mass as the initial manifestation of a previously unrecognized lung cancer. The autopsy incidence of metastatic pituitary disease varies depending on the series cited. Reports from unselected autopsies document as little as 0.14% to 1.0% involvement, while autopsy series on cancer patients have demonstrated up to a 28% incidence (1,2). Cancer of almost every type has been described as a source of metastases to the pituitary (6). Among the solid tumors, breast and lung cancer predominate. Metastases from lung cancer account for up to 60% of pituitary metastases among men (1,4,6), and breast cancer metastases account for up to 70% of pituitary metastases among women (4).

There is a predilection for pituitary metastases to occur in the neurohypophysis (1,3,5,7). A review of the literature by Max et al (3) revealed that of 178 reported cases of pituitary metastases, the neurohypophysis was involved alone in 52% and the adenohypophysis was involved alone in only 21%. The reason for this discrepancy in presentation is thought to be inherent in the blood supply to the pituitary gland. Arterial blood is supplied directly to the neurohypophysis by the inferior hypophyseal arteries. The blood supply to the anterior pituitary is predominantly postcapillary blood from the hypothalamus via the long portal veins. This would suggest that the higher frequency of metastases to the posterior pituitary than to the anterior pituitary is due to the predominantly portal (postcapillary) type of blood supply to the anterior pituitary. Supporting evidence for this hypothesis is provided by Goldman and Sapirstein's studies (8) in which radioactive microspheres injected into the carotid arteries of rats localized in the neurohypophysis and hypothalamus, and not in the adenohypophysis.

The anterior pituitary is involved in up to 67% of the cases of pituitary metastases evaluated at autopsy, but anterior pituitary dysfunction is rare (1,3,4). In reviewing 28 previously reported cases of symptomatic pituitary metastases in which sufficient endocrinological data were available, Max et al (3) found that only three of the patients had anterior pituitary deficiency. In 20 of 88 cases of pituitary metastases, Teears and Silverman (4) found discrete anterior pituitary infarcts which were postulated to have occurred secondary to neurohypophyseal involvement with metastatic disease compromising the blood supply to the adenohypophysis. None of the infarcts resulted in anterior pituitary dysfunction. The low frequency of anterior pituitary dysfunction in pituitary metastasis may be best explained by the fact that a large portion of the anterior pituitary must be destroyed before clinical hypopituitarism is evident (1,9,10).

The majority of cases of pituitary metastases recognized antemortem have diabetes insipidus (3). The presence of diabetes insipidus early in the course of metastasis to the pituitary is illustrated in Kimmel and O'Neill's series (6). In 11 of 25 patients, diabetes insipidus as a complication of metastatic disease was the initial symptom of the malignancy. That diabetes insipidus is a clue to the presence of metastasis is also shown by the same report. In 100 consecutive cases of diabetes insipidus at Mayo Clinic, 14% were secondary to metastatic disease, 27% were secondary to trauma, and 25% were idiopathic (6). Most cases of pituitary metastases are asymptomatic, however. Teears and Silverman (4) found that only 7% of 88 patients with pituitary metastases had diabetes insipidus.

Because of the origin of antidiuretic hormone in the supraoptic and paraventricular nuclei, hypothalamic dysfunction must be present in patients with persistent diabetes insipidus. In our patient, symptoms of impaired smell, taste, libido, and mentation were present in addition to diabetes insipidus. These symptoms indicate effects of the metastasis beyond its immediate sellar-suprasellar location.

The most common cause of an intrasellar mass is a pituitary adenoma in patients with or without malignancy (3). Adenomas usually manifest with anterior pituitary dysfunction or visual field defects (3). Diabetes insipidus is typically a late finding (11). In metastatic disease to the pituitary, diabetes insipidus is usually the first noted abnormality of pituitary function with anterior pituitary deficiency occurring after the onset of diabetes insipidus (3,4), as in the present case.

Treatment of an intrasellar mass in cancer patients depends upon the clinical picture. If it is that of a benign mass and survival with the cancer is expected to be six or more months, treatment should be the same as for patients without cancer. If, however, the clinical picture is that of a pituitary metastasis, treatment should be irradiation to the pituitary. Surgical intervention would be indicated if the discovery of metastasis would influence therapy of the primary cancer or if surgery would be sight-saving.

If surgical intervention is indicated, the transfrontal or the transsphenoidal route may be used for tissue diagnosis and optic apparatus decompression. Although in our patient the dumbbell shape of the lesion might not have lent itself to thorough decompression through the sphenoid, either method can be effective in decompressing the optic apparatus. The transsphenoidal route is safer, easier for the patient to tolerate, and has the theoretical advantage of not opening the intracranial space to tumor

dissemination, as may be the case if transfrontal craniotomy is employed.

In conclusion, pituitary metastasis is a frequent complication of malignancy but is infrequently recognized antemortem. When clinically evident, it will likely present as diabetes insipidus. Symptoms or signs of anterior pituitary deficiency and hypothalamic dysfunction may subsequently occur. Patients may present with pituitary metastasis as the initial manifestation of malignancy. An intrasellar mass which presents with diabetes insipidus or anterior pituitary deficiency preceded by diabetes insipidus should suggest the presence of a metastasis to the pituitary. Appropriate treatment of a pituitary metastasis is usually irradiation. Surgical treatment is indicated if definite demonstration of metastatic disease would affect therapy of the primary tumor or if surgery would be sight-saving.

## References


1. Kovacs K. Metastatic cancer of the pituitary gland. Oncology 1973; 27:533-42.

2. Roessmann U, Kaufman B, Friede RL. Metastatic lesions in the sella turcica and pituitary gland. Cancer 1970;25:478-80.

3. Max MB, Deck MDF, Rottenberg DA. Pituitary metastasis: Incidence in cancer patients and clinical differentiation from pituitary adenoma. Neurology 1981;31:998-1002.

4. Teears RJ, Silverman EM. Clinicopathologic review of 88 cases of carcinoma metastatic to the pituitary gland. Cancer 1975;36:216-20.

5. Kistler M, Pribram HW. Metastatic disease of the sella turcica. AJR 1975;123:13-21.

6. Kimmel DW, O'Neill BP. Systemic cancer presenting as diabetes insipidus: Clinical and radiographic features of 11 patients with a review of metastatic-induced diabetes insipidus. Cancer 1983;52:2355-8.

7. Piney A, Coates I. Metastatic carcinoma of the pituitary gland and diabetes insipidus. J Path & Bacteriol 1924;27:211-5.

8. Goldman H, Sapirstein L. Nature of the hypophysial supply in the rat. Endocrinology 1962;71:857.

9. Sheehan HL. Atypical hypopituitarism. Proc Roy Soc Med 1961;54:43-8.

10. Sheehan HL, Stanfield JP. The pathogenesis of postpartum necrosis of the anterior lobe of the pituitary gland. Acta Endocr (Coph) 1961;37:479-510.

11. Hollenhorst RW, Younge BR. Ocular manifestations produced by adenomas of the pituitary gland—analysis of 1000 cases. In: Kohler PO, Ross GT, eds. Diagnosis and treatment of pituitary tumors. Amsterdam: Excerpta Medica, 1973:53.