Case report

Sid, 68, presented with marked lethargy, fatigue, anorexia, and frontal headaches with nausea. These symptoms had been ongoing for the last eight weeks. His past history includes non-small cell lung cancer and hypertension in remission.

Sid had noticed a progressive decline in potency and libido for the last year, and he had been experiencing frontal headaches and diplopia lately. His medical examination was significant for an alert patient with lower than baseline blood pressure of 94/70 mmHg without an orthostatic drop (despite being off his usual anti-hypertensives). Sid had bitemporal visual field defects by confrontation and mild gynecomastia, but normal general androgenization.

Investigations

- Prolactin 8.3 ng/mL (normal < 17)
- Thyroid-stimulating hormone 0.06 mIU/L (normal 0.40 to 5.50)
- Free thyroxine 10.1 pmol/L (normal 11 to 23)
- Total testosterone < 1.00 mmol/L (normal 8.40-27.70)
- Inappropriately low gonadotropins,
- Growth hormone < 0.05 µg/L (normal 0.06 to 5.00),
- Sometomedin-C 36 ug/L (96 to 243),
- Adrenocorticotropic hormone < 2.2 pmol/L (normal 2.2 to 10.2)
- Sodium 140 mmol/L (normal 136 to 145)
- Potassium 5.1 mmol/L (3.5 to 5)

Sid was discovered to have a 1.8 x 2.4 x1.6 cm pituitary macroadenoma (Figure 1) on magnetic resonance imaging of pituitary with suprasellar extension, optic chiasm compromise, and cerebellar metastasis on workup.

Obviously, labs and imaging studies were compatible with nonfunctional macroadenoma with panhypopituitarism (cosyntropin test revealed subnormal response also). Steroids were instituted immediately. He was started on multiple hormone replacement (hydrocortisone first then levothyroxine sodium tablets, intramuscular testosterone, and 1-deamino-8-D-arginine vasopressin) after biochemical confirmation of anterior and posterior hypopituitarism, with marked improvement in his symptoms.

A successful debulking transphenoidal surgery was undertaken. A pathologic report was consistent with metastatic adenocarcinoma of bronchogenic origin. Figure 2 shows a high-power microscopic view of a section from the tissue of the pituitary mass showing adenocarcinoma. A cluster of abnormal glandular lumina borders the areas of necrosis.

Discussion

Lung cancer is a common disease mainly among older patients. An advanced process can cause metastases to various organs. Pituitary metastases are rare, but represent an important differential
The possibility of a secondary location must always be considered in a differential diagnosis when treating an intrasellar lesion in a patient with a documented primary malignancy. Hypopituitarism, due to pituitary metastasis, is a rare complication of lung cancer. The overall reported incidence of pituitary metastasis is 1% to 6%.\(^1\)\(^2\) It may be the initial presentation of an unknown primary malignancy.\(^3\) Breast cancer is the most common malignancy to metastasize to pituitary in females. Amongst the same population, adenocarcinoma of the cervix represents another potential source.\(^4\) A clinical triad of headache, extraocular nerve palsy, and diabetes insipidus are highly suggestive of a sellar lesion. Diabetes insipidus is an aftermath of interruption of hypophyseal system due to posterior pituitary metastases.\(^5\)\(^6\)

Pituitary apoplexy is an uncommon clinical presentation with visual deficits, headache, ophthalmoplegia, and altered mental status caused by sudden hemorrhage or infarction of the pituitary gland.\(^7\) Pituitary apoplexy has been reported following metastasis of bronchogenic adenocarcino-

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ma to a pre-existing prolactinoma. Pituitary apoplexy is an emergent situation and requires prudent use of steroids even before imaging confirmation of diagnosis. Surgical decompression, especially in the presence of visual compromise, is mandatory to enable apoplexy or macroadenoma to ameliorate of disabling symptoms, including painful ophthalmoplegia and visual field deficits.

Case Revisited

Sid underwent immediate debulking transphenoidal surgery for this sellar mass arising due to lung cancer metastasis. He remained on maintenance hormones for secondary hypogonadism (with osteoporosis), central hypothyroidism, hypoadrenalism, and diabetes insipidus with resolution of most of his symptoms. Unfortunately, he had progressive loss of his left eye visual acuity and later had complete loss of vision. Growth hormone deficiency was not checked due to the baseline history of cancer. Unfortunately, he expired in palliative care seven and half months after the above diagnosis, due to metastatic lung cancer.

References