



Invasive Giant Prolactinoma

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Key words

- Dopamine
- Hormonal
- Pituitary
- Prolactinoma

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CASE DESCRIPTION

A 64-year-old man presented with an 8-year history of decreased libido, impotence, and blurred vision. The neurologic examination revealed a mild left facial weakness (House-Brackman II). Visual acuity was decreased bilaterally (20/60). A right homonymous hemianopia was noted. There were no other cranial nerve palsies.

Laboratory investigation of serum prolactin was 7896 ng/mL (reference range: 2–20 ng/mL), testosterone was 0.35 nmol/L (reference range: 4.41–35.38 nmol/L), free T4 was 6.3 pmol/L (reference range: 9–19 pmol/L), and adrenocorticotrophic hormone was 1.97 ng/L (reference range: 7.2–63.3 ng/L). The remaining pituitary hormonal values were within normal limits. Magnetic resonance imaging of the brain showed a giant sellar/suprasellar lesion with extension to the cavernous sinus, left Meckel cave, and third ventricle, compressing on the brainstem and extending to the cerebellopontine angle (Figure 1).

A diagnosis of panhypopituitarism secondary to a giant macroprolactinoma was made. The patient was offered surgery because he had progressive visual loss.

A 64-year-old man presented with an 8-year history of decreased libido, impotence, and blurred vision. The neurologic examination revealed a mild left facial weakness, visual acuity of (20/60), and right homonymous hemianopia. Laboratory investigation of serum prolactin was 7896 ng/mL. Testosterone, free T4, and adrenocorticotrophic hormone were all decreased. Brain magnetic resonance imaging showed a giant sellar/suprasellar lesion, compressing on the brainstem and extending to the cerebellopontine angle. A diagnosis of panhypopituitarism secondary to a giant macroprolactinoma was made. The patient was started on hormonal replacement therapy and underwent craniotomy and tumor resection. The histopathologic findings were compatible with prolactinoma. Invasive giant prolactinomas are defined as 1) tumor size of >4 cm in diameter; 2) serum prolactin of >1000 ng/mL; and 3) mass effect or hyperprolactinemia-induced symptomatology. The management of invasive giant prolactinoma commonly comprises a multimodal approach of both medical treatment and surgical intervention.

However, he refused any surgical intervention and was started on hormonal replacement including selective dopamine agonist therapy. His prolactin level after 6 months on medical therapy decreased significantly to 369.655 ng/mL (reference range: 2–20 ng/mL).

A year following the diagnosis, the patient developed obstructive hydrocephalus secondary to the lesion. He underwent external ventricular drain insertion, followed by a left-sided craniotomy and tumor resection.

The histopathologic findings were compatible with prolactinoma. The neoplastic cells were immunopositive for prolactin (polyclonal, while immunonegative for adrenocorticotrophic hormone, thyroid-stimulating hormone, growth hormone, follicle-stimulating hormone, and luteinizing hormone. The Ki-67 proliferation index was 1%–2%.

The patient was discharged with periodic clinicoradiologic follow-up. His visual acuity remained unchanged. The patient was kept on hormonal replacement therapy.

DISCUSSION

Invasive giant prolactinomas are defined as 1) tumor size of >4 cm in diameter; 2)

serum prolactin of >1000 ng/mL; and 3) mass effect or hyperprolactinemia-induced symptomatology.^{1–3} They are frequently diagnosed in males more than females (male-to-female ratio: 9:1).^{2–4}

According to the current World Health Organization classifications of central nervous system tumors, prolactinomas are categorized under PITr-lineage tumors.⁵ They are interchangeably known as lactotroph adenomas/tumors.⁵ PITr-lineage tumors are the most complex group within pituitary neuroendocrine cell tumors.⁵

Invasive giant prolactinomas are extremely rare and account for 1%–5% of all prolactinomas.⁴ Management of invasive giant prolactinoma commonly comprises a multimodal approach of both medical treatment and surgical intervention.^{2,6} The current article presents the clinical and radiologic appearance of a patient with invasive giant prolactinoma.

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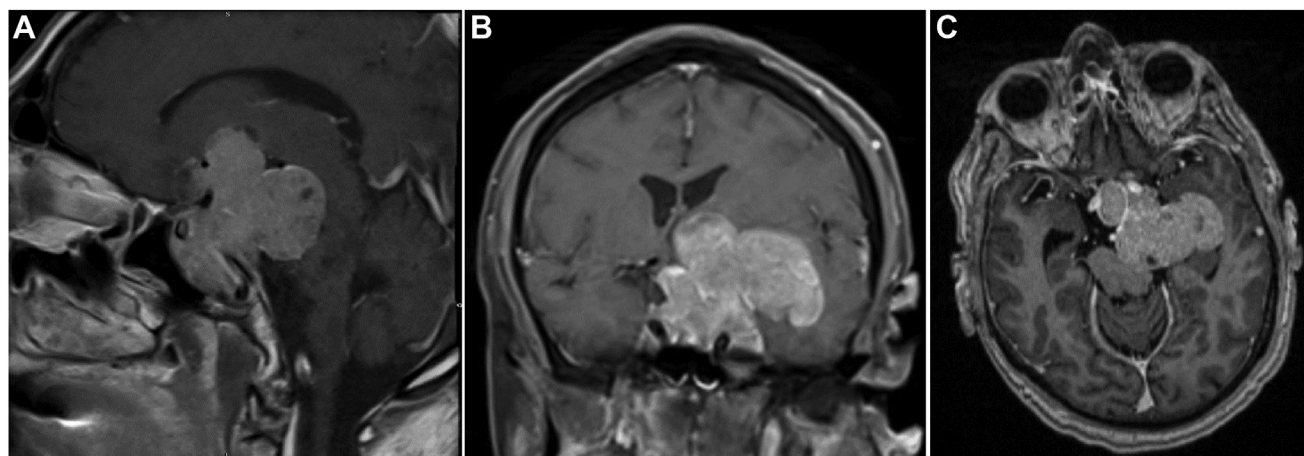


Figure 1. (A–C) Sagittal, coronal, and axial brain magnetic resonance imaging with contrast demonstrating a giant sellar and suprasellar lesion. It is well defined, lobulated in shape, and approximately $5.8 \times 6 \times 5.5$ cm in anteroposterior, transverse, and craniocaudal diameters, respectively. There is associated significant sellar expansion. The pituitary gland is indistinguishable from the lesion. The lesion is extending to and distending the left cavernous sinus, Meckel cave, and left middle cranial fossa with small extension to the right cavernous sinus. There is also extension to the

posterior fossa to the prepontine cistern on the left side, as well as smaller extension to the anterior cranial fossa along the dorsum sellae on the left side. It encases both internal carotid arteries and contains areas of calcification and hemorrhagic components. The cisternal segments of the optic nerves and most of the optic chiasm are engulfed by the lesion and not clearly visualized. The lesion is causing significant mass effect on the hypothalamus, third ventricle, frontal horn of the left lateral ventricle, left temporal lobe midbrain, and pons.

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