

Giant Prolactinoma Presenting as Stroke: An Unusual Case of Facial Paralysis and Hemiparesis

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Abstract

Giant prolactinomas are rare pituitary tumors, usually responsible for compressive symptoms, but they may exceptionally present with neurological deficits mimicking an acute cerebrovascular event.

We report the case of an 88-year-old man admitted for right facial palsy associated with right hemiparesis, which prompted radiological investigations to rule out an ischemic stroke. Hypothalamic-pituitary magnetic resonance imaging (MRI) revealed a large central sellar mass suggestive of an invasive macroadenoma measuring 30 × 28 × 44 mm, with suprasellar extension and involvement of the third ventricle, resulting in active biventricular hydrocephalus. Serum prolactin levels were markedly elevated (600 ng/mL). Visual field examination demonstrated a right homonymous hemianopia.

Treatment with cabergoline (0.5 mg) was initiated, leading to significant clinical improvement within a few months. After twelve months of therapy, tumor volume had decreased by more than 30%, and serum prolactin levels were reduced by 96%.

This case highlights an unusual neurological presentation of a giant prolactinoma, occurring in the absence of both cerebrovascular accident and pituitary apoplexy.

Introduction

Giant prolactinomas are pituitary adenomas that secrete prolactin, defined by a tumor diameter greater than 40 mm, representing a rare but clinically significant entity. They are more frequently observed in men and are typically associated with markedly elevated prolactin levels, often exceeding 1,000 ng/mL [1,2].

Clinical manifestations are mainly driven by the mass effect of the tumor, including headaches and visual disturbances related to optic chiasm compression, as well as features of hyperprolactinemia-induced hypogonadotropic hypogonadism, such as infertility and menstrual disturbances in women, or gynecomastia and decreased libido in men [3].

Dopamine agonists, particularly bromocriptine and cabergoline, represent the first-line treatment and generally lead to a significant reduction in both tumor volume and prolactin levels [4]. Neurological complications secondary to local tumor extension have been reported, especially oculomotor cranial nerve

palsies (III, IV, and VI), whereas facial nerve palsy associated with hemiparesis mimicking an acute cerebrovascular event remains exceptional and diagnostically misleading [5].

Case Presentation

We describe the unusual case of an elderly man presenting with facial paralysis and hemiparesis, initially suggestive of an acute ischemic stroke. Paraclinical investigations revealed a large sellar lesion, and the diagnosis of a giant macroprolactinoma was subsequently established in the setting of marked hyperprolactinemia. Treatment with cabergoline was initiated, leading to a gradual improvement in neurological deficits over the following months.

An 88-year-old man with no significant past medical history presented with right-sided hemiparesis and facial paralysis. On clinical examination, he was conscious and oriented, with central facial palsy and right-sided motor weakness, without sensory impairment. Visual field examination revealed temporal hemianopsia, and fundoscopic examination showed bilateral optic disc cupping. Magnetic resonance imaging (MRI) of the hypothalamic-pituitary region demonstrated a large invasive sellar mass measuring $30 \times 28 \times 44$ mm, consistent with a giant macroadenoma, associated with biventricular hydrocephalus and compression of the optic chiasm (Figure 1). The lesion was classified as Knosp grade 4 on the left side and grade 3b on the right. Hormonal evaluation revealed significant hyperprolactinemia, while the remaining pituitary hormone levels were within normal ranges. A diagnosis of giant prolactinoma was therefore established.

The patient was treated with cabergoline at a dose of 1 mg per week. After one month of treatment, serum prolactin levels decreased by 78%. Over the following months, partial improvement in neurological symptoms and visual field deficits was observed. Follow-up MRI performed 12 months later demonstrated a 30% reduction in tumor size (Figure 2), with persistent hydrocephalus.

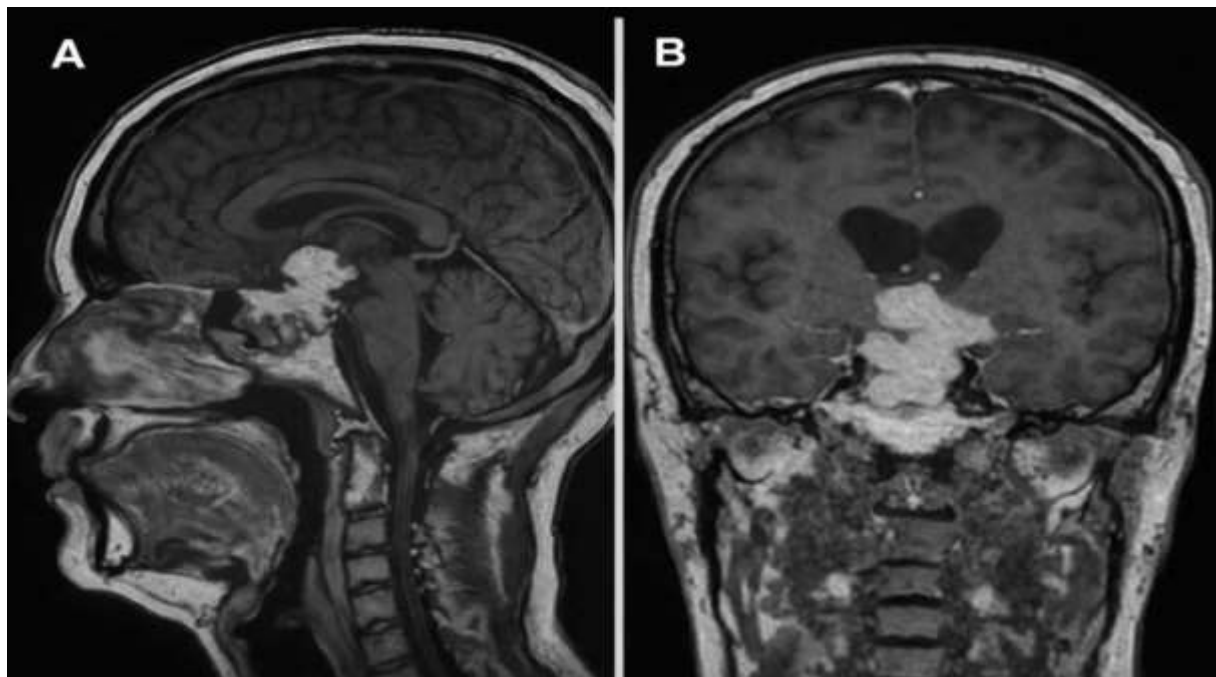


Figure 1: Hypothalamic–pituitary MRI showing an invasive macroprolactinoma, classified as Knosp grade 4 on the left side and grade 3b on the right side, measuring $30 \times 28 \times 44$ mm in diameter. Sagittal and coronal views.

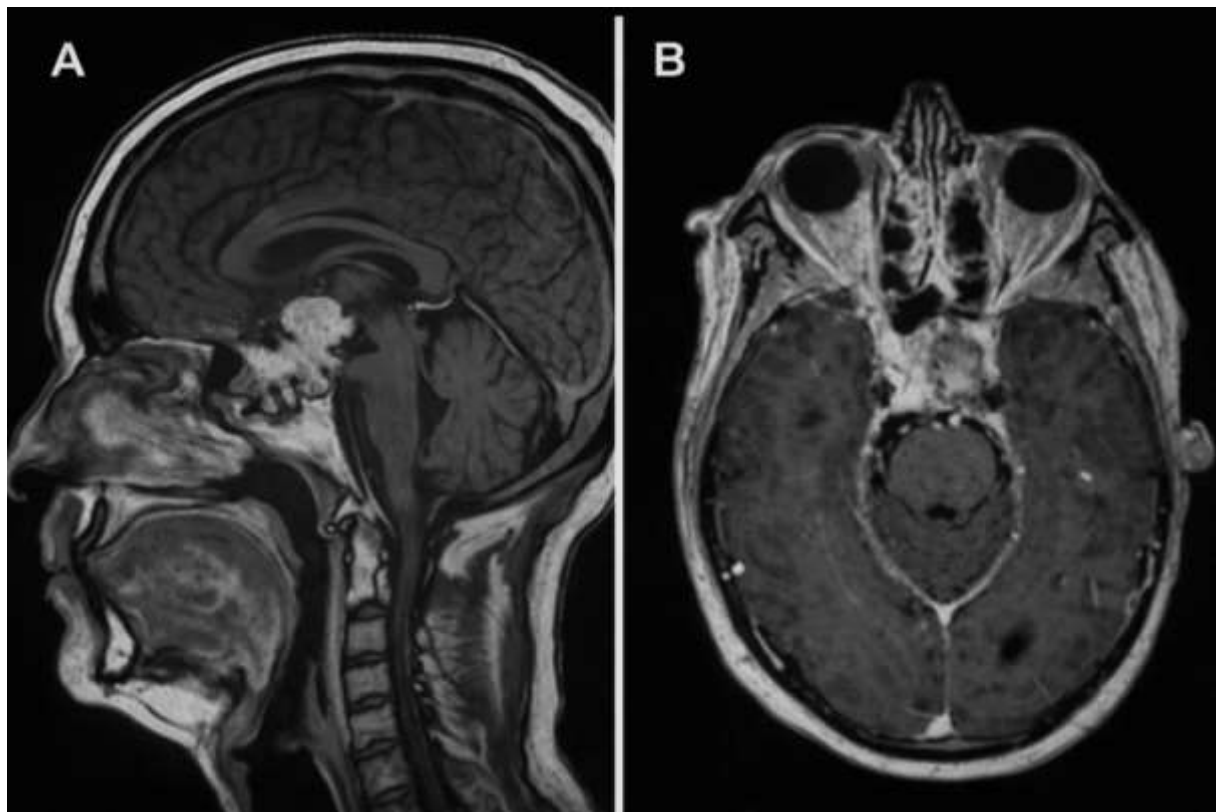


Figure 2: Hypothalamic–pituitary MRI showing an invasive macroprolactinoma, classified as Knosp grade 3a, measuring 30 × 28 × 44 mm in diameter.

Discussion

Giant prolactinomas are rare pituitary tumors, defined by a diameter exceeding 40 mm, and are most often associated with marked hyperprolactinemia. Their management is challenging because of their large size, invasive behavior, and the wide spectrum of clinical manifestations they induce. The mass effect represents the main pathophysiological mechanism, leading to compression of adjacent structures, particularly the optic chiasm. The most frequently reported symptoms are visual disturbances (54.9%) and headaches (42.3%) [1,2].

Endocrine disorders are also common, especially hypogonadotropic hypogonadism, which in men manifests as decreased libido and erectile dysfunction. Beyond these classical features, giant prolactinomas may be associated with rarer neurological manifestations, including cranial nerve palsies, epileptic seizures, cognitive decline, or psychiatric symptoms [1,3].

Cranial nerve III, IV, V, and VI palsies have been described in association with cavernous sinus invasion [1]. In contrast, involvement of the facial nerve (cranial nerve VII) remains exceptional in this context. Hemiplegia is also rarely reported and usually occurs in the setting of pituitary apoplexy or an associated cerebrovascular accident [3]. In our patient, the combination of facial paralysis and hemiplegia appeared to be related to the tumor's mass effect, with compression of motor pathways and corticobulbar fibers, in the absence of clinical or radiological signs of pituitary apoplexy or stroke. The progressive improvement of these neurological deficits following initiation of cabergoline therapy supports this hypothesis.

Giant prolactinomas are generally associated with very high prolactin levels. However, no consistent correlation has been demonstrated between serum prolactin concentration and tumor size [3]. Dopamine

agonists, particularly bromocriptine and cabergoline, represent the first-line treatment because of their proven efficacy and favorable safety profile. Cabergoline, a long-acting dopamine D2 receptor agonist, is preferred due to its superior effectiveness in normalizing prolactin levels and reducing tumor volume [2,4]. Surgery is reserved for cases requiring rapid decompression of the optic chiasm in the presence of severe visual impairment or for tumors resistant to medical therapy [2].

In our case, surgery was contraindicated because of the patient's advanced age. Cabergoline therapy was therefore initiated at a dose of 1 mg per week, under close clinical, biochemical, and radiological monitoring. This cautious approach was justified by the theoretical risk of ischemic events reported in some patients with large tumors or rapid tumor shrinkage under dopamine agonist therapy [5]. After 12 months of treatment, serum prolactin levels had decreased by 96%, and follow-up MRI demonstrated significant tumor regression without associated ischemic complications.

Overall, giant prolactinomas generally respond favorably to dopamine agonists, with rapid improvement of mass effect-related symptoms within days and progressive regression of other clinical manifestations over several months, often preceding complete normalization of prolactin levels [2,4]. Recovery of gonadal function has been reported in approximately 67% to 80% of treated men. Nevertheless, cabergoline dosing must be individualized to optimize efficacy while minimizing adverse effects.

In resistant forms, prolactin levels may remain elevated despite high doses of cabergoline or bromocriptine. Patients with extremely high baseline prolactin levels may sometimes exhibit a more pronounced biochemical response, although this is not consistently correlated with tumor volume reduction [3]. In our study, a rapid and marked decrease in prolactin levels was observed within the first month of treatment. However, tumor volume reduction was more modest (30% at 12 months) compared with the 60-80% reductions reported in the literature, leading to maintenance of the initial cabergoline dose because of the potential risk of adverse effects.

This case is remarkable for the patient's advanced age, the rare association of facial paralysis and hemiplegia, and the absence of pituitary apoplexy, a complication described in some aggressive forms of giant prolactinomas [3]. The resolution of neurological symptoms with medical therapy alone highlights the effectiveness of dopamine agonists, even in atypical clinical presentations.

Giant prolactinomas require long-term follow-up to monitor tumor evolution, hormonal function, visual fields, and the development of potential complications. A substantial proportion of patients with very large or aggressive tumors fail to achieve complete normalization of prolactin levels despite often significant clinical improvement [4,5]. In our case, the patient experienced marked clinical improvement accompanied by a progressive decline in prolactin levels, justifying continued clinical, hormonal, and radiological surveillance.

In aggressive or treatment-resistant cases, alternative therapies such as pasireotide or temozolomide have shown promising results [6]. Genetic testing may be considered in the presence of suggestive family history or suspicion of an underlying hereditary syndrome.

Discontinuation of dopamine agonist therapy is generally discouraged in giant prolactinomas due to the high risk of recurrence. The main predictors of recurrence are initial tumor size and baseline prolactin levels. Cabergoline dose adjustment must be performed cautiously, as rapid tumor shrinkage may lead to rare but serious complications, such as cerebrospinal fluid rhinorrhea or ischemic events related to bone erosion. Finally, long-term cabergoline therapy requires regular echocardiographic monitoring to screen for potential valvular heart disease [5].

Conclusions

Giant prolactinomas may exceptionally present with acute neurological deficits mimicking a cerebrovascular accident. This case highlights the importance of considering pituitary pathology in the differential diagnosis of sudden facial palsy and hemiparesis, even in the absence of pituitary apoplexy. Treatment with cabergoline resulted in significant biochemical and radiological improvement, confirming the effectiveness of dopamine agonists even in atypical clinical presentations. Early diagnosis and appropriate management are essential to prevent irreversible complications and to optimize patient prognosis.

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