

Delayed Diagnosis of a Giant Pituitary Adenoma with Irreversible Blindness and Ischemic Stroke

Zara Martínez¹ and Gabriela Mintegui^{2*} 

¹Assistant Professor, Academic Unit of Endocrinology and Metabolism, School of Medicine, Universidad de la República. Montevideo, Uruguay

²Associate Professor, Academic Unit of Endocrinology and Metabolism, School of Medicine, Universidad de la República. Montevideo, Uruguay

Citation: Martinez Z, Mintegui G. Delayed Diagnosis of a Giant Pituitary Adenoma with Irreversible Blindness and Ischemic Stroke. *Medi Clin Case Rep J* 2026;4(2):1740-1742. DOI: doi.org/10.51219/MCCRJ/Gabriela-Mintegui/473

Received: 27 May, 2026; **Accepted:** 29 May, 2026; **Published:** 01 June, 2026

***Corresponding author:** Associate Professor, Gabriela Mintegui, Academic Unit of Endocrinology and Metabolism, School of Medicine, Universidad de la República. Montevideo, Uruguay, Email: gabyming2@gmail.com

Copyright: © 2026 Mintegui G, et al., This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

ABSTRACT

Giant pituitary adenomas are uncommon tumors that may cause severe neurological complications due to mass effect on adjacent structures. We report the case of an 83-year-old institutionalized woman with a five-year history of progressive bilateral blindness without previous etiological evaluation. She presented with a 48-hour history of cognitive decline, left hemiparesis, and facial paralysis.

Brain computed tomography revealed a right frontal cortico-subcortical hypodense lesion in the anterior cerebral artery territory, consistent with acute-subacute ischemic stroke. In addition, a large sellar and suprasellar mass measuring 58 × 46 × 43 mm was identified, with parasellar extension, bone erosion, and close relationship to adjacent vascular structures, including the cavernous segments of both internal carotid arteries.

The lesion was interpreted as a giant pituitary adenoma with optic chiasm compression causing irreversible blindness and possible association with ischemic cerebrovascular disease through vascular compression or hemodynamic compromise.

Giant pituitary adenomas may lead to irreversible visual impairment and, rarely, ischemic stroke due to compression or displacement of intracranial arteries. This case highlights the severe consequences of delayed diagnosis of a potentially treatable condition and underscores the importance of early evaluation of progressive visual symptoms, even in elderly and institutionalized patients, to prevent irreversible neurological sequelae.

Keywords: Giant pituitary adenoma; Blindness; Ischemic stroke; Optic chiasm; Pituitary tumors

Introduction

Pituitary adenomas are benign tumors arising from the anterior pituitary gland and account for approximately 10-15% of intracranial neoplasms^{1,2}. Giant pituitary adenomas, defined as tumors measuring 4 cm or more in diameter, represent an uncommon subgroup associated with increased morbidity due to their expansive growth and involvement of adjacent structures, particularly the optic chiasm and intracranial vascular structures.

Compared with smaller macroadenomas, giant pituitary adenomas more frequently exhibit suprasellar and parasellar extension, cavernous sinus invasion, and compression of critical neurovascular structures. Clinically, they usually present with symptoms related to mass effect, especially progressive visual impairment and neurological deficits, whereas endocrine manifestations may be subtle or absent, particularly in nonfunctioning tumors^{3,4}.

Clinical manifestations range from incidental findings to severe hormonal dysfunction, headache, and progressive visual loss. Early diagnosis is essential because prolonged tumor growth without treatment may result in permanent neurological sequelae.

Case Presentation

An 83-year-old institutionalized woman with a history of arterial hypertension presented with progressive bilateral blindness over a five-year period without previous etiological evaluation or medical follow-up.

She was admitted with a 48-hour history of cognitive deterioration, left hemiparesis, and left facial palsy.

Brain computed tomography revealed a right frontal cortico-subcortical hypodense lesion within the anterior cerebral artery territory, consistent with acute-subacute ischemic stroke. In addition, a large solid sellar mass with suprasellar and parasellar extension measuring 58 × 46 × 43 mm was identified. The lesion caused erosion of the sella turcica and sphenoid sinus and showed close relationship to the cavernous segments of both internal carotid arteries and the anterior cerebral arteries.

A diagnosis of giant pituitary adenoma was considered, with optic chiasm compression explaining the irreversible blindness and possible association with the ischemic cerebrovascular event. No previous imaging studies or endocrine evaluation were available at the time of diagnosis (**Figure 1**).



Figure 1: Axial non-contrast brain computed tomography demonstrating a large heterogeneous sellar and suprasellar mass centered in the pituitary region, with expansive features and extension into adjacent structures, consistent with a giant pituitary adenoma

Discussion

Giant pituitary adenomas are rare tumors characterized by aggressive expansive growth, frequent suprasellar extension, cavernous sinus invasion, and compression of adjacent neurovascular structures. Visual impairment is one of the most common manifestations at diagnosis because of chronic optic chiasm compression⁵.

Visual loss is typically progressive and may be reversible when diagnosed early. However, prolonged compression can lead to permanent blindness, as observed in the present case. The slow-growing nature of these tumors may allow progressive patient adaptation⁶, contributing to delayed medical consultation and diagnosis, especially in vulnerable populations such as institutionalized elderly patients.

Vascular involvement is uncommon but clinically significant. Proposed mechanisms include direct arterial compression, vascular displacement, and secondary hemodynamic alterations caused by tumor mass effect. In this patient, ischemic stroke in the anterior cerebral artery territory suggests a possible association with vascular compromise secondary to the giant sellar lesion, although this presentation remains exceptionally rare⁷.

From an endocrinological perspective, giant pituitary adenomas may be functioning or nonfunctioning, with nonfunctioning tumors being more common among large lesions. The absence of overt endocrine manifestations may contribute to delayed diagnosis, with neurological and compressive symptoms predominating at presentation.

Management of giant pituitary adenomas requires a multidisciplinary approach. Surgical resection is generally considered the treatment of choice; however, visual prognosis largely depends on the duration and severity of preexisting optic pathway damage. In this case, therapeutic options were limited because of advanced disease, prolonged visual impairment, and the patient's overall clinical condition^{8,9}.

This case highlights the severe consequences of delayed diagnosis of a potentially treatable condition and emphasizes the importance of early investigation of progressive visual symptoms, even in elderly or institutionalized individuals, in order to prevent irreversible neurological complications.

The absence of magnetic resonance imaging and endocrine evaluation represents a limitation of this report.

Progressive visual loss in older patients should not be attributed solely to aging without structural evaluation.

Conclusion

Delayed diagnosis of giant pituitary adenomas may lead to severe and irreversible neurological complications. Early recognition and timely evaluation of progressive visual symptoms are essential to improve functional outcomes and prevent permanent neurological sequelae, even in elderly and vulnerable patients.

References

1. Melmed S, Kaiser UB, Lopes MB, Bertherat J, Syro LV. Pituitary tumors. *Nat Rev Dis Primers* 2017;3:17001.
2. Molitch ME. Diagnosis and treatment of pituitary adenomas: a review. *JAMA* 2017;317(5):516-524.

3. Dekkers OM, Pereira AM, Romijn JA. Treatment and follow-up of clinically nonfunctioning pituitary macroadenomas. *J Clin Endocrinol Metab* 2008;93(10):3717-3726.
4. Nomikos P, Buchfelder M, Fahlbusch R. The outcome of surgery in 668 patients with giant pituitary adenomas. *J Neurosurg* 2004;100(4):628-635.
5. Couldwell WT. Transsphenoidal and transcranial surgery for pituitary adenomas. *J Neurooncol* 2004;69(1-3):237-256.
6. Chanson P, Salenave S. Diagnosis and treatment of pituitary adenomas. *Minerva Endocrinol* 2004;29(4):241-275.
7. Jane JA Jr, Laws ER Jr. Surgical treatment of pituitary adenomas. In: Feingold KR, Anawalt B, Blackman MR, et al., editors. *Endotext*. South Dartmouth: MDText.com, Inc 2022.
8. Melmed S. Pituitary-tumor endocrinopathies. *N Engl J Med* 2020;382(10):937-950.
9. Micko ASG, Wöhrer A, Wolfsberger S, Knosp E. Treatment strategies for giant pituitary adenomas in the era of endoscopic transsphenoidal surgery. *J Neurosurg* 2021;136(3):776-785.