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Giant Plurihormonal Pit-1 Pituitary Neuroendocrine Tumor: A Case Report Highlighting Diagnostic, Surgical, And Therapeutic Complexities

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ABSTRACT

Giant pituitary adenomas represent a major clinical challenge due to their invasive nature and the serious complications caused by their mass effect on adjacent structures. This report describes the case of a 35-year-old patient presenting with a massive 60x34 mm tumor, revealed by intracranial hypertension and bilateral blindness, whose histopathology confirmed a Pit-1 lineage plurihormonal PitNET. Despite emergency surgical resection aimed at decompressing the optic pathways and restoring cerebrospinal fluid circulation, resection remained subtotal due to adhesion to critical structures such as the cavernous sinus. Postoperative management required a complex multimodal strategy, combining hormone replacement therapy with targeted medical therapies using dopamine agonists and somatostatin analogues, in the face of a persistent invasive tumor remnant. This case illustrates the therapeutic complexities and fragile prognosis inherent to these neoplasms, highlighting the imperative of multidisciplinary management and rigorous molecular characterization to optimize follow-up and treatment strategies in these high-risk patients.

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KEYWORDS: Giant PitNETs, Knosp Grade 4, Cavernous sinus invasion, Somatostatin analogues, Dopamine agonists, Multidisciplinary management, Optic chiasm compression

INTRODUCTION

Pituitary neuroendocrine tumors are generally benign neoplasms of the anterior pituitary gland. [1] However, tumors exceeding a maximum diameter of 40 mm are classified as "giant" pituitary adenomas, representing a rare subgroup (approximately 10 to 15% of macroadenomas) associated with pronounced surgical morbidity and therapeutic resistance. [2]

Furthermore, truly plurihormonal tumors, specifically those co-secreting growth hormone (GH), prolactin (PRL), and thyroid-stimulating hormone (TSH), account for less than 1% of all anterior pituitary tumors. These mixed-secretion tumors derive from the Pit-1 cell lineage. [3] The management of giant plurihormonal pituitary tumors raises major therapeutic challenges and carries a complex prognosis, requiring a rigorous multidisciplinary approach.

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Tumor volume gives rise to severe neurological complications including obstructive hydrocephalus, cranial nerve involvement, and blindness, while multi-axis hormonal dysregulation represents a major challenge for perioperative therapeutic optimization and long-term follow-up. [1]

Through the study of this case, we examine the diagnostic complexities, histopathological features, and multimodal management of a giant Pit-1 lineage plurihormonal tumor, drawing on current international recommendations in endocrinology.

PATIENT ET OBSERVATION

35-year-old patient was admitted to the emergency department with a syndrome of intracranial hypertension that had been evolving over five days, characterized by helmet-pattern headaches of increasing intensity, projectile vomiting, and a sudden and profound decrease in visual acuity. His notable medical history included insulin-dependent type 1 diabetes and Hashimoto's thyroiditis under balanced hormone replacement therapy.

On physical examination, the patient was severely agitated with a Glasgow Coma Scale (GCS) score of 14. Ophthalmological evaluation revealed bilateral decreased visual acuity resulting in complete blindness. No clinical

signs of classic acromegaly or overt thyrotoxicosis were visually dominant at presentation. An emergency brain MRI revealed a massive, lobulated, and highly invasive sellar and suprasellar mass measuring 60 × 34 mm, establishing the definitive diagnosis of a giant pituitary tumor. The tumor caused upward displacement and distortion of the optic chiasm, secondary obstructive hydrocephalus due to compression of the third ventricle, and significant compression of the V3 segment in Meckel's cave.

Mass dimensions: 60 mm (craniocaudal) × 34 mm (transverse).

Extension: Severe suprasellar invasion, significant compression of the third ventricle, and bilateral cavernous sinus invasion (Knosp Grade 4).

The preoperative endocrine workup revealed mixed pituitary dysfunction, characterized by multi-axis tumor secretion:

Suppressed TSH with proportionally altered free thyroid hormones, significantly confounded by the pre-existing Hashimoto's thyroiditis and levothyroxine intake.

Elevated IGF-1 levels relative to age-adjusted reference values, indicative of autonomous GH secretion.

Moderately elevated serum prolactin (112 ng/mL), raising a differential diagnosis between true tumor co-secretion and the "stalk effect" (disruption of dopamine-mediated inhibitory tone caused by compression of the pituitary stalk).

Here is the translation:

Given the clinical picture of obstructive hydrocephalus and the compromised visual functional prognosis, urgent neurosurgical intervention was indicated. The transsphenoidal endoscopic approach had as its primary objective the decompression of the optic-chiasmatic junction. The intraoperative finding of a fibrous, hypervascularized tumor strongly adherent to critical skull base structures and the cavernous sinus limited the possibilities of total resection. A subtotal resection was successfully carried out, restoring CSF circulation and relieving the optic chiasm from the mechanical compression it was subjected to.

Histopathological examination confirmed the diagnosis of a pituitary neuroendocrine tumor (PitNET). Immunohistochemical analysis was decisive in characterizing the tumor lineage, revealing strong and diffuse nuclear immunoreactivity for the Pit-1 transcription factor. The immunohistochemical profile, combining the concomitant expression of GH, PRL, and TSH, established the definitive diagnosis of a mature Pit-1 lineage plurihormonal PitNET.

In the immediate postoperative period, the patient developed central diabetes insipidus, confirmed by hypernatremia and high serum osmolarity, which was rapidly controlled by the administration of desmopressin. This phase was complicated by severe panhypopituitarism, including acute adrenal insufficiency and central hypothyroidism, requiring replacement corticotherapy and hormonal rebalancing.

The three-month follow-up MRI revealed persistent invasive tumor remnant (44 × 36 × 36 mm) infiltrating the cavernous sinuses and encasing the internal carotid arteries. Faced with this sizeable residual tumor and persistent hormonal activity, a multimodal strategy was implemented, combining medical therapy with dopamine agonists and somatostatin analogues, supplemented by planned iterative surgery followed by adjuvant stereotactic radiotherapy.

Unfortunately, before the therapeutic strategy could bear fruit, the patient died from acute cardiovascular collapse resulting from major brainstem compression, illustrating the clinical aggressiveness and high mortality of these giant pituitary tumors.

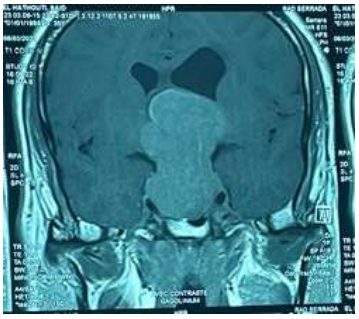

Figure 1

DISCUSSION

The 2022 WHO classification replaced the outdated concept of "atypical adenoma" with a nomenclature based on pituitary transcription factors (Pit-1, T-Pit, and SF-1). Plurihormonal tumors co-expressing GH, PRL, and TSH belong exclusively to the Pit-1 lineage, which regulates the differentiation of somatotrophs, mammotrophs, and thyrotrophs. While these tumors sometimes follow an indolent course, a structural subgroup displays an aggressive clinical phenotype, marked by rapid growth, early cavernous sinus invasion, and increased resistance to conventional therapeutic strategies. [4]

Giant PitNETs (≥ 40 mm) pose a major therapeutic dilemma. Complete resection is rarely achievable via the standard transsphenoidal approach, due to frequent cavernous sinus invasion, encasement of the internal carotid arteries, and extension toward the anterior or posterior cranial fossae. [1] As illustrated by this case, subtotal resection is often the only option for vital decompression, leaving sizeable tumor remnants that require complex and prolonged multimodal management.

In accordance with the guidelines of the European Society of Endocrinology (ESE) and the Pituitary Society, the management of aggressive residual PitNETs requires a proactive therapeutic strategy. When surgical resection fails, rapid medical optimization must be initiated, combined with adjuvant radiotherapy or, in cases of documented rapid progression, chemotherapy with alkylating agents (temozolomide), as recommended in the context of aggressive tumors. [2]

For Pit-1 lineage tumors expressing GH and TSH, the therapeutic standard relies on first-generation somatostatin

analogues (octreotide or lanreotide), allowing biochemical control in more than 90% of classic thyrotrophinomas. [3] However, giant forms frequently exhibit pharmacological resistance mechanisms linked to reduced SSTR2 receptor density or alterations in intracellular signaling pathways, promoting persistent tumor expansion. [2]

In these refractory cases, current recommendations advocate a multidisciplinary approach combining a thorough evaluation of the receptor profile — sometimes justifying the use of second-generation analogues such as pasireotide — with early stereotactic radiotherapy to stabilize tumor volume.

CONCLUSION

This case highlights the diagnostic and therapeutic challenges posed by giant Pit-1 lineage PitNETs. Despite management combining surgical resection and targeted medical therapies, the unfavorable clinical outcome serves as a reminder of the aggressive nature and high mortality of these tumors. To improve the prognosis of these patients, an early multidisciplinary strategy is essential, incorporating rigorous molecular characterization as well as close coordination between neurosurgeons, endocrinologists, and radiation oncologists.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

AUTHOR CONTRIBUTIONS

All authors have contributed to the conduct of this work. All authors also declare that they have read and approved the final version of the manuscript.

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