

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

Treatment-resistant Cushing disease and acromegaly in a young woman: A case of functional pituitary macroadenoma[☆]

Ibrahim Khalil, MBBS^{a,*}, Md. Imran Hossain, MBBS^b^aDhaka Medical College and Hospital, Dhaka, Bangladesh^bFaculty of Medicine, Dhaka University, Dhaka, Bangladesh

ARTICLE INFO

Article history:

Received 4 September 2024

Revised 9 December 2024

Accepted 3 January 2025

Keywords:

Hormone

Pituitary

Macroadenoma

Microadenoma

Cushing

Acromegaly

ABSTRACT

Cushing disease and acromegaly are common endocrine disorders caused by excessive cortisol and growth hormone production, respectively. Both conditions can co-occur due to functioning pituitary adenomas, which are typically benign pituitary gland tumors. This report discusses a 30-year-old woman with hyperpituitarism leading to treatment-resistant Cushing disease and acromegaly caused by a functional pituitary macroadenoma. A 30-year-old woman presented with a history of excessive weight gain, facial puffiness, fatigue, persistent headaches, and visual disturbances. Clinical examination revealed features consistent with Cushing disease and acromegaly, including a moon face, central obesity, and large hands and feet—the ophthalmologic evaluation identified bitemporal hemianopia, suggesting optic chiasm compression. Laboratory results showed elevated ACTH, IGF-1, and prolactin levels, alongside confirmed hypercortisolism. The patient also had secondary diabetes mellitus and galactorrhea—initial treatment with octreotide provided limited benefit, with persistent hormone elevations and insufficient symptom control. The patient underwent endonasal endoscopic transsphenoidal resection of the pituitary macroadenoma, leading to marked symptomatic and hormonal improvements. This underscores the diagnostic challenge and treatment complexity of such cases. Early diagnosis is critical for optimizing outcomes in patients with hyperpituitarism and mitigating complications. This case highlights the importance of multidisciplinary management and the necessity of long-term follow-up to monitor for recurrence and ensure sustained remission.

© 2025 The Authors. Published by Elsevier Inc. on behalf of University of Washington.

This is an open access article under the CC BY-NC-ND license

(<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

[☆] Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

* Corresponding author.

E-mail address: ibrahim124904@gmail.com (I. Khalil).

<https://doi.org/10.1016/j.radcr.2025.01.010>

1930-0433/© 2025 The Authors. Published by Elsevier Inc. on behalf of University of Washington. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

Introduction

Pituitary adenomas are benign tumors arising from the pituitary gland, often referred to as the "master gland" due to its central role in regulating key physiological processes such as growth, metabolism, and reproduction [1,2]. These tumors are classified by size into microadenomas (<10 mm) and macroadenomas (≥ 10 mm) and by hormonal activity into functioning and nonfunctioning adenomas. Functioning adenomas actively secrete hormones, leading to distinct syndromes such as prolactinomas, acromegaly (from growth hormone overproduction), and Cushing disease (from excess ACTH). In contrast, nonfunctioning adenomas do not secrete hormones but may cause symptoms due to mass effects, such as visual disturbances or hypopituitarism [3–5].

The simultaneous occurrence of Cushing disease and acromegaly is rare and presents a significant diagnostic and therapeutic challenge. Both conditions stem from hyperpituitarism, typically due to a functional pituitary adenoma [6,7]. Cushing disease results from ACTH hypersecretion, causing excessive cortisol production and features such as central obesity, hypertension, hyperglycemia, and muscle weakness [8–10]. Prolonged cortisol exposure can lead to severe complications, including cardiovascular diseases and osteoporosis. Acromegaly, on the other hand, arises from growth hormone overproduction, leading to elevated IGF-1 levels and characteristic features such as enlarged extremities, facial changes, and systemic complications like insulin resistance and joint abnormalities [11–13].

The coexistence of Cushing disease and acromegaly within the same affected person is extraordinarily rare, making this particular case record particularly noteworthy [14,15]. The simultaneous presentation of these 2 endocrine problems in a young lady because of a hormonally functioning pituitary macroadenoma presents a unique scientific venture [16,17]. The pituitary macroadenoma, defined as a tumor more than 10 mm in diameter, can compress adjoining structures within the sella turcica and enlarge into surrounding areas, leading to signs and symptoms with complications, visible disturbances, and hyperpituitarism. In this case, the patient presented with both Cushing disease and acromegaly, at the same time symptoms as a result of the mass impact of the macroadenoma.

The case of a 30-year-old female with hyperpituitarism, characterized with the aid of drug-resistant Cushing disease and acromegaly, highlights the complexities intricately associated with the analysis and control of a couple of endocrine issues bobbing up from a single pituitary macroadenoma. Her medical presentation changed into one marked by a history of noticeable weight gain, facial puffiness, fatigue, chronic complications, and visual disturbances. A thorough physical exam found traits consistent with each Cushing disorder and acromegaly, which include a moon face, vital weight problems, and enlarged arms and toes. The ophthalmologic exam confirmed bitemporal hemianopia, indicative of optic chiasm compression with the aid of the pituitary macroadenoma. Early recognition and multidisciplinary management are essential to mitigate the significant morbidity associated with these conditions. This case report highlights a rare instance of concurrent Cushing disease and acromegaly due to a func-

tional pituitary macroadenoma, underscoring the importance of timely diagnosis and treatment.

Case presentation

This case of a 30-year-old female highlights the complexities of diagnosing and managing a functional pituitary macroadenoma presenting with overlapping features of Cushing disease and acromegaly, along with secondary diabetes mellitus.

The patient demonstrated classic signs of hypercortisolism, including central obesity with a "moon face" and "buffalo hump," skin thinning, easy bruising, and muscle weakness. Cortisol's catabolic effects were evident in her limb wasting and truncal obesity. Metabolic complications included hypertension and secondary diabetes mellitus, supported by elevated random blood sugar (22 mmol/L) and postprandial blood sugar levels (27 mmol/L). Laboratory findings showed significantly elevated ACTH levels (670 pg/mL; normal: 10–60 pg/mL) and increased morning urine cortisol levels.

The patient also exhibited hallmark features of acromegaly, including enlarged hands and feet, necessitating larger shoe and glove sizes, and distinct facial changes such as mandibular prognathism, frontal bossing, and nasal broadening. Soft tissue swelling and fatigue were also noted, alongside joint pain likely resulting from cartilage and bone overgrowth. Her IGF-1 levels were markedly elevated (798 ng/mL; normal: 100–300 ng/mL).

Hyperprolactinemia (643 ng/mL; normal: 5–25 ng/mL) caused galactorrhea, likely resulting from tumor compression of the pituitary stalk or direct prolactin secretion. Diabetes mellitus, secondary to insulin resistance driven by excess cortisol and growth hormone, further complicated her clinical picture (Table 1).

Secondary diabetes mellitus is a common trouble in sufferers with Cushing disease and acromegaly, stemming from the insulin resistance brought about by persistent hypercortisolism and hypersecretion of GH. This patient's multiplied blood sugar also reflects tremendous impairment in glucose metabolism. Polyuria, polydipsia, and unexplained weight loss are classic signs of diabetes that could have been found in her clinical history but are frequently overshadowed by the traits of the more distinguished functions of her endocrine disorders. The affected person additionally experienced galactorrhea, an odd milk discharge from the breasts, that's on account of her expanded prolactin levels (643 ng/mL, ordinary range: 2–29 ng/mL). Hyperprolactinemia inside the context of a pituitary macroadenoma can result from the tumor's direct secretion of prolactin or from the stalk effect, where the tumor compresses the pituitary stalk, disrupting dopamine inhibition of prolactin secretion.

MRI was the primary imaging modality, revealing a large pituitary macroadenoma centered within the sella turcica and extending suprasellar. The tumor demonstrated homogeneous postcontrast enhancement and exerted mass effects, including optic chiasm compression correlating with bitemporal hemianopia. Other modalities, such as CT, were not considered due to MRI's superior resolution for pituitary evaluation.

Table 1 – Markedly elevated hormone levels preoperatively and their postoperative normalization.

Hormone	Patient's level (Preoperative)	Postoperative levels	Normal reference value
ACTH	670 pg/mL	90 pg/mL	10–60 pg/mL
IGF-1	798 ng/mL	280 ng/mL	100–300 ng/mL (age-dependent)
Prolactin	643 ng/mL	42 ng/mL	5–25 ng/mL
Morning Urine Cortisol	Elevated	Normal	<50 mcg/24 h
Random Blood Sugar	22 mmol/L	6.5 mmol/L	4.0–7.8 mmol/L
2-Hour Postprandial Blood Sugar	27 mmol/L	7.0 mmol/L	<7.8 mmol/L
TSH (Thyroid-Stimulating Hormone)	0.8 mIU/L	1.2 mIU/L	0.5–5.0 mIU/L
FT3 (Free Triiodothyronine)	4.5 pmol/L	4.0 pmol/L	3.5–7.7 pmol/L
FT4 (Free Thyroxine)	15 pmol/L	16 pmol/L	12–22 pmol/L

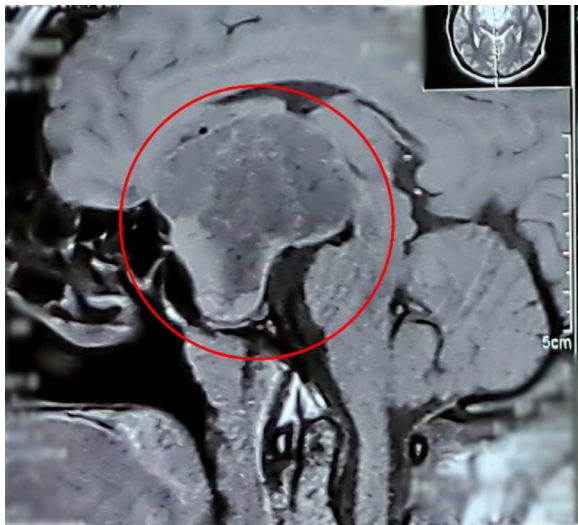


Fig. 1 – This sagittal T1-weighted postcontrast MRI of the brain, specifically focusing on the sella turcica region, reveals a large, homogeneously enhancing mass centered within the sella turcica, consistent with a pituitary macroadenoma. The mass exhibits clear, well-defined borders and appears to expand the sella, with extension into the suprasellar region (marked by circle).

The MRI scans of the patient reveal a large, well-defined pituitary macroadenoma centered within the sella turcica, exhibiting significant suprasellar extension. On sagittal T1-weighted postcontrast imaging (Fig. 1), the lesion demonstrates homogeneous enhancement with clear, well-defined borders, expanding superiorly into the suprasellar region. Coronal T2-weighted images (Fig. 2) further delineate this suprasellar extension, with the mass exerting mass effect on adjacent structures.

Additional sagittal T1-weighted postcontrast imaging (Fig. 3) confirms the uniform enhancement of the macroadenoma, filling the sella turcica and extending upward. Coronal T2-weighted MRI (Fig. 4) reveals the lesion as hyperintense, extending into the suprasellar region and displacing the optic chiasm. The imaging highlights the well-defined borders of the mass and the potential mass effect on adjacent structures.

Axial T2-weighted MRI images (Fig. 5) depict a hyperintense lesion in the basal ganglia and thalamus, appearing as a

bright, well-defined signal. This finding suggests a potential coexisting pathology affecting deep brain structures, which may or may not be related to the primary pituitary lesion. The characteristics and location of the pituitary macroadenoma correspond with the patient's clinical presentation of bitemporal hemianopia, likely caused by compression of the optic chiasm.

The overall imaging features, including homogeneous enhancement, well-defined borders, and suprasellar extension, are hallmark characteristics of pituitary macroadenomas. The potential lateral extension toward the cavernous sinus warrants further evaluation, while the hyperintense lesion in the basal ganglia and thalamus may indicate secondary effects or unrelated CNS pathology.

The imaging findings collectively support the diagnosis of a large, functioning pituitary macroadenoma, exceeding 10 mm in diameter. The mass's size and anatomical impact align with the patient's clinical presentation, which includes headaches, visual field deficits, and hormonal imbalances. The documented compression of the optic chiasm and possible involvement of the cavernous sinus provide a radiological explanation for the patient's visual symptoms and hormonal disruptions. This MRI assessment substantiates the diagnosis of a pituitary macroadenoma with significant suprasellar extension and compression effects, consistent with the patient's symptomatology and clinical findings.

The conglomeration of her clinical presentations, elevated hormone levels, and MRI findings of a big suprasellar mass pretty suggestive of a pituitary macroadenoma showed the analysis of a functioning pituitary adenoma. The preliminary treatment control with octreotide, a somatostatin analog, aimed to control both acromegaly and Cushing disorder by inhibiting GH and ACTH secretion. However, the suboptimal reaction highlighted the undertaking of achieving hormone manipulation in sufferers with massive, competitive adenomas.

Given the patient's persistent symptoms and the insufficient biochemical response to medical therapy, surgical intervention was considered imperative. The patient underwent endonasal endoscopic transsphenoidal resection of the pituitary gland, a minimally invasive surgical approach targeting the tumor via the nasal passages. This approach was preferred over traditional craniotomy due to its demonstrated efficacy in reducing tumor size and lowering elevated hormone levels with fewer complications, reduced morbidity, shorter hospital stays, and faster recovery.

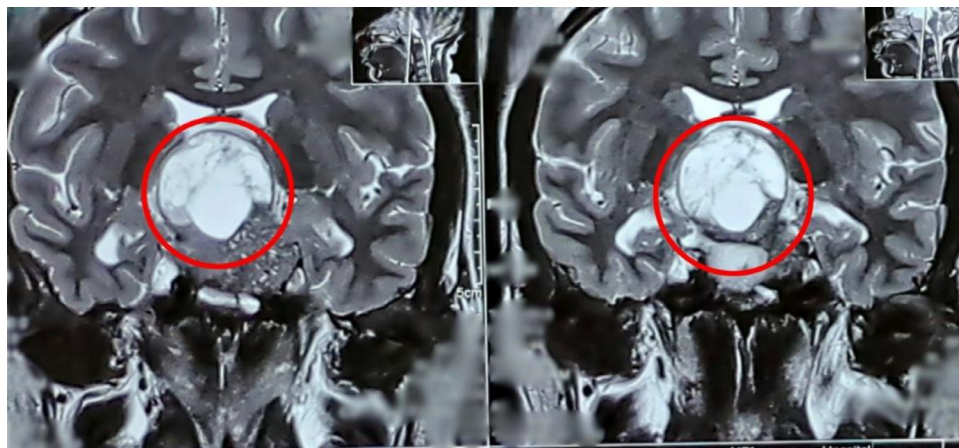


Fig. 2 – This image shows MRI scan of the brain in coronal T2-weighted images which reveals large suprasellar mass (marked by circles).

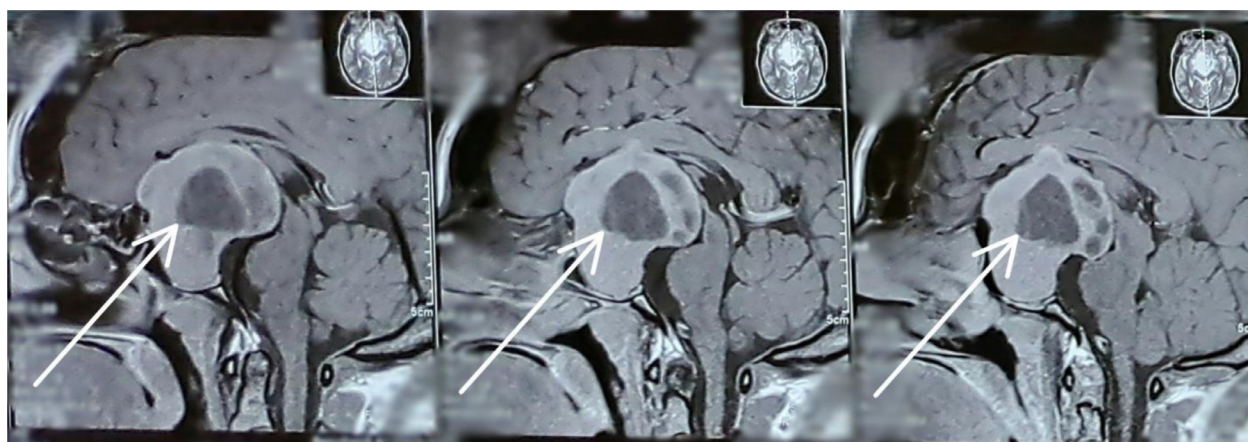


Fig. 3 – Sagittal T1-weighted postcontrast MRI depicting a large, homogeneously enhancing pituitary macroadenoma within the sella turcica, expanding into the suprasellar region with well-defined borders (marked by arrows).

ery times. Additionally, the endoscopic technique offers superior visualization of the surgical field, which aids in precise tumor resection and preservation of normal pituitary tissue.

During the surgery, the tumor was noted to be soft and well-circumscribed, with no significant adherence to adjacent structures such as the cavernous sinus or optic chiasm. This facilitated a complete resection of the tumor, minimizing the risk of residual disease. There were no notable intraoperative complications, such as cerebrospinal fluid leakage or significant bleeding, underscoring the safety and efficacy of the chosen approach. Postoperatively, the patient demonstrated marked clinical improvement in her symptoms, accompanied by a significant reduction in hormone levels to within normal reference ranges. This confirmed the diagnosis and highlighted the effectiveness of the surgical intervention. Specifically, there was a substantial decrease in ACTH, IGF-1, and prolactin levels, leading to clinical remission of Cushing disease and acromegaly.

In the postoperative period, the patient did not require immediate hormone replacement therapy, as her endocrine functions remained stable. However, long-term monitoring is planned to assess for potential hormone deficiencies, disease recurrence, or other complications. The follow-up plan includes regular clinical evaluations, hormonal assays, and periodic imaging studies to ensure sustained remission and to promptly address any residual or recurrent tumor growth. This case highlights the crucial role of surgical intervention in managing functional pituitary macroadenomas, particularly when medical therapy fails. The successful outcome underscores the importance of a multidisciplinary approach and the need for lifelong surveillance to optimize long-term outcomes for such patients. This case scenario also underscores the complexities interwoven in diagnosing and coping with hyperpituitarism because of a pituitary macroadenoma, emphasizing the warrant for a complete and multidisciplinary approach. Early recognition of symptoms, correct diagnostic workup, and timely endocrine disorders.

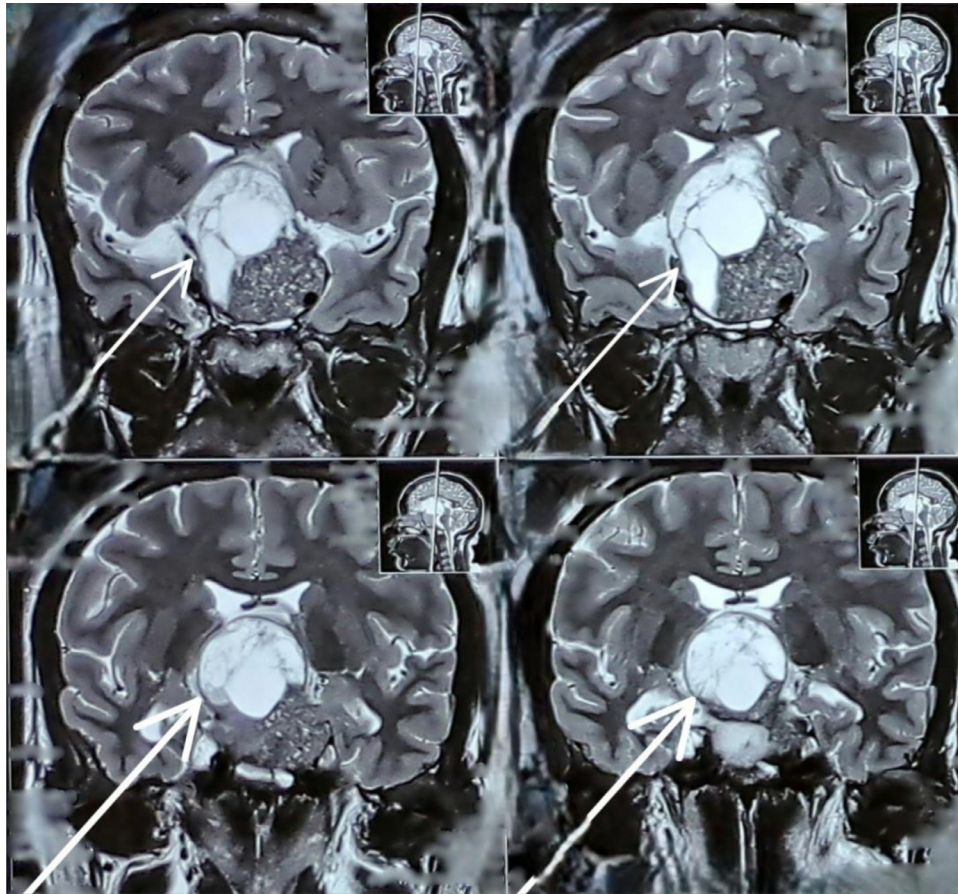


Fig. 4 – Coronal T2-weighted MRI demonstrating a large, hyperintense pituitary macroadenoma within the sella turcica, extending into the suprasellar region (marked by arrows). The lesion displaces the optic chiasm and exhibits well-defined borders, suggesting potential mass effect.

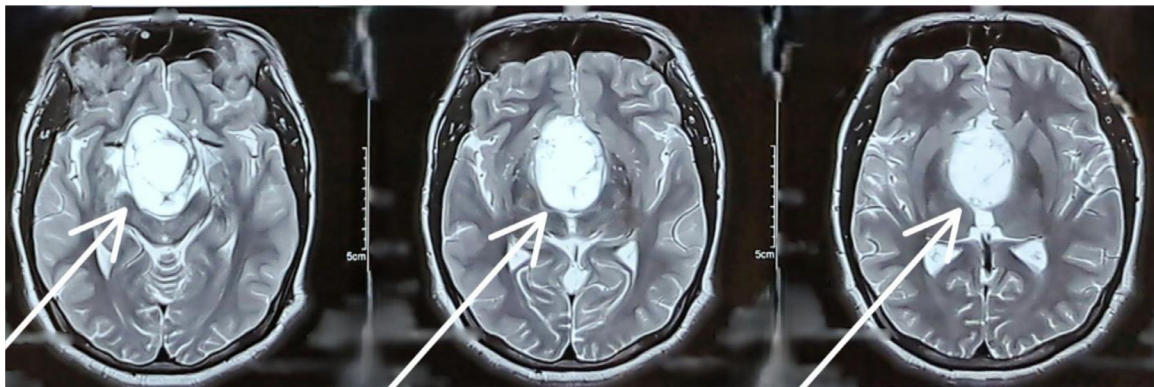


Fig. 5 – Axial T2-weighted MRI images of the brain showing a hyperintense lesion in the region of the basal ganglia and thalamus, indicated by white arrows. The lesion appears as a well-defined, bright signal, suggestive of a pathology affecting deep brain structure.

Discussion

The case of this 30-year-old woman with concurrent refractory Cushing disease and acromegaly due to a functional pi-

pituitary macroadenoma highlights the challenges inherent in diagnosing and managing multiple endocrine disorders. Recognizing overlapping clinical features was central to reaching the diagnosis. Classic symptoms of Cushing disease, such as a moon face and central obesity, coupled with acromegalic fea-

tures, including enlarged extremities, underscored the complexity of the case. The presence of bitemporal hemianopia further pointed to a large pituitary mass compressing the optic chiasm, necessitating imaging studies for confirmation. This case underscores the need for clinicians to remain vigilant when evaluating overlapping endocrine features to avoid delays in diagnosis and treatment [18–20].

Laboratory evaluations were pivotal, revealing markedly elevated ACTH, IGF-1, and prolactin levels, in addition to evidence of hypercortisolism and secondary diabetes mellitus. These findings highlighted the intricate interplay of hypersecreted pituitary hormones and the systemic consequences of unregulated hormone production. MRI findings of a large suprasellar pituitary tumor were instrumental in confirming the diagnosis of a functional macroadenoma and guided subsequent treatment decisions.

The patient's suboptimal response to octreotide therapy underscored the limitations of medical treatments in addressing aggressive, hormone-secreting pituitary macroadenomas. While somatostatin analogs are effective in many cases of acromegaly and can provide symptomatic relief, their efficacy is limited in patients with large adenomas and significant hormonal hypersecretion. This case highlights the necessity of early consideration of definitive surgical intervention when medical therapy fails to achieve adequate biochemical control [21–23].

Endonasal endoscopic transsphenoidal surgery was selected for this patient due to its minimally invasive approach, superior visualization of the sellar region, and lower complication rates compared to traditional craniotomy. Intraoperatively, the tumor's soft consistency and lack of adherence to adjacent structures facilitated a complete resection. Notably, the absence of significant complications, such as cerebrospinal fluid leakage or vascular injury, reflected the safety and precision of this surgical approach [24–26].

Postoperatively, the patient experienced substantial improvement in symptoms, with normalization of ACTH, IGF-1, and prolactin levels. This outcome underscores the efficacy of surgical intervention in achieving hormonal remission and alleviating symptoms in patients with functional macroadenomas. The resolution of her secondary diabetes mellitus and galactorrhea further reinforced the success of treatment [27–29].

Managing such complex endocrine disorders necessitates a multidisciplinary approach, with endocrinologists, radiologists, and neurosurgeons collaborating to ensure accurate diagnosis and effective treatment planning. Radiologists play a critical role in identifying and characterizing pituitary tumors, while endocrinologists monitor hormonal responses and guide perioperative management [30–32]. Neurosurgeons provide expertise in resecting these challenging lesions and optimizing patient outcomes.

The prognosis for patients undergoing surgical resection of functional pituitary macroadenomas is generally favorable when hormonal remission is achieved. However, long-term follow-up is critical to monitor for potential disease recurrence and manage any residual hormone deficiencies. Lifelong surveillance, including periodic hormonal assays and imaging studies, is recommended. Although the patient did not require immediate hormone replacement therapy, ongoing as-

essment of endocrine function remains essential to address emerging deficiencies promptly [33–36].

This case exemplifies the importance of integrating current evidence-based practices into patient care. Recent guidelines and studies emphasize the role of endoscopic surgery as the preferred approach for resecting pituitary tumors due to its high success rates and reduced morbidity compared to older techniques.

Conclusion

This case highlights the pivotal role of surgical intervention in managing hormone-resistant pituitary macroadenomas underscoring the role of a multidisciplinary approach involving endocrinology, radiology, and neurosurgery, demonstrating its effectiveness in resolving hormonal overproduction and alleviating symptoms. Long-term follow-up is indispensable to monitor for recurrence, address emerging complications, and ensure sustained remission, reinforcing the need for vigilance and specialized endocrine care in managing these complex disorders.

Patient consent

Written informed consent for publication of this case report was obtained from the patient(s). The patient(s) were provided with sufficient information regarding the nature of the publication, including the details to be disclosed and potential implications. The patient(s) have confirmed their understanding and voluntarily agreed to the publication of this case report.

REFERENCES

- [1] Ershadinia N, Tritos NA. Diagnosis and treatment of acromegaly: an update. *Mayo Clin Proc* 2022;97(2):333–46. doi:10.1016/j.mayocp.2021.11.007.
- [2] Melmed S. Pituitary-tumor endocrinopathies. *N Engl J Med* 2020;382(10):937–50. doi:10.1056/NEJMra1810772.
- [3] Chanson P, Raverot G, Castinetti F, Cortet-Rudelli C, Galland F, Salenave S French Endocrinology Society non-functioning pituitary adenoma work-group. Management of clinically non-functioning pituitary adenoma. *Ann Endocrinol (Paris)* 2015;76(3):239–47 Epub 2015 Jun 10. doi:10.1016/j.ando.2015.04.002.
- [4] Melmed S. Acromegaly pathogenesis and treatment. *J Clin Invest* 2009;119(11):3189–202 Epub 2009 Nov 2. doi:10.1172/JCI39375.
- [5] Gadelha MR, Wildemberg LE, Kasuki L. The future of somatostatin receptor ligands in Acromegaly. *J Clin Endocrinol Metab* 2022;107(2):297–308. doi:10.1210/clinem/dgab726.
- [6] Gadelha MR, Kasuki L. Refractory somatotroph adenomas. *Pituitary* 2023;26(3):266–8 Epub 2023 Jun 15. doi:10.1007/s11102-023-01324-5.
- [7] Lamas C, García-Martínez A, Cámara R, Fajardo-Montanana C, Viguera L, Aranda I. Silent somatotrophinomas. *Minerva Endocrinol* 2019;44(2):137–42 Epub 2018 Dec 7. doi:10.23736/S0391-1977.18.02946-2.

- [8] CDC Kamilaris, Faucz FR, Voutetakis A, Stratakis CA. Carney complex. *Exp Clin Endocrinol Diabetes* 2019;127(2–03):156–64 Epub 2018 Nov 14. doi:[10.1055/a-0753-4943](https://doi.org/10.1055/a-0753-4943).
- [9] Carroll P.V., Joshi M.N. Acromegaly. In: Feingold KR, Anawalt B, Blackman MR, Boyce A, Chrousos G, Corpas E, de Herder WW, Dhatariya K, Dungan K, Hofland J, Kalra S, Kaltsas G, Kapoor N, Koch C, Kopp P, Korbonits M, Kovacs CS, Kuohung W, Laferrère B, Levy M, McGee EA, McLachlan R, New M, Purnell J, Sahay R, Shah AS, Singer F, Sperling MA, Stratakis CA, Trencé DL, Wilson DP, editors. *Endotext* [Internet] MDText.com, Inc.; South Dartmouth (MA): 2000.
- [10] Biermasz NR. The burden of disease for pituitary patients. *Best Pract Res Clin Endocrinol Metab* 2019;33(2):101309 Epub 2019 Aug 2. doi:[10.1016/j.beem.2019.101309](https://doi.org/10.1016/j.beem.2019.101309).
- [11] Walz PC, Drapeau A, Shaikhouni A, Eide J, Rugino AJ, Mohyeldin A, et al. Pediatric pituitary adenomas. *Childs Nerv Syst* 2019;35(11):2107–18 Epub 2019 Jul 13. doi:[10.1007/s00381-019-04293-y](https://doi.org/10.1007/s00381-019-04293-y).
- [12] Ben-Shlomo A, Cooper O. Silent corticotroph adenomas. *Pituitary* 2018;21(2):183–93. doi:[10.1007/s11102-018-0864-8](https://doi.org/10.1007/s11102-018-0864-8).
- [13] Greco DS. Feline acromegaly. *Top Companion Anim Med* 2012;27(1):31–5. doi:[10.1053/j.tcam.2012.05.004](https://doi.org/10.1053/j.tcam.2012.05.004).
- [14] Mehta GU, Jane JA Jr. Pituitary tumors. *Curr Opin Neurol* 2012;25(6):751–5. doi:[10.1097/WCO.0b013e3283587bed](https://doi.org/10.1097/WCO.0b013e3283587bed).
- [15] Mosbah H, Brue T, Chanson P. Acromégalie : améliorer la prise en charge : acromégaly: improving care. *Ann Endocrinol (Paris)* 2019;80(Suppl 1):S10–18 French. doi:[10.1016/S0003-4266\(19\)30112-X](https://doi.org/10.1016/S0003-4266(19)30112-X).
- [16] Labadzhyan A, Melmed S. Molecular targets in acromegaly. *Front Endocrinol (Lausanne)* 2022;13:1068061. doi:[10.3389/fendo.2022.1068061](https://doi.org/10.3389/fendo.2022.1068061).
- [17] Valassi E. Pituitary disease and pregnancy. *Endocrinol Diabetes Nutr (Engl Ed)* 2021;68(3):184–95. doi:[10.1016/j.endien.2020.07.002](https://doi.org/10.1016/j.endien.2020.07.002).
- [18] Iglesias P, Rodríguez Berrocal V, Díez JJ. Giant pituitary adenoma: histological types, clinical features and therapeutic approaches. *Endocrine* 2018;61(3):407–21. doi:[10.1007/s12020-018-1645-x](https://doi.org/10.1007/s12020-018-1645-x).
- [19] Chanson P, Salenave S, Kamenicky P, Cazabat L, Young J. Pituitary tumours: acromegaly. *Best Pract Res Clin Endocrinol Metab* 2009;23(5):555–74. doi:[10.1016/j.beem.2009.05.010](https://doi.org/10.1016/j.beem.2009.05.010).
- [20] Dhaneshwar S, Shandily S, Tiwari V. Growth hormone excess: implications and management. *Endocr Metab Immune Disord Drug Targets* 2023;23(6):748–63. doi:[10.2174/1871530322666221012155533](https://doi.org/10.2174/1871530322666221012155533).
- [21] Giantini-Larsen AM, Uribe-Cardenas R, Juthani RG. Acromegaly: mmedical and surgical considerations. *Otolaryngol Clin North Am* 2022;55(2):331–41 Epub 2022 Mar 4. doi:[10.1016/j.otc.2021.12.006](https://doi.org/10.1016/j.otc.2021.12.006).
- [22] Auriemma RS, Grasso LF, Pivonello R, Colao A. The safety of treatments for prolactinomas. *Expert Opin Drug Saf* 2016;15(4):503–12 Epub 2016 Mar 7. doi:[10.1517/14740338.2016.1151493](https://doi.org/10.1517/14740338.2016.1151493).
- [23] Mehta GU, Lonser RR. Management of hormone-secreting pituitary adenomas. *Neuro Oncol* 2017;19(6):762–73. doi:[10.1093/neuonc/now130](https://doi.org/10.1093/neuonc/now130).
- [24] Loughrey PB, Korbonits M. Genetics of pituitary tumours. *Exp Suppl.* 2019;111:171–211. doi:[10.1007/978-3-030-25905-1_10](https://doi.org/10.1007/978-3-030-25905-1_10).
- [25] Bray DP, Mannam S, Rindler RS, Quillin JW, Oyesiku NM. Surgery for acromegaly: iindications and goals. *Front Endocrinol (Lausanne)* 2022;13:924589. doi:[10.3389/fendo.2022.924589](https://doi.org/10.3389/fendo.2022.924589).
- [26] Ambrosio MR, Gagliardi I, Chiloiro S, Ferreira AG, Bondanelli M, Giampietro A, et al. Acromegaly in the elderly patients. *Endocrine* 2020;68(1):16–31. doi:[10.1007/s12020-020-02206-7](https://doi.org/10.1007/s12020-020-02206-7).
- [27] Bray DP, Rindler RS, Dawoud RA, Boucher AB, Oyesiku NM. Cushing disease: mmedical and surgical considerations. *Otolaryngol Clin North Am* 2022;55(2):315–29 Epub 2022 Mar 4. doi:[10.1016/j.otc.2021.12.006](https://doi.org/10.1016/j.otc.2021.12.006).
- [28] Cooper O, Greenman Y. Dopamine agonists for pituitary adenomas. *Front Endocrinol (Lausanne)* 2018;9:469. doi:[10.3389/fendo.2018.00469](https://doi.org/10.3389/fendo.2018.00469).
- [29] Haberbosch L, Strasburger CJ. Efficacy and safety of Pegvisomant in the treatment of acromegaly. *Arch Med Res* 2023;54(8):102884 Epub 2023 Sep 1. doi:[10.1016/j.arcmed.2023.102884](https://doi.org/10.1016/j.arcmed.2023.102884).
- [30] González-Virla B, Vargas-Ortega G, Romero-Gameros CA. Radiotherapy and mortality in pituitary adenomas. *Arch Med Res* 2023;54(8):102900 Epub 2023 Nov 6. doi:[10.1016/j.arcmed.2023.102900](https://doi.org/10.1016/j.arcmed.2023.102900).
- [31] Ioachimescu AG. Sociodemographic factors in pituitary adenomas. *Endocrinol Metab Clin North Am* 2023;52(4):705–17 Epub 2023 Jun 7. doi:[10.1016/j.ecl.2023.05.008](https://doi.org/10.1016/j.ecl.2023.05.008).
- [32] Donoho DA, Bose N, Zada G, Carmichael JD. Management of aggressive growth hormone secreting pituitary adenomas. *Pituitary* 2017;20(1):169–78. doi:[10.1007/s11102-016-0781-7](https://doi.org/10.1007/s11102-016-0781-7).
- [33] Faltermeier CM, Magill ST, Blevins LS Jr, Aghi MK. Molecular biology of pituitary adenomas. *Neurosurg Clin N Am* 2019;30(4):391–400 Epub 2019 Jul 5. doi:[10.1016/j.nec.2019.05.001](https://doi.org/10.1016/j.nec.2019.05.001).
- [34] Bianchi A, Chiloiro S, Giampietro A, Gaudino S, Calandrelli R, Mazzarella C, et al. Multidisciplinary management of difficult/aggressive growth- hormone pituitary neuro-endocrine tumors. *Front Endocrinol (Lausanne)* 2023;14:1123267. doi:[10.3389/fendo.2023.1123267](https://doi.org/10.3389/fendo.2023.1123267).
- [35] Hu M, Tomlinson B. Pharmacokinetic evaluation of lanreotide. *Expert Opin Drug Metab Toxicol* 2010;6(10):1301–12. doi:[10.1517/17425255.2010.513700](https://doi.org/10.1517/17425255.2010.513700).
- [36] Minniti G, Clarke E, Scaringi C, Enrici RM. Stereotactic radiotherapy and radiosurgery for non-functioning and secreting pituitary adenomas. *Rep Pract Oncol Radiother* 2016;21(4):370–8. doi:[10.1016/j.rpor.2014.09.004](https://doi.org/10.1016/j.rpor.2014.09.004).