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Pituicytoma Presenting With Hypogonadotropic Hypogonadism in a Highly Vascular Sellar and Suprasellar Mass: A Case Report

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Abstract

Pituicytoma is a rare low-grade tumor arising from the pituicyte lineage of the posterior pituitary and infundibular region. Because its clinical and magnetic resonance imaging (MRI) features overlap with those of more common sellar tumors and because it is often highly vascular, preoperative diagnosis and surgical management can be challenging.

We presented a 45-year-old man who presented with four years of fatigue, reduced libido, erectile dysfunction, and intermittent frontal headache. Early-morning biochemical testing showed low total testosterone (8.9 nmol/L), with inappropriately normal luteinizing hormone (LH) and follicle-stimulating hormone (FSH), consistent with secondary hypogonadism. Pituitary magnetic resonance imaging showed a 25 × 28 × 27 mm sellar and suprasellar mass with cavernous sinus extension and superior displacement of the optic chiasm; radiologically, the lesion was initially interpreted as a pituitary macroadenoma. Because of concern regarding vascularity, computed tomography (CT) cerebral angiography was performed and demonstrated a hypertrophied right meningo-hypophyseal trunk supplying the tumor.

Due to the anticipated risk of major intraoperative hemorrhage, partial surgical debulking was performed. Histopathology and immunohistochemistry supported the diagnosis of pituicytoma. Testosterone replacement increased serum testosterone. Six weeks after surgery, the patient developed secondary adrenal insufficiency and was commenced on hydrocortisone replacement. Radiotherapy was planned for the substantial residual tumor.

This case highlights the importance of considering pituicytoma in the differential diagnosis of an enhancing sellar or suprasellar mass with pituitary dysfunction, particularly when marked vascularity is suspected. Preoperative vascular assessment and multidisciplinary management are important when complete resection may carry substantial hemorrhagic risk.

Categories: Oncology, Neurosurgery, Endocrinology/Diabetes/Metabolism
Keywords: hypervascular tumor, hypogonadotropic hypogonadism, pituicytoma, pituitary tumor, secondary adrenal insufficiency, sellar mass

Introduction

Pituicytoma is a rare, low-grade neoplasm of the pituicyte lineage arising in the posterior pituitary and infundibular region. In the fifth edition of the World Health Organization classification of endocrine and neuroendocrine tumors, pituicytoma belongs to the pituicyte tumor family, which also includes granular, oncocytic, and ependymal forms. These tumors are uncommon and may present with headache, visual symptoms, or pituitary hormone abnormalities [1,2].

Preoperative diagnosis is difficult because pituicytomas can appear as solid, strongly enhancing sellar or suprasellar masses and may closely mimic a pituitary neuroendocrine tumor (PitNET), historically termed pituitary adenoma [2-4]. Marked vascularity is a clinically important feature and can result in major intraoperative bleeding, limiting the extent of resection [3,5,6]. Histopathology is therefore central to diagnosis, with a characteristic spindle-cell morphology and nuclear TTF-1 expression supporting pituicyte lineage [4,7].

We report a highly vascular pituicytoma presenting predominantly with symptomatic hypogonadotropic hypogonadism. The case illustrates the diagnostic value of considering structural hypothalamic-pituitary disease even when testosterone is above the threshold commonly associated with pituitary imaging and the importance of preoperative vascular characterization when a sellar mass is suspected.

Case Presentation

A 45-year-old man was referred to endocrinology with a four-year history of progressive fatigue, lethargy,

reduced libido, and erectile dysfunction. Symptoms had worsened over the preceding months and were affecting his quality of life. He also reported intermittent sharp frontal headaches without a consistent precipitating factor. There was no reported visual disturbance.

His medical history included familial hypercholesterolemia, prediabetes, depression, and post-traumatic stress disorder. Medications were simvastatin and ezetimibe. He denied anabolic steroid, long-term opioid, or glucocorticoid use. On examination, testicular volume was approximately 15 mL bilaterally, with normal secondary sexual characteristics and no gynecomastia. Bedside visual-field examination was normal.

Early-morning fasting biochemical testing demonstrated low total testosterone and calculated free testosterone, with luteinizing hormone (LH) and follicle-stimulating hormone (FSH) within their laboratory reference ranges but inappropriately normal for the degree of testosterone reduction. This was consistent with hypogonadotropic hypogonadism (Table *1*). Prolactin, thyroid function, and insulin-like growth factor 1 were within the laboratory reference ranges. The 9 AM cortisol was low at 189 nmol/L, prompting assessment of the hypothalamic-pituitary-adrenal axis.

Investigation	Patient value	Reference range*
Total testosterone	8.9 nmol/L	10-30 nmol/L
Calculated free testosterone	0.220 nmol/L	>0.226 nmol/L
Sex hormone-binding globulin	20.4 nmol/L	17.3-65.8 nmol/L
Prolactin	364 mIU/L	45-375 mIU/L
Follicle-stimulating hormone (FSH)	2.6 IU/L	1.6-11.0 IU/L
Luteinizing hormone (LH)	1.6 IU/L	1.3-8.3 IU/L
Thyroid-stimulating hormone (TSH)	2.54 mIU/L	0.27-4.2 mIU/L
Free T4	12.6 pmol/L	10-21 pmol/L
Insulin-like growth factor 1 (IGF-1)	19.2 nmol/L	9-27 nmol/L
9 AM serum cortisol	189 nmol/L	>300 nmol/L
Adrenocorticotropic hormone (ACTH)	38 ng/L	7-63 ng/L
Prostate-specific antigen (PSA)	0.6 µg/L	<2.5 µg/L
Hematocrit	0.40 L/L	0.40-0.54 L/L

TABLE 1: Initial biochemical investigations

*Reference ranges are those provided by the reporting laboratory.

A short Synacthen test was performed, which showed an increase in serum cortisol from 141 nmol/L at baseline to 619 nmol/L at 30 minutes and 656 nmol/L at 60 minutes, demonstrating an adequate adrenal response. ACTH was normal at 38 ng/L (reference range, 7-63 ng/L).

Pituitary magnetic resonance imaging (MRI) was nevertheless arranged because of the biochemical evidence of secondary hypogonadism and the presence of intermittent headache. Although the total testosterone concentration of 8.9 nmol/L was above the 5.2 nmol/L threshold cited in the Endocrine Society diagnostic pathway, that guideline also identifies new-onset headache and other symptoms or signs of mass effect as indications for pituitary imaging.

Contrast-enhanced pituitary MRI demonstrated a well-defined, smoothly marginated 25 × 28 × 27 mm sellar and suprasellar mass occupying the pituitary fossa and extending into the suprasellar cistern. The lesion was initially favored radiologically to represent a pituitary macroadenoma (Figure *1*). There was posterior erosion of the dorsum sellae, inferior erosion of the sellar floor, and left cavernous sinus extension with partial involvement of the cavernous internal carotid artery. Superior extension displaced the prechiasmatic optic nerves laterally and the optic chiasm superiorly. Formal ophthalmological assessment with Octopus perimetry showed no visual-field defect.

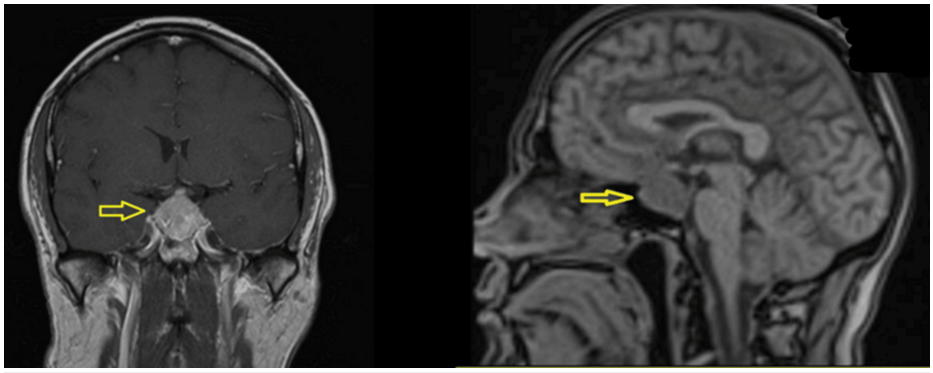

FIGURE 1: Preoperative coronal (left) and sagittal (right) pituitary MRI images demonstrating the sellar and suprasellar mass (arrows).

The case was reviewed at the pituitary multidisciplinary team meeting. Because of concern about the lesion's vascularity, computed tomography (CT) cerebral angiography was obtained. It confirmed the enhancing sellar and suprasellar mass and demonstrated a hypertrophied right meningo-hypophyseal trunk supplying the tumor. No other abnormal intracranial vessels were identified. The prominent feeding vessel was considered to confer a substantial risk of intraoperative hemorrhage (Figure *2*).

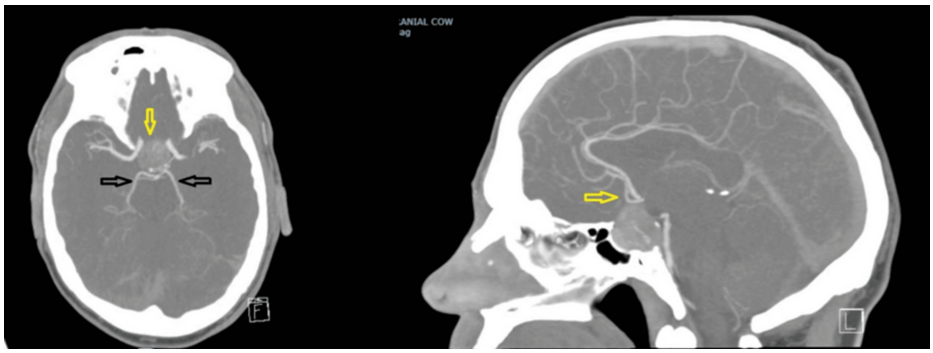

FIGURE 2: Axial computed tomography angiography (left) showing a pituitary mass (yellow arrow) abutting the right and left cavernous segments of the internal carotid arteries without significant narrowing (black arrow). The sagittal view (right) shows a hypertrophied meningo-hypophyseal trunk (yellow arrow).

The treatment options discussed were partial surgical debulking followed by radiotherapy or radiotherapy alone. After discussion of the potential benefits and risks, the patient elected to proceed with partial surgical debulking followed by radiotherapy.

Intramuscular testosterone undecanoate was commenced before surgery. Total testosterone subsequently increased from 8.9 to 12.4 nmol/L, with suppression of LH to <0.1 IU/L and FSH to <0.3 IU/L, consistent with the expected pharmacological effect of testosterone replacement.

At surgery, only minimal tissue could be obtained because of significant intraoperative bleeding, and complete resection was not attempted. Postoperative pituitary MRI showed persistent substantial residual tumor with no meaningful reduction in overall tumor volume (Figure *3*).

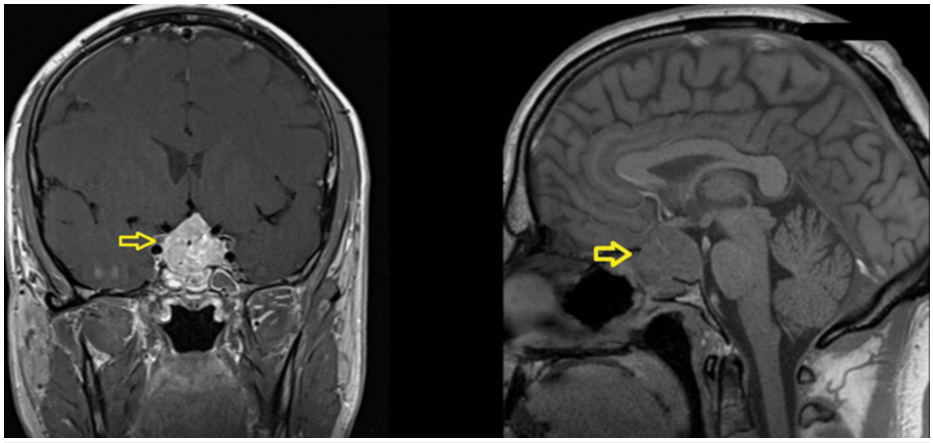

FIGURE 3: Postoperative coronal (left) and sagittal (right) views of pituitary MRI demonstrating persistent residual tumor (arrows).

Histopathological examination showed a tumor composed predominantly of bland spindle cells with focal epithelioid morphology and a suggestion of fascicular architecture. No readily identifiable mitotic figures or necrosis were seen. Immunohistochemistry demonstrated positivity for thyroid transcription factor 1 (TTF-1), glial fibrillary acidic protein (GFAP), S100, and CD56. Most tumor cells were negative for synaptophysin, chromogranin, and CAM5.2. The Ki-67 proliferative fraction was low. The morphologic and immunophenotypic findings supported the diagnosis of pituicytoma (Figure *4*). Granular pituicytoma and oncocytic pituicytoma/spindle cell oncocytoma were considered in the differential diagnosis; however, the specimen lacked prominent granular or oncocytic cytoplasmic features.

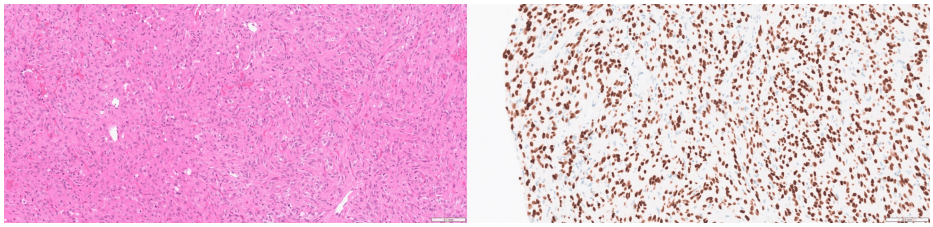

FIGURE 4: Histopathology images showing hematoxylin and eosin-stained section showing spindle cell tumor morphology (left). Immunostained section showing nuclear TTF1 staining (right).

Six weeks after surgery, repeat short Synacthen testing was done, which demonstrated inadequate adrenal response. Serum cortisol increased from 192 nmol/L at baseline to 292 nmol/L at 30 minutes and 334 nmol/L at 60 minutes (satisfactory response: >450 nmol/L at 60 min). The ACTH was at the lower end of the reference range at 13 ng/L (reference range, 7-63 ng/L). Secondary adrenal insufficiency was diagnosed, and hydrocortisone replacement was commenced. Prolactin, IGF-1, TSH, and free T4 remained within the laboratory reference ranges. LH and FSH remained suppressed during testosterone therapy (Table *2*). Postoperative visual-field assessment remained normal.

Investigation	Patient value	Reference range*
Prolactin	300 mIU/L	45-375 mIU/L
Follicle-stimulating hormone	<0.3 IU/L	1.6-11.0 IU/L
Luteinizing hormone	<0.1 IU/L	1.3-8.3 IU/L
Thyroid-stimulating hormone	1.70 mIU/L	0.27-4.2 mIU/L
Free thyroxine	13.0 pmol/L	10-21 pmol/L
Insulin-like growth factor 1	24.8 nmol/L	9-27 nmol/L
Adrenocorticotropic hormone	38 ng/L	7-63 ng/L

TABLE 2: Postoperative pituitary function

*Reference ranges are those provided by the reporting laboratory.

Luteinizing hormone (LH) and follicle-stimulating hormone (FSH) were measured while the patient was receiving testosterone replacement.

The case was subsequently re-discussed at the pituitary multidisciplinary team meeting. Given the substantial residual tumor, the marked vascularity, and the limited tissue obtained at surgery, repeat surgery was not recommended. The patient was referred back to oncology, with a plan for six weeks of radiotherapy followed by MRI brain imaging at four months to assess treatment response.

Discussion

The principal diagnostic challenge in this case was the close clinical and radiological resemblance between pituicytoma and a non-functioning pituitary macroadenoma. Pituicytomas are rare tumors of the posterior pituitary and infundibular region and may present with headache, visual symptoms, and anterior pituitary dysfunction [2-4]. MRI commonly shows a solid, strongly enhancing lesion, and imaging findings may overlap substantially with those of PitNETs and other sellar masses [2,4]. In the present case, the lesion was therefore reasonably interpreted preoperatively as a macroadenoma, and the definitive diagnosis was established only after limited tissue sampling.

The endocrine presentation is also notable. The patient had symptomatic secondary hypogonadism with low total testosterone and inappropriately normal gonadotropins, while most other pituitary axes were initially preserved. This pattern can occur with partial hypothalamic-pituitary dysfunction and does not by itself distinguish pituicytoma from other sellar lesions. The Endocrine Society guideline identifies severe secondary hypogonadism, panhypopituitarism, persistent hyperprolactinemia, or symptoms/signs of mass effect such as new-onset headache as situations in which pituitary MRI should be performed [8]. In this case, headache provided an additional clinical reason for imaging despite the testosterone concentration being above 5.2 nmol/L.

Marked vascularity is a recognized feature of pituicytoma and can substantially complicate surgical resection [3,5,6]. In the present case, CT angiography demonstrated a hypertrophied right meningo-hypophyseal trunk supplying the lesion, providing important preoperative information that influenced the surgical strategy. The subsequent intraoperative hemorrhage and limited tissue acquisition were consistent with the anticipated vascular risk. This experience supports obtaining detailed vascular imaging when a sellar or suprasellar lesion has imaging characteristics that raise concern for a hypervascular pituitary tumor.

Although gross-total resection is associated with lower reported recurrence or progression rates, the decision to pursue complete resection must be balanced against the potential for major hemorrhage and injury to the optic apparatus, cavernous sinus structures, pituitary stalk, or residual pituitary tissue [5,6]. In a pooled analysis of 115 patients, progression occurred in 4.9% of patients after gross-total resection compared with 35.2% after non-gross-total resection [6]. These figures are derived from retrospective and heterogeneous case-based literature and should not be interpreted as individualized risk estimates.

Histologically, pituicytoma is typically composed of bland spindle cells arranged in fascicles or other compact patterns, with low mitotic activity. TTF-1 nuclear positivity is a useful diagnostic marker of pituicyte lineage, while S100 and variable GFAP expression are also characteristic [4,7]. The immunophenotype in this case-TTF-1, GFAP, and S100 positivity with low Ki-67 activity-supported pituicytoma. The differential diagnosis includes other members of the pituicyte tumor family, particularly granular and oncocytic forms, as well as other spindle-cell lesions of the sellar region [1,4,7].

The limited specimen in this case is an important diagnostic consideration. Because substantial hemorrhage prevented extensive sampling, the diagnosis relied on the combination of morphology and immunophenotype rather than a large resection specimen. The absence of prominent granular or oncocytic features favored conventional pituicytoma, although the small sample necessarily limits assessment of intratumoral heterogeneity.

The optimal management of residual pituicytoma remains uncertain because evidence is limited to retrospective series and pooled case reports. Complete resection is generally associated with durable local control when it can be achieved safely, whereas residual disease carries a higher risk of subsequent progression [5,6]. Radiotherapy has been used for residual or recurrent disease, including after subtotal resection, but the available evidence is insufficient to define a universal postoperative radiation strategy [5,6]. In this case, the decision to plan radiotherapy reflected the substantial residual tumor and the high hemorrhagic risk associated with repeat surgery rather than an assumption that radiotherapy is required for all pituicytomas.

This report has the limitations inherent to a single case. The clinical course cannot establish the diagnostic yield of pituitary imaging in patients with secondary hypogonadism, determine the comparative effectiveness of surgery versus radiotherapy, or provide a generalizable estimate of recurrence risk. Follow-up after planned radiotherapy was not available in the source case at the time of manuscript preparation; therefore, long-term tumor control and endocrine outcome cannot be reported.

Conclusions

Pituicytoma should be included in the differential diagnosis of an enhancing sellar or suprasellar mass accompanied by pituitary dysfunction, particularly when imaging or operative findings suggest marked vascularity. In patients with secondary hypogonadism, the presence of headache or other features suggestive of mass effect may justify pituitary imaging even when testosterone is above the threshold commonly used as an indicator for pituitary imaging.

Preoperative vascular assessment can materially influence surgical planning in suspected hypervascular sellar tumors. When complete resection is unsafe because of hemorrhagic risk or critical neurovascular relationships, management should be individualized through a multidisciplinary pituitary team, with consideration of radiotherapy and long-term radiological and endocrine surveillance.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Kanza Wasim, Waqar Ahmad

Acquisition, analysis, or interpretation of data: Kanza Wasim, Otite Akpovona, Waqar Ahmad

Drafting of the manuscript: Kanza Wasim, Otite Akpovona

Critical review of the manuscript for important intellectual content: Kanza Wasim, Otite Akpovona, Waqar Ahmad

Supervision: Waqar Ahmad

Disclosures

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