

hormone (12.1 mIU/mL) with normal testosterone levels. Scrotal ultrasound showed bilateral testicular involution (right: 8.66 mL, left: 7.21 mL), contradicting the expected macroorchidism of FGPA. Although the patient felt his vision was normal, a confrontation test revealed temporal visual field defects, indicating chiasmatic compression. This case presents a striking clinical discordance where tumor-induced gonadotropin hypersecretion provided a supra-physiological drive to the involuting testes, maintaining testosterone levels and masking andropause. The primary indication for transsphenoidal surgery is the introduction of evidence of visual field defects. However, removing the adenoma will abruptly eliminate this “hormonal drive.” Postoperative management will focus on rigorous hormonal monitoring. Testosterone replacement therapy is not immediately indicated; rather, it will be carefully planned only if postoperative evaluations demonstrate a significant hormonal decline accompanied by symptomatic hypogonadism. This tailored approach ensures that the benefits of intervention outweigh the potential risks in an elderly patient.

CONCLUSION

In elderly patients, FGPAs can mask age-related hormonal decline. In this case, clinicians must treat the patient, not just the tumor. Anticipating a potential decline in postoperative stamina is as crucial as the surgery itself. Comprehensive hormonal evaluation and a personalized approach to therapy are essential to preserve quality of life after the “masking” effect is removed.

EP_A119

WHEN HYPERPROLACTINEMIA FAILS TO SUPPRESS: THE SILENT GONADOTROPH IN A PITUITARY MACROADENOMA

Min Jing Choo¹ and Liang Wei Wong²

¹Department of Internal Medicine, Hospital Taiping

²Endocrine Institute, Hospital Putrajaya

INTRODUCTION

Pituitary macroadenomas may present with mass effects, hypopituitarism, or hormone hypersecretion. Hyperprolactinemia, resulting from a prolactin-secreting tumor or stalk compression, typically suppresses gonadotropins. Thus, elevated follicle-stimulating hormone (FSH) and luteinizing hormone (LH) with low testosterone in this context are unusual. We report a macroprolactinoma with a clinically non-functioning gonadotroph adenoma.

CASE

A 69-year-old male presented with acute giddiness and headache while in Vietnam. He reported reduced libido but no visual symptoms or galactorrhea. Examination showed no neurological deficits, normal visual fields, and secondary sexual characteristics; bilateral testes volume of 25 mL. Magnetic resonance imaging (MRI) brain revealed a 1.6 × 2.2 × 2.1 cm pituitary macroadenoma compressing the optic chiasm and pituitary stalk.

Initial pituitary evaluation demonstrated hyperprolactinemia (prolactin >200 µg/L; normal 2.4–13.1), central hypothyroidism (T4 6.8 pmol/L [7.8–14.4], thyroid-stimulating hormone 2.3 mIU/L [0.38–5.3]), elevated FSH (21.5 IU/L [1.2–19.2]) and LH (191 IU/L [1.24–8.62]), low testosterone (8.88 nmol/L), normal insulin-like growth factor 1 (IGF-1) (68 µg/L [46.5–191.9]), and cortisol 237 nmol/L. Cabergoline was initiated at 0.25 mg twice weekly and titrated to 1 mg twice weekly over 4 months, alongside levothyroxine 25 µg daily.

Repeated MRI brain 2 months after cabergoline initiation showed a persistent macroadenoma (1.7 × 2.2 × 2.2 cm) with bilateral cavernous sinus extension. Prolactin decreased to <170 µg/L, central hypothyroidism persisted; levothyroxine was optimized, and hydrocortisone was initiated.

At the 5-month follow-up, prolactin further improved to 57 µg/L. Central hypothyroidism and borderline adrenal function persisted, requiring continued replacement therapy. Testosterone remained low-normal (10.2 nmol/L) despite elevated FSH (15.4 IU/L) and LH (92 IU/L). Follow-up MRI and hormonal reassessment were planned in May 2026.

CONCLUSION

Profoundly elevated prolactin level >200 µg/L and response to cabergoline reflect true prolactinoma rather than stalk effect. Persistently low testosterone with discordant high FSH/LH suggests a non-functioning gonadotroph component producing biologically inactive gonadotropins. Definitive diagnosis requires histopathological confirmation, while serial biochemical and radiological follow-up guides management and clarifies tumor subtype.