

Her clinical course was complicated by severe hospital-acquired infection leading to septic shock, requiring intensive care support, mechanical ventilation, and renal replacement therapy. She was commenced on desmopressin, with subsequent clinical and biochemical improvement, alongside multidisciplinary management.

CONCLUSION

In patients with type 2 diabetes mellitus, persistent polyuria may obscure an underlying water balance disorder. This case highlights the importance of considering alternative causes of polyuria and a systematic endocrine approach to ensure accurate diagnosis and appropriate management.

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RARE CASE: EMPTY SELLA SYNDROME, GROWTH HORMONE DEFICIENCY, AND HASHIMOTO'S THYROIDITIS IN THALASSEMIA SPECTRUM

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INTRODUCTION

Thalassemia is a hemoglobin synthesis disorder associated with chronic anemia and transfusion-related iron overload, which predisposes patients to multiple endocrine complications. Iron deposition in the pituitary and gonads may result in growth hormone deficiency (GHD) and hypogonadism. Empty sella syndrome (ESS), characterized by herniation of the subarachnoid space into the sella turcica, may also contribute to hypopituitarism. The coexistence of thalassemia-related endocrinopathies, ESS, and autoimmune thyroid disease is rare and presents significant diagnostic complexity.

CASE

A 27-year-old female with transfusion-dependent thalassemia on deferiprone presented with secondary amenorrhea and short stature. Her height was 145 cm (below the target range of 140.5–157.5 cm), body mass index 17.3 kg/m², and Tanner stage M3P1.

Laboratory evaluation revealed elevated thyroid-stimulating hormone (10.92 mIU/L) with low-normal free thyroxine 4 (11.4 pmol/L), consistent with primary hypothyroidism. Thyroid ultrasound demonstrated

features of chronic thyroiditis, and anti-thyroid peroxidase antibodies (8.18 IU/mL) supported a diagnosis of Hashimoto's thyroiditis. Insulin-like growth factor-1 was markedly reduced (68 ng/mL), indicating GHD. Morning cortisol was within normal range (14.5 µg/dL). Gonadotropins were inappropriately low-normal (follicle-stimulating hormone 7.42 mIU/mL, luteinizing hormone 11.41 mIU/mL) with low estradiol (26.49 pg/mL), suggesting hypogonadotropic hypogonadism.

Skeletal survey showed thalassemia-related bone changes, including trabecular coarsening and metaphyseal widening, with a predicted adult height of 142.7 cm. Pituitary magnetic resonance imaging revealed an empty sella.

CONCLUSION

Thalassemia must be recognized not only as a primary hematologic condition but also as a complex multisystem disorder with profound endocrine implications. Furthermore, the co-occurrence of these conditions with rare manifestations such as ESS and Hashimoto's thyroiditis underscores the diagnostic complexity faced by clinicians. Therefore, early detection and rigorous, regular endocrine screening are essential to optimize management strategies and improve the long-term quality of life for patients with thalassemia.

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THYROID-STIMULATING HORMONE (TSH)-SECRETING MACROADENOMA PRESENTING WITH RECURRENT ATRIAL FIBRILLATION IN FAILURE

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INTRODUCTION

Accounting for less than 2% of all pituitary adenomas, TSH-secreting pituitary adenomas (TSHoma) are an uncommon cause of hyperthyroidism. Majority are macroadenomas with delayed diagnosis as most patients are unwittingly treated for primary hyperthyroidism. Recurring discordant thyroid function test (TFT) with elevated thyroid-stimulating hormone (TSH) and Free T4 is a hint and warrants additional investigation to facilitate diagnosis.

CASE

We report a case of a 48-year-old female who was treated for primary hyperthyroidism since her late 20s with multiple admissions for recurrent congestive heart failure

and atrial fibrillation. The cardiac issue was preceded by worsening thyrotoxicosis. Previous thyroid autoantibodies were negative. Of note, she had recurring discordant TFT results from two different assays (TSH:84.7, free thyroxine 4 [FT4]:75.43) (TSH:37.86, FT4:27.34) during admission, excluding assay interference and prompting toward TSHoma or Resistance to Thyroid Hormone (RTH). Examination revealed a large goiter (10 × 8 cm) hard in consistency, and pansystolic murmur over tricuspid area. No thyroid eye disease nor bitemporal hemianopia or clinical sign of acromegaly.

Echocardiography showed dilated left atrium and mild to moderate tricuspid regurgitation with preserved ejection fraction. Thyroid-releasing hormone (TRH) stimulation test demonstrated blunted TSH response confirming TSHoma. Anterior pituitary hormone profile revealed normal insulin-like growth factor-1 level with suppressed sex hormones and prolactin, thus excluding co-secreting hormone. Sex hormone-binding globulin level, α -subunit, and T3 suppression test were not done due to unavailability. Magnetic resonance imaging pituitary uncovered pituitary mass measuring (2.6 × 3.3 × 2.2 cm) suggestive of macroadenoma with encasement of cavernous internal carotid arteries and cavernous sinus compression. Computed tomography neck revealed diffuse thyroid enlargement with compressive mass effects onto adjacent structure with trachea narrowing. After discussing with a multidisciplinary team, we planned her for total thyroidectomy followed by transsphenoidal surgery.

CONCLUSION

Late presentation and diagnosis in TSHoma remain a major challenge. TFT interpretation is fundamental in identifying the causes of secondary hyperthyroidism to avert detrimental sequelae and to guide optimal treatment.

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SMALL LESION, BIG IMPACT: EUS LOCALIZATION AND ABLATION OF A CT-OCCULT INSULINOMA

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INTRODUCTION

Insulinoma is a rare functioning pancreatic neuroendocrine tumor and the most common cause of endogenous hyperinsulinemic hypoglycemia. Although biochemical confirmation is usually straightforward, tumor localization

may be difficult when lesions are small and not detected on conventional cross-sectional imaging. In such cases, endoscopic ultrasound (EUS) plays an important role in identifying occult lesions and facilitating definitive treatment.

CASE

A 52-year-old female was admitted in April 2025 with recurrent seizures secondary to hypoglycemia for 3 years, with increasing frequency over time. She fulfilled Whipple's triad, with documented capillary glucose of 1.8 mmol/L during an episode and symptom resolution following glucose administration. A supervised prolonged fasting test confirmed endogenous hyperinsulinemic hypoglycemia, with plasma glucose 1.4 mmol/L, insulin 122 pmol/L, and C-peptide 1,010 pmol/L. Short Synacthen test demonstrated adequate adrenal reserve. Due to persistent hypoglycemia, she required high-dose diazoxide.

Contrast-enhanced computed tomography abdomen did not reveal a pancreatic lesion but incidentally detected a right ovarian teratoma. She underwent total abdominal hysterectomy and bilateral salpingo-oophorectomy in June 2025, with histopathology confirming a mature cystic teratoma without malignancy. However, hypoglycemic episodes persisted. Further evaluation with EUS in July 2025 identified a highly vascular isoechoic 8 × 8 mm lesion in the pancreatic body. Fine-needle biopsy confirmed a well-differentiated neuroendocrine tumor (WHO grade 1) with Ki-67 index of 2%. She subsequently underwent EUS-guided radiofrequency ablation in August 2025. Follow-up EUS in December 2025 showed post-ablation change, and her hypoglycemic episodes resolved completely, allowing diazoxide to be discontinued.

CONCLUSION

This case highlights the diagnostic challenge of occult insulinoma in the presence of negative conventional imaging. EUS was pivotal for tumor localization and tissue diagnosis, while EUS-guided radiofrequency ablation provided effective minimally invasive treatment in a carefully selected patient.