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BIOCHEMICAL DISCORDANCE IN ACROMEGALY COMPLICATED BY PITUITARY APOPLEXY AND SEVERE INSULIN RESISTANCE

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INTRODUCTION

Acromegaly is usually diagnosed by elevated age- and sex-adjusted insulin-like growth factor-1 (IGF-1) levels reflecting chronic growth hormone (GH) excess. IGF-1 is preferred as a screening biomarker due to its longer half-life and reduced pulsatility compared with GH. However, IGF-1 levels may be disproportionately low or only modestly elevated in certain clinical contexts, leading to diagnostic uncertainty. Pituitary apoplexy is one such condition in which acute tumor hemorrhage or infarction may disrupt sustained GH secretion and attenuate IGF-1 production.

CASE

A 48-year-old female with hypertension, type 2 diabetes mellitus, and dyslipidemia presented with a 2-day history of severe headache, vomiting, and visual disturbance, on a background of progressive acral enlargement over 2 years. Examination revealed coarse facial features, prognathism, enlarged hands, and cranial nerve involvement. Magnetic resonance imaging demonstrated an invasive sellar–suprasellar pituitary macroadenoma with optic chiasmal compression and cavernous sinus encasement. Intravenous dexamethasone was initiated pre-operatively due to a significant mass effect.

Biochemical evaluation showed markedly elevated random GH levels (>50 ng/mL) with only mildly elevated IGF-1 at 1.19 times the upper limit of normal, below the threshold at which confirmatory oral glucose tolerance testing may be omitted according to current guidelines. Other pituitary axes suggested evolving hypopituitarism. During admission, she developed severe hyperglycemia with marked insulin resistance, requiring high-dose insulin therapy (approximately 1.5 U/kg/day). She underwent urgent transsphenoidal surgery, with histopathology confirming a pituitary neuroendocrine tumor with extensive hemorrhage and infarction, consistent with pituitary apoplexy. Postoperatively, GH levels were suppressed to <5 ng/mL, insulin requirements decreased markedly, and hormone replacement was initiated for secondary adrenal insufficiency and central hypothyroidism.

CONCLUSION

This case highlights that IGF-1 levels below conventional diagnostic thresholds do not exclude clinically significant acromegaly, particularly in the setting of pituitary apoplexy. Integration of clinical phenotype, GH levels, and imaging findings is essential to avoid diagnostic delay and ensure timely management.

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GROWTH AGAINST THE CLOCK: HORMONAL THERAPY IN LATE-DIAGNOSED MOSAIC TURNER SYNDROME

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INTRODUCTION

Short stature and delayed puberty characterize Turner syndrome (TS). The 2024 international clinical practice guidelines recommend growth hormone (GH) for late-diagnosed patients if epiphyses remain open. For patients with remaining growth potential, initiating GH alongside low-dose estrogen effectively balances linear growth with the need for timely pubertal induction.

CASE

A 15-year-old female, born prematurely at 6 months gestation, presented with delayed puberty, primary amenorrhea, and short stature. Examination revealed a height of 122 cm (<5th percentile), weight of 26 kg, with no syndromic facies, and Tanner stage 1. Investigations confirmed hypergonadotropic hypogonadism. Metabolic screening, including thyroid, renal, and liver profile, was normal. Her baseline insulin-like growth factor 1 (IGF-1) was low at 117.5 ng/mL (127.5–541.5). Karyotyping confirmed mosaic TS (45,X/46,Xr). Her skeletal bone age was delayed at 12 years, indicating a viable window for linear growth prior to complete epiphyseal fusion.

Subcutaneous GH was initiated at 0.3 mg up titrated to 1.2 mg (0.45 µg/kg) daily over 4 weeks, then 1.35 mg (50 µg/kg) daily at month 5. Low-dose oral estradiol (0.5 mg three times weekly) was introduced for pubertal induction at month 4. After 9 months of combined GH and estrogen therapy, the patient achieved a height increment of 6 cm, reaching 128 cm without an adverse event. To achieve the clinical target of a 10–15 cm increment in the first year, her GH dose was further increased to 1.50 mg (55 µg/kg) daily. She showed an appropriate biochemical response with IGF-1 increased to 40.2 nmol/L (16.4–67.8) with a total height gain of 6 cm over the first 9 months of GH therapy.