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BEWARE OF TALL STATURE: GROWTH HORMONE EXCESS ASSOCIATED WITH A RARE PARASELLAR EXTRA-AXIAL TUMOUR—DIAGNOSTIC AND MANAGEMENT CHALLENGES IN A RESOURCE-LIMITED SETTING

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ABSTRACT

Introduction: Tall stature can be the sole indicator of growth hormone (GH) excess, which causes pituitary gigantism before epiphyseal fusion. In developing regions like Indonesia, a lack of systematic screening often delays diagnosis. While classic presentations involve accelerated linear growth, atypical cases may be dominated by neurological signs, such as seizures secondary to tumour mass effect.

Case description: A 14-year-old boy presented with a 2-year history of rapidly accelerating growth velocity, increasing shoe size, and a 6-month history of severe morning headaches accompanied by drug-resistant focal seizures. Endocrine evaluations revealed significantly elevated basal GH (4.27 ng/ml) and insulin-like growth factor 1 (IGF-1) levels (536 ng/ml). Brain magnetic resonance imaging identified a large parasellar extra-axial tumour with suprasellar extension. The patient underwent a craniotomy and tumour excision; histopathology subsequently confirmed a rare, aggressive malignant mesenchymal tumour. Postoperatively, the patient achieved seizure resolution and normalization of GH and IGF-1 levels. However, he developed evolving pituitary dysfunction consistent with multiple pituitary hormone deficiency (MPHD), specifically central hypothyroidism, necessitating targeted hormonal replacement therapy and continuous clinical follow-up.

Conclusion: GH excess and gigantism can arise secondary to parasellar pathology rather than intrinsic pituitary disease, with neurological features like refractory epilepsy occasionally preceding classic endocrine manifestations. Rapid adolescent growth paired with neurological signs warrants urgent structural neuroimaging. Meticulous perioperative pituitary monitoring and early multidisciplinary involvement are essential to optimize long-term patient outcomes

KEYWORDS

Pituitary gigantism, growth hormone excess, refractory epilepsy, parasella extra-axial tumour

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LEARNING POINTS

- Pituitary gigantism can be driven by extra-axial parasellar masses rather than intrinsic pituitary adenomas, operating via mass-effect compression that disrupts hypothalamic somatostatin inhibition.
- Drug-resistant epilepsy and severe headaches caused by local tumour infiltration can dominate the clinical picture, meaning rapid adolescent growth paired with neurological symptoms mandates urgent magnetic resonance imaging of the brain.
- Postoperative multiple pituitary hormone deficiency often evolves silently over weeks or months, requiring lifelong, multidisciplinary surveillance to detect late-stage deficiencies like secondary adrenal insufficiency

INTRODUCTION

Tall stature is defined as a height greater than +2 standard deviations or >97th percentile for age, sex, and population reference[1]. While usually benign (constitutional or familial), it can be the earliest sign of underlying endocrine, genetic, or neoplastic disorders. Among endocrine aetiologies, growth hormone (GH) excess is rare but critical. GH hypersecretion prior to epiphyseal fusion results in pituitary gigantism, characterized by excessive linear growth, whereas post-fusion onset leads to acromegaly without height changes[2].

Epidemiologically, both conditions are exceedingly rare; acromegaly has an annual incidence of 2.8 to 4 cases per million population, while only several hundred cases of pituitary gigantism are documented worldwide. Approximately 10% of patients with acromegaly present with excessive tall stature, indicating hypersecretion initiated prior to epiphyseal closure[2]. Although over 95% of GH excess cases stem from GH-secreting pituitary tumours, rare ectopic or parasellar lesions can drive complex, atypical presentations[3].

Epidemiology data on tall stature and GH excess is scarce in developing countries like Indonesia, where the national growth surveillance system concentrates on undernutrition and stunting. Since tall stature is infrequently evaluated, it is often misconstrued as normal variation, resulting to under- or delayed GH excess diagnosis[4]. Most cases of GH excess may be caused by pituitary adenomas. This delay in diagnosis is due to a failure to detect an expanding cerebral mass early

Moreover, problems in systemic identification and treatment of cerebral tumour and late stage presentation have limited the management of cerebral tumour in Indonesia. Most publications have focused on big cities like Jakarta where 42% of cases of paediatric brain tumours are astrocytomas[5]. National epidemiological registries of these tumours are still scant and the real extent of the disease is masked. Patients with suspected lesions face many barriers to care, including unequal access to sophisticated neuroimaging a shortage of skull-base neurosurgeons outside of major metropolitan areas, and limited availability of therapeutic modalities and adjuvant therapies.

The clinical GH excess spans classic and atypical presentations. Rapid linear growth, acral and facial enlargement, headaches, visual problems, arthralgia, hyperhidrosis, and cardiometabolic issues are typical. Seizures and refractory epilepsy, rare but confirmed indications of tumour mass effects or parasellar lesions, might dominate the clinical picture[6].

We present a rare case of a child presenting with refractory seizures and extreme tall stature who was found to have a large parasellar extra-axial tumour with suprasellar extension, emphasising the necessity of thorough diagnostics in atypical GH excess.

CASE DESCRIPTION

A 14-year-old boy born to average-stature parents presented with a 2-year history of accelerated growth velocity and increasing shoe size, without early pubertal signs or a family history of overgrowth. Over the prior 6 months, he experienced daily, drug-resistant focal seizures

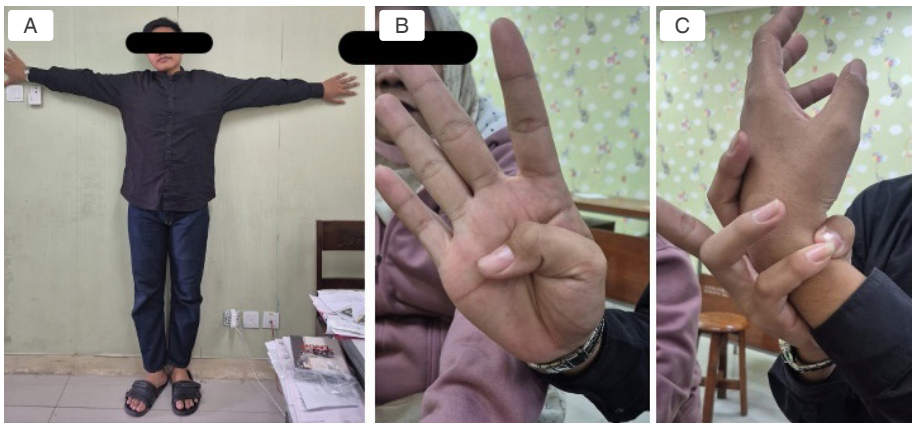


Figure 1. Physical examination of the patient. A) A full-body photograph demonstrates marked tall stature with increased arm span; B) thumb sign; C) wrist sign.

(up to three times daily, lasting 10–60 seconds). Episodes featured a prominent aura (headache, nausea, vomiting, and ictal premonition) followed by behavioural arrest, hand tremors, drooling, lip-smacking automatisms, and postictal somnolence with amnesia. Seizures worsened on combination therapy of carbamazepine, phenytoin, valproic acid and levetiracetam. The patient also reported severe morning headaches and non-alimentary vomiting, indicative of increased intracranial pressure.

Physical examination revealed disproportionate linear overgrowth with an increased arm span and a reduced upper-to-lower segment ratio *(Fig. 1)*. Centers for Disease Control and Prevention growth charts confirmed extreme tall stature (height >97th percentile) with a diverging trajectory; body mass index percentiles supported true linear overgrowth rather than obesity-related weight gain *(Fig. 2)*. Pubertal development, facial features, and skeletal alignments were age-appropriate, with no signs of arachnodactyly, scoliosis, or joint laxity *(Table 1)*. Longitudinal tracking over 2 years demonstrated a phase of rapid height velocity followed by a plateau *(Fig. 3)*. Neurological evaluation, including cranial nerve and visual field testing, was completely normal.

Endocrine and metabolic panels showed elevated basal GH (4.72 ng/ml; ref range: 0.02–3.81 ng/ml) and insulin-like growth factor 1 (IGF-1) levels (536 ng/ml; ref range: 117–507 ng/ml) *(Table 2)*. Biochemical analysis of the hormones confirmed central hypothyroidism, while the adrenal gland was still working properly. The first electroencephalogram (EEG) showed generalized intermittent slow activity suggestive of mild diffuse encephalopathy, whereas a second repeat EEG was normal. Skeletal maturation assessment revealed bone age in agreement with chronological age with open epiphyseal growth plates *(Fig. 4)*. Magnetic resonance imaging (MRI) of the brain revealed a large right sphenoid extra-axial mass measuring approximately 5.7 × 8.5 × 4.6 cm with suprasellar extension. The lesion was interpreted as an extra-axial parasellar tumour based on its radiological features and parasellar location, meningioma being the most likely differential diagnosis *(Fig. 5)*.

Parameter	Finding
Height (cm)	184
Weight (kg)	90
BMI-for-height (percentile)	90th–95th
Target parental height (cm)	164–181
Arm span (cm)	193
Upper/lower segment ratio	Decreased
Pubertal stage	Tanner G4P4
Testes (ml)	10/10
Urethral meatus	At penile tip
Chordee	Absent
Stretched penile length (cm)	7
Neurological examination	No focal deficit

Table 1. *Anthropometric and physical examination findings at endocrine evaluation.*
Abbreviations: BMI, body mass index.

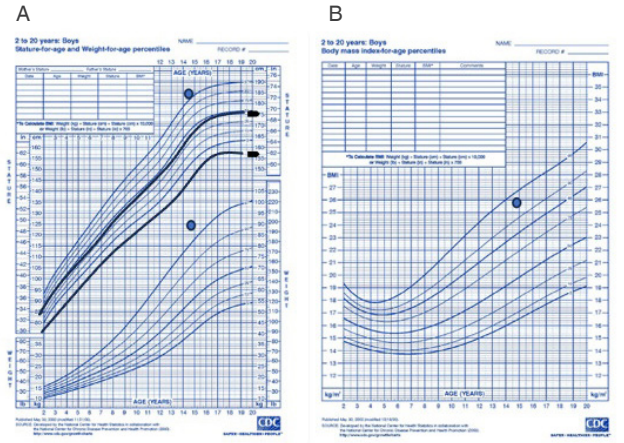


Figure 2. *Growth chart of the patient. A) Height and weight for age; B) Body mass index for age.*

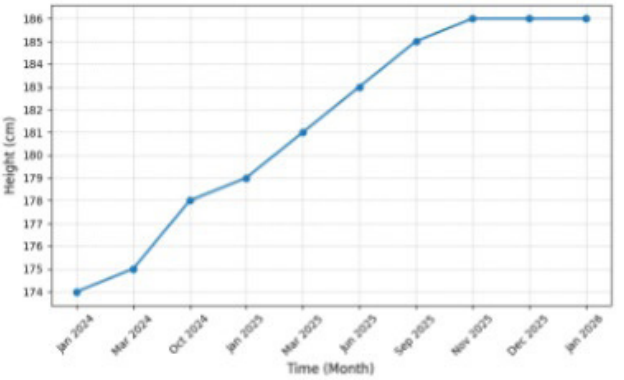


Figure 3. *Height of the patient.*



Figure 4. *Bone age of the patient.*

The patient had a craniotomy for tumour excision and decompression. Histopathology of the excised tissue showed a malignant mesenchymal tumour. Follow-up MRI

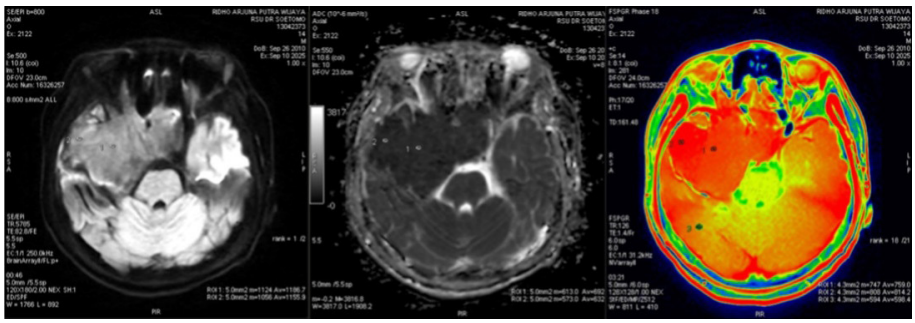


Figure 5. Brain MRI of the patient.

Test	Preoperative	Postoperative	Reference range
Basal growth hormone (ng/ml)	4.72	0.628	0.02–3.81
IGF-1 (ng/ml)	536	181	117–507
FT4 (ng/dl)	0.59	0.71	0.8–1.9
TSH (ultrasensitive) (µIU/ml)	2.879	2.726	0.7–5.7
Morning cortisol (ng/ml)	64.1	77.6	45.5–208.2

Table 2. Preoperative and postoperative laboratory findings. Abbreviations: IGF, insulin-like growth factor; FT4, free thyroxine; TSH, thyroid- stimulating hormone.

and angiography showed extensive local gliosis and a rim-enhancing seroma as postoperative inflammatory changes and residual or recurrent tumour masses involving the right temporal convexity and right cavernous sinus. Vascular complications consisted of a subacute thrombotic infarct in the right temporo-occipital region and long segment stenosis of the right internal carotid artery.

Postoperatively, the patient was stable and neurologically intact with complete resolution of headaches and seizures. Labs confirmed normalized serum GH (0,628 ng/ml) and IGF-1 (181 ng/ml) levels, despite persistent central hypothyroidism (free thyroxine: 0.71 ng/dl; ref range: 0.83–1.43; thyroid- stimulating hormone: 2.726 µIU/ml; ref range: 0.51–4.94) and mild hyponatremia (Na: 134.5; ref range: 135-145 mmol/l *(Table 2)*. Adjuvant radiotherapy was scheduled as the sole post-therapeutic management plan. Levothyroxine was initiated using an alternating regimen of 150 µg (Mondays, Wednesdays, Fridays) and 125 mg on the remaining days. In coordination with paediatric neurology, antiepileptic therapy was continued with phenytoin (200 mg twice daily), valproic acid (1000 mg twice daily), and levetiracetam (500 mg twice daily). The patient was closely followed by multidisciplinary team of paediatric endocrinology, neurology and neurosurgery.

DISCUSSION

This case illustrates an unusual presentation of pituitary gigantism and drug-resistant epilepsy due to a malignant mesenchymal parasellar tumour instead of a classical pituitary adenoma. Paediatric tall stature can be caused by benign familial variation, genetic overgrowth diseases like Beckwith-Wiedemann Syndrome, and uncommon endocrine problems such GH overload[2]. Indonesia, with limited access, has significant systemic challenges diagnosing these disorders. Because national healthcare networks tackle stunting, excessive linear growth is rarely analysed and frequently mistaken as favourable. Neuroimaging is delayed until advanced neurological signs like refractory epilepsy occur, creating this blind gap.

Over 95% of paediatric GH excess cases are primary adenomas, but this patient's MRI showed a structurally normal pituitary and a massive right sphenoid extra-axial mass with suprasellar extension[1,6]. Functional GH hypersecretion via hypothalamic disinhibition is hypothesised. Hypothalamic somatostatin normally inhibits GH secretion. These inhibitory mechanisms may have been disrupted by structural compression or tumour infiltration, causing somatotrophs to hypersecrete GH without an underlying pituitary lesion. The patient's central hypothyroidism, part of the global hypothalamic-pituitary axis dysfunction, supports this[7].

A distinguishing feature of this case is the dominance of neurological manifestations, specifically a 6-month history of drug-resistant focal epilepsy that escalated despite aggressive antiseizure polytherapy. Seizures are an exceedingly rare manifestation of GH excess and typically arise from the direct mass effect or local infiltration of a parasellar tumour into adjacent temporal structures[6]. Chronological analysis demonstrated that rapid linear growth preceded the onset of epilepsy, mapping a clear timeline of tumour progression from subclinical endocrine dysregulation to macro-structural neurological irritation[8].

Managing GH excess from and aggressive parasellar lesion requires a multidisciplinary, integrated approach that addresses simultaneously the tumour mass, endocrine deficits and neurological symptoms. Surgical resection is the first-line intervention for tissue decompression, refractory seizure control, and definitive histopathological diagnosis. This patient's histopathology showed a malignant mesenchymal tumour with extremely infiltrative growth, high recurrence, and secondary vascular problems. On follow-up imaging, postoperative internal carotid artery stenosis and localised infarction showed how aggressive this cancer is.

Central hypothyroidism and abnormal mineral metabolism were due to multiple pituitary hormone deficiency (MPHD) as a consequence of the mechanical and inflammatory injury of tumour removal. This serious dysfunction is due to tumour compression, vascular compromise and surgical manipulation and postoperative inflammation in the delicate hypothalamic-pituitary region[9]. The weak neuronal and circulatory connections of the pituitary stalk are susceptible to ischemia or strain injury after surgery, therefore hypothalamic regulatory disruption commonly occurs over weeks to months. Thus, even if a patient's adrenal function was initially normal, late-stage secondary adrenal insufficiency can develop.

The patient is at significant risk for delayed-onset MPHD. Thus, regular monitoring allows for the timely beginning and exact adjustment of targeted hormone replacement therapy and prevents life-threatening adrenal crises by verifying adrenal reserve[1]. Beyond neurosurgery, parasellar tumours have complex endocrine and oncological effects that require continuing therapeutic monitoring and multidisciplinary care[10]. Prognosis is a long-term emotional, financial, and logistical hardship on families. To manage complex home therapies and follow-up care, family-centred assistance, education, and coordination are essential.

In conclusion, parasellar disease may cause GH excess and gigantism and neurological symptoms such as seizures may precede endocrine problems. Early multidisciplinary perioperative management enhances diagnostic precision and outcomes. The use of standardised growth velocity charts and early baseline assessment for tertiary referral may help reduce the delay in diagnosis by primary care providers.

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