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MRI Findings of the Pituitary Gland in Children with Growth Hormone Deficiency in Sulaimaniyah City – Iraq

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ABSTRACT

Abstract

Introduction:

One of the most common referrals to pediatric endocrinology clinics is for short stature, with the aim of ruling out growth hormone deficiency (GHD). This study aims to investigate MRI Findings of the Pituitary Gland in Children with GHD, in Sulaimaniyah City – Iraq.

Materials and Methods:

A retrospective cross-sectional study was conducted at Dr. Jamal Ahmed Rashid Pediatric Teaching Hospital, including 90 children (aged 2–16 years) diagnosed with GHD between 2020 and 2024. Demographic, clinical, hormonal, and MRI data were collected and analyzed using SPSS v28.0.

Results:

Demographic information of 90 children showed that Mean ± SD age of children was 9.47 ± 2.68 years. In terms of sex, 42 (46.7%) children were male and 48 (53.3%) children were female. The mean ± SD age at diagnosis of GHD in the children studied was 6.80 ± 1.92 years. MRI findings showed that the pituitary gland in the pediatric sample was normal in 77 (85.6%) cases. In 3 (3.3%) subjects pituitary hypoplasia was identified. In addition, mixed anomalies including pituitary hypoplasia in association with ectopic posterior pituitary, and pituitary microadenoma were also found in 2 (2.2%) children. MRI findings (brain abnormalities) were normal in 83 (92.2%) children, structural abnormalities in the brain were seen in 5 (5.6%) and midline defects were seen in 2 (2.2%) children.

Conclusions:

Most children with GHD had normal pituitary MRI, but structural abnormalities correlated with more severe hormonal deficiency and earlier, more severe clinical presentation. MRI is a valuable adjunct but should be interpreted alongside clinical and biochemical assessments.

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Introduction

One of the most common referrals to pediatric endocrinology clinics is for short stature, with the aim of ruling out growth hormone deficiency (GHD). Short stature has non-endocrine or endocrine causes, including GHD (1). GHD is a syndrome with various causes that leads to changes in growth, body composition, and metabolic changes (2). GHD, defined as suboptimal GH secretion, includes a group of disorders with different causes. GH is produced in a pulsatile manner from somatotrope cells in the anterior pituitary, peaking at night during sleep. GH secretion is controlled by growth hormone-releasing hormone (GHR) from the hypothalamus and somatostatin from the pituitary gland (1).

GHD can be congenital, genetically and/or due to malformation, or acquired (due to trauma, tumor, brain infection, inflammation, or radiotherapy), scattered or familial, independently or in association with other pituitary hormone deficiencies (including multiple pituitary hormone deficiencies (MPHD)) (3), and temporary or permanent, however in most cases, isolated GHD (IGHD) occurs idiopathically (4).

The incidence of GHD varies considerably between countries, and precise incidence rates based on standard criteria have not yet been established. However, the most common prevalence of short stature due to GHD in childhood has been reported to be between one in 3,000 and one in 10,000 live births (5). IGHD is the most common type of hypopituitarism, with an incidence of between 1/4,000 and 1/10,000 live births (5). The incidence of GHD has been reported in more recent studies to range from 1:5000 to 18,646, indicating a relatively constant incidence rate (6).

In children, the GHD diagnosis is made through physical examinations, medical history, biochemical and histological examinations, radiological assessment of skeletal maturation, pituitary neuroimaging (7), and genetic analysis (4). GHD diagnosis in infancy can be confirmed by measuring GH, especially during a period of hypoglycemia, from the infant's plasma or serum during the first week of life (8). However, GH secretion may be affected by factors such as sex, age, obesity, puberty, and malnutrition (9). In cases where biochemical tests are positive, brain magnetic resonance imaging (MRI) should be performed (4).

The advent of MRI has significantly improved the understanding of the pathogenesis of disorders affecting the hypothalamic-pituitary region, especially congenital hypopituitarism (10). MRI enables detailed and anatomical examination of the pituitary gland, distinguishing between the anterior and posterior lobes of the pituitary gland. MRI identification of pituitary hypertrophy in the posterior sella, which indicates functional integrity of the neurohypophysis, provides significant findings for the diagnosis and understanding of anterior and posterior pituitary diseases (11).

GHD results in growth retardation, short stature, metabolic changes, and if left untreated, is associated with significant psychological, social, and physical complications (12, 13). The diagnosis of GHD is based on a combination of clinical, laboratory, and imaging evaluations. Among them, MRI plays a significant role in examining the anatomical structure of the hypothalamus-pituitary axis and can identify congenital abnormalities such as pituitary hypoplasia, thin pituitary stalk, ectopic posterior pituitary, and acquired lesions such as tumors, cysts, inflammatory, and vascular lesions. Many studies have investigated the relationship between MRI findings and clinical manifestations in children with GHD. However, epidemiological and imaging data in these patients are limited and scattered in specific regions such as Sulaymaniyah, in the Kurdistan Region of Iraq. Investigating the imaging characteristics of the pituitary in children with GHD in this region can help to more accurately understand the morphological and pathophysiological patterns of this disorder. Also, recognizing MRI abnormalities can play an effective role in differentiating between acquired and congenital causes of GHD, determining prognosis, and even making treatment decisions. This is the first attempt at systematic characterization of pituitary magnetic resonance imaging findings in children with biochemically confirmed growth hormone deficiency living in

Sulaimaniyah, Iraq, a place where published material on pediatric hypothalamic-pituitary imaging is scarce. Given the lack of endocrine and neuroimaging services in this environment, our study provides invaluable baseline radiology information that can be used to shape local diagnostic regimens and can guide clinical decision-making. The key objective of the research was to define the range of structural malformations of the pituitary gland and the surrounding cerebral structures on MRI in children with isolated or multiple growth hormone deficiency and to determine the correlation between the radiological results and the related clinical, auxological, and hormonal variables at the time of the first diagnosis.

Material and Methods

Study setting

The study was conducted at Endocrinology Unit, Dr. Jamal Ahmed Rashid Pediatric Teaching Hospital, Sulaimaniyah, Iraq, a tertiary care facility serving a diverse pediatric population.

Study design

Retrospective observational cross-sectional study design was employed aiming to analyze pituitary MRI findings in children diagnosed with GHD in Sulaimaniyah, Iraq. This design was chosen to document and classify the spectrum of pituitary gland abnormalities on MRI in children diagnosed with GHD.

Study population

The study population included pediatric patients (aged 2 to 16 years) who were referred to Endocrinology Unit, Dr. Jamal Ahmed Rashid Pediatric Teaching Hospital in Sulaimaniyah, Iraq for evaluation of short stature and were subsequently diagnosed with GHD based on clinical and biochemical criteria and underwent pituitary MRI during 2020–2024.

A non-probability consecutive sampling method was employed, whereby all children meeting the inclusion criteria within the defined study period were included. The sample size of 90 was determined by the total number of eligible cases identified during the six-year study period.

Inclusion criteria

Children were included in the study if they had a confirmed diagnosis of GHD based on standard hormonal stimulation tests (such as insulin tolerance test or clonidine stimulation test), availability of high-resolution pituitary MRI, and complete clinical and laboratory documentation.

Exclusion criteria

Exclusion criteria were strictly applied to minimize confounding factors. Children were excluded if they had secondary causes of GHD, such as a history of cranial trauma, brain tumors, or previous neurosurgical interventions. Additionally, children with incomplete imaging or clinical data, as well as those with syndromic features or multi-endocrine disorders (unless specified), were excluded from the study.

Data collection

Data collection for this study was conducted through a comprehensive review of patient medical records, radiology reports, and laboratory databases. All children diagnosed with growth hormone deficiency (GHD) who underwent pituitary MRI at Sulaimaniyah Teaching Hospital between January 2018 and December 2023 were identified. Demographic information, including age, sex, and place of residence, was extracted from patient case files and electronic health records. Clinical data such as height, weight, height standard deviation score (SDS), bone age, and growth velocity at diagnosis were obtained from growth charts and clinical notes maintained by the pediatric endocrinology team.

Hormonal profiles were collected from laboratory records, focusing on peak growth hormone (GH) levels during stimulation tests, insulin-like growth factor 1 (IGF-1) levels, and other relevant pituitary hormone measurements. The age at diagnosis, age at initiation of treatment, and follow-up outcomes were also documented to provide a comprehensive clinical overview. All data were recorded using a standardized data collection form to ensure consistency and accuracy across all cases.

Radiological data were obtained from high-resolution pituitary MRI scans, which were reviewed and interpreted by experienced pediatric radiologists. Every MRI pituitary examination was conducted with a 1.5T Siemens MAGNETOM Avanto scanner (Siemens Healthineers, Erlangen, Germany), which was equipped with a specifically

eight-channel head coil. The imaging protocols included sagittal and coronal T1-weighted spin-echo (TR/TE: 500/12ms; slice thickness: 3mm; no gap), coronal T2-weighted turbo spin-echo (TR/TE: 3500/100ms; slice thickness: 3mm) and axial T2-weighted FLAIR T2 sequences to evaluate the related cerebral parenchymal abnormalities. Pre-contrast T1-weighted images were obtained in sagittal and coronal planes in each scenario. Post-contrast images were taken in the same planes after intravenous injection of gadoterate meglumine (Dotarem 0.1 mmol/kg) at a concentration of 0.1mmol/kg. Dynamic contrast-enhanced imaging was not routinely done, but in two suspected cases of pituitary stalk interruption, delayed imaging at 5 and 10 minutes after injection was also added to look at an ectopic posterior pituitary bright spot. Two pediatric radiologists who were 8 and 12 years experienced interpreted all the examinations, and the readers were not informed of the hormonal condition of the patients. MRI findings were classified as normal or abnormal, with abnormal findings further categorized into specific structural abnormalities such as pituitary hypoplasia, ectopic posterior pituitary, pituitary stalk interruption syndrome (PSIS), empty sella, and other relevant anomalies. The size and morphology of the anterior pituitary gland, integrity and thickness of the pituitary stalk, and the position and signal intensity of the posterior pituitary were systematically assessed according to established pediatric radiological criteria. All collected data were anonymized and entered into a secure database for subsequent statistical analysis.

Ethical consideration

Ethical approval was obtained from the University of Sulaimani Ethics Committee, University of Sulaimani (Reference: ABC). Data were anonymized, with identifiers removed during analysis. Data protection was carried out in line with institutional and national guidelines.

Statistical analysis

Data were analysed using SPSS v28.0. Descriptive statistics (means, frequencies) summarized socio-demographic, anthropometric data, and frequencies of imaging abnormalities. Chi-square tests assessed associations between categorical variables (e.g., socio-economic status and frequencies of imaging abnormalities). Pearson/Spearman assessed correlation between MRI abnormalities and hormone levels/growth outcomes. Statistical significance was set at $p < 0.05$.

Results

Demographic information of 90 children with GHD is presented in Table 4.1. Mean ± SD age of children was 9.47 ± 2.68 years. In terms of sex, 42 (46.7%) children were male and 48 (53.3%) children were female.

The place of residence of 72 children (80%) was urban and 18 children (20%) were rural. The mean ± SD age at diagnosis of GHD in the children studied was 6.80 ± 1.92 years. The mean ± SD current height of the children was 126.62 ± 16.41 cm, height at diagnosis was 103 ± 14.85 cm, and target height (based on parental height) was 164.50 ± 4.98 cm.

Mean ± SD growth velocity at diagnosis in all children studied was 4.207 ± 0.98 cm/year. Also, mean ± SD GH provocation test peak in all children was 3.438 ± 1.12 ng/ml. (Table 1).

Table 1. Demographic, Diagnosis and Growth measurements Characteristics in Children with GHD in Sulaymaniyah, Iraq

Variable		Frequency
Age		9.472 ± 2.687
Age of diagnosis of GHD (years)		6.802 ± 1.924
Current height(cm)		126.62 ± 16.414
Height at diagnosis(cm)		103 ± 14.858
Target height (based on parental height)		164.50 ± 4.988
Growth velocity at the time of diagnosis (cm/year)		4.207 ± 0.983
GH provocation test peak(ng/ml)		3.438 ± 1.127
Sex	Female	42 (46.7%)
	Male	48 (53.3%)
Place of residence	Rural	18 (20%)
	Urban	72 (80%)

MRI findings (pituitary gland abnormalities) in children with GHD are presented in Table 4.3. Normal pituitary gland was seen in 77 (85.6%) children, indicating that MRI findings were normal in these children. Hypoplastic pituitary gland was seen in 3 (3.3%) children, Hypoplastic pituitary gland, Ectopic posterior pituitary gland and Pituitary micro adenoma were seen in 2 (2.2%) children each. Hypoplastic pituitary gland, Absent pituitary stalk, Absent pituitary stalk, Ectopic posterior pituitary gland, Absent pituitary stalk, Ectopic posterior pituitary gland, thinning pituitary stalk, Hypoplastic pituitary gland (anterior), thickening pituitary stalk and Thinning pituitary stalk were seen in 1 (1.1%) child each.

MRI findings (brain abnormalities) in children with GHD are presented in Table 4.4. MRI findings (brain abnormalities) were normal in 83 (92.2%) children, structural abnormalities in the brain were seen in 5 (5.6%) and midline defects were seen in 2 (2.2%) children. (Table 2)

Table 2. MRI findings (pituitary gland abnormalities/ brain abnormalities) in Children with GHD in Sulaymaniyah, Iraq

Variable		Frequency
MRI findings (pituitary gland abnormalities)	Normal pituitary gland	77 (85.6%)
	Hypoplastic pituitary gland, Absent pituitary stalk	1 (1.1%)
	Hypoplastic pituitary gland	3 (3.3%)
	Absent pituitary stalk	1 (1.1%)
	Ectopic posterior pituitary gland, Absent pituitary stalk	1 (1.1%)
	Hypoplastic pituitary gland, Ectopic posterior pituitary gland	2 (2.2%)
MRI findings (brain abnormalities)	None	83 (92.2%)
	Structural abnormalities in the brain	5 (5.6%)
	Midline defects	2 (2.2%)

Mean ± SD Growth velocity at the time of diagnosis (cm/year) by type of MRI findings (pituitary gland abnormalities) is presented in Table 4.9. Mean ± SD Growth velocity at the time of diagnosis (cm/year) in children whose findings were Normal was 4.388 ± 0.814. Mean ± SD Growth velocity at the time of diagnosis (cm/year) was 2.800 in Hypoplastic pituitary gland, Absent pituitary stalk, 3.700 ± 1.352 in Hypoplastic pituitary gland, Absent pituitary stalk, 2.100 in Ectopic posterior pituitary gland, Absent pituitary stalk, 2.200 in Hypoplastic pituitary gland, Ectopic posterior pituitary gland, 2.250 ± 1.060 in Ectopic posterior pituitary gland, thinning pituitary stalk, 2.200 in Hypoplastic pituitary gland (anterior), thickening pituitary stalk, 5 in Pituitary micro adenoma, 4.250 ± 1.060 in Thinning pituitary stalk, 2.500. And the mean growth velocity at the time of diagnosis (cm/year) was statistically significantly different among different MRI findings (pituitary gland abnormalities) (P≤0.001). (Table 3)

Table 3. Mean ± SD Growth velocity at the time of diagnosis (cm/year) by type of MRI findings (pituitary gland abnormalities)

Growth velocity at the time of diagnosis (cm/year)		Mean ± SD	P-value*
MRI findings (pituitary gland abnormalities)	Normal pituitary gland	4.388 ± 0.814	0.001
	Hypoplastic pituitary gland, Absent pituitary stalk	2.800	
	Hypoplastic pituitary gland	3.700 ± 1.352	
	Absent pituitary stalk	2.100	
	Ectopic posterior pituitary gland, Absent pituitary stalk	2.200	
	Hypoplastic pituitary gland, Ectopic posterior pituitary gland	2.250 ± 1.060	
	Ectopic posterior pituitary gland, thinning pituitary stalk	2.200	
	Hypoplastic pituitary gland (anterior), thickening pituitary stalk	5	
	Pituitary micro adenoma	4.250 ± 1.060	
	Thinning pituitary stalk	2.500	
*P-value based on one-way ANOVA			

Mean ± SD Growth velocity at the time of diagnosis (cm/year) by type of MRI findings (brain abnormalities) is presented in Table 4.11. Mean ± SD Growth velocity at the time of diagnosis (cm/year) in children whose findings were Normal was 4.310 ± 0.899. Mean ± SD Growth velocity at the time of diagnosis (cm/year) in the finding of Structural abnormalities in the brain was 3.440 ± 1.078 and in the finding of Midline defects was 1.850 ± 0.495. The mean Growth velocity at the time of diagnosis (cm/year) was statistically significantly different among different MRI findings (brain abnormalities) (P≤0.001). (Table 1)

Table 4. Mean ± SD Growth velocity at the time of diagnosis (cm/year) by type of MRI findings (brain abnormalities)

Growth velocity at the time of diagnosis (cm/year)		Mean ± SD	P-value*
MRI findings (brain abnormalities)	None	4.310 ± 0.899	0.001
	Structural abnormalities in the brain	3.440 ± 1.078	
	Midline defects	1.850 ± 0.495	
*P-value based on Spearman correlation			

The correlation between Growth velocity at the time of diagnosis (cm/year) and MRI findings (pituitary gland abnormalities) was an indirect and significant correlation (r = -0.360, P≤0.001), This means that cases with pituitary gland abnormalities have a lower mean growth velocity compared to those with normal MRI. The correlation between Growth velocity at the time of diagnosis (cm/year) and MRI findings (brain abnormalities) was an indirect and significant correlation (r = -0.306, P≤0.003). While the correlation between Growth velocity at the time of diagnosis (cm/year) and Growth hormone therapy was not statistically significant.

The correlation between GH provocation test peak (ng/ml) and MRI findings (pituitary gland abnormalities) was an indirect and significant correlation (r = -0.373, P≤0.001), This means that patients with pituitary gland abnormalities had significantly lower mean peak GH levels compared to those with normal MRI (Table 6).

Table 6. Correlation between Growth velocity at the time of diagnosis (cm/year) and GH provocation test peak (ng/ml) with MRI findings (pituitary gland abnormalities), MRI findings (brain abnormalities)

Variable	Growth velocity at the time of diagnosis (cm/year)	GH provocation test peak (ng/ml)
MRI findings (pituitary gland abnormalities)	r = -0.360 P≤0.001	r = -0.373 P≤0.001*
MRI findings (brain abnormalities)	r = -0.306 P≤0.003	r = -0.004 P≤0.966
*P-value based on Spearman correlation		

Discussion

The present study aimed to investigate MRI imaging findings in children with GHD in Sulaymaniyah, Iraq, seeking to document the imaging patterns of the pituitary gland, assess the correlation between radiological findings and clinical and hormonal parameters, and determine the role of imaging in clinical judgment and treatment planning.

This study included 90 children aged 2 to 16 years with a diagnosis of GHD. MRI findings (pituitary gland abnormalities) showed that the vast majority of 77 (85.6%) had normal MRI, indicating a high prevalence of idiopathic or mild GHD in this population. Abnormalities included hypoplastic pituitary gland, hypoplastic pituitary gland, ectopic posterior pituitary gland and pituitary micro adenoma, hypoplastic pituitary gland, absent pituitary stalk, absent pituitary stalk, ectopic posterior pituitary gland, absent pituitary stalk, ectopic posterior pituitary gland, thinning pituitary stalk, hypoplastic pituitary gland (anterior), thickening pituitary stalk and thinning pituitary stalk.

MRI findings (brain abnormalities) were normal in 83 (92.2%) and Structural abnormalities in the brain and Midline defects were seen. These findings indicate a limited but important role of brain MRI in

identifying abnormalities associated with GHD. The findings showed a significant inverse relationship between age at diagnosis, lower growth velocity at diagnosis, and lower peak GH levels on stimulation tests with pituitary and brain abnormalities, indicating that children with pituitary abnormalities were usually diagnosed earlier and had lower growth velocity and peak GH levels, indicating a significant relationship between MRI findings and clinical parameters. Multiple pituitary hormone deficiency was observed in 6 (6.7%) children, while IGHD was more common. Statistically significant differences in growth velocity and peak levels were observed based on MRI findings, indicating the influence of MRI abnormalities on treatment response.

Examination of brain abnormalities in this study showed that most children had normal brain MRI findings. Structural brain abnormalities were the most common finding, and midline defects were the next most common finding. The finding of a normal pituitary gland on MRI is very significant. This rate is consistent with the study by Ö Kara et al. (14) in Turkey and the study by RNA AlJurayyan et al. (15) in Saudi Arabia, which reported normal MRI in most IGHD patients. These regional similarities suggest a common pattern for idiopathic GHD, in which structural abnormalities are less common. While in the study by M Yakubu et al. (16) a wide range of MRI abnormalities was reported.

The lower prevalence of abnormalities in the present study may be partly due to exclusion criteria such as excluding children with secondary causes of GHD due to trauma, tumor, surgery, or syndromic multiglandular disorders except in specific cases, which means that the study was mainly focused on idiopathic GHD, in which a normal MRI is often expected. The high percentage of normal MRI findings suggests that idiopathic GHD accounts for a significant proportion of GHD cases. This means that in many children in this region, the underlying cause of GHD may not be a major structural abnormality detectable by standard MRI, but rather a functional or microstructural defect, or a genetic cause not associated with overt morphological changes (17). This finding is consistent with the global understanding that idiopathic GHD is the most common type.

A statistically significant inverse correlation was observed between growth velocity at diagnosis and pituitary gland abnormalities. Similarly, a significant inverse correlation was found between growth velocity and brain abnormalities. These significant correlations between structural abnormalities of the pituitary and brain with slower growth velocity clearly indicate that structural defects are associated with more severe growth failure. Structural abnormalities of the hypothalamic-pituitary axis or broader brain structures can directly impair GH production and/or secretion or interfere with its downstream effects, leading to more pronounced growth failure (18,19). This finding is consistent with previous studies that have suggested that structural hypothalamic-pituitary abnormalities play a significant role in predicting growth response (20,21). Thus, MRI is not only a diagnostic tool for identifying anatomical defects but also a prognostic indicator for the severity of growth failure (22,23), which can guide clinicians in predicting the extent of growth delay and possibly influencing the urgency and intensity of intervention.

A statistically significant inverse correlation was observed between peak GH stimulation test and pituitary abnormalities. This is to be expected, as the pituitary is the primary site of GH production (24).

However, it is noteworthy that no statistically significant correlation was observed between peak GH stimulation test and brain abnormalities. This distinction is important. Although structural pituitary abnormalities are strongly associated with impaired GH secretion, more widespread brain abnormalities may affect growth through mechanisms other than direct impairment of GH secretion, such as affecting IGF-1 sensitivity, general metabolic pathways, or even nutritional intake due to neurological problems (25). They may also represent more complex syndromes of which GHD is only one component, but not necessarily the most severe hormone deficiency as measured by peak GH (26). A statistically significant inverse correlation

was observed between the age of diagnosis of GHD and both pituitary and brain abnormalities. This means the presence of pituitary and brain abnormalities suggests that structural GHD, that is GHD with detectable abnormalities on MRI, tends to present at a younger age (27). Structural abnormalities, especially congenital abnormalities, often present with more severe or obvious clinical signs of GHD, such as more pronounced short stature, more rapid growth retardation, or associated neurological symptoms (28), leading to earlier medical evaluation and diagnosis compared with idiopathic cases without obvious structural defects.

In the present study, six cases of MPHD were identified among the participants. Each of these MPHD cases was associated with distinct pituitary gland abnormalities identified by MRI. The distribution of MRI findings between MPHD and IGHD reinforces the role of MRI beyond simply diagnosing GHD. While a normal MRI does not rule out the presence of GHD (especially IGHD), the presence of specific and severe structural pituitary abnormalities, such as an absent stalk or significant hypoplasia, strongly increases the likelihood of MPHD (29). This makes MRI a vital tool for predicting the complexity of hormonal dysfunction, not just the presence of GHD.

Conclusion

The results indicate that GHD in children is predominantly associated with normal MRI findings of the pituitary gland, reflecting the high prevalence of idiopathic GHD in the region, where major structural abnormalities are infrequently observed. Nevertheless, a significant subset of patients exhibited various pituitary abnormalities, including hypoplasia, ectopic posterior pituitary, stalk abnormalities, and microadenomas, with brain abnormalities being rare. The statistically significant correlation between specific MRI abnormalities and reduced peak growth hormone levels during stimulation tests suggests that MRI can serve as an important complementary tool in assessing the severity and etiology of GHD. However, given the high frequency of normal MRI findings, imaging alone is insufficient for diagnosis and should be integrated with thorough clinical and hormonal evaluations. Additionally, both pituitary and brain abnormalities were associated with an earlier age at diagnosis and more severe short stature at presentation, underscoring the importance of early medical assessment.

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Conflict of interest:

All authors assert the absence of any conflict of interest.

Data availability:

Upon reasonable request, the data from the research may be obtained from the corresponding author.

Authors' contributions:

Each author made an equal contribution to this research work.

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