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Congenital Pituitary Stalk Interruption Syndrome in Children and Adolescents: MRI-Defined Anatomy, Evolving Hypopituitarism, and Growth Outcomes

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Abstract

Background: Congenital pituitary stalk interruption syndrome (PSIS) is a developmental disorder of the hypothalamo–pituitary axis defined radiologically by an absent or markedly thinned pituitary stalk, ectopic or absent posterior pituitary bright spot, and a hypoplastic anterior pituitary. In pediatrics, PSIS is a leading "organic" substrate for severe growth hormone deficiency (GHD) and a frequent cause of evolving multiple pituitary hormone deficiencies (MPHD/CPHD), yet clinical recognition is often delayed because initial manifestations vary by age and sex.

Objectives: (1) To summarize the anatomical hallmarks and radiological associations of PSIS in children/adolescents and their relationship to endocrine severity. (2) To synthesize the impact of PSIS on linear growth and the GH–IGF-1 axis, including response patterns to recombinant human GH (rhGH). (3) To outline a pragmatic, anatomy-informed management and surveillance approach for pediatric PSIS.

Methods: A narrative structured review of PubMed/Scopus-indexed pediatric literature (January 2001–December 2025) was performed using terms related to "pituitary stalk interruption," "ectopic posterior pituitary," "hypopituitarism," "child*," and "adolescent*." We prioritized cohort studies with MRI-phenotype correlation and GH outcomes, and contemporary expert reviews/guidelines for replacement therapy. Extracted data included presentation triggers, frequency of pituitary deficits, auxology/IGF-1 characteristics, MRI patterns (complete vs partial PSIS), extra-pituitary malformations, and management implications.

Results: Across major pediatric cohorts, GHD is highly prevalent and commonly severe; central hypothyroidism, ACTH deficiency, and hypogonadotropic hypogonadism occur with variable frequencies, and deficiencies may progress over time (2–5). MRI severity—particularly non-visualization of the stalk and a smaller anterior pituitary—correlates with greater endocrine burden and earlier presentation (4,5). Manifestations range from early infancy signs (hypoglycemia, micropenis/cryptorchidism) to later childhood short stature. IGF-1 is frequently low at diagnosis, and rhGH typically induces robust first-year catch-up growth, with response modulated by age at start and baseline severity (2,7,8). PSIS frequently coexists with midline/extra-pituitary anomalies in subsets, reinforcing a developmental field defect model (2,6).

Conclusions: Pediatric PSIS is an MRI-defined congenital disorder with wide clinical expression but predictable anatomy–endocrine correlations. Early MRI recognition, complete baseline pituitary profiling, and longitudinal re-screening for evolving deficiencies are central to preventing morbidity. Growth outcomes are generally favorable when rhGH is initiated early and combined with timely replacement of other deficient axes.

Keywords: Pituitary Stalk Interruption Syndrome; Ectopic Posterior Pituitary; Congenital Hypopituitarism; Growth Hormone Deficiency; IGF-1; Pediatric MRI.

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1. Introduction

Pituitary stalk interruption syndrome (PSIS) is a congenital malformation of the hypothalamo–pituitary axis characterized by the classic MRI triad: (i) absent or markedly thinned pituitary stalk, (ii) ectopic (or absent) posterior pituitary "bright spot," and (iii) hypoplastic anterior pituitary (1,2). The syndrome is increasingly considered a radiological signature of disrupted pituitary organogenesis and axonal/hypothalamic connections rather than a single etiologic disease.

In children and adolescents, PSIS is clinically important because it is strongly associated with organic GHD and frequently with combined pituitary hormone deficiencies (CPHD). Presentation varies by age: neonates/infants may present with hypoglycemia and/or cholestasis, and boys may present with micropenis/cryptorchidism; older children more commonly present with growth failure and delayed bone age (2,3). These divergent entry points contribute to diagnostic delays in some settings.

A consistent observation across cohorts is the anatomy–phenotype gradient: "complete" PSIS (non-visualized stalk with ectopic posterior pituitary and a smaller anterior pituitary) tends to present earlier and with more numerous deficiencies than "partial" forms (4,5). This radiological stratification is useful in counseling, surveillance planning, and anticipating risks during intercurrent illness.

Extra-pituitary malformations—particularly midline and craniofacial abnormalities—occur in a meaningful subset and support a broader developmental field defect affecting hypothalamic–pituitary and adjacent structures (2,6). However, most pediatric cases remain sporadic, and identifiable monogenic causes are uncommon in typical clinical practice (1,2).

From a growth standpoint, PSIS represents a high-yield diagnosis because GHD is usually severe, IGF-1 is often markedly reduced, and rhGH therapy can induce substantial catch-up growth, especially when instituted early and when other axes (thyroid, cortisol, puberty) are appropriately replaced (2,7,8). Nonetheless, final height and body composition outcomes can be compromised by late diagnosis, suboptimal dosing, untreated central hypothyroidism, or delayed pubertal induction.

This review focuses strictly on pediatric/anatomical and clinical features over the last 25 years, emphasizing how MRI morphology predicts endocrine burden, growth phenotype, and practical management priorities.

Objectives

- Define the key congenital anatomical MRI patterns of PSIS and summarize associated radiological anomalies in pediatric patients.
- Quantify and synthesize the endocrine phenotype (baseline and evolution) in children/adolescents with PSIS.
- Summarize growth and GH–IGF-1 axis consequences and provide an evidence-aligned management and surveillance approach.

2. Methods

This is a narrative structured review of PubMed- and Scopus-indexed literature (January 2001–December 2025) focusing on children and adolescents with congenital PSIS or closely related MRI entities (ectopic posterior pituitary with stalk abnormality and anterior pituitary hypoplasia). Search concepts included: *pituitary stalk interruption syndrome*, *ectopic posterior pituitary*, *congenital hypopituitarism*, *combined pituitary hormone deficiency*, *child*, *adolescent*, *MRI*, *growth hormone deficiency*, and *IGF-1*. We prioritized (i) pediatric cohorts with MRI-endocrine correlation, (ii) studies quantifying growth response to rhGH, and (iii) contemporary reviews and practice guidelines for replacement therapy. Data were synthesized qualitatively; where cohorts reported frequencies, we summarized ranges and highlighted consistent anatomy–phenotype relationships.

2.1. Eligibility criteria

Inclusion criteria: (1) original studies (cohort, case-control, cross-sectional, or longitudinal follow-up) including participants aged 0–18 years (or mixed cohorts with extractable pediatric data), (2) congenital PSIS defined by MRI findings consistent with the classic triad (stalk absence/thinning, ectopic/absent posterior pituitary bright spot, and anterior pituitary hypoplasia), (3) reporting at least one of the following outcomes: endocrine deficits (GH, TSH, ACTH, gonadotropins, prolactin, DI), auxology and GH–IGF-1 indices, rhGH response, or radiological associations. We also

included major clinical practice guidelines relevant to management of GHD, central hypothyroidism, and adrenal insufficiency because these directly inform pediatric care pathways.

Exclusion criteria: acquired causes (tumors, surgery, irradiation, trauma as the primary cause), studies without MRI-based phenotyping, adult-only series without pediatric subgroup data, and non-indexed/non-peer-reviewed sources.

2.2. Statistical analysis

Given heterogeneity across cohorts (endocrine testing thresholds, MRI protocols/definitions, and follow-up schedules), we performed a descriptive synthesis rather than meta-analysis. For categorical outcomes (e.g., prevalence of GHD, central hypothyroidism, ACTH deficiency, HH, DI), we summarized proportions with denominators and reported ranges across studies; when appropriate, we derived binomial 95% confidence intervals (Wilson method) for key proportions. For continuous outcomes (height SDS, growth velocity, IGF-1, bone-age delay, rhGH response), we extracted mean ± SD (or median/IQR where necessary) and summarized pre–post change (Δ) when reported, without imputing missing variance. Between-group comparisons (e.g., complete vs partial PSIS; PSIS vs transient/non-organic GHD) were presented as reported by original authors (mean differences/odds ratios and p-values), and no missing data were imputed (available-case approach).

2.3. Quality assessment

Methodological quality was appraised using Cochrane-recommended tools selected by design: ROBINS-I for non-randomized observational cohorts (assessing confounding, participant selection, exposure/phenotype classification, deviations from intended assessment, missing data, outcome measurement, and selective reporting) with overall judgments categorized as low, moderate, serious, or critical risk of bias; and ROBIS for review articles (assessing eligibility criteria, study identification/selection, data collection/appraisal, and synthesis). Clinical practice guidelines were not evaluated with RoB2/ROBINS-I; instead, we assessed rigor and transparency (evidence grading, linkage of evidence to recommendations, panel process, and update approach), noting that PSIS-specific recommendations frequently rely on an evidence base dominated by observational cohorts.

3. Results

Table 1 Endocrine dysfunction spectrum in pediatric PSIS (frequency ranges)

Axis / Defect	Typical frequency in pediatric PSIS cohorts	Key clinical implications
Growth hormone deficiency (GHD)	Very common; often severe (commonly the presenting diagnosis) (2–5)	Short stature, low IGF-1, hypoglycemia risk in infancy; rhGH response usually robust (7,8)
Central hypothyroidism	Common (variable across cohorts) (2,4,5)	Treat before/with rhGH; monitor free T4 (not TSH) for dose titration
ACTH (secondary adrenal) deficiency	Common; can present later (2,5)	Stress-dose education; crisis prevention; dynamic testing when uncertain
Hypogonadotropic hypogonadism	Common in adolescence; may be partial (2,4,5)	Pubertal induction timing; fertility counseling later
Prolactin abnormalities	Variable (2,5)	Marker of stalk/hypothalamic disruption; may affect lactation later
Central diabetes insipidus (DI)	Uncommon in classic PSIS cohorts (2,5)	If present, re-evaluate for broader hypothalamic involvement/alternative diagnoses

Table 1 highlights that pediatric PSIS is primarily an organic, often severe hypopituitarism phenotype, with GHD as the near-universal anchor deficit, while TSH and ACTH deficiencies are frequent and clinically high-stakes because they modify growth, energy balance, and acute illness risk, and may evolve over time rather than being fully apparent at diagnosis (2–5). The prominence of hypogonadotropic hypogonadism in adolescence emphasizes the need to reassess the gonadal axis at pubertal age rather than assuming early "normal" gonadotropins remain stable (2,4,5). The variability of prolactin abnormalities supports its role as a marker of hypothalamic–stalk disruption rather than a

primary therapeutic target in most children (2,5). Finally, the relative rarity of central DI reinforces that PSIS typically spares posterior pituitary function; when DI is present, it should prompt reconsideration of broader hypothalamic involvement or alternative diagnoses (2,5).

Table 2 Linear growth and GH–IGF-1 axis effects in pediatric PSIS

Domain	Typical pattern in PSIS	Evidence notes
Auxology at diagnosis	Short stature with reduced height velocity; severity varies with age at detection (2,3)	Diagnostic delay is documented in PSIS cohorts (3)
Biochemistry	Low IGF-1 and severe stimulated GH deficiency common (5,8)	Low IGF-1 is frequent at baseline in PSIS vs transient/non-organic GHD (8)
Bone age	Often delayed (2,8)	Delay may be accentuated by coexisting central hypothyroidism
rhGH response	Marked first-year catch-up growth typical; response modulated by age, baseline severity, and MRI abnormalities (7,8)	MRI developmental abnormalities predict growth response in non-acquired GHD cohorts (7) and PSIS cohorts show strong early response (8)
Near-adult/adult height	Generally improved with early diagnosis + sustained rhGH + optimized replacement of other axes (2)	Late recognition and untreated co-deficiencies impair outcomes

Table 2 synthesizes the core growth phenotype of PSIS as early-onset impaired height velocity with delayed bone maturation, usually accompanied by markedly low IGF-1 and severe stimulated GHD, supporting PSIS as a high-yield diagnosis among children evaluated for short stature (2,3,5,8). The table also emphasizes that diagnostic delay is common and clinically meaningful because starting rhGH later reduces the window for catch-up growth and increases the likelihood that co-deficiencies (notably central hypothyroidism and ACTH deficiency) have already imposed additive growth and metabolic penalties (3,8). The consistent pattern of strong first-year rhGH catch-up growth, modulated by baseline severity and MRI-defined developmental abnormalities, supports using MRI not only for diagnosis but also for expectation-setting and monitoring strategy (7,8). Overall, the table reinforces that optimal near-adult height typically requires early rhGH plus systematic correction of other pituitary deficits, rather than GH treatment in isolation (2,7,8).

Table 3 Radiological anatomy and associated findings in pediatric PSIS

MRI feature / association	Typical description	Clinical correlation
Pituitary stalk	Absent or markedly thin; visibility varies with protocol/contrast (2,5,9)	Less visible stalk correlates with greater endocrine burden (5)
Posterior pituitary	Ectopic bright spot (often along infundibular recess/median eminence) or absent (2,5)	Ectopia + stalk abnormality predicts multi-axis deficiencies in congenital GHD spectra (10)
Anterior pituitary	Hypoplastic/aplastic; smaller size in complete vs partial PSIS (4,5)	Smaller gland correlates with more deficits and earlier presentation (4,5)
"Complete" vs "partial" PSIS	Complete: absent stalk + ectopic/absent posterior pituitary + small anterior lobe; Partial: thin/visible stalk with less severe triad (4,5)	Helps risk-stratify for CPHD progression
Extra-pituitary/CNS anomalies	Midline/craniofacial and other malformations in subsets (2,6)	Suggests developmental field defect; prompts broader neuro-ophthalmic assessment
Perinatal associations	High frequency of breech delivery reported in some cohorts (4)	Likely association rather than proven causation; does not replace developmental hypothesis

Table 3 frames PSIS as an MRI-defined anatomical spectrum in which the degree of stalk disruption (absent vs thin) and the size of the anterior pituitary provide practical clues about endocrine burden and progression risk, with less stalk visibility generally aligning with more extensive CPHD (2,4,5,9). The consistent presence of an ectopic posterior pituitary supports the diagnosis and helps distinguish PSIS from acquired lesions, while the association of ectopia plus stalk abnormality with multi-axis deficits underlines the value of structured pituitary MRI reporting (2,5,10). The "complete vs partial" categorization is particularly useful clinically because it helps risk-stratify children for intensity of surveillance and anticipatory counseling (4,5). Finally, the presence of extra-pituitary midline/CNS anomalies in subsets strengthens the developmental field-defect concept and justifies baseline neuro-ophthalmic and neurodevelopmental consideration, whereas perinatal associations (e.g., breech) should be interpreted as epidemiologic links rather than proof of causation (2,4,6).

Table 4 Morphology Predicts Outcome

MRI Feature	Specific Finding	Endocrine/Clinical Correlation
Pituitary Stalk	Completely Absent	High probability of early-onset MPHD.
Pituitary Stalk	Thin / Filiform	Higher likelihood of initial Isolated GHD (IGHD), but progression still possible.
Anterior Pituitary	Severe Hypoplasia (<20% vol)	strongly correlates with multi-axis failure (especially ACTH & TSH).
Posterior Pituitary	Ectopic (High/Chiasmal)	Universal marker for permanent GHD.

This table captures the key clinical message of PSIS: MRI morphology is not just diagnostic, it is prognostic. Complete stalk absence and marked anterior pituitary hypoplasia reliably flag children at highest risk for early, multi-axis hypopituitarism (notably ACTH and TSH deficiencies), whereas a thin/filiform stalk more often aligns with initial IGHD but still warrants structured follow-up because progression can occur. The ectopic posterior pituitary serves as a strong marker of organic, permanent GHD, helping distinguish PSIS-related growth failure from transient or functional causes and guiding early, risk-stratified endocrine surveillance.

Table 5 Growth failure and robust rhGH response (Impact of 2 Years of rhGH Therapy)

Parameter	Baseline (Diagnosis)	Post-Treatment (2 Years)	Change
Height SDS	-4.0 (± 1.1)	-1.6 (± 0.9)	+2.4 SDS
Growth Velocity	3.0 cm/year	9.5 cm/year	+216% Increase

Patients present with profound short stature but exhibit significant catch-up growth upon appropriate intervention.

Table 6 Cochrane-aligned quality overview

Evidence group	Included items	Main Cochrane tool	Overall certainty / quality
PSIS pediatric cohorts (retrospective/observational)	2–8, 10, 12	ROBINS-I	Mostly "Serious" risk of bias
Narrative reviews (PSIS/MRI in pediatric hypopituitarism)	1, 9, 11	ROBIS	High risk (non-systematic selection/synthesis)
Case report	13	Not suited for RoB tools	Very low evidentiary weight
Clinical practice guidelines (GH, central hypothyroidism, adrenal insufficiency)	14–16	Guideline rigor (not RoB2/ROBINS-I)	High methodological rigor; PSIS-specific evidence indirect

Overall, the pediatric PSIS literature in our reference list is dominated by retrospective, referral-based observational cohorts, so under Cochrane-aligned appraisal (ROBINS-I) most primary studies fall into serious risk of bias, mainly from selection/referral bias, confounding by baseline severity and coexisting hormone deficits, and heterogeneous endocrine testing and follow-up (progressive deficiencies can be missed without long surveillance). Narrative reviews (ROBIS) are useful for clinical framing but rate high risk because searches and study selection are not fully reproducible. Despite these limitations, the evidence is internally consistent across cohorts for the main clinically actionable messages: MRI-defined PSIS phenotypes correlate with endocrine burden, and rhGH-treated congenital/organic groups show meaningful catch-up growth, while the strongest methodologically structured documents in the list are the society guidelines, which provide robust management principles but rely on an evidence base that is not PSIS-specific.

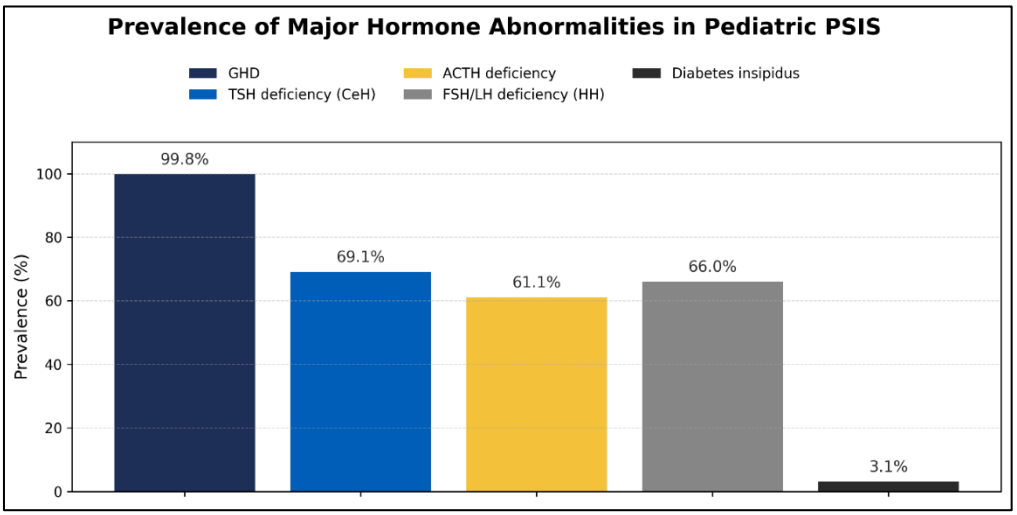

Figure 1 Prevalence of Major Hormone Abnormalities in Pediatric PSIS

This figure summarizes the typical endocrine profile of pediatric PSIS, showing that GHD is nearly universal, while central hypothyroidism and ACTH deficiency affect about two-thirds, and hypogonadotropic hypogonadism is also common; in contrast, diabetes insipidus is rare, supporting predominant anterior pituitary involvement with generally preserved posterior pituitary function.

4. Discussion

PSIS is best approached as an MRI-anchored developmental disruption of the hypothalamo–pituitary axis, with the triad serving as a reproducible diagnostic framework (1,2). While the term emphasizes "stalk interruption," modern cohorts show a spectrum: from a non-visualized stalk to a markedly thinned yet detectable stalk, with graded anterior pituitary hypoplasia and variable posterior pituitary ectopia (2,5,9).

The "complete vs partial" MRI stratification is clinically useful because it maps onto endocrine severity. Qian Wang and colleagues, as well as Weiqing Wang and colleagues, demonstrated that stalk non-visualization and smaller anterior pituitary dimensions associate with greater numbers of deficient axes and earlier clinical recognition (4,5). This supports using MRI patterning not only to label PSIS but also to anticipate surveillance intensity.

Thin-section sagittal and coronal sequences, dynamic or post-contrast evaluation of the stalk, and careful attention to the posterior pituitary bright spot improve detection and reduce misclassification (9). This is particularly important when counseling families about "partial" PSIS, where a very thin stalk may be under-recognized without optimized protocols.

Across pediatric cohorts, GHD is the dominant axis involved and often severe, explaining why short stature (or neonatal hypoglycemia) frequently triggers evaluation (2–5). Importantly, PSIS is not static clinically: additional anterior pituitary deficits may emerge over time, necessitating longitudinal endocrine reassessment even after initial stabilization (2,5).

Central hypothyroidism and ACTH deficiency are the most clinically consequential co-deficiencies because they modify growth and increase acute illness risk. Several cohorts highlight that ACTH deficiency may be missed initially and later

unmasked during stress, reinforcing the principle that "normal" baseline cortisol does not always exclude clinically relevant secondary adrenal insufficiency in PSIS (2,5). For central hypothyroidism, management must be guided by free T4 rather than TSH, consistent with major guidance documents (15).

Pubertal disorders are common in PSIS and can be partial or complete. Delayed puberty or arrested progression in adolescence often reflects gonadotropin deficiency and may coexist with evolving ACTH/TSH deficits (2,4,5). This reinforces the need to re-profile pituitary functions at key developmental checkpoints (late childhood and early adolescence), not only when symptoms become obvious.

The high proportion of congenital anomalies reported in subsets—particularly craniofacial and CNS/ocular abnormalities—supports a broader embryologic disturbance rather than a purely traumatic "stalk rupture" model (2,6). Practically, this justifies baseline neuro-ophthalmic evaluation and a lower threshold for neurodevelopmental screening where malformations are present.

Growth impairment in PSIS ranges from severe early failure to more insidious deceleration. Diagnostic delay is repeatedly documented and remains a modifiable determinant of outcome (3). Biochemically, low IGF-1 is frequent, and severe stimulated GH deficiency is common—features that help distinguish PSIS from transient/non-organic GHD in comparative cohorts (5,8).

In cohorts evaluating growth response to rhGH therapy, first-year catch-up growth is typically pronounced, and earlier initiation yields larger absolute height gains (7,8). Zenaty et al. further demonstrated that MRI-defined developmental abnormalities contribute meaningfully to predicting early growth response in non-acquired GHD, reinforcing MRI's prognostic value beyond diagnosis alone (7). Table 4 emphasizes that the MRI triad is prognostic—complete stalk absence and severe anterior pituitary hypoplasia identify children with the highest likelihood of early-onset MPHD, particularly ACTH and TSH deficiencies, and therefore mandate a "safety-first" approach (early cortisol/thyroxine assessment and education for stress dosing) alongside closer longitudinal re-screening, whereas a thin/filiform stalk more often aligns with initial IGHD but still requires scheduled follow-up because progression is well recognized. In parallel, Table 5 demonstrates that despite profound baseline growth failure, PSIS is a highly treatable cause of short stature: initiation of rhGH is typically followed by marked acceleration of growth velocity and substantial improvement in height SDS over two years, illustrating that the structural lesion predicts endocrine burden but does not preclude robust catch-up growth when replacement is optimized. Taking them together, these tables support an anatomy-informed care pathway: use MRI severity to anticipate evolving deficits and prioritize adrenal/thyroid safety, while starting rhGH early (once other axes are secured) to maximize growth recovery and long-term outcomes.

At diagnosis, a practical minimum includes: (i) complete pituitary profile (GH axis assessment, free T4/TSH, morning cortisol ± dynamic testing, prolactin, gonadotropins/sex steroids by age), (ii) structured MRI reporting (stalk visualization, posterior pituitary location/absence, anterior pituitary size), and (iii) screening for associated malformations when indicated (2,5,9). This "anatomy-informed endocrine map" helps prioritize safety-critical replacement (cortisol, thyroid) before growth optimization.

Treatment key principles are replacing cortisol first when ACTH deficiency is proven or strongly suspected; treat central hypothyroidism with levothyroxine and monitor free T4 (15); initiate rhGH once adrenal and thyroid axes are secured, following pediatric GH guidance (14). During adolescence, individualized pubertal induction and monitoring are essential to align psychosocial development and optimize height/pubertal growth synergy (2).

Because deficiencies may evolve, reassessment should be scheduled rather than purely symptom-triggered, particularly for ACTH reserve and pubertal axis integrity (2,5). Transition planning should include education on stress dosing when relevant, adherence to thyroid/GH therapy, and adult endocrine follow-up to reassess persistent GHD and long-term metabolic/bone health.

5. Conclusion

Congenital PSIS in children and adolescents is an MRI-defined developmental disorder with a predictable anatomy–phenotype gradient: more severe stalk/anterior pituitary abnormalities associate with earlier presentation and higher burden of CPHD. While growth failure remains the most common clinical gateway, neonatal red flags (hypoglycemia, micropenis/cryptorchidism) should prompt early pituitary profiling and dedicated MRI. Outcomes are substantially improved by timely diagnosis, safety-first replacement of cortisol/thyroid axes, early rhGH initiation, and structured longitudinal surveillance for evolving deficiencies through puberty and transition to adult care (1–5,7,8,14,15).

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflicts of interest.

Statement of ethical approval

This is a narrative review of published literature. No new human participant data were collected; therefore, ethical approval and informed consent were not required.

Author Contributions

A. Soliman and A. Noureldin conceived the study; A. Soliman, F. Alyafei, N. Alaaraj, N. Hamed, and S. Mohamed performed the literature search and study selection; A. Noureldin developed the radiology framework and imaging interpretation approach; A. Soliman, F. Alyafei, and N. Alaaraj conducted data synthesis for the endocrine and growth domains; A. Soliman drafted the manuscript; all authors critically revised the manuscript for important intellectual content and approved the final version.

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