A Study to Evaluate Clinical and Hormonal Profile in Short Statured Children of Age Between 6 to 12 Years at a Tertiary Care Centre

Mohd Azmaan Parvez [1*], Bhawna Kohli [2], Yogesh K Goel [3], Subhash Sylonia [4], K Rimi Singh [5]
[1] PG Resident, Deptt of Pediatrics, SIMS, UP, India
[2] Professor, Deptt of Pediatrics, SIMS, UP, India
[3] Professor & HOD, Deptt of Pediatrics, SIMS, UP, India
[4] Professor & HOD, Deptt of Radio Diagnosis, SIMS, UP, India
[5] Assistant Professor, Deptt of Pediatrics, SIMS, UP, India

* Corresponding Author: **Mohd Azmaan Parvez**

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Abstract

Background: It is not established which routinely available clinical parameter best separates endocrine from non-endocrine short stature. We characterised the clinical, anthropometric and hormonal profile of short-statured pre-pubertal children and tested which parameters discriminate between etiological groups.

Methods: Prospective observational study of 100 children aged 6–12 years with height-for-age below the 3rd percentile, over 18 months at a tertiary care centre. All underwent structured history, anthropometry (segment ratios, mid-parental height, height velocity), thyroid function tests, serum IGF-1, cortisol and radiographic bone age; growth hormone (GH) stimulation testing and karyotyping were performed on defined indications. Comparisons used one-way ANOVA with Tukey post-hoc testing, Kruskal–Wallis, t-tests and chi-square/Fisher exact tests.

Results: Mean age was 8.61 ± 1.54 years and 64% were male. Causes were normal variant in 36 (constitutional growth delay 23, familial short stature 13), endocrine in 28, nutritional in 21, chronic/systemic in 9 and genetic in 6; 64% therefore had a pathological cause. GH deficiency was the commonest endocrine diagnosis (18/28, 64.3%). Attained height lay 1.62 SDS below the mid-parental target (95% CI 1.51–1.73; paired $t = 28.99$, $p < 0.001$), with 86/100 more than 1 SDS below. Height SDS did not differ across etiological groups ($F = 0.931$, $p = 0.449$), but height velocity did ($F = 10.49$, $p < 0.001$, $\eta^2 = 0.31$): the endocrine group grew more slowly (3.09 ± 0.71 cm/year) than every other group on Tukey testing (all $p \leq 0.010$), with no other pairwise difference. Peak GH separated GH-deficient children from normal responders (5.10 ± 1.86 vs 12.05 ± 2.65 ng/mL, $p < 0.001$); IGF-1 SDS did not (−2.29 ± 0.54 vs −2.13 ± 0.57, $p = 0.452$).

Conclusion: Severity of height deficit did not identify the underlying cause; height velocity did, and was selectively reduced in endocrine disease. A single IGF-1 measurement did not distinguish GH-deficient children from normal responders. Height velocity should drive the decision to investigate, and GH stimulation testing remains necessary for diagnosis.

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Keywords: Short stature, height velocity, growth hormone deficiency, insulin-like growth factor 1, mid-parental height, bone age

1. Introduction

Short stature — height below −2 standard deviations or the 3rd percentile for age and sex — is among the commonest reasons for referral to paediatric endocrine services. It is a clinical sign rather than a diagnosis, arising from normal growth variants such as familial short stature and constitutional growth delay as well as from endocrine, nutritional, systemic and genetic disease [1, 2]. The clinical task is to separate children who need only reassurance and monitoring from those with a treatable pathological

cause, because growth hormone (GH) deficiency and hypothyroidism respond to replacement therapy and delayed recognition costs adult height [3, 4].

That separation is difficult in practice. Referral cohorts are already selected for severe height deficit, so degree of shortness is unlikely to remain informative once a child has crossed the referral threshold, and there are limited region-specific data on which routinely available parameter actually discriminates between etiological groups. We therefore studied consecutive short-statured children aged 6 to 12 years — a pre-pubertal window in which growth velocity should be steady and endocrine disease is most readily detectable — with the objectives of defining the etiological and hormonal profile of this population and determining which clinical, anthropometric, hormonal and radiological parameters distinguish endocrine from non-endocrine causes.

2. Materials and Methods

2.1. Design, setting and participants

This prospective observational study was conducted in the Department of Paediatrics, Saraswathi Institute of Medical Sciences, Hapur, over 18 months. Children aged 6–12 years with height-for-age below the 3rd percentile on WHO growth charts were enrolled consecutively from the outpatient department and wards. Children with systemic cardiovascular, renal or hepatobiliary disease, those with contractures or kyphoscoliosis preventing height measurement, and those already on treatment were excluded. Informed written parental consent was obtained. Sample size was calculated as $n = Z^2Pq/E^2$ ($Z = 1.96$, $P = 5.5\%$, $q = 0.945$, $E = 5\%$), giving 83.16; adding 20% for non-response gave a target of 100.

2.2. Assessment and investigations

A structured proforma recorded demographic details, birth history, family history of short stature or pubertal delay, consanguinity, diet and chronic illness. Height was measured to the nearest 0.5 cm on a fixed height board and weight on a calibrated scale. The lower segment was measured from pubic symphysis to heel and the upper segment derived by subtraction; the upper-to-lower segment ratio classified stature as proportionate or disproportionate. Mid-parental height was calculated in every child and expressed as a standard deviation score (SDS), height velocity recorded in cm/year, and height, weight and BMI converted to SDS on CDC references. Pubertal status was assigned by Tanner staging. First-line investigations in all children were complete blood count with ESR, urine examination, serum creatinine, SGPT, thyroid function tests, serum IGF-1, serum cortisol and radiographic bone age (Greulich–Pyle). GH stimulation testing was performed for poor height velocity, low IGF-1 or severe short stature, with GH deficiency defined as peak GH below 10 ng/mL; karyotyping and skeletal survey were performed on clinical indication. Each child received a single final diagnosis and was grouped as normal variant, endocrine, nutritional, chronic/systemic or genetic.

2.3. Statistical analysis

Analyses were performed on patient-level data. Continuous variables are given as mean ± SD with median and range, categorical variables as counts and percentages. Groups were compared by one-way ANOVA with Levene's test for homogeneity of variance, Kruskal–Wallis testing as a distribution-free check and Tukey HSD for post-hoc comparison, with effect size as η^2. Attained and mid-parental height SDS were compared within each child by paired t-test and Wilcoxon signed-rank test. Two-group comparisons used independent-samples or Welch t-tests, and categorical associations the Pearson chi-square test with the Fisher–Freeman–Halton exact test where expected counts were below five. Correlations are Pearson r with Spearman ρ. Two-sided $p < 0.05$ was considered significant.

3. Results

3.1. Cohort characteristics and etiological distribution

One hundred children were enrolled with complete data. Mean age was 8.61 ± 1.54 years, 64 (64.0%) were male and all were pre-pubertal. Mean height SDS was −2.89 ± 0.38, with 38 (38.0%) below −3 SDS, and stature was proportionate in 90 (90.0%). Undernutrition was documented in 79 children but judged the primary cause in only 21, making it a coexisting rather than causative finding in 58 of the 79 (73.4%). Mean height velocity was 3.75 ± 0.79 cm/year, with 95 children (95.0%) growing at under 5 cm/year and 57 (57.0%) under 4 cm/year, confirming active growth faltering (Table 1).

Table 1: Demographic, anthropometric, hormonal and radiological characteristics (N = 100). Values are mean ± SD or n.

Variable	Value
Age, years	8.61 ± 1.54 (median 9; range 6–12)
Age 6–7 / 8–9 / ≥10 years, n	22 / 47 / 31
Male / female, n	64 / 36
Low birth weight / preterm, n	29 / 10
Birth asphyxia / neonatal jaundice, n	4 / 9
Family history of short stature / consanguinity, n	34 / 19
Vegetarian diet / undernutrition documented, n	36 / 79
Height, cm	111.72 ± 7.75 (range 94.1–128.5)
Height SDS	−2.89 ± 0.38 (range −3.74 to −2.09)
Height SDS −2.5 to −3.0 / < −3.0, n	62 / 38
Weight SDS / BMI SDS	−2.40 ± 0.49 / −1.65 ± 0.47
Mid-parental height SDS	−1.26 ± 0.45 (range −2.30 to −0.38)
Height velocity, cm/year	3.75 ± 0.79 (range 1.8–5.8)
Proportionate short stature / Tanner stage I, n	90 / 100
IGF-1 SDS	−2.21 ± 0.64 (range −3.74 to −0.35)
Subclinical hypothyroidism / low serum cortisol, n	8 / 2
Bone age / bone age delay, years	6.28 ± 2.12 / 2.33 ± 1.08
GH stimulation test / karyotype performed, n	28 / 10

Normal variant short stature was the largest category (36, 36.0%), comprising constitutional growth delay in 23 and familial short stature in 13. Endocrine causes accounted for 28 (28.0%), nutritional for 21 (21.0%), chronic or systemic illness for 9 (9.0%) and genetic causes for 6 (6.0%); 64 children (64.0%, 95% CI 53.8–73.4%) therefore had a pathological cause. Within the endocrine group GH deficiency was commonest (18/28, 64.3%), followed by hypothyroidism (6/28) and multiple pituitary hormone deficiency (2/28) (Table 2, Figure 1). Sex was not associated with etiological category ($\chi^2 = 2.688$, df = 4, p = 0.611; Fisher exact p = 0.631).

Table 2: Etiological categories and final diagnoses (N = 100).

Etiological category and diagnosis	n	%
Normal variant	36	36.0
Constitutional growth delay	23	23.0
Familial short stature	13	13.0
Endocrine	28	28.0
Growth hormone deficiency	18	18.0
Hypothyroidism	6	6.0
Multiple pituitary hormone deficiency	2	2.0
Other endocrine	2	2.0
Nutritional	21	21.0
Chronic / systemic illness	9	9.0
Genetic	6	6.0

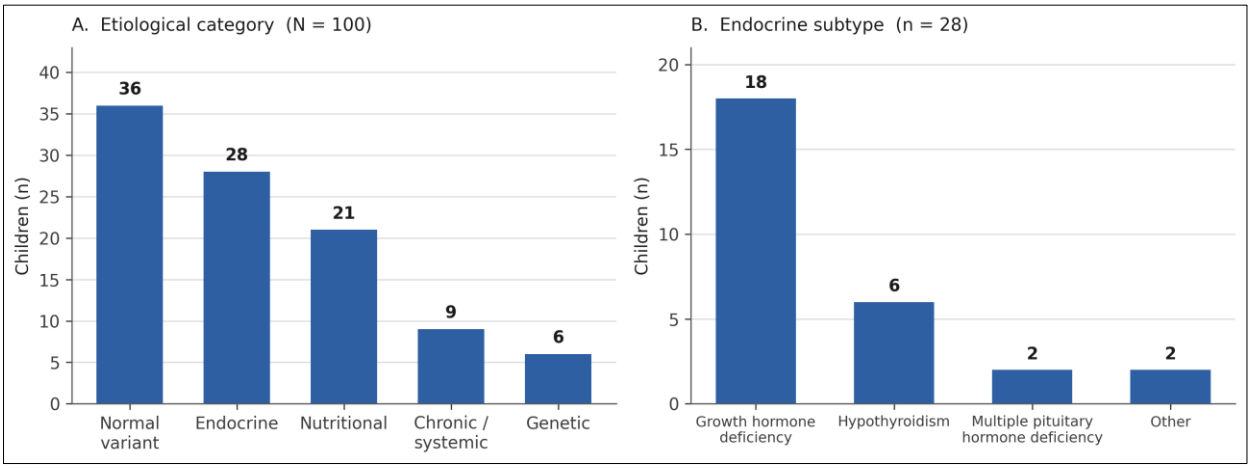

Fig 1: (A) Etiological categories in the whole cohort. (B) Diagnostic subtypes within the endocrine group.

3.2. Attained height relative to genetic target

Mean mid-parental height SDS was −1.26 ± 0.45 against a mean attained height SDS of −2.89 ± 0.38. The within-child deficit relative to genetic target averaged 1.62 SDS (95% CI 1.51–1.73; paired t = 28.99, df = 99, p < 0.001; Wilcoxon p < 0.001; Cohen's d = 2.90). Eighty-six children (86.0%) were more than 1 SDS below target and 32 (32.0%) more than 2 SDS below, confirming that the cohort was short relative to family background and not only relative to the population (Figure 3A). Mid-parental height SDS itself differed across etiological groups (F = 3.220, p = 0.016), being lowest in the normal variant group (−1.39 ± 0.44) and highest in the nutritional group (−0.99 ± 0.49).

3.3. Which parameters distinguish the etiological groups?

Height SDS did not differ across the five etiological categories (F(4,95) = 0.931, p = 0.449, η^2 = 0.038), and neither did IGF-1 SDS (F = 1.456, p = 0.222) or deficit from target (F = 1.863, p = 0.123). Bone age delay was borderline by ANOVA (F = 2.073, p = 0.090) but significant on Kruskal–Wallis testing (H = 10.64, p = 0.031). Height velocity, by contrast, differed markedly (F(4,95) = 10.494, p < 0.001, η^2 = 0.306; Kruskal–Wallis H = 28.90, p < 0.001), with homogeneous variances (Levene p = 0.984). On Tukey post-hoc testing the endocrine group (3.09 ± 0.71 cm/year) grew more slowly than the normal variant (4.14 ± 0.62, p < 0.001), genetic (4.15 ± 0.52, p = 0.007), chronic/systemic (3.96 ± 0.81, p = 0.010) and nutritional groups (3.74 ± 0.68, p = 0.010), with no other pairwise difference significant (Table 3, Figure 2). Comparing normal variants against all pathological causes combined, height velocity again separated the groups (4.14 ± 0.62 vs 3.53 ± 0.79 cm/year, t = 4.30, p < 0.001) whereas height SDS (p = 0.744), weight SDS (p = 0.254), BMI SDS (p = 0.707) and bone age delay (p = 0.704) did not.

Table 3: Comparison of growth, hormonal and skeletal parameters across etiological categories (one-way ANOVA; df 4,95). On Tukey post-hoc testing of height velocity the endocrine group differed from every other group (all $p \leq 0.010$).

Parameter	Normal variant (n = 36)	Endocrine (n = 28)	Nutritional (n = 21)	Chronic/systemic (n = 9)	Genetic (n = 6)	F	p
Height SDS	−2.90 ± 0.42	−2.88 ± 0.36	−2.84 ± 0.35	−3.06 ± 0.31	−2.70 ± 0.41	0.931	0.449
Mid-parental height SDS	−1.39 ± 0.44	−1.25 ± 0.36	−0.99 ± 0.49	−1.37 ± 0.41	−1.41 ± 0.42	3.220	0.016
Deficit from target, SDS	−1.52 ± 0.64	−1.64 ± 0.47	−1.85 ± 0.52	−1.70 ± 0.47	−1.29 ± 0.53	1.863	0.123
Height velocity, cm/year	4.14 ± 0.62	3.09 ± 0.71	3.74 ± 0.68	3.96 ± 0.81	4.15 ± 0.52	10.494	< 0.001
IGF-1 SDS	−2.37 ± 0.66	−2.24 ± 0.55	−2.06 ± 0.75	−1.99 ± 0.42	−1.92 ± 0.65	1.456	0.222
Bone age delay, years	2.38 ± 0.93	2.03 ± 1.19	2.70 ± 0.91	2.63 ± 1.59	1.63 ± 0.24	2.073	0.090

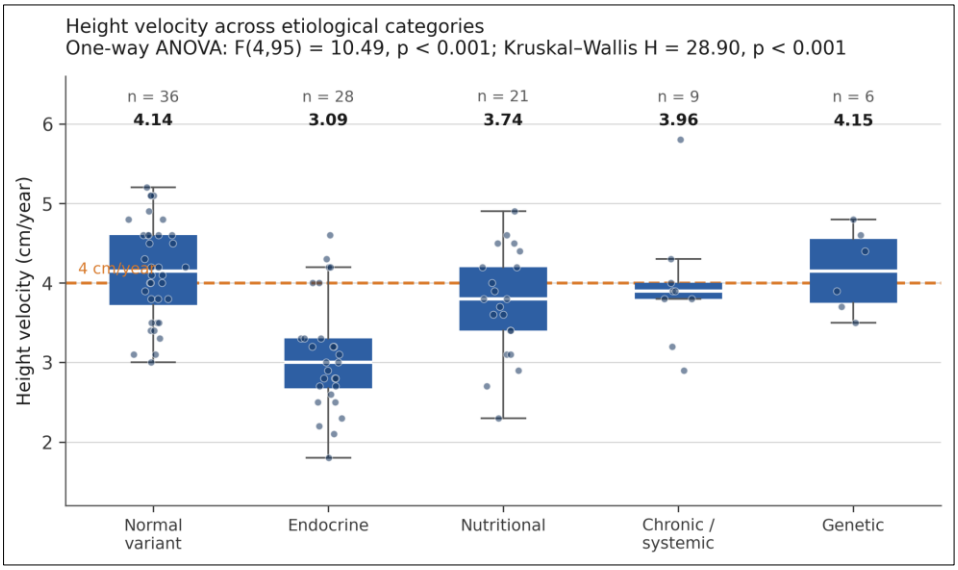

Fig 2: Height velocity across etiological categories. Boxes show median and interquartile range; points are individual children; the dashed line marks 4 cm/year.

3.4. Hormonal evaluation and growth hormone testing

Mean IGF-1 SDS was −2.21 ± 0.64. Thyroid function was normal in 92 children, with subclinical hypothyroidism in 8, all of whom fell within the endocrine group (6 hypothyroidism, 2 multiple pituitary hormone deficiency). Serum cortisol was low in only 2 children, both with multiple pituitary hormone deficiency. GH stimulation testing was performed in 28 children (28.0%) for poor height velocity (12), low IGF-1 (9) or severe short stature (7), confirming GH deficiency in 18 — an overall yield of 64.3% (95% CI 44.1–81.4%), similar across the three indications (66.7%, 55.6% and 71.4%). Peak GH clearly separated the outcome groups (5.10 ± 1.86 vs 12.05 ± 2.65 ng/mL; Welch t = 7.34, p < 0.001; Figure 3B), but IGF-1 SDS did not differ between GH-deficient children and normal responders (−2.29 ± 0.54 vs −2.13 ± 0.57; t = −0.76, df = 26, p = 0.452; Mann–Whitney p = 0.457) and correlated only weakly with peak GH (r = 0.220, p = 0.260). Across the cohort height SDS correlated with neither bone age delay (r = 0.119, p = 0.239) nor IGF-1 SDS (r = −0.036, p = 0.719); age correlated inversely with bone age delay (r = −0.295, p = 0.003).

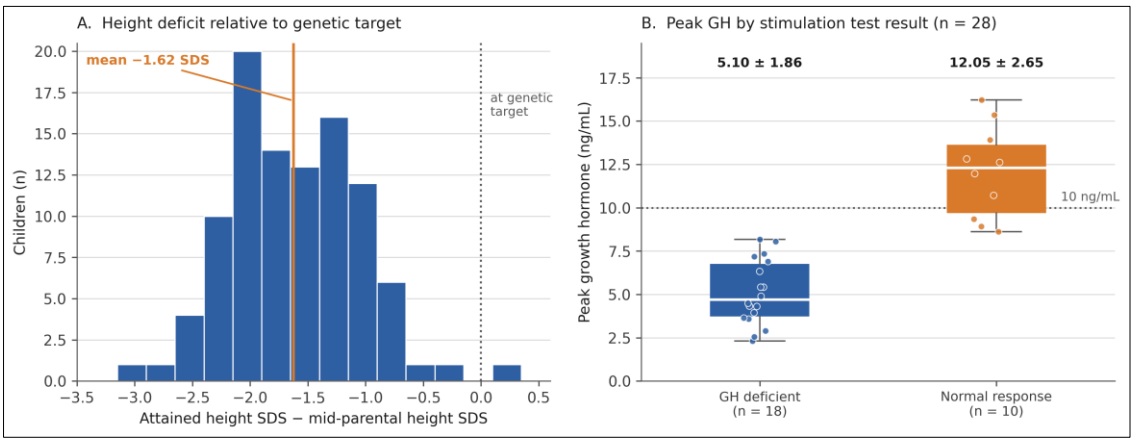

Fig 3: (A) Within-child height deficit relative to mid-parental target; the dotted line marks the genetic target and the solid line the mean deficit. (B) Peak growth hormone by stimulation test result; the dotted line marks the 10 ng/mL threshold.

4. Discussion

The central finding is that the severity of a child's height deficit carried no information about its cause, whereas height velocity did. Height SDS was almost identical across all five etiological categories (group means −2.70 to −3.06, p = 0.449), and neither weight SDS, BMI SDS, IGF-1 SDS nor deficit from genetic target separated normal variants from pathological causes. Height velocity produced a large and specific effect ($\eta^2 = 0.306$) that post-hoc testing localised entirely to the endocrine group, which grew about 1 cm/year more slowly than every other group. This is physiologically coherent: once a referral threshold of the 3rd percentile has been applied, attained height is a cumulative measure already constrained by that selection criterion, whereas velocity reflects the growth process at the time of assessment and remains free to vary with active endocrine disease. The practical implication is that velocity, not degree of shortness, should drive the decision to investigate — and indeed poor height velocity was the commonest trigger for GH stimulation testing here, yielding a diagnosis in 8 of 12 children so tested. Because 95% of the cohort was already growing at under 5 cm/year, absolute cut-offs derived from healthy populations were of limited use; the discriminating signal lay in the relative reduction between groups.

The second clinically consequential finding concerns IGF-1. Mean IGF-1 SDS was low across the whole cohort (−2.21 ± 0.64), including in children with normal variants, and it did not distinguish GH-deficient children from those with a normal stimulation response (p = 0.452), nor did it correlate meaningfully with peak GH (r = 0.220). Peak GH separated the two groups cleanly. IGF-1 is therefore useful for selecting children for dynamic testing but cannot substitute for it, and a low IGF-1 in isolation should not be read as evidence of GH deficiency — consistent with published guidance that biochemical confirmation requires stimulation testing [5,6].

Comparing attained with mid-parental height added a third dimension: children fell 1.62 SDS below their genetic target on average, with 86% more than 1 SDS below, so the cohort was genuinely short for its families rather than short in a familial context. Mid-parental height SDS was lowest in the normal variant group, as expected where familial short stature contributes, and highest in the nutritional group, where an environmental insult has displaced a child from comparatively normal genetic potential. Bone age delay behaved less consistently, reaching significance only on non-parametric testing; the inverse correlation between age and bone age delay (r = −0.295, p = 0.003) indicates that delay was proportionally greater in younger children and cautions against interpreting an absolute delay in years without reference to chronological age. The etiological distribution — 36% normal variants against 64% pathological causes, GH deficiency commonest among the latter — is broadly consistent with other Indian tertiary-centre series [7,8] and confirms that referral to such a centre substantially enriches for treatable disease. The gap between documented undernutrition (79%) and nutritional short stature as a primary diagnosis (21%) is a reminder that undernutrition frequently coexists with, and may be secondary to, another cause, and should not close the diagnostic process [9].

This study is limited by its single-centre, referral-based design and by small numbers in the chronic/systemic (n = 9) and genetic (n = 6) groups, which leaves those comparisons underpowered; the absence of a difference there should not be read as evidence of equivalence. Height velocity was recorded over a single interval rather than a prospectively standardised observation period, karyotyping was performed selectively in 10 children so genetic causes may be underestimated, and there was no follow-up relating the baseline profile to treatment response or adult height. Strengths are the prospective design, a uniform diagnostic protocol applied to every child, complete data capture, and analysis at the level of individual patient records.

5. Conclusion

Almost two-thirds of pre-pubertal children referred with height below the 3rd percentile had a pathological cause, and growth hormone deficiency was the commonest endocrine diagnosis. The degree of height deficit did not indicate the underlying cause, but height velocity did, and was selectively reduced in endocrine disease — making it the parameter that should determine which children are investigated further. A single IGF-1 measurement did not separate growth hormone deficient children from normal responders and cannot replace stimulation testing. Assessment of short stature in this age group should rest on serial height velocity and comparison with mid-parental target, with dynamic endocrine testing reserved for children in whom velocity is disproportionately reduced.

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