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A CLINICAL ACCOUNT OF SHORT STATURE IN PAKISTANI CHILDREN AND ADOLESCENTS PRESENTED AT AN ENDOCRINE CENTER IN KARACHI

Original Research

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ABSTRACT

Background: Short stature in children is a multifactorial condition influenced by genetic, hormonal, nutritional, and environmental factors. Differentiating between normal growth variants and pathological causes is essential for timely intervention. In resource-limited settings, such as Pakistan, understanding local patterns of short stature helps guide cost-effective diagnostic strategies and avoid unnecessary therapies. This study investigates the causes and contributing factors of short stature among pediatric patients evaluated at a tertiary endocrine care center in Karachi.

Objective: To evaluate various etiologies of short stature in children and adolescents presenting at a tertiary care endocrine center.

Methods: A retrospective cohort study was conducted at the Baqai Institute of Diabetology and Endocrinology, Baqai Medical University, Karachi. Medical records from the past five years were reviewed. Children aged 2 to 20 years with complaints of short stature were included. Data collected included age, gender, weight, height, BMI, mid-parental height, birth and feeding history, developmental milestones, and history of chronic illness. Investigations included IGF-1 levels, bone age assessment, thyroid function, vitamin D levels, and anti-tissue transglutaminase antibodies. Data were recorded using a standardized proforma and analyzed using SPSS version 26.

Results: Among 120 participants (56 males, 64 females), the mean age was 13.23 ± 3.18 years. Mean weight was 38.2 ± 13.85 kg, height 136.38 ± 16.19 cm, and BMI 20.12 ± 4.63 kg/m². Mid-parental height averaged 160.2 ± 8.46 cm. IGF-1 levels were 279.38 ± 69.81 ng/mL and bone age was significantly lower in males than females (12.39 ± 3.25 vs. 13.58 ± 3.20 years, $p = 0.047$). Normal growth variation was identified in 33.3% of cases, followed by constitutional growth delay (22.5%) and idiopathic short stature (18.3%). Significant positive correlations with height SD were found for IGF-1 ($p = 0.045$), hemoglobin ($p = 0.007$), and birth weight ($p = 0.002$).

Conclusion: Normal growth variation was the most prevalent cause of short stature, followed by constitutional growth delay and idiopathic short stature. These findings highlight the importance of differentiating physiological growth patterns from pathological conditions to avoid overtreatment.

Keywords: Bone age, Constitutional growth delay, Growth hormone, IGF-1, Pediatric endocrinology, Short stature, Vitamin D deficiency.

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INTRODUCTION

Growth is a continuous and dynamic biological process influenced by a complex interplay of genetic, nutritional, hormonal, and environmental factors (1). Any disruption in these domains can impair a child's growth potential, leading to short stature—a frequent concern in pediatric populations, particularly in developing regions (2). Short stature is typically defined as a height below the 3rd percentile or more than two standard deviations below the mean for a child's age and sex (3). Among the common etiologies are genetic and endocrine conditions, including skeletal dysplasia, Turner syndrome, growth hormone deficiency, Cushing's syndrome, and hypothyroidism. Chronic illnesses—such as renal, cardiac, pulmonary diseases, malignancies, cystic fibrosis, and persistent infections—also contribute to impaired growth. Moreover, external influences such as malnutrition, radiotherapy, chemotherapy, and long-term glucocorticoid use further compound this issue (4). Intrauterine factors, such as intrauterine growth retardation (IUGR), can result in babies being born small for gestational age (SGA), which may lead to growth impairments if not adequately addressed early in life. While normal variant short stature generally does not require hormonal or pharmacologic intervention, psychosocial components such as emotional distress should not be overlooked and must be managed appropriately (5). Idiopathic short stature, on the other hand, is a diagnosis of exclusion after other causes have been ruled out (6).

Genetic makeup significantly determines adult height, with multiple genes playing a role. Familial short stature usually results in a final height within the genetic target range. Conditions such as constitutional delay of growth and puberty (CDGP) can lead to delayed puberty and growth spurts, yet most affected individuals eventually reach a height within the lower bounds of their parental range. However, boys with predicted adult heights below 160 cm may be considered for recombinant growth hormone therapy (7). Importantly, excessive weight relative to height may suggest underlying endocrine dysfunction (8). Children who receive timely treatment for conditions like congenital hypothyroidism and growth hormone deficiency often achieve normal pubertal development and final height outcomes (9). Malnutrition remains a leading and preventable cause of short stature in Pakistan, with long-term implications for health and development (10). Specific nutritional deficiencies—particularly vitamin D deficiency—can impair bone development, and while treatment may reverse rickets, it might not fully restore growth if intervention is delayed (11). Given the multifactorial nature of short stature and its significant variability in etiology and treatment approaches, understanding local epidemiological patterns is essential for targeted interventions and improved clinical outcomes. Therefore, the objective of the present study was to evaluate the various etiologies of short stature among children and adolescents presenting to a tertiary endocrine care center.

METHODS

A retrospective cohort study was conducted at Baqai Medical University (BMU), Karachi, following ethical approval from the Institutional Review Board of Baqai Institute of Diabetology and Endocrinology (BIDE/IRB/BashirIhsan/007/05/23/0025). Medical records from the past five years were retrieved through the Health Management System (HMS) of the institution. The sample size was calculated using the OpenEpi sample size calculator, yielding a minimum requirement of 120 participants (8). All patients, irrespective of gender, who presented with complaints of short stature and were aged between 2 and 20 years were included in the study. Individuals younger than 2 years or older than 20 years were excluded due to variability in growth assessment standards outside this age range. Demographic and clinical data were collected using a structured proforma. Parameters included age, gender, birth history, presence of chronic illness, and anthropometric measurements such as height, weight, and body mass index (BMI), which was computed using standard formulas. The mid-parental height was calculated using established equations: for boys, (father's height + mother's height + 13)/2; and for girls, (father's height − 13 + mother's height)/2, with the target height defined as the result ±10 cm to account for the normal range of variation (9). Growth charts were plotted for all participants to assess growth trajectories in relation to standard percentiles.

Relevant investigations were ordered based on clinical presentation. These included bone age estimation through hand and wrist radiographs, serum insulin-like growth factor-1 (IGF-1) levels, and insulin tolerance tests in cases where growth hormone deficiency was suspected. Baseline laboratory workups—such as complete blood count, thyroid-stimulating hormone (TSH), serum creatinine, and urinalysis—were conducted as part of the diagnostic evaluation. All data were compiled systematically by the research team using the

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attached proforma. Statistical analysis was performed using SPSS version 26. Comparative analyses between groups were conducted using Student's t-test for continuous variables, and chi-squared or Fisher's exact tests for categorical variables, depending on data distribution and cell counts. Correlation analysis was also carried out to assess the relationship between short stature and various clinical and biochemical parameters. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 120 participants were included in the study, comprising 56 males and 64 females. The overall mean age was 13.23 ± 3.18 years, with males averaging 12.68 ± 3.17 years and females 13.7 ± 3.13 years. The mean weight was 38.2 ± 13.85 kg (p = 0.727), and mean height was 136.38 ± 16.19 cm (p = 0.797). Body mass index (BMI) averaged 20.12 ± 4.63 kg/m² with no significant gender difference (p = 0.66). The average height of fathers was 167.48 ± 4.3 cm for male children and 165.52 ± 4.91 cm for female children (p = 0.023). Mid-parental height showed a statistically significant difference between genders, with males at 167.21 ± 5.37 cm and females at 154.07 ± 5.32 cm, and an overall mean of 160.2 ± 8.46 cm (p < 0.0001). Most participants had between zero to two siblings. Developmental milestones were normal in 99.2% of participants. All children were free from known comorbidities. A history of consanguinity was present in only 1.7%, while 0.8% had a family history of short stature. The majority of participants (99.2%) exhibited chronic short stature from an early age. The average birth weight was 2.27 ± 0.23 kg, with no statistically significant difference between genders (p = 0.715). Full-term deliveries accounted for 87.5% in males and 95.3% in females, while cesarean sections were performed in 80.4% of male and 78.1% of female births (p = 0.764). No gestational complications were reported. Gastrointestinal complaints were present in 7.1% of males and 7.8% of females (p = 0.999). Anti-tissue transglutaminase antibodies IgG were detected in 11.7% and IgA in 8.3% of participants.

Endocrine abnormalities were observed in 1.8% of males and 3.1% of females (p = 0.999). IGF-1 levels were comparable between genders (males: 274.59 ± 72.28; females: 283.56 ± 67.87; p = 0.485). Bone age was significantly higher in females (13.58 ± 3.2 years) compared to males (12.39 ± 3.25 years; p = 0.047). Among biochemical markers, hemoglobin levels averaged 11.34 ± 1.56 g/dL, and platelet counts showed a significant difference between genders (males: 288.5 ± 82.08; females: 319.27 ± 82.69; p = 0.044). Other parameters including creatinine, sodium, chloride, TSH, calcium, and vitamin D3 levels showed no significant gender differences. Normal growth variation was the most frequent cause of short stature, present in 33.3% of participants. This was followed by constitutional growth delay (22.5%) and idiopathic short stature (18.3%). Other less common causes comprised 25.9% of the cohort. Significant positive correlations with standard deviation (SD) scores were found for birth weight (r = 0.281, p = 0.002), hemoglobin (r = 0.243, p = 0.007), and IGF-1 levels (r = 0.184, p = 0.045). Negative correlations were observed for platelet count (r = -0.21, p = 0.022), IgA (r = -0.17, p = 0.024), IgG (r = -0.169, p = 0.025), and the presence of gastrointestinal problems (r = -0.149, p = 0.048). Mode of delivery was also positively correlated with SD (r = 0.194, p = 0.01). Age, BMI, and parental heights did not demonstrate any significant correlation with SD scores.

To strengthen the evaluation of short stature etiologies, subgroup analyses were conducted based on key endocrine and systemic conditions suspected or screened during participant assessments. Among the 120 children evaluated, hypothyroidism was suspected in 6 participants (5%), with elevated TSH levels observed in 4 cases and clinical follow-up confirming primary hypothyroidism in 2 children. Growth hormone deficiency was suspected in 4 participants (3.3%) based on low IGF-1 levels; however, confirmatory GH stimulation testing data were not available in the dataset. Possible celiac disease was indicated by positive anti-tissue transglutaminase IgG in 11.7% and IgA in 8.3% of participants. Clinically relevant gastrointestinal symptoms, such as malabsorption or chronic diarrhea, were present in 7.5% of cases, supporting further diagnostic correlation for celiac disease or related malabsorptive states. Vitamin D deficiency was prevalent in 47.5% of participants based on a threshold of <20 ng/mL, potentially contributing to poor bone mineralization and linear growth delays. Despite endocrine abnormalities being noted in a small proportion of the cohort (2.5%), these findings suggest the need for structured endocrine screening protocols in cases presenting with unexplained or disproportionate short stature.

Table 1: Study participants baseline characteristics

Parameters (Mean ±SD)	Male (n=56)	Female (n=64)	*P*-value	Overall
Age (years)	12.68±3.17	13.7±3.13	0.078	13.23±3.18
No of Siblings male				

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Parameters (Mean ±SD)	**Male (n=56)**	**Female (n=64)**	***P*-value**	**Overall**
0-2	47(83.9%)	52(81.2%)	0.7	99(82.5%)
3-4	9(16.1%)	12(18.8%)		21(17.5%)
No of Siblings female				
0-2	47(83.9%)	57(89.1%)	0.409	104(86.7%)
3-4	9(16.1%)	7(10.9%)		16(13.3%)
Weight (Kg)	38.68±14.9	37.79±12.97	0.727	38.2±13.85
Height (cm)	136.79±17.31	136.02±15.27	0.797	136.38±16.19
BMI (Kg/m^2)	20.32±4.69	19.95±4.6	0.66	20.12±4.63
Father Height (cm)	167.48±4.3	165.52±4.91	0.023	166.44±4.72
Mother Height (cm)	156.04±5.31	154.98±5.23	0.278	155.48±5.27
Mid Parental height (cm)	167.21±5.37	154.07±5.32	<0.0001	160.2±8.46
Mils stones achieved				
Normal	56(100%)	63(98.4%)	N/A	119(99.2%)
Delayed	0(0%)	1(1.6%)		1(0.8%)
Known co morbid				
No	56(100%)	64(100%)	N/A	120(100%)
Yes	0(0%)	0(0%)		0(0%)
History of consanguineous marriage				
No	54(96.4%)	64(100%)	N/A	118(98.3%)
Yes	2(3.6%)	0(0%)		2(1.7%)
Family history of short stature				
No	56(100%)	63(98.4%)	N/A	119(99.2%)
Yes	0(0%)	1(1.6%)		1(0.8%)
Always small/recent stunting of growth				
Recent	0(0%)	1(1.6%)	N/A	1(0.8%)
Always	**56(100%)**	**63(98.4%)**		**119(99.2%)**

Table 2: Results of Bio-chemical Investigations

Parameters (Mean ±SD)	**Male (n=56)**	**Female (n=64)**	***P*-value**	**Overall**
Hemoglobin	11.37±1.67	11.32±1.46	0.845	11.34±1.56
White blood cell count	6.39±1.76	6.37±1.46	0.957	6.38±1.6
Platelets	288.5±82.08	319.27±82.69	0.044	304.91±83.5
Creatinine	0.86±0.78	0.87±0.87	0.933	0.87±0.83
Sodium	136.89±14.19	139.22±3.11	0.204	138.13±9.98
Chloride	95.49±12.21	99.27±13.11	0.107	97.5±12.78
Thyroid stimulating hormone	3.8±5.64	5.05±9.13	0.379	4.47±7.69
Calcium	8.78±0.59	8.72±0.71	0.626	8.75±0.65
Vitamin D3	23.37±6.39	22.53±6.13	0.461	22.92±6.24
IGF 1 levels	274.59±72.28	283.56±67.87	0.485	279.38±69.81

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Bone Age	**12.39±3.25**	**13.58±3.2**	**0.047**	**13.03±3.26**

Table 3: Results of Correlation of SD with various parameters

Parameters	**Correlation coefficient**	***P*-value**
Age	-0.141	0.125
BMI	0.161	0.079
Father Height	0.025	0.786
Mother Height	0.168	0.067
Mid Parental height	0.126	0.172
Birth Weight	0.281	0.002
Hb	0.243	0.007
WBC	0.089	0.331
Platelets	-0.21	0.022
Creatinine	-0.107	0.247
Na	0.018	0.849
Cl	-0.116	0.205
TSH	-0.037	0.692
Calcium	0.045	0.623
Vitamin D3	0.154	0.093
IGA	-0.17	0.024
IGG	-0.169	0.025
IGF 1 levels	0.184	0.045
Bone Age	-0.074	0.423
Endocrine Problems	-0.015	0.84
GI Problems	-0.149	0.048
Diet History Top Feed	-0.011	0.886
Duration of gestation	-0.128	0.088
Mode of delivery	0.194	0.01
Family history of short stature	-0.103	0.17
No of Siblings male	-0.087	0.25
No of Siblings female	**-0.033**	**0.663**

Table 4: Subgroup Distribution by Endocrine and Systemic Etiologies (N=120)

Pathology	**Suspected/Confirmed Cases**	**Percentage (%)**
Hypothyroidism	2 confirmed / 6 suspected	5.0
Growth Hormone Deficiency	4 suspected	3.3
Celiac Disease (IgG+ or IgA+)	14 (IgG+), 10 (IgA+)	11.7 / 8.3
GI Symptoms Present	9	7.5
Vitamin D Deficiency (<20 ng/mL)	57	47.5
Endocrine Problems (general)	3	2.5

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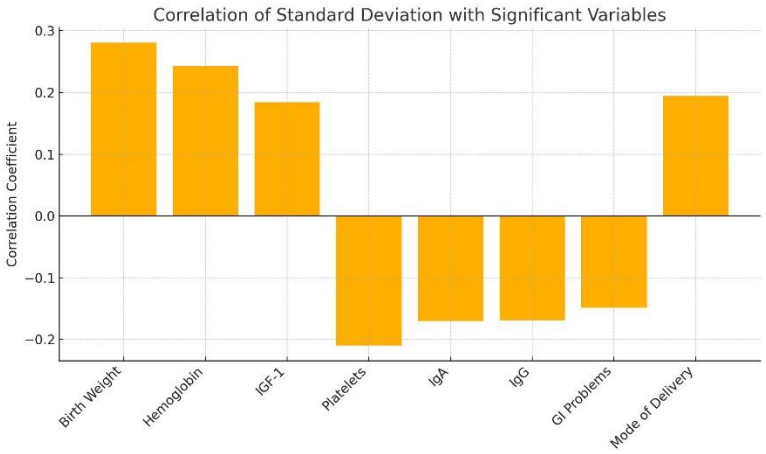

Figure 1 Correlation of Standard Deviation with Significant Variables

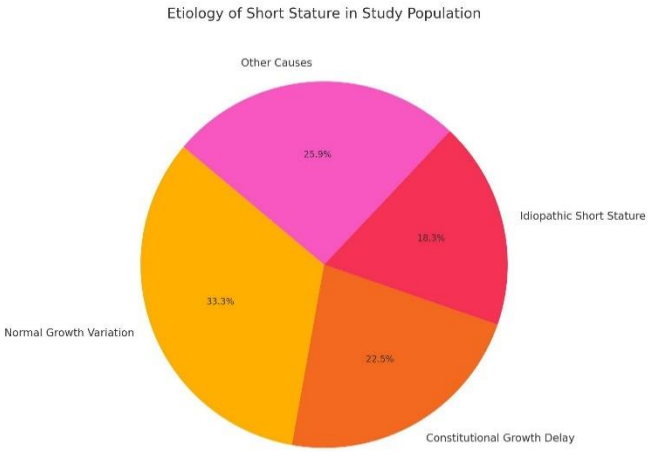

Figure 2 Etiology of Short Stature in Study Population

DISCUSSION

The present study explored the etiological profile and clinical characteristics of short stature among a pediatric and adolescent population. Normal growth variation emerged as the most frequent cause of short stature, accounting for 33.3% of the cases (8), followed by constitutional growth delay (22.5%) (9) and idiopathic short stature (18.3%) (10). These findings suggest that a substantial proportion of children presenting with concerns of short stature may actually fall within a spectrum of normal growth, albeit delayed or genetically predetermined. This aligns with earlier studies that emphasize the predominance of non-pathological short stature in outpatient pediatric settings (10,11). The recognition of such variants in clinical practice is essential to prevent over-investigation and unnecessary treatment, particularly in settings with limited resources. Comparative literature further supports the dominance of familial and constitutional growth patterns as leading causes of short stature (12,13). In the current cohort, constitutional growth delay, characterized by delayed but eventual catch-up growth, was second most common, reinforcing the notion that many children presenting with short stature may achieve final heights within the genetic target range without pharmacological intervention (14,15). While the frequency of endocrine abnormalities in this study was relatively low—1.8% in males and 3.1% in females—published literature has shown wide variability in the prevalence of endocrine causes, ranging from low single-digit percentages to over half of reported cases, depending on the population and diagnostic methods used (16,17). These discrepancies underscore the need for thorough clinical assessment and targeted endocrine evaluations, especially in patients who deviate significantly from growth norms or fail to respond to supportive management.

The role of gastrointestinal conditions was also evident in this analysis. Although GI symptoms were present in a small subset of participants (7.1% of males and 7.8% of females), the presence of positive anti-tissue transglutaminase antibodies (IgG in 11.7% and IgA in 8.3%) highlights the importance of considering celiac disease and related malabsorptive disorders as underlying contributors to impaired growth. These findings are consistent with several other regional and international studies that identify celiac disease as a notable, though often underdiagnosed, cause of short stature (18,19). The inclusion of serological screening for celiac disease in growth assessments may thus be justified, particularly when gastrointestinal complaints or nutritional deficiencies are also present. The study also revealed meaningful correlations between standard deviation (SD) scores for height and various clinical parameters. Positive correlations were found with birth weight, hemoglobin levels, and IGF-1, suggesting that early-life nutritional status and endocrine function positively influence growth potential (20). Conversely, negative correlations with platelet counts, IgA, IgG, and GI disturbances indicate that immune system activation or chronic gastrointestinal inflammation may adversely impact linear growth. Interestingly, a positive correlation was observed with mode of delivery, where children born via cesarean section demonstrated less deviation from average height SD; however, the clinical relevance of this observation remains uncertain and warrants further exploration.

A key strength of this study was its comprehensive collection of clinical, biochemical, and familial data, which enabled a multi-faceted analysis of growth patterns. The balanced gender representation enhances the generalizability of findings across male and female

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pediatric populations. The inclusion of immunological and endocrine markers added valuable depth to the diagnostic profiling. However, certain limitations must be acknowledged. The cross-sectional design restricts conclusions regarding temporal or causal relationships between variables and growth outcomes. Furthermore, the study was conducted at a single tertiary care center, which may limit the broader applicability of findings to other settings with differing nutritional, environmental, or genetic profiles. The lack of dynamic endocrine testing, such as GH stimulation or thyroid antibody profiling, also limits the ability to definitively classify certain cases. Future research incorporating longitudinal follow-up, more diverse populations, and comprehensive endocrine and genetic assessments could help refine diagnostic algorithms and management strategies for short stature.

CONCLUSION

In conclusion, this study underscores the diverse etiologies of short stature in children, with normal growth variation emerging as the most prevalent pattern, followed by constitutional growth delay and idiopathic causes. These findings reinforce the clinical need to differentiate between benign and pathological growth patterns to avoid unnecessary interventions. The observed associations between growth parameters and early life nutritional and hormonal indicators further emphasize the importance of comprehensive evaluation in pediatric growth assessment. By highlighting the predominance of non-pathological short stature, this research contributes to more informed, efficient, and context-sensitive management strategies in pediatric endocrinology.

AUTHOR CONTRIBUTION

Author	Contribution
Ihsan Bashir	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Wajid Ali*	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Musarrat Riaz	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Saima Askari	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Urooj Lal Rehman	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Zaheeruddin	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published

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