Causes and Etiologies of Short Stature in Pediatric Endocrine Clinic in Basrah, Southern Iraq during 2024

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***Abstract*—Child growth is a key indicator of overall health and depends on adequate nutrition and coordinated hormonal regulation, including growth hormone, insulin-like growth factor-1, thyroid hormones, and gonadal steroids. Short stature, typically defined as height more than two standard deviations below the mean, is the most common reason for referral to pediatric endocrinology clinics. This retrospective cross-sectional study aimed to identify the underlying causes of short stature among children attending the Pediatric Endocrinology Clinic at Al-Faiha Specialized Diabetes, Endocrine, and Metabolism Center in Basrah, South Iraq, and to evaluate the relative frequency of etiologies alongside demographic and anthropometric factors. The study reviewed electronic medical records of patients aged 2–18 years seen between January 1 and December 31, 2024, and included 618 eligible cases.**

Adolescents aged 12–18 years constituted the largest group (57.5%), and males accounted for 56.8% of cases ($p<0.05$). Most patients (57.3%) were from rural areas of Basra. Pathological causes were the predominant category, representing 65.6% of cases. Among normal variants, familial short stature was the most frequent aetiology (18.1%, $p<0.001$), followed by idiopathic short stature (11%), constitutional growth delay (4%), and small for gestational age (1.3%). Within pathological causes, growth hormone deficiency (GHD) was the leading diagnosis, accounting for 30.6% of cases ($p=0.004$), and was more common in males (60.8%) than females (39.2%), although this difference was not statistically significant ($p=0.12$). Overall, the findings indicate that pathological short stature—particularly due to GHD—predominates in this population, and that many children in Basrah are identified at a relatively late age, potentially limiting the benefits of early therapeutic intervention.

***Keywords*—Short stature; Growth hormone deficiency; Pediatric endocrinology; Familial short stature; Basrah.**

INTRODUCTION

Child growth is a fundamental indicator of overall health and reflects the combined effects of adequate nutrition and coordinated hormonal regulation, including growth hormone (GH), insulin-like growth factor-1 (IGF-1), thyroid hormones, and gonadal steroids (1,2). Monitoring growth is therefore an essential component of pediatric care and preventive health programs. Standard anthropometric measurements—particularly height, weight, and growth velocity—are widely used to detect deviations from normal development, which are typically expressed as standard deviations from population means (1,3). Short stature (SS) is the most common reason for referral to pediatric endocrinology services (4) and is generally defined as height below −2 standard deviations for age and sex (5). Advances in molecular genetics have expanded understanding of the etiologies of SS and shifted clinical attention toward growth plate biology and targeted therapies (6). Early identification of abnormal growth is crucial because untreated short stature may adversely affect both physical and psychosocial well-being (3).

Normal linear growth is regulated through the hypothalamic–pituitary–GH–IGF-1 axis. Growth hormone-releasing hormone stimulates pulsatile GH secretion from the anterior pituitary, while somatostatin provides inhibitory control (7). GH promotes hepatic production of IGF-1, which in turn stimulates epiphyseal cartilage proliferation and bone elongation (6,7). Growth follows predictable phases—infancy, childhood, and puberty—with variation in timing between sexes. Early growth is strongly influenced by maternal and intrauterine factors, whereas later growth increasingly reflects genetic

potential (8). During puberty, synergistic increases in GH and sex steroids produce the characteristic growth spurt (9).

Short stature results from impaired linear growth at some stage of development (5). Globally, stunting affects more than 162 million children under five years of age (10), while approximately 2.3% of any general population falls below the −2 SD threshold (11). Reported prevalence of pathological short stature varies widely (1.3–19.8%) depending on referral patterns and diagnostic criteria (12). Worldwide, the most common causes include constitutional delay of growth and puberty (CDGP), familial short stature (FSS), and growth hormone deficiency (GHD) (8). Prevalence also varies geographically, with reported rates of 4.9% in Jordan (14), 33.68% in Taif, Saudi Arabia (13), 1.93% in England (15), and 2.88% in China (16). Data from Iraq remain limited; however, a recent study in Dhi Qar reported a stunting frequency of 3.6% among primary school children (18).

Etiologically, stature is genetically determined but modified by environmental, nutritional, and hormonal influences (11,19). Physiological variants—primarily FSS and CDGP—are the most frequent causes and typically present with normal growth velocity despite reduced height percentiles (11). In contrast, pathological short stature is more likely when height falls below −3 SD and may result from chronic systemic disease, endocrine disorders, skeletal dysplasias, genetic syndromes, or children born small for gestational age (11). Endocrine causes include GH deficiency, hypothyroidism, and panhypopituitarism (20–23), while metabolic conditions such as poorly controlled diabetes can also impair growth through disruption of the GH–IGF axis (24). Genetic syndromes including Turner, Noonan, and Prader–Willi syndromes represent important contributors (25). Nutritional rickets and other metabolic bone diseases further highlight the broad differential diagnosis (26,27).

Accurate evaluation of children with SS requires detailed clinical assessment, family and perinatal history, and careful anthropometry (4,28). Initial investigations typically include screening laboratory tests, thyroid function, celiac screening, and bone age radiography (11,29). Additional studies—such as karyotyping in girls, GH stimulation testing, IGF-1/IGFBP-3 measurement, and brain MRI—are guided by clinical suspicion (30,31). Management depends on the underlying cause and aims to optimize growth velocity and final adult height. Recombinant human growth hormone therapy has revolutionized treatment, particularly for GHD and several non-GHD conditions including Turner syndrome, SHOX deficiency, and children born small for gestational age (8,32). Many children with physiological variants require only reassurance and monitoring, whereas pathological causes necessitate targeted therapy and multidisciplinary care (33).

Delayed growth remains a major parental concern and a frequent reason for endocrine referral. Given the wide range of potential etiologies and limited regional data, particularly in Iraq, systematic evaluation of affected children is essential.

This study aims to identify and analyze the underlying etiologies of short stature among pediatric patients attending the endocrine clinic in Basrah, South Iraq, and to assess the relative frequency of different causes alongside associated demographic and anthropometric factors.

METHODS

This study was designed as a retrospective, record-based cross-sectional analysis conducted at the Pediatric Endocrinology Clinic of Al-Faiha Specialized Diabetes, Endocrine, and Metabolism Center (FDEMC), a tertiary referral center in Basrah, Iraq. The study targeted children and adolescents aged 2–18 years who attended the clinic for the first time in 2024 and were diagnosed with short stature. Data collection was performed between February 1 and June 30, 2025, using electronic medical records. Short stature was defined as height below the third percentile or more than two standard deviations (SDS) below the mean for age and sex (5).

A total population sampling approach was used. The final sample included 618 children aged 2–18 years who met the inclusion criteria between January 1 and December 31, 2024. Eligible participants were those presenting for the first time with short stature confirmed by CDC growth chart standards, while

cases with incomplete records or prior treatment for short stature were excluded. Ethical approval and official endorsement were obtained from the Ministry of Health–Iraq, Basrah Health Directorate, and FDEMC, and all data were handled according to confidentiality and privacy standards.

Extracted data included demographic characteristics (age, sex, and residence), anthropometric measurements, pubertal assessment, and etiological classification. Age was categorized into three groups (2–5 years, 6–11 years, and 12–18 years), sex was recorded as male or female, and residence was classified as urban, rural, or other governorates. Height was measured in centimeters and interpreted using the 2000 CDC growth charts. Pubertal status was assessed clinically using Tanner staging, with children younger than 8 years in girls and 9 years in boys considered prepubertal (34).

Height-for-age Z-scores were calculated using CDC standards (35), and the severity of short stature was categorized as mild (−2 to −3 SDS), moderate (−3 to −4 SDS), severe (−4 to −5 SDS), and extreme (< −5 SDS). For consistency, Z-scores exactly at cutoff values were included in the lower severity category. Etiologies of short stature were classified into normal variants—familial short stature (FSS), constitutional delay of growth and puberty (CDGP), idiopathic short stature (ISS), and small for gestational age (SGA)—and pathological causes, including growth hormone deficiency, chronic diseases, genetic syndromes, malnutrition, psychological causes, endocrine/metabolic disorders, and combined etiologies.

Statistical analysis was performed using SPSS version 30.0 and Microsoft Excel. Continuous variables were expressed as mean ± standard deviation or median as appropriate, while categorical variables were presented as frequencies and percentages. Group comparisons were conducted using the unpaired t-test, Chi-square test, and one-way ANOVA where applicable. A p-value of less than 0.05 was considered statistically significant.

RESULTS

The study covered 618 cases in total. The predominant age group was 12–18 years (57.5%), followed by 6–11 years age group at (37.5%). This difference was statistically significant ($p<0.05$). Males represented 56.8% of the participants, that was also statistically significant ($p<0.05$). According to Table 1, the majority of participants (57.3%) were from Basra's rural areas, while only 1.3% was from other governorates. This difference was statistically significant ($p<0.05$).

Table 1: Demographic distribution of study sample

Variable	Demographic profile	Frequency of Short Stature	percentage	P-value
Age (year)	2-5	31	5.0%	<0.001
	6-11	232	37.5%	
	12-18	355	57.5%	
Sex	Male	351	56.8%	<0.001
	Female	267	43.2%	
Residence	Urban (Basra)	256	41.4%	<0.001
	Rural (Basra)	354	57.3%	
	Other Governorate	8	1.3%	
Total		618	100%	

The study population's average age was 11.7 ± 3.61 years, with a median age of 12 years old, as in Table 2.

Table 2: Distribution of study population by age characteristics

	Age (years)
Mean Age ± SD cm	11.7 ± 3.615
Median	12

In particular, total number was 14 with the mean age 4.2 ± 0.97 for males and total number 18 with mean age 3.9 ± 1.02 for females in 2–5 years age group it was statistical insignificance ($p>0.05$), in the 6–11 years age group the total number 114 with the mean age 8.6 ± 1.72 for males and 118 with mean age 8.9±1.82 for females and this was not statistically significant ($p>0.05$), also according 12–18 years age group total number was 223 with the mean age 14.6 ± 1.61 for males and 131 with mean age 13.8 ± 1.59 for females and this was statistically significant ($p<0.05$). Overall, the mean age of the males included in the study was 12.3 ± 3.64 While the mean age of the

females was 11.0 ± 3.48, There was a statistically significant difference (p<0.05), as shown in Table 3.

Table 3: Distribution of the study population by sex and the mean age according to their age groups.

Age (year)	Male No. Mean Age ± SD	Female No. Mean Age ± SD	p-value *
2-5	14 4.2 ± 0.97	18 3.9 ± 1.02	0.403
6-11	114 8.6 ± 1.72	118 8.9 ± 1.82	0.19
12-18	223 14.6± 1.61	131 13.8 ±1.59	<0.001
Total	351 12.3± 3.64	267 11.0± 3.48	<0.001

An evaluation of the average height z-scores between both gender across various age groups, as shown in Table 3.4, revealed that females had lower mean z-score than males in all age groups, as noted there was no statistical significance in this difference (p>0.05) in all age group 2-5 ,6-11 and 12-18 years. In total there was statistical significance p <0.05 between males and females in severity of short stature according to mean z-score.

Table 3.4: Comparison means Z-score of height between males and females by age groups

Age (year)	Male	Z-score	Mean Height ± SD cm	Female	Z-score	Mean Height ± SD cm	p-value*
2-5	14	-3.95 ±1.86	87.02 ± 10.8	18	-4.57 ±1.22	82.59 ± 7.68	0.29
6-11	114	-3.14 ± 1.04	112.76 ± 11.43	118	-3.38 ±1.10	112.81 ± 10.21	0.08
12-18	223	-3.05 ± 0.71	140.94 ± 9.69	131	-3.17 ±0.97	136.31 ± 9.12	0.21
Total	351	-3.12 ± 0.91	129.63 ± 18.78	267	-3.35 ±1.09	122.45 ± 18.13	0.01

The bulk of the study population (42.3%) were of -2 to -3 z-score (mild short stature) followed (40.5%) by -3 to -4 z-score (moderate short stature) and the remainder had severe form of short stature. Male participants were almost equally spread between the z-score ranges of -2 to -3 and -3 to -4, with proportions of (43.8%) and (43.6%) respectively. while Females show mostly (40.4%) mild severity short stature -2 to -3 followed by moderate severity in about (36.4%); additionally, females had the severest form of short stature comparing to males and this was significant statistically (p<0.05) as shown in Table 5.

Table 5: Distribution of study population by severity of short stature

Z-Score Range	Male %	Female %	Total %	P-value*
-2 to -3	154 43.8	108 40.4	262 42.3	0.001
-3 to -4	153 43.6	97 36.4	250 40.5	
-4 to -5	34 9.7	38 14.2	72 11.7	
Below -5	10 2.9	24 9.0	34 5.5	
Total	351 100	267 100	618 100	

Regarding pubertal status, only 19.7% of males and 19.9% of females were in the prepubertal stage. Most of the study population had passed through puberty, but this difference was statistically insignificant (p<0.05), as shown in Table 6 and Figure 1.

Table.6: Distribution of the study population by gender and pubertal status

Puberty	Male	%	Female	%	P value*
prepubertal	69	19.7	53	19.9	1.0
pubertal	282	80.3	214	80.1	
Total	351	100	267	100%	

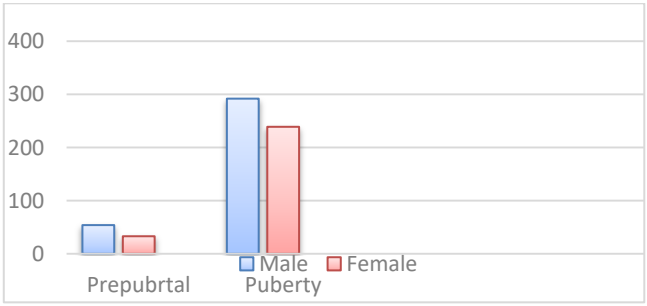


Figure 1: Distribution of the study population by pubertal status

Pathological causes (65.6%) were the leading cause of Short Stature among the study population, including various pathological conditions, while normal variants structured 34.4%, as shown in Figure 2.

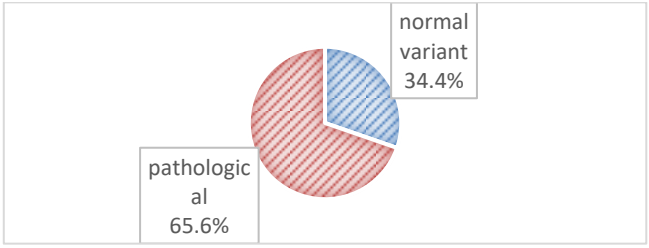


Figure 2: Classification of main causes of Short Stature

Pathological short stature (65.6%) was significantly more common than normal variants (34.4%) among the studied population (p < 0.001). Although no statistically significant correlation was seen between age groups and the underlying cause of short stature (P = 0.1). However, a higher quantity of pathological causes 244 (68.8%) was observed in older age group (12–18 years), while the normal variant causes were relatively more common in the 6–11 age group 92 (39.6%). This may indicate a possible trend, although it was not statistically significance. There was a statistically significant association between gender and the underlying cause of short stature (P = 0.033). Although pathological causes were predominant in both sexes (69.3% for males and 60.7% for females). Females had a relatively higher proportion of normal variant short stature compared to males (39.3% vs. 30.7 in males %). Regarding place of residence, both urban and rural participants showed a predominance of the pathological causes of short stature (63.3% and 67.8%), respectively. In contrast, cases belonging to other governorates were ordered as having a normal variant as the cause of their short stature, and this was not statistically significant, as shown in Table 7.

Table 7: Distribution of study population by demographic profile in correlation with the main causes of Short Stature.

		Normal variant	Pathological	Total	p-value
Age		No. %	No. %	No. %	
2-5 **6-11** **12-18**		10 32.2 92 39.6 111 31.2	21 67.8 140 60.4 244 68.8	31 100 232 100 355 100	0.1
Sex	Male	108 30.7	243 69.3	351 100	0.03
	Female	105 39.3	162 60.7	267 100	
Residence					0.12
Urban		94 36.7	162 63.3	256 100	
Rural		114 32.2	240 67.8	354 100	
Other Governorate		5 62.5	3 37.5	8 100	
Total		213 34.4	405 65.6	618 100	<0.001

In Table 8, considering the pubertal status of the study population and the main causes of short stature was assessed separately for males and females we found that, the majority of pubertal boys 73.1% having pathological cause for their short stature compared to 47.1% in prepubertal group, which was a statistically significant association (P = 0.001). conversely, more than half of prepubertal males (52.6%) had normal variant short stature. In female, although pathological causes were more frequent in both pubertal 63.6% and prepubertal (56.6%) groups, the difference was not statistically significant in females (P = 0.438).

Table 8: Distribution of study population by Pubertal status in correlation with the main causes of Short Stature

	Normal variant	Pathological	Total	p- value
Pubertal status	No. %	No. %	No %	
Male: **Prepubertal** **Puberty**	36 52.9 76 26.9	33 47.1 206 73.1	69 100 282 100	0.001
Female **Prepubertal** **Puberty**	23 43.4 78 36.4	30 56.6 136 63.6	53 100 214 100	0.438
Total	213 34.4	405 65.6	618 100	

In Table 9, and Table 10 outlines the specific etiologies of short stature, categorized into normal variant and pathological causes. About normal variants, familial short stature (FSS) represented the most frequent etiology (18.1%) among the other causes, and it was statistically significant p value <0.001, and also showed a significant association with female gender (56% females vs. 44% males; p = 0.002). In contrast, other normal variants including idiopathic short stature (ISS)11%, constitutional growth delay (CGD) 4%, and small for gestational age (SGA) 1.3%, and did not demonstrate any significant gender association (p > 0.05). Regarding pathological causes which represent the majority (65.6%) of cases. growth hormone deficiency (GHD) was the leading cause, accounting for 30.6% of cases and it was statistically significant p-value=0.004. It was more prevalent in males (60.8%) than in females (39.2%), with no significant p-value of (0.12). Other pathological categories including metabolic, genetic, chronic diseases, and psychological causes did not show significant gender differences, likely due to smaller subgroup sizes. Notably, cases with combined etiologies were significantly more common in males (76.2%) than females (23.8%), with a highly significant p-value of (0.001). In details Diabetes

mellitus accounts for (10%), with nearly equal representation among males and females. Moreover, CD structured about 8.2%, sickle cell anemia and thalassemia contribute 2.8%, while rickets account for 1.5%, skeletal dysplasia for 0.5%, hypothyroidism, Turner syndrome, anemia, and malnutrition each for 0.3%, and finally Russell syndrome, Noonan syndrome, precocious puberty and Autism is 0.2%. However, none were statistically significant.

Table 9: Distribution of short stature according to etiological cause

Normal variant	frequency	p-value
Familial SS	112 (18.1%)	<0.001
Constitutional GD	25 (4 %)	
SGA	8 (1.3%)	
Idiopathic	68 (11.0%)	
Subtotal	213	
Pathological:		
Endocrine		
GHD	189 (30.6%)	P = 0.004
Hypothyroidism Precocious puberty	2 (0.3%) 1 (0.2%)	
Metabolic		
DM	62 (10%)	
Genetic Syndromes		
Russel	1 (0.2%)	
Noonan	1 (0.2%)	
Turner	2 (0.3%)	
Chronic Diseases		
Celiac	51 (8.2%)	
SCA and Thalassemia	17 (2.8%)	
IDA	2 (0.3%)	
Malnutrition	2 (0.3%)	
Ricket	9 (1.5%)	
Skeletal dysplasia	3 (0.5%)	
Neurodevelopmental		
Autism	1 (0.2%)	
Combination of causes	62 (10%)	
Subtotal	405	
Grand total	618 (100)	

Table 10: Etiological classification of Short Stature with gender distribution

Causes	Frequency (%)	Male	Female	P value
Normal variant				
Familial SS	112 (18.1%)	49 (44%)	63 (56%)	0.002
Constitutional GD	25 (4 %)	12 (48%)	13 (52%)	1.00
SGA	8 (1.3%)	5 (62.5%)	3 (37.5%)	0.94
Idiopathic	68 (11.0%)	36 (52.9%)	32 (47.1%)	0.49
Subtotal	213	102	111	
Pathological:				
Endocrine				
GHD	189 (30.6%)	115 (60.8%)	74 (39.2%)	0.12
Hypothyroidism	2 (0.3%)	1(50%)	1 (50%)	-----
Precocious puberty	1 (0.2%)	0	1(100%)	
Metabolic				
DM	62 (10%)	32 (51.6%)	30(48.4%)	
Genetic Syndromes				
Russel	1 (0.2%)	1(100%)	0	
Noonan	1 (0.2%)	1(100%)	0	
Turner	2 (0.3%)	0	2(100%)	
Chronic Diseases				
Celiac	51 (8.2%)	34(66.6%)	17(33.3%)	0.109
SCA and Thalassemia	17 (2.8%)	10 (58.8%)	7(41.2%)	0.94
IDA	2 (0.3%)	0	2(100%)	------
Malnutrition	2 (0.3%)	1(50%)	1(50%)	------
Ricket	9 (1.5%)	4 (44.4%)	5(55.6%)	------
Skeletal dysplasia	3 (0.5%)	1(33.3%)	2(66.7%)	------
Neurodevelopmental				
Autism	1 (0.2%)	1(100%)	0	------
Combination of causes	62 (10%)	48 (76.2%)	15 (23.6%)	0.001
Subtotal	405	249	156	
Grand total	618 (100)	351(56%)	267(44%)	

Figure 3.3: The following chart illustrates the distribution of the causes of short stature in the hierarchy

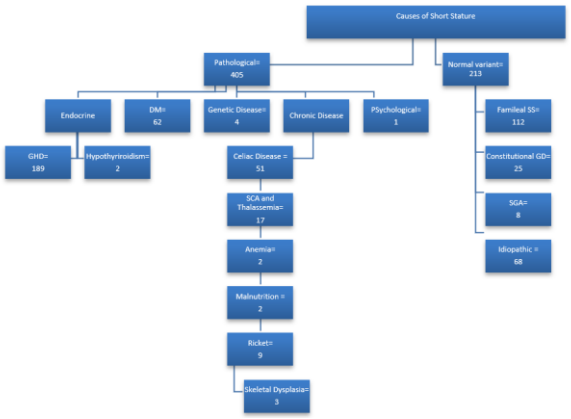
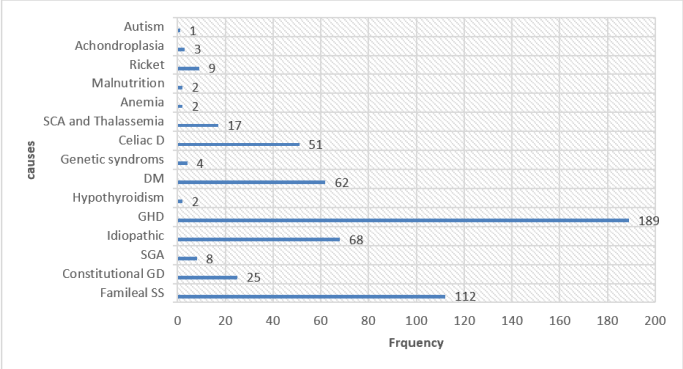


Figure 3.4: Causes of Short Stature in the study population



Children with familial short stature and constitutional GD had the mildest degree of severity, with a mean z-score of -2.46 and -2.85, respectively. Additionally, moderate severity was associated with GHD, DM, Celiac disease, SCA, and Thalassemia, with mean z-scores ranging from -3.1 to -3.67. Other pathological causes, such as Rickets mean z-score -4.13, genetic syndromes -5.48, and Achondroplasia -5.71, were of a severe degree of short stature; this was statistically significant by using the ANOVA test.

Table 11: Distribution of the causes of short stature by their mean Z-score and degree of severity

Causes	Mean Z-score	Sample size (n)	Degree of severity
1. Familial SS	-2.46	112	Mild
2. Constitutional GD	-2.85	25	Mild
3. GHD	-3.1	189	Moderate
4. DM	-3.22	62	Moderate
5. Celiac	-3.43	51	Moderate
6. SCA and Thalassemia	-3.67	17	Moderate
7. Ricket	-4.13	9	Severe
8. Genetic syndromes	-5.48	4	Severe
9. Achondroplasia	-5.71	3	Severe

The ANOVA test revealed a significant difference in mean Z-scores between the 9 groups of causes of short stature ($F=6.11$, $p<<0.001$); this suggests that at least one group has a mean Z-score differs significantly from others that is statistically significant. As shown in Table 12.

Table 12: ANOVA test results

Group	Comparison Mean Difference	p-value
Achondroplasia	3.25	<0.001
Genetic syndromes	3.02	<0.001

DISCUSSION

Delayed growth in children is a major parental concern and may result from multiple underlying conditions; delayed diagnosis and treatment can reduce the likelihood of achieving genetic height potential, making early identification and management essential (36). In 2024, the center recorded approximately 35,000 pediatric visits, yet only 618 cases met the diagnostic criteria for short stature after applying exclusion rules. This highlights the importance of strict diagnostic assessment to prevent overdiagnosis and suggests that many clinic attendees may not have true short stature. Similar patterns have been reported elsewhere; Song et al. found that 89.4% of hospitalized children had normal height, reflecting increasing societal concern about stature (37). In Kuwait, Al-Abdulrazzaq et al. reported that 78.3% of children referred for short height met criteria, meaning a substantial minority were referred despite normal stature, underscoring the need to strengthen referral pathways (34).

The mean age at presentation in this cohort was 11.7 ± 3.61 years, with males presenting at an older mean age than females, and the predominant age group being 12–18 years. This likely reflects delayed recognition until growth failure becomes more apparent with age and pubertal expectations. Sex-related differences in growth patterns may also contribute, as males typically achieve greater adult height due to longer prepubertal growth and a more prolonged pubertal growth spurt, whereas estrogen accelerates epiphyseal closure in females; genetic influences may also differ by sex (38–40). Comparable age trends have been described in India and Jordan, with Rajput et al. reporting a similar pattern of younger female presentation and older male presentation (36), and Alassaf et al. reporting similar mean ages between sexes (41). Sharma et al. likewise noted that short stature tends to be detected more frequently in older children (42). However, some studies show younger age at presentation, such as Hussein et al. in Egypt (43), Al-Abdulrazzaq et al. in Kuwait (34), and Karim et al. in Bangladesh (44), likely reflecting differences in access to care, referral practices, and awareness.

Males accounted for 56.8% of cases, and this male predominance may reflect referral bias and sociocultural pressures for boys to be taller, which can influence help-seeking behavior and the likelihood of receiving interventions such as recombinant human growth hormone (45). Other studies have reported either a similar male excess or no significant sex difference, including Rajput et al. (45), Alassaf et al. (41), Hussein et al. (43), and Al-Abdulrazzaq et al. (34). In contrast, Karim et al. reported a female predominance, highlighting that referral and cultural patterns vary across settings (44). Most participants were from rural Basrah, consistent with findings from Bangladesh, and this may relate to differences in nutrition, access to healthcare, and delayed recognition in rural communities (44).

Most children in this study had mild to moderate short stature, but females had a higher proportion of severe cases, which may reflect delayed presentation and differing social expectations. Findings from Tunisia and Kuwait demonstrate variability in height SDS patterns between sexes and settings (46,34), and some differences may reflect parental concern and referral patterns rather than biological differences alone (41). Severity was also associated with etiology: normal variants tended to have milder deficits, while severe

forms were more common with syndromic and skeletal causes, supporting observations that lower height SDS increases the likelihood of pathological disease (37). Similar associations between etiology and severity have been reported in Kuwait and Jordan, where children with normal variants were taller than those with GHD or Turner syndrome, and GHD was prominent in severe short stature (34,41).

Most participants had already passed puberty at presentation, contrasting with studies in Kuwait and Jordan where most referrals were prepubertal (34,41). This is clinically important because earlier detection—especially before puberty—improves response to treatment in conditions such as GHD, where height gain correlates with younger age and prepubertal initiation of therapy (47,48). Pathological causes were more frequent than normal variants in this cohort, and GHD was the leading diagnosis. Similar patterns have been reported in Jordan and Korea, where pathological etiologies were slightly more common and GHD featured prominently (41,37), and in Tunisia where pathological causes were overwhelmingly dominant and endocrine disorders were a major contributor (46). Differences from other reports, including Kuwait where normal variants were most common (34), may reflect referral-center bias, diagnostic practices, availability of parental height data (which affects classification of FSS), and regional population differences (36). Normal variant patterns were broadly consistent with other studies, with FSS being the most common normal variant in several cohorts, while the proportion of ISS differed between studies, likely due to differing definitions and limited access to advanced genetic testing (43,34,50,37,41).

Within pathological causes, GHD was the most frequent diagnosis, followed by chronic disease and combined etiologies. Several studies similarly report GHD as a leading endocrine cause, although the relative ranking varies by population; Hussein et al. reported GHD followed by hypothyroidism and diabetes (43), while Song et al. and Kuwait data show differing proportions of GHD, SGA, and Turner syndrome (37,34). Papadimitriou et al. also reported that a notable minority meet auxological and biochemical criteria for GHD (50). In contrast, Rajput et al. found hypothyroidism to be the most common endocrine cause and reported a different distribution of GHD and diabetes, illustrating how etiological profiles vary with referral criteria, healthcare systems, and population characteristics (36).

CONCLUSIONS AND RECOMMENDATIONS

This study demonstrates that congenital adrenal This study demonstrates that pathological short stature constitutes the majority of cases, with growth hormone deficiency (GHD) identified as the leading underlying cause. Despite this, a substantial proportion of children referred for evaluation had normal growth patterns, highlighting the need to strengthen referral pathways and avoid overdiagnosis. Children in Basrah tend to present at a relatively late age, potentially reducing the benefits of early intervention, particularly for GHD. The higher proportion of diagnosed males may reflect referral bias influenced by sociocultural preferences regarding male height. Most patients had mild to moderate short stature, but those with height below −3 to −4 SDS were more likely to have pathological causes, underscoring the importance of careful evaluation in this group. Accordingly, routine growth monitoring should be reinforced within primary healthcare—especially in rural areas—alongside improved maternal and child nutrition. Clear referral guidelines, increased awareness among parents and healthcare providers, and timely evaluation for GHD are essential. Wider adoption of Z-score–based prioritization, ensuring availability of key diagnostic tests, cautious use of GH testing based on strict criteria, and expansion of genetic evaluation services are recommended to improve early detection, diagnostic accuracy, and overall management of short stature.

Conflicts of Interests: None

Funding: No funding body was involved in this study.

Ethical Approvals: Ethical approval for the study was obtained from the relevant institutional review board, and informed consent was acquired from all participants prior to their inclusion in the study.

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