Al Ameen J Med Sci 2021;14(4):298-304 ● US National Library of Medicine enlisted journal ● ISSN 0974-1143

ORIGINAL ARTICLE CODEN: AAJMBG

A study of short stature among adolescents in the rural tertiary center- A prospective observational study

Dixa Shah[1], Sunil Pathak[2], Prasad Muley[3], Prashant Modi[3*], Naveed M Ahmed[2] and Vivek Bhatt[2]

[1]Consultant Pediatrician, Divine Plus Child Care Hospital, Tagore Road, Near Nagar Boarding, Rajkot-360001, Gujarat, India, [2]Department of Pediatrics, Smt. B.K. Shah Medical Institute & Research Center, Sumandeep Vidyapeeth, Waghodia, Vadodara-391760, Gujarat, India and [3]Department of Pediatrics, Parul Institute of Medical Sciences & Research, Parul University, Waghodia, Vadodara-391760, Gujarat, India

Received: 04th November 2020; ***Accepted:*** 22nd September 2021; ***Published:*** 01st October 2021

Abstract: *Background:* Long-term morbidities during the adolescence period cause short stature which is permanent, once epiphysis fuse. *Objectives:* To evaluate etiology in short stature adolescents and its relation with HSDS (Height Standard Deviation Score) and BA (Bone Age) delay. *Methods:* The prospective observational study was conducted at a rural tertiary hospital. Anthropometry, BA, and etiology of short stature in hospitalized adolescents (10 to 18 years) were assessed. *Results:* The mean CA (Chronologic Age), mean HA (Height Age), and mean BA were 12.82 (2.20) years, 8.55 (1.92) years, and 10.76 (3.18) years, respectively. Overall non-endocrinal diseases (54%) were most prevalent followed by idiopathic short stature (20%), endocrinal diseases (16%), and normal variants (10%). The mean BA was the least delay in normal variants 0.60 (1.63) years in comparison to endocrinal diseases 2.81 (4.47) years. Severe short stature (mean HSDS ≤ – 3) was commonly seen in pathologic short stature than normal variants (mean HSDS between -2 to -3). *Conclusion:* The common etiology of short stature amongst adolescents were chronic systemic diseases followed by idiopathic short stature, endocrinal diseases, and normal variants. The more delay in BA and severe short stature (HSDS < -3), the pathological short stature is more likely.

Keywords: Adolescents, Malnutrition, Pathological short stature, Standard deviation score

Introduction

Adolescents are adults of the future and comprise one-fifth (18%) of the total population of India [1]. Adolescence is the second most important period of rapid growth in humans. Linear growth halts at the end of puberty, once epiphysis fuses [2]. Any significant morbidity especially of long duration during the pubertal period falters body growth and causes short stature; which is irreversible if there is a delay in diagnosis and intervention. Body image perception and physical fitness greatly affect behavior and self-esteem of adolescents [3-4].

Short stature remarkably influences psychosocial aspects like social competence, behavior problems, self-esteem, and family functioning; which end in poor self-rated health and decreased life satisfaction in children and adolescents [5-6]. Jia Ma et al have reported the prevalence of short stature of 3.87% among adolescents [7]. While studies from India have reported variable prevalence of short stature like 5.6% and 13.8% [8-9]. Short stature in children may be normal variant of growth or maybe pathologic resulting from a variety of etiology like endocrinal diseases and many non-endocrinal diseases. Non-endocrinal diseases include chronic systemic diseases, malnutrition, chromosomal disorders, skeletal developmental disorders, etc. Hence restoration of ultimate adult height by early intervention is crucial for improvement in self-esteem and life satisfaction [10].

Therefore, with this viewpoint, the study was planned to assess growth and to evaluate etiology of short stature amongst adolescents and its correlation with HSDS (Height

Standard Deviation Score) and BA (Bone Age) delay. The final results may contribute to available data and helps in building management guidelines on short stature in adolescents.

Material and Methods

This is a prospective observational study; in which 50 adolescent children with short stature (length/ height for chronological age less than 3 percentile or minus 2 SD as per standard growth chart) [11], hospitalized in the pediatric ward of Dhiraj Hospital during December 2017 to July 2019, were included after approval from the institutional ethics committee. All children were enrolled after taking written informed consent from parents or guardians and also separate assent from children. While children and/ or those whose parents or guardians did not give consent to participate were excluded.

The purposive sampling was done where adolescent children seeking health care were enrolled during the study period. Data was collected as per predesigned proforma.

Tools used in the study were

- Suzuki K9 electronic weighing scale for weight measurement with an accuracy of 10 gms.
- A wall mounted stadiometer for height measurement.
- The gender-specific standard IAP growth charts.
- Non-stretchable clinical measuring tape for the upper segment (US) and lower segment (LS) measurement.
- Tanner staging chart for assessment of puberty.

Weight, height, BMI, and US: LS were determined as per standard methods and formulas [11]. Furthermore HSDS (Height Standard Deviation Score) was derived by using the following formula: HSDS= (Observed height- Mean height) / 1SD, where 1 SD was calculated from height for age growth charts [12]. All the enrolled participants were investigated and treated as per standard protocol of the diagnosed disease. Bone age was assessed by doing an age-appropriate skeletal x-ray and using Greulich and Pyle's Standards. Serum TSH and free-T4 were done by using the chemiluminescent immunoassay (CLIA) method. Epi Info Ver-7 software was used to analyze data statistically. Quantitative data are presented as frequency, proportion, ratio, and percentage.

Results

In the present study, 50 adolescents (10-18 years of age) were enrolled. Amongst them 21 (42%) were girls and 29 (58%) were boys with the M: F ratio of 1.38:1.

Table-1: Gender specific descriptive statistics of anthropometry of subjects

Parameters	Female (n=21)	Male (n=29)	Total (n=50)
Mean CA* (SD) Years	12.71 (1.95)	12.89 (2.39)	12.82 (2.20)
Mean HA† (SD) Years	8.42 (1.72)	8.63 (2.08)	8.55 (1.92)
Mean BA‡ (SD) Years	11.40 (2.61)	10.30 (3.51)	10.76 (3.18)
Mean Weight (SD) Kgs	24.84 (8.90)	25.62 (8.40)	25.29 (8.53)
Mean Height (SD) cms	127.88 (10.26)	129.62 (11.80)	128.89 (11.11)
Mean BMI§ (SD) Kgs/m^2	14.83 (3.37)	15.18 (4.21)	15.03 (3.85)
Mean HSDS¶	– 3.11 (0.97)	– 3.14 (1.08)	– 3.12 (1.03)

** CA: Chronological Age, † HA: Height Age, ‡ BA: Bone Age, § BMI: Body Mass Index, ¶ HSDS: Height Standard Deviation Score*

As shown in Table-1, the overall mean CA, mean HA, and mean BA were12.82 (2.20) years, 8.55 (1.92) years, and 10.76 (3.18) years, respectively. The mean CA and HA were marginally high in boys than in girls; while the mean BA was high in girls than in boys. The overall mean

value of anthropometry parameters like weight, height, and BMI were 25.29 (8.53) kgs, 128.89 (11.11) cms, and 15.03 (3.85) kgs/m^2, respectively. The mean values of all anthropometry parameters were slightly higher in the boys than in girls. The mean HSDS was – 3.12 (1.03) without any gender predilection.

Table-2: Gender-Etiology wise distribution of participants (n=50)

Primary	Female (n=21)	Male (n=29)
Normal Variants (5, 10%) • Constitutional short stature (2, 4%) • Familial short stature (3, 6%)	**1 (4.7%)**	**4 (13.8%)**
Endocrinal Diseases (8, 16%)	**4 (19.1%)**	**4 (13.8%)**
Non-Endocrinal Diseases (37, 74%)	**16 (76.2%)**	**21 (72.4%)**
• Idiopathic (10, 20%)	6 (28.5%)	4 (13.8%)
• Respiratory diseases (6, 12%)	3 (14.4%)	3 (10.4%)
• Gastrointestinal diseases (5, 10%)	3 (14.4%)	2 (6.9%)
• Renal diseases (5, 10%)	0	5 (17.4%)
• Hematologic diseases (3, 6%)	1 (4.7%)	2 (6.8%)
• Neurologic diseases (3, 6%)	2 (9.5%)	1 (3.4%)
• Systemic inflammatory disorders (2, 4%)	0	2 (6.09%)
• Skeletal deformity (2, 4%)	1 (4.7%)	1 (3.4%)
• Syndromic short stature (1, 2%)	0	1 (3.4%)

In the present study, the incidence of normal variants and pathologic short stature were 10% (5) and 90% (45), respectively (shown in Table-2). Amongst 45 pathologic short stature children, endocrinal diseases, non-endocrinal diseases, and Idiopathic short stature were seen in 8 (17.7%), 27 (60%), and 10 (22.3%) adolescents, respectively. The gender-wise distribution of etiology revealed that renal diseases (17.4%), endocrine disorders (13.8%), idiopathic short stature (13.8%), etc. were common in boys. Similarly, idiopathic short stature (28.5%), endocrine disorders (19.1%), respiratory disorders (14.4%), gastrointestinal diseases (14.4%) and neurologic diseases (9.5%) were more common in girls.

Etiological distribution as per nutritional stature showed 17 (63%) children of non-endocrinal diseases, 1 (12.5%) child of endocrinal diseases and 6 (60%) children of idiopathic short stature were thin with an overall incidence of the thinness of 24 (53.33%) in 45 adolescents with pathological short stature. The incidence of obesity among pathologic short stature adolescents were 2 (4.44%).

The constitutional short stature and familial short stature were seen in 2 (4%) and 3 (6%) children, respectively. The proportion of common causes in the present study was idiopathic short stature 10 (20%), hypothyroidism 7 (14%), asthma (6%), tuberculosis (6%), chronic kidney diseases (6%), ulcerative colitis (4%), sickle cell diseases (4%), etc. (Table-3).

Table-3: Causes of pathologic short stature

Idiopathic short stature - 10 (20%)
Endocrine disorders • Hypothyroidism - 7 (14%) • Cushing syndrome - 1 (2%)
Respiratory diseases • Asthma - 3 (6%) • Tuberculosis- 3 (6%)
Renal diseases • Chronic kidney disease- 3 (6%) • Distal renal tubular acidosis - 1 (2%) • Frequent relapse nephrotic syndrome - 1 (2%)

Cont...Table-3: Causes of pathologic short stature
Gastrointestinal diseases • Ulcerative colitis - 2 (4%) • Functional abdominal pain - 1 (2%) • Juvenile polyposis syndrome - 1 (2%) • Wilsons disease with rickets - 1 (2%)
Systemic inflammatory disorders • Rheumatoid arthritis - 1 (2%) • Takayasu arteritis - 1 (2%)
Neurologic diseases • Cerebral palsy - 1 (2%) • Intellectual disability- 2 (4%)
Hematologic diseases • Sickle cell disease with b thalassemia trait - 1 (2%) • Sickle cell disease - 2 (4%)
Skeletal deformity • Atlanto-axial subluxation with kyphosis - 1 (2%) • Dorso-lumbar severe kyphosis - 1 (2%)
Syndromic short stature • Seckel syndrome - 1 (2%)

In the current study, the overall mean BA delay was 1.95 (2.79) years and gender-wise statistics demonstrated more delayed BA in boys than girls (Table-4). The etiology specific distribution of mean BA delay revealed significant BA delay in endocrinal diseases 2.81 (4.47) years followed by non-endocrinal diseases 1.95 (2.44) years and normal variants 0.60 (1.63) years.

Table-4: Mean BA delay– etiology and –gender specific distribution statistics

	Mean BA Delay (SD) Years		
Etiology	**Overall**	**Female**	**Male**
Endocrinal diseases (8)	2.81 (4.47)	1.5 (0.91)	4.12 (6.42)
Non-endocrinal diseases (37)	1.95 (2.44)	1.03 (1.59)	2.65 (2.77)
Normal variants (5)	0.60 (1.63)	0.5	0.62 (1.88)
Total (50)	1.95 (2.79)	1.09 (1.44)	2.57 (3.34)

As shown in Table-5, the mean HSDS in normal variants was slightly low (-2.32); while in pathologic short stature the mean HSDS was significantly low (below -3). The mean HSDS was around -3.53 in neurologic diseases, renal diseases, and endocrinal diseases.

Table-5: Mean HSDS – etiology distribution statistics of participants

Etiology	Mean	Std Dev
Neurologic diseases	-3.55	0.66
Renal diseases	-3.53	1.51
Endocrine disorders	-3.5	1.18
Gastrointestinal diseases	-3.26	0.91
Idiopathic	-3.22	0.88
Respiratory diseases	-3.16	1.18
Systemic inflammatory disorders	-3.05	1.58
Syndromic short stature	-2.9	NaN
Skeletal deformity	-2.55	0.2
Hematologic diseases	-2.4	0.39
Normal variants	-2.32	0.32

Discussion

In our study, the M: F ratio was 1.38:1; while Su Wu et al have reported M: F ratio was 1.59:1 [13]. Other similar studies had also reported M: F ratio of 1.28:1; [14-15] which was similar to the present study. On the contrary, Melody Seb Rengma et al study with a total of 1818 participants reported the M: F ratio of 0.84:1 [16].

In the current study, the mean CA, mean HA, and mean BA were 12.82 (2.20) years, 8.55 (1.92) years, and 10.76 (3.18) years, respectively. The mean CA and mean HA were slightly higher in boys than girls. The mean BA in girls was advanced by 1.1 (0.9) years than boys. In the Mehboob Sultan et al study with participants' age range of 2-15 years, the mean CA, mean HA, and mean BA were 6.1 (3.1) years, 4.3 (2.6) years, and 5.0 (2.8) years, respectively [17].

The possible reason for this may be a difference in the age group of enrolled participants. In a similar study by Fahim Ullah et al, the mean CA was 11.75.3 (4.06) years with a range of 2-20 years and the mean BA

was 8.56 (4.03) years [14]. Other studies have reported the range of the mean BA from 6.94 (3.04) to 7.8 (2.8) years [13, 18]. In these last two studies, the prevalence of endocrinal diseases was higher than non-endocrinal diseases; which may be a possible reason for less BA than the present study [13, 18].

In the current study, the value of mean weight, mean height and mean BMI, were 25.29 (8.53) kgs, 128.89 (11.11) cms, and 15.03 (3.85) kgs/m^2, separately. The boys were taller and heavier than the girls. In Su Wu et al study, the mean weight, mean height and mean BMI was24.03 (8.13) kgs, 120.20 (15.35) cms, and 16.66 kgs/m^2, respectively; [13] which was similar to the current study. A large cross-sectional survey of adolescents also revealed similar findings [16]. The study mean height SDS was -3.12 (1.03); while in Su Wu et al study mean height SDS was -2.37 (1.05) [13]. This difference was because of a high incidence of pathological short stature than normal variants in the present study, which was opposite to Su Wu et al study.

In the present study, non-endocrinal diseases (54%) were more prevalent followed by idiopathic short stature (20%), endocrinal diseases (16%), and normal variants (10%). There was no difference in the distribution of etiology across both genders. Su Wu et al have reported endocrinal diseases (50.43%) commonest etiology followed by idiopathic short stature (41.40%), non-endocrinal diseases (4.56%), and normal variants (3.59%) with no difference in distribution across gender [13]. Other studies have reported Constitutional short stature as the commonest cause in males and Familial short stature as a commonest cause in female subjects [17, 19].

In the Shazia Kulsom Lashari et al study, normal variants were the most common group (55%) followed by endocrinal diseases (28%) and non-endocrinal diseases (17%) [20]. Bhadada SK et al reported normal variants in 36.1%, endocrinal diseases in 30.09%, and non-endocrinal diseases in 33.81% cases [19]. In a similar Pankaj Garg et al study, the incidence of normal variants, endocrinal etiology and non-endocrinal etiology was 24.4%, 4.7% and 70.9%, respectively [21]. The present study was done at a tertiary hospital with an enrolment of those adolescents who were admitted to the pediatric ward, which resulted in a high incidence of pathologic short stature than normal variants.

The study incidence of hypothyroidism and chronic renal failure was 14% and 6%, respectively. In other studies, the incidence of hypothyroidism ranges from 13.7% - 15% [19-20, 22]. The incidence of thinness among pathological short stature was 54.44%. (24/45) and of these, isolated malnutrition causing severe short stature was seen in 6/45 (13.33%) adolescents i.e. adolescents having idiopathic short stature. It means malnutrition was directly or indirectly a major contributing factor to short stature. The other similar studies have reported incidence of malnutrition as the only cause for short stature was ranging between 5.1% - 7.8% [19, 22-23]. The majority of participants of the present study were belonging to the rural area having a high prevalence of malnutrition; which may be the possible reason for this difference in malnutrition incidence.

The overall mean BA delay was 1.95 (2.79) years and gender-wise the mean BA was more delay in boys than girls. While Fahim Ullah et al has reported the mean bone age delay of 3.23 (1.94) years [14]. The possible reasons for this difference were (i) Age of participants: In Fahim Ullah et al study, participants' age range was 2-12 years; while in the present study it was 10-18 years and (ii) High proportion of normal variants (35.61%) in Fahim Ullah et al study in comparison to that of the present study (10%). Etiology-wise, the mean BA delay was least in normal variants and high in endocrinal diseases because linear growth almost ceases in Endocrinal diseases in contrast to normal variants in which skeletal growth continues at a slow rate.

In the present study, severe short stature (mean HSDS ≤ -3) was seen in neurological diseases, renal diseases, endocrine diseases, gastrointestinal diseases, idiopathic short stature, and systemic inflammatory disorders. While moderate short stature (mean HSDS between -2 and -3) was observed in normal variants, hematologic diseases, skeletal deformity, and syndromic shorts stature. In

the Tansit Saengkaew et al study, the common etiology in severe short patients was syndromic short stature (16.2%) and Growth Hormone Deficiency (14.1%) and in the moderate short stature group, constitutional delay of growth and puberty was the most common etiology (34.1%) [24]. The limitations of this study were: (i) Karyotyping could not be done in all idiopathic short stature girls. (ii) Few adolescents with idiopathic short stature could not be evaluated after first-line investigations due to financial constraints and 4/10 (40%) of them left against medical advice.

Conclusion

Short stature is slightly more common in adolescent boys than girls. Pathologic short stature is more common than normal variants in hospitalized adolescents. Chronic systemic diseases amongst non-endocrinal diseases and hypothyroidism amongst endocrinal diseases are the commonest cause of short stature in adolescents. Malnutrition is a major direct or indirect cause of short stature. The more delay in BA and severe short stature (HSDS < -3), the chances of having pathological short stature especially endocrinal diseases are more. The group of adolescents with Idiopathic short stature needs special attention and approach for better management.

Acknowledgment

We acknowledge the support provided by Dr. Manish Rasania, Dr. BakulJavadekar, and colleagues. We thank our patients and their parents for their participation in the study.

Financial Support and sponsorship: Nil

Conflicts of interest: There are no conflicts of interest.

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Cite this article as: Shah D, Pathak S, Muley P, Modi P, Ahmed NM, Bhatt V. A study of short stature among adolescents in the rural tertiary center- A prospective observational study. *Al Ameen J Med Sci* 2021; 14(4): 298-304.

*All correspondences to: Dr. Prashant Modi, Associate Professor, Department of Pediatrics, Parul Institute of Medical Sciences & Research, Parul University, Waghodia, Vadodara-391760, Gujarat, India. E-mail: dpmm408@gmail.com