

## A study of clinical profile of children with short stature

Kumar Angadi<sup>1</sup>, Sharanagouda Patil<sup>2</sup>, Mohammed Mukeeb Ahmed<sup>3</sup>, Shivanand S Bhimalli<sup>4</sup>From <sup>1</sup>Associate Professor, <sup>2</sup>Professor and HOD, <sup>3</sup>Junior Resident, <sup>4</sup>Professor, Department of Paediatrics, Mahadevappa Rampure Medical College, Kalaburagi, Karnataka, India

## ABSTRACT

**Background:** Short stature is one of the common concerns among the Indian children. However, community-based studies in Indian context are rare compared to studies performed in Western countries. **Aim:** Here, we aimed to study the clinical profile of children with short stature and the different causes of it. **Methods:** This study was a hospital-based prospective study that included 65 children  $\leq 18$  years (female: 57%, male: 43%) who met the inclusion criteria. Detailed history, physical examination, anthropometry, laboratory tests, bone age, and chromosomal analysis were included for evaluation. **Results:** The most common age group affected was 11–15 years (40%) followed by 6–10 years (34%) and  $< 5$  years (21.5%). Growth hormone (GH) deficiency (21.4%), followed by malnutrition (18.5%), familial short stature (13.9%), hypothyroidism (10.8%), turner syndrome (10.8%), constitutional delay of growth and puberty (9.2%), systemic diseases (6.2%), and miscellaneous (6.1%) were identified as the common causes of short stature. **Conclusion:** Commonly observed etiology of short stature was identified in this study. Proportionate and disproportion short statures were 90.7% and 9.3%, respectively. About 23% of the cases were of physiological short stature and 77% were of pathological short stature.

**Key words:** Familial short stature, Growth hormone deficiency, Short stature

Short stature (SS) is one of the most common causes for referral to the pediatric endocrinology clinic [1]. Altered growth potential may result from disturbances of the endocrine system, skeletal system, altered nutrition, or chronic diseases [2]. Normal growth is considered to be an indirect indicator of the overall well-being of the child, any alteration or disturbance in the linear growth is found to be a concern to the family. Approximately 3% of children in any population are found to be short, among which half of them will be physiological (familial or constitutional) half will be pathological [3]. Although SS is not a disease *per se*, it is a manifestation of several diseases. Its early diagnosis and treatment are the most of the time rewarding [4]. Moreover, many reports indicated that children with SS were academically and socially handicapped. While western literature is replete with studies on SS, there are very few studies from the Indian subcontinent. The current study was undertaken to enrich the literature about the SS among the Indian children.

## MATERIALS AND METHODS


The present study was carried out in the Department of Pediatrics, Mahadevappa Rampure Medical College, Kalaburagi. Patients' attendees were informed about the purpose of the study and

written consent was taken before initiation of the study. Ethical clearance was obtained by the ethical clearance committee of the institution. The study of clinical profile of children with SS was carried out during the period from October 2018 to April 2020.

Inclusion criteria were children  $< 18$  years of age of either sex with height  $< 3^{\text{rd}}$  centile according to the revised growth chart of Indian Academy of Pediatrics. The exclusion criteria were height  $> 3^{\text{rd}}$  percentile.

Data were analyzed by IBM SPSS 20.0 software. For qualitative data analysis, we applied the Chi-square test while Student's t-test and ANOVA test were used for quantitative data analysis.  $p < 0.05$  was considered statistically significant.

The etiological evaluation of 65 children was done by detailed history, physical examination, anthropometry, routine investigations such as complete blood count, renal function tests, liver function tests, urine routine, thyroid function tests, bone age [5] by Tanner white house 3, karyotyping, magnetic resonance imaging brain, and clonidine stimulation test. Cases were recruited based on inclusion and exclusion criteria after obtaining informed parental consent. Systemic diseases, nutritional disorders, and chronic infections were the symptoms to rule out. Cases with the history of consanguinity, family history of SS, mode of delivery, any antenatal events, postnatal complications, development history, dysmorphic features, constipation, history suggestive

Access this article online	
Received - 15 June 2021 Initial Review - 28 June 2021 Accepted - 20 August 2021	Quick Response code 
DOI: 10.32677/IJCH.2021.v08.i09.3022	

**Correspondence to:** Dr. Kumar Angadi, Associate Professor, Department of Paediatrics, Mahadevappa Rampure Medical College, Gulbarga, Karnataka, India. E-mail: drkumarangadi@gmail.com

© 2021 Creative Commons Attribution-NonCommercial 4.0 International License (CC BY-NC-ND 4.0).

CNS infection/raised ICP, and genitalia examination were also ruled out.

Growth formula used is as follows: Height age (HA)<weight age (WA)<bone age (BA)=chronological age (CA) – Familial SS; HA<WA<BA<=CA – CGD; WA<HA<BA<CA-Malnutrition; BA<=HA<WA<=CA-endocrine disease. Growth is strongly related to genetic potential. A child’s mid parental height is calculated as follows: Girl=(mother’s height in cm+father’s height in cm)/2–6.5 cm. Boy=(mother’s height in cm+father height in cm)/2+6.5 cm.

**RESULTS**

A total of 65 cases (female: 37 (57%) and male: 28 (43%); age: 11–15 years) were studied of here. The age and etiology profile among the study population are shown in Fig. 1.

In female, common cause of SS include familial SS(FSS)(12.3%), followed by turner syndrome (10.8%), growth hormone (GH) deficiency (9.2%), hypothyroidism (9.2%), and malnutrition (7.7%). In male, GH deficiency (12.8%) is the most common cause of SS followed by malnutrition (10.8%), constitutional delay of growth and puberty (CDGP) (6.1%), systemic diseases (4.6%), and FSS (1.5%). Table 1 shows the family history of SS and etiology and Table 2 shows the etiology as per the birth weight.

Table 3 shows the etiology as per the growth formula and Table 4 shows the results of the clonidine stimulation test.

Among 65 cases, 20 cases underwent CST. Out of them, 14 cases were having low basal and post-CST value and were diagnosed as GH deficiency, that is, failure to reach 7 ng/ml after clonidine stimulation.

**DISCUSSION**

This hospital-based prospective study included 65 children with SS with an aim to find out the demographic characteristics, clinical profile, and causes of SS in children up to 18 years of age attending the endocrine outpatient department.

Hussein et al. [6] reported normal variant SS (63.6%) with CGD (15.8%) and FSS (42%) being the most common causes of SS followed by endocrine causes (26%) and systemic diseases (9%). In our study, endocrine causes of SS are most common followed by normal variants (FSS 13.8% and CDGP 9.2%), malnutrition, turner syndrome, and systemic disease. Hussein et al. further

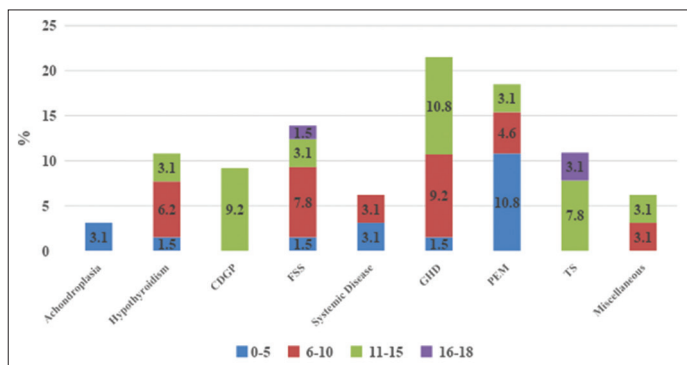


Figure 1: The age and etiology profile among the study population

reported that, in endocrine causes of SS, GH deficiency is the most common etiology followed by hypothyroidism which is comparable with our study.

Bhadada et al. [4] reported that 30% of cases of SS are due to endocrine diseases and 7% are due to systemic diseases. In our study, 32% of cases are of endocrine diseases and 6% of cases are due to systemic diseases which are comparable with the study.

Ramagopal and Vasudevan [7] reported that 20% of cases of SS are due to malnutrition which is comparable with our study (18%). The author also reported that 58% were of proportionate SS and 42% were of disproportionate SS. In our study, 90.7% were of proportionate SS and 9% were of disproportionate SS.

In our study, endocrine cause of SS is more common than normal variants. Among the endocrine causes, GH deficiency is the common, which is correlated with that reported by Shiva and Nikzad [8], Othman et al. [9], and Hussein et al. [6]. Moreover, the current study noted female preponderance comprising 57%, the common age of presentation was 11–15 years followed by 33.8 years, <5 years, which is correlated with that reported by Bhadada et al. [4].

Zargar et al. [10] reported that BA was close to HA in GH deficiency. Similar findings are seen in the present study. Al-Jurayyan NA et al. [11] reported that intrauterine growth retardation (23.8%) was the most common cause of SS in non-endocrine etiology. Similar findings are seen in the present study. However, in the present study, the sample size is small and it was done at a tertiary care center. Hence, the study population does not reflect the general population.

Table 1: Family history of SS and etiology

Causes of SS	Frequency (n)	Percentage
FSS	09	42.8
CDGP	06	28.6
GHD	06	28.6
Total	21	100

SS: Short stature, FSS: Familial short stature, CDGP: Constitutional delay of growth and puberty, GHD: Growth hormone deficiency

Table 2: Birth weight and etiology

Etiology	Low birth weight (LBW), n (%)	Normal weight, n (%)	Total, n (%)
Achondroplasia	0	2 (3.1)	2 (3.1)
Hypothyroidism	0	7 (10.8)	7 (10.8)
CDGP	0	6 (9.2)	6 (9.2)
FSS	6 (9.2)	3 (4.6)	9 (13.8)
Systemic disease	0	4 (6.2)	4 (6.2)
GHD	2 (3.1)	12 (18.4)	14 (21.5)
Protein-energy malnutrition (PEM)	1 (1.5)	11 (16.9)	12 (18.4)
Turner syndrome (TS)	3 (4.6)	4 (6.2)	7 (10.8)
Miscellaneous	0	4 (6.2)	4 (6.2)
Total	12 (18.5)	53 (81.5)	65 (100)

SS: Short stature, FSS: Familial short stature, CDGP: Constitutional delay of growth and puberty, GHD: Growth hormone deficiency

**Table 3: Growth formula and etiology**

Etiology	CA (Mean±SD)	HA Mean±SD)	BA (Mean±SD)	WA (Mean±SD)
Achondroplasia	1.83±0.70	0.50±0.23	0.91±0.82	0.83±0.82
Hypothyroidism	8.44±2.54	3.36±1.71	3.98±2.41	3.90±2.39
CDGP	11.90±1.41	8.04±1.18	8.09±3.61	8.86±1.18
Familial SS (FSS)	8.35±4.14	5.78±3.07	7.66±4.10	5.43±3.24
Systemic disease	4.81±4.54	2.06±1.85	3.60±3.70	2.10±2.96
GHD	9.25±2.37	4.35±1.76	4.75±1.95	4.88±1.78
Protein-energy malnutrition (PEM)	5.53±3.74	3.40±2.67	3.88±2.83	2.63±2.45
Turner syndrome (TS)	12.65±4.0	6.93±1.88	10.01±3.05	7.96±2.66
Miscellaneous	9.64±3.20	5.95±3.30	6.12±3.83	5.64±4.51
Total	8.59±4.08	4.87±2.91	5.99±3.65	4.75±3.25

SS: Short stature, FSS: Familial short stature, CDGP: Constitutional delay of growth and puberty, GHD: Growth hormone deficiency, CA: Chronological age, HA: Height age, BA: Bone age, WA: Weight age

**Table 4: Clonidine stimulation test (CST)**

Name of the values	GHD Mean±SD	Non-GHD Mean±SD	p-value
GH basal value	0.22±0.20	3.75±1.20	0.001*
GH post-clonidine stimulation	4.17±3.25	16.91±2.40	0.001*

GH: Growth hormone, GHD: Growth hormone deficiency, \*:Statistically significant

## CONCLUSION

The most common age of presentation is 11–15 years and incidence was more in females compared to males (ratio 1.3:1). Most commonly observed etiology of SS was GH deficiency followed by malnutrition, FSS, hypothyroidism, turner syndrome, CDGP, systemic diseases, and miscellaneous. In female, common cause of SS include FSS followed by turner syndrome, GH deficiency, hypothyroidism, and malnutrition. In male, GH deficiency is the most common cause of SS followed by malnutrition, CDGP, systemic diseases, and FSS.

## REFERENCES

1. Cakan N, Kamat D. Short stature in children: A practical approach for primary care providers. *Clin Pediatr (Phila)* 2007;46:379-85.
2. Kamboj M. Short stature and growth hormone. *Indian J Pediatr* 2005;72:149-57.

3. Short Stature: Evaluation and Management; 2016. Available from: <http://www.apiindia.org/medicine> update. [Last accessed on 2016 Jul 12].
4. Bhadada SK, Agrawal NK, Singh SK, Agrawal JK. Etiological profile of short stature. *Indian J Pediatr* 2003;70:545-7.
5. Spampinato C, Palazzo S, Giordano D, Aldinucci M, Leonardi R. Deep learning for automated skeletal bone age assessment in X-ray images. *Med Image Anal* 2017;36:41-51.
6. Hussein A, Farghaly H, Askar E, Askar E, Metwalley K, Saad K, *et al.* Etiological factors of short stature in children and adolescents: Experience at a tertiary care hospital in Egypt. *Ther Adv Endocrinol Metab* 2017;8:75-80.
7. Ramagopal G, Vasudevan J. Clinical and etiological profile of children with pathological short stature. *Int J Contemp Pediatr* 2017;4:73-7.
8. Shiva S, Nikzad A. Etiology of short stature in Tabriz University of medical college Iran. *Iran J Pediatr* 2009;19:35-40.
9. Othman RE, Naous A, Habr G, Rajab M. Short stature identified in children presenting at Makassed General Hospital. *Eur J Biomed Pharm Sci* 2018;5:34-40.
10. Zargar AH, Laway BA, Masoodi SR, Wani AI, Salahuddin M. An aetiological profile of short stature in the Indian subcontinent. *J Paediatr Child Health* 1998;34:571-6.
11. Al-Jurayyan NA, Mohamed S, Al Otaibi HM, Al Issa ST, Omer HG. Short stature in children: Pattern and frequency in a pediatric clinic, Riyadh, Saudi Arabia. *Sudan J Paediatr* 2012;12:79-83.

*Funding: None; Conflicts of Interest: None Stated.*

**How to cite this article:** Angadi K, Patil S, Ahmed MM, Bhimalli SS. A study of clinical profile of children with short stature. *Indian J Child Health*. 2021; 8(9):328-330.