

# A Hospital Based Study of Short Stature among children in India from the Eastern UP Population

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## **Abstract**

**Introduction:** Accurate anthropometric measurements and critical analysis of growth data allow the clinician to promptly recognize children with short stature. The aim of this study was to determine the frequency of etiological factors causing short stature among children referred to the department of pediatric and endocrinology, Institute of medical Science, Banaras Hindu University.

**Design:** Retrospective population-based study.

**Methods:** To assess the prevalence of hospitalization, a questionnaire-based study was conducted on subjects suffering from Short Stature to record the details of their lifestyle, habits and familial history. All subjects were of Indian ethnicity from Eastern Uttar Pradesh and Bihar, the two states of northern Indian population. Patients were characterized in terms of their socio-demographic and clinically diagnosed characteristics, Evaluation included: detailed medical history, physical examination, laboratory tests, bone age and chromosomal analysis.

**Results:** Endocrinological causes accounted for 20% of short stature of them, 10.9% had growth hormone deficiency (GHD), 58.6% had normal variants of growth [of them, 42% had familial short stature (FSS), 14.18% had constitutional growth delay (CGD) and 4.4% a combination of both]. Interestingly, celiac disease (CD) constituted 6.6% of children with short stature in our cohort.

**Conclusions:** Although potentially treatable causes such as GHD, hypothyroidism and CD accounted for a considerable percentage of short stature in our study, the majority of short stature in children had normal variations of growth. Growth hormone treatment in children, however, should be promptly initiated with specific clinical indications. CD is a not uncommon cause of short stature.

**Keywords:** constitutional growth delay, growth hormone deficiency, short stature

### **Introduction:**

Short stature is a common pediatric endocrine problem. Short stature is an adult height that is more than 2 standard deviations (SD) under the mean for age and gender; also, it corresponds to the shortest 2.3% of individuals since normal growth is a barometer of health in childhood, any child who is growing normally, virtually excludes chronic physical or mental illnesses. Hence, yearly evaluation of height and weight of all children is mandatory to assess their growth potential. The short stature, although not a disease per se, is a manifestation of several diseases. Its early diagnosis and treatment is most of the time, rewarding. Literature is replete with studies on short stature; there are very few studies from Indian subcontinent. Since multiple factors viz. genetic, prenatal, postnatal and local environmental factors, affect the growth, their relative significance would be variable in different populations.

The study are design on the basis of physical and hormonal imbalance and etiological factor are associated with short stature patient.

### **Factors affecting human growth:**

Linear growth of an individual and final adult height is determined by his/her genetic potential. However true realization of one's growth potential depends upon general wellbeing, nutrition and hormonal milieu like growth hormone, thyroid hormone, insulin like growth factor 1,2, IGFBP 3. From weeks 4 to 8, there is rapid growth and differentiation to form all the major organ systems in the body. In the second trimester,

the fetus undergoes major cellular hyperplasia and in the third, organ systems mature in preparation for extrauterine life. Throughout gestation, fetal growth is constrained by maternal factors and placental function but is coordinated by growth factors [2]. These can act locally in a paracrine manner [eg. IGF-I and IGF-II, fibroblast growth factor, epidermal growth factor, transforming growth factors  $\alpha$  and  $\beta$ ] or as endocrine hormones (e.g. insulin). Nutrition from the mother plays a rate-limiting role. During the first year, infants grow rapidly but at a sharply decelerating rate [1]. A similar pattern is observed for weight gain. It has been postulated that nutritional input is the principal regulator of growth over this period with minimal contribution from growth hormone. Data from humans and transgenic animal models suggest that the hormones and receptors within the GH-IGF axis also play their part in this early phase of growth. Nevertheless, it is during this period that alterations in dietary intake are likely to have the greatest impact on growth.

Although growth charts give the impression that growth is linear, most studies of short-term growth (day to day, week to week) find it to be non-linear. By 4 years of age, average growth velocity has declined to 7 cm/year and remains relatively steady until adolescence, the prepubertal nadir in average velocity being 5 to 5.5 cm/year. On an individual basis, there is a well-recognized mid-childhood growth spurt. Additionally, if an individual is measured throughout childhood, oscillations in growth velocity of variable amplitude are observed with a periodicity of approximately 2 years [5].

During childhood, growth hormone, in addition to thyroid hormone, is the major determinant of growth. It is therefore the time when dysfunction in the GH axis may be recognized. There is also wide variation in pubertal timing within each sex. The later onset of puberty in boys gives them two additional years of prepubertal growth compared with girls. The height gained in this time (8–10 cm), in addition to the greater amplitude of pubertal growth in boys (3–5 cm

more than the female growth spurt), gives rise to the 12.5 cm difference in adult height between the sexes.

Constitutional delay of growth and puberty is common and can be considered a variant of normal. The condition can be associated with chronic disease, for example atopy, but more often occurs in isolation. Pubertal development commences late and the growth spurt is blunted. Although constitutional delay in growth and puberty may actually present in the pubertal years, some children may have shown slow growth much earlier in childhood. The corollary is constitutional tall stature and early puberty, which is associated with more intense pubertal growth than normal. The net result is that both early and late maturers should achieve a comparable height.

The degree of physical development and the timing of the pubertal growth spurt complicate assessment of growth velocity around puberty (6). It is this variation that gives rise to the wide variation in peak height velocity on growth charts.

<b>Table 8. Physiologic factors that affect GH secretion.</b>	
<b>Factors that stimulate GH secretion</b>	<b>Factors that suppress GH secretion</b>
Exercise	Hypothyroidism
Stress	Obesity
Hypoglycemia	Hyperglycemia
Fasting	High carbohydrate meals
High protein meals	Excess glucocorticoids
Sleep	

## **Epidemiology**

### **United States**

By definition, 2.5% of the population is short. However, the number of children with poor linear growth is higher given the frequency of chronic diseases of childhood. The Utah Growth Study is the largest population-based survey of growth in children published to date. These investigators assessed height and growth velocity in nearly 115,000 American children. Among the 555 children with short stature (defined as height below the third percentile) and poor growth rate (defined as growth velocity < 5 cm annually), only 5% had an endocrine disorder. In addition, 48% of the children with growth hormone deficiency (GHD) or Turner syndrome (TS) in this large cohort had been undiagnosed or untreated.

Parents often suspect an endocrine disorder (eg, GHD) as the major cause of short stature in their child. In fact, the Utah Growth Study confirms that most (95%) children with poor growth (velocity < 5 cm/y) do not have an endocrine disorder.

## **India**

A Study in Indian population on 15644 children belonging to 23 schools were evaluated, and 448 (2.86%) children had SS. Etiological evaluation was further performed in 87 randomly assigned children, and it is identified that familial SS or constitutional delay in growth was the most common cause of SS in the study population (66.67%). Hypothyroidism and growth hormone deficiency were the two most common pathological causes of SS seen in 12 (13.79%) and 8 (9.20%) children, respectively. Malnutrition was the cause of SS in 6 (6.9%) children and cardiac disorders, psychogenic SS, and skeletal dysplasia were other identified causes of SS in the study.

## **International**

Unfortunately, malnutrition remains the most common cause of GF worldwide. Supporting lay and professional efforts to reverse this preventable cause of short stature in besieged communities must be a high priority of all governments and health care professionals.

## **Materials and methods**

The present study was conducted at Department of department of pediatric and endocrinology, Sir Sunderlal Hospital, Institute of Medical Sciences, Banaras Hindu University, Varanasi. The study was questionnaire-based and the details of the lifestyle, habits, and familial history of patients were recorded. All subjects were of Indian ethnicity from eastern Uttar Pradesh and Bihar, the two states of northern India. The study was approved by the Institutional Medical Ethical Committee (**No. Dean/2020/EC/2035**). All the subjects were informed about the study and their consents were taken prior to the start of the study.

## **Inclusion and exclusion criteria**

Recruitment for the study was based on following inclusion criteria: Indian children of both sexes, aged from 4 to 18 years. The exclusion criteria were: children less than 4 years or more than 18 years old, children whose mothers/guardians or caregivers refused medical examination and investigation, receiving drugs known to cause short stature including chronic use of steroids, attention-deficit/hyperactivity disorder medications and anticonvulsants during the time of examination.

The mother/guardian or caregiver of each participating child was interviewed for potential determinants of child nutritional status including socio-demographic characteristics.

Socioeconomic standard (SES) was assessed by updated scaling score of SES developed by Fahmy et al., 2015. Physical assessment and hemoglobin (Hb) level assessment were carried out to all children.

It was measured on a height scale with heels, buttocks, shoulders and occiput against a vertical board and the head is positioned in Frankfurt plane (outer canthi of eyes at horizontal plane with upper border of tragus. The children were drawn upto their full height by upward pressure on the mastoids).

## **Arm Span:**

It was measured with the child standing, arms fully extended parallel to the floor and palms facing forward. With parent's assistance, the distance between middle finger was measured using tape.

## **Upper Segment, Lower Segment:**

The upper segment extends from vertex to pubis, lower segment extends from pubis to heel. First lower segment was measured and upper segment derived by subtracting LS from

standing height.

**Height Age:**

Height age means the age which corresponds to the Ht. in cm along 50th centile curve.

**Bone Age:**

Bone age assessment was done on skiagram of pelvis, knees, left hand wrist, and left elbow following a standard chart based on a study at Radiology department of Institute of Child Health and Hospital for Children, Chennai.

**Growth Formula:**

Height age < bone age < chronological age – Familial SS Bone age = height age < chronological age – CGD

Bone age < height age < chronological age – GH Deficiency Target height:

Growth is strongly related to the genetic potential. A child's midparental height is calculated as follows:

**Girl = (height of mother in inches + height of father in inches)/2 - 2.5 inches**

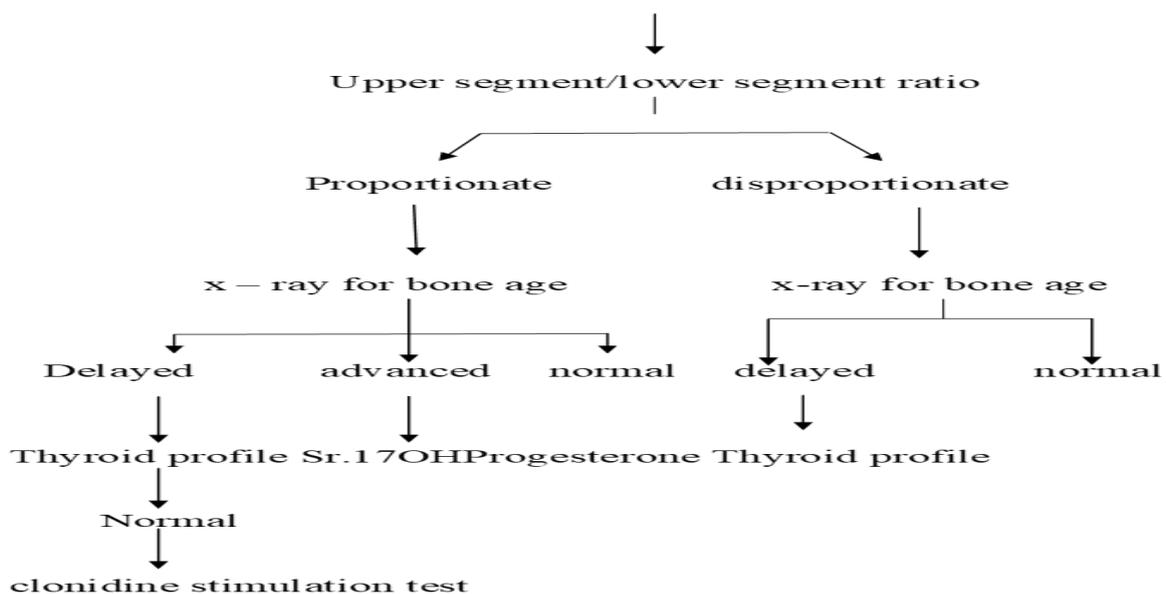
**Boy = (height of mother in inches + height of father in inches)/2 + 2.5inches.**

This value plotted as adult height at 18 years and the spread for target range is 6 cm on either side of the target height. This then becomes target range and if the child's height is within these percentiles, it is considered as normal. A short child who is growing close to his/her target height percentile is likely to have familial short stature. [15].

The child's present height was projected along the percentile curve to get the anticipated adult height and correlated with mid parental height. Growth deceleration during the first 2 years followed by a normal growth velocity, with acceleration late in adolescence, leading to a final height that is close to the target height suggests constitutional delay in growth and development. Auxological data mainly target height, child's current height, height velocity and body proportions are some of the important tools for proper evaluation and management of SS. Judicious use of these techniques will reduce the cost of subsequent investing.

**Anthropometric measurements**

(Standing height, upper segment, lower segment, arm span, S height age and bone age)



### **Clonidine stimulation test:**

In the morning after overnight fasting, 5 ml of venous sample was drawn for basal level of GH estimations. Then the child was given oral clonidine 4 microgram/kg body weight. Blood samples were drawn at 30 mins, 60 mins, and 90 mins in three separate non-heparinised tubes. During the procedure, the subjects were kept in recumbent position and blood pressure were recorded half hourly but no fall in BP were noted.

### **Hormone assay:**

Sera separated aliquated and stored at -20° C in a deep freezer until assayed. GH estimation was done at pediatric APOLLO Hospital using Radio-immunoassay (RIA) kit supplied by BARC. The tests cover a range of 0-40 ng of GH per ml of serum with an intra-assay and inter assay variability ranging between 5-10%. Only basal and post clonidine values were taken into account. Peak value >7 ng/ml is normal. Value <7 ng/ml-partial GHD and <3.5 ng/ml-complete GH deficiency.

TSH assay was done in all subjects using the TSH Immuno Radiometric Assay (IRMA) kit supplied by GB Aura Chennai. The sensitivity is 0.07 microunits/ml. The TSH values taken as normal in ICH, Egmore is 0.2-4 microunits /ml. IGF1 : IGF1 estimation was done to rule out pure GH deficiency, Larons dwarfism. MRI Brain: MRI Brain was done to rule out CNS pathology.

### **Statistical analysis**

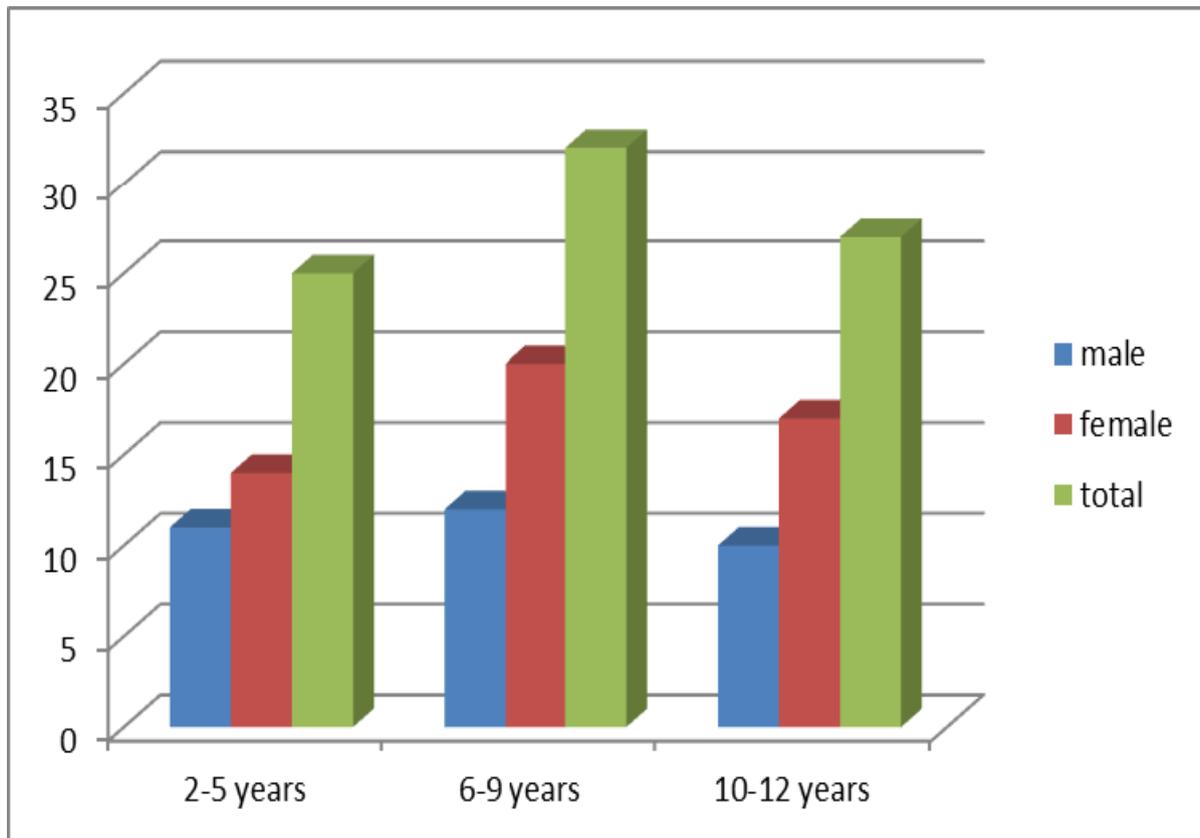
Distributions of data sets obtained in the study were checked for normality using Kolmogorov-Smirnoff test. Means were separated using Tukey's test when data were normally distributed and variances were homogeneous (Bartlett's test for equal variances). The data were presented as Mean±SD along with their Inter Quartile Range (IQR) for continuous variable and frequency with their respective percentages for categorical variables. Chi-Square test was performed to analyze the associations between categorical variables. One-way ANOVA test was performed to for the statistical significance of the difference in mean values of variables among several groups. The P-values less than 0.05 were considered statistically significant. All statistical analyses were performed using the Statistical Package for Social Sciences (SPSS) software, version 23.0.

### **Result**

Totally 84 children with short stature were studied in the age group less than 18years. Demographic characteristics of children with short stature. Total no. of males = 33(39.3%) Total no. of females = 51(60.7%) Female predilection was observed. The study population comprises 33 (39.3%) males and 51 (60.7%) females, there is no significant difference (p = 0.052) in male female distribution among the study population. Out of study population of 84 cases, most commonly observed etiology among endocrine causes was GH deficiency (53%) followed by constitutional growth delay (18%), hypothyroidism (11%), larons dwarfism (9%) & familial short stature (9%). Of the total study population, clustering (38.1%) of short stature noted in 6 to 9 years age group. Comparatively lesser number of cases were affected with SS in 2 to 5 years (29.8%).

**Fig. AGE AND SEX DISTRIBUTION OF SHORT STATURE**

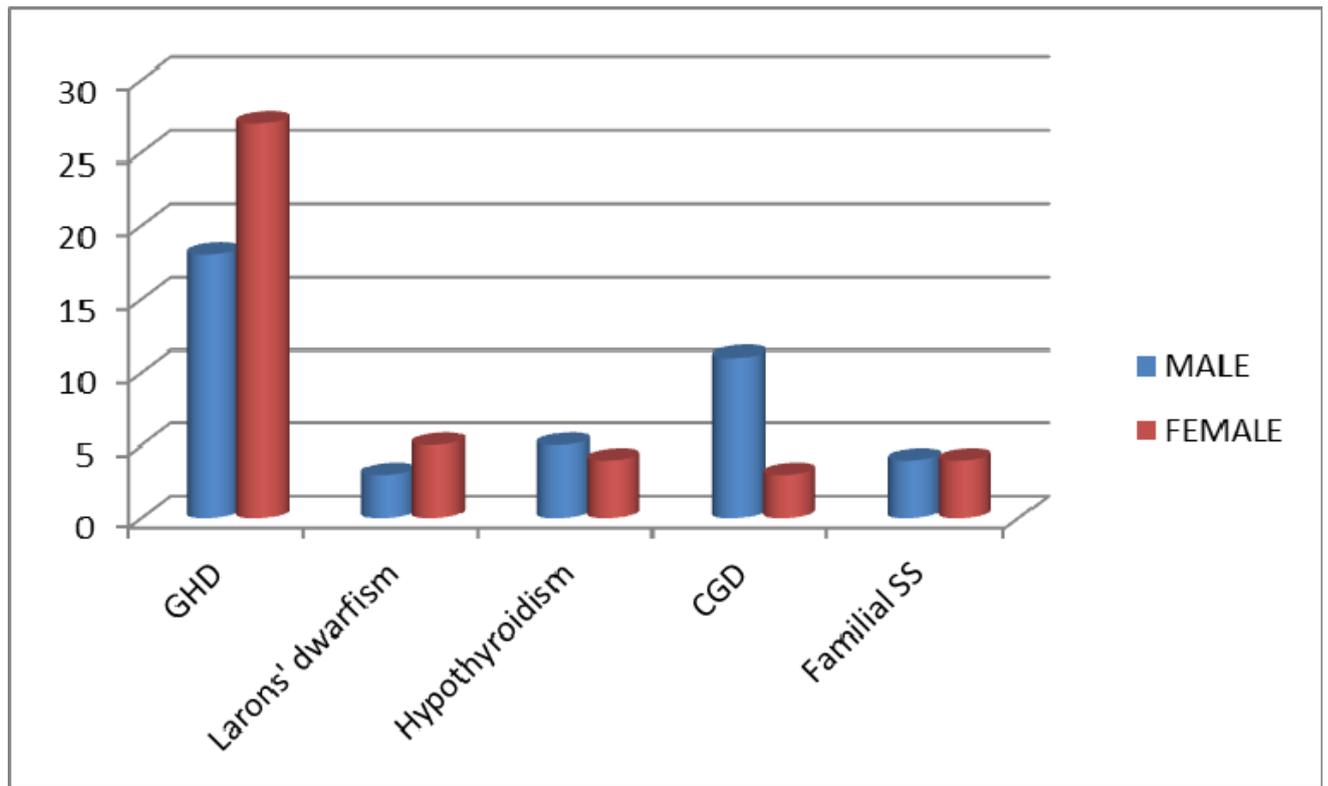
Maximum number of short statures were observed in 6 to 9 years group followed by 10 to 12



years group. Whereas maximum cases of GH deficiency are seen in 6 to 9 years (43.4%) group followed by 10 to 12 years group with 30.2% and finally 2 to 5 years group with 26.4%. The observed difference is statistically not significant ( $p=0.413$ ).

Out of 53 children with GH deficiency, 14 (26.4%) were less than 5 year of age, 23(43.2%) belong to the age group 6 to 9 years, 16 (18.2 %) belongs to the age group 10 to 12 years. Out of 21 children with non-GH deficiency causes, 11 (35.5%) were less than 5 year of age, 9(29%) belong to the age group 6 to 9 years, 11 (35.5 %) belongs to the age group 10 to 12 years. There is no significant difference in the distribution between these groups.

In this study of short stature, we had seen wide variations in age distribution ranging from 4years to 18years. Of total cases of short stature, mean age of presentation was  $7.25 \pm 3.33$ . It was almost similar in both sexes i.e. male- $7.24 \pm 3.26$  and in female SS children  $7.25 \pm 3.41$ .



In GH deficiency, females were dominantly affected than males. Similar finding was observed in Larons dwarfism. Males were predominantly affected with hypothyroidism, constitutional growth delay. Both male and female were equally affected in familial short stature.

**Table. Perinatal features & clinical symptom among children presented with short stature**

SYMPTOMS	N	Percentage
Increased ICP	1	1.9%
APH	5	9.4%
Maternal Fever	9	16.9%
Normal delivery	42	79.2%
Pre Term	15	28.3%
Birth asphyxia	33	39.3%

<b>Breech</b>	58	69%
<b>NNH</b>	15	17.9%
<b>Neonatal hypoglycaemia</b>	12	14.2%
<b>Consanguinity</b>	49	58.3%
<b>Family history</b>	8	9.5%
<b>Parental history of delayed puberty</b>	14	16.7%
<b>Microphallus</b>	8	9.5%
<b>Hyper pigmented skin</b>	10	11.9%
<b>Dysmorphic facies</b>	41	48.8%
<b>Hypothyroidism</b>	12	14.2%
<b>MPHD</b>	10	11.9%

The study population was analysed based on their clinical symptoms and presentation. We observed raised ICP in 1(1.9%), antepartum hemorrhage in 9(9.4%), maternal fever in 9 (16.9%)cases, normal delivery in 42 (79.2%), preterm delivery in 15 (28.3%), birth asphyxia in 33 (39.3%), breech presentation in 58 (69%), NNH in 15 (17.9%), neonatal hypoglycemia in 12 (14.2%), consanguinity in 49 (58.3%), family history in 8 (9.5%),parental history of delayed puberty in 14 (16.7%), microphallus in 8 (9.5%), hyperpigmented skin in 10 (11.9%), dysmorphic facies in 41 (48.8%), hypothyroidism in 12 (14.2%) and MPHD in 10 cases(11.9%).

## **DISCUSSION**

This descriptive study was conducted at Institute of Child Health to find out the demographic characteristics, clinical and etiological profile of short stature in children between 4 to 18 years of age attending endocrine OPD. 84 children between 4 to 18 years of age getting admitted at Institute of medical science Banaras Hindu University who met the inclusion and exclusion criteria were recruited.

2500 children admitted in hospitals. Khadgawat et al<sup>45</sup> have reported 7% prevalence among 280 normal school children. Another observation made by Colaco revealed a prevalence of 10% short stature in children utilizing outpatient services. However, Garg<sup>46</sup> in his study of short stature in Indian Children had reported 13.8% prevalence of short stature, lesser than that of our study. In this study, the prevalence of short stature was 15.6%.

#### **Conflict of interest**

Authors have declared that and research was conducted in and the absence of any commercial or financial relationships without any conflict of interest.

#### **Acknowledgment**

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#### **Author Contributions**

R.S., R., A. contributed to the conception, design, and writing of the study protocol and the design of search strategies; N.K.S., A K.Y., M.K. located and obtained reports, helped to select and assess cases, conducted the data analysis, and drafted and approved the final paper. All authors contributed to the conception, design, and writing of the study protocol, conducted data analysis and revised and approved the final paper.

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