e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(10); 337-345

Original Research Article

Clinical Profile and Impact of Intervention in Children with Stunting Attending a Tertiary Care Hospital in Kerala-A Prospective Study

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Received: 13-08-2025 / Revised: 12-09-2025 / Accepted: 13-10-2025

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Conflict of interest: Nil

Abstract:

Background: Stunting is a major public health concern associated with adverse health and developmental outcomes. Hospital-based data on the etiological profile and response to intervention in stunted children in Kerala remain limited.

Objectives: (1) To describe the clinical profile of children with stunting attending the paediatric outpatient department (OPD) of a tertiary care hospital in Kerala. (2) To estimate the impact of interventions, based on etiology, in children with treatable causes of stunting at the end of a two-year follow-up.

Methods: This prospective observational study was conducted at paediatric OPD of Government Medical College, Ernakulam. Children aged 2–15 years with stunting were enrolled consecutively over six months. Children with prior diagnosis or treatment for growth failure were excluded. Relevant data was collected using structured proforma. Anthropometry, bone age, laboratory investigations, and relevant endocrine/genetic tests were performed. Etiology-specific interventions were initiated, and children were followed up for two years.

Results: Of 150 children enrolled with stunting, 108 completed follow up. The mean age was 6.2±3.1 years, with a male predominance (60.2%). Majority (57.4%) were less than 7 years age. Severe stunting was present in 21.3% and wasting in 67.6%. Familial short stature (FSS) (36.1%) and constitutional delay of growth and puberty (CDGP) (34.3%) were the most common diagnoses. Endocrine causes identified were hypothyroidism (2.8%), growth hormone (GH) deficiency (5.6%). GH stimulation test was done for 17, of which 9 were abnormal. Only 4 initiated GH treatment due to cost constraints. Severe bone age delay was significantly associated with poor height velocity (p=0.04).

Conclusions: FSS and CDGP remain predominant causes of stunting, but endocrine, genetic, and nutritional etiologies are also important. Early identification, growth monitoring, and low-cost interventions can improve outcomes, while access to advanced diagnostics and growth hormone therapy remains a challenge.

Keywords: Stunting, Nutrition, Height Velocity, Growth Hormone.

Introduction

Stunting reflects chronic undernutrition and is associated with impaired cognitive and physical development. Although Kerala has better health indicators compared to other Indian states, it has prevalence of stunting of 23.4% among under-five children.[1] Hospital-based studies describing the etiological spectrum of stunting in Kerala are scarce.

Short stature may result from familial or constitutional factors, nutritional deficiencies, chronic illnesses, endocrine disorders, or genetic syndromes. [2,3] Distinguishing benign variants from pathological causes requires careful evaluation of growth patterns, bone age, and targeted investigations.

This study aimed to describe the clinical and etiological profile of children presenting with stunting in a tertiary care hospital in Kerala, India and to assess the impact of appropriate interventions over a two-year follow-up.

Materials & Methods

Study Design and Setting: Prospective observational study conducted at the paediatric OPD, Government Medical College, Ernakulam, Kerala, India for a period of Six-month recruitment

(2022), with two-year follow-up. The current study was approved by Institutional Ethics Committee. Informed consent from parents for age <12 years and written informed consent for age >12 years.

Inclusion Criteria: Children aged 2–15 years with stunting.

Exclusion Criteria: Children previously diagnosed or under treatment for growth failure or chronic illness and those with global developmental delay were excluded from the study. Children who were lost to follow up during study period were also excluded.

Definitions

- Stunting: Height for age <-2 standard deviation (SD) and Severe stunting: < –3 SD according to World Health Organization (WHO) Z- score charts up to 5 years age and Indian Academy of Paediatricians (IAP) charts for >5 years age.[4] Serial plotting data and height velocity of each participant during follow up, every 6 month for two years, was saved in a mobile application (IAP growth chart plotter).
- Age group-Early childhood: 2–7 years; middle childhood: 7–11 years; adolescence: ≥12 years.
- Socioeconomic status (SES) was classified based on Modified Kuppuswamy scale.[5]
- Stormy perinatal period- morbidity requiring admission in Neonatal Intensive Care Unit (NICU) for > 2 weeks after birth.
- Low birth weight (LBW)-Birth weight less than 2.5 kg.
- Small for gestational age (SGA)-Birth weight/length smaller than expected for the gestational age and sex.
- Midparental height (MPH) was calculated as: Average of father's height and mother's height +6.5 cm for boys and -6.5 cm for girls. MPH is plotted at 18 years age in WHO-IAP centile charts. Target height range is plotted as MPH +/- 8 cm
- Sexual Maturity Rating (SMR) was done for all children >7 years age based on Tanner staging.
- Bone age (BA): based on Greulich–Pyle atlas[6] – classified as normal (BA = chronological age), mild delay (BA delay<2 years) or severe (BA delay >2 yrs).
- Height velocity (HV)-Rate of increase in height over time (expressed as cm/year). Interpreted based on normal HV for age.[7]
- FSS- Those with MPH<3rd centile in WHO-IAP centile charts, and child's target height within target range with normal height velocity.
- CDGP-Those with history of CDGP in either parent, prepubertal on Sexual maturity rating (SMR) and adequate height velocity on follow up.
- Idiopathic short stature (ISS)- height <-2.25 SD from the mean (or < 1.2 percentile) for age and gender with all investigations, including Growth hormone stimulation test (GHST) normal, and poor height velocity.
- Multiple pituitary hormone deficiency (MPHD)-Deficiency in atleast 2 pituitary hormones.

Sample Size and Sampling: Based on NFHS-5 prevalence of stunting (23.4%), 1200 children more than 2 years age, who came to OPD consecutively during first 6 months of study were screened for stunting. 150 children with stunting were enrolled in the study, but 42 were lost to follow up during study period and were excluded. 108 children were evaluated during the study period (N=108).

Data Collection: Structured proforma captured sociodemographic, perinatal, nutritional, and family history. History and clinical examination of signs of chronic illness and micronutrient deficiency, anthropometry, and laboratory evaluation were performed.

Anthropometry included height in cm (using stadiometer), weight in kg, Body mass index (BMI) in kg/m2, Upper segment: Lower segment ratio. References for normal cutoffs were based on WHO-IAP growth charts and guidelines.[8]

Screening investigations included Level 1(for all children) to identify secondary/modifiable causes of stunting -Bone age, Complete blood count, serum electrolytes, serum calcium, serum phosphorous, serum Alkaline phosphatase (ALP), blood glucose, renal/liver function tests (RFT/LFT), thyroid profile, chest x-ray, urine analysis, including urine pH, stool examination (for parasites, occult blood, steatorrhea). These were done free of cost in the hospital.

Abnormal results were defined as follows: haemoglobin (Hb) < –2 SD for age and sex (anaemia), serum 25-hydroxy vitamin D <12 ng/mL (deficiency) and <20 ng/mL (insufficiency), Thyroid- stimulating hormone (TSH) above the upper limit of reference range for age (primary hypothyroidism) , free Thyroxine(FT4) below the lower limit of normal with normal/low TSH(central hypothyroidism).The cut-off values were based on IAP consensus guidelines.[9]

All children with normal Level 1 investigations and normal BA/mild BA delay were kept under follow up for height velocity.

Level 2- based on affordability, for those with severe bone age delay or poor height velocity - Karyotype in girls, celiac serology and venous blood gas (VBG) in those with associated malnutrition.

Level 3-targeted tests-GHST for those with severe BA delay/poor height velocity on follow up. GHST

was done after clonidine stimulation, with prior sex steroid (oestradiol valerate) priming when required (done for pre pubertal girls >10 years age and boys >11 years age. We used peak GH values below 7 µg/L as indicative of GH deficiency.[10] Genetic analysis by whole/clinical exome sequencing was done based on clinical suspicion.

Intervention: Growth velocity was monitored at 6-month intervals for next 2 years. Children with modifiable causes received appropriate treatment (diet modification, deworming, micronutrient supplementation, thyroxine, treatment for chronic illness, growth hormone).

Statistical Analysis: Data were analysed using jamovi software (Version 2.3, The Jamovi project, Sydney, Australia).11 Frequencies, means, and proportions were calculated. Associations were tested using chi-square and t-tests. $p<0.05$ was considered significant.

Results

Study population: Of 150 children identified with stunting, 42 were lost to follow-up; 108 were evaluated till end of study period (N=108). 84.3% came to OPD for complaints other than short stature. Mean age was 6.2±3.1 years (range 2–15), with median of 5 years. Male:female ratio of study population was 65:43. Majority (55.6%) were from upper low SES. Demographic profile detailed in Table 1.

Table 1: Demographic profile of the study population (N = 108)

Age		
Variable	**N (%)**	**Mean ± SD / Median (Range)**
Age group (years)	108	6.2 ± 3.1 / 5 (2–15)
Early childhood (2–7)	62 (57.4)	
Middle childhood (7–11)	40 (37.0)	
Adolescents (>_12)	6 (5.6)	
Sex		
Male	65 (60.2)	
Female	43 (39.8)	
Socioeconomic status		
Low	12 (11.1)	
Upper low	60 (55.6)	
Lower middle	29 (26.9)	
Upper middle	7 (6.5)	

Table 2: Clinical profile of the study population (N = 108)

Variable	N (%)
Breastfeeding pattern	
Exclusive BF ≥6 months & continued till 2 years	27 (25.0)
Exclusive BF for 6 months but discontinued before 2 years	73 (67.6)
BF <6 months	8 (7.4)
Anthropometry	
Stunting with wasting	34 (31.5)
Stunting with wasting	73 (67.6)
Stunting with obesity	1 (0.9)
Dysmorphism	
Present	9 (8.3)
Absent	99 (91.7)
Body proportions	
Proportionate short stature	100 (92.6)
Disproportionate short stature	8 (7.4)
Micronutrient deficiency signs	
Present	67 (62.0)
Absent	41 (38.0)
Severity of short stature	
> –3 SD	23 (21.3)
–2 to –3 SD	85 (78.7)
Height velocity catch-up	
Good	80(74.1)
Inadequate	28 (25.9)

- Preterm -13(12%), Term-95(88%)-Term LBW-19(20%). All babies born term with LBW were considered SGA (20%). Gestation age for preterms could not be uniformly assessed from records.
- Stormy perinatal history: 11.1%.
- Dysmorphic features: In 9 children (8.3%) →Whole exome sequencing (WES) done to diagnose Noonan syndrome (2), Kabuki syndrome (1), Pseudohypoparathyroidism (1), skeletal dysplasia (2), minor chromosomal abnormality (2). Karyotyping was done for 22 girls - abnormal for 1 child (45 XO suggestive of Turner syndrome).
- Prepubertal- 85.8%.1 child with hypothyroidism had precocious puberty (Van Wyk-Grumbach syndrome).
- 17(15.7%) had extreme stressors in family because of parental loss, parental conflicts or lack of care in family and this was associated with malnutrition /micronutrient deficiency.
- There was significant association between breastfeeding and severity of stunting (p-value=0.027) (Figure 1)

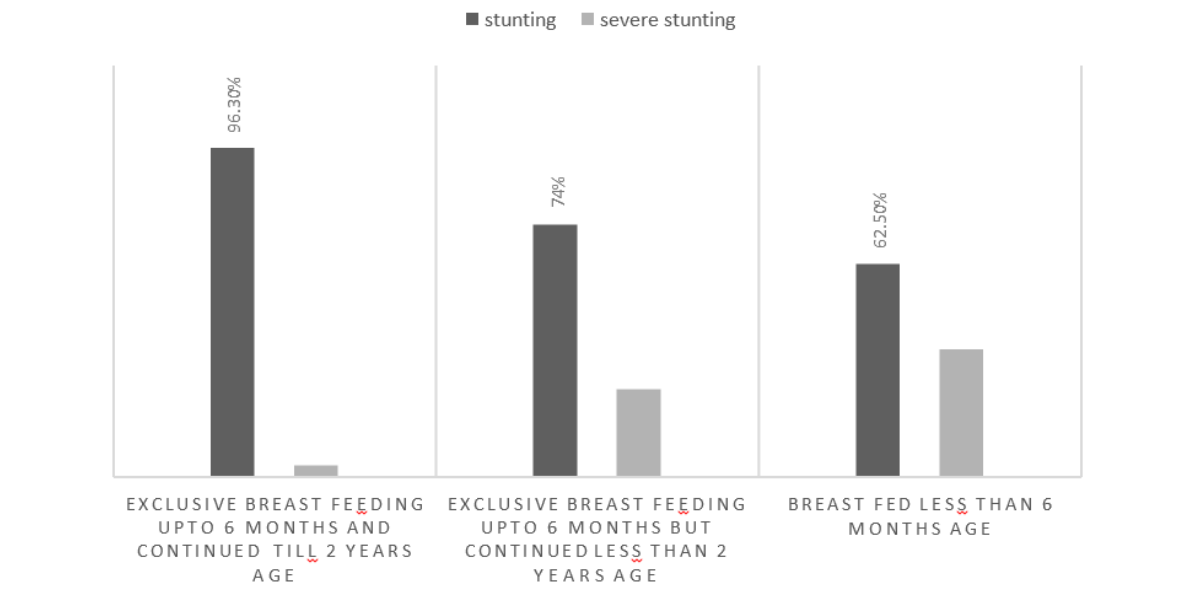

Figure 1: Association between breastfeeding practices and severity of short stature

$\chi^2 = 7.21$; df = 2; p = 0.027

Table 3: Investigations

Investigation	N (%)
General screening	
Normal	70 (64.8)
Anaemia	16 (14.8)
Investigation	**N (%)**
Abnormal thyroid function test (TFT)	8 (7.4)
Low vitamin D3	8 (7.4)
Abnormal calcium/phosphorus/ALP	4 (3.7)
Abnormal renal function test (RFT)	1 (0.9)
Abnormal venous blood gas (VBG)	1 (0.9)
Growth hormone stimulation test (GHST)	
Not required	72 (66.7)
Done – normal	8 (7.4)
Done – abnormal	9 (8.3)
Not done (financial constraints)	19 (17.6)
Karyotyping	
Not done	86 (79.6)
Normal	21 (19.4)
Abnormal	1 (0.9)
Celiac work-up	
Not required	82(75.9)
Done-Normal	15 (13.9)
Not done (financial constraints)	11(10.2)

- Of those with low Vitamin D (7.4%)- 1 had deficiency, others insufficiency. VBG showed normal anion gap metabolic acidosis in 1 child, later confirmed to have Renal tubular acidosis (RTA) after genetic testing.1 child with low serum calcium and high serum phosphorous with abnormal thyroid profile was later diagnosed to have Pseudohypoparathyroidism (PHP) and 1 to have chronic kidney disease (CKD).
- Celiac work up was done for 15 children with associated wasting, and inadequate weight catch up during follow up- all tested negative.
- Magnetic resonance imaging (MRI) brain done in those with GH deficiency: pituitary hypoplasia (2), pituitary stalk interruption syndrome (2), normal (2).
- Of 6 children referred with low IGF-1 values- 3 had normal GHST.
- Mild BA delay (<2 years): 46.3%, Severe BA delay:13.9%. Severe BA delay was significantly associated with inadequate height velocity (p=0.04) (figure 2)

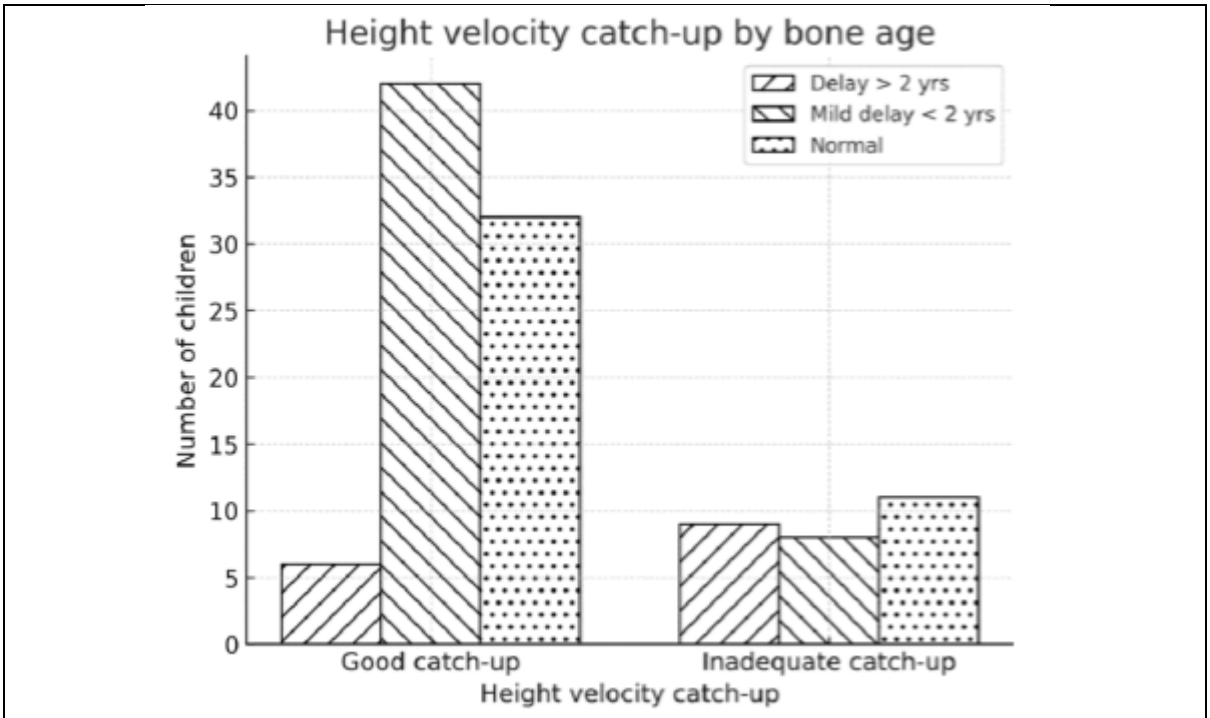

Figure 2: Height velocity catch-up in children stratified by bone age delay

χ^2=18.6 df=2 p-value =0.04

Treatment and follow-up

- Nutritional/dietary counselling and deworming: 67.6 %.
- Micronutrient supplementation and emphasis on Weekly Iron-folic acid supplementation (WIFS): 67.6 %
- Steps were taken to give psychology counselling for those with psychosocial deprivation risk factors.
- Iron therapy for anaemia: 16(14.8%).
- Vitamin D supplementation was started for vitamin D deficiency and insufficiency.
- Thyroxine for hypothyroidism: 8 (7.4%).
- Apart from GH deficiency diagnosed by GHST, GH treatment was also advised in those with Turner syndrome, Noonan syndrome and SGA with poor catch up. Only 4 (3 with GH deficiency and 1 with Turner syndrome) could afford to start the same.
- Appropriate treatment was started for RTA, PHP and CKD.

Table 4: Final diagnoses

Final diagnosis	N (%)
Familial short stature	39 (36.1)
Constitutional delay of puberty and growth (CDPG)	37 (34.3)
Small for gestational age (SGA) with poor catch-up	5 (4.6)
Multiple pituitary hormone deficiency (MPHD)	4 (3.7)
Other syndromes	3 (2.8)
Idiopathic short stature	3 (2.8)
Hypothyroidism	3 (2.8)
Under follow-up (etiology not yet established)	3 (2.8)
Noonan syndrome	2 (1.9)
Skeletal dysplasia	2 (1.9)
Growth hormone deficiency (isolated)	2 (1.9)
Turner syndrome	1 (0.9)
Fanconi anaemia	1 (0.9)
Renal tubular acidosis (RTA)	1 (0.9)
Pseudohypoparathyroidism (PHP)	1 (0.9)
Chronic kidney disease (CKD)	1 (0.9)

Child with Turner syndrome had associated hypothyroidism also. 4 children had primary hypothyroidism and 3 central hypothyroidisms as part of MPHD. 3 children below 7 years age with improving but still inadequate height velocity, who could not afford to do Level 2 /3 investigations were kept under follow up. Those with ISS were also kept under follow up, for probable GH requirement.

Associations: Exclusive breastfeeding till 6 months after birth and continuation of breastfeeding till 2 years of life had significant association with severity of stunting(p=0.027). Severe BA delay was significantly associated with inadequate height velocity (p=0.04)

Discussion

Short stature is very prevalent in India but there are very few studies on it. Busy OPDs, especially with limited staff, defer paediatricians from recording height of children coming to OPD for various ailments, as prescription of drug doses requires weight recording only. Though birth weight is always recorded for babies, birth length often goes unrecorded. Early screening of short stature can go a long way in early identification of causes and early intervention can contribute to regaining the height potential of children, who otherwise would remain stunted. In our study, majority (84.3%) of children diagnosed to have stunting were brought for unrelated complaints, which supports the fact that stunting as a major public health problem is still under recognized.[12,13] This prospective study provides valuable insights into the clinical profile, etiological spectrum, and impact of interventions in children presenting with stunting in a tertiary care setting in Kerala and underscores the need for routine anthropometric screening in all paediatric OPDs.

The mean age of presentation was 6.2 years. Similar to previous studies from India and other resource-limited settings, boys were more frequently affected than girls, suggesting either a biological susceptibility or gender-related differences in health-seeking behavior.[13]

Lack of exclusive breast feeding for 6 months and not continuing breast feeds till 2 years age was significantly associated with severe stunting (p value=0.027). 25% babies with stormy post-natal period had severe stunting, but was not significant in our study (p-value=0.7).Many studies report how in utero factors and post-natal factors, including adequate breast feeding contribute to stunting till 2 years of age, after which proper diet, environment, physical and mental well-being, prompt treatment of any acute or chronic illness can help a child to catch up in height velocity.[12-14]

67.6 % had associated wasting and 62% had clinical signs of micronutrient deficiencies. They were advised dietary modification and given deworming with multivitamin supplementation. Anganwadi workers in the child's locality were alerted to provide healthy diet for children <5 years age with associated wasting. They have been found to have a significant role in preventing malnutrition-related stunting. [12-14] Anaemia was present only in 16(14.8%), may be due to the WIFS carried out through schools and anganwadis in Kerala. This is consistent with NFHS-5 data, which showed that despite improvements in health indicators, Kerala continues to report a prevalence of stunting around 23%. [1,13,15] Our findings emphasize that malnutrition and micronutrient deficiencies remain major contributors even in relatively well-resourced states.

Age group classification was done keeping in mind the need for examining for pubertal onset after 7

years age. Optimum mean age of pubertal onset in Indian children is between 10-11 years age, and stunting is associated with delayed puberty. In our study, majority (84.85%) were prepubertal. Various studies show how prepubertal height deficits may be carried into adult height.[16]

BA is an important tool to predict later height velocity. Importantly, our study showed significant association between normal BA/mild BA delay and adequate height velocity on follow up (Fig 1). This supports the utility of bone age assessment in differentiating benign variants from pathological causes.[17] We used Greulich-pyle charts to assess BA, which is proven reliable though subjective method of assessment.[18]

Affordability for tests like celiac serology, IGF-1were a limiting factor in our study. While Turner syndrome can present with short stature only, and all girls with stunting and normal screening investigations should be evaluated for Turner syndrome according to guidelines, we did karyotype for girls with clinical suspicion and with inadequate height velocity.

Although GHST was indicated in 36(33%) of children, affordability constraints meant that only 17(15.7%) underwent testing, of whom 9(53%) had confirmed deficiency of GH. This reflects the practical challenges in applying guideline-based evaluation in low- and middle-income settings.

Genetic and endocrine etiologies were also identified. Dysmorphic features were noted in 9(8.3%) children, with confirmed diagnoses of Turner, Noonan, Kabuki, and skeletal dysplasias.

Hypothyroidism (3.7%), MPHD (3.7%), and isolated GH deficiency (1.9%) were less common but clinically significant. As per Growth Hormone Research Society Consensus Guidelines, MRI with 2 mm slices should be done in all with confirmed isolated GH deficiency or MPHD, to note the height/volume of anterior pituitary, position of posterior pituitary and anatomy of the stalk.[19] MRI was done free of cost at the hospital for all children with GH deficiency, of which 4 were abnormal. Several guidelines support stepwise evaluation in stunting, since targeted investigations are limited by cost-constraints. [11,19,20]

The final diagnostic distribution showed FSS (36.1%) and CDGP (34.3%) as the most common causes. There are prior hospital-based studies, which consistently report physiological and nutritional causes account for more than half of cases of short stature. [21,22] Rajput et al. (2021) and Penugonda et al. (2020) observed a high proportion of endocrine causes among children with short stature. [23,24]

Although malnutrition contributed significantly (67.6%) to the presentation of stunting in our study, it was cross-present with other etiologies. As weight catch-up was achieved following dietary modification, malnutrition was not considered a distinct diagnosis in this cohort.

Besides GH deficiency, some FDA-approved indications for GH are Turner syndrome, Noonan syndrome, CKD, ISS, SGA with poor catch up. In our study,17(15.7%) children were advised GH therapy, but only 4 could afford treatment. This reflects the prohibitive costs of therapy and indicates the need for policy measures to improve access to growth hormone in eligible children.[25] On the other hand, simple interventions such as dietary counselling/support, regular deworming, micronutrient supplementation, treatment for anaemia and other chronic illness and thyroid hormone replacement showed measurable benefits and remain critical, low-cost strategies. Many studies have shown significant improvement in height velocity after starting GH in indicated causes. [26,27]

Our study highlights that early recognition, systematic evaluation, and etiology-specific management can improve outcomes in children with stunting. The findings reinforce the need for strengthening growth monitoring at the community level, improving parental awareness, and ensuring better access to diagnostic and therapeutic facilities.[28]

Strengths and Limitations: A key strength of this study is the comprehensive, prospective evaluation of children with stunting, integrating clinical, nutritional, endocrine, and genetic assessments. The longitudinal follow-up over two years, along with timely identification and treatment of modifiable risk factors/illness, provides insight into the impact of interventions. However, the study is limited by attrition (42 children lost to follow-up), small sample size, and affordability barriers that restricted advanced investigations and growth hormone therapy in many cases. Being a single-centre hospital-based study, the findings may not be fully generalizable to the community.

Conclusions

Familial short stature and constitutional delay are common causes of stunting in Kerala. Nutritional, endocrine, and genetic causes are also significant. Early recognition and simple interventions such as dietary modification and micronutrient supplementation are effective. Documentation of birth length and later height at each anganwadi/hospital/immunization visit at regular intervals is imperative in early diagnosis of stunting. However, cost-related barriers limit advanced diagnostics and growth hormone therapy, underscoring the need for improved health system support.

Contributions of Authors:

Bifina Beegum M[1]- 1st author and corresponding author- identified children with stunting as per inclusion criteria, prepared structured proforma, collected details and did clinical examination, including anthropometry, interpreted laboratory investigations, diet advice and counselling given for those with malnutrition, treatment started for any illness identified after screening, kept under follow up for height velocity and decided on GH need.

Peter P. Vazhayil[2] - 2nd author-guided throughout the study process regarding the stepwise evaluation as needed and developer of mobile application, IAP growth chart plotter, in which details of serial height monitoring over two-year study period was saved and plotted on growth charts.

Aswathi P.R.[3]- 3rd author-examined children at review for height velocity, interpreted repeat investigations as needed, and documented the details of height velocity and targeted investigations.

Acknowledgements: The authors sincerely thank Professor Dr Shiji K Jacob, Head of the Department of Paediatrics, Government Medical College, Ernakulam, for encouragement and institutional support during the conduct of this study.

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