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Calcium and Vitamin D Serum Level of Stunting and Severe Stunting Children Aged 12-23 Months in the Pauh Health Center, Padang City

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ABSTRACT

Stunting remains a significant public health challenge, particularly in developing countries. Vitamin D and calcium play crucial roles in linear growth and bone mineralization during early childhood. This study aimed to analyze the levels of serum vitamin D and calcium in stunted and severely stunted children aged 12–23 months in the working area of Pauh Health Center, Padang City, West Sumatra. This cross-sectional analytical study was conducted from February to May 2025, involving 43 children aged 12–23 months (33 stunted and 10 severely stunted). Data were analyzed using the chi-square test and independent-samples t-test. Most children exhibited hypovitaminosis D (62.7%), with 9.3% deficiency and 53.4% insufficiency. Serum calcium levels were predominantly normal (85.1%). No significant differences were found in vitamin D levels ($p > 0.05$) or calcium levels ($p > 0.05$) between stunted and severely stunted children. The mean vitamin D level was 12.14 ± 1.97 ng/mL in stunted children and 12.16 ± 1.63 ng/mL in severely stunted children. The mean serum calcium levels were 9.14 ± 1.97 mg/dL in stunted children and 9.16 ± 1.63 mg/dL in severely stunted children, respectively. Despite the high prevalence of vitamin D insufficiency, there were no significant differences in serum vitamin D and calcium levels between stunted and severely stunted children aged 12–23 months. The body's homeostatic mechanisms maintain normal serum calcium levels despite low vitamin D status, potentially at the expense of bone density. Further research is needed to identify other contributing factors to stunting severity.

KEYWORDS stunting, vitamin D, serum calcium, children under two years, malnutrition

INTRODUCTION

Stunting remains a serious unresolved health problem, with prevalence rates still quite high. It represents a global burden, as 80% of cases occur in developing countries, where Southeast Asia has the second-highest prevalence, with 15.6 million stunted children (29.4%). Weight faltering marks the onset of stunting and can appear as early as the first six months, even among breastfed children (20%), followed by the complementary feeding period (50%) and children aged >3 years (10%). Aguayo et al. reported that stunting prevalence in India is higher among children aged 12-23 months than those aged 0-11 months (34.8% vs. 11.7%) (Aguayo et al. 2016). Similar findings emerged from Torlesse et al., who identified the highest stunting prevalence (37.7%) in children aged 12-23 months (Torlesse et al. 2016). The 2018 *Survei Kesehatan Dasar* (*Riskesdas*) National Basic Health Research in Indonesia reported stunting and severe stunting prevalence rates of 17.1% and 12.8%, respectively, among children under two years (*baduta*) (Riskesdas 2018).

Stunting occurring early in life, during periods of rapid growth, causes detrimental short- and long-term effects that are irreversible. These impacts include hindered linear growth, reduced cognitive ability, increased mortality, and degenerative diseases (Black et al. 2013; WHO 2020). Prevention involves two main strategies: first, providing complete nutrition—both macro- and micronutrients—in sufficient amounts; and second, conducting routine anthropometric measurements to detect early weight faltering and address it promptly.

Nutritional interventions supply calories, animal protein, and key micronutrients supporting linear growth, such as iron, zinc, calcium, and vitamin D, to facilitate catch-up growth. The primary source of vitamin D (80-90%) is ultraviolet B (UVB) sunlight exposure, with the remainder from foods rich in vitamin D, such as oily fish, mushrooms, and fortified milk (Beal et al. 2018; Sari et al. 2016).

Animal protein provides insulin-like growth factor 1 (IGF-1) in sufficient amounts to support linear growth (Semba et al. 2016). One function of IGF-1 is to regulate the enzyme 1α-hydroxylase, which converts inactive vitamin D (25(OH)D) to its active form ($1,25(OH)_2D$/calcitriol). Calcitriol enhances calcium absorption in the digestive tract, ensuring adequate calcium availability for bone mineralization. Additionally, vitamin D supports the immune system to prevent recurrent infections, a key stunting contributor (Herber et al. 2020). Milk and eggs are prime sources of animal protein, offering complete essential amino acids (EAAs) along with vitamin D and calcium (Semba et al. 2016).

Vitamin D deficiency poses a major public health issue, particularly among at-risk groups like toddlers. It is prevalent even in sunny equatorial countries like Indonesia. Holick and Chen (2008) and Kang et al. (2018) linked high deficiency rates to children's habits of wearing closed clothing, limited outdoor activity, and sunblock use, reducing UVB exposure. Adequate vitamin D levels promote intestinal calcium absorption; low 25(OH)D impairs formation of $1,25(OH)_2D$ (active vitamin D), hindering bone growth (Fleet 2017). Alshammari et al. (2017) found that stunted children had lower intakes of calories, zinc, vitamin A, vitamin D, and calcium than normal children. Genetic factors, including the vitamin D receptor (*VDR*), contribute 43-80% to vitamin D levels, explaining deficiencies despite sufficient sunlight and diet (Schöttker 2012; Angeline 2018).

The *VDR* gene is expressed across human cells and tissues, especially in osteoblasts, chondrocytes, and immune cells (Shea et al. 2009; Kongsbak et al. 2013). The vitamin D receptor binds calcitriol ($1,25(OH)_2D$) to induce transcription of genes encoding calcium transport channels. Elevated calcitriol stimulates intestinal calcium absorption via genomic mechanisms, transporting it into enterocytes and the nucleus to interact with nuclear *VDR*. Calcitriol regulates genes for proteins involved in calcium absorption and transport, such as calcium channel transporters, Ca^{2+}-ATPase, and claudins (Gropper 2010). Two single nucleotide polymorphisms (SNPs) in the *VDR* promoter region—rs11568820 (*Cdx2*) and rs4516035 (*GATA*)—influence height and vitamin D response (D'Alesio et al. 2005; Arai et al. 2001).

VDR polymorphisms affect vitamin D function in three ways: first, by altering *VDR* gene expression efficiency and receptor availability; second, by changing receptor affinity for vitamin D; and third, by modifying cellular responses like calcium metabolism, immune function, or inflammation. They can also disrupt corepressor dissociation, impairing transcriptional inhibition (Uitterlinden et al. 2004; Bikle 2017).

Micronutrient deficiencies (zinc, iron, copper, calcium, vitamin D) directly impair epiphyseal growth plate function, affecting chondrocyte differentiation and proliferation (Inzaghi et al. 2022). Vitamin D facilitates intestinal calcium absorption to maintain blood levels for normal bone mineralization (WHO 2020). Active vitamin D ($1,25(OH)_2D$), with parathyroid hormone and calcitonin, regulates calcium/phosphorus homeostasis, renal calcium reabsorption, and skeletal calcium mobilization (Holick et al. 2011). Hossein-Nezhad and Holick (2013) emphasized vitamin D's role in calcium/phosphorus metabolism for growth and mineralization.

West Sumatra is among 20 Indonesian provinces with relatively high stunting prevalence. The 2018 *Riskesdas* reported a national rate of 30.8% (*Riskesdas* 2018). The 2022 Indonesian Nutrition Status Survey (*SSGI*) showed improvement: 24.1% nationally, 23.3% in West

Sumatra, and 19.5% in Padang City (Ministry of Health RI 2021). These data highlight the persistent malnutrition burden and need for targeted interventions.

No official Ministry of Health data on vitamin D deficiency prevalence among Indonesian children under five appears in the 2022 Indonesian Health Profile or *SSGI*. However, a Sijunjung Regency study found 50% of stunted children under five had vitamin D intake deficiency, versus 13.64% of non-stunted children (Marsellinda and Ferilda 2023). Using 2018 *Riskesdas* data, 8.2% of children under five had serum calcium deficiency (<9.0 mg/dL), but no specific data exist for West Sumatra or Padang City (*Riskesdas* 2018).

Despite vitamin D and calcium's established roles in linear growth and mineralization, their links to stunting severity remain underexplored in Indonesia, especially Padang City. Prior studies emphasized dietary intake over serum levels, creating a physiological status gap. *VDR* polymorphisms' interactions with nutrition also merit study. With no prior research in this equatorial, high-sun-exposure area, this study fills a key literature gap. Understanding stunted children's biochemical profiles is vital for targeted interventions and policies. Thus, this study determines and compares serum vitamin D and calcium levels in stunted and severely stunted children aged 12-23 months at the Pauh Health Center, Padang City, to inform evidence-based strategies against childhood malnutrition.

METHOD

This study employed an analytical design with a cross-sectional approach and was conducted in the working area of the Pauh Health Center, Padang City, from February to May 2025. Research permission and ethical clearance were obtained from the Health and Medical Research Ethics Committee of Andalas University (letter number KE/FK/953/EC/2015). Subjects were stunted children aged 12-23 months residing in the study area who met inclusion criteria: parental consent (via signed informed consent) and stunting status, defined as height-for-age below -2SD according to the WHO 2006 growth chart, with weight age (WA) less than length age (LA) and chronological age (WA < LA < chronological age). Exclusion criteria included severe diseases (parathyroid, kidney, or cancer), congenital abnormalities (e.g., congenital heart disease), physical disabilities, and chronic infections (e.g., tuberculosis).

The sample size was calculated using the formula for proportions at a 95% confidence level, with $P = 27\%$(stunting prevalence in Pauh sub-district), precision $d = 10\%$, and a 20% contingency, yielding 42 children. Data collection involved gathering mothers of stunted children at the health center and home visits. The final sample comprised 43 respondents (33 stunted and 10 severely stunted children) from the Pauh Health Center area, recruited directly.

Independent variables were serum vitamin D and calcium levels; the dependent variable was stunting status. Stunting was defined as short stature in children aged 12-23 months based on height-for-age (HAZ) z-scores per WHO 2006 standards: stunting (z-score <-2SD) and severe stunting (z-score <-3SD), with WA < LA < chronological age.

Serum 25(OH)D levels were measured at a private laboratory using chemiluminescent microparticle immunoassay (CMIA), reported in ng/mL, and categorized as: deficiency (<20 ng/mL), insufficiency (20–<30 ng/mL), or sufficiency (30–<100 ng/mL). Total serum calcium was assessed via photometric method with arsenazo III at Prodia Laboratory (Padang City), reported in mg/dL: normal (8.8–10.8 mg/dL) or low (<8.8 mg/dL).

External variables included age, sex, parental education, birth weight, exclusive breastfeeding history, and energy intake. Child age was calculated as months between birth and measurement dates, grouped as 12–17 months or 18–23 months. Parental education was the highest formal level attained by either parent: low (< high school/SMA) or high (≥ SMA). Birth weight was sourced from the Maternal and Child Health (KIA) Book and classified as low birth weight (LBW; <2,500 g).

Univariate analysis described data characteristics and distributions. Bivariate analysis examined independent-dependent variable associations using chi-square tests and independent-samples t-tests. Data collection was supported by enumerators with D3-level health qualifications: one public health specialist for anthropometry, one nutritionist for dietary interviews, and one laboratory technician for phlebotomy.

RESULTS AND DISCUSSION

Forty-three study subjects consisted of 33 stunted children and 10 severely stunted children. The number of boys was nearly equal to that of girls, and most of the children were aged 18-23 months. Most of the mothers and fathers were highly educated (high school graduates), with mothers generally working as housewives and fathers working as laborers. Most of the subjects were born with normal birth weight (72.1%), more than half were not exclusively breastfed (53.5%), and most of the children had incomplete immunizations (65.1%). (Table 1).

Table 1. Distribution of Research Subject Characteristics

Variable	n (43)	%
Stature		
- Severe Stunting	10	23.3
- Stunting	33	76.7
Age		
- 12-17 months	14	32.6
- 18-23 months	29	67.4
Gender		
- Male	20	46.5
- Female	23	53.5
Mother's education		
- Low educated	12	27.9
- Highly educated	31	72.1
Father's education		
- Low educated	14	32.6
- Highly educated	29	67.4
Mother's occupation		
- Not working	41	95.3
- Working	2	4.7
Father's occupation		
- Laborer	34	79.1
- Farmer	2	4.7
- Private sector worker	7	16.3
Birth Weight		
- Low birth weight	12	27.9
- Normal	31	72.1
Exclusive breastfeeding		
- No	23	53.5
- Yes	20	46.5
Immunization status		
- Incomplete	28	65.1
- Complete	15	34.9

Table 2. Gene Percentage CDX2 in Stunting and Severe Stunting Children

Gene	Stunting n (%)	Severe Stunting n (%)	Total n (%)
rs11568820			
Alel CC = Wild Type	6 (66,7)	3 (33,3)	9 (100)
Alel CT = Heterozygote Mutation	13 (72,2)	5 (27,8)	18 (100)
Alel TT = Homozygote Mutation	10 (91,0)	1 (9,0)	11 (100)

The TT genotype (homozygous mutation) had the highest proportion of stunting (91%) and the lowest proportion of severe stunting (9%). Conversely, the CC (wild type) genotype showed a higher proportion of severe stunting (33.3%) than the TT genotype. This may indicate that the homozygous mutation in rs11568820 may not always worsen the condition—there may be biological compensation or other mechanisms that protect against severe stunting in the TT genotype.

Table 3. Gene Percentage GATA in Stunting and Severe Stunting Children

Gene	Stunting n (%)	Severe Stunting n (%)	Total n (%)
rs4516035			
Alel CC = Wild Type	30 (76,9)	9 (23,1)	39 (100)
Alel CT = Heterozygote Mutation	2 (66,7)	1 (33,3)	3 (100)
Alel TT = Homozygote Mutation	1 (100)	0(0)	1 (100)

The CC genotype (homozygous mutation) was present in only one case of stunting, and none in severe stunting. The TC genotype (heterozygous mutation) had the highest proportion of severe stunting (33.3%) compared to the wild type (23.1%). No significant difference was observed between TT and TC in children with stunting (76.9% vs. 66.7%).

Table 4. Bivariate Analysis of Vitamin D Levels in Stunting and Severe Stunting Children

Variable	Stunting		Severe Stunting		p
	n	%	n	%	
Vitamin D levels					0,124
- Deficiency	2	6,1	2	20	
- Insufficiency	20	60,6	3	30	
- Sufficiency	11	33,3	5	50	
Total	33		10		

The results of the study show that most vitamin D levels in stunting subjects are categorized as hypovitaminosis D (66.7%) with details of 6.1% deficiency and 60.6% insufficiency. However, in subjects with severe stunting, the number of those with hypovitaminosis D (deficiency and insufficiency) and normal (sufficiency) is the same (50%). Based on bivariate analysis, it is known that there is no significant difference between vitamin D levels in stunting and severe stunting children ($p>0.05$). The average vitamin D level in stunting children is 12.14±1.97 mg/dL, while in severe stunting children it is 12.16±1.63 mg/dL, and the t-test results show that there is no significant difference ($p>0.05$).

Table 5. Bivariate Analysis of Calcium Levels in Stunting and Severe Stunting Children

Variable	Stunting		Severe Stunting		p
	n	%	n	%	
Calcium levels					0,529
- Low	4	12,1	2	20	
- Normal	29	87,9	8	80	
Total	33		10		

The results of the study show that the majority of serum calcium levels in subjects based on stunting and severe stunting are categorized as normal (87.9% and 80%). Based on bivariate analysis, it is known that there is no significant difference between serum calcium levels in stunting and severe stunting children (p>0.05). The average serum calcium level in stunting children is 12.14±1.97 mg/dL, while in severe stunting children it is 12.16±1.63 mg/dL, and the t-test results show that there is no significant difference (p>0.05).

Table 6. Bivariate Analysis of Vitamin D Levels by Nutritional Status

	Well-nourished		Malnutrition		
Variable	**n**	**%**	**n**	**%**	**p**
Vitamin D levels					0,042
- Hypovitamin D	17	77,3	10	47,6	
- Normal	5	22,7	11	52,4	
Total	22		21		

The results of the study show that the majority (77.3%) of vitamin D levels in subjects with good nutritional status are low (hypovitamin D), conversely, more than half (52.4%) of malnourished children have normal vitamin D levels. Based on bivariate analysis, it is known that there is a significant difference between vitamin D levels and nutritional status (p>0.05). The average vitamin D level in children with good nutrition is 12.14±1.97 mg/dL, while in malnourished children it is 12.16±1.63 mg/dL, the t-test results show that there is no significant difference (p>0.05).

Table 7. Bivariate Analysis of Calcium Levels by Nutritional Status

	Well-nourished		Malnutrition		
Variable	**n**	**%**	**n**	**%**	**p**
Calcium levels					0,077
- Low	5	22,7	1	4,8	
- Normal	17	77,3	20	95,2	
Total	22		21		

The results of the study show that serum calcium levels in subjects with good nutritional status and malnutrition are mostly normal, with 77.3% for good nutrition and almost all subjects with malnutrition having normal calcium levels at 95.2%. Based on bivariate analysis, it is known that there is no significant difference between serum calcium levels and nutritional status (p>0.05). The average serum calcium level in children with good nutrition is 12.14±1.97 mg/dL, while in malnourished children it is 12.16±1.63 mg/dL, the t-test results show that there is no significant difference (p>0.05).

Discussion

Vitamin D participates in various metabolic processes in the body, including calcium and bone metabolism (classification and ossification processes). The main function of 1,25 dihydroxy vitamin D as the active metabolite of vitamin D is to control the absorption of

calcium and phosphate in the intestines by stimulating the synthesis of calcium and calcium binding protein at the brush border of the intestinal mucosa, with the aim of maintaining blood calcium concentration so that bone mineralization is well preserved. If there is a deficiency of vitamin D, this homeostasis process will be affected (Javorsky et al. 2006; Kimball, Fuleihan, and Vieth 2008). Calcium absorption is influenced by the adequacy of vitamin D levels, where if vitamin D levels are sufficient, namely 25 (OH) D >50 nmol/L, calcium absorption can reach 30%. Meanwhile, during periods of rapid growth, calcium absorption can reach 60% to 80% (Holick et al. 2011).

Indonesia is a tropical country with abundant sunlight exposure; however, the prevalence of vitamin D deficiency is quite high. This occurs due to a lack of sunlight exposure (ultraviolet B) caused by habits of wearing closed clothing or using sunblock, compounded by a low intake of vitamin D-rich foods. The South East Asian Nutrition Surveys (SEANUTS) in 2011, which included a recap of data from 300 children aged 2 to 12 years, representing 48 districts/cities in Indonesia, found that Indonesian children experience calcium and vitamin D deficiency due to insufficient consumption of calcium and vitamin D-rich food sources, which is lower than the recommended daily intake (AKG). In the SEANUTS study, it was found that vitamin D in the blood has a significant correlation with bone mass and tibia bone density ($p<0.05$). This study recommends that calcium and vitamin D fortification in food is important to improve the nutritional status of Indonesian children and 20% RDA per serving size is recommended (Valentina et al. 2014).

The vitamin D requirements based on recommended dietary allowances (RDAs) are listed in micrograms (mcg) and international units (IU), where 1 mcg of vitamin D is equal to 40 IU. The vitamin D requirement for children aged 0 to 12 months is 10 mcg (400 IU) and for ages 1 to 13 years is 15 mcg (600 IU). Vitamin D supplementation is recommended for children at risk of deficiency, such as the child who minimally receives sunlight exposure, or vitamin D intake from low food. Supplementation aims to maintain serum 25(OH)D levels above 20 ng/mL (50 nmol/L) to support bone growth. European Food Safety Authority issued safety limit for vitamin D consumption which is set at 1000 IU/day for infants, 2000 IU/day for children aged 1 year, 4000 IU/day for children and adolescents aged 11-17 years (Grossman et al. 2017).

Numerous studies have demonstrated that the activity of 1,25-dihydroxyvitamin D (RVD) results in various biological activities in tissues through genomic and non-genomic pathways (Haussler et al., 2010). The RVD gene is located primarily in the cell nucleus, where it acts as a transcription factor that regulates gene expression/transcription (genomic pathway) that controls specific cell biological functions (Uitterlinden et al. 2004; Bikle D, 2017). The Vitamin D receptor (RVD) plays a role in bone metabolism, which is important for linear growth, including intestinal calcium absorption and modulation of the immune response. Nuclear expression and activation of the vitamin D receptor are required for the effects of vitamin D. The presence of the RVD is an absolute requirement for efficient calcium absorption. Mutations in this gene are associated with vitamin D-resistant rickets type II (Fleet, 2017).

The finding that vitamin D levels showed no significant difference between stunted and severely stunted children was unexpected given the well-established role of vitamin D in linear growth. This contrasts with several previous studies that have documented associations between vitamin D deficiency and stunting severity. For instance, a study by Chairunnisa et al. (2018) in Semarang found significant differences in vitamin D intake between stunted and non-stunted children. However, our findings align more closely with research by Sari et al. (2016), which also found no significant differences in certain micronutrient levels between stunting categories. This discrepancy may be explained by several factors unique to our study population and setting.

The levels of vitamin D in stunted and severely stunted children in this study were divided into three: 9.3% of children experienced deficiency, more than half (53.4%) suffered from insufficiency, and one-third of children (37.2%) had sufficient vitamin D status. This condition may be caused by children receiving sufficiently high ultraviolet sunlight exposure, considering that Padang City is located on the equatorial line with high sunlight exposure, where the largest source of vitamin D (up to 80%) is obtained from ultraviolet sunlight. Padang City is one of the tropical areas with relatively high air temperatures accompanied by high humidity. The average daytime temperature in Padang City ranges from 23°C to 32°C, with a monthly maximum temperature reaching up to 34.0°C, and a minimum temperature of around 22.4°C. The relative humidity in Padang City averages between 80% to 85% throughout the year, and the average daily sunlight exposure ranges from 4.2 to 4.6 kWh/m² per day, equivalent to 35-38% of the total duration of daylight (Bappeda Padang, 2018).

Vitamin D3 is produced in the skin from 7-dehydrocholesterol through a non-enzymatic process, utilizing ultraviolet B (UVB) light energy. Abundant sunlight leads to relatively high serum 25-hydroxycholecalciferol levels, resulting in high vitamin D levels. Ultraviolet B is very important in this reaction, stimulated in the kidneys by 1α-hydroxylase, an enzyme that converts 25-hydroxycholecalciferol into 1,25-dihydroxycholecalciferol or 1,25(OH)2D3, which is the active form of vitamin D. The increased synthesis of 1,25(OH)2D3 can further contribute to the increase in serum calcium levels (Cline J, 2012).

Research results show that the majority (77.3%) of vitamin D levels in subjects with good nutritional status were found to be low (hypovitaminosis D), conversely, children suffering from malnutrition more than half (52.4%) had normal vitamin D levels. Fat-soluble vitamin D has different distributions in children with various nutritional statuses. Children with good nutritional status or mild obesity have more adipose tissue than malnourished children. Vitamin D tends to be retained in adipose tissue (lipophilic), so its blood levels can be low, leading to hypovitamin D. Conversely, in thin or malnourished children, due to the lack of adipose tissue, more vitamin D circulates in the blood, so serum levels can appear normal even though total reserves are low (Moon et al., 2019).

In this study, calcium levels in stunted children and severe stunting are generally normal even though vitamin D levels are low. Low vitamin D levels will be related to decreased calcium absorption in the intestines, which impacts the active vitamin D (calcitriol) working more to increase calcium absorption in the intestines. Normal serum calcium levels can occur because the body prioritizes maintaining serum calcium levels so that serum calcium remains within normal levels. If calcium absorption from food also decreases, the body detects the drop in calcium, then the parathyroid glands will increase the secretion of parathyroid hormone (PTH) to maintain serum calcium levels by stimulating bone resorption so that calcium is released from bones into the blood, increasing calcium reabsorption in the kidneys to reduce calcium lost through urine, activating the enzyme 1α hydroxylase in the kidneys with the aim of increasing the production of active vitamin D (calcitriol), even though its initial levels are low. This process keeps serum calcium normal thanks to the work of PTH and the release of reserves from bones, but this will sacrifice bone density, especially if it lasts a long time.

Serum calcium levels in this study show that in all stunted children, generally (85.1%) are normal and only 13.9% have low calcium levels. Bivariate analysis results show that there is no significant difference between serum calcium levels in stunted children and severe stunting. The absence of significant differences indicates that the body always maintains serum calcium levels. Serum calcium levels are kept very tightly (homeostasis) where the body must maintain serum calcium levels within a narrow range (around 8.5 – 10.5 mg/dL) due to the very important role of calcium. If calcium intake is low, the body will increase calcium absorption from the intestines (with the help of active vitamin D), take calcium reserves from bones (bone resorption), and reduce calcium excretion by the kidneys. This condition is what

causes the average calcium serum levels to not differ significantly. This compensation keeps serum calcium levels normal, even when bone reserves are depleted.

This study found no significant relationship between serum calcium levels and nutritional status. A child's nutritional status can indirectly influence calcium levels, or rather, it does not linearly reflect a person's nutritional status. Serum calcium levels are not a sensitive indicator of nutritional status, as the body maintains calcium levels strictly for vital cellular functions. However, if malnutrition persists (chronic), calcium deficiency can occur, and the body can no longer compensate. Decreased serum calcium levels occur, especially when accompanied by deficiencies in vitamin D, magnesium, or animal protein. This condition usually occurs in children with severe malnutrition (marasmic malnutrition or kwashiorkor) or suffering from serious illnesses that cause metabolic disorders, not just stunting with mild to moderate malnutrition. Stunted children often have low calcium and vitamin D intakes, but serum calcium levels remain normal because calcium is drawn from the bones to maintain vital organ function. The above explains the largely normal serum calcium levels in stunted children in the Pauh Community Health Center (Puskesmas) area, Padang City.

CONCLUSION

This study revealed no significant differences in serum vitamin D or calcium levels between stunted and severely stunted children aged 12-23 months in the Pauh Community Health Center (Puskesmas) area of Padang City, despite low levels of these micronutrients inhibiting linear growth by disrupting growth hormone and bone-regulating signals—particularly critical during the first 1000 days of life (HPK). For future research, investigators should explore additional contributors to stunting severity, such as genetic factors (e.g., VDR polymorphisms), dietary patterns, infection burden, or socioeconomic determinants through longitudinal cohort studies with larger samples.

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