Vitamin D in children with primary hypertension

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Abstract

Introduction: Recent evidence suggests that vitamin D plays a role in pathogenesis of arterial hypertension. The aim was to assess vitamin D in children and adolescents with arterial hypertension.

Material and methods: In 49 children (14.29 ± 3.17 years) with arterial hypertension we evaluated vitamin D status (according to Polish 2018 Guidelines), serum calcium, phosphorus, parathormone, alkaline phosphatase, office blood pressure, ABPM (including ambulatory arterial stiffness index [AASI]), BMI, GFR, uric acid, lipids and albuminuria. None of the children were supplemented with vitamin D.

Results: Mean vitamin D concentration was 19.74 ± 9.68 ng/mL. Vitamin D severe deficiency (0–10 ng/mL) was found in 5 (10.2%), deficiency (> 10–20 ng/mL) in 29 (49.0%), suboptimal concentration (> 20–30 ng/mL) in 17 (34.7%), optimal concentration (> 30 to 50 ng/mL) in 1 (2.0%), and high concentration (> 50 to 100 ng/mL) in 2 (4.1%) children. Vitamin D was higher in spring-summer vs. autumn-winter (21.79 ± 10.19 vs. 15.53 ± 7.08 ng/mL, $p = 0.03$). Vitamin D correlated with height Z-score ($r = 0.39$, $p < 0.01$), BMI Z-score ($r = –0.34$, $p = 0.02$), uric acid ($r = -0.31$, $p = 0.04$), triglycerides ($r = -0.37$, $p = 0.01$), but not with office blood pressure and ABPM parameters except for heart rate ($r = -0.38$, $p < 0.01$). In 24 children treated with antihypertensive medications vitamin D correlated with AASI ($r = 0.50$, $p = 0.04$).

Conclusions:

1. Inadequate vitamin D supply is ubiquitous in children with arterial hypertension.
2. Vitamin D deficiency should be suspected especially in autumn-winter period and among obese and short children.
3. The relation between vitamin D status and ambulatory arterial stiffness index suggests negative influence of vitamin D on arterial wall but requires further investigations.

Key words: children, adolescents, primary hypertension, vitamin D, ambulatory blood pressure monitoring, arterial stiffness

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Introduction

Arterial hypertension (AH) is found in 3–5% of children and adolescents. Primary hypertension (PH) is a dominant form of AH in teenagers and its prevalence in paediatric population is increasing due to outbreak of obesity and excessive salt intake [1, 2].

Twenty-four hour ambulatory blood pressure monitoring (ABPM) allows earlier detection of abnormal blood pressure and shows better correlation with target organ damage compared to office measurements. High blood pressure variability and disturbed circadian blood pressure rhythm were found

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to be risk factors for the development of target organ damage including left ventricular hypertrophy [3]. Based on ABPM data [4], the ambulatory arterial stiffness index (AASI) was proposed as a marker of arterial compliance.

Vitamin D is a group of fat-soluble sterols with most important compounds being vitamin D_3 (cholecalciferol) and vitamin D_2 (ergocalciferol). Ingested vitamin D is hydroxylated in liver to calcifediol (25(OH)D), which is the most abundant metabolite of vitamin D, and its serum concentration defines the status of vitamin D supply. Calcifediol is further hydroxylated by kidneys to calcitriol ($1,25(OH)_2D$), the biologically active form of vitamin D [5, 6].

Vitamin D deficiency affects general population irrespective of latitude, age, sex and race [5]. In Poland vitamin D deficiency was found in 90% of adults, children and adolescents [7, 8]. Vitamin D deficiency may be associated with well-known calcaemic effects as well as with a broad spectrum of other effects, e.g. increased risk of neoplasms, asthma, autoimmune disorders and cardiovascular diseases [5, 6].

The relation between vitamin D status and risk of arterial hypertension has been thoroughly studied in recent years [9–14]. Experimental studies indicate that vitamin D deficiency may contribute to blood pressure elevation by activation of renin-angiotensin-aldosterone system, generation of reactive oxygen species with resulting endothelial dysfunction and impaired secretion of nitric oxide [6, 15].

In observational studies an inverse relation between vitamin D status and arterial hypertension was found [11–14] but interventional trials have not confirmed unequivocally the impact of vitamin D supplementation on reducing blood pressure [9].

There are only single studies on vitamin D in paediatric patients with hypertension. Płudowski did not find significant differences in vitamin D status between children with prehypertension, hypertension stage 1, and stage 2. Also these authors [16] did not find relation between vitamin D supply and carotid intima media thickness and left ventricular mass. Kao [17] found that vitamin D status is inversely related to systolic and diastolic blood pressure in overweight and obese adolescents.

To the best of our knowledge there are no data on relation between vitamin D status and blood pressure evaluated by ABPM and ABPM-derived indices (blood pressure dipping, blood pressure variability, ambulatory arterial stiffness index).

The aim of our study was to assess vitamin D status in children and adolescents with primary hypertension and to find relations between vitamin D, blood pressure assessed in office measurement and ABPM, and selected clinical and biochemical parameters.

Material and methods

In this prospective cross-sectional single-centre study we analysed data of 49 children and adolescents with primary hypertension. Children who were supplemented with vitamin D during last 12 months were excluded from the study.

In all children hypertension was confirmed using Polish Society of Arterial Hypertension and Polish Society of Paediatric Nephrology guidelines [1, 2]. Secondary forms of hypertension were excluded by medical history and physical examination and by means of additional tests: renal function tests (creatinine, urea), serum ions (sodium, potassium, and calcium), capillary blood gases, urinalysis, and abdominal ultrasonography with evaluation of kidneys, urinary tract, adrenal glands, and blood flow in renal arteries. Detailed diagnostic tests for secondary forms of hypertension were performed in all children younger than 10 years and with grade II hypertension [1, 2].

In all patients we assessed following clinical parameters: sex, age [years], duration of arterial hypertension [months], height [cm], weight [kg], BMI [kg/m^2]. Anthropometric variables were compared with Polish normative data and expressed as Z-score [18]. Overweight and obesity were defined according to World Health Organization definitions as BMI Z-score values > 1 and > 2, respectively. We assessed also antihypertensive medications and time period (month) of clinical and biochemical evaluation.

Following biochemical parameters were evaluated: serum creatinine [mg/dL], uric acid [mg/dL], total cholesterol [mg/dL], triglycerides [mg/dL], and daily urinary albumin loss [mg/24h]. Glomerular filtration rate (GFR) was calculated according to Schwartz formula [19]. Hyperuricaemia was defined as uric acid > 6.0 mg/dL, hypercholesterolaemia as cholesterol ≥ 200 mg/dL, hypertriglyceridaemia as triglycerides ≥ 100 mg/dL (children aged 0–9 years) or ≥ 130 mg/dL (children aged 10–19 years), and abnormal urinary albumin excretion as albuminuria > 30 mg/24h [1, 2].

Following parameters of calcium-phosphorus metabolism were evaluated: 25(OH)D [ng/mL], calcium [mg/dL], inorganic phosphorus [mg/dL], alkaline phosphatase [IU/L], and intact parathormone (PTH) [pg/mL]. According to Polish recommendations, vitamin D concentrations were defined as: severe

deficiency (0–10 ng/mL), deficiency (> 10–20 ng/mL), suboptimal (> 20–30 ng/mL), optimal (> 30–50 ng/mL), high (> 50–100 ng/mL), and toxic (> 100 ng/mL) levels [5]. Normal concentrations of calcium (8.8–10.7 mg/dL), phosphorus (2.8–5.6 mg/dL), alkaline phosphatase (45–515 IU/L), and parathormone (12–95 pg/mL) were taken from normative values in accordance with producer's recommendations. Vitamin D was assessed by chemiluminescence (ARCHITECT i1000SR, Abbott, USA), parathormone by chemiluminescence (IMMULITE 2000XPi, Healthcare-in-Europe, Germany), all other biochemical parameters by dry chemistry (VITROS 5600, Ortho Clinical Diagnostics, USA).

Peripheral blood pressure was measured using oscillometric device (Welch Allyn VSM Patient Monitor 300, Welch Allyn, USA). Obtained values were expressed as [mm Hg] and were compared with normative values [20] and expressed as Z-score.

24-hour blood pressure measurement was performed using a SUNTECH OSCAR 2 device (SunTech Medical, Inc., Morrisville, NC, USA) and interpreted according to the American Heart Association (AHA) guidelines. Monitors were programmed to measure blood pressure every 15 minutes from 6 AM to 10 PM and every 30 minutes from 10 PM to 6 AM. Periods of night-time rest and daytime activity were determined individually by providing data in a diary. Following parameters were evaluated based on ABPM: systolic, diastolic, and mean arterial pressure (SBP, DBP, MAP) during 24 hours [mm Hg], systolic and diastolic blood pressure load during 24 hours [%], nocturnal blood pressure dip [%], blood pressure variability, and AASI. Blood pressure load was calculated as a percentage of measurements ≥ 95th percentile during 24 hours and increased load was defined as > 25% [3]. AASI was calculated as 1 minus correlation coefficient between diastolic and systolic blood pressure values over 24 hours in ABPM [4]. Nocturnal systolic and diastolic blood pressure dip (SBP DIP, DBP DIP, respectively) was calculated as difference between daytime pressure and nighttime pressure expressed as a percentage of the day value. Disturbed circadian blood pressure rhythm was defined as nocturnal systolic or diastolic blood pressure dip less than 10%. Blood pressure variability was calculated as standard deviation (SD) from mean of all systolic or diastolic blood pressure values over 24 hours (SD SBP/24h, SD DBP/24h) [3].

The research project was approved by local Ethics Committee (approval No. KB/58/2016). All procedures were performed in accordance with the Declaration of Helsinki on the treatment of human subjects. Informed consent was obtained from all participants (≥ 16 years) and their representatives included in the study.

Statistical analysis

Statistical elaboration was performed using Dell Statistica 13.0 PL software (Dell Inc., AlisoViejo, CA, USA). Variables were presented as the mean ± standard deviation (SD). Normality of data distribution was tested using the Shapiro-Wilk test. Differences between normally distributed data were tested using the Student t-test, whereas differences between non-normally distributed data using the U Mann-Whitney test. Correlations between parametric and non-parametric variables were evaluated using the Pearson or the Spearman rank correlation, respectively. A p-value < 0.05 was considered statistically significant.

Results

Clinical characteristics of the study group together with biochemical parameters were presented in Table I. In the study group, about two third of the patients were boys. Mean age of the studied children was about 14 years; 5 (10.2%) children were younger than 10 years. Twenty-nine (59.2%) patients were

Table I. Clinical and biochemical characteristics of the study group

Parameter	Value
Number of patients (n)	49
Boys/girls (n, %)	34/15 (69.4%/30.6%)
Age [years]	14.29 ± 3.17 (from 5.58 to 18.0)
Duration of arterial hypertension [months]	19.26 ± 20.52
BMI Z-score	1.19 ± 0.79
Overweight (n, %)	14 (28.6%)
Obesity (n, %)	15 (30.6%)
Antihypertensive medications (n, %)	24 (49.0%)
Number of antihypertensive medications: 1 medication 2 medications 3 medications	 14 9 1
GFR ac. to Schwartz [mL/min/1.73 m^2]	99.41 ± 20.95
Total cholesterol [mg/dL]	163.86 ± 37.46
Triglycerides [mg/dL]	94.19 ± 32.42
Uric acid [mg/dL]	5.53 ± 1.23
Albumin urinary excretion [mg/24h]	23.68 ± 31.54

BMI — body mass index; GFR — glomerular filtration rate

Table II. Parameters of calcium-phosphorus metabolism in children and adolescents with primary hypertension

Parameter	Value
Calcium [mg/dL]	9.99 ± 0.27
Phosphorus [mg/dL]	4.54 ± 0.68
Calcium phosphorus product [mg^2/dL^2]	45.22 ± 7.04
Parathormone [pg/mL]	19.74 ± 9.68
Alkaline phosphatase [IU/L]	126.73 ± 61.44
25(OH)D [ng/mL]	19.74 ± 9.68 (from 6.1 to 55.3)
Vit. D severe deficiency (25(OH)D: 0–10 ng/mL)	5 (10.2%)
Vit. D deficiency (25(OH)D: < 20 ng/mL)	29 (49.0%)
Vit. D suboptimal status (25(OH)D: 20–30 ng/mL)	17 (34.7%)
Vit. D optimal status (25(OH)D: >30–50 ng/mL)	1 (2.0%)
Vit. D high supply (25(OH)D: > 50–100 ng/mL)	2 (4.1%)

overweight or obese. Half of the patients were already treated with antihypertensive medications during the evaluation. All children had normal kidney function. Hyperuricaemia was recognized in 13 (26.5%) patients with highest uric acid concentration 8.2 mg/dL. Hypercholesterolaemia was found in 6 (12.2%), and hypertriglyceridaemia in 7 (14.3%) children. Elevated urinary albumin excretion was found in 8 (16.3%) patients.

Parameters of calcium-phosphorus metabolism were presented in Table II. In all the studied children calcium, phosphorus, calcium-phosphorus product, alkaline phosphatase, as well as intact parathormone were within normal limits. Vitamin D deficiency and suboptimal concentration were revealed in 46 out of 49 patients (93.9%).

Blood pressure measurements were presented in Table III. In 24-hour ABPM performed at the day of blood sampling, systolic hypertension was found in 34 patients (69.4%), and diastolic hypertension in 12 children (24.9%); 21 patients (42.9%) had elevated mean arterial pressure. Abnormal systolic, diastolic or mean blood pressure was found in all 25 children with untreated arterial hypertension. Disturbed circadian blood pressure rhythm was found in 16 children (32.7%).

Vitamin D concentration was significantly higher in spring-summer period compared to autumn-winter period (21.79 ± 10.19 vs. 15.53 ± 7.08 ng/mL, p = 0.03). We found no differences in vitamin D concentration between boys and girls (20.14 ± 11.13 vs. 18.83 ± 5.25 ng/mL, p = 0.97) and between children treated and not treated with antihypertensive medications (20.72 ± 12.71 vs. 18.80 ± 5.53 ng/mL, p = 0.65) (Figure 1).

In group of 49 children vitamin D concentration correlated negatively with height Z-score (r = –0.39, p < 0.01), BMI Z-score (r = –0.34, p = 0.02),

Table III. Blood pressure measurements in the studied children

Office blood pressure	
Systolic blood pressure [mm Hg] Systolic blood pressure Z-score	136.04 ± 13.76 2.14 ± 0.98
Diastolic blood pressure [mm Hg] Diastolic blood pressure Z-score	81.47 ± 9.98 1.38 ± 0.79
24-hour ambulatory blood pressure monitoring	
Systolic blood pressure during 24h [mm Hg]	130.82 ± 10.72
Diastolic blood pressure during 24h [mm Hg]	71.88 ± 6.85
Mean blood pressure during 24h [mm Hg]	91.51 ± 7.51
Mean blood pressure during 24h Z-score	1.49 ± 1.32
SBP load during 24h [%]	47.08 ± 22.02
DBP load during 24h [%]	26.96 ± 19.88
SBP variability	13.44 ± 2.64
DBP variability	11.32 ± 2.56
SBP dipping [%]	11.68 ± 5.75
DBP dipping [%]	17.78 ± 7.45
Ambulatory arterial stiffness index	0.38 ± 0.12

SBP — systolic blood pressure; DBP — diastolic blood pressure

serum triglycerides (r = –0.37, p = 0.01), and serum uric acid (r = –0.31, p = 0.04). No statistically significant correlations between vitamin D and other parameters of calcium-phosphorus metabolism were revealed (Table IV). Vitamin D did not correlate significantly with office blood pressure, ambulatory blood pressure and ABPM-derived blood pressure indices (blood pressure load, blood pressure variability, blood pressure dipping, and ambulatory arterial stiffness index) (Table V). We found only a negative correlation between vitamin D concentration and mean 24-hour heart rate (r = –0.38, p < 0.01) without significant relation between vitamin D and age (r = –0.22, p = 0.12).

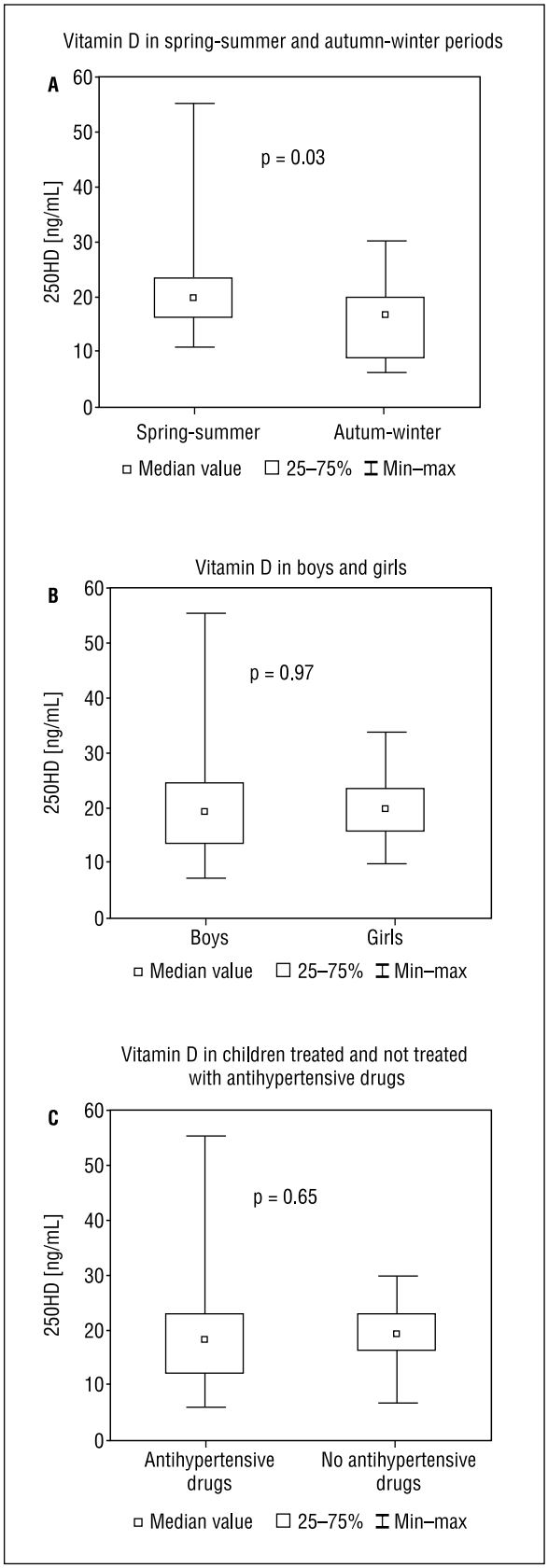

Figure 1. Vitamin D status in children and adolescents with primary arterial hypertension: spring-summer and autumn-winter period (**A**), boys and girls (**B**), and patient treated and not treated with antihypertensive medications (**C**)

Twenty-four out of 49 children (49.0%) were treated with antihypertensive medications during evaluation of vitamin D status. In this subgroup there were 18 boys and 6 girls, and mean age of this cohort was 14.68 ± 3.13 years. Mean duration of hypertension was 23.54 ± 21.65 months and was significantly longer compared to 25 not-treated patients (14.59 ± 18.57 months, p = 0.02). The treated and not treated children did not differ in terms of clinical and biochemical parameters and in ABPM parameters including AASI (0.384 ± 0.095 vs. 0.373 ± 0.139, p = 0.58). Following antihypertensive medications were used in these 24 patients: calcium channel blockers in 18 (75.0%), angiotensin converting enzyme inhibitors in 12 (50.0%), angiotensin receptor blockers in 2 (8.3%), diuretics, beta- and alpha-adrenolytics, each in one (4.2%) patient. In this subgroup of 24 children we found a positive correlation between ambulatory arterial stiffness index and vitamin D status (r = 0.50, p = 0.04).

Discussion

Our results revealed that both vitamin D deficiency and vitamin D suboptimal concentration are abundant among paediatric patients with primary hypertension. Only 3 patients had adequate or high supply of vitamin D. Our results are in consistence with other epidemiological studies assessing vitamin D in Polish children [7, 8]. Also, very similarly to our results, Płudowski [16] found 25(OH)D levels lower than 30 ng/mL in 91%, and lower than 20 ng/mL in 71% of paediatric patients with primary hypertension.

In our cohort risk factors for low vitamin D status were autumn-winter period, short stature, high BMI, hypertriglyceridaemia, and higher uricaemia. Decreased vitamin D concentration in autumn and winter is a consequence of diminished skin production of cholecalciferol from 7-dehydrocholesterol [5, 6]. Severe vitamin D deficiency leads to rickets with negative impact on height but the impact of moderately lowered vitamin D level in adolescents on final height remains unclear [21]. The negative relation between 25(OH)D status and BMI is consistent with results of studies by Turer [22], Kao [17], and Płudowski [16]. According to Polish guidelines, both short stature and obesity are conditions associated with risk for vitamin D deficiency and indications for assessment of vitamin D status [5].

In case of vitamin D deficiency, initially, a compensatory increase of PTH secretion to sustain normocalcaemia is observed. However, a resistance to

Table IV. Correlations of vitamin D concentration with parameters of calcium-phosphorus metabolism

Parameter	r	p
Serum calcium [mg/dL]	r = -0.09	p = 0.57
Serum phosphorus [mg/dL]	r = 0.00	p = 1.00
Calcium phosphorus product [mg^2/dL^2]	r = 0.01	p = 0.95
Parathormone [ng/mL]	r = -0.20	p = 0.19
Alkaline phosphatase [IU/L]	r = 0.05	p = 0.73

Table V. Correlations of vitamin D concentration with office blood pressure and ambulatory blood pressure parameters

Parameter	r	p
Office blood pressure		
SBP [mm Hg]	r = –0.14	p = 0.33
SBP Z-score	r = –0.15	p = 0.30
DBP [mm Hg]	r = –0.19	p = 0.18
DBP Z-score	r = –0.21	p = 0.15
Ambulatory blood pressure monitoring		
SBP during 24h [mm Hg]	r = –0.14	p = 0.34
DBP during 24h [mm Hg]	r = –0.17	p = 0.25
MAP during 24h [mm Hg]	r = –0.15	p = 0.29
MAP during 24h Z-score	r = –0.23	p = 0.11
SBP load during 24h [%]	r = –0.18	p = 0.21
DBP load during 24h [%]	r = –0.13	p = 0.36
SBP variability	r = –0.21	p = 0.14
DBP variability	r = –0.23	p = 0.12
SBP dipping [%]	r = –0.15	p = 0.30
DBP dipping [%]	r = –0.21	p = 0.14
Ambulatory arterial stiffness index	r = 0.26	p = 0.11

SBP — systolic blood pressure; DBP — diastolic blood pressure; MAP — mean arterial pressure

PTH may develop resulting in decreased calcium concentrations and increased phosphorus concentrations and symptoms of hypocalcaemia together with osteopenia. When severity of vitamin D deficiency progresses, PTH resistance gets overcome leading to improved calcaemia but also to hypophosphataemia and clinical and radiological manifestation of rickets. Alkaline phosphatase increases, whereas $1,25(OH)_2D$ is normal or increased. Finally, when vitamin D deficiency becomes very severe, $1,25(OH)_2D$ synthesis is inhibited with subsequent impairment of calcium and phosphorus absorption, along with persistent elevation of PTH [5, 21]. Interestingly, despite extremely common low levels of vitamin D we found no other abnormalities in calcium-phosphorus metabolism in our cohort. Calcium, phosphorus, PTH, and alkaline phosphatase remained normal in all studied children. In addition, no significant correlation was found between 25(OH)D and these variables. In a study by Płudowski only a weak negative correlation between parathormone and 25(OH)D was revealed (r = –0.24, p = 0.03) [16]. We hypothesize that negative findings of our study may be result of small sample size as we found trend towards higher PTH level in more vitamin D deficient subjects (r = –0.20, p = 0.19).

The fact that primary hypertension is more commonly seen in higher latitudes, and in those with deep skin pigmentation living far from the equator triggered speculations that vitamin D deficiency contributes to increased prevalence of primary hypertension. Krause [10] treated adults with PH with UVB irradiation causing increase in 25(OH)D levels and drop in blood pressure in vitamin D-deficient individuals. This finding published already 20 years ago raised research interest in the relationship between

vitamin D and PH. Most of the cross-sectional studies showed an inverse relation between 25(OH)D status and systolic blood pressure or prevalence of primary hypertension [11, 12]. In addition, most prospective observational studies [13, 14] with observation period up to 15 years [13] revealed increased risk for developing PH in case of vitamin D deficiency.

Unfortunately, interventional trials on vitamin D supplementation in adults with arterial hypertension bring inconsistent results. Some studies show beneficial impact of vitamin D supplementation [23], while other not [9]. Chen [6] states that inconsistent results of these trials were caused by short observation period and vitamin D supply not adjusted to actual vitamin D status. The ongoing prospective 5-year VITAL study investigating impact of 2000 IU/24h of cholecalciferol on cardiovascular morbidity is hoped to bring final conclusions on this topic.

In our study we found no significant relation between blood pressure measured both in office and in ABPM and vitamin D status. As blood pressure in paediatric population is dependent on anthropometric variables (especially height) [1, 2, 20] it is recommended to index measurements to normative values. When expressed as Z-score the relation was stronger but still did not reach statistical significance. Oscillometric devices measure directly mean arterial pressure and MAP during 24 hours seems to most precisely reflect cardiovascular burden as showed in ESCAPE study in children with chronic kidney disease [24]. We observed only a trend towards a negative correlation between MAP 24h and vitamin D status ($r = –0.23$, $p = 0.11$). We hypothesize that this relation might reflect findings from adult observational studies [11–14] and could become statistically significant in larger patient cohort.

Similarly, Płudowski [16] did not find differences in vitamin D status between prehypertension, stage 1 hypertension, and stage 2 hypertension paediatric patients. On the other hand, in a study by Kao [6] lower vitamin D concentrations were related to both higher systolic and diastolic blood pressure in overweight and obese adolescents. Noteworthy, in contrast to our study, Kao [17] did not use ABPM, which more precisely reflects blood pressure compared to office measurements.

Ambulatory arterial stiffness index is an ABPM-derived marker of arterial compliance [4]. Its clinical value in children remains unclear, though it was found to be elevated in children with hypertension [25] and IgA nephropathy [26]. In our cohort we observed a tendency towards positive correlation between AASI and vitamin D status. When we analysed a subgroup of treated children, who were characterized by more severe hypertension and longer duration of hypertension, this relation became statistically significant.

The relation between arterial stiffness and vitamin D remains unclear. Experimental studies suggest that stimulation of vitamin D receptor (VDR) by $1,25(OH)_2D$ enhances nitric oxide production and decreases arterial stiffness [6, 15]. In adults both negative relation between arterial stiffness and vitamin D status was found [27] but also lower pulse wave velocity (PWV) in vitamin D deficient individuals was revealed [28]. In addition, both positive [29] and neutral [30] effect of vitamin D supplementation on arterial compliance was found. A negative relation between PWV and augmentation index and vitamin D was found in healthy adolescents [31], adolescents with diabetes mellitus [31] and children with chronic kidney disease [32]. Also, contrary to our results, Turkish authors [33] found a negative relation ($r = –0.385$, $p < 0.001$) between vitamin D status and AASI in adults with newly diagnosed hypertensive adults.

Limitation of our study is small patient sample which could have precluded disclosure of some relations between vitamin D and analysed parameters. We also did not evaluate $1,25(OH)_2D$ concentration which is a direct stimulator of VDR in arterial wall and did not assess "golden standard" of arterial compliance — pulse wave velocity. We are convinced that further studies on impact of vitamin D deficiency and vitamin D supplementation on development of arterial hypertension and arterial wall in children are necessary.

Conclusions

1. Inadequate vitamin D supply is ubiquitous in children with arterial hypertension.
2. Vitamin D deficiency should be suspected especially in autumn-winter period and among obese and short children.
3. The relation between vitamin D status and ambulatory arterial stiffness index suggests negative influence of vitamin D on arterial wall but requires further investigations.

Conflict of interest

The authors declare that they have no conflict of interest.

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