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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Vitamin D insufficiency is associated with coronary artery tortuosity

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Abstract

Background: Increasing evidence suggests a relationship between vitamin D (VD) insufficiency and cardiovascular disease.

Aim: We aimed to investigate the association between serum 25-hydroxyvitamin D (25-OH VD) with coronary tortuosity (CT) in patients with normal or near-normal (< 40% stenosis) coronary arteries.

Methods: The present study was cross-sectional and observational. We enrolled 356 consecutive patients who had undergone coronary angiography for suspected ischaemic heart disease and were found to have normal or near-normal coronary arteries. Patients were categorised as VD insufficient (< 30 ng/mL) or VD sufficient (≥ 30 ng/dL). CT was defined as the presence of ≥ three bends (defined as ≥ 45° change in vessel direction) along the main trunk of at least one coronary artery, present both in systole and in diastole.

Results: The study populations were divided into two groups according to the presence of CT: patients with CT (n = 103, 29%) and patients without CT (NCT; n = 253, 71%). CT is more frequently seen in elderly women and is positively correlated with hypertension. The incidence of VD insufficiency was significantly higher in the CT group (n = 46, 45%) than in the NCT group (n = 90, 36%; p = 0.005). In further multivariate logistic regression analyses, adjustment for major clinical parameters affecting CT showed statistically significant correlations between 25-OH VD and CT (odds ratio = 0.77, 95% confidence interval 0.66–0.98, p = 0.006).

Conclusions: Vitamin D insufficiency was independently associated with coronary tortuosity.

Key words: vitamin D insufficiency, coronary tortuosity, coronary artery disease, biomarkers, coronary angiography

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INTRODUCTION

Coronary artery disease (CAD) is the leading cause of mortality and morbidity worldwide. Coronary angiography is still the gold standard for the diagnosis of CAD, and coronary artery tortuosity (CT) is a common coronary angiography (CAG) finding [1–3]. The aetiology and clinical importance of CT are still unclear. CT is generally attributed to age-dependent, pathologic changes of elastic material in the vessel, and hypertension due to elongation and dilatation of the arteries with left ventricular hypertrophy [3, 4]. A recent clinical trial showed that CT was associated with subclinical atherosclerosis and increased coronary artery calcification, even in the absence of significant obstructive lesion [5].

Several clinical studies have reported a high prevalence of vitamin D (VD) deficiency in patients with hypertension, diabetes mellitus, CAD, peripheral artery disease, and stroke, and an association between low VD levels and cardiovascular mortality has been shown [6–9]. Postulated mechanisms for these presumed cardiovascular effects include endothelial dysfunction, inflammation, reduced vessel compliance, detrimental effects via bone proteins such as osteoprotegerin, as well as dysregulation of the renin–angiotensin system and arterial wall calcification because of increased parathyroid hormone levels [10, 11]. Additionally, VD effectively preserved the structure of elastic fibres and the ratio of elastic fibres to collagen fibres in the artery media [12]. Given the adverse

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effects of VD insufficiency on the cardiovascular system, VD insufficiency may be associated with CT. We aimed to determine the relationship between CT and VD in patients with chronic stable angina and normal or near normal coronaries using CAG. To our knowledge, this is the first study of 25-OH VD with CT in patients with normal or near-normal coronary arteries (< 40% stenosis).

METHODS

Patients

The present observational cross-sectional study included 356 patients who had undergone CAG for suspected stable ischaemic heart disease between December 2014 and February 2015 and were found to have normal or near-normal coronary arteries. Age, sex, body mass index, and information on the following CAD risk factors were recorded: hypertension (self-report, blood pressure > 140/90 mm Hg, or use of an antihypertensive drug), diabetes mellitus (self-report, fasting glucose > 126 mg/dL, or use of oral hypoglycaemic agents or insulin), dyslipidaemia (self-report, low-density lipoprotein > 130 ng/dL, total cholesterol > 200 ng/dL), and nicotine use (within one year). The use of cardio-protective drugs such as angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, statins, beta-blockers, and calcium channel blockers on admission was recorded. Blood samples were obtained to measure 25-OH VD3 levels. All patients underwent echocardiography to assess left ventricular systolic function and dimensions. The study was approved by the Local Ethics Committee. All patients provided written, informed consent.

Exclusion criteria were: previous myocardial infarction, cardiomyopathy, ejection fraction less than 50%, severe valvular disease, chronic renal or liver disease, active malignancy, and recent VD replacement therapy. There was no regional left ventricle contractile abnormality in the study subjects.

Laboratory methods

The study was performed in Adana, Turkey, which has a Mediterranean climate, with hot, humid summers and a mild climate in winter. Rainfall occurs during spring and autumn. Average temperatures of 41°C in summer and 9°C in winter were recorded. All blood samples were collected during the winter months (December–February) to avoid seasonal variations. Serum was separated and stored at –70°C within 30 min of collection. Serum 25(OH)D concentrations were measured using an automated Vitamin D2–D3 HPLC Analyzer (25-OH Vitamin D2–D3 HPLC Analysis Kit; Zivak Technologies, Kocaeli, Turkey). The intra-assay and interassay coefficients of variation were 6% and 9%, respectively. The detection limit for 25-OH VD was 3 ng/mL. Patients were categorised as VD insufficient (< 30 ng/mL) or VD sufficient (≥ 30 ng/dL).

Coronary angiography

Patients underwent elective CAG using the standard Judkins technique. Iohexol (Omnipaque; Nycomed Ireland Ltd., Cork, Ireland) was used as the contrast agent during angiography in all patients. The coronary arteries were visualised in the left and right oblique planes using cranial and caudal angulation, at a rate of 15 frames per second. Cine-angiography was performed by hand injection through a 6 Fr Judkins diagnostic catheter. Two independent observers evaluated the coronary angiograms. A normal or near-normal coronary artery was defined as having less than 40% stenosis. The thrombolysis in myocardial infarction (TIMI) frame count was determined for each major coronary artery in each patient by two independent observers using a previously reported technique [13]. Frame counts in the left anterior descending coronary artery were divided by 1.7 to correct for its longer length. Coronary slow flow phenomenon (CSFP) was defined as a TIMI frame count greater than 27/frame. Tortuosity was identified by the presence of ≥ three bends (defined as ≥ 45° change in vessel direction) along the main trunk of at least one coronary artery, present both in systole and in diastole [3]. The tortuosity score was calculated as the sum of all bends on the main trunk, as one point for each bend [3].

Statistical analysis

All statistical tests were performed using SPSS for Windows version 19.0 (SPSS Inc., Chicago, Illinois, USA). Continuous data are expressed as mean ± standard deviations and categorical data are expressed as percentages. The χ^2-test was used to assess differences in categorical variables between groups. The relationships among parameters were assessed using Pearson's correlation analysis. The Shapiro-Wilk W test was performed for testing normality. According to the results of a normality test, statistically significant differences between two groups of continuous variables were determined using the independent t-test and Mann-Whitney U test, as appropriate. Multivariable logistic regression analyses were performed to assess the relationships between coronary artery tortuosity and VD insufficiency and risk factors (age, sex, smoking, diabetes mellitus, hypertension, and dyslipidaemia). The results are expressed as relative risk and 95% confidence interval (CI). A p value less than 0.05 was considered statistically significant.

RESULTS

A total of 1327 patients underwent diagnostic left heart catheterisation for stable ischaemic heart disease over a three-month period. Of those, 971 were excluded because they had significant stenosis in at least one major coronary artery or fulfilled other exclusion criteria; thus, 356 patients with normal or near-normal coronary arteries were included in our study. The mean 25-OH VD level was 40.1 ng/mL

Table 1. Baseline characteristics of study patients

	CT (n = 103)	NCT (n = 253)	p
Age [years]	62.3.2 ± 10.2	52.4 ± 11.9	0.008
Body mass index [kg/m^2]	25.2 ± 4.5	26.1±4.9	NS
GFR [mL/min]	88.6 ± 17	85.7±14	NS
Female sex	43.6%	31%	0.006
Diabetes mellitus	13.8%	15.1%	NS
Hypertension	56.9%	29.3%	< 0.005
Hyperlipidaemia	22.1%	21.5%	NS
Smoking	33.6%	32.2%	NS
Biochemical parameters:			
Creatinine [mg/dL]	0.93 ± 0.47	0.98 ± 0.36	NS
Glucose [mg/dL]	93 ± 23	89 ± 34	NS
Haemoglobin [/dL]	13.7 ± 1.6	13.5 ± 1.8	NS
Total cholesterol [mg/L]	172 (107–305)	185 (121–323)	NS
LDL-C [mg/dL]	116 (55–237)	114 (59–263)	NS
HDL-C [mg/dL]	38.2 ± 10.3	37.3 ± 14.1	NS
Triglycerides [mg/dL]	138 (82–355)	144 (71–442)	NS
25-OH VD [ng/mL]	23.9 ± 7.8	33.5 ± 10.6	< 0.005

25-OH VD — 25-hydroxyvitamin D; CT — patients with coronary tortuosity; GFR — glomerular filtration rate; HDL-C — high-density lipoprotein cholesterol; LDL-C — low-density lipoprotein cholesterol; NCT — patients without coronary tortuosity; NS — non-significant

(range, 3–86 ng/mL), and 25-OH VD levels were insufficient (< 30 ng/mL) in 38% (n = 136) of the patients. The study populations were divided into two groups according to the presence of CT; patients with CT (n = 103, 29%) and patients without CT (NCT; n = 253, 71%). Table 1 shows baseline characteristics of CT and NCT groups. The average 25-OH VD level of the patients with CT was lower than the average 25-OH VD level of the NCT group (23.9 ± 7.8 ng/mL vs. 33.5 ± 10.6 ng/mL, p < 0.005). The incidence of VD insufficiency was significantly higher in the CT group (n = 46, 45%) than in the NCT group (n = 90, 36%; p = 0.005). There was no significant difference between both groups regarding smoking, presence of diabetes mellitus, and dyslipidaemia (regarding HDL and LDL levels). However, there was a highly significant difference between both groups regarding hypertension, age, and female sex. The CT group was older, included more women, and was more hypertensive (Table 1). The incidence of CSFP was significantly higher in the CT group (n = 7, 7%) than in the NCT group (n = 10, 4%, p = 0.01). Additionally, a strong association was found between VD insufficiency and CSFP (8% in the VD insufficient group vs. 3% in the VD sufficient group; p = 0.008). The incidence of CT was similar between patients with normal coronary arteries and near-normal coronary arteries (< 40% stenosis). The pattern of CT is indicated in Table 2. The average CT score was 6.4 ± 2.1 in the CT group. There was a significant difference between the VD sufficient and insufficient groups regarding average CT score (5.7 ± 1.9 vs. 7.3 ± 2.3, p = 0.006). There was a significant negative correlation between tortuosity score and 25-OH VD (r = –0.45, p < 0.005). Age was significantly correlated with tortuosity score (r = 0.26, p = 0.008).

Table 2. Pattern of coronary tortuosity

Pattern (tortuosity)	Number of patients (%)
LAD	16 (15%)
Cx	18 (17%)
RCA	10 (10%)
LAD and Cx	23 (22%)
LAD and RCA	17 (17%)
Cx and RCA	11 (11%)
LAD and Cx and RCA	8 (8%)

LAD — left anterior descending artery; Cx — circumflex artery; RCA — right coronary artery

Univariate and multivariate analyses for CT

Several variables were found to be significant in our univariate analysis, including age, female sex, hypertension, and 25-OH VD level, which were entered into a multivariate logistic regression analysis. The data indicate that, age, female sex, hypertension, and 25-OH VD level (Wald = 7.2; odds ratio [OR] = 0.77, 95% CI 0.66–0.98, p = 0.006) were independently correlated with the presence of CT (Table 3).

Table 3. Predictors of angiographic coronary tortuosity in univariate and multivariate logistic regression analysis

Variables	Odds ratio	95% CI	p
Univariate analysis			
Female sex	1.36	1.21–1.97	0.023
Age	2.32	1.44–3.21	< 0.005
Hypertension	2.43	1.8–3.42	< 0.005
25-OH VD level	0.86	0.72–0.94	0.005
Multivariate logistic regression analysis			
Female sex	1.50	1.1–2.2	0.03
Age	1.35	1.02–2.3	< 0.005
Hypertension	1.84	1.78–2.89	< 0.005
25-OH VD level	0.77	0.66–0.98	0.006

CI — confidence interval; 25-OH VD — 25-hydroxyvitamin D

DISCUSSION

The main findings of the present study were that: (1) patients with CT were older, female, hypertensive, and had more VD insufficiency compared with patients in the NCT group; and (2) 25-OH VD level was independently associated with CT in the multiple linear regression analysis.

Coronary tortuosity is a phenomenon often encountered by cardiologists performing CAG. Tortuosity was identified by the presence of ≥ three bends (defined as ≥ 45° change in vessel direction) along the main trunk of at least one coronary artery, present both in systole and in diastole [3]. There was an alternative method defining CT such as tortuosity index (TI). TI was defined as the percentage ratio of the shortest distance divided by the total length of the coronary artery [14]. The aetiology and clinical importance of CT are still unclear. CT is associated with increased acute occlusion of coronary arteries, diabetes, and coronary artery fistula [4, 15]. CT hampers ventricular function and has been proposed as an indicator of ventricular dysfunction [16]. Of the three coronary arteries, the circumflex artery is most often affected, especially when associated with hypertension [1–3]. There have been case reports of looping in proximal portions of major epicardial arteries causing chest pain and ischaemia on myocardial scintigraphy in patients who had no obstructive CAD [17]. The authors of this case report previously proposed two possible mechanisms for the ischaemia; first was axial torsion during systole, and second, increased smooth muscle reactivity [17, 18]. Additionally, Nie et al. [19] showed that CSFP was associated with higher TI and more distal branches in coronary arteries, indicating that the slow flow could also play a role in the chest pain in patients with CT. However, any causative interrelationship between CT and ischaemia needs to be clarified. In order to determine the impact of CT on coronary perfusion, coronary flow reserve or fractional flow reserve calculations can be used in further studies.

Vitamin D is a steroidal hormone that has relation between almost all metabolic pathways. VD insufficiency is an under-recognised problem in adults in the world. National population statistics from the Third National Health and Nutrition Examination Survey (NHANES III) indicate that 41% of men and 53% of women in the United States have hypovitaminosis D [20]. Increasing interest has focused on the role of VD as it relates to the cardiovascular system. Recent clinical studies have associated low levels of 25-OH VD with hypertension, coronary artery calcification, and cardiovascular diseases such as myocardial infarction, acute stroke, congestive heart failure, and diabetes mellitus [6–9]. The mechanisms underlying the role of VD in the prevention of cardiovascular disease are not known; however, they may involve diverse physiological actions on the vascular wall, including reduction of smooth muscle cell proliferation, reduction of secretion of the pro-inflammatory cytokines interleukin-6 and tumour necrosis factor-alpha by macrophages, and increased secretion of the anti-inflammatory cytokine interleukin-10, all acting to reduce vascular inflammation [10, 11]. Given its relationship with cardiovascular disease, VD insufficiency may be associated with CT. The result of our study is in line with this hypothesis. The present study showed that VD was independently associated with CT. The present study is the first study in the literature to evaluate the association between VD and CT.

Several potential mechanisms could explain the higher risk of CT associated with lower 25-OH VD concentrations. Traction and pressure in the lumen are suggested as two forces that tend to lengthen a vessel. These two forces are opposed by retractive forces. Under normal conditions, the retractive force is equal and opposite to the sum of the traction and pressure forces, resulting in a stable length of the vessel [21]. It has been suggested that the retractive force is generated by elastin. Thus, degeneration of elastin in the arterial wall may lead to aneurysmal dilatation and the de-

velopment of arterial tortuosity. Molecular and cell biology research has shown VD receptors to be present on vascular smooth muscle, endothelium, and cardiomyocytes, and VD influences cardiomyocytes and vascular smooth muscle cell function by exerting anti-proliferative effects [22, 23]. A recent trial has shown its participation in regulating the expression of many proteins with vascular action, such as vascular endothelial growth factor, type 9 metalloproteinase, myosin, elastin, type 1 collagen, and gamma-carboxyglutamic acid, the latter a protein that protects the vessel against parietal calcification, and also in the suppression of pro-inflammatory cytokines, including interleukin-6 and tumour necrosis factor-alpha *in vitro* and *in vivo* [22–24]. Moreover, VD suppresses degeneration of elastin in the arterial wall [24].

Li et al. [15], found that CT was positively correlated with essential hypertension. Tortuosity of coronary arteries is also a common finding seen with hypertension due to elongation and dilatation of the arteries associated with left ventricular hypertrophy [4, 15]. The authors assumed that arteries may become tortuous due to reduced axial strain and hypertensive pressure in an elastic cylindrical arterial model. Thus, coronary tortuosity might be one of the forms of artery remodelling induced by hypertension due to increased coronary pressure and blood flow. This is consistent with the finding of the current work. We found a highly significant difference between the CT and NCT groups regarding the presence of hypertension. Experimental studies have suggested that VD may be involved in the regulation of blood pressure and the pathogenesis of hypertension through its effects on calcium homeostasis, vascular smooth muscle cells, and endothelial cells, as well as activity of the renin–angiotensin system [25]. Human studies have shown a possible link between inadequate VD status and elevated blood pressure or higher prevalence of hypertension [6].

Davutoglu et al. [26] found that CT was strongly associated with subclinical atherosclerosis indicated by carotid intima–media thickness (CIMT). Oz et al. [27] showed that VD insufficiency was associated with subclinical atherosclerosis, assessed using CIMT. Additionally, recent clinical trials have shown that CT was associated with subclinical atherosclerosis and increased coronary artery calcification (CAC) score even in the absence of significant obstructive lesions [5]. VD insufficiency was independently associated with CAC score and subclinical atherosclerosis [28]. Taken together, these findings show that decreased 25-OH VD might be involved in the development of CT via CAC, hypertension, elastin degeneration, and inflammation.

Most studies have reported a higher prevalence of VD insufficiency among elderly people [20]. In a study based on data from the National Health and Nutrition Examination Survey III by Looker et al. [29], the prevalence of VD deficiency and insufficiency among the adolescent and adult population of the United States was observed more frequently in women. Women were found to have more CT and smaller heart dimensions in comparison with men, and this was thought to be responsible for this trend [4, 15]. A higher proportion of elderly people affected with CT has also been documented [3]. In accordance with previous studies, female sex and increased age were correlated with CT in our study. However, classic risk factors and other baseline clinical characteristics of subjects with and without tortuosity were similar. Accordingly, female sex and increased age may be associated with CT because they have more prevalent VD insufficiency.

Limitations of the study

Our study has several limitations, including a relatively small sample size limited to patients in a stable condition. Furthermore, the cross-sectional and observational nature of our study does not allow us to determine cause and affect relationships. The half-life of VD is approximately three weeks, but serum VD levels may change throughout the day and seasons of the year. Therefore, a single measurement may not reflect actual VD status. 1,25 OH VD3 is the active metabolite and was not measured in the present study.

CONCLUSIONS

Coronary tortuosity is more frequently seen in elderly women and is positively correlated with hypertension. A strong association was found between VD insufficiency and CT, indicating that VD status could also play a role in the pathogenesis of CT. Further studies that investigate the effects of VD replacement therapy on the CT development are warranted.

Conflict of interest: *none declared*

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Związek między niedoborem witaminy D a krętym przebiegiem tętnic wieńcowych

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Streszczenie

Wstęp: Coraz więcej danych wskazuje na związek niedoboru witaminy D (VD) z chorobami sercowo-naczyniowymi.

Cel: Badanie przeprowadzono w celu oceny zależności między stężeniem 25-hydroksywitaminy D (25-OH VD) w surowicy a krętym przebiegiem tętnic wieńcowych u chorych z prawidłowymi lub nieznacznie zmienionymi (zwężenie < 40%) tętnicami wieńcowymi.

Metody: Badanie miało charakter przekrojowy i obserwacyjny. Włączono do niego 356 kolejnych pacjentów poddanych koronarografii z powodu podejrzenia choroby niedokrwiennej serca i u których stwierdzono prawidłowy lub nieznacznie zmieniony obraz tętnic wieńcowych. Chorych podzielono na dwie grupy: osoby z niedoborem VD (< 30 ng/ml) oraz osoby z prawidłowym stężeniem VD (≥ 30 ng/dl). Kręty przebieg tętnic wieńcowych definiowano jako obecność ≥ 3 zagięć (określonych jako zmiana kierunku przebiegu naczynia o ≥ 45°) w głównej części co najmniej jednej tętnicy wieńcowej, widocznych zarówno w czasie skurczu, jak i rozkurczu.

Wyniki: Badaną populację podzielono na dwie grupy w zależności od występowania krętego przebiegu naczyń: osoby z krętym przebiegiem tętnic wieńcowych (CT; n = 103, 29%) i osoby bez krętego przebiegu tętnic wieńcowych (NCT; n = 253, 71%). Kręty przebieg tętnic wieńcowych występuje częściej u kobiet w podeszłym wieku i jest dodatnio skorelowany z nadciśnieniem tętniczym. Niedobór VD występował istotnie częściej w grupie CT (n = 46, 45%) niż w grupie NCT (n = 90, 36%; p = 0,005). W wieloczynnikowej analizie regresji logistycznej wykazano, po skorygowaniu względem najważniejszych parametrów klinicznych wpływających na krętość tętnic, statystycznie istotną korelację między stężeniem 25-OH VD a krętym przebiegiem tętnic wieńcowych (iloraz szans = 0,77; 95% przedział ufności 0,66–0,98; p = 0,006).

Wnioski: Niedobór VD był niezależnie związany z krętym przebiegiem tętnic wieńcowych.

Słowa kluczowe: niedobór witaminy D, kręty przebieg tętnic wieńcowych, choroba wieńcowa, biomarkery, koronarografia

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