Association between parathyroid hormone levels and the extensiveness of coronary artery disease

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Abstract

Objective: Previous studies have suggested that there is a relationship between coronary artery disease (CAD) and parathyroid hormone (PTH) levels. Here, we aimed to evaluate the association between PTH levels and severity of CAD.
Methods: Patients were divided into two groups based on their serum PTH values. Patients with PTH levels ≤72 pg/mL were accepted as Group 1 (n=568) and >72 pg/mL as Group 2 (n=87). Gensini score system and >50% stenosis in any coronary artery with conventional coronary angiography were used to determine the extensiveness of CAD. This study was designed as a prospective and cross-sectional study.
Results: Baseline characteristics except for age, gender, and blood pressure were similar between groups. Mean serum PTH levels of the entire cohort was 43.4±29.5 pg/mL. Median Gensini score was 19.5 in Group 1 and 14.5 in Group 2 (p=0.75). On the other hand, PTH levels were weakly correlated with Gensini score (Spearman's Rho=0.11, p=0.003). Additionally, we did not observe a statistically significant difference between PTH levels and the number of stenotic vessels (p=0.14). This study was designed as a prospective and cross-sectional study.
Conclusion: There is no association between serum PTH levels and extensiveness of CAD. *(Anatol J Cardiol 2016; 16: 839-43)*
Keywords: atherosclerosis, coronary artery disease, parathyroid hormone, Gensini score

Introduction

Atherosclerotic cardiovascular diseases (CVDs) remain the leading cause of mortality around the world. Many studies are being conducted to define new prognostic factors for coronary artery disease (CAD). In this context, previous studies suggest that impaired calcium and bone metabolism, such as primary or secondary hyperparathyroidism and chronic kidney disease, are related with a higher cardiovascular morbidity and mortality (1–3). These studies showed that elevated parathyroid hormone (PTH) levels in chronic renal failure have a positive correlation with increased all-cause and cardiovascular mortality (4); also, some of these studies have been claimed this situation in patient without renal disease (5, 6). Moreover, it has been proven that elevated PTH levels are related to CVD as long as they are within the normal range (7). This situation was explained by PTH potentially having direct or indirect effects on the cardiovascular system through several mechanisms beyond regulation of bone and calcium metabolism.

PTH receptors were shown in various locations, such as the brain, adrenal glands, vascular smooth muscle cells, endothelium, and myocardium; this possibly explains the direct effect of PTH on the cardiovascular system (8, 9). Elevated PTH levels have been associated with increased blood pressure, cardiac contractility, cardiomyocyte hypertrophy, and apoptosis, as well as structural and functional changes in the vascular system (10–14). Valvular, myocardial and vascular ectopic calcifications may be considered structural effects of hyperparathyroidism (15), whereas elevated aortic pulse pressure and lessened large artery elasticity, impaired endothelial functions, and local regulation of vascular tone (vasoconstriction or vasodilation) may be considered functional effects of PTH on the cardiovascular system (14, 16, 17). Finally, PTH may also affect the cardiovascular system via proinflammatory pathways, stimulating cytokine release from inflammatory cells; thus, it is important in atherosclerosis (7, 18). Given these findings, many studies investigated PTH as a risk factor for CAD, but conflicting results were obtained (3, 19).

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We aimed to evaluate serum PTH levels and their relationship with severity and extensiveness of CAD in 655 patients referred for coronary angiography.

Methods

Study population

A total of 655 patients who underwent elective (positive cardiac stress test, ischemia in myocardial perfusion scintigraphy, patients with recently detected left ventricular wall motion abnormalities, or patients with stable angina pectoris, etc.) or emergency (acute coronary syndromes) coronary angiographies were enrolled into the our prospective and cross-sectional study. Exclusion criteria were elevated serum creatinine levels, an active malignancy, presence of primary and secondary hyperparathyroidism, acute or chronic inflammatory disease, or a known calcium hemostasis disorder and chronic renal disease. Likewise, those receiving replacement thyroid hormones, immunosuppressive treatments, or drugs affecting bone metabolism such as hormone replacement therapy, calcium, or vitamin D supplements were excluded.

The study was conducted according to the recommendations of the Declaration of Helsinki on Biomedical Research Involving Human Subjects. The Institutional Ethics Committee approved the study protocol, and each participant provided written informed consent.

Coronary angiography

Standard Judkins technique was used for coronary artery visualization, and the coronary angiograms were reviewed by two physicians blinded to the patients to assess the Gensini score. Gensini score system was used to assess the severity and extensiveness of CAD (20). This score was designed according to severity and anatomical location of stenosis. The severity of CAD was scored as 1 for 1–25% narrowing, 2 for 26–50%, 4 for 51–75%, 8 for 76–90%, 16 for 91–99%, and 32 for a completely occluded artery. The score is then multiplied by a factor as to the importance of the coronary artery as previously described. This system was chosen to determine the severity and extensiveness of CAD due to most commonly used system according the literature (21). Stenotic CAD was defined as >50% stenosis in any main coronary artery (i.e., left anterior descending, circumflex, or right coronary artery), and the number of diseased vessels was recorded.

Routine laboratory examinations

Routine hematologic and biochemical tests were performed before elective coronary angiographies, but not in emergency cases. Samples of peripheral venous blood were drawn after 12-h of fasting from the antecubital vein on admission and studied at the laboratory within 1–3 h. Laboratory parameters [including complete biochemistry panel (creatinine, uric acid, albumin, calcium, etc.), blood count, and lipid parameters] of all the participants were recorded. Siemens ADVIA Centaur® and ADVIA Centaur XP Automated Chemiluminescence® Systems were used to evaluate plasma or serum PTH levels. The accepted reference range for intact PTH is 14–72 pg/mL (1.48–7.63 pmol/L) within normal blood calcium levels in this laboratory measurement. Patients were distributed to two groups as regards the PTH level: Group 1 with PTH <72 pg/mL and Group 2 with all the others.

Statistical analysis

In all statistical analysis, SPSS 17.0 Statistical Package Program for Windows (SPSS Inc., Chicago, IL, USA) was used. The Kolmogorov–Smirnov test was used to test normality of distribution. Quantitative variables with a normal distribution were specified as the mean±standard deviation, and those with non-normal distribution were specified with median [interquartile range (IQR)]; categorical variables were specified with number and percentage values. One-way ANOVA or Kruskal–Wallis tests were used to compare continuous variables according to the Gensini score. Baseline characteristics were compared using the independent sample t-test, Mann–Whitney U test, chi-square test, multibox chi-square test, or Fisher's exact test where appropriate. The Spearman rank correlation was used to assess the correlation between PTH levels and Gensini scores. When p was <0.05, statistical significance was accepted.

Results

Baseline characteristics and laboratory parameters of the study groups were presented in Table 1.

The 655 patients were aged 61.1±11.8 years, and 215 (32.8%) were women. Group 1 comprised 568 patients, and Group 2 comprised 87. The mean PTH level was 43.4±29.5 pg/mL (range, 4.5–234 pg/mL); among women, it was 48.8±32 pg/mL, and among men, it was 40.6±27 pg/mL.

There were no significant differences between the groups in terms of HT, DM, smoking, or family history of CAD when compared using conventional cardiovascular risk factors (p>0.05). There were no statistically significant difference between groups in terms of ACS (p=0.147; Table 2).

Median Gensini score was 18.5 (4–50 IQR) in the study population. It was 19.5 (4–49 IQR) in Group 1 and 14.5 (4–53 IQR) in Group 2, without significant difference between the groups (p=0.752). Moreover, there was a significant but weak correlation between serum PTH levels and Gensini score (Spearman's Rho=0.11, p=0.003). Even though patients with ACS were excluded, there was no association between PTH level and Gensini score (p=0.403). Similar results were obtained between PTH levels and ACS subtypes (p=0.137).

Patients were divided into four groups (no-, one-, two-, or three-vessel disease) to assess the association between PTH levels and stenotic CAD (Fig. 1). There was no statistically significant difference between PTH levels and the number of stenotic vessels (p=0.147).

Table 1. Baseline characteristics of the study population

Variables	Overall (n=655)	Parathyroid hormone ≤ 72 pg/mL (n=568)	Parathyroid hormone >72 pg/mL (n=87)	*P*
Age, years, mean±SD	61.1±11.8	60.7±11.7	64.2±12.1	**0.010**
Gender, male, n (%)	440 (67.2)	391 (68.8)	49 (56.3)	**0.021**
BMI, kg/m^2	27.8 (25.8–31.0)	27.8 (25.9–31.1)	28.2 (25.6–30.9)	0.874
Hypertension, n, %	416 (63.6)	360 (63,5)	56 (64.4)	0.873
SBP, mm Hg (mean±SD)	131±21	132±27	126±21	**0.019**
DBP, mm Hg (mean±SD)	80±12	81±12	76±12	**0.001**
Diabetes, n (%)	217 (33.1)	198 (34.9)	19 (21.8)	0.016
Hyperlipidemia, n (%)	278 (42.4)	245 (43.1)	33 (37.9)	0.362
Smoking, n (%)	280 (42.7)	244 (43.0)	36 (41.4)	0.781
Family history, n (%)	130 (19.8)	109 (19.2)	21 (24.1)	0.284
FBG, mg/dL, (mean±SD)	123±63	126±65	103±42	0.003
BUN, mg/dL, (mean±SD)	16.6±5.4	16.6±5.5	16.8±5.0	0.863
Creatinine, mg/dL, (mean±SD)	1.2±5.8	1.2±6.2	0.9±0.2	0.616
HbA1c (%) (mean±SD)	7.2±1.8	7.3±1.9	6.5±1.1	0.184
TC, mg/dL, (mean±SD)	191±52	191±53	190±48	0.953
HDL, mg/dL, (mean±SD)	42±10	41±10	43±11	0.184
LDL, mg/dL, (mean±SD)	120±46	119±47	122±43	0.551
TG, mg/dL, (mean±SD)	131 (94–196)	133 (95–199)	114 (88–170)	0.096
TB, mg/dL, (mean±SD)	0.62±0.33	0.61±0.32	0.68±0.35	0.120
DB, mg/dL, (mean±SD)	0.31±0.15	0.33±0.16	0.17±0.08	0.691
AST (U/L) (median-IQR)	22.0 (18–27)	22.0 (18.0–28.0)	21.0 (17.0–26.0)	0.544
ALT (U/L) (mean±SD)	23±13	24±13	22±12	0.287
ALP (U/L) (mean±SD)	84±27	83±27	88±30	0.143
Albumin, g/dL, (median-IQR)	4.2 (4.0–4.4)	4.2 (4.0–4.4)	4.1 (3.9–4.3)	0.276
Calcium, mg/dL, (mean±SD)	9.4±0.5	9.4±0.5	9.3±0.65	0.332
Phosphor, mg/dL, (mean±SD)	3.5±0.8	3.5±0.8	3.4±0.5	0.489
Hemoglobin, g/dL, (mean±SD)	14.1±1.7	14.1±1.7	13.7±2	0.043
Platelet x 1000 K/µL (median-IQR)	229.0 (194.0–275.0)	231.0 (195.0–274)	220.0 (188–276.0)	0.513
EF, %, (mean±SD)	56±11	57±11	56±12	0.624
Gensini score, median-IQR	18.5 (4–50)	19.5 (4–49)	14.5 (4–53)	0.752
Parathyroid hormone, pg/mL, (mean±SD)	43.4±29.5	35.0±17.9	98.5±31	<0.001

*ALP - alkaline phosphatase; ALT - alanine transaminase; AST - aspartate aminotransferase; BMI - body mass index; BUN - blood urea nitrogen; DB - direct bilirubin; DBP - diastolic blood pressure; EF - ejection fraction; FBG - fasting blood glucose; HbA1c - hemoglobin A1C; HDL - high density lipoprotein cholesterol; LDL - low density lipoprotein cholesterol; SBP - systolic blood pressure; TB - total bilirubin; TC - total cholesterol; TG - triglyceride

Discussion

This study demonstrated that there is no association between PTH levels and the severity and extensiveness of CAD. On the other hand, there was a statistically significant but weak correlation between PTH levels and Gensini score. Moreover, there was no relationship between PTH levels and the number of stenotic vessels. It is well known that PTH regulates blood calcium and phosphorus concentrations. Elevated calcium is a risk factor for vascular disease, and it is associated with increased vascular stiffness and high pulse pressure (22). Previous studies demonstrated that elevated PTH levels are related to impaired vascular function, increased vascular stiffness, carotid and brachial artery intima–media thickness, and increased systolic and diastolic blood pressure (22–24). Previous studies showed that increased levels of PTH are associated with high risks for cardiovascular mortality and morbidity (1–3). It has also been suggested that elevated PTH levels may be related to nonfatal atherosclerotic CVD and development of atherosclerosis in peripheral and central large arteries (25). Moreover, elevated PTH

Table 2. The relationship between parathyroid hormone levels and acute coronary syndrome

Variable (n/%)	Parathyroid hormone ≤72 pg/mL (n=568)	Parathyroid hormone >72 pg/mL (n=87)	*P*
Normal coronary artery	53 (9.3)	7 (8)	0.147
Unstable angina pectoris	81 (14.3)	9 (10.3)	
Non-ST elevation myocardial infarction	29 (5.1)	2 (2.3)	
ST-elevation myocardial infarction	58(10.2)	4 (4.6)	
Stable angina pectoris	347 (61.1)	65 (74.7)	

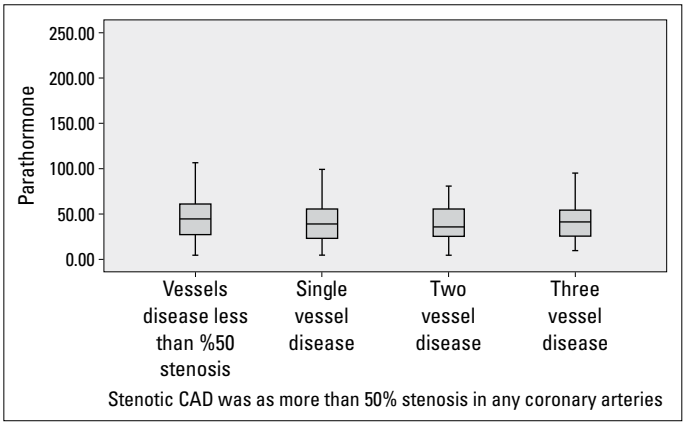

Figure 1. The relationship between parathyroid hormone and the number of stenotic vessels disease

levels have been associated with cardiac contractility, cardiomyocyte hypertrophy, and apoptosis, as well as structural and functional changes in the vascular system and low left-ventricular ejection fraction (10–14, 26). Additionally, elevated circulating levels of PTH are associated with significantly decreased functional capacity and advanced heart failure (27).

According to recent data, PTH is suggested to affect the cardiovascular system through various mechanisms (10–18). Many studies investigated the relationship between CAD and PTH, but conflicting results were obtained (19, 28, 29). Kamysheva et al. (3) suggest that CAD was associated with higher, rather than normal or lower, PTH levels. Öztürk et al. (19) investigated the relationship between serum PTH levels and severity of CAD in patients with stable angina pectoris (n=260) and also used the Gensini score to evaluate the atherosclerotic burden. They speculated that serum PTH level was not associated with the severity and extensiveness of CAD (19). We found a similar result, but our study population was more heterogeneous (with not only stable angina pectoris) and we investigated more patients (n=655). When patients with ACS (n=183) were excluded from study, our results were still found to be similar. In a recent study, Shekarkhar et al. (26) demonstrated an association between PTH levels and the number of stenotic coronary arteries. This study also showed that patients with high PTH levels were more hypertensive and had lower ejection fraction. Our results were inconsistent with the findings of this study. We did not find significant differences between PTH levels and the number of stenotic coronary arteries or ejection fraction or a correlation between PTH level and Gensini score. We also could not demonstrate an association between PTH level and ACS or its subgroups. The strict exclusion criteria for our study population may have influenced the results. Moreover, we accept that our study population and groups were not homogeneous; but when we evaluated the relationship between the groups separately, the results did not change. However, more accurate results could be obtained by evaluating patients according to their glomerular filtration rate and PTH levels.

The relationship between PTH and CVD was shown particularly in patients with chronic renal disease (27). Also, higher PTH levels may be related with the risk of sudden cardiac death in elderly populations without CVD (30). However, some of studies have claimed that elevated PTH levels are associated with increased cardiovascular mortality and morbidity in patients without renal disease (5, 6). Our study was a cross-sectional study and has no long-term mortality or morbidity data; therefore, our results cannot provide additional data on this issue.

Study limitations

This study had some important limitations. First of all, this was a cross-sectional study and PTH levels were measured only once. Exclusion of patients with chronic kidney disease, which causes secondary hyperparathyroidism, may be an important limitation, as it has been shown to be related with cardiovascular mortality.

Coronary angiography is only a lumenography and cannot detect coronary microcalcifications and cannot rule out the presence of atheromatous plaques; thus, had we used optical coherence tomography, intravascular ultrasonography, or coronary computed tomography, we might have had more satisfactory results. A coronary artery calcium scoring system in combination with computed tomography may reflect more accurate results in patients without chronic kidney disease. Another important limitation was non-homogeneous distribution and the number difference between the study groups. There were significant differences between the groups in terms of age, sex, and blood pressure status, but we thought that adjustment the study population according to these variables may cause a selection bias.

Conclusion

In conclusion, there is no association between PTH levels and the extensiveness of CAD in our study. Therefore, the relationship between PTH levels and CAD is going to be a matter of debate in the future. It is difficult to argue that PTH has no effect on atherosclerosis with the available data. Our results failed to

certify a positive or negative link between serum PTH levels and the severity and extent of CAD. We need to conduct further studies to evaluate the effect of PTH on atherosclerosis at the cellular and molecular levels.

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