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Predictors of coronary tortuosity in patients with chronic coronary syndrome

Aim	Coronary artery tortuosity is a common coronary angiographic finding. This tortuosity can cause myocardial ischemia even in the absence of significant coronary artery stenosis. Our aim was to compare the demographic, clinical and echocardiographic features of patients with chronic coronary syndrome (CCS) and with and without coronary artery tortuosity.
Material and methods	361 patients who underwent elective coronary angiography (CAG) due to CCS were included in the study. These patients divided into two groups, those with coronary tortuosity (Group 1) and those without (Group 2). Univariable and multivariable logistic regression analysis was performed to identify predictors associated with coronary artery tortuosity.
Results	The mean age of the 361 CCS patients (44% female; 56% male) was 56.7±11.5 years. In the univariable regression analysis, age, female sex, hypertension (HT), PR interval, QTc interval, ST/T segment changes, left ventricle diastolic dysfunction (LVDD), left ventricle hypertrophia (LVH) were identified as predictors of coronary tortuosity. In the multivariable regression analysis, age (OR: 1.059; 95%CI: 1.032–1.087, p<0.001) and hypertension (OR: 0.484; 95%CI: 0.278–0.843, p=0.01) were identified as independent predictors of coronary tortuosity.
Conclusion	Coronary artery tortuosity is an angiographic finding that develops as a result of adaptive mechanisms in the heart and can cause myocardial ischemia. Predictors of coronary artery tortuosity in patients with CCS were long PR and QTc intervals, ST/T segment changes, LVH, LVDD, advanced age, and female gender. Evaluation of these demographic, electrocardiographic, and echocardiographic data may help clinicans to anticipate coronary artery tortuosity in patients with CCS and to be precautious for PCI.

Keywords	Chronic coronary syndrome; coronary tortuosity; coronary angiography; clinical features, predictors
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Introduction

Coronary artery disease (CAD), characterized by accumulation of atherosclerotic plaque in the epicardial arteries, is one of the leading causes of morbidity and mortality worldwide [1]. Patients with stable angina pectoris or with angina equivalent symptoms/signs as defined in the 2019 ESC Chronic Coronary Syndrome (CCS) Guidelines are referred to as having CCS [1].

Coronary angiography (CAG) is considered the gold standard test for the diagnosis of CAD. CAG shows that approximately half of patients with evidence of myocardial ischemia, typically chest pain, have no occlusive lesion in their coronary arteries or have less than 40% stenosis of a coronary artery [2]. It is thought that coronary tortuosity alters coronary flow, and reduces perfusion pressure distal to the tortuous segment. This may subsequently lead to myocardial ischemia [3, 4]. However, the mechanisms related to how tortuosity develops in the coronary arteries have not been fully elucidated [5].

Coronary tortuosity correlates with clinical conditions such as CAD, diabetes mellitus (DM), HT, and age [6]. The relationship between atherosclerosis and coronary artery tortuosity has been investigated, and it was observed that shear stress caused by blood flow flowing through the tortuous segment accelerated atherosclerosis and increased plaque in that segment [7]. In another study, tortuous and non-tortuous coronary artery segments were compared, and no difference in atherosclerotic stenosis was detected [8]. The current study compared the demographic, clinical, and

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echocardiographic characteristics of CCS patients with and without coronary artery tortuosity.

Material and methods

Study design and inclusion-exclusion criteria

This retrospective, observational study initially included 680 patients who applied to cardiology outpatient clinics between August, 2022 and January, 2023 and who underwent CAG with the diagnosis of CCS based on non-invasive tests. A total of 361 patients >18 yrs of age and with optimal CAG images were finally included in the study. Patients <18 yrs of age, with obstructive stenosis (>50%) on CAG, with a history of coronary artery bypass graft (CABG), percutaneous transluminal coronary angioplasty (PTCA), or severe kidney and liver failure were excluded from the study. Written, informed consent was obtained from all subjects.

The Non-Interventional Ethics Committee approved the study (decision number 2022/828). The patient inclusion flowchart is shown in Figure 1.

Smoking was defined as active use according to the patient's history. Hypertension (HT), which was one of the comorbid diseases, was defined as blood pressure (BP) above 140/90 mmHg with repeated measurements or the use of oral antihypertensive drugs. Chronic renal failure (CRF) was defined as glomerular filtration rate below 60 ml/min, and hyperlipidemia (HPL) was defined as total cholesterol >200, LDL >130, or triglyceride >150 mg/dl.

Laboratory definitions

BP, measured by digital sphygmomanometer, and standard electrocardiogram (ECG) recorded at the outpatient admission were noted from hospital records. Heart rate, PR interval, QTc interval ($QT/\sqrt{RR}$), presence of bundle branch block, and ST/T segment changes were evaluated from the ECG. Body mass index was calculated as weight/height^2.

In all patients, the CAG procedure was performed with the standard Judkins technique. CAG images were evaluated on the ExtremePACS system retrospectively by two cardiologists who were blinded to the study. The left coronary arteries were observed from the right cranial and caudal positions, and the right coronary artery from the left oblique and cranial position. Obstructive CAD was defined as more than 50% stenosis in any coronary artery. Coronary artery tortuosity was defined as three or more curves with an angle >45 degrees in the trunk of any of the epicardial coronary arteries at any time during the cardiac cycle [9]. Coronary angiography images and ECGs of two patients with and without coronary tortuosity are shown in Figures 2 and 3, respectively.

Figure 1. Patient inclusion flowchart

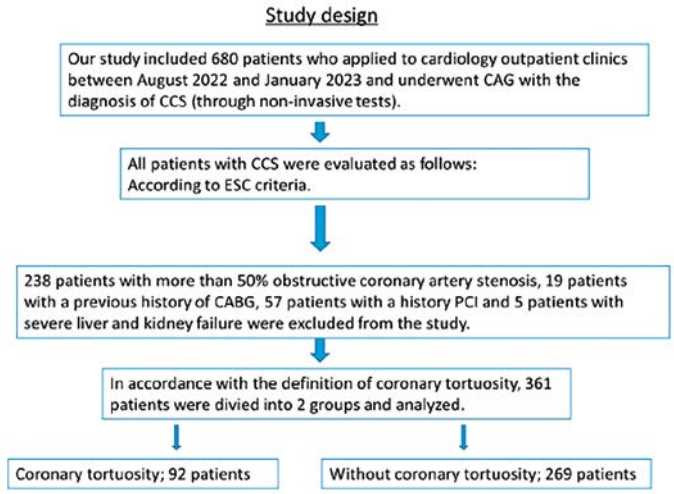


Figure 2. Demonstrative angiography and ECG image of a patient with coronary tortuosity

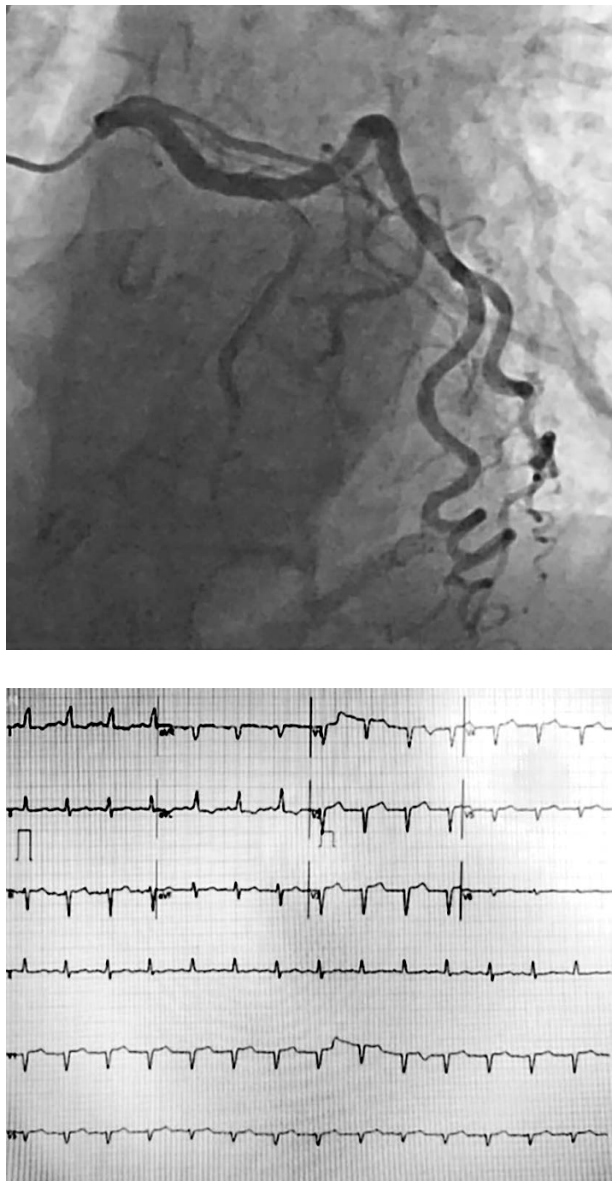
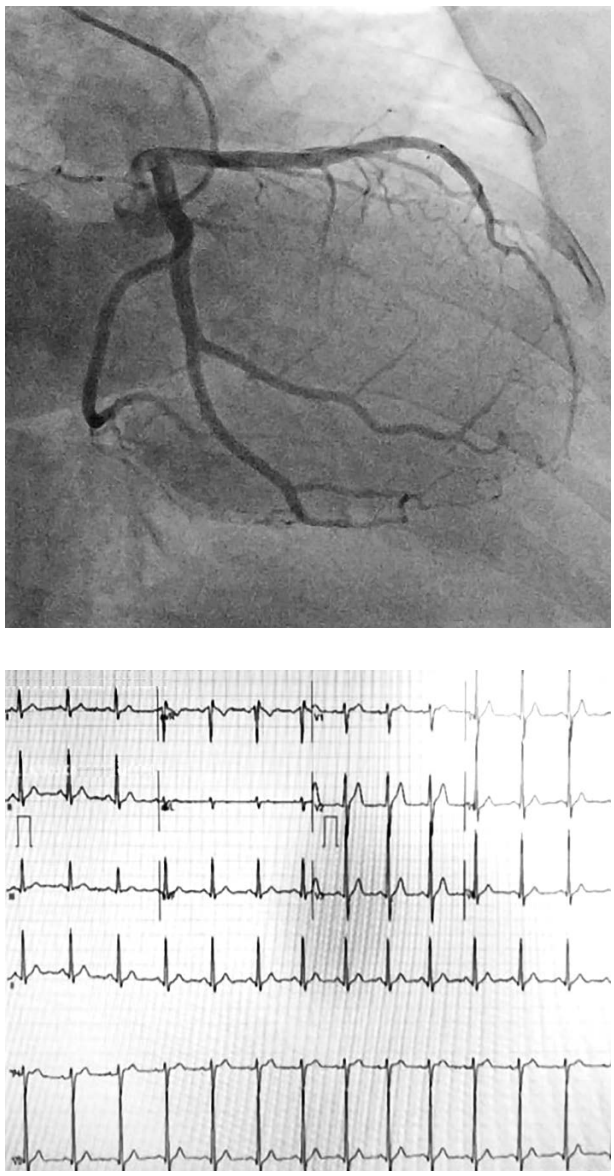


Figure 3. Demonstrative angiography and ECG image of a patient without coronary tortuosity



Demographic data, comorbid disease information, routine blood test values, and transthoracic echocardiography data were recorded from the hospital data record system. The patients divided into two groups: Group 1, with coronary tortuosity; Group 2, without coronary tortuosity.

Statistical analysis

IBM SPSS Statistics 24.0 Program SPSS (Inc., Chicago, IL, USA) was used for statistical analyses. Distribution normality analysis of continuous variables was evaluated according to Kolmogorov Smirnov and Shapiro-Wilk tests. Normally distributed, continuous variables were analyzed with Student's t test and are presented as mean and standard deviation (SD). Continuous variables that are not normally distributed are presented as median and interquartile range (IQR) (Q1-Q3) and compared with the Mann-Whitney U test. Categorical variables are reported as numbers and frequencies and were analyzed with Pearson Chi-square and Fisher's exact tests. Univariate and multivariate logistic regression analyses were performed for prediction of coronary artery tortuosity. p values < 0.05 were considered significant.

Results

The clinical, demographic, and comorbid conditions of the patients are presented in Table 1. The mean age of the 361 CCS patients (44% female) was 56.7±11.5 yrs. The mean age of the Group 1 patients was significantly greater than that of the Group 2 patients (63.1±11.5 yrs vs 54.5±10.6 yrs, p<0.001). There were more females in Group 1 than in Group 2 (54% vs 41%, p=0.02). No statistically significant differences were found between the two groups for clinical and demographic characteristics. Of the comorbid diseases, only HT was significantly higher in Group 1 than in Group 2 (45% vs 25%, p<0.001). The PR and QTc intervals were significantly longer in Group 1 than in Group 2 (152 msec vs 142 msec, p=0.007 and 419 msec vs 408 msec, p=0.003). ST/T segment changes was seen significantly more frequently in Group 1 than in Group 2 (37% vs 26%, p=0.04). LVH and LVDD were present significantly more frequently in Group 1 than in Group 2 (34% vs 22%, p=0.03 and 51% vs 32%, p=0.001). Among the left ventricular wall thickness measurements, the posterior wall thickness was significantly thicker in Group 1 than in Group 2 (11 mm vs 10 mm, p=0.03). The drugs used by the patients during the study, and the results of routine hematological and biochemical blood analyses are presented in Table 2.

Results of the univariate and multivariable regression analyses for predicting coronary tortuosity in patients with CCS are shown in Table 3. In the univariate regression analysis, age, female sex, HT, PR interval, QTc interval, ST/T segment changes, LVDD, and LVH were detected as predictors of coronary tortuosity. In the multivariable regression analysis, age, female sex, HT, PR interval, ST/T segment changes, LVDD, and LVH were found to be independent predictors of coronary tortuosity.

Discussion

The main finding of this study is, as a result of multivariate logistic regression analysis, HT and age are predictors of coronary tortuosity in patients with CCS. Although the pathophysiological mechanisms of coronary tortuosity have not been clearly explained by previous studies, coronary tortuosity is an angiographic finding encountered frequently (approximately 12–40%) to be neglected [10, 11]. While Gaibazzi et al. [12] found the prevalence of coronary tortuosity to be 27.3%, Yang et al. [13] found it to be 37.5%.

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Table 1. Demographic data, comorbidities and clinical features of the patients

Variable	Group 1 (n=92)	Group 2 (n=269)	Total (n=361)	p value
Age, yrs	63.1±11.5	54.5±10.6	56.7±11.5	<0.001
Females	50 (54)	109 (41)	159 (44)	0.02
Comorbidities				
Hypertension	41 (45)	67 (25)	108 (30)	<0.001
Diabetes mellitus	28 (30)	64 (24)	92 (25)	0.21
Hyperlipidemia	36 (39)	94 (35)	130 (36)	0.47
CRF	7 (8)	8 (3)	15 (4)	0.054
Smoking	32 (35)	69 (26)	101 (28)	0.09
CVD or TIA	6 (7)	7 (3)	13 (4)	0.08
CHF	10 (11)	27 (10)	37 (10)	0.82
Anemia	5 (5)	14 (5)	19 (5)	0.93
Systolic BP, mmHg	128 (118-134)	128 (117-135)	128 (117-135)	0.71
Diastolic BP, mmHg	72 (68-80)	70 (65-80)	72 (65-80)	0.32
BMI, kg/m^2	25.6 (23.5-27.9)	25.8 (22.4-29.1)	25.8 (22.8-29)	0.99
ECG findings				
Heart rate, beat/min	74 (69-83)	74 (67-82)	74 (68-80)	0.58
RBBB	12 (13)	22 (8)	34 (9)	0.17
LBBB	7 (8)	21 (8)	28 (8)	0.95
PR interval, ms	152 (137-166)	142 (130-160)	146 (130-162)	0.007
QTc interval, ms	419 (402-446)	408 (390-426)	410 (393-436)	0.003
ST/T segment changes	34 (37)	69 (26)	103 (29)	0.04
Echocardiographic findings				
LVEF, %	60 (54-60)	60 (55-60)	60 (55-60)	0.72
LVH	31 (34)	60 (22)	91 (25)	0.03
LVDD	47 (51)	86 (32)	133 (37)	0.001
LVEDD, mm	48 (46-52)	48 (45-51)	48 (45-51)	0.31
LVESD, mm	30 (25-36)	29 (25-33)	29 (25-33)	0.34
IVS thickness, mm	11 (10-12)	10 (9-12)	10 (10-12)	0.14
PW thickness, mm	11 (10-13)	10 (9-12)	10 (9-12)	0.03

Data are number (percentage), mean±SD, or median and IQR (Q1-Q3). Group 1, coronary tortuosity (+); Group 2, coronary tortuosity (-). n, number of patients; SD, standard deviation; CRF, chronic renal failure; CVD, cerebrovascular disease; TIA, transient ischemic attack; CHF, chronic heart failure; BMI, body mass index; ECG, electrocardiogram; RBBB, right bundle branch block; LBBB, left bundle branch block; LVEF, left ventricle ejection fraction; LVH, left ventricle hypertrophy; LVDD, left ventricle diastolic dysfunction; LVEDD, left ventricle end diastolic diameter; LVESD, left ventricle end systolic diameter; IVS, interventricular septum; PW, posterior wall.

Table 2. Hematological-biochemical laboratory values of the patients and their medications

Variable	Group 1 (n=92)	Group 2 (n=269)	Total (n=361)	p value
Prescribed drugs				
Beta-blockers	41 (45)	100 (37)	141 (39)	0.21
ACE inhibitors	28 (30)	87 (32)	115 (32)	0.74
AT receptor blockers	17 (18)	41 (15)	58 (16)	0.47
Non-dhp CCBs	6 (7)	16 (6)	22 (6)	0.84
Dhp CCBs	20 (22)	44 (16)	64 (18)	0.24
Oral anticoagulants	10 (11)	24 (9)	34 (9)	0.58
Statins	31 (34)	99 (37)	130 (36)	0.59
Antithrombotic agents	52 (57)	127 (47)	179 (50)	0.12
Furosemide	9 (10)	13 (5)	22 (6)	0.09
Digoxin	2 (2)	3 (1)	5 (1)	0.38
Aldactone	7 (8)	11 (4)	18 (5)	0.18
Oral antidiabetics	29 (32)	63 (23)	92 (25)	0.12
Insulin's	11 (12)	20 (7)	31 (9)	0.18
Laboratory findings				
WBCs, ×10^3/l	7.8 (6.6-9.0)	8 (7.0-9.1)	7.9 (6.9-9.1)	0.25
Neutrophils, ×10^3/l	4.6 (3.9-5.8)	4.7 (3.7-5.9)	4.6 (3.7-5.9)	0.87
Lymphocytes ×10^3 /l	2.1 (1.7-2.7)	2.2 (1.7-2.7)	2.1 (1.7-2.7)	0.43
Monocytes, ×10^3 /l	0.5 (0.4-0.8)	0.5 (0.4-0.8)	0.5 (0.4-0.8)	0.38
Hemoglobin, gr/dl	13.5 (12.7-14.4)	13.9 (12.7-15.0)	13.8 (12.7-14.9)	0.06
Hematocrit, %	41.2 (38.6-44.3)	42.7 (38.8-46.5)	42.3 (38.7-46)	0.06
MPV, fl	8.9 (8.1-9.5)	8.6 (8.1-9.4)	8.7 (8.1-9.5)	0.25
Platelet count, ×10^3/l	261 (219-295)	263 (219-314)	263 (218-309)	0.19
Glucose, mg/dl	104 (92-115)	103 (97-112)	104 (96-112)	0.79
Urea, mg/dl	30 (24-39)	31 (26-37)	31 (25-38)	0.98
Creatinine, mg/dl	0.86 (0.75-0.98)	0.84 (0.73-0.96)	0.84 (0.73-0.96)	0.19
Albumin, mg/dl	3.7 [0.5]	3.6 [1.0]	3.6 [0.9]	0.30
Total protein, mg/dl	6.9 (6.4-7.3)	6.7 (6.1-7.4)	6.8 (6.1-7.4)	0.23
Total cholesterol, mg/dl	190 (159-229)	186 (160-210)	186 (160-211)	0.37
Triglyceride, mg/dl	133 (104-185)	144 (105-214)	140 (105-202)	0.17
High density lipoprotein cholesterol, mg/dl	45.8 [10.1]	43 [12.4]	43.7 [11.9]	0.05
Low density lipoprotein cholesterol, mg/dl	112 (92-141)	107 (89-132)	108 (90-133)	0.39

Data are number (percentage). Group 1, coronary tortuosity (+); Group 2, coronary tortuosity (-). n, number of patients; ACE, angiotensin converting enzyme; AT, angiotensin; Dhp CCB, dihydropyridine calcium channel blocker; WBC, white blood cell; MPV, mean platelet volume; TSH, thyroid stimulation hormone.

Table 3. Results of univariate and multivariate logistic regression analysis for predicting coronary tortuosity in patients with CCS

Variable	Univariate Logistic Regression			Multivariate Logistic Regression		
	OR	95% CI	p	OR	95% CI	p
Age	1.076	1.050-1.102	<0.001	1.059	1.032-1.087	<0.001
Female sex	1.747	1.085-2.816	0.02	1.637	0.953-2.811	0.07
Hypertension	0.413	0.251-0.677	<0.001	0.484	0.278-0.843	0.01
PR interval	1.012	1.002-1.022	0.02	1.010	0.999-1.021	0.07
QTc interval	1.006	1.000-1.012	0.07		–	
ST/T segment changes	0.589	0.356-0.974	0.04	0.790	0.447-1.397	0.42
LVDD	0.450	0.278-0.729	0.001	0.610	0.347-1.071	0.09
LVH	0.565	0.336-0.949	0.03	0.679	0.374-1.236	0.20

LVDD, left ventricle diastolic dysfunction; LVH, left ventricle hypertrophy; OR, odds ratio; CI, confidence interval.

The current study found a 25% rate of coronary tortuosity, in agreement with the literature.

However, HT is one of the most common causes of tortuosity due its mechanical effect on the arterial wall. Kamiya and Togawa [14] showed that an adaptive process developed against parietal stress (nearly 10–20 dynes/m^2) to detect rapid changes in arterial dimensions. As a result, an transverse and longitudinal increase in arterial dimensions and an increase tortuosity were observed. At the microscopic level, it was shown that endothelial and smooth muscle cells proliferated and the elastic layer degenerated [15].

The report of a small-scale study [16] mentioned a positive correlation between HT and coronary tortuosity. The patients were divided into three groups, angina, LVH, and neither, and prospectively examined to compare the results of exercise ECG, CAG, and ventriculography. Coronary tortuosity was observed to a large extent (about 84%) in the group with LVH. In 62 patients, Jakob et al. [17] examined the results of chronic pressure and volume overload of the coronary arteries and found that HT, female gender, and age was predictor of coronary tortuosity. Eleid et al. [18] examined the angiographic features and clinical effects of coronary tortuosity in spontaneous coronary artery dissection. Age and HT were found to be significantly higher in the group with coronary tortuosity.

In a previous study that examined the relationship between coronary tortuosity and left ventricular mass index (LVMI), patients in the coronary tortuosity group were older and had a higher rate of HT and DM [19]. If we consider that LVMI is calculated from left ventricular wall thickness, cavity diameters, and body surface area, we can interpret that the group with high LVMI has more wall thickness due to high blood pressure. Coronary tortuosity can be considered a result of adaptive mechanisms in the morphological structure and functional characteristics of the heart. Through this adaptive mechanism, which is the shear stress effect created by high blood pressure on the arterial wall, the morphological structure and functional characteristics of the heart change, and coronary tortuosity occurs. This coronary tortuosity phenomenon is more common in the elderly population with diastolic dysfunction that accompanies left ventricular hypertrophy [20]. In our study, left ventricular hypertrophy and diastolic dysfunction were more common in the group with coronary tortuosity.

Gaibazzi et al. [12] found more ST segment changes in patients with myocardial bridge and coronary tortuosity in a study of 400 patients who underwent CAG for chest pain. In that study, ST/T segment changes, which are thought to be mostly due to left ventricular hypertrophy, were observed more frequently in the group with coronary tortuosity. The reason why the PR interval was longer, another ECG finding, in the group with coronary tortuosity may be age-related AV nodal degeneration [21]. The QTc interval was aimed to investigate the relationship between coronary tortuosity and ECG findings, but no correlation was found between QTc prolongation and coronary tortuosity. Only a incidental increase in QTc interval was observed, if this value is carefully examined, as we could not obtain the prolonged QTc over 450 milliseconds.

Limitations

This study has some limitations. It was a single-center, retrospective study. In addition, the degree of coronary artery tortuosity and atherosclerotic lesions were not evaluated with intravascular ultrasound (IVUS) or optical coherence thomography (OCT), but were only visually interpreted.

Conclusions

Coronary artery tortuosity is an angiographic finding that develops as a result of adaptive mechanisms in the heart and can cause myocardial ischemia. Predictors of coronary artery tortuosity in patients with chronic coronary syndrome: long PR and QTc intervals, ST/T segment changes, LVH, LVDD, advanced age, and female gender. Evaluation of these demographic, electrocardiographic, and echocardiographic data may help clinicians anticipate coronary artery tortuosity in patients with CCS and to be precautious for PCI.

No conflict of interest is reported.

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