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Original article

Association of prediabetes with diffuse coronary narrowing and small-vessel disease

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ABSTRACT

Background: A significant number of patients may not benefit from conventional techniques of myocardial revascularization due to diffuse coronary artery disease (CAD) or small coronary arterial sizes because of smaller arteries causing anastomotic technical difficulties and poor run-off. Diabetic patients have a more severe and diffuse coronary atherosclerosis with smaller coronary arteries limiting the possibility to perform a successful and complete revascularization, but this has not been examined in prediabetics.
Objective: To evaluate whether there is an association between prediabetes and the coronary arterial size.
Methods: We prospectively studied 168 consecutive patients with CAD and 172 patients with normal coronary artery anatomy (NCA). Patients were divided into three groups according to hemoglobin (Hb) A1c levels as "normal," "prediabetic," and "diabetic" groups, and the coronary artery sizes and Gensini scores were analyzed.
Results: There were 78 female patients and 90 male patients in the CAD group, and 87 female patients and 85 male patients in the NCA group. There was a statistically significant difference in distal and proximal total coronary arterial size among the CAD and NCA groups for both genders. There was a positive correlation between the HbA1c subgroups and Gensini score (Spearman's ρ: 0.489, $p < 0.001$ in female group; Spearman's ρ: 0.252 $p = 0.016$ in male group).
Conclusion: We found that prediabetic patients have a smaller coronary size and diffuse coronary narrowing for both genders, particularly in distal coronary arterial tree of left anterior descending coronary artery. The early detection of prediabetes in daily cardiology practice may provide more appropriate coronary lesion for percutaneous or surgical revascularization.

Introduction

Cardiovascular diseases are the leading cause of morbidity and mortality in insulin-resistant individuals with glycemic disorders. The risk for death among people with diabetes is about twice that of people of similar age but without diabetes [1,2]. However, epidemiologic evidence suggests this morbidity–mortality relationship begins early in the progression from normal glucose tolerance to overt diabetes. Since both increased insulin resistance and impaired β-cell function are present long before overt hyperglycemia becomes evident, indeed, most diabetic patients already show signs of cardiovascular disease upon diagnosis [1,2]. Overt diabetes is usually preceded by a condition known as "prediabetes," which refers to an intermediate stage in which individuals have blood glucose or glycated hemoglobin (hemoglobin A1c, HbA1c) levels higher than normal but not high enough to be classified as diabetes [2]. Subjects with prediabetes have an increased risk for future overt diabetes with a conversion rate of ~5–10% per year [3,4]. This risk increase has found to be a continuum and begins at a level below the cut point for impaired fasting glucose (IFG, 100 mg/dl) [5]. The prevalence of diabetes and prediabetes is substantial among adults with coronary artery disease (CAD) and likely underestimated because of suboptimal screening [6,7]. In 2010, the American Diabetes Association (ADA) proposed a "diabetes" diagnosis based on an HbA1c ≥ 6.5%, and 'prediabetes' [including IFG or impaired glucose tolerance (IGT)] as an HbA1c 5.7–6.4% [2].

Diabetic patients have a more severe, extensive-diffuse and rapidly progressive form of coronary atherosclerosis with an unfavorable angiographic anatomy limiting the possibility to perform

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a successful and complete revascularization [3,8–10]. Compared to those without diabetes, diabetics have had worse outcomes with percutaneous transluminal coronary angioplasty, bare-metal stents, and drug-eluting stents. The reasons for this trend are because patients with diabetes have smaller-caliber vessels, a diffuse disease that often progresses rapidly, a greater burden of atherosclerotic disease, and exaggerated neointimal hyperplasia. Patients with small vessels present a higher risk for an adverse outcome after percutaneous coronary intervention (PCI) [10,11], because of a higher incidence of restenosis and an increased risk of major adverse cardiac events [12,13] and a higher risk after coronary artery bypass grafting (CABG) due to more technically challenging operative procedures and lower long-term patency rates [14–17] due to difficulties in anastomoses between saphenous vein grafts or internal mammary conduits to small caliber native coronary arteries particularly in diabetics [18] or women [19–21]. In the Coronary Artery Surgery Study, small body size and coronary artery caliber were the strongest predictors of perioperative mortality [22].

In general, CAD in diabetic patients is detected at an advanced stage, whereas the disease in its premature (prediabetes) or asymptomatic stages (undiagnosed diabetics) remains unfortunately undetected [23,24]. Such observations impose an aggressive approach to diagnostic strategies in diabetic patients to detect CAD at an early asymptomatic stage, which is probably characterized by a more favorable coronary vessel anatomy. Despite the remarkable advances in revascularization strategies made during the past decade, a significant proportion of patients are excluded from either PCI or CABG because of unsuitable or ungraftable coronary anatomy including diffuse severe CAD, extremely calcified vessels, chronic total occlusions, or small vessel caliber. In diabetics, the coronary arteries and their branches have been shown to have smaller diameters than normal subjects [25,26], but also appear to be narrower in prediabetic patient in daily cardiology practice. We aimed to evaluate whether there was an association between prediabetes and diffuse coronary narrowing and/or coronary artery sizes.

Methods

We planned to compare the coronary artery sizes separately in patients with normal coronary artery (NCA) findings in all 3 major epicardial and the left main coronary arteries, and patients with CAD. We prospectively studied 172 consecutive patients with NCA anatomy, and 168 consecutive CAD patients referred for elective coronary angiography. Those with known valvular heart disease, congenital heart disease, chronic kidney disease, anemia, or hemoglobinopathies were excluded from the study [27–29]. Patients were classified according to HbA1c levels in accordance with 2010 ADA Guidelines [2] into three groups: HbA1c lower than 5.7% (normal control group), HbA1c: 5.7–6.4% (prediabetic group), and HbA1c higher than 6.4% (diabetic group), and the coronary artery sizes and Gensini scores were analyzed. All measurements were performed by the same cardiology specialist blinded to the subjects' clinical and laboratory status.

Fasting blood specimens were collected to measure fasting plasma glucose (FPG), lipid profile, creatinine, and HbA1c levels. Other risk factors for cardiovascular disease and demographic parameters (age, gender, and body mass index) at the moment of enrollment in the study were evaluated by history-taking and physical examination results.

Selective coronary angiography was performed with the standard Judkins approach. Significant CAD was defined as the presence of >50% luminal diameter narrowing of one or more major epicardial arteries or its major branches. Segments of each epicardial coronary artery were measured in locations defined by Dodge et al. [30] and Mosseri et al. [25] as follows:

(a) The left main artery (LM) was measured at its midpoint,
(b) the left anterior descending artery (LAD) was divided into three segments, the proximal LAD (pLAD) was measured at its midpoint between its origin and the first branch (first septal-1S or diagonal-1D) of the pLAD, the mid-LAD (mLAD) was measured between 1S and 1D, and the distal LAD (dLAD) was measured after the diagonal branch of the LAD, apical LAD was measured in its distal 1.0 cm before the distal bifurcation, commonly referred to as the "pitchfork," "moustache," or "whale's tail",
(c) the circumflex (Cx) was also divided into two segments, the proximal Cx (pCx) was measured at its midpoint between its origin and the first obtuse marginal (1 M), the distal Cx (dCx) was measured at the origin of the second obtuse marginal branch (2 M); and finally the 1 M was measured at its origin,
(d) the right coronary artery (RCA) was divided into two segments: the proximal RCA (pRCA) was measured 15 mm from the ostium and the distal RCA (dRCA) was measured at the ostium of the posterior descending artery (PDA). In the CAD group, measurements of artery size were done on the most proximal disease-free part of each segment. In this respect, totally occluded segments were not evaluated in statistical analysis. Intracoronary nitrate was not administered to patients before the coronary angiography. Quantitative coronary angiographic analysis of all three coronary arteries was performed using the edge-detection method. The diameter of the catheter after contrast filling was used as a reference for calculating true arterial diameters. Measurements were taken in two orthogonal views for each of the major epicardial coronary arteries. The average of the two measurements was used for each coronary artery.

The sum of the pLAD, pCx, and pRCA was calculated and defined as total proximal coronary diameter (pTCD). The sum of diameters of the distal segments including dLAD, dCx, and dRCA was calculated and defined as total distal coronary diameter (dTCD).

The SPSS statistical software package (version 16.0; SPSS Inc., Chicago, IL, USA) was used to perform all statistical calculations. Continuous variables were expressed as mean ± SD. Since the coronary diameters in men are greater than women [31], all comparisons were made separately for both genders. The analysis of variance (ANOVA) with post hoc Tukey's HSD or Chi-square test was used for the statistical analysis of the results. Relationships between the continuous variables were evaluated by Pearson's correlation analysis when data were normally distributed or by Spearman's correlation analysis when they were not normally distributed. For all tests, a value of $p < 0.05$ was considered significant.

Results

There were 78 female patients and 90 male patients in the CAD group (Table 1) and 87 female patients and 85 male patients in the NCA group (Table 2). There were no statistically significant differences at baseline in any demographic or baseline variables between the groups (Tables 1 and 2). There were statistically significant differences in proximal and distal total coronary arterial sizes between the CAD (Table 1) and NCA (Table 2) groups for both genders particularly in LAD. There was a positive correlation between the patient subgroups (diabetic, prediabetic, and normal groups) and Gensini score (Spearman's ρ: 0.489, $p < 0.001$ in the female group; Spearman's ρ: 0.252, $p = 0.016$ in the male group).

Table 1
Comparison of the coronary sizes among patients with coronary artery disease. BMI, body mass index; LDL, low density lipoprotein; LMCA, left mean coronary artery; Prox-LAD, proximal left anterior descending artery; OM1, obtus marginal; RCA, right coronary artery; PDA, posterior descending artery; TCD, total coronary diameter.

Groups	Diabetes (HbA1c > 6.4)	Prediabetes (HbA1c: 5.7–6.4)	Normal (HbA1c < 5.7)	*p* value
Female	(*n* = 32)	(*n* = 22)	(*n* = 24)	
Age (year)	60.8 ± 7.3	57.5 ± 8.4	57.5 ± 8.0	0.190
BMI (kg/m^2)	32.8 ± 5.1	29.7 ± 3.6	31.6 ± 5.1	0.145
Family history (%)	34%	59%	58%	0.058
Hypertension (%)	75%	50%	62%	0.167
Current smoke (%)	19%	14%	37%	0.119
Creatinine (mg/dl)	0.86 ± 0.18	0.77 ± 0.09	0.81 ± 0.11	0.118
LDL cholesterol (mg/dl)	153.4 ± 39.7	158.0 ± 37.9	145.6 ± 30.4	0.844
Angiographic findings				
LMCA (mm)	3.94 ± 0.64	4.29 ± 0.63	4.38 ± 0.63	0.028
Prox-LAD (mm)	2.96 ± 0.34	3.26 ± 0.35	3.64 ± 0.46	<0.001
Mid-LAD (mm)	2.39 ± 0.46	2.70 ± 0.33	3.13 ± 0.59	<0.001
Distal-LAD (mm)	1.98 ± 0.40	2.18 ± 0.35	2.60 ± 0.55	<0.001
Apex (mm)	1.15 ± 0.44	1.20 ± 0.34	1.83 ± 0.42	<0.001
Prox-Cx (mm)	2.62 ± 0.60	3.00 ± 0.58	3.27 ± 0.65	0.007
Distal-Cx (mm)	2.19 ± 0.57	2.41 ± 0.56	2.52 ± 0.72	0.232
OM1-Cx (mm)	1.59 ± 0.51	1.82 ± 0.51	1.94 ± 0.59	0.022
Prox-RCA (mm)	3.08 ± 0.53	3.29 ± 0.57	3.68 ± 0.82	0.160
Mid-RCA (mm)	2.60 ± 0.50	2.85 ± 0.63	3.39 ± 0.80	0.012
PDA-RCA (mm)	1.61 ± 0.29	1.96 ± 0.58	2.08 ± 0.53	0.011
Proximal TCD (mm)	8.67 ± 1.20	9.54 ± 0.95	10.08 ± 1.32	<0.001
Distal TCD (mm)	5.75 ± 0.88	6.55 ± 0.48	6.95 ± 0.98	<0.001
Gensini score	62.5 ± 50.4	45.0 ± 29.8	33.1 ± 30.0	<0.001
Male	(*n* = 34)	(*n* = 31)	(*n* = 25)	
Age (year)	60.4 ± 7.3	55.7 ± 7.7	58.1 ± 9.3	0.070
BMI (kg/m^2)	31.6 ± 4.8	31.2 ± 3.8	29.2 ± 3.4	0.075
Family history (%)	26%	19%	40%	0.225
Hypertension (%)	47%	23%	40%	0.114
Current smoke (%)	62%	65%	56%	0.806
Creatinine (mg/dl)	0.88 ± 0.18	0.82 ± 0.16	0.94 ± 0.09	0.178
LDL cholesterol (mg/dl)	136.5 ± 42.3	140.8 ± 26.6	145.6 ± 30.4	0.649
Angiographic findings				
LMCA (mm)	4.00 ± 0.66	4.27 ± 1.09	4.55 ± 0.77	0.055
Prox-LAD (mm)	3.01 ± 0.58	3.23 ± 0.83	3.70 ± 0.55	0.001
Mid-LAD (mm)	2.59 ± 0.51	2.75 ± 0.65	3.15 ± 0.59	0.002
Distal-LAD (mm)	2.06 ± 0.34	2.24 ± 0.56	2.60 ± 0.55	<0.001
Apex (mm)	1.25 ± 0.47	1.45 ± 0.45	1.83 ± 0.42	<0.001
Prox-Cx (mm)	2.78 ± 0.54	3.02 ± 0.96	3.27 ± 0.65	0.046
Distal-Cx (mm)	2.28 ± 0.45	2.46 ± 0.76	2.52 ± 0.72	0.325
OM1-Cx (mm)	1.75 ± 0.53	1.91 ± 0.72	1.94 ± 0.59	0.442
Prox-RCA (mm)	3.19 ± 0.72	3.44 ± 0.73	3.68 ± 0.82	0.053
Mid-RCA (mm)	2.81 ± 0.75	2.85 ± 0.63	3.39 ± 0.80	0.006
PDA-RCA (mm)	1.65 ± 0.46	1.73 ± 0.42	2.08 ± 0.53	0.002
Proximal TCD (mm)	8.98 ± 1.30	9.69 ± 1.82	10.65 ± 1.67	0.047
Distal TCD (mm)	5.99 ± 0.75	6.42 ± 1.10	7.20 ± 1.41	0.025
Gensini score	51.4 ± 33.3	43.0 ± 32.0	33.1 ± 20.2	0.040

Discussion

In the present study, prediabetes was found to be associated with diffuse coronary narrowing and small vessel disease particularly in distal coronary arteries for both genders, particularly in the LAD (Tables 1 and 2). Since patients with small vessels present a higher risk for an adverse outcome after PCI [10,11] or CABG especially in the LAD coronary artery [14–17], this finding is particularly important for early detection of prediabetes in daily cardiology practice.

The role of cardiologists in the management of patients with diabetes is evolving owing to the close relationship between diabetes and CAD. Indeed, a large percentage of patients with diabetes present with a first coronary event before their diabetes are diagnosed. Therefore, because the cardiologist often may be the first clinician to diagnose a patient with diabetes, it is incumbent upon practitioners of this specialty to understand the disease process and the interventions necessary to improve outcomes [32]. The worldwide increase in the incidence of diabetes and prediabetes as a result of global dietary changes and reduced physical activity should be of special concern to cardiologists and patient management necessitates transprofessional collaboration between cardiologists and diabetologists to be successfully accomplished [33]. The early detection of prediabetes may also provide more appropriate coronary lesions for percutaneous or surgical revascularization and identifies people at highest risk of developing overt diabetes and CAD [34].

Traditionally, diagnosis of diabetes was based on symptoms due to hyperglycemia, but during the past decades much emphasis has been placed on the need to identify diabetes and other forms of glucose abnormalities such as prediabetes in asymptomatic subjects [3,4]. All things considered, it is clear that that prediabetes is not a benign condition [35–42]. In addition to the risk of progression to overt diabetes, prediabetes has been reported to increase the risk for certain microvascular and macrovascular complications that are typically associated with diabetes [3,35,36]. Evidence suggests that most diabetic patients have the condition for between 9 and 12 years before the diagnosis [37] and about 50% of patients already have diabetic tissue damage such as retinopathy, heart disease, or microalbuminuria at the time they are diagnosed [38]. The risk for

Table 2
Comparison of the coronary sizes among patients with angiographically normal coronary arteries. BMI, body mass index; LDL, low density lipoprotein; LMCA, left mean coronary artery; Prox-LAD, proximal left anterior descending artery; OM1, obtus marginal; RCA, right coronary artery; PDA, posterior descending artery; TCD, Total coronary diameter.

Groups	Diabetes (HbA1c > 6.4)	Prediabetes (HbA1c:5.7–6.4)	Normal (HbA1c < 5.7)	*p* value
Female	(*n* = 31)	(*n* = 26)	(*n* = 30)	
Age (year)	58.4 ± 8.3	58.1 ± 8.1	56.1 ± 7.9	0.486
BMI (kg/m^2)	34.2 ± 5.2	32.4 ± 4.7	32.9 ± 7.8	0.489
Family history (%)	13%	31%	17%	0.211
Hypertension (%)	52%	50%	33%	0.292
Current smoke (%)	7%	23%	13%	0.193
Creatinine (mg/dl)	0.73 ± 0.16	0.67 ± 0.13	0.68 ± 0.13	0.194
LDL cholesterol (mg/dl)	132.4 ± 30.6	148.5 ± 33.9	141.2 ± 28.3	0.194
Angiographic findings				
LMCA (mm)	3.91 ± 0.66	3.90 ± 0.61	4.26 ± 0.94	0.127
Prox-LAD (mm)	3.14 ± 0.48	3.29 ± 0.57	3.61 ± 0.75	0.012
Mid-LAD (mm)	2.64 ± 0.62	2.65 ± 0.61	3.00 ± 0.61	0.039
Distal-LAD (mm)	1.99 ± 0.58	2.14 ± 0.50	2.43 ± 0.47	0.006
Apex (mm)	1.14 ± 0.48	1.11 ± 0.37	1.36 ± 0.32	0.031
Prox-Cx (mm)	2.83 ± 0.55	3.03 ± 0.54	3.25 ± 0.74	0.039
Distal-Cx (mm)	2.29 ± 0.46	2.50 ± 0.51	2.60 ± 0.68	0.102
OM1-Cx (mm)	1.71 ± 0.52	1.57 ± 0.43	1.79 ± 0.71	0.348
Prox-RCA (mm)	3.31 ± 0.62	3.52 ± 0.87	3.56 ± 0.44	0.303
Mid-RCA (mm)	2.91 ± 0.49	3.22 ± 0.49	3.16 ± 0.79	0.125
PDA-RCA (mm)	1.62 ± 0.46	1.99 ± 0.38	1.99 ± 0.59	0.004
Proximal TCD (mm)	9.28 ± 1.32	9.88 ± 1.15	10.38 ± 2.04	0.028
Distal TCD (mm)	5.90 ± 1.17	6.64 ± 0.91	7.01 ± 1.28	0.001
Male	(*n* = 28)	(*n* = 28)	(*n* = 29)	
Age (year)	53.5 ± 6.3	54.4 ± 8.5	54.8 ± 9.4	0.824
BMI (kg/m^2)	32.1 ± 5.9	30.6 ± 2.3	29.7 ± 3.8	0.081
Family history (%)	36%	18%	41%	0.140
Hypertension (%)	43%	18%	38%	0.108
Current smoke (%)	46%	36%	48%	0.589
Creatinine (mg/dl)	0.89 ± 0.17	0.93 ± 0.14	0.84 ± 0.15	0.101
LDL cholesterol (mg/dl)	140.4 ± 47.3	126.8 ± 35.7	133.2 ± 37.7	0.471
Angiographic findings				
LMCA (mm)	4.03 ± 0.89	4.36 ± 1.06	4.60 ± 0.89	0.081
Prox-LAD (mm)	3.34 ± 0.60	3.56 ± 0.61	3.84 ± 0.80	0.025
Mid-LAD (mm)	2.69 ± 0.64	3.06 ± 0.58	3.31 ± 0.78	0.003
Distal-LAD (mm)	2.19 ± 0.56	2.43 ± 0.50	2.73 ± 0.69	0.004
Apex (mm)	1.36 ± 0.47	1.34 ± 0.35	1.67 ± 0.51	0.012
Prox-Cx (mm)	3.10 ± 0.58	3.31 ± 0.70	3.51 ± 0.69	0.066
OM1-Cx (mm)	1.82 ± 0.48	1.87 ± 0.64	2.07 ± 0.80	0.324
Distal-Cx (mm)	2.57 ± 0.62	2.42 ± 0.61	2.96 ± 0.76	0.008
Prox-RCA (mm)	3.68 ± 0.81	3.80 ± 0.70	3.82 ± 1.05	0.800
Mid-RCA (mm)	3.30 ± 0.81	3.34 ± 0.78	3.46 ± 1.11	0.798
PDA-RCA (mm)	2.01 ± 0.50	2.16 ± 0.52	2.10 ± 0.77	0.651
Proximal TCD (mm)	10.12 ± 1.46	10.67 ± 1.61	11.18 ± 1.85	0.059
Distal TCD (mm)	6.77 ± 1.22	7.00 ± 1.11	7.79 ± 1.36	0.007

diabetic retinopathy has been found to be highest at an HbA_{1c} level of 6.0–6.4% for whites and 5.5–5.9% for blacks [39]. Data from the Nurses' Health Study [40] suggest that women destined to develop diabetes experienced a 3-fold increased risk of cardiovascular disease 10–15 years before the onset of diabetes, corroborating the hypothesis that the "clock starts ticking" very early [41–43].

All these studies showing increased risks for glycemic progression and microvascular and macrovascular complications in prediabetes strengthen the rationale for early intervention and detection of diabetes in its premature stage. There is a need to simplify screening tests for glycemic disorders so patients can be identified earlier and more efficiently. Growing evidence strongly suggests that the assessment of HbA1c levels has advantages over measurement of glucose levels or oral glucose tolerance test (OGTT) in predicting the risk of developing diabetes or CAD [3,44]. The HbA1c result reflects longer term glycemia and is less affected by recent physical/emotional stress. Appointments do not need to be limited to the morning also. However, there is some debate as to whether HbA1c should replace FPG or the OGTT [3,45,46]. As the two tests detect different people, some individuals with diabetes detected on OGTT will no longer be classified as having type 2 diabetes using HbA1c ≥6.5% criteria. Kumaravel et al. reported that current ADA definitions of prediabetes based on HbA1c would fail to detect almost 40% of people currently classified as IFG [45]. More recent studies have shown that HbA1c may be insensitive in diagnosing IFG and IGT, with sensitivities ranging from 27% to 47% [46]. Furthermore, some medical conditions can result in HbA1c assay measurements not reflecting glycemic control over the previous 2–3 months; these include hematological disorders, renal failure, and chronic excess alcohol consumption. Although there is still some debate, HbA1c has been recommended in 2010 by the ADA as a diagnostic tool for detecting type diabetes and prediabetes [2]. Efficacy of the new HbA1c 5.7–6.4% criterion will improve as a screening test and will be more predictive when used with IFG criterion for identification of individuals at substantially increased risk of developing diabetes [2]. Using both HbA1c and IFG for screening together could increase diagnostic accuracy [2,47].

Previous studies have reported that female gender is associated with worse clinical and revascularization outcomes following coronary revascularization compared to men [19,31,32]. The observed less favorable clinical outcomes in women may be due to smaller vessel size in women. On average, women have proximal coronary

arteries that were 0.30 mm smaller than men in previous studies [32,48]. The smaller coronary artery vessel size in women may explain some, but not all excess gender-related risk with coronary artery revascularization [27,29].

Several limitations should be considered. Firstly, this study is limited by its cross-sectional nature of patients referred for coronary angiography and its results may not be generalizable to the population as a whole. Secondly, the effect of factors that might change HbA1c levels independently of glycemia, such as anemia, or hemoglobinopathies, should be considered. A third possible limitation was the lack of intravascular ultrasound usage. It must always be remembered that a coronary angiogram is a "luminogram" and cannot be used to assess changes in wall thickness, a cardinal feature of atherosclerosis. With the use of traditional coronary angiography, diffuse atherosclerosis and positive remodeling that may not be apparent with lumen-limited angiography may preclude accurate measurement of the true vessel wall dimensions, because the normal size of the coronary artery for its distal myocardial bed size is not known and cannot be measured directly with diffuse involvement of the artery, therefore, intravascular ultrasound could be a more effective method [49]. The other main limitation of the study included the inability to examine the effect of the duration of a specific HbA_{1c} level. Again, we only included patients with completely normal creatinine levels. The higher creatinine levels in diabetics could have been affected by the coronary artery sizes.

In conclusion, we found that the prediabetes was associated with diffuse coronary narrowing and small vessel disease. Since small vessel disease has a higher risk for an adverse outcome after PCI because of a higher incidence of restenosis and after CABG because of smaller arteries causing anastomotic technical difficulties and poor run-off, this finding is particularly important for early detection of prediabetes in daily cardiology practice, which may provide more appropriate coronary lesions for percutaneous or surgical revascularization. Cardiologists must be aware of these trends and understand the influence of prediabetes on CAD.

Acknowledgment

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