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Impaired fasting glucose and the prevalence and severity of angiographic coronary artery disease in high-risk Chinese patients

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Abstract

We assessed the relation between different fasting plasma glucose (FPG) levels of 5.6 to 6.9 mmol/L and the prevalence and severity of angiographic coronary artery disease (CAD) in high-risk Chinese patients. Among 512 subjects who were to undergo coronary angiography for the confirmation of suspected myocardial ischemia, 409 subjects were enrolled and categorized into 3 groups based on FPG levels: (1) ≤5.5 mmol/L, (2) 5.6 to 6.0 mmol/L, and (3) 6.1 to 6.9 mmol/L. Each of these groups was further divided into subgroups by sex; the second and third groups were combined as an additional group according to the 2003 definition of impaired fasting glucose (FPG at 5.6-6.9 mmol/L). We analyzed the coronary artery stenosis score, the prevalence of angiographic CAD, and the percentage of stenosis in the 3 main arteries among the groups and examined the risk factors for angiographic CAD prevalence by logistic regression analysis. A higher correlation was observed between angiographic CAD prevalence and FPG levels of 6.1 to 6.9 mmol/L as compared with FPG levels ≤5.5 mmol/L (adjusted odds ratio [OR], 2.67; 95% confidence interval [CI], 1.72-4.10; $P = .011$). The FPG levels of 5.6 to 6.9 mmol/L (adjusted OR, 2.57; 95% CI, 1.65-4.02; $P < .001$) and 5.6 to 6.0 mmol/L (adjusted OR, 2.33; 95% CI, 1.58-3.49; $P = .008$) were modestly correlated with angiographic CAD prevalence. The angiographic CAD prevalence, coronary artery stenosis score, and the percentage of stenosis in the left anterior descending branch increased corresponding to increasing FPG levels from ≤5.5 mmol/L to 5.6 to 6.0 mmol/L to 6.1 to 6.9 mmol/L. We concluded that FPG levels of 5.6 to 6.9 mmol/L as well as of 6.1 to 6.9 mmol/L may be an independent risk factor for angiographic CAD; furthermore, there was a progressive and graded relation between FPG levels of 5.6 to 6.9 mmol/L and angiographic CAD prevalence and severity in high-risk Chinese patients.

1. Introduction

It has been recognized that impaired glucose tolerance is a risk factor for cardiovascular disease, but its diagnosis requires an oral glucose tolerance test (OGTT), which many physicians and patients are reluctant to perform [1]. As a more convenient alternative, the American Diabetes Association has emphasized screening by fasting plasma glucose (FPG) and lowered the cutoff point for abnormal FPG levels progressively from 7.0 to 6.0 to 5.6 mmol/L, resulting in the new definition of impaired fasting glucose (IFG) with the FPG lowered to 5.6 to 6.9 mmol/L [2]. This modification has provoked great controversy, particularly regarding the relation between different levels of FPG less than 7.0 mmol/L and coronary artery disease (CAD) mortality or angiographic CAD. The European Diabetes Epidemiology Group refused to accept this new definition and recommended that the values of all categorical definitions of nondiabetic hyperglycemia be reconsidered [3]. In reality, a continuum of CAD risk and mortality with increasing postchallenge glucose levels has been consistently demonstrated in multiple studies [4,5]; nevertheless, the association of this risk with FPG has never been examined in detail. Furthermore, the glucose thresholds used for diagnosing diabetes were largely based on the risks of eye and kidney

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impairments [6,7], with a relatively lesser focus on the CAD risk. There is a dearth of data regarding IFG in high-risk populations, such as those scheduled for coronary angiography, particularly in Chinese patients.

Recently, the results from the International Collaborative Study of Cardiovascular Disease in Asia [8] showed that in a nationally representative sample of 15 540 adults, the prevalence of self-reported diabetes, undiagnosed diabetes, and IFG (FPG at 6.1-6.9 mmol/L) was 1.3%, 4.2%, and 7.3%, respectively, among whom approximately 60% had cardiovascular disease. An even higher prevalence of undiagnosed diabetes or IFG was found in patients with suspected CAD [9,10], which highlights the necessity to measure the glucose concentration in high-risk patients without known diabetes. Considering the enormous medical expenses of more than 25 million patients with CAD along with the insufficient medical resources in China, FPG—which is greatly practicable, convenient, and cost-effective—is recommended over OGTT for use in epidemiological studies and individual diagnosis. Because clarifying the clinical value and association of different cutoffs of IFG definitions with CAD would exert an important impact on policy-making and would have important implications for clinical practice, we assessed the relation between FPG levels of 5.6 to 6.9 mmol/L and the prevalence and degree of angiographically determined CAD in high-risk Chinese patients.

2. Study design and methods

2.1. Subjects

A total of 512 consecutive Chinese patients who were scheduled for coronary angiography to confirm suspected myocardial ischemia (positive stress test or signs of ischemia in the resting electrocardiogram and/or typical angina) at Huashan Hospital in Shanghai were enrolled in the study. Eighty patients with self-reported diabetes, 3 patients with FPG levels ≥7.0 mmol/L without previously known diabetes, and 20 patients with valvular disease and cardiomyopathy were excluded from the study. Based on the FPG level, the remaining 409 patients were categorized into 3 groups, as follows: ≤5.5, 5.6 to 6.0, and 6.1 to 6.9 mmol/L, each of which was further divided into subgroups according to sex. According to the new definition of IFG (FPG at 5.6 to 6.9 mmol/L), an additional group was formed to examine its association with CAD prevalence. The subjects were interviewed for the documentation of medical histories (eg, duration of hypertension) and smoking habits (current, former, or never). During the physical examination, body weight and height were examined for measuring the body mass index (BMI). Blood pressure was measured by standard methods in the sitting position, and the mean systolic and diastolic blood pressure values of 2 measurements were recorded. A fasting venous blood sample was also obtained. The present study was approved by the Ethics Committee of Huashan Hospital, and informed consent was obtained from all the subjects.

2.2. Laboratory assays

Venous blood was collected after an overnight fast. The FPG concentration was estimated by the glucose oxidase procedure (Hitachi 7600-020 Automated Analyzer; Hitachi, Tokyo, Japan). Serum total cholesterol, high-density lipoprotein (HDL) cholesterol, and triglyceride levels were measured by an enzymatic method using a chemistry analyzer (Hitachi 7600-020). Day-to-day coefficients of variation for all analyses were 1% to 2% at the central laboratory in our hospital. Low-density lipoprotein (LDL) cholesterol was calculated using the Friedewald formula.

2.3. Angiographic studies

A catheterizing cardiologist, a qualified and experienced angiographer who was blinded to the study, performed the angiographic assessments. Coronary angiography was performed by the Judkins technique, and only the angiographically satisfactory images were used in the study. Angiographic CAD was diagnosed by a stenotic lesion of at least 50% in one or more coronary arteries as described previously [11]. We used a coronary artery stenosis score—obtained by adding the stenosis scores of the 3 main coronary arteries (the left anterior descending artery, left circumflex artery, and right coronary artery) together—to quantitate the severity of angiographic CAD according to the method described by Austen et al [12]. Furthermore, the percentage of stenosis in each main branch was documented as another index of the severity of angiographic CAD.

2.4. Statistical analysis

Data were expressed as mean ± SD for continuous variables or as percentages for categorical variables. The χ^2 statistic was used to determine the differences in the categorical variables between groups; the repeated measures of analysis of variance, to determine the significance of any trend across the groups. The analysis of covariance was performed to compare the variables between groups when the potential confounders had been controlled, which we determined a priori: sex, age, smoking, hypertension, triglycerides, and LDL levels. Logistic regression analysis was applied to examine the independent risk factors for angiographic CAD prevalence and to estimate its odds ratio (OR) with different levels of FPG (5.6-6.0, 6.1-6.9, and 5.6-6.9 mmol/L). Those with FPG ≤5.5 mmol/L were considered as the reference group. The adjusted OR and 95% confidence intervals (CIs) were calculated as the presence of dichotomous risk factors (eg, current or former smoking) and each SD increase or decrease in continuous risk factors (eg, increase of 3 kg/m^2 in BMI, increase of 10 years in age, increase of 1.0 mmol/L in the triglyceride level, and decrease of 0.3 mmol/L in the HDL cholesterol level). *P* values were based on 2-sided tests, and the cutoff

Table 1
Clinical characteristics of the studied groups with different FPG levels

	FPG categories			*P*
	≤5.5 mmol/L	5.6-6.0 mmol/L	6.1-6.9 mmol/L	
Women				
n	79	26	16	
Age (y)	63.9 ± 9.6	64.5 ± 7.6	67.9 ± 10.4	.447
BMI (kg/m^2)	24.3 ± 3.2	25.5 ± 3.3	25.1 ± 3.2	.399
Duration of smoking (y)	0.1 ± 1.1	0.8 ± 3.9	3.8 ± 10.9	.031[a]
Duration of hypertension (y)	9.7 ± 10.6	7.8 ± 9.5	11.1 ± 11.8	.755
FPG (mmol/L)	5.1 ± 0.3	5.7 ± 0.2	6.4 ± 0.2	<.001[a]
Cholesterol (mmol/L)	5.1 ± 1.1	5.5 ± 1.2	5.4 ± 1.4	.474
Triglyceride (mmol/L)	1.9 ± 1.0	2.2 ± 1.4	2.7 ± 2.1	.041[a]
HDL (mmol/L)	1.3 ± 0.4	1.3 ± 0.3	1.1 ± 0.2	.228
LDL (mmol/L)	4.2 ± 10.9	3.4 ± 1.3	3.3 ± 1.2	.971
Lipoprotein (α) (mmol/L)	245.4 ± 243.9	231.8 ± 197.2	221.4 ± 203.7	.972
Men				
n	179	63	46	
Age (y)	62.5 ± 11.6	64.6 ± 10.2	63.4 ± 7.8	.396
BMI (kg/m^2)	24.3 ± 3.2	24.8 ± 2.9	25.2 ± 3.5	.155
Duration of smoking (y)	15.3 ± 17.5	13.6 ± 16.8	16.4 ± 17.0	.693
Duration of hypertension (y)	5.8 ± 8.2	9.3 ± 10.5	9.6 ± 11.3	.005[b]
FPG (mmol/L)	5.0 ± 0.4	5.8 ± 0.1	6.4 ± 0.3	<.001[b]
Cholesterol (mmol/L)	4.5 ± 1.0	4.7 ± 0.9	4.8 ± 1.0	.111
Triglyceride (mmol/L)	1.5 ± 0.9	1.6 ± 0.9	1.9 ± 1.2	.045[b]
HDL (mmol/L)	1.1 ± 0.3	1.2 ± 0.3	1.1 ± 0.3	.426
LDL (mmol/L)	2.8 ± 0.9	2.9 ± 0.8	2.9 ± 0.9	.477
Lipoprotein (α) (mmol/L)	221.6 ± 188.4	243.9 ± 215.1	171.3 ± 118.8	.129

Data are mean ± SD.
[a] $P < .05$, significantly different among 3 female groups.
[b] $P < .05$, significantly different among 3 male groups.

point for statistical significance was .05. The SPSS 10.0 software (SPSS, Chicago, IL) was used for all the statistical calculations.

3. Results

3.1. Clinical characteristics of the studied groups

There were no significant differences among the 3 groups of our study with respect to age, BMI, cholesterol, HDL cholesterol, LDL cholesterol, and lipoprotein (a) levels (Table 1). However, among the 3 groups, the triglyceride levels (in the male and female subjects) and the duration of hypertension (in the male subjects) or smoking (in the female subjects) increased significantly (Table 1).

Table 2
Estimates of the relative risk of angiographic CAD prevalence

Risk factors	OR	*P*	95% CI
Age: 10 y	1.07	<.001	1.04-1.08
Male:female	3.06	<.001	1.84-4.61
Duration of hypertension: 10 y	1.04	.025	1.01-1.06
Current or former smoking: never smoking	1.02	.041	1.00-1.04
Triglyceride: 1.0 mmol/L	1.83	<.001	1.31-2.59
BMI: 3 kg/m^2	1.11	.001	1.04-1.18
HDL: 0.3 mmol/L	0.12	<.001	0.08-0.38
FPG at 5.6-6.0 mmol/L[a]: FPG ≤5.5 mmol/L	2.33	.008	1.58-3.49
FPG at 5.6-6.9 mmol/L[a]: FPG ≤5.5 mmol/L	2.57	<.001	1.65-4.02
FPG at 6.1-6.9 mmol/L[a]: FPG ≤5.5 mmol/L	2.67	.011	1.72-4.10

[a] Adjusted for age, sex, BMI, smoking habits, lipoproteins, and hypertension duration.

3.2. Relative risks of angiographic CAD prevalence

The risk factors of angiographic CAD prevalence were obtained through logistic regression analysis (Table 2). Compared with the reference group with an FPG level ≤5.5 mmol/L, the group with FPG levels of 6.1 to 6.9 mmol/L demonstrated the most significant association with angiographic CAD prevalence after controlling for age, sex, BMI, smoking habits, lipoproteins, and hypertension duration (adjusted OR, 2.67; 95% CI, 1.72-4.10; $P = .011$). A less significant association was noted in the group with FPG levels of 5.6 to 6.9 mmol/L (adjusted OR, 2.57; 95% CI, 1.65-4.02; $P < .001$). Interestingly, the group with FPG levels of 5.6 to 6.0 mmol/L persistently presented as an independent risk factor for angiographic CAD prevalence (adjusted OR, 2.33; 95% CI, 1.58-3.49; $P = .008$). Other risk factors included age, sex (male), smoking, hypertension duration, BMI, triglycerides, and HDL levels.

3.3. Comparison of CAD prevalence and severity among groups with different FPG levels

As the FPG level increased, so did the prevalence of angiographic CAD (54% ± 48% vs 65% ± 48% vs 72% ± 46%, $P_{trend} < .05$) (Fig. 1). Similarly, the coronary artery stenosis score (3.4 ± 3.9 vs 4.0 ± 3.5 vs 5.5 ± 4.3, $P_{trend} < .01$)

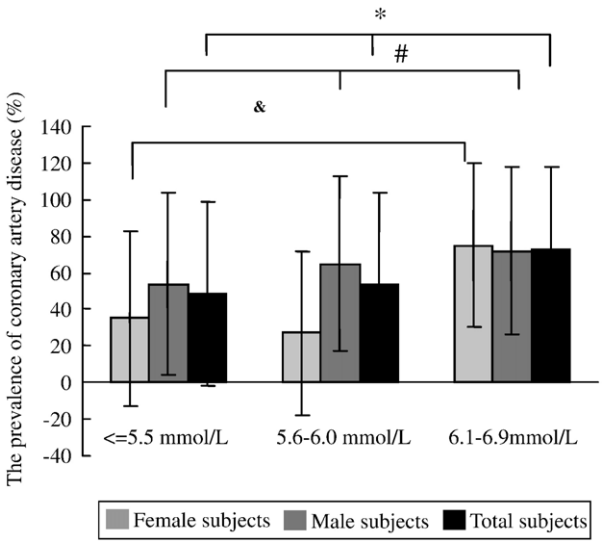

Fig. 1. Prevalence of CAD among different FPG levels in male and female subjects. Data are mean ± SD. *$P_{trend} < .05$, significantly different among 3 groups with FPG levels ≤5.5, 5.6 to 6.0, and 6.1 to 6.9 mmol/L in total subjects; #$P_{trend} < .05$, significantly different among 3 groups with FPG levels ≤5.5, 5.6 to 6.0, and 6.1 to 6.9 mmol/L in male subjects; &$P < .05$, significantly different between the group with FPG levels ≤5.5 mmol/L and that with FPG levels of 6.1 to 6.9 mmol/L in female subjects.

correspondingly increased with the FPG level (Fig. 2). These trends were graded and were observed to be statistically significant in the male subjects. In the female subjects, those with FPG levels of 6.1 to 6.9 mmol/L showed a considerably higher stenosis score (3.7 ± 3.3 vs 2.1 ± 3.7, $P < .01$) and CAD prevalence (75% ± 45% vs 35% ± 50%, $P < .01$) as compared

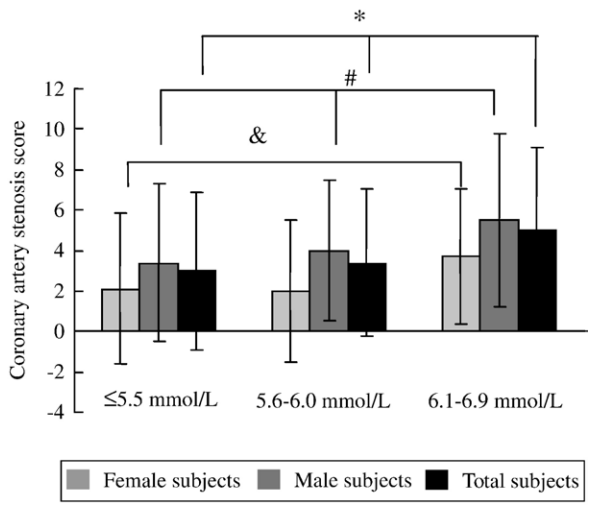

Fig. 2. Comparison of coronary artery stenosis score with different fasting glucose levels in men and women. Data are mean ± SD, adjusted for age, smoking, hypertension, triglycerides, and LDL levels. *$P_{trend} < .05$, significantly different among 3 groups with FPG levels ≤5.5, 5.6 to 6.0, and 6.1 to 6.9 mmol/L in total subjects; #$P_{trend} < .05$, significantly different among 3 groups with FPG levels ≤5.5, 5.6 to 6.0, and 6.1 to 6.9 mmol/L in male subjects; &$P < .05$, significantly different between the group with FPG levels ≤5.5 and that with FPG levels of 6.1 to 6.9 mmol/L in female subjects.

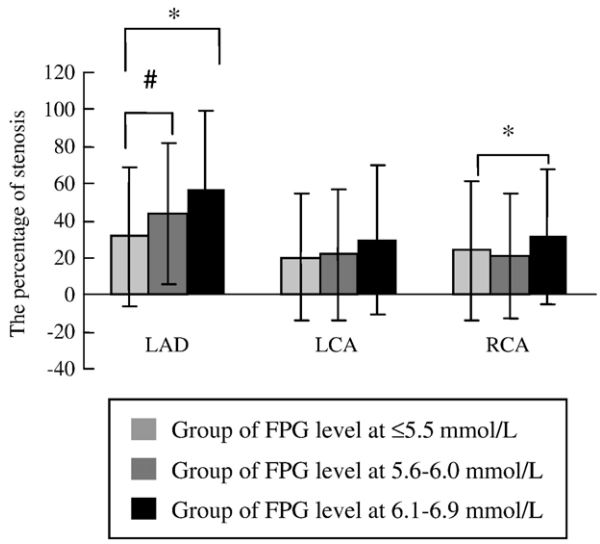

Fig. 3. Comparison of the percentage of stenosis with different FPG levels in the 3 main branches of coronary arteries. Data are mean ± SD, adjusted for sex, age, smoking, hypertension, triglycerides, and LDL levels. *$P < .05$, significantly different between the group with FPG levels of 6.1 to 6.9 mmol/L and that with FPG levels ≤5.5 mmol/L; #$P < .05$, significantly different between the group with FPG levels of 5.6 to 6.0 mmol/L and that with FPG levels ≤5.5 mmol/L. LAD indicates left anterior descending artery; LCA, left circumflex artery; RCA, right coronary artery.

with those with an FPG level ≤5.5 mmol/L. However, subjects with FPG levels of 5.6 to 6.0 mmol/L failed to demonstrate a significant difference in the stenosis score and the CAD prevalence in comparison with those with an FPG level ≤5.5 mmol/L (2.1 ± 3.7 vs 2.0 ± 3.5 and 35% ± 50% vs 27% ± 45%, respectively) (Figs. 1 and 2).

After controlling for the relevant covariates such as sex, age, smoking, hypertension, triglycerides, and LDL levels, the percentage of stenosis in the left anterior descending branch increased from 31.3% to 43.5% and to 56.8% ($P_{trend} < .05$), corresponding to the increase in FPG levels in all the subjects (Fig. 3). In the male subjects, the percentage of stenosis in the left anterior descending branch increased from 35.8% to 52.6% and to 59.3% ($P_{trend} < .05$); however, this result was not observed in the female subjects (data not shown). The percentage of stenosis in the left circumflex and right coronary arteries showed no graded relation with the FPG level, although the percentage of stenosis in the right coronary artery elevated significantly, corresponding to the increase in the FPG level from ≤5.5 mmol/L to 6.1 to 6.9 mmol/L (Fig. 3).

4. Discussion

Our present study, in which the criterion standard of coronary angiography was applied as the criteria for evaluating the prevalence and severity of CAD, demonstrated a progressive and graded relation between FPG levels varying from 5.6 to 6.9 mmol/L and angiographic CAD in high-risk Chinese patients for the first time.

There is universal agreement that diabetes is associated with a markedly increased risk of cardiovascular disease [13]. Most studies have indicated that this risk cannot be explained by conventional cardiovascular risk factors alone, suggesting that plasma glucose concentration, including higher euglycemic values, may be cardiovascular risk factors. Coutinho and his colleagues [14] conducted a metaregression analysis of the published data from 20 studies of 95 783 individuals, who had 3707 cardiovascular events over 12.4 years, and demonstrated that a fasting and postchallenge glucose level of 6.1 and 7.8 mmol/L was associated with relatively higher cardiovascular risks as compared with a glucose level of 4.2 mmol/L. This study indicated a progressive relation between glucose levels and cardiovascular risks extending below the diabetic threshold, which was well in line with our findings.

Furthermore, our study revealed that an FPG level between 6.1 and 6.9 mmol/L was an independent risk factor of CAD according to the 1997 IFG definition. Moreover, after controlling for other cardiovascular risks, FPG level of 5.6 to 6.9 mmol/L also presented as a significant risk factor of CAD according to the 2003 IFG definition. We also evaluated the effect of FPG level of 5.6 to 6.0 mmol/L and found it to be a risk factor for CAD prevalence, although with less statistical significance.

The 1997 definition of IFG was established to clarify a state of nondiabetic hyperglycemia at FPG level of 6.1 to 6.9 mmol/L. In 2003, the American Diabetes Association recommended that this diagnostic threshold be lowered to 5.6 mmol/L [2]; this has, however, been questioned. The Diabetes Epidemiology Collaborative Analysis of Diagnostic Criteria in Europe study among 22 European cohorts reported that there was no glycemic threshold for either fasting or postchallenge glucose levels above which the total mortality increased sharply [15]. Considerably low and high FPG levels were both associated with total and CAD mortality with a J-shaped curve; however, there was a graded association for the relation between postchallenge glucose levels and CAD mortality. Further evidence against the modification of IFG threshold came from a Taiwanese study [16]. Impaired fasting glucose, when defined as 6.1 to 6.9 mmol/L, was associated with a significant increase in CAD and/or diabetes mortality. However, the predictive value of IFG was lost when it was defined as 5.6 to 6.9 mmol/L, with the mortality risks diminishing substantially because of the inclusion of the 5.6- to 6.1-mmol/L group. Nevertheless, a recent meta-analysis of 38 prospective studies revealed the association between FPG and CAD to be nonlinear and the possible threshold to be approximately 5.6 mmol/L [17]. These studies chose surrogate markers, self-reporting questionnaires, or end-stage events for CAD; however, owing to its objectiveness and precision, it is vitally critical to use the criterion standard of angiographic CAD as the definition for CAD. Our study demonstrated the strongest association between FPG levels of 6.1 to 6.9 mmol/L and the prevalence of angiographic CAD; furthermore, FPG levels of 5.6 to 6.9 mmol/L or 5.6 to 6.0 mmol/L were still a significantly independent risk factor for CAD, suggesting the clinical significance of the lowered cutoff point of IFG. It should be noted that in our study, the criterion standard of angiographic CAD was used; furthermore, all the patients were hospitalized so that the fasting state and the precise measurement of glucose and lipid levels could be ensured.

A large prospective cohort study by Kanaya et al [18] reported that women with IFG according to the 1997 definition were at an increased risk for any CAD event, whereas those with IFG according to the 2003 definition were not. In particular, those with FPG levels of 5.6 to 6.0 mmol/L did not demonstrate an increased risk for CAD. Our data confirmed these results; that is, the female subjects with FPG levels of 5.6 to 6.0 mmol/L failed to show a significant difference in the stenosis score and CAD prevalence when compared with the values obtained for these parameters at an FPG level $\leq$5.5 mmol/L, whereas the male subjects with FPG levels of 5.6 to 6.0 mmol/L presented a greater severity and increased prevalence of CAD as compared with the controls. It appeared that there might be differences in angiographic CAD prevalence and severity between men and women with different FPG levels. Such investigations may have important clinical implications in handling male patients with a high risk for CAD even with borderline high FPG levels. However, whether men are more sensitive to increasing FPG levels needs to be further investigated and validated in large-scale study.

Finally, we compared the severity of stenosis in the 3 major branches among the different FPG groups and found that the percentage of stenosis corresponding to increasing FPG levels was more obvious in the left anterior descending branch than in the left circumflex branch or the right coronary artery. Certain previous studies have indicated that the severity of stenosis in the proximal left anterior descending artery was a stronger predictor of CAD prognosis than stenoses elsewhere in the major coronary arteries [19]. Our data revealed the close association between the stenosis in this anatomical location and FPG, further confirming the relation between FPG levels and CAD.

The limitation of this study was that because OGTT was not conducted, we could not compare the impact of different FPG levels and postchallenge glucose values on angiographic CAD. Wascher et al [20] observed that 30% of patients with suspected CAD had impaired glucose tolerance and highlighted the importance of OGTT. Satoh et al [21] maintained that postchallenge hyperinsulinemia rather than hyperglycemia was more likely to be associated with the number of diseased coronary arteries. Although IFG was reportedly less sensitive in its predictive value for diabetes or CAD than impaired glucose tolerance [22,23], FPG has been universally recognized as the preferred mode of diabetes screening, with the test being easier to perform, more convenient and acceptable to patients, more reproducible, and, most importantly, less expensive [24]. Meanwhile, OGTT has been reported to have poor reproducibility

[25,26], with an intraindividual coefficient of variation of 16.7% for postchallenge glucose values after OGTT vs 6.5% for FPG in the Hoorn Study [25]. In view of its advantages, particularly its relatively low cost, FPG is preferable in China, which is still a developing country. In addition, because FPG values are less variable and more convenient, the estimates of the glucose–cardiovascular risk relation based on FPG studies may be more acceptable than those based on combined studies using different glucose loads and glucose sampling times. Furthermore, this is a cross-sectional and non–population-based study (high-risk Chinese patients), which limits the generalizability of our findings.

In summary, there was a progressive and graded relation between FPG levels of 5.6 to 6.9 mmol/L and angiographic CAD prevalence and severity in high-risk Chinese patients who were scheduled for coronary angiography. Those with FPG levels of 6.1 to 6.9 mmol/L demonstrated an increased risk for CAD according to the 1997 IFG definition, and those with FPG levels of 5.6 to 6.9 mmol/L also had a less but significant CAD risk according to the 2003 definition. Identifying individuals using the 2003 definition of IFG may be very important for high-risk Chinese patients.

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