Isolated Post-Challenge Hyperglycemia in Patients With Normal Fasting Glucose Concentration Exaggerates Neointimal Hyperplasia After Coronary Stent Implantation

Nobuo Nakamura, MD; Yuji Ueno, MD; Yasuko Tsuchiyama, MD; Yoshimitsu Koike, MD; Masahiro Gohda, MD; Osamu Satani, MD

Postprandial hyperglycemia has been shown to increase the risk of cardiovascular disease as much as overt diabetes mellitus does. The aim of this study was to determine whether isolated post-challenge hyperglycemia during an oral glucose tolerance test (OGTT) is related to exaggerated neointimal proliferation after coronary stent implantation. Forty-seven coronary lesions treated with stents in 40 patients who had normal fasting glucose levels (<110 mg/dl) were categorized into the following 2 groups according to the results of a 75-g OGTT: 29 lesions in 24 patients with normal glucose tolerance (NGT group) and 18 lesions in 16 patients with abnormal glucose tolerance (AGT group). Although there were no differences in angiographic characteristics before and immediately after stenting between the 2 groups, the minimal lumen diameter was significantly smaller (p=0.04) and the degree of stenosis and late loss were also significantly greater (p=0.01 and p=0.047) in the AGT group than in the NGT group at 6-month follow-up. Multiple regression analysis including the insulin concentrations during an OGTT revealed that the 120-min plasma glucose concentration after glucose load significantly correlated with late loss (p=0.0018) and the degree of stenosis (p=0.0100) at follow-up. It is concluded that isolated post-challenge hyperglycemia exaggerates neointimal hyperplasia after coronary stent implantation.

Key Words: Coronary stent implantation; Isolated post-challenge hyperglycemia; Neointimal hyperplasia
patients with 168 lesions underwent coronary stent implantation at Seiyu Memorial Hospital. We studied 47 lesions from 40 patients with a fasting plasma glucose <110 mg/dl. Exclusions included (1) patients with previously treated DM (oral hypoglycemic agents or insulin); (2) restenosed lesions; (3) lesions with chronic total occlusion; (4) ostial or bifurcational lesions; and (5) patients who were unwilling to undergo the follow-up angiography approximately 6 months after the coronary stent implantation. There were 27 men and 13 women (mean age, 59±13 years). The study was approved by the institutional Ethics Committee and written informed consent was obtained from all patients.

Body mass index (BMI) was calculated and a 75-g OGTT was carried out by all 40 patients before follow-up angiography. After fasting overnight, each patient supplied blood samples at baseline and 30, 60, and 120 min after the glucose load. The study group was divided into 2 subgroups on the basis of the results: 24 patients with 29 stented lesions had normal glucose tolerance (NGT group) and 16 patients with 18 stented lesions had abnormal glucose tolerance (AGT group). Normal or abnormal glucose tolerance was determined by World Health Organization diagnostic criteria for DM:11 for the NGT group, 120-min plasma glucose concentration ≥140 mg/dl; for AGT group, 120-min plasma glucose concentration after the glucose load ≥210 mg/dl. The sum of plasma glucose (ΔPlasma glucose = fasting plasma glucose + 30-min plasma glucose+60-min plasma glucose+120-min plasma glucose) was calculated. The fasting immunoreactive insulin concentration and the sum of insulin ([Insulin]=fasting insulin+30-min insulin+60-min insulin+120-min insulin) were also measured. Homeostasis model assessment insulin resistance (HOMA-R) was calculated as (fasting plasma glucose×fasting insulin)/405.12 Plasma glucose concentrations were measured by an enzymatic method using QuickAuto II GLU-HK(s) (Shino-Test, Tokyo, Japan), and the immunoreactive insulin concentrations were measured by radioimmunoassay using Insulin Riabead II (Dainabot, Tokyo, Japan). Analyses of lipid concentration were performed at the same time: total cholesterol (TC) and triglyceride (TG) concentrations were measured by enzymatic methods using L type Wako Cholesterol and L type Wako TG-H, respectively (both from Wako Pure Chemical Industries, Osaka, Japan); the concentrations of low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C) were also measured by enzymatic methods using Cholestest LDL and Cholestest N HDL, respectively (both from Daichi Pure Chemicals, Tokyo, Japan).

Implantation of Stent and Follow-up Angiography

Stent implantation was performed according to standard protocols and in all cases conventional balloon angioplasty was the only procedure performed before stent placement. Balloon size and pressure were at the surgeon’s discretion. Multiple stents were deployed if necessary to cover the full extent of the target lesion or a dissection if it occurred. Adequacy of the final result was based solely on angiographic assessment. All patients received 162 mg of aspirin and 200 mg of ticlopidine, the latter for at least 4 weeks. Follow-up angiography was performed 6.0±0.8 months after stenting.

Coronary Angiographic Evaluation

Quantitative angiographic analysis was performed using a caliper method with the contrast-filled, nontapered catheter tip as the calibration. The measurements were done before coronary intervention and immediately after stent placement, and on the angiogram recorded at follow-up. Coronary reference segments were selected from sites both proximal and distal to the lesion, showing a smooth edge on contour with homogeneous filling of contrast medium. The reference diameter of the vessel, the minimal lumen diameter (MLD), the degree of stenosis expressed as a percentage of the diameter of the vessel, and the lesion length were calculated. The reference diameter was determined by the mean of the proximal and distal references of the stenotic lesion. The balloon/artery ratio was calculated as diameter of the inflated balloon divided by the coronary reference diameter. Acute gain was defined as MLD immediately after stent implantation minus MLD before intervention, and the percentage of acute gain was defined as the degree of stenosis immediately after stent implantation minus the degree before intervention. Late loss was calculated as MLD immediately after stent implantation minus MLD at follow-up, and the loss index was calculated as late loss divided by acute gain. Net gain was calculated as MLD at follow-up minus MLD before intervention. Restenosis was defined as stenosis of 50% or more at follow-up examination. To avoid inter- and intra-observer differences, angiograms were analyzed by at least 3 experienced angiographers who were unaware of the OGTT results.

Data Analysis

Categorical variables are reported as counts (percentage) and continuous variables as means±SD. Differences in categorical variables were analyzed by the chi-square test with Yates’s correction or the Fisher exact probability test, and differences in continuous variables were analyzed by the Mann-Whitney U-test. Clinical, angiographic, and procedural characteristics were determined by a lesion-based analysis. Previous studies have shown that luminal narrowing in stented lesions occurs at independent rates within the same patient when multiple lesions are dilated13 Multivariate regression analysis was used to determine the factors that correlated well with late loss, MLD, and the degree of stenosis on the follow-up angiogram. Factors with a p<0.10 in linear regression analysis were entered into the multivariate model. Statistical significance was defined as a p<0.05.

Results

The patients’ clinical characteristics and laboratory results are shown in Table 1. There were no differences in the clinical characteristics (ie, sex, age, hypertension, hyperlipidemia, previous myocardial infarction, acute myocardial infarction, and medications) except for current smoking, which was significantly greater in the AGT group (p=0.02). No differences were seen in TC, HDL-C, LDL-C and TG between the 2 groups. Although there were no differences in fasting plasma glucose, 30-min plasma glucose, fasting insulin, 30-min insulin, 60-min insulin, and ΔInsulin between the 2 groups, 60-min plasma glucose, 120-min plasma glucose, ΔPlasma glucose, and 120-min insulin were significantly greater in the AGT group. There was no significant difference in HOMA-R between the 2 groups.

Table 2 shows the angiographic characteristics before and immediately after stent implantation. There were no differences between the 2 groups in target vessel, modified

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Table 1 Clinical Characteristics and Laboratory Results

<table>
<thead>
<tr>
<th>M/F</th>
<th>NGT group (29 lesions)</th>
<th>AGT group (18 lesions)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>59.8±13.1</td>
<td>57.9±11.9</td>
<td>0.48</td>
</tr>
<tr>
<td>Smoker (%)</td>
<td>38</td>
<td>78</td>
<td>0.02</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>41</td>
<td>67</td>
<td>0.16</td>
</tr>
<tr>
<td>Hypoeylipidemia (%)</td>
<td>41</td>
<td>33</td>
<td>0.81</td>
</tr>
<tr>
<td>Previous myocardial infarction (%)</td>
<td>14</td>
<td>17</td>
<td>1.00</td>
</tr>
<tr>
<td>Acute myocardial infarction (%)</td>
<td>55</td>
<td>50</td>
<td>0.96</td>
</tr>
<tr>
<td>BMI</td>
<td>23.2±2.6</td>
<td>24.7±2.9</td>
<td>0.12</td>
</tr>
</tbody>
</table>

Laboratory results
- Cholesterol (mg/dl): 217±36 vs. 206±28 (p=0.36)
- High-density lipoprotein cholesterol (mg/dl): 48±10 vs. 44±13 (p=0.84)
- Low-density lipoprotein cholesterol (mg/dl): 142±38 vs. 124±26 (p=0.15)
- Triglyceride (mg/dl): 111±60 vs. 132±81 (p=0.10)
- Fasting plasma glucose (mg/dl): 94±18 vs. 97±28 (p=0.20)
- 30-min plasma glucose (mg/dl): 156±22 vs. 153±25 (p=0.83)
- 60-min plasma glucose (mg/dl): 168±35 vs. 191±29 (p=0.017)
- 120-min plasma glucose (mg/dl): 109±19 vs. 164±27 (<0.001)
- Plasma glucose (mg/dl): 527±56 vs. 605±74 (<0.001)
- Fasting insulin (µU/ml): 6±3 vs. 7±2 (p=0.24)
- 30-min insulin (µU/ml): 52±15 vs. 46±42 (p=0.19)
- 60-min insulin (µU/ml): 74±26 vs. 69±45 (p=0.69)
- 120-min insulin (µU/ml): 39±25 vs. 85±59 (<0.001)
- Insulin (µU/ml): 171±69 vs. 208±130 (p=0.45)
- HOMA-R: 1.38±0.71 vs. 1.65±0.63 (p=0.082)

Therapy at discharge (%)
- Statins: 24 vs. 22 (p=0.84)
- β-blockers: 0 vs. 11 (p=0.14)
- Angiotensin-converting enzyme inhibitors: 31 vs. 44 (p=0.35)

Table 2 Angiographic Characteristics at Baseline

<table>
<thead>
<tr>
<th>Target vessel</th>
<th>NGT group (29 lesions)</th>
<th>AGT group (18 lesions)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAD:LCX:RCA</td>
<td>14:6:9</td>
<td>8:4:6</td>
<td>0.97</td>
</tr>
<tr>
<td>No. of diseased vessels</td>
<td>1:2:3</td>
<td>22:4:3</td>
<td>10:6:2</td>
</tr>
<tr>
<td>Reference diameter (mm)</td>
<td>3.07±0.62</td>
<td>3.05±0.39</td>
<td>0.48</td>
</tr>
<tr>
<td>Lesion length (mm)</td>
<td>13.35±6.65</td>
<td>13.91±5.70</td>
<td>0.61</td>
</tr>
<tr>
<td>Stent type</td>
<td>Terumo:S670:NIR:GFX</td>
<td>4:8:10:11</td>
<td>2:7:10:2</td>
</tr>
<tr>
<td>No. of implanted stents</td>
<td>1.14±0.35</td>
<td>1.17±0.38</td>
<td>0.79</td>
</tr>
<tr>
<td>Balloon/artery ratio</td>
<td>1.09±0.15</td>
<td>1.04±0.12</td>
<td>0.35</td>
</tr>
<tr>
<td>Maximal inflation pressure (atm)</td>
<td>12.2±1.3</td>
<td>12.8±1.9</td>
<td>0.25</td>
</tr>
<tr>
<td>Minimal lumen diameter (mm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before procedure</td>
<td>0.52±0.44</td>
<td>0.62±0.44</td>
<td>0.41</td>
</tr>
<tr>
<td>After stenting</td>
<td>3.00±0.48</td>
<td>2.93±0.36</td>
<td>0.56</td>
</tr>
<tr>
<td>Degree of stenosis (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before procedure</td>
<td>82.0±14.9</td>
<td>79.1±14.2</td>
<td>0.48</td>
</tr>
<tr>
<td>After stenting</td>
<td>1.7±2.7</td>
<td>3.2±13.6</td>
<td>0.48</td>
</tr>
</tbody>
</table>

AGT, abnormal glucose tolerance; NGT, normal glucose tolerance.

ACC/AHA lesion classification14 the number of diseased vessels, reference diameter, lesion length, MLD before procedure, the degree of stenosis before procedure, stent type, the number of implanted stents, balloon/artery ratio, and maximal balloon inflation pressure. After stent implantation, there were also no differences in MLD, the degree of stenosis, and acute gain between the 2 groups.

The angiographic measurements at follow-up are shown in Table 3. There was no difference in reference diameter between the 2 groups, but the MLD in the AGT group was significantly smaller than that in the NGT group (p=0.04), and the degree of stenosis at follow-up in the AGT group was significantly greater (p=0.01). Late loss in the AGT group was significantly greater than in the NGT group (p=0.047), and net gain in the AGT group was significantly smaller than that in the NGT group (p=0.04).
Fig 1 shows that the MLDs before and immediately after stent implantation were similar for the 2 groups at baseline and there was similar angiographic gain after stenting; however, at follow-up there was greater late loss of luminal diameter in the AGT group (1.34±0.81 vs 0.88±0.47 mm, p=0.047).

Factors that tended to correlate with late loss, MLD at follow-up, and the degree of stenosis in lesions in the abnormal glucose tolerance (AGT) group were entered into the multivariate regression analysis (p<0.10) were entered into the multivariate regression analysis (Table 4). Lesion length, maximal inflation pressure, and TG concentration significantly correlated with the late loss only (p=0.0299, p=0.0272, and p=0.0479, respectively). The degree of stenosis immediately after stenting correlated only with the degree of stenosis at follow-up (p=0.0197). The 120-min plasma glucose concentration significantly correlated with not only late loss (p=0.0018), but also the degree of stenosis at follow-up (p=0.0100), and moreover had a tendency to correlate with MLD at follow-up (p=0.0751).

Discussion

In this study, which only enrolled patient with NFG (<110 mg/dl), we demonstrated that greater late loss and greater degree of stenosis at the site of stent implantation occurred in the AGT group, defined by an OGTT, than in the NGT group, and that the AGT group had a trend of higher rates of restenosis and TLR than the NGT group. Because stents do not recoil and in-stent restenosis is the result of neointimal proliferation, our results indicate that exaggerated neointimal hyperplasia in stented lesion occurs in patients with AGT.

Otsuka et al have reported that an important determinant for long-term prognosis after coronary balloon angioplasty is the presence of AGT, and recently, Takagi et al reported that patients with IGT defined by an OGTT had greater neointimal tissue proliferation after coronary stent implantation than those with NGT; however, their study included patients with a fasting plasma glucose <126 mg/dl (ie, patients with NFG and IFG). Ko et al have reported that subjects with IFG progress to DM more frequently than those with NFG, and that IFG status is an independent risk factor for progression to diabetes. In the present study, we excluded patients with IFG, and our study is the first to demonstrate that the NGT patients with AGT have more exaggerated neointimal hyperplasia in the stented lesion compared with the NGT patients with NGT; that is, abnormal glucose metabolism that cannot be detected by only measuring the fasting plasma glucose concentration might progress the in-stent restenosis independently of other risk factors.

The 120-min plasma glucose concentration had a strong positive association with late loss and the degree of stenosis at follow-up, and had a tendency to associate with MLD at follow-up. In other words, there was a positive correlation between 120-min plasma glucose concentration and neointimal hyperplasia in the stented lesion. It has been reported that carotid IMT in patients with isolated post-challenge hyperglycemia during an OGTT is greater than that in non-diabetic patients and that 120-min post-challenge glucose concentration correlated more closely to carotid IMT than fasting plasma glucose concentration. The Diabetes Intervention Study (DIS) indicated that post-
Isolated Post-Challenge Hyperglycemia and In-Stent Restenosis

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Table 4  Multiple Regression Analysis of Predictors of Late Loss, MLD at Follow-up, and Degree of Stenosis at Follow-up

<table>
<thead>
<tr>
<th></th>
<th>Linear regression</th>
<th>Multiple regression analysis</th>
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</thead>
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<tr>
<td></td>
<td>Regression p value</td>
<td>Regression coefficient Standard error of coefficient</td>
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<tr>
<td>Association with late loss</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lesion length</td>
<td>0.0886</td>
<td>0.025</td>
</tr>
<tr>
<td>Balloon/artery ratio</td>
<td>0.0974</td>
<td>−0.925</td>
</tr>
<tr>
<td>Maximal inflation pressure</td>
<td>0.0977</td>
<td>0.133</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>0.033</td>
<td>0.002</td>
</tr>
<tr>
<td>Fasting plasma glucose</td>
<td>0.0222</td>
<td>0.004</td>
</tr>
<tr>
<td>120-min plasma glucose</td>
<td>0.014</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Association with MLD at follow-up

Reference diameter | 0.0854 | −0.299 | 0.663 | −0.452 | 0.6544 |
MLD after stenting | 0.0006 | 0.57   | 0.44  | 1.295  | 0.2038 |
Acute gain (mm)    | 0.0056 | 0.359  | 0.604 | 0.594  | 0.5562 |
Acute gain (%)     | 0.0439 | −0.006 | 0.019 | −0.291 | 0.7726 |
Triglyceride       | 0.0648 | −0.001 | 0.002 | −0.697 | 0.4907 |
High-density lipoprotein cholesterol | 0.0182 | 0.012 | 0.013 | 0.97    | 0.3389 |
Fasting plasma glucose | 0.0501 | −0.028 | 0.021 | −0.337 | 0.1898 |
Fasting insulin    | 0.0644 | −0.046 | 0.066 | −0.695 | 0.4919 |
120-min plasma glucose | 0.0123 | −0.005 | 0.003 | −1.834 | 0.0751 |
HOMA-R             | 0.0873 | 0.215  | 0.29  | 0.741  | 0.4636 |

Association with degree of stenosis at follow-up

MLD after stenting | 0.0751 | −3.146 | 6.779 | −0.464 | 0.6453 |
Degree of stenosis after stenting | 0.0027 | 0.641 | 0.263 | 2.437 | 0.0197 |
Acute gain (%)     | 0.0211 | −0.036 | 0.192 | −0.186 | 0.8537 |
Triglyceride       | 0.0094 | 0.035  | 0.047 | 0.739  | 0.4644 |
High-density lipoprotein cholesterol | 0.0348 | −0.286 | 0.3 | −0.954 | 0.3464 |
Fasting plasma glucose | 0.0333 | 0.518 | 0.455 | 1.137 | 0.2627 |
Fasting insulin    | 0.0682 | 0.49   | 1.154 | 0.424  | 0.6738 |
120-min plasma glucose | 0.0019 | 0.223 | 0.082 | 2.714  | 0.0089 |

MLD, minimal lumen diameter.

prandial hyperglycemia, but not fasting hyperglycemia, was an independent risk factor for myocardial infarction based on an 11-year follow-up study of 1,139 patients with newly diagnosed non-insulin-dependent DM.18 Furthermore, the DECODE (Diabetes Epidemiology: Collaborative analysis Of Diagnostic criteria in Europe) Study Group found that the plasma glucose concentration at 120-min after an OGTT was a better predictor of death from cardiovascular disease than was the fasting plasma glucose.19 These previous studies suggest more rapid progression of coronary atherosclerosis in patients with postprandial hyperglycemia and we have also demonstrated that the degree of neointimal hyperplasia in stented lesions might depend on the postprandial glucose concentration in patients with NFG. It appears that isolated postprandial hyperglycemia accelerates not only the long-term progression of coronary atherosclerotic change, but also the acute hyperglycemic response to the vessel injury that accompanies stent implantation. Management of the postprandial glucose concentration in patients who undergo stenting of a coronary artery might be important to prevent exaggerated neointimal hyperplasia in the stented lesion even if the fasting glucose concentration is normal.

There are several mechanisms that induce an exaggerated neointimal hyperplasia in the hyperglycemic state. First, production of advanced glycosylation end products (AGEs) in the exposed vessel wall could be accelerated by high glucose concentration. AGEs can mediate inflammatory cell recruitment and activation, and stimulate smooth muscle cell proliferation and abnormal matrix production.20,21 Moreover, AGEs have been shown to rapidly inactivate endothelium-derived relaxing factor, which inhibits smooth muscle cell proliferation.22,23 Second, hyperglycemia itself could affect the expression of several growth factors, such as basic fibroblast growth factor and transforming growth factor-alpha, which induce the proliferation of smooth muscle cells and extracellular matrix synthesis.24,25 In addition, hyperglycemia could result in a decrease in the de novo synthesis of extracellular matrix-associated heparan sulfate, which is a potent inhibitor of smooth muscle cell proliferation and plays an important role in the regulation of smooth muscle cell growth in injured arteries.26–29 Decreased heparan sulfate concentration could result in a loss of tonic growth inhibition and facilitation of smooth muscle cell proliferation.

Several recent studies have demonstrated that hyperinsulinemia during an OGTT is a predictor for restenosis after coronary angioplasty.16,30,31 and physiologic concentrations of insulin are known to stimulate the proliferation of cultured smooth muscle cells and fibroblasts.32,33 Although insulin itself is a poor mitogen, it can potentiate the expression of more potent mitogens such as platelet-derived growth factor and insulin-like growth factor.34,35 In the present study, the 120-min insulin concentration during an OGTT in the AGT group was significantly higher than in the NFG group, but in multiple regression analysis, there were no associations between all insulin concentrations during an OGTT and the parameters representing neointimal hyperplasia. Secretion of intrinsic insulin from the pancreas changes according to the phase of the glucose metabolic disorder and in general, the circulating insulin concentration in patients with IGT is high. However, the present AGT group included not only IGT but also DM because the definition of the group was a 120-min plasma glucose concentration during an OGTT of ≥140 mg/dl. The tendency of insulin secretion in the AGT group was not
homogeneous and it is possible that there would have been significant associations between any insulin concentration and the parameters of neointimal hyperplasia if we had excluded the patients with DM.

Cigarette smoking impairs endothelial function and is one of the major risk factors for atherosclerosis and coronary heart disease.36,37 We found in the present study that the patients with AGT had a significantly greater history of current smoking than those with NFG. Nakanishi et al have reported that current smokers have a significantly greater risk developing impaired fasting glucose and type 2 DM, and its risk is associated with the number of cigarettes smoked daily and the number of pack-years of exposure.38 Recently, Ko et al demonstrated that male smokers had higher plasma glucose concentrations 60 min after an OGTT and more incidence of DM than male non-smokers.39 Although there is no evidence that cigarette smoking contributes to an adverse clinical outcome after coronary intervention,40-42 it is possible that it may be indirectly related to the restenosis after coronary stent implantation through development of AGT.

Study Limitations
There are some potential limitations in this study. First, it was a single-center study with a relatively small number of patients. Second, quantitative angiographic analysis was not performed using computer-assisted. However, the measurement was always repeated and evaluated by at least 3 experienced angiographers, and we believe that an error of measurement was always repeated and evaluated by at least 3 experienced angiographers, and we believe that an error of measurement is unlikely to have influenced the results. Third, we did not directly measure the area of the neointimal hyperplasia in the stented lesions using serial intravascular ultrasound (IVUS), but we did use 3 parameters (late loss, MLD at follow-up, and the degree of stenosis at follow-up) that are thought to express the degree of the neointimal hyperplasia in stented lesions. We analyzed the participation of various clinical characteristics to those 3 parameters in multiple regression analysis. We are firmly persuaded that there is no advantage of IVUS against the certain added cost and risk related to that technique. Fourth, our study included patients with acute myocardial infarction whose target lesions have some amount of thrombus that might influence smooth muscle cell proliferation.43 However, there was no significant difference in the incidence of acute myocardial infarction between the 2 study groups, and acute myocardial infarction had no association with late loss, MLD at follow-up, or the degree of stenosis at follow-up in regression analysis. It is unlikely that our results would change if we excluded the patients with acute myocardial infarction. Fifth, we used various types of stents and so a further study using a single type of stent is necessary to minimize the influence of the properties of the stent.

Conclusions
Isolated hyperglycemia 120 min after an oral 75-g glucose load exaggerates neointimal hyperplasia after coronary stent implantation in patients with NFG. Clinicians need to be aware of postprandial hyperglycemia in patients after coronary stenting therapy and should manage it, even if the patient has NFG, in order to prevent exaggerated neointimal hyperplasia in the stented lesions.

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