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Residual Angina Following Elective Percutaneous Coronary Intervention in Patients with Diabetes

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Abstract

Background—Prior studies suggest that among patients with stable coronary artery disease (CAD), patients with diabetes mellitus (DM) have less angina and more silent ischemia as compared with those without DM. However, the burden of angina in diabetic versus nondiabetic patients following elective percutaneous coronary intervention (PCI) has not been recently examined.

Methods and Results—In a 10-site US PCI registry, we assessed angina before and at 1, 6, and 12 months after elective PCI with the Seattle Angina Questionnaire angina frequency score (range 0–100, higher=better). We also examined the rates of antianginal medication prescriptions at discharge. A multivariable, repeated measures Poisson model was used to examine the independent association of DM with angina over the year after treatment. Among 1080 elective PCI patients (mean age 65, 74.7% male), 34.0% had DM. At baseline and at each follow-up, patients with DM had similar angina prevalence and severity as those without DM. Patients with DM were more commonly prescribed calcium channel blockers and long-acting nitrates at discharge (DM vs. not: 27.9% vs. 20.9% [p=0.01] and 32.8% vs. 25.5%, [p=0.01], respectively), while beta-blockers and ranolazine were prescribed at similar rates. In the multivariable, repeated measures model, the risk of angina was similar over the year following PCI in patients with vs. without DM (RR 1.04, 0.80–1.36).
Conclusions—Patients with stable CAD and DM exhibit a burden of angina that is at least as high as those without DM, despite more antianginal prescriptions at discharge. These findings contradict the conventional teachings that patients with DM experience less angina due to silent ischemia.

Keywords
angina; diabetes; percutaneous coronary intervention

Patients with diabetes (DM) generally have more diffuse coronary artery disease (CAD) than patients without DM, greater progression of CAD, and a higher risk of restenosis after percutaneous coronary intervention (PCI). However, the degree to which these anatomic differences translate into differences in ischemic symptoms is not clear. Prior studies comparing the burden of angina by DM status in patients with CAD have yielded contradictory results. Earlier studies suggested that patients with DM experienced less angina than those without DM, theoretically due to silent ischemia as a consequence of autonomic neuropathy. Conversely, more recent work has shown more angina in patients with DM, both before and after acute myocardial infarction. However, the prevalence of residual angina after PCI in patients with DM and stable CAD has not been examined.

This is particularly relevant as the primary benefit of PCI in patients with stable ischemic heart disease is amelioration of angina. Residual angina after PCI is associated with impaired quality of life and is associated with repeat hospitalizations. As such, a better understanding of the burden of residual angina is needed among patients with DM and stable ischemic heart disease—patients who are often challenging to treat interventionally due to the diffuse nature of their atherosclerosis and increased risk of restenosis after successful PCI. To address this gap in knowledge, we compared angina over the year following elective PCI among patients with and without DM in a large, multicenter US PCI registry.

METHODS

Study Design and Participants

Our analytic population was derived from the Outcomes of PCI Study (OPS)/Personalized Risk Information Services Manager™ (PRISM) prospective study of patients undergoing PCI. Briefly, from May 26, 2009 to October 21, 2011, consecutive patients undergoing PCI for all indications at 10 US hospitals were invited to participate in OPS/PRISM at the time of PCI. Baseline data were obtained through a combination of chart abstraction and a detailed interview performed by trained study coordinators at each site. For the purposes of this analysis, only patients who underwent PCI electively for stable angina were included. Detailed telephone follow-up interviews were attempted on all surviving patients at 1, 6, and 12 months following index PCI by a specialized team at the coordinating center. If patients were unavailable by phone, follow-up interviews were mailed to the patients with a pre-paid return envelope. During follow-up interviews, patients were asked to report interval hospitalizations since their last study contact, which were confirmed with hospitalization records review. Each participating site obtained Institutional Research Board approval, and all patients provided informed consent for baseline and follow-up assessments.
Definition of DM and Angina

DM was defined as a chart-derived diagnosis of either type 1 or type 2 DM. Angina was assessed during the PCI hospitalization and at each follow-up interview using the Seattle Angina Questionnaire (SAQ). The SAQ is a reliable, responsive, and valid 19-item questionnaire with a 4-week recall that assesses 5 clinically important domains of health in patients with CAD: angina frequency, angina stability, quality of life, physical limitations and treatment satisfaction. The scores for each of the SAQ domains are transformed, with a range from 0 to 100, with higher scores indicating less angina and better health status. For this study, we focused on the SAQ angina frequency domain (SAQ AF), which has been shown to correlate well with patient-reported daily diaries of angina. To facilitate interpretation of the SAQ AF scores, we mirrored prior work by categorizing scores into daily (SAQ AF score 0–30), weekly (SAQ AF score 31–60), monthly (SAQ score 61–99), and no angina (SAQ AF score=100).

Statistical Analysis

Baseline characteristics were compared between patients with and without DM using chi-square tests for categorical variables and t-test for continuous variables. The prevalence (SAQ AF score <100 vs. 100) and severity of angina (category of SAQ AF [daily, weekly, monthly, none]) were compared between groups at each follow-up time point using chi-square tests. Mean scores on all SAQ domains were compared at baseline and at 12 months between groups using t-tests. A hierarchical, multivariable repeated measures Poisson model was then used to assess the independent association between DM and angina over the year following index PCI. This allowed us to integrate multiple follow-up time points into a single estimation of risk over time and also to estimate relative risks (RR) directly, to avoid overestimating the effect size. Covariates for the model were selected a priori based on literature review and clinical judgment, balancing adjustment with over-fitting, and included age, sex, race, current smoking, dyslipidemia, hypertension, history of myocardial infarction, history of PCI, history of coronary artery bypass grafting, and multivessel disease on the current angiogram (defined as ≥70% stenosis in 2 or more major epicardial coronary arteries or ≥50% stenosis of the left main coronary artery). Hospital was entered in the model as a random effect to adjust for patient clustering by site.

The association of residual angina at 1-month post-PCI with subsequent all-cause rehospitalizations was examined in patients with and without DM using Kaplan-Meier methods. A Cox proportional hazards model was used to examine whether the association of residual angina (yes/no), DM, and the interaction of DM*1-month angina.

Baseline data were generally complete, with a mean number of missing items per patient of 0.03. Supplemental Table 1 describes baseline characteristics of patients missing from analytic cohort (n=42) compared to patients included in overall cohort. These data were estimated with a single imputation dataset using IVEware (University of Michigan’s Survey Research Center, Institute for Social Research, Ann Arbor, MI). All remaining analyses were conducted using SAS v9.3 (SAS Institute, Inc., Cary, NC), and statistical significance was determined by a 2-sided p-value of <0.05.
RESULTS

Study Population

From 2009–2011, 3299 patients who underwent PCI at 10 US sites were enrolled in OPS/PRISM. We excluded 1230 patients who underwent PCI for an acute coronary syndrome, 947 patients whose PCI was performed urgently or emergently, and 42 (3.7%) patients who were missing either baseline or follow-up angina assessments (Figure 1). Our final analytic cohort thus consisted of 1080 patients who underwent PCI for stable ischemic heart disease, of whom 367 (34.0%) had DM. The mean age of the cohort was 65 years, 74.7% were male, 93.8% were of white race, and 92.6% had at least a high school education (Table 1). An established diagnosis of CAD was common, with 23.2% having a history of MI, 41.8% with a history of PCI, and 19.0% with a history of CABG.

Patients with DM (vs. those without) were less likely to be white (89.8% vs. 95.9%, p<0.001; Table 1) and were more likely to report avoiding care due to costs (11.5% vs. 7.0%, p=0.013). They had a higher mean body mass index (32.9 vs. 29.4, p<0.001), more frequent clinical diagnosis of heart failure (12.3% vs. 5.3%, p<0.001), and had had a prior PCI (48.5% vs. 38.3%, p=0.001) more often than patients without DM. Angiographic findings, however, were comparable between patients with and without DM (no difference in number of patients with multivessel disease, and no numerical difference in number of diseased vessels (neither ≥50%, nor ≥70% stenoses, nor number of vessels with chronic total occlusion). There was, however, a non-significant trend toward more obstructive CAD among patients with DM.

Burden of Angina

Over the 4 weeks prior to PCI, patients with and without DM reported similarly high angina burden (Table 2), with over one-third of patients reporting daily or weekly angina (DM vs. non-DM: 37.1% vs. 36.2%, p=0.952; Figure 2). Patients with DM were more commonly discharged on antianginal medication as compared with patients without DM (81.6% vs. 74.7%, p=0.011; Table 2) and were more likely to be discharged on 2 or more antianginal medications (31.2% vs. 23.1%, p=0.004). These differences were primarily driven by increased use of calcium channel blockers and long-acting nitrates in patients with DM, as beta-blocker and ranolazine prescription rates were similar between groups. There was no significant difference in the number of antianginals in those with and without HF.

Over the 12 months following PCI, patients with DM reported nominally higher but statistically similar rates of angina as compared with patients without DM, both in prevalence of angina and severity of angina (Figure 2). One year after PCI, 17.4% of patients with DM reported residual angina vs. 16.0% of patients without DM (p=0.577). Other SAQ domains were also similar between those with and without DM with the exception of SAQ physical limitations scores, which were statistically lower (worse) at 12 months in patients with DM (DM vs. non-DM: 95.1 vs. 96.5, p=0.047), although this mean difference is not generally considered clinically important. After adjusting for demographic and clinical characteristics, including multivessel disease, patients with DM had a similar risk of residual angina over the year following PCI (RR 1.04, 0.80–1.36).
Rehospitalization

At 1-month after PCI, 24.0% of patients reported residual angina (DM: 27.7%; no DM: 21.9%). Residual angina was associated with a greater risk of rehospitalization after PCI in both patients with and without DM (Figure 3). In the Cox model that included DM, residual angina was associated with a 1.74-increased hazard of rehospitalization (95% CI 1.23–2.47, p=0.002). However, this increased hazard did not vary by DM status (DM*angina interaction p=0.808). As patients with DM were more likely to have HF and rehospitalizations were not restricted to those for angina, we also examined whether a diagnosis of HF impacted the association between residual angina and risk of rehospitalization. The addition of HF to the model did not impact the HR for residual angina (HR 1.37, 95% CI 0.74–2.56).

DISCUSSION

In a large, contemporary multicenter US PCI registry, we found that angina burden was similar between those with and without DM, both at the time of elective PCI and over the following year, despite more aggressive treatment of diabetic patients with antianginal medications. Furthermore, we found that residual angina after PCI was associated with an increased risk of rehospitalization—a risk that was similar among patients with and without DM—highlighting the clinical importance of residual angina. These findings support results from recent studies demonstrating that patients with DM and CAD (stable or unstable) frequently experience symptomatic ischemia by extending these observations to a post-PCI population.

Earlier studies suggested that patients with DM incur a large burden of silent ischemia due to autonomic neuropathy. As such, the prevailing wisdom has been that patients with DM and CAD primarily experience myocardial ischemia silently and therefore have less angina as compared CAD patients without DM. However, more contemporary work explicitly investigating the rates of symptomatic myocardial ischemia in patients with and without DM demonstrated consistent results to ours, namely that patients with DM experience angina at similar or even higher rates as patients without DM.

Furthermore, the concept that patients with DM and CAD have autonomic dysfunction and therefore more silent ischemia than patients without DM has also been challenged. In the Asymptomatic Cardiac Ischemia Pilot (ACIP) Study of patients with stable ischemic heart disease, patients with DM had a similar prevalence of asymptomatic ischemia during exercise treadmill testing as compared with patients without DM. Our results therefore add to a growing body of studies demonstrating that patients with DM and CAD may experience myocardial ischemia symptomatically, and not just silently, and that angina (and mechanisms to treat angina) remains a substantial clinical concern in this population. In fact, a recent large clinical trial of an antianginal medication focused specifically on patients with DM, with the idea that these patients are particularly challenging to treat. Further defining the high burden of angina among patients with DM and CAD should encourage continued efforts to identify mechanisms by which to treat these symptoms of ischemia, particularly given the implications of this angina in terms of rehospitalizations and quality of life. Furthermore, these data should encourage clinicians to continue to probe about the presence of angina (or anginal equivalent) in their patients with diabetes.
Limitations

Our findings should be considered in the context of the following potential limitations. First, while the severity of anatomic CAD was similar between groups, we did not have functional assessments of ischemia at baseline or at follow-up. These would have allowed us to determine the proportion of ischemia over follow-up that was experienced as angina (vs. silent ischemia). While this could be an interesting future study, our study was designed to compare the prevalence and burden of angina after PCI between patients with and without DM—not to compare the amount of residual ischemia. Angina, as assessed with the SAQ, is associated with lower quality of life, an increased risk of hospitalization, and higher healthcare costs, and as such, we believe it is an important patient-centered outcome.

Second, our population included only patients who presented for PCI, most of who had some degree of symptomatic ischemia on presentation. As such, we cannot generalize our results to the general population of patients with CAD. Third, DM classification was based only on chart-derived diagnosis. This potentially misses a healthy minority of patients who may have previously undiagnosed DM as studies have shown that a substantial amount of patients presenting with coronary disease become newly diagnosed with DM. Another consideration is that HF was more common in patients with DM, and some antianginal medications are not recommended in HFrEF (e.g., verapamil, diltiazem, propranolol, nebivolol). As such, there is a possibility that differences in antianginal medications may have been influenced by the differing rates of HF between groups. However, all 4 classes of antianginal medications have options that are safe and effective even in HFrEF, and so it is unlikely that this was a major contributor to the observed differences in use in antianginal medications between groups. Finally, although we adjusted for a number of demographic and clinical characteristics, as an observational analysis, residual confounding is possible.

Conclusions

Among patients undergoing PCI for stable CAD, patients with DM report a burden of angina that is at least as high as those without DM—both before and up to 1 year after PCI—despite more aggressive antianginal prescription. These findings lend support to recent studies demonstrating that patients with DM have a high burden of symptomatic chest pain as opposed to only silent ischemia. Continued efforts to reduce the burden of angina in patients with DM, including novel interventional and surgical techniques and antianginal medications, are needed.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Dr. Arnold has received advisory board income from Novartis.

References


What is Known

- The prevailing wisdom has been that patients with diabetes (DM) and CAD primarily experience myocardial ischemia silently and therefore have less angina as compared CAD patients without DM.

- However, more contemporary work investigating the rates of symptomatic myocardial ischemia in patients with and without DM demonstrated that patients with DM experience angina at similar or even higher rates as patients without DM.

What the Study Adds

- We found that angina burden was similar between those with and without DM, both at the time of elective PCI and over the following year.

- Residual angina after PCI was associated with an increased risk of rehospitalization—a risk that was similar among patients with and without DM—highlighting the clinical importance of residual angina.

- Our findings support recent studies demonstrating that patients with DM have a high burden of symptomatic chest pain as opposed to only silent ischemia. Continued efforts to reduce the burden of angina in patients with DM, including novel interventional and surgical techniques and antianginal medications, are needed.
Figure 1.
Flow chart of the analytic cohort

PCI patients enrolled in OPS/PRISM (n=3299)

- Patients with ACS (n=1230)
- Patients with urgent/emergent PCI (n=947)

PCI for stable ischemic heart disease (n=1122)

- Missing baseline SAQ angina frequency score (n=2)
- Missing follow up SAQ angina frequency score (n=40)

Analytic cohort (n=1080)

Diabetes (n=367)
34.0%

No diabetes (n=713)
66.0%
Figure 2. Angina burden at baseline and over the year following PCI in patients with and without DM.
As assessed with the Seattle Angina Questionnaire angina frequency domain. Scores 0–30 indicate daily angina; 31–60 indicate weekly angina; 61–99 indicate monthly angina.
Figure 3.
Kaplan-Meier curve depicting rehospitalization after PCI in patients with and without DM stratified by presence or absence of residual angina at 1 month following index PCI
### Table 1
Baseline characteristics of analytic cohort from the OPS-PRISM registry

<table>
<thead>
<tr>
<th></th>
<th>Overall n=1080</th>
<th>Diabetes n=367</th>
<th>No Diabetes n=713</th>
<th>P - Value</th>
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<tr>
<td><strong>Socio-demographics</strong></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Age (years)</td>
<td>65.1±10.4</td>
<td>65.3±9.9</td>
<td>65.0±10.6</td>
<td>0.692</td>
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<tr>
<td>Male sex</td>
<td>74.7%</td>
<td>71.7%</td>
<td>76.3%</td>
<td>0.097</td>
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<td>White race</td>
<td>93.8%</td>
<td>89.8%</td>
<td>95.9%</td>
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</tr>
<tr>
<td>High school education</td>
<td>92.6%</td>
<td>90.9%</td>
<td>93.4%</td>
<td>0.143</td>
</tr>
<tr>
<td>Insurance for medications</td>
<td>95.5%</td>
<td>97.0%</td>
<td>94.7%</td>
<td>0.084</td>
</tr>
<tr>
<td>Self-reported avoidance of care due to cost</td>
<td>8.6%</td>
<td>11.5%</td>
<td>7.0%</td>
<td>0.013</td>
</tr>
<tr>
<td><strong>Clinical characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body mass index (kg/m$^2$)</td>
<td>30.6 ± 6.1</td>
<td>32.9±6.6</td>
<td>29.4±5.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>History of MI</td>
<td>23.2%</td>
<td>26.4%</td>
<td>21.6%</td>
<td>0.075</td>
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<td>History of PCI</td>
<td>41.8%</td>
<td>48.5%</td>
<td>38.3%</td>
<td>0.001</td>
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<td>History of CABG</td>
<td>19.0%</td>
<td>18.8%</td>
<td>19.1%</td>
<td>0.914</td>
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<td>Chronic heart failure</td>
<td>7.7%</td>
<td>12.3%</td>
<td>5.3%</td>
<td>&lt;0.001</td>
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<tr>
<td>Peripheral artery disease</td>
<td>8.7%</td>
<td>10.9%</td>
<td>7.6%</td>
<td>0.066</td>
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<td>Chronic lung disease</td>
<td>9.4%</td>
<td>11.2%</td>
<td>8.4%</td>
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<tr>
<td>Depression</td>
<td>6.9%</td>
<td>6.2%</td>
<td>6.9%</td>
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<td><strong>Angiographic characteristics</strong></td>
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<tr>
<td>Multivessel disease*</td>
<td>39.4%</td>
<td>42.7%</td>
<td>37.7%</td>
<td>0.112</td>
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<tr>
<td># Patients with diffuse disease</td>
<td>9.3%</td>
<td>11.5%</td>
<td>8.1%</td>
<td>0.073</td>
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<td>Residual # of diseased vessels post PCI†</td>
<td>0.4±0.7</td>
<td>0.4±0.6</td>
<td>0.4±0.7</td>
<td>0.206</td>
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<td>Vessels with ≤50% stenoses</td>
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<td>0</td>
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<td>11.7%</td>
<td>12.7%</td>
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<td>Vessels with ≤70% stenoses‡</td>
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</tr>
<tr>
<td>3</td>
<td>11.6%</td>
<td>12.4%</td>
<td>11.3%</td>
<td></td>
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<tr>
<td>Vessels with chronic total occlusions</td>
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<tr>
<td>0</td>
<td>84.9%</td>
<td>83.9%</td>
<td>85.4%</td>
<td>0.538</td>
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<tr>
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<td>0.6%</td>
<td>0.3%</td>
<td>0.7%</td>
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</table>

MI: myocardial infarction, PCI: percutaneous coronary intervention, CABG: coronary artery bypass graft surgery
Data are presented as mean±standard deviation of %

* Defined as ≥70% stenosis in ≥2 major epicardial coronary arteries or ≥50% stenosis of the left main coronary artery

† Defined as number of vessels with ≥70% stenosis (including LM >= 50%) minus number of vessels with successful PCI

‡ Includes left main stenosis ≥50%
Table 2
Health status and antianginal medication use, stratified by DM status

<table>
<thead>
<tr>
<th>SAQ domain scores</th>
<th>Diabetes n=367</th>
<th>No Diabetes n=713</th>
<th>P-value</th>
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<td>Baseline</td>
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<tr>
<td>Angina frequency</td>
<td>73.2±24.8</td>
<td>73.8±24.6</td>
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<tr>
<td>Quality of life</td>
<td>58.4±24.3</td>
<td>60.1±25.9</td>
<td>0.278</td>
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<tr>
<td>Physical limitation</td>
<td>77.1±23.1</td>
<td>78.4±23.3</td>
<td>0.396</td>
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<tr>
<td>Treatment satisfaction</td>
<td>94.3±9.7</td>
<td>94.1±9.9</td>
<td>0.720</td>
</tr>
<tr>
<td>12 months</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angina frequency</td>
<td>94.9±13.2</td>
<td>95.2±13.6</td>
<td>0.754</td>
</tr>
<tr>
<td>Quality of life</td>
<td>81.8±18.3</td>
<td>83.7±16.7</td>
<td>0.106</td>
</tr>
<tr>
<td>Physical limitation</td>
<td>95.1±13.0</td>
<td>96.5±10.5</td>
<td>0.047</td>
</tr>
<tr>
<td>Treatment satisfaction</td>
<td>94.4±9.8</td>
<td>93.7±11.7</td>
<td>0.333</td>
</tr>
</tbody>
</table>

Antianginal medications at discharge

<table>
<thead>
<tr>
<th>Number of medications</th>
<th>Diabetes</th>
<th>No Diabetes</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>11.5%</td>
<td>16.1%</td>
<td>0.040</td>
</tr>
<tr>
<td>1</td>
<td>51.4%</td>
<td>59.7%</td>
<td>0.008</td>
</tr>
<tr>
<td>2+</td>
<td>37.2%</td>
<td>24.1%</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Class of medications

<table>
<thead>
<tr>
<th>Class of medications</th>
<th>Diabetes</th>
<th>No Diabetes</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beta-blocker</td>
<td>81.1%</td>
<td>76.4%</td>
<td>0.077</td>
</tr>
<tr>
<td>Calcium channel blocker</td>
<td>27.9%</td>
<td>20.9%</td>
<td>0.010</td>
</tr>
<tr>
<td>Long-acting nitrate</td>
<td>32.8%</td>
<td>25.5%</td>
<td>0.012</td>
</tr>
<tr>
<td>Ranolazine</td>
<td>3.8%</td>
<td>3.0%</td>
<td>0.233</td>
</tr>
</tbody>
</table>