Differences in atherosclerotic plaque burden and morphology between type 1 and 2 diabetes mellitus as assessed by multi-slice computed tomography

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Objective: It is unclear whether coronary atherosclerotic plaque burden is similar in patients with type 1 and type 2 diabetes. Using multi-slice CT (MSCT) the presence, degree and morphology of coronary artery disease (CAD) in type 1 and type 2 diabetes were compared.

Research design and methods: Prospectively, coronary artery calcium (CAC) scoring and MSCT coronary angiography were performed in 135 asymptomatic patients (65 patients with type 1 and 70 patients with type 2 diabetes). The presence and extent of coronary atherosclerosis as well as plaque phenotype were assessed and compared between groups.

Results: No difference was observed in average CAC score (217±530 vs. 174±361) nor the prevalence of coronary atherosclerosis (65% vs. 71%) in type 1 and type 2 diabetes. However, the prevalence of obstructive atherosclerosis was higher in patients with type 2 diabetes (n=24; 34%) as compared to type 1 diabetes (n=11; 17%) (P=0.02). Also, higher mean number of atherosclerotic and obstructive plaques was observed in type 2 diabetes. In addition, the percentage of non-calcified plaques was higher in type 2 diabetes (66%) versus type 1 diabetes (27%)(P<0.001), resulting in a higher plaque burden for each CAC score as compared to type 1 diabetic patients.

Conclusions: Although CAC scores and prevalence of coronary atherosclerosis were similar between type 1 and type 2 diabetes, CAD was more extensive in the latter. Also, a relatively higher proportion of non-calcified plaques was observed in type 2 diabetes. These observations may be valuable in the development of targeted management strategies adapted to diabetes type.
Cardiovascular disease, and coronary artery disease (CAD) in particular, constitute a major cause of morbidity and mortality in patients with diabetes mellitus (DM) (1). However, management of this patient population remains challenging. Current European guidelines regard type 2 diabetes as a CAD equivalent, whereas type 1 diabetes is considered a high risk state only in presence of microalbuminuria (2). In contrast, US guidelines on primary prevention recommend stringent pharmacological therapy with lipid and blood pressure goals comparable to those in secondary prevention in all diabetic patients regardless of type (3). Notably, these guidelines are based on clinical trials in type 2 diabetes. However, CAD in type 1 diabetes, which has been studied less extensively, may have a distinct pathophysiology from type 2 diabetes. As a result, caution is indicated when extrapolating clinical observations obtained in type 2 diabetes to patients with type 1 diabetes. To date, it is unclear whether asymptomatic type 1 diabetic patients equally benefit from the current preventive treatment strategies.

In order to optimize guidelines for type 1 diabetic patients, more detailed understanding of coronary atherosclerosis in type 1 diabetes is required. Thus far, most studies have evaluated the complications and risk factors associated with microvascular disease in this population (4). Studies of CAD have mainly focused on type 1 diabetic patients with kidney failure undergoing coronary angiography preceding kidney transplantation (5). Limited information is available on the presence and morphology of CAD in asymptomatic patients with type 1 diabetes in daily clinical practice (6).

Multi-slice Computed Tomography (MSCT) allows evaluation of coronary artery calcium (CAC) score and direct assessment of coronary artery integrity. Importantly, in diabetic patients the diagnostic accuracy of MSCT coronary angiography for the detection of significant stenoses has been shown to be similar to the general population (7). In addition, the technique provides information on atherosclerotic plaque burden and to some extent on plaque composition (8-10). Previous studies with MSCT revealed an increased prevalence of non-calcified coronary plaques, which have been linked to unstable CAD, in type 2 diabetes (11, 12). However, thus far no studies have addressed plaque morphology in patients with type 1 diabetes. To improve understanding of potential differences in pathophysiology and atherosclerotic patterns as well as for development of more targeted management strategies, the evaluation of differences in plaque composition on MSCT may provide valuable information.

The purpose of the present study was therefore to explore and compare the extent, degree and morphology of coronary atherosclerosis in asymptomatic patients with type 1 and type 2 diabetes recruited from a regular diabetes clinic, by MSCT.

**RESEARCH DESIGN AND METHODS**

**Patients and design:** Hundred-thirty-five consecutive asymptomatic patients with DM were prospectively included from an ongoing registry of new patients at the diabetes outpatient clinic. Diabetic patients were referred to the cardiology outpatient clinic for cardiovascular screening. Patients were stratified as having type 1 or type 2 diabetes according to the ADA criteria (13). Plasma levels of C-peptide and auto-antibodies to islet cells, insulin and glutamic-acid-decarboxylase (GAD) were determined to distinguish between primary insulinopenia and immune destruction of beta pancreas cells (type 1 diabetes) and insulin resistance (type 2 diabetes).

Asymptomatic status was confirmed using the Rose questionnaire for angina (14). A structured interview, physical examination and laboratory analysis were acquired in all
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patients. Cardiovascular risk factors were assessed according to the following criteria: 1. positive family history of CAD (CAD in first degree family members <55 (men) or <65 (women) years of age) 2. smoking (current smoking or smoking in the last 2 years) 3. hypertension (blood pressure >140/90 mmHg or treatment with antihypertensive medication) 4. hypercholesterolemia (total cholesterol level >5.0 mmol/L or use of cholesterol lowering medication) 5. obesity (estimated by body mass index [BMI=Kg/m^2]) 6. level of glycemic control defined by plasma HbA1c (mmol/L) 7. diabetic nephropathy (urine albumin/creatinine ratio ≥ 35 mg/mmol) and 8. renal function (estimated by glomerular filtration rate [GFR]).

Non-invasive MSCT coronary angiography was performed in all patients as part of a clinical protocol. Exclusion criteria consisted of ventricular and supraventricular arrhythmia and contraindications for the use of iodinated contrast media.

**MSCT data acquisition:** Imaging was performed with a 64-slice MSCT scanner (Toshiba Medical Systems, Tokyo, Japan). In case of a heart rate ≥ 65 beats per minute, oral beta-blocking medication (metoprolol 50 mg or 100 mg) was provided 1 hour preceding the scan, if tolerated. First, a prospective CAC scan without contrast enhancement was performed, followed by MSCT coronary angiography according to protocols described previously (15).

**Assessment of CAD by MSCT:** CAC score - CAC score was assessed using dedicated software (Vitrea2, Vital Images, USA). CAC was identified as a dense area in the coronary artery exceeding the threshold of 130 Hounsfield units. Total Agatston score was determined for each patient.

**Coronary atherosclerosis** - All MSCT coronary angiograms were interpreted by two experienced observers blinded to the patients characteristics. Discrepancies in interpretation were immediately resolved by consensus. The presence of coronary atherosclerosis was evaluated by scrolling through axial images, followed by visual assessment of curved multiplanar reconstructions in at least two orthogonal planes. Coronary plaques were defined as structures >1 mm^2 within and/or adjacent to the coronary artery lumen, which could be clearly distinguished from the vessel lumen and the surrounding pericardial tissue (10).

Firstly, the presence of any atherosclerosis (≥1 plaque in the coronary tree), multi-vessel atherosclerosis (≥1 plaque in minimum two coronary arteries), and that of obstructive atherosclerosis (luminal narrowing ≥ 50%) were evaluated at patient level, in type 1 and type 2 diabetes. Thereafter, a more extensive analysis of plaque burden was obtained by registering the total number of atherosclerotic plaques and obstructive plaques for each patient (10).

In addition, plaques were classified according to phenotype: 1. non-calcified plaques (plaques with lower density than contrast-enhanced lumen), 2. calcified plaques (plaques with higher density than contrast-enhanced lumen), and 3. mixed plaques (plaques with components of low- and high density plaques) (8-10).

**Statistical Analysis:** Continuous variables were expressed as means ± standard deviation and compared between the group of patients with type 1 and type 2 diabetes, with the two-tailed independent t-test. Categorical variables were expressed as numbers (percentages) or medians (lower-quartile, upper-quartile) and compared with a χ^2-test.

Separate multivariate regression analyses with backward elimination were performed correcting for all baseline clinical characteristics including age, male gender, BMI, smoking, positive family history of CAD, hypercholesterolemia, hypertension, HbA1c, GFR and type of diabetes, to identify
independent predictors of each coronary atherosclerosis variable on MSCT.

The relationship between CAC scores and extent of coronary atherosclerosis was compared in type 1 and type 2 diabetes. Patients were further classified according to CAC scores: 1. patients without coronary calcium, 2. patients with a CAC score in the range 1-100 and 3. patients with a CAC score >100. The mean number of atherosclerotic lesions was determined for each CAC score category.

Statistical analyses were performed using SPSS software (version 12.0.1, Inc., Chicago, Illinois) and SAS software (version 6.12. SAS Institute Inc., Cary, North Carolina). P values <0.05 were considered statistically significant.

RESULTS

Patient characteristics: Hundred-thirty-five asymptomatic diabetic patients were included in the study. Mean age was 48±10 years and 79 patients (59%) were male. The study population consisted of 65 patients with type 1 and 70 patients with type 2 diabetes. Baseline characteristics are provided in Table 1. Importantly, age and gender distribution were comparable in patients with type 1 and type 2 diabetes. However, patients with type 2 diabetes had a significantly higher BMI and HbA1c and a shorter duration of diagnosed DM. Other baseline cardiovascular risk factors were similar in the two groups.

Assessment of CAD by MSCT: CAD at patient level - Results of CAD assessment by MSCT are illustrated in Table 2. Mean CAC score and prevalence of atherosclerosis were similar among patients with type 1 and type 2 diabetes. However, the prevalence of multi-vessel atherosclerosis was higher in type 2 diabetes (N=41; 59%) than in type 1 diabetes (N=19; 29%) (P=0.001). Finally, obstructive stenosis was more prevalent in type 2 diabetes (N=24; 34%) than in type 1 diabetes (N=11; 17%) (P=0.02).

Plaque analysis - Quantification of the total number of atherosclerotic plaques for each patient revealed a significantly higher mean number of lesions in patients with type 2 diabetes (9.9±11.9) as compared to patients with type 1 diabetes (3.4±4.8)(P<0.001)(Fig 1A). In addition, the mean number of obstructive plaques was significantly higher in type 2 diabetes (1.7±3.9) than in type 1 diabetes (0.5±1.4) (P=0.02) (Fig 1A).

Analysis of plaque phenotype showed a higher mean number of non-calcified plaques in type 2 diabetes (6.5±9.5) versus type 1 diabetes (1.0±1.3)(P<0.001), whereas the mean number of mixed- and calcified plaques was not significantly different. Accordingly, a higher proportion of non-calcified plaques was observed in type 2 diabetes (66%) in comparison with type 1 diabetes (27%)(P<0.001)(Fig 1B).

Relation between type of diabetes and the presence and extent of CAD: To correct for baseline characteristics, the relation between type 2 diabetes (as compared to type 1 diabetes) and the presence and extent of CAD was evaluated using multivariate regression analyses (Table 3). While type of diabetes was not related to the presence of any atherosclerosis, type 2 diabetes strongly related to the extent and degree of coronary atherosclerosis after correction for all other risk factors. In addition, an independent association was observed between the presence of type 2 diabetes and increased number of non-calcified coronary plaques.

Relation between CAC scores and atherosclerosis: Comparison of the CAC score versus coronary angiography showed that the number of atherosclerotic lesions paralleled the increase in coronary calcium (Fig 1C). However, for each CAC score category the mean number of atherosclerotic lesions was significantly higher in patients with type 2 diabetes (Fig 1C).
Similarly, the prevalence of obstructive atherosclerosis increased per CAC score category (Fig 1D). Importantly, absence of coronary calcium excluded the presence of obstructive atherosclerosis in type 1 diabetes, whereas obstructive atherosclerosis was identified in 3 patients with type 2 diabetes (9%). In patients with a CAC score ≤100, the prevalence of obstructive atherosclerosis was only 2 (4%) in type 1 diabetes, whereas 10 (19%) type 2 diabetes patients showed obstructive CAD.

CONCLUSIONS

In the present study, no significant difference was observed in the prevalence of atherosclerosis in asymptomatic patients with type 1 and type 2 diabetes. However, in type 1 diabetes, multi-vessel disease was less prevalent and a lower atherosclerotic plaque burden was observed. Moreover, the number of obstructive coronary lesions was significantly lower in patients with type 1 diabetes. Presence of type 2 diabetes (as opposite to type 1 diabetes) was shown to be an independent predictor of extent and degree of coronary atherosclerosis on MSCT angiography. Secondly, for each CAC score category a higher atherosclerotic plaque burden was observed in type 2 diabetes. This observation was explained by the high proportion of non-calcified plaques in asymptomatic patients with type 2 diabetes as compared to a high proportion of calcified plaques in type 1 diabetes.

Plaque burden: Most previous studies on the prevalence of coronary atherosclerosis have used conventional coronary angiography to examine patients with clinical suspicion of CAD and observed more extensive CAD in diabetic patients as compared to their non diabetic counterparts (16-17). Limited studies have been performed in asymptomatic diabetic patients. MSCT provides accurate non-invasive evaluation of the extent and degree of coronary atherosclerosis and may be used in patients with lower likelihood of CAD. Importantly, excellent sensitivity, specificity and negative predictive values for detection of significant stenosis have also been shown in diabetic patients (7). Thus far, the technique has been used in several studies to explore the presence and extent of CAD in patients with type 2 diabetes. Scholte et al observed a high prevalence of coronary atherosclerosis (80%), which predominantly involved more than 1 coronary artery (74%), in asymptomatic type 2 diabetic patients (12). Obstructive CAD was observed in 26% of patients, similar to observations in the current study. Thus far, no MSCT studies have reported on the presence of CAD in asymptomatic type 1 diabetic patients.

However, using magnetic resonance imaging, Kim et al previously evaluated CAD in asymptomatic patients with type 1 diabetes and observed a higher atherosclerotic plaque burden in presence of diabetic nephropathy as compared to normoalbuminuria (6). In that particular study, absence of diabetic nephropathy excluded presence of subclinical obstructive CAD. In our current study, prevalence of nephropathy was low (1%) in type 1 diabetic patients, suggesting that these patients were at relatively low risk. Interestingly however, absence of nephropathy did not exclude subclinical obstructive CAD on MSCT. Nevertheless, when compared to type 2 diabetes, the extent of atherosclerosis was less severe with a lower prevalence of multi-vessel disease and a smaller number of lesions. Moreover, a smaller proportion of patients with type 1 diabetes had obstructive atherosclerosis. Importantly, in this study presence of type 2 diabetes remained a significant predictor of the severity of atherosclerosis after correction for traditional cardiovascular risk factors including obesity, glycemic control and renal function. Accordingly, the higher atherosclerotic plaque burden in patients with
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Type 2 diabetes may warrant more aggressive anti-atherosclerotic treatment. On the other hand, MSCT coronary angiography excluded atherosclerosis in approximately 30% of both type 1 and type 2 diabetic patients. This finding raises the question whether anti-atherosclerotic medical therapy should be initiated in all asymptomatic diabetic patients and to which extent. Possibly, MSCT may be used to identify or exclude the presence of atherosclerosis and provide a basis for individually tailored therapy.

**Plaque morphology and relation with CAC:** In addition to increased severity of atherosclerosis, we also observed differences in relative plaque composition in patients with type 2 diabetes with a higher percentage of non-calcified plaques. This finding is in line with a previous study in which non-calcified plaques comprised 41% of atherosclerotic plaques in asymptomatic patients with type 2 diabetes (12). In retrospective studies, a higher proportion of non-calcified plaques has been associated with unstable CAD. Preliminary prognostic data also suggest that the presence of substantial non-calcified plaque indeed confers worse outcome (18). Accordingly, it has been suggested that these plaques represent more active stages of CAD and may be more prone to rupture (15,19). Notably, the distribution of coronary plaque phenotype has not been previously examined in type 1 diabetes. In contrast with type 2 diabetes, we found a higher proportion of calcified plaques and lower proportion of non-calcified plaques in patients with type 1 diabetes, despite similar CAC scores. As a result, plaque burden was higher for each CAC score category in type 2 diabetes as compared to type 1 diabetes.

Assessment of CAC score has been suggested as a primary step in cardiovascular risk stratification and screening of asymptomatic diabetic patients (20) as the presence of elevated CAC scores has been associated with a higher likelihood of myocardial ischemia (21). However, our current observations suggest that CAC score assessment may be more effective in identifying CAD in patients with type 1 diabetes. Indeed, absence of coronary calcium accurately excluded presence of obstructive coronary atherosclerosis in patients with type 1 diabetes. In contrast, this relation was distorted in patients with type 2 diabetes with a higher prevalence of obstructive CAD in patients without or only minor calcium. As both the prevalence of obstructive CAD and extent of CAD per CAC category were higher, it appears that CAC scores may underestimate CAD in patients with type 2 diabetes, in line with previous comparisons with non-diabetic patients (22). Accordingly, strategies using CAC scores to identify diabetic patients at higher risk should be developed with caution and should potentially be adjusted for type of diabetes.

**Study limitations:** Several limitations need to be acknowledged. The current analysis was restricted to evaluation of coronary atherosclerosis in type 1 and type 2 diabetes and the pro-atherogenic processes involved in each form of diabetes were not investigated. Also, the lack of a control group without diabetes should be acknowledged. As MSCT coronary angiography involves radiation exposure it is not feasible to perform a similar assessment in asymptomatic subjects free of cardiovascular risk. Furthermore, MSCT coronary angiography requires administration of potentially nephrotoxic contrast media, rendering the technique unsuitable for use in asymptomatic diabetic patients with severe renal dysfunction. Finally, no follow-up data were available. Indeed, the prognostic implications of our observations should be evaluated in prospective follow-up studies.

In conclusion, although CAC scores and prevalence of coronary atherosclerosis were similar between type 1 and type 2
diabetes, CAD was more extensive in the latter. Also, a relatively higher proportion of non-calcified plaques was observed in type 2 diabetes. These observations may be valuable in the development of targeted management strategies adapted to diabetes type. Possibly, MSCT angiography may be useful to identify or exclude the presence of atherosclerosis and provide a basis for individually tailored therapy.

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REFERENCES


### Table 1: Characteristics of the study population (n=135)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>DM1 (n=65)</th>
<th>DM2 (n=70)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>46.0 (38.0-54.5)</td>
<td>49.5 (45.0-57.0)</td>
<td>0.08</td>
</tr>
<tr>
<td>Men</td>
<td>42 (65%)</td>
<td>37 (53%)</td>
<td>0.17</td>
</tr>
<tr>
<td>BMI (Kg/m²)</td>
<td>23.8 (22.2-26.6)</td>
<td>28.2 (24.9-33.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Smokers</td>
<td>17 (26%)</td>
<td>14 (20%)</td>
<td>0.40</td>
</tr>
<tr>
<td>Family history of CAD</td>
<td>30 (46%)</td>
<td>37 (53%)</td>
<td>0.44</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>41 (63%)</td>
<td>50 (71%)</td>
<td>0.30</td>
</tr>
<tr>
<td>Hypertension</td>
<td>32 (49%)</td>
<td>43 (61%)</td>
<td>0.22</td>
</tr>
<tr>
<td>Duration of DM (years)</td>
<td>23.0 (9.5-33.0)</td>
<td>7.5 (2.0-13.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HbA1c (mmol/L)</td>
<td>7.6 (6.6-8.6)</td>
<td>8.3 (7.0-9.5)</td>
<td>0.04</td>
</tr>
<tr>
<td>GFR (mL/min/1.73m²)</td>
<td>101.6 (84.7-122.8)</td>
<td>98.4 (81.8-124.8)</td>
<td>0.88</td>
</tr>
<tr>
<td>Albuminuria*</td>
<td>1 (2%)</td>
<td>4 (6%)</td>
<td>0.20</td>
</tr>
</tbody>
</table>

Data are medians (lower quartile, upper quartile) or number of patients (%). BMI = body mass index, CAD = coronary artery disease, DM = diabetes mellitus. *Albuminuria was defined by a urine albumin/creatinine ratio ≥35 mg/mmol.

### Table 2: Results of MSCT coronary angiography

<table>
<thead>
<tr>
<th>Patients</th>
<th>DM1</th>
<th>DM2</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery calcium scores</td>
<td>217 ± 530</td>
<td>174 ± 361</td>
<td>0.59</td>
</tr>
<tr>
<td>Atherosclerosis</td>
<td>42 (65%)</td>
<td>50 (71%)</td>
<td>0.40</td>
</tr>
<tr>
<td>Atherosclerosis Multi-vessel</td>
<td>19 (29%)</td>
<td>41 (59%)</td>
<td>0.001</td>
</tr>
<tr>
<td>Obstructive Atherosclerosis*</td>
<td>11 (17%)</td>
<td>2 (34%)</td>
<td>0.02</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Plaques</th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>No of plaques</td>
<td>3.4 ± 4.8</td>
<td>9.9 ± 11.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No of obstructive plaques</td>
<td>0.5 ± 1.4</td>
<td>1.7 ± 3.9</td>
<td>0.02</td>
</tr>
<tr>
<td>No of non-calcified plaques</td>
<td>1.0 ± 1.3</td>
<td>6.5 ± 9.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No of mixed plaques</td>
<td>0.7 ± 1.3</td>
<td>1.1 ± 1.9</td>
<td>0.25</td>
</tr>
<tr>
<td>No of calcified plaques</td>
<td>1.8 ± 3.6</td>
<td>2.2 ± 3.9</td>
<td>0.52</td>
</tr>
</tbody>
</table>

Data are mean ± standard deviation, median (lower quartile, upper quartile) or number of patients (%). * Obstructive Atherosclerosis defined as luminal narrowing ≥50%.
Table 3: Presence of type 2 diabetes (not type 1 diabetes) as a predictor of MSCT variables. Results of multivariate analysis in a backward regression model. Predictive value of type 2 diabetes was tested in a separate multi-variate regression model for each MSCT variable.

<table>
<thead>
<tr>
<th>Patients</th>
<th>HR or β (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery calcium score*</td>
<td>-</td>
<td>NS</td>
</tr>
<tr>
<td>Atherosclerosis†</td>
<td>-</td>
<td>NS</td>
</tr>
<tr>
<td>Atherosclerosis Multi-vessel†</td>
<td>4.16 (1.76-9.93)</td>
<td>0.001</td>
</tr>
<tr>
<td>Obstructive Atherosclerosis‡†</td>
<td>4.01 (1.38-11.60)</td>
<td>0.01</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Plaques</th>
<th>HR or β (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of plaques*</td>
<td>6.82 (3.51-10.13)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No of obstructive plaques*</td>
<td>1.40 (1.32-2.48)</td>
<td>0.01</td>
</tr>
<tr>
<td>No of non-calcified plaques*</td>
<td>6.27 (3.60-8.94)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No of calcified plaques*</td>
<td>-</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS = Not significant.
* Results of analysis in a multivariate linear regression model.
† Results of analysis in a multivariate binary logistic regression model.
‡ Obstructive Atherosclerosis defined as luminal narrowing ≥50%.
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**Fig 1A:** Clustered columns demonstrating average number of lesions and obstructive lesions in type 1 and type 2 diabetes. A significantly higher mean number of lesions and obstructive lesions were observed in type 2 diabetes.

**Fig 1B:** Bargraph illustrating plaque phenotype in type 1 and type 2 diabetes. A higher percentage of non-calcified plaques was observed in type 2 diabetes.

**Fig 1C:** Clustered bargraph illustrating the increase in number of lesions for each CAC score category among patients with type 1 and type 2 diabetes. Plaque burden was significantly higher in type 2 diabetes for each CAC score category.

**Fig 1D:** Clustered bargraph demonstrating the increase in prevalence of obstructive atherosclerosis for each CAC score category among patients with type 1 and type 2 diabetes. Absence of coronary calcium excluded obstructive atherosclerosis in type 1 diabetes, but not in type 2 diabetes. Prevalence of obstructive atherosclerosis was higher in type 2 diabetes for each CAC score category.