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Difference of coronary artery disease severity, extent and plaque characteristics between patients with hypertension, diabetes mellitus or dyslipidemia

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Abstract The purpose of this study was to investigate the difference of coronary artery disease (CAD) severity and extent as well as plaque characteristics between patients with either one of hypertension (HT), diabetes mellitus (DM) or dyslipidemia (DL). We retrospectively reviewed the records of 1,161 patients (HT 442, DM 77, DL 248, no disease 394) who underwent coronary computed tomography angiography. Stenosis severity was classified as normal, non-obstructive (1–49 % stenosis), moderate (50–69 % stenosis) or severe (≥70 % stenosis). Segment involvement score (SIS) and segment severity score (SSS) was calculated. We defined patients at risk as patients with obstructive CAD or non-obstructive CAD with extensive disease (SIS ≥ 5). Plaque characteristics were evaluated including positive remodeling, low attenuation and spotty calcification. Obstructive CAD was most frequent in DM patients, followed by HT and DL patients (34, 19 and 15 %, respectively, $p < 0.0001$). DM patients had more extensive disease than HT and DL patients (SIS 3.1 vs 2.1 vs 1.4, SSS 4.0 vs 2.7 vs 2.0). DM patients were more at risk than HT and DL patients ($p < 0.05$). The prevalence of positive remodeling, low attenuation and spotty calcium were all highest in DM patients ($p < 0.005$, vs HT and DL), while low attenuation was more frequent in DL than HT patients ($p < 0.005$). The median calcium score of HT and DM patients were higher than DL patients ($p < 0.01$ and $p < 0.005$, respectively), while no significant difference was observed between HT and DM patients. In conclusion, DM patients possessed more high risk plaque and obstructive as well as extensive CAD compared with HT and DL patients. Coronary calcification was similarly high in HT and DM patients. Low attenuation plaque was more frequent in DL than HT patients.

Keywords Coronary artery disease · Diabetes mellitus · Dyslipidemia · Hypertension · Plaque

Introduction

Acute coronary syndrome is one of the leading causes of mortality in Japan as well as in western countries. Hypertension (HT), diabetes mellitus (DM) and dyslipidemia (DL) have been identified as risk factors for coronary artery disease (CAD) or the progression of vulnerable plaques [1–4]. Rupture of vulnerable plaques followed by severe stenosis of coronary arteries result in acute coronary syndrome [2].

Recent advances in computed tomography (CT) enabled the visualization of vulnerable plaques. These high risk plaques include positive remodeling, low attenuation and spotty calcification [5]. Growing evidence have shown that especially DM is related with the progression of high risk plaques [2–4, 6, 7]. However, most patients in these studies also have other risk factors such as HT and DL. The differences in the effect of HT, DM and DL on CAD and plaque characteristics have not been investigated.

Thus the purpose of this study was to evaluate the difference of CAD severity and extent as well as plaque characteristics between patients who have either one of HT, DM or DL. We also investigated patients without these diseases as a reference standard.

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Methods

This retrospective study was approved by the local ethics committee, and the requirement for informed consent to participate in this study was waived.

Patients

The records of 2,199 consecutive patients who underwent coronary CT angiography (CTA) from December 2012 to March 2014 were retrospectively examined. These patients had several risk factors of CAD with chest pain and/or dyspnea, abnormal results of electrocardiogram, cardiac echo or treadmill test. The exclusion criteria were as follows: patients with known CAD (n = 171); poor image quality due to severe artifacts (n = 9); risk factors unknown (n = 11). Of the 2,008 patients, 847 patients had more than one of HT, DM or DL. The final study group included 1,161 patients (Fig. 1).

Clinical history was obtained by the referring physician, and the following conventional cardiovascular risk factors were assessed: (1) HT (blood pressure ≥140/90 mmHg and/or use of antihypertensive drugs); (2) DM (fasting glucose level ≥126 mg/dl or HbA1c ≥6.5 % or need for insulin or oral antidiabetic drugs) [8]; (3) DL (low-density lipoprotein ≥140 mg/dl or high-density lipoprotein <40 mg/dl or triglyceride ≥150 mg/dl or need for anti-lipidemic drugs) [9]; (4) smoking (currently or previously); (5) family history of CAD. The pretest cardiovascular risk was assessed by the Morise pre-test score [10]. The Morise pre-test score is a clinical risk score including age, gender, angina history, estrogen status in women, HT, DM, DL, smoking, family history and obesity.
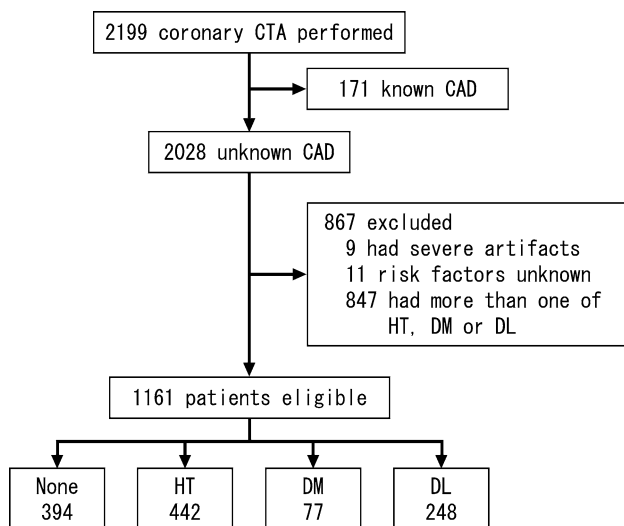

Fig. 1 A total of 2,199 patients were screened and 1,161 patients were included in the final study group. *CAD* coronary artery disease, *CTA* computed tomography angiography, *DL* dyslipidemia, *DM* diabetes mellitus, *HT* hypertension

CT data acquisition

All patients underwent CTA with the 64-row CT (Brilliance 64; Philips, Tokyo, Japan). Each patient first underwent unenhanced prospectively electrocardiogram-gated calcium scoring with 120 kVp, 196 mA and 2.5 mm collimation. Images were obtained at 40 % or 70 % of the R–R interval depending on the heart rate and were reconstructed with a slice thickness of 2.5 mm and increment of 2.5 mm.

Coronary CTA was performed by retrospectively electrocardiogram-gated helical scan. The scanning parameters were as follows: detector configuration, 64 × 0.625 mm; tube potential, 120 kVp; tube current–time product, 800–1,050 mAs, depending on the body weight; gantry rotation time, 420 ms; and helical pitch, 0.2. The dose length product was derived from the CT console.

The patients received 21.0 mg I/kg/s of iopamidol 370 mg I/ml (Iopamiron 370; Bayer, Osaka, Japan). Contrast medium was injected for acquisition duration plus 7 s, followed by a 30-ml saline flush. Bolus tracking method was performed to determine the scan timing. The scan started 6 s after the descending aorta reached 100 Hounsfield Unit (HU).

Eighty-two patients were receiving an oral β-blocker as part of baseline medication, and an oral β-blocker (20 mg of metoprolol) was administered to 683 outpatients with heart rate >65 beats per minute (bpm). The patients were told to take the medicine 1 h prior to CTA. If the heart rate was over 65 bpm, as many as 12.5 mg of landiolol (Corebeta; Ono Pharmaceutical, Tokyo, Japan) was given intravenously. No patient had contraindications for β-blockers, and no β-blocker side effects were observed or reported. All patients received 0.3 mg sublingual nitroglycerin (Nitropen; Nippon Kayaku, Tokyo, Japan) before imaging.

For each patient, a senior technologist determined the phase with minimum artifacts at the CT console. Multiple phases were reconstructed when artifacts resisted in the image. The reconstructed slice thickness was 0.67 mm, and the increment was 0.33 mm. Images were reconstructed by using a cardiac sharp kernel. For processing, images were transformed to a workstation (Syanpse Vincent; Fuji, Tokyo, Japan).

Image analysis of CTA

Calcium scoring

Calcified lesions with a minimum area of three pixels and a minimum CT number of 130 HU were scored by using the algorithm developed by Agatston et al. [11] Lesion scores from the left main, left anterior descending, left circumflex

and right coronary arteries were summed to determine a total calcium score.

Stenosis analysis

The Society of Cardiovascular Computed Tomography 18-segment classification was applied for analysis of CTA data [12]. All segments with a diameter of at least 1.5 mm at their origin were included and each lesion was visually classified into four categories: normal, non-obstructive (1–49 % stenosis), moderate (50–69 % stenosis) and severe (≥70 % stenosis). Obstructive CAD was defined by a presence of ≥50 % stenosis in 0, 1, 2 or 3 coronary artery vessels. We also evaluated the impact of the extent of non-obstructive and obstructive CAD [13]. Segment involvement score (SIS) was calculated as a measure of overall coronary artery plaque distribution. SIS was defined as the total number of coronary segments with plaque, irrespective of the degree of luminal stenosis within each segment (minimum = 0; maximum = 18). Segment severity score (SSS) was also calculated as a measure of the overall coronary artery plaque burden. SSS was defined as the sum of each coronary segment as none to severe plaque (0–3) based on the extent of obstruction with a total score ranging from 0 to 54. We defined patients at risk as patients with obstructive CAD or non-obstructive CAD with extensive disease (SIS ≥ 5) [14].

Plaque analysis

Atherosclerotic plaque was defined as any clearly discernable structure larger than 1 mm^2 which could be assigned to the coronary artery wall in at least two independent image planes. Remodeling index was defined by dividing the vessel diameter at the plaque site by the diameter at the reference site. Positive remodeling was reported when the remodeling index was larger than 1.1. Minimum CT number of the plaque was measured using several circular regions of interest (area of 1 mm^2) at each plaque area. When the minimum CT number was <30 HU, the plaque was referred to as low attenuation plaque [15]. Calcification was classified as spotty when the size was <3 mm and occupied only one side on cross sectional images [5]. We assessed all plaques in the coronary arteries and analysis was performed by a per-patient basis.

Statistical analysis

Continuous variables are shown as mean ± standard deviation and categorical variables as number (%). The analysis of variance and Tukey's HSD test were used to compare continuous variables. The Chi square (χ^2) test and Steel–Dwass test were used to compare categorical variables or skewed variables.

Logistic regression analysis was used to assess the relationship of HT, DM and DL with plaque characteristics and stenosis severity and extent.

All statistical analyses were performed using JMP software (version 9.0.0; SAS, Cary, NC). A p value <0.05 was deemed to indicate significance.

Results

Of the 1,161 patients, 442 patients (38 %) had HT, 77 patients (7 %) had DM, 248 patients (21 %) had DL and the remaining 394 patients (34 %) had no disease (Table 1). All patients in the DM group were type 2 DM. Patients with HT and DM were more often male and older compared with the remaining groups. Body weight was higher when any disease existed. The status of smoking did not differ between the groups. Patients were more symptomatic in the DL group. The Morise risk score was higher when any disease existed. About 80 % of the patients had an intermediate risk.

The average and standard deviation of the dose length product was 1,137 ± 156 mGy cm. This was quite high because we used an earlier scanner thus dose reducing techniques such as prospective triggering, dose modulation or iterative reconstruction were not available.

Stenosis analysis

Single-vessel disease was present in 104 patients (9 %), 2-vessel disease was present in 60 patients (5 %), and 3-vessel or left main disease was present in 23 patients (2 %). The presence of any disease resulted in higher prevalence of obstructive CAD (Table 2, $p < 0.0001$). Obstructive CAD was most frequent in the DM group (34 %), while the proportion of obstructive CAD was similar between the HT and the DL group (19 and 15 %, respectively). The SSS and SIS were highest in the DM group, followed by HT group, DL group and patients without disease with a significant difference ($p < 0.0001$). The proportion of patients at risk was highest in the DM group (45 %), followed by the HT group (25 %, $p < 0.05$, vs DM), DL group (18 %, $p < 0.05$, vs DM) and patients without disease (6 %, $p < 0.0005$, vs DM) (Table 2; Fig. 2). When no disease existed, 94 % of the patients had no obstructive or extensive CAD.

Logistic regression analysis showed that HT, DM and DL were all related with obstructive CAD ($p < 0.0001$) and patients at risk ($p < 0.0001$) (Table 3). DM was highly associated with obstructive CAD and patients at risk (12.0 and 13.4, $p < 0.0001$). The relationship of HT and DL with obstructive CAD and patients at risk was similar with slightly higher odds ratio in HT patients (5.7 vs 4.4, $p = 0.22$ and 5.5 vs 3.5, $p = 0.02$, respectively).

Table 1 Patient demographics

Parameter	None	HT	DM	DL	P
Number of patients	394	442	77	248	
Male/female	196/198	249/193	47/30	102/146	0.0004*
Age (years)	61.4 ± 0.6	67.5 ± 0.6	67.3 ± 1.4	63.4 ± 0.8	<0.0001*
Body weight (kg)	57.7 ± 0.6	60.4 ± 0.6	61.6 ± 1.3	59.1 ± 0.7	0.003*
Body mass index (kg/m²)	22.2 ± 0.2	23.2 ± 0.2	23.7 ± 0.4	23.1 ± 0.2	<0.0001*
Heart rate (bpm)	60.5 ± 0.6	62.6 ± 0.5	61.9 ± 1.3	61.1 ± 0.7	0.06
Coronary risk factor					
Smoking	123 (31)	152 (34)	29 (38)	86 (35)	0.61
Family history	67 (17)	62 (14)	8 (10)	65 (26)	0.0003*
Chest pain					
Typical	13 (3)	14 (3)	3 (4)	17 (7)	0.01*
Atypical	79 (20)	90 (20)	13 (17)	59 (24)	
Noncardiac	94 (24)	77 (17)	14 (18)	62 (25)	
Asymptomatic	208 (53)	261 (59)	47 (61)	110 (44)	
Morise risk score	10.0 ± 0.1	12.0 ± 0.1	13.0 ± 0.3	12.1 ± 0.2	<0.0001*
Low (0–8)	87 (22)	30 (7)	2 (3)	21 (8)	
Intermediate (9–15)	298 (76)	374 (85)	63 (82)	200 (81)	
High (16–24)	9 (2)	38 (8)	12 (15)	27 (11)	
Medication					
Aspirin	10 (3)	62 (14)	4 (5)	19 (8)	
Beta blocker	0 (0)	82 (19)	0 (0)	0 (0)	
Calcium blocker	0 (0)	217 (49)	0 (0)	0 (0)	
ACE inhibitor	0 (0)	17 (4)	0 (0)	0 (0)	
ARB	0 (0)	171 (39)	0 (0)	0 (0)	
Nitrol	0 (0)	23 (5)	0 (0)	0 (0)	
Diuretics	0 (0)	53 (12)	0 (0)	0 (0)	
Statin	0 (0)	0 (0)	0 (0)	75 (30)	
Insulin	0 (0)	0 (0)	9 (12)	0 (0)	

Numbers are reported as average ± standard deviation or N (%)

ACE angiotensin converting enzyme, *ARB* angiotensin receptor blocker, *DL* dyslipidemia, *DM* diabetes mellitus, *HT* hypertension

* Statistically significant, $p < 0.05$

Plaque analysis and calcium score

High risk plaque increased when any disease existed. DM patients had the highest prevalence of positive remodeling (43 %), low attenuation (28 %) and spotty calcium (64 %) ($p < 0.005$, vs all groups). The frequency of positive remodeling and spotty calcification was similar between HT and DL, while low attenuation was more frequent in DL compared with HT (11 % vs 5 %, $p < 0.005$) (Table 4). When no disease existed, positive remodeling or low attenuation appeared in only 1 % of the patients.

Regression analysis showed that DM was highly related with positive remodeling (odds ratio 58.4, $p < 0.0001$) and low attenuation plaque (odds ratio 29.2, $p < 0.0001$) (Table 5). The relationship of HT and DL was similar for positive remodeling ($p = 0.77$). DL was more related with low attenuation plaque compared with HT (odds ratio 9.9 vs 3.7, $p = 0.001$). Although DM and HT were related with spotty calcium, the relationship was lower than positive remodeling and low attenuation.

The calcium score increased when any disease existed (Table 4; Fig. 3). The median calcium score of HT and DM were higher than DL ($p < 0.01$ and $p < 0.005$, respectively), while no significant difference was observed between HT and DM.

Discussion

The present study evaluated the difference of CAD severity and extent as well as plaque characteristics between patients who have either one of HT, DM or DL. DM was highly related with obstructive and extensive CAD. DM was also highly related with high risk plaque. Although the prevalence and extent of CAD was slightly higher in HT patients than DL patients, low attenuation plaque was more frequent in DL patients than HT patients. Calcium score was similarly high in HT and DM patients compared with DL patients.

Obstructive CAD detected by coronary CTA is related with increased mortality [16]. Recent studies have shown

Table 2 Comparison of distribution and severity of CAD between patients with or without HT, DM or DL

	None	HT	DM	DL
Per vessel CAD*				
0VD	378 (96)	356 (81)	51 (66)	209 (85)
1VD	11 (2)	53 (12)	17 (22)	23 (9)
2VD	2 (1)	20 (4)	5 (7)	13 (5)
3VD or left main	3 (1)	13 (3)	4 (5)	3 (1)
Disease extent				
SIS	0.7 ± 1.5	2.1 ± 2.9[†,‡,§]	3.1 ± 3.5[‡]	1.4 ± 2.3[†,‡]
SSS	0.8 ± 1.8	2.7 ± 4.3[†,‡,§]	4.0 ± 4.8[‡]	2.0 ± 3.7[†,‡]
Disease severity and extent*				
No CAD	273 (69)	191 (43)	21 (27)	140 (56)
Non-obstructive, SIS <5	98 (25)	139 (32)	21 (27)	64 (26)
Non-obstructive, SIS ≥5	9 (2)	28 (6)	9 (12)	5 (2)
Obstructive, SIS <5	11 (3)	35 (8)	13 (17)	17 (7)
Obstructive, SIS ≥5	3 (1)	49 (11)	13 (17)	22 (9)
Patients at risk[‖]	23 (6)	112 (25)[†,‡]	35 (45)[‡]	44 (18)[†,‡]

Numbers are reported as N (%) or average ± standard deviation

CAD coronary artery disease, *DL* dyslipidemia, *DM* diabetes mellitus, *HT* hypertension, *SIS* segment involvement score, *SSS* segment severity score, *VD* vessel disease

* Difference in distribution was statistically significant, $p < 0.0001$

[†] $p < 0.05$ versus DM

[‡] $p < 0.0005$ versus none

[§] $p < 0.005$ versus DL

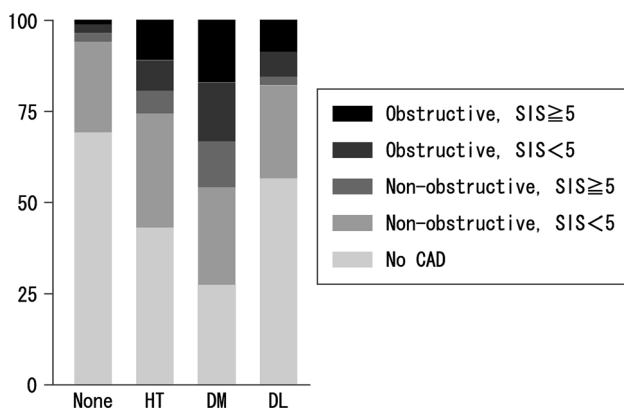[‖] Patients with obstructive CAD or non-obstructive CAD with SIS ≥ 5

Fig. 2 Prevalence of obstructive CAD as well as non-obstructive CAD with SIS ≥ 5 was highest in patients with DM. The frequency of obstructive CAD or non-obstructive CAD with SIS ≥ 5 was significantly different ($p < 0.05$) for all pairwise comparisons except for HT and DL. *CAD* coronary artery disease, *DL* dyslipidemia, *DM* diabetes mellitus, *HT* hypertension, *SIS* segment involvement score

that among patients with non-obstructive CAD, patients with extensive disease experienced a higher rate of mortality compared with non-extensive disease [14]. The hazard ratio for major adverse cardiovascular events in patients with non-obstructive extensive CAD was similar with patients having obstructive non-extensive CAD (3.1 vs 3.0, patients with no CAD as a reference). The present study showed that both obstructive disease and extensive disease were more frequent in DM patients compared with HT and DL patients. A total of 45 % of the DM patients had either obstructive or extensive CAD and were potentially at risk for cardiovascular events.

Although HT, DM and DL were all independent predictors of low attenuation plaque, the relationship of DM was stronger compared with HT and DL. Low attenuation plaques are related with acute coronary syndrome [17], and statin have been known to lower the progression of these plaques [18]. CT could further differentiate DM or HT patients who might benefit from statin treatment to prevent acute coronary syndrome even when the serum lipid level is within normal limit.

Kamimura et al. [2] showed that the prevalence of either low attenuation plaque or positive remodeling was 27 % in asymptomatic DM patients. Another study by Loffroy et al. [4] showed that the prevalence of low attenuation plaque was 7.5 % in asymptomatic DM patients. However, about 70 % of these patients also had HT or DL. Because we only included patients with only one disease, the direct relationship of each disease on the characteristics of plaque could be investigated by the present study. The present

Table 3 Association between HT, DM and DL with obstructive CAD and patients at risk

| | Odds ratio | p | | | |
	(95 % CI)	Versus without disease	Versus HT	Versus DM	Versus DL
Obstructive CAD					
HT	5.7 (3.4–10.3)	<0.0001[†]	N/A	0.007*	0.22
DM	12.0 (6.1–24.4)	<0.0001[†]	0.007*	N/A	0.0009*
DL	4.4 (2.4–8.3)	<0.0001[†]	0.22	0.0009*	N/A
Patients at risk*					
HT	5.5 (3.5–9.0)	<0.0001[†]	N/A	0.0005*	0.02*
DM	13.4 (7.3–25.2)	<0.0001[†]	0.0005*	N/A	<0.0001*
DL	3.5 (2.1–6.0)	<0.0001[†]	0.02*	<0.0001*	N/A

Odds ratio were calculated as patients with no disease as a reference standard

Obstructive CAD was defined as CT stenosis ≥50 %

CAD coronary artery disease, *CI* confidence interval, *DL* dyslipidemia, *DM* diabetes mellitus, *HT* hypertension, *N/A* not available

* Patients with obstructive CAD or non-obstructive CAD with SIS ≥ 5

[†] Statistically significant

Table 4 Comparison of plaque characteristics and calcium score between patients with or without HT, DM or DL

	None	HT	DM	DL
Plaque characteristic				
Positive remodeling	5 (1)	38 (9)*	33 (43)[†]	23 (9)*
Low attenuation	5 (1)	20 (5)*,[‡]	21 (28)[†]	28 (11)*
Spotty calcium	109 (28)	189 (43)*	49 (64)[†]	85 (35)
Any of the three	112 (28)	206 (47)*	57 (74)[†]	99 (40)*
Calcium score	0 (0–14.7)	7.6[§] (0–149.3)	33.3[‖] (0–236.7)	0* (0–62.8)

Numbers are reported as *N* (%) or median (interquartile range)

DL dyslipidemia, *DM* diabetes mellitus, *HT* hypertension

* $p < 0.05$ versus none

[†] $p < 0.005$ versus none, HT, DL

[‡] $p < 0.005$ versus DL

[§] $p < 0.01$ versus none, DL

[‖] $p < 0.005$ versus none, DL

study showed that 43 % of DM patients had positive remodeling and 28 % had low attenuation plaque, which was more frequent than the previous studies. This might be because the present study included symptomatic patients. Amano et al. [19] showed that metabolic syndrome was related with increased lipid rich plaque (odds ratio 4.00). The odds ratio for low attenuation plaque in our study ranged from 3.7 with HT to 29.2 with DM, which was higher than the results of Amano et al. Metabolic syndrome was defined as a complex of HT, DM and DL. Patients without metabolic syndrome might have had one of HT, DM or DL hence the odds ratio of metabolic syndrome might have been lower than the present study.

A number of risk factors including HT, DM, DL and age are known to contribute to the development of coronary artery calcification [20]. This is compatible with our results

which showed that patients with HT, DM or DL had higher calcium score than patients without. The present study further showed HT and DM patients were more at risk for coronary calcification than DL patients. Increased circumferential wall tension by HT cause intimal calcification [20], which might play a role for the progression of atherosclerosis [21]. In diabetic patients, advanced glycation end-products could promote mineralization of microvascular pericytes, which results in both intimal and medial calcification [20].

Our study possessed some limitations. The groups were fairly unbalanced in sample size. However, selecting patients matched with other variables might cause unexpected selection bias. Secondly, the stenosis severity was not tested by coronary angiography. However, we believe that the diagnostic accuracy of 64-row CT is optimal with

Table 5 Association between HT, DM and DL with high risk plaque

	Odds ratio	p			
	(95 % CI)	Versus without disease	Versus HT	Versus DM	Versus DL
Positive remodeling					
HT	7.3 (3.1–21.4)	<0.0001*	N/A	<0.0001*	0.77
DM	58.4 (23.5–177)	<0.0001*	<0.0001*	N/A	<0.0001*
DL	8.0 (3.2–24.0)	<0.0001*	0.77	<0.0001*	N/A
Low attenuation					
HT	3.7 (1.5–11.2)	0.004*	N/A	<0.0001*	0.001*
DM	29.2 (11.4–90.2)	<0.0001*	<0.0001*	N/A	0.001*
DL	9.9 (4.1–29.5)	<0.0001*	0.001*	0.001*	N/A
Spotty calcium					
HT	2.0 (1.5–2.6)	<0.0001*	N/A	0.0007*	<0.0001*
DM	4.6 (2.8–7.7)	<0.0001*	0.0007*	N/A	<0.0001*
DL	1.3 (0.97–1.9)	0.07	<0.0001*	<0.0001*	N/A

Odds ratio are calculated as patients without any disease as a reference standard

CI confidence interval, *DL* dyslipidemia, *DM* diabetes mellitus, *HT* hypertension, *N/A* not available
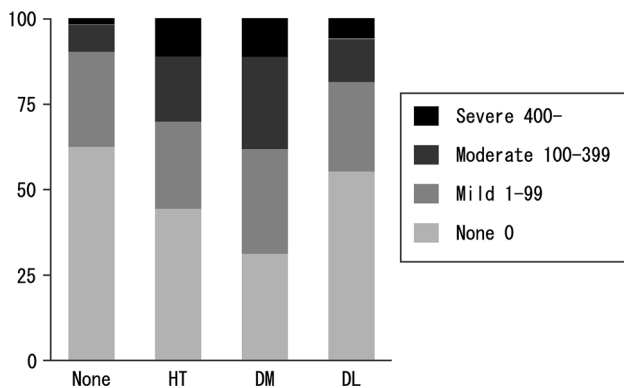
* Statistically significant

Fig. 3 The calcium score category was higher in HT and DM compared with the remaining two groups. The categories were similar in patients with HT or DM and patients with DL or with no disease. *DL* dyslipidemia, *DM* diabetes mellitus, *HT* hypertension

its high negative predictive value. Thirdly, we determined low attenuation plaque based on the density of plaque components. The density of lipid core might change with reconstruction kernel, slice thickness [22] or lumen attenuation [23], thus it is difficult to exclude contamination of fibrous or thrombus components in low attenuation plaques. Finally, because all patients in this study received coronary CTA only once, it is difficult to know the progression of each CAD over time. Also, disease duration and severity might be different between HT, DM and DL because long standing HT may lead to symptoms even in the absence of obstructive coronary disease whereas DM may have no angina despite obstructive disease. However, the results of this study show the presence of CAD at the presence of the outpatient department.

In conclusion, DM patients possessed more high risk plaque and obstructive as well as extensive CAD compared with HT and DL patients. Coronary calcification was similarly high in HT and DM patients. Low attenuation plaque was more frequent in DL patients than HT patients.

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