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Research paper

Percent atheroma volume: Optimal variable to report whole-heart atherosclerotic plaque burden with coronary CTA, the PARADIGM study

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ABSTRACT

Background and aims: Different methodologies to report whole-heart atherosclerotic plaque on coronary computed tomography angiography (CCTA) have been utilized. We examined which of the three commonly used plaque burden definitions was least affected by differences in body surface area (BSA) and sex.
Methods: The PARADIGM study includes symptomatic patients with suspected coronary atherosclerosis who underwent serial CCTA > 2 years apart. Coronary lumen, vessel, and plaque were quantified from the coronary tree on a 0.5 mm cross-sectional basis by a core-lab, and summed to per-patient. Three quantitative methods of plaque burden were employed: (1) total plaque volume (PV) in mm^3, (2) percent atheroma volume (PAV) in %

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[which equaled: PV/vessel volume * 100%], and (3) normalized total atheroma volume (TAV_{norm}) in mm^3 [which equaled: PV/vessel length * mean population vessel length]. Only data from the baseline CCTA were used. PV, PAV, and TAV_{norm} were compared between patients in the top quartile of BSA vs the remaining, and between sexes. Associations between vessel volume, BSA, and the three plaque burden methodologies were assessed.

Results: The study population comprised 1479 patients (age 60.7 ± 9.3 years, 58.4% male) who underwent CCTA. A total of 17,649 coronary artery segments were evaluated with a median of 12 (IQR 11–13) segments per-patient (from a 16-segment coronary tree). Patients with a large BSA (top quartile), compared with the remaining patients, had a larger PV and TAV_{norm}, but similar PAV. The relation between larger BSA and larger absolute plaque volume (PV and TAV_{norm}) was mediated by the coronary vessel volume. Independent from the atherosclerotic cardiovascular disease risk (ASCVD) score, vessel volume correlated with PV ($P < 0.001$), and TAV_{norm} ($P = 0.003$), but not with PAV ($P = 0.201$). The three plaque burden methods were equally affected by sex.

Conclusions: PAV was less affected by patient's body surface area then PV and TAV_{norm} and may be the preferred method to report coronary atherosclerotic burden.

1. Introduction

Coronary computed tomography angiography (CCTA) allows for accurate evaluation of atherosclerotic plaque, and its presence, extent and severity are strongly related to future major adverse cardiovascular events.[1] More recently, studies have demonstrated the ability of CCTA to accurately quantify the total coronary plaque burden (whole-heart) and to assess changes in plaque burden on CCTA over time.[2–5]

Plaque burden with intravascular ultrasound (IVUS) has traditionally been reported using percent atheroma volume (PAV)—defined as the total plaque volume (PV)/vessel volume x 100%—or total atheroma volume normalized to vessel length (TAV_{norm})—defined as PV/vessel length x mean population vessel length.[6,7] Normalization is performed to adjust for differences in the length of the interrogated coronary segments. Compared with IVUS, CCTA offers the ability to quantify and characterize atherosclerotic plaque from all coronary artery segments simultaneously, albeit at a lower spatial resolution. This allows for whole-heart atherosclerotic plaque assessment by total PV without normalization for vessel length or volume.[2,3] To date, no study—either by CCTA or IVUS—has examined how PV, PAV or TAV_{norm} are influenced by scenarios that affect vessel length and vessel volume. Previous histological and imaging data has shown that the size of arteries is dependent on body surface area (BSA) and sex.[8,9]

The ideal method for determining atherosclerotic plaque burden would be least affected – most stable – to patient demographics. PV (no normalization of plaque), PAV (normalization for vessel volume), and TAV_{norm} (normalization for vessel length) may vary according to BSA and sex – which are known to affect vessel size (length and volume). In a large, prospective CCTA study with whole-heart quantification of coronary plaque, lumen and vessel wall, we aimed to determine the optimal way to report coronary atherosclerotic plaque burden.

2. Methods

2.1. Patients

The PARADIGM (Progression of AtheRosclerotic PlAque DetermIned by Computed TomoGraphic Angiography IMaging) study is a dynamic, multicenter, observational registry which prospectively collects clinical, procedural, and follow-up data for MACE from patients undergoing clinically indicated CCTA.[5] The study was approved by the institutional review board of all participating centers. The PARADIGM registry enrolled 2252 patients from 13 sites in 7 countries (Brazil, Canada, Germany, Italy, Portugal, South Korea and the United States) between 2003 and 2015. Patients underwent a baseline and follow-up CCTA at least 2 years apart, using ≥64-slice CT-scanners for the evaluation of CAD. For the current analysis, patients with previous coronary revascularization or uninterpretable CCTA for quantitative analysis were excluded, leaving 1479 patients in the current study. Only data from the baseline CCTA were used.

2.2. CCTA imaging and analysis protocol

CCTAs were acquired using ≥64 detector row CT scanners, with image acquisition protocols in accordance with the Society of Cardiovascular Computed Tomography guidelines, including the systematic administration of beta-blocker and nitroglycerin before acquisition.[10] CCTA DICOM files from each participating site were transferred to a Central Core Laboratory for blinded image analysis. Level III experienced CTA readers from the Central Core Laboratory, masked to patient clinical and demographical characteristics, performed qualitative and semi-quantitative CCTA analysis using dedicated software (QAngioCT Research Edition v2.1.9.1; Medis Medical Imaging Systems, Leiden, the Netherlands).[5] CCTA imaging analysis methodology included routine measures of inter- and intra-observer variability, as has been described previously: intra-class correlation coefficient 0.992 for inter-observer, and 0.996 for intra-observer plaque volume repeatability.[5,11] In brief, measures of coronary plaque, lumen and vessel wall were assessed in all coronary segments ≥2 mm in diameter, using standardized width/level display settings adjusted for aortic contrast attenuation. Contours were manually adjusted where needed. Coronary plaque was defined as any tissue ≥1 mm^2 within or adjacent to the lumen that can be distinguished from the surrounding pericardial tissue, epicardial fat or the coronary artery lumen.[1] Measurements were performed per-segment on a 0.5 mm cross-sectional basis according to a modified 16-segment coronary tree model.[12] Lumen and vessel wall contours were overlapping in segments without plaque. To obtain per-patient measures, the data for all coronary artery segments were summed.

2.3. Quantitative plaque burden reporting

Coronary artery plaque burden was reported according to the following three previously described definitions in the CCTA and IVUS literature: Plaque volume (PV), percent atheroma volume (PAV) and total atheroma volume normalized for vessel length (TAV_{norm}), with definitions as described below.[2–4,6,13–17]

Plaque volume (PV) = Summation of plaque from all segments

Percent atheroma volume (PAV) = (Plaque volume / Vessel volume) * 100%

Total atheroma volume normalized (TAV_{norm}) = (Plaque volume) / Vessel length) * mean population vessel length

2.4. PV, PAV, and TAV_{norm}

PV is summation of plaque from all analyzable segments from the 16-segment coronary tree. PAV and TAV_{norm} are similar to PV but have been normalized for either the length of the vessel (TAV_{norm}) or the volume (PAV) respectively.

We examined how differences in vessel volume, body surface area (BSA), and sex affect PV, PAV, and TAV_{norm}. Previous imaging and histopathological data have demonstrated that the size (volume and length) of arteries is larger with increasing BSA and male sex.[8,9] Given the different types of normalization (or no normalization for PV), alterations in BSA and sex may affect PV, PAV, and TAV_{norm}. An example of how PV, PAV and TAV_{norm} are affected by vessel volume, BSA, and sex is shown in Fig. 1.

2.5. Statistical analysis

Continuous variables were expressed as mean ± standard deviation (SD) or median (25th – 75th interquartile range) if non-normally distributed and categorical variables as counts (percentage). Because of their left skewed distribution, all plaque data are provided as medians with interquartile range. Normality of data distribution was assessed by review of histograms and Q-Q plots. Continuous variables were compared with the independent T-test or Mann-Whitney *U* test as appropriate. Categorical variables were compared with the chi-square test as appropriate. The variability of PV, PAV, and TAV_{norm} was assessed according to vessel volume, BSA, and sex, with dichotomizing vessel volume and BSA according to their 75th and 50th percentile. PV, PAV, and TAV_{norm} were compared between the BSA and sexes. The association between BSA and vessel volume was examined with a scatter plot and the Pearson's correlation coefficient was determined. The atherosclerotic cardiovascular disease score (ASCVD) was calculated.[18] Linear regression analyses were performed between vessel volume and the 3 plaque burden methodologies (log-transformed) – adjusting for ASCVD score – to evaluate the effect of vessel size with plaque burden defined by the 3 different methods. Two-sided P-values < 0.05 were considered statistically significant. All analyses were performed with SAS (version 9.4; SAS Institute Inc., Cary, NC) and R 3.3.0 (R Development Core Team, 2016).

3. Results

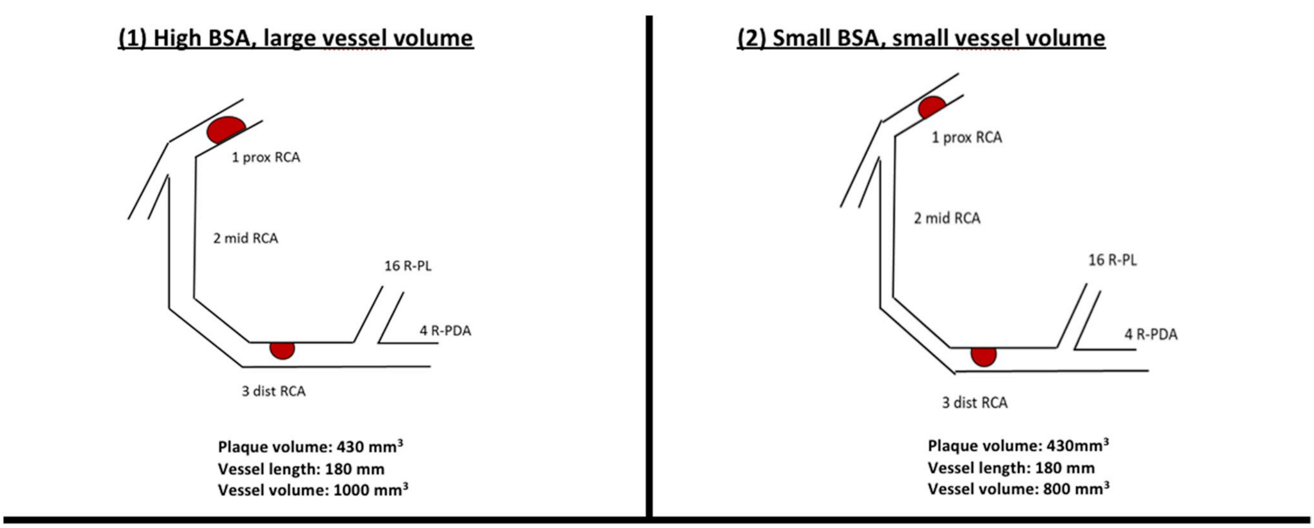
3.1. Patients

In total, 1479 patients were included with a mean age of 60.7 ± 9.3 years and 58.4% was male. A median of 12 (IQR 11–13) coronary segments were analyzed per patient (17,649 segments in total). The clinical indication for CCTA was evaluation of chest pain in 83.1% (Table 1) The mean height, weight, BMI, and BSA were 167 ± 9.7 cm, 71 ± 13 kg, 25.3 ± 3.3 kg/m^2, and 1.81 ± 0.21 m^2, respectively. Most patients had non-obstructive CAD (62.5%) and 24.0% of the patients had more than 5 diseased coronary artery segments (Table 1).

3.2. PV, PAV, and TAV_{norm} according to BSA, and sex

Patients in the top quartile of vessel volume, compared with the remaining patients, had similar PAV, but greater PV and a trend towards greater TAV_{norm}: 2.0% (IQR 0.32–5.7) vs 2.4% (IQR 0.0–7.1) P = 0.445, 73.8 mm^3 (IQR 9.8–197.6) vs 40.7 mm^3 (IQR 0.0–118.7) P < 0.001, and 49.2 mm^3 (IQR 7.2–143.4) vs 42.9 mm^3 (IQR 0.0–135.1) P = 0.075, respectively (Table 2). Similarly, when categorizing patients into the top quartile of BSA vs the remaining patients, PAV was similar, but PV and TAV_{norm} were significantly larger in patients with the largest BSA, Table 3. The results remained unchanged when vessel volume and BSA were dichotomized by the 50th percentile (Appendix Tables 1 and 2).

PV, PAV, and TAV_{norm} were similarly affected by sex: plaque burden by all 3 methodologies was higher in men, Table 4. In patients at low risk (ASCVD score < 7.5) plaque burden by all 3 methodologies was



Right panel RCA vs left panel RCA:

Plaque volume (PV)	=
Percent atheroma volume (PAV)	↑
Vessel length normalized total atheroma volume (TAV_{norm})	=

Fig. 1. An example of how vessel volume and BSA influence PV, PAV, TAV_{norm}. The left panel shows an RCA with 2 lesions (red coded coronary plaques) in a patient with a high BSA and vessel volume. The right panel shows an RCA with similar plaque volume (430 mm^3) but smaller vessel volume. As a result of the normalization for vessel volume, PAV is larger in the RCA in the right panel, while PV and TAV_{norm} are similar. BSA, body surface area; PAV, percent atheroma volume; PV, plaque volume; RCA, right coronary artery; R-PL, right postero-lateral branch; R-PDA, right posterior descending artery; TAV_{norm}, total atheroma volume normalized for vessel length. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

Table 1
Baseline characteristics.

Clinical	Value (N = 1479)	CCTA	Value (N = 1479)
Demographics		Extent of CAD	
Age, years	60.7 ± 9.3	No CAD, n	334 (22.6)
Male, n	864 (58.4)	Non-obstructive CAD, n	924 (62.5)
Height, cm	167 ± 9.7	Obstructive CAD, n	221 (14.9)
Weight, kg	71.3 ± 13.2		
Body mass index, kg/m^2	25.3 ± 3.3	**Number of diseased segments**	
Body surface area, m^2	1.81 ± 0.21	0, n	334 (22.6)
		1-5, n	790 (53.4)
Ethnicity		> 5, n	335 (24.0)
Caucasian, n	402 (27.2)		
East-Asian, n	1013 (68.5)	**Plaque quantification**	
Other, n	64 (4.3)	PV, mm^3	47.1 (3.9–138.7)
		PAV, %	2.3 (0.13–6.8)
Cardiac symptoms		TAV_{norm}, mm^3	44.0 (2.6–137.6)
No chest pain, n	251 (16.9)	Vessel volume, mm^3	2282 ± 1024
Non-anginal, n	136 (9.2)	Vessel length, mm	396 ± 123
Atypical, n	1021 (69.0)	Lumen volume, mm^3	2174 ± 991
Typical, n	71 (4.8)		
Cardiovascular risk factors			
Diabetes, n	303 (20.6)		
Hypertension, n	790 (53.8)		
Dyslipidemia, n	576 (39.2)		
Familial history for CAD, n	432 (29.2)		
Currently smoking, n	263 (17.9)		
Medications			
Aspirin, n	559 (38.4)		
ACE-I/ARB, n	420 (29.0)		
Beta blocker, n	421 (29.0)		
Calcium channel blocker, n	325 (22.5)		
Statin, n	591 (42.2)		
ASCVD score, %	9.8 (4.9–18.7)		

Data provided as mean (SD), median (25th, 75th interquartile range), or counts (%).
ACE-I, angiotensin converter enzyme inhibitor; ARB, angiotensin receptor blocker; CAD, coronary artery disease; PAV, percent atheroma volume; PV, plaque volume; TAV_{norm}, total atheroma volume normalized for vessel length.

Table 2
Effect of vessel volume on plaque burden measurements.

	Vessel volume > 75th percentile (N = 369)	Vessel volume < 75th percentile (N = 1110)	P-value
CCTA			
PV, mm^3	73.8 (9.8–197.6)	40.7 (0.0–118.7)	< 0.001
PAV, %	2.0 (0.32–5.7)	2.4 (0.0–7.1)	0.445
TAV_{norm}, mm^3	49.2 (7.2–143.4)	42.9 (0.0–135.1)	0.075
Vessel volume, mm^3	3660 ± 744	1823 ± 609	< 0.001
Vessel length, mm	509 ± 88	358 ± 109	< 0.001
BSA, m^2	1.93 ± 0.19	1.77 ± 2.0	< 0.001
Male sex, n	258 (69.9)	606 (54.6)	< 0.001

BSA, body surface area; CCTA, coronary computed tomography angiography; PAV, percent atheroma volume; PV, plaque volume; TAV_{norm}, total atheroma volume normalized for vessel length.

Table 3
Effect of body surface area on plaque burden measurements.

	Above 75th percentile (N = 369)	below 75th percentile (N = 1110)	P-value
CCTA			
PV, mm^3	65.2 (13.8–149.2)	41.2 (0.0–133.3)	0.001
PAV, %	2.5 (0.53–6.5)	2.1 (0.0–7.0)	0.225
TAV_{norm}, mm^3	59.3 (12.9–148.6)	41.0 (0.0–134.2)	0.005
Vessel volume, mm^3	2781 ± 1135	2111 + 932	< 0.001
Vessel length, mm	419 ± 132	388 ± 120	< 0.001
BSA, m^2	2.06 (2.00–2.16)	1.74 (1.62–1.84)	< 0.001
Male sex, n	324 (88.8)	528 (48.4)	< 0.001

BSA, body surface area; CCTA, coronary computed tomography angiography; PAV, percent atheroma volume; PV, plaque volume; TAV_{norm}, total atheroma volume normalized for vessel length.

Table 4
Effect of sex on plaque burden measurements.

	Male (N = 864)	Female (N = 615)	P-value
CCTA			
PV, mm^3	61.4 (10.-155.1)	29.6 (0.0–102.5)	< 0.001
PAV, %	2.8 (0.44–7.4)	1.5 (0.0–5.7)	< 0.001
TAV_{norm}, mm^3	58.2 (9.2–154.4)	28.3 (0.0–110.4)	< 0.001
Vessel volume, mm^3	2428 ± 1066	2075 ± 922	< 0.001
Vessel length, mm	404 ± 123	384 ± 123	< 0.001
BSA, m^2	1.90 ± 0.19	1.69 ± 0.17	< 0.001

BSA, body surface area; CCTA, coronary computed tomography angiography; PAV, percent atheroma volume; PV, plaque volume; TAV_{norm}, total atheroma volume normalized for vessel length.

similar between sexes, Appendix Table 3.

3.3. BSA and vessel volume

A larger BSA was significantly associated with a larger vessel volume (Fig. 2): Pearson's R = 0.53 $P < 0.001$. Every 0.1 m^2 increase in BSA related to an approximately 250 mm^3 larger vessel volume. Further, a larger vessel volume was associated with increasing PV ($P < 0.001$, $R^2 = 0.121$) and TAV_{norm} ($P = 0.003$, $R^2 = 0.111$) after adjustment for ASCVD score, but no independent association between vessel volume and PAV was observed ($P = 0.201$, $R^2 = 0.116$), appendix Table 4.

3.4. Discussion

In the current study, we aimed to determine which plaque burden methodology is most stable – shows the least amount of variability - according to differences in BSA and sex. PV and TAV_{norm} were both larger in patients with the highest BSA, while PAV was not affected by BSA. This relationship between larger BSA and larger PV and TAV_{norm} was mediated by coronary vessel volume, which showed a parallel increase with larger BSA (Pearsons R = 0.52, $P < 0.001$).

3.5. PV, PAV, and TAV_{norm} to measure atherosclerotic burden

The clinical expert consensus documentation on reporting plaque burden with IVUS recommends to use either PAV or TAV_{norm}.[19] CCTA differs compared to IVUS by the ability to quantify plaque from all coronary segments which resulted in the use of PV: summation of whole-heart atherosclerotic plaque without normalization.[2,4] PV, PAV, and TAV_{norm} will differ between each other when the length or the volume of coronary arteries are not exactly similar (Fig. 1), stressing the need for standardization in reporting. Previous imaging and histological studies showed that BSA and sex affect vessel size (function of vessel

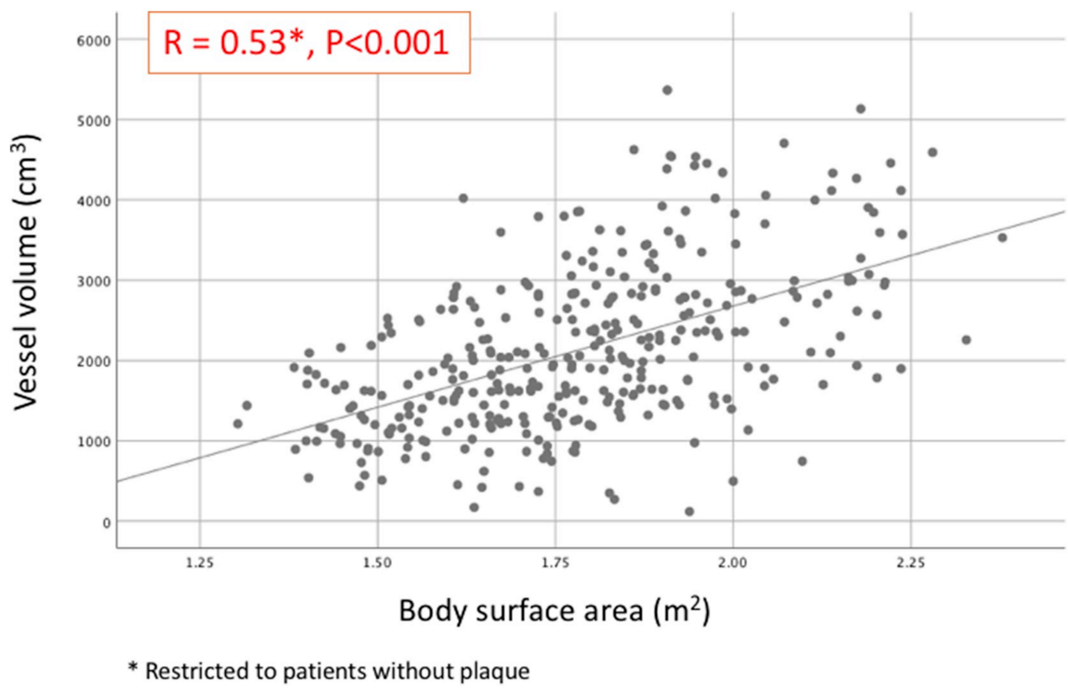


Fig. 2. Scatter plots between per patient vessel volume and BSA. Among patients without coronary plaque (N = 359), increasing BSA was associated with increasing vessel volume (Pearson's correlation coefficients 0.53, P < 0.001). The equation of the linear fitting line was −2360 + 2520*BSA. As such, a 0.1 m^2 increase in BSA relates to a 252 m^3 higher vessel volume. Restricted was to patients without coronary plaque to avoid the enlarging effect of plaque on vessel volume (positive remodeling).

length and area). We studied how PV, PAV, and TAV_{norm} are affected by BSA and sex, while the optimal way is most stable – least variable – according to differences in BSA and sex.

3.6. PV, PAV, and TAV_{norm} according to vessel volume, BSA, and sex

We observed that increasing vessel volume relates to larger absolute plaque volume as measured by PV and TAV_{norm}. Patients in the top quartile of vessel volume had greater PV and TAV_{norm} compared with the remaining patients, while PAV was similar because of its normalization. Given the correlation between vessel volume and BSA (correlation coefficient: 0.53), similar results were observed when PV, PAV, and TAV_{norm} were compared between patients in the top quartile of BSA and the remaining. These findings indicate that plaque volume increases proportionally with the size of the vessel, and indirectly with BSA. BSA has been previously associated with aortic dimensions[9] or left atrium,[20] and these structures are usually adjusted for BSA to reduce variability in the measurement and define 'normal' values. Pathological data in explanted hearts demonstrated a similar relationship between a larger BSA and increasing coronary vessel size.[8] It seems therefore apparent that coronary atherosclerosis demonstrates a similar positive correlation with the size of the vessel, or indirectly BSA. In coronary arteries, a similar volume of plaque results in greater relative reductions in coronary lumen in smaller coronary arteries compared to larger arteries. Similarly, a larger volume of plaque is needed to provide a similar relative reduction in lumen in larger vessels compared with smaller vessels. PAV is a function of vessel volume and may therefore better reflect the clinical importance of coronary atherosclerosis than absolute plaque quantification definitions (PV and TAV_{norm}). In line with this, Kishi et al. demonstrated that PAV showed the highest accuracy to identify hemodynamically significant stenosis compared with several other plaque measurements including TAV_{norm}.[21] Further, low variability is important for standardization in reporting of plaque burden and endpoint selection in the design of trials to reach statistical significance. In addition, multiple IVUS trials have observed lower variability of PAV compared to TAV_{norm},[6,22,23] supporting our findings that PAV is superior to report coronary plaque burden.

Although men had a significantly larger vessel volume than women, no differences were observed between the 3 plaque burden methodologies when comparing men vs women. This suggests that PV, PAV, and TAV_{norm} are equally influenced by the sex of a patient.

3.7. Limitations

Ideally, histologically measured plaque burden would serve as gold standard to determine the accuracy of overall plaque burden measurements with PV, PAV and TAV_{norm}. Vessel volume increases with growth of coronary plaque (positive remodeling).[24] Hence, when dividing plaque volume by vessel volume as done in PAV, the true plaque burden will be underestimated. Future studies should evaluate and define the true volume of diseased coronary arteries as if there was no plaque (i.e. not affected by positive remodeling) which can be used to normalize the quantified plaque volume for. Most patients were low risk and had East-Asian ethnicity which might limit generalizability of the findings. No comparisons of prognostic value between the plaque burden methodologies were performed. Finally, differences in vasodilatory response to pre-CT use of nitroglycerine use may have affected vessel volumes.

4. Conclusion

We observed that PAV was less affected by patient's body surface area then PV and $TAV_{norm.}$ PAV may be the preferred method to report coronary atherosclerotic burden in CCTA.

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Declaration of competing interest

Dr. James K. Min receives funding from the Dalio Foundation, National Institutes of Health, and GE Healthcare. Dr. Min serves on the scientific advisory board of Arineta and GE Healthcare, and has an equity interest in Cleerly. Dr. Habib Samady serves on the scientific advisory board of Philips, has equity interest in Covanos Inc., and has a research grant from Medtronic. Dr. Kavitha Chinnaiyan is a non-compensated medical advisory board member of Heartflow Inc. The remaining authors have no relevant disclosures.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jcct.2020.01.012.

APPENDIX

Table 1
Effect of vessel volume on plaque burden measurements

	Vessel volume > 50th percentile (N = 740)	Vessel volume < 50th percentile (N = 739)	P-value
CCTA			
PV, mm^3	64.1 (7.1–173.0)	33.2 (0–105.2)	< 0.001
PAV, %	2.2 (0.22–6.3)	2.3 (0–7.4)	0.504
TAV_{norm}, mm^3	47.7 (5.2–139.7)	39.3 (0–133.1)	0.060
Vessel volume, mm^3	3067 ± 806	1496 ± 460	< 0.001
Vessel length, mm	473 ± 90	319 ± 103	< 0.001
BSA, m^2	1.89 ± 0.19	1.74 ± 0.20	< 0.001
Male sex, n	479 (64.7)	385 (52.1)	< 0.001

BSA, body surface area; CCTA, coronary computed tomography angiography; PAV, percent atheroma volume; PV, plaque volume; TAV_{norm}, total atheroma volume normalized for vessel length.

Table 2
Effect of body surface area on plaque burden measurements

	BSA > 50th percentile (N = 728)	BSA < 50th percentile (N = 727)	P-value
CCTA			
PV, mm^3	55.8 (7.5–150.1)	40.6 (0–124.6)	0.004
PAV, %	2.3 (0.34–6.6)	2.3 (0–7.1)	0.652
TAV_{norm}, mm^3	47.2 (6.7–141.9)	39.4 (0–128.9)	0.040
Vessel volume, mm^3	2634 ± 1097	1924 ± 812.2	< 0.001
Vessel length, mm	419 ± 129	373 ± 113	< 0.001
BSA, m^2	1.98 ± 0.13	1.65 ± 0.11	< 0.001
Male sex, n	583 (80.1)	269 (37.0)	< 0.001

BSA, body surface area; CCTA, coronary computed tomography angiography; PAV, percent atheroma volume; PV, plaque volume; TAV_{norm}, total atheroma volume normalized for vessel length.

Table 3
Effect of sex on plaque burden measurements in low risk patients (ASCVD score < 7.5%)

	Male (N = 256)	Female (N = 328)	P-value
CCTA			
PV, mm^3	25.4 (0.0–117.2)	17.6 (0.0–72.8)	0.145
PAV, %	1.3 (0.0–4.7)	0.93 (0.0–3.7)	0.256
TAV_{norm}, mm^3	24.1 (0.0–97.2)	15.8 (0.0–63.1)	0.198
Vessel volume, mm^3	2497 ± 1026	2139 ± 967	< 0.001
Vessel length, mm	429 ± 110	403 ± 119	0.009
BSA, m^2	1.91 ± 0.18	1.70 ± 0.16	< 0.001

ASCVD, atherosclerotic cardiovascular disease; BSA, body surface area; CCTA, coronary computed tomography angiography; PAV, percent atheroma volume; PV, plaque volume; TAV_{norm}, total atheroma volume normalized for vessel length.

Table 4
Linear association between vessel volume and plaque burden

	Standardized beta-coefficient for PV*	P-value	Standardized beta-coefficient for TAVnorm*	P-value	Standardized beta-coefficient for PAV*	P-value
ASCVD score, %	0.307	< 0.001	0.327	< 0.001	0.338	< 0.001
Vessel volume, mm^3	0.173	< 0.001	0.074	0.003	−0.031	0.201

*Log-transformed.
ASCVD, atherosclerotic cardiovascular disease; BSA, body surface area; CCTA, coronary computed tomography angiography; PAV, percent atheroma volume; PV, plaque volume; TAV_{norm}, total atheroma volume normalized for vessel length.

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