Cardiothoracic Imaging

Quantitative plaque analysis with A.I.-augmented CCTA in end-stage renal disease and complex CAD

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ABSTRACT

Background: Adverse cardiovascular events are a significant cause of mortality in end-stage renal disease (ESRD) patients. High-risk plaque anatomy may be a significant contributor. However, their atherosclerotic phenotypes have not been described. We sought to define atherosclerotic plaque characteristics (APC) in dialysis patients using artificial-intelligence augmented CCTA.

Methods: We retrospectively analyzed ESRD patients referred for CCTA using an FDA approved artificial-intelligence augmented-CCTA program (Cleerly). Coronary lesions were evaluated for APCs by CCTA. APCs included percent atheroma volume (PAV), low-density non-calcified-plaque (LD-NCP), non-calcified-plaque (NCP), calcified-plaque (CP), length, and high-risk-plaque (HRP), defined by LD-NCP and positive arterial remodeling >1.10 (PR).

Results: 79 ESRD patients were enrolled, mean age 65.3 years, 32.9% female. Disease distribution was non-obstructive (65.8%), 1-vessel disease (21.5%), 2-vessel disease (7.6%), and 3-vessel disease (5.1%). Mean total plaque volume (TPV) was 810.0 mm$^3$, LD-NCP 16.8 mm$^3$, NCP 403.1 mm$^3$, and CP 390.1 mm$^3$. HRP was present in 81.0% patients. Patients with at least one >50% stenosis, or obstructive lesions, had significantly higher TPV, LD-NCP, NCP, and CP. Patients >65 years had more CP and higher PAV.

Conclusion: Our study provides novel insight into ESRD plaque phenotypes and demonstrates that artificial-intelligence augmented CCTA analysis is feasible for CAD characterization despite severe calcification. We demonstrate elevated plaque burden and stenosis caused by predominantly non-calcified-plaque. Furthermore, the quantity of calcified-plaques increased with age, with men exhibiting increased number of 2-feature plaques and higher plaque volumes. Artificial-intelligence augmented CCTA analysis of APCs may be a promising metric for cardiac risk stratification and warrants further prospective investigation.

1. Introduction

End-stage renal disease (ESRD) is a widespread global health problem that carries significant cardiovascular morbidity and mortality. Patients frequently develop advanced coronary atherosclerosis since they have clustering of traditional atherosclerotic risk factors, such as diabetes, systemic inflammation, and altered mineral metabolism.

Patients with ESRD are 8 times more likely to die compared with the general U.S. population, and cardiovascular causes account for >40% of all deaths. In this population, the prevalence of obstructive coronary artery disease (CAD) on coronary angiography exceeds 50%. Despite having extensive CAD and vascular disease, patients with ESRD often do not present with classic anginal symptoms. They often are asymptomatic because of impaired exercise capacity and diabetic or

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uremic neuropathy. Predictive accuracy for future cardiac events using traditional diagnostic screening tools, including conventional risk prediction models, is limited in ESRD patients. Comorbidity, poor exercise capacity, and a high prevalence of cardiovascular abnormalities limit the diagnostic accuracy of traditional ischemia-driven tests. Coronary computed tomographic angiography (CCTA) represents a promising method for the noninvasive detection of coronary artery disease (CAD). It may provide a much-needed improvement in the cardiovascular assessment of ESRD patients.

Prior studies using CCTA in patients with ESRD have shown that mild and moderate pre-dialysis CKD are independent risk factors for CAD. Despite the expected high prevalence of calcified plaque, Jug et al. showed high per-patient diagnostic performance for detecting significant stenosis using CCTA in ESRD patients compared to conventional coronary angiography. Recent developments have allowed for a more accurate quantitative assessment of both the type and volume of coronary atherosclerosis. Recent single and multicenter studies have shown that quantitative evaluation of coronary artery plaque on CCTA can identify patients at risk of subsequent cardiac events and culprit lesions in patients who subsequently experience a myocardial infarction. In addition, recent demonstration of artificial intelligence augmented CCTA shows that it allows for rapid accurate evaluation of CAD and plaque characteristics. Therefore, based on all available publications to date, we are the first group to perform artificial intelligence assisted quantitative plaque analysis in ESRD patients on dialysis (cohort of 79 CCTA exams), to further define the CAD characteristics in this population.

2. Methods

2.1. Study population

The study population was pooled the joint database of CCTA diagnostic procedures performed at two medical centers in Los Angeles county between 2007 and 2017 (retrospective cohort). For the retrospective cohort, patients were included in the analysis if they had ESRD and referred for a chest pain diagnostic workup and had available CCTA. Patients with a history of coronary artery bypass grafting, irregular heartbeat, or intolerance to iodinated contrast agents were excluded.

2.2. CCTA exams

All CCTA scans were performed using a 64-multidetector row scanner (GE Healthcare, Milwaukee, Wisconsin, USA). Accordingly, data were acquired with collimation of 64 × 0.625 mm and a tube rotation time of 350 ms. The tube current was 300–400 mA at 100–120 kV for patients based on their body size. Individuals presenting with baseline heart rates of >65 beats/min were administered oral beta-blocker therapy as the preferred method for slowing down the heart rate. Intravenous administration was allowed in the protocol, using metoprolol at 5 mg increments to a total possible dose of 25 mg to achieve a resting heart rate of <65 beats/min. Nitroglycerine 0.4 mg sublingually was administered immediately before contrast. During 64-slice multidetector-row computed tomography, 40 to 60 ml of contrast material was administered into the antecubital vein, depending on the total scan time, at a flow rate of 4 ml/s, followed by a contrast saline mixture at 4 ml/s and saline flush at 4 ml/s. Images were acquired using either prospective ECG triggering at 75% of the RR interval or by retrospective ECG gating with images constructed at 5% intervals from 5 to 95% of the RR interval.

Because all patients had ESRD, the CCTA be performed as close to the next dialysis session as possible—that is, in the afternoon of the day before morning dialysis or in the morning of the same day of an afternoon dialysis session. Patients provided informed consent to undergo the CCTA (as part of the clinical workup). We obtained approval from the institutional review board of each center to review the medical records of these individuals by maintaining patient records confidentiality.

2.3. CCTA analysis

Coronary segments with a diameter ≥ 2 mm were included in the analysis using the modified 18-segment SCCT model. Each segment was evaluated for the presence or absence of coronary atherosclerosis, defined as any tissue structure >1 mm² within the coronary artery wall that was differentiated from the surrounding epicardial tissue, epicardial fat, or the vessel lumen itself. The following atherosclerotic plaque characteristics (APCs) were evaluated (Fig. 1).

Quantitative atherosclerosis characterization was performed for every coronary artery and its branches using an automated artificial intelligence (AI)-enabled web-based software platform (Cleerly Labs, Cleerly Inc., New York, New York) with protocols as described in further detail in the CLARIFY trial. Plaque volumes (mm³) were calculated for each coronary lesion and then summed to compute the total plaque volume at the patient level. This provided data for analysis on both the lesion and patient level. Plaque volume was categorized using Hounsfield unit (HU) ranges, with NCP defined as HU between 30 and 350; LD-NCP defined as plaques <30 HU; CPs defined as >350 HU. Coronary plaque burden was normalized to vessel volume to account for natural variation in coronary artery volume. Plaque burden was reported as percent atheroma volume (PAV), which was calculated as Plaque Volume / Vessel Volume × 100%. Arterial remodeling was calculated by examining the lesion diameter divided by the normal reference diameter. PR was defined as a ratio ≥ 1.10. RR was defined as a ratio of <0.95. IR was a ratio between 0.95 and 1.10. HRPs were defined as coronary lesions with both LD-NCP and PR. Plaque length measured uninterrupted plaque along the length of a vessel. Plaque diffusivity was the percent plaque along the vessel's length divided by the total vessel length.

2.4. Safety in ESRD patients

Safety concerns of CCTA in ESRD predominantly pertain to contrast-related volume overload and exposure to radiation. As a precaution, our study protocol recommended that CCTA be performed as close to the next dialysis session as possible. More importantly, the average amount of contrast volume was very low (i.e., averaging 67 ml) and did not represent a clinically significant volume overload for ESRD patients in our study. No incidents of contrast induced nephropathy were reported. As for concerns related to sources of ionizing energy, conventional radiation doses in the range of 20 mSv have been reduced recently by step-and-shoot acquisition with adaptive ECG triggering (i.e., prospective ECG-gated CCTA) to doses as low as 3–4 mSv. This is similar in magnitude to the average annual background radiation and compares favorably to radioisotope stress perfusion imaging and conventional coronary angiography.

2.5. Statistical analysis

All statistical analyses were performed using SAS version 9.4 (SAS, Cary, NC). Continuous data are reported as mean ± standard deviation, and categorical variables are presented as absolute numbers with corresponding frequencies. Student’s t-test, Mann-Whitney test, chi-square, and Fisher exact tests were used to compare the distribution of continuous and categorical variables, respectively.

3. Results

3.1. Study population

A total of 79 patients with ESRD were enrolled in the study, mean age
of 65.3 (±12.5) years 26 (32.9%) female. Demographics, medical history, and calcium scores are shown in Table 1. All patients had ESRD and were undergoing either hemodialysis (n = 74), peritoneal dialysis (n = 4) or were recommended for HD and declined and went to hospice (n = 1). Risk factors and comorbidities were common, including diabetes (60.8%), hypertension (89.7%), hyperlipidemia (53.3%), smoking history (52.9%), and family history of heart disease (35.1%). Baseline demographic data are presented in Table 1. The average duration of dialysis relative to when the CCTA was acquired was 52.3 (±40.7) months, comparable to prior studies.

History of previous cardiovascular events included prior MI (37.5%), stroke (19.6%), and current chest pain (24.7%).

### 3.2. Disease prevalence and phenotype

Disease prevalence was high in this population (Table 2). CAC scores were available in 75 (94.9%) of 79 subjects, and only 6 (8.0%) had a calcium score of zero. As expected, there was a high mean coronary calcium Agatston score of 2221 (±1666), range 0–9217 (Quartiles: Q1 389, Q2 1567, Q3 3808). The mean total plaque volume was 810.0 (±552.0) mm$^3$, LD-NCP volume was 16.8 (±52.9) mm$^3$, NCP volume was 403.1 (±294.6) mm$^3$, and the CP volume was 390.1 (±341.5) mm$^3$ (Table 2). High-risk plaques (LD-NCP plus PR) were present in 64 (81.0%) of patients and 43 (5.6%) of individual plaques. Positive remodeling (>1.1) was present in 64 (81.0%) of patients and 409 (53.8%) of plaques. A total of 760 individual coronary plaques were identified in 79 patients (9.62 plaques per patient). Obstructive disease was present in 27 (34.2%) patients. The distribution of disease was non-obstructive in 52 (65.8%) patients. 1-vessel disease 17 (21.5%), 2-vessel disease 6 (7.6%), and 3-vessel disease 4 (5.1%).

### 3.3. Relationship of APCs by CCTA to stenosis

Per-patient plaque volumes and characteristics differed between patients with and without stenosis (Table 2). Patients with at least one >50% stenosis had significantly higher plaque volume 1082.7 (529.4) mm$^3$ vs 466.8 (513.1) mm$^3$ (p = 0.0012). They also demonstrated more low density non-calcified plaque (LD-NCP) 31.5 (88.2) vs 9.1 (12.2) (p = 0.025), non-calcified plaque (NCP) 517.6 (341.7) vs 343.6 (250.2) (p = 0.0075), and total calcified plaque (CP) 533.5 (309.3) mm$^3$ vs 315.7 (336.3) mm$^3$ (p = 0.0053). The maximum remodeling index was also

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**Table 1**

<table>
<thead>
<tr>
<th>Variable</th>
<th>All (N = 79)</th>
<th>Non-obstructive (N = 52)</th>
<th>Obstructive (&gt;50%) (N = 27)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>65.3 (12.5)</td>
<td>65.2 (13.5)</td>
<td>65.5 (10.7)</td>
<td>0.9232</td>
</tr>
<tr>
<td>Female</td>
<td>26 (32.9%)</td>
<td>22 (42.3%)</td>
<td>4 (14.8%)</td>
<td>0.0219</td>
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<tr>
<td>Hypertension (%)</td>
<td>70 (89.7%)</td>
<td>46 (88.5%)</td>
<td>24 (92.3%)</td>
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</tr>
<tr>
<td>Dyslipidemia (%)</td>
<td>41 (53.3%)</td>
<td>25 (49.0%)</td>
<td>16 (61.5%)</td>
<td>0.3411</td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td>48 (60.8%)</td>
<td>28 (53.9%)</td>
<td>20 (74.1%)</td>
<td>0.0943</td>
</tr>
<tr>
<td>Family history (%)</td>
<td>27 (35.1%)</td>
<td>14 (27.5%)</td>
<td>13 (52.0%)</td>
<td>0.0776</td>
</tr>
<tr>
<td>Tobacco use, ever (%)</td>
<td>37 (52.9%)</td>
<td>28 (57.1%)</td>
<td>9 (42.9%)</td>
<td>0.2725</td>
</tr>
</tbody>
</table>

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**Fig. 1.** Example of plaque analysis in a 63-year-old female with ESRD. Three groups of two images of the RCA, LM/LAD, and circumflex are presented. Each group’s left image is a curved multiplanar planar reformat (MPR) of the CCTA, and the right images are straightened MPR with color plaque overlay (red LD-NCP, yellow NCP, blue CP). A 20 × 3.0 mm stent is present in the proximal RCA and was excluded from QCT analysis (purple overlay with “N” markers, second image from left). This is the only exclusion in this patient, and it represented 2.82% of the coronary vessels measuring 2 mm or greater. Note that a 51% stenosis was depicted distal to the stent (arrow). Two moderate stenoses of 61 and 55% were depicted in the mid LAD (4th image, arrows). Non-obstructive disease was present in the circumflex. The patient's plaque volume was high at 1289.4 mm$^3$, CP 384.8 mm$^3$, NCP 904.6, total PAV was 29.8%, and 4 two-feature HRP were depicted.
higher in patients with obstructive disease 1.7 (0.3) vs 1.4 (0.3) (p < 0.0001). Plaques were also longer in patients with obstructive disease 136.3 (61.0) mm vs 96.2 (65.0) mm, respectively.

We then compared the APCs of individual atherosclerotic plaques, comparing those with (n = 36) and without (n = 724) stenosis of >50%. Obstructive lesions had greater total plaque volume 154.5 (150.7) mm$^3$ vs 62.0 (91.3) mm$^3$ (p = 0.0004), LD-NCP 5.4 (11.3) mm$^3$ vs 1.3 (7.0) mm$^3$ (p = 0.0049), NCP 81.5 (88.6) mm$^3$ vs 30.4 (48.2) mm$^3$ (p = 0.0003), and CP 65.7 (76.5) mm$^3$ vs 29.8 (52.7) mm$^3$ (p = 0.0096). Obstructive lesions had a higher percentage of 2-feature HRP (LD-NCP + PR) 18 (50%) vs 205 (28.3%) (p = 0.0248) as well as longer lesion length 24.2 (16.1) mm vs 12.8 (11.2) mm (p < 0.0001).

### 3.4. Relationship of APCs by CCTA to gender

There were per-patient gender differences in APCs identified (Table 3). Men had higher total plaque volume 909.4 (545.6) mm$^3$ vs 607.4 (517.4) mm$^3$ (p = 0.0267), higher LD-NCP 22.2 (63.8) mm$^3$ vs 5.7 (9.9) mm$^3$ (p = 0.0003), and higher NCP 461.0 (302.5) mm$^3$ vs 284.9 (242.5) mm$^3$ (p = 0.0054). Men also exhibited a higher prevalence of any positive remodeling 52 (98.1%) vs 20 (76.9%) (p = 0.0044). There were no significant differences in CP volume, 2-feature HRP or plaque length.

### 3.5. Relationship of APCs by CCTA to age

Age related differences in APCs were also present (Table 3). Patients >65 years had more CP 456.3 (331.0) mm$^3$ vs 307.0 (340.9) mm$^3$ (p = 0.0175). The percentage of a plaque that was calcified increased significantly with age, 51.7 (19.9) % for those >65 years vs 32.9 (23.6) % (p = 0.001). The per patient percent CP increased with age; correlation coefficient 0.45 (p < 0.0001) the slope of the regression line was

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**Table 2**

Per-patient APCs by CCTA for all patients as well as by the degree of stenosis.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total (N = 79)</th>
<th>Non-obstructive (&lt;50%) (N = 52)</th>
<th>Obstructive (50%) (N = 27)</th>
<th>P-value (obs vs. non-obs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atherosclerosis volume</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plaque volume, mm$^3$</td>
<td>810.0 (552.0)</td>
<td>668.4 (513.1)</td>
<td>1092.7 (529.4)</td>
<td>0.0012</td>
</tr>
<tr>
<td>LD-NCP, mm$^3$</td>
<td>16.8 (12.2)</td>
<td>9.1 (12.2)</td>
<td>31.5 (88.2)</td>
<td>0.0250</td>
</tr>
<tr>
<td>NCP, mm$^3$</td>
<td>403.1 (294.6)</td>
<td>343.6 (250.2)</td>
<td>517.6 (341.7)</td>
<td>0.0075</td>
</tr>
<tr>
<td>CP, mm$^3$</td>
<td>390.1 (341.5)</td>
<td>315.7 (336.3)</td>
<td>533.5 (309.3)</td>
<td>0.0053</td>
</tr>
<tr>
<td>Arterial remodeling</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Remodeling index</td>
<td>1.52 (0.30)</td>
<td>1.4 (0.3)</td>
<td>1.7 (0.3)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Positive remodeling &gt;1.10</td>
<td>72 (91.1%)</td>
<td>45 (86.5%)</td>
<td>27 (100%)</td>
<td>0.0484</td>
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<tr>
<td>Negative remodeling &lt;0.95</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Intermediate remodeling</td>
<td>7 (8.9%)</td>
<td>7 (13.5%)</td>
<td>0</td>
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<tr>
<td>Other APCs</td>
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<tr>
<td>High-risk plaque (LD-NCP + PR), %</td>
<td>64 (81.0%)</td>
<td>40 (76.9%)</td>
<td>24 (88.9%)</td>
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</tr>
<tr>
<td>Lesion length, mm</td>
<td>109.9 (66.1)</td>
<td>96.2 (65.0)</td>
<td>136.3 (61.0)</td>
<td>0.0073</td>
</tr>
</tbody>
</table>

**Table 3**

Per-patient APCs by CCTA for all patients by gender, age, and diabetes.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Gender</th>
<th>Age</th>
<th>Diabetes</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Males (N=63)</td>
<td>Females (N=26)</td>
<td>&lt;65 (N=35)</td>
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<tr>
<td>Plaque volume, mm$^3$</td>
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<td></td>
<td></td>
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<tr>
<td>LD-NCP, mm$^3$</td>
<td></td>
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<td></td>
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<tr>
<td>NCP, mm$^3$</td>
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<td>CP, mm$^3$</td>
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<td>% of Plaque that is</td>
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<tr>
<td>Calculated</td>
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<tr>
<td>PAV</td>
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<tr>
<td>PAV LD-NCP</td>
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<tr>
<td>PAV NCP</td>
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<tr>
<td>PAV CP</td>
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<tr>
<td>Remodeling index</td>
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<td></td>
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<tr>
<td>Positive Remodeling &gt;1.10</td>
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<tr>
<td>Negative Remodeling &lt;0.95</td>
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<tr>
<td>Intermediate Remodeling</td>
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<tr>
<td>HRP (LD-NCP + PR), %</td>
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<tr>
<td>Lesion length, mm</td>
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</tbody>
</table>

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3.6. Relationship of APCs by CCTA to diabetes

Diabetes mellitus (DM) related differences in APCs were also noted (Table 3). Patients with DM had more total plaque PAV 19.4 (15.9) % vs 21.3 (15.8) % (p = 0.0045), NCP PAV 14.2 (7.0) % vs 10.3 (6.7) % (p = 0.0139) and a higher mean remodeling index 1.6 (0.3) vs 1.4 (0.3) (p = 0.0248). There were no significant differences in CP, CP PAV, LD NCP, LD NCP PAV, number of HRP, or mean plaque length.

4. Discussion

ESRD patients have a high prevalence of obstructive CAD. The presence of epicardial coronary stenosis (>50%) using invasive angiography has been reported in 50% to 70% of asymptomatic patients at the start of dialysis, with multivessel involvement in 25% to 40% of studied populations. The incidence and types of atherosclerotic plaque characteristics however have not been defined previously in this ESRD population, for which we sought to investigate using artificial intelligence augmented CCTA.

The main challenge of interpreting CCTA in patients with ESRD is to overcome the adverse effect of the high calcific burden. The role of CCTA in patients with ESRD was initially limited by early studies suggesting that a small but significant portion of studies (10%) and vessels (14%) were not interpretable due to imaging artifacts, particularly dense coronary calcium.

Despite these limitations CCTA has been shown to be useful in ESRD. Bie et al. found after 2 years of follow-up, 36% of those with CAD had a cardiac event compared with 0% of the patients with no significant CAD. Also, in the absence of CAD determined by CCTA, Budoff et al. demonstrated that CCTA performed well in detecting/excluding coronary stenosis when directly compared to invasive angiography with high negative predictive value (>94%) with excellent short to intermediate term prognosis. Jug et al. assessed the diagnostic performance of 64-slice CCTA comparing ESRD patients with non-ESRD patients undergoing CCTA and invasive coronary angiography. On a patient-based model, the sensitivity, specificity, and positive and negative predictive values to detect at least 50% and 70% stenosis were 100, 78, 92, and 100% and 100, 91, 95, and 100%, respectively, for ESRD patients, and 97, 83, 87, and 96% and 94, 87, 85, and 95%, respectively, for non-ESRD controls. There were no statistically significant differences between ESRD and non-ESRD participants in diagnostic performance measures. Of interest, the specificity (91% per patient) remained high despite extensive plaque calcification. Roy et al. used a visual plaque assessment method to generate a total plaque score (TPS), segment involvement score (SIS) and a segment stenosis score (SSS); segment plaque quantity was graded on a scale of 1–3. Patients with mild chronic kidney disease had a mean TPS 2.2 points higher than those with the referent normal GFR (P = 0.001), and patients with moderate CRD had a mean TPS 5.0 points higher than the referent (P < 0.001). The SIS and SSS scores also increased with mild and moderate chronic renal disease. Overall, these studies demonstrate the accuracy of CAD analysis between CCTA correlating to coronary angiogram - and subsequently with studies such as the CLARIFY trial, also shows that A.I. augmented CCTA is as accurate as CCTA performed by human expert readers.

In view of the severe and complex disease in ESRD patients with advanced lesions that are difficult to characterize, we took the approach of quantitative atherosclerosis characterization performed using an automated artificial intelligence (AI)-enabled software. The results demonstrate that CAD analysis using CCTA can be reliably performed to characterize atherosclerosis in the vast majority of dialysis dependent ESRD patients (94.3% analyzable studies). Furthermore, via this methodology, which the CLARIFY trial has shown to be reproducible for plaque evaluation, our novel findings showed that the incidence of coronary disease assessed by CAC was high, occurring in 75 (94.9%) of 79 subjects (Table 2) with a mean Agatston score of 2221 and mean total plaque volume of 810.0 mm³. In particular, high-risk plaques were prevalent in this group (64 patients, 81.0%), congruent with the high MACE in the ESRD population and CAD burden described by prior studies.

Men had increased total plaque volume, as well as higher LD-NCP, NCP, and positive remodeling (Table 3). We observed that the presence of ESRD in patients >65 years old independently manifested increased CP and atheroma volume, and that the per-patient percentage of CP positively correlated with age (regression line slope 0.91 (p < 0.0001)) (Fig. 3). We also found that patients with DM had more total plaque PAV, NCP, and a higher mean remodeling index (Table 3).

Evaluating APCs of individual atherosclerotic plaques, compared to those without CAD, patients with obstructive disease (>50% stenosis) demonstrated significantly elevated levels of several APC parameters, including higher plaque volume 1082.7 (529.4) mm³ vs 466.8 (513.1) mm³ (p = 0.0012) along with significantly increased LD-NCP, NCP, and total calcified plaque. Furthermore, ESRD patients with obstructive CAD manifested higher remodeling indices, increased 2-feature HRP’s, and longer plaque length (Table 2). These finding are consistent with an increased frequency of myocardial infarction demonstrated by other studies, and higher cardiovascular events observed in ESRD patients.

Fig. 3. The per-patient percent of plaque that was calcified increased with age.
4.1. Success of quantitative plaque quantification

The length and percentage of excluded segments was recorded. Exclusions were placed because of an inability to make a diagnosis due to motion or calcium-related artifacts, poor opacification, or the presence of coronary stents (Fig. 1). Exclusions were present in 41 (51.9%) patients. The excluded lengths totaled 2217.3 mm (5.71%) of 38,828 mm of total coronary vessel length. On a per-patient basis, there were no exclusion in 38 (48.1%), and the remaining exclusions totaled 1–4.9% of the total vessel length in 11 (13.9%), 5–9.9% in 9 (11.4%), 10–19.9% in 10 (12.6%), 20–29.9% in 5 (6.3%), and >30% in 6 (7.6%) (Fig. 2). Overall, our exclusion criteria are based on larger CCTA trials (CONFIRM, SCOT-HEART, CLARIFY) which have similar criteria and percentages of exclusion.

4.2. Limitations

First, the study population was sampled retrospectively and subject to the limitations inherent in all such study designs—namely, a high risk of referral, spectrum, and ascertainment bias. For the accuracy of AI CCTA in CAD analysis, direct comparison between invasive coronary angiography, automated artificial intelligence (AI)-enabled web-based software platform, and standard interpretation was not performed, but direct coronary angiogram to CCTA correlation has been performed to demonstrate correlation/accuracy previously in non-AI CCTA studies, and subsequently AI CCTA to human CCTA, which has been done by our group and co-authors, and given that there were patients in these studies with higher plaque burden (similar to our patient population), these results should be generalizable, but indeed that further studies in this specific ESPR dialysis population are needed to confirm this with certainty. Finally, and most importantly, the prognostic implications and clinical relevance of CCTA detection of coronary atherosclerosis in this population are yet to be determined prospectively.

5. Conclusion

Despite significant CAD burden, quantitative plaque analysis in ESPR patients is possible in a high percentage of coronary vessels using artificial intelligence enhanced CCTA. Notably, we have revealed a phenotype with a high plaque burden, a high percentage of calcified plaque, and frequent stenoses caused by predominantly non-calcified plaque. Men exhibited higher plaque volumes and more two-feature plaque, and frequent stenoses caused by predominantly non-calcified plaque on coronary computed tomography angiography predicts myocardial infarction: results from the multicenter SCOT-HEART trial (Scandinavian Computed Tomography of the Heart). Circulation 2020 May 5;141(18):1452-62.

Author contributions

Study conception/design: GWC, ADC, JKM, RPK, JPE; data collection: GWC, AGK, TRC, RSJ, ADC; data analysis/interpretation: GWC, ADC, MJB, JKM, RPK, JPE; manuscript preparation: GWC, ADC, JKM, RPK, JPE. All authors have approved the final version of this manuscript.

Declaration of competing interest

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