High incidence of newly diagnosed obstructive coronary artery disease regardless of chest pain detected on pre-procedural cardiac computed tomography angiography in patients undergoing atrial fibrillation ablation

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\textbf{Background} Cardiac computed tomography (CT) is often performed before catheter ablation of atrial fibrillation to map atrial and pulmonary anatomy. Incident coronary artery disease (CAD) may also be diagnosed during cardiac CT angiography (CTA). Our aim was to assess whether coronary CTA might be able to identify a significant proportion of patients with obstructive CAD prior to their catheter ablation procedure event, even in asymptomatic patients.

\textbf{Methods} Consecutive patients undergoing pre-ablation coronary CTA for atrial fibrillation between 2013 and 2020 were retrospectively selected. Patients with previously diagnosed CAD were excluded. Obstructive CAD was defined as $\geq$50% luminal stenosis. We analyzed the relationship between obstructive CAD, any chest pain, and traditional risk factors.

\textbf{Results} Overall, 2321 patients (median age 63.0 (54.4–69.2), 1052/2321 (45.3%) female) underwent coronary CTA and 488/2321 (21.0%) were diagnosed with obstructive CAD. There was no difference regarding the rate of obstructive CAD in patients with any chest pain compared to patients without any chest pain [91/404 (22.5%) vs. 397/1917 (20.7%), $P=0.416$, respectively]. The following parameters were associated with obstructive CAD: age $>65$ years (odds ratio (OR)=2.51; 95% confidence interval (CI), 2.02–3.13; $P<0.001$), male sex (OR=1.59; 95% CI, 1.28–1.98; $P<0.001$), hypertension (OR = 1.40; 95% CI, 1.28–1.98; $P=0.011$), diabetes (OR=1.50; 95% CI, 1.13–1.99; $P=0.006$), dyslipidaemia (OR = 1.33; 95% CI, 1.07–1.66; $P=0.011$) and history of smoking (OR=1.34; 95% CI, 1.07–1.68; $P=0.011$).

\textbf{Conclusions} The high prevalence of obstructive CAD even in patients without chest pain highlights the importance of additional coronary artery diagnostics in patients undergoing left atrial CTA awaiting catheter ablation for atrial fibrillation. These patients regardless of chest pain thus may require further risk modification to decrease their potential ischemic and thromboembolic risk.

\textbf{Keywords:} ablation, atrial fibrillation, chest pain, coronary artery disease, coronary computed tomography angiography

Introduction Cardiac computed tomography (CT) angiography (CTA) before catheter ablation of atrial fibrillation provides important information on the anatomy of the left atrium and pulmonary veins to tailor the ablation strategy [1–4]. It has been argued, that cardiac CTA results in increased cumulative radiological exposure [5,6]. Nevertheless, if we gain more data from the cardiac CTA images, like incidental extracardiac alterations or information on coronary artery disease (CAD), patients may benefit from these new findings [6–8]. The endeaveour for the holistic strategy of atrial fibrillation management – the Atrial fibrillation Better Care (ABC) management described in the 2021 European Society of Cardiology’s Guidelines on cardiovascular disease prevention in clinical practice – lowers the risk of all-cause and cardiovascular death, first hospitalization and cardiovascular events as well [9]. This guideline underlines the importance of identification and management of risk factors and concomitant diseases, such as CAD [9]. Since symptoms suggesting ischemic heart disease, such as chest pain could be observed in atrial fibrillation patients also without CAD, predicting...
CAD in atrial fibrillation patients is often challenging [10]. However, an incidentally found obstructive CAD may require a new preventive strategy to decrease potential ischemic events. Moreover, the reclassification of the thrombotic risk stratification (CHA$_2$DS$_2$-VASc score) potentially affects the anticoagulant strategy of atrial fibrillation patients as well [8].

Multiple studies have shown that at least 10%, or in some publications even 70% of patients with atrial fibrillation have CAD [11–14]. Most of these referred publications used different definitions to determine the presence of CAD with lower spatial resolution CT scans assessing only a few hundred patients [8,11–14].

Our aim was to present a precise evaluation of CAD in a large cohort of consecutive atrial fibrillation patients undergoing high-spatial resolution coronary CTA before catheter ablation. We hypothesised that coronary CTA might be able to identify a significant proportion of patients with obstructive CAD prior to their catheter ablation procedure event, even in patients without symptoms.

Materials and methods

Study population

In our descriptive study, we screened all the consecutive patients undergoing coronary CTA imaging retrospectively between 2013 and 2020. Inclusion criteria were (1) coronary CTA indicated to tailor ablation strategy, (2) history of atrial fibrillation, (3) patients ≥18 years old. Exclusion criteria were the following: (1) previously known CAD (history of acute myocardial infarction and coronary revascularization, known CAD treated conservatively), (2) coronary CTA image quality non-diagnostic for coronary artery luminal stenosis assessment, (3) and repeated examinations of the same patient (in this case we involved the first good-quality pre-ablation scan).

Data regarding demographics, medical history and symptoms were collected from the questionnaires filled out by patients and other medical reports collected prior to coronary CTA. Overweight was defined as BMI above 25 kg/m$^2$. Two groups were identified based on whether the patients had reported any type of chest pain or had no chest pain. Afterwards, we analyzed the relationship between these collected data and novel obstructive CAD diagnosed by coronary CTA.

All patients, who were enrolled in this study, gave written informed consent. The study protocol was reviewed and approved by the Local Research Ethics Committee (SE RKEB: 142/2019) and was in accordance with the Declarations of Helsinki.

Cardiac computed tomography angiography – imaging

Cardiac CT examinations were performed on a 256-slice scanner (Brilliance iCT 256, Philips Healthcare, Best, The Netherlands) with prospective ECG-triggered axial acquisition mode. For coronary artery calcium score (CACS) a 120-kV tube voltage with a 30–50 mAs tube current was used. For coronary CTA, a 100–120 kV with a 200–300 mAs tube current depending on patient anthropometrics was set. Image acquisition was performed with 128x0.625 mm detector collimation, and 270 ms gantry rotation time. For heart rate control metoprolol was given, if necessary. In patients with a heart rate of <80 beats per minute (bpm), mid-diastolic triggering was chosen with 3–5% padding (73–83% of the R–R interval), and in those with ≥80 bpm, systolic triggering was applied (35–45% of the R–R interval). Iomoprol contrast material (Iomeron 400, Bracco Ltd, Milan, Italy) was used with 85–95 mL contrast material at a flow rate of 4.5–5.5 mL/s from antecebral vein access using a four-phasic protocol. Bolus tracking in the left atrium was applied to obtain proper scan timing. Sublingual nitroglycerin was given between these collected data and novel obstructive CAD.

Image analysis

Evaluation of cardiac CTA images was performed offline by 22 cardiologist and radiologist experts. In case of any uncertainty, the images were reviewed and re-evaluated. Coronary artery status was analyzed using commercially available semi-automated software (HeartBeat-CS, Philips IntelliSpace Portal, Philips Healthcare). Stenosis severity was qualitatively reported according to the current Society of Cardiovascular Computed Tomography Guidelines: normal: absence of plaque and no luminal stenosis; minimal: 1–25% stenosis; mild: 25–49% stenosis; moderate: 50–69% stenosis; severe: 70–99% stenosis; occluded: 100% stenosis. In this current analysis, we defined obstructive CAD as a lesion with ≥50% stenosis [15]. In case of the presence of multiple lesions, the most severe stenosis was considered. Furthermore, total CACS was determined as well.

Statistical analysis

Categorical variables were presented as numbers and percentages. As continuous variables showed

Table 1 Characteristics of the studied population

| Parameters                                      | n=2321 |
|------------------------------------------------|--------|
| Age (years)                                    | 63.0   |
| Female sex                                     | 1052   |
| BMI (kg/m$^2$)                                 | 28.7   |
| Hypertension                                   | 1580   |
| Diabetes                                       | 313    |
| Dyslipidaemia                                  | 803    |
| History of smoking                             | 724    |
| Positive family history of cardiovascular disease | 401    |
| Peripheral vascular disease                    | 108    |
| Prior stroke/TIA                               | 126    |
| Any chest pain                                 | 404    |

Continuous variables are presented as median and interquartile ranges and categorical variables as numbers and percentages.

TIA, transient ischemic attack.
Results

Studied population

We included 3,335 pre-ablation coronary CTA examinations, from which 570 were excluded due to non-diagnostic images, and 169 patients because of previously known CAD. Additional 274 images were excluded, where multiple coronary CTA examinations were performed. In this case, the first good-quality image, only one for each patient, was included. Finally, 2,321 patients with paroxysmal and persistent atrial fibrillation were included in our study population (Fig. 1).

The median age of the included patients was 63.0 (54.4–69.2) with the main characteristics shown in Table 1. The population was rather overweight: the median BMI was 28.7 (25.8–31.9) kg/m^2. 68.1% (1,580/2,321) of the patients had hypertension, 34.6% (803/2,321) dyslipidaemia and 31.2% (724/2,321) history of smoking. 17.3% (401/2,321) of the studied group reported having a positive family history of cardiovascular diseases. Furthermore, 13.5% (313/2,321) of the involved ones suffered from diabetes. There were 404/2,321 (17.4%) patients who mentioned any chest pain before the coronary CTA examination without previously known CAD.

Comparison of patients with and without any chest pain

Patients with and without any chest pain were compared (Table 2). Patients with chest pain were older [64.8 (58.0–70.3) vs. 62.6 (53.5–68.9), P < 0.001], majority female [68.1% (1,580/2,321) vs. 42.5% (814/1,917), P < 0.001], having hypertension [72.3% (292/404) vs. 67.2% (1,288/1,917), P = 0.048], reporting positive family history for cardiovascular disease [24.3% (98/404) vs. 15.8% (303/1,917), P < 0.001] and having peripheral vascular disease [7.7% (31/404) vs. 4.0% (77/1,917), P < 0.001]. Obstructive CAD was in equal distribution in both groups [22.5% (91/404) vs. 20.7% (397/1,917), P = 0.416].

According to our multivariable analysis, factors associated with any chest pain were age >65 years (OR = 1.30; 95% CI, 1.03–1.64; P = 0.028), female sex (OR = 1.84; 95% CI, 1.47–2.30; P < 0.001), positive cardiovascular family history (OR = 1.70; 95% CI, 1.30–2.22; P < 0.001) and peripheral vascular disease (OR = 1.74; 95% CI, 1.11–2.75; P = 0.016). CAD was not associated with the symptom of chest pain (OR = 1.06; 95% CI, 0.80–1.39; P = 0.693) (Table 3).

Coronary artery status of the studied population

We analyzed the stenosis severity on all coronary artery segments. In our patient population, 577/2,321 (24.9%) had no stenosis, in 573/2,321 (24.7%) minimal, and in 683/2,321 (29.4%) mild stenosis was observed. Moderate stenosis was found in 311/2,321 (13.3%) of the patients, severe stenosis in 151/2,321 (6.5%) and in 26/2,321 (1.1%) occluded coronary arteries were diagnosed (Fig. 2). In total, 488 (21.0%) patients were diagnosed with obstructive CAD (≥50% luminal stenosis). The total CACS was median 17.8 [0.0–168.6].

Next, we determined different factors associated with prevalent obstructive CAD by uni- and multivariable tests (Table 4). In multivariable analysis age >65 years (OR = 2.51; 95% CI, 2.02–3.13; P < 0.001), male sex (OR = 1.59; 95% CI, 1.28–1.98; P < 0.001), hypertension (OR = 1.40; 95% CI, 1.08–1.81; P = 0.012), diabetes (OR = 1.50; 95% CI, 1.13–1.99; P = 0.006), dyslipidaemia (OR = 1.33; 95% CI, 1.07–1.66; P = 0.011) and smoking history (OR = 1.34; 95% CI, 1.07–1.68; P = 0.011) were associated with obstructive CAD.
patients admitted to the hospital due to chest pain and relevant CAD [16]. Brown et al., presented, that 140 atrial fibrillation patients with chest pain syndromes had no increased risk for acute coronary syndrome compared to 683 matched control subjects (11.4% vs. 10.8%) [17]. Graf et al., studied 79 patients with typical chest pain (without reporting cardiac rhythm) and normal epicardial coronary arteries, where 65% had reduced coronary flow reserve [18]. They established as well, that clinical cardiac risk factor analysis may help in prediction of the individual probability of microvascular dysfunction [18].

In our cohort, chest pain was not related to obstructive CAD. Elderly, female patients, ones with a positive family history of cardiovascular disease, and patients suffering from peripheral vascular disease or hypertension were more likely to have chest pain. The difference between the associated factors for chest pain and obstructive CAD suggests that the reported chest pains are rather noncardiac or related to atrial fibrillation. These findings highlight the importance of coronary diagnostics in patients undergoing pre-ablation cardiac CTA, while patients without any symptoms could have hidden CAD as well.

**Table 2** Comparison of traditional cardiovascular risk factors of patients with and without any chest pain determined by Mann–Whitney U-test (number, percentage) and Chi-square test (median, interquartile range)

| Parameters                      | Any chest pain (n=404) | Without chest pain (n=1917) | P     |
|---------------------------------|------------------------|-----------------------------|-------|
| Age (years)a                    | 64.8 (58.0–70.3)       | 63 (54–69)                  | <0.001|
| Female sexb                     | 238 (58.9%)            | 814 (42.5%)                 | <0.001|
| BMI (kg/m²)b                    | 28.7 (25.9–32.1)       | 28.7 (25.6–31.6)            | 0.679 |
| Hypertensionb                   | 252 (72.3%)            | 1288 (67.2%)                | 0.048 |
| Diabetesb                       | 53 (13.1%)             | 260 (13.6%)                 | 0.807 |
| Dyslipidaemiaa                  | 145 (36.0%)            | 658 (34.4%)                 | 0.534 |
| History of smokingb             | 127 (31.7%)            | 597 (31.3%)                 | 0.886 |
| Positive family history of CVd  | 98 (24.3%)             | 303 (15.8%)                 | <0.001|
| Peripheral vascular diseaseb    | 31 (7.7%)              | 77 (4.0%)                   | 0.001 |
| Prior stroke/TIA                 | 25 (6.2%)              | 101 (5.3%)                  | 0.460 |
| Obstructive CAD                  | 91 (22.5%)             | 397 (20.7%)                 | 0.416 |

CAD, coronary artery disease; CV, cardiovascular; TIA, transient ischemic attack.

**Table 3** Factors associated with any chest pain reported as determined by uni- and multivariable analysis, using logistic regression

| Parameters                      | Univariate OR (95% CI) | P     | Multivariable OR (95% CI) | P     |
|---------------------------------|------------------------|-------|---------------------------|-------|
| Age >65years                    | 1.43 (1.15–1.77)       | 0.001 | 1.30 (1.03–1.64)          | 0.028 |
| Female sex                      | 1.94 (1.56–2.42)       | <0.001| 1.84 (1.47–2.30)          | <0.001|
| BMI >25kg/m²                    | 0.97 (0.74–1.28)       | 0.837 | 0.98 (0.72–1.37)          | 0.758 |
| Hypertension                    | 1.27 (1.00–1.61)       | 0.048 | 1.20 (0.92–1.56)          | 0.170 |
| Diabetes                        | 0.96 (0.70–1.32)       | 0.807 | 0.81 (0.58–1.14)          | 0.234 |
| Dyslipidaemia                   | 1.07 (0.86–1.34)       | 0.535 | 0.93 (0.73–1.18)          | 0.563 |
| History of smoking              | 1.02 (0.81–1.28)       | 0.886 | 0.97 (0.76–1.24)          | 0.822 |
| Positive family history of CV   | 1.71 (1.31–2.21)       | <0.001| 1.70 (1.30–2.23)          | <0.001|
| Peripheral vascular diseaseb    | 2.00 (1.30–3.07)       | 0.002 | 1.74 (1.11–2.75)          | 0.016 |
| Prior stroke/TIA                | 1.19 (0.76–1.87)       | 0.460 | 0.94 (0.59–1.51)          | 0.803 |
| Obstructive CAD                 | 1.11 (0.86–1.44)       | 0.416 | 1.06 (0.80–1.39)          | 0.693 |

CAD, coronary artery disease; CI, confidence interval; CV, cardiovascular; OR, odds ratio; TIA, transient ischemic attack.

**Table 4** Factors associated with prevalent obstructive coronary artery disease as determined by uni- and multivariable analysis, using logistic regression

| Parameters                      | Univariate OR (95% CI) | P     | Multivariable OR (95% CI) | P     |
|---------------------------------|------------------------|-------|---------------------------|-------|
| Age >65years                    | 2.68 (2.19–3.30)       | <0.001| 2.51 (2.02–3.13)          | <0.001|
| Female sex                      | 0.78 (0.64–0.96)       | 0.018 | 0.63 (0.50–0.78)          | <0.001|
| BMI >25kg/m²                    | 1.14 (0.88–1.48)       | 0.323 | 1.00 (0.75–1.32)          | 0.994 |
| Hypertension                    | 1.97 (1.56–2.50)       | <0.001| 1.40 (1.08–1.81)          | 0.012 |
| Diabetes                        | 2.07 (1.59–2.68)       | <0.001| 1.50 (1.11–1.99)          | 0.006 |
| Dyslipidaemia                   | 1.67 (1.36–2.04)       | <0.001| 1.33 (1.07–1.66)          | 0.011 |
| History of smoking              | 1.23 (1.00–1.52)       | 0.054 | 1.34 (1.07–1.68)          | 0.011 |
| Positive family history of CV   | 1.11 (0.85–1.43)       | 0.444 | 1.10 (0.83–1.45)          | 0.516 |
| Peripheral vascular diseaseb    | 2.04 (1.34–3.05)       | <0.001| 1.40 (0.90–2.18)          | 0.136 |
| Prior stroke/TIA                | 1.67 (1.12–2.46)       | 0.010 | 1.39 (0.92–2.12)          | 0.121 |
| Any chest pain                  | 1.11 (0.86–1.44)       | 0.416 | 1.08 (0.81–1.40)          | 0.676 |

CI, confidence interval; CV, cardiovascular; OR, odds ratio; TIA, transient ischemic attack.

**Discussion**

**Main findings**

The main finding of our study is the relatively high incidence (21.0%) of coronary artery luminal stenosis in atrial fibrillation patients regardless of the reported symptoms. The incidence of obstructive CAD was similar in patients with and without any chest pain (22.5% vs. 20.5%). Factors associated with chest pain differed from the parameters associated with obstructive CAD.

**Relationship of chest pain and coronary artery disease in atrial fibrillation patients**

There is still scarce evidence about the relationship between chest pain and atrial fibrillation. In a recent publication, Rottlander et al., conclude that there might be only a weak association between atrial fibrillation

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Relevant stenosis (≥75% luminal reduction) was observed in 27.7% of the 238 patients with atrial fibrillation with the exclusion of myocardial ischemia [20]. In multiple studies ≥ or >50% luminal stenosis of main coronary arteries was defined as CAD: Weijs et al. found underlying CAD in 49% of paroxysmal atrial fibrillation population including 390 patients [14], while Nucifora et al. reported similar, 41% obstructive CAD cases among 150 patients with atrial fibrillation [21]. In a recent study involving 94 patients, only 26% had obstructive CAD on coronary CTA [13]. In our current study, the most widely used definition of CAD, as 50% equal or more of luminal stenosis was applied. CAD was found with a high-resolution CT scanner even in one-fifth of our studied population involving more than 2000 patients.

Risk factors for CAD vary extensively. The CADAF-CT trial identified male sex, high number of co-existing coronary risk factors, elevated BNP levels, enlarged left atrial volume, high CACS, as independent risk factors of myocardial ischemia in 757 patients with atrial fibrillation [11,22]. Weijs et al. reported similar predictors for luminal stenosis while comparing 115 paroxysmal atrial fibrillation patients with ones with constant sinus rhythm [14]. Also, the presence of atrial fibrillation was named as a risk factor for obstructive CAD [14,21]. Besides male sex, age, diabetes, Framingham score and CHA2DS2-VASc score proved to be good predictors for CAD in a retrospective analysis of Rottlander et al. involving 566 paroxysmal or newly diagnosed atrial fibrillation patients [16]. However, according to Chen et al., CACS was also observed without any conventional cardiovascular risk factors in 58% of the studied 324 patients [8].

In our study, several parameters were associated with obstructive CAD in atrial fibrillation patients. Obstructive CAD is threefold more likely among patients >65 years old, and nearly twofold more likely in males and in patients with diabetes as well. Hypertension, dyslipidemia and a history of smoking are also significant variables anticipating obstructive CAD. Interestingly, positive family history, and obesity did not show any correlation with CAD.

**Clinical impact of our study**

Our recent investigation underlines the importance of defining the coronary artery status in atrial fibrillation patients. The determined associated factors and relatively high number of novel obstructive CAD suggest that it is worth extending the routine pre-ablation left atrial CT examination with characterization for coronary artery stenosis even for patients with no chest pain. For atrial fibrillation treatment, the holistic ‘ABC’ pathway is recommended by the European Society of Cardiology’s new prevention guideline [9]. The identification and management of concomitant diseases and cardiometabolic risk factors (‘C’) plays an equal role as the anticoagulation (‘A’) and better symptom (‘B’) management [9]. Since a newly diagnosed obstructive CAD could raise the ischemic risk of atrial fibrillation patients, lifestyle changes, closer clinical follow-up, altered medical treatment (e.g. statins, antiplatelets), further investigations (e.g. stress echocardiography, coronarography) could be needed based on other individual risk factors and chronic diseases [9].

A newly diagnosed obstructive CAD would reevaluate the CHA2DS2-VASc score as well, which potentially would modify the anticoagulation strategy in patients with atrial fibrillation. One observational retrospective review concluded that atrial fibrillation patients with obstructive CAD had a higher incidence of thromboembolic events (ischemic stroke and systemic thromboembolism) compared to controls adjusted for CHA2DS2-VASc score components and relevant variables [23]. In multiple studies, after finding incidental CAD by coronary CTA, CHA2DS2-VASc reclassification was needed in 20–50% of the involved atrial fibrillation patients [8,13,24]. Thus, anticoagulation treatment needed to be modified in 20%
of the cohort according to the observation of Wang et al. [24].

Limitations
The most important limitation is the retrospective nature of the study. Unfortunately, there was a significant lack of follow-up of the patients regarding the results of further investigations advised based on the coronary CTA image. Thus, we could not demonstrate our investigation’s ultimate clinical impact. Additionally, some patients were excluded from our study, due to the poor quality caused by tachyarrhythmia. It is possible, it added a selection bias to our study.

Conclusions
The incidence of obstructive CAD (≥50% luminal stenosis) among patients awaiting ablation for atrial fibrillation is high (21.0%). Any kind of chest pain was not related to the incidence of obstructive CAD. Our study highlights the importance of CAD diagnostics in patients awaiting ablation regardless even in patients experiencing no chest pain. A newly diagnosed obstructive CAD could raise the ischemic and thromboembolic risk affecting further medical treatment strategies.

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Conflicts of interest
G.S. reports personal fees from Abbott, Bayer, Boston Scientific and Johnson and Johnson Medical outside the submitted work. The remaining authors have no conflicts of interest.

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