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Evaluation of coronary stenosis versus plaque burden for atherosclerotic cardiovascular disease risk assessment and management

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Abstract

Purpose of review—To provide a summary of recent literature on the relative impact of luminal stenosis versus atherosclerotic plaque burden in atherosclerotic cardiovascular disease (ASCVD) risk stratification and management.

Recent findings—Recent results from both randomized controlled clinical trials as well as observational cohort studies have demonstrated that ASCVD risk is mediated mainly by the extent of atherosclerotic disease burden rather than by the presence of coronary stenosis or inducible ischemia. Although patients with obstructive CAD are generally at higher risk for ASCVD events than patients with nonobstructive CAD, this is driven by a higher plaque burden in those with obstructive CAD. Accordingly, the ASCVD risk for a given plaque burden is similar in patients with and without obstructive CAD. Accompanying these observations are randomized controlled trial data, which show that optimization of medical therapy instead of early revascularization is most important for improving prognosis in patients with stable obstructive CAD.

Summary—Emerging evidence shows that atherosclerotic plaque burden, and not stenosis per se, is the main driver of ASCVD risk in patients with CAD. This information challenges the current paradigm of selecting patients for intensive secondary prevention measures based primarily on the presence of obstructive CAD.

Keywords

computed tomography; computed tomography angiography; coronary artery calcium; coronary heart disease; luminal stenosis

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Conflicts of interest

There are no conflicts of interest.

INTRODUCTION

Coronary artery disease (CAD) is characterized by atherosclerotic plaque accumulation in the coronary arteries, which is a prolonged and indolent process that may or may not progress towards luminal obstruction [1]. Historically, evaluation and definition of CAD has mainly focused on the presence of at least one coronary stenosis at least 50% or provokable ischemia on stress testing. The reason for this historical focus is easy to understand. First, the explicit role of obstructive CAD in causing cardiac ischemia and angina has created a large interest in revascularization procedures with the aim to relieve symptoms and improve quality of life. Second, patients with obstructive CAD have a high risk for subsequent atherosclerotic cardiovascular disease (ASCVD) events, and therefore, qualify for intensive preventive interventions – so-called secondary prevention [2–4]. Together, this has generated the widespread belief that it is the stenotic atherosclerotic lesions that are responsible for the high risk of ASCVD events in patients with obstructive CAD. This long-held belief, however, has been challenged by randomized controlled trials of revascularization (‘removing the stenosis’) in patients with stable obstructive CAD, such as COURAGE (Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation) [5], FAME-II [6], and ISCHEMIA [7], which have not been able to show reductions of hard endpoints, such as myocardial infarction, stroke or cardiovascular death compared with medical therapy alone. The failure of revascularizations to reduce hard events have come as a surprise to many in the cardiovascular community but the explanation may be quite simple; the presence and extent of obstructive CAD is strongly correlated with the total atherosclerotic plaque burden, that is, plaque burden is generally higher in patients with obstructive CAD than in those with no or nonobstructive CAD. Likewise, patients with multivessel obstructive CAD generally have a higher atherosclerotic plaque burden than patients with single vessel obstructive CAD. As a consequence, the high risk of ASCVD events in patients with obstructive CAD, may therefore, be because of the high plaque burden with multiple atherosclerotic lesions distributed throughout the coronary arteries rather than by the solitary stenotic lesion per se [8]. In the present review, we summarize recent evidence that shows that the main determinant of ASCVD risk in patients with obstructive CAD is in fact the extent of plaque burden. This information is important for several reasons. First, it challenges the current prevention paradigm that are largely based on the presence of obstructive CAD or not. Further, it demonstrates that the risk for ASCVD is not uniformly high in patients with obstructive CAD; those with low plaque burden have low risk for ASCVD despite obstructive CAD. Finally, patients with nonobstructive CAD may have higher ASCVD risk than patients with obstructive CAD if their plaque burden is higher. Together, this suggests that guidelines should focus more on plaque burden than on the presence of obstructive CAD when selecting patients for intensive preventive interventions.

CORONARY PLAQUE BURDEN, LUMINAL STENOSIS, AND ATHEROSCLEROTIC CARDIOVASCULAR DISEASE RISK

Coronary computed tomography angiography (CTA) has helped to generate a new stage in ASCVD risk assessment, enabling the simultaneous evaluation of coronary artery lumen and

total coronary atherosclerotic plaque burden [9]. Assessment of the coronary lumen allows for the diagnostic evaluation of CAD in symptomatic patients by identifying obstructive atherosclerotic lesions that are responsible for the reported symptoms, and thus, may be targeted with revascularization to provide symptom relief. Atherosclerotic plaque burden allows for the prognostic evaluation of CAD and can be measured on noncontrast CT using the coronary artery calcium (CAC) score and/or by CTA that also visualizes noncalcified plaque. Although the optimal method for quantification of atherosclerosis burden is not clear, assessment of calcified and noncalcified plaque may be complimentary to each other whenever evaluating plaque burden and individual plaques. Higher calcium density is associated with plaque stability and the temporal length of disease [10], whereas spotty calcification, noncalcified, and low attenuation plaques correlate with lesion vulnerability and early stages of the atherosclerotic process [11].

Although calcified and noncalcified plaque represent distinct stages of the atherosclerotic process [12], both measures robustly associate with long-term ASCVD risk. In particular, an abundance of evidence has shown that CAC is one of the most robust noninvasive markers of long-term ASCVD risk [13]. In one of the largest studies of its kind completed to date, Mortensen *et al.* evaluated the value of CAC on noncontrast CT, independent of lumen stenosis on CTA, for predicting incident myocardial infarction, stroke, and/or all-cause mortality among nearly 24 000 symptomatic participants of the Western Denmark Heart Registry who were followed for over 4 years [8[■]]. Here, there was a strong association between CAC and obstructive disease, as the prevalence of obstructive CAD and number of vessels with at least 50% stenosis increased across higher CAC scores. Although there was a graded increase in major ASCVD events and all-cause death with both higher CAC scores and obstructive CAD severity (Fig. 1), there were no significant differences in the composite outcome between individuals with obstructive versus no or nonobstructive CAD within a given CAC burden (Fig. 2). Furthermore, the findings remained consistent after excluding individuals who underwent revascularization within 90 days of coronary CTA testing. In totality, the results of this study completed in the Western Denmark Heart Registry demonstrated that calcific plaque burden is as strongly associated with ASCVD risk irrespective of luminal stenosis, and that patients with nonobstructive CAD but high CAC scores carry similar risk for ASCVD events as patients with obstructive CAD.

Similar observations have been noted among more than 27 000 patients without chest pain in the CONFIRM (Coronary CT Angiography Evaluation for Clinical Outcomes) study. Investigators in CONFIRM specifically found that the addition of coronary CTA measures to a model including traditional ASCVD risk factors and CAC did not significantly improve all-cause mortality discrimination, providing less than a 0.01 increment increase in the C-statistic [14]. In addition, there was limited ability of coronary CTA to improve reclassification for all-cause mortality and nonfatal myocardial infarction when added to a model including traditional risk factors and CAC. In contrast, the addition of CAC scores to models containing traditional risk factors significantly improved risk discrimination and net reclassification for all-cause mortality and composite ASCVD events. Although the findings in CONFIRM study demonstrated that both CAC and coronary CTA independently predict mortality and nonfatal myocardial infarction beyond traditional risk factors, there were limited improvements in risk prediction when adding coronary CTA data to the evaluation of

CAC and traditional risk factors. Overall, these findings suggest that information on luminal stenosis does not provide prognostic value beyond its association with plaque burden [13].

Although the above studies performed in the Western Denmark Heart Registry and CONFIRM assessed plaque burden using the CAC score, additional prognostic information may be derived by assessing noncalcified (fibrous and lipid-rich) plaque burden and location. For example, low attenuation plaque (<30 Hounsfield Units) detected on coronary CTA is more strongly associated with future risk of myocardial infarction than CAC, traditional risk factors, and stenosis severity among persons with stable angina. In particular, the presence of a low-attenuation plaque burden of greater than 4% was independently associated with an approximate five-fold increase for fatal and nonfatal myocardial infarction [11]. Hou and colleagues like-wise found that coronary CTA measures including the composition of the plaques (calcified, noncalcified, or mixed), location (left main disease), and extent of disease provided further incremental improvement (AUC 0.93) for the prediction of major adverse cardiovascular events when added to traditional risk factors and CAC (AUC = 0.82) among more than 5000 patients with suspected obstructive CAD [15]. The 3-year probability of a major adverse cardiovascular event were also higher for noncalcified and mixed plaque morphologies versus calcified plaques [15]. Importantly, a reduction in lipid-rich necrotic core volume on coronary CTA is associated with plaque stability, and statin therapy have been shown to reduce noncalcified plaque lesions while increasing calcified plaque burden [16].

Although the prognostic information of plaque burden can easily be used to improve management of patient's with nonobstructive CAD by identifying those at high risk who needs intensive preventive therapies, the situation is more challenging in patients with obstructive CAD because of collinearity of plaque burden and stenosis. Indeed, coronary plaque burden and stenosis are strongly associated with one another [17]. Among persons with stable chest pain, the correlation between low-attenuation plaque burden, and stenosis severity are 0.83 and a similarly strong association has been observed for CAC burden and the presence of at least 50% stenosis [8[■],11]. The strength of the relationship between plaque burden and stenosis becomes particularly problematic to disentangle in risk prediction modeling even with multivariable adjustment as event rates increase across both parameters of interest. Thus, although plaque burden is the most important determinant of ASCVD risk, much of the information that plaque burden provide are indirectly used when assessing whether patients with obstructive CAD have either one-vessel, two-vessel, or three-vessel disease. Therefore, future studies that assess the associations of noncalcified and calcified plaque burden versus stenosis on the per-vessel level while utilizing additional parameters, such as the regional distribution and location of lesions [18,19], may help further improve downstream ASCVD risk beyond overall plaque burden and the number of vessels with obstructive disease.

IMPLICATIONS FOR CLINICAL MANAGEMENT

Most coronary events arise from patients without obstructive CAD and even in patients with known obstructive CAD, the majority of lesions giving rise to acute coronary events are located in vessel segments without significant stenosis prior to the event [20–23].

For instance, more than one-half of all major cardiovascular events in the Prospective Multicenter Imaging Study for Evaluation of Chest Pain (PROMISE) occurred among participants who had no evidence of provokable ischemia [24] and approximately 65% of events in the Western Denmark Registry analysis were among participants without obstructive disease [8[■]]. Therefore, identifying patients who have significant nonobstructive CAD should become a greater focus of guideline strategies aimed at reducing the ASCVD burden in the population. Given that plaque burden, and not stenosis per se, is the major determinant of ASCVD risk, the current paradigm where intensive secondary preventive therapies are mainly reserved for patients with obstructive CAD miss great opportunities for ASCVD prevention. Indeed, patients with extensive nonobstructive CAD are rarely considered candidates for intensive preventive treatments even though they may be at higher risk than many patients with known obstructive CAD. However, there is a need to better clarify how the quantity of plaque burden (e.g. CAC scores) should affect the intensity of preventive interventions, including life-style changes and pharmacotherapies. Initial work in this area has, for example, indicated that the utility of aspirin therapy for primary prevention outweighs the risk of major adverse bleeding events among individuals with CAC at least 100 [25,26]. Likewise, among individuals without clinical ASCVD referred for CAC scoring, statin therapy is associated with a significantly lower risk (24%) of major adverse cardiovascular events *only* in persons with prevalent CAC, and the number needed to treat (NNT) to prevent one event was significantly associated with CAC severity (NNT = 100 for CAC 1–100, and NNT = 12 for CAC >100) [27]. Collectively, these findings suggest that decisions regarding the initiation of preventive pharmacotherapies should, at least partly, be determined by the presence of subclinical atherosclerosis whereas the intensity of treatment should be guided by the extent of atherosclerotic plaque (i.e. burden and location).

The utility of coronary CTA in guiding clinical management arises from its ability to simultaneously assess stenosis and atherosclerotic plaque burden, which can thus be applied to individuals with both obstructive and nonobstructive disease. Among patients with stable CAD, the utilization of coronary CTA has been shown to reduce coronary events and/or nonfatal myocardial infarction mortality by 41% over a 5-year follow-up compared with standard therapy alone in the SCOT-HEART (Scottish Computed Tomography of the Heart) trial [28]. Approximately 35% of patients in the CTA group had nonobstructive CAD, and the risk reduction was mediated by changes in management as individuals randomized to coronary CTA were 40% more likely to have started preventive therapies compared with those in the standard therapy group. Importantly, the lower rates of events in the CTA group were independent of coronary angiography and/or revascularization, and these results support the broader clinical trial data, which show that early revascularization does not lead to better outcomes among individuals with stable CAD compared with optimal medical therapy alone [5,7[■],29]. In the COURAGE trial [5], for example, percutaneous coronary intervention did not significantly reduce the risk of death, myocardial infarction or stroke when added to medical therapy alone for individuals with stable CHA who had objective evidence of ischemia, and these results are supported by the more recent FAME-2 (The Fractional Flow Reserve versus Angiography for Multivessel Evaluation 2) [6] and ISCHEMIA (International Study of Comparative Health Effectiveness with Medical and Invasive Approaches) trials. Specifically, ISCHMIEA showed a lack of utility

of early angiography and/or revascularization when compared with optimal medical therapy in the same patient population [7[■]]. Overall, these results further underline the role of atherosclerotic plaque burden in driving acute coronary syndromes irrespectively of the presence of coronary stenosis or not. Indeed, unstable atherosclerotic lesions that cause acute coronary syndrome most often arise from atherosclerosis in nonobstructive coronary segments [12,29–31].

COMPUTED TOMOGRAPHY VERSUS COMPUTED TOMOGRAPHY ANGIOGRAPHY FOR EVALUATION OF CORONARY ARTERY DISEASE

The utilization of cardiac CT versus coronary CTA should consider both the symptoms and acuity of each particular patient. In symptomatic patients, coronary CTA is the preferred imaging modality as it provide both diagnostic (is there stenosis?) and prognostic (plaque burden) information regarding CAD severity [11,15,16,32]. Quantification of CAC on noncontrast cardiac CT does not provide information on lumen stenosis, and is therefore, not recommended as the test of choice during the evaluation of symptomatic patients, as absence of CAC does not exclude the presence of obstructive CAD. Specifically, a significant proportion of symptomatic younger patients with CAC = 0 may still have noncalcified plaque and/or significant stenosis [8[■],11] that may require revascularization to relive symptoms. Thus, one of the limitations of CAC scoring is that calcification reflects one of the last stages of the atherosclerotic process and may not be the most sensitive test in younger patients or symptomatic patients with early obstructive CAD. Accordingly, recent European Society of Cardiology guidelines provided coronary CTA and CAC testing a class I and class II, respectively, for their use in low–moderate risk symptomatic patients with suspected obstructive CAD [2].

CONCLUSION

Emerging evidence from both observational and randomized controlled trials demonstrate that ASCVD risk is mainly mediated by the extent of atherosclerosis burden and not by the presence of stenosis per se. Thus, for a given atherosclerotic plaque burden, ASCVD risk is similar regardless of the presence of obstructive CAD or not. For clinicians, it is important to understand the differential diagnostic and prognostic value of coronary stenosis versus coronary plaque burden when evaluating CAD. Although revascularization of coronary stenoses is important for relieving symptoms in symptomatic patients with obstructive CAD, the prognosis is mainly determined by the extent of coronary atherosclerosis, which can help guide treatment intensity. Taken together, this challenges the current preventive paradigm where patients are selected for intensive secondary prevention measures based mainly on the presence of obstructive CAD and suggest that guidelines should shift focus to plaque burden for better capturing patients at high ASCVD risk irrespectively of the presence of stenoses.

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■ of special interest

■ ■ of outstanding interest

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KEY POINTS

- Plaque burden is the primary driver of ASCVD risk among patients with obstructive and nonobstructive CAD.
- The ASCVD risk for a given plaque burden is similar in patients with and without obstructive CAD.
- Among patients with stable obstructive CAD, early revascularization fails to reduce major ASCVD events and optimization of medical therapy is preferred.
- Independent of luminal stenosis, plaque burden may help to identify the patients most likely to derive maximal benefit from both primary and secondary prevention treatments.

FIGURE 1.

Major atherosclerotic cardiovascular disease event and all-cause mortality event rate across increasing coronary artery calcium burden and stenosis severity. *Myocardial infarction, stroke, and all-cause mortality.

FIGURE 2.

Hazard for myocardial infarction, stroke, and all-cause mortality for persons with and without obstructive coronary artery disease, stratified by coronary artery calcium score.