

The Power and the Limits of Zero: Strengths and Pitfalls of CT Coronary Artery Calcium Score in Risk Assessment

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Abstract

Assessing cardiovascular risk by measuring coronary artery calcium (CAC) scoring obtained by computed tomography is well understood and is an accepted practice. It determines the amount of calcium in the atherosclerotic plaque present in the coronary arteries with significant accuracy and establishes the risk of suffering a future cardiac event. CAC scoring enhances the value of traditional risk factors, facilitating improved patient management. In this review, we examined a case with a CAC scoring result and analysed the case to see how it influenced the decision-making process. The case illustrates how decision-making is assisted by CAC scoring in comparison with other cardiac tests, particularly as a basis for initiating preventive therapies, such as statins. Although of such great importance, CAC also has some limitations. CAC cannot identify non-calcified plaque, and in some cases, it can be misleading. This narrative review, supplemented from a practical standpoint, suggests that CAC scanning is generally cost-effective in intermediate-risk groups because it avoids unnecessary lifelong statin therapy in low-risk individuals while targeting therapy to those with demonstrable atherosclerosis. However, considerations of radiation (approximately 1 mSv per scan) and potential downstream testing from incidental findings must be weighed. Most guidelines recommend. From a practical aspect, CAC scanning is generally cost-effective in intermediate-risk groups because it avoids unnecessary lifelong statin therapy in low-risk individuals while targeting therapy to those with demonstrable atherosclerosis. However, considerations of radiation (approximately 1 mSv per scan) and potential downstream testing from incidental findings must be weighed. Most guidelines recommend the selective use, rather than widespread population screening, of cardiovascular risk assessment

to balance cost, benefit, and safety.

Keywords

Coronary Artery Calcium Score, Cardiovascular Risk Assessment, Agatston Score, Statins, Coronary Artery Atherosclerosis

1. Introduction

Cardiovascular disease remains one of the leading causes of death around the globe, and determining the individual risk level is critical for preventing it [1]. Traditional approaches, like the Framingham risk score and ASCVD risk estimator, include variables like age, cholesterol, blood pressure, diabetes, and smoking status, which are helpful, but patients may be misclassified, as the approaches do not indicate whether atherosclerosis is present [1]. In the last twenty years, the measurement of coronary artery calcium (CAC) has emerged as a valuable imaging technique for determining the presence of early coronary atherosclerosis and improving risk prediction. Non-contrast cardiac CT is used to assess a patient's coronary arteries for calcified plaques and subsequently determines the burden using the Agatston score [1]. The score, expressed in Agatston units, indicates the entire burden of calcified plaques in the arteries. The higher the score, the more calcified plaque is present in the arteries, leading to an increased risk of coronary events [2]. In contrast, a score of zero (no calcification) is a very strong negative marker associated with a very low risk in the short term. This is often called the "power of zero" [2].

Clinically, CAC scores are stratified into risk categories: 0 (no calcium), 1 - 99 (mild), 100 - 399 (moderate), and ≥ 400 (high/severe) [1]. These categories correlate with gradations of 10-year coronary heart disease risk, ranging from well below 5% when CAC is zero to significantly higher rates when CAC is very elevated [3]. Guidelines in preventive cardiology have incorporated CAC scoring as an optional tool to improve risk stratification, particularly in patients at borderline or intermediate risk where treatment decisions (such as initiating statin therapy) are uncertain [3]. CAC testing can thus refine risk estimates by identifying individuals who harbor substantial atherosclerosis despite few risk factors, or conversely those with zero calcium whose risk might be overestimated by algorithms [2] [3]. This expanded risk insight allows more personalized preventive strategies.

2. Case Report

A European woman aged 67 with hyperlipidemia and a family history of premature coronary artery disease underwent cardiovascular risk evaluation. The patient had an LDL cholesterol level of 4.2 mmol/L (~160 mg/dL) and was an ex-smoker (30 pack-year history, quit 13 years prior). She had no history of hypertension or diabetes but had a BMI of 33. The patient denied typical angina, re-

porting only mild shortness of breath on exertion and no chest pain. A family history of premature coronary artery disease (a sibling required coronary stenting at age 48) and high cholesterol levels warranted a stress echocardiogram to screen for occult ischemia. She exercised for 6 minutes (achieving ~7 METs) without chest discomfort. The stress ECG showed no ischemic ST changes, and echocardiographic imaging demonstrated normal left ventricular systolic function with no wall motion abnormalities induced by exercise. The stress test was thus negative for myocardial ischemia at a moderate workload.

Despite the reassuring stress echo, the patient's cardiologist and general practitioner (GP) remained concerned about her cardiovascular risk, given her risk factors, and recommended that she commence using a lipid-lowering agent. For more than a decade, she was reluctant to begin statin therapy due to fear of side effects from statins. To further clarify her coronary risk, a CT coronary calcium score test was obtained. The non-contrast CT scan revealed extensive coronary calcifications, with a total Agatston CAC score of 465. The scan was acquired using a non-contrast, ECG-gated multidetector CT protocol with 3-mm slice thickness, which is consistent with standard acquisition methods for calcium scoring. This score is markedly elevated (indicating severe atherosclerotic plaque burden) for a woman in her sixties. The calcium was noted predominantly in the right coronary artery distribution, while it was reported as zero in the left coronary artery distribution. In light of this finding, the patient was started on intensive preventive therapy, including aspirin and rosuvastatin, a statin lipid-lowering medication. However, only five days after the commencement of taking rosuvastatin, the patient reported intolerance to it, and the GP thus switched it over to ezetimibe. Shortly thereafter, she reported some worsening exertional dyspnea and occasional jaw discomfort with exercise, raising concern for significant coronary artery disease that had not been apparent on the stress test.

The patient underwent invasive coronary angiography to obtain a definitive evaluation. Angiography revealed multi-vessel obstructive coronary disease with severe calcific stenosis in the mid right coronary artery, high-grade stenosis in the obtuse marginal and diagonal branches, and significant lesions (60% - 70% stenosis) in the mid to distal left anterior descending artery. Due to the diffuse, calcified nature of her disease, the joint cardiology-cardiothoracic team advised surgical revascularization. The patient was then able to undergo a triple coronary artery bypass graft surgery with successful grafting to the diseased regions of the coronary arteries. Post-surgery, the patient recovered uneventfully. This case exemplifies a paradox where the left coronary artery distribution reported a CAC score of zero, despite the total Agatston score being 465, alongside a normal echocardiogram. It reveals the pitfalls of calcium scoring in risk assessment. A zero score, even with non-invasive contouring of the arteries and the absence of ischemia on a stress test, does not necessarily rule out significant disease.

3. Clinical Application of Calcium Scores

As illustrated in **Table 1**, CAC scoring has become an important adjunct in car-

diovascular risk assessment when compared to other cardiac investigations, especially for guiding primary prevention strategies. Current guidelines endorse selective use of CAC testing in asymptomatic adults at borderline or intermediate 10-year risk (generally between ~5% and 20% 10-year ASCVD risk) when the decision to initiate therapies like statins is uncertain [3]. In many patients, the CAC score helps adjust risk estimates and guide management.

CAC = 0 (no calcification): This indicates very low short-term risk. Preventive therapy can often be deferred. The 2018 ACC/AHA Cholesterol Guideline and the 2021 European Society of Cardiology (ESC) Prevention Guideline recommend withholding statin treatment when CAC is 0, provided no high-risk features are present, and reassessing in 5 - 10 years [3] [4]. A zero score “de-risks” the patient, as large studies show 10-year coronary event rates of about 1% - 5% in this group [3]. This is known as the “power of zero,” which reflects a favourable prognosis over the next 5 - 15 years [2]. Important exceptions include patients with diabetes, persistent smoking, or strong family history, in whom preventive therapy may still be advised despite CAC 0 [3].

CAC 1 - 99 (mild calcification): This range signals early atherosclerosis. Guidelines suggest that any CAC > 0 supports at least considering statin therapy, especially in middle-aged or older patients [3]. The risk increases approximately 2- to 3-fold compared to CAC 0 [4]. In practice, lifestyle measures are reinforced, and moderate-intensity statin therapy is usually recommended, particularly in those over 55 years [3].

CAC 100 - 399 (moderate calcification): This level reflects a substantial plaque burden. Patients in this category often have a 10-year risk greater than 7.5% - 10% and gain clear benefit from intensive prevention. Statin therapy is strongly indicated. Additional measures, such as aspirin, may be considered in select cases, although the routine use of aspirin in primary prevention is debated. In some cases, a CAC in this range may also necessitate further anatomical evaluation, such as CT angiography, particularly if the patient is experiencing symptoms.

CAC ≥ 400 (severe calcification): This denotes extensive coronary calcification and is linked with a high probability of obstructive coronary disease. Scores above 400, and especially above 1000, are associated with annual event rates similar to those in secondary prevention populations [1]. Ten-year CHD risk in this group is often well above 20% [3]. Aggressive risk factor management is essential, including high-intensity statin therapy and strict control of blood pressure and other modifiable risks. Some guidelines consider a CAC ≥ 300 - 400 a threshold for further imaging, even in asymptomatic patients, due to the high likelihood of significant disease. In symptomatic patients, a very high CAC score increases the pre-test probability of obstructive CAD, though heavy calcification can reduce the accuracy of CT angiography due to blooming artifact.

The Multi-Ethnic Study of Atherosclerosis (MESA) and other cohorts showed that adding the CAC score to risk factor models significantly improves the prediction of coronary events [2]. In intermediate-risk individuals, the CAC score

has been found to be one of the strongest predictors of 10-year coronary outcomes, stronger than many individual risk factors [2]. Each incremental rise in CAC is associated with a progressively higher risk; for example, in one analysis, every doubling of the calcium score corresponded to approximately a 14% increase in the relative risk of a cardiac event [3]. At the extremes, CAC scores in the thousands are linked with annual event rates similar to those seen in patients with established coronary disease [1]. In contrast, patients with CAC = 0 may have annual cardiovascular event rates as low as 0.1% - 0.3% [1] [2]. CAC, therefore, acts as a calibrated measure of atherosclerotic burden and short-term risk, offering clearer stratification than risk equations alone.

The most common use of the CAC score is guiding statin therapy in primary prevention. A CAC \geq 100 provides strong justification for starting statins, as evidence shows that these patients benefit from LDL-lowering therapy that slows atherosclerosis and prevents events [3]. Even a low score (>0) supports consideration of statins if risk factors are present, since any calcification indicates underlying plaque. By contrast, a CAC of 0 can support deferring statins in borderline-risk patients, avoiding unnecessary therapy when no atherosclerosis is detected [3]. This approach is now endorsed in prevention guidelines worldwide [3]. CAC may also guide therapy intensity. For example, a very high score can prompt the use of high-intensity statins and stricter control of blood pressure and other associated risks. Some clinicians use CAC to inform aspirin use in primary prevention or to determine whether further evaluation is necessary, although practices vary. Communicating CAC findings to patients is also valuable. Showing evidence of calcified plaque often reinforces the reality of the disease and motivates adherence to lifestyle measures and medications.

Table 1. Comparison of ECG, echocardiogram, CT calcium score, and CT angiogram.

Modality	Mechanism	Indications	Diagnostic Utility
Electrocardiogram (ECG)	Measures the electrical activity of the heart via skin electrodes. Identifies conduction abnormalities, arrhythmias, and ischemic ST-T changes.	Initial evaluation for chest pain or suspected ischemia; basic cardiovascular screening; monitoring for arrhythmias.	Can suggest acute or prior myocardial infarction (e.g., ST changes, Q waves) and ischemia during stress testing. However, resting ECG has low sensitivity for stable CAD; a normal ECG cannot rule out coronary disease.
Echocardiogram	Ultrasound imaging of the heart to visualize chambers, valves, and wall motion. A stress echocardiogram adds exercise or pharmacologic stress to detect inducible wall motion abnormalities.	Assess cardiac structure and function (e.g., ejection fraction, valvular disease, cardiomyopathy). Stress echo is indicated for evaluation of exertional chest pain or ischemia in those who can exercise and have an interpretable ECG.	Excellent for diagnosing structural heart disease (e.g., valve lesions, cardiomyopathy). In CAD, stress echo detects ischemia via new wall motion abnormalities, with moderate sensitivity and high specificity for obstructive CAD. Provides real-time functional assessment of myocardial ischemia.

Continued

CT Calcium Score (CAC)	Non-contrast cardiac CT scan quantifying calcified coronary plaque (Agatston score). Calcium appears as high-density areas on CT; the score sums areas of calcification weighted by density.	Risk stratification in asymptomatic or primary prevention patients at intermediate risk to guide prevention (e.g., statin decision). Occasionally used in low-risk patients with atypical chest pain to exclude significant atherosclerosis. Not indicated for acute chest pain or in known CAD (since calcium is expected).	Prognostic tool: Reflects total coronary atherosclerotic burden. A high CAC implies the presence of coronary plaque and a higher likelihood of future coronary events [2]. A CAC score of 0 carries a very low short-term risk of MI [2]. However, CAC does not reveal the location or severity of specific blockages (no luminal information). It is purely an anatomic risk marker, not a test for ischemia.
CT Coronary Angiogram (CCTA)	A contrast-enhanced CT scan produces detailed 3D images of the coronary artery lumen and wall. Identifies both calcified and soft (non-calcified) plaques and any stenoses.	Evaluation of chest pain in low-to-intermediate risk patients as a non-invasive alternative to invasive angiography [1]. Useful when a functional test is inconclusive or contraindicated. Not usually for widespread screening due to radiation and contrast.	Anatomic diagnostic test: High sensitivity to visualize coronary artery stenosis; effectively rules out CAD if normal [1]. Can detect and characterize plaques (calcified vs soft) and the degree of lumen narrowing. Negative CCTA has a very high negative predictive value for MI risk [1]. Limitations: moderate radiation exposure and need for iodinated contrast; accuracy can be reduced by extensive calcifications or rapid heart rate.

4. Limitations of CT Calcium Scores

Despite its value in risk assessment, CT calcium scoring has important limitations, as illustrated in **Table 2**. First, CAC scoring detects only calcified plaque, so it underestimates total atherosclerotic burden in patients with predominantly non-calcified (“soft”) plaques. Early or unstable coronary lesions (common in younger individuals and in acute coronary syndromes) may be non-calcified and thus yield a CAC score of zero despite the presence of significant plaque. In other words, a CAC score of 0 does not equate to the absence of coronary atherosclerosis or risk. Studies have shown that a small percentage of individuals with zero calcium can still harbor obstructive coronary disease. For example, among symptomatic patients, approximately 1% - 5% may demonstrate significant coronary narrowing on CT angiography despite a CAC of 0. Furthermore, some studies report that up to 15% - 20% of cardiovascular events occur in individuals with a CAC of zero, particularly in younger patients or those with non-calcified plaque [5]. This zero-score “blind spot” illustrates that CAC = 0 is not a free pass rule in the context of strong clinical suspicion or high-risk features. In these scenarios, further assessment (or even empirical treatment) may be necessary—even in the presence of a zero-calcium score.

Table 2. Benefits and limitations of CT coronary calcium scoring.

Benefits of CAC Scoring	Limitations of CAC Scoring
<p>Improved risk stratification: Provides direct evidence of coronary atherosclerosis, enhancing the prediction of heart attacks beyond traditional risk factors [2]. Identifies individuals (especially intermediate risk) who may benefit from more aggressive prevention or, if CAC = 0, those who can safely avoid unnecessary medication [3].</p>	<p>Misses non-calcified plaque: A zero CAC score does not rule out soft plaques or even obstructive disease in rare cases [5]. Patients (particularly at younger ages) can have significant non-calcified atherosclerosis with a CAC of 0.</p>
<p>Power of zero: A CAC score of 0 is a strong negative prognostic marker, associated with very low 5 - 10-year event rates [2]. This can reassure truly low-risk patients and help avoid over-treating them with drugs or invasive tests.</p>	<p>No functional or anatomical detail: CAC scoring reveals nothing about luminal stenosis or ischemia. It cannot localize lesions or assess their severity. Thus, it cannot diagnose angina causes or guide revascularization decisions—further testing is needed if symptoms are present.</p>
<p>Quick, non-invasive, low radiation: The scan is fast (10 minutes), does not require contrast injection, and imparts relatively low radiation (~1 mSv) [6]. It is widely available and cost-effective for the information gained.</p>	<p>Incidental findings and downstream testing: Up to 40% of scans uncover incidental abnormalities (lungs, etc.) [6]. These can lead to anxiety, additional imaging or biopsies, and potential harm if they prompt unnecessary interventions. A high CAC may also lead to a cascade of cardiac tests (stress tests, angiography) that might not yield net benefit in an asymptomatic person [6].</p>
<p>Motivational tool for patients: Visual proof of calcified plaque can improve patient understanding of risk. Patients with a high CAC often become more adherent to statins and lifestyle changes, while a zero CAC can reinforce the importance of maintaining healthy habits to keep risk low.</p>	<p>Not indicated for all patients: CAC scoring is most useful in specific risk groups (e.g., intermediate risk). It has little utility in very low-risk young individuals (where a zero score is expected and does not change management) or in high-risk patients (who need aggressive therapy regardless of CAC). It is contraindicated in pregnancy and generally avoided in patients with known CAD (where it adds no new information).</p>
<p>Guiding therapy personalization: Helps resolve uncertainty in preventive therapy decisions. For example, it can identify candidates for statin or aspirin therapy who otherwise might not be treated, and conversely identify low-risk individuals who can avoid lifelong medications [3]. This tailoring can improve the cost-effectiveness of prevention.</p>	<p>Lack of outcome trials: Management guided by CAC score is supported by observational evidence but lacks randomized trial proof of improved outcomes. There is a risk of over-reliance on CAC in lieu of treating risk factors, e.g., a high-risk patient with diabetes should be treated intensively even if CAC is 0. Inappropriate deference to a CAC = 0 (“zero means zero risk”) or overreaction to a high CAC without context are pitfalls to avoid.</p>

Another key limitation is that the CAC score provides no information on the luminal diameter or ischemic significance of any particular plaque. It is a measure of disease burden, not a direct test for obstructive disease. A patient with a very high calcium score is likely to have atherosclerosis, but the score itself will not reveal whether there is a critical narrowing in a specific artery. As the case report demonstrated, a patient can have extensive calcification (high CAC) yet pass a stress test if no single lesion is severe enough to induce ischemia until later. Conversely, a patient with a relatively low CAC could still, in rare instances, have a focal non-calcified

plaque causing a significant stenosis. Thus, CAC cannot replace functional testing or anatomical imaging when those are indicated to diagnose ischemia or guide revascularization. It is primarily a *risk marker*. Clinicians must be cautious not to over-interpret a high CAC as evidence of immediate danger requiring intervention, nor a zero CAC as proof of health; context and clinical correlation are required.

Technical and practical limitations also exist. Radiation exposure, although low (on the order of 1 mSv per scan, equivalent to approximately 6 months of background radiation) [6], is non-zero; unnecessary or overly frequent scanning should be avoided. Cost and availability can be considered, although CAC scans are relatively inexpensive compared to other cardiac tests and increasingly available. CAC scans often detect incidental findings in the lungs, mediastinum, or other areas, given the field of view. Studies report that up to ~40% of calcium scans reveal incidental extracardiac findings [6], such as pulmonary nodules, masses, or other abnormalities, which can trigger additional workup. While occasionally this leads to important diagnoses, more often it can lead to anxiety and unnecessary tests for findings that ultimately prove benign. This “collateral damage” is a recognized downside of widespread imaging.

Furthermore, CAC scoring lacks prospective outcomes data from randomized trials. Its use is grounded in observational studies showing association with events [2], but it has not been definitively proven that acting on CAC scores (versus traditional risk factors alone) improves hard outcomes. For instance, the strategy of withholding statins if CAC = 0 is based on observational data showing low event rates, rather than trials demonstrating that such a strategy is safe in the long term [6]. Thus, some experts caution that a zero CAC result should not unduly dissuade us from treating a high-risk patient. Another limitation is that calcium score cut-offs are not one-size-fits-all. The significance of a given score depends on age and sex; for example, a score of 100 in a 45-year-old man is far above average (in the very high percentile) and indicates a higher risk, whereas a score of 100 in a 75-year-old man might be around the population average for that age. Clinicians should consider CAC percentiles for age/gender when counseling patients, in addition to absolute scores. However, percentile reference data may vary by population, and current evidence suggests that the absolute score correlates better with outcomes than age-percentile [1].

The progression of CAC over time deserves mention. CAC score tends to increase as a person ages (by ~20 - 30 units or 17% - 24% per year on average [7], faster if risk factors are uncontrolled) [8]. Serial CAC scans have been used in research to track the progression of atherosclerosis, but this is not part of standard care. A notable paradox is that effective therapies like statins can increase the calcified component of plaques (stabilizing them) [9], so a patient on intensive statin therapy might see their CAC score rise despite risk reduction [10]. Thus, changes in CAC over time are hard to interpret on an individual level, and serial scanning is not generally recommended for monitoring therapy efficacy [11]. In summary, CT calcium scoring is a powerful risk assessment tool, but it has inherent blind

spots and should complement, not replace, thorough clinical evaluation and sound judgment. From a practical standpoint, CAC scanning is generally cost-effective in intermediate-risk groups because it avoids unnecessary lifelong statin therapy in low-risk individuals while targeting therapy to those with demonstrable atherosclerosis. However, considerations of radiation (approximately 1 mSv per scan) and potential downstream testing from incidental findings must be weighed. Most guidelines recommend selective use, not widespread population screening, to balance cost, benefit, and safety.

5. Clinical Pitfalls that Should Be Avoided

When using CAC scoring in practice, clinicians should be mindful of several pitfalls to avoid misapplication or misinterpretation of results:

- **Using CAC in lieu of appropriate testing for symptoms:** Relying on a calcium score to rule out coronary disease in a symptomatic patient can be misleading. For example, patients with active chest pain or high pre-test likelihood of CAD should undergo definitive diagnostic evaluation (such as stress imaging or CT angiography) rather than a calcium scan alone. A zero CAC in a patient with anginal symptoms does not exclude coronary artery disease and could create false reassurance [5]. Always choose the test that answers the clinical question—CAC is a risk tool, not a diagnostic test for obstructive CAD.
- **Overriding clinical risk due to a zero score:** A critical pitfall is failing to treat high-risk individuals because of a CAC score of 0. Guidelines emphasize that patients with diabetes, significant smoking history, or strong premature family history should generally be managed aggressively, even if CAC is zero [3]. These patients have an elevated baseline risk, where a zero score, while reassuring, does not eliminate risk. In such cases, life-saving therapies like statins should not be withheld improperly. The calcium score should augment, not replace, clinical judgment in patients with major risk factors.
- **Automatically pursuing invasive interventions for high CAC:** Routinely sending asymptomatic patients with elevated CAC scores for invasive coronary angiography or stenting is not evidence-based and can be harmful. A high CAC score should prompt risk factor optimization first and foremost, not necessarily invasive investigation in the absence of other indications. Unnecessary downstream testing and procedures in low-symptom, high-CAC patients can lead to overtreatment [6]. Avoid the “oculostenotic reflex” (seeing a high number and reflexively looking for stents); instead, interpret the score in the context of the patient’s clinical picture.
- **Neglecting the age and context of the patient:** The significance of a given CAC score varies with patient age and demographics. A moderate score in a 40-year-old warrants far more concern than the same score in an 80-year-old, due to differing baseline risk and plaque expectations. Clinicians should avoid a one-size-fits-all reaction to a number. Always contextualize CAC results with the patient’s overall profile—including age, sex, ethnicity (since calcium prev-

alence can vary), and clinical risk factors—before altering management.

- **Serial scanning as a substitute for therapy:** Repeating CAC scans at short intervals to monitor therapy response is a pitfall. Calcium scores will typically increase over time as a part of plaque maturation, and effective therapies may even accelerate calcification of plaques (stabilizing them). Thus, an increase in CAC on a follow-up scan does not necessarily mean treatment failure; it could be a byproduct of plaque healing. Conversely, focusing on the score's progression might distract from established therapeutic targets (LDL, blood pressure, etc.). Follow-up CAC testing should be infrequent (every 5 years or more, if at all) and only considered if it is likely to change management. Do not chase a “zero CAC” as a treatment goal—it is usually not achievable once any atherosclerosis exists.

By being cognizant of these pitfalls, practitioners can use CAC scoring wisely, maximizing its benefits in risk stratification and patient motivation, while avoiding scenarios where it might mislead or inappropriately influence management. The overarching principle is that CAC results should inform a comprehensive clinical assessment, not dominate it. When applied to the right patient at the right time, with an understanding of what the calcium score can and cannot reveal, CAC scoring is a powerful tool in cardiovascular prevention. But beyond the score itself, it is the integration of that information into holistic patient care that ultimately determines outcomes.

6. Conclusion

The CT coronary artery calcium scoring has become an important element when assessing cardiovascular risks as it provides a patient's atherosclerotic burden. Predictive risk therapy, which goes beyond traditional factors, is refined with CAC, particularly guided therapy, which is often statins. In certain scenarios, it can lead to uncovering high-risk coronary disease, yet functional tests fail to capture it, thus allowing for earlier intervention. Despite these positives, CAC is not an isolated examination. It does not exhibit a plaque's lesion severity, nor does it illustrate the non-calcified plaques. Over-reliance on CAC tests, or their misuse, could lead to misplaced treatment or a failure to diagnose conditions. Other diagnostic tools are also necessary to determine the full clinical context. Used in moderation, it enhances risk prediction and aids in improved patient outcomes. It is most valuable when used to complement clinical judgment, rather than substitute for it. Preventive cardiology benefits most from CAC when viewed as one of many elements within the broader clinical context.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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