Complementary Roles of SPECT and CT

Myocardial perfusion imaging (MPI) with single-photon computed tomography (SPECT) and coronary artery calcium (CAC) measurement with multidetector spiral computed tomography (CT) or electron-beam CT (EBCT) are useful strategies in the noninvasive evaluation of patients with suspected coronary artery disease (CAD).[1] Coronary calcification is a highly sensitive marker of overall atherosclerotic burden but is much less specific for significant stenosis, while SPECT is useful for detecting stenotic lesions but may miss non-flow-limiting lesions.[2] The different information obtained with each of these techniques makes them highly complementary.[1]

PROGNOSTIC VALUE OF CAC SCORING

CAC assessment has been shown to have prognostic value in both symptomatic[3] and asymptomatic[4] patients. However, while SPECT MPI can provide useful information for patients with known CAD as well as for those with suspected CAD, the primary prognostic value of CAC scoring likely lies in the assessment of asymptomatic patients with a low-to-intermediate likelihood of CAD by Framingham score.[4,5]

In CAC scoring, the coronary arteries are imaged, and a score is assigned based on the amount of calcium buildup seen on the coronary walls (Figure 1). CAC score has been shown to be an independent predictor of mortality, with the 5-year survival rate being significantly lower for patients with a CAC score >1000 (95%) compared with those who have a score <10 (99%) ($P < .001$).[6] Risk-adjusted relative risk values increased significantly with increasing CAC scores.[6]

PROGNOSTIC VALUE OF SPECT

SPECT MPI is commonly used and well established for risk stratification.[5] SPECT has been shown to provide incremental prognostic information beyond clinical data, patient history, and exercise treadmill testing alone.[7] SPECT enhances prognostic risk stratification across the complete spectrum of scan results,[8] and such information can be used to help guide clinical management decisions.[7,9]

CORRELATION OF CAC SCORE AND SPECT RESULTS

CAC scores have been shown to correlate with ischemic SPECT results. In one study of 1195 patients without known coronary disease, ischemic results occurred in <2% of those with a CAC score <100, regardless of the presence or absence of clinical symptoms (although a trend toward more ischemia was present in symptomatic patients with a CAC score of 10 to 99).[10] As the CAC score increased beyond 100, the frequency of ischemic SPECT results increased progressively, with 20% of patients with CAC scores >1000 having ischemic scans (Figure 2).[10] A subsequent study found even higher rates of abnormal SPECT results in patients with moderate (CAC score 100 to 400) and severe (CAC score ≥400) atherosclerosis—18% and 45%, respectively.[11]

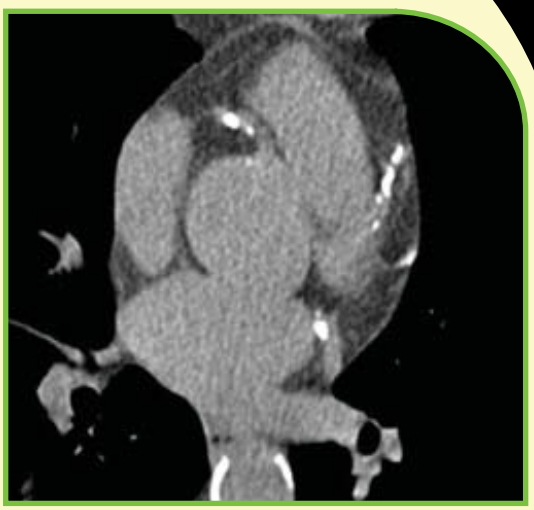
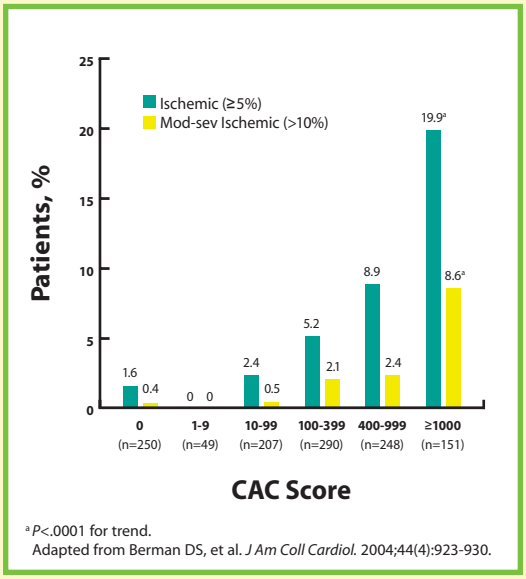


Figure 1. EBCT showing severe calcification (white areas) of all 3 major coronary arteries and the descending aorta, for a total CAC score of 1612.



[a] P<.0001 for trend.
Adapted from Berman DS, et al. *J Am Coll Cardiol.* 2004;44(4):923-930.

Figure 2. Frequency of ischemia and moderate-to-severe ischemia by SPECT MPI by CAC score.

COMPLEMENTARY VALUE OF CAC SCORING AND SPECT RESULTS

Patients with both severe CAC scores and high-risk SPECT results have a higher 10-year mortality risk than those with lower risk by either test.[2] Also, most studies have shown that at least half of patients with a CAC score of ≥400 have no evidence of ischemia.[1] In such cases, it does not appear to be appropriate to use only the SPECT results to determine the need for aggressive medical management,[1] as patients with a CAC score >400 have been shown to have twice the 5-year mortality rate than those with a score <400 (1.0% vs 0.5%, respectively).[2]

A number of strategies that include both CAC scoring and imaging for ischemia have been proposed for CAD assessment in asymptomatic patients.[5,12,13] The WISE[b] study strategy includes CAC scoring as an option in asymptomatic women with an intermediate-to-high (ie, ≥6%) Framingham 10-year risk of cardiac events.[13] The SHAPE[c] guidelines suggest measuring CAC or carotid intima-media thickness and carotid plaque in all asymptomatic men aged 45 to 75 and women aged 55 to 75, except those defined as very low risk.[12] In both strategies, patients with high risk by CAC score would undergo ischemia imaging, with SPECT as an option.

The algorithm in Figure 3 illustrates the strategy proposed by Berman et al[5] that includes utilizing both CAC scoring and nuclear imaging (SPECT or positron-emission tomography [PET]) in CAD diagnosis and risk stratification. For symptomatic patients, Berman et al recommend CT angiography, when available, over CAC scoring prior to nuclear imaging.[5]

[b] WISE = Women's Ischemia Syndrome Evaluation.

[c] SHAPE = Screening for Heart Attack Prevention and Education.

CAC SCORING *AFTER* SPECT MPI

A potential role for CAC assessment after SPECT MPI has also been suggested.[10,14] Patients with normal MPI results frequently have significant atherosclerosis by CAC scoring.[10,14] In one study, 17.5% of patients with normal MPI scans were subsequently shown to have CAC scores >100, allowing intervention with aggressive medical therapy.[14] Future studies may determine which patients with normal MPI results would be candidates for subsequent CAC assessment.[10]

CONCLUSIONS

Both SPECT and CAC scoring with CT are useful for prognosis and risk stratification, but the information they provide is very different. SPECT appears to be an effective tool for assessing short-term risk, thereby helping to guide decisions regarding revascularization.[5] CAC scoring, on the other hand, may be more useful in providing longer-term risk assessment and can help determine the need for aggressive medical prevention measures.[5] The results of these modalities may be used together to enhance CAD assessment, and strategies for their integration continue to evolve.

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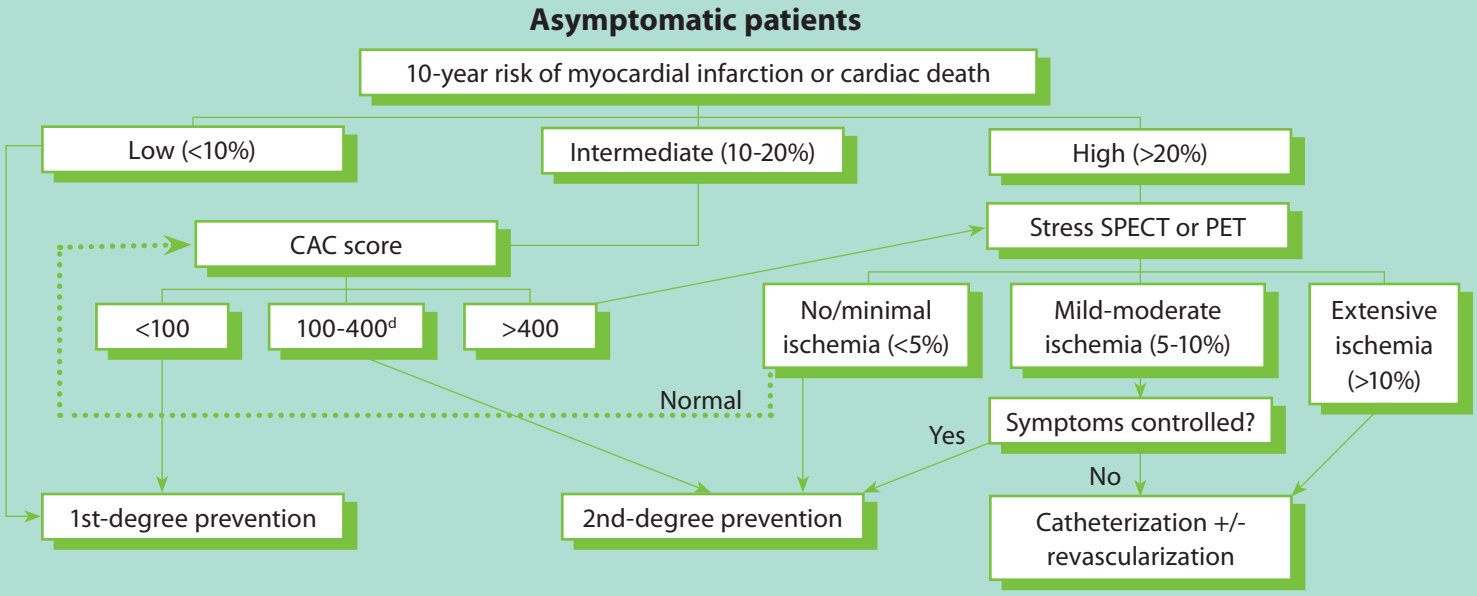

Figure 3. Approach to use of CAC scoring and nuclear imaging in CAD screening in asymptomatic patients.

[d] Consider SPECT or PET in higher-risk subsets.

Adapted from Berman DS, et al. *J Nucl Med*. 2006;47(7):1107-1118.

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