Niels Ramsing Holm, MD
Hans Erik Bøtker, MD, DMSc
Per Ivarsen, MD, PhD
Morten Bøttcher, MD, PhD

*Department of Cardiology
Aarhus University Hospital
Brendstrupgaardsvej 100
DK-8200 Aarhus
Denmark
E-mail: sw@dadlnet.dk

http://dx.doi.org/10.1016/j.jcmg.2015.06.027

Please note: The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

REFERENCES

1. Winther S, Svensson M, Jorgensen HS, et al. Diagnostic performance of coronary CT angiography and myocardial perfusion imaging in kidney transplantation candidates. J Am Coll Cardiol Img 2015;8:553–62.

2. Task Force Members, Montalescot G, Sechtem U, et al. 2013 ESC guidelines on the management of stable coronary artery disease: the task force on the management of stable coronary artery disease of the european society of cardiology. Eur Heart J 2013;34:2949–3003.

3. De Lima JJ, Sabbaga E, Vieira ML, et al. Coronary angiography is the best predictor of events in renal transplant candidates compared with noninvasive testing. Hypertension 2003;42:263–8.

Coronary Artery Calcium Scanning: Past, Present, and Future

Because I am a clinical cardiologist with a major interest in ischemic heart disease, this elegantly written and comprehensive assessment of coronary artery calcium scanning attracted my attention.

I do not think anyone would argue that the presence of calcium in the coronary artery equals coronary atherosclerosis but does not define the degree of stenosis. At the moment, the blooming artifact makes it difficult to assess lumen size even when computed tomography (CT) angiography is done. Likewise, a properly done negative calcium scan does not equate with the absence of any plaques in the coronary circulation, for example, noncalcified plaques, which are also risk factors.

Those interested in this subject and working in this field believe that there is evidence suggesting that asymptomatic individuals with an "intermediate" Framingham Risk Score may be "'reasonable candidates for using coronary artery calcification scanning as a potential means of modifying risk predictions and altering therapy." Unfortunately, no risk score is useful clinically if there is no intervention that might alter the clinical outcome.

It is interesting to reflect on the historical aspects of the identification of coronary artery calcium. Many years ago, coronary artery calcium was reported using simple cine fluoroscopy. Unfortunately, the sensitivity of the fluoroscopic technique is less than the CT technique, and CT can quantitate the amount of calcium, whereas fluoroscopy cannot.

In this paper, Hecht (1) compares early detection of coronary artery calcium with the early detection of breast cancer, colon cancer, lung cancer, and aortic aneurysm. I do not think that this is a fair comparison because these 4 conditions, if detected early, can initiate immediate curative (or at least palliative) therapy. In contrast, early detection of coronary atherosclerosis by calcium scanning in a normotensive, nonsmoking, exercising, nondiabetic patient with normal lipid levels offers no advantage to the worried well because there is nothing to treat. In addition, the lack of insurance coverage for CT scanning can be a problem for several low-income patients.

I would like to propose a theoretical patient and ask what therapy should be recommended. The patient is a 55-year-old asymptomatic man who has mildly elevated blood pressure, moderately elevated cholesterol, moderately elevated low-density lipoprotein, and low high-density lipoprotein. This patient could be considered at intermediate risk by Framingham criteria (9% estimated risk at 10 years) and a finding of calcium in the coronaries would increase this risk (20% risk at 10 years), thus justifying aggressive medical management. I agree and cannot argue that point, but I think most cardiologists (including me) would be fairly aggressive with blood pressure management and lipid management in this particular patient whether or not coronary artery calcium is present.

I have no idea whether there is calcium in my coronary arteries (at age 80, there probably is) and if I did know, I am not sure what I would do about it other than what I am doing without knowing. I guess it would scare me a little, particularly if calcium was present in high concentration. However, I would also be concerned if I had uncontrolled hypertension, diabetes, obesity, renal disease, or severe hypercholesterolemia. I can do nothing about my age or my sex, so why worry about that?

Along with many others, I firmly believe that the presence of coronary artery calcium heightens the level of awareness of potential major adverse cardiac events, and it certainly should stimulate the physician and the patient to control other known clinical problems such as hypertension, smoking, diabetes, obesity, hyperlipidemia, lack of exercise, and so forth.

I am in full agreement with Hecht's proposal that if the presence of calcium in the coronary artery can be used to motivate patients to adhere to a program of risk factor reduction, then patients with known risk

factors should have a coronary artery calcium scan. If the scan does not motivate the patient, then I see no reason to do it. I guess one can do the scan and see what happens to patient adherence.

C. Richard Conti, MD*

*Health Science Center
University of Florida
Room M-438
1600 SW Archer Road
P.O. Box 100277
Gainesville, Florida 32610-0277
E-mail: conticr@medicine.ufl.edu

http://dx.doi.org/10.1016/j.jcmg.2015.06.031

Please note: Dr. Conti has reported that he has no relationships relevant to the contents of this paper to disclose.

REFERENCE

1. Hecht HS. Coronary artery calcium scanning; past, present and future. J Am Coll Cardiol Img 2015;8:579–96.

THE AUTHOR REPLIES:

Dr. Conti's letter is greatly appreciated because it so clearly illustrates the educational gap and misconceptions that have contributed to the slow acceptance of coronary artery calcium (CAC). His major concern is that "no risk score is useful clinically if there is no intervention that might alter the clinical outcome."

Let us examine Dr. Conti's 3 clinical scenarios:

1. "Early detection of coronary atherosclerosis by calcium…because there is nothing to treat." In fact, this patient has atherosclerosis to treat, and, depending on the degree of CAC, will be at higher risk than implied by the absence of risk factors. In 542,008 patients presenting with a first myocardial infarction, 48.5% had ≤1 risk factor (1) and would not have qualified for statin treatment unless CAC had been detected, as is the case in 95% of acute events (1). If there is high risk by CAC, the patient will benefit from statin therapy irrespective of low-density lipoprotein.
2. "The patient is a 55-year-old asymptomatic man who has mildly elevated…but I think most cardiologists (including me) would be fairly aggressive…whether or not coronary artery calcium is present." In reality, the extent of CAC determines whether the risk is increased, decreased, or remains the same. The outcome based net reclassification indexes of the Framingham Risk Score by CAC from 3 major trials (MESA [1], Heinz-Nixdorf [1], and Rotterdam [1]), ranged from 52% to 65.6% in the intermediate-risk group. The clinician's ability to accurately assess risk in this group is worse than a flip of the coin, and CAC avoids the mistakes inherent in extrapolating risk classification from large population groups to the individual. Thus, high-risk CAC will mandate aggressive lipid treatment and 0 CAC may obviate the need for statin therapy because there are no data that statins can lower the risk below the 1.0% 10-year event rate of the 0 CAC group (1). Simply because most cardiologists would be fairly aggressive in this case does not mean that they are correct, assuming that treatment should be data driven.
3. "I have no idea whether there is calcium in my coronary arteries,…and if I did know, I am not sure what I would do about it…" That depends on the CAC results and what you are already doing. If you had extensive CAC, more aggressive lipid lowering than you may have already achieved would be appropriate if you believe lower low-density lipoprotein is better, assuming you are on a statin; if you are not, then you should be. If you were statin intolerant and had a 0 or low score, the statin could be stopped.

Thus, in an era of increasing concern over the benefit versus harm of any therapy, including statins, the use of CAC will routinely alter treatment by tailoring it to the patient's accurately determined risk rather than guessing on the basis of inaccurate risk factor-based paradigms.

Harvey S. Hecht, MD*

*Icahn School of Medicine at Mount Sinai
Saint Luke's Mount Sinai Medical Center
1111 Amsterdam Avenue
New York, New York 10025
E-mail: harvey.hecht@mountsinai.org

http://dx.doi.org/10.1016/j.jcmg.2015.06.032

Please note: Dr. Hecht is a consultant for Philips Medical Systems and HeartFlow.

REFERENCE

1. Hecht HS. Coronary artery calcium scanning: past, present and future. J Am Coll Cardiol Img 2005;8:579–96.