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Electron beam computed tomography with suspected CAD: the preferred initial diagnostic test in clinically stable patients

C.A. Geluk, F. Zijlstra

The presence of coronary atherosclerosis in clinically stable patients with suspected coronary artery disease (CAD) is initially evaluated by clinical, ECG and laboratory characteristics. In Europe and the Netherlands, the exercise tolerance test was traditionally the first choice of additional testing in these patients. In the last decade, a promising new noninvasive imaging modality has been developed, namely electron beam computed tomography (EBCT). EBCT is a quick and noninvasive procedure requiring less than five minutes of scan time and with a low dose of radiation exposure (<1.0 mSv). EBCT detects coronary calcifications and may represent a superior first-line diagnostic tool in the evaluation of suspected CAD. We will explain the rationale of EBCT as a first-line diagnostic tool in the evaluation of suspected CAD. Pathophysiological as well as clinical arguments support the concept that EBCT is superior to exercise tolerance testing as initial diagnostic test.

The first question to be answered is how the current concepts of diagnostic tests are related to the pathophysiological atherosclerotic process. Coronary atherosclerosis results from a consecutive process of endothelial dysfunction, development of fatty streaks (type I-III plaque according to AHA/ACC criteria I-VI) and of atheroma including a lipid core (type IV plaque) and fibrotic and/or calcified tissue (type V plaque). Coronary calcifications are 100% specific for the atherosclerotic process. Although the precise mechanism of calcium deposition is still unclear, it has been shown that matrix glycoproteins play a part in this process. The amount of coronary calcification is correlated with total plaque volume in autopsied hearts. In addition, amounts of coronary calcifications detected by EBCT are associated with the presence of obstructive CAD at coronary angiography. Both type IV and V plaques are prone to rupture (resulting in a type VI plaque) and may result in an acute coronary syndrome. In these consecutive processes (I-VI), narrowing of the coronary luminal diameter occurs only in the later stages, since the first stages of the atherosclerotic process are associated with positive remodelling. Noninvasive stress tests evaluate whether myocardial ischaemia occurs with increasing myocardial oxygen demands in the presence of significant obstructive CAD. Therefore, stress tests are only able to detect CAD in the advanced stages of the atherosclerotic process. Detection of coronary calcifications by EBCT, however, yields a much greater diagnostic sensitivity when compared with noninvasive stress testing due to detection of CAD in the earlier stages of the atherosclerotic process. Therefore, the pathophysiology behind the atherosclerotic processes favours the use of EBCT as a first-line diagnostic tool in the evaluation of suspected CAD.

The second question is whether detection of CAD in the early stages of the atherosclerotic process predicts the occurrence of major adverse cardiac events and should have consequences for the clinical management in patients with suspected CAD. It has been shown that traditional evaluation of low-risk patients with suspected CAD without ST-segment changes or elevated cardiac enzymes should be improved, since 5% of these patients will experience a major adverse cardiac event within two weeks after presentation. This observation confirms the difficulty in risk stratification, based on clinical variables, into patients with high, intermediate and low risk of a future coronary event. Although more obstructive CAD is associated with even worse clinical outcome, the majority of acute
coronary syndromes result from plaque ruptures or erosions at locations with nonsignificant CAD. Therefore, a diagnostic tool used to improve cardiac risk stratification should not only prove the presence of obstructive CAD, but should evaluate the total atherosclerotic burden and thus the vulnerability for plaque rupture. The detection of coronary calcifications by EBCT fulfils this prerequisite. EBCT has a high positive predictive value for the occurrence of major adverse cardiac events in large patient groups. 6,7 Therefore, in any patient with suspected CAD and presence of coronary calcifications, coronary risk factors should be evaluated and minimised and, if indicated, use of aspirin and statins should be instituted. 8 In the clinical management of patients with suspected CAD, EBCT therefore shows a major advantage when compared with exercise tolerance testing.

EBCT, as currently used without radiographic contrast agents, does not provide information on obstructive CAD. However, increasing amounts of calcifications are associated with obstructive CAD. 4 Evidence of a large atherosclerotic burden and the anatomical localisation of calcifications (left main stem!) may identify potential candidates for a revascularisation procedure. In patients presenting with typical anginal complaints, one may question whether performance of EBCT prior to coronary angiography is useful, since in these patients the pretest probability is very high and may not be changed by the amount of coronary calcifications (Bayes' theorem). However, the majority of patients presenting to the emergency department with suspected CAD do not have typical angina, but show atypical or nonanginal complaints. In these patients EBCT may be helpful in the risk stratification prior to coronary angiography. It has been reported that in Europe up to 50% or more of coronary angiographies are not followed by a revascularisation procedure. This observation raises questions about the use of coronary angiography in patients with suspected CAD, since this procedure is expensive and associated with a low but significant risk of complications. Coronary angiography is often performed to evaluate the presence of CAD in patients with a low likelihood of CAD, but with an abnormal or nonconclusive exercise tolerance test result. Exercise tolerance testing frequently shows false-positive results, especially in women and those with a low pretest likelihood of CAD. If EBCT is used as a first-line diagnostic tool prior to CAG, the performance of coronary angiography in patients with normal coronary arteries will for a large part be prevented.

Furthermore, EBCT is very useful in patients with suspected CAD because of its high negative predictive value. 5,7 A considerable number of patients with suspected but unproven CAD continue to have symptoms and require resources resulting in increased medical costs. The absence of coronary calcifications virtually excludes the presence of clinically significant CAD and the occurrence of major adverse cardiac events in the future. Preliminary data on the EBCT findings in patients with suspected CAD, but a nondiagnostic ECG and negative cardiac markers in the University Medical Centre of Groningen, show that over 50% of patients had a calcium score <10. During three months of follow-up no coronary events or revascularisation procedures occurred. These results are promising, and need to be confirmed by longer follow-up, in larger cohorts of patients.

We have brought up both pathophysiological and clinical arguments that favour the use of EBCT as a first-line diagnostic tool in the evaluation of suspected CAD. The remaining question is how EBCT should be implemented in a clinical decision protocol for patients with suspected CAD. We suggest first evaluating the presence of CAD by EBCT in all clinically stable patients with suspected CAD. In the absence of coronary calcifications, patients can be discharged safely. When coronary calcifications are present, evaluation of obstructive CAD should take place to decide whether a patient should undergo coronary angiography and a revascularisation procedure if indicated.

References