LETTERS TO THE EDITOR

Does coronary CT angiography improve risk stratification over coronary artery calcium scoring in symptomatic patients with a low pre-test probability of coronary artery disease and a CAC of zero?

I read with great interest the article entitled ‘Does coronary CT angiography improve risk stratification over coronary calcium scoring in symptomatic patients with suspected coronary artery disease? Results from the prospective multicenter international CONFIRM registry’ from Al-Mallah et al.1 published in European Heart Journal Cardiovascular Imaging. Perhaps, an unanswered question that could be addressed by the same group would be: Does coronary computed tomography angiography (CCTA) improve risk stratification over coronary artery calcium (CAC) scoring in symptomatic patients with a low pre-test probability of coronary artery disease and a CAC of zero?

The authors acknowledged the overall low event rate in symptomatic individuals with absent CAC and stated ‘it remains unclear when one should proceed to CCTA in all symptomatic patients with a zero calcium score’. Indeed, in a report from the same registry, there was no difference in mortality among patients with a CAC score of zero irrespective of obstructive coronary artery disease (CAD).2 In addition, in the same study, the majority of patients with a CAC score of zero and obstructive disease on CCTA had single-vessel disease, a cohort in which coronary revascularization has not been shown to improve survival.3 It is well known that the pre-test probability of CAD is an essential factor when assessing the prognostic performance of CCTA vs. CAC scoring in symptomatic patients. Therefore, combining patients with a low pre-test risk with those with an intermediate or even high pre-test probability of obstructive CAD might not be the best strategy. In this context, the recent 2013 ESC guidelines on the management of stable coronary artery disease3 considered CCTA useful when performed in patients within the lower range of intermediate pre-test probability of CAD (not in the low risk category). In the study by Al-Mallah et al., 49% of patients had a low pre-test probability of CAD and 56% of all patients had a CAC score of zero. How many of those classified as low risk had no CAC and what was the utility of adding CCTA to study these subjects remain unclear. Avoiding or reducing the need of CCTA in at least part of this large group of individuals could reduce costs and radiation exposure, if CAC scoring could be demonstrated as a guarantor of good prognosis. Perhaps, data from the study by Al-Mallah et al.1 could help on answering these questions.

Conflict of interest: None declared.

References


Felipe S. Torres1,2,*
1. Postgraduate Studies Program in Cardiology, School of Medicine, Universidade Federal do Rio grande do Sul, Ramiro Barcelos, 2400, Porto Alegre, RS 90.035-003, Brazil.
2. Department of Radiology, Hospital de Clínicas de Porto Alegre, Porto Alegre, RS, Brazil.
*Corresponding author. Tel: +55 51 33165604; Fax: +55 51 33165614; Email: felpesonarestorres@gmail.com

References


We thank Dr Torres for his interest in our paper.1 We agree that patients with zero coronary calcium score (CCS) have a low prevalence of coronary artery disease (CAD) as well as a very low event rate, as has been shown before by our group and others.2,3 We further analysed our data to answer the question whether coronary computed tomography angiography (CCTA) adds an incremental prognostic value over CCS in patients with a zero CCS and a low pre-test probability. Among the 4860 symptomatic patients with a zero CCS in our study, 3209 (66%) patients had a low pre-test likelihood. In this group, 1% of the patients had obstructive CAD and 12% had non-obstructive CAD, while the rest had normal coronaries. At the end of the follow-up duration, 16 patients experienced death/myocardial infarction. The event rate was 0.4% among patients with normal coronaries, 0.8% among patients with non-obstructive CAD, and 3.4% among patients with obstructive CAD (trend P = 0.052). Given the small number of events in this group, an appropriate multivariable analysis is not possible. These findings confirm our prior conclusion that it remains unclear when one should proceed to CCTA in symptomatic patients with a zero calcium score even among those patients with a low pre-test likelihood. A larger sample size or longer follow-up of these patients is needed to answer this clinically important question. Until this evidence is available, the decision to proceed to CCTA among patients with a zero CCS and a low pre-test likelihood should be individualized.

Conflict of interest: None declared.

References


Published online 11 November 2013

Published online 28 November 2013
Letters to the Editor

The intriguing issue of genetic predisposition and the importance of identification of pre-clinical markers of endothelial damage in radiotherapy-induced cardiotoxicity

We have read with great interest the article ‘Expert consensus for multi-modality imaging evaluation of cardiovascular complications of radiotherapy in adults: a report from the European Association of Cardiovascular Imaging and the American Society of Echocardiography’ by Lancellotti et al.

Late cardiovascular complications after chest radiotherapy (RT), even modern RT techniques, are a remarkably increasing problem. Care of cancer survivors is becoming an emergent and topical issue especially after chest irradiation and more so for left breast cancer patients. In this subset of patients, RT should be addressed as a risk factor for coronary artery disease (CAD) and since vascular endothelium seems to be the first target of radiation, it should be our duty to detect early marker of endothelial damage long before clinical coronary events. In our institution, we are enrolling early left breast cancer patients in a protocol study to estimate coronary flow reserve (CFR) by 99mTc-sestamibi SPECT Myocardial Perfusion Imaging before and after RT. Regional CFR is defined as the ratio between dipyridamole and baseline myocardial blood flow.  In preliminary data, we have found ST segment and T wave of ventricular repolarization abnormalities registered soon after RT, coupled with myocardial perfusion defects (mostly in the apical region of the left ventricle) and with reduction of estimated values of CFR even in patients with no other risk factors for CAD besides chest RT. Our patients were all treated according to the QUANTEC constraints. We do not know yet the predicting role of CFR reduction for clinical coronary events, but while following up very closely our patients, we are aggressively treating their risk factors for CAD.

We are also intrigued by the genetic issue of RT-induced cardiotoxicity, and we are also trying to identify the genetic marker of increased risk. We have read the editorial by Kelsey et al. and the paper by Hilbers et al. about the association between genetic variants in Transforming Growth Factor-β-1 and Plasminogen activator inhibitor-1 and an increased risk for cardiovascular diseases after RT for breast cancer. The authors say that, for the great majority of individuals, the normal tissue toxicity is influenced by the cumulative effect of multiple genetic polymorphisms. If these assumption are proved to be true, then we will be able to predict which patient is more exposed to toxicity and we can improve our ‘tailored therapies’ maximizing the therapeutic ratio of cancer therapies.

We would like to ask two questions:

(1) What is your opinion on genetic determinants of RT-induced toxicity? The search for polymorphisms should be encouraged in Oncology Departments to modify therapeutic strategies. (For example, left mastectomy instead of breast-conserving surgery plus adjuvant RT if the risk of RT-induced cardiotoxicity is genetically increased.)

(2) Do you think it is worthy to search for a suitable early marker of endothelial damage? And do you think CFR reduction could be such a preclinical marker? Would you suggest an ECG recording soon after RT to screen high-risk patients?

Conflict of interest: None declared.

References


Giuseppina Gallucci1,2* Gianni Storto2
1Cardiology Unit, IRCCS-CROB Centro di Riferimento Oncologico della Basilicata, via Padre Pio, 1, Rionero in Vulture, Potenza 85028, Italy
2Radiotherapy Unit, IRCCS-CROB Centro di Riferimento Oncologico della Basilicata, via Padre Pio, 1, Rionero in Vulture, Potenza 85028, Italy*Corresponding author. Tel: +39 3336337080; Email: pin.gallucci@iscali.it, giuseppina.gallucci@crob.it

doi:10.1093/ehjci/jet248
Published online 24 November 2013

The intriguing issue of genetic predisposition and the importance of identification of pre-clinical markers of endothelial damage in radiotherapy-induced cardiotoxicity: reply

We thank Dr Gallucci for her letter about the joint EACVI/AESE expert consensus for multi-modality imaging evaluation of cardiovascular complications of radiotherapy in adults. As underlined, there is compelling evidence that chest radiotherapy can increase the risk of heart disease. Although modern radiotherapy techniques are likely to reduce the prevalence and severity of radiation-induced heart disease (RHD), the incidence of RHD is expected to...