CT perfusion angiography; beware of artifacts!

E. E. van der Wall · J. D. Schuijf · J. J. Bax · J. W. Jukema · M. J. Schalij

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Over the past years, myocardial perfusion imaging has been established as the reference standard for prognosis and clinical decision making of patients with coronary artery disease (CAD). Myocardial perfusion has predominantly been assessed by single photon emission tomography (SPECT) [1–7] and, more recently, by positron emission tomography (PET) and magnetic resonance imaging (MRI) [8–16]. Amongst the advanced cardiac imaging modalities, multi detector computed tomography (CT) angiography has emerged as a reliable non-invasive method for the assessment of coronary anatomy, CAD, and cardiac function [17–22]. Multiple studies involving over several thousands of patients have established that CT angiography is highly accurate for delineation of the presence and severity of coronary atherosclerosis [23–30]. CT angiography may also reveal the total plaque burden, i.e. both calcified and non-calcified components, for individual patients with coronary atherosclerosis [31–40].

The advent of prospectively gated acquisition techniques for 64-slice CT angiography has allowed a significant reduction in dose exposure. Consequently, a combined approach of CT coronary angiography and myocardial perfusion imaging with CT angiography might potentially become feasible at a total radiation dose of less than 10 mSv, particularly for the assessment of patients with established CAD, who are likely to have diffuse calcification [40–47].

Since myocardial perfusion by CT angiography is based on myocardial signal density, it is crucial to determine the normal values of myocardial signal density and to identify potential mechanisms of misinterpretation of perfusion defects. In routine CT angiography acquisitions, there might be a considerable signal density drop at the posterobasal wall resembling perfusion defects possibly being attributed to beam hardening artifacts.

In the current issue of the International Journal of Cardiovascular Imaging Rodríguez-Granillo et al. [48] investigated normal myocardial signal density levels during CT angiography and evaluated the impact of artifacts due to beam hardening. A group of 36 consecutive asymptomatic patients with a low probability of CAD were referred for CT angiography because of inconclusive or discordant functional tests. Perfusion defects were defined as a myocardial segment having a signal density two standard deviations below the average myocardial signal density for the 16 left ventricular American Heart Association (AHA) segments. Signal density was evaluated in 576 American Heart Association (AHA) segments
and 36 posterobasal segments. The mean myocardial signal density at the posterobasal segment was 53.5 ± 35.1 Hounsfield Units, whereas the mean myocardial signal density at the basal, mid and apical myocardium was 97.4 ± 17.3 Hounsfield Units, with significant differences between posterobasal and all AHA segments. Posterobasal perfusion defects were identified in 26 (72%) patients. The only variable associated to the presence of posterobasal perfusion defect was heart rate, whereas body mass index, blood signal density of the left and right ventricles, contrast-to-noise ratio, and the extent of atherosclerosis were not related to the presence of perfusion defects. The main findings of the study were that (1) beam hardening artifacts are a common finding at CT angiography of asymptomatic patients affecting predominantly the posterobasal wall, (2) perfusion defects at the short axis plane including the mitral valve and the left ventricular outflow tract are not related to technical issues whereas heart rate may be associated with this finding, (3) perfusion defects can be also identified at the inferior and anteroapical segments but this occurs less often.

Occurrence of attenuation artifacts during radionuclide SPECT perfusion imaging has been considered an important limitation of the technique [49–51]. Apical thinning due to the overlying diaphragm and the occurrence of anteroseptal defects as a result of breast attenuation are very common causes for unwanted perfusion deficits, leading to image misinterpretation and potentially a wrong diagnosis. The present study [48] indicates that perfusion artifacts also occur at CT perfusion angiography and could therefore be a reason for misinterpretation. These perfusion defects can largely be ascribed to beam hardening artifacts most likely from the spine for posterobasal segments and from the sternum for anteropical segments.

In radionuclide myocardial perfusion SPECT imaging, successful attenuation correction programs have been developed in order to discriminate between true and false perfusion defects [52–55]. Similarly, correction algorithms are currently being designed for CT angiography with the same purpose. Recently, So et al. [56] designed phantoms to simulate the beam hardening artifacts encountered in cardiac CT perfusion studies of humans and animals. These phantoms were used to investigate whether beam hardening artifacts could be reduced with this approach and to determine the optimal settings of the correction algorithm for patient and animal studies, which depend upon the anatomy of the scanned subject. The correction algorithm was also applied to correct beam hardening in a clinical study to further demonstrate the effectiveness of this technique.

To summarize, the study by Rodríguez-Granillo et al. [48] convincingly shows that in an asymptomatic population with no history of CAD, who undergo CT perfusion angiography, artifacts in the posterobasal wall are a common finding in more than two-thirds of patients. This phenomenon of pseudo-perfusion defects may considerably affect proper image interpretation and should be taken into account in the judgment of CT perfusion images. In the near future, correction algorithms for CT perfusion angiography will assist in identifying the true nature of the defects in order to establish the correct diagnosis.

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