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Multidetector computed tomography predictors of late ventricular remodeling and function after acute myocardial infarction

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ABSTRACT

Background: Despite advent of rapid arterial revascularization as 1st line treatment for acute myocardial infarction (AMI), incomplete restoral of flow at the microvascular level remains a problem and is associated with adverse prognosis, including pathological ventricular remodeling. We aimed to study the association between multidetector row computed tomography (MDCT) perfusion defects and ventricular remodeling post-AMI.

Methods: In a prospective study, 20 patients with ST-elevation AMI, treated by primary angioplasty, underwent arterial and late phase MDCT as well as radionuclide scans to study presence, size and severity of myocardial perfusion defects. Contrast echocardiography was performed at baseline and at 4 months follow-up to evaluate changes in myocardial function and remodeling.

Results: Early defects (ED), late defects (LD) and late enhancement (LE) were detected in 15, 7 and 16 patients, respectively and radionuclide defects in 15 patients. The ED area ($r = 0.74$), and LD area ($r = 0.72$), and to a lesser extent LE area ($r = 0.62$) correlated moderately well with SPECT summed rest score. By univariate analysis, follow-up end-systolic volume index and ejection fraction were both significantly related to ED and LD size and severity, but not to LE size or severity. By multivariate analysis, end-systolic volume index was best predicted by LD area ($p < 0.05$) and ejection fraction by LD enhancement ratio.

Conclusions: LD size and severity on MDCT are most closely associated with pathological ventricular remodeling after AMI and may thus play a role in early identification and treatment of this condition.

1. Introduction

Rapid and complete revascularization of occluded coronary arteries, responsible for acute ST-elevation myocardial infarction (STEMI), has revolutionized the treatment and outcome of this condition over the past decade. Nevertheless, a sizable proportion of patients do not restore myocardial perfusion, despite epicardial coronary artery reperfusion, a phenomenon known as no-reflow and usually related to microvascular obstruction [1,2]. This condition is associated with a worse prognosis and has been attributed to several potential causes, such as distal embolization, ischemia–reperfusion injury with associated myocyte swelling, infiltration and activation of neutrophils and platelets and deposition of fibrin. Patients with no-reflow exhibit a higher prevalence of early post-infarction complications, adverse ventricular remodeling, heart failure and mortality [1]. Early detection and treatment of no-reflow may thus have important implications.

Various modalities are available to diagnose and quantify this condition, including ECG ST resolution, contrast echocardiography, magnetic resonance imaging (MRI), and radionuclide studies [2]. Recently multidetector row computed tomography (MDCT) has been shown to be capable of visualizing myocardial microvascular blood volume [3–9]. Scanning at different time periods after injection permits estimation of the severity of the microvascular dysfunction in patients with myocardial infarction; myocardial hypoenhancement in the first-pass scan immediately following contrast medium injection, known as early defect (ED) is a sign of slow or reduced microvascular blood flow occurring in resting ischemia or infarction, similar to a resting myocardial perfusion

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scan, whereas persistence of a defect in a late scan performed 5–10 min later, known as late defect (LD) depicts regions of no-reflow, usually associated with late enhancement (LE), a sign of necrosis. All of these defects have been shown to be related to late myocardial function and functional recovery [10–13].

We performed a detailed analysis of a group of revascularized STEMI patients with the aim of studying the relationship between both size and severity of different myocardial defects on MDCT and follow-up myocardial function and volumes evaluated by contrast echocardiography, as measures of LV remodeling. We also compared findings with radionuclide imaging, as a reference standard for infarct size and viability.

2. Materials and methods

2.1. Study population and design

Twenty consecutive patients with a first acute STEMI undergoing primary angioplasty and who agreed to participate were prospectively included in the study. The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a priori approval by the institution's human research committee and all patients signed informed consent. The study was registered in the NIH Clinical Trials Registry (ID NCT00285064). Exclusion criteria were renal failure (creatinine >1.3 mg/dl), known allergy to iodine, arrhythmia, and unstable clinical condition. Patients underwent contrast enhanced ECG-gated MDCT 3–4 days post-infarction, including a "1st pass" early scan immediately after injection and a late scan 6 min post injection. Trans-thoracic echocardiography (TTE) was performed within 24 h of STEMI and full contrast-enhanced TTE performed 3–5 days after MI and again 4 months later to evaluate changes in regional and global myocardial function and volumes. Radionuclide imaging was also performed 3–5 days post MI. Scans were performed immediately following injection of thallium-201 to evaluate perfusion and again at 24 h to assess viability.

2.2. MDCT

ECG-gated MDCT angiography was performed on a Brilliance16-slice scanner ($n = 4$) and a Brilliance 64-slice scanner ($n = 16$, Philips Healthcare, Cleveland, Ohio), following IV injection of 80 ml contrast at a rate of 5 ml/s, followed by 40 ml saline at the same rate. The scan was triggered by automatic bolus tracking at 130HU. Scanning parameters for the early scan were: detector collimation 16 mm × 0.75 mm or 64 mm × 0.625 mm, pitch 0.2, gantry rotation time 420 ms, tube voltage 120 kV, and tube–current–time product 600–800 mAs. For the late scan, we used either 16 mm × 1.5 mm or 32 mm × 1.25 mm detector collimation, pitch 0.2, gantry rotation time 420 ms, tube voltage 80 kV, and tube–current–time product 400–600 mAs with tube current modulation. Effective radiation dose was approximately12 mSv for the early scan and 1.5 mSv for the late scan.

2.3. Invasive coronary angiography

This was performed on admission to identify the infarct-related artery and successful angioplasty was performed in all patients resulting in TIMI 3 flow.

2.4. Echocardiography

TTE was performed on either Acuson Sequoia (Siemens Medical Systems, Mountain View, California), or Vivid-7 (GE Healthcare, Milwaukee, WI). A preliminary routine examination was performed during the 1st 24 h following admission, concentrating on 2D imaging of left ventricular (LV) segmental function, including parasternal long-axis and short-axis views and apical 4-chamber, 2-chamber and 3-chamber views. This examination was performed mainly as a precaution not to miss any significant LV dysfunction, which may have improved between the 1st and 2nd examinations. During days 3–5, within 24 h of the MDCT scan, a 2nd TTE examination was performed concentrating on the same views as before, with and without intravenous injection of echo contrast (Definity, Lantheus Medical Imaging, N. Billerica, Massachusetts) to better define endocardial borders. The use of contrast echocardiography has been shown to enable accurate calculation of LV volumes in comparison to MRI, which is regarded as the accepted gold standard [14]. A 3rd examination was performed 4 months later, in an identical way to the 2nd examination in order to evaluate changes in segmental and global left ventricular function and volumes. All TTE's were stored digitally for later processing. For each examination, each of 16 segments was scored by a single observer, giving a score of 1 (normal), 2 (mildly hypokinetic), 2.5 (severely hypokinetic) or 3 (akinetic or dyskinetic, these were combined since the number of dyskinetic segments was very small).

Functional recovery of a segment was defined as any segment having an abnormal score at baseline and a normal score at 4 months. The wall motion score (WMS) defined as the sum of scores of all segments was calculated as a measure of overall segmental myocardial dysfunction. A 2nd observer calculated the end-diastolic and end-systolic volumes and ejection fraction (EF), using the biplanar area-length method, for the 2nd and 3rd examinations only (Fig. 1). The volumes were expressed as indices by normalizing for body surface area.

2.5. Radionuclide imaging (Fig. 2a)

Nuclear studies were performed on a single photon emission computed tomography (SPECT)/low dose CT device (Millennium VG or Infinia & Hawkeye; GE Healthcare) using a rest only single-isotope protocol using thallium-201 in all patients. Imaging was performed at 15 min and again at 24 h after the intravenous injection to assess possible redistribution. Standardized 17 myocardial segments were scored from 0 (normal) to 4 (absent perfusion) (SPECT segment score), although the apical segment was not used in analysis in order to correspond with the analysis of the MDCT data. A summed score of all segments (SPECT summed rest score) was calculated as a representative of infarct size [15].

2.6. Post-processing and analysis of MDCT data (Fig. 2b and c)

For each of 3 cardiac phases – 0%, 75% and the end-systolic phase, 6 equally spaced short-axis reformations of 5 mm thickness were obtained from about 1 cm from the apex until the most basal slice having a complete rim of myocardium. In addition, horizontal and vertical long-axis reformations were obtained. These slices were loaded into the Extended Brilliance Workspace (Philips Healthcare, Ohio) for analysis. A single user, blinded to all other data related to the patient, measured the area and thickness of any defect present in each of 16 AHA segments (excluding segment 17) by planimetry, using a technique previously described [10]. Early defects were defined as confluent regions of myocardium with a CT value at least 20HU below that of the normal myocardium, having a subendocardial or transmural distribution in the early scan. Late defects were similarly defined when identified on the late scan. Late enhancement was defined as confluent regions of myocardium having at least 20HU higher CT value than normal myocardium in the late scan. The phase with the best image quality (usually 75%) was used for evaluation. For potential enhancement defects to be read as a true enhancement defects, as opposed to imaging artifacts, they