Novel MRI and CT Approaches for the Characterization of Myocardial Infarct

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Abstract Accurate myocardial viability assessment is essential to optimize revascularization and/or medical treatment in patients with myocardial infarct. To date, cardiac magnetic resonance imaging (MRI) is the widely accepted clinical standard for visualization, characterization and quantification of myocardial viability. Multidetector computed tomography (CT) is reported by numerous authors as an emerging imaging modality for myocardial viability assessment. This review article summarizes cardiac MRI and CT modalities for myocardial viability assessment.

Keywords Myocardial viability · Cardiac MRI · Cardiac CT

Introduction

Prolonged myocardial ischemic injury due to significant coronary artery disease leads to irreversible tissue damage

and myocardial cell loss that manifest as myocardial infarction (MI). Infarcted cells lose their integrity and initiate cascades of responses to ischemic injury. In the acute phase of ischemia, vital ion channels are blocked. Failure of the sodium potassium ion channel pump in the cellular membrane induces cellular edema, cellular swelling. On a macroscopic scale, after the first few days of acute coronary artery occlusion, the infarct volume can double in size due to predominantly edematous changes within infarcted heart [1]. Experimental results indicate that edema impedes ventricular contraction and relaxation, induces additional necrosis and may initiate arrhythmias [2, 3].

In the subacute phase of MI, a process of resorption starts to restore edematous changes, and subsequently necrotic tissue forms that is replaced by condensed scar tissue in the chronic phase of infarct. During the next 4–6 weeks after MI, infarct volume can diminish to about 25 % of its size in the acute phase as necrotic myocytes are replaced by scar tissue. In the months after acute MI, wall thinning of the infarct area and adjacent myocardium can be observed [4].

Even after restoring coronary flow in the acute phase, an area of residual myocardial perfusion abnormality may remain, called microvascular obstruction (MO). The presence of MO results in a more extensive final infarct size, left ventricular remodeling, and lack of functional recovery [5], and is related to a worse prognosis [6–8]. MO serves as an important prognostic factor for future functional recovery and morbidity. Presence of no-reflow zone predicts left ventricle (LV) remodeling, late wall thinning, lack of functional recovery and poor cardiovascular outcomes [7].

Cardiac magnetic resonance imaging (MRI) plays an important and essential role to monitor the tissue changes associated with infarction related cardiac injury, including edematous, necrotic infarcted myocardium, microvascular

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obstruction and infarcted chronic scar tissue. Delayed enhancement MRI is unsurpassed in its ability to differentiate viable from nonviable myocardial tissue, whether in the acute, subacute, or chronic phase of MI [9]. Delayed enhancement cardiac viability magnetic resonance imaging technique was established in the 1980s, when initial reports confirmed the delayed gadolinium contrast enhancement on T1-weighted magnetic resonance images in 1984 [10, 11]. Cardiac MR imaging, either by late gadolinium enhancement (LGE) or low-dose dobutamine challenge, is currently the “gold standard” noninvasive technique for evaluating myocardial viability and infarct morphology [12–14]. The transmural extent of delayed enhancement was found to be strongly related to the probability of improved contractility after revascularization: segments showing delayed enhancement of more than 75 % of myocardial thickness were unlikely to benefit from revascularization [15]. Just like necrotic myocardium, scarred myocardium does not regain functionality after revascularization. The size of the final infarct is related to the extent of LV remodeling and LV dysfunction [16].

Iodinated contrast enhancement within myocardial infarction was first recognized on computed tomography (CT) in the late 1970s [17, 18]. Among others, Sato et al. published a cardiac CT viability paper with 102 patients. Delayed iodine contrast enhancement (DE) was investigated by CT. Myocardial DE size was a significant, independent predictor of for cardiac events. Myocardial contrast DE size on multi detector CT (MDCT) immediately after primary percutaneous intervention may provide promising information for predicting clinical outcome in patients with acute MI [19].

To date, MDCT for identification and quantification of infarcted myocardium uses early arterial phase acquisition and/or delayed MDCT acquisition (DE-MDCT) with single or dual energy, utilizing similar contrast agent myocardial tissue kinetics between iodinated and gadolinium-based contrast media. DE-MDCT acquisition, similarly to delayed enhanced cardiac MRI (DE-CMR), delineates acute, subacute and chronic MI. Investigators recently provided evidence to use DE-MDCT shortly after cardiac catheterization, since both X-ray based imaging modalities use iodinated contrast agent. Without administering additional contrast agent, patients can be scanned by MDCT immediately after catheterization for reperfusion, which allows detection of hyperenhancing areas without administering additional contrast medium [20].

Cardiac MRI for Myocardial Viability—Present Clinical Application

MI often induces LV systolic dysfunction. LV function predicts worst outcome after acute coronary syndrome in

terms of mortality and morbidity. Long standing LV dysfunction is associated with development of chronic heart failure and poor survival. The recently published Surgical Treatment for Ischemic Heart Failure (STICH) trial reported that coronary artery bypass graft (CABG) resulted in 19 % lower risk of cardiovascular death compared with medical therapy in a large patient population ($n = 1212$) with ischemic heart failure and ejection fraction of 35 % or less [21]. Of these, 601 patients underwent viability testing and were studied in the recently reported STICH Viability sub-study [22]. In this study, it was reported that the presence of myocardial viability did not appear to affect outcome. However, there are a number of significant limitations with this study. Firstly, the role of myocardial viability was not a predefined outcome, and viability testing was performed solely at the clinicians’ discretion in a nonrandomized manner. Secondly, the decision to perform CABG was not based on the presence of viable myocardium. Finally, during STICH, single-photon emission computed tomography and/or dobutamine stress echocardiography was performed for myocardial viability assessment. These techniques have lower sensitivity and specificity than Cardiac MRI. Due to these significant limitations, the results of the study should be taken with caution, and further trials are needed to address this important clinical question.

LV dysfunction in association with myocardial infarction is induced by an irreversible process including scar formation, necrosis or by reversible process due to stunning or hibernation or a combination of both. In an early report, Baker et al. [23] showed that patients with reversible LV dysfunction benefit from revascularization, yielding symptomatic improvement, improved contractile function and increased survival.

Myocardial viability assessment aims to identify patient who can have significant benefit from revascularization. Allman et al. used myocardial viability assessment to show the survival benefit. Annual mortality rates for patients with viable myocardium and who underwent revascularization show significant decrease in mortality compared to the patient group with medical treatment (three vs. 16 %, respectively) [24].

MRI Techniques

T2-weighted Imaging Study

The presence of edema, assessed by T₂ weighted MRI, has been utilized to differentiate acute MI from chronic MI [25]. Non-contrast enhanced black blood double or triple inversion recovery sequences are applied to acquire T2-weighted images with an echo time that is usually between 70 and 100 ms [26].

T2-weighted images frequently have low signal-to-noise ratio and are less specific for detection of myocardial viability compared to late gadolinium enhancement by contrast-enhanced MRI [27]. Increased signal intensity on T2-weighted images can indeed show edema in association with a myocardial infarct. Increased signal intensity area was accurately correlated with region at risk, which accumulated at the infarcted region and surrounding region of injury as edematous myocardium [28].

Animal experimental studies showed that the automated threshold method is able to delineate edematous myocardium, which is usually defined as at least two standard deviations above the signal intensity of healthy myocardium. T2-weighted signal intensity enhancement alone is not a precise indicator of myocardial viability. It is important to note that reports confirmed high T2-weighted signal intensity values with flow of 25 % in cardiac microcirculation [29].

T2-weighted imaging, especially so-called T2* imaging, can also be used to detect hemorrhage. Intramyocardial hemorrhage is associated with more severe infarct-related injury [30•].

Perfusion Imaging—Role of Microvascular Obstruction

Fast single shot images acquisition using recently introduced echo planar image (EPI) or hybrid EPI sequences can generate good quality perfusion images. Rapid first pass administration of Gd-DTPA contrast agent at rest and under pharmaceutical stress using Adenosine or Regadenosine to delineate fixed and stress-induced reversible ischemia. In acute myocardial infarction, prolonged reduced signal intensities can be appreciated in the center of infarction, showing the presence of previously emphasized microvascular obstruction or no reflow zone. Multiple factors have been suggested to play a role in the manifestation of no-reflow phenomena, including microvascular spasm, endothelial dysfunction, inflammation, edema, embolization of thrombus and plaque [31].

Cardiac MR assessment of MO is based on first-pass perfusion and late gadolinium enhancement (LGE). Early MO is defined as a prolonged (~60 s) perfusion deficit in first-pass images. Late MO appears as a hypo-intense infarct core on LGE images acquired 10 min after contrast injection. In a recent study, Dennis et al. [32] compared the angiographic parameters of MO with first-pass perfusion cardiac MRI for the assessment of early MO and LGE as markers for late MO in 40 patients following primary percutaneous intervention. Late MO correlated better than the perfusion MRI parameters of early MO with left ventricular ejection fraction (LVEF) at 90 days. Multivariate analysis of the data showed that of all the angiographic and MRI variables, LGE-derived late MO was the strongest

predictor of LVEF at 90 days ($p = 0.004$) following ST segment elevation myocardial infarction (STEMI). Natale et al. [33] further investigated the role of first-pass and delayed enhancement for the assessment of segmental functional recovery in 46 patients treated with either primary percutaneous intervention ($n = 40$) or thrombolysis ($n = 6$) after acute myocardial infarction (AMI). These patients underwent cardiac MR imaging within the first week to assess edema, MO, function and viability, and then again after 4–6 months to assess functional recovery and scar. After the first MR examination, post-contrast images were divided into three patterns. Pattern 1 (normal first pass and late hyper-enhancement < 50 % thickness) identified viable myocardium, whereas pattern 2 (late hyper-enhancement > 50 % thickness, with or without first-pass perfusion defect) and pattern 3 (perfusion defect at first pass and late hyper-enhancement) recognized nonviable myocardium, with 93 % sensitivity, 75 % specificity, 92 % positive predictive value and 78 % negative predictive value for identifying viable tissue. Further, they divided pattern 2 into two sub-patterns, 2A and 2B, based on the absence or presence of MO in > 50 % trans-mural infarcts. Through this approach, they identified the nonviable segments with a 1.39 relative risk of failed recovery. They concluded that not all infarcts with > 50 % transmural were nonviable, and MO detected with first pass assessment could help to stratify these cases better. Early MO, assessed by first-pass cardiac MR, has also been found to be an independent long-term prognostic indicator after acute MI [34].

The recently published CE-MARC study established the superior diagnostic accuracy of cardiac MR over Single-photon emission computed tomography (SPECT) and further cost-effectiveness analysis also proved its superiority as a diagnostic tool for ischemic heart disease [35].

CINE Imaging

Steady State Free Precession sequence-based cine imaging study is routinely used for myocardial global and segmental function analysis. Areas of hypokinesia, akinesia, or even dyskinesia are reported as a consequence of ischemic myocardial injury or MI. Acute MI shows normal or thickened myocardial areas depending on the edema content of the infarcted segment. LV remodeling process can produce thin myocardium with condensed scars that occasionally express dyskinesia with aneurysm formation. Romero et al. reported a cutoff value of more than 5.5 or 6 mm of the myocardium that recovers function after revascularization [36]. Cine image analysis plays an important role of integrated viability cardiac MRI study analysis, since it refines viability assessment and helps to accurately measure end diastolic wall thickness.

Fig. 1 Cardiac MRI study of acute myocardial infarction. Patient is a 60-year-old gentleman with chest pain. Cine imaging studies in end diastole (ED) (**a**) and end systole (ES) (**b**) show the presence of normal wall motion. T2-weighted imaging study in dedicated short axis view (**c**) shows the presence of a bright signal in the edematous infero-lateral myocardial segment. The corresponding short axis viability image confirms the presence of viable infarcted myocardium, with less than 50 % transmural extent of hyperenhancement in the same segment. It is noteworthy that the size of infarction in the LGE image is significantly smaller than the area of signal intensity enhancement, due to edema in T2-weighted image. T2-weighted (**e**) and LGE (**d**) two-chamber long axis views delineate edematous changes with associated acute infarction, respectively

◀ **Fig. 2** Cardiac MRI study of subacute myocardial infarction. Patient is a 47-year-old male with chest pain. Cine imaging study in ED (a) and ES (b) show extensive akinesis without the presence of end systolic thickening in septal, apical and lateral segments. Corresponding viability LGE images in four chambers delineate the presence of infarcted myocardium with full thickness transmurality. Microvascular obstruction is noted as non enhanced areas within the center of the infarct, depicted as a low signal intensity region surrounded by bright infarct tissue

the clinical standard for myocardial viability assessment [37].

Several investigators showed that Gd-based contrast agent has a late accumulation phase in T1-weighted post contrast images after 8–10 min of contrast agent administration. Late gadolinium enhancement (LGE) depicts both acute and chronic MI [38, 39]. Inversion recovery sequence is used to acquire T1-weighted LGE images that have better signal and contrast-to-noise ratio than fast perfusion imaging. The crucial point is to identify the tau0 point when the longitudinal, T1-weighted magnetization reaches its null point in healthy myocardium. “Nulling” the myocardium results in normal myocardium appearing dark while infarcted areas due to LGE appear to be bright. Inversion time for nulling of normal myocardium differs per patient, and sometimes has to be optimized during the acquisition of multiple slices.

In an early landmark paper studying myocardial viability with CMR, Kim et al. [15] reported that recovery of myocardial function is closely related to and depends on transmural extent of infarction. A transmural extent of less than 50 % of nonviable infarcted myocardium shows a correlation with recovery in contractile functional reserve (Figs. 1, 2).

The probability of functional recovery is around 65 % in cases with 1–25 % transmural late enhancement. Importantly, if transmural extent is 26–50 %, the probability of functional recovery is decreased to 43 % [15, 40]. Recovery of function after revascularization appears to be related to the ratio of viable-to-scarred myocardium within dysfunctional myocardial segments. Different cutoff values for transmural extent of hyperenhancement have been applied to determine whether or not functional recovery post-revascularization can be expected, ranging from > 0 % to > 75 % [35]. Due to its superior spatial resolution, LGE by MRI is better than SPECT and Positron emission tomography (PET) at identifying regions of subendocardial scar [41]. Nagel et al. [42] incorporated these fundamental findings and established the presently widely accepted algorithm of myocardial viability based on cardiac MRI.

Delayed Enhancement MR Imaging

After the important reclassification of the role of cardiac MRI in early 2000, the modality became

Threshold Based Viability Assessment

The threshold method is widely used to determine the size of enhanced areas. Myocardial infarct delineated by

Fig. 3 Threshold method in animal model. Pig myocardial infarction was created with ligation of the left anterior descending artery. Late contrast enhancement is visible in anterior territory (a) with isolated microvascular obstruction in the center of the infarct. Corresponding binary image is calculated and delineated using the two standard

deviation threshold method. Signal intensities two standard deviations above remote, healthy myocardium delineate infarcted area in anterior segments. *Bright color* shows infarct and *grey color* shows the territory of normal myocardium. The center of infarct is also *grey*, due to the presence of microvascular obstruction

Fig. 4 Cardiac CT study of acute myocardial infarction—first pass arterial phase imaging. Patient is a 40-year-old gentleman with atypical chest pain and borderline Troponin elevation. ECG-gated cardiac CT angiography shows the presence of occluded proximal left anterior descending artery (a) arrows, and corresponding apical anterior infarction (hypoattenuation) is noted in dedicated four chamber view

contrast accumulation in LGE images is defined as a signal intensity increased by at least two standard deviations above that of normal myocardium [43], although there is no universal agreement between investigators, as some groups use six standard deviations, and others use a user specified threshold [44]. Using the threshold method, a binary image is created and each volume element of the image is either infarcted or viable non-infarcted. Unfortunately, this method entirely disregards areas where infarction is not confluent, and solid and viable regions are mixed with nonviable islets. The early-on presence of viable islets, especially at the rim of the infarction, is extensively studied in reperfused postinfarct myocardium [45]. An area of partly viable infarction is usually the region where salvage is most likely to occur upon reperfusion [46].

Similar to the threshold based method, manual planimetry incorporates areas of mixed viability into infarcted territory. Both threshold and planimetry methods tend to overestimate true infarct size [47]. In chronic MI, this overestimation is not as significant as it is with acute or subacute MI, where infarct evolution and remodeling actively influence the presence of partially viable myocardium in the periphery of infarct zone (Fig. 3).

T1-Mapping and R1-Based Percent Infarct Mapping

The statement of “bright is dead”, reflecting an LGE image finding of nonviable, hyperenhanced myocardium is not a precise description of myocardial viability. Edematous myocardium also accumulates contrast agent with still

◀ **Fig. 5** Cardiac CT study of chronic myocardial infarction—arterial phase imaging. The patient is a 60-year-old man with known past medical history of ischemic heart disease and myocardial infarction. Retrospective ECG-gated cardiac CT images are used for myocardial function reconstruction. Two-chamber MPR view in ED (**a**) and ES (**b**) delineate dyskinetic apex due to left ventricle apical aneurysm. Four-chamber MPR reconstruction delineates the presence of thin myocardium with hypoattenuation and calcification as an imaging manifestation of chronic myocardial infarction and left ventricle remodeling

Longitudinal relaxation time (T_1) and consequently longitudinal relaxation rate ($R_1 = 1/T_1$) mapping-based visualization and characterization of MI is to date one of the most promising future tools to accurately identify nonviable myocardium with appropriate exclusion of partly viable and salvageable cell islets. Messroghli et al. proposed the use of a modified look locker sequence to collect serial images for mapping voxel by voxel T_1 values in healthy and infarcted myocardium. The inevitable limitation of the technique is its time-consuming nature and the difficulty in co-registration of corresponding viability images voxel by voxel in the relatively short period of contrast-enhanced phase [48].

Suranyi et al. used a myocardial tissue intrinsic physical parameter, R_1 , which is enhanced in linear proportion with the contrast agent concentration. With the help of using paramagnetic relaxation rate enhancement, ΔR_1 (the difference between inverse T_1 with and without the presence of contrast agent), a continuous scale is assigned in each infarcted voxel. Based on contrast accumulation of the infarcted volume element of interest, a Percent Infarct Map (PIM) can be created [49, 50]. PIM can not only assess the infarct size, or the global parameter called “infarction fraction”, but also the density (i.e. the percent of infarcted cells per myocardial volume) of infarct with a resolution determined by the number of myocardial volumes that MRI is able to provide. In contrast to signal intensity-based image analysis, PIM is not influenced by inhomogeneity of the MRI coil, which artificially imparts varying signal intensities to different parts of the heart depending on their distance from, and relative position to, the coil.

Cardiac CT for Myocardial Viability—Technical Points

Cardiac CT has a rather low signal-to-noise ratio, but better spatial resolution and consequently less prominent partial volume effect compared to cardiac MRI. CT generally is not felt to be capable of accurately characterizing myocardial tissue, at least when performed using a single-energy technique. Low kilovoltage acquisition image protocols have been described to improve the detection of areas of delayed contrast enhancement within the myocardium [51, 52]. However, image quality is tempered by high levels of image noise when imaging obese patients.

viable but stunned and/or hibernating myocytes. The rim of the infarction can also contain partially viable cell islets with late gadolinium enhancement that remain viable with the potential for full functional recovery [15].

Fig. 6 Dual energy cardiac CT study of myocardial infarction—late iodine enhancement imaging patient is a 55-year-old patient with recurrent chest pain and positive family medical history of ischemic heart disease. Short axis MRI image **a** show late gadolinium

enhancement in inferior myocardial segment delineating myocardial infarction. Corresponding short axis CT image **b** in the same patient delineates late iodine enhancement in the same inferior territory in good agreement with clinical standard MRI finding.

Fig. 7 A 68-year-old female patient presenting with shortness of breath. (**a**, **b**) DECT coronary angiography with perfusion imaging depicts an inducible defect in the antero-septal and inferior apical

wall. (**c**, **d**) SPECT examination confirms the presence of an apical inducible ischemia. (**e**) A severe stenosis of the mid left descending coronary artery was demonstrated at catheter angiography

Iodinated contrast material is usually administered as a bolus during coronary CT angiography (CTA) or dedicated myocardial blood pool assessment. Investigators [53] reported greater dynamic range in attenuation values between infarcted tissue and normal myocardium when applying a combination of a bolus and subsequent longer low-flow injection (30 mL at 0.1 mL/s).

The optimal timing of delayed acquisition is still subject to debate. Delayed acquisition should be triggered when

the accumulation of iodine contrast is highest in infarcted myocardium, with the highest absolute attenuation values in infarct and delineating differences of contrast between healthy and infarcted myocardium. A delay time of 5–15 min after contrast medium administration is frequently reported [54].

According to a previous report by Blankstein et al., delayed enhancement CT studies are best viewed as thick (5 or 10 mm) multiplanar reformations in general, with a

Table 1 Summary of stress agents

	Adenosine	Regadenoson	Dobutamine	Dipyridamole
Mechanism	Adenosine receptor agonist	A _{2A} selective adenosine receptor agonist	Stimulation of β_1 receptors	Increase endogenous levels of adenosine blocking cellular uptake
Effects	Vasodilator	Vasodilator	Increases heart rate and myocardial contractility	Indirect vasodilator
Half-Life	< 10 s	2 min	2 min	30 min
Dose	140 $\mu\text{g/kg/min}$ for 3–6 min	0.4 mg at 5 ml/s	High-dose protocol: IV dobutamine infusion at 3 min stages (10, 20, 30, 40 $\mu\text{g/kg/min}$) Low-dose protocol: 5–10 $\mu\text{g/kg/min}$	0.56 mg/Kg IV over 4 min
Contraindications	(1) High-grade AV block; (2) asthma or COPD; (3) sinus bradycardia; (4) systemic hypotension (BP < 90 mmHg); (5) severe carotid stenosis	(1) High-grade AV block; (2) sinus bradycardia. (3) systemic hypotension (BP < 90 mmHg); (4) severe carotid stenosis	(1) Severe hypertension (> 220/120 mmHg); (2) congestive heart failure; (3) unstable angina; (4) Aortic valve stenosis (peak gradient > 50 mmHg); (5) HCM; (6) complex arrhythmias; (7) myocarditis; (8) pericarditis	(1) High-grade AV block; (2) asthma or COPD; (3) sinus bradycardia; (4) systemic hypotension (BP < 90 mmHg); (5) severe carotid stenosis

narrow window width and level (e.g., width, 200 HU; level, 100 HU) or as maximum intensity projections [55].

First Pass Arterial Phase Imaging

Electrocardiogram (ECG) synchronized first pass coronary CT angiography acquisition allows us to not only visualize the coronary artery tree and consequently grade coronary stenosis, but also to characterize areas of decreased myocardial blood flow. Decreased perfusion, shown as hypoattenuated areas on CT images, may be secondary to a critical coronary artery stenosis or occlusion, microvascular obstruction, or myocardial scar. It is difficult to distinguish between these causes, but the diagnostic value of delineating the areas with deteriorated blood flow (hypoattenuation) is important and likely to be of clinical significance [56].

On contrast-enhanced cardiac CT angiography, chronic MI can ordinarily be recognized as a hypoattenuating region (> 50 % HU decrease compared with surrounding myocardium) [57] in a subendocardial or transmural distribution that persists in systole and diastole and is concordant with a coronary territory [58]. Cardiac CT is sensitive to delineate calcification within chronic myocardial infarction as an end-product of infarct remodeling (Figs. 4, 5).

In 2004, animal experiments provided evidence of not only delineation but also quantification of hypoattenuated

areas within acutely infarcted myocardium. The area of low attenuation within porcine heart shows good correlation with myocardial blood flow reduction and absolute MI size [59].

Nicolaou et al. reported clinical correlation in 30 patients. First pass CT angiography was able to detect all but one (ten out of 11) MI compared with DE-CMR. By quantifying blood flow defect representing MI, a significant underestimation (19 %) was found compared to true infarct size determined by DE-CMR.

Differentiation between ischemia and MI is not possible by using first pass CTA acquisition alone [60]. Shapiro et al. [61] investigated the prognostic value of the extent of hypodense areas in patients with MI. It was found that prognostic relevance of hypoperfused areas as determined from arterial-phase CT remains unclear, because decreased myocardial attenuation on arterial-phase CT imaging is not solely indicative, and more importantly, not specific to MI. However, patients with MI often show hypoattenuation after first pass iodinated contrast injection in the infarcted area.

Delayed Enhancement CT Imaging

Cardiac contrast-enhanced MRI uses GdDTPA contrast agent, which has similar tissue kinetics in the myocardium as iodinated contrast agent. Thus iodinated contrast material accumulates in the infarcted cardiac segments, and a

Fig. 8 A 76-year-old male patient with occluded left anterior descending (LAD) and patent left internal mammary artery (LIMA) CABG. (a) Dynamic perfusion stress CT examination demonstrates an extensive perfusion deficit in the antero-septal mid-apical wall with reduced blood flow (b), blood volume (c), and volume transfer constant (d)

similar late enhancement phenomenon appears to occur, as visualized by delayed cardiac CT. DE-MDCT acquisition after iodinated contrast agent administration is capable of delineating MI as an area of hyperattenuation; i.e., myocardial region with high Hounsfield unit values. Late scan acquisition is usually considered within 5–15 min after the administration of contrast agent.

In acute MI, myocyte necrosis results in interstitial edema and membrane rupture, which allows iodinated contrast agents and gadolinium chelates to diffuse into the intracellular space [62].

Dual Energy CT Technique for Myocardial Viability

Tissues in the human body show different absorption characteristics when penetrated with different X-ray spectra, which are typically generated by different kV settings of the X-ray tube. With the help of using different energies, tissue differentiation by X-ray-based imaging modalities has been reported to be possible [63]. It has also been recognized that iodine-based contrast media have unique X-ray absorption characteristics at different kV levels [64]. Animal studies proved that identifying and quantifying MI with dual energy DE-MDCT is feasible [65•].

Using dual energy acquisition for quantification of MI size in human infarcted myocardium helps to combine

information of low kV (80 or 100 kV) in fine visualization of contrast accumulation differences within infarcted myocardium, and high kV (140 kV) in visualization of sharp endo and epicardial borders.

Kang et al. reported close, linear correlation ($r = 0.9$) between DE-CMR and DE-MDCT for determining percent infarct per slice (PIS) in human infarcted hearts ($n = 40$). Dual energy DE-MDCT may represent an important alternative for MI quantification, and correlation statistics yield good agreement in quantification of infarct size between delayed enhancement DE-MDCT and DE-CMR [66] (Fig. 6).

With the help of dual energy CT acquisition under stress and at rest, reversible myocardial ischemia can be identified. Reversible ischemia is detected when the myocardium only expresses ischemic features i.e. hypoperfusion (low iodine uptake) under stress conditions. A viable but ischemic myocardium shows reversible ischemia (Fig. 7). Several pharmacological stressors can be used to generate sufficient cardiac stress and help to delineate stress-induced ischemia (Table 1).

Novel cardiac CT imaging application is the dynamic, real time myocardial perfusion. The feasibility of this CT-based technique was published recently and proposes an accurate delineation of reversible but viable myocardium [67] (Fig. 8).

Conclusion

The assessment of myocardial viability is essential in patients with previous myocardial infarction, and provides both prognostic information as well as guiding revascularization. Cardiac MRI is considered to be the modality of choice, and novel techniques allow for more accurate differentiation of viable from nonviable myocardium. Multi-detector CT, using single and dual energy, is a rapidly involving modality for viability assessment.

Compliance with Ethics Guidelines

Conflict of Interest U. Joseph Schoepf receives honoraria from Siemens, Bayer, GE Medrad, and Bracco. Sukumaran Binukrishnan, Vishal Sharma, Abishek Kumar, Carlo Nicola De Cecco, and Balazs Ruzsics declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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