Evaluation of poor left ventricle: multislice computer tomography in the assessment of myocardial viability

Kötü ventrikülün değerlendirilmesi: Miyokardiyal canlılığın araştırılmasında
çok kesitli bilgisayarlı tomografi

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ABSTRACT

The evaluation of left ventricular function and viability is critical in patients with suspected or documented ischemic heart disease. Recently, cardiac multislice computed tomography (MSCT) has emerged as a powerful modality for cardiac imaging. In addition to coronary artery assessment, contrast enhanced MSCT can provide reliable information about myocardial function, perfusion and viability. This review will focus on the pathophysiological mechanisms and clinical applications of contrast enhanced myocardial function and viability MSCT imaging.
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Key words: Left ventricle, multislice computed tomography, viability

ÖZET

Sol ventrikül fonksiyonlarının değerlendirilmesi şüpheli veya kanıtlanmış kalp hastalığı olan hastaların takibinde kritik öneme sahiptir. Kardiyak çok kesitli bilgisayarlı tomografi (ÇKBT) yeni ve güçlü bir tetkik olarak yakın zamanda kardiyak görüntüleme alanında kullanıma girmiştir. Kardiyak çok kesitli bilgisayarlı tomografi ile koroner arterlerin değerlendirilmesinin yanında, sol ventrikül fonksiyonu, perfüzyonu ve canlılık değerlendirmesi de güvenilir bir şekilde yapılabilmektedir. Bu makalede kontrastlı ÇKBT ile miyokardiyal fonksiyon ve canlılık değerlendirmesinin patofizyolojik mekanizması ve klinik uygulamaları tartışılacaktır.
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Anahtar kelimeler: Sol ventrikül, çok kesitli bilgisayarlı tomografi, canlılık

Introduction

A precise assessment of cardiac function is critical in patients with suspected or documented heart disease. The evaluation of left ventricular (LV) function provides valuable diagnostic and prognostic information. Prognosis after myocardial infarction is closely related to the extent of myocardial necrosis and the degree of LV contractile dysfunction (1). Therefore, distinction between nonviable and dysfunctional but viable myocardium is a major concern in the settings of acute and chronic myocardial ischemia. Viable dysfunctional myocardium can be broadly defined as any region that directionally improves contractile function after coronary revascularization (2, 3). In patients with acute myocardial infarction, revascularization of viable myocardium can improve ventricular dysfunction and long-term survival (4-6). Similarly, in the presence of chronic myocardial injury, the detection of myocardial scar extension enables to identify patients with hibernating myocardium who may achieve functional systolic recovery with revascularization. In both settings however, revascularization of the nonviable myocardium increases exposure to unnecessary risk of invasive procedures and late mortality. Therefore, there has been an increasing emphasis to accurately define viable myocardium over the last decade.

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Currently, the available imaging techniques for determining myocardial viability are radionuclide techniques, low-dose dobutamine stress echocardiography, and cardiac magnetic resonance imaging (MRI). Except for cardiac MRI, none of these techniques allows the differentiation between transmural and non-transmural infarction. Cardiac MRI not only allows for detailed visualization of viable and nonviable myocardium, but also enables the assessment of transmurality of myocardial infarction, thus accepted as the gold standard for myocardial viability imaging (7-10).

In recent years, the temporal resolution of computed tomography has improved and contrast-enhanced multi-slice spiral computed tomography (MSCT) has become a useful tool for cardiac imaging. The aim of this article is to introduce the pathophysiological basics of MSCT viability imaging and to review the current status of technique in different settings of ischemic myocardium.

Metabolic and functional consequences of myocardial ischemia

Irreversible injury and myocyte death

The temporal evolution and extent of irreversible tissue injury after coronary occlusion is variable and dependent on transmural location, residual coronary flow, and the hemodynamic determinants of oxygen consumption. Irreversible myocardial injury begins after 20 minutes of coronary occlusion in the subepicardium and progresses as a wave front over time, from the subendocardial layers to subepicardial layers (11). In experimental infarction, the entire subendocardium is irreversibly injured within 1 hour of occlusion and the transmural progression of infarction is largely completed within 4 to 6 hours after coronary occlusion. Although revascularization of the coronary arteries is necessary before transmural infarction develops for myocardial salvage, restoration of blood flow in the coronary arteries does not always result in successful reperfusion at the microvascular level (12).

Within 6 weeks of myocardial infarction, the necrotic myocardium, is replaced by scar tissue. Several factors such as the magnitude of residual coronary flow through collaterals or through a subtotal coronary occlusion are the most important determinants of the actual time course of irreversible injury and myocardial remodeling. Hibernating myocardium can be broadly defined as viable but dysfunctional myocardium with chronically impaired regional blood flow. Contractile function in hibernating myocardium is diminished as a result of reduced myocyte metabolism due to prolonged hypoperfusion and it can be salvaged to some extent with revascularization (13, 14).

MSCT imaging of myocardial viability

Up to now, the key clinical applications of cardiac computed tomography have been largely limited to coronary angiography and coronary calcium detection (15-17). The improvement in temporal resolution, multi-planar and three-dimensional reconstruction, and the ability of combining other protocols with MSCT angiography while still using a single dose of contrast medium widened its application beyond coronary artery imaging. Recently, several groups showed the reliability of MSCT for the evaluation of the cardiac veins, heart valves, ventricular volumes and ejection fraction, and finally myocardial perfusion and viability (18-21).

Pathophysiological mechanisms of early and late phase contrast enhancement on MSCT

As computed tomography (CT) contrast agents have similar extracellular distribution and kinetics to those of magnetic resonance (MR) contrast agents (22), theoretically, the mechanism of myocardial hyperenhancement and hypoenhancement in injured myocardium after iodinated contrast administration is similar to that proposed for first-pass and delayed gadolinium-enhanced MRI (23).

Contrast medium is thought to reach the microvascular bed in the early phase after an intravenous administration. Therefore, myocardial enhancement during this phase reflects the volume of the vascular bed and is directly related to local myocardial perfusion and vascular integrity (24). As myocardial infarction is associated with a lack of perfusion, the hypoenhanced myocardium during arterial phase is related to epicardial coronary artery stenosis or occlusion, obstruction at microvascular level, or chronic myocardial scar (Fig. 1).

After the contrast medium reached the microvascular bed, it gradually flows into the extracellular space, stays for a certain time and then is washed out. Therefore, myocardial enhancement in the late phase mainly reflects the interstitial characteristics of myocardium (24).

Figure 1. Two cases with varying degrees of LAD stenosis The white arrows indicate LAD lesions (A and C). Arterial phase MSCT imaging presents myocardial hypoenhancement at the LAD territory (arrowheads) (B and D).

LAD - left anterior descending artery, MSCT - multislice computed tomography

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The increased myocardial enhancement of infarcted myocardium on delayed enhancement MSCT imaging is a consequence of loss of cellular membrane integrity and subsequent enlargement of the interstitial space, and the slow wash-in and wash-out of the contrast media in the injured myocardium (25). In normal functioning myocytes, sarcolemmal membranes serve to exclude iodine from the intracellular space. After myocyte necrosis, however, membrane dysfunction ensues, and iodine molecules are able to penetrate the cell. Because the majority of the total myocardial volume is intracellular, large increases in the volume of distribution are achieved, which results in marked hyperenhancement relative to the normal myocytes.

Persistent hypoenhanced areas on delayed phase MSCT imaging is thought to correspond to regions with microvascular obstruction. The red blood cells and necrotic debris blocks intramyocardial capillaries and these obstructed capillaries do not allow contrast media to flow into the damaged bed, which results in a region of low signal intensity compared with normal myocardium (26). The persistent hypoenhancement in non-perfused infarcted myocardium is explained by the lack of collateral flow to that region (27). However, in the reperfused myocardium, even in the presence of a certain extent of microvascular obstruction, contrast material is able to penetrate the necrotic myocytes that reside in that myocardial territory over time. Then the reperfused infarction will present as hyperenhancement with a hypoenhanced 'no-reflow' core as described in the 'wave front' phenomenon (28).

Clinical applications of arterial phase imaging

The detection of acute and chronic myocardial infarction with computed tomography was first tested in the late 1970s and early 1980s in experimental animal models and clinical settings. The results of these studies were promising as they proposed that it is possible to detect myocardial infarction with electron-beam computed tomography (EBCT) or single-slice CT (29-32).

With the recent advents in CT technology, there has been a number of studies proving the feasibility of arterial phase MSCT imaging for the detection of myocardial infarction. In a study by Nikolaou et al. (33), the presence of myocardial infarction was detected by MSCT against coronary angiography. When myocardial early hypoenhancement and regional wall thinning was considered as myocardial infarction, MSCT detected infarcted myocardium with high accuracy (90%), sensitivity (85%) and specificity (91%) (33). The same group reported similarly high sensitivity and specificity for the detection of myocardial infarction with MSCT when validated against MR imaging. Mahnken et al. (34) found a sensitivity of 83% and specificity of 91% for the detection of myocardial infarction with arterial phase MSCT imaging. The extent of early hypoenhancement on arterial phase MSCT showed a good agreement with the extent of first-pass perfusion defect on MR imaging (34).

Differentiation of acute from chronic myocardial infarction is also possible with MSCT imaging. Although both are expected to appear as hypoenhanced regions on arterial phase imaging,

myocardium with chronic infarction will present with wall thinning due to scar formation (Fig. 2). Nikolaou et al. (33), in the study where they assessed the presence of myocardial infarction with arterial phase MSCT imaging, also showed that chronic infarcts can be subjectively differentiated from acute infarcts.

However, MSCT infarct assessment based on regional hypoenhancement from arterial phase imaging has important limitations. The size of chronic myocardial scar can be underestimated with arterial phase imaging when compared with delayed contrast enhanced MR imaging. Mahnken et al. (34) and Sanz et al. (35) showed that myocardial infarcts can be accurately detected on arterial phase MSCT imaging, but the infarct size measured on this phase is smaller than with delayed contrast enhanced MRI. Another important drawback of arterial phase MSCT imaging is that the hypoenhancement detected during this phase is not specific to myocardial infarction. Indeed, regions of hypoenhancement may indicate myocardial segments that are under-perfused due to an obstructed coronary artery, no-reflow phenomenon after revascularization of acute occlusion, or diminished capillary density associated with scar formation chronically (36). The inability of distinguishing non-viable tissue from viable hypoperfused myocardium is a major limitation for using arterial phase MSCT imaging in clinical routine.

Clinical applications of delayed enhancement

The first attempts to detect myocardial infarction with a prototype cardiac CT by using late enhancement protocol showed that contrast media accumulates in the regions of acutely infarcted myocardium (37, 38).

Shortly after that, several studies assessed the potential of delayed enhancement imaging with EBCT, however the use of the technique could not be implemented in clinical routine (39, 40).

The introduction of recent MSCT technology however, has changed the previous observations about the value of the late enhancement CT imaging for myocardial infarction detection. In a short period of time, number of experimental and clinical
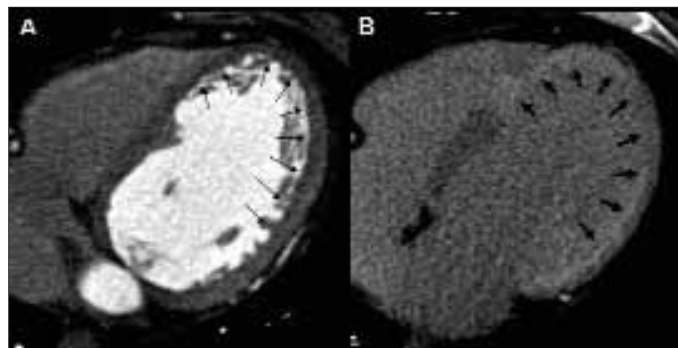

Figure 2. A case of 3-year-old septal and 6-month-old anterolateral myocardial infarction. At the arterial phase, basal and mid septum was normally enhanced with normal wall-thickness, whereas apical septal wall revealed a marked thinning with myocardial hypoenhancement. The entire lateral wall also showed hypoenhancement (tiny arrows) (A). Basal and mid septum presented as normoenhanced myocardium also at the delayed-phase imaging, while hypoenhanced regions showed hyperenhancement (thick arrows) (B).

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studies have been performed and all have validated the reliability of MSCT late enhancement against MRI and pathologic assessment (41-43).

The authors compared the accuracy of infarct sizing on MSCT with triphenyltetrazolium chloride (TTC) and contrast MRI delayed enhancement (43, 44), and found equivalent results with late enhancement MSCT imaging. There was however, a difference in signal to noise ratio (CNR) in favor of MRI when compared with MSCT (41), which can be attributed to the non-linear relationship between the contrast media and the MRI signal that provides a high contrast-to-noise ratio.

The persistence of no-reflow was also successfully demonstrated in experimental and clinical settings by using late enhancement MSCT imaging (41, 42). The no-reflow regions were presented as hypoenhanced area at the core of the enhanced myocardium (Fig. 3).

Recent studies showed the reliability of late enhancement MSCT imaging in predicting the short- and long-term functional recovery of injured myocardium as well. In a clinical study, Habis et al. (46) proved that the magnitude of the hyperenhanced myocardium could predict the functional recovery after acute myocardial infarction, which is tested with low-dose dobutamine stress echocardiography at the 4th week. Koyama et al. (26) and very recently Lessick et al. (47) also showed that there is a close relationship between the extent of myocardial hyperenhancement and functional recovery following myocardial infarction.

Dual-phase MSCT imaging

Combining the data from arterial phase imaging with late enhancement MSCT, provides a more comprehensive assessment of myocardial function and viability following myocardial infarction. Koyama and his co-workers (26) defined three groups of patients regarding to different myocardial enhancement patterns on dual-phase MSCT imaging. The first group showed normal enhancement on arterial phase and presented hyperenhancement on the late phase. The second and third group both presented hypoenhancement on arterial phase, whereas group 2 presented hyperenhancement without persistent hypoenhancement and group 3 presented hyperenhancement with accompanying persistent hypoenhancement region on the late phase MSCT imaging (Fig. 4). They have further classified the late phase hyperenhancement pattern as subendocardial and transmural. The follow-up of the patients showed that Group 3 revealed the largest decrease in myocardial wall thickness, and poorest functional recovery (26).

Technical aspects of MSCT contrast enhancement imaging

As the early phase of MSCT imaging is used for the assessment of coronary arteries as well as myocardial perfusion, a higher spatial and contrast resolution is required for this acquisition. Unlike arterial phase imaging, delayed contrast enhancement acquisition does not require as high
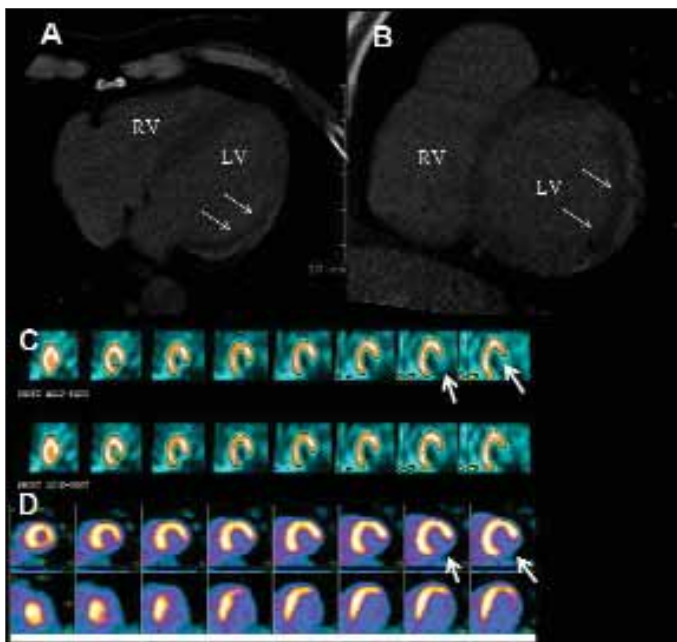

Figure 3. A case of acute left circumflex artery occlusion, which was treated by primary PTCA after 10 hours of angina onset. Delayed phase images revealed a persistent subendocardial hypoenhancement with surrounding hypoenhanced myocardium that corresponds to the infarct related artery territory (white arrows) (A, B). 99mTc-sestamibi single photon emission tomography (SPECT) (C) and ¹⁸F-fluorodeoxyglucose positron emission tomography (PET) (D) showed no viability at this myocardial region (white arrows)

PET - positron emission tomography, PTCA - percutaneous transluminal coronary angioplasty, SPECT - single photon emission tomography
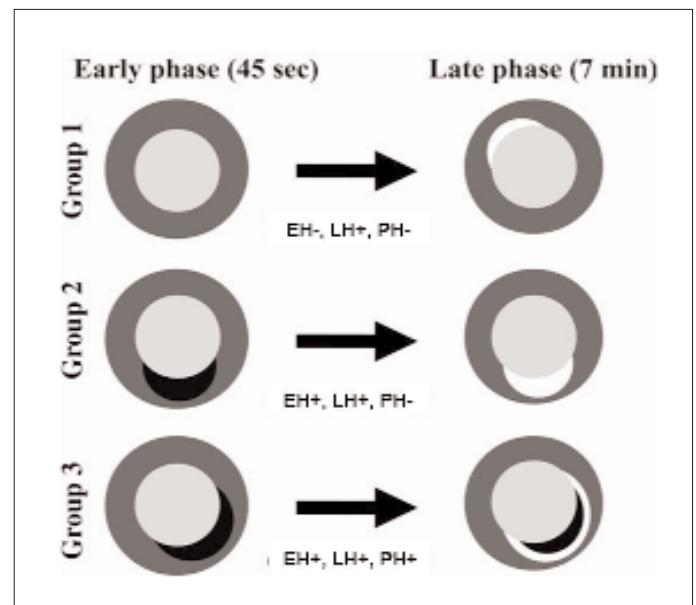

Figure 4. Three different enhancement patterns: Group 1,- no hypoenhancement in the early arterial phase and hyperenhancement in the delayed phase; Group 2 - early hypoenhancement in the arterial phase and hyperenhancement in the delayed phase; Group 3 -early hypoenhancement in the arterial phase and hyperenhancement in the delayed phase with accompanying persistent hypoenhancement.

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spatial resolution and the radiation exposure can be decreased with different adjustments of scanning parameters. Currently there is no established scanning settings for a suitable protocol for delayed enhancement MSCT imaging. Paul et al. (48) performed late enhancement MSCT imaging with different adjustments such as decreasing the tube voltage, using prospective acquisition, and adapting the tube voltage and current according to patient's body morphology.

Another concern about the late enhancement MSCT imaging is the contrast injection protocol and the optimal timing for the late enhancement images. Late enhancement imaging requires a larger dose of contrast medium compared to arterial phase imaging. In most of the studies, a single dose injection have been used in various total amounts. Experimental studies performed by using high dose contrast media showed that increasing the iodine concentration consequently increased the CNR ratio and contrast resolution (44, 49), however these doses can not be applied to clinical use. Although there is no recommended injection dose in the literature, usually in total, more than 50 g iodine should be used in order to provide sufficient quality of images. In most of the clinical studies, 0.6-0.7 mg of iodine per kg was used for delayed enhancement MSCT imaging. Yet, there is no consensus on the injection rationale, however in an experimental study, comparison of single bolus injection versus continuous injection resulted in higher CNR when using the latter approach (50).

A variety of time delays were used for the acquisition of late enhancement images after the contrast injection. In general a time delay ranging from 5 to 15 minutes were suggested to acquire adequate quality of images (42, 46, 51). Habis et al. (46) delayed the acquisition time up to 24±11 minutes in the setting of revascularization after acute myocardial infarction, where they have injected the contrast media directly to coronary arteries during the angiography. Broedfel et al. (52), tested different time delays of late image acquisition and achieved the highest image quality with 5, 10 and 15 minutes of time delay after injection. Several experimental studies also confirmed the rationale that the best quality of late enhancement images were acquired with 2 to 6 minutes of time delay after contrast injection (27, 41, 44).

Conclusion

Multislice computed tomography is not recommended as the first-line modality for solely LV function assessment in the routine clinical practice yet. However, the combination of noninvasive coronary artery imaging and the assessment of LV function, perfusion and viability with a single study enables a comprehensive diagnostic work-up and more conclusive cardiac evaluation in patients with ventricular dysfunction. With the improvements in temporal resolution, new generation MSCT system appears as a powerful alternative imaging modality to MRI particularly in patients with contraindications to MRI imaging.

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