Recent developments in cardiac magnetic resonance (CMR) imaging have led to a tremendous breakthrough in functional imaging and tissue characterisation of the left ventricular (LV) myocardium. Over the past few decades, numerous studies have shown significant improvement in CMR imaging of acute myocardial ischaemia and myocardial infarction (MI). Advances in hardware, acquisition sequences and coil technology have greatly contributed to the improvement of image quality while simplifying cardiac examinations. Cine-CMR allows for accurate time-resolved imaging of global and segmental LV function with high spatial resolution. Dynamic multi-slice CMR of myocardial perfusion is now widely available, allowing for the detection of microvascular obstruction after MI or adding significant diagnostic value over usual clinical and biological markers after non-ST elevation acute coronary syndromes (ACS). Direct high-resolution CMR imaging of MI with the so-called delayed-enhancement technique after gadolinium contrast injection is now well standardised and carries important clinical implications for the diagnosis of myocardial viability.

More recently, CMR has shown great potential for non-invasive imaging of acute myocarditis, showing particular patterns of delayed enhancement when performed 10–15 minutes after contrast injection. Besides, multi-slice CT (MSCT) was initially developed for non-invasive coronary angiography and has great negative predictive value in selected patients for exclusion of coronary artery disease (CAD). Cine–MSCT of the heart allows accurate assessment of global and segmental LV function with high spatial resolution. When performed five minutes after iodine injection, MSCT also has the potential for depicting myocardial damage associated with MI or myocarditis. In other words, recent improvements in MSCT have made it capable of challenging CMR in the characterisation of myocardial tissue. This paper provides a concise overview of the technical aspects, advantages and limits of CMR and MSCT for non-invasive imaging and characterisation of MI and myocarditis.

Imaging of Myocardial Infarction

CMR

Besides the accurate assessment of LV function by cine-CMR, acute infarcts may be recognised as a hyperintense signal on breath-hold electrocardiogram (ECG)-gated T2-weighted fast spin-echo CMR images. This technique, however, does not identify chronic infarcts and may overestimate infarct size by including area at risk. In addition, T2-weighted images often have a low signal:noise ratio. Gadolinium-chelate enhanced imaging provides high-contrast and high-resolution images in which the infarct appears as a hyperenhanced region relative to non-infarcted tissue on inversion–recovery images acquired 10–15 minutes after contrast injection. Indeed, the differences observed after MI in myocardial wash-in/wash-out kinetics of gadolinium enable differentiation of three patterns:

- normal non-ischaemic myocardium is characterised by rapid wash-in, of the contrast agent on first-pass images (<30 seconds) with progressive wash-out over the subsequent minutes;
- conversely, infarcted myocardium is characterised by a slower wash-in but more importantly by a delayed wash-out (>30 minutes) due in large part to an increase of the distribution volume. Therefore, and because of greater gadolinium content, the signal is enhanced (bright) on delayed images compared with non-infarcted tissue; and
- the third pattern corresponds to microvascular obstruction when perfusion is not adequate at the tissue level despite reopening of the culprit coronary artery. In this case, gadolinium wash-in is dramatically delayed and the signal is very low (black) on first-pass images relative to non-ischaemic or necrotic but reperfused myocardium.

It is now well established from contrast-echocardiography and contrast-enhanced CMR that microvascular obstruction identified at the tissue level represents a marker of subsequent poor...
outcome and adverse LV remodelling. Delayed-enhanced CMR has important clinical implications for detection of infarct size, which is the strongest determinant of prognosis in these patients, and for detection of myocardial viability. Infarct imaging by CMR (see Figure 1) is very sensitive and can depict subtle non-transmural infarcts or even infarcllets. The method has been validated against positron emission tomography (PET) for detection of viability and has been recognised as the standard of reference for viability detection by the European Society of Cardiology (ESC) Consensus Panel report.

**MSCT**

Helical MSCT of the heart was initially developed for non-invasive coronary imaging. This technique has great diagnostic value and may be used in selected low to intermediate risk profile patients to rule out significant CAD with high negative predictive value. Besides, MSCT has the capability to accurately assess LV volumes and function through the use of cine-MSCT, which is of great prognostic value in patients with acute MI or ischaemic cardiomyopathy. Recently, experimental and clinical studies have shown that when acquired five minutes after iodine contrast injection, delayed contrast-enhanced images of the LV may accurately depict MI as a hyperintense signal compared with remote tissue. Similar to contrast-enhanced CMR, MI corresponds to a linear single hyperenhanced area that predominates in the subendocardium and matches a pre-defined coronary territory. Similar to CMR, microvascular obstruction may be identified as a hypodense area on images acquired early after contrast injection.

Finally, common features of infarct imaging by CMR and MSCT are characterised by the presence of delayed enhancement of the infarcted tissue, which occurs in a pre-defined coronary territory and predominates in the subendocardium. The delayed enhancement of the myocardium corresponds typically to a single linear area of hypersignal that extends towards the subepicardium as a wavefront. The transmural extent of MI serves as an index of tissue viability, with graded probability of subsequent recovery after revascularisation for a given myocardial segment, which is inversely proportional to the initial transmural extent.

**Imaging of Acute Myocarditis**

Myocarditis corresponds to an acute aggression of the myocardium, resulting in various degrees of myocyte necrosis associated with cellular infiltration, inflammation and oedema. In contrast to MI, myocyte necrosis preferentially develops in the subepicardial layers and tends to diffuse transmurally.

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**Figure 1: Magnetic Resonance Imaging of a Patient with Myocardial Infarction**

(a) The short axis first-pass perfusion view of the mid left ventricle demonstrates a subendocardial perfusion defect of the anterior wall and interventricular septum corresponding to an anteroseptal acute myocardial infarction. (b) Delayed-enhancement sequence in short axis, (c) long axis and (d) and four-chamber planes displays a subendocardial linear hyperenhancement in the same area indicating myocardial viability.

**Figure 2: Acute Viral Myocarditis**

(a) A short axis T2-weighted view at the level of mid left ventricle shows a transmural area of increased signal of the anterior wall suggesting myocardial oedema. (b) There is a homogenous of the myocardium on the short axis first-pass perfusion view at the same level. (c) Pre-contrast cine-MR confirms the relative high signal intensity of this area as well as of the subepicardium of the inferior and lateral wall, (d) as well as delayed-enhancement inversion-recovery prepared T1-weighted gradient echo sequence in short axis, whereas (e) long axis and (f) four-chamber views show diffuse nodular enhancing nodules in particular of the apex.
at the subacute stage. The myocardial areas involved by the pathologic process do not correspond to any pre-defined coronary territory. Myocarditis can be caused by a variety of diseases, but is primarily due to viruses. The clinical features are often limited to minor signs such as fatigue, palpitations in the days following an acute episode of fever and/or angina. In approximately 10% of cases, acute myocarditis can lead to acute heart failure. It may also have a subacute course such as rapidly progressive dilated cardiomyopathy with subsequent heart failure. The disease can evolve to chronic features and persistent dilated cardiomyopathy. In a recent study of 2,231 unexplained primary cardiomyopathies, 111 (9%) were secondary to myocarditis. It also can be revealed by an acute chest pain mimicking ST-elevation or non-ST-elevation ACS. In both cases, troponin I measurements may be increased. The presumed diagnosis of myocarditis is often difficult to confirm. The clinical presentations, ECG, laboratory tests and echocardiography are not specific. Coronary angiography may serve to eliminate an unstable coronary stenosis. Endomyocardial biopsy is the most specific examination and has been considered as the method of reference. In clinical practice it is often skipped because of its invasive property and the risk of false negatives due to the patchy and heterogeneous distribution of myocardial tissue damage. Indeed, its sensitivity is low and has been estimated in to be the range of 50–65%. The sensitivity of $^{67}$Gallium myocardial scintigraphy is relatively poor. Myocardial scintigraphy with $^{111}$Indium-labelled antimyosine monoclonal antibodies, which are fixed specifically to intracellular myosine within the damaged cells, carries higher sensitivity but low specificity. Therefore, the need for reliable diagnostic tools is of great importance.

**CMR**

Several preliminary studies have shown the capability of CMR to image myocardial damage during the course of acute myocarditis. One of the main interests of this technique relies on its sensitivity to rapid changes in tissue composition and its ability to visualise the entire myocardium, which is required for the accurate detection of a patchy and sometime diffuse inflammatory pathologic process. The CMR examination is well standardised and quite similar to that used in particular for the evaluation of ischaemic cardiomyopathy and viability assessment. It includes breath-hold ECG-gated black-blood T2-weighted sequence, steady-state free precession cine-CMR for assessment of LV function, dynamic first-pass perfusion myocardial imaging during the minute following 0.05–0.1 mmol.kg$^{-1}$ gadolinium-chelate injection and delayed-enhanced inversion-recovery T1-weighted imaging 10 minutes after injection. CMR features vary according to the time elapsed from the onset of symptoms to the CMR study. Although still debated, one can schematically distinguish a focal form of acute myocarditis within the first five days that may evolve towards a more diffuse disease. CMR is able to detect on-going inflammation, its extent and severity, and to differentiate myocardial involvement from that of acute or chronic MI.

During the first days of the disease, myocardial oedema is present in about 30% of cases and appears as a hypersignal on T2-weighted images. Oedema involves predominantly the inferolateral wall with or without increased wall thickening. Pericardial effusion is noted in approximately 20% of cases and is generally moderate. Cine-CMR may reveal wall motion abnormalities that are usually diffuse with a global hypokinesia in approximately 30% of cases. In the most severe forms (fulminant myocarditis), LV ejection fraction (LVEF) is severely decreased without LV dilation. Segmental wall motion abnormality may be present in myocardial segments that can be different from those exhibiting myocardial damage on delayed enhancement sequence. In contrast to microvascular obstruction frequently observed after acute MI, there is no perfusion defect on contrast-enhanced first-pass perfusion imaging. Areas of delayed contrast-enhancement are frequent, either nodular predominating in the subepicardium or showing up as thick bands preferentially at mid-wall. Myocyte membrane rupture leading to increased extracellular space, oedema related to the inflammatory phenomenon with capillary compression, increased vascular permeability responsible for an increased distribution volume, along with decreased gadolinium clearance, may explain gadolinium accumulation in regions involved in the pathologic process of acute myocarditis. These lesions occur in the same territory as oedema and do not correspond to a known coronary territory (see Figure 2). These abnormal areas of delayed enhancement are very often localised in the inferolateral wall. More subtle patterns such as micronodular lesions can be observed. These abnormal delayed-enhancement patterns have low sensitivity (around 60%) but a high specificity (97–100%), underscoring the need for using a combination of different sequences to improve diagnostic accuracy. Recent comparison data on the use of endomyocardial biopsy showed positive biopsies in more than 90% of delayed-enhanced areas, against 9% in non-enhanced areas. After 10 days, subacute forms are more difficult to pick by CMR because of the diffusion of the viral process in the myocardium. The oedema may become more diffuse, as well as wall motion abnormality. Delayed enhancement may be difficult to highlight...
because of a more diffuse process. Several longitudinal studies have followed patients up to three months. A favourable outcome was observed when LV contractile function improved and paralleled a significant decrease or involution of damaged delayed-enhanced myocardial tissue. Although still controversial, early hyperenhancement occurring four minutes after gadolinium injection at the acute phase and persisting up to one month after the onset of symptoms could be indicative of poor outcome.

**MSCT**

Recent works including only limited numbers of patients showed that delayed-enhanced ECG-gated cardiac MSCT acquired five minutes after injection can provide similar information to that of CMR, with an excellent correlation in terms of location and patterns of myocardial lesions. Also, limited numbers of clinical observations have been so far reported in the literature indicating that MSCT could accurately depict myocardial damage in acute myocarditis. Although the contrast between damaged myocardium and remote tissue seems less pronounced with MSCT than CMR, this technique offers the advantage of non-invasive coronary angiography during the same examination, which may be of crucial importance for the exclusion of ACS in patients presenting with acute chest pain.

**Myocarditis or Acute Coronary Syndrome?**

It is well known that acute myocarditis can masquerade as acute MI. In the setting of acute chest pain with concomitant ST-segment elevation on at least two contiguous ECG leads, guidelines for management of ST-segment elevation MI should be applied. When patients have a low risk profile for CAD and particularly if they have a recent history of fever or flu, the authors suggest that invasive coronary angiography should be the preferred method when rapidly available in order to avoid potentially inappropriate thrombolytic therapy. CMR should play an important role when coronary angiography rules out significant coronary stenosis in these cases and has the potential to confirm the diagnosis of myocarditis. On the other hand, in the setting of acute chest pain without ST-segment elevation, CMR, if rapidly available, may become the first-line imaging study, especially in those patients with low risk profile and/or recent history of flu.

Myocardial oedema may be depicted by T2-weighted CMR during the acute phase of MI. Although myocardial distribution of oedema is theoretically different, it is often difficult to distinguish between myocarditis and acute MI based on T2-weighted CMR images. First-pass myocardial perfusion imaging may help distinguish the two diseases as there is no early subendocardial defect in myocarditis, whereas it is very common in acute MI despite prompt reopening of the infarct-related-artery (30–60% of cases). As previously described, patterns of delayed hyper-enhancement are very distinct and help discriminate between acute MI and acute myocarditis (see Table 1).

In conclusion, functional CMR has routine clinical applications in the setting of myocardial ischaemia and infarction. Besides accurate assessment of LV function and cardiac anatomy with cine-CMR, the study of myocardial perfusion permits the detection of microvascular obstruction after acute MI, which carries important prognostic implications. Contrast-enhanced CMR has become the clinical reference method for detection of myocardial viability after MI or in chronic ischaemic LV dysfunction. In addition, CMR is also becoming a reference diagnostic tool in suspected myocarditis. The evolution of CMR patterns during the course of myocarditis may be of great interest for the establishment of prognosis especially in patients with initial LV dysfunction, heart failure, or with familial history of cardiomyopathy. MSCT emerges as a novel promising technique for imaging of MI and myocarditis. Because of its great value for non-invasive coronary angiography, it is mandatory to prospectively evaluate different diagnostic strategies using these techniques alone or in combination in patients with suspected acute myocarditis.

**Table 1: Discriminating Acute Myocardial Infarction and Myocarditis Based on Delayed-enhanced CMR and MSCT**

<table>
<thead>
<tr>
<th>Contrast-enhanced CMR or MSCT patterns</th>
<th>Acute MI</th>
<th>Myocarditis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type</td>
<td>One focus</td>
<td>Multiple foci, patchy</td>
</tr>
<tr>
<td>Shape</td>
<td>Linear</td>
<td>Nodular or bands</td>
</tr>
<tr>
<td>Contrast</td>
<td>High</td>
<td>Moderate</td>
</tr>
<tr>
<td>Topography</td>
<td>Coronary territory</td>
<td>Non-coronary territory</td>
</tr>
<tr>
<td>LV distribution</td>
<td>Depends on infarct related artery</td>
<td>Inferolateral wall</td>
</tr>
<tr>
<td>Transmural distribution</td>
<td>Subendocardium</td>
<td>Subepicardium, mid-wall</td>
</tr>
<tr>
<td>Extension</td>
<td>Towards subepicardium</td>
<td>Towards mid-wall and subendocardium</td>
</tr>
<tr>
<td>Evolution</td>
<td>Persistent, shrinkage</td>
<td>Less visible after 10 days</td>
</tr>
<tr>
<td>Accompanying oedema</td>
<td>Hardly visible on T2-W CMR</td>
<td>More pronounced</td>
</tr>
<tr>
<td>Early perfusion defect</td>
<td>Often present</td>
<td>Absent</td>
</tr>
<tr>
<td>Wall motion abnormality</td>
<td>Segmental, concordant</td>
<td>More diffuse</td>
</tr>
</tbody>
</table>

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