Non-invasive imaging in acute chest pain syndromes

Udo Sechtem¹, Stephan Achenbach¹,², Matthias Friedrich¹,³,⁴,⁵,⁶,⁷, Frans Wackers¹,⁸,⁹,¹⁰, and José L. Zamorano¹,¹¹

¹Robert-Bosch-Krankenhaus, Auerbachstr. 110, 70376 Stuttgart; ²Department of Cardiology, University of Giessen, Klinikstrasse 33, 35392 Giessen, Germany; ³CMR Centre at the Montreal Heart Institute, Université de Montréal, QC, Canada; ⁴Cardiovascular Imaging, Université de Montréal, QC, Canada; ⁵Department of Cardiology, Université de Montréal, QC, Canada; ⁶Department of Cardiac Sciences, University of Calgary, 5000 Rue Belanger, Montréal, QC, Canada; ⁷Department of Radiology, University of Calgary, 5000 Rue Belanger, Montréal, QC, Canada; ⁸Le Buc, France, 82120; ⁹Department of Diagnostic Radiology and Medicine, Yale University School of Medicine, New Haven, CT, USA; ¹⁰Section of Cardiovascular Medicine, Yale University School of Medicine, New Haven, CT, USA; and ¹¹Cardiovascular Institute, University Clinic, San Carlos, Plaza de Cristo Rey 28040, Madrid, Spain

This review has the purpose of informing the reader about the current use of imaging techniques in patients presenting with acute chest pain to the emergency department. We will focus on three aspects of managing the patient with acute chest pain:

• Imaging to increase the number of correct diagnoses in the acute situation;
• Imaging to rule out other than coronary causes of chest pain;
• Use of imaging for risk stratification once myocardial infarction has been ruled out in the CPU.

Special emphasis is given to how these management aspects are discussed in current guidelines on the management of patients with acute chest pain or acute coronary syndrome.

Keywords

- Acute chest pain
- CMR
- Echocardiography
- Computed tomography coronary angiography
- Nuclear cardiology
- Pericarditis
- Aortic dissection
- Pulmonary embolism

Imaging in the clinical context

The challenge to clinicians in emergency departments (EDs) is the rapid identification of those patients with chest pain who require admission and urgent management and those with low clinical risk who can be discharged safely directly from the ED. Although it is possible to identify patients with <5% probability of myocardial infarction (MI) on the basis of simple clinical and ECG variables, today’s diagnostic process has to be more precise as misdiagnoses carry a risk of serious adverse events for the patients but also substantial liability for the physician or the hospital. An improved triage process is expected with the increasing use of chest pain units (CPUs), risk scores, accelerated diagnostic protocols, and non-invasive imaging.

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• Imaging to increase the number of correct diagnoses in the acute situation;

Imaging techniques in patients suspected to have chest pain of ischaemic origin

To aid in the diagnosis of ACS, imaging modalities can evaluate either myocardial perfusion/ventricular function or coronary anatomy. Objective evidence of ischaemia or coronary obstruction by cardiac imaging may reduce the time to treatment in patients with suspected ACS, whereas exclusion of ischaemia will speed the time necessary to safely discharge the patient. Imaging strategies to accelerate diagnosis of ACS include rest echocardiography, ...
rest myocardial perfusion imaging (MPI), computed tomography coronary angiography (CTCA), and cardiac magnetic resonance (CMR) imaging. All have very high negative predictive values for excluding ACS in patients with or shortly after an episode of acute chest pain in whom the ECG is non-diagnostic.

In the acute situation imaging is often necessary to exclude other potentially serious causes of chest pain such as aortic dissection or pulmonary emboli. However, as patients sent home after ruling out these diagnoses tend to have recurrent symptoms, it is desirable to provide an explanatory diagnosis. This may improve quality of life, help to develop appropriate long-term treatment, and prevent unnecessary returns to the ED. Accordingly, the current ESC guidelines on ACS recommend that ‘in patients without recurrence of pain, normal ECG findings, negative troponin tests, and a low risk score, a non-invasive stress test for inducible ischemia’ should be performed. The current ACC/AHA guidelines for stress testing as well as those for management of non-ST segment elevation ACS specifically recommend ECG exercise testing (EET) without imaging in patients who can exercise and do not have substantial baseline ECG changes that preclude interpretation. They reserve imaging stress testing only for patients who do not fulfil those criteria.

### Echocardiography

The current ESC guidelines on ACSs put special emphasis on the early use of echocardiography in patients with acute chest pain. They state: ‘Among non-invasive imaging techniques, echocardiography is the most important modality in the acute setting because it is rapidly and widely available. LV systolic function is an important prognostic variable in patients with CAD and can be easily and accurately assessed by echocardiography. In experienced hands, transient segmental hypokinesia or akinesia may be detected during ischaemia. Furthermore, differential diagnoses such as aortic dissection, pulmonary embolism, aortic stenosis, hypertrophic cardiomyopathy, or pericardial effusion may be identified’. Echocardiography has been used for diagnosis and immediate risk assessment in patients presenting to the ED with symptoms that suggest ACS on the basis of its high degree of reliability in identifying regional wall-motion abnormalities (RWMA). A normal resting echocardiogram in patients admitted to a CPU indicates a lower clinical risk. Ischaemic RWMA can be detected by echocardiography almost immediately after the onset, preceding EKG alterations and biomarker elevation. Of course, RWMA do not only occur during acute MI, but also during severe ischaemia not associated with marker elevation (unstable angina). Therefore, echocardiography may also identify such patients when performed during chest pain or shortly thereafter. However, persistence of RWMA after an attack of resting angina is highly variable which will influence the accuracy of echocardiography to identify acute ischaemic episodes. Moreover, RWMA may be pre-existing.

In a busy ED, echocardiography will seldom be used for the diagnosis of RWMA as patients can be easily triaged using clinical information and an interpretable resting ECG. However, new RWMA seen by echocardiography may be the clue to the correct diagnosis of ACS in patients with non-diagnostic ECG changes (left bundle-branch block or paced rhythm) and persistent chest discomfort.

However, in the latter group of patients, it may be difficult to distinguish between RWMA induced by the electrical conduction abnormality and additional ones induced by ischaemia. Finally, it must be remembered that the accuracy of echocardiography is highly dependent on the skill of the personnel performing the examination.

The use of intravenous contrast agents may improve the quality of resting two-dimensional echocardiography for detection of resting RWMA by providing better endocardial border detection. Myocardial contrast echocardiography performed early in patients with on-going chest pain may be as accurate as resting MPI for early detection of acute MI or unstable angina requiring urgent revascularization.

The current ESC guidelines on the management of ACSs give a recommendation that ‘in patients without recurrence of pain, normal ECG findings, negative troponin tests, and a low risk score, a non-invasive stress test for inducible ischaemia is performed before deciding on an invasive strategy’. Stress echocardiography is well suited and better than basic clinical indicators to distinguish between low-risk patients who can be safely sent home from those in whom further work-up is required because they harbour flow-limiting stenoses in coronary arteries perfusing a large area of myocardium. Its advantages include immediate availability, no risk from radiation exposure, and information both on structural and functional data.

Sensitivity and specificity of stress echocardiography for obstructive CAD have been reported as 86 and 81%, respectively. Although stress echocardiography can be performed by exercise, an alternative method in CPU patients is using dobutamine. Dobutamine stress echocardiography has demonstrated generally excellent negative predictive value for obstructive CAD in CPU patients and has also provided important prognostic information regarding early and late cardiac events. The negative predictive value of DSE at 6-month follow-up has been reported between 91 and 96%.11

### Nuclear imaging

The clinical usefulness of rest radionuclide MPI with either technetium-99m-sestamibi or tetrofosmin in patients with acute chest pain is well validated. Rest MPI has the unique characteristic that images are abnormal at the very moment that regional myocardial blood flow is disturbed. The extent of perfusion abnormality correlates well with the anatomic extent of myocardial ischaemia.

The sensitivity of rest imaging for detecting acute MI is high (>90%) provided that imaging is performed <6 h of the onset of pain (Figure 1). The 80% specificity of abnormal rest imaging for acute MI can be explained readily because regional myocardial perfusion is also abnormal in conditions with severely reduced resting myocardial blood flow without myocardial necrosis, i.e. in unstable angina, which is the other entity of ACS. Furthermore, one single rest MPI one cannot distinguish between ACS and reduced regional blood flow due to prior infarction.

Very relevant for the clinical use of resting MPI in an ED is the excellent negative predictive value, which consistently has been reported to exceed 99%. This implies that, if a patient with on-going chest pain has completely normal rest MPI, it is highly
unlikely that the patient has an ACS. In addition to the diagnostic value of rest MPI, it also has important short-term prognostic value. Patients with normal rest MPI have a very low (≤1%) 30-day cardiac event rate, whereas patients with abnormal rest MPI have a 10–30% 30-day cardiac event rate.13,14

Because many patients no longer have ongoing chest pain at the time of evaluation in the ED, another novel approach has been investigated in recent years.15 This involves the evaluation of myocardial metabolism. During ischaemia, myocardial metabolism shifts from aerobic free-fatty acid utilization to anaerobic glucose utilization. Even when myocardial blood flow is improved and ischaemia no longer is present, the return to free fatty acid metabolism is slow and may take as much as 30 h. Decreased free fatty acid metabolism can be visualized using I-123 β-iodophenyl-pentadecanoic acid (BMIPP) imaging. This is often referred to as ‘ischaemic-memory imaging’.

In a recent multicentre trial involving 448 patients, I-123-BMIPP was injected on an average of 12 h after cessation of symptoms.15 BMIPP imaging had overall a similar sensitivity (~90%) and specificity (~80%) as rest MPI for detecting ACS. Importantly, when early (0–12 h after resolution of symptoms) and late (12–30 h) metabolic imaging was compared, sensitivity and specificity were similar.15 Thus, the unique niche for I-123-BMIPP imaging appears to be in patients who have no longer chest pain.

Although rest MPI appears an ideal and effective imaging strategy in a CPU, in reality it is infrequently used. The reason for this is that in clinical practice at the time of evaluation in a CPU, the majority of patients have become pain-free. Once a high-risk condition has been excluded, patients may already enter the risk stratification path which prescribes exercise testing as the next step.

Patients in whom a high-risk ACS can be ruled often have a low pre-test likelihood of CAD. If in such a patient, the EET is normal at good workload, one has virtually excluded significant CAD and these patients have a favourable outcome. However, if the EET is abnormal, there is a 50% chance that it is a false-positive result. To further elucidate this, the EET can be followed by exercise MPI because of its much higher specificity for significant epicardial stenosis. Because of the low-risk nature of the CPU population, most exercise ECGs can be expected to be normal and a step-wise approach, i.e. exercise ECG first followed by exercise MPI, is cost effective.16

If patients cannot perform adequate (≥7 METS) physical exercise, pharmacological vasodilator stress is appropriate. Because most of the stress MPI in these patients will be normal, rest MPI is not needed in the majority of patients. Thus, the imaging protocol can be relatively short (stress-only) and the patient is exposed to less radiation.

Coronary computed tomography

Currently, 64-slice CT systems with gantry rotation times between 330 and 420 ms constitute the widely accepted minimum requirement for coronary CT angiography. Despite substantial technical progress over the past years, the rapid motion of the coronary arteries remains a limitation of cardiac CT and image quality is not equal to invasive coronary angiography because the latter has a better temporal resolution. Low heart rates substantially improve image quality and it is currently recommended to lower the patients’ heart rate below 60 bpm in order to achieve optimal image quality.17 In patients with higher heart rates, with arrhythmias, severe obesity, or difficulty to follow breath-hold commands, the use of CT for cardiac imaging should be reconsidered.

Calcium scoring

Coronary artery calcifications can be detected and quantified in ‘native’ acquisitions, without the use of contrast agent (Figure 2). Coronary calcium is always caused by coronary atherosclerosis (with the only exception of patients on dialysis, in whom coronary arteries can calcify independently from atherosclerosis). The

![Figure 1](image1.png) Sensitivity, specificity, and negative predictive value (neg pred value) of rest MPI (thallium-201, Tc-99m-sestamibi, or Tc-99m-tetrofosmin) for the detection of ACS within 6 h of onset of chest pain.13 Reproduced from (13) with permission.

![Figure 2](image2.png) Detection of coronary calcium by computed tomography (CT) in a non-enhanced study. Calcifications are visible in the left main ostium and at the bifurcation of the left main coronary artery (arrows).
extent of coronary calcium is a surrogate marker for the extent of atherosclerotic plaque in the coronary, and numerous studies have convincingly demonstrated this to be a very good prognostic marker concerning future cardiac events, such as MI and cardiac death in the primary prevention setting. However, coronary calcium detection by CT is not useful in patients with acute chest pain because the absence of calcium does not permit to rule out ACS. In addition, the prevalence of calcium in the population is so high that the positive predictive value would be extremely low.

Computed tomography coronary angiography

Patients must be appropriately selected and CTCA may not be a useful option in patients with arrhythmias, inability to perform adequate breath hold, or severe obesity. If performed appropriately and by sufficiently trained physicians, CTCA provides reliable coronary visualization in the majority of patients as well as a high degree of confidence to detect and especially to rule out coronary artery stenosis (Figure 3). In two multi-centre trials both of which included patients with low risk ACS, CTCA demonstrated a sensitivity of 95–99%, a specificity of 64–83%, and a negative predictive value of 97–99% to identify patients with at least one coronary artery stenosis. The positive predictive value was lower (64 and 86%). This is due to a tendency to overestimate stenosis degree in CTCA especially at sites with calcifications.

Few studies have specifically addressed the diagnostic accuracy of CTCA in patients with acute chest pain. In 104 patients with non-ST-elevation ACSs, Meijboom et al. demonstrated a sensitivity of 100% (88/88) and a specificity of 75% (12/16) to identify patients with coronary lesions ≥50% luminal stenosis.

Ruling out obstructive coronary artery stenoses by CTCA has a very good prognostic value in patients with acute chest pain. A recently published, randomized 16-centre trial of more than 700 patients confirmed that the use of coronary CT angiography is a safe and cost-effective tool to evaluate patients with low-risk acute chest pain. Therefore, both an expert consensus document for the appropriate use of cardiac CT as well as the most recent ESC guidelines for the management of patients with non-ST segment elevation ACSs suggest that CTCA may be appropriate to rule out coronary stenoses in such patients. Strong emphasis on the use of CTCA in patients with acute chest pain and a low pre-test probability of obstructive coronary disease (>10% but <30% based on the algorithm provided in the guideline) is also given in the recent NICE guideline on ‘Assessment of recent onset chest pain or discomfort of suspected cardiac origin’.

Under ideal conditions, CTCA permits rather detailed visualization of coronary atherosclerotic plaque, both in stenotic and non-stenotic lesions. Lesions associated with ACSs often display large plaque volumes—typically predominantly non-calcified—as well as pronounced positive remodelling, low CT attenuation within the non-calcified plaque material, and a circular pattern of contrast enhancement (Figure 4). While it has been proposed that such parameters may be useful in the identification of high-risk individuals in primary prevention, no specific clinical value of visualizing plaque has been demonstrated in patients with acute chest pain.

Contrast-enhanced computed tomography imaging can also be used to visualize myocardial perfusion. Several early trials which were recently reviewed have demonstrated the feasibility of stress MPI by CT and evaluated the accuracy for the detection of coronary artery stenoses in stable chest pain. Early data indicate that resting myocardial perfusion defects in CT may be associated with myocardial necrosis in ACS (Figure 5), but confirmatory proof is missing. Although a combination of CTCA and CT perfusion imaging is potentially attractive, it has not yet been shown to be clinically useful in patients following an episode of acute chest pain.

Cardiac magnetic resonance

Technical advances in CMR imaging make this technique now also suitable for applications in the ED. In patients with suspected ACS, CMR may contribute important information regarding the
correct diagnosis and risk stratification once a high-risk scenario has been excluded. CMR employing resting perfusion imaging, functional cine imaging, and late gadolinium enhancement (LGE) imaging is able to accurately identify patients at high risk for short-term adverse events. Similar to echocardiography, CMR can detect RWMA at rest. As noted above, both echocardiography and nuclear imaging cannot distinguish between old and new RWMA or new and old resting perfusion defects. In contrast, CMR is able to distinguish between acute and chronic MI on the basis of T2-weighted oedema imaging. Although logistics of performing CMR in the ED are more challenging than those of performing echocardiography or CTCA, cine and LGE CMR plus T2-weighted oedema imaging can be performed within 30 min in low-risk patients. This CMR protocol identifies patients with ACS more reliably than clinical variables. Similar to echocardiography, CMR can diagnose diseases such as aortic dissection of hypertrophic cardiomyopathy which may imitate the clinical presentation of ACS. Moreover, CMR has unique capabilities of identifying patients with myocarditis who may present as ACS (see below).

CMR has a very good safety profile in patients evaluated for the presence of myocardial ischaemia following an attack of acute chest pain. In clinical practice, CMR stress testing is always performed using a pharmacological ‘stress’ agent. CMR protocols using dobutamine-increasing doses and—if required—added atropine accurately predict the presence of flow-limiting stenosis. In low-risk patients, a negative CMR dobutamine stress test has an excellent negative predictive value with very low subsequent event rates.

An alternative and more commonly used CMR protocol tracks the myocardial inflow of a contrast agent. Images are evaluated either visually or using quantitative analysis tools. A regionally

**Figure 4** CTCA in a patient with an acute coronary syndrome (NSTEMI). (A) Two-dimensional reconstruction of displaying the left main and left circumflex coronary artery. A high grade stenosis is present, the lesion is predominantly non-calcified, and displays substantial positive remodeling (arrows). (B) Corresponding invasive coronary angiogram (arrow = stenosis).

**Figure 5** Resting myocardial perfusion defect in a patient with ACS. Left: occlusion of the left circumflex coronary artery (arrow) in a 53-year-old patient with NSTEMI. CT before reperfusion shows obvious hypoperfusion in the lateral wall (right, arrows).
diminished perfusion reserve in response to the vasodilating agent adenosine is easily identified (Figure 6). This test is particularly safe in the setting of ACS since vasodilators do not induce myocardial stress. It can accurately predict coronary artery stenoses and prognosis at 1 year. In the latter study, none of the 105 patients with normal adenosine perfusion studies suffered a coronary event over the mean follow-up time of 1.3 years.

Microvascular dysfunction including microvascular spasm may be another source of acute resting chest pain. Adenosine CMR may demonstrate a circular subendocardial delay of gadolinium inflow in patients with microvascular abnormalities (Figure 7). Further research is required to validate findings at first-pass perfusion for this application.

Non-ischaemic sources of chest pain

Pulmonary embolism

All symptoms in patients with confirmed PE including chest pain are non-specific as they tend to occur as frequently in patients in whom PE is suspected but can be ruled out. If chest pain is pleuritic in character differentiation of PE from ACS is usually straightforward. However, chest pain in PE may also be retrosternal and oppressive in character which combined with a frequently elevated troponin may lead to the erroneous diagnosis of ACS. Imaging in the form of CT angiography or transthoracic or transoesophageal echocardiography (TEE) (in patients too sick to leave the intensive care unit for the CT suite) enters the diagnostic pathway early if the patient is hypotensive or already in cardiogenic shock. Once PE has been confirmed in such patients (directly by CT or TEE or indirectly by transthoracic echocardiography demonstrating right heart compromise), thrombolytic therapy will be instituted.

If patients with suspected PE are stable, further diagnostic steps depend on their pre-test probability of indeed having PE. Common protocols rely on CT angiography. Lung scintigraphy is a valuable alternative in those who should not be exposed to ionic contrast material, but CMR pulmonary angiography may also be employed. Right ventricular performance is important for placing patients in the low or intermediate risk categories. In general, echocardiography will identify right ventricular dysfunction with high accuracy but some patients may require CMR for this purpose.

Acute aortic syndromes

Acute aortic syndromes encompass a spectrum of acutely life-threatening diseases such as aortic dissection, intramural haematoma, penetrating atherosclerotic ulcer, and completed or imminent rupture of an aortic aneurysm. Imaging is required to identify the disease and to provide a basis for decision making concerning treatment options which may be surgical (typically if the ascending aorta is involved), interventional, or conservative. Hence, imaging of the entire aorta down to the iliac arteries is desirable, especially if a percutaneous intervention for dissection of the descending aorta is considered.

Echocardiography, magnetic resonance imaging, and computed tomography can be used to diagnose acute aortic syndromes. A normal chest X-ray does not exclude aortic disease and should not delay definitive aortic imaging. Transthoracic
echocardiography reliably identifies pericardial effusion, which in combination with aortic valve regurgitation is a hallmark of type A aortic dissection in patients with acute chest pain. TEE can identify ascending aortic dissection and intramural haematoma, but only partly visualizes the aortic arch and descending aorta. The ability to reliably rule out aortic dissection may be limited by reverberation artefacts and expertise is required. Both CMR and multi-detector row CT permit detailed imaging of the entire ascending and descending aorta and identification of intramural haematoma, dissection, rupture, and ulcer (Figure 8). The CT imaging sequence should contain a pre-contrast acquisition to identify subtle increases in mural contrast attenuation as to diagnose intramural haematoma. Although CT, TEE, and CMR are all reliable tools for diagnosing acute aortic syndromes and especially aortic dissection, CT imaging is most often used in the ED as it is usually logistically easier to obtain than TEE. However, TEE is the tool of choice in haemodynamically unstable patients in the intensive care unit. CMR is only rarely used in the emergency situation. Current guidelines recommend that a second imaging study should be obtained if clinical suspicion is high and a first test remains negative.

**Pericarditis**

Acute pericardial disease can be diagnosed by echocardiography, CT, and CMR. However, echocardiography is initially employed in a patient in whom a pericardial cause of acute chest pain is suspected. Echocardiography provides a rapid and accurate assessment of the pericardium and underlying cardiac function; also allows diagnosis of the presence of pericardial effusion and semi-quantify its severity and its physiological consequences. Pericarditis is the likely diagnosis in a patient with pericardial effusion and absence of WMA in a patient with non-diagnostic ECG changes and persistent chest discomfort, but uncomplicated acute pericarditis may be associated with normal echocardiographic findings. If the acoustic window is suboptimal, CT will demonstrate pericardial effusion and absence of WMA in a patient with non-diagnostic ECG changes and persistent chest discomfort, but uncomplicated acute pericarditis may be associated with normal echocardiographic findings.

Myocarditis and other cardiac sources of CP

In patients with persistent acute chest pain and ECG changes, coronary angiography is often performed even in patients without a typical risk factor constellation. If CAD is not found, the search for a potential origin of the chest pain must continue. Myocarditis...
is a differential diagnosis which must be considered especially in young men with intermittent severe chest pain, ST elevation or ST depression, and an elevated troponin. The imaging modality of choice for diagnosing or excluding myocarditis is CMR. In order to achieve the best diagnostic accuracy, CMR should examine the presence of (i) oedema, (ii) inflammatory hyperaemia, and (iii) irreversible inflammatory injury (‘Lake Louise Criteria’) (Figure 9). In patients with a history and presentation suggestive of myocarditis, two out of three positive criteria are considered indicative of active myocardial inflammation. The evaluation should be performed using quantitative signal intensity measurements. The presence of reduced left ventricular function and/or pericardial effusion is supportive of the diagnosis. A CMR scan providing information on all five criteria can be performed in less than 25 min. These criteria await further validation in larger trials especially trials with endomyocardial biopsy as the gold standard, but have yielded a good diagnostic accuracy in single-centre trials when a final clinical diagnosis of myocarditis was the diagnostic gold standard. While specificity and positive predictive value are high, these criteria have a limited sensitivity (67%) and therefore a negative scan may not allow for reliably excluding active myocardial inflammation. Interestingly, positive CMR markers suggesting myocardial inflammation have also been found in patients with dilated cardiomyopathy, a condition associated with angina chest pain in one-third of the patients.

Data on the prognostic value of CMR criteria are scarce, but the presence of inflammation and irreversible injury demonstrated by CMR may predict worsening of LV function. Although there is currently no established form of therapy for myocarditis, confirmation of active inflammation is nevertheless very helpful for patient management, avoiding additional diagnostic procedures and unnecessary treatment but alerting the physician to the need of continued surveillance of these patients. As a completely non-invasive imaging modality, CMR is well suited for patient follow-up and may show resolution of acute signs of inflammation.

**Imaging in patients with acute chest pain**

A possible scenario is given in the recent NICE guideline on management of chest pain of recent onset. In patients in whom anginal pain cannot be diagnosed or excluded on the basis of clinical parameters alone, further diagnostic steps are based on the pre-test probability that indeed the chest pain is caused by coronary artery disease (see Table 1 in the NICE guideline). Efficacy and cost considerations led NICE to advise against the use of EET in these patients. NICE sees the use of stress imaging mainly in patients with a pre-test probability between 30 and 60% (see flow chart #2 on ‘Diagnostic testing for people in whom stable angina cannot be diagnosed or excluded by clinical assessment alone’ in the Stable chest pain pathway in reference). Patients in whom the pre-test probability is between 10 and 30% are recommended to have CTCA. If the pre-test probability is below 10% or above 90%, the NICE guideline sees no incremental value in performing any kind of test to confirm or exclude the diagnosis of CAD as the source of chest pain. Direct referral for coronary angiography is recommended in those patients in whom the pre-test probability is between 60 and 90%. Once the diagnosis of stable angina has been made, further steps can be planned according to the most recent ESC guideline on chronic stable angina. Although the NICE guideline clearly recommends a rather aggressive diagnostic strategy in patients with acute chest pain, they also emphasize the important role of clinical judgement for making decisions (and modifying recommendations) in individual patients.

The scenario advocated by the current ESC guidelines on ACS for using imaging techniques can be summarized as follows: during initial management of the patient with non-ST-elevation, ACS.
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Figure 10 Decision-making algorithm in ACS. Echocardiography is mandatory in each patient, whereas other forms of imaging are optional (framed green in the validation box). ACS, acute coronary syndrome; CAD, coronary artery disease; CT, computed tomography; ECG, electrocardiogram; GRACE, Global Registry of Acute Coronary Events; MRI, magnetic resonance imaging; STEMI, ST-elevation MI. Modified from Hamm et al.1 with permission.

transthoracic echocardiography at rest is now mandatory to assess LV function as an important prognostic parameter. Echocardiography may also show RWMA or identify differential diagnoses. Although the potentials of CMR and nuclear imaging are recognized, both are not given a high priority in the initial management due to limited availability on a 24/7 basis in most hospitals. However, they may be used as options in selected patients where available (Figure 10). Next to echocardiography, CT is recognized as the preferred imaging test for ruling out pulmonary embolism. CTCA is given a Ia-B recommendation to ‘be considered as an alternative to invasive angiography to exclude ACS when there is a low to intermediate likelihood of CAD and when troponin and ECG are inconclusive’. Performing a non-invasive stress test for inducible ischaemia is a I-A recommendation in patients without recurrence of pain, normal ECG findings, negative troponins tests, and a low-risk score, before deciding on an invasive strategy. The guideline does not differentiate between EET and imaging stress testing nor is a preference given for any of the above-mentioned stress imaging modalities. The ESC guideline leaves more space for clinical judgment and is more in line with current clinical practice than the new NICE guideline.

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