Prognostic value of adenosine stress myocardial perfusion by cardiac magnetic resonance imaging in patients with known or suspected coronary artery disease

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Summary

Background: Cardiac magnetic resonance imaging (CMR) has been intensely researched in recent years, and its high diagnostic accuracy for myocardial ischemia has been demonstrated. However, its prognostic information is very limited.

Aim: We sought to assess the value of adenosine stress myocardial perfusion by CMR in predicting cardiac events in patients with known or suspected coronary artery disease (CAD).

Design: Retrospective study.

Methods: From January 2003 to December 2008, we retrospectively reviewed consecutive patients with or without history of CAD referred for evaluation of suspected myocardial ischemia who had undergone adenosine stress CMR in our hospital. End points were cardiac death or non-fatal myocardial infarction (MI).

Results: After a mean follow-up of 3.2 ± 1.6 years in 203 patients, 15 (7.4%) cardiac events occurred. The 4-year event-free survival was 96.2% for patients with normal stress CMR perfusion and 71.5% for those with abnormal stress CMR perfusion. Univariate analysis showed that both adenosine-induced reversible perfusion defect and delayed gadolinium enhancement by CMR were significant predictors of cardiac events [Hazard ratio (HR) 9.31; 95% Confidence Interval (95% CI) 3.18–27.3; and HR 9.24; 95% CI 3.27–26.08; P < 0.001, respectively]. By multivariate analysis, adenosine-induced reversible perfusion defect remained an independent predictor of cardiac events (HR 7.77; 95% CI 2.50–24.18; P < 0.001). In a stepwise multivariate model (Cox regression), an abnormal stress CMR perfusion result had significant incremental predictive value over clinical risk factors and resting regional wall motion abnormality (RWMA) (P < 0.001).

Conclusion: In patients with known or suspected CAD, adenosine stress CMR could be used to identify patients at high risk for subsequent cardiac death or nonfatal MI. A normal CMR perfusion was associated with a very low long-term event rate and excellent long-term prognosis. In addition, stress CMR perfusion provided important incremental prognostic information over clinical risk factors and RWMA.

Introduction

While coronary angiography remains the gold standard for diagnosis of coronary artery disease (CAD),¹ non-invasive tests for CAD remain an integral part of clinical cardiology.

Currently, non-invasive imaging for evaluation of CAD is largely performed by either anatomical imaging or functional imaging. Anatomical imaging includes coronary computer tomography directly visualize the coronary arteries. Functional testing, such as single-photon emission computed...
tomography (SPECT) or stress echocardiography, evaluates the hemodynamic effect of coronary obstructive disease.

Cardiac magnetic resonance (CMR) imaging for the evaluation of CAD has tremendous potential because of its high-spatial resolution, image contrast and lack of ionizing radiation and excellent depiction of wall motion. Indeed, CMR stress testing has been intensely researched in recent years, and its high-diagnostic accuracy for CAD has been demonstrated. However, the prognostic information, especially in adenosine perfusion CMR, is very limited. In contrary, prognostic value of other stress test including SPECT, stress echocardiography and treadmill is well-established in large prospective studies. Therefore, the role of CMR in the evaluation of patients with CAD remains to be fully defined.

Prognostic studies using CMR are limited in number. Hundley et al. were the first who found that dobutamine/atropine CMR had good prognostic value in their report of 279 patients in 2002. After an average follow-up of 20 months, the annualized hard cardiac events rate was around 1% in patients without ischemia and left ventricular ejection fraction (LVEF) >40%. Recent literature extended this prognostic value to adenosine stress CMR. In 2009, Steel et al. reported that in 254 patients referred for symptoms of myocardial ischemia, CMR imaging provided robust risk stratification. Patients with normal CMR examination had a 98.1% negative annual event rate for death or myocardial infarction (MI).

However, the above studies only dealt with specific subsets of patient population (e.g. patients presented with acute chest pain). Moreover, they examined the ability of stress CMR to predict composite adverse outcome that included ‘soft’ cardiac end points like revascularization and incidence of heart failure.

The objective of present study was to assess the value of adenosine stress CMR perfusion in predicting cardiac events (cardiac death or non-fatal MI) in patients with known or suspected CAD. The incremental prognostic value of CMR stress result over traditional cardiac risk factors would be determined.

Methods

Study population

This was a single-centre retrospective study to determine the outcomes of patients who had undergone adenosine stress CMR. The study was approved by the local research ethics committee.

From January 2003 to December 2008, we retrospectively reviewed consecutive patients referred for evaluation of suspected myocardial ischemia who had undergone adenosine stress CMR in our hospital. Patients were eligible if they had suspected or known CAD (with or without prior percutaneous or surgical revascularization).

Exclusion criteria

Patients were excluded from the study if the referring indication for CMR was for viability testing or functional assessment of a known intermediate coronary artery stenosis. They were excluded because of the high probability of myocardial ischemia before CMR examination.

Patients referred for adenosine CMR because of suspected hypertrophic obstructive cardiomyopathy or myocarditis was also excluded, due to co-existing pathologies apart from CAD.

Patients were excluded if the CMR examination was failed. Either due to development of side effects during the examination, or patients failed to hold breath.

Clinical data collection

Cohort data and outcome were collected by detailed review of medical notes. Electronic patient record data was also retrieved for analysis. Demographic and clinical data, as well as stress CMR results, were collected.

Clinical data definition

Patients referred for evaluation of chest pain were classified as having typical angina, atypical angina or non-cardiac chest pain as previously described. In patients without history of CAD, the pretest probability of CAD was assessed according to a previously validated score described by Morise et al. Patients were classified as low pretest probability for a score of 0–8 points, intermediate for 9–15 points, and high for points >15.

A history of CAD was defined as previous MI, previous coronary revascularization, or previous angiographic documentation of any significant (>50%) coronary stenosis. Clinical variables were defined according to the Framingham Risk Score assessment.

In addition, the total number of cardiac risk factors (range from 0 to 7) was determined by the number of the following risk factors: hypertension, hyperlipidaemia, diabetes mellitus (DM), age (>45 years for men, >55 years for women), current or prior smoking history, a history of CAD and a family history of CAD.
CMR protocol

CMR studies were performed with the patient in the supine position using the Siemens Magnetom Sonata machine. Cardiac synchronization was performed with four electrodes placed on the left anterior hemithorax.

Cine imaging at rest was acquired by the standard short axis and three long axis views (two-, three- and four-chamber) using the true fast imaging with steady precession sequence.

Perfusion stress imaging was acquired using the standard short axis view. After infusion of adenosine at a constant rate of 140 μg/kg/min over 4 min, first pass kinetic of a gadolinium-based contrast agent was measured during breath holding.

After waiting for 15 min for contrast to clear, a second perfusion in the same orientation and with the same setting was performed at rest. Five to 10 min after this second bolus, late gadolinium enhancement images were acquired by using the short axis view and three long axis views.

One experienced consultant radiologist in our hospital evaluated all the CMR studies. For analysis of first-pass myocardial perfusion images, the left ventricle was divided into the standard 16-segment model (Figure 1). Both perfusion images and late enhancement images were assessed visually. Ischemia was defined as adenosine-induced reversible perfusion defect (abnormal stress CMR perfusion).

Follow-up was completed in December 2009. Follow-up was obtained by review of hospital databases, medical records and death certificates. The diagnoses of cardiac events were retrospectively verified by cardiologists conducting this study. End points were cardiac death and non-fatal MI. Cardiac death was defined as death due to acute MI, congestive heart failure, life-threatening arrhythmias or cardiac arrest. Unexpected and unexplained sudden death was also considered cardiac death. MI was defined by the presence of new symptoms of myocardial ischemia, or ischemic electrocardiogram (ECG) changes, accompanied with increase in markers of myocardial necrosis.

Revascularization procedures during follow-up were collected although they were not considered ‘events’ as CMR results may influence patients’ management and decision for revascularization.

Statistical analysis

Categorical variables were reported as percentages and comparison between groups based on the χ²-test or Fisher’s exact test. Continuous variables were reported as mean ± SD, and differences were assessed with the Independent samples t-test or Mann–Whitney test.

Cumulative event curves were calculated by the Kaplan–Meier method and compared by the log-rank test. Patients were censored at the time of development of cardiac events or non-cardiac death.

Univariate and multivariate associations of clinical and CMR variables with the end points were assessed with Cox’s proportional hazards models. The Cox model was used to estimate the risk of a given variable as expressed by a hazard ratio (HR) with corresponding 95% confidence interval (CI). To build the best final model for cardiac death and non-fatal MI, we performed multivariable Cox regression analysis using a step-wise forward selection with P = 0.01 as the criteria for model entry or stay.

In addition, to investigate the prognostic value of stress CMR incremental to clinical data, a three-step modeling procedure was performed, with variables included in the model in the same order as in clinical practice. First, the χ² value for patients with more than three clinical risk factors was calculated. Then the second step was to add CMR cine imaging results, i.e. regional wall motion abnormality (RWMA) at rest. The third analysis was conducted by adding adenosine-induced perfusion defect to the second step. The incremental prognostic value of the added variable was determined by comparison of the global χ² value.

Statistical analyses were performed with the use of SPSS software (version 13.0)

Results

Two Hundred and Sixty patients had undergone adenosine stress CMR during the study period. The indication of CMR included viability testing in 27 (10.4%) patients, functional testing of a known intermediate coronary artery stenosis in six (2.3%) patients and suspected hypertrophic obstructive cardiomyopathy/myocarditis in seven (2.7%) patients. They were excluded from the study population.

Figure 1. Standard 16-segment model for myocardial perfusion imaging.
Another 17 (6.5%) patients were excluded due to incomplete CMR examination after development of side effects during examination or failed the breath-holding instruction. In two of these 17 patients, CMR were not performed because of claustrophobia. Another six of them failed the breath-holding instruction resulting in poor image quality. Five patients had very frequent ventricular ectopic beats at baseline. Two patients developed adenosine-induced dyspnoea, which lead to early termination of the protocol. Two patients developed adenosine-related bradycardia or atrio-ventricular heart block. Side effects resolved quickly after stopping adenosine. The remaining 203 patients were enrolled for analysis.

**Baseline characteristics**

For the study population, mean age was $62.1 \pm 11.6$, and 58.6% of patients were men. The mean follow-up period was $3.2 \pm 1.6$ years (median, 3.1 years; range, 0.3–6.8 years). The indications for CMR included investigation for patients presented with chest pain in 89.2% of cases, investigation for patients presented with shortness of breath or heart failure symptoms in 4.4% of cases, 6.4% were for investigation of incidental abnormal ECG or treadmill finding. In 35.8% of cases, prior stress testing was done before CMR.

Clinical and demographic characteristics of the 203 patients are summarized in Table 1. Overall, 43 patients (21.2%) had abnormal stress CMR perfusion. They had evidence of ischemia on CMR as defined by adenosine-induced reversible perfusion defect.

Patients with abnormal stress CMR perfusion were older, were more likely to be male or having hypertension, hyperlipidaemia, smoking history or history of CAD. Total number of risk factors was also greater in patients with abnormal perfusion images.

**CMR results**

CMR derived left ventricular parameters were LVEF $65\% \pm 13\%$ (median 67.6%), end-systolic volume

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**Table 1** Baseline clinical characteristics

<table>
<thead>
<tr>
<th>All patients (n=203)</th>
<th>Normal CMR perfusion (n=160)</th>
<th>Abnormal CMR perfusion (n=43)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>58.6</td>
<td>54.4</td>
<td>74.4</td>
</tr>
<tr>
<td>Age (years)</td>
<td>$62.1 \pm 11.6$</td>
<td>$60.7 \pm 11.8$</td>
<td>$67.3 \pm 9.0$</td>
</tr>
<tr>
<td>Current/exsmoker</td>
<td>29.1</td>
<td>25.0</td>
<td>44.2</td>
</tr>
<tr>
<td>DM</td>
<td>30</td>
<td>28.8</td>
<td>34.9</td>
</tr>
<tr>
<td>Hypertension</td>
<td>69.5</td>
<td>64.4</td>
<td>88.4</td>
</tr>
<tr>
<td>Hyperlipidaemia</td>
<td>45.8</td>
<td>40.6</td>
<td>65.1</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>1.54 $\pm 1.15$</td>
<td>1.47 $\pm 1.13$</td>
<td>1.81 $\pm 1.17$</td>
</tr>
<tr>
<td>HDL</td>
<td>1.45 $\pm 0.43$</td>
<td>1.50 $\pm 0.43$</td>
<td>1.25 $\pm 0.36$</td>
</tr>
<tr>
<td>LDL</td>
<td>2.71 $\pm 0.82$</td>
<td>2.73 $\pm 0.85$</td>
<td>2.63 $\pm 0.73$</td>
</tr>
<tr>
<td>History of CAD</td>
<td>15.8</td>
<td>11.9</td>
<td>30.2</td>
</tr>
<tr>
<td>Family history of CAD</td>
<td>3.9</td>
<td>3.8</td>
<td>4.7</td>
</tr>
<tr>
<td>Prior MI</td>
<td>9.9</td>
<td>9.4</td>
<td>11.6</td>
</tr>
<tr>
<td>History of heart failure</td>
<td>11.8</td>
<td>8.1</td>
<td>25.6</td>
</tr>
<tr>
<td>Body mass index</td>
<td>25.0 $\pm 4.0$</td>
<td>24.9 $\pm 4.0$</td>
<td>25.2 $\pm 3.9$</td>
</tr>
<tr>
<td>Prior coronary revascularization</td>
<td>12.3</td>
<td>10.0</td>
<td>20.9</td>
</tr>
<tr>
<td>PCI</td>
<td>3.4</td>
<td>3.1</td>
<td>4.7</td>
</tr>
<tr>
<td>CABG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretest probability</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>16.1</td>
<td>19.7</td>
<td>0</td>
</tr>
<tr>
<td>Intermediate</td>
<td>51.6</td>
<td>50.4</td>
<td>57.1</td>
</tr>
<tr>
<td>High</td>
<td>32.3</td>
<td>29.9</td>
<td>42.9</td>
</tr>
<tr>
<td>Total number of risk factors</td>
<td>2.8 $\pm 1.5$</td>
<td>2.5 $\pm 1.4$</td>
<td>3.7 $\pm 1.0$</td>
</tr>
<tr>
<td>Anti-platelet</td>
<td>61.6</td>
<td>56.3</td>
<td>81.4</td>
</tr>
<tr>
<td>Betablocker</td>
<td>52.2</td>
<td>50.0</td>
<td>60.5</td>
</tr>
<tr>
<td>ACEI</td>
<td>43.3</td>
<td>41.3</td>
<td>51.2</td>
</tr>
<tr>
<td>Statin</td>
<td>43.3</td>
<td>40.0</td>
<td>55.8</td>
</tr>
</tbody>
</table>

C, indicates $\chi^2$-test; F, Fisher’s exact test; T, independent samples t-test; M, Mann–Whitney test. The bold values indicate variables that were statistically significant ($P<0.05$).
LVESV (46 ± 29 g), end-diastolic volume 122 ± 38 g and mass 105 ± 42 g.

RWMA at rest, late gadolinium enhancement and abnormal stress CMR perfusion was present in 25 patients (12.3%), 27 patients (13.3%) and 43 patients (21.2%), respectively. An example of abnormal CMR perfusion was shown in figure 2.

Univariate analysis of potential predictors of cardiac events

After a mean follow up of 3.2 ± 1.6 years, non-cardiac death occurred in six patients. These patients were censored at the time of death. Fifteen cardiac events were observed (7.4%), including four cardiac deaths and 11 non-fatal MI.

Overall, cardiac catheterization was performed in 52 patients (25.6%) and significant CAD was diagnosed in 30 cases (57.7%). Twenty-two cases (42.3%) underwent revascularization procedures [all patients received PCI except one patient received coronary artery bypass grafting (CABG)] during follow-up period. For the 43 patients who had abnormal stress CMR perfusion, cardiac catheterization was performed in 27 patients (62.8%) with 21 cases (77.8%) diagnosed to have significant CAD.

Univariate analysis of potential predictors of cardiac events was listed in Table 2. A significant association was found for age, hyperlipidaemia, DM, known CAD and total number of cardiac risk factors >3. Presence of RWMA at rest, LVEF, LVESV, delayed gadolinium enhancement and adenosine-induced perfusion defect in CMR were all significant predictors of cardiac events.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Crude HR</th>
<th>95% CI</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (per decade)</td>
<td>2.12</td>
<td>1.20–3.74</td>
<td>0.01</td>
</tr>
<tr>
<td>Gender, male</td>
<td>1.05</td>
<td>0.37–2.95</td>
<td>0.929</td>
</tr>
<tr>
<td>Smoking history</td>
<td>1.39</td>
<td>0.47–4.07</td>
<td>0.55</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.52</td>
<td>0.45–5.51</td>
<td>0.11</td>
</tr>
<tr>
<td>Hyperlipidaemia</td>
<td>3.38</td>
<td>1.07–10.62</td>
<td>0.038</td>
</tr>
<tr>
<td>DM</td>
<td>2.93</td>
<td>1.06–8.07</td>
<td>0.038</td>
</tr>
<tr>
<td>Known CAD</td>
<td>3.58</td>
<td>1.27–10.08</td>
<td>0.016</td>
</tr>
<tr>
<td>Total risk factor &gt;3</td>
<td>3.54</td>
<td>1.26–9.96</td>
<td>0.017</td>
</tr>
<tr>
<td>LVEF</td>
<td>0.94</td>
<td>0.92–0.97</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVESV</td>
<td>1.02</td>
<td>1.01–1.03</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RWMA at rest</td>
<td>5.67</td>
<td>2.04–15.76</td>
<td>0.001</td>
</tr>
<tr>
<td>Delayed gadolinium enhancement</td>
<td>9.24</td>
<td>3.27–26.08</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Adenosine-induced reversible perfusion defect</td>
<td>9.31</td>
<td>3.18–27.3</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

The bold values indicate variables that were statistically significant (P<0.05).

Multivariate analysis of potential predictors of cardiac events

In multivariate analysis, adenosine-induced perfusion defect and LVEF remained independent predictors of cardiac events (Table 3). Variables that were not significant were age, hyperlipidaemia, DM, known CAD, total number of cardiac risk factors >3, presence of RWMA at rest, LVESV and delayed enhancement.
The Kaplan–Meier curves of event-free survival were illustrated in Figure 3. The 4-year event-free survival was 96.2% in patients with normal stress CMR perfusion vs. 71.5% in patients with abnormal test. Patients with normal stress CMR perfusion had a 99% negative annual event rate for cardiac death/MI.

**Incremental value of stress CMR**

Cox regression models were fit to test the incremental value of adenosine-induced perfusion defect over clinical variables (total number of cardiac risk factors >3). The presence of RWMA at rest increased the likelihood of cardiac events over the analysis of clinical risk factors ($\chi^2$, 14.0 vs. 6.5; $P=0.002$). An abnormal result of adenosine-induced perfusion defect further increased the $\chi^2$ from 14.0 to 24.5 ($P<0.001$).

**Discussion**

The principle findings of our study were:

(i) adenosine stress CMR perfusion could be used to identify patients at risk for future cardiac death or MI;
(ii) a normal adenosine stress CMR identified a low-risk population, with 4-year event-free survival of 96.2% in patients with normal CMR perfusion; and
(iii) adenosine stress CMR perfusion provided incremental prognostic value over clinical valuables in predicting cardiac events.

Our result was comparable to the prognostic data observed in previous studies. We had shown adenosine-induced perfusion defect was a good predictor of cardiac events, which carried a ~9-fold increased risk of future cardiac events. In the most recently published report by Steel et al. in 2009, they showed that a patient with ischemia detected by stress perfusion CMR had a ~7-fold increased risk for experiencing a cardiac event.

In our study, the annualized cardiac event rate was around 1% in patients with normal myocardial perfusion, a figure which was comparable to that reported by Jahnke et al., indicating good prognosis in patients with normal adenosine CMR. They also showed CMR stress testing provided important incremental information over clinical risk factors.

Although recent literature indicated the possible prognostic value of adenosine stress CMR, some previous studies dealt with specific subsets of patient populations (e.g. presenting with acute chest pain). The strength of our study was that we extended the findings to routine patients referred to a general hospital for CAD evaluation, predominantly with intermediate to high pretest probability.

Another strength was that we included ‘hard’ cardiac events only (i.e. cardiac death and non-fatal MI) as our study end point. Some previous studies included only ‘soft’ end points including hospitalization due to cardiac events and revascularization.

**Table 3** Multivariate predictors of cardiac death and non-fatal MI in patients undergoing adenosine stress CMR

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Adjusted HR</th>
<th>95% CI</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF</td>
<td>0.92</td>
<td>0.88–0.056</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Adenosine-induced reversible perfusion defect</td>
<td>7.77</td>
<td>2.50–24.18</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

The bold values indicate variables that were statistically significant ($P<0.05$).
Our study also provided long-term follow-up results as compared to 1–2 years follow-up result in previous studies, which is important in terms of prognostic information and implication.

Stress tests for prognosis in patients with suspected CAD should be performed in those with intermediate or high-pretest probability. In this group of patients, stress tests can identify patients at different degrees of risk, which can be used to guide further evaluation and therapy, including the choice between medical therapy and revascularization in patients with stable angina.

The role of stress CMR remains to be determined in this aspect. The ACC/AHA 2002 guidelines\(^\text{14}\) on management of stable angina suggest treadmill, stress echocardiography or radionuclide perfusion imaging for risk stratification, depends on patients’ ability to exercise and their baseline ECG. These recommendations were not changed in the 2007 ACC/AHA focused update on stable angina.\(^\text{15}\)

Our study suggested a good prognostic value of adenosine stress CMR; it might hold a similar prognostic value as other stress imaging and might equally be used for identifying a low-risk patient group. Large scale prospective study is warranted in this aspect.

Prognostic accuracy of a stress test has a growing importance with the concept of an increasing demand for non-invasive assessment of the indication for coronary angiography.\(^\text{16}\) Previous registries suggested an unsatisfactory diagnostic yield of only 60–70\(^\%\)\(^\text{17}\) in invasive coronary angiography. A study using radionuclide myocardial perfusion imaging showed that a non-invasive gatekeeper approach could make about half of catheterizations redundant.\(^\text{18}\) The approach of an ischemia-guided (by nuclear medicine perfusion scans) selective catheterization has been shown to potentially contribute to saving health expenses.\(^\text{19}\) Further study is warranted to investigate the role of CMR as a ‘gatekeeper approach’ for selective coronary angiography.

In patients who present with stable angina, stress imaging can also guide therapeutic decision making apart from risk stratification. In the nuclear substudy of the Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation (COURAGE) trial,\(^\text{20}\) Shaw et al. showed that adding percutaneous coronary intervention to optimal medical therapy resulted in a greater reduction in ischemia than did medical therapy alone. Moreover, they also found that the magnitude of residual ischemia on follow-up was proportional to the risk of death or MI, and a \(\geq5\%\) reduction in ischemia was associated with a significant reduction in risk, particularly if baseline ischemia was moderate to severe. In this study, \(\geq10\%\) ischemic myocardium identified by SPECT was considered moderate to severe ischemia.

Although the results of CMR seem promising, its role is still unclear in the diagnostic workup of patients presenting with cardiac symptoms. The ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR 2006 Appropriateness Criteria for Cardiac Computed Tomography and CMR indicates that the use of CMR stress testing is appropriate in individuals with intermediate pretest probability of CAD and those with an uninterpretable pretest probability of CAD and those with an uninterpretable pretest probability of CAD and those with an uninterpretable pretest probability of CAD and those with an uninterpretable pretest probability of CAD and those with an uninterpretable pretest probability of CAD.\(^\text{21}\)

Some limitations apply to our study. This was a retrospective study, which was prone to confounding factors and bias. For the representativeness of our patient group, the study population came from a tertiary referral centre, having patients with a higher prevalence of CAD and other cardiovascular risk factors. Thus our finding might not be applicable to the general population. Moreover, selection bias existed from the pattern for CMR referral, with choice of stress testing according to the personal preference of the responsible physician. For CMR imaging interpretation, only one single radiologist interpreted all the images by visual assessment (qualitative assessment), and he was not blinded to the clinical history, baseline LVEF and RWMA.

In conclusions, in patients with known or suspected CAD, adenosine stress CMR can be used to identify patients at high risk for subsequent cardiac death or non-fatal MI. Adenosine-induced perfusion defect is an independent predictor of cardiac events after adjustment of effects of late gadolinium enhancement and other clinical risk factors. A normal stress CMR perfusion is associated with a very low long-term event rate and excellent long-term prognosis. Our results indicates that adenosine-stress CMR holds a similar prognostic value as other perfusion imaging such as SPECT scan and may equally be used for identifying the low-risk patient group. Verification of our findings in large-scale, prospective, randomized, multicentered trials dealing with more general population may be warranted.

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References