Comparison of the Prognostic Value of Normal Regadenoson With Normal Adenosine Myocardial Perfusion Imaging With Propensity Score Matching

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OBJECTIVES The aim of this study was to test the hypothesis that patients with normal regadenoson myocardial perfusion imaging (MPI) have a low rate of cardiac events, similar to patients with normal adenosine MPI.

BACKGROUND Regadenoson, a new selective adenosine A2A receptor agonist, is now a widely used stress agent for MPI. The low rate of cardiac events in patients with normal adenosine MPI is well-documented, but the prognostic implications of a normal regadenoson MPI have not been examined and compared with those with adenosine.

METHODS Data on primary composite endpoint (cardiac death, myocardial infarction, and coronary revascularization) were collected for 2,000 patients (1,000 regadenoson, and 1,000 adenosine stress) with normal myocardial perfusion and left ventricular ejection fraction referred for vasodilator MPI. In addition, propensity scores were used to assemble a balanced cohort of 505 pairs of patients who were balanced on 36 baseline characteristics.

RESULTS The primary endpoint occurred in 21 (2.1%; 1.1%/year) patients in the regadenoson group and 33 (3.3%; 1.7%/year) patients in the adenosine group (hazard ratio [HR] for regadenoson vs. adenosine: 0.62; 95% confidence interval [CI]: 0.36 to 1.08; p = 0.090). In the propensity-matched pairs, the primary endpoint occurred in 7 (1.4%; 0.7%/year) patients in the regadenoson group and 13 (2.6%; 1.3%/year) patients in the adenosine group (matched HR: 0.58; 95% CI: 0.23 to 1.48; p = 0.257). Cardiac deaths were infrequent in the entire sample and in the propensity-matched groups; the cardiac death rate was 0.9%/year and 1.15%/year in the regadenoson and adenosine groups (HR: 0.77; 95% CI: 0.42 to 1.43; p = 0.404) in the pre-match sample and 0.5%/year and 0.7%/year in the matched groups, respectively (HR: 0.83; 95% CI: 0.25 to 2.73; p = 0.763).

CONCLUSIONS Major cardiac events are infrequent in patients with normal regadenoson MPI. These findings provide assurance that normal MPI using a simpler stress protocol with regadenoson provides prognostic data similar to normal adenosine MPI. (J Am Coll Cardiol Img 2012;5:1014–21) © 2012 by the American College of Cardiology Foundation

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Regadenoson is a selective adenosine $A_{2A}$ receptor agonist and is now the stress agent most widely used in the United States, despite the short time since its approval by the Food and Drug Administration (FDA). Unlike adenosine, regadenoson is given as a bolus rather than as an infusion, simplifying the testing protocol, and is better-tolerated by patients (12–14). Although randomized phase 3 multicenter trials have demonstrated that regadenoson is noninferior to adenosine for the detection of reversible perfusion abnormalities (15,16), there are no data on risk prediction, especially the prognostic value of normal regadenoson MPI. This information is needed, because of the concern that normal images might be falsely normal, because regadenoson is given as a bolus rather than as infusion and therefore the timing of tracer injection in relation to the bolus injection is crucial to the image results. Unlike adenosine, regadenoson is given as a fixed dose that is not adjusted for weight, raising concerns for suboptimal dosing in overweight individuals. Furthermore, it is inherently important to perform prognostic studies with new stress agents to confirm the implications of using these agents in practice, because the prognostic data are of more relevance to the clinician than the diagnostic data.

The objective of the current study is to compare outcomes of patients with normal regadenoson and adenosine MPI. Considering the diagnostic equivalence of these 2 agents, we hypothesized that patients with normal regadenoson MPI will have similar prognosis to patients with normal adenosine MPI.

**METHODS**

**Study population.** Data were collected on 2,000 patients who had normal vasodilator-only MPI. Of these, 1,000 consecutive patients had normal regadenoson MPI performed between July 2008 and June 2009, and another 1,000 consecutive patients had normal adenosine MPI performed between July 2006 and June 2007 (before our center switched from adenosine to regadenoson). All patients had normal left ventricular ejection fraction (LVEF). The institutional review board for human research at the University of Alabama at Birmingham approved this study.

**MPI.** Adenosine was administered intravenously as an infusion (140 μg/kg/min for 5 min). Technetium-99m-sestamibi was injected at 3 min into the infusion. Regadenoson was administered as a single peripheral intravenous bolus of 0.4 mg, followed by saline flush. Technetium-99m-sestamibi was administered intravenously, 10 to 20 s after the saline flush. All the studies were done in the absence of accompanying exercise.

Gated single-photon emission computed tomography images were acquired 1 h after tracer injection with a dual-head detector gamma camera with a low-energy, high-resolution collimator with a $64 \times 64$ matrix. The cameras operated in an elliptical 180° acquisition orbit with 32 projections and 30 s/project. A 15% energy window centered on the 140-keV gamma peak was used for imaging. Gating was done with 8 to 16 frames/RR cycle. Butterworth filtering followed by filtered back projection reconstruction was performed, and image interpretation was done without attenuation or scatter correction. All MPI were interpreted by readers blinded to subsequent events. Rest images were obtained whenever there was uncertainty in the interpretation of the stress images as previously described (17). The images were interpreted visually and aided by automated polar maps with the 4DM program (18–20). The LVEF and end-diastolic and end-systolic volumes were measured from stress gated images on the basis of a method previously described by Germano et al. (21). For purposes of this study, normal MPI was defined as LVEF $\geq 50\%$ and normal perfusion pattern.

**Other baseline characteristics.** Variables abstracted from the medical records of patients included patient demographic data; comorbidities including prior myocardial infarction (MI), diabetes mellitus, hypertension, hyperlipidemia, and stroke; prior cardiovascular interventions, such as percutaneous coronary intervention or coronary artery bypass grafting; history of tobacco use; medication usage at the time of MPI; and laboratory results (serum creati-
nine and lipid panel). The estimated glomerular filtration rate (eGFR) was calculated according to the Modification of Diet in Renal Disease study formula (22). Patients were considered to have chronic kidney disease if their eGFR was 15 to 60 ml/min/1.73 m². Patients were considered to have end-stage renal disease if their eGFR was <15 ml/min/1.73 m² or if they were receiving renal replacement therapy.

Outcomes variables. The primary outcome of the study is a composite endpoint of major adverse cardiovascular events (MACE) occurring within 2 years of the normal MPI. These include cardiac death, nonfatal MI (as documented by appropriate combination of symptoms, electrocardiography, and enzyme changes), and coronary revascularization with either percutaneous coronary intervention or coronary artery bypass grafting. Cardiac mortality is defined as death resulting from fatal arrhythmias, MI, or heart failure as determined by reviewing electronic medical records and verified with Social Security Death Index database up to June 13, 2011. Cardiac catheterization and coronary revascularization data were verified by review of hospital records. Time to event is defined as days from baseline to first MACE event, noncardiovascular death, or end of 2 years of follow-up. Secondary endpoints are cardiac and all-cause mortality.

Assembly of a balanced study cohort. Baseline characteristics of patients with normal regadenoson versus normal adenosine MPI were compared with Pearson chi-square, Student t, or Wilcoxon rank sum tests as appropriate (23). Considering the significant differences in distribution of key baseline characteristics between the groups, we used propensity scores to assemble a cohort in which the 2 groups are balanced on all measured baseline characteristics. We began by estimating propensity scores for the receipt of regadenoson with a non-parsimonious logistic regression model (24). In the model, receipt of regadenoson was the dependent variable, and baseline characteristics displayed in Figure 1 were entered as covariates. With a “greedy” matching protocol; we matched patients in the regadenoson group with those in the adenosine group who had similar propensity scores (24). Absolute standardized differences that directly quantify bias in the means (or proportions) of

![Image of Love Plot of Selected Variables]

**Figure 1. Love Plot of Selected Variables**

Absolute standardized differences before and after propensity score matching for patients receiving regadenoson versus adenosine. BP = blood pressure; CABG = coronary artery bypass grafting; CCB = calcium channel blocker; CHF = congestive heart failure; CKD = chronic kidney disease; CVA = cerebrovascular accident; FRS = Framingham Risk Score; HR = heart rate; LVEF = left ventricular ejection fraction; MI = myocardial infarction; PCI = percutaneous coronary intervention; PVD = peripheral vascular disease; TIA = transient ischemic attack.
covariates across the treatment groups were estimated, and findings were expressed as a percentage of pooled SDs were presented as Love plots (25).

**Statistical analysis.** Baseline characteristics of matched patients were compared with McNemar, paired t, and Wilcoxon rank sum tests, as appropriate. Cox proportional hazards models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs), comparing primary and secondary outcomes in the regadenoson group with those in the adenosine group. A conditional Cox model was used to estimate the HR for the propensity score-matched pairs. In consideration of the low number of events, a separate multivariable-adjusted Cox regression model adjusting for the 36 covariates was not attempted. Instead, we repeated our initial analysis in the 2,000 pre-match participants, adjusting for propensity scores. Event-free survival curves were constructed with the Kaplan–Meier method, and differences were estimated by the log-rank test for the pre-match groups. All tests were 2-tailed, and a p value <0.05 was considered statistically significant. All statistical analyses were carried out with PASW version 18 (SPSS, Inc., Chicago, Illinois).

**RESULTS**

**Baseline characteristics.** Overall, patients (n = 2,000) had an age of 59 ± 12 years; 55% were women, and 35% were African Americans. Imbalances in baseline characteristics before matching and balances achieved after matching between the 2 study groups are displayed in Table 1 and Figure 1. Before matching, patients in the regadenoson group had a higher proportion of men (47% vs. 43%, p = 0.04) and more tests ordered for pre-transplant evaluations (41% vs. 19%, p < 0.001) than patients in the adenosine group. The regadenoson group had fewer patients with prior coronary interventions (14% vs. 17%, p = 0.06), hyperlipidemia (45% vs. 51%, p = 0.03), and history of prior MI (5% vs. 9%, p < 0.001) than the adenosine group. Medication use was similar between the groups, except that more patients in the regadenoson group were receiving calcium channel blockers (31% vs. 26%, p = 0.02) and potassium sparing diuretics (11% vs. 7%, p = 0.002) (Online Table 1). After matching, only transplant evaluation as an indication for MPI maintained an imbalance, occurring more often in the regadenoson group (5% vs. 2%, p = 0.07).

**Primary outcomes.** Among the 2,000 pre-match patients, the primary endpoint (cardiac death, MI, and coronary revascularization) occurred in 21 (2.1%; 1.1%/year) and 33 (3.3%; 1.7%/year) patients in the regadenoson and adenosine groups, respectively (HR for regadenoson: 0.62; 95% CI: 0.36 to 1.08; p = 0.09) (Table 2), during a mean follow-up of 22 ± 5 months (23 ± 4 months for regadenoson; 22 ± 5 months for adenosine). After adjustment for propensity score in the entire cohort, there was no significant difference associated with stress agent used (propensity-adjusted HR: 0.63; 95% CI: 0.32 to 1.22; p = 0.169). Kaplan–Meier analysis with MACE as the endpoint was not statistically different between the groups (log-rank p = 0.087) (Fig. 2). In the propensity score-matched pairs, MACE occurred in 7 (1.4%) and 13 (2.6%) patients in the regadenoson and adenosine groups, respectively (matched HR for regadenoson vs. adenosine: 0.58; 95% CI: 0.23 to 1.48; p = 0.257) (Table 3). This association remained unchanged after additional adjustment for transplantation as an indication for MPI, because this was a remaining unbalanced covariate in the matched sample (transplant indication-adjusted HR: 0.55; 95% CI: 0.22 to 1.38; p = 0.201).

**Other outcomes.** Overall, 236 patients died during 2 years of follow-up after index MPI. Common causes of noncardiac death were cancer and sepsis. Among the 2,000 pre-match patients, cardiac death occurred in 18 (1.8%; 0.9%/year) and 23 (2.3%; 1.15%/year) patients in the regadenoson and adenosine groups, respectively (HR: 0.77; 95% CI: 0.42 to 1.43; p = 0.404) (Table 2). These associations remained unchanged after adjustment for propensity score (propensity-adjusted HR: 0.88; 95% CI: 0.42 to 1.86; p = 0.736).

Among the 2,000 pre-match patients, a combination of cardiac death and nonfatal MI occurred in 18 (1.8%; 0.9%/year) and 25 (2.5%; 1.25%/year) patients in the regadenoson and adenosine groups, respectively (HR: 0.71; 95% CI: 0.39 to 1.30; p = 0.261). These associations remained unchanged after adjustment for propensity score (propensity-adjusted HR: 0.80; 95% CI: 0.38 to 1.66; p = 0.547).

In the propensity score-matched pairs, cardiac death occurred in 5 (1.0%) and 7 (1.4%) matched patients in the regadenoson and adenosine groups, respectively (matched HR: 0.83; 95% CI: 0.25 to 2.73; p = 0.763) (Table 3). These associations remained unchanged after additional adjustment for transplantation as an indication for MPI (transplant indication-adjusted HR: 0.72; 95% CI: 0.23 to 2.28; p = 0.576).
After 2 years of follow-up from the index MPI in the 2,000 pre-match patients, there were 106 deaths (5.3%/year) in the regadenoson group and 130 (6.5%/year) in the adenosine group (p = 0.091). After propensity score matching, there were 52 all-cause deaths (5.15%/year) in the regadenoson

### Table 1. Baseline Characteristics by Stress Agent Before and After Propensity Score Matching

<table>
<thead>
<tr>
<th></th>
<th>Before Propensity Score Matching</th>
<th>After Propensity Score Matching</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Adenosine (n = 1,000)</td>
<td>Regadenoson (n = 1,000)</td>
</tr>
<tr>
<td>Age</td>
<td>60 ± 12</td>
<td>59 ± 12</td>
</tr>
<tr>
<td>Male</td>
<td>426 (43)</td>
<td>474 (47)</td>
</tr>
<tr>
<td>Race</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>646 (65)</td>
<td>624 (62)</td>
</tr>
<tr>
<td>Black</td>
<td>339 (34)</td>
<td>357 (36)</td>
</tr>
<tr>
<td>Other</td>
<td>15 (2)</td>
<td>19 (2)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>786 (79)</td>
<td>773 (77)</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>505 (51)</td>
<td>454 (45)</td>
</tr>
<tr>
<td>DM</td>
<td>378 (38)</td>
<td>358 (36)</td>
</tr>
<tr>
<td>CKD</td>
<td>288 (29)</td>
<td>245 (25)</td>
</tr>
<tr>
<td>CHF</td>
<td>72 (7)</td>
<td>62 (6)</td>
</tr>
<tr>
<td>PVD/CVA/TIA</td>
<td>162 (16)</td>
<td>180 (18)</td>
</tr>
<tr>
<td>CAD</td>
<td>239 (24)</td>
<td>242 (24)</td>
</tr>
<tr>
<td>Prior MI</td>
<td>88 (9)</td>
<td>45 (5)</td>
</tr>
<tr>
<td>Prior PCI</td>
<td>134 (13)</td>
<td>97 (10)</td>
</tr>
<tr>
<td>Prior CABG</td>
<td>66 (7)</td>
<td>65 (7)</td>
</tr>
<tr>
<td>FRS</td>
<td>11 ± 4</td>
<td>11 ± 4</td>
</tr>
<tr>
<td>Former smoker</td>
<td>260 (26)</td>
<td>278 (28)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>177 (18)</td>
<td>185 (19)</td>
</tr>
<tr>
<td>Inpatient testing</td>
<td>386 (39)</td>
<td>43 (4)</td>
</tr>
</tbody>
</table>

### Resting parameters

- **Resting heart rate**: 72 ± 13, 72 ± 13 (0.9) 72 ± 13, 72 ± 14 (0.9)
- **Resting systolic BP**: 132 ± 21, 132 ± 22 (0.8) 131 ± 20, 130 ± 22 (0.6)
- **Resting diastolic BP**: 74 ± 11, 75 ± 11 (0.002) 75 ± 10, 75 ± 11 (0.9)
- **Heart rate response**: 29 ± 21, 32 ± 20 (0.001) 33 ± 22, 32 ± 19 (0.2)
- **LV ejection fraction**: 67 ± 10, 67 ± 9 (0.3) 68 ± 9, 68 ± 9 (0.6)
- **Serum creatinine**: 2.46 ± 3.00, 2.83 ± 3.31 (0.009) 1.55 ± 1.97, 1.71 ± 2.30 (0.2)
- **Estimated GFR**: 59 ± 34, 58 ± 39 (0.6) 70 ± 28, 71 ± 35 (0.9)

### Indication for testing

- **Chest pain/dyspnea**: 518 (52), 405 (41) <0.0001 330 (65), 348 (69) (0.5)
- **Transplant evaluation**: 186 (19), 408 (41) <0.0001 24 (5), 12 (2) (0.07)
- **Pre-operative**: 39 (4), 47 (5) 0.4 29 (6), 37 (7) (0.4)
- **Other**: 257 (26), 140 (14) <0.0001 122 (22), 108 (21) (0.4)

### Table 2. Association Between Stress Agent Used and Outcomes Among the 2,000 Patients

<table>
<thead>
<tr>
<th>Events, % (n)</th>
<th>Regadenoson (n = 1,000)</th>
<th>Adenosine (n = 1,000)</th>
<th>Absolute Difference, %</th>
<th>Unadjusted HR (95% CI)</th>
<th>Propensity-Adjusted HR* (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MACE</td>
<td>2.1 (21)</td>
<td>3.3 (33)</td>
<td>−1.2</td>
<td>0.62 (0.36–1.08); p = 0.090</td>
<td>0.63 (0.32–1.22); p = 0.169</td>
</tr>
<tr>
<td>Cardiac mortality</td>
<td>1.8 (18)</td>
<td>2.3 (23)</td>
<td>−0.5</td>
<td>0.77 (0.42–1.43); p = 0.404</td>
<td>0.88 (0.42–1.86); p = 0.736</td>
</tr>
<tr>
<td>All-cause mortality</td>
<td>10.6 (106)</td>
<td>13 (130)</td>
<td>−2.4</td>
<td>0.80 (0.62–1.04); p = 0.091</td>
<td>0.73 (0.53–1.00); p = 0.048</td>
</tr>
</tbody>
</table>

*Additional adjustment for propensity score.

CI = confidence interval; HR = hazard ratio; MACE = major adverse cardiac events.
group and 64 (6.35%/year) in the adenosine group (p = 0.122) (Table 3).

**Side effects.** Side effects occurred significantly more frequently in the adenosine group at the time of testing. Chest pain, dyspnea, and bronchospasm were the most common adverse effects (Online Table 2).

**DISCUSSION**

The findings of the current study demonstrate that patients with normal regadenoson MPI (normal perfusion and LVEF) have low incident cardiovascular events during a 2-year follow-up that did not exceed those encountered with adenosine. There is no statistically significant difference in these events in a cohort of propensity-matched patients. To the best of our knowledge, this is the first study of the prognostic value of normal regadenoson MPI. These findings are important, because they suggest that a normal regadenoson MPI is as effective as a normal adenosine MPI in identifying patients at low risk of MACE (defined here as cardiac death, nonfatal MI, and coronary revascularization).

The unadjusted near-significant lower MACE in the regadenoson group is likely due to confounding by selection bias. Patients in the regadenoson group were younger and had fewer cardiovascular risk factors such as hyperlipidemia, renal insufficiency, and prior MI. These patients were less likely to be referred due to chest pain and more likely to be referred for pre-transplant evaluation. However, when adjusted for imbalances in baseline characteristics, in a propensity-matched cohort as well as adjusting for propensity scores in the original cohort, there was no significant difference in the primary endpoint, suggesting no independent association between regadenoson and cardiac outcomes. Importantly, there was no significant difference in cardiac mortality between the 2 groups. It should be emphasized that, before matching, the events rates were lower and not higher in the regadenoson group. This is an important observation, because our concern was that the bolus method of injection might conceal true abnormalities and hence increase the rate of false negative scans. The fact that with or without matching, the event rates were not higher in the regadenoson group is therefore quite reassuring.

Regadenoson is a selective adenosine A2A receptor agonist and has much lower affinity for non-A2A adenosine receptor subtypes, which are believed to be associated with adverse effects attributed to nonselective agonists such as adenosine. Coronary vasodilation is a physiological response that has a very large A2A receptor reserve (26). Regadenoson is given intravenously as a bolus at a dose of 0.4 mg with no weight adjustment, simplifying administration and mitigating errors in dose calculations. Regadenoson induces peak hyperemia that starts within 30 s of injection and lasts 2 to 3 min (27–29). The A2A receptor prevalence in the heart, its importance to coronary vasodilation, and the rapid onset and peak of action of regadenoson allow it to be given as a bolus but still provide adequate vasodilation for stress MPI.

Since its approval by the FDA in 2008, regadenoson has become the most commonly used vasodilator stress agent with MPI. In the United States,
as of February 2011, regadenoson is used in 68%, adenosine in 15%, and dipyridamole in 13% of vasodilator MPI (the remaining studies use dobutamine) (29). The FDA approval of regadenoson was based on results of the ADVANCE MPI (Adenosine versus Regadenoson Comparative Evaluation for Myocardial Perfusion Imaging) trials (15,16,30), which demonstrated no clinical differences in efficacy or safety between adenosine and regadenoson in a wide spectrum of patients (30). The concordance between regadenoson and adenosine was better by automated (polar maps) than by visual analysis (number of reversible defects) (16). The ADVANCE MPI trials did not provide prognostic data on the regadenoson MPI, although there is a large body of evidence in support of the prognostic usefulness of adenosine MPI (6–8,31,32).

Adenosine MPI has been well-accepted in risk stratification of many patient groups, and therefore it is imperative that any new stress agent not only have similar diagnostic accuracy but also confers the same prognostic information. In the current era of healthcare reform and cost-saving measures, appropriate application of testing and post-test management requires knowledge of the value of a test after consideration of the other clinical and historical factors that are known to the physician at the time of testing (8,33). With this approach, the statistical and clinical incremental value for adenosine single-photon emission computed tomography MPI has been demonstrated (4,8,33,34). The current study extends these findings to regadenoson MPI as shown by several models used in our analysis in which the results were independent of stress agent.

Prior studies suggest that patients undergoing vasodilator MPI are, as a group, at a higher risk for future MACE than those undergoing exercise MPI and that the event rate in patients with a normal adenosine MPI is higher than patients with normal exercise MPI, reflecting the differences in baseline risk (5). The outcome of patients who received adenosine in our study is consistent with previously reported data (1,6). The use of all-cause mortality in some of the earlier studies might explain the higher-than-expected event rates in patients with normal adenosine images (3,5,32). Regadenoson was associated with fewer adverse effects, compared with adenosine in our population and is, in general, better-tolerated by patients (12,13,15,30).

**Study limitations.** The results were obtained in a single tertiary care academic institution and might not be generalizable, and the findings need to be replicated in other patient populations. The study was not randomized. However, we used propensity scores to assemble a cohort in which the 2 groups were balanced on most key baseline confounders. Despite the large sample size, these results should be considered exploratory in nature. Finally, we did not examine the prognostic value of abnormal MPI, which needs to be addressed in future studies.

**Conclusions**

The 2-year incidence of MACE is low in patients with normal regadenoson MPI and does not exceed that seen in patients with normal adenosine MPI.

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**References**


Key Words: myocardial perfusion imaging • prognosis • regadenoson • single photon emission computed tomography • stress testing • vasodilator.

Appendix
For supplementary tables, please see the online version of this article.