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Importance of Measuring Glycosylated Hemoglobin in Patients with Myocardial Infarction and Known Diabetes Mellitus

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Abstract

Although medical comorbidities commonly affect clinical outcomes after acute myocardial infarction (AMI), current performance measures of AMI quality focus exclusively on management of the AMI itself. However, AMI patients frequently present with other comorbidities such as diabetes mellitus (DM) that also warrant assessment and management. To date, the quality of DM evaluation among patients presenting with an AMI has not been described. Between 1/2003–6/2004, the PREMIER-QI registry enrolled 3953 AMI patients at 19 U.S. centers. Frequency of glycosylated hemoglobin (A1C) assessment, either during the hospitalization or documented in the chart from the preceding 3 months, was prospectively evaluated. Among 1168 AMI patients with pre-existing DM, only 47% had recent A1C levels available, with marked variability in A1C assessment between hospitals (range 7%–81%). Among those with available A1C, 39% had good control (A1C <7), 36% suboptimal control (A1C 7–9), and 25% poor control (A1C >9). Patients with suboptimal and poor control were more likely to have their DM treatment intensified than those without A1C assessment (RR 1.38 [CI 1.03–1.85] for A1C 7–9; RR 2.20 [CI 1.68–2.88] for A1C >9). Similarly, patients with DM who had A1C measured were more likely to receive instructions on DM disease management prior to discharge. In conclusion, assessment of chronic glycemic control is highly variable among AMI patients with DM. Since much of this variability occurs at the hospital level, evaluation of DM control could represent an additional quality indicator and an opportunity to advance patient-centered AMI care.

Keywords

Myocardial infarction; diabetes mellitus; glycosylated hemoglobin

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Although the prognostic and therapeutic implications of diabetes mellitus (DM) at the time of acute myocardial infarction (AMI) have been well recognized,¹⁻⁵ the assessment of DM control at the time of AMI has generated little attention. Furthermore, while aggressive inpatient treatment of hyperglycemia during AMI hospitalization continues to be controversial,^{6,7} the benefits of chronic glycemic control on the microvascular complications of DM are well established.^{8,9} Given the increasing focus on managing multiple coexisting illnesses affecting cardiovascular patients,¹⁰ assessment of glycosylated hemoglobin (A1C) in AMI patients with DM could be an important opportunity to improve care for this important subset of patients. In addition, the relationship between A1C assessment and medication titration among patients with inadequate DM control remains unclear. To address these issues, we used a multicenter registry to a) examine the frequency of A1C assessment among AMI patients with DM, b) describe patterns of glycemic control in these individuals, and c) assess the clinical response to elevated A1C levels.

Methods

PREMIER-QI (Prospective Registry Evaluating Myocardial Infarction patients: Events and Recovery-Quality Improvement) is an observational registry of consecutive patients hospitalized for AMI, in which detailed clinical data were abstracted on 3953 patients between January 1, 2003 and June 28, 2004 at 19 U.S. centers.¹¹ Criteria for inclusion were age ≥ 18 years, prolonged (>20 minutes) signs or symptoms of myocardial ischemia or ischemic electrocardiographic changes, and biochemical evidence of myocardial necrosis. Patients transferred from other institutions were included only if they were transferred within 24 hours of symptom onset, and patients who developed elevated cardiac enzymes as a complication of elective coronary revascularization were not included. No identifying patient information was collected for this registry. Institutional Research Board approval was obtained at each participating center.

For the purposes of the current study, only patients with an established diagnosis of diabetes at hospital admission were included. Both Type I and Type II DM were included because the recommendations for regular monitoring of A1C are the same for both etiologies.⁸ To avoid confounding associated with small sample sizes, centers with <10 diabetic patients were excluded from the analyses. The primary outcomes were A1C assessment and the introduction or intensification of DM therapy by hospital discharge.

Patients were classified as having A1C assessment if A1C was measured during the hospitalization or if the chart documented A1C results within 3 months prior to admission. These data were prospectively collected throughout the study. To define the treating physician's response to a clinically-available measurement of A1C, admitting and discharge medications were compared and treatment was categorized as increased, unchanged, or decreased. Patients were considered to have increased DM treatment if the doses of oral antihyperglycemic agents were intensified, if a new antihyperglycemic agent was added to the medical regimen, if chronic insulin therapy was initiated, or if the cumulative daily insulin dose was increased by $\geq 20\%$. The DM regimen was considered to be unchanged if no changes were made in antihyperglycemic therapy, if one oral medication was changed to a different oral medication, or if the insulin formulation was changed (e.g. NPH to glargine) and the total daily units of insulin varied by $<20\%$. DM therapy was considered to be decreased if oral agents remained the same but the dose was decreased, if an insulin regimen was changed to oral agents alone, if oral agents or insulin were stopped and not restarted by hospital discharge, or if the total daily insulin dose was lowered by $>20\%$.

Patients with DM were dichotomized into those whose glycemic control was assessed (i.e., A1C level available) and those who did not have a recent A1C reported. We then compared

patient demographics, clinical characteristics, admission glucose concentrations, cardiac treatments, and diabetic discharge instructions of patients who had A1C assessed and those who did not. Between-group differences were assessed with chi-square tests for categorical variables and *t*-tests for continuous variables.

Variation in A1C assessment across hospitals was examined both by crude rates and via estimated random effects from a hierarchical regression model. To better quantify this variation, we calculated the median rate ratio (MRR) after adjusting for patient characteristics. The MRR describes the likelihood that a single patient, if presenting to 2 random hospitals in our study, would have had an A1C assessment at one of the hospitals as compared with the other. The MRR was adjusted for individual patient demographics (age, gender, race, body-mass index, insurance status), medical history (hypertension, dyslipidemia, prior coronary disease, heart failure, prior stroke, renal failure), and clinical status at presentation (ST- or non-ST-elevation AMI, left ventricular systolic function, admission glucose).

To evaluate the consequences of A1C assessment on subsequent DM management, we examined the change in medication regimen and the provision of discharge instructions between those who did and did not have A1C assessed. Among patients who had recent assessment, A1C levels were categorized as good control (<7%, the American Diabetes Association recommendation at the time of this study),¹² suboptimal control (A1C 7–9%), or poor control (A1C >9%). Rate ratios (versus the reference group of patients without A1C assessed) were estimated using modified Poisson regression models adjusting for hospital.

Missing values were present in <1% of the data; these were imputed using expectation-maximization methods (which estimate conditional means for missing values given observed values of the other covariates) to allow all patients to be retained in the analysis. *P*-values <0.05 were considered statistically significant. All analyses were performed using SAS version 9.1 (SAS Institute, Inc., Cary, NC) and R version 2.7.2 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Of 3953 patients enrolled in PREMIER-QI, 1201 (30.4%) had known DM at admission. After excluding 5 centers with low enrollment of patients with DM (*n*=33 patients), the final analytic cohort consisted of 1168 patients from 14 enrolling hospitals (range 22 to 172 from each hospital). Mean age was 64 ± 13 years, 702 (60.1%) were male, and 716 (61.7%) were Caucasian. Only 547 patients with DM (46.8%) had a recent A1C level available to treating physicians during their AMI hospitalization.

Patient characteristics, stratified by whether A1C was assessed, are shown in Table 1. Individuals undergoing A1C assessment were slightly younger (mean age 63 vs 65 years, *p*<0.001) and less likely to be Caucasian (56% vs 67%, *p*<0.001), but otherwise there were no significant differences in gender, health insurance, body mass index, cardiovascular history, or clinical presentation of AMI. Patients who had A1C measured had higher admission glucose levels than those who were not assessed (225 ± 126 mg/dL vs 207 ± 114 mg/dL, *p*=0.012). The medical therapy for AMI and multidisciplinary discharge planning are listed in Table 2. Quality of AMI care, as assessed by the appropriate use of evidence-based medical therapies for AMI, did not differ between those patients with vs without A1C assessment. However, intensification of DM medication was more likely when a current A1C level was available (33% vs 26%, *p*=0.018). Nonpharmacologic interventions such as dietary counseling (78% vs 63%, *p*<0.001) and disease-management instructions for DM

(41% vs 29%, $p<0.001$) were also more likely to be provided to patients with known A1C levels.

There was substantial variability in A1C assessment between the enrolling sites in PREMIER-QI, ranging from 7% to 73%. Frequency of A1C assessment across hospitals is illustrated in the Figure. Unadjusted MRR was 1.60 (95% CI 1.35–2.17), which remained unchanged after multivariable adjustment (MRR 1.60, 95% CI 1.34–2.19). This suggests that the typical relative difference in A1C assessment between hospitals is 60%, which represents a large amount of hospital-level variation when managing AMI patients with DM.

Among the patients with A1C assessment, only 39% were found to have good glycemic control, while 36% had suboptimal control (A1C 7–9) and 25% had poor control (A1C >9). When proportions of patients with poor control were examined across treatment centers in PREMIER-QI, little variability was found (median 28% of diabetic patients with known A1C level, range 17%–33%). Treatment intensification for DM—both pharmacologic and nonpharmacologic—is shown in Table 3. Patients with suboptimal and poor control were significantly more likely to have their DM medication intensified than those not assessed (RR 1.38 [CI 1.03–1.85] for suboptimal control; RR 2.20 [CI 1.68–2.88] for poor control; $p<0.001$ for both comparisons). Similarly, patients with poor long-term DM control (A1C >9) were more likely to receive DM management instructions at discharge.

Discussion

Despite widespread recognition of the importance of long-term glycemic control in preventing the microvascular complications of DM, we found that fewer than half of patients with DM had their A1C value known or measured at the time of their AMI hospitalization. Hospital site was strongly predictive of A1C assessment, even after adjusting for a multitude of patient characteristics in the detailed PREMIER-QI registry. We also observed marked variability in the frequency of A1C assessment across hospitals (range 7%–81%). Among those who had their A1C assessed, only 38% had good glycemic control (A1C <7%), indicating a potential opportunity to optimize the care of >60% of AMI patients with diabetes. In addition, availability of A1C was associated with higher rates of adjustments in patients' DM medication regimens, and with higher rates of disease management instructions at hospital discharge. To our knowledge, this is the first study to document the prevalence, variability, and opportunity to improve DM care at the time of AMI hospitalization.

Poor chronic glycemic control is an important risk factor for adverse outcomes in patients with DM.¹³ This is a completely distinct aspect of care than acute hyperglycemia at the time of an AMI, for which prognosis is worsened^{14–16} but evidence supporting tight inpatient glucose control remains controversial.¹⁷ In contrast, epidemiologic studies suggest that an increase in A1C by 1% is associated with a 20% increased risk in subsequent mortality for patients with coronary disease and concurrent DM.^{13,18} Despite recent data questioning the benefits of intensive glycemic management for preventing cardiovascular disease,^{19,20} better long-term glycemic control consistently prevents the progression of microvascular complications in multiple studies—an important outcome with major implications for patients with DM.^{20–22} Since A1C assessment was significantly associated with pharmacologic and nonpharmacologic intensification of DM therapies, a sizable proportion of diabetic AMI patients with suboptimal and poor glycemic control would likely benefit from A1C measurement and subsequent modification of DM therapy prior to hospital discharge. In addition, other disease management instructions including exercise and weight counseling were documented at remarkably low rates during the AMI hospitalization, and were not related to the assessment of A1C. While low, these recommendations are for *all*

patients recovering from an AMI (regardless of DM management strategy), and it is therefore not surprising that the rates were similar between those with and without an A1C assessment. As such, A1C assessment (and possibly other nonpharmacologic measures of DM care) could serve as future quality indicators of DM management at the time of AMI—particularly given the recent focus on the multiple comorbidities affecting AMI patients.¹⁰ This approach would allow the AMI hospitalization to become an opportunity to assess overall health status, including an assessment of risk factors that underlie the AMI itself, in order to provide individualized care for both the AMI and for other comorbid medical conditions.

While some may argue that addressing glycemic control during AMI hospitalization is unnecessary, and could be deferred to the outpatient setting, several studies have demonstrated improved medication adherence when therapies are prescribed at discharge.^{23–26} For diabetic patients, the American Diabetes Association recommends that A1C be assessed (if not drawn in the previous 2–3 months) in all patients with DM admitted to the hospital in order to facilitate discharge planning and to improve the quality of their DM care.⁸ Our study clearly documents that this is not the current standard of care at many hospitals, since fewer than half of the patients in PREMIER underwent assessment of A1C. Adopting this practice as a quality indicator could potentially improve the transition of DM management from hospitalization to the outpatient setting. As with other risk factors, this approach would allow for the adoption of multifaceted care, beyond treating just coronary stenoses, and would emphasize individualized, patient-centered management strategies designed to reduce the complications of chronic comorbidities such as DM. In addition, while other performance measures for AMI (e.g. aspirin, beta blocker use) have been widely adopted by most hospitals, A1C assessment and subsequent DM management could emerge as measures for which even the “highest performers” have significant room for improvement. Finding such a large opportunity to improve the current practice of DM management at the time of AMI suggests that relatively simple, system-level interventions (e.g. AMI care paths that include routine A1C assessment) may improve the care given to patients’ DM and can lay the foundation for overcoming clinical inertia.²⁷

The results of our study should be considered in the context of the following potential limitations. First, only patients with known DM prior to admission were included, and the analysis did not include patients with pre-diabetic states or with newly-diagnosed DM. Prior studies have shown that 14–31% of non-diabetic patients with acute coronary syndromes are ultimately diagnosed with DM,^{28,29} and therefore our study may have underestimated the proportion of individuals who could benefit from inpatient A1C assessment. In addition, opportunities to improve glycemic control were only identified among patients with DM who had their A1C levels recorded. We were unable to estimate the proportion of patients whose A1C was not assessed who may have been eligible for more intensive DM care. Finally, we did not have access to follow-up A1C levels, and we do not know whether outpatient follow-up specifically addressed DM therapy in these patients. As a result, future outpatient assessment and titration of DM medications after hospital discharge may have occurred, and the ultimate effect of A1C assessment on long-term glycemic control is unknown.

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References

1. Haffner SM, Lehto S, Ronnema T, Pyorala K, Laakso M. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. *N Engl J Med* 1998;339:229–234. [PubMed: 9673301]
2. Morocutti G. The BARI Investigators. Bypass treatment is better than angioplasty in reducing long-term mortality in diabetic patients with multivessel coronary disease. *Ital Heart J Suppl* 2000;1:951–952. [PubMed: 10935745]
3. Schwartz L, Kip KE, Frye RL, Alderman EL, Schaff HV, Detre KM. Coronary bypass graft patency in patients with diabetes in the Bypass Angioplasty Revascularization Investigation (BARI). *Circulation* 2002;106:2652–2658. [PubMed: 12438289]
4. Berger PB, Velianou JL, Aslanidou Vlachos H, Feit F, Jacobs AK, Faxon DP, Attubato M, Keller N, Stadius ML, Weiner BH, Williams DO, Detre KM. Results from the Bypass Angioplasty Revascularization Investigation (BARI). Survival following coronary angioplasty versus coronary artery bypass surgery in anatomic subsets in which coronary artery bypass surgery improves survival compared with medical therapy. *J Am Coll Cardiol* 2001;38:1440–1449. [PubMed: 11691521]
5. Donahoe SM, Stewart GC, McCabe CH, Mohanavelu S, Murphy SA, Cannon CP, Antman EM. Diabetes and mortality following acute coronary syndromes. *Jama* 2007;298:765–775. [PubMed: 17699010]
6. Malmberg K, Ryden L, Efendic S, Herlitz J, Nicol P, Waldenstrom A, Wedel H, Welin L. Randomized trial of insulin-glucose infusion followed by subcutaneous insulin treatment in diabetic patients with acute myocardial infarction (DIGAMI study): effects on mortality at 1 year. *J Am Coll Cardiol* 1995;26:57–65. [PubMed: 7797776]
7. Malmberg K, Ryden L, Wedel H, Birkeland K, Bootsma A, Dickstein K, Efendic S, Fisher M, Hamsten A, Herlitz J, Hildebrandt P, MacLeod K, Laakso M, Torp-Pedersen C, Waldenstrom A. Intense metabolic control by means of insulin in patients with diabetes mellitus and acute myocardial infarction (DIGAMI 2): effects on mortality and morbidity. *Eur Heart J* 2005;26:650–661. [PubMed: 15728645]
8. Standards of medical care in diabetes--2008. *Diabetes Care* 2008;31:S12–S54. [PubMed: 18165335]
9. Skyler JS, Bergenstal R, Bonow RO, Buse J, Deedwania P, Gale EA, Howard BV, Kirkman MS, Kosiborod M, Reaven P, Sherwin RS. Intensive Glycemic Control and the Prevention of Cardiovascular Events: Implications of the ACCORD, ADVANCE, and VA Diabetes Trials: A Position Statement of the American Diabetes Association and a Scientific Statement of the American College of Cardiology Foundation and the American Heart Association. *Circulation* 2009;119:351–357. [PubMed: 19095622]
10. Werner RM, Greenfield S, Fung C, Turner BJ. Measuring quality of care in patients with multiple clinical conditions: summary of a conference conducted by the Society of General Internal Medicine. *J Gen Intern Med* 2007;22:1206–1211. [PubMed: 17516106]
11. Spertus JA, Peterson E, Rumsfeld JS, Jones PG, Decker C, Krumholz H. The Prospective Registry Evaluating Myocardial Infarction: Events and Recovery (PREMIER)--evaluating the impact of myocardial infarction on patient outcomes. *Am Heart J* 2006;151:589–597. [PubMed: 16504619]
12. Standards of medical care in diabetes. *Diabetes Care* 2005;28:S4–S36. [PubMed: 15618112]
13. Gerstein HC. Glycosylated hemoglobin: finally ready for prime time as a cardiovascular risk factor. *Ann Intern Med* 2004;141:475–476. [PubMed: 15381522]
14. Capes SE, Hunt D, Malmberg K, Gerstein HC. Stress hyperglycaemia and increased risk of death after myocardial infarction in patients with and without diabetes: a systematic overview. *Lancet* 2000;355:773–778. [PubMed: 10711923]
15. Kosiborod M, Rathore SS, Inzucchi SE, Masoudi FA, Wang Y, Havranek EP, Krumholz HM. Admission glucose and mortality in elderly patients hospitalized with acute myocardial infarction: implications for patients with and without recognized diabetes. *Circulation* 2005;111:3078–3086. [PubMed: 15939812]
16. Stranders I, Diamant M, van Gelder RE, Spruijt HJ, Twisk JW, Heine RJ, Visser FC. Admission blood glucose level as risk indicator of death after myocardial infarction in patients with and without diabetes mellitus. *Arch Intern Med* 2004;164:982–988. [PubMed: 15136307]

17. van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, Vlasselaers D, Ferdinande P, Lauwers P, Bouillon R. Intensive insulin therapy in the critically ill patients. *N Engl J Med* 2001;345:1359–1367. [PubMed: 11794168]
18. Saydah SH, Fradkin J, Cowie CC. Poor control of risk factors for vascular disease among adults with previously diagnosed diabetes. *Jama* 2004;291:335–342. [PubMed: 14734596]
19. Gerstein HC, Miller ME, Byington RP, Goff DC Jr, Bigger JT, Buse JB, Cushman WC, Genuth S, Ismail-Beigi F, Grimm RH Jr, Probstfield JL, Simons-Morton DG, Friedewald WT. Effects of intensive glucose lowering in type 2 diabetes. *N Engl J Med* 2008;358:2545–2559. [PubMed: 18539917]
20. Patel A, MacMahon S, Chalmers J, Neal B, Billot L, Woodward M, Marre M, Cooper M, Glasziou P, Grobbee D, Hamet P, Harrap S, Heller S, Liu L, Mancia G, Mogensen CE, Pan C, Poulter N, Rodgers A, Williams B, Bompoint S, de Galan BE, Joshi R, Travert F. Intensive blood glucose control and vascular outcomes in patients with type 2 diabetes. *N Engl J Med* 2008;358:2560–2572. [PubMed: 18539916]
21. Nathan DM, Cleary PA, Backlund JY, Genuth SM, Lachin JM, Orchard TJ, Raskin P, Zinman B. Intensive diabetes treatment and cardiovascular disease in patients with type 1 diabetes. *N Engl J Med* 2005;353:2643–2653. [PubMed: 16371630]
22. UK Prospective Diabetes Study (UKPDS) Group. Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). *Lancet* 1998;352:854–865. [PubMed: 9742977]
23. Fonarow GC. In-hospital initiation of statin therapy in acute coronary syndromes: maximizing the early and long-term benefits. *Chest* 2005;128:3641–3651. [PubMed: 16304326]
24. Fonarow GC. Practical considerations of beta-blockade in the management of the post-myocardial infarction patient. *Am Heart J* 2005;149:984–993. [PubMed: 15976779]
25. Fonarow GC. When to initiate beta-blockers in heart failure: is it ever too early? *Curr Heart Fail Rep* 2005;2:94–99. [PubMed: 16036057]
26. Fonarow GC, Wright RS, Spencer FA, Fredrick PD, Dong W, Every N, French WJ. Effect of statin use within the first 24 hours of admission for acute myocardial infarction on early morbidity and mortality. *Am J Cardiol* 2005;96:611–616. [PubMed: 16125480]
27. Phillips LS, Branch WT, Cook CB, Doyle JP, El-Kebbi IM, Gallina DL, Miller CD, Ziemer DC, Barnes CS. Clinical inertia. *Ann Intern Med* 2001;135:825–834. [PubMed: 11694107]
28. Conaway DG, O'Keefe JH, Reid KJ, Spertus J. Frequency of undiagnosed diabetes mellitus in patients with acute coronary syndrome. *Am J Cardiol* 2005;96:363–365. [PubMed: 16054458]
29. Norhammar A, Tenerz A, Nilsson G, Hamsten A, Efendic S, Ryden L, Malmberg K. Glucose metabolism in patients with acute myocardial infarction and no previous diagnosis of diabetes mellitus: a prospective study. *Lancet* 2002;359:2140–2144. [PubMed: 12090978]

Figure 1. Variation of hemoglobin A_{1c} (A1C) assessment in diabetic patients with acute myocardial infarction across 14 institutions participating in the PREMIER-QI registry. Solid bars represent point estimates and 95% confidence intervals of estimated assessment rates of A1C.

Table 1

Baseline patient characteristics, stratified by assessment of A1C

Characteristics	A1C assessment		P-value
	No (n = 621)	Yes (n = 547)	
Age (yrs)	65 ± 13	63 ± 12	<0.001
Male	61%	59%	0.490
Caucasian	67%	56%	<0.001
Insurance for health care	93%	90%	0.115
Prior myocardial infarction	32%	29%	0.275
Prior percutaneous coronary intervention	23%	23%	0.939
Prior coronary bypass surgery	20%	20%	0.862
History of heart failure	26%	22%	0.073
History of hypertension	81%	82%	0.798
History of hypercholesterolemia	58%	56%	0.529
Peripheral arterial disease	16%	13%	0.199
Prior cerebrovascular event	14%	11%	0.166
Chronic lung disease	16%	15%	0.775
Smoking status			0.413
Current (<30 days)	21%	24%	
Former (≥30 days)	29%	28%	
Never (or <100 total cigarettes)	51%	48%	
Diabetes subtype			0.720
Type 1	56	41	
Type 2	511	463	
Unknown	54	43	
Clinical presentation			
ST-elevation myocardial infarction	32%	29%	0.262
Left ventricular systolic function			0.347
Normal	48%	50%	
Mildly reduced	19%	19%	
Moderately reduced	16%	19%	
Severely reduced	16%	13%	
Admitting glucose (mg/dL)	207 ± 114	225 ± 126	0.012

Data are expressed as percentages or mean ± standard deviation.

A1C = glycosylated hemoglobin.

Table 2

Medical therapies and discharge planning, stratified by assessment of A1C

Therapies	A1C assessment		P-value
	No (n = 621)	Yes (n = 547)	
Initial cardiac treatment			
Received aspirin at arrival *	94%	96%	0.062
Beta-blocker within 24 hrs *	88%	90%	0.392
Anti-platelet within 24 hrs	45%	42%	0.330
Anti-thrombin	78%	82%	0.060
Glycoprotein IIb/IIIa inhibitor	44%	48%	0.100
Reperfusion for ST-elevation myocardial infarction *	57%	50%	0.146
Discharge cardiac medications			
Aspirin	85%	87%	0.582
Beta-blocker	82%	82%	0.794
ACE inhibitor or ARB	66%	68%	0.333
Statin	71%	74%	0.175
Discharge planning			
Diabetes medication intensified prior to hospital discharge	26%	33%	0.018
Diabetes disease management instructions	29%	41%	<0.001
Exercise counseling	39%	43%	0.132
Dietary counseling	63%	78%	<0.001
Weight management instructions	3.7%	2.0%	0.086

* Among patients meeting appropriateness criteria defined by the Centers for Medicare & Medicaid Services (CMS) regarding quality-of-care measures for myocardial infarction.

A1C = glycosylated hemoglobin; ACE = angiotensin converting enzyme; ARB = angiotensin receptor blocker.

Table 3

Diabetes treatment intensification according to AIC level, compared to patients without AIC assessment

	No AIC assessment	AIC level			Trend P-value
		<7% (n = 214)	7–9% (n = 195)	>9% (n = 138)	
Medical therapy					
Diabetes medication increased	26%	19% RR 0.76 (0.52–1.10)	34% RR 1.38 (1.03–1.85)	54% RR 2.20 (1.68–2.88)	<0.001
Discharge instructions					
Diabetes management	29%	34% RR 1.14 (0.93–1.41)	39% RR 1.52 (1.24–1.86)	56% RR 1.82 (1.51–2.19)	<0.001
Exercise counseling	39%	42% RR 1.10 (0.94–1.29)	47% RR 1.09 (0.92–1.27)	41% RR 1.04 (0.85–1.26)	0.86
Dietary counseling	63%	77% RR 1.08 (0.99–1.19)	79% RR 1.10 (1.00–1.20)	78% RR 1.12 (1.01–1.24)	0.17
Weight management	3.7%	2.3% RR 0.59 (0.22–1.55)	2.1% RR 0.56 (0.18–1.74)	1.5% RR 0.35 (0.08–1.46)	0.62

AIC = glycosylated hemoglobin; RR = rate ratio (95% confidence interval).