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Effect of Hospital Admission on Glycemic Control One Year after Discharge

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Abstract

Objective—We assessed the effect of hospital admission on glycemic control in patients with diabetes up to 1 year after discharge.

Methods—We retrospectively studied 826 adults with diabetes admitted to a tertiary care medical center and with available hemoglobin A1c (A1C) values prior to admission and 1 year after discharge. We compared them to 826 age-, sex-, race-, co-morbidity- and baseline A1C-matched non-hospitalized adults with diabetes. We determined change in A1C relative to hospitalization and baseline A1C using multivariate random effects models for repeated measures. Logistic regression was used to determine predictors of achieving recommended A1C levels at 1 year.

Results—Patients with baseline A1C $\geq 9\%$ (adjusted mean A1C 9.90% [95% CI 9.25, 10.55]) had an adjusted change in A1C of -0.10% per month ([95% CI -0.18 , -0.022], $p < 0.01$) over the course of 1 year without differences between hospitalized and non-hospitalized patients in the mean rate of change. However, hospitalized patients were less likely to achieve an A1C $< 7\%$ at one year (OR 0.68 [95% CI 0.55, 0.86], $p < 0.01$) or A1C $< 8\%$ at one year (OR 0.62 [95% CI 0.48, 0.81], $p < 0.01$) compared to non-hospitalized patients.

Conclusions—Despite an overall trend towards improved glycemia over time, hospitalized patients with uncontrolled diabetes were less likely to achieve glycemic targets at 1 year compared to matched non-hospitalized patients. These results suggest a missed opportunity to improve long-term glycemic control in hospitalized patients with diabetes.

Keywords

hemoglobin A1c; hospitalization; glycemic control

Introduction

Long-term glycemic control reduces the risk of microvascular complications in type 1 and type 2 diabetes but can be difficult to achieve (1, 2). Given the intense daily exposure to

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clinical care teams, hospital admissions have been proposed as an opportunity to improve metabolic control, even for diabetic patients not admitted primarily for metabolic control (3). One-quarter of hospitalized patients have diabetes, with average A1C at the time of admission ranging from $7.4 \pm 1.7\%$ to $8.5 \pm 2.6\%$ in recently published studies (4–6). Most studies of inpatient interventions on glycemic control have focused on immediate outcomes, such as in-hospital morbidity/mortality and glycemic excursions, and not on its impact on subsequent outpatient glycemic control (7–9).

Given current advances in healthcare reform, with a new focus on medical homes and development of accountable care organizations (10), resource-intensive hospital admissions present an important opportunity both to address immediate medical problems and to improve the treatment of chronic conditions which, left uncontrolled, could lead to increased future resource utilization and cost. In addition, more clinical focus is being placed on improving transitions of care from the inpatient to outpatient setting to reduce the risk associated with hospital discharge itself (7, 11, 12). In this context, the impact of hospitalization on long-term glycemic control could be used as a measure of the effect of clinical interventions (e.g. medication initiation and titration, teaching) begun during hospitalizations (13).

To determine whether interventions in the inpatient setting improve long-term glycemic control, we need first to understand the natural course of glycemic changes in patients who have been hospitalized. Hemoglobin A1c (A1C) is the standard clinical measure and quality metric of long-term glycemic control. It reflects the average level of glycemia integrated over the 120-day lifecycle of the red blood cell, assuming a normal red blood cell aging process and hemoglobin profile (14). Hemoglobin A1c level measured approximately 3 months after hospitalization can serve as a measure of overall glucose control post-discharge, albeit with less reliability in the setting of possible blood loss and transfusions, among other factors, during a hospitalization that may make an A1C value difficult to interpret.

The effect of hospitalization on long-term glycemic control is poorly characterized. The disruption in routine and stress of treating conditions leading to hospitalization might transiently raise A1C levels, while inpatient management of glycemic control may lead to treatment intensification and subsequent improvement in A1C. Whether putative short-term effects of hospitalization on glycemic control are sustained after discharge is unknown. We sought to determine the impact of hospital admission on change in A1C and achievement of recommended target A1C levels over 1 year in an observational cohort of patients admitted to medical and surgical services compared to matched non-hospitalized diabetes patients. We hypothesized that hospitalization, with presumed non-standardized in-hospital intensification of glycemic control, would be associated with greater sustained improvement in A1C over 1 year than usual outpatient care.

Patients and Methods

Setting and Participants

The study was conducted at Massachusetts General Hospital, a 900-bed tertiary care academic medical center in Boston, Massachusetts. Using a previously described prospective diabetes cohort (N=12,233 in the years 2005–2007) that receives continuous primary care within the hospital system (15), we identified patients with type 2 diabetes admitted to the hospital between January 1, 2005 and December 31, 2007, not previously admitted within 12 months of the index admission, who had hemoglobin A1c data available within 6 months prior to admission and 12 months after discharge. One thousand four hundred and twenty-eight hospitalized patients fulfilled these eligibility requirements. After

excluding patients with diagnosis codes indicating chronic kidney disease, dialysis, gastrointestinal bleeding, cancer, or bone marrow disorders (to decrease spurious A1C results owing to abnormal red cell kinetics), we were left with 1,009 hospitalized patients with type 2 diabetes who fulfilled the eligibility criteria.

Using the same diabetes cohort, each case was matched to a non-hospitalized patient meeting all of the five following criteria: age group- (<35, 35–44, 45–54, 55–64, 65 years), gender-, race- (white, non-white), baseline A1C group- 7%, 7–9%, 9%), co-morbidity score-, and year of admission. We matched 859 non-hospitalized patients to serve as a control group. After excluding controls with missing baseline or one-year A1C results, there were 826 matched controls for 975 cases. To maintain a 1:1 case:control match, we excluded 149 cases where more than one case matched to the same control. The final analysis was performed with 826 cases and 826 matched controls. These patients were stratified by degree of baseline glycemic control into three groups: A1C 7%, 7–9%, and 9%.

Variables and Outcomes

All A1C data were obtained from MGH laboratory records; all measurements were performed with an HPLC method that is DCCT-aligned and serves as one of the primary reference methods for the National Glycohemoglobin Standardization Program (16). The assay has intra- and inter-assay coefficients of variation less than 2%.

Baseline A1C was defined as the A1C value within 6 months prior to the admission date, including the date of admission. Baseline A1C in the matched control group was defined as the first A1C in the matched year; the observation period for the control group was 12 months from the date of the first A1C. All hospitalized patients had at least three A1C measurements (one pre/peri-admission and two within 12 months of discharge). All non-hospitalized matched controls had at least two A1C measurements in 12 months; 675 had 3 A1C measurements. Target outcomes of 7% and <8% were chosen based on current American Diabetes Association (ADA) outcomes-based and National Committee for Quality Assurance (NCQA)-Healthcare Employer Information Data Set (HEDIS) practice quality guidelines (17, 18). Both targets were assessed given the ongoing debate regarding ideal degree of glycemic control for patients with multiple comorbidities.

Age, sex, race, discharge diagnosis, admitting service (medicine or surgery), length of stay (LOS), insurance status, and number of A1C tests in the year of interest were obtained from hospital administrative data. Cumulative score of the nine most significant cost-related co-morbid conditions including depression, diabetes, heart failure, stroke, hypertension, coronary artery disease, osteoarthritis, COPD or asthma, and atrial fibrillation (co-morbidity score) (19) was obtained from the diabetes cohort database. CHF and depression diagnoses were identified separately from the co-morbidity score because those conditions have specifically been shown to affect diabetes care (20, 21). Number of outpatient visits with and connectedness of the patient to a specific primary care physician (PCP) were obtained from the diabetes cohort database. PCP connectedness, a measure of how closely patients are followed by their PCPs, was determined by a previously validated algorithm (15). Race was dichotomized to white and non-white. Insurance was coded into three categories: Medicare, private insurance, and Medicaid/Free Care. Diabetes was considered to be the primary cause of admission if the primary discharge diagnosis was listed as diabetes (ICD9 codes 249, 250, 251). Data on duration of diabetes were not available. Approximately 5% of adult inpatients with diabetes receive an endocrine consult for diabetes at our institution; we were unable to exclude these patients. There were no ongoing standardized diabetes management or education programs during the study period.

Statistical Methods

We compared baseline unadjusted characteristics using t-tests for continuous variables, Fisher's exact tests for dichotomous variables and χ^2 tests for categorical variables.

Analysis of change in A1C was performed using A1C values relative to the baseline A1C. A random effects model with repeated measures was used to model change in A1C over time in months. We first modeled the rate of A1C change and the effects of hospitalization and baseline A1C group for the entire cohort, controlling for baseline A1C group (7%, 7–9%, 9%), CHF, depression, PCP-connectedness, and type of insurance. We then modeled each baseline A1C group separately and looked for additional effect modification of CHF, PCP-connectedness, and type of insurance on the rate of A1C change.

Logistic regression was used to model predictors for achieving A1C <7% at 1 year with hospitalization as the independent variable and baseline A1C group (7%, 9% vs. 7–9% referent group), CHF, depression, type of health insurance, and PCP-connectedness as covariates. A second logistic regression was used to model predictors for achieving goal A1C <8% with the same independent variable and covariates. All analyses were performed with SAS version 9.1.3. The protocol was approved by the Partners Healthcare Institutional Review Board.

Results

Participants

The unadjusted demographic, health, and healthcare characteristics of the 826 patients with type 2 diabetes admitted to the hospital and 826 matched non-hospitalized patients are shown in Table 1. The groups were well-matched on most variables except that the hospitalized patients had significantly more outpatient visits in the 12 months after discharge compared to the non-hospitalized patients in the 12 months following baseline A1C, and slightly, albeit significantly, fewer A1C results in the same time period (2.8 vs. 3.0, $p<0.01$). Of the hospitalized patients, the mean ($\pm$ SD) length of stay was 4.2 ± 4.2 days, with median stay of 3 days. Sixty-seven percent ($N=557$) were admitted to the medicine service and 5.3% ($N=44$) were admissions primarily for uncontrolled diabetes.

Duration between baseline A1C and first follow-up A1C measurement were similar between hospitalized and non-hospitalized patients. Hospitalized patients had longer overall follow-up with mean duration between baseline and last follow-up A1C (mean months $\pm$ SD), 11.7 ± 2.4 compared with 9.4 ± 1.8 in the matched controls. Among hospitalized patients, the first follow-up A1C was 2.5 ± 1.8 months after hospital discharge and the last follow-up A1C was 9.4 ± 2.0 months after hospital discharge.

Rate of Change in A1C over 1 year

In the random effects model with all A1C data points from 1652 patients, the adjusted rate of A1C change was $-0.013\%/month$ ([95% CI $-0.023, -0.002$], $p=0.016$) (Table 2). The rate of A1C change was significantly different between patients with baseline A1C <7%, 7–9%, and $\geq 9\%$, after adjusting for baseline A1C, CHF, depression, type of insurance, and PCP-connectedness.

Analysis of A1C results from patients with baseline A1C <7% ($N=368$ controls, 361 cases) found a non-significant rise in A1C change of $0.014\%/month$ ([95% CI $-0.004, 0.03$], $p=0.12$), after controlling for CHF, depression, type of insurance, and connectedness to PCP. Hospitalization, CHF, depression, type of insurance and PCP-connectedness did not significantly alter the rate of A1C change.

Patients with baseline A1C 7–9% (N=331 controls, 330 cases) had a non-significant A1C rate change of $-0.019\%/month$ ([95% CI $-0.039, 0.0013$], $p=0.067$), after adjusting for CHF, depression, insurance, and loyalty to PCP. Although the overall rate of A1C change in the group with baseline A1C 7–9% did not meet significance, the diagnosis of CHF had a significantly negative impact on the rate of change in A1C (interaction term $p=0.002$). Hospital admission, depression, type of insurance, and PCP-connectedness were not found to significantly interact with the rate of A1C change.

Patients with poorly controlled diabetes at baseline (A1C $\geq 9\%$, N=127 controls, 135 cases) had a significant A1C rate change of $-0.10\%/month$ ([95% CI $-0.18, -0.022$], $p=0.012$), after adjusting for CHF, depression, type of insurance and loyalty to PCP. Hospitalization, CHF, and depression did not significantly alter the rate of A1C change. Higher patient-PCP connectedness was associated with a steeper decline in A1C in this poorly controlled subgroup ($p=0.023$).

Hospitalization and achievement of target A1C at 1 year

Hospitalization (OR 0.68 [95% CI 0.55, 0.86], $p<0.01$) and baseline A1C $\geq 9\%$ (relative to A1C 7–9%; OR 0.45 [0.32, 0.63], $p<0.01$) significantly decreased the likelihood of achieving target A1C $\leq 7\%$ at 1 year controlling for CHF, depression, type of insurance, and connectedness to PCP (Figure 1) compared to matched non-hospitalized patients. Similarly, hospitalized patients (OR 0.62 [95% CI 0.48, 0.81], $p<0.01$), baseline A1C $\geq 9\%$ (OR 0.25 [95% CI 0.18, 0.34], $p<0.01$), and patients with depression (OR 0.71 [95% CI 0.52, 0.96], $p=0.027$) were less likely to achieve a more conservative target A1C of $<8\%$ at 1 year.

Discussion

This is the first observational study to look at glycemic control after hospitalization in patients with diabetes. Although there was no significant difference in overall mean A1C change for hospitalized compared to matched non-hospitalized patients, hospitalization was associated with decreased odds of achieving target A1C $\leq 7\%$ and A1C $<8\%$. These results disproved our initial hypothesis that hospitalization would be associated with greater A1C improvement over 1 year than usual outpatient care and suggest that hospitalization and immediate post-hospitalization care represent an unrealized opportunity to improve long-term glycemic control in patients who are likely to be at high risk by virtue of their hospital admission.

Although there was significant decline in A1C at 1 year in patients with baseline A1C $\geq 9\%$, the majority (63.2% in hospitalized patients, 65.9% in non-hospitalized patients) failed to achieve an A1C $<8\%$ at 1 year ($p=0.13$). The decline in A1C at 1 year in patients with A1C $\geq 9\%$ was similar to the findings in an observational study of French patients with type 2 diabetes started on insulin during hospitalization and followed for 1 year. The majority of the French patients also did not achieve goal A1C $<7\%$; further, they had little intensification of insulin therapy after discharge by their outpatient providers (12). Taken together, these findings raise questions regarding continuity of diabetes management and intensification of therapy in the outpatient setting after hospitalization, and is worthy of future study and intervention. Appropriately, outpatient post-hospitalization care may also initially focus more on the acute medical conditions that resulted in admission than on management of chronic conditions like diabetes. While it may be argued that some patients with chronic co-morbidities may not be candidates for intensive glycemic control, these findings were observed in patients with degrees of hyperglycemia that are likely to be detrimental to current as well as long-term health outcomes.

Our results must be considered within the limitations of the study design. Our study was observational and as such our findings represent correlative associations. It is also limited to care at a single large tertiary hospital and its associated outpatient clinics, and may not be generalizable to other settings. Sicker patients are under-represented in the final analysis given inability to find adequate number of individually matched controls. However, equivalently sick patients are included in the matched analysis; removing some portion of the sickest hospitalized patients has left a study group that is closely matched to outpatient controls, and therefore more likely to warrant similar attention to glycemia. Data on duration of diabetes and glycemia-related medication changes were not available for this study. There was no ongoing structured inpatient glycemic management program (diabetes management service, standardized diabetes education program, or standardized diabetes discharge instructions) during the study period, though some subjects may have received diabetes education as recipients of non-standard diabetes education or endocrinology consults.

Ongoing health care reform with the creation of medical homes and accountable care organizations supports increased continuity between inpatient and outpatient care. In diabetes management, it is likely to fuel interest in research on the impact of inpatient diabetes management on outpatient outcomes and overall cost-effectiveness (7, 12, 22). Previous studies have shown that sustained reductions in A1C of >1% for over 1 year are associated with significant cost savings within 1 to 2 years of improvement, regardless of baseline complications, and that every 1% increase in A1C above 7% significantly increases medical costs over the following 3 years (23, 24). Similarly, higher mean A1C levels have been associated with increased estimated cost for broadly defined “diabetes-related” hospitalizations, especially in those with mean A1C >10% over a 4 year period (2002–2006) (25).

As focus on the inpatient-to-outpatient transition increases (as has already begun with efforts to reduce readmissions and acute-care utilization) and movement towards accountable care organizations results in greater global care initiatives, measures of long-term glycemia in hospitalized patients will likely become more important. Glycemic control in the outpatient setting has long been a target for value-driven health care initiatives, using A1C as the marker of glycemic control. The National Committee for Quality Assurance (NCQA)-Healthcare Employer Information Data Set (HEDIS) defines good glucose control as A1C <7% for individuals <65 years without CV disease, end-stage diabetic complications or dementia, and <8% otherwise (17). These guidelines are similar to the recent ADA/AACF/AHA consensus and American Association of Clinical Endocrinologists (AACE) statements which recommend a goal A1C of <7% (AACE goal 6.5%) for non-pregnant adults in general to prevent microvascular and neuropathic complications in type 1 and type 2 diabetes (18, 26).

Our study suggests that in an population of primary care patients with diabetes who would be included in an ACO, patients undergoing hospitalization are less likely to attain even the less stringent HEDIS goal of A1C<8%. Moreover, this study provides data regarding the expected glycemic trajectory after hospital discharge stratified by baseline A1C. These findings may be used to inform research and quality improvement study design and demonstrate that any measurement of inpatient interventions, such as inpatient glycemic management programs, on long-term glycemic control, will need to account for the significant long-term change in A1C that occurs under usual care.

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Figure 1.

Predictors for achieving target hemoglobin A1c values.

Matching variables of age, sex, race, and comorbidity score were not included in the logistic regression models.

*Reference group – baseline A1C 7–9%

†Reference group – Medicare insurance

Table 1

Baseline characteristics of hospitalized patients and matched non-hospitalized controls

	Hospitalized patients	Non-Hospitalized patients	p-value
N (%)	826	826	
Age, mean years (SD) *	65.0 (13.3)	64.6 (12.8)	0.48
Female, N (%) *	394 (48)	388 (47)	0.81
Race, N (%) *			
White	627 (76)	631 (76)	
Non-White	199 (24)	195 (24)	0.33
Black	82 (10)	63 (8)	
Hispanic	77 (8)	88 (11)	
Other	40 (5)	44 (5)	
Comorbidity sum, mean (SD) *	3.17 (1.2)	3.15 (1.2)	0.7
CHF, N (%)	124 (15)	111 (13)	0.4
COPD, N (%)	110 (13)	124 (15)	0.4
Depression, N (%)	186 (23)	193 (23)	0.73
High PCP-connectedness, N (%)	718 (87)	689 (83)	0.052
Health Center, N (%)	297 (36)	348 (42)	0.012
Number of PCP clinic visits in 1 year, mean (SD)	6.96 (4.2)	6.15 (3.8)	<0.001
Number of A1C tests checked in 1 year, mean (SD)	2.8 (0.9)	3.0 (1.0)	<0.001
	Hospitalized patients	Non-Hospitalized patients	p-value
Months from baseline A1C to first follow-up A1C, mean (SD)	4.8 (2.08)	4.8 (2.1)	0.99
Months from baseline A1C to last follow-up A1C, mean (SD)	11.7 (2.4)	9.4 (1.8)	<0.001
Insurance, N (%)			
Medicare	451 (54)	413 (50)	
Private	254 (31)	260 (31)	
Medicaid/Free Care	121 (15)	153 (19)	0.065
Baseline A1C, mean % (SD) *	7.7 (1.7)	7.6 (1.6)	0.2
Baseline A1C 7%, N (%)	361 (44)	368 (45)	0.77
Mean % (SD)	6.41 (0.5)	6.38 (0.5)	0.33
Baseline A1C 7–9%, N (%)	330 (40)	331 (40)	1.0
Mean % (SD)	7.8 (0.5)	7.7 (0.5)	0.1
Baseline A1C 9%, N (%)	135 (16.3)	127 (15.4)	0.64
Mean % (SD)	10.8 (1.6)	10.6 (1.3)	0.45

* Variables used for case-control matching

CHF = congestive heart failure, COPD = chronic obstructive pulmonary disease, PCP = primary care physician, A1C = hemoglobin A1c

Table 2

Estimated rate of AIC change (% per month) from random effects models.

	All patients			Hospitalized patients			Non-hospitalized patients			Interaction of hospitalization and AIC change	
	AIC change (%/month)	95% CI	p-value	AIC change (%/month)	95% CI	p-value	AIC change (%/month)	95% CI	p-value	p-value	p-value
All patients	-0.013	-0.023, -0.002	0.016	-0.010	-0.022, 0.0009	0.072	-0.0099	-0.022, 0.0021	0.11	0.42	0.42
Baseline AIC 9% *	-0.10	-0.18, -0.02	0.012	-0.10	-0.19, -0.022	0.01	-0.10	-0.18, -0.02	0.02	0.12	0.12
Baseline AIC 7-9% †	-0.019	-0.04, 0.001	0.067	-0.013	-0.04, 0.013	0.33	-0.029	-0.054, -0.004	0.03	0.78	0.78
Baseline AIC 7%	0.014	-0.004, 0.03	0.12	0.012	-0.012, 0.03	0.35	0.021	0.0014, 0.04	0.04	0.61	0.61

* Adjusted for congestive heart failure (CHF), Depression, health insurance, and connectedness to primary care provider (PCP).

† Model of all patients also included interaction term for baseline hemoglobin A1c (A1C).

‡ AIC stratified models also included interaction terms for rate of change and CHF, depression, health insurance and PCP-connectedness.

* Connectedness to PCP had significant advantageous interaction with rate of AIC change (p 0.023), with those loyal to PCP having greater decrease in AIC over time (months).

† Having CHF had a significant detrimental interaction with rate of AIC change (p 0.002), with those with CHF having less dramatic decrease in AIC over time (months).