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Factors Influencing the Increasing Disparity in LDL Cholesterol Control between White and Black Patients with Diabetes in a Context of Active Quality Improvement

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Abstract

After implementing a multifaceted physician-directed quality improvement (QI) initiative, we observed an increased disparity in LDL cholesterol control between white and black diabetes patients. To examine possible causes, we performed a retrospective analysis of 962 black and white patients treated continuously between 2008 and 2010. At baseline, 55.0% of whites and 49.8% of blacks were controlled (5.2% disparity). The disparity increased, with 61.8% of whites and 44.6% of blacks having control in 2010 (17.2% disparity). Among patients uncontrolled at baseline, blacks were less likely to become controlled. Among patients controlled at baseline, blacks were less likely to remain controlled. Accounting for patient characteristics and changes in lipid-lowering drug prescription regimens did not attenuate these relationships. Physician-facing, general QI interventions may be insufficient to produce equity in LDL cholesterol control. Helping patients maintain prior success controlling cholesterol appears as important in addressing this disparity as is helping uncontrolled patients achieve control.

Health information technology (HIT) and performance measurement are increasingly used to drive quality improvement (QI). Previous studies have shown that interventions such as measurement and feedback of performance to providers, point-of-care clinical decision support and the use of registries can improve overall quality of care but general, practice-level QI interventions do not clearly lead to reduced racial disparities in quality.[1–6] Understanding how generalized QI interventions influence racial disparities in quality may lead to more effective strategies to reduce disparities.

The UPQUAL (Using Precision Performance Measurement to Conduct Focused Quality Improvement) project was an example of a HIT-supported practice-wide QI initiative. It was implemented in February 2008 in the Northwestern Medical Faculty Foundation General Internal Medicine practice.[7] The intervention sought to improve performance for multiple

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chronic disease and preventative care quality measures simultaneously using rigorous performance measurement from electronic health record (EHR) data, physician-directed point-of-care computerized clinical decision support, and audit and feedback of performance data to primary care providers.

A previous study on the impact of UPQUAL observed racial disparities prior to implementing these quality improvement methods.[1] Some disparities persisted even as overall quality improved. Of seven performance measures for which baseline racial disparities were present prior to the start of this intervention in 2008, disparities declined for only two measures, remained stable for four, and increased for one measure – control of low-density lipoprotein (LDL) cholesterol in diabetic patients.[1]

These results illustrate the need to examine whether this quality improvement intervention may have affected patients from different racial groups in different ways. We sought to perform an in depth study of how LDL cholesterol control status and treatment plans differed by race before and during this quality improvement intervention in order to better understand the causes of this increasing racial disparity in LDL cholesterol control.

Methods

Study population

The study was conducted at the Northwestern Medical Faculty Foundation General Internal Medicine practice, a large primary care practice in an urban setting affiliated with an academic medical center. Northwestern University's institutional review board approved the study. The study population included all patients aged 18 to 89 with diabetes mellitus (based on a diagnosis code for diabetes mellitus on their past medical history or problem list) diagnosed before the start of the study period (prior to 2/1/2008), who had at least one office visits in each of 2008 and 2009 (2008 defined as 2/1/2008 – 1/31/2009, 2009 defined as 2/1/2009 – 1/31/2010), and whose race was recorded in the EHR as either white or black.

Measurements

Patient characteristics—Data regarding patient age, sex, race, health insurance, comorbidities, LDL cholesterol values, and lipid lowering medications were obtained using Structured Query Language queries from the Northwestern University Enterprise Data Warehouse which contains data copied from the EHR. Insurance type was categorized as commercial, Medicare, Medicaid, other, and uninsured. The number of major comorbidities included eight of the nine categories included in the 2008 Dartmouth Atlas of Health Care: cancer, chronic pulmonary disease, coronary artery disease, congestive heart failure, peripheral vascular disease, severe chronic liver disease, renal failure, and dementia.[8] Diagnosis of ischemic vascular disease included ICD-9 diagnoses of coronary artery disease, peripheral artery disease, and ischemic cerebrovascular disease. Patient income and education were estimated by mapping patient addresses to US census block aggregate socioeconomic data from 5-year estimates from the American Community Survey (2006 – 2010) using ArcGIS 10 software (ESRI, Redlands, CA).

LDL cholesterol control—LDL cholesterol control was assessed at baseline (2/1/2008) and two year follow up (2/1/2010). LDL cholesterol control was defined as having the most recent LDL cholesterol test result < 100 mg/dL performed within the previous year. This measure is similar to the National Committee for Quality Assurance's Healthcare Effectiveness Data and Information Set measure.[9] Patients who did not meet the measure either had their last LDL-cholesterol level ≥ 100 mg/dL or did not have an LDL cholesterol test done in the preceding 1 year.

Lipid-lowering drug regimens—Lipid-lowering drugs (LLD) included HMG-CoA reductase inhibitors ("statins"), bile acid sequestrants, niacin, ezetimibe, and fibrates. The LLD regimen at baseline and follow up was defined as the drug or drugs that were active on the patient's EHR medication list on the date 6 weeks prior to the date of the LDL cholesterol test used to determine LDL control status for that date. We chose these criteria because we sought to determine LLD regimens that were prescribed at the time their LDL cholesterol levels were measured. When there was no LDL cholesterol value measured in the preceding year, the active medication list on February 1, 2008 or February 1, 2010 was used to indicate the LLD regimen for baseline and follow up, respectively. We defined high-potency statins as atorvastatin ≥40 mg or rosuvastatin ≥ 20 mg, based on the ability of the daily doses of these medications to reduce LDL cholesterol > 50%, and defined all others as lower-potency statins.[10, 11] We defined combination LLD therapies as being prescribed both a statin and a non-statin concurrently. We considered patients to have had an increase in LLD regimen between 2008 and 2010 if any LLD was added (or substituted) or the dose of a previously prescribed LLD was increased. Decreases in regimen from 2008 to 2010 were defined as removal of any LLD, or a decrease in the daily dosage of a LLD.

Statistical analyses

We used descriptive statistics (Chi-square test for categorical variables and students t-test for normally distributed continuous variables) to compare baseline patient characteristics of white and black patients.

We first used univariable logistic regression to assess the unadjusted association between race and baseline LDL control. We then performed multivariable logistic regression with a model that included patient characteristics (gender, age, neighborhood education, ischemic vascular disease, Dartmouth Atlas comorbidity count, number of primary care visits in prior year, and insurance status). The third model added the presence of a LLD on the medication list at baseline.

We performed three regression models to assess the relationship between race and achieving LDL control at follow up, stratified by baseline LDL cholesterol control: 1) black race as the only independent variable, 2) after adding variables to indicate lack of baseline LLD prescription and changes in LLD treatment regimens, and 3) adding gender, age, neighborhood education, ischemic vascular disease, Dartmouth Atlas comorbidity count, number of primary care visits in prior year, and insurance status to the second model. Analyses were performed using STATA version 12.1 (StataCorp, College Station, TX).

Chart review for patient adherence

We performed retrospective review of EHR charts for patients whose LDL cholesterol was not controlled in 2010 to identify physician-documented reasons why patients may have remained uncontrolled or fallen out of control. We defined physician-documented non-adherence to a LLD as any mention in the chart of the patient not taking a prescribed LLD for any reason other than under the direction of a healthcare professional. We classified different kinds of physician-documented reasons for non-adherence into a priori categories: the patient experienced an adverse drug event, had a financial barrier to accessing medication, perceived the risk of taking medication to be too great, or the patient preferred to not use medications in general. More than one reason for non-adherence could be present for each patient. One investigator (RZ) reviewed all internal medicine, endocrinology, and cardiology outpatient visit, and telephone call notes from 2/1/2008 to 2/1/2010. A second investigator (SP) reviewed a subset of 10 random records. Adjudication of any non-adherence was in complete agreement with the first reviewer. There was a single discrepancy in categorization of non-adherence type.

Results

Baseline characteristics and LDL control

There were 962 patients who met inclusion criteria for this study, of whom 56% were white and 44% were black. Table 1 presents baseline patient characteristics by race. Black patients were more likely than white patients to be female, were less likely to be diagnosed with ischemic vascular disease, and made more general internal medicine visits in the prior year. Black patients were more likely than white patients to have Medicaid insurance and were less likely to have commercial insurance. Most patients (79.4%) had their LDL tested in the past year. The mean of the most recent LDL cholesterol levels among patients tested in the prior one year was significantly higher for black patients than white patients (96.4 vs. 88.3 mg/dL). Neighborhood socioeconomic characteristics, measured by the proportion of individuals in the neighborhood with an education reported as high school or less and by neighborhood median income, did not differ significantly between black and white patients. Black patients were less likely to have any LLD on their medication list at baseline and were less likely to be prescribed combination lipid lowering drug therapy. Table 2 presents prescribed lipid-lowering regimens by race.

In 2008, 55.0% of white patients and 49.8% of black patients had controlled LDL cholesterol, a 5.1% difference between groups that was not statistically significant; univariable odds ratio for LDL control for black patients compared to white patients 0.81 (95% confidence interval [CI] 0.63 – 1.04). This relationship was attenuated by adding patient characteristics to the model (adjusted OR 0.88 [95% CI 0.67 – 1.17]), and baseline LLD prescription (adjusted OR 0.94 [95% CI 0.70 – 1.26]).

Changes in LDL cholesterol control and racial disparity 2008 to 2010

The racial disparity in LDL cholesterol control increased following the implementation of the multimodality QI intervention. As of February 1, 2010, 61.8% of whites and 44.6% of blacks met this measure, a 17.2% difference in between groups and a 12.1% increase in the

racial disparity between 2008 and 2010. Most of the cohort had an LDL test during the year prior to 2/1/2010, and this did not differ significantly by race (75.7% of white patients and 72.4% of black patients, p=0.25). Among patients with controlled LDL cholesterol at baseline, 73.3% of white patients and 56.4% of black patients remained controlled (p <0.001). Among patients with uncontrolled LDL cholesterol at baseline, 47.9% of white patients and 32.9% of black patients became controlled (p=0.001).

Black patients were significantly less likely than white patients to have any LLD regimen on their active medication list in both 2008 (70% vs. 80%) and 2010 (69% vs. 79%) (p<0.001 for both comparisons), but there was no appreciable change in the proportion of each group treated with a LLD during the study period (Table 2). For patients on a LLD regimen, regimens differed significantly by race in both 2008 (p = 0.028) and 2010 (p = 0.017).

During the two-year intervention period, a majority of uncontrolled patients had no change in their LLD regimens (Table 3). Patients who were uncontrolled at baseline were significantly more likely to have their LLD regimen changed than patients controlled at baseline (38.5% of uncontrolled patients, 30.8% of controlled patients, p = 0.012). There was no statistically significant difference in types of changes during the two-year period in LLD regimens between white and black patients, for either the controlled or uncontrolled at baseline groups.

Table 4 presents results for models of LDL cholesterol control in 2010 stratified by control status in 2008. In the unadjusted models, black race was associated with lower odds of having LDL control in 2010 for patients uncontrolled in 2008; odds ratio (OR) of 0.53 (95% confidence interval [CI] 0.36 – 0.78), and for patients controlled in 2008; OR of 0.47 (95% CI 0.32 – 0.69). Adjusting for lack of baseline LLD prescription and treatment changes (Model B) and adding patient demographic and clinical characteristics (Model C) did not attenuate the effect of race. Not having any LLD drug on the medication list was significantly associated with subsequent lack of control for both controlled and uncontrolled patients at baseline. Having the regimen decreased by 2010 was significantly associated with subsequent lack of control for patients who were controlled at baseline.

Reasons for not achieving/losing LDL cholesterol control

Among patients with uncontrolled LDL cholesterol in 2010, structured chart review demonstrated that 40% of black patients and 29% of white patients had physician-documented non-adherence to LLD (p = 0.019). The reasons for non-adherence were not significantly different between black and white patients (Table 5). The most commonly documented reasons for non-adherence were patient experienced adverse drug events (32% for black patients, 35% for white patients) and patient preference to not use medication (27% for black patients, 35% for white patients). Less commonly documented reasons included financial barriers (24% for black patients, 18% for white patients), and perceived risks of taking medication (4% for black patients, 8% for white patients).

Discussion

We sought to examine why the racial disparity in LDL cholesterol control among adults with diabetes cared for in the same care setting increased during a period of active quality improvement. In a continuously followed cohort, performance for this measure improved by 6.8% for white patients and declined by 5.2% for black patients. Black patients were less likely to have a LLD on their medication list at both time points, and this difference did not change appreciably. Accounting for differences in patient characteristics and in drug treatments prescribed did not alter the observed racial difference in LDL cholesterol control. Black patients were more likely than white patients both to remain uncontrolled (among those uncontrolled at baseline) and to become uncontrolled (among those controlled at baseline).

Other studies addressing racial differences in LDL cholesterol control in diabetes have shown varying patterns of change during times of general quality improvement. One study conducted in the Veterans Affairs health care system and another from an integrated healthcare system in Michigan showed that during periods of considerable quality improvement, racial differences in LDL control in diabetes were not reduced.[12, 13] Another study from an integrated health care system in Massachusetts showed a slight reduction in the black-white disparity, however, this was done at a time when baseline levels of LDL cholesterol testing, and LDL control were low (1997–2001), and the amount of change in the racial disparity was small (2.5% reduction).[3] General QI interventions directed at patients such as individualized case management programs using teams may be among the more potent interventions for improving control of chronic conditions.[14] This approach has led to similar improvement in disease control for diabetes across different racial groups.[15]

We examined whether changes in drug prescribing, which may have occurred in response to the QI efforts, differed by race and could potentially account for the increasing disparity. White patients were more likely than black patients to be prescribed any LLD drug regimen in 2008 (80% vs. 70%), and this factor seemed to have a small influence on the relationship between race and LDL control in 2008. However, this difference did not change by 2010 (79% vs. 69), and accounting for intensification or reduction in LLD regimens did not attenuate the relationships between LDL control and race in 2010. Therefore, we do not find evidence to support the notion that differences in prescribed LLD treatments over time (as represented by the medication list in the EHR) caused the racial disparity to increase. There are limitations to the medication data available through electronic health records that must be kept in mind when interpreting these data. While treatment intensification is associated with subsequent LDL control,[15] we do not know how often a lack treatment intensification in response to uncontrolled LDL cholesterol is due to providers' inaction and how much is driven by patients' actions or preferences. We also lack reliable information on adherence to medications listed in the EHR, so we are unable to parse out the extent to which non-adherence contributes to the observed findings. From chart review, we observed that physicians commonly documented patient non-adherence to prescribed LLD regimens (40% of black patients, 29% of white patients), suggesting that patient behaviors, preferences, and ability to tolerate medication are major determinants of whether prescribed LLDs are used. QI interventions should be tested that not only focus on providers' prescribing actions, but

also monitor and support patient medication usage behaviors and proactively identify and address ameliorable adverse drug events when they occur.

A large part of the increase in the racial disparity that we observed was due to a disproportionally large number of black patients with previously controlled LDL cholesterol who did not remain controlled during follow up. Point-of-care clinical decision support that calls attention to patients who were not currently meeting the quality measure, such as what was used here, does not do anything to encourage physicians to assess patients' current medication use, detect medication discontinuation proactively, and formulate new care plans before a patient returns for periodic laboratory monitoring that shows inadequate risk factor control. System-level interventions to promote long term persistence with important medical therapies among both patients who are and are not currently meeting quality of care goals should be tested. The incorporation of pharmacy data into clinics' information systems so that it could be used to identify when patients stop refilling medications and trigger prompt action holds promise as a way to address the quality challenge we observed.[17, 18]

These findings should be viewed in the context of several additional limitations. The study was conducted at one site and the findings may not be generalizable to other settings. We used prescription drug data from electronic health records which may be a better reflection of clinician action than pharmacy claims data, but this data cannot show us which patients filled prescriptions. Socioeconomic differences were measured using geospatial coding to neighborhood characteristics. Had we had patient specific data, we may have found more of the observed differences were mediated by socioeconomic differences. The size of the patient population available for study was 962 patients, therefore some estimates are imprecise. Chart reviews are a very limited way to obtain information about patients' medication use. Standardized patient interviews may provide more reliable measures of patient behaviors and attitudes pertaining to medications.

Ultimately, in a fixed cohort of black and white patients with diabetes receiving care from the same group of providers, taking into consideration clinical and demographic characteristics and changes in prescribed lipid-lowering drug regimens, we were unable to account for the increasing racial disparity in LDL control that occurred during a 2-year period of active quality improvement. This finding does not support the notion that general QI initiatives that rely on provider-directed interventions like clinical decision support and audit and feedback will improve equity by race in the control of intermediate clinical outcomes.

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Table 1

Characteristics of Diabetes Patients, February 1, 2008

Characteristic	White (N = 538)	Black (N = 424)	*P-value*
Female, n (%)	242 (45%)	295 (70%)	<0.001
Age, mean (SD)	64.4 (10.7)	63.3 (10.9)	0.13
Neighborhood percentage with low education level, (SD)	41.6 (19.4)	41.1 (20.8)	0.7
Neighborhood median income, $1000s (SD)	58.8 (29.2)	60.0 (29.0)	0.5
Tested in last year, %	77.3	82.1	0.07
Latest LDL value, mg/dl (SD)	88.3 (31.5)	96.4 (35.0)	0.001
GIM visits past year, n (IQR)	3.9 (2–5)	4.41 (2–6)	0.004
DA comorbidities	2.69 (1.09)	2.63 (1.07)	0.4
IVD	184 [34%]	104 [25%]	0.001
Type of Insurance			
Medicaid	4%	12%	<0.001
Medicare	43%	44%	0.8
Other	1%	1%	0.9
Commercial	51%	43%	0.014
Uninsured	1%	1%	0.9
Control in 2008			
LDL controlled	296 (55.0%)	211 (49.8%)	0.11
LDL not controlled			0.002*
LDL > 100	120 (22%)	137 (32%)	
LDL not tested in past year	122 (23%)	76 (18%)	

Low education level: calculated from geospatially neighborhood data, represents percent of individuals in the neighborhood with high school education or below.

GIM: general internal medicine

DA: Dartmouth Atlas comorbidities, includes cancer, chronic pulmonary disease, coronary artery disease, congestive heart failure, peripheral vascular disease, severe chronic liver disease, diabetes, renal failure, and dementia.

IVD: ICD-9 diagnosis of ischemic vascular disease, includes coronary artery disease (CAD), peripheral artery disease (PAD), and cardiovascular disease (CVD)

*P-value comparing LDL-cholesterol testing rates among uncontrolled patients only

Table 2

Lipid-Lowering Drug Regimens Recorded in Patients' Medical Records

	2008			2010		
	White	**Black**	**P-value**	**White**	**Black**	**P-value**
Any LLD drug regimen	429 (80%)	295 (70%)	< 0.001	426 (79%)	293 (69%)	< 0.001
Regimen composition						
Lower-potency statin*	263 (61%)	201 (68%)	0.06	257 (60%)	197 (67%)	0.059
High-potency statin†	59 (14%)	45 (15%)	0.3	78 (19%)	63 (22%)	0.3
Non-statin only	25 (6%)	17 (6%)	0.9	15 (4%)	5 (2%)	0.15
Combination	82 (19%)	32 (10%)	0.003	71 (17%)	28 (9%)	0.007
Total	429	295		426	293	

LLD: lipid lowering drug

*Lower-potency statins are those predicted reduce average LDL cholesterol < 50%

†High-potency statins are those predicted reduce average LDL cholesterol > 50%

Table 3

Changes in Lipid-Lowering Drug Regimens from 2008 to 2010

	Uncontrolled in 2008			Controlled in 2008		
	White	**Black**	***P-value***	**White**	**Black**	***P-value***
			0.3			0.9
Regimen increased	56 (23%)	61 (29%)		47 (16%)	30 (14%)	
Regimen decreased	35 (23%)	23 (11%)		45 (19%)	34 (16%)	
No change	151 (54%)	129 (60%)		204 (65%)	147 (70%)	
Total	242	213		296	211	

Table 4

Multivariable Regression Results for Predicting LDL Cholesterol Control in 2010, by Baseline Patient Control Status

		Black Race, OR (95% CI)	No LLD at baseline OR (95% CI)	Regimen Increased, OR (95% CI)	Regimen Decreased, OR (95% CI)
Uncontrolled in 2008	Model A	0.53 (0.36 – 0.78)			
	Model B	0.52 (0.35 – 0.76)	0.59 (0.39 – 0.89)	1.60 (1.03 – 2.50)	0.53 (0.28 – 0.99)
	Model C	0.52 (0.34 – 0.80)	0.67 (0.43 – 1.05)	1.59 (1.01 – 2.53)	0.62 (0.32 – 1.22)
Controlled in 2008	Model A	0.47 (0.32 – 0.69)			
	Model B	0.49 (0.34 – 0.73)	0.56 (0.33 – 0.96)	1.13 (0.66 – 1.94)	0.39 (0.23 – 0.65)
	Model C	0.46 (0.30 – 0.70)	0.54 (0.31 – 0.96)	1.11 (0.62 – 1.98)	0.42 (0.24 – 0.72)

Model A: unadjusted

Model B: adjusted for lipid-lowering drug (LLD) at baseline and changes in treatment. Regimen increased includes addition of any LLD or increase in dosage of current LLD. Regimen decreased includes removal of any LLD or decrease in dosage of current LLD.

Model C: adjusted for variables in model B and patient characteristics (gender, age, neighborhood education, ischemic vascular disease, Dartmouth Atlas comorbidities, number of general medicine visits, insurance status)

Table 5

Documentation of Non-adherence to Lipid Lowering Medication among Patients with Uncontrolled LDL Cholesterol at Follow Up

	White N = 205	Black N = 235	P
Any documented non-adherence to LLD	60 [29%]	94 [40%]	0.019
Reason for non-adherence			0.7
Financial	11 [18%]	23 [24%]	
Experienced adverse drug event	21 [35%]	30 [32%]	
Patient preference not to take medication	21 [35%]	25 [27%]	
Perceived risk of medication	5 [8%]	4 [4%]	
Unspecified reason	10 [17%]	24 [26%]	