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Race/Ethnic Disparities in Risk Factor Control and Survival in the Bypass Angioplasty Revascularization Investigation 2 Diabetes (BARI 2D) Trial

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Abstract

This study sought to evaluate the impact of race/ethnicity on cardiovascular risk factor control and on clinical outcomes in a setting of comparable access to medical care. The BARI 2D trial enrolled 1,750 participants from the United States and Canada that self-reported either White non-Hispanic (n = 1,189), Black non-Hispanic (n = 349), or Hispanic (n = 212) race/ethnicity. Participants had type 2 diabetes and coronary artery disease and were randomized to cardiac and glycemic treatment strategies. All patients received intensive target-based medical treatment for cardiac risk factors. Average follow-up was 5.3 years. Kaplan-Meier survival curves and Cox proportional hazards regression models were constructed to assess potential differences in mortality and cardiovascular outcomes across racial/ethnic groups. Long-term risk of death and death/myocardial infarction/stroke did not vary significantly by race/ethnicity (5-year death: 11.0% Whites, 13.7% Blacks, 8.7% Hispanics, p = 0.19; adjusted hazard ratio 1.18 Black versus White, 95% confidence interval 0.84 to 1.67, p = 0.33 and 0.82 Hispanic versus White, 95% confidence interval 0.51 to 1.34, p = 0.43). Among the 1,168 patients with suboptimal risk factor control at baseline, the ability to attain better risk factor control during the trial was associated with higher 5-year survival (71%, 86% and 95% for patients with 0 or 1, 2, and 3 factors in control, respectively, p <0.001); this pattern was observed within each race/ethnic group. In conclusion, significant race/ethnic differences in cardiac risk profiles that persisted during follow-up did not translate into significant differences in 5-year death or death/MI/stroke.

The Bypass Angioplasty Revascularization Investigation 2 Diabetes (BARI 2D) clinical trial was undertaken to compare treatment strategies for patients with both type 2 diabetes

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Disclosures

The authors have no conflicts of interest to disclose.

mellitus and stable coronary heart disease in the presence of intensive risk factor control. Neither all-cause mortality nor major cardiovascular events, defined as the composite end point of death, myocardial infarction, and stroke, differed among the randomized treatment strategy groups in the overall trial. At study entry, self-reported race/ethnicity predicted important differences in demographic, clinical history, biochemical parameters, and burden of coronary artery disease (CAD) among patients with longstanding type 2 diabetes mellitus enrolled in the BARI 2D trial.[1] Given the systematic approach of risk factor control with intensive medical therapy for all study participants, we hypothesized that race/ethnic disparities in risk factor control would be attenuated during follow-up. We also postulated that although race/ethnicity would be an important determinant of long-term clinical outcomes among patients treated in the BARI 2D study, the observed disparities in outcome would be explained by differences in baseline risk factors and burden of CAD. Thus, in the setting of comparable access to intensive medical care and treatment of cardiac risk factors, we sought to evaluate the impact of race/ethnicity on long-term risk factor control and cardiovascular outcomes among patients with both type 2 diabetes mellitus and documented stable CAD enrolled in the BARI 2D clinical trial.

Methods

The trial design, patient characteristics, and primary results of BARI 2D have been described in previous publications.[2,3] Patients with type 2 diabetes mellitus and documented stable CAD with ≥1 coronary lesion with ≥50% stenosis appropriate for elective revascularization were enrolled from 49 clinical sites in 6 countries between January 2001 and March 2005. Participants were randomized in a 2-by-2 factorial design to a strategy of intensive medical therapy (MED) with deferred revascularization if clinically indicated versus prompt revascularization (REV) (coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI)) with intensive medical therapy, and, concurrently, a glycemic treatment strategy of insulin sensitization (IS) versus insulin provision (IP). Before randomization, the enrolling physician determined whether CABG or PCI was more suitable based on the patient's coronary anatomy and clinical profile, and the randomization was stratified by this intended method of revascularization. IS medications included metformin and thiazolidinediones, and IP medications included sulfonylureas and insulin. The primary trial end point was all-cause mortality, and the principal secondary end point was a composite of death, myocardial infarction (MI), and stroke. The average follow-up per patient was 5.3 years for mortality and 4.5 years for other outcomes. BARI 2D was approved by the internal review board at the coordinating center and each clinical site, and all participants gave informed consent.

Study clinic visits occurred on a monthly basis for the first 6 months and every 3 months thereafter. All patients received intensive treatment for cardiac risk factors including hypertension, dyslipidemia, obesity, and smoking. A medication in most diabetes and cardiac drug classes was available free of charge through the BARI 2D trial, and centralized management centers monitored patient risk factor control. The target risk factor goals for the trial were based on concurrent national guidelines (American Diabetes Association, National Cholesterol Education Program, Adult Treatment Panel III, and Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure 7),[4] and these treatment goals were hemoglobin A1c (HbA1c) <7.0%, low-density lipoprotein cholesterol (LDL) <100 mg/dl, and blood pressure ≤130/80 mm Hg. According to the BARI 2D protocol, trial management centers monitored patient-level and site-level risk factor control over the course of the study, and the clinical site investigators were contacted when individual patients had risk factor values above "threshold" levels set at HbA1c ≥8.0%, LDL ≥115 mg/dl, and blood pressure > 140/90 mmHg.

HbA1c, lipid levels, and urine albumin and creatinine were analyzed at the BARI 2D Central Biochemistry Laboratory at the University of Minnesota. HbA1c was measured at baseline, 1 month, 3 months, 6 months, 1 year, and semiannually thereafter, and lipids were measured at baseline, 6 months, 1 year, and annually thereafter. Urine was collected at baseline and at each annual follow-up. Blood pressure and angina status were recorded by the site at every clinic visit. A core laboratory at Stanford University analyzed coronary angiograms for all patients at study entry. A lesion was counted if the measured stenosis was ≥20%, a significant lesion was defined as a lesion with angiographic stenosis ≥50%, and the Myocardial Jeopardy Index was calculated as the percentage of myocardium jeopardized by the significant lesions. The total number of lesions and the Myocardial Jeopardy Index capture the extent and severity of CAD, respectively.

Race and Hispanic ethnicity were self-reported by participants at study entry. Patients who replied “Yes” to “Are you Hispanic or Latino?” were classified as having Hispanic ethnicity for this analysis. Other patients were considered non-Hispanic and were classified according to their self-reported race: (1) American Indian or Native Alaskan, (2) Asian, (3) Black or African-American, (4) Native Hawaiian or Other Pacific Islander, (5) White, or (6) Other. Because the concept of race/ethnicity and the health barriers associated with race/ethnicity vary by country, this analysis was restricted to patients from the BARI 2D sites in the United States and Canada.

Baseline patient characteristics were compared by race/ethnicity group. Continuous variables were presented as means and standard deviations or as medians and inter-quartile ranges and were compared with analysis of variance or Kruskal-Wallis tests; categorical variables were presented as percentages and compared with chi-square tests. Analyses were performed at baseline and at the Year 3 follow-up visit, so chosen to ensure completeness of data for participants recruited later in the study who may not have 4 full years of follow-up.

In this article, overall risk factor control per patient was defined by glycemic control (HbA1c <8.0%), lipid control (non–high-density lipoprotein [HDL] cholesterol ≤130 mg/dl), and blood pressure control (blood pressure ≤140/90 mm Hg). This definition was based on the trial monitoring threshold values, and non-HDL cholesterol was used rather than LDL to minimize the amount of missing data. Patients who attained all 3 goals were deemed to have “good” control, those who attained 2 goals had “moderate” control, and those who attained 0 or 1 goal had “poor” control. Goals were measured at baseline and using area under the curve methods for study follow-up (from baseline to Year 5). Area under the curve values were calculated by drawing lines between each follow-up measurement of the respective risk factors and calculating the average value.

The 5-year rates of death, death/MI/stroke, and subsequent revascularization for the race/ethnicity groups were estimated using Kaplan-Meier survival curves and compared with log-rank statistics. For patients assigned to REV, revascularization procedures that occur after the assigned revascularization procedure are categorized as subsequent revascularization procedures, whereas for patients assigned to MED, all revascularization procedures during the trial are categorized as a subsequent procedures. Cox proportional hazards regression models were used to estimate the hazard ratios for the outcomes of death and death/MI/stroke between the respective race/ethnic categories. The adjusted Cox model included the baseline covariates: age, gender, education, insurance status, albumin creatinine ratio >30, smoking history, body mass index, history of MI, congestive heart failure, duration of diabetes, baseline insulin use, number of diseased coronary regions, number of lesions with ≥20% stenosis, and randomized glycemic and cardiovascular treatment assignment. A second adjusted Cox model included these same baseline factors as well as time-varying covariates of HbA1c, systolic blood pressure, and non-HDL cholesterol. Interactions

between race/ethnicity and glycemic treatment and race/ethnicity and cardiovascular treatment were tested and were included only if statistically significant. A 2-sided alpha = 0.05 was considered statistically significant for all analyses.

Results

Among the 2,368 patients randomized in the BARI 2D clinical trial, 1,852 patients were enrolled in the 45 U.S. and Canadian clinical sites. Based on self-reported Hispanic ethnicity and race respectively, patients were categorized as Hispanic (n = 212), and the non-Hispanic patients were categorized as White (n = 1,189), Black (n = 349), American Indian/Native Alaskan (n = 11), Asian (n = 89), Native Hawaiian/Pacific Islander (n = 1), or Other-race (n = 1). Given the small numbers of patients in several categories, only White non-Hispanic (White), Black non-Hispanic (Black), and Hispanic race/ethnicity groups are included in this analysis.

There were numerous differences in the baseline characteristics of patients in the 3 race/ethnicity categories (Table 1). There were differences in age, gender, education, health insurance status, body mass index, duration of diabetes, myocardial jeopardy index, and number of significant coronary lesions. There were no imbalances regarding the randomization to the cardiac (REV and MED) or glycemic (IS vs IP) treatment groups. For a majority of the U.S. and Canadian BARI 2D patients, the intended revascularization procedure was PCI rather than CABG, and PCI was more frequently selected for Black participants compared with White and Hispanic participants.

Among patients assigned to revascularization (Table 1), Black patients had significantly fewer lesions attempted with PCI on average, consistent with the fact that Blacks had fewer significant lesions at study entry. The use of multi-vessel PCI as well as the use of drug-eluting and bare-metal stents did not differ significantly by race/ethnicity. When CABG was the initial assigned procedure, the use of arterial conduits (internal mammary grafts), number of bypass grafts and conduits, and utilization of off-pump CABG were similar for the 3 race/ethnicity groups.

Medication use by race/ethnicity at baseline and at the 3-year follow-up is presented in Table 2. At study entry, Black patients were more likely to receive insulin and less likely to receive metformin for glycemic control than White or Hispanic patients. Race/ethnic differences in diabetes drug use decreased during the trial, but differences in insulin use remained statistically significant at Year 3. Utilization of cardiac drugs was fairly similar among race/ethnic groups; however, White patients were more likely to receive aspirin at baseline and Black patients were more likely to receive nonsublingual nitrates at Year 3.

Significant differences in cardiovascular risk factors were noted between White, Black, and Hispanic patients at study entry (Table 2). After entry into the trial, there were marked improvements in mean HbA1c, blood pressure, and lipid profiles within each of the 3 race/ethnicity groups. The magnitude of the change from baseline to the 3-year follow-up was similar for systolic blood pressure, LDL, and non-HDL cholesterol, whereas the magnitude of change was different across racial/ethnic categories for HbA1c, diastolic blood pressure, and triglycerides. The proportions of patients who were above the “monitoring threshold” targets set by the BARI 2D trial decreased in all 3 race/ethnicity categories (Figure 1). A consistently lower proportion of White patients were above the glycemic and the renal targets compared with Black and Hispanic patients, and a consistently higher proportion of Black patients were above the cholesterol and blood pressure targets compared with White and Hispanic patients.

Among patients randomized to medical therapy, the subsequent revascularization rate during the trial was similar for the 3 race/ethnic groups (5-year Kaplan-Meier subsequent procedure rates: 46.0% White; 48.9% Black; 46.4% Hispanic; p = 0.90), whereas among those randomized to revascularization therapy, the subsequent revascularization rate was higher for Black patients for the duration of follow-up (5-year Kaplan-Meier subsequent procedure rates: 24.1% White; 37.4% Black; 19.4% Hispanic; p = 0.002).

Regarding good (3 of 3), moderate (2 of 3), and poor (0 or 1 of 3) control of HbA1c, non-HDL, and systolic blood pressure, 35.2% of White patients, 23.5% of Black, and 29.9% of Hispanic patients were in good control at study entry (p <0.001). During the trial follow-up period, good control was achieved by 59.9% of White, 38.9% of Black, and 43.1% of Hispanic patients, and moderate control was achieved by 30.3% of White, 37.4% of Black, and 34.1% of Hispanic patients (p <0.001). Among the 1,168 patients with suboptimal risk factor control at study entry (<3 risk factors in control), the ability to attain better risk-factor control during the trial was associated with higher 5-year survival (5-year survival was 71% for patients with poor follow-up control, 86% for moderate follow-up control, and 95% for good follow-up control, p <0.001). This pattern was observed within all 3 race/ethnic groups (Figure 2).

The 5-year survival rate was 91.3% for Hispanics, 89.0% for White patients, and 86.3% for Black patients (Figure 3, log-rank p = 0.19); and 81.3% of Hispanic, 77.4% of White, and 72.5% of Black patients were free from death, MI, and stroke (Figure 3, log-rank p = 0.11). Although the risks of death and death/MI/stroke were highest for Black patients, the hazard ratios by race/ethnicity were not statistically significant with or without adjustment for baseline risk factors using Cox proportional regression (Table 3). Moreover, the race/ethnicity estimates did not change substantially after adjusting for follow-up risk factor control (Table 3). The effect of race/ethnicity on death and on death/MI/stroke did not vary significantly according to treatment assignment in the overall trial or within the PCI or CABG strata (interaction p >0.10).

Discussion

The impact of race/ethnicity on long-term risk factor control and cardiovascular outcomes among patients with type 2 diabetes mellitus and stable ischemic heart disease was evaluated in the BARI 2D trial. Clinically and statistically significant disparities in cardiovascular risk factors among the 3 race/ethnic groups were present at baseline and during the follow-up period, such that Black and Hispanic patients had worse risk profiles throughout the trial. There were marked improvements in risk profiles within each of the 3 patient groups after study entry, and the extent of these changes was similar across racial/ethnic groups.

In the setting of intensive medical management offered through the trial, the risk of death and death/MI/stroke did not differ significantly by race/ethnicity with or without adjustment for baseline risk factors. The risk of death and cardiovascular events was estimated to be 20% higher in Black participants than White participants when adjusting for potential confounders, but this difference was not statistically significant and is less than the estimated 30% difference nationwide. This suggests that providing intensive medical therapy with a goal of uniform risk factor control may reduce racial/ethnic disparities in outcomes.

Although risk factor control improved in all race/ethnic groups in BARI 2D, interesting patterns emerged. Control of cholesterol and blood pressure tended to improve progressively across the 5 years, whereas HbA1c control gains were most marked from baseline to year 1, with little improvement in subsequent years. In the Hispanic participants, there was little

change in glycemic control, with 35% to 40% of individuals retaining HbA1c values >8%. This is consistent with nationally representative data showing rates of optimal glycemic control in Hispanics (34%) lower than Whites (48%), and slightly lower than Blacks (36%).[5]

Major disparities in risk factor control and death rates by race/ethnicity are well documented in the United States, with CAD death rates 30% higher and diabetes-related mortality roughly 50% and 100% higher in Hispanics and Blacks, respectively, compared with Whites.[6] National data suggest that risk factor control in persons with diabetes is generally challenging, and especially so in minorities.[7] According to the 2003 Institute of Medicine report,[8] disparate care can be defined as "racial and ethnic differences in health care that are not otherwise attributable to known factors such as access to care (insurance/ability to pay)." The structure of the BARI 2D trial largely removed access to care as an explanation, and indeed the trial appears to have closed some of the race/ethnicity differential observed at the national level. The study protocol, however, did not remove all inherent causes of disparate care. For example, language literacy has been shown to be associated with diabetes self-care[9] and may have influenced the risk factor control of Hispanic patients. Despite trial guidelines and monitoring, treatment decisions were ultimately made by the study investigators along with their patients, and thus we cannot rule out the potential for disparate care at the provider level.

Previous literature suggests that Blacks with coronary disease are significantly less likely than Whites to undergo coronary revascularization.[10–13] In the present study, similar treatment was available to all participants, and by design, the assignments to cardiac (REV vs MED) and glycemic (IS vs IP) treatment strategies were balanced across the race/ethnicity groups. PCI was selected as the intended revascularization procedure by the BARI 2D cardiologist rather than CABG for the majority of U.S. and Canadian BARI 2D patients. Among the revascularization group, Blacks were more likely to undergo PCI rather than CABG; this is consistent with previous literature and may be explained by less severe coronary disease, as measured by lower myocardial jeopardy and fewer significant coronary lesions, observed in Black patients at study entry.[12] It is possible that the higher proportion of Blacks assigned to PCI (relative to Whites and Hispanics) rather than CABG in BARI 2D may have affected the racial/ethnic relationships mortality due to slightly lower coronary disease burden among Blacks. However, we present results adjusted for several variables representing extent of disease, such as number of diseased coronary regions and number of total lesions, in which the hazard ratios comparing racial/ethnic groups remain fairly consistent. Also notable, it has been previously reported that Blacks undergoing revascularization with PCI have worse long-term outcomes, stent thrombosis[14] and mortality compared with whites[15,16]; no such difference was found in BARI 2D, although Black participants assigned to REV did require subsequent revascularizations more frequently than white and Hispanic participants.

There is a growing literature describing interventions to reduce cardiovascular and diabetes disparities,[17,18] particularly because minorities may fare less well than Whites, even when cared for in similar high-quality ambulatory settings in the community.[19] Recent work suggests that emphasis on dietary interventions and more collaborative goal setting between patients and clinicians may provide more effective cardiovascular risk reduction in Black individuals.[20,21] Interventions to improve glycemic control in Hispanic patients are also urgently needed given the increasing prevalence of both diabetes and diabetic complications in this population.[22]

National efforts to improve adherence to cardiac care guidelines have demonstrated important gains in the past decade.[23] The strong relationship between risk factor burden and cardiovascular events suggests that aggressive risk factor modification has the potential to

close the gap in racial differences in cardiovascular events.[24] Our study of a well-characterized population with both type 2 diabetes mellitus and CAD offers further evidence that such efforts are likely to reduce disparities in cardiovascular outcomes, including mortality.

The present analysis from BARI 2D suggest that Blacks entered the study with a worse risk-factor profile and poorer risk-factor control, but at follow-up, no statistically significant differences in outcomes in mortality or death/MI/stroke were evident. It should be noted that the study population was categorized into race/ethnicity groups according to self-report data, which may introduce possible bias if 1 race/ethnic group or if participants of mixed race are more likely to identify as 1 racial/ethnic group, and also it should be noted that enrollees in a randomized trial may not be entirely representative of the general population. Finally, there may not have been enough minorities enrolled to determine whether risk factor modification truly moderated race/ethnic differences in cardiovascular outcome. Although BARI 2D did have sufficient power to detect differences of magnitude seen in previous studies, BARI 2D had limited power to detect statistical significance of smaller differences such as the observed 20% relative increase in death for Black compared with White patients in BARI 2D. The observed differences might have been statistically significant if BARI 2D had enrolled more patients or had longer follow-up.

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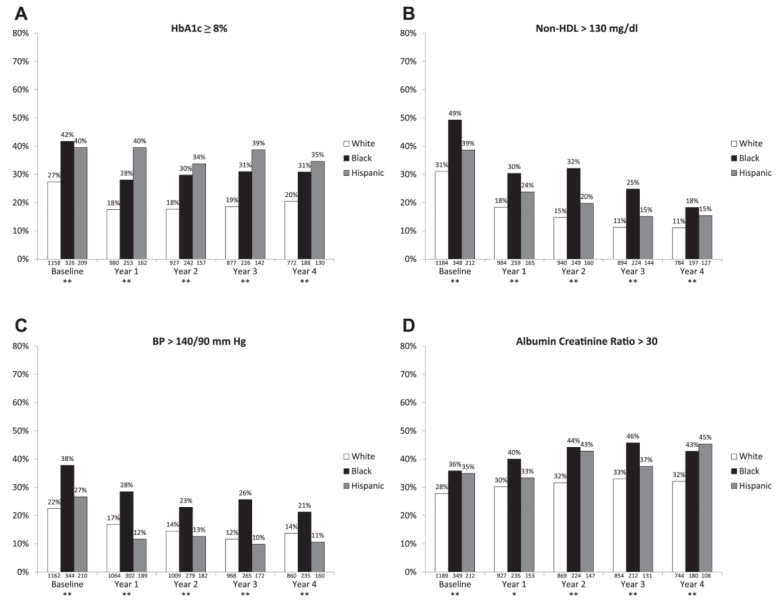

Figure 1.

The percent of patients above risk factor thresholds for HbA1c, non-HDL cholesterol, blood pressure (BP), and albumin creatinine ratio (ACR) by race/ethnicity and follow-up year. * p <0.05; ** p <0.01.

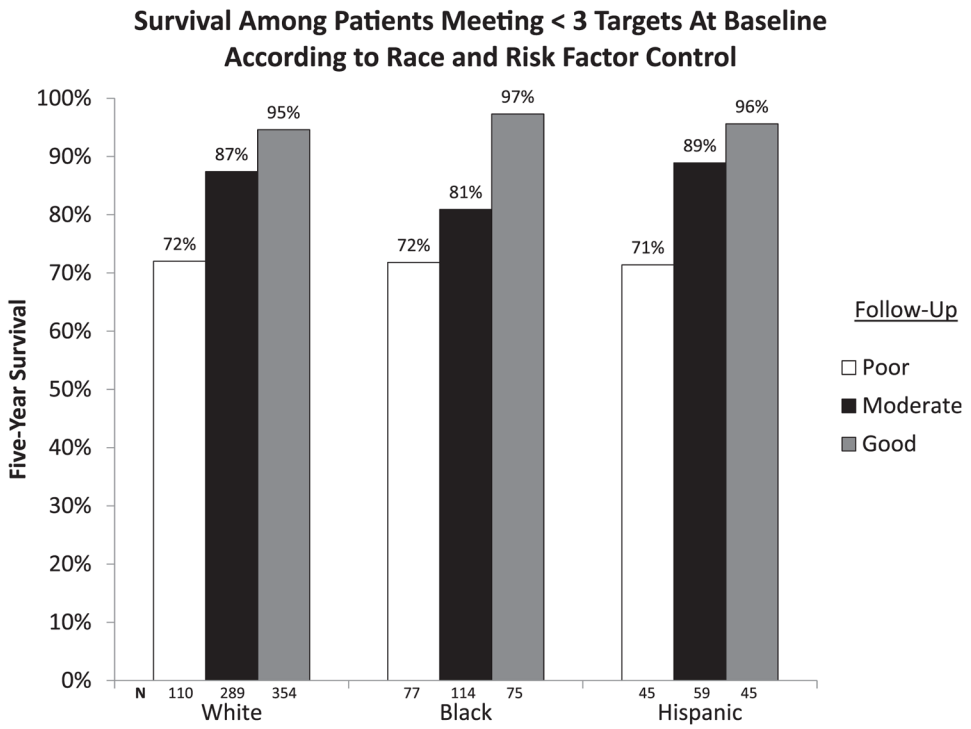

Figure 2.

The 5-year Kaplan-Meier estimated survival rate for patients entering the trial with <3 of the key risk factors in control (HbA1c <8.0%, non-HDL cholesterol ≤130 mg/dl, and systolic blood pressure ≤140 mm Hg) according to race/ethnicity and number of follow-up risk factors in control over the trial follow-up period.

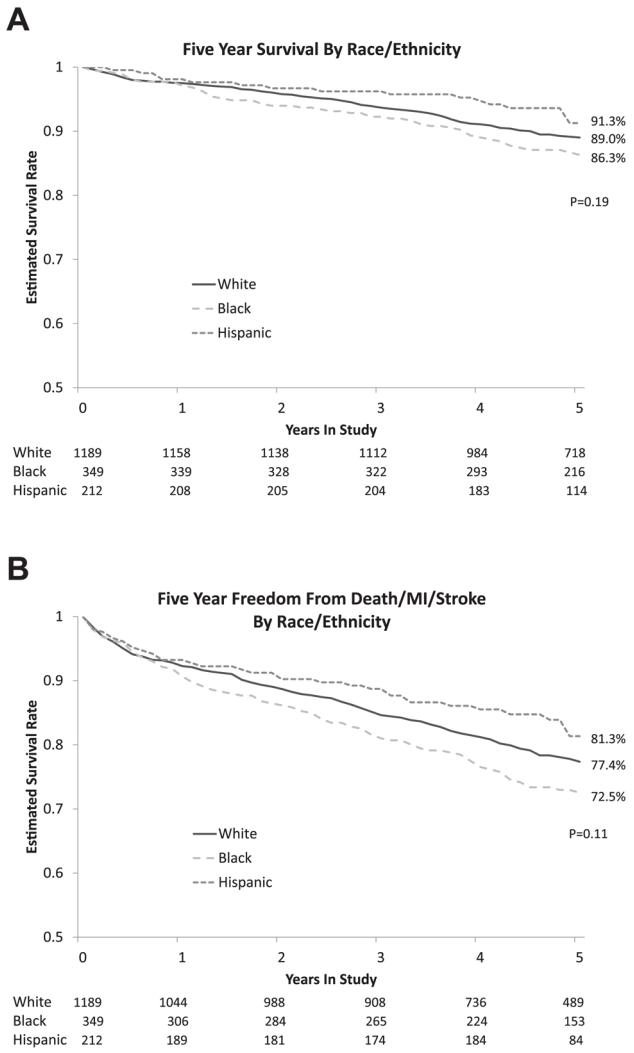

Figure 3.

Kaplan-Meier estimated rates of survival *(A)* and freedom from death/MI/stroke *(B)* by race/ethnicity after randomization in the BARI 2D trial.

Table 1

Baseline characteristics and characteristics for assigned revascularization procedures by race/ethnicity

Patient Baseline Characteristics	White (n = 1,189)	Black (n = 349)	Hispanic (n = 212)	p Value
Male	77.5%	51.0%	67.9%	<0.001
Age at study entry (yrs)	63.5, 8.8	61.5, 9.6	61.2, 8.5	<0.001
Primary insurance				
None	3.4%	10.1%	7.1%	<0.001
Medicare/public	62.8%	52.1%	65.6%	
Private	33.8%	37.8%	27.4%	
Education				
Less than high school degree	20.2%	32.1%	46.4%	<0.001
High school degree, but less than college degree	56.8%	57.8%	45.0%	
College degree or higher	23.0%	10.1%	8.6%	
Body mass index (kg/m^2)	32.7, 5.9	33.2, 7.0	31.6, 5.4	0.009
MI (history)	31.2%	27.2%	26.8%	0.221
Heart failure requiring therapy (history)	7.3%	10.8%	10.1%	0.073
Peripheral or carotid artery disease	24.7%	27.2%	30.7%	0.163
Duration of diabetes mellitus (yrs)	10.3, 8.7	11.8, 9.5	11.4, 8.8	0.012
Number of coronary regions with ≥50% stenosis	1.9, 0.9	1.8, 0.9	1.8, 0.9	0.088
Myocardial Jeopardy Index	43.8, 24.4	38.5, 23.5	40.0, 25.0	0.001
Number of coronary lesions	4.9, 2.3	4.6, 2.3	4.7, 2.2	0.023
Number of significant coronary lesions (≥50% stenosis)	2.7, 1.9	2.4, 1.9	2.6, 1.9	0.016
Treatment assignment and stratification				
MED (vs REV) assignment	51.1%	49.0%	49.1%	0.711
IS (vs IP) assignment	49.2%	51.9%	47.6%	0.523
PCI (vs CABG) stratum	73.8%	85.1%	73.1%	<.001

Index Percutaneous Coronary Intervention	White (n = 414)	Black (n = 149)	Hispanic (n = 78)	p Value
Number of lesions attempted	1.5, 0.8	1.3, 0.6	1.5, 0.7	0.043
Drug-eluting stent	36.4%	30.9%	39.7%	0.337
Bare-metal stent	54.1%	61.1%	52.6%	0.291
No stent	9.0%	8.0%	7.7%	0.824

Index Coronary Artery Graft Surgery	White (n = 173)	Black (n = 36)	Hispanic (n = 29)	p Value
Off-pump use	16.2%	22.2%	13.8%	0.609
Internal mammary arterial conduit	94.8%	94.4%	93.1%	0.933
Arterial conduits	2.8, 0.9	2.8, 0.9	2.7, 1.0	0.729
Distal anastomoses	3.3, 1.1	3.3, 1.0	3.0, 1.0	0.291

Data separated by a comma indicates mean, standard deviation.

Table 2

Medication use and risk factors at baseline and year 3 by race/ethnicity

Medication Use	Baseline				Year 3			
	White (n = 1,189)	Black (n = 349)	Hispanic (n = 212)	p Value	White (n = 971)	Black (n = 266)	Hispanic (n = 172)	p Value
Biguanide (metformin)	56.8%	47.1%	56.1%	0.01	41.5%	38.5%	46.3%	0.26
Thiazolidinone	24.5%	19.5%	27.4%	0.15	31.7%	34.2%	35.6%	0.50
Sulfonylurea	52.5%	49.4%	53.3%	0.58	39.3%	32.0%	41.2%	0.06
Insulin	26.6%	42.0%	33.0%	<.001	44.3%	52.7%	51.4%	0.005
Beta blocker	72.3%	75.1%	71.7%	0.55	84.9%	89.4%	91.5%	0.02
Angiotensin converting enzyme inhibitor	64.9%	70.2%	66.5%	0.18	64.6%	63.1%	62.1%	0.77
Angiotensin receptor blocker	15.8%	18.3%	13.2%	0.26	35.6%	38.7%	36.2%	0.64
Nonsublingual nitrate	28.1%	29.5%	32.1%	0.47	21.7%	30.3%	19.2%	0.005
Aspirin	90.1%	82.4%	84.0%	<.001	93.6%	92.3%	95.5%	0.41
Statin	77.8%	77.1%	78.7%	0.91	94.5%	93.1%	96.0%	0.40

Risk Factors	Baseline				Difference Between Year 3 and Baseline			
	White (n = 1,189)	Black (n = 349)	Hispanic (n = 212)	p Value	White (n = 971)	Black (n = 266)	Hispanic (n = 172)	p Value
HbA1c (%)	7.4, 1.4	8.0, 1.8	7.8, 1.6	<.001	−0.2, 1.4	−0.5, 1.6	−0.1, 1.6	0.014
Sitting systolic BP (mm Hg)	129.0, 17.5	136.0, 20.5	132.1, 21.0	<.001	−4.0, 19.6	−4.8, 25.0	−6.8, 20.7	0.265
Sitting diastolic BP (mm Hg)	71.4, 10.1	76.4, 10.6	73.8, 10.7	<.001	−3.6, 11.1	−4.5, 12.7	−6.0, 12.0	0.038
Total cholesterol (mg/dl)	163.9, 39.9	174.8, 43.0	168.3, 36.7	<.001	−16.7, 42.8	−17.7, 49.0	−17.6, 41.0	0.939
LDL (mg/dl)	90.3, 30.8	105.7, 38.0	95.3, 31.0	<.001	−13.6, 34.0	−18.9, 42.2	−18.0, 34.5	0.091
HDL (mg/dl)	36.5, 9.0	43.8, 13.4	39.1, 10.5	<.001	2.4, 8.7	2.0, 10.5	0.9, 8.7	0.178
Non-HDL (mg/dl)	127.4, 40.2	130.9, 41.5	129.2, 36.2	0.326	−19.0, 42.2	−19.7, 46.4	−18.5, 40.3	0.964
Triglycerides (mg/dl) *	156 (109,225)	110 (71,159)	150 (101,218)	<.001	−18.5 (−77,32)	−6.0 (−42,23)	−3.0 (−48,35)	0.008
Albumin creatinine ratio *	11.2 (5,42)	18.7 (6,79)	16.9 (7,72)	<.001	1.8 (−4,15)	0.5 (−17,26)	1.1 (−5,15)	0.445
eGFR, (ml/min/1.73m^2) *	73.5 (61,90)	81.4 (63,98)	78.2 (63,93)	0.001	−7.4 (−21,4)	−10.1 (−22,4)	−6.4 (−19,6)	0.510

eGFR = estimated glomerular filtration rate.

* Triglycerides, albumin-creatinine ratio, and eGFR are reported as median (first quartile–third quartile); all others reported as mean, SD.

Table 3

Unadjusted and adjusted hazard ratios (HR) for death and death/myocardial infarction/stroke by race/ethnicity (n = 1,750 patients)

	Unadjusted HR	95% CI	p Value	Adjusted HR*	95% CI	p Value	Adjusted HR†	95% CI	p Value
Death (n = 227)									
Black	1.20	0.89, 1.63	0.24	1.18	0.84, 1.67	0.33	1.21	0.85, 1.72	0.38
Hispanic	0.77	0.49, 1.21	0.26	0.82	0.51, 1.34	0.43	0.80	0.48, 1.32	0.29
White (reference)	1.00			1.00			1.00		
Death/MI/stroke (n = 416)									
Black	1.16	0.92, 1.46	0.22	1.08	0.84, 1.40	0.54	1.07	0.82, 1.40	0.62
Hispanic	0.78	0.56, 1.09	0.15	0.77	0.54, 1.09	0.14	0.75	0.52, 1.09	0.14
White (reference)	1.00			1.00			1.00		

CI = confidence interval.

*Adjusted for baseline variables of age, gender, education, insurance status, non-HDL cholesterol, systolic blood pressure, HbA1c, albumin creatinine ratio >30, smoking history, body mass index, history of myocardial infarction, history of congestive heart failure, duration of diabetes, current insulin use, number of diseased coronary regions, total number of lesions, randomized glycemic treatment, and cardiovascular treatment.

†Adjusted for baseline variables in above plus annually updated non-HDL cholesterol, systolic blood pressure, and HbA1c.