Racial disparities in the prevalence of cardiovascular disease among incident end-stage renal disease patients

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Abstract

Background. Prevalence of coronary heart disease (CHD) and heart failure (HF) is higher among blacks as compared with whites in general population. This study describes unexpected racial differences in the prevalence of CHD and HF among incident dialysis patients, with whites being at a disadvantage.

Methods. Data were obtained from Centers for Medicare and Medicaid Services (CMS) 2728 form for incident dialysis patients in Georgia, North Carolina and South Carolina in 1995–2003. The CHD and HF prevalence between races were compared using adjusted odds ratios (ORs). The potential for case ascertainment bias was assessed.

Results. Compared with whites (n = 23,951), black patients (n = 32,642) had lower prevalence of CHD (15.7 vs 31.2%) and HF (28.1 vs 34.1%). After controlling for age, gender, diabetes, hypertension and smoking, the association of race with CHD varied by gender and diabetes status: OR ranged from 0.36 (0.34–0.39) for non-diabetic males to 0.57 (0.53–0.61) for diabetic females. Racial differences were not fully explained by case ascertainment bias. The race-HF association varied by age, gender and diabetes: among patients aged < 55, blacks tended to have higher prevalence than whites (OR ranged from 0.99 (0.90–1.09) for diabetic males to 1.25 (1.13–1.39) for non-diabetic females), but among those aged above 55, blacks were less likely to HF (OR ranged from 0.62 (0.58–0.67) for diabetic males to 0.79 (0.73–0.85) for non-diabetic females).

Conclusions. Substantial racial disparities exist in CHD/HF prevalence among incident dialysis patients that persist after controlling for confounders and cannot be fully explained by disease misclassification.

Keywords: coronary heart disease; end-stage renal disease; heart failure; race

Introduction

Cardiovascular disease (CVD) is highly prevalent among patients with chronic kidney disease and patients with end-stage renal disease (ESRD) have cardiovascular mortality rates 10–30 times greater than the general population [1–7]. According to the United States Renal Data System (USRDS), which captures information on over 90% of incident ESRD cases, in 2002, 24.8% of the new dialysis patients had a prior history of ischaemic heart disease, which included coronary artery bypass graft (CABG), angioplasty and diagnoses of coronary artery disease (CAD)/coronary heart disease (CHD) [8]. Validation ofUSRDS data with clinical records showed a significant underreporting of comorbidities within the system, so that the true prevalence of ischaemic heart disease prior to the onset of renal replacement therapy might be as high as 42% [9].

Racial disparities in the prevalence of CVD among incident ESRD patients in the US have been reported, with blacks having lower age-adjusted estimates than whites [8,10–12]. These observations in the US ESRD population stand in contrast to the age-adjusted prevalence of CHD and heart failure (HF) in the general population which is higher in blacks [13]. The National Health and Nutrition Examination Survey (NHANES) and NHANES epidemiological follow-up studies suggest that the CHD prevalence is higher among blacks compared with white women, while among men it is comparable or slightly lower for blacks [13–16]. Even considering possibly lower prevalence of CHD in blacks compared with white males in the general population, finding significantly lower prevalence of CHD in blacks compared with whites in both gender groups in the incident ESRD population is unexpected.
Among the four studies that have reported disparities in CVD prevalence among dialysis patients, two used small or non-representative samples of the ESRD population [11,12]. The reports from USRDS [8], should also be interpreted with caution, since racial disparities in the classification of CVD on the medical evidence report form used by USRDS have been reported [9]. Therefore, we sought to confirm the findings of racial differences in the prevalence of CVD among ESRD patients after accounting for the possibility that misclassification of CVD can explain these disparities.

Subjects and methods

Study population and data

We used de-identified data on all ESRD patients in Georgia, North Carolina and South Carolina who initiated dialysis between 1995 and 2003. The data were collected by dialysis facilities using the Medical Evidence Report form (CMS-2728), which is completed by treatment centre staff on each incident ESRD patient entering the Medicare programme. The following information was obtained from this form: date of birth, sex, race; weight and height; primary cause of renal failure; comorbidities (including HF; CAD; myocardial infarction (MI); cardiac arrest, cardiac dysrhythmia; pericarditis; cerebrovascular disease; peripheral vascular disease; history of hypertension; diabetes); smoking status; erythropoietin administration prior to dialysis initiation and laboratory testing results (creatinine clearance, haemoglobin, and serum albumin at the initiation of treatment).

The CVD was considered to be present if noted on the Medical Evidence Report form and, if not recorded, to be absent. No independent validation or verification of the recorded history was obtained. We coded as yes/no a history of pre-existing HF, MI and CAD, defined as prior CABG, angioplasty, or diagnoses of CAD. A composite variable, CHD, was created to indicate a history of CAD and/or MI. Presence of CHD and HF were considered the main outcome variables in the analysis. Race was categorized as white or black and we excluded patients from the analysis whose race was neither black nor white.

Diabetes mellitus and hypertension were considered present if listed as either primary cause of ESRD or comorbidity on the CMS-2728. Body mass index was calculated using weight and height measurements recorded on the CMS-2728. We computed a residual glomerular filtration rate (GFR) for each patient using the four-variable MDRD study equation:

$$GFR = 186 \times (\text{creatinine})^{-1.154} \times (\text{age})^{-0.203} \times (0.742, \text{ if female}) \times (1.210, \text{ if black})$$

Statistical data analysis

We compared gender–race-specific prevalence of CVD among incident ESRD patients and examined the race-specific prevalence of CHD and HF across the 5-year age groups. Means and proportions were used to describe incident ESRD patient characteristics by presence of CHD and HF.

Independent associations of race with CHD and HF were assessed by fitting multiple logistic regression models including potential confounders [age, gender, diabetes, hypertension, smoking status, BMI, laboratory testing results (GFR, serum albumin, haemoglobin) and history of erythropoietin use]. All the variables were forced in the models and the confounding factor was evaluated by looking at the change in the parameter estimate for race effect after removing variables one at a time from the model. If removal of the variable did not result in significant change of parameter estimate (>10%) then the variable was dropped from the model. Interactions between race and other patient’s characteristics were assessed by including the product terms of race with each of the other predictors in the models. The interaction terms that were statistically significant at 0.05 level were kept in the final models.

Since the data on laboratory testing results were not recorded on CMS-2728 form uniformly [serum creatinine measurements were missing for 1235 patients (2.2%), albumin—10 403 patients (18.4%), haemoglobin—4067 patients (7.2%)], the models including these variables were fitted on the subset of patients with no missing data. The laboratory measurements did not confound the relationship of interest and the final models without these variables were run on the full dataset.

All analyses were performed using SAS computer software version 8.2 [17].

Misclassification bias

We assessed the magnitude of bias potentially caused by differential accuracy of a diagnosis of CAD among blacks and whites within a surveillance system by correcting our analyses for race-specific diagnostic specificity and sensitivity for the form CMS-2728. Comorbidity reporting on form CMS-2728 is voluntary, thus the prevalence of CVDs assessed using the form may be underestimated by as high as 50%. A previous study found that the adjusted sensitivity of a diagnosis of comorbid CAD on the form CMS-2728 was 0.34 for blacks and 0.49 for whites (P-value < 0.05) [9]. The specificity for both races was higher than 0.95. Lower sensitivity of documenting CVDs in blacks could explain their lower reported prevalence of these conditions compared with whites in our study. To assess the extent to which differential misclassification explains the detected racial disparities in CAD prevalence, we computed OR estimates corrected for misclassification of comorbid CAD using the previously reported race-specific sensitivities and specificities ranging from 0.95 to 1.0. We then compared the corrected OR with the observed OR to evaluate the magnitude and the direction of bias.

Results

There were 58 464 patients who started dialysis in Georgia, North Carolina and South Carolina between January 1995 and September 2003. After patients whose race was neither black nor white were excluded, a total of 56 593 individual records were available for the analysis.

The overall mean age of the patients was 60.3 years (SD: 15.7). There were approximately equal
proportions of males and females (49.8 and 50.2%, respectively). About 58% of the patients were black. Compared with whites, African Americans were younger, had higher mean body mass index (BMI), and were more likely to be males, less likely to be smokers, and more likely to have a history of diabetes and hypertension (Table 1). White patients were more likely to receive erythropoietin before dialysis initiation compared with black patients. In addition, black patients had lower values of serum albumin, haemoglobin, and GFR (Table 1).

The prevalence of CHD among incident ESRD patients was 22.3%; 19.7% of patients had a history of CAD and 7.9% a history of MI. The HF prevalence was 30.7% (Table 2). The prevalence of CVD was substantially lower in blacks than in whites. In the univariate analysis, the black to white OR (95% CI) for CHD was 0.41 (0.40, 0.43). Comparable measures for CAD were 0.41 (0.39, 0.43), for previous MI 0.45 (0.42, 0.48), and for HF 0.76 (0.73, 0.78). There were also gender differences in prevalence within the race groups. In general, males were less likely than females to have CVD, among blacks, but more likely to have CVD among whites. Nevertheless, within both genders, blacks had lower disease prevalence than whites (Table 2).

Within all age strata, blacks were less likely to have CHD as compared with whites (Figure 1).

Table 1. Socio-demographic and clinical characteristics of study patients, by race

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Race</th>
<th>P-value/OR (95% CI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Black (n = 32 642)</td>
<td>White (n = 23 951)</td>
</tr>
<tr>
<td>Mean age (SD)</td>
<td>58.1 (15.8)</td>
<td>63.5 (15.2)</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td></td>
<td>&gt;0.0001</td>
</tr>
<tr>
<td>Albumin (g/dl)</td>
<td>3.2 (1.0)</td>
<td>3.3 (0.9)</td>
</tr>
<tr>
<td>Haemoglobin (g/dl)</td>
<td>9.7 (4.1)</td>
<td>10.2 (3.6)</td>
</tr>
<tr>
<td>GFR (ml/min)</td>
<td>8.4 (5.0)</td>
<td>9.1 (5.6)</td>
</tr>
<tr>
<td>Mean BMI (SD)</td>
<td>27.6 (7.1)</td>
<td>26.5 (6.8)</td>
</tr>
<tr>
<td>Male gender (%)</td>
<td>56.0</td>
<td>45.4</td>
</tr>
<tr>
<td>Smoking (%)</td>
<td>7.0</td>
<td>8.8</td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td>53.6</td>
<td>49.8</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>88.0</td>
<td>80.3</td>
</tr>
<tr>
<td>EPO (%)</td>
<td>22.5</td>
<td>27.2</td>
</tr>
</tbody>
</table>

*Unadjusted odds ratios, interpreted as the odds of a particular characteristic (e.g. male gender) in blacks divided by the odds of that characteristic in whites.

Table 2. Prevalence of comorbid CVDs in incident ESRD patients, by gender-race group

<table>
<thead>
<tr>
<th>All Patients %</th>
<th>Black (n = 32 642)</th>
<th>White (n = 23 951)</th>
<th>Unadjusted B:W OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All (%)</td>
<td>Males (%)</td>
<td>Females (%)</td>
<td>M:F OR (95% CI)</td>
</tr>
<tr>
<td>CHD (CAD/MI)</td>
<td>22.3</td>
<td>15.7</td>
<td>14.8</td>
</tr>
<tr>
<td>CAD</td>
<td>19.7</td>
<td>13.6</td>
<td>12.7</td>
</tr>
<tr>
<td>MI</td>
<td>7.9</td>
<td>5.4</td>
<td>5.4</td>
</tr>
<tr>
<td>HF</td>
<td>30.7</td>
<td>28.1</td>
<td>25.1</td>
</tr>
</tbody>
</table>

The age-specific black–white OR varied from 0.36 (95% CI, 0.28–0.48) for patients aged 40–44 to 0.53 (95% CI, 0.47–0.59) for patients aged 60–64. Compared with whites, blacks tended to have higher HF prevalence at earlier ages (<55), but had significantly lower prevalence later in life (Figure 2).

Results of multivariable analysis for CHD are summarized graphically in Figure 3. Serum albumin, haemoglobin, GFR, erythropoietin use and BMI did not appear to confound the association between race and CHD and were dropped from the model. The final model included race, age, gender, diabetes, hypertension, smoking status and two interaction terms—race–gender and race–diabetes. The racial differences in CHD prevalence were more pronounced among non-diabetics than diabetics and among males compared with females: diabetic males—black–white OR = 0.41 (95% CI, 0.39–0.44), diabetic females—OR = 0.57 (95% CI, 0.53–0.61), non-diabetic males—OR = 0.36 (95% CI, 0.34–0.39) and non-diabetic females—OR = 0.50 (95% CI, 0.46–0.55).

Results of multivariable analysis for HF are summarized in Figure 4. As in the CHD analysis, laboratory measurements, erythropoietin and BMI were omitted from the model since these variables were not found to confound the association of interest. The final model included race, age, gender, diabetes, hypertension, smoking status and three interaction terms—race–gender, race–age and race–diabetes. First, among patients <55 years of age blacks, were slightly more likely to have HF as compared with whites. Second, within younger age group, patients with diabetes exhibited less racial discrepancy in the prevalence of HF. The black–white OR for HF among younger diabetic males was 0.99 (95% CI, 0.90–1.09) and among younger diabetic females 1.11 (1.01–1.22) compared with 1.11 (1.01–1.22) and 1.25 (1.13–1.39), respectively for non-diabetic males and females. Among those aged 55 and older, comparable values were 0.62 (0.58–0.67) and 0.70 (0.66–0.75) for diabetic males and females and 0.70 (0.65–0.75) and 0.79 (0.73–0.85) for non-diabetic males and females.

Misclassification bias

Depending on the value of specificity chosen, the bias for reporting CAD in blacks and whites could be either away from the null or towards the null (Table 3).
The corrected OR varied from 0.32 to 0.62 compared with the observed value of 0.41. However, even under the most extreme situation (specificity for whites = 0.95 and specificity for blacks = 1.00), the OR was still significantly less than 1 (Table 3).

**Discussion**

Our study brings attention to the substantial racial disparities in the prevalence of CVD among incident ESRD patients in the US. In our study, black patients had a significantly lower prevalence of CVD compared with white patients. This association persisted after controlling for the potential confounders including age, gender, comorbid diabetes and hypertension, and smoking status. Further, the racial disparity did not seem to reflect misclassification bias due to inaccurate diagnosis of CVD.

This finding is intriguing since the increased prevalence of traditional Framingham risk factors and uraemia-specific CVD risk factors among black ESRD
patients in our study would suggest a higher, not lower, CVD prevalence compared with whites. This is the case for the general US population where blacks are more likely to have adverse CVD risk factor profiles [18] and have higher prevalence and poorer control of hypertension [19–21] and diabetes mellitus [22–24]. With the exception of younger age, and slightly lower prevalence of smoking, a similar higher prevalence of traditional CVD risk factors among blacks was observed in our study. Black incident ESRD patients were more likely to be males, to have diabetes and to be hypertensive. Albumin level, which was reported to be inversely related to CVD incidence [25], was lower in black than in white patients in our population. Blacks had lower GFR in our study sample, again not suggesting their lower CVD prevalence. Finally, anaemia, a risk factor for CVD [26–28], was more severe in black patients in our study than in whites at the initiation of dialysis.
Racial differences in CVD among incident ESRD patients

Table 3. Racial differences in prevalence of CAD among incident ESRD patients corrected for disease misclassification*  

<table>
<thead>
<tr>
<th>Specificity</th>
<th>Prevalence of CAD (%)</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black</td>
<td>White</td>
<td>Black</td>
</tr>
<tr>
<td>0.95</td>
<td>1.0</td>
<td>29.74</td>
</tr>
<tr>
<td>0.95</td>
<td>0.95</td>
<td>29.74</td>
</tr>
<tr>
<td>0.98</td>
<td>0.98</td>
<td>36.32</td>
</tr>
<tr>
<td>1.0</td>
<td>1.0</td>
<td>40.07</td>
</tr>
<tr>
<td>1.0</td>
<td>0.95</td>
<td>40.07</td>
</tr>
</tbody>
</table>

*Based on sensitivity = 0.34 for blacks, and sensitivity = 0.49 for whites and different sets of specificities.

Young et al. [12] has previously reported that the prevalence of MI or ischaemic heart disease, angina, coronary atherosclerosis, cardiac dysrhythmias and HF in the population of diabetic veterans with ESRD was lower for blacks compared with whites (51.6 vs 65.4%, respectively). Although bringing attention to the racial difference in CVD prevalence, the findings of Young et al. [12] could not be generalized to the population of incident ESRD patients since the authors studied a very special group of individuals (veterans with diabetes). Our results generalize the findings by Young et al. [12] to the population of incident ESRD patients regardless of their diabetes status.

Our study findings are also consistent with the reports of Stack and Bloembergen [10] and Cheung et al. [11]. Stack and Bloembergen [10] used data from the dialysis morbidity and mortality study wave 2, a national random sample of incident ESRD cases in the US in 1996–97, and noted lower prevalence of CAD in blacks compared with whites (37 vs 43%, respectively). After adjustment for conventional CAD risk factors as well as uraemic factors, geographic region, elements of pre-ESRD care and comorbidities, whites had 1.85 times greater odds of having CAD compared with blacks. Cheung et al. [11] focused their analysis on 936 haemodialysis patients enrolled in the baseline phase of the haemodialysis study and noted that after adjusting for traditional Framingham risk factors, blacks had lower odds of having CHD compared with whites (OR = 0.64, P-value = 0.017). We confirm here the findings by Stack and Bloembergen [10] and Cheung et al. [11] by using a bigger sample of incident dialysis patients, including more recent data. Finally, the USRDS has reported racial differences in CVD prevalence among incident dialysis patients [8]. Our study extends USRDS findings by addressing the issue of under-reporting and possible differential misclassification of disease status on the Medical Evidence Report form (CMS-2728).

Our analysis found that the racial differences in CAD prevalence persisted after correcting for potential bias caused by differential ascertainment of CAD in blacks and whites on the CMS-2728 form. These results suggest that while the strength of association might be underestimated in our study, misclassification bias is unlikely to explain completely the observed racial differences in CVD prevalence. A potential limitation of our approach to correcting for misclassification is that we could not account for the uncertainty in the specificity/sensitivity estimates, since the confidence intervals were not reported in the validation study. Longenecker et al. [9] presented race-specific sensitivities for CAD diagnoses adjusted for age, gender, modality, number of comorbid conditions, calendar time and the presence of diabetes. To obtain the sensitivities, they used generalized estimating equation models taking into account dialysis centre effects (clustering of coding patterns within centre) on sensitivity. Given the data provided in the article, we could not reproduce authors’ analysis to obtain the confidence intervals for sensitivity of CAD diagnoses for blacks and whites. There is also a possibility that misclassification bias was not fully eliminated since CHOICE Study estimates of sensitivity and specificity obtained using the sample of patients treated in 19 states were applied to the data originated from three southern states. The race-specific patient mix in our study could be different from the CHOICE patient mix. In addition, even regardless of patient mix there could be different coding patterns within facilities in our study and CHOICE Study, resulting in different sensitivity/specificity of diagnoses of comorbid conditions. We were unable to estimate the magnitude of these possible differences and acknowledge this as a limitation of our approach to addressing disease misclassification. Independent validation of diagnoses of comorbid conditions, a gold standard approach, could not be performed due to lack of resources.

The observations reported here are of interest, in part, for the questions they generate. For example, are the ethnic differences we note for the US population observed in other multiracial populations, including those of African descent? Absence of these differences across populations would provide further support for the possibility that variations in prevalent CVD among incident ESRD patients reflects differences in healthcare and survival of African-Americans prior to the onset of ESRD. In contrast, persistent differences would provide support for recent conjectures that kidney disease progresses more rapidly in African-Americans with CKD [29]. Further, can the lower prevalence of cardiovascular conditions in black as compared with white incident ESRD patients be attributable to differences in pre-ESRD survival among different race groups with CVD? It is possible that blacks with chronic kidney disease are more likely to die of CVD before reaching ESRD than whites. This would leave a healthier pool of black patients initiating renal replacement therapy and black race would appear protective on CVDs, especially in the older age groups. This possibility is consistent with our observations for HF. We found that higher HF prevalence in whites as compared with blacks was evident only in patients >55 years, and among younger patients blacks tended to have higher HF prevalence. Unfortunately, with the prevalence data available to us we were unable to address the possible differential survival in the analysis.
and the published literature is contradictory on this point. In the analysis of the pooled data from ARIC Study, Cardiovascular Health Study, Framingham Heart Study and Framingham Offspring Study, Weiner et al. [6] found that among patients with impaired renal function free of CVD at baseline, blacks had a significantly higher 10-year probability of developing MI, fatal CHD, fatal and nonfatal stroke or dying from any cause than whites. These findings would be supportive of the hypothesis of differential survival to ESRD among blacks and whites with CKD. On the contrary, Smith et al. [30] found that black patients had lower mortality risk than white patients at every level of kidney function in a cohort of Medicare patients hospitalized with HF. Similar findings were reported by Newsome et al. [31] for a nationally representative cohort of patients with acute MI—among those with GFR 15–30 ml/min, blacks had significantly better survival than whites. The findings by Smith et al. [30] and Newsome et al. [31] would not suggest the higher mortality rates in blacks. These conflicting results support the need for further mortality studies assessing the race-specific CVD mortality among patients at different stages of chronic kidney disease, which would eventually bring further insight into differential survival hypothesis.

Another possibility for the observed racial differences in CVD prevalence is that, blacks may be exposed to risk factors that promote more rapid progression to ESRD while delaying the development of CVD. For example, it was noted that hyperexpression of the cytokine TGF-β playing a role in the progression of kidney disease [32,33] is more frequent in blacks than whites [34–36]. It has also been suggested that TGF-β can function as an inhibitor of atherosclerosis [32,37,38]. Thus, increased expression of TGF-β in blacks might pre-dispose them to more aggressive loss of renal function, and at the same time might have a protective effect on the development of atherosclerosis.

It is also possible that there are other factors that could be responsible for the observed racial differences in CVD. For example, our findings that black subjects had lower baseline haemoglobin, but were less likely to receive erythropoietin in pre-ESRD stage, and that they were less likely to start dialysis with lower residual GFR as compared with whites may be suggestive of the racial differences in access to care. If black patients had worse access to healthcare, than they may have been less likely to be diagnosed with CVD. This would bias our results away from the null. On the other hand, as compared with whites, black patients can be expected to be using less antihypertensive, cholesterol lowering or other medications with cardiovascular effects. We could not adjust for medications use other than erythropoietin in our study due to the lack of data. However, most likely this limitation had resulted in more conservative estimates of black–white differences in CVD prevalence (bias toward the null).

In addition, we could not control for hypercholesterolaemia, an important Framingham risk factor, and several non-traditional risk factors which may be important predictors of an excess CVD risk among ESRD patients (e.g. homocysteine, C-reactive protein). One of the future research steps could be to examine the distribution of these non-traditional risk factors by race and to see to what extent, if any, they are responsible for the observed racial differences in CVD prevalence among ESRD patients.

The major strength of our study is that it used the data from a population-based surveillance system and addressed the issue of disease underreporting and misclassification within the system. One limitation is that patients from only three US states (Georgia, North Carolina and South Carolina) were included, and about 58% of the patients in the sample were black. In contrast, about 30% of the US dialysis population is black [8]. Although this limits the generalizability of our findings to the US ESRD population, our results are consistent with the reports or studies based on the national data [8,10].

In conclusion, racial differences in CVD prevalence exist in ESRD patients, with whites having a higher prevalence of CHD and HF. The observed disparities are not likely to be explained by disease misclassification. Our findings generate hypotheses for explaining racial differences in CVD prevalence in ESRD population. One hypothesis that should be investigated in further studies is that differential survival in pre-ESRD stage is responsible for the lower prevalence of CVD in black compared with white ESRD patients.

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Conflict of interest statement. None declared.

References
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