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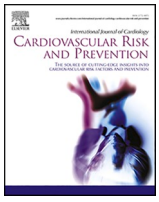

Depression and incident cardiovascular disease among patients with chronic kidney disease

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ABSTRACT

Background: Depression is associated with an increased risk of cardiovascular disease (CVD) and is prevalent among patients with chronic kidney disease (CKD). We aimed to identify the association of depression with incident CVD.

Methods: We studied patients with CKD stages 2–4 enrolled in the Chronic Renal Insufficiency Cohort (CRIC) and excluded participants with preexisting CVD. The Cox proportional hazard model was used to examine the association between baseline depression [Beck's Depression Inventory (BDI) score ≥11] and incidence of CVD (cerebrovascular accident, myocardial infarction, heart failure, or peripheral artery disease). Models were adjusted for age, sex, race, estimated glomerular filtration rate (eGFR), urine albumin-creatinine ratio (UACR), systolic and diastolic blood pressure, and 10-year estimated CVD risk.

Results: Among 2585 CRIC study participants, 640 (25%) patients had depression at study baseline. Compared to patients without depression, patients with depression were more likely to be women (56% vs. 46%), non-White (68% vs. 53%), with household income <$20,000 (53% vs. 26%), without a high school degree (31% vs. 15%), uninsured (13% vs. 7%), with lower eGFR (42 vs. 46 ml/min/1.73 m (Palmer et al., 2013 Jul)22), and with higher UACR (90 vs. 33 mg/g). In multivariate analyses, depression was associated with a 29% increased risk of developing CVD (adjusted hazard ratio 1.29, 95% confidence interval 1.03–1.62, p = 0.03). BDI (as a continuous variable) was associated with CVD (adjusted hazard ratio 1.017, 95% confidence interval 1.004–1.030, p = 0.012).

Conclusions: Among patients with CKD stages 2–4 enrolled in CRIC without preexisting CVD, depression was associated with a 29% increased risk of incident CVD.

1. Introduction

Depression, chronic kidney disease (CKD), and cardiovascular disease (CVD) are common major causes of morbidity and mortality in the United States. While the prevalence of depression among the general United States population is approximately 5% [1], the estimated prevalence of depression specifically among patients with CKD is 25% [2], and among patients with CVD is as high as 45% [3].

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Depression is a known risk factor for CVD, yet clinically significant depression is often unrecognized or goes untreated until a cardiac event requires hospitalization [3]. Among patients diagnosed with depression during hospitalization for a cardiac event, about 50–70% had preceding signs [4]. CKD is also a known risk factor for CVD, as up to 50% of patients with CKD have concomitant CVD compared to 26% in controls without kidney disease [5]. Because over 40% of mortality among patients with CKD is related to cardiovascular causes [6], it is important to identify additional predisposing risk factors to CVD among patients with CKD. Previous studies have evaluated the cardiovascular risk of depression among patients with severe CKD [7–9], but less is known about the association between depression and CVD among patients with mild-to-moderate CKD. The aim of this study was to assess the prevalence of depression and its association with incident CVD over time, in a large national cohort of individuals with CKD. We hypothesized that depression is associated with higher incidence of CVD among patients with CKD.

2. Methods

The Chronic Renal Insufficiency (CRIC) study is a prospective longitudinal cohort study that recruited participants aged 21–74 years old from 7 centers (13 clinical sites) with an estimated glomerular filtration rate (eGFR) of 20–70 ml/min/1.73 m [2] starting in 2003. Participants are seen at annual clinic visits and contacted by telephone every 6 months [10].

Patients with pre-existing CVD, as indicated on the past medical history questionnaire, were excluded. Self-reported history of any CVD at baseline included previous myocardial infarction, heart failure, stroke, or peripheral arterial disease. Patients with missing data on depression scores were also excluded. The remaining patients were divided into 2 groups based on their responses to the validated Beck's Depression Inventory (BDI), in which higher scores indicate more severe depression [11]. Because scores ≥11 indicate clinically significant depression [12], the 2 groups were defined as: 1) BDI≥11 to represent patients with depression and 2) BDI<11 to represent patients without depression.

Data collected for each patient included patient-reported past medical history, behavioral health assessments, and anthropometric data including calculated body mass index (BMI). Demographic characteristics collected for each patient included age at enrollment, sex, race and ethnicity, annual household income, education level, health insurance status at the initial visit, and the Kidney Disease Quality of Life (KDQOL) self-reported quality of life assessment specific to patients with kidney disease [13]. Additional baseline characteristics included: systolic and diastolic blood pressure, BMI, eGFR, urine albumin to creatinine ratio (UACR), history of tobacco use, and history of diabetes and hypertension. At baseline and follow-up, the estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI creatinine equation [14]. The Framingham 10-year CVD risk was calculated for each patient [15].

Outcomes were adjudicated by 2 physicians using standardized definitions [10]. The primary outcome was incident composite cardiovascular events (myocardial infarction, cerebrovascular accident, heart failure, and peripheral arterial disease) ascertained during the course of the study by asking participants about hospitalizations during the clinic or phone visit. Secondary outcomes included incidence of each of the individual CVD components listed above, in addition to atrial fibrillation.

Standard descriptive statistics were used to characterize the study cohort at CRIC study baseline, stratified by depression groups. Continuous variables were expressed with median values (interquartile range [IQR]) and compared using Wilcoxon rank-sum tests. Categorical variables were expressed as frequencies or proportions and compared with the Chi-squared test. Then, using *a priori* determined clinically significant variables, multivariate Cox proportional hazard ratios were used to determine the association between depression and incident composite CVD in Model 1. Variables used for adjustments included: sex, age, race, eGFR, UACR, systolic blood pressure, diastolic blood pressure, and Framingham 10-year CVD risk. Additionally, using the same covariates as in Model 1, hazard ratios were generated analyzing the association between BDI≥11 and each of the secondary outcomes.

Subgroup analysis was performed in the following subgroups to determine the association of BDI ≥11 with the primary outcome of CVD: age <65, age ≥65, male sex, female sex, White race, Non-White race, eGFR ≥45 ml/min/1.73 m [2], eGFR <45 ml/min/1.73 m [2], Framingham Risk Score <20%, and Framingham Risk Score ≥20%.

One multivariate model was built similar to Model 1 in which BDI was analyzed as a continuous rather than binary categorical value. A supplemental model included all the variables of Model 1 plus self-reported anti-depressant usage at the time of enrollment. These models were compared using the likelihood ratio test, and the hazard ratio ascribed to depression was calculated for each model.

Kaplan Meier survival curves were generated for incident CVD and compared between patients with BDI≥11 and BDI<11 as well as for each BDI quartile. Kaplan Meier survival curves also compared incident CHF, atrial fibrillation, MI, PAD, and CVA between the 2 groups. Statistical analysis was performed using R Statistical Computing Environment (R Foundation for Statistical Computing, Vienna, Austria) version 4.1.2 [16].

3. Results

The CRIC study enrolled 3939 patients. Of these, 1316 (33%) were excluded due to pre-existing CVD, and 38 were excluded due to missing BDI scores, leaving 2585 to be included in the final study population. Of the 2585 study patients, 640 (25%) were included in the group with depression while 1945 (75%) were in the group without depression.

Compared to patients without depression, patients with depression were significantly more likely to be women (56% vs. 46%; $p < 0.001$), non-White (68% vs. 53%; $p < 0.001$), with annual household income <$20,000 (53% vs 26%; $p < 0.001$), without a high school education (31% vs. 15%; $p < 0.001$), and without health insurance (13% vs. 7%; $p < 0.001$).

Patients with depression were more likely to have comorbid hypertension (87% vs. 82%; $p = 0.002$) and diabetes (51% vs. 39%; $p < 0.001$) with their CKD. Additionally, patients with depression self-reported their general health as worse than patients without depression ($p < 0.001$). Data indicated that patients with depression had higher rates of obesity ($p < 0.002$), lower median eGFR (42 vs. 46 ml/min/1.73 m [2], $p < 0.001$), and higher UACR (90 vs. 33 mg/g, $p < 0.001$).

After a median follow up of 8.01 years, 398 participants developed incident CVD, including 124/640 patients (19%) with depression and 274/1945 (14%) patients without depression. After adjustment for age, race, GFR, UACR, systolic and diastolic blood pressure, and Framingham 10-year risk score, participants with a BDI ≥11 were associated with a 29% higher risk of developing incident CVD (HR: 1.29, 95% CI: 1.03–1.62; $p = 0.03$). Of the analyzed covariates, depression was associated with the largest risk of incident CVD. Using Model 1, but with BDI included as a continuous instead of categorical variable, BDI was still significantly associated with increased risk of incident CVD (HR: 1.02, 95% CI: 1.00–1.03; $p = 0.01$).

Regarding secondary outcomes, 248 (9.6%) participants had incident CHF, 204 (7.9%) had incident atrial fibrillation, 134 (5.2%) had incident MI, 69 (2.7%) had incident PAD, and 65 (2.5%) had incident CVA. After multivariable adjustments, BDI ≥11 was associated with a HR of 1.30 for incident CHF (95% CI: 0.98–1.73, $p = 0.07$), 1.18 for incident MI (95% CI: 0.79–1.76, $p = 0.42$), 1.605 for incident PAD (95% CI: 0.94–2.73, $p = 0.08$), 1.83 for incident CVA 1.83 (95% CI: 1.06–3.17, $p = 0.03$), and 1.14 for incident atrial fibrillation (95% CI: 0.80–1.62, $p = 0.47$) (Supplemental Table 1).

Upon subgroup analysis by age, sex, race, eGFR, and Framingham risk score, BDI ≥11 was consistently associated with increased risk of

composite CVD, but the results were not always significant (Supplemental Table 2). Depression remained a significant risk factor for CVD among patients younger than 65, patients of any sex or race, patients with eGFR<45, and patients with Framingham Risk Scores ≥20%. Depression was most strongly associated with incident CVD among patients with Framingham Risk Scores ≥20% (HR 1.43, 95% CI: 1.07–1.92; p = 0.02), age <65 (HR: 1.41, 95% CI: 1.08–1.83; p = 0.01), and eGFR <45 (HR 1.37, 95% CI: 1.05–1.79; p = 0.02).

In a supplemental model, antidepressant medications had no significant association with the risk of incident CVD (HR: 1.11, 95% CI: 0.83–1.48; p = 0.50), but the risk of incident CVD among patients with BDI ≥11 remained largely the same (HR 1.28, 95% CI: 1.01–1.62; p = 0.04). The likelihood ratio test demonstrated that Model 1 had a better fit than the supplemental model (p = 0.59, null hypothesis that Model 1 has better fit was not rejected).

Kaplan Meier plots illustrating CVD-free survival are shown in Figs. 1 and 2. Composite-outcome disease-free survival curves are significantly different from each other when compared between patients with BDI <11 and ≥ 11 (p < 0.001) or when compared between patients of each BDI quartile (p < 0.05) (Fig. 1).

Individual-outcome disease-free survival curves were significantly different between patients with BDI <11 or BDI ≥11 when assessing for heart failure (p < 0.001), CVA (p = 0.003), and PAD (p = 0.006) but no significant difference was seen while assessing for MI (p = 0.085) or atrial fibrillation (p = 0.81).

4. Discussion

In a large cohort of adults with CKD, participants with a BDI≥11 had 29% higher risk of incident CVD when accounting for demographics, comorbidities, medications and kidney function. The results were consistent in subgroup analyses and most significant in patients age <65, with eGFR <45, or Framingham Risk Score of 20% or greater. Our results add to existing literature that demonstrates a significant association between depression and CVD and is among the first with an emphasis on mild-to-moderate CKD.

The association between depression and CVD has been demonstrated among several sociodemographic populations including women, men, the elderly, and people who identify as Black [17–19]. Additionally, the association between depression and CVD has been shown among patients with a variety of chronic illnesses including hypertension [20], diabetes [21], and obesity [22]. Regarding the association of depression and CVD among patients with CKD, most prior studies have either focused exclusively on African-Americans or patients with an end-stage renal disease requiring dialysis [7,8]. The increased risk of CVD we demonstrated among patients with CKD and depression, indicates that depression is an independent and additive risk factor for the development of CVD in CKD. We found that of all the variables included in our multivariate analysis, depression carried the largest magnitude of risk of incident CVD. This result is similar to a prior study that found depression to carry a greater risk of CVD than other, more traditional, risk factors [17]. As depression is a potentially modifiable risk factor, these results have important clinical implications for the screening and management of depression in patients with CKD.

Interestingly, we found that the addition of antidepressant medication to the multivariate model had no major effect on the relationship between depression and incident CVD. While our data does not reflect more granular data such as whose depression improved with antidepressant medication, or the length or type of antidepressant medication they were taking, this result suggests that antidepressant medication alone may not be enough to mitigate the risk of depression in the development of CVD. A prior prospective cohort study suggests that antidepressant medication (particularly tricyclic antidepressants) carries an increased risk of incident major adverse cardiovascular events [23], and prior randomized control trials have found that antidepressant medication does not protect patients with pre-existing congestive heart failure from hospitalization or all-cause mortality [24,25]. While our results, taken in the context of these prior studies, do not endorse

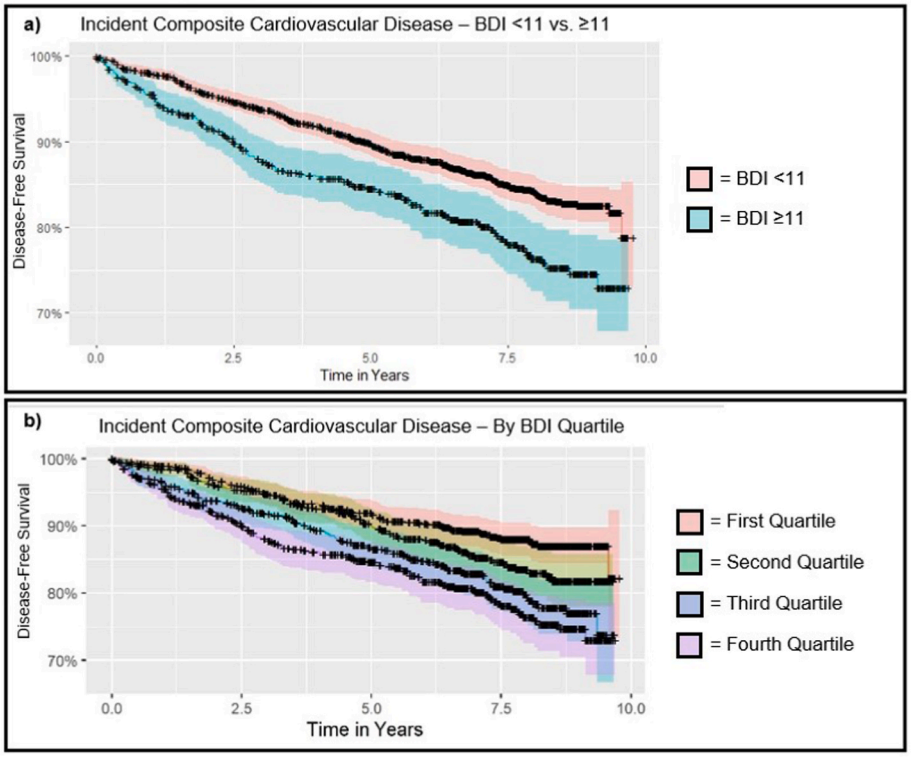

Fig. 1. Composite-outcome disease-free survival curves, **a)** compared between patients with BDI <11 vs BDI ≥11 (p < 0.001), **b)** compared between patients in each quartile of BDI score (p = 0.049).

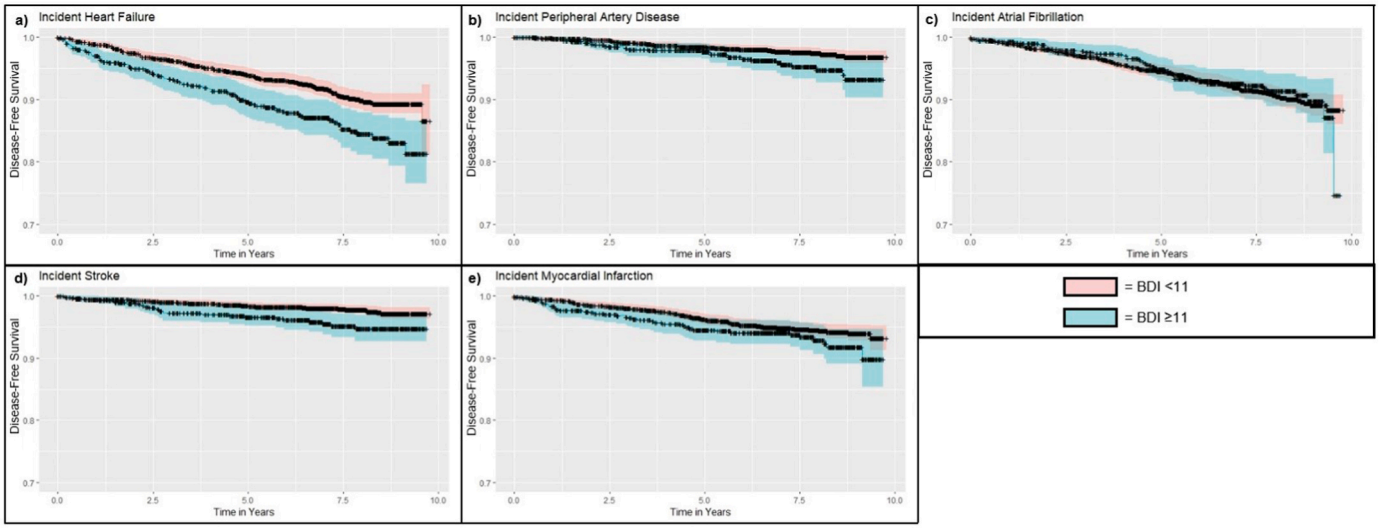

Fig. 2. Individual-outcome disease-free survival curves, compared between patients with BDI <11 vs BDI≥11. **a)** Incident heart failure ($p < 0.001$), **b)** incident peripheral artery disease ($p = 0.006$), **c)** incident atrial fibrillation ($p = 0.810$), **d)** incident cerebrovascular accident ($p = 0.003$), **e)** incident myocardial infarction ($p = 0.085$).

antidepressant medication as protective against heart disease, it should be noted that antidepressant medication is effective in the treatment of depression alone, and that other modalities including psychotherapy and access to social services are also effective in the treatment of depression but have not been extensively studied in their relationship to CVD risk in the setting of CKD.

Importantly, our results corroborate many socioeconomic disparities associated with depression, including lower educational attainment, lower household income, and increased likelihood of being uninsured [26]. These results support the likely multifactorial contribution of depression to heart disease. Many physiological and behavioral hypotheses have been studied to explain the potential mechanisms by which depression increases the likelihood of CVD [26]. From a physiologic standpoint, studies have demonstrated increased autonomic activity, increased inflammation, and accelerated atherosclerotic changes among patients with depression. Behaviorally, patients with depression are less likely to maintain a healthy diet, engage in regular exercise, or adhere to medication recommendations. The adverse socioeconomic conditions associated with depression have the potential to fuel both deleterious physiologic stress responses and behavioral adaptations that contribute to heart disease. Furthermore, lower educational attainment has been directly linked with lower compliance with medical recommendations, likely leading to accelerated progression of predisposing chronic illnesses [27]. Thus, these results suggest why antidepressants alone may be insufficient in the treatment of depression and its associated risk of incident heart disease.

Given the high prevalence of depression among patients with CKD and the additive risk of depression for the development of CVD, our results suggest that patients with CKD may benefit from regular depression screening initiated early in the CKD course. This is especially true among patients with more advanced CKD, or patients with other CVD risk factors, as our subgroup analyses demonstrated a stronger association of depression and incident CKD among these patients. Screening can be accomplished with an efficient 2-question survey that is roughly 80% sensitive and 90% specific for the detection of clinical depression and is recommended by the United States Preventive Services Task Force (and comparable to the BDI in a review among cardiac patients) [28]. While depression is often exacerbated by upstream socioeconomic factors, screening can be efficacious in connecting patients to appropriate social and medical resources and reducing morbidity and mortality related to depression and other chronic medical conditions [29,30].

This study has a few important strengths. It represents a comprehensive analysis of the association between depression screening and rigorously adjudicated cardiac outcomes in patients with CKD stages 2–4. Additionally, the large and racially diverse patient population, long duration of follow-up, comprehensive covariate measurements, and large subgroups allowed for robust subgroup analyses.

4.1. Study limitations

Our study also has several limitations. There are many differences between the 2 patient groups studied. While we attempted to control for confounding variables, it is possible the significant baseline differences between the 2 groups affected our final analysis. Additionally, we are unable to glean from the data how long patients have had depression and to what extent they are engaged in medical or psychotherapy. While we did attempt to control for this by including antidepressant medication in our supplemental multivariate model, we were unable to capture the type, dose, or response of each medication. Finally, while the data for this study are collected prospectively, our analysis was retrospective, necessarily predisposing our analysis to additional confounding variables.

5. Conclusions

Among patients with mild to moderate CKD enrolled in CRIC without preexisting CVD, depression was associated with a 29% increased risk of developing CVD after accounting for socioeconomic and medical factors. Clinicians should regularly incorporate screening for depressive symptoms in order to identify potential remedies that may mitigate risk of incident CVD in individuals with CKD.

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Declaration of competing interest

none.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcrp.2023.200199.

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