Published in final edited form as:

Health Psychol. 2010 November ; 29(6): 594–600. doi:10.1037/a0021235.

Association Between Depressive Symptoms and Mortality Risk in Chronic Kidney Disease

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Abstract

Objective—Previous examinations of depression as a predictor of mortality in end-stage renal disease have yielded inconsistent findings. We sought to clarify the possible link with mortality by assessing depression at an earlier stage of renal impairment before the uremic disease state and depressive symptoms become highly confounded, and then following patients during the period of disease progression.

Design—Prospective design using an assessment of depression prior to initiation of renal replacement therapy to predict mortality status an average of 81 months later in patients in the early stages of chronic kidney disease.

Main Outcome Measures—Mortality status.

Results—After controlling for relevant mortality risk factors (i.e., age, gender, presence of diabetes and cardiovascular disease, and potassium level), results of Cox regression analyses indicated that higher levels of nonsomatic depression symptoms were predictive of an increased mortality risk, $\chi^2(1, N=359) = 8.02, p = .005$. Patients with nonsomatic depression scores one standard deviation above the mean had an estimated mortality rate 21.4% higher than average scorers in this sample.

Conclusion—Clinical implications of these findings point to the importance of assessment and treatment of depressive symptoms in patients with chronic kidney disease.

Keywords

chronic kidney disease; depression; mortality

Chronic kidney disease (CKD) affects approximately 31 million people in the United States, at varying stages of severity (U.S. Renal Data System, 2008). CKD is associated with significant morbidity and mortality and, in many cases, eventually progresses to “end-stage”

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renal failure requiring renal replacement intervention (i.e., renal dialysis or transplantation) to sustain life. Given that CKD typically develops as a complication secondary to diabetes or hypertension, poor control of blood glucose and blood pressure are significant predictors of morbidity in this population and often accelerate the progression of renal failure to end-stage disease (National Kidney Foundation, 2002). Cardiovascular events are also highly correlated with mortality in CKD (Anderson et al., 2009; U.S. Renal Data System, 2008). In addition, nutritional factors, as measured by low levels of serum albumin (Menon et al., 2005), increases in serum phosphate (Kestenbaum et al., 2004), and potassium levels (National Kidney Foundation, 2002), have been found to predict higher risk of all-cause mortality in patients with early stage CKD.

Despite the increasing attention CKD has recently received in the literature due to recognition of its significant public health burden, there remains a paucity of research on the psychosocial factors that may be related to outcomes in this population. Although considerable research has examined the influence of psychological variables on patients in the end-stage of renal disease (e.g., Kimmel, 2002; Kimmel, Weihs, & Peterson, 1993), only a few such studies have involved patients with earlier stages of CKD who are not yet dependent on renal replacement therapy (e.g., Christensen et al., 2002; Cvengros, Christensen, & Lawton, 2005; Shidler, Peterson, & Kimmel, 1998). With the exception of one study that examined the effects of depression on 12-month mortality on inpatients with severe CKD and comorbid congestive heart failure (Hedayati et al., 2004), little to no work has been done on the influence of depressive symptoms on patient mortality in this population.

CKD, Depression, and Mortality

Although depression is thought to be the most common psychological problem in patients with end-stage renal disease (ESRD), it is under-diagnosed and has received relatively little empirical attention (Drayer et al., 2006; Kimmel, 2002). Under-recognition of depression in this population may be attributed to the overlap in symptoms of uremia (kidney failure) with the somatic or vegetative indicators of depression, such as fatigue, appetite disturbance, loss of energy, and cognitive impairments (Drayer et al., 2006). Given that the etiology of somatic symptoms is often unclear, it may be important to focus on the cognitive and/or affective (i.e., nonsomatic) symptoms of depression as predictors of morbidity and mortality in ESRD.

Studies that have tested an association between depression and mortality in patients receiving hemodialysis have reported contradictory findings. For example, early work by Peterson and colleagues (1991) found that cognitive symptoms of depression as measured by the Beck Depression Inventory were predictive of shorter survival time in hemodialysis patients, yet Devins et al. (1990) did not find evidence for this link. Many of the studies examining the association between depression and mortality in ESRD patients have been flawed with methodological limitations, including a lack of control for other relevant risk factors (e.g., demographic, disease and treatment-related variables), and inappropriate use of data analytic procedures (Kimmel, 2002). However, even some well-designed studies have been unable to demonstrate that depressive affect was predictive of mortality risk in ESRD patients receiving hemodialysis (e.g., Kimmel et al., 1998).

The development of ESRD is typically a gradual process, and patients may progress through the early stages of CKD (as measured by laboratory abnormalities) over the course of several years before the severity of disease requires initiation of treatment. Patients only require initiation of treatment in the form of renal replacement therapy (e.g., dialysis or transplantation) when they have reached end-stage disease. Earlier stages of CKD are more

difficult to recognize clinically and some patients may present emergently with a need for dialysis. Thus, this important population has been understudied and there is little research on the prevalence of depression and its effects across the early stages of the CKD continuum. There is evidence to suggest that severity of predialysis CKD is associated with increased depressive symptoms, and that the point prevalence of major depressive disorder in patients with more advanced CKD is 21.6%, which is similar to reports of clinical depression in ESRD patients (20–30%) and more than five times the rate of depression in the general population (2–4%; Hedayati et al., 2004). Given that the tested associations between depression and mortality in patients with ESRD have yielded contradictory findings, examining depression at an earlier stage of renal impairment before the uremic disease state and symptoms of depression become highly confounded, and then following patients during the period of disease progression, may clarify the possible link between depression and mortality in this population (Christensen & Ehlers, 2002).

The Present Study

The purpose of the present study was to investigate the influence of depressive symptoms on mortality in patients with chronic kidney disease over an average of a seven-year follow-up period. Depressive symptoms were assessed at a relatively early stage of renal impairment, prior to the start of dialysis or renal transplantation, and the effects of a range of clinical and demographic background characteristics were adjusted for in the analyses. This study is the first to examine depressive symptoms as a risk factor for later mortality in patients in the early stages of renal impairment.

Method

Patient Sample

Research participants were recruited from the renal medicine clinic at the University of Iowa Hospitals and Clinics between August 1994 and December 2002 as part of a larger study on quality of life in renal disease. Previous work using this data set has been published by Christensen et al. (2002), Cvengros, Christensen, and Lawton (2005), and Hoth et al. (2007). This study was approved by the University of Iowa's institutional review board for the protection of human research participants and patients were compensated \$15 for completing the study measures. Criteria for eligibility included age over 18 years, English speaking, absence of severe cognitive impairment, presence of a progressive form of renal disease (i.e., diagnosis of CKD), and a serum creatinine level greater than 2.5 mg/dL, reflecting moderate impairment in renal function. A staff member screened all of the patients being seen in the renal medicine clinic during the study period for eligibility via medical record review. Those patients who met the aforementioned criteria were mailed a letter informing them about the study and requesting return of the signed consent document and completed questionnaire if they were interested in participating. At the time of enrollment in the study, none of the patients had progressed to later stage renal disease and therefore had not initiated treatment in the form of dialysis or transplantation.

Based on available information from the primary data set, approximately 520 patients met the aforementioned inclusion criteria following medical record reviews and were contacted about participating in the study. Three hundred seventy-five patients (72%) agreed to be enrolled and completed the study protocol. Sixteen patients were excluded from the statistical analyses due to missing data on depressive symptoms, clinical, or laboratory measures, resulting in a final patient sample of 359. Mortality status was determined in December 2009 (84 months after the last participant was enrolled) through cross-examination of hospital records and data from the Social Security Administration death index. Thus, patients were followed for an average of 81.2 months ($SD = 51.2$; range = 2 –

184 months), with elapsed follow-up time (i.e., time until death or December 2009 if not deceased) dependent on the enrollment date. At follow-up, 39.8% (N = 143) patients were surviving and 60.2% (N = 216) of patients were deceased. Cause of death was determined through an examination of death codes provided by the Iowa Department of Public Health Vital Records Office. The causes of death were as follows: cardiovascular disease (22.6%), other diabetes complications (23.1%), other kidney disease complications (18.8%), cancer (10.2%), infection (9.1%), cerebrovascular disease (4.8%), pulmonary disease (2.7%), liver disease (2.2%), accident (2.2%), gastrointestinal hemorrhage (1.6%), and other (2.7%).

Measures

Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961)—Symptoms of depression were measured with the Beck Depression Inventory. This commonly used self-report instrument has demonstrated adequate reliability and validity (Beck, Steer, & Garbin, 1988). The BDI consists of 21 multiple choice items reflecting participants' experience of depression over the previous week. Questions represent components of both cognitive/affective depression (e.g., feeling sad, hopeless, worthless, guilty) and somatic/vegetative depression (e.g., loss of energy, fatigue, sleep and appetite disturbance). In order to account for the overlap between symptoms of somatic depression and symptoms of CKD, we conducted separate analyses first using the total depression score and then using the sum of only the 14 nonsomatic items as predictors of mortality risk. Several other studies examining depression in patients with kidney disease have utilized this shortened BDI, and the reliability and validity of the measure remain adequate (e.g., Cvengros, Christensen, & Lawton, 2005; Hoth et al., 2007; Kimmel et al., 2000; McDade et al., 2006). The nonsomatic subset of BDI items demonstrated good internal consistency for this sample ($\alpha = .89$), as did the total BDI scale ($\alpha = .88$).

Demographic and Clinical Variables—Sociodemographic information, including age, gender, marital status, and level of education was collected concurrent with the self-reported symptoms of depression at the time of enrollment in the study. Baseline information on relevant clinical variables previously found to be associated with mortality risk in this population (Levin, 1999; Rahman & Smith, 1998) was ascertained from the patients' medical record. These included presence of diabetes and cardiovascular disease, as well as blood pressure, albumin, potassium, creatinine, hemoglobin, and blood urea nitrogen levels. For continuous variables, an average of two values in close temporal proximity to the baseline assessment (one prior to and one following the questionnaire completion date, in order to capture fluctuations in these values around this time) was computed and used in the analyses. The demographic and clinical characteristics of the sample grouped by follow-up survival status are presented in Table 1. In sum, patients were predominantly Caucasian, male, married, ranged in age from 19–92, and had some college education. Serum creatinine levels ranged from 2.5–13.5mg/DL, suggesting that there was considerable variability in severity/stage of CKD at the time of the assessment.

Results

Statistical Procedure

Multivariate survival analyses were performed with the Cox proportional hazard regression program in SPSS for Windows, Release 17.0. This program allows for the inclusion of both categorical and continuous predictor variables, as well as censoring of the outcome variable if the event (e.g., death) has not occurred (Cox, 1972). The categorical predictor variables were dummy coded (i.e., gender: female = 0 and male = 1; diabetes and cardiovascular disease status: absence of disease = 0 and presence of disease = 1). Elapsed survival time was calculated as the time from the initial assessment to the time of death (when relevant)

and was used as the dependent variable in all analyses. It was censored at the follow-up date (December 1, 2009) for patients alive at follow-up.

Preliminary Analyses

In order to examine the association of clinical and demographic variables and patient mortality risk, a preliminary Cox regression procedure was conducted. The predictor variables included in the model were patient age, gender, marital status, years of education, diabetic status, presence of cardiovascular disease, systolic and diastolic blood pressure readings, and a number of measures of disease severity including serum creatinine, blood urea nitrogen, albumin, potassium, and hemoglobin levels. All variables were entered into the regression model simultaneously to determine their unique associations with mortality risk. These results can be found in Table 2. The overall regression model was significant, $\chi^2 = (13, N = 359) = 122.87, p < .001$. Age, gender, diabetic status, presence of cardiovascular disease, and potassium level were all significant unique predictors of all-cause patient mortality. The pattern of results indicated that older patient age, male gender, presence of diabetes, cardiovascular disease, and decreased serum potassium were all associated with an increase in risk of death. Given that these covariates were all predictive of mortality risk, they were included in the primary survival analyses along with the depression variable.

Primary Analyses

In order to determine the unique effects of depression on all-cause mortality risk, the significant clinical and demographic characteristics were entered into the first step of the Cox regression, followed by the depression variable in the second step of the model for the primary analyses. We first ran the analyses with the total BDI score, including the somatic items. These results can be found in Table 3. The goodness-of-fit statistic for likelihood ratios was significant for the overall regression model, $\chi^2 = (6, N = 359) = 109.33, p < .001$. A unique association was found for patient depression, $\chi^2 = (1, N = 359) = 10.65, p = .001$. The hazard ratio (HR: 1.027) indicated that a 1-point increase in the total depression score was related to a 2.7% increase in mortality risk. Patients with total depression scores one standard deviation above the sample mean (mean = 13.58, SD = 8.91) had an estimated mortality rate that was 24% higher than average scorers on this measure. Notably, all of the aforementioned clinical and demographic variables that were predictive of mortality in the preliminary analyses remained significant in this model¹. Figure 1 depicts the estimated survival functions for high and low total depression scores.

Considering the overlap between somatic depression symptoms and uremia in CKD, we repeated the same analysis and entered the nonsomatic depression score into the second step of the Cox regression model. These results can be found in Table 4. The overall model was also significant, $\chi^2 = (6, N = 359) = 106.57, p < .0001$, and a unique effect was found for nonsomatic depression, $\chi^2 = (1, N = 359) = 8.02, p = .005$. Examination of the hazard ratio in this analysis (HR: 1.034) suggested that a 1-point increase in nonsomatic depression was associated with a 3.4% higher risk of death. For patients with relatively high nonsomatic depression (i.e., one standard deviation above the sample mean; mean = 6.63, SD = 6.30), the estimated mortality rate was 21.4% higher than for those who scored at the mean. Figure 2 depicts the estimated survival functions for high and low scores on nonsomatic depression.

¹An additional set of Cox regression analyses was conducted for both the total and nonsomatic depression scores with the subsample of patients whose death was associated with CKD or related complications (i.e., cardiovascular disease, diabetes, or infection), so as to compare these results to those for all-cause mortality. For this subsample, the pattern of results was unchanged and depression remained a significant predictor of mortality for both the total ($\chi^2 = (1, N = 281) = 8.88, p = .003$) and nonsomatic ($\chi^2 = (1, N = 281) = 7.54, p = .006$) BDI scores.

Discussion

The present study examined the association between symptoms of depression and mortality risk an average of seven years after an assessment of depression in a sample of patients with chronic kidney disease. These patients had not yet progressed to ESRD and did not have significant symptoms of uremia at the time of depression assessment, so we were able to demonstrate the unique effects of depressive symptoms on later patient survival and eliminate potential confounding associated with advanced renal impairment and dialysis treatment itself. Results indicated that both total depression scores on the BDI and the nonsomatic symptom scores were significantly associated with higher mortality risk, after controlling for the significant effects of demographic (i.e., age, gender) and clinical (i.e., presence of diabetes and cardiovascular disease, potassium level) predictors of patient survival.

This research had important strengths and limitations. Demographic data on patients who declined participation in the study was not available; thus, although we had a reasonably high response rate of 72%, it is possible that selection biases may have occurred that could affect generalizability of the present results. Assessment of depression was limited by use of a self-report instrument and may have been enhanced by use of a structured clinical interview to examine differences between subthreshold depressive symptoms and diagnosable disorders. Also, given that these analyses include data from only one time point, it would be important for future research to assess depression at multiple time points prospectively so as to examine how dynamic change in depressive symptoms over time relate to mortality risk in patients with earlier stage CKD (Kimmel et al., 2000). It would also be interesting for future research to examine whether receipt of mental health treatment affects variability in patient outcomes.

It is noteworthy that 54% of our sample had total depression scores greater than 10 on the BDI, indicating that more than half of the patients were experiencing at least mild symptoms of depression at the time of the assessment. Specifically, 29% of patients scored in the mild-moderate depression range (total scores between 10 and 18), 18.4% in the moderate-severe range (total scores between 19 and 29), and 6.6% of the sample reported severe depression symptoms (total scores between 30 and 63), based on the cut off scores used to discern symptom severity from the original measure (Beck et al., 1961; Kendall, Hollon, Beck, Hammen, & Ingram, 1987). Previous research has suggested that these BDI cutoff scores normed on the general population are appropriate for use with CKD patients (Hedayati et al., 2009). Patients in this study endorsed more depressive symptomatology compared to the ranges of BDI scores reported by Oliver and Simmons (1984) on a general population sample. In their noninstitutionalized adult random sample, 19.8% of participants reported mild depression, 10.7% were moderately depressed, and 4.0% scored in the severely depressed range on the BDI.

Additional strengths of the study include control for several important demographic and clinical variables that have been previously associated with mortality risk in patient populations. Finally, the average follow-up period was seven years, which is substantially longer than what has been reported in previous studies examining survival status. Slightly more than half of our sample was deceased at the end of the follow-up assessment, effectively increasing the statistical power of our survival analyses and helping to avoid a limitation of earlier work.

It has been postulated that depression may affect risk for early mortality in renal dialysis patients with ESRD through various mechanisms, including effects on immune system functioning, proper dietary regulations, and adherence to fluid-intake restrictions and the

dialysis treatment schedule (Kimmel, 2002; Kimmel, Weihs, & Peterson, 1993). Similar mechanisms could explain the present depression symptom effects. However, given that little work has been devoted to psychosocial predictors of outcome in patients with early stage CKD, mediators of the association between depressive symptoms and shortened patient survival in this population require further delineation.

This work extends previous findings that personality traits (i.e., neuroticism and conscientiousness) assessed in CKD patients are associated with mortality (Christensen et al., 2002) and shows that we have identified another potential, early risk factor of later patient mortality in this population. Unlike many risk factors for mortality in this population (e.g., medical comorbidities), depression symptoms are potentially modifiable. Indeed, there is quite strong evidence that both pharmacologic and psychological interventions for depression are efficacious in ESRD (e.g., Cohen, Norris, Acquaviva, Peterson, & Kimmel, 2007; Cukor, Peterson, Cohen, & Kimmel 2006; Duarte, Miyazaki, Blay, & Sesso, 2009; Whooley & Simon, 2000), and would likely also improve outcomes in patients with earlier stage CKD. Future research is needed to determine whether a reduction in depression symptoms is associated with a later reduction in mortality risk. Nevertheless, the results of the present study highlight the importance of quick and inexpensive depression screening being incorporated into the comprehensive assessment and risk stratification of patients with CKD.

Acknowledgments

The views expressed are those of the authors and do not necessarily represent the views of the Department of Veterans Affairs. Work on this article was supported by the National Institute of Diabetes and Digestive and Kidney Diseases, National Institute of Health Grant DK072325 to Alan J. Christensen.

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Figure 1. Estimated Survival Functions by Level of Total Depression

Note. High and low depression represents total depression scores one standard deviation above and below the mean, respectively, for this sample.

Figure 2. Estimated Survival Functions by Level of Nonsomatic Depression

Note. High and low depression represents nonsomatic depression scores one standard deviation above and below the mean, respectively, for this sample.

Table 1

Baseline Characteristics of the Sample Grouped by Follow-Up Survival Status

Variable	Surviving (n = 143)		Deceased (n = 216)	
	Number	%	Number	%
Male	76	53.1	124	57.4
Female	67	46.9	92	42.6
Married	78	54.5	139	64.4
Caucasian	130	90.9	197	91.2
African American	8	5.6	10	4.6
Other Race	5	3.5	9	4.1
Diabetes	57	39.9	125	57.9
Cardiovascular Disease	45	31.5	131	60.6

	Surviving (n = 143)		Deceased (n = 216)	
	Mean	SD	Mean	SD
Age (years)	46.68	14.19	59.39	15.31
Education (years)	13.26	2.90	12.37	2.48
Body Mass Index (kg/m ²)	29.03	7.48	29.65	6.81
Albumin (g/dL)	4.05	0.45	3.84	0.52
Potassium (mEq/dL)	4.81	0.92	4.62	0.57
Creatinine (mg/dL)	4.85	1.94	4.90	1.70
Hemoglobin (g/dL)	11.13	1.49	10.86	2.04
BUN (mg/dL)	64.16	24.78	68.62	21.79
Systolic BP (mmHg)	144.94	19.58	151.21	19.17
Diastolic BP (mmHg)	79.98	11.50	77.38	11.80
Total Depression Score	12.86	8.78	14.05	8.98
Nonsomatic Depression	6.57	6.44	6.67	6.23

Note. BUN = blood urea nitrogen; BP = blood pressure.

Table 2
Results of the Preliminary Cox Regression Analysis for Clinical and Demographic Variables

Variable	β^a	SE (β)	Hazard (e^{β})	χ^2 (N = 359) ^b	P
Age	.047	.007	1.048	50.98	<.0001
Gender	.310	.160	1.363	3.76	.053
Marital status	-.210	.157	.811	1.78	.182
Race	-.088	.259	.916	.116	.734
Years of education	-.029	.027	.971	1.16	.281
Diabetic status	.584	.169	1.793	11.92	.001
Cardiovascular disease	.548	.158	1.730	12.02	.001
Creatinine (mg/dL)	.040	.053	1.041	.587	.444
BUN (mg/dL)	.000	.004	1.000	.014	.905
Albumin (g/dL)	-.616	.146	.540	1.72	.200
Potassium (mEq/dL)	.353	.125	.703	7.98	.005
Hemoglobin (g/dL)	-.072	.043	.931	2.78	.096
Systolic BP (mmHg)	-.007	.004	.993	2.19	.139
Diastolic BP (mmHg)	.014	.008	1.014	2.67	.103
Overall model				122.87	<.0001

^aCoefficients reported are from the final regression model.

^bdf = 1 for all but final model; df = 13 for final (overall) model.

Table 3

Results of the Primary Cox Regression Survival Analysis for Total Depression

Variable	β^a	SE (β)	χ^2 (N = 359) ^b	P	Hazard (e^{β})	95% CI for e^{β}
Age	.041	.005	57.21	<.0001	.1041	1.031 1.052
Gender	.308	.146	4.45	.035	1.360	1.022 1.810
Diabetic status	.576	.148	15.18	<.0001	1.779	1.331 2.376
Cardiovascular disease	.404	.152	7.05	.008	1.497	1.112 2.017
Potassium (mEq/dL)	-.259	.117	4.92	.027	.772	.614 .970
Total BDI score	.027	.008	10.65	.001	1.027	1.011 1.044
Overall model			109.33	<.001		

^a Coefficients reported are from the final regression model.^b df = 1 for all but final model; df = 6 for final (overall) model.

Table 4
Results of the Primary Cox Regression Survival Analysis for Nonsomatic Depression

Variable	β^a	SE (β)	χ^2 (N = 359) ^b	P	Hazard (e^{β})	95% CI for e^{β}
Age	.041	.005	56.03	<.0001	.1042	1.031 1.053
Gender	.294	.146	4.06	.044	1.341	1.008 1.785
Diabetic status	.581	.148	15.40	<.0001	1.787	1.337 2.389
Cardiovascular disease	.417	.152	7.56	.006	1.518	1.127 2.043
Potassium (mEq/dL)	-.271	.117	5.35	.021	.762	.606 .960
Nonsomatic BDI score	.033	.012	8.02	.005	1.034	1.010 1.058
Overall model			106.57	<.0001		

^a Coefficients reported are from the final regression model.

^b df = 1 for all but final model; df = 6 for final (overall) model.