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BRIEF COMMUNICATION

Chronic Kidney Disease

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The association of post-traumatic stress disorder with glomerular filtration rate decline

Farrukh M. Koraishy[1,2] | Beth E. Cohen[3,4] | Jeffery F. Scherrer[5] | Mary Whooley[3,4] | Janos Hajagos[6] | Cassianne Robinson-Cohen[7] | Wei Hou[8]

[1]Division of Nephrology, Department of Medicine, Stony Brook University, Stony Brook, New York, USA

[2]Department of Medicine, Northport VA Medical Center, Northport, New York, USA

[3]Department of Medicine, University of California, San Francisco, California, USA

[4]Department of Medicine, San Francisco VA Health Care System, San Francisco, California, USA

[5]Department of Family and Community Medicine, Saint Louis University, St. Louis, Missouri, USA

[6]Department of Medical Bioinformatics, Stony Brook University, Stony Brook, New York, USA

[7]Division of Nephrology, Department of Medicine, Vanderbilt University Medical Center, Nashville, Tennessee, USA

[8]Department of Family, Population, and Preventive Medicine, Program in Public Health, Stony Brook University, Stony Brook, New York, USA

Correspondence
Farrukh M. Koraishy, Department of Medicine/Nephrology, Stony Brook University, 100 Nicolls Road, HSCT16-080 E, Stony Brook, NY, USA.
Email: farrukh.koraishy@stonybrookmedicine.edu

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Abstract

While major depression is known to be associated with glomerular filtration rate (GFR) decline, there is a lack of data on the association of other mental illnesses like posttraumatic stress disorder (PTSD) with kidney disease. In 640 adult participants of the Heart and Soul Study (mean baseline age of 66.2 years) with a high prevalence cardiovascular disease, hypertension and diabetes, we examined the association of PTSD with GFR decline over a 5-year follow-up. We observed a significantly greater estimated (e) GFR decline over time in those with PTSD compared to those without (2.97 vs. 2.11 ml/min/1.73 m^2/year; $p = .022$). PTSD was associated with 91% (95% CI 12%–225%) higher odds of 'rapid' versus 'mild' (>3.0 vs. <3.0 ml/min/1.73 m^2/per year) eGFR decline. These associations remained consistent despite controlling for demographics, medical comorbidities, other mental disorders and psychiatric medications. In conclusion, our study provides evidence that PTSD is independently associated with GFR decline in middle-aged adults with a high comorbidity burden. This association needs to be examined in larger cohorts with longer follow-ups.

KEYWORDS
chronic kidney disease, epidemiology, glomerular filtration rate

Summary at a glance

There is lack of data on the association of posttraumatic stress disorder (PTSD) with kidney disease. In middle aged adults with a high comorbidity burden, we observed a significantly greater eGFR decline over time in those with PTSD compared to those without after controlling for demographics, medical comorbidities, other mental disorders and psychiatric medications.

1 | INTRODUCTION

Chronic kidney disease (CKD) is a common condition associated with high morbidity, mortality and health-care costs.[1] While numerous risk factors for CKD progression are known,[2] emerging evidence indicates that mental health disorders are highly comorbid with CKD.[3] Major depression is known to be associated with CKD progression[4] and with estimated glomerular filtration rate (eGFR) decline in the general population.[5] A recent study found that receiving a 'stress-related disorder (SRD)' diagnosis was associated with a 23% higher risk of CKD progression.[6]

We recently reported that post-traumatic stress disorder (PTSD) was associated with decline in eGFR among World Trade Center (WTC) responders.[7] This cohort consisted of relatively young participants (mean age 53.1 years), mostly Caucasians, with low prevalence of diseases associated with CKD like diabetes mellitus (DM), hypertension (HTN) and cardiovascular disease (CVD).[7] Whether PTSD is also an independent risk factor for GFR decline in older people, with diverse racial backgrounds and a greater comorbidity burden is not known. To address this gap in the field, we estimated the association of PTSD with eGFR decline in the Heart and Soul Study. This is a well-characterized cohort of older adults with coronary heart disease and a high prevalence of HTN and DM, where data on renal measures, mental health disorders and psychiatric medications was collected as part of the study protocol.

TABLE 1 Clinical characteristics of all subjects in the cohort and comparision of those with and without PTSD

	All subjects $N = 640$	No PTSD $N = 580$	PTSD $N = 60$	p value
Demographics				
Baseline age	66.18 (10.13)	66.63 (10.06)	61.82 (9.83)	**.0006**
Male	532 (83.1%)	487 (84.0%)	45 (75.0%)	.078
White	378 (59.1%)	345 (59.5%)	33 (55.0%)	.501
Married	288 (45.1%)	267 (46.2%)	21 (35.0%)	.097
Co-morbidities				
BMI	28.50 (5.09)	28.58 (5.12)	27.69 (4.83)	.180
HTN	444 (69.4%)	400 (69.0%)	44 (73.3%)	.485
DM	151 (23.6%)	134 (23.1%)	17 (28.3%)	.364
Stroke	78 (12.2%)	69 (11.9%)	9 (15.0%)	.487
CHF	94 (14.8%)	77 (13.3%)	17 (28.8%)	**.001**
Angina	231 (36.1%)	199 (34.3%)	32 (53.3%)	**.003**
LVH	342 (53.9%)	318 (55.2%)	24 (40.7%)	**.033**
Renal measures				
Y1 SCr	1.09 (0.55)	1.10 (0.56)	1.03 (0.48)	.274
Y6 SCr	1.24 (0.62)	1.24 (0.61)	1.23 (0.71)	.912
Y1 eGFR (ml/min/1.73 m^2) (Cr-CKD EPI 2021)	77.02 (18.92)	76.43 (18.88)	82.72 (18.52)	**.015**
Y6 eGFR (ml/min/1.73 m^2) (Cr-CKD EPI 2021)	66.06 (18.85)	65.87 (18.90)	67.88 (18.35)	.423
eGFR decline (ml/min/1.73 m^2/year)	2.19 (2.71)	2.11 (2.70)	2.97 (2.70)	**.022**
Psychosocial				
Alcohol abuse (score)	2.35 (2.43)	2.39 (2.44)	1.98 (2.40)	.218
Smoking (pack/years)	18.22 (20.44)	17.27 (20.01)	27.29 (22.37)	**.001**
Mental health disorders				
MDD	112 (17.5%)	90 (15.5%)	22 (36.7%)	**<.0001**
GAD	62 (10.2%)	44 (7.9%)	18 (34.6%)	**<.0001**
Psychiatric medication use				
SSRIs	51 (8.0%)	41 (7.1%)	10 (16.7%)	**.01**
TCAs	18 (2.8%)	16 (2.8%)	2 (3.3%)	.807
Other antidepressants	42 (6.6%)	30 (5.2%)	12 (20.0%)	**<.0001**
Any antidepressant	97 (15.3%)	79 (13.7%)	18 (30.0%)	**.0009**

Note: Mean (standard deviation) and *N* (%) are presented for continuous and categorical variables respectively.
Abbreviations: BMI, body mass index; CHF, congestive heart failure; CKD-EPI 2021, Chronic Kidney Disease Epidemiology Collaboration 2021 equation; DM, diabetes mellitus; eGFR, estimated glomerular filtration rate; GAD, generalized anxiety disorder; HTN, hypertension; LVH, left ventricular hypertrophy; MDD, major depressive disorder; PTSD, post-traumatic stress disorder; SCr, serum creatinine; SSRIs, selective serotonin reuptake inhibitors; TCAs, tricyclic antidepressants; Y1, year 1, baseline; Y6, year 6, follow-up.
Values in bold were statistically significant.

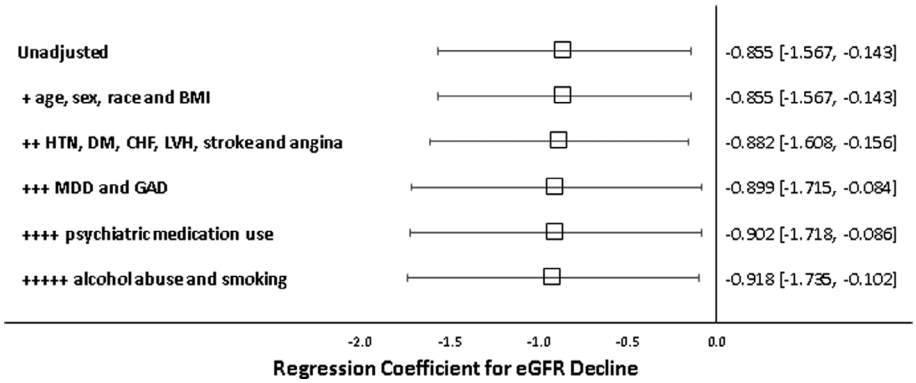

FIGURE 1 Association of post-traumatic stress disorder (PTSD) with the slope of estimated glomerular filtration rate (eGFR) decline. Forest Plot to display to association between PTSD and slope/trajectory of eGFR decline unadjusted [Model 1]. In addition, after sequential adjustment for demographics (age, gender and race) [Model 2], medical comorbidities (BMI, HTN, DM, CHF, LVH, stroke and angina) [Model 3], psychiatric diagnoses (MDD and GAD) [Model 4], psychiatric medication use [Model 5], and finally alcohol abuse and smoking [Model 6]. Regression coefficient (β statistic) and 95% confidence interval (CI) is shown for each model. BMI, body mass index; CHF, congestive heart failure; DM, diabetes mellitus; GAD, generalized anxiety disorder; HTN, hypertension; LVH, left ventricular hypertrophy; MDD, major depressive disorder

2 | METHODS

2.1 | Heart and Soul (H&S) study cohort

This prospective cohort study enrolled 1024 participants with coronary heart disease from multiple medical centers in San Francisco, USA, between 1 September 2000 and 31 December 2002.[8] The study was approved by the institutional review board (IRB) of the University of California, San Francisco and all participants provided written informed consent. The 640 participants who completed a second in-person visit approximately 5 years after their baseline visit and with complete data on all variables were included in this study. No participant in the study had end-stage kidney disease (ESKD). Use of existing H&S data for the present study was approved by the Stony Brook University IRB (#2019-00732).

2.2 | PTSD measure

PTSD was assessed with the Computerized Diagnostic Interview Schedule (CDIS) for DSM-IV.[9]

2.3 | Covariates

All baseline (indexed at the individual's first eGFR observation) covariates known to be associated with CKD[2] were included in models: demographic factors (age, race and gender) and co-morbidities (DM, HTN, body mass index [BMI] and CVD [left ventricular hypertrophy [LVH], angina and stroke]). Psychiatric diseases associated with PTSD[10] (major depressive disorder [MDD] and generalized anxiety disorder [GAD]); psychiatric medications (selective serotonin reuptake inhibitors [SSRIs], tricyclic antidepressants [TCAs] and 'others' [not classified]), substance abuse (smoking [pack years] and alcohol intake [alcohol score]) were also included. MDD was defined by a 9-item Patient Health Questionnaire (PHQ9) score ≥10[11] and GAD was determined by the CDIS using DSM-IV criteria.[9]

2.4 | Outcome measures (eGFR decline)

Serum creatinine (SCr) was measured at baseline (year 1) and 5-year follow-up visits (year 6). eGFR was calculated using the 2021 CKD-EPI equation.[12] We reported the mean rate of eGFR decline (ml/min/1.73 m^2/year) defined as the total change in eGFR (from baseline to follow-up) divided by the years of follow-up. We further sub-divided the cohort into subjects with 'rapid' versus 'mild' (>3.0 vs. <3.0 ml/min/1.73 m^2/year) eGFR decline.[13]

2.5 | Statistical analysis

Baseline characteristics were described using means and standard deviations (SD) or frequencies (percentages) in each category, as appropriate. The baseline characteristics and renal profile were compared between subjects with 'PTSD' versus 'no PTSD' and between those with 'rapid' versus 'mild' eGFR decline using Δ^2-tests for categorical variables and t-tests for continuous variables. We used generalized estimating equations to estimate the association of PTSD diagnosis with slope of eGFR decline using the following six sequential adjustment models: unadjusted [Model 1] followed by sequential adjustment for demographics (age, gender, race) [Model 2], medical comorbidities (BMI, HTN, DM, CHF, LVH, stroke and angina) [Model 3], other psychiatric diagnoses (MDD and GAD) [Model 4], psychiatric medication use [Model 5], and finally alcohol intake and smoking [Model 6]. We used logistic regression to estimate the association

TABLE 2 Comparsion of the chatacteristics of subjects with "Mild" versus "Rapid" eGFR decline

	'Mild' eGFR decline (<3.0 ml/min/1.73 m^2/year) $N = 420$	'Rapid' eGFR decline (>3.0 ml/min/1.73 m^2/year) $N = 220$	*p* value
Demographics			
Baseline age	66.66 (10.20)	65.25 (9.95)	.093
Male	350 (83.3%)	182 (82.7%)	.846
White	261 (62.1%)	117 (53.2%)	**.029**
Married	198 (47.3%)	90 (41.1%)	.138
Co-morbidities			
BMI	28.30 (4.89)	28.88 (5.46)	.190
HTN	273 (65.0%)	171 (77.7%)	**.0009**
DM	81 (19.3%)	70 (31.8%)	**.0004**
Stroke	43 (10.3%)	35 (15.9%)	**.038**
CHF	55 (13.1%)	39 (17.9%)	.108
Angina	158 (37.6%)	73 (33.2%)	.267
LVH	219 (52.3%)	123 (56.9%)	.263
Renal measures			
Y1 SCr (mg/dl)	1.16 (0.66)	0.98 (0.20)	**<.0001**
Y6 SCr (mg/dl)	1.18 (0.65)	1.36 (0.55)	**.0002**
Y1 eGFR (Cr-CKD EPI 2021)	73.59 (19.58)	83.59 (15.67)	**<.0001**
Y6 eGFR (Cr-CKD EPI 2021)	69.95 (18.88)	58.63 (16.43)	**<.0001**
eGFR decline (ml/min/1.73 m^2/year)	0.73 (1.76)	4.99 (1.86)	**<.0001**
Psychosocial			
Alcohol abuse (score)	2.35 (2.46)	2.35 (2.40)	.991
Smoking (pack/year)	17.47 (19.99)	19.65 (21.24)	.211
Mental health disorders			
MDD	68 (16.2%)	44 (20.0%)	.228
Anxiety disorder	42 (10.5%)	20 (9.7%)	.739
PTSD	31 (7.4%)	29 (13.2%)	**.017**
Psychiatric medication use			
SSRIs	28 (6.7%)	23 (10.5%)	.096
TCAs	7 (1.7%)	11 (5.0%)	**.016**
Other antidepressants	25 (6.0%)	17 (7.8%)	.398
Any antidepressant	54 (13.0%)	43 (19.6%)	**.027**

Note: Mean (standard deviation) and *N* (%) are presented for continuous and categorical variables respectively.
Abbreviations: BMI, body mass index; CHF, congestive heart failure; CKD-EPI 2021, Chronic Kidney Disease Epidemiology Collaboration 2021 equation; DM, diabetes mellitus; eGFR, estimated glomerular filtration rate; GAD, generalized anxiety disorder; HTN, hypertension; LVH, left ventricular hypertrophy; MDD, major depressive disorder; PTSD, post-traumatic stress disorder; SCr, serum creatinine; SSRIs, selective serotonin reuptake inhibitors; TCAs, tricyclic antidepressants; Y1, year 1, baseline; Y6, year 6, follow-up.
Values in bold were statistically significant.

between PTSD and odds of 'rapid' versus 'mild' eGFR decline with the six sequential adjustment models. Unadjusted and multivariable-adjusted β statistics & odds ratio (OR), 95% confidence intervals, and p-values were reported. All analyses were performed using SAS v9.4 (the SAS Institute, Cary, NC).

3 | RESULTS

Baseline cohort characteristics overall and by PTSD status are shown in Table 1. Of a total of 640 individuals, the mean age was 66.18 (SD 10.13) years, and those with PTSD were significantly younger. 83.1% were males, 59.1% were Whites participants, mean BMI was 28.50 (5.09). 69.4% had HTN and 23.6% DM. These characteristics were similar between those with or without PTSD. Those with PTSD had a higher prevalence of CHF and angina but a lower prevalence of LVH. Subjects with PTSD were more likely to be smokers, have comorbid MDD and GAD and be on anti-depressants compared to those without PTSD (Table 1).

No differences were noted in the mean baseline (Y1) and final (Y6) SCr between the two groups, however those with PTSD had a higher mean eGFR at baseline than those without PTSD (82.72 vs. 76.43 ml/min/1.73 m^2). The overall decline in eGFR over time was significant greater is those with PTSD compared to those without (2.97 vs. 2.11 ml/min/1.73 m^2/year; $p = .022$).

3.1 | Association of PTSD with the trajectory of GFR decline

As shown in Figure 1, PTSD was associated with a significantly greater GFR decline over time (β statistic −0.855 [95% CI −1.567 to −0.143]; $p = .018$) and this association remained significant after sequential adjustment for demographics followed by medical comorbidities followed by MDD and GAD and then psychiatric medications and substance abuse (−0.918 [−1.734 to −0.101], $p = .027$).

3.2 | Comparison of eGFR decline categories and association of PTSD with 'rapid' GFR decline

The baseline characteristics and renal profile of subjects with 'mild' versus rapid' eGFR decline (<3.0 vs. >3.0 ml/min/1.73 m^2) per year are shown in Table 2. Subjects with 'rapid' eGFR decline were more likely to be hypertensive and diabetic and be on TCAs. As shown in Figure 2, PTSD was significantly associated with 91% (95% CI 12%–225%) higher odds of 'rapid' versus 'mild' eGFR decline ($p = .018$). This association remained statistically significant after adjustment for demographics, comorbidities, mental disorders and psychiatric medications (OR = 1.98 [1.00–3.77], $p < .05$). The significance was borderline after further adjustment for alcohol abuse and smoking even though the odds ratio was similar (OR = 1.94 [1.00–3.77], $p = .05$).

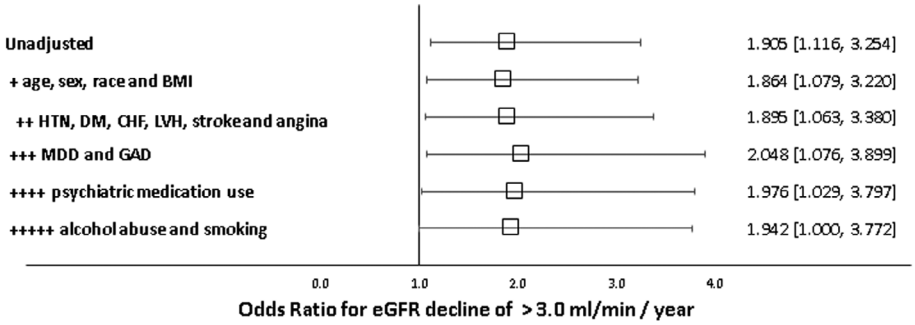

FIGURE 2 Association of post-traumatic stress disorder (PTSD) with 'rapid' (>3.0 ml/min/1.73 m^2/year) versus 'mild' (<3.0 ml/min/1.73 m^2/year) estimated glomerular filtration rate (eGFR) decline. Forest Plot to display to association between PTSD with 'rapid' compared to 'mild' eGFR decline unadjusted [Model 1]. In addition, after sequential adjustment for demographics (age, gender and race) [Model 2], medical comorbidities (BMI, HTN, DM, CHF, LVH, stroke and angina) [Model 3], psychiatric diagnoses (MDD and GAD) [Model 4], psychiatric medication use [Model 5], and finally alcohol abuse and smoking [Model 6]. Odds ratios (OR) and 95% confidence interval (CI) is shown for each model. BMI, body mass index; CHF, congestive heart failure; DM, diabetes mellitus; GAD, generalized anxiety disorder; HTN, hypertension; LVH, left ventricular hypertrophy; MDD, major depressive disorder

4 | DISCUSSION

A major increase in PTSD has been noted since the start of the COVID-19 pandemic[14] and the clinicians need to be cognizant of the potential impact of PTSD on the kidney. In a cohort of middle aged and elderly subjects we observed that PTSD was significantly associated with greater decline in eGFR over time after sequential adjustment for traditional CKD risk factors including demographics and medical comorbidities and further adjustment for other mental disorders, psychiatric medications and finally smoking and alcohol use.

PTSD was also significantly associated with higher odds of 'rapid' versus 'mild' (> 3.0 vs. <3.0 ml/min/1.73 m^2) eGFR decline. This association also remained statistically significant after adjusting for all covariates except became borderline significant at the final step of adjustment for smoking and alcohol use. This could potentially be related to our limitations of the small sample and lower proportion (34.4%) of patients with rapid eGFR decline.

Our results are consistent with our previous report of the association of PTSD with GFR decline in WTC responders[7] that were young and relatively healthy. Compared to the WTC cohort,[7] the H&S cohort participants were older (mean baseline age 66.2 vs. 53.1 years), more likely to be non-White (40.9% vs. 10.9%) and with greater prevalence of HTN (69.4% vs 29.7%) and DM (23.6% vs. 8.6%) and lower baseline renal function (77.02 vs. 90.42 ml/min/1.73 m^2). The mean rate of GFR decline over time was greater in this study compared to the WTC cohort: 2.19 versus 1.51 ml/min/1.73 m^2/year. Moreover, compared to the large portion (25%) of subjects in the WTC cohort who had a significant rise in GFR overtime (>1.0 ml/min/1.73 m^2/year) this proportion was much smaller (8.5%) in the present cohort. Finally, this study had a longer follow-up time of 5 years compared to 2 years.[7] Therefore the current study extends the evidence of the association of PTSD with eGFR decline to an older, more racially diverse population with additional comorbidities.

Mechanisms for the association between PTSD and increased rates of GFR decline remain uncertain. In this study the participants with PTSD were younger, had a higher baseline eGFR and a similar prevalence of the major risk factors of CKD i.e. HTN and DM compared to those without PTSD diagnosis. PTSD could potentially cause kidney damage via systemic pathophysiological mechanisms like inflammation,[15] altered hypothalamic–pituitary axis,[16] endothelial dysfunction[17] or premature aging.[18] Depression has been postulated to accelerate CKD progression through similar mechanisms and PTSD and depression share pathophysiology.[19] However our findings remained significant even after controlling for MDD and antidepressant medications suggesting an independent association of PTSD with CKD. Besides the potential of direct kidney damage as a result of systemic pathophysiology in patients with PTSD, an alternate hypothesis is that patients with mental health disorders like PTSD have medical non-compliance[20] that has been associated with adverse outcomes including CKD progression.[21] However this hypothesis was not tested in this study.

4.1 | Limitations

This is an observational study that did not test for causality. The study was limited to a small sample size and relatively small numbers of subjects with PTSD compared to those without. We did not have data on proteinuria and other nephrotoxic drugs on all patients. We also did not have complete data on medications associated with renal protection like blood pressure and diabetic medications. eGFR progression was based only on two time points, which is suboptimal to describe trajectories. Due to relatively short follow-up and normal baseline GFR of our subjects, we could not test for the association of PTSD with advanced renal outcomes like incident CKD, doubling of serum creatinine or end stage kidney disease. Finally, the low proportion of females and limited geographic region of the H&S cohort limits generalizability.

In conclusion, our study provides further evidence that PTSD is a potential risk factor for GFR decline. This association needs to be

tested in in larger cohorts with longer follow-ups for advanced renal outcomes.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

ORCID

Farrukh M. Koraishy https://orcid.org/0000-0001-6974-5674

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