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ORIGINAL ARTICLE

Relationship between trajectory of sleep quality and short-term changes in residual renal function in stage 3–5 chronic kidney disease patients

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Abstract

Background Chronic kidney disease (CKD) is commonly associated with sleep disturbance. However, the relationship between the trajectory of sleep quality and short-term residual renal function is not clear. Thus, this study aimed to investigate such relationship.

Methods In total, 132 patients with CKD stage 3–5 were prospectively enrolled. All participants were followed-up for 6 months. The Pittsburgh Sleep Quality Index (PSQI) questionnaire was used to assess sleep quality. The longitudinal PSQI and estimated glomerular filtration rate (eGFR) were measured at baseline, the 3rd month and 6th month. The participants were stratified into three groups according to the PSQI trajectories. The primary outcome was set as the eGFR change among 6 months less than the median.

Results Sixty nine participants showed PSQI ≤ 5 at baseline and 15 patients had increased scores > 5 at 3rd month among them. 63 participants showed PSQI > 5 at baseline and 11 patients had decreased scores ≤ 5 at 3rd month. Only in patients whose baseline PSQI ≤ 5 but increased to > 5 at 3rd month presented a longitudinal decline in eGFR at both 3rd month and 6th month compared with baseline eGFR. Linear regression analysis for eGFR change showed no significant association between eGFR change and PSQI score. Logistic regression revealed worsen sleep quality will deteriorate renal function.

Conclusion A relationship was observed between worsening sleep quality and eGFR decline in non-dialysis CKD patients.

Keywords Chronic kidney disease · Pittsburgh sleep quality index (PSQI) · Sleep disturbance · Estimated glomerular filtration rate

Introduction

A variety of studies have been performed to explore the potential risk factors of chronic kidney disease (CKD) progression and the development of end-stage renal disease (ESRD), which cause a significant burden on health and economy worldwide [1, 2]. CKD clinics primarily aim to control the factors that enhance the development of CKD and to inhibit the pathways underlying ESRD. However, although various recommendations are provided in the clinical guidelines for management in CKD, ESRD incidence remains high globally [3–5]. Prior clinical studies have found a significant association between sleep disturbance and CKD [6–8]. One study has reported that 36.2% of non-dialysis CKD patients present with sleep disturbance [9]. Meanwhile, other investigators have found that patients with CKD present with some psychological problems (i.e., depression and anxiety), and such problems affect sleep rhythm. Comorbidities in patients with CKD, which include diabetes, hypertension, cardiovascular diseases, and obesity, are associated with sleep disturbance and present a complex interaction that deteriorate the residual renal function of patients with CKD [6, 10–12]. Clinically, improved sleep quality can be achieved by modifying clinical factors, such as pain control and treating depression and systemic

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inflammation [8, 13, 14]. Given above evidence, recommendation in clinical guideline stated that treatment of sleep disorders in CKD could halt the deterioration in residual renal function. We hypothesized that improved sleep quality can halt CKD progression. Thus, a prospective study was performed to investigate the impact of the trajectory of sleep disturbance on residual renal function in patients with CKD.

Material and methods

Participants

Adult non-dialysis patients with stage 3–5 CKD and followed-up at the out-patient clinic from November 2017 to December 2018 at Tianjin First Center Hospital in China were enrolled in this prospective study. The exclusion criteria were patients aged below 18 or above 80 years who were undergoing renal replacement therapy or using sedatives and hypnotics or those who were not willing to participate in the study and those with mental or psychiatric illness or unstable medical condition, which makes them unable to complete the questionnaire. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee (Tianjin First Center Hospital, 2015008S) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Data collection

The participants were followed-up from the beginning of the study until 6 months after the initiation of the study or dialysis, death, and unavoidable reason for finish the follow-up. Demographic and laboratory data were collected during the study period. The estimated glomerular filtration rate (eGFR) was calculated using the CKD Epidemiology Collaboration (CKD-EPI) equation [15]. All of the participants were estimated eGFR at baseline, 3rd month and 6th month. The definition of eGFR change was derived from comparable eGFR values at 6th months and baseline.

Measurement of sleep status

The measurement of sleep status was conducted using the Chinese version of the Pittsburgh Sleep Quality Index (PSQI) questionnaire [16, 17], which contains 19 self-rated questions grouped to seven sleep components (subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction). Each component was scored from 0 to 3, thereby yielding a total score of 0–21. Higher scores indicate lower quality of sleep. The assessment of sleep quality was conducted at the initiation of the study, 3rd month and 6th month. In this study, sleep disturbance was defined based on a total PSQI score > 5. The participants were first stratified into two groups according to the baseline PSQI scores (PSQI ≤ 5 and PSQI > 5).The longitudinal PSQI and eGFR were measured at the 3rd month and 6th month. The changes of eGFR from baseline to the 6th month were calculated. According to the median eGFR change among 6 months, the primary outcome was set as the eGFR change less than the median which represents the deterioration of residual renal function.

Statistical analysis

Normally distributed continuous variables were presented as mean ± standard deviation (SD), none normally distributed continuous variables as medians and interquartile ranges, and categorical variables as percentages. The comparison of continuous variables between two groups were analyzed using independent sample *T* test or Mann–Whitney *U* test accordingly. The chi-square test was used for categorical variables. The estimating of longitudinal changes of PSQI scores and eGFR values at 3rd and 6th month with baseline in each group were compared by paired *T* test. Univariable and multivariable linear regression analyses were performed to obtain the associations of longitudinal PSQI scores with eGFR change at 6th month from baseline. According to the PSQI trajectories, patients were then allocated to Worsen, Improved or Stable groups. Logistic regression was performed to evaluate the PSQI trajectories as categorical parameters on predicting the primary outcome. All statistical analyses were conducted using the Statistical Package for the Social Sciences software version 22 (IBM, Japan) and STATA version 14 (StataCorp LP, College Station, TX, the USA). A p value < 0.05 was considered statistically significant.

Results

Characteristics of the participants

Figure 1 shows the flow diagram of the participant recruitment. In total, 132 patients with CKD stage 3–5 were prospectively enrolled. All participants were followed-up for 6 months. The mean age of the participants was 54.2 ± 15.4 years, and 67.4% (89/132) of the participants were men. The prevalence of diabetes was 17.4% (23/132) in this CKD cohort. The mean baseline eGFR of the participants was 32.8 ± 15.4 mL/min/1.73 m^2. The mean baseline PSQI score was 6.46. Sixty-nine participants showed PSQI score ≤ 5 at baseline and 15 patients had increased scores > 5 at 3rd month among them. Sixty-three participants showed PSQI score > 5 at baseline and

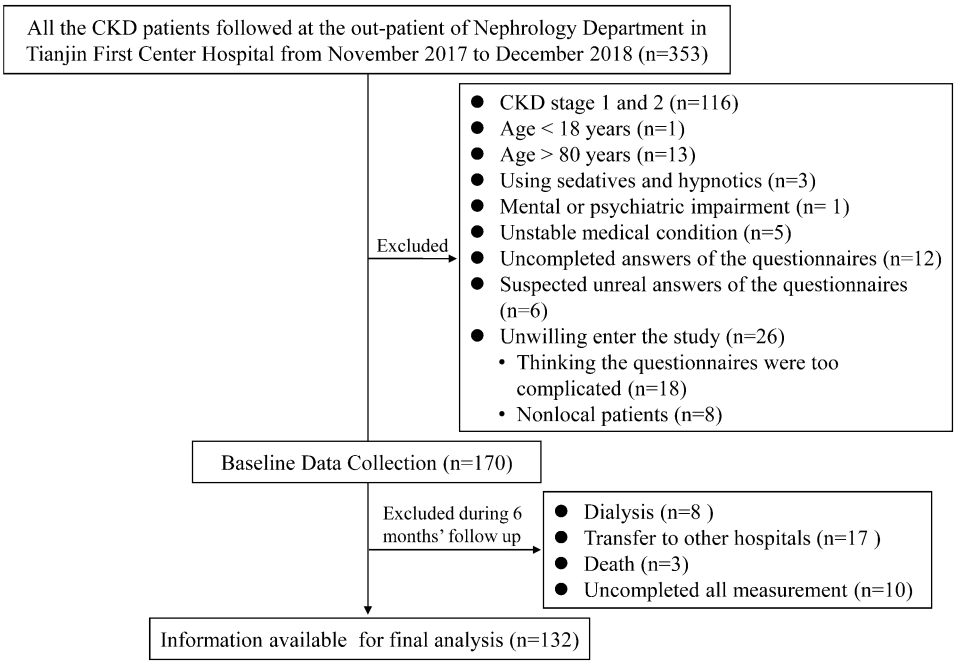

Fig. 1 Flow diagram of participant recruitment

among them 11 patients had decreased scores ≤ 5 at 3rd month. Table 1 shows the demographic characteristics and laboratory parameters of all the participants at baseline separated by PDSI scores. In both two groups stratified by baseline PDSI scores, the demographic and clinical parameters did not significantly differ between patients whose PSQI scores changed or sustained at the 3rd month from baseline except renin-angiotensin system inhibitor (RASi) usage in the group of baseline PSQI score ≤ 5 and Hb level, calcium channel blocker (CCB) usage in the group of baseline PSQI score > 5.

PSQI score and variations in sleep status

The distributions of PSQI scores at baseline, 3rd month and 6th month were plotted in Fig. 2. Figure 3 presents the longitudinal PSQI scores and eGFR values at three time points based on patient stratification with PSQI scores at baseline. In participants whose PSQI scores ≤ 5 at baseline and increase to > 5, eGFR presented significant decline in 3rd and 6th month ($p = 0.028$ and $p = 0.016$ by paired T test) compared with baseline eGFR, whereas, participants with constant PSQI score were not. In contrast, participants with PSQI score > 5 at baseline, longitudinal eGFR remained relative constant no matter longitudinal changes in PSQI scores.

Associations of eGFR change at 6th month from baseline with PSQI scores

By linear regression analysis, eGFR change from baseline to the 6th month showed no association with PSQI score at baseline, 3rd or 6th month in both univariate models and multivariate models adjusting for age, sex, systolic blood pressure, diabetes, hemoglobin, serum albumin and RASi treatment (Table 2). The univariate associations between PSQI score and eGFR change among 6 months were plotted in Fig. 4.

Associations of eGFR change at 6th month from baseline with PSQI scores

According to the PSQI trajectories, patients were then allocated to Worsen, Improved or Stable groups. Those patients whose PSQI ≤ 5 at baseline then increased to > 5 at 3rd month were allocated to Worsen group; Patients whose PSQI > 5 at baseline then decreased to ≤ 5 at 3rd month were allocated to Improve group; Patients whose PSQI ≤ 5 at baseline then still ≤ 5 at 3rd month and patients whose PSQI > 5 at baseline then still > 5 at 3rd month were allocated to Stable group. As shown in Table 3, Worsen group had higher risk on the deterioration of residual renal function compared with Improve group and Stable group by univariate and multivariate logistic regression analysis.

The score change in each sleep component of PSQI global score was also analyzed. In Improved group, the subjective sleep quality score change occurred in 50% cases; sleep latency score change occurred in 62.5% cases, sleep duration score change occurred in 50% cases, habitual sleep efficiency score change occurred in 62.5% cases, sleep disturbances score change occurred in 50% cases, use of sleeping medication score didn't change, and daytime dysfunction score change occurred in 75% cases. In Worsen group, the

Table 1 Demographic characteristics and laboratory parameters at baseline

Characteristic	PSQI ≤ 5 at baseline ($n=69$)			PSQI > 5 at baseline ($n=63$)		
	PSQI ≤ 5 at 3rd month ($n=54$)	PSQI > 5 at 3rd month ($n=15$)	p value[a]	PSQI ≤ 5 at 3rd month ($n=11$)	PSQI > 5 at 3rd month ($n=52$)	p value[a]
Age, year	52.1 ± 16.1	52.8 ± 16.2	0.875	50.1 ± 13.5	57.6 ± 14.4	0.116
Sex, male, *n* (%)	36 (66.7%)	11 (73.3%)	0.759	10 (90.9%)	32 (61.5%)	0.082
DM, *n* (%)	9 (16.7%)	3 (20.0%)	0.715	1 (9.1%)	10 (19.2%)	0.284
Smoking history, *n* (%)	21 (38.9%)	6 (40.0%)	0.886	3 (27.3%)	16 (30.8%)	0.363
Drinking history, *n* (%)	15 (27.8%)	5 (33.3%)	0.702	4 (36.4%)	18 (34.6%)	0.903
Original kidney disease			0.535			0.598
Glomerulonephritis, *n* (%)	23 (42.6%)	6 (40.0%)		5 (45.5%)	30 (57.7%)	
Diabetic nephropathy, *n* (%)	10 (18.5%)	1 (6.7%)		1 (9.1%)	7 (13.5%)	
Hypertensive nephrosclerosis, *n* (%)	18 (33.3%)	6 (40.0%)		4 (36.4%)	11 (21.2%)	
Others, *n* (%)	3 (5.6%)	2 (13.3%)		1 (9.1%)	4 (7.7%)	
SBP, mmHg	138.5 ± 19.7	135.2 ± 14.6	0.549	141.3 ± 13.7	139.7 ± 15.3	0.747
BMI, kg/m^2	25.4 ± 4.6	24.1 ± 4.0	0.303	25.3 ± 4.2	24.3 ± 4.3	0.505
Blood parameters						
Hb, g/dL	12.7 ± 1.8	12.8 ± 2.0	0.783	13.7 ± 1.8	11.9 ± 2.5	0.027
Alb, g/dL	4.03 ± 0.57	4.30 ± 0.55	0.109	4.38 ± 0.35	4.16 ± 0.52	0.236
BUN, mg/dL	11.1 ± 4.9	13.0 ± 7.5	0.247	9.6 ± 4.5	13.2 ± 7.5	0.132
Cr, mg/dL	2.41 ± 1.05	2.66 ± 1.76	0.491	2.25 ± 1.20	2.77 ± 1.58	0.304
Ca, mEq/L	2.28 ± 0.17	2.32 ± 0.19	0.479	2.17 ± 0.64	2.23 ± 0.19	0.612
P, mEq/L	1.20 ± 0.22	1.21 ± 0.21	0.913	1.23 ± 0.19	1.24 ± 0.23	0.896
K, mEq/L	4.69 ± 0.69	4.87 ± 0.62	0.375	5.04 ± 0.86	4.53 ± 0.64	0.034
UA, mg/dL	4.58 ± 1.09	4.59 ± 1.05	0.968	4.83 ± 1.65	4.30 ± 1.11	0.188
CO_2, mEq/L	25.4 ± 2.6	23.8 ± 4.2	0.137	26.9 ± 2.9	24.1 ± 3.5	0.109
iPTH, pg/mL	2.00 [1.60, 2.75]	1.80 [1.55, 2.40]	0.263	2.30 [1.80, 2.65]	2.20 [1.60, 2.60]	0.802
Treatment						
RASi, *n* (%)	23 (42.6%)	1 (6.7%)	0.001	4 (36.4%)	13 (25.0%)	0.469
CCB, *n* (%)	33 (61.1%)	11 (73.3%)	0.546	3 (27.3%)	33 (63.5%)	0.043
β-blocker, *n* (%)	13 (24.1%)	8 (53.3%)	0.054	3 (27.3%)	19 (36.5%)	0.733
Diuretic, *n* (%)	3 (5.6%)	2 (13.3%)	0.297	1 (9.1%)	2 (3.8%)	0.443

DM diabetes mellitus; *SBP* systolic blood pressure; *BMI* body mass index, *Hb* hemoglobin; *Alb* albumin; *BUN* blood urea nitrogen; *Cr* creatinine; *Ca* calcium; *P* phosphorus; *K* potassium; *UA* uric acid; CO_2 venous carbon dioxide; *iPTH* intact parathyroid hormone; *RASi* RAS inhibitor; *CCB* calcium channel blocker; *β* blocker, *β* receptor blocker.

[a]Independent sample *T* test, Mann–Whitney *U* test (iPTH) or Chi-square test (Sex, DM, Smoking history, Drinking history, Original kidney disease, RASi, CCB, β-blocker and Diuretic treatment) as appropriate

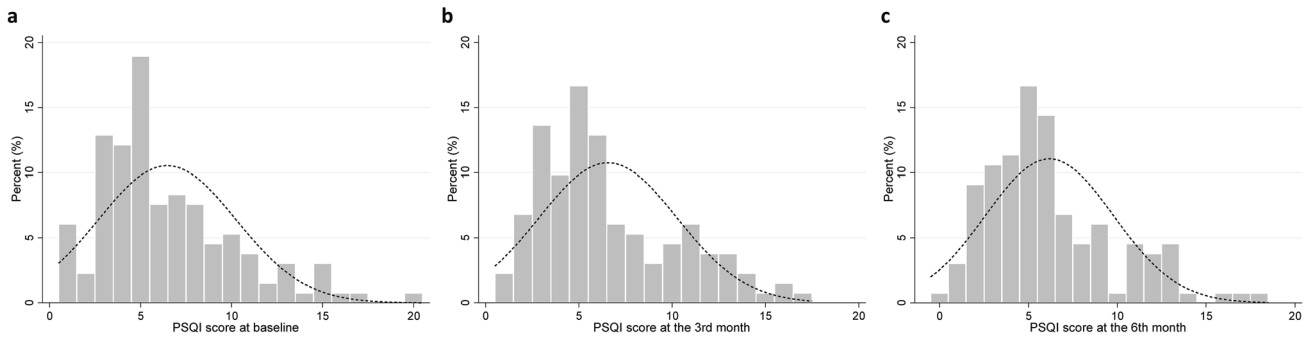

Fig. 2 Histogram of the PSQI score. **a** PSQI score at baseline; **b** PSQI score at the 3rd month; **c** PSQI score at the 6th month

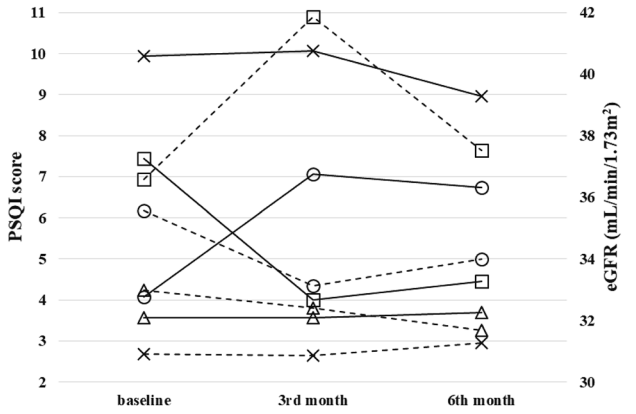

Fig. 3 Longitudinal PSQI scores and eGFR values at three time points. Solid line present the longitudinal PSQI score value; dash line present the longitudinal eGFR value. (△) Patients whose PSQI ≤ 5 at baseline and PSQI ≤ 5 at 3rd month; (×) Patients whose PSQI > 5 at baseline and PSQI > 5 at 3rd month; (○) Patients whose PSQI ≤ 5 at baseline and PSQI > 5 at 3rd month; (□) Patients whose PSQI > 5 at baseline and PSQI ≤ 5 at 3rd month

score change occurred in 30%, 20%, 40%, 40%, 20%, 0% and 50% cases separately.

Discussion

To the best of our knowledge, this study first examined the relationship between the trajectory of sleep disturbance and residual renal function in patients with non-dialysis CKD. Our study found that patients with CKD usually have poor sleep quality, which is comparable to that in previous studies [6, 9, 10]. During the 6-month study period, a significant relationship was observed between worsened sleep disturbance and decline in residual renal function in our cohort.

Compelling studies have reported that decreased sleep quality is associated with the development and progression of CKD. In patients with non-apnea sleep disorders (NASDs), the risk of developing CKD increased by 14% [18]. A nationwide study performed in Taiwan using data from the National Health Insurance Research Database included 4674 patients who were newly diagnosed with sleep apnea (SA) and 23,370 patients without SA as controls [19]. Results showed a 1.9-fold increase in the incidence of CKD in patients with SA compared with the controls. In addition, the incidence of ESRD increased to 2.2-fold in the population with SA. A similar result was also found in another population-based cohort study that included 4319 patients with obstructive sleep apnea (OSA) but excluded those with comorbidities, such as hypertension and diabetes [20]. The hazard ratio for the development of CKD was 1.37 in the cohort with OSA.

Table 2 Linear regression for eGFR change with Pittsburgh Sleep Quality Index scores across six month

Characteristic	PSQI ≤ 5 at baseline						PSQI > 5 at baseline					
	Model 1			Model 2			Model 1			Model 2		
	R^2	β (95% CI)	*p* value	R^2	β (95% CI)	*p* value	R^2	β (95% CI)	*P* value	R^2	β (95% CI)	*P* value
PSQI score (baseline)	0.001	− 0.11 (− 1.17 to 0.96)	0.845	0.062	− 0.10 (− 1.37to 1.1)	0.875	0.007	− 0.19 (− 0.80 to 0.42)	0.541	0.201	− 0.26 (− 0.95 to 0.42)	0.443
PSQI score (3rd month)	0.023	− 0.48 (− 1.28 to 0.31)	0.230	0.080	− 0.48 (− 1.44 to 0.48)	0.319	0.027	− 0.35 (− 0.91 to 0.20)	0.209	0.203	− 0.29 (− 1.00 to 0.41)	0.406
PSQI score (6th month)	0.025	− 0.37 (− 0.96 to 0.21)	0.208	0.084	− 0.38 (− 1.07 to 0.30)	0.266	0.018	− 0.28 (− 0.82 to 0.26)	0.307	0.200	− 0.27 (− 0.97 to 0.44	0.455

Model 1: Univariate

Model 2: Adjusted for age, sex, systolic blood pressure, diabetes, hemoglobin, serum albumin and renin-angiotensin system inhibitors (RASi) treatment

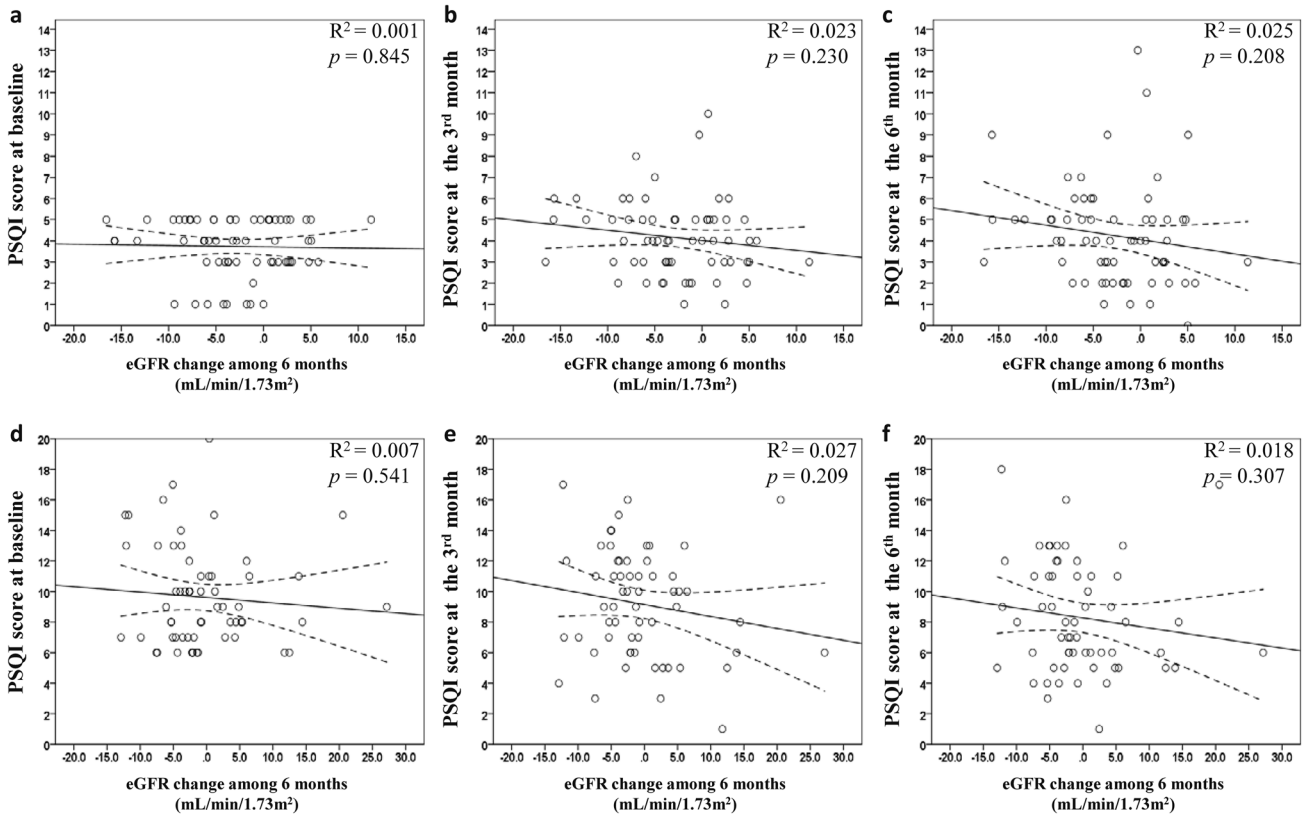

Fig. 4 The univariate associations between PSQI score and eGFR change among 6 months. **a** The association between PSQI score at baseline and eGFR change in patients whose PSQI score at baseline ≤ 5. **b** The association between PSQI score at the 3rd month and eGFR change in patients whose PSQI score at baseline ≤ 5. **c** The association between PSQI score at the 6th month and eGFR change in patients whose PSQI score at baseline ≤ 5. **d** The association between PSQI score at baseline and eGFR change in patients whose PSQI score at baseline > 5. **e** The association between PSQI score at the 3rd month and eGFR change in patients whose PSQI score at baseline > 5. **f** The association between PSQI score at the 6th month and eGFR change in patients whose PSQI score at baseline > 5

Table 3 Logistic regression for 3 groups according to PSQI trajectory on eGFR change among 6 months

Groups	Model 1			Model 2		
	OR	95% CI	*p* value	OR	95% CI	*p* value
Worsen group vs. Improve group	7.333	1.272–42.294	0.026	9.187	1.159–72.844	0.036
Stable group vs. Improve group	2.568	0.646–10.212	0.181	3.029	0.532–17.250	0.212
Worsen group vs. Stable group	2.856	0.855–9.539	0.088	3.033	0.788–11.670	0.107

Stable group as PSQI ≤ 5 at baseline + PSQI ≤ 5 at 3rd month and PSQI > 5 at baseline + PSQI > 5 at 3rd month; Worsen group as PSQI ≤ 5 at baseline + PSQI > 5 at 3rd month; < Improve group as PSQI > 5 at baseline + PSQI ≤ 5 at 3rd month

Model 1: Univariate

Model 2: Adjusted for age, sex, systolic blood pressure, diabetes, hemoglobin, serum albumin and renin-angiotensin system inhibitors (RASi) treatment

The mechanisms underlying sleep disorders with the progression of CKD and ESRD may be via the direct and indirect pathways [6]. Sleep disorders are always accompanied by activated sympathetic nervous system and renin–angiotensin–aldosterone system [21, 22]. This activation does not only induce hypertension but also directly affects renal outcome by causing glomerular capillary hypertension and endothelial dysfunction [23, 24]. The indirect effects are via the various risk factors of CKD and ESRD (e.g., hypertension, diabetic mellitus, obesity, and cardiovascular diseases). In a study conducted on 1137 employees aged 30–64 years, sleep disorder was found to be significantly associated with hypertension and dyslipidemia [25]. The incidence rate of diabetes was significantly higher in patients with NASDs

than in age- and sex-matched controls [26]. Sleep disorder increases the risk of developing vascular diseases, such as ischemic stroke and acute coronary syndrome [27, 28]. A higher body mass index is associated with short sleeping time and development of CKD based on prior studies [29–32]. Thus, impaired sleep quality can initiate and enhance CKD progression via various pathogenic pathways.

In the present study, we found that subjects who presented PSQI worsen to > 5 from ≤ 5 at baseline demonstrated declined eGFR at 6th month. However, eGFR in subjects who had PSQI improved to ≤ 5 from > 5 at baseline were not changed at 6th month. In addition, subjects who had persisted PSQI > 5 at baseline and 6th month were not worsen their eGFR at 6th month. Due to small number of diabetic subjects in our study, we did not further examine the relationship between diabetes and sleep quality and residual renal function decline. In addition, natural course of diabetic kidney disease is in wide-range variation and may be modified by intervention tools in the clinical practice [33]. Nevertheless, it is worthy to investigate these associations in the future study. In summary, rigorous program to improve sleep disturbance cannot eliminate the possibility of eGFR decline across 6 month when patients already existed poor sleep quality. In contrast, patients who did not present poor PSQI scores, measures to maintain their sleep quality could avoid their eGFR decline across 6 months.

The strength of the present study was the use of a prospective design to assess the relationship between sleep disturbance and changes in residual renal function in a cohort of patients with CKD stage 3–5. To examine the impact of sleep disturbance, we used the longitudinal PSQI scores within 6 months to predict eGFR changes during the course. The trajectory of the consecutive PSQI scores provided an insight about the risk of eGFR decline associated with sleep disturbance at 6-month interval. Of note, our results indicated that sleep disturbance played an important role in the deterioration of residual renal function and more marked in the subjects with continuously deteriorated sleep quality. The current study also had some limitations. First, several comorbidities could have affected sleep disturbance and residual renal function. However, these complex interactions were not adequately examined in our study. Thus, a large-scale population-based study must be conducted. Second, our study included a small number of patients from a single hospital. Therefore, our results may be not generalizable to other patients in other CKD care facilities. Moreover, a cause-effect relationship between sleep disturbance and CKD progression could not be identified using our data. Third, we used the trends of PSQI scores at baseline, 3rd month and 6th month as predictors of changes in residual renal function across 6 months. The cut-off time points were arbitrary. We hypothesized that the treatment measures for improving sleep disturbance can be effective at least after 3 months. However, we did not compare the advantages and disadvantages of using other cut-off time points in predicting consecutive eGFR changes. Therefore, the best cut-off time point that can be used to observe changes in PSQI score was not identified in our study. Finally, the observational duration was short, sleep disturbance on the long term renal outcome deserved to be investigated. In the present study, we did not use intervention tools that can improve sleep disturbance in our participants. The participants found their own coping measures to overcome sleep disturbance during the study period. Thus, the actual influence of these components on residual renal function was not identified in our study.

In conclusion, a relationship was observed between worsening sleep disturbance and eGFR decline in non-dialysis CKD patients. Integrated management need to be implemented on CKD patients to avoid renal dysfunction progression including maintaining a good sleep quality.

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Compliance with Ethical Standards

Conflict of interest All the authors have declared no competing interest.

Informed consent Informed consent was obtained from all individual participants included in the study.

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