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CLINICAL STUDY

Predictors of poor sleep quality and excessive daytime sleepiness in peritoneal dialysis patients

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Abstract

To explore the possible impact factors on daytime sleepiness among peritoneal patients from a single center in China. A cross-sectional study was conducted in 98 prevalent peritoneal dialysis (PD) patients using both the Pittsburgh Sleep Quality Index (PSQI) questionnaire of sleep quality and the Epworth Sleepiness Scale (ESS) questionnaire of excessive daytime sleepiness (EDS). Biochemical differences between daytime sleepiness and non-daytime sleepiness population were evaluated, following univariate and multivariable analysis to find the risk factors on sleep disturbance. The prevalence of ''poor sleep quality'' (PSQI >5) was 74.49%, while daytime sleepiness (ESS ≥ 9) occurred in 22.45%. Mean PSQI was 9.06 ± 4.60 and EES was 6.31 ± 4.98. Compared to non-EDS cases, patients with ESS ≥ 9 had worse residual renal function (RRF), higher serum creatinine, higher serum magnesium and elevated serum ferritin. In univariate analysis, ESS correlated with serum albumin ($r = 0.346$, $p = 0.015$), phosphate ($r = 0.313$, $p = 0.029$), magnesium ($r = 0.376$, $p = 0.008$) and urinary Kt/V ($r = -0.341$, $p = 0.029$). Finally, multivariable linear regression indicated that urinary Kt/V, PSQI and magnesium were independent predictors of ESS score. EDS does exist in PD patients and is associated both with poor nighttime sleep quality and lower RRF. Hypermagnesemia may be a treatable risk factor to improve daytime tiredness.

Keywords

Daytime sleepiness, peritoneal dialysis, quality of life, residual renal function

History

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Introduction

Sleep complaints and sleep disorders had been reported quite prevalent in dialysis cases,[1–3] and poor sleep quality was independently linked to higher mortality rate in hemodialysis (HD) patients.[4] Due to the lots of pattern of sleep disorder and many modes of dialysis, it was hard to achieve a well-recognized theory to elucidate the data. Comparing with that in HD, fewer studies focused on the sleep disorder in peritoneal dialysis (PD) patients, not to mention works in certain type of sleep, i.e., insomnia, sleep apnea, excessive daytime sleepiness (EDS) and restless legs syndrome. In aged population, EDS was associated high rate of cardiovascular disease,[5] and the clinical significant EDS might be more frequent, comparing with aged population. Only two single-center researches investigated this symptom with relative small sample-size in PD patients in latest years.[6,7] Thus, by the self-reported instruments, the aim of this study was to identify the prevalence of global and certain aspect of sleep quality in PD patients from eastern China as well as clinical correlates.

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Materials and methods

Subjects

From January 2010 to March 2010, we screened 110 PD patients and enrolled 98 from the Dialysis Unit of Changhai Hospital, Shanghai, China. Exclusion criteria were PD history of less than three months, congestive heart failure, liver cirrhosis, severe chronic obstructive pulmonary disease, mental or psychiatric impairment or unwillingness to participate in the study. The study protocol was approved by the local ethics committee, and written informed consent was obtained from each participant. Enrolled patients completed two questionnaires during in hospital dialysis session or while waiting for a consultation. Before starting, the patients had each questionnaire explained, and assistance was also provided during the filling out, if needed.

Questionnaires

For assessment of general sleep quality, the Pittsburgh sleep quality index (PSQI) was used, which had been developed by Buysse et al.[8] Furthermore, the Chinese version of the PSQI had also been validated.[9] It was a valid and standardized measurement for sleep quality evaluation and it could make a reliable semi-quantification of sleep during the past one month period. The questionnaire contains 19 self-rated questions yielding seven specific patient components.

Each component was scored from 0 to 3, yielding a global PSQI score between 0 and 21, with higher scores indicating lower quality of sleep. The obtained data are translated into the overall sleep quality as good (0–5 points) and poor (6–21 points). Although PSQI achieved worldwide good performance of sleep assessment, the index could not indicate the certain type of the prevalence of sleep disorders. Thus some specific questionnaires were introduced into the clinical research of certain type of sleep quality.

In general, daytime sleep function was assessed subjectively by the Epworth Sleepiness Scale (ESS). The ESS asked patients to rank their likelihood of dozing in eight different situations, with ''0'' indicating ''would never doze'' and ''3'' indicating ''high chance of dozing''. The total score ranges from 0 to 24, with more than 9 indicating the presence of EDS.[10] The validity and reliability of the Chinese version of ESS had also been conducted.[11]

A review of patients' charts was carried out within one month of recruitment, and relevant clinical data were also recorded. Adequacy of dialysis was assessed by Kt/V. The total weekly urea Kt/V (total Kt/V) and the normalized protein catabolic rate (nPCR) were analyzed based on 24 hours peritoneal effluent and urine collection. The contribution of total Kt/V by PD (Kt/V PD) and residual renal function (RRF), defined as urinary Kt/V (KRU) were estimated separately. All of the bio-fluid samples were collected according the standard protocol, and the specimen was preserved well under the circumstance of four degree before analyzed by automatic biochemical analyzer (HITACHI 7600; Hitachi Co., Tokyo, Japan).

Statistical analysis

Data was expressed as mean ± SD or as median (25%–75% quartiles), as appropriate. Comparisons between groups were done by an unpaired Student's *t* test. Nonparametric data were compared using a Mann–Whitney *U* test. For normally distributed variables, Pearson's correlation test was used; for non-normally distributed variables, Spearman's rank correlation test was used. Factors reaching statistical significance and the traditional risk factors were then included in a multivariable linear regression analysis with stepwise model. All *p* values were two-tailed, and values of <0.05 were considered to indicate statistical significance. All analyses were performed using the SPSS statistical software for Windows (version 17.0, SPSS Inc., Chicago, IL).

Results

The characteristics of the 98 patients are listed in Table 1, with differences between groups shown in Table 2. The average age was 57.12 years, and 56.12% were male. The median duration of CAPD was 16 months. The average PSQI score and ESS score for study participants were 9.06 ± 4.60 and 6.31 ± 4.98, separately. Twenty-five patients (25.51%) were classified as good sleepers (PSQI ≤ 5) and 73 patients (74.49%) were bad sleepers (PSQI > 5). The average score of PSQI in good and bad sleepers were 3.50 and 10.86, respectively. Compared with bad sleepers, good ones had significantly higher KRU ($p = 0.046$). Patients having ESS score ≥9 (indicating the presence of EDS) were 22.45% of the total sample. Compared with patients ESS ≥ 9, patients with EDS had a worse RRF (KRU 0.31 ± 0.30 vs. 0.59 ± 0.40, $p = 0.037$) significantly higher serum creatinine (1010.73 ± 340.03 vs. 776.16 ± 323.12 mmol/L, $p = 0.041$), higher serum magnesium (1.09 ± 0.37 vs. 0.91 ± 0.18 mmol/L, $p = 0.034$) and an elevated serum ferritin (575.45 vs. 332.90, $p = 0.010$).

Table 1. Characteristics of peritoneal dialysis patients recruited in the cross-sectional study.

Parameters	PD patients
Age, years	57.12 ± 14.90
Male/female	55/43
Duration of PD, months	16 (1026.50)
BMI, kg/m^2	23.60 ± 3.67
Hemoglobin, g/L	104.57 ± 22.44
Serum albumin, g/L	34.29 ± 4.61
BUN, mmol/L	19.12 ± 7.01
Creatinine, μmol/L	808.82 ± 338.15
Corrected calcium, mmol/L	2.36 ± 0.24
Phosphate, mmol/L	1.96 ± 0.69
Ca × P product, mg^2/dL^2	54.49 ± 19.79
Magnesium, mmol/L	0.95 ± 0.24
Total cholesterol, mmol/L	4.73 ± 1.25
Triglyceride, mmol/L	1.95 ± 1.13
HDL cholesterol, mmol/L	0.99 ± 0.36
LDL cholesterol, mmol/L	2.77 ± 0.97
TSAT,%	29.72 ± 14.82
Ferrtin, ng/mL	357.55 (198.55–590.53)
Intact-PTH, pg/mL	186.15 (82.20–447.23)
hs-CRP, mg/L	3.22 (3.22–5.43)
Total Kt/V	2.04 ± 0.47
Kt/V PD	1.56 ± 0.42
KRU	0.49 ± 0.40
NPCR (g/kg/day)	0.88 ± 0.24
Global PSQI	9.06 ± 4.60
ESS	6.31 ± 4.98

Notes: Data expressed as mean ± SD or as median (first, third quartile), as appropriate. PD, peritoneal dialysis; HDL cholesterol, high-density lipoprotein cholesterol; LDL cholesterol, low-density lipoprotein cholesterol; TSAT, transferrin saturation; PTH, parathyroid hormone; hs-CRP, high-sensitivity C-reactive protein; nPCR, normalized protein catabolic rate; total Kt/V, total weekly Kt/V urea; Kt/V PD, peritoneal Kt/V; KRU, urinary Kt/V.
Statistical significance was set at $p < 0.05$.

Univariate correlates of PSQI and ESS are listed in Table 3. Briefly, PSQI correlated with a low KRU ($r = -0.392$, $p = 0.032$), while ESS correlated with serum albumin ($r = 0.346$, $p = 0.015$), phosphate ($r = 0.313$, $p = 0.029$), magnesium ($r = 0.376$, $p = 0.008$) and KRU ($r = -0.341$, $p = 0.029$). The relationships between PSQI and KRU, as well as ESS between KRU are illustrated in Figure 1.

Table 4 lists independent predictors of PSQI and ESS. Briefly, PSQI was predicted by KRU ($\beta = -0.406$, $p = 0.029$) and ESS ($\beta = 0.546$, $p < 0.001$), while ESS was predicted by KRU ($\beta = -0.296$, $p = 0.047$), magnesium ($\beta = 0.314$, $p = 0.019$) and PSQI ($\beta = 0.496$, $p < 0.001$).

Discussion

This study confirmed previous data suggesting a high prevalence of poor sleep quality amongst patients undergoing PD.[12–15] We also found that EDS is associated both with a poor nighttime sleep quality and a lower RRF.

Table 2. Characteristics of groups with different sleep quality among the 98 PD patients.

	PSQI			ESS		
Variables	≤5	>5	*p* Value	<9	≥9	*p* Value
Score	3.50 ± 1.38	10.86 ± 3.72	<0.001*	4.21 ± 2.59	13.55 ± 4.46	<0.001*
Age, years	54.00 ± 11.53	58.14 ± 15.84	0.409	58.26 ± 15.34	53.18 ± 13.10	0.324
Male/female	15/10	40/33		41/35	14/8	
Duration of PD, months	20	23	0.655	19	22	0.601
BMI, kg/m^2	23.26 ± 3.07	23.71 ± 3.88	0.712	23.56 ± 3.76	23.73 ± 3.54	0.894
Hemoglobin, g/L	99.75 ± 22.12	106.14 ± 22.62	0.397	104.42 ± 22.16	105.09 ± 244.51	0.932
Serum albumin, g/L	34.50 ± 4.60	34.22 ± 4.67	0.855	34.21 ± 4.30	34.55 ± 5.77	0.834
BUN, mmol/L	21.62 ± 9.46	18.37 ± 5.96	0.165	18.18 ± 6.99	22.57 ± 6.16	0.066
Creatinine, μmol/L	819.82 ± 367.29	831.70 ± 333.48	0.918	776.16 ± 323.12	1010.73 ± 340.03	0.041*
Corrected calcium, mmol/L	2.32 ± 0.26	2.37 ± 0.23	0.542	2.34 ± 0.23	2.41 ± 0.25	0.371
Phosphate, mmol/L	2.09 ± 0.87	1.91 ± 0.63	0.444	1.90 ± 0.70	2.17 ± 0.65	0.252
Ca × P product, mg^2/dL^2	56.76 ± 21.67	53.75 ± 19.40	0.652	52.36±19.79	61.84 ± 18.81	0.164
Magnesium, mmol/L	0.97 ± 0.22	0.94 ± 0.26	0.696	0.91 ± 0.18	1.09 ± 0.37	0.034*
Total cholesterol, mmol/L	4.53 ± 1.12	4.80 ± 1.30	0.531	4.79 ± 1.34	4.55 ± 0.94	0.582
Triglyceride, mmol/Ll	2.02 ± 0.94	1.63 ± 1.19	0.352	1.93 ± 0.78	2.01 ± 1.97	0.843
HDL cholesterol, mmol/L	0.89 ± 0.25	1.02 ± 0.38	0.263	0.99 ± 0.53	0.99 ± 0.39	0.975
LDL cholesterol, mmol/L	2.54 ± 1.02	2.85 ± 0.95	0.345	2.86 ± 1.03	2.48 ± 0.62	0.260
TSAT,%	26.44 ± 9.46	30.71 ± 16.08	0.409	28.95 ± 13.35	32.53 ± 19.94	0.504
Ferrtin, ng/mL	334.50 (153.31–833.73)	357.55 (206.63–582.13)	0.728	332.90 (153.05–508.35)	575.45 (442.70–947.25)	0.010*
Intact-PTH, pg/mL	271.00 (93.59–454.78)	168.30 (66.43–446.95)	0.627	172.30 (78.49–468.75)	315.90 (61.65–424.50)	0.870
hs-CRP, mg/L	3.22 (3.22–3.77)	3.59 (3.22–5.81)	0.301	3.22 (3.22–4.79)	4.33 (3.00–6.08)	0.795
Total Kt/V	1.97 ± 0.47	2.07 ± 0.47	0.530	2.11 ± 0.39	1.85 ± 0.66	0.126
Kt/V PD	1.39 ± 0.39	1.62 ± 0.42	0.138	1.56 ± 0.30	1.54 ± 0.70	0.867
KRU	0.58 ± 0.43	0.47 ± 0.39	0.046*	0.59 ± 0.40	0.31 ± 0.30	0.037*
nPCR (g/kg/day)	0.87 ± 0.17	0.88 ± 0.27	0.909	0.88 ± 0.25	0.88 ± 0.22	0.997

Note: Statistical significance was set at *$p < 0.05$.

Table 3. Variables correlation with global PSQI score and ESS score.

	PSQI		ESS	
Variables	*r*	*p*	*r*	*p*
Age, years	0.092	0.530	−0.118	0.421
Duration of PD, months	0.091	0.534	0.047	0.749
BMI, kg/m^2	0.071	0.627	−0.017	0.910
Hemoglobin, g/L	−0.199	0.170	0.273	0.057
Serum albumin, g/L	−0.107	0.464	0.346	0.015*
Creatinine, μmol/L	0.103	0.483	−0.045	0.670
Corrected calcium, mmol/L	0.497	0.099	0.144	0.325
Phosphate, mmol/L	0.102	0.486	0.313	0.029*
Ca × P product, mg^2/dL^2	0.099	0.497	0.335	0.019*
Magnesium, mmol/L	0.034	0.818	0.376	0.008*
Total cholesterol, mmol/L	0.169	0.246	−0.026	0.860
Triglyceride, mmol/Ll	0.786	0.078	0.089	0.543
HDL cholesterol, mmol/L	0.034	0.818	−0.066	0.654
LDL cholesterol, mmol/L	0.045	0.761	−0.067	0.649
TSAT,%	0.009	0.953	−0.045	0.764
Ferrtin, ng/mL	0.127	0.421	0.244	0.102
Intact-PTH, pg/mL	−0.028	0.852	−0.047	0.757
hs-CRP, mg/L	0.019	0.899	0.134	0.392
Total Kt/V	−0.150	0.336	−0.201	0.202
Kt/V PD	0.179	0.262	0.083	0.606
KRU	−0.392	0.032*	−0.341	0.029*

Note: Statistical significance was set at *$p < 0.05$.

Daytime sleepiness is not studied well in PD patients, but the few papers suggest that the prevalence may vary greatly between populations. Indeed, our finding of a 22% prevalence was similar to that reported by Chen et al.[16] (17%) in 710 Taiwanese HD patients, but higher than that reported by Merlino et al.[17] (12%) in 883 Italian dialysis patients. Sabbatini et al. reported a much higher prevalence (41%) in 694 Italian HD patients,[18] while other studies also reported different prevalence of EDS in HD or mixed patients.[19,20] This large variation may reflect both ethnic and cultural differences in subjective reporting, as well as different impacts of dialysis modality and dose, comorbidities and medication use. Meanwhile, the etiology of sleep disorders among dialysis patients are not well understood and are likely to be multifactorial. Old age, smoking, alcohol consumption caffeine intake, pruritus, bone pain, discontinuing dialysis and neuropathy have all been related to insomnia or sleep quality in dialysis patients.[1,2,11,17]

To these data, our study added the importance of nighttime sleep for daytime wakefulness, as well as the novel relationship between serum magnesium and sleep quality. Sleep apnea and low O_2 saturation levels could conceivably be caused by hypermagnesemia due to shallow breathing,[21] which may be one explanation. Furthermore, and not surprisingly, we find that patients with better preserved RRF tend to be less tired during the day. Though the NECOSAD study reported an important contribution of RRF to most dimensions of quality of life by the means of another questionnaire, SF-36, involving sleep disorders.[22] To the best of our knowledge, it was the first study to find the possible link between RRF and EDS in PD patients. Uremic encephalopathy is well described in ESRD, and daytime sleepiness might share some of these characteristics.[23] Preservation of RRF was of clinical relevance in PD, which can bring many benefits, such as reduction in blood pressure and left ventricular hypertrophy, increased sodium removal, improved fluid condition, increased β2-microglobulin clearance and higher serum hemoglobin levels, better nutritional status and decreased circulating inflammatory markers,[24] though most of the specific underlying mechanisms are still poorly understood. Recently, Ignace et al. found that a preserved RRF is associated with lower levels of oxidative stress

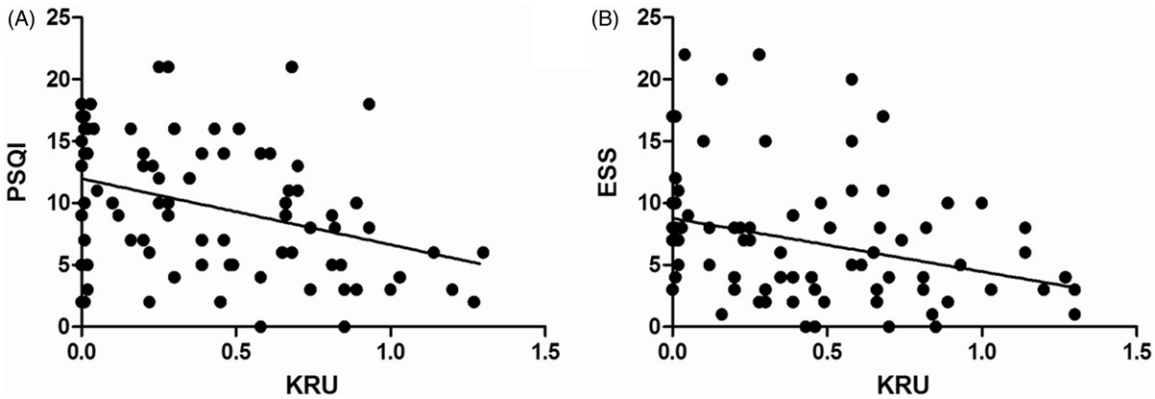

Figure 1. Correlation between Pittsburgh Sleep Quality Index and urinary Kt/V (KRU) (A), Epworth Sleepiness Scale (B) and urinary Kt/V (KRU).

Table 4. Multivariable linear regression analysis for the global PSQI score and ESS score.

Variables	β Value	*p* Value
PSQI		
KRU	−0.406	0.029*
ESS	0.546	<0.001*
ESS		
KRU	−0.296	0.047*
Magnesium	0.314	0.019*
PSQI	0.496	<0.001*

Note: Statistical significance was set at $*p < 0.05$.

markers in stable PD patients.[25] It is possible that lower RRF is the link between sleep disturbance and reduced survival in PD patients. Unlike the data in HD patients,[26] we did not find any relationship of inflammation (measured as high-sensitivity C-reactive protein and interleukin 1β) with sleep quality or daytime tiredness (data not shown). This may be explained as RRF was relatively better preserved in PD than HD patients. Latest report indicated that residual diuresis had a beneficial effect on the left ventricular function in HD patients,[27] and fluid overload and micro-inflammation were associated with left ventricular hypertrophy.[28] Thus we speculated that the better RRF was associated with better left ventricular function, which reduced fluid overload and micro-inflammation. Resulting of lower fluid overload and inflammation status improved EDS quality were found in better RRF ones.

In the other hand, the relationship between calcium–phosphate metabolism and ESS had been also demonstrated in this work. Just like the inflammatory status, the potential link could be found between good RRF and calcium–phosphate balance. Meanwhile the higher phosphate level, the higher risk for arterial calcification and endothelial dysfunction, while would be the source of insomnia, cognitive impairment and daytime sleepiness.

Also of interest was the relationship of ESS with PSQI. It was conceivable that patients with poor sleep quality at night might experience excessive tiredness during the day. Thus, these patients might also require taking a longer daytime nap to compensate for the nocturnal sleep deprivation.[12] Recently, ESS had been suggested to reflect sleep apnea,[29] a risk factor for cardiovascular morbidity and mortality in PD patients,[30] while the complication was potentially treatable. Tang et al. reported improving sleep with transfer to nocturnal cycler-assisted PD.[31]

Our work has several limitations that should be kept in mind. First, PSQI and ESS were subjective and semi-quantitative measurements. Objective and quantitative tools like comprehensive polysomnography and the Multiple Sleep Latency Test were not performed. Second, certain medications such as beta-blockers, statins, sleep medications and anti-epileptics, which may influence sleep quality, were not controlled. Finally, this cross-sectional study can not reveal the causality in observed relationships.

In conclusion, we report a high prevalence of poor sleep quality in PD patients and the correlation of daytime tiredness with low RRF, poor nighttime sleep quality and hypermagnesemia in PD patients, suggesting that interventions aimed at increasing daytime alertness could be one way to improve the quality of life in PD patients.

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Declaration of interest

All the authors declared no competing interests.

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