Original Article

Analysis of Early Kidney Injury-Related Factors in Patients with Hypertension and Obstructive Sleep Apnea Hypopnea Syndrome (OSAHS)

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Abstract

Objective: The aim of this study was to analyze the related factors of obstructive sleep apnea hypopnea syndrome (OSAHS) that would affect early kidney injury in patients with hypertension (Hyp).

Methods: A total of 457 Hyp patients with nocturnal snoring were selected for polysomnography (PSG). The patients were divided into four groups according to the apnea hypopnea index (AHI), and the related factors that would impact blood urea, creatinine, 24 hr urinary protein (24 hr UTP), 24 h urinary microalbuminuria and serum cystatin C (Cyst C) were analyzed in the groups.

Results: Severe OSAHS (OR = 4.880, 95% CI = 1.577~15.099) was the influencing factor for 24 hr UTP; blood pressure control (OR = 2.335, 95% CI = 1.326~4.112) and Obesity (OR = 2.072, 95% CI = 1.236~3.474) were the influencing factors for 24 hr urinary microalbuminuria; age (OR = 1.996, 95% CI = 1.366~2.917), blood pressure control (OR = 2.895, 95% CI = 1.267~6.615) and severe OSAHS (OR = 6.093, 95% CI = 1.267~29.303) were the influencing factors for Cyst C. As for the Hyp patients associated with OSAHS, severe OSAHS were the influencing factors for 24 hr UTP; blood pressure control and Obesity were the influencing factors for 24 hr urinary microalbuminuria; age, blood pressure control and severe OSAHS were the influencing factors for Cyst C.

Conclusions: OSAHS is a risk factor of early kidney injury.

Keywords: Hypertension, kidney injury, obstructive sleep apnea hypopnea syndrome

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Introduction

The obstructive sleep apnea hypopnea syndrome (OSAHS) refers to a respiratory sleep disorder in which the patients exhibit recurrent apnea or hypopnea during sleeping because of complete or partial stenosis in upper airway. The recurrence times per night are more than 30 times, or the sleep apnea hypopnea index (AHI) is ≥5 times/hour, and the patients are also afflicted with such clinical symptoms as lethargy, etc. It is a common clinical disease that seriously threatens human health. The prevalence of this disorder in North American and Chinese cohorts is shown to be 24% and 18.8% (AHI > 5) and 9% and 5.3% (AHI > 15), respectively.[1–3] OSHAS may cause intermittent hypoxia during sleep, increase the excitability of chemoreceptors and intrathoracic negative pressure, microarousal during sleeping, sympathetic overactivity,[4] excessive releasing of renin-angiotensin-aldosterone hormone,[5] insulin resistance, inflammation and endothelial dysfunctions,[6] etc., thus leading to multiple organ damage. With gradual worsening of OSAHS conditions, the patients might be at increased risk for such serious complications as pulmonary heart disease,[7] hypertension,[8,9] coronary heart disease,[10] arrhythmias[11] and stroke.[12] Clinical studies have found that with OSAHS severity increasing, the early renal damages of patients are aggravated.[13] Zhang showed that AHI was significantly and positively associated with serum cystatin C (Bate(B) = 0.284, $P = 0.007$). Cystatin C was associated with the severity of OSAHS in younger men.[14] Daskalopoulou reported that following continuous positive airway pressure (CPAP) treatment, urinary albumin excretion is reduced in OSAHS patients during sleep.[15] Kanbay, *et al*. reported that[16] OSAHS is a risk factor for the progression of chronic kidney disease, which is a growing health problem. In recent years, epidemiological studies have shown that 30% to 50% of hypertension (Hyp) patients are associated with OSAHS, and 50% to 60% of OSAHS patients are associated with hypertension.[17] The coexistence of hypertension and OSHAS increases the severity of patients' organ damage. The hypertensive renal damage is one of the complications in patients with essential hypertension, while whether OSAHS could increase the hypertensive kidney damages has not been studied much yet.[18] This study retrospectively analyzed the data from 457 Hyp patients, confirmed by the First Affiliated Hospital of Xinjiang Medical University, preliminarily studied the influencing factors of OSAHS on renal functions of Hyp patients, aiming to provide evidence to prevent, diagnose and treat the renal dysfunctions of Hyp patients associated with OSAHS.

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Materials and Methods

Subjects

A total of 475 Hyp patients (aged from 20 to 78 years, young

people: 202 people, mid adult: 203 people, old people: 52 people), confirmed in the First Affiliated Hospital of Xinjiang Medical University from January 2011 to January 2014 who complained of nocturnal snoring, were selected and underwent polysomnography (PSG). The patients were divided into 4 groups according to "Obstructive sleep apnea"[19]: The control group [pure hypertension group, with AHI < 10/hr, n = 106], the mild group (with AHI 10~15/hr, n = 102), the moderate group (with AHI > 15~30/hr, n = 153) and the severe group (with AHI >30/hr, n = 96). The diagnosis of hypertension was based on-JNC8[20]: Hypertension was defined as (1) a systolic blood pressure (SBP) ≥ 140 mmHg and/or a diastolic blood pressure (DBP) ≥ 90 mmHg, (2) use of antihypertensive medication, or (3) physician-diagnosis of hypertension as per clinical history. The diagnosis of OSAHS referred to "Obstructive sleep apnea",[19] which was mainly diagnosed based on the disease history, signs and PSG results. The clinical diagnostic criteria included the typical nocturnal snoring associated with apnea, daytime sleepiness [Epworth Sleepiness Score, ESS) ≥9 points] and other symptoms, the physical examination revealed stenosis obstruction of any part of airway, AHI ≥ 5/hr. Patients with 1 or more complications, such as obvious daytime sleepiness (ESS < 9 points), AHI ≥ 10 or ≥5 times/hr, cognitive dysfunction, hypertension, coronary heart disease, cerebrovascular disease, diabetes and insomnia, could also be diagnosed with the condition. This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of Xinjiang Medical University. Written informed consent was obtained from all participants.

Exclusion criteria

1) With secondary hypertension except for OSAHS; 2) with central and mixed sleep apnea syndrome; 3) with unstable lung disease; 4) with urinary system diseases: nephrotic syndrome, glomerular nephritis, acute and chronic renal failure, gout, urinary tract inflammation and urinary calculi; 5) with endocrine system diseases, including diabetes, hyperthyroidism and hypothyroidism psychosis; 6) with acute and chronic heart, liver and kidney dysfunctions, chronic wasting diseases and cancers; 7) with valvular heart disease, congenital heart disease and primary cardiomyopathy.

Overnight Embletta monitoring

Embletta (Embla Systems incorporated (Inc), The United States) was used for 7-hr monitoring and analysis during night. Caffeine, sedatives, hypnotics and alcohol should be prohibited on the examination day. The monitoring indicators included AHI and the time points of mean oxygen saturation degree ($MSaO_2$), lowest oxygen saturation ($LSaO_2$) and oxygen saturation <85% (TS < 85%). Height and weight were measured in the evening before the examination, and body mass index (BMI) was then calculated [BMI = body weight (kg) / height^2 (m^2)].

Blood pressure measurements

The dynamic blood pressure monitor ABPM6100 (Welch Allyn, USA) was used, with the blood pressure measurements every 30 min during daytime and every 60 min at night. At the end of monitoring, we recorded the 24 hr mean systolic blood pressure (24 hr SBP), 24 hr mean diastolic blood pressure (24 hr DBP) and 24 hr mean pulse pressure (24 hr PP). According to the standards of 2014 Guideline for Management of High Blood Pressure,[20] hypertension was defined as (1) a systolic blood pressure (SBP) ≥140mmHg and/or a diastolic blood pressure (DBP) ≥90 mmHg, (2) use of antihypertensive medication, or (3) physician-diagnosis of hypertension as per clinical history.

Determination of renal functions and other indicators (Roche Diagnostics GmbH, Germany)

All patients included fasted for 8 hr; then, venous blood was drawn in the next morning for the measurement of fasting blood glucose (FBG), triglyceride (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), lipoprotein a [LP (a)], urea, creatinine and serum cystatin C (Cyst C) by Roche C8000 automatic biochemical analyzer. The Beckman automatic biochemical analyzer Dxc800 was used to analyze the 24 hr urinary total protein (24 hr UTP) and 24 hr urinary microalbuminuria.

Diagnostic criteria of kidney damage

Diagnostic criteria: 1) diagnosis of kidney damage: 24 hr UTP was increased to ≥0.15 g/24 hr; 24 hr urinary microalbuminuria was increased to ≥30 mg/24 hr[21]; Cyst C was ≥1.03 mg/L.[22]

Statistical Analysis

SPSS17.0 statistical software was used for statistical analysis of all data. Quantitative data with normal distribution were expressed as mean ± standard deviation ($\bar{x} \pm s$). The intergroup comparison used one-way analysis of variance (ANOVA), the pairwise comparison used SNK test. The quantitative data with skewed distribution were expressed as median and interquartile range, and analyzed with the Wilcoxon test. The qualitative data were expressed by constituent ratio, and analyzed with the Chi-square test. The 24 hr UTP, 24 hr urinary microalbuminuria and Cyst C were considered as dependent variables, while the remaining various related factors were considered as the independent variables for the Logistic regression analysis. Meanwhile, the AHI group was considered as the dummy variable, with $P < 0.05$ considered as statistically significant.

Results

General conditions and PSG monitoring

Such baseline data as age, gender ratio, BMI, 24 hr SBP and fasting blood sugar in the 4 groups were statistically different ($P < 0.05$). The Hyp patients associated with various degrees of OSAHS had higher male proportion than the pure hypertension group ($P < 0.05$); the age of the moderate group was higher than the mild group and the control group ($P < 0.05$). BMI and 24 hr SBP of the severe group were higher than other groups; BMI of the moderate group was higher than the control group ($P < 0.05$); fasting blood glucose of the severe group was higher than the other three groups ($P < 0.05$); while hypertension duration, 24 hr DBP, triglyceride, total cholesterol, HLD-C, LDL-C, medications and medication types among the four groups had no significant difference ($P > 0.05$, Table 1).

Changes of renal functions

Urea and creatinine among the four groups had no statistically significant difference ($P > 0.05$). The 24 hr UTP and 24 hr urinary microalbuminuria of the severe group were higher than the control group and the mild group (all $P < 0.05$); Cyst C of every OSAHS group was higher than the control group ($P < 0.05$, Table 2).

Table 1. General information ($\bar{x}$ ±s).

		Control	OSAHS			*P*-Values
			Mild	Moderate	Severe	
Cases (M/F)		54/52	65/37[a]	99/54[b]	75/21[bcf]	<0.001
Age (years)		44.01±10.32	44.97±10.39	48.46±10.17[bc]	47.48±10.98	<0.001
BMI(kg/m^2)		26.35±3.19	27.38±3.68	27.60±3.27[a]	29.78±3.53[bdf]	<0.001
Duration of hypertension * (months)		36.00(12.00, 72.00)	36.00(11.00, 87.00)	60.00(12.00, 120.00)	36.00(12.00, 96.00)	0.250
24hSBP(mmHg)		129.25±15.78	129.66±14.44	130.03±14.24	135.33±16.91[ace]	0.015
24hDBP(mmHg)		81.43±12.57	80.48±11.49	80.76±10.35	83.08±10.70	0.344
AHI* (events/h)		7.95(6.45, 9.20)	13.00(11.65, 14.10)[b]	22.00(18.00, 25.95)[bd]	42.75(34.00, 54.52)[bdf]	<0.001
FBG * (mmol/L)		4.55(4.25, 4.90)	4.42(4.22, 4.86)	4.57(4.27, 4.95)	4.76(4.41, 5.23)[bde]	<0.001
TG * (mmol/L)		1.73(1.19, 2.41)	1.80(1.38, 2.36)	1.74(1.26, 2.40)	1.87(1.27, 3.02)	0.298
TC (mmol/L)		4.34±0.88	4.45±0.92	4.55±0.92	4.61±0.92	0.130
HDL-C* (mmol/L)		1.02(0.88,2.41)	0.97(0.81,1.14)	0.99(0.84,1.16)	0.96(0.78,1.11)	0.110
LDL-C (mmol/L)		2.63±0.70	2.74±0.77	2.81±0.74	2.79±0.83	0.280
Number of medication[*n* (%)]	none	27(25.47)	25(24.51)	30(19.61)	16(16.67)	0.363
	1	65(61.32)	52(50.98)	91(59.47)	50(52.08)	0.310
	2	12(11.32)	19(18.62)	22(14.37)	23(23.95)	0.080
	3	2(1.89)	6(5.88)	10(6.53)	7(7.29)	0.253
Medication[*n* (%)]	Calcium antagonists	71(66.98)	69(67.65)	101(66.01)	73(76.04)	0.380
	ACEI or ARB	16(15.09)	23(22.54)	38(24.83)	23(23.95)	0.274
	β-blockers	8(7.55)	15(14.71)	20(13.07)	17(17.71)	0.182
	Diuretics	1(0.94)	2(1.96)	6(3.92)	4(4.16)	0.390

Note: * = expressed by median (P25, P75); control group = AHI <10 times/hr; mild group = AHI 10 ~ 15 times/hr; moderate group = AHI> 15 ~ 30 times/hr; severe group = AHI > 30 times/hr. OSAHS = obstructive sleep apnea hypopnea syndrome; BMI = body mass index; 24 hSBP = 24 hr mean systolic blood pressure; 24 hDBP = 24 hr mean diastolic blood pressure; AHI = apnea-hypopnea index; HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; TC = total cholesterol; TG = triacylglycerol; FBG = fasting blood glucose. Compared with the control group, [a]$P < 0.05$, [b]$P < 0.01$. Compared with the mild group [c]$P < 0.05$, [d]$P <$ 0.0. Compared with the moderate group [e]$P < 0.05$, [f]$P < 0.0$.

Table 2. Renal functional indexes of simple hypertension group and Hyp-OSAHS association groups ($\bar{x}$±s).

Group	Cases	Urea (mmol/L)	Creatinine (mmol/L)	24 hr UTP* (g/24hr)	24 hr urinary microalbuminuria * (mg/24 hr)	Cyst C* (mg/mL)
Control	106	5.29±1.54	64.05±14.07	0.06(0.04,0.10)	5.20(2.18,14.93)	0.75(0.69,0.86)
OSAHS						
Mild	102	4.93±1.28	66.22±15.67	0.07(0.04,0.10)	4.63(2.13,17.65)	0.80(0.70,0.91)[a]
Moderate	153	5.11±1.23	67.39±14.68	0.07(0.05,0.11)	7.59(2.76,22.63)	0.83(0.74,0.93)[b]
Severe	96	5.15±1.36	68.75±15.12	0.09(0.05,0.14)[bd]	13.03(2.90,37.88)[bc]	0.83(0.75,0.90)[b]
P		0.222	0.132	0.005	0.010	<0.001

Note: * = expressed by median (P25, P75); control group = AHI < 10 times/hr; mild group = AHI 10 ~ 15 times/h; moderate group = AHI>15 ~ 30 times/h; severe group = AHI>30 times/hr. 24 hr UTP: 24 hr urinary protein; Cyst C = serum cystatin C; OSAHS = obstructive sleep apnea hypopnea syndrome. Compared with the control group, [a]$P < 0.05$, [b]$P < 0.01$. Compared with the mild group [c]$P < 0.05$, [d]$P < 0.0$.

Table 3. Variable assignment.

Risk factors	Variable assignment
Sex	Female = 0,Male = 1
Age(years)	<20 = 0,20 ~ <30 = 1,30 ~ <40 = 2,40 ~ <50 = 3,50 ~ <60 = 4,60 ~ <70 = 5, ≥70 = 6
Duration of hypertension (months)	<120 = 0,120 ~ <240 = 1,240 ~ <360 = 2,360 ~ <480 = 3, ≥480 = 4
Situation of blood pressure	Controlled = 0，Uncontrolled = 1
BMI(kg/m^2)	<28 = 0, ≥28 = 1
AHI(times/h)	<10 = 0, 10 ~ 15 = 1,15 ~ 30 = 2, >30 = 3
FBG (mmol/L)	<6.1 = 0, ≥6.1 = 1
TG (mmol/L)	<1.7 = 0, ≥1.7 = 1
TC (mmol/L	<5.72 = 0, ≥5.72 = 1
HDL-C(mmol/L)	<1.16 = 1, ≥1.16 = 0
LDL-C(mmol/L) Urea(mmol/L) Creatinine(mmol/L) 24 hr UTP(g/24 hr) 24 hr urinary microalbuminuria(mg/L) CystC(mg/L)	<3.1 = 0, ≥3.1 = 1 <8.2 = 0, ≥8.2 = 1 <115=0, ≥115=1 <0.15 = 0, ≥0.15 = 1 <30 = 0, ≥30 = 1 <1.03 = 0, ≥1.03 = 1

Logistic regression analysis

The 24 hr UTP, 24 hr urinary microalbuminuria and Cyst C were considered as the dependent variables, while gender, age, hypertension duration, blood pressure, Obesity, AHI, fasting plasma glucose, triglyceride, total cholesterol, HLD-C and LDL-C were considered as the independent variables for the Logistic regression. The assignments are shown in Table 3. The analysis showed that severe OSAHS was the influencing factor for 24 hr UTP in Hyp patients with OSAHS; blood pressure control and Obesity were the influencing factors for 24 hr microalbuminuria; age, blood pressure control and severe OSAHS were the influencing factors for Cyst C (Table 4).

Discussion

OSAHS-related kidney damage has been reported several times.[23,24] Basic research has demonstrated[25] that OSAHS could cause edema, degeneration and abnormalities of kidney tissues' ultrastructures, thus causing damage to kidney function. Epidemiological studies have shown that the coexistence of OSAHS and hypertension is high. The majority of OSAHS patients are obese, normally exhibit insulin resistance, dyslipidemia and other phenomena. However, it has not been determined whether OSAHS is the risk factor, independent from blood pressure, blood glucose, lipids and other reasons causing kidney injuries, as well as what the influencing factors of OSAHS-related kidney injuries may be. This study explores the relevant factors of renal functions when the Hyp patients are afflicted with various degrees of OSAHS.

This study selected Urea, creatinine,[26] 24 hr UTP, 24 hr urinary microalbuminuria[27] and Cyst C as indicators for evaluating renal function, among which 24 hr UTP, 24 hr urinary microalbuminuria and Cyst C[28–30] were the sensitive markers to evaluate early kidney damage, while the glomerular filtration rates of urea and creatinine would drop to 1/2 and 1/3 of normal values, respec-

Table 4. The Logistic regression analysis of the renal function in Hyp-OSAHS patients (n = 457).

	B	SE	Wald	*P*	OR	95% CI
24h UTP						
OSAHS						
Mild	0.566	0.608	0.868	0.351	1.762	0.535~5.795
Moderate	0.759	0.575	1.744	0.187	2.136	0.692~6.591
Severe	1.585	0.576	7.566	0.006	4.880	1.577~15.099
CystatinC						
Age	0.691	0.194	12.753	<0.001	1.996	1.366~2.917
Situation of blood pressure	1.063	0.422	6.357	0.012	2.895	1.267~6.615
OSAHS						
Mild	1.595	0.817	3.813	0.051	4.929	0.994~24.440
Moderate	1.292	0.790	2.675	0.102	3.640	0.774~17.121
Severe	1.807	0.801	5.085	0.024	6.093	1.267~29.303
24 hr urinary microalbuminuria						
Situation of blood pressure	0.848	0.289	8.624	0.003	2.335	1.326~4.112
Obesity	0.728	0.264	7.630	0.006	2.072	1.236~3.474

Note：24 hr UTP , 24 hr urinary microalbuminuria and Cyst C as the dependent variables, logistic regression model(backward wald) with indicator variables for gender, age, hypertension duration, blood pressure, Obesity, AHI, fasting plasma glucose, triglyceride, total cholesterol, HLD-C and LDL-C were set as the independent variables.

tively, then begin to rise,[31] and they are the indicators commonly used to assess kidney function when seriously damaged. In this study, the comparison of renal function index in early renal injury among the four groups showed that urea and creatinine among the 4 groups had no statistically significant difference ($P > 0.05$). The 24 hr UTP and 24 hr urinary microalbuminuria of the severe group were higher than the mild group; Cyst C in all OSAHS groups was higher than the control group ($P < 0.05$), indicating that as OSAHS severity increased, the extent of early kidney damage was also increased, while the renal function showed no significant difference. However, the results of basic information comparison showed that as OSAHS severity increased, age, gender ratio, BMI, 24 hr SBP and fasting blood sugar also increased, and the differences were statistically significant ($P < 0.01$), which is consistent with "Obstructive sleep apnea"[19] and clinical studies.[32,33] Furthermore, the results of logistic regression analysis of 24 hr UTP, 24 h urinary microalbuminuria and Cyst C showed that uncontrolled blood pressure and Obesity were the risk factors of 24 hr urinary microalbuminuria; age, uncontrolled blood pressure and severe OSAHS were the risk factors for Cyst C; and severe OSAHS was the risk factor for 24 hr UTP. Based on the above results, we could conclude that OSAHS is independent from age, sex, BMI, blood pressure and blood sugar, and is the risk factor for 24 hr UTP and Cyst C. Also, the fact that the hypertension-OSAHS patients had higher 24 hr urinary microalbuminuria than the patients with pure hypertensive was obviously mainly related with BMI and blood pressure.

Dickson and Wagner reported that when proteins passed glomerular filtration membrane, only a very small part of small molecule proteins, with positive charges, could filter through the glomerulus, while most proteins that filtered into the original urine were reabsorbed inside the proximal tubule, and the urine content was very low, therefore, the increased urinary protein prompted early renal damage.[34] Cyst C is a non-glycosylated basic protein, which could be expressed in almost every nucleated cells of human body, its daily secretion is relatively constant, and it could freely penetrate the glomerular filtration membrane. Cyst C in the original urine is almost completely uptaken and decomposed by the epithelial cells of proximal convoluted tubule, while it would not enter the blood, and trace quantity of Cyst C is eliminated in urine, thus the serum Cyst C is a sensitive and specific marker in judging early renal injuries.[28–30] The results of this study showed that severe OSAHS was the independent risk factor for 24 hr UTP and Cyst C. The specific reasons which might be considered are the following: 1) OSAHS the intermittent hypoxia might stimulate the sympathetic activity to increase, thus further activating the renin-angiotensin-aldosterone system, and generating powerful vasoconstrictor effects, the effects of contracting the renal efferent glomerular arteries are greater than those on the afferent glomerular arteries, so that the intrarenal pressures are increased, and the renal glomerulus is overloaded,[35] which leads to the leakage of urinary protein and the increasing of Cyst C filtration; 2) recurrent apnea and breathlessness of OSAHS patients result in the increasing intrathoracic pressures, the heart would then be dragged, so the atrium-released atrial natriuretic peptide is increased, which relaxes the afferent glomerular arteries, and contracts the efferent glomerular arteries, resulting in a state of high filtration in the glomerulus , as well as the filtration of large proteins and Cyst C; 3) the intermittent hypoxia of patients with OSAHS during night is similar to renal ischemia reperfusion. The kidney is a very sensitive organ towards hypoxia which could inhibit renal metabolism, produce oxygen free radicals, and cause damages to cells and cel-

lular skeletons, as well as apoptosis; thus, the renal structures and functions are damaged, resulting in the increased protein leakage and Cyst C filtration, followed by the formation of proteinuria[36] and Cyst C elevation in urine. Because the OSAHS patients are commonly associated with such metabolic syndromes as obesity, hypertension, diabetes and hyperlipidemia, which could all cause or aggravate the kidney damage, it might obscure the OSAHS-caused kidney damage. Therefore, OSAHS could be deduced as the risk factor for the early renal damage independently from age, gender, BMI, blood pressure and blood sugar.

Our study has some limitations. The first limitation is that few indicators for tubular damage were included, thus the specific damaged sites caused by the association of hypertension and OSAHS could not be further confirmed. The second limitation pertains to the fact that this study was only a cross-sectional study, and a further longitudinal study was not performed. The third limitation is that the number of patients in the present study is small. More supportive evidence might be expected in future, so that the possible mechanisms of hypertension-OSAHS-related kidney damage could be further explored.

In summary, the hypertension-OSAHS patients showed more severe early kidney damage than the pure Hyp patients, among which severe OSAHS was the risk factor of 24 hr UTP; blood pressure and obesity were the risk factors of 24 hr urinary microalbuminuria; age, uncontrolled blood pressure and severe OSAHS were the risk factors of Cyst C; and severe OSAHS was the independent risk factor for early kidney damage. Therefore, in clinical practice, the hypertension-OSAHS patients should reduce their body weight much more actively, lower their blood pressure and improve OSAHS condition to control the risk factors of kidney damage, and undergo regular measurements of 24 hr UTP, 24 hr urinary microalbuminuria and Cyst C to evaluate the kidney damage degrees caused by hypertension and OSAHS, thus achieving early prevention, diagnosis and treatment.

Conflict of interest

All authors have no conflict of interest regarding this paper.

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