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Clinical Presentation of Obstructive Sleep Apnea in Patients with End-stage Renal Disease

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Study Objectives: Obstructive sleep apnea (OSA) is common in patients with end-stage renal disease (ESRD) and is largely underrecognized. Our objective was to determine whether the presentation of OSA in patients with ESRD differs from the stereotypical presentation in the general population (loud snoring, witnessed apnea, and daytime sleepiness in overweight, middle-aged men).

Methods: Seventy-six chronic dialysis patients with OSA were compared to 380 OSA patients with normal renal function who were matched for apnea severity (apnea-hypopnea index). All patients underwent overnight polysomnography and completed the Epworth Sleepiness Scale and a questionnaire to assess symptoms of OSA.

Results: Age and gender distribution were similar between groups, however, body mass index was lower in the ESRD group (28 ± 5 vs. 33 ± 14 kg/m^2). Patients with ESRD were less likely to report snoring (80% vs. 98%), witnessed apnea during sleep (32% vs. 58%), unrefreshing sleep (55% vs. 73%), and morning headaches (15% vs. 27%). Overnight polysomnography revealed less intense snoring and more sleep disturbance in patients with ESRD. The prevalence and severity of self-reported daytime sleepiness was similar between groups.

Conclusions: The presenting symptoms of patients with ESRD and documented OSA differed from a control group of OSA patients matched for AHI. This suggests that the presentation of ESRD patients with OSA may differ from the general population, and this should be appreciated to avoid underdiagnosis of this important comorbidity.

Keywords: End-stage renal disease; dialysis; obstructive sleep apnea; polysomnography; symptoms; snoring

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Sleep apnea is a common and important comorbidity in patients with end-stage renal disease (ESRD).[1] In addition to causing sleep disruption, sleep apnea has the potential to increase the risk of cardiovascular disease, which is a common complication of chronic renal failure. In patients with ESRD, hypoxemia during sleep is associated with nocturnal hypertension, left ventricular hypertrophy, impaired sympathovagal balance, and an increased risk of cardiovascular complications.[2-5] Recently, it has been suggested that sleep apnea contributes to an increased mortality risk in patients with ESRD.[6] Sleep apnea can be effectively treated in many patients with continuous positive airway pressure,[7] and consequently it is important that the condition be recognized and diagnosed appropriately.

The stereotypical presentation of obstructive sleep apnea (OSA) consists of a history of loud snoring, witnessed apnea during sleep, and daytime sleepiness in an overweight male patient.[8,9] However, the clinical features of OSA in patients with ESRD may differ from this classic presentation for several reasons. First, previous studies in patients with ESRD have found weak or no association between traditional risk factors for sleep apnea, such as age, gender, and body mass index (BMI), and the presence of OSA.[10-14] Consequently, the characteristics of ESRD patients who present with sleep apnea may differ from the general sleep apnea population. Second, although apneas and hypopneas are predominantly obstructive,[10,11,15,16] many ESRD patients with sleep apnea also have central or mixed respiratory events,[7,13,14,17-20] which may change the pattern and intensity of snoring. Third, patients with ESRD have other potential causes of daytime sleepiness[21] which may reduce the reliability of daytime sleepiness as a sign of sleep apnea. Finally, patients with ESRD frequently have other sleep disorders such as insomnia and restless legs syndrome,[22,23] which may alter their clinical presentation. Accordingly, the presentation of sleep apnea in patients with ESRD may be atypical and, consequently, underrecognized.

The objective of this study was to describe the clinical features of OSA in a large group of patients with ESRD and to determine if the presentation differed from that in the general sleep apnea population. We addressed these questions by comparing the clinical and polysomnographic features of OSA in patients with ESRD to those in OSA patients with normal renal function.

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METHODS

Patient Recruitment

Patients receiving treatment with chronic dialysis who were referred to the Sleep Laboratory at St. Michael's Hospital in Toronto for suspected sleep apnea were enrolled in the study. OSA was defined as ≥ 10 apneas and hypopneas per hour of sleep (apnea-hypopnea index [AHI]) in which > 50% of apneas and hypopneas had obstructive features. Non-apneic patients and patients with predominantly central sleep apnea (> 50% of apneas and hypopneas with central features) were excluded. Control patients with no history of kidney disease who were referred to the sleep laboratory for suspected OSA during the same time period were randomly selected from the clinical database. Control patients were matched 1:5 with ESRD patients, based on AHI. Selection and matching was performed while blinded to other polysomnographic parameters, patient demographics, and questionnaire data. Data for renal patients were obtained from participation in other clinical research studies which had been approved by the Research Ethics Board of St Michael's Hospital.

Sleep History Questionnaire

All patients completed a sleep history questionnaire, which included a history of snoring, nocturnal choking and witnessed apnea, frequent awakenings, unrefreshing sleep, excessive daytime sleepiness, morning headaches, and memory impairment (Appendix). Subjective snoring intensity was assessed by asking patients to rate the loudness of their snoring and how disruptive it was to their bed partner's sleep, using a 4-point Likert scale. In addition, patients were asked whether their snoring was supine dependent and if their snoring caused their bed partner to sleep in a separate room.

Epworth Sleepiness Scale

All patients completed the Epworth Sleepiness Scale (ESS).[24] The ESS is a self-administered questionnaire designed to measure the general level of daytime sleepiness. Patients rate on a scale of 0-3 how likely they are to fall asleep in 8 different situations commonly encountered in daily life. Total ESS score ranges from 0-24; higher scores indicate more subjective sleepiness.[24] All questionnaires were completed on the evening of polysomnography.

Polysomnography

All patients underwent diagnostic overnight polysomnography, which was performed and scored by registered polysomnographic technologists in a standardized fashion.[25] Recordings were performed by continuous monitoring of the electroencephalogram, electrooculogram, and submental electromyogram, electrocardiogram, nasal pressure (Ultima Dual Airflow Pressure Transducer, Braebon Medical Corporation, Kanata, ON), chest and abdominal respiratory movements (Respitrace, Ambulatory Monitoring; Ardsley, NY), oximetry (Mallinckrodt/Nellcor Puritan Bennett, Hazelwood, MO), anterior tibialis electromyogram, body position, and sound intensity (CEL-231 Digital Sound Survey Meter, CEL Instruments Bedford, England).

Total recording time was defined as the time from lights out in the evening to lights on the following morning. Total sleep time (TST) was defined as the duration of sleep from the beginning to the end of the polysomnographic recording. Sleep efficiency was defined as TST expressed as a proportion of the total recording time. Sleep was scored as NREM and REM sleep. NREM sleep was further scored as stage 1, 2, and slow wave sleep. Sleep onset latency was defined as the time from lights out to the first epoch of sleep. Wakefulness after sleep onset was the total amount of time awake from sleep onset to the end of the polysomnographic recording. Apnea was defined as absence of airflow > 10 sec, and hypopnea was defined as any reduction in airflow for ≥ 10 sec associated with an arousal and/or reduction in oxygen saturation > 3%. Apneas and hypopneas were further classified as central if abdominal and ribcage movements were synchronous, obstructive if the movements were paradoxical, and mixed if a central event had terminal obstructive features. Periodic limb movements (PLM) were defined as ≥ 4 consecutive leg movements, reflected by activation of the anterior tibialis electromyogram, lasting 0.5 to 5 sec, with 5 to 90 sec between each movement. Arousal was defined as a sudden increase in electroencephalogram frequency to ≥ α, accompanied by electromyogram activation and eye movements lasting 3 to 15 sec. Awakening was scored if these changes persisted > 15 sec. An arousal was further categorized as a respiratory arousal if it occurred within 3 sec of an apnea or hypopnea and as a PLM arousal if it occurred within 3 sec of a limb movement. The maximum decibel level recorded on the sound meter during each 30-sec epoch of the polysomnogram was identified; the mean value of this measurement (mean maximum decibel level) during different sleep states and body positions was used to determine snoring intensity. We used the mean maximum decibel level to classify snoring as mild (40-50 dB), moderate (50-60 dB), or severe (> 60 dB). This classification was based on a validation study (unpublished) in a separate group of patients, in which we correlated the sleep laboratory technologist's subjective assessment of snoring intensity with the recorded decibel level.

Analysis

Mean data and standard deviations were analyzed using independent samples *t*-test. Nonparametric data were analyzed using Mann-Whitney U test and χ^2 analysis. All statistical analysis was performed using computer software (SPSS 14.0, SPSS Inc., Chicago, IL). All p values < 0.05 were considered statistically significant.

RESULTS

Patient Demographics

One hundred thirty-four ESRD patients were reviewed. Eighty-one of these patients met our criteria for a diagnosis of sleep apnea (AHI > 10). Five patients with > 50% central apneas and hypopneas were excluded. The remaining 76 ESRD patients with OSA and 380 controls (OSA with normal renal function) matched for AHI were enrolled in the study. Sixty-five (86%) ESRD patients were receiving treatment with hemodialysis, and 11 (14%) were on peritoneal dialysis. Age and gender dis-

Table 1—Patient Demographics

	ESRD	Control	p value
Patients, #	76	380	
Age, years	53 ± 13	51 ± 13	NS
Gender, % male	73	71	NS
BMI, kg/m^2	28.1 ± 5.3	33.0 ± 13.8	0.003
Neck circumference, cm	39.5 ± 4.1	41.1 ± 4.4	0.006
Neck: Height	0.23 ± 0.02	0.24 ± 0.03	0.039

BMI = body mass index; ESRD = end-stage renal disease; Neck: Height = neck circumference indexed to height. Data are mean ± standard deviation.

Table 2—Medications

	ESRD	Control	p value
Cardiac			
ACE inhibitors	30 (40)	63 (17)	< 0.001
β blockers	36 (47)	47 (12)	< 0.001
Angiotensin II receptor blockers	20 (26)	28 (7)	< 0.001
Calcium channel blockers	31 (41)	38 (10)	< 0.001
CNS			
Benzodiazepines	7 (9)	16 (4)	NS
Zopiclone	3 (4)	2 (1)	0.009
Other sedatives	2 (3)	0 (0)	0.002
Tricyclic antidepressants	4 (5)	8 (2)	NS
SSRI	2 (3)	44 (12)	0.018
Other antidepressants	0 (0)	7 (2)	NS

ESRD = end-stage renal disease; ACE = angiotensin-converting enzyme; CNS = central nervous system; SSRI = selective serotonin reuptake inhibitor. Data are number (percentage) of patients within group.

tribution were similar between ESRD and control groups (Table 1). BMI and neck circumference were significantly greater in the control group. A higher proportion of patients in the ESRD group were taking cardiac medications (Table 2). The use of central nervous system (CNS) medications was quite low in both groups; the main difference was the higher prevalence of selective serotonin reuptake inhibitors (SSRIs) in patients with normal renal function.

Sleep History

Snoring, witnessed apnea, unrefreshing sleep, and morning headaches were reported less often by patients with ESRD than control patients (Table 3). The proportion of patients who reported nocturnal choking, restless sleep, memory impairment, and daytime sleepiness was not significantly different between groups.

Polysomnography

TST and sleep efficiency were reduced in patients with ESRD compared to controls (Table 4). Patients with ESRD had more wakefulness after sleep onset and a greater proportion of stage 2 NREM sleep. The proportion of stage 1 NREM, slow wave, and REM sleep were similar between groups. Patients with ESRD had a higher frequency of PLM and PLM-related arousals. The total number of arousals and awakenings per hour of sleep was elevated[26] but was not significantly different between groups. These findings indicate that ESRD patients with OSA suffer greater sleep loss and sleep disruption than patients with normal renal function.

Table 3—Sleep History Questionnaire

	ESRD	Control	p value
History of snoring, %	80	98	< 0.001
Witnessed apnea, %	32	58	< 0.001
Nocturnal choking sensation, %	34	36	NS
Daytime sleepiness, %	47	44	NS
Restless sleep, %	45	49	NS
Unrefreshing sleep, %	55	73	0.002
Morning headaches, %	15	27	0.022
Memory impairment, %	51	49	NS

ESRD = end-stage renal disease. Data are percentage within each group.

By definition, total AHI was similar between ESRD and control groups, as was the frequency of respiratory-related arousals. Mean oxygen saturation (SpO_2) and the percentage of TST with SpO_2 < 90% were not significantly different between groups. Although respiratory events were predominantly obstructive, as determined by enrollment criteria, ESRD patients had a relatively greater number of central and mixed apneas and hypopneas and a smaller number of obstructive events (Table 5). There was no difference between group in the frequency of respiratory events indexed to the supine and lateral positions.

Snoring Intensity

Patients with ESRD reported snoring less frequently than did controls, and snorers rated their snoring as being quieter and less disruptive to their bed partner than did control patients. A greater percentage of patients with ESRD indicated that they snored only in the supine position. Mean maximum sound intensity was significantly lower in patients with ESRD than control patients in all sleep states and body positions (Table 6); there was a greater percentage of mild snorers and a smaller percentage of severe snorers in the ESRD group than the control group. All of these subjective and objective indices of snoring indicate that it was a less prominent feature of OSA in patients with ESRD.

Daytime Sleepiness

Each component of the ESS and the total ESS score were not significantly different between ESRD and control groups (Table 7), and the proportion of patients with total ESS score indicating excessive daytime sleepiness (> 10)[24] was similar between groups (ESRD vs. control, 28% vs. 34%).

DISCUSSION

Previous studies have reported a high prevalence of OSA in patients with ESRD.[10,11,13-20,27-29] Many of these studies have relied on research protocols with objective nocturnal monitoring to diagnose OSA regardless of the patients' sleep related symptoms.[10,14-20,28] However, in clinical practice, the diagnosis of OSA is usually prompted by the clinical presentation, which typically consists of loud snoring, witnessed apnea during sleep, and daytime sleepiness in overweight men.[8,9] To date, we do not

Table 4—Polysomnography

	ESRD	Control	p value
Total recording time, h	7.5 ± 0.9	7.8 ± 0.7	0.005
Total sleep time (TST), h	4.9 ± 1.6	5.6 ± 1.2	0.001
Sleep efficiency, %	71.4 ± 20.4	77.6 ± 15.3	0.015
Sleep onset latency, min	17.1 ± 25.8	20.6 ± 26.8	NS
Wake after sleep onset, min	98.5 ± 70.6	79.6 ± 58.3	0.03
Stage 1, %TST	17.1 ± 18.1	14.4 ± 12.5	NS
Stage 2, %TST	52.2 ± 15.8	56.8 ± 13.1	0.015
Slow wave, %TST	15.0 ± 10.7	13.6 ± 10.7	NS
REM, %TST	15.9 ± 8.8	15.2 ± 7.3	NS
AHI	44.2 ± 27.7	44.2 ± 27.6	NS
Mean SpO_2, %	94.0 ± 2.6	93.7 ± 2.9	NS
SpO_2 < 90%, %TST	8.9 ± 15.8	10.1 ± 20.0	NS
PLM, /h	25.2 ± 35.3	11.5 ± 21.5	0.002
PLM arousals, /h	8.0 ± 15.1	3.0 ± 8.2	0.006
Respiratory arousals, /h	39.6 ± 28.5	40.5 ± 27.2	NS
Spontaneous arousals, /h	6.2 ± 6.1	4.8 ± 4.2	NS
Total arousals, /h	53.8 ± 29.8	48.3 ± 25.8	NS
Awakenings, /h	8.6 ± 8.6	6.6 ± 6.1	NS

AHI = apnea-hypopnea index; ESRD = end-stage renal disease; PLM = periodic limb movements; SpO_2 = oxyhemoglobin saturation; TST = total sleep time. Data are mean ± standard deviation.

Table 5—Respiratory Events (Apneas and Hypopneas) during Polysomnography

	ESRD	Control	p value
Obstructive			
Events (/h)	32.8 ± 22.9	41.3 ± 26.4	0.005
Events occurring in REM (/h)	35.2 ± 29.0	42.1 ± 25.6	0.035
Events occurring in NREM (/h)	31.6 ± 24.4	40.3 ± 28.5	0.007
% of total events	81 ± 27	95 ± 13	0.002
Central			
Events (/h)	4.9 ± 8.5	1.4 ± 4.1	0.001
Events occurring in REM (/h)	1.6 ± 5.0	0.8 ± 3.2	NS
Events occurring in NREM (/h)	5.3 ± 9.2	1.4 ± 4.4	0.001
% of total events	9.4 ± 12.9	3.0 ± 7.5	0.002
Mixed			
Events (/h)	5.4 ± 12.3	1.6 ± 6.4	0.01
Events occurring in REM (/h)	5.1 ± 13.3	1.0 ± 5.0	0.01
Events occurring in NREM (/h)	5.4 ± 12.7	1.7 ± 6.7	0.014
% of total events	8.2 ± 16.5	2.5 ± 8.4	0.002
Total			
Events (/h)	44.2 ± 27.7	44.2 ± 27.6	NS
Events occurring in REM (/h)	43.2 ± 29.7	43.9 ± 26.0	NS
Events occurring in NREM (/h)	43.4 ± 29.6	43.4 ± 29.9	NS
Events while supine (/h)	61.1 ± 33.8	57.9 ± 36.9	NS
Events while non-supine (/h)	34.5 ± 31.4	35.1 ± 30.3	NS

ESRD = end-stage renal disease. Data are mean ± standard deviation.

know if ESRD patients with OSA conform to this stereotypical presentation. This is the first study to systematically evaluate the presentation of OSA in ESRD patients by comparison to OSA patients with normal renal function. We found that the clinical features of OSA in patients with ESRD differ from the general sleep apnea population. It is important that these differences are appreciated so as to avoid underrecognition and undertreatment of OSA in this patient population, particularly as it can increase both sleep related symptoms and cardiovascular morbidity and mortality.[2-6,8]

Advancing age, male gender, and excessive weight are established risk factors for the development of OSA in the general population.[30] Some investigators have described similar findings in patients with ESRD,[15,18,31] while others have reported these associations are weak in this patient population.[10-14] Both our ESRD and control groups were predominantly male, and both age and gender distribution were similar between groups, suggesting the mechanisms by which advancing age and male gender predispose to the development of OSA in the general population are similar in patients with ESRD. However, our ESRD patients were generally not obese; BMI was significantly lower than control patients. This is consistent with previous reports, which have described a weak association between BMI and the prevalence and severity of OSA in patients with ESRD.[10,12-14,32] These findings support the notions that obesity is not required for ESRD patients to develop OSA and that the pathogenesis of OSA in this patient population

Table 6—Snoring Intensity

	ESRD	Control	p value
Subjective (Sleep History Questionnaire)			
Present, %	80	98	< 0.001
Intensity rating	2.6 ± 1.1	3.1 ± 0.9	< 0.001
Disturbance of bed partner	2.3 ± 1.0	3.0 ± 1.0	< 0.001
Partner moved to separate bedroom, %	25	38	NS
Supine dependent, %	60	36	0.001
Objective (Polysomnography)			
Mean maximum intensity, dB			
Total sleep time	49.0 ± 5.2	55.0 ± 6.7	< 0.001
NREM	49.1 ± 5.3	55.4 ± 6.9	< 0.001
REM	48.8 ± 5.9	53.3 ± 6.4	< 0.001
Non-supine	47.9 ± 5.2	53.9 ± 7.2	< 0.001
Supine	51.0 ± 5.5	56.1 ± 6.7	< 0.001
Classification, %			
Mild (40-50 dB)	58	21	< 0.001
Moderate (50-60 dB)	37	52	NS
Severe (> 60 dB)	5	27	0.002

ESRD = end-stage renal disease. Data are mean ± standard deviation unless otherwise indicated.

Table 7—Epworth Sleepiness Scale

	ESRD	Control	p value
Sitting and reading	1.3 ± 1.1	1.5 ± 1.1	NS
Watching television	1.7 ± 1.0	1.7 ± 1.0	NS
Sitting in public	0.7 ± 0.9	0.9 ± 1.0	NS
Passenger in car	0.9 ± 1.0	1.2 ± 1.1	NS
Lying down in afternoon	2.2 ± 1.0	2.1 ± 1.0	NS
Talking to someone	0.2 ± 0.6	0.2 ± 0.5	NS
Sitting after lunch	1.2 ± 1.0	1.0 ± 1.0	NS
Stopped in traffic	0.1 ± 0.4	0.2 ± 0.6	NS
Total score	8.2 ± 4.6	8.8 ± 4.9	NS

ESRD = end-stage renal disease. Data are mean ± standard deviation.

is related to factors unique to ESRD, such as pharyngeal narrowing associated with fluid overload.[33]

Although a higher proportion of patients in the ESRD group were taking cardiac medications (Table 2), this is not surprising in view of the greater prevalence of hypertension and cardiac disease in this patient population. However, we do not believe that a higher prevalence of cardiac medications in patients with ESRD accounts for the differences we found in the clinical presentation of obstructive sleep apnea. The use of CNS medications was relatively low in both groups. The main difference was the higher prevalence of SSRIs in patients with normal renal function. Once again, we do not think that these differences contributed significantly to our findings.

Patients with ESRD reported fewer of the typical symptoms associated with OSA, namely loud snoring, witnessed apnea, and morning headaches. Those who reported a history of snoring described their snoring as less intense and less disruptive to their bed-partner's sleep than patients with normal renal function. These findings were confirmed by objective measurement of snoring intensity during polysomnography, which indicated quantitatively and categorically less intense snoring (Table 6).

Previous investigators have suggested that snoring alone may be an unreliable indicator of OSA in ESRD patients.[32,34] Our data extend these findings by demonstrating that snoring is less common and less severe in patients with ESRD *who are known to have OSA*, and that other symptoms of OSA, such as witnessed apnea and morning headaches, may also be less reliable diagnostic criteria. One possible explanation for these findings is the smaller number of obstructive respiratory events and greater number of central events in ESRD patients. Central sleep apnea can be associated with less intense snoring,[35,36] and thus an increased frequency of central events might contribute to the lower prevalence and intensity of snoring in patients with ESRD. The potential mechanisms responsible for the development of central sleep apnea in patients with ESRD include stimulation from pulmonary mechanoreceptors associated with pulmonary edema and enhanced ventilatory sensitivity to hypercapnia,[10] both of which could destabilize the central control of breathing.

Overnight polysomnography revealed markedly disturbed sleep in both ESRD and control groups, reflected by the high frequency of arousals and awakenings, poor sleep efficiency, and reduced proportion of slow wave sleep and REM sleep. However, sleep efficiency was significantly worse in patients with ESRD, which may be attributed in part to the higher frequency of PLM and PLM-related arousals.[8] Reduced sleep efficiency may also be related to the high prevalence of restless legs syndrome and primary insomnia in ESRD patients.[22,23] These findings highlight the fact that patients with ESRD often have multiple sleep disorders, which may complicate the presentation and interpretation of their sleep complaints.

Although polysomnographic recordings indicated poorer sleep in ESRD patients, they reported a similar degree of daytime sleepiness as OSA patients with normal renal function. Daytime sleepiness is common in patients with ESRD, and its etiology is multifactorial.[21] Although some investigators have described an association between sleep apnea and excessive daytime sleepiness in patients with ESRD,[14,21] this association is relatively weak,[21] and other factors such as comorbid disease, medication, and uremia itself are likely to contribute. Excessive daytime sleepiness has also been linked to PLM in patients with ESRD.[37] Given these considerations, we anticipated that subjective daytime sleepiness, reflected by ESS scores, would be more severe in patients with ESRD. There are a number of possible explanations for this discrepancy. First, daytime dialysis sessions afford patients with ESRD more opportunity to nap during the day thereby alleviating sleepiness. Second, central sleep apnea is not strongly associated with daytime sleepiness[35,36]; the relatively higher proportion of central respiratory events in ESRD patients may have contributed to less daytime sleepiness than anticipated. Third, patient perception of daytime sleepiness may be altered by uremia, and patients may confuse sleepiness with fatigue or attribute it to the general symptomatology of their ESRD. This possibility is supported by the poor association between objective and subjective measurements of daytime sleepiness in this patient population.[21,37] Regardless of the explanation, the cumulative evidence indicates that daytime sleepiness is not a reliable diagnostic criterion for OSA in patients with ESRD, and that the absence of daytime sleepiness should not dissuade the clinician from considering a diagnosis of OSA in this patient population.

The study has some limitations, which should be addressed. First, we studied patients who were referred for suspected sleep apnea, which raises the possibility of referral bias. Despite the clinical suspicion of sleep apnea by the referring physician, we found significant differences in the presentation of OSA between ESRD patients and those with normal renal function. In fact, a referral bias may have lead to an underestimation of these differences, which could have been greater if all patients from a dialysis clinic were included. Second, since our patients were selected from a sleep clinic population, it is unclear how representative these results are of the larger dialysis and community based populations. Third, we did not study ESRD patients *without* suspected OSA; therefore the extent to which OSA is underdiagnosed in patients with ESRD because of differences in clinical presentation cannot be addressed. Future research is required to determine predictors of OSA in patients with ESRD and to develop and validate appropriate screening tools for this patient population.

In conclusion, the clinical presentation of OSA in patients with ESRD is different from the general sleep apnea population. Many of the stereotypical symptoms of OSA are less common, which may lead to underdiagnosis of this important comorbidity. Increased awareness of the atypical presentation of OSA in ESRD patients may address this concern.

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Appendix—Sleep History Questionnaire (modified to include only questions regarding symptoms of obstructive sleep apnea listed in Table 3)

PATIENT HISTORY QUESTIONNAIRE

NAME: _____ DATE:_____

1.	Do you snore?	□ Yes □ No
2.	How loud would you, or your bed partner, rate your snoring? (please circle, 1=mild, 2=moderate, 3=loud, 4=very loud)	1 2 3 4
3.	How disturbed is your bedpartner by your snoring? (please circle, 1=not at all, 2=mildly, 3=moderately, 4=severely)	1 2 3 4
4.	Does your snoring cause him or her to sleep in a separate room?	□ Yes □ No
5.	Do you only snore if you are lying on your back?	□ Yes □ No
6.	Has anyone ever told you that you stop breathing while you sleep?	□ Yes □ No
7.	Do you ever wake up with the feeling that you are choking?	□ Yes □ No
8.	Have you been told that you are a restless sleeper?	□ Yes □ No
9.	Do you feel refreshed when you wake up?	□ Yes □ No
10.	Do you often wake up with headaches in the morning?	□ Yes □ No
11.	Do you have trouble concentrating or remembering things?	□ Yes □ No