Study of Sleep Disorders in Resistant Hypertensive Patients on Conventional Hemodialysis

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Abstract: Background: Every year more people suffer from end stage renal disease (ESRD). Sleep disturbances have been reported to be frequent in dialysis patients. Sleep disturbances could be sleep apnea, periodic limb movements in sleep or restless leg syndrome. **Patients and methods:** Sixty patients were chosen from Ain Shams University Hospitals dialysis units, between July 2010 and March 2012. Patients were divided into 6 groups: **Group A:** 10 patients with resistant HTN and ESRD on conventional HD having a PSQI score > 5. **Group B:** 10 patients with resistant, HTN and ESRD on conventional HD having a PSQI score < 5. **Group C:** 10 patients with ESRD only on conventional HD having a PSQI score > 5. **Group D:** 10 patients having ESRD only on conventional HD having a PSQI score < 5. **Group E:** 10 patients having resistant HTN normal renal function. They had PSQI score > 5. **Group F:** 10 patients having resistant HTN and normal renal function. They had PSQI score < 5.For all groups, creatinine, BUN, Hb, albumin, Na, K, PO_4, Ca and uric acid were done. Polysomnography was done for bad sleeper groups (group A, group C and group E). **Results:** Creatinine and BUN were significantly higher in bad sleeper groups than good sleeper groups (group A than group B and group C than group D). Nearly all polysomnographic parameters were abnormal in group A, group C and group E. In group A and group C, creatinine was positively correlated to parameters of obstructive sleep apnes OSA and periodic limb movement PLM ($P < 0.05$). In group A and group E, SBP and DBP were positively correlated to parameters of OSA and PLM. **Conclusion:** ESRD induces sleep disorders exacerbated by resistant HTN. Also resistant HTN alone can induce sleep disorders.

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1. Introduction

Sleep plays an important role in workers lives, allowing them to relax, restore, and revitalize their bodies, minds and emotions every 24 hours ***(Ohlman* and O'Sullivan ., *2009)***

Sleep is more than the absence of being awake; it is a homeostatically regulated process ***(Kotronoulas et al., 2009).***

The average sleep duration of adults is approximately 7 hours. National sleep foundation found the average sleep duration on work days in 44% of people to be shorter than this. Different studies indicate that too short a sleep duration is associated with a number of negative health outcomes, including higher risk for hypertension and cardiovascular disease ***(Portaluppi et al., 2009).***

Patients with common medical disorders often complain to their physician about sleep problems, and these patients are often referred to sleep specialists for evaluation and diagnosis. If the quality of sleep is improved, subjective symptoms related to the disease may improve ***(Parish, 2009).***

Obstructive sleep apnea (OSA), is the most common form of sleep disordered breathing ***(Hoffman et al., 2004)***

High blood pressure and obstructive sleep apnea are closely related, and the latter is considered to induce hypertension ***(Sharabi et al., 2004).***

Resistant hypertension is a common medical problem. Secondary causes of hypertension, such as obstructive sleep apnea require investigations and effective treatment if present ***(Pisoni et al., 2009).***

Hypertension is a well-known cause of renal impairment, and impaired renal function is a well known cause of hypertension; therefore the two conditions constitute a vicious circle resulting in the progressive worsening of each. This relationship is very prominent in end-stage renal disease ***(Portaluppi et al., 2009).***

Patients with end stage renal disease (ESRD) have a considerable symptom burden, among which sleep related symptoms are highly prevalent. Sleep disorders, such as restless legs, periodic limb movements and sleep apnea, and sleep complaints such as insomnia and day time sleepiness are very common in ESRD patients despite treatment with 3 times a week conventional hemodialysis ***(Hanly, 2009).***

Aim Of The Work

To assess the pattern of sleep disorders in resistant hypertensive patients on regular hemodialysis.

2. Patients and Methods:

Our study was conducted in Ain Shams University Hospital in Cairo between July 2010 and March 2012. Our study included 60 patients divided into 6 groups:

Group A: 10 patients with resistant hypertension and chronic renal failure on regular HD 3 times a week. Their pittsburg sleep quality Index (PSQI) score was > 5, known as (Bad sleepers). They were subjected to do polysomnogram study to assess their sleep pattern.

Group B: 10 patients with resistant hypertension and chronic renal failure on regular hemodialysis 3 times a week. Their PSQI score was < 5 and known as (good sleepers). They didn't do polysomnogram study as they didn't have sleep problems according to PSQI.

Group C: 10 patients with normal blood pressure and chronic renal failure on regular 3 times a week hemodialysis. Their PSQI score was > 5 and known as (Bad sleepers). They were subjected to do polysomnogram study to assess their sleep pattern.

Group D: 10 patients with normal blood pressure and chronic renal failure eon regular HD 3 times a week. Their PSQI score was < 5 and known as (good sleepers). They didn't do polysomnogram study as they didn't have sleep problems according to PSQI.

Group E: 10 patients with resistant hypertension and normal kidney function. Their PSQI score was > 5 known as (bad sleepers). They were subjected to do polysomnogram study to assess their sleep pattern.

Group F: 10 patients with resistant hypertension and normal kidney function. Their PSQI score was < 5 and known as (good sleepers). They didn't do polysomnogram study as they don't have sleep problems according to PSQI.

We excluded from the study patients with frank psychiatric problems, patients who are taking drugs that affect sleep quality like (antihistaminics or B_2 agonists), and antihypertensive drugs that are centrally acting like (α-methyldopa or lipid soluble beta-blockers).

We also excluded patients with renal failure secondary to other systemic disease like (SLE or DM), patients with multi-advanced chronic organ failure. Smokers and diabetic patients were also excluded.

All patients included in the study were subjected to complete clinical examination with calculation of body mass index (BMI), serum creatinine and BUN, Hb, Na and K according to conventional methods in Ain Shams University hospitals, Ca, Ph and serum albumin, body mass index (BMI; weight/height squared) was calculated according to the World Health Organization classification ***(Executive Summary, 1998).***

Assessment of sleep quality using the Pittsburgh sleep quality index (PSQI), which is a score derived by a self rated questionnaire, consisting of 19 questions which assess a wide variety of factors related to sleep quality in the previous month. The 19 questions are grouped into seven components scores, each weighted equally on 0-3 scale. The seven components are summed to yield a global PSQI score (range: 0-21) higher scores indicate worse sleep quality ***(Buysse et al., 1989)***. The seven components included: subjected sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medications, and daytime dysfunction.

Patients with global PSQI score > 5 are conventionally defined as" poor sleepers", whereas those with a score < 5 are considered "good sleepers". Patients with poor sleep quality will be subjected to assessment of their sleep pattern using polysomnography in the sleep lab. Of neuropsychiatry department, for assessment of the etiology of the sleep disorder.

The polysomnography recording started at the patient's usual bedtime and was carried out using a polysmith neurotronics Inc. system, with 14 channels disturbed as follows: 3 for electroencepha-logrpay, 2 for oculogram, 1 for chin electromyography, 1 for tibial electromyography, 1 for electrocardiography, 1 for airflow, 2 for thoracic-abdominal movements, 1 for detection of body position . The system automatically analyses sleep stages using the ***Rechtshaffen and Kales (1968)*** criteria and arousals according to scoring rules of *Sleep Dis*orders *Atlas Task Force of the* ***American Sleep Disorders Association (1992)***

Polysomnography also known as a sleep study, a multiparametric test used in the study of sleep and as a diagnostic tool in sleep medicine. The test result is called a polysomnogram, also abbreviated PSG ***(Rechtschaffen and Kales, 1968).***

The patients underwent HD the day before the polysomnographic evaluation. Before the polysomnographic evaluation, clinical examination was made and weight (in kilograms), height (in meter), and the average of three systolic blood pressure (SBP, in mm Hg) measurements and three diastolic blood pressure (DBP, in mmHg) measurements were recorded, according to the recommendations of the VI report of the Joint National Committee on Prevention, Detection, Evaluation and treatment of high blood pressure ***(Joint National Committee, 1997).***

Terms of sleep study:

Apnea: was defined as the complete cessation of respiration or a > 50% reduction in airflow lasting longer than 10 seconds accompanied by persistent respiratory effort, with or without oxygen desaturation or arousal ***(Torre-Bouscoulet et al., 2007).***

Hypopnea: Defined as a reduction in airflow of < 50% for longer than 10 seconds, detected by thermistor or nasal cannula, accompanied by oxygen desaturation of > 4% or an arousal of 1.5 seconds or longer ***(Torre-Bouscoulet et al., 2007).***

Apnea-Hypopnea index (AMI)

Is an index used to assess the severity of sleep apnea based on the total number of complete cessations (apnea) and partial obstructions (hypopnea) of breathing occurring per hour of sleep. These pauses in breathing must last for 10 seconds and are associated with a decrease in oxygenation of the blood. AHI can be used to classify the severity of disease in events/ hour (mild 5-15, moderate 15-30, and severe greater than 30 ***(Peters, 2011).***

Oxygen saturation:

Normal oxygen saturation (SaO_2) is > 94% "oxygen saturation: oxygen content of blood divided by oxygen capacity and expressed in volume percent" ***(American Sleep Apnea Association, 2012).***

Desaturation Index

Another scoring criteria used is the oxygen desaturation index which is defined as the number of total oxygen desaturations of 3% or more divided by the total sleep time ***(Torre-Bouscoulet et al., 2007).***

Arousal

An abrupt change from a "deeper" stage of non-REM (NREM) sleep to a "lighter" stage, or from REM sleep towards wakefulness, with the possibility of awakening as the final outcome. Arousal may be accompanied by increased tonic electromyographic activity and heart rate, as well as by an increased number of body movements.

Spontaneous arousal index

The number of spontaneous arousals (eg arousals not related to respiratory events, limb movements, snoring, etc) multiplied by the number of hours of sleep. An arousal is a wake or "alpha" pattern for 3 to 15 seconds ***(American Sleep Apnea Association, 2012).***

Total wake episodes

It means total number of patient's wake times or episodes during the whole night sleep ***(American Sleep Apnea Association, 2012).***

Snoring episodes number

It means total number of patient's snoring times or episodes during the whole night sleep ***(American Sleep Apnea Association, 2012)***.

Periodic limb movement episodes number

Periodic limb movements defined as sets of muscle contractions occurring at intervals of < 90 seconds during the night, may cuase sleep disturbance or be associated with other sleep disorders such as apnea ***(Kataria and Vaughn, 2010).*** Here we mean how many times it occurs during whole night sleep latency.

Sleep latency :Means the duration of time from lights out or bedtime, to the onset of sleep.

Normal sleep latency is about 15 minutes, REM latency is 90 minutes, so these are OK ***(American Sleep Apnea Association, 2012).***

REM latency

After a person falls a sleep, the amount of time it takes for the first onset of REM sleep. Normal REM latency is 90 minutes ***(Gonzalez-Mayo and Shaner, 2005).***

Sleep efficiency

Sleep efficiency (or sleep efficiency index): the proportion of sleep in the episode potentially filled by sleep (i.e., the ratio of total sleep time to time in bed) or sleep efficiency = total sleep time multiplied by time in bed. Normal sleep efficiency is at least 85% (a sleep 85% of the night) ***(American Sleep Apnea Association, 2012).***

In this study, we have used the following abbreviations: BMI: body mass index, A/H index: Apnea/hypopnea index, Des. Index, desaturation index, TWE = total waking episode, PLM = periodic limb movement, TTPLM: Total time periodic limb movement. The normal values used for analyses of sleep variables were those proposed by ***Carskadon and Dement (2000)***.

The respiratory events (apnea and hypopnea) were classified according to the ***American Academy of Sleep Medicine Task Force Criteria (1999)***.

Resistant HTN is defined as blood pressure that remains alone (140/90) inspite of concurrent use of three anti-hypertensive agents of different classes, one of which being a diuretic, all agents should be prescribed at optimal doses ***(Demede et al., 2011).***

Statistical methods:

P-value

* P-value was considered significant if < 0.05 (S).
* P-value was borderline significance if < 0.1 (BS).
* P-value was highly significant if < 0.01 (HS).
* P-value was considered non significant if > 0.1 (NS).

Statistical package of social science (SPSS) version 15.0 was used for analysis of data.

Data was summarized as mean and standard deviation, t-test was used for analysis of two quantitative data and non parametric test (Mann-Whitney U) was used when data was not symmetrically distributed. Also Pearson linear correlation test was used in study of SBP, DBP and creatinine to different laboratory and polysomnogram items.

We also used chi-square test for multiple variables comparison.

3. Results

The percentage of good sleepers among our 60 patients was 50% and bad sleepers were 50%.

Among the studied 6 groups (group A, B, C, D, E and F), we didn't find a statistical significant difference as regards age and gender (P > 0.05). Group A constituted of 8 males (80% and 2 females (20%).

Group B constituted of males (100%) and no females, group C comprised 7 males (70%) and 3 females (30%). Group D comprised 10 males (100%) and no females. Group E constituted of 8 males (80%) and 2 females (20%). Group F constituted of 10 males (100%) and no females.

Table (1): Comparison between group A and B as regards different variables:

Variables	*Group A N = 10*	*Group B N = 10*	*t**	p
SBP(mmHg)	139±7	140±6	0.8	>0.05
DBP (mmHg)	103±8	98±5	1.9	>0.05
Creatinine (mg/dl)	11.1±0.6	9.7±1.2	3	<0.001
BUN (mg/dl)	91±26	88±16	0.9	>0.05
HB (gm/dl)	8.1±0.6	8±0.7	0.7	>0.05
Albumin (gm/dl)	3.8±0.9	3.9±0.8	1.2	>0.05
Na (mEq/L)	140±2.4	139±3	1.7	>0.05
K (mEq/L)	5.4±0.9	5.2±0.7	2.2	<0.05
Po4 (mg/dl)	96.5±0.2	6.9±0.3	1.2	>0.05
Ca (mg/dl)	7.5±0.9	8±0.7	0.9	>0.05
Uric acid (mg/dl)	6.5±1.2	6.1±1.2	1.1	>0.05

*unpaired t-test

Serum creatinine was significantly higher in bad sleeper patients with hypertension and ESRD (group A).

Table (2): Comparison between group A and B as regards BMI and questionnaire

Variables	Group A N = 10	Group B N = 10	t*	p
BMI	36.8±3	32±5	0.8	>0.05
PSQI	14.9±2	3.9±1.1	21	<0.001

* Unpaired t-test

There was a significant difference between group A and B as regards PSQI.

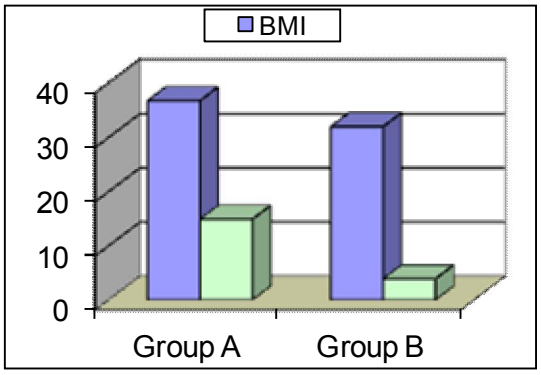

Figure (1): The diagram showing that patient within group A have BMI and PSQI score higher than group B.

Table (3): Comparison between group C and D as regards different variables:

Variables	Group C N = 10	Group D N = 10	T+	p
SBP(mmHg)	109±11	109±6	0.16	>0.05
DBP (mmHg)	69±5	97±6	1.5	>0.05
Creatinine (mg/dl)	9.9±1.3	9.9±5	0.12*	>0.05
BUN (mg/dl)	95±13	75±12	3.5	<0.001
HB (gm/dl)	8.2±0.4	8.1±5	0.2*	>0.05
Albumin (gm/dl)	3.9±0.2	3.9±0.5	0.4	>0.05
Na (mEq/L)	140±4	137±3	0.6	>0.05
K (mEq/L)	5.3±0.09	5.1±0.07	0.9	>0.05
Po4 (mg/dl)	6.5±0.6	6.1±0.5	1.1	>0.05
Ca (mg/dl)	7.5±0.3	7.5±0.2	0.03	>0.05
Uric acid (mg/dl)	4.7±0.8	6.2±1.5	1.6	>0.05

+ unpaired t-test * Mann Whitney test

Bad sleeper patients with normal blood pressure and ESRD (group C) had higher BUN than good sleeper patients with normal blood pressure and ESRD (group D).

Table (4): Comparison between group C and D as regards BMI and questionnaire

Variables	Group C N = 10	Group D N = 10	t*	p
BMI	28±3	30±3	1.2	>0.05
PSQI	13.3±1.2	2.6±0.7	23	<0.001

* Unpaired t-test

Group C had higher PSQI than group D.

Table (5): Comparison between group E and F as regards different variables:

Variables	Group E N = 10	Group F N = 10	t+	p
SBP(mmHg)	139±7	135±5	1.1	>0.05
DBP (mmHg)	98±6	95.5±5	0.5	>0.05
Creatinine (mg/dl)	0.99±0.19	0.92±0.19	0.10	>0.05
BUN (mg/dl)	11.6±1.6	13±1.1	1.3	>0.05
HB (gm/dl)	10.5±0.9	11±1.5	1.4	>0.05
Albumin (gm/dl)	4±0.5	4.2±0.18	0.9	>0.05
Na (mEq/L)	139±1.6	139.5±1.7	0.03	>0.05
K (mEq/L)	4.6±1	4.6±0.5	0.05	>0.05
Po4 (mg/dl)	3.8±0.5	3.9±0.4	0.6	>0.05
Ca (mg/dl)	8.7±0.9	9.5±0.4	1.4	>0.05
Uric acid (mg/dl)	5.9±1.3	6±0.99	1.1	>0.05

* unpaired t-test

Bad sleeper patients with normal kidney function and hypertension (group E) didn't have any significant difference as regards SBP, DBP and laboratory parameters when compared to good sleeper patients with normal kidney.

Table (6): Comparison between group E and F as regards BMI and questionnaire

Variables	Group C N = 10	Group D N = 10	t*	p
BMI	35.5±14	32.4	1.1	>0.05
PSQI	12.7±1.7	2.6±0.84	15	<0.001

* Unpaired t-test

Group E had higher PSQI than group F.

Table (7): Comparison between group A and C as regards different variables:

Variables	Group A N = 10	Group C N = 10	t+	p
SBP(mmHg)	139±7	109±11	6	<0.001
DBP (mmHg)	103±8	69±5	12	<0.001
Creatinine (mg/dl)	11.1±0.6	9.9±1.3	3	<0.001
BUN (mg/dl)	91±26	95±13	1.2	>0.05
HB (gm/dl)	8.1±0.6	8.2±0.4	1	>0.05
Albumin (gm/dl)	3.8±0.9	3.9±0.2	0.9	>0.05
Na (mEq/L)	140±2.4	140±4	0.8	>0.05
K (mEq/L)	5.4±0.9	5.3±0.09	2.2	<0.05
Po4 (mg/dl)	6.5±0.2	6.5±0.6	0.7	>0.05
Ca (mg/dl)	7.5±0.9	7.5±0.3	0.9	>0.05
Uric acid (mg/dl)	6.5±1.2	4.7±0.8	3.3	<0.05

* unpaired t-test

Patients with hypertension and chronic renal failure (group A) had higher SBP, DBP , creatinine, K and uric acid than patients with normal blood pressure and chronic renal failure (group C).

Table (8): Comparison between group A and C as regards questionnaire and different sleep parameters and BMI

Variables	Group A N = 10	Group C N = 10	t+	p
BMI	36.8±3	28±3	3	<0.05
PSQI	14.9±2	13.3±1.2	2.5	<0.05
A/H index	41.6±8	14.4±3	3	<0.001
Des. Index	35.6±8	11.5±2	2	<0.05
TWE	17.9±4	12.5±3	1.7	>0.05
Arousal index	24.8±4	20.7±4	1**	>0.05
Total number of snoring episodes	141±30	86±16	1.9*	>0.05
Mean duration of snoring episode (mns)	8.8±2.1	6.8±1.2	0.7*	>0.05
Snoring episode %	38±8	32±7	0.8*	>0.05
Duration of snoring (mns)	7.5±1.5	5.6±1	0.6*	>0.05
Total number of PLM	54.7±10	26±6	1.8	>0.05
Duration of PLM (mns)	113±23	95±20	1.7*	>0.05
Duration of PLM %	106±20	32±7	2	<0.05
TT PLM (mns)	25.4±6	8±5	1.9	>0.05
Sleep latency (mns)	28.8±6	36±8	0.7	>0.05
REM latency (mns)	165±35	105±29	1.8*	>0.05
Sleep efficiency (%)	0.67±0.10	0.75±0.09	2.1*	<0.05

+ unpaired t-test

Group A showed higher BMI, PSQI, A/H index, Des Index, duration of PLM and sleep efficiency than group C.

Table (9): Comparison between group A and E as regards different variables:

Variables	Group A N = 10	Group E N = 10	t+	p
SBP(mmHg)	139±7	139±7	0.6	>0.05
DBP (mmHg)	103±8	98±6	0.7	>0.05
Creatinine (mg/dl)	11.1±0.6	0.99±0.19	53	<0.001
BUN (mg/dl)	91±26	11.6±1.6	15	<0.001
HB (gm/dl)	8.1±0.6	10.5±0.9	6	<0.001
Albumin (gm/dl)	3.8±0.9	4±0.5	1.1	>0.05
Na (mEq/L)	140±2.4	139±1.6	0.6	>0.05
K (mEq/L)	5.4±0.9	4.6±1	3.5	<0.05
Po4 (mg/dl)	6.5±0.2	3.8±0.5	14	<0.001
Ca (mg/dl)	7.5±0.9	8.7±0.9	0.8	>0.05
Uric acid (mg/dl)	6.5±1.2	5.9±1.3	0.6	>0.05

* unpaired t-test

Bad sleeper patients with hypertension and ESRD (group A) had higher creatinine , BUN, HB, K and PO_4 as compared to bad sleeper patient with only hypertension (group E).

Table (10): Comparison between group A and E as regards questionnaire and different sleep parameters and BMI.

Variables	Group A N = 10	Group C N = 10	t+	p
BMI	36.8±3	35.5±7	0.4	>0.05
PSQI	14.9±2	12.7±1.7	2.9	<0.05
A/H index	41.6±8	29±5	1.9	>0.05
Des. Index	35.6±8	26±6	1.6	>0.05
TWE	17.9±4	16.7±3	0.7	>0.05
Arousal index	24.8±4	30.7±8	3.5	<0.05
Total number of snoring episodes	141±30	138±40	0.6	>0.05
Mean duration of snoring episode (mns)	8.8±2.1	8.3±1.5	0.09	>0.05
Snoring episode %	38±8	25.8±6	1.5	>0.05
Duration of snoring (mns)	7.5±1.5	6.4±1.5	1.1	>0.05
Total number of PLM	54.7±10	21.4±5	1.9	>0.05
Duration of PLM (mns)	113±23	174±34	1.9	>0.05
Duration of PLM %	106±20	108±20	0.3	>0.05
TT PLM (mns)	25.4±6	24.7±5	0.5	>0.05
Sleep latency (mns)	28.8±6	36.8±8	1.1	>0.05
REM latency (mns)	165±35	139±39	1.4	>0.05
Sleep efficiency (%)	0.67±0.10	0.74±0.15	0.7	>0.05

+ unpaired t-test

Group A had higher PSQI and arousal index than group E.

Table (11): Comparison between bad sleeper groups as regard diagnosis

Diagnosis	Group A N=10	Group C N=10	Group E N=10	X^{2*}	P
Mild OSA	3 (30%)	9 (90%)	4 (40%)		
Moderate OSA	2 (20%)	2 (20%)	2 (20%)		
Severe OSA	6 (60%)	0	2 (20%)		
PLM	4 (40%)	0	2 (20%)		16<0.001
Reduced sleep efficiency	0	1 (10%)	0		
Bruxism	0	1 (10%)	0		
Insomnia	0	1 (10%)	0		
Sleep induced OSA	0	0	1 (10%)		

* chi-square test

This table shows highly significant difference between the bad sleeper groups as regard diagnosis

Table (12): Correlation between SBP, versus different variables among group A

Variables	SBP r	SBP P
Creatinin	0.09	>0.05
BUN	0.11	>0.05
HB	-0.68	<0.05
Albumin	-0.65	<0.05
Na	0.60	<0.05
K	0.08	>0.05
Po_4	-0.11	>0.05
Ca	-0.17	>0.05
Uric acid	-0.15	>0.05
BMI	0.03	>0.05
PSQI	0.13	>0.05
A/H index	0.23	>0.05
Des. Index	-01.12	>0.05
TWE	0.19	>0.05
Arousal index	0.59	<0.05
Total number of snoring episodes	0.19	>0.05
Mean duration of snoring episode	0.18	>0.05
Snoring episode %	0.27	<0.05
Duration of snoring	0.37	<0.05
Total number of PLM	0.18	>0.05
Duration of PLM	0.17	>0.05
Duration PLM%	0.15	>0.05
TT PLM	0.46	<0.05
Sleep late	0.12	>0.05
REM	0.03	>0.05
Sleep eff.	0.18	>0.05

*Spearman correlation test

There was a negative correlation between SBP and each of Hb and albumin, and positive correlation between SBP and each of Na, arousal index, snoring episode%, duration of snoring, and TTPLM in bad sleeper patients with hypertension and ESRD (group A).

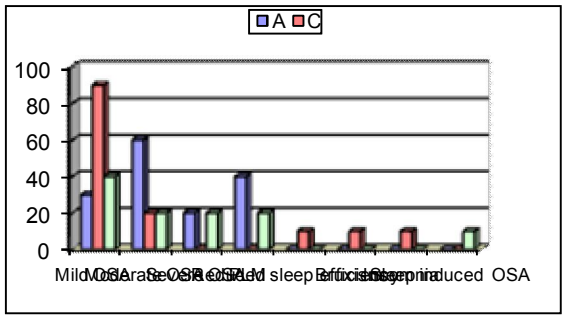

Figure (2): Comparison of bad sleeper groups as regards of different diagnosis of sleep disorders.

Table (13): Correlation between DBP, versus different variables among group A

Variables	DBP	
	r	P
Creatinin	0.17	>0.05
BUN	0.03	>0.05
HB	-0.19	>0.05
Albumin	-0.18	>0.05
Na	0.23	>0.05
K	0.26	>0.05
Po_4	0.15	>0.05
Ca	0.17	>0.05
Uric acid	-0.13	>0.05
BMI	0.20	>0.05
PSQI	0.24	<0.05
A/H index	0.04	>0.05
Des. Index	0.24	<0.05
TWE	0.15	>0.05
Arousal index	0.12	>0.05
Total number of snoring episodes	-0.13	>0.05
Mean duration of snoring episode	0.10	>0.05
Snoring episode %	0.62	<0.05
Duration of snoring	0.13	>0.05
Total number of PLM	0.17	>0.05
Duration of PLM	-0.13	>0.05
Duration PLM%	-0.19	>0.05
TT PLM	0.04	>0.05
Sleep late	0.22	<0.05
REM	0.18	>0.05
Sleep eff.	0.23	<0.05

*Spearman correlation test

There was a negative correlation between SBP and each of Hb and albumin, and positive correlation between SBP and each of Na, arousal index, snoring episode%, duration of snoring, and TTPLM in bad sleeper patients with hypertension and ESRD (group A).

Table (14): Correlation between creatinine, versus different variables among group A

Variables	Creatinine	
	r	P
SBP	0.23	>0.05
DBP	0.18	>0.05
Creatinin	-0.12	>0.05
BUN	0.04	>0.05
HB	0.22	>0.05
Albumin	0.09	>0.05
Na	0.14	>0.05
K	0.03	>0.05
Po_4	0.02	>0.05
Ca	0.18	>0.05
Uric acid	0.22	>0.05
BMI	-0.21	>0.05
PSQI	0.66	<0.05
A/H index	0.13	>0.05
Des. Index	0.32	>0.05
TWE	0.19	>0.05
Arousal index	-0.23	>0.05
Total number of snoring episodes	0.17	>0.05
Mean duration of snoring episode	0.18	>0.05
Snoring episode %	0.66	<0.05
Duration of snoring	0.16	>0.05
Total number of PLM	0.43	<0.05
Duration of PLM	0.17	>0.05
Duration PLM%	0.02	>0.05
TT PLM	0.23	>0.05
Sleep latency	0.74	<0.05
REM latency	0.65	<0.05
Sleep efficiency	0.45	<0.05

*Spearman correlation test

We found a positive correlation between serum creatinine and PSQI, snoring episode%, total number of PLM.

Table (15): Correlation between creatinine versus different variables among group C

Variables	Creatinine	
	r	P
SBP	0.20	> 0.05
DBP	0.10	> 0.05
Creatinin	-0.16	> 0.05
BUN	0.08	> 0.05
HB	0.75	<0.05
Albumin	0.03	> 0.05
Na	0.11	> 0.05
K	0.72	< 0.05
Po_4	0.07	> 0.05
Ca	0.14	> 0.05
Uric acid	0.20	> 0.05
BMI	-0.11	> 0.05
PSQI	0.44	< 0.05
A/H index	0.17	> 0.05
Des. Index	0.30	> 0.05
TWE	0.09	> 0.05
Arousal index	0.42	< 0.05
Total number of snoring episodes	-0.10	> 0.05
Mean duration of snoring episode	0.13	> 0.05
Snoring episode %	0.16	> 0.05
Duration of snoring	0.49	< 0.05
Total number of PLM	-0.20	> 0.05
Duration of PLM	-0.14	> 0.05
Duration PLM%	0.06	> 0.05
TT PLM	0.20	> 0.05
Sleep latency	0.14	> 0.05
REM latency	0.12	> 0.05
Sleep efficiency	0.45	< 0.05

*Spearman correlation test We found a positive correlation between creatinine level in the blood and each of HB, K, PSQI.

Table (16): Correlation between SBP versus different variables among group E

Variables	SBP	
	r	P
SBP	0.11	> 0.05
DBP	0.16	> 0.05
Cratinine	-0.11	> 0.05
BUN	0.06	> 0.05
HB	0.15	> 0.05
Albumin	0.07	> 0.05
Na	0.19	> 0.05
K	-0.08	> 0.05
Po_4	0.07	> 0.05
Ca	0.13	> 0.05
Uric acid	0.27	> 0.05
BMI	-0.13	> 0.05

PSQI	0.50	< 0.05
A/H index	0.45	> 0.05
Des. Index	0.35	< 0.05
TWE	0.13	> 0.05
Arousal index	0.52	< 0.05
Total number of snoring episodes	0.36	< 0.05
Mean duration of snoring episode	0.15	> 0.05
Snoring episode %	0.57	< 0.05
Duration of snoring	0.12	> 0.05
Total number of PLM	-0.10	> 0.05
Duration of PLM	-0.04	> 0.05
Duration PLM%	0.02	> 0.05
TT PLM	0.49	< 0.05
Sleep late	0.12	> 0.05
REM	0.16	> 0.05
Sleep eff.	0.27	> 0.05

*Spearman correlation test

We found a positive correlation between SBP and each of PSQI, desaturation index, arousal index, total number of snoring episodes, snoring episode % and TT PLM in bad sleeper patients with normal kidney function and hypertension (group E).

Table (17): Correlation between DBP, versus different variables among group E

Variables	DBP	
	r	P
SBP	0.17	>0.05
DBP	0.10	>0.05
Creatinine	-0.14	>0.05
BUN	-0.04	>0.05
HB	0.14	>0.05
Albumin	0.08	>0.05
Na	0.14	>0.05
K	-0.04	>0.05
Po_4	0.01	>0.05
Ca	0.13	>0.05
Uric acid	0.20	>0.05
BMI	-0.13	>0.05
PSQI	0.46	<0.05
A/H index	0.46	<0.05
Des. Index	0.33	>0.05
TWE	0.19	>0.05
Arousal index	-0.11	>0.05
Total number of snoring episodes	-0.13	>0.05
Mean duration of snoring episode	0.19	>0.05
Snoring episode %	0.37	<0.05
Duration of snoring	0.16	>0.05
Total number of PLM	-0.17	>0.05
Duration of PLM	-0.14	>0.05
Duration PLM%	0.06	>0.05
TT PLM	0.39	<0.05
Sleep late	0.15	>0.05
REM	0.12	>0.05
Sleep eff.	-0.20	>0.05

*Spearman correlation test There was a positive correlation between DBP and each of PSQI, A/H index, snoring episode % and TT PLM in group E.

4. Discussion

The relation between sleeping, waking and uremia has long been of interest, and ***Schreiner (1959)*** described the uremic patient's drowsiness by day with insomnia at night and the atypical responses to sedatives ***(Daly and Hassal, 1970)***. ***Gonzales et al. (1963)***, reported insomnia in his patients being treated with hemodialysis, thought to be of functional origin. ***Shea et al. (1965)***, however noted that Soporific effects of dialysis occurred in every patient unless the patients was particularly anxious, a view that was supported by ***Menzies and Stewart (1968)***.

An objective study of sleep and dialysis was made by ***Passouant et al. (1967)***, using EEG recordings, they found that before dialysis the total duration of sleep was diminished, with myocolonic jerks and periods of wakefulness occurring throughout the night. Paradoxical (REM) sleep was diminished and occurred at irregular intervals. After dialysis, the sleep cycles became regular. Sleep quality is a major concern as regard the quality of life in patients with resistant hypertension and chronic renal failure. Sleep disorders affect the majority of chronic kidney disease (CKD) patients. Some investigators hypothesized that end stage renal disease (ESRD) directly influences the quality of sleep. Interestingly, 80% of hemodialysis patients suffer from sleep abnormalities ***(Sabry et al., 2010; Parish, 2009; Haba-Rubio et al., 2011; Ibrahim and Omar, 2011).***

In chronic renal failure patients, an increased prevalence of sleep apnea, restless leg syndrome, and periodic limb movement during sleep has been reported. Epidemiology, pathophysiology and treatment of sleep disorders in CRF and dialysis patients are still unclear and requires further research ***(Haba-Rubio et al., 2011).***

Our patients were relatively young as compared to other studies which stated that the sleep the general population as reported by ***Makhlouf et al. (2007); Colbay et al. (2007); Mavanur et al. (2010); Glass et al. (2005); Guney et al. (2010); Elias et al. (2009); Roumeliote et al. (2011); Sabbattini et al. (2003); Sakkas et al. (2008).***

This was not observed in HD population, on the contrary younger age ranges were reported by ***Koch et al. (2008) and Musci et al. (2004)***.

We didn't find significant difference as regards age between the six groups of our study which mean that age difference didn't affect our results.

Also, we didn't find a significant difference as regards sex distribution between the six groups of our study.

Most of our study patients were males with nearly absent female gender in some groups.

Colby et al. (2007); Bilgic et al. (2007) and Perl et al. (2006) reported that female gender has been found to be a good sleeper. ***Elias et al. (2009)***; ***Unruh et al. (2003)***; ***Argekar et al. (2007) and Walker et al. (1995)*** found that male sex was associated with more sleep complaints. Other studies, however, found no association between gender and sleep disturbances ***(Merlino et al., 2006; Musci et al., 2004; Sabbattini et al., 2002; and Koch et al., 2008)*** and even more frequent insomnia symptoms were reported in female patients ***(Edinger and*** **Means** ***2005; Pai et al., 2007)***.

The average BMI of our patients was 27.11 kg/m^2 which is normal or predisposing to mild obesity and it was not considered a contributing factor to poor sleep quality in our study.

BMI was significantly higher (P < 0.05) in bad sleepers of group A with both resistant hypertension and ESRD when compared with bad sleepers of group C with ESRD only, which reflects BMI importance in ESRD patients as a contributing factor for developing poor sleep quality and resistant hypertension.

The same result was obtained when comparing bad sleepers of group A with resistant hypertension and ESRD to good sleepers of group D with ESRD only.

Abdel-Kader et al. (2012), has stated that obesity has been independently associated with higher rates of uncontrolled HTN in both the general population and among those with kidney disease.

Among risk factors for sleep disordered breathing (SDB), obesity and neck circumference are considered as reported by ***Iseki et al.(2007); Elias et al. (2009); Roumelioti et al. (2011)*** and also increased BMI as reported by ***Guney et al. (2010) Elder et al. (2008), Tada et al. (2007) and Unruh et al. (2008)***.

Moving a BMI > 30 was associated with a higher probability of sleep apnea ***(Argekar et al., 2007; Sakkas et al., 2008).***

The threshold for obstructive sleep apnea development among patients on hemodialysis is lower than in the general population (BMI in HD of 26-28) as compared to > 30 to > 42 in the general population ***(Rodrigues et al., 2005).***

Multivariate analysis showed that BMI was independently associated with occurrence of Sleep Apnea (OR 1.20, 95% CI 1.05-1.38, p = 0.008).

Mavanur et al. (2010); ***and Beecroft et al. (2007),*** stated that unlike the general population, obesity was not consistently associated with Sleep Disordered Breathing in dialysis patients.

In our study, we have used PSQI questionnaire to divide our patients into good sleepers and bad sleepers. Using PSQI questionnaire was used by different studies in assessment of sleep quality with many other disorders, by ***Huang et al. (2011); Al-Jadahli (2011); Sabbattini et al. (2003); Iliescu et al. (2003); Cengic and Resik (2012); Loewen et al. (2009); and Araujo et al. (2010).***

In our study, we have performed laboratory studies for good sleeper groups as well as for bad sleep groups.

In our study, serum creatinine was higher in bad sleepers of group A with resistant HTN and ESRD when compared to good sleepers of group B with resistant HTN and ESRD (P < 0.001).

Also creatinine was significantly higher (P < 0.001) in group A than in group C with ESRD only, both groups being bad sleeper groups.

BUN levels were significantly higher (P < 0.001) in bad sleepers of group C with ESRD only, as compared to good sleepers of group D with ESRD only.

Our results suggests that dialysis inefficiency is, at least in part, a cause of sleep abnormality and this means that uremia does affect the patients sleep pattern.

As regards other laboratory parameters, especially in bad sleepers with ESRD (group A and group C), serum potassium and uric acid were significantly higher in bad sleepers of group A with resistant HTN and ESRD as compared to good sleepers of group B" with resistant HTN and ESRD, and also in bad sleepers of group C with ESRD only as compared to good sleepers of group D with ESRD only.

Lee et al. (2009), in their study found that uric acid was correlated positively with total sleep time (TST) (r = 0.407) and negatively with apnea hypopnea index (AHI) and oxygen –desaturation index (ODI) (r = -0.377,r = -0.405).

Hsu et al. (2004), have suggested a positive effect of uric acid related to its anti-oxidant properties, predicting high mortality in HD patients with lower uric acid levels.

We have also found significantly lower Hb (P < 0.001) and higher phosphorous (P < 0.001) in bad sleepers of group A with resistant HTN and ESRD as compared to bad sleepers of group E with resistant HTN only and this confirms our opinion about inefficient dialysis as a cause of sleep disturbances.

Haba-Rubio et al. (2011); and Ibrahim and Omar (2011) reported that sleep disorders were common in patients receiving hemodialysis. Studies have suggested that the very high prevalence of sleep disorders (especially sleep apnea) in ESRD reflects suboptimal dialysis ***(Pai et al., 2007; Hanly et al., 2009; Perl et al., 2006; Unruh, 2007; Ibrahim and Omar, 2011; Bastros et al., 2007; Koch et al., 2008; Mavanur et al., 2010; Elias et al., 2009; Guney et al., 2010; Argekar et al., 2007; Al- Jadahli, 2011; and Deloach and Berns, 2009).***

On the contrary; ***Elder et al. (2008)***; ***Musci et al. (2004); Sakkas et al. (2008); Beecroft et al. (2007); and Lerma (2011)*** stated that poor sleep quality was not affected by Kt/v, serum albumin or treatment time.

Attempts in optimizing uremia control in the form of nocturnal hemodialysis (NHD) and renal transplantation have shown early clinical success in treating sleep disorders as stated by ***Unruh (2007) and Tang et al. (2006).***

Al-Jadahli (2011); ***Sabry et al. (2010); Kovacs et al. (2011); Elder et al. (2008); Sabbattini et al. (2008); Koch et al. (2008)*** in their studies reported that the risk factors for insomnia were inadequate dialysis, anemia, hypoalbuminemia and hyperphosphatemia.

Haba-Rubio et al. (2011); Ibrahim and Omar (2011); Al-Jadahli (2011); Araujo et al. (2010) and Gigli et al. (2004) stated that insomnia, restless leg syndrome (RLS) and obstructive sleep apnea were significantly associated with these mentioned risk factors in patients with ESRD on regular HD.

Koch et al. (2008), found that in patients with ESRD, the circadian sleep-wake rhythm can be disrupted by both internal factors (biochemical parameters and melatonine) and external factors (dialysis and medications).

Polysomnography is the gold standard for diagnosing sleep disorders, as reported by ***Argekar et al. (2007)***; ***Unruh et al. (2007); Ballard (2005); Lee et al. (2009); Loewen et al. (2009); Liakopoulos et la. (2008); Enomoto et al. (2008); Roumelioti et al. (2011); Elias et al. (2009); Iseki et al. (2007); Tang et al. (2010); Rodrigues et al. (2005); Koch et al. (2008); Mavanur et al. (2010); Tada et al. (2007); Miskowiec et al. (2006).***

Sleep efficiency under normal circumstances, is recorded best by means of polysomnogrpahy ***(Kushida et al., 2005)***.

Hanly et al. (2009) in their study using polysomnography found a high frequency of arousals in HD patients (up to 30/h), resulting in a sleep efficiency that ranged from 66% to 85%.

In our study, we found that nearly all polysomnogram parameters were abnormal in the three bad sleeper groups (group A, group C, and group E).

In our study, the most prevalent sleep disorder was obstructive sleep apnea , ranging from 20 to 60% in our bad sleeper groups (group A, group C, and group E), and to a much less extent periodic limb movement , ranging from 20% to 40% in the same mentioned groups.

Ibrahim and Omar (2011) in their study, found that prevalence of sleep disorders was 61.4% of their HD patients, the survey included insomnia (57.6%), RLS (56.4%) and OSA (21.2%).

Al-Jadahli (2011), reported that 46% of HD patients included in his study suffered from RLS, 67% suffered from sleep apnea, and 59% suffered from insomnia.

Loewen et al. (2009), has reported sleep apnea in up to 50-70% of patients with ESRD.

Elias et al. (2009), has reported sleep-disordered breathing ranging from 50 to 73% and the majority of respiratory events were represented by obstructive sleep apnea.

Lee et al. (2009), reported sleep apnea ranging from 50 to 70% of ESRD patients, which was more than 10 times the range in general population, 60% of patients had PLM and 50% of patients had both Sleep Apnea Syndrome and PLM.

Prevalence of sleep apnea with ***Deloach and Berns (2009)*** was 30 to 80% of HD patients, and in ***Argekar et al. (2007)*** study, ranging from 50-60%.

In ***Hanly et al. (2009) and Rodrigues et al. (2005)*** studies, OSA was the predominant type of sleep apnea in ESRD patients.

Gaetano et al. (2010), found that RLS affected 31% of his studied HD population, and in ***Araujo et al. (2010)*** study, RLS prevalence was 21.5%.

On comparing abnormal polysomnograms of bad sleeper of group A with HTN and ESRD to bad sleeper of group C with ESRD only, apnea/hypopnea index was found to be higher than normal in group A patients than group C ($P < 0.001$).

Also desaturation index was much higher in bad sleepers of group A than bad sleepers of group C ($P < 0.05$).

Duration of PLM% was much higher in bad sleeper of group A with HTN and ESRD as compared to bad sleeper of group C with ESRD only ($P < 0.05$).

Sleep efficiency was worse in bad sleepers of group A as compared to bad sleepers of group C ($P < 0.05$).

This means that ESRD does affect sleep disordered breathing and periodic limb movement and not simply inability to initiate or maintain sleep.

But coexistence of HTN, especially resistant hypertension, does exacerbate these disorders.

In bad sleepers of group A with HTN and ESRD, we found a positive correlation between creatinine and each of PSQI score ($P < 0.05$), snoring episode ($P < 0.05$), total number of PLM ($P < 0.05$), sleep latency ($P < 0.05$), REM latency ($P < 0.05$), and sleep efficiency ($P < 0.05$).

This shows the impact of high creatinine level on sleep pattern in ESRD patients.

A study made by ***Loewen et al. (2009)*** showed that patients treated with conventional hemodialysis had significantly greater odds of short sleep (OR 3.27 [1.16-9.25]) and less efficient sleep (OR 5-5 [1.5-19.6]) than a matched control group.

Anastassov and Trigger (1998); Al-Jadahli (2011); Abdel-Kader et al. (2012); Mavanur et al. (2010); and Sim et al. (2010), stated that sleep disordered breathing is highly prevalent in patients with CKD and may be due to volume overload, upper airway narrowing, older age and other co-morbidities.

Loewen et al. (2009) and Beecroft et al. (2009) found that obstructive sleep apnea was the most frequent type of sleep disordered breathing in ESRD patients.

Langevin et al. (2010), stated that during metabolic acidosis, compensatory hyperventilation leads to hypocapnia, thus more easily reaching the apneic threshold at sleep onset, generating periodic respiration and destabilization of respiratory control.

In a study of ***Beecroft et al. (2006)***, 49 ESRD patients with sleep apnea were found to have a higher apneic threshold and a higher sensitivity to hypercapnia. Their results suggested that the sensitivities of both central and peripheral chemoreceptors are increased with sleep apnea and ESRD.

Sleep apnea is characterized by abnormal respiratory patterns during sleep (apnea, hypopnea) and disturbed sleep (snoring, restlessness, resulting in daytime sleepiness and fatigue which may reduce quality of life

(QOL) and functional capacity ***(Deloach and Berns, 2009)***.

Roumelioti et al. (2011), in their study found that median AHI was higher in HD group as compared to control group.

Conventional HD group in ***Unruh (2007)*** study, had significantly less sleep time, more frequent arousals, increased number of apneas/hypopneas (> 30 respiratory events per hour and more severe nocturnal oxyhemoglobin desaturation).

Roumelioti et al. (2011),found that AHI was much higher (18.2) in HD patients that CKD patients (8.8) and controls (8.6) ($p = 0.002$). ***Loewen et al. (2009),***found that conventional HD patients were more likely to have severe sleep apnea (AHI > 30) with an odds ratio of 4.07 (95% CI 1.83 to 9.07) as compared to normal kidney function controls.

Tada et al. (2007) found that the number of obstructive apnea events per hour was significantly correlated with creatinine ($P < 0.05$, $r = 0.418$) and BUN ($r = 0.490$, $p < 0.01$). Also creatinine and BUN were correlated with OSA index ($P < 0.05$ and $P < 0.01$ respectively).

Nearly the same results were found by ***Iseki et al. (2007)***. Increasingly it is believed that "uremic factors" may play a role in the pathophysiology of sleep apnea in ESRD ***(Deloach and Berns, 2009).***

The dose of hemodialysis was significantly and inversely associated with OSA and remained significant in multivariate analysis ***(Elias et al., 2009)***. On contrary in ***Tada et al. (2007)*** study, patients with sleep apnea (SA) tended to have a larger means dialysis dose than patients without SA ($P = 0.056$).

Unruh (2007), Beecroft (2009) and Deloach and Berns (2009) reported improvement in sleep apnea following conversion to nocturnal HD.

Beecroft et al. (2008), demonstrated that improvement in sleep apnea symptoms was associated with an increase in pharyngeal cross-sectional area and that was related to nocturnal HD. ***Beecroft et al. (2007)*** found that pharyngeal area was smaller in ESRD patients.

Periodic limb movement syndrome (PLMS) is a common dysfunction of motor control during night time sleep, and it is frequently associated with signs of arousal in the electroencephalogram (EEG) plus activation of the autonomic nervous system ***(Portaluppi et al., 2009).***

Hanly (2009), ***Gigli et al. (2004), Araujo et al. (2010) and Al-Jadahli (2011),***reported that potential risk factors for RLS and PLM disorders in HD patients included peripheral and central nervous system abnormalities and hyperparathyroidism besides the previously mentioned risk factors of sleep disorders.

An association between RLS and cardiovascular disease has been observed in two epidemiologic studies on the general population ***(Ulfberg et al., 2001; Winkelman et al., 2008)a***nd one community based study of large middle age and elderly cohort of the sleep heart health study ***(Winkelman et al., 2008)***.

Portaluppi et al. (2009), reported that the prevalence of restless leg syndrome (RLS), which is high in dialysis patients would play a role in the pathogenesis of sleep-hypertension in renal patients.

The association between RLS and PLMS and the risk of hypertension, however, is controversial ***Ohayon and Roth,(2002)***; ***Ulfberg et al. (2001***);W***inkelman et al. (2008)***.

Sforza et al. (1999) and ***Siddiqui et al. (2007)***, have reported that episodes of PLM are associated with significant repetitive sleep time elevations of heart rate plus systolic blood pressure (SBP) and diastolic blood pressure (DBP).

Because of the importance of the relationship between PLM and respiratory events, as indicated by the finding of a strong linear correlation of rise in BP with the duration of respiratory related limb movements, and it seemed possible that some PLM may be in fact respiratory-related and connected to BP rise in other ways than previously postulated ***(Siddiqui et al., 2007).***

The resulting over activity of the sympathetic nervous system seemed to be responsible for non dipping BP pattern associated with PLM ***(Siddiqui et al., 2007).***

Benz et al. (2000) reported that, in renal patients with sleep disorders, the parameters used to quantify the severity of PLMs (PLM index, arousing PLM index, and arousal index), as determined by polysomnography, have been found to be associated with mortality.

In ***Enomoto et al. (2008)*** study, using polysomnography, the uremic RLS group had a higher arousal index and a higher sleep stage I percentage than controls. Uremic RLS patients were more vulnerable to the appearance of PLM, not only while asleep, but also while awake.

On comparing polysomnograms of bad sleepers of group A with HTN and ESRD to bad sleepers of group E with resistant HTN alone, arousal index was found to be higher in group E than in group A, both having an arousal index higher than normal ($P < 0.05$). This means that resistant hypertension alone can affect sleep process and causes sleep disorders. All other polysomnogram parameters were abnormal in both group A and group E.

Without significant difference between both groups. This shows how much resistant hypertension is deleterious for sleep quality, although bad sleepers of group E had normal kidney function.

In bad sleepers of group A with HTN and ESRD systolic blood pressure (SBP) had a negative correlation with each of Hb ($P < 0.05$), and albumin ($P < 0.05$). Also, SBP had a positive correlation with each of Na ($P < 0.05$), Arousal index ($P < 0.05$), Snoring episode % ($P < 0.05$), duration of snoring ($P < 0.05$), and total time PLM ($P < 0.05$).

Diastolic blood pressure (DBP) in bad sleepers of group A, had a positive correlation with each of PSQI score ($P < 0.05$), desaturation index ($P < 0.05$), snoring episode % ($P < 0.05$),

Sleep latency ($P < 0.05$) and sleep efficiency ($P < 0.05$).

In bad sleepers of group E with resistant hypertension only, SBP had a positive correlation with each of PSQI score ($P < 0.05$), arousal index ($P < 0.05$), total number of snoring episodes ($P < 0.05$), snoring episode ($P < 0.05$) and total time PLM ($P < 0.05$). DBP in bad sleepers of group E had a positive correlation with each of PSQI scores ($P < 0.05$), A/H index ($P < 0.05$), snoring episode % ($P < 0.05$) and total time PLM ($P < 0.05$).

Our results showed the relationships of resistant HTN with polysomnogram parameters in bad sleeper groups with hypertension, whether having ESRD or normal renal function.

People are sleeping less in modern societies. It is said that short sleep duration could play a role in the etiology of HTN ***(Sasanabe and Shiomi, 2009)***.

Experimentally induced sleep deprivation can raise blood pressure and worsen HTN.

Short sleep duration is associated with resistant hypertension, independently of OSA, which can itself disrupt and shorten sleep ***(Friedman et al., 2010).***

All hypertensive patients should be routinely screened for possible sleep disorders ***(Sasanabe and Shiomi, 2009).***

Huang et al. (2011) found that non dippers with poor sleep quality had an increased activity of sympathetic nervous system and a more stressful status. The decline of SBP and DBP at night was inversely related to PSQI score.

The contribution of sleep disorders to resistant HTN remains incompletely characterized. Studies examining the link between OSA and resistant HTN have explicitly excluded patients with moderate to severe chronic kidney disease or have had relatively few patients with advanced CKD ***(Abdel-Kader et al., 2012).***

Friedman et al. (2010), reported a significantly higher prevalence of obstructive sleep apnea (OSA) and reduced rapid eye movement (REM) sleep time in drug resistant hypertensives compared to controlled hypertensives. The hypoxemia, arousals and intrathoracic pressure changes associated with sleep apnea leads to sympathetic nervous system activation, endothelial dysfunction, inflammation and oxidative stress, which may be associated with arterial HTN and other adverse cardiovascular outcomes ***(Elias et al., 2009; Deloach and Berns, 2009; Hanly, 2009 and Unruh, 2007).***

Obstructive sleep apnea (OSA) is common in patients with HTN with a prevalence of up to 50% ***(Abdel-Kader et al., 2012).***

Patients with OSA needed significantly more pills than patients without OSA for optimal BP control ***(Elias et al., 2009)***

Rodrigues et al. (2005) found an impressive correlation between sleep apnea and blood pressure in dialysis patients independent of antihypertensive treatment.

In the prospective Wisconsin sleep cohort study ***(Peppard et al., 2000)***, observed that in the general population, the presence of sleep disordered breathing was associated with the development of HTN four years later, suggesting a cause effect result. Other investigators ***(Fletcher et al., 1985; Garcia-Rio et al., 2000; Lavie et al., 2000),***also have demonstrated an association between sleep disorders and hypertension in prospective or cross-sectional studies.

Abdel-Kader et al. (2012) observed that resistant HTN and severe OSA (AHI > 30) significantly more prevalent in patients with advanced kidney disease (30% resistant HTN, 25% severe OSA). ESRD patients with severe OSA were seven fold more likely to have resistant HTN than their counterparts.

Rodrigues et al. (2005), found that systolic, diastolic and mean blood pressure were higher in patients with AHI > 5 and were the only parameters significantly and positively correlated with AHI.

Demede et al. (2011) showed that patients with resistant HTN were nearly 2.5 times more likely to be at high OSA risk, relative to those with controlled HTN.

Bansil et al. (2011), in their study, found no relationship between having a sleep disorder and hypertension if short sleep or poor sleep was not also present.

Araujo et al. (2010), stated that hypertension was found associated with moderately severe RLS.

Canadian investigators have published in the journal (neurology) a study showing that patients with RLS demonstrated increase in systolic blood pressure averaging 22 mmHg and in diastolic pressure averaging 11 mmHg, and further more these blood pressure spikes occurred repeatedly every 20 to 40 seconds throughout the night ***(Forogos, 2011)*** Also ***Araujo et al. (2010)***, stated that systolic and diastolic predialysis blood pressures tended to be higher in RLS cases. Odds ratio for development of hypertension in RLS patients are well over 2 fold ***(Pennestri et al., 2007).***

4. Conclusion:

ESRD and hemodialysis, through different pathophysiology mechanisms are enough to cause sleep disorders and poor sleep quality, with their effects exacerbated by the presence of resistant hypertension. Also resistant hypertension alone is enough to disrupt normal sleep pattern away from OSA or RLS.

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