Sleep Disorders in Peripheral Neuropathy: A Clinical and Polysomnographic Study

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ABSTRACT

Background and Purpose: *A fair number of sleep disorders are associated with peripheral neuropathies due to various mechanisms. Our* ***aim*** *was to assess different patterns of sleep disturbances in polyneuropathy patients.* ***Methods:*** *We studied the sleep architecture and sleep abnormalities in 20 polyneuropathy patients of different etiologies and 20 healthy control subjects. All patients and controls underwent clinical assessment and electromyographic and polysomnographic testing.* ***Results*** *revealed 40% of the patients reported positive sleep complaints mostly in the form of muscle cramps, numbness and tingling sensations in lower limbs and leg restlessness. The percentages of stages 1& 2 were significantly increased and SWS was significantly reduced in patients compared to controls, the apnea /hypopnea index were increased specially in REM and 25% of the patients had a significant pathological periodic limb movement index (PLMI). We* ***concluded*** *that sleep disordered breathing and periodic leg movements during sleep are not uncommon and important to recognize in patients with polyneuropathy and can cause frequent and increased awakenings.* **(Egypt J. Neurol. Psychiat. Neurosurg., 2008, 45(2): 375-385)**

INTRODUCTION

Peripheral neuropathy refers to a scope of clinical syndromes affecting a variety of peripheral nerve cells and fibers, including motor, sensory, and autonomic fibers. Most generalized disorders conform to a polyneuropathy syndrome, which usually implies both sensory- and motor-fiber involvement in a relatively symmetric fashion and typically with a distal-to-proximal gradient of involvement, which represent the most common form of peripheral neuropathy.[1]

In general, patients with peripheral nervous system dysfunction complain of sensory disturbance, motor weakness or both. The most common generalized polyneuropathy is diabetic sensory-motor polyneuropathy, which occurs more in type 1 than in type 2 diabetic patients [2]. Peripheral nerve pains are often more active at night. [3]

Typically, symptoms of sensory disturbance range along a continuum from "negative" phenomena as numbness, loss of sensation to "positive" phenomena as tingling, burning, "pins and needles," bands of tightness, and stabbing or shooting pain or both. Sensory symptoms may be subtle and are not always present during physical examinations, particularly when it is early in the progression of peripheral nervous system dysfunction.[3]

Deep-tendon reflexes are often decreased or absent in peripheral nervous system dysfunction. Hyporeflexia implies dysfunction of large myelinated fibers representing the afferent limb of muscle spindle–initiated reflexes. If the pathologic process is limited strictly to small sensory fibers, deep-tendon reflexes may not be affected.[4]

Motor symptoms are usually found in combination with sensory deficits. Isolated motor findings do occur and suggest that the disease process may be limited to the ventral horn or roots. Respiratory muscles and the diaphragm may be involved which can cause sleep and respiratory dysfunctions[5]. Such findings may represent acute inflammatory demyelinating polyradiculoneuropathy, or amyotrophic lateral sclerosis.[6]

Most of the sleep disturbances in neuromuscular disorders are secondary to sleep-related respiratory dysfunction. However, other factors as pain, muscle cramps, muscle immobility,

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joint pains, contractures, kyphoscoliosis, obesity due to sedentary lifestyle, craniofacial abnormalities, anxiety and depression may further contribute to sleep dysfunction (e.g. insomnias and excessive daytime sleepiness).[7]

The single most important laboratory test in patients with sleep complaints secondary to neuromuscular disease is polysomnographic recording. Polysomnography refers to the monitoring during sleep of many biological variables including brain activity, respiration, heart rhythm, oxygen saturation, muscle tone, eye movement, and body movement and the evaluation of their relationships with specific states of alertness.[8]

The aim of the present study is to assess the various patterns of sleep disturbances in peripheral neuropathies and to compare the polygraphic sleep measures in patients and healthy age matched controls.

SUBJECTS AND METHODS

Subjects

Twenty patients (five females and fifteen males) presenting with peripheral neuropathy were included in this study. They were recruited from the Neurology department and out patient clinics of Cairo University Hospital in the period from July 2006 to June 2007. The study also included twenty healthy subjects matching for age, sex and body mass index (5 females and 15 males) as a control group.

Excluded from this study were patients suffering from severe medical illness affecting cardiac or respiratory systems, and obese subjects with body mass index>30. [9]

Methods

Patients were submitted to the following***:***

1. **Detailed history taking**: with special stress on the onset, course and duration of sensory, motor and autonomic symptoms of peripheral neuropathy, drug intake which may affect sleep architecture, and family history of similar conditions.
2. **Sleep history** A check list was introduced to help identify sleep dysfunctions with special emphasis on total nocturnal sleep time, sensory manifestations (paresthesias, dysesthesias) or motor events (cramps, leg restlessness) interfering with sleep pattern, autonomic events such as (sweating, piloerection, or tachycardia), sleep atone related events as sleep paralysis or cataplexy, insomnia, and respiratory complaints. The sleep check list was applied for both patients and controls.
3. **Thorough general and neurological examination** according to the peripheral neuropathy sheet of the neurology department of Cairo University Hospital.
4. **Routine laboratory investigations** were carried out as complete blood count, Erythrocyte sedimentation rate, serum glucose levels, and liver and renal function tests to help detecting the etiology of polyneuropathy.
5. **Nerve conductions and electromyography studies**: The modalities of electromyography and nerve conductions were done to verify the diagnosis of peripheral neuropathy and detect the type of neuropathy axonal or demylination using a Neuropack mini four apparatus. Nerve conduction studies were carried out for motor conduction in the left median, right ulnar, left common peroneal and the right posterior tibial nerves. Sensory conduction studies were carried out for the right ulnar and medial planter nerves. Conventional EMG examination was done for the right gluteus medius, the left quadriceps, the right tibialis anterior, the left medial head of gastrocnemius, the right extensor digitorum brevis, the left abductor digiti minimi and the extensor digitorum communis muscles.
6. **Polysomnographic study:** Overnight polysomnography was performed to both patients and controls in the Department of Neurology, Clinical Neurophysiology unit, using a *Schwarzer*. *Epos 32 GmpH*, medical diagnostic polysomnogram, *Schwarzer*, *Germany*. The software used was *Somnologica* version 3.1.

The subject was prepared about one hour prior to the recording by attaching electrodes using average 12-18 channels. The recording time ranging from 6 to 8 hours, the following components were recorded according to Rechtschaffen and Kales[10] Electroencephalography (EEG) four channel recording, Electrooculogram (EOG) two channels, two submental electrodes, respiratory effort

measurement using two elastic belts placed around the thorax and abdomen, airflow cannula placed in the nostrils and over the mouth, a channel for upper airway sound detection, pulse oximeter, and body position sensor.

The recording was scored manually for sleep stages according to the standard scoring system for sleep stages.[10]

The following parameters were measured: Sleep efficiency, number of awakenings, attacks of obstructive and central apnea per hour, oxygen desaturation per hour, percentage of wake state per total sleep period, the percentage of REM and NREM stages per total sleep period, REM onset latency, leg movement index (LMI) with regard to time in bed. LMI >15 h is pathological and periodic limb movement index (PLM) which equals the number of PLM episodes (stereotyped leg movements involving one or both limbs, 0.5 to 5 seconds in duration that recurs primarily in stages 1 and 2 of sleep) during the night/ TST. A PLM index above 5 per hour is considered pathologic[10].

Statistical Methods:

Data were tabulated and statistically analyzed to evaluate the difference between the groups under study as regards the various parameters. The statistical analysis included: arithmetic mean, standard deviation. Student's "t" test was used to test the significance of differences between two mean values. Pearson's correlation test was used to detect if change in one variable is accompanied by changes in the other variable. The results were considered significant if P-value<0.05, while P-value>0.05 indicates non significant and P<0.01 highly significant values.

RESULTS

The present study is a cross sectional case-control study conducted on 20 patients with polyneuropathy and 20 healthy control subjects. The age of patients ranged from 20 to 70 years with a mean age of 46.8±16.5 years. The age of the controls ranged from 20 to 70 years with a mean of 40.3±17.3 (p > 0.05).

The duration of polyneuropathy ranged between 9 months to 98 months with a mean of 36.9±5.3 months.

Clinical Results:

Neurological history and examination:

The etiology of polyneuropathy included seven patients 35% with diabetic polyneuropathy, six patients 30% with hereditary polyneuropathy, four patients 20% with hepatic polyneuropathy and three patients 15% with uremic polyneuropathy. Neurological examination revealed signs of bilateral combined sensory motor neuropathy affecting both upper and lower limbs, superficial sensory affection in the form of glove and stocking were detected in 16 patients 80% while deep sensory affection were in nine patients 45% the neuropathy were mainly sensory in diabetic patients and in patients with chronic renal failure . Autonomic neuropathy was detected in five patients 25% in the form of sweating, postural hypotension, impotence, and tachycardia.

Sleep history:

Only eight patients 40% reported positive sleep complaints while twelve patients 60% did not report history of any sleep disturbance. The sleep complaints were mainly muscle cramps, numbness and tingling sensations in lower limbs and leg restlessness interfering with sleep, feeling of not getting enough sleep and feeling drowsy during the day. None of the controls reported any sleep complaints.

According to nerve conduction studies patients were classified into patients with predominantly axonal polyneuropathy (4 patients purely axonal and 10 patients had mixed mainly axonal (70%)) and patients with predominantly demyelinating polyneuropathy (6 patients (30%)).

Polysomnographic Results:

The minimum, maximum and mean values of sleep architectures in patients and controls are shown in Table (1).

The number of awakening was significantly higher in patients compared to controls P = 0.0001. No significant differences were detected regarding other sleep parameters (Table 1).

The minimum, maximum and mean values of sleep stages in patients and controls are shown in Table (2).

The percentages of stages 1&2 were significantly increased in patients compared to controls P=0.0015, P=0.0025 respectively, while the percentage of SWS was significantly reduced P=0.0284 (Table 2).

The mean and range of the values of different sleep abnormalities in the patients and controls are shown in Table (3).

All sleep abnormalities measured were affected in our patients (Table 3). Five patients 25% had a significant pathological PLM>5/hour. One had hereditary polyneuropathy, two were diabetic and two had uremic polyneuropathy, while nine patients 45% had an increase in the leg movement index; LMI>15/hour who were subsequently categorized into periodic, and nonperiodic movements (defined as neither periodic nor related to a respiratory event).

The two types of apnea (central and obstructive) and the hypopnea indices were significantly increased P=.02, P=.04, and P=.0000 respectively. Also the apnea /hypopnea indices were increased in REM and non REM stages and the O2 saturation levels were significantly reduced in REM and non REM stages in our patients.

No statistical significant differences were detected as regards the sleep architecture parameters between patients having axonal polyneuropathy type or demyelinating polyneuropathy type (p>0.05) (Table 4).

No statistically significant differences were detected in the percentages or latencies of sleep stages between patients having axonal polyneuropathy type or demyelinating polyneuropathy type (p>0.05) (Table 5).

The leg movement (LM) index, periodic limb movement (PLM) index, hypopnea index, oxygen desaturation index and average of oxygen saturation in REM and non REM in axonal polyneuropathy patients were all non significantly increased compared to demyelinating polyneuropathy patients, while the central apnea index was non significantly increased in the latter group (Table 6).

Correlating the age of the patients with different sleep parameters showed that there were a high positive correlation between the age and the percentage of stage 1 sleep (r=0.57), and a significant negative correlation between the age and the percentage of stage 2 of sleep (r=0.49) and the latency of slow wave sleep (r=0.519).

No correlation was detected between age of the patients and OSA, or PLMI. No correlation was found between sex and the different parameters of sleep abnormalities.

No statistical significant correlation could be detected between the duration or the type of the polyneuropathy and the different sleep parameters.

Table 1. Values of mean and P values in patients and controls regarding sleep architectures.

tnemerusaeM	Patient			Control			
	niM	xaM	Mean±SD	niM	xaM	Mean±SD	eulav-P
TST	129.5	479	99.1 ±352	353	477	388.8±35.2	0.1256
tesno peelS	2.2	83.6	20.5±19.9	0.5	59	13.2±13.1	0.2257
Sleep efficiency (%)	45.9	98.7	82.1±14.6	81.8	94.5	88.8±3.58	0.0597
No of Awakenings	2	30	12.5±7.51	1	9	4.6±2.76	0.0001**

TST= Total sleep time **= highly significant

Table 2. Values of mean and P values in patients and controls regarding sleep stages.

tnemerusaeM	Patient			Control			
	niM	xaM	Mean±SD	niM	xaM	Mean±SD	P value
S1 latency (min)	2.2	83.6	20.5±19.9	0.5	59	13.2±13.1	0.2257
S2 latency (min)	3.2	133.5	47.2±35.7	7.3	177	45.1±44.5	0.8797
SWS latency (min)	29	295.7	117.2±80.7	13.8	222	81.5±59.6	0.1270
REM latency (min)	23.5	400	148.8±104	43.5	358	136.4±86.4	0.8794
S1 %	7.8	77.9	34.2±19.4	0.5	33.7	13.9±10	0.0015**
S2 %	3.6	53.2	34.8±14.6	17.9	61.8	49.0±9.5	0.0025**
SWS %	0	32.2	15.7±11.9	4.5	66.5	25.6±13.5	0.0284*
REM %	0	47.9	14.9±11.0	4.3	31.2	13.2±6.2	0.5327

*= significant **= highly significant

Table 3. The range, mean±SD and P values of sleep abnormalities in the patients and controls.

Measurements	Patient		Control		P value
	Range	Mean±SD	Range	Mean±SD	
LMI	0.6 –36.4	14.3±11.3	0 – 14	3.1±4.7	0.0002**
PLMI	0 – 18.4	4.1± 6.0	0	0	0.0037**
AHI	2.8 – 130.7	23.4±28.2	0 – 3.6	0.7±0.9	0.0009**
AHI in REM	0 – 95.5	24.5±20	0 – 10.9	0.8±2.4	0.0000**
AHI in non REM	1.2 – 138.6	23.3±30.3	0 – 4	0.7±1.03	0.0019**
Central apnea	0.3 – 50.7	7.2±13.3	0 – 1.8	0.18±0.5	0.0221*
Obstructive apnea	0 – 22	3.6±7.5	0 – 1.2	0.11±0.3	0.0414*
Hypopnea	1.5 – 21.1	9.8±5.5	0 – 2.1	0.4±0.5	0.0000**
Oxygen desaturation	0 – 25.3	8.1±8.0	0 – 6.4	1.5±1.9	0.0011**
Lowest O_2 saturation	50 – 94	78.1±12.7	81 – 97	91.4±4.7	0.0001**
O_2 saturation in REM	88 – 98.2	94.7±3.0	92 – 99	96.6±1.92	0.0239*
O_2 saturation in non REM	85.6 – 98	94.7±3.2	92 – 98.5	97±1.72	0.0102*

LMI=leg movement index, PLMI= periodic limb movement index, AHI= apnea/hypopnea index.
* = significant. **= highly significant

Table 4. The mean±SD and P value of different parameters of sleep architecture in patients with axonal and demyelinating polyneuropathy.

Sleep architecture	Type of Polyneuropathy	Mean	± SD	F value	P value
TST	Axonal n=14	355.00	118.59	0.197	0.662
	Demyelinating n=6	350.52	92.29		
Sleep onset	Axonal	15.04	13.83	3.449	0.797
	Demyelinating	23.45	22.57		
Sleep efficiency	Axonal	83.77	16.82	0.128	0.7243
	Demyelinating	81.20	13.96		
Number of awakenings	Axonal	12.29	9.96	0.496	0.49
	Demyelinating	12.62	6.31		

Table 5. The mean±SD and P value of sleep stages latency (in minutes) and percentages in patients with axonal and demyelinating polyneuropathy.

Sleep stage	Group	Mean	± SD	F value	P value
S1 latency (min)	Axonal n=14	15.04	13.83	3.449	0.797
	Demyelinating n=6	23.45	22.57		
S2 latency (min)	Axonal	30.07	20.83	0.209	0.652
	Demyelinating	56.45	39.23		
SWS latency (min)	Axonal	121.10	77.74	0.050	0.825
	Demyelinating	115.31	86.27		
REM latency (min)	Axonal	79.50	58.91	0.024	0.878
	Demyelinating	180.85	107.4		
S1%	Axonal	39.91	13.00	3.943	0.625
	Demyelinating	31.23	22.10		
S2%	Axonal	28.03	14.71	2.862	0.107
	Demyelinating	38.53	13.72		
SWS%	Axonal	13.40	11.47	0.554	0.466
	Demyelinating	16.95	12.49		
REM%	Axonal	18.73	16.39	0.003	0.954
	Demyelinating	12.88	6.77		

Table 6. The mean±SD and P value of sleep abnormalities in patients with axonal and demyelinating polyneuropathy.

Sleep abnormality	Group	Mean	± SD	F value	P value
LMI	Axonal n=14	17.04	12.2	0.023	0.879
	Demyelinating n=6	12.87	11.0		
PLMI	Axonal	5.31	7.01	0.03	0.862
	Demyelinating	3.55	5.63		
AHI	Axonal	17.93	11.9	0.864	0.364
	Demyelinating	26.42	34.1		
AHI REM	Axonal	22.70	11.3	0.627	0.438
	Demyelinating	25.52	23.7		
AHI in non REM	Axonal	17.59	13.1	0.899	0.355
	Demyelinating	26.48	36.6		
Central apnea index	Axonal	3.40	4.56	0.852	0.368
	Demyelinating	9.37	16.0		
Obstructive apnea index	Axonal	3.23	7.35	1.068	0.314
	Demyelinating	3.92	7.95		
Hypopnea index	Axonal	11.30	5.88	0.063	0.804
	Demyelinating	9.09	5.37		
Oxygen desat. Index	Axonal	7.91	8.63	0.004	0.947
	Demyelinating	8.28	8.14		
Lowest O_2 saturation	Axonal	82.00	10.1	0.136	0.715
	Demyelinating	76.08	13.9		
O_2 saturation in REM	Axonal	94.96	3.06	0.121	0.731
	Demyelinating	94.68	3.21		
O_2 saturation in non REM	Axonal	95.27	3.01	0.032	0.858
	Demyelinating	94.51	3.50		

DISCUSSION

Neuromuscular diseases are well known to cause sleep related dysfunctions among which sleep-disordered breathing are the commonest disorders. A common complication and consequence of undiagnosed, untreated sleep disordered breathing is development of chronic respiratory failure. Thus it is of utmost importance to detect, diagnose and treat the sleep disorders and not just focus on the primary disease[7].

Recent experimental evidence documenting the effect of peripheral neuropathy on sleep pattern were conducted by Andersen and Tufik[11], who studied sleep patterns in a model induced peripheral neuropathy by inducing sciatic nerve constriction in rats and showed that chronic constrictive injury induced sleep alterations; such as reduced sleep efficiency and increased number of arousals, especially during the light period. Additionally, latency to the first paradoxical sleep episode was reduced in both light and dark period recordings particularly during the first week of the condition.

In our study we aimed to assess various patterns of sleep disturbances in polyneuropathy patients and compare their polysomnographic sleep measures with healthy age-matched control group.

Forty percent of our patients complained of sleep related symptoms; the sleep complains were mainly muscle cramps, numbness and tingling sensations in the lower limbs and leg restlessness interfering with sleep, some patients reported feeling that they did not get enough sleep at night and lack of concentration and drowsiness in the morning. This was similar to what had been reported in a large survey conducted by Gore and colleague[12], who recruited 265 patients suffering of diabetic

polyneuropathy. Patients in the study reported greater sleep problems compared to the general U.S. population; The mean quantity of sleep among study subjects was only 6 hours each night (SD = 1.5) and less than a quarter (23.1%) had optimal sleep (7 to 8 hours of sleep each night). More than half (51.8%) felt that their sleep was not quiet, 41.3% did not get enough sleep to feel rested upon waking, 53% felt drowsy during the day, 44.7% had trouble falling asleep, 44.3% reported awakening during the night and having trouble falling back to sleep.

The most frequent cause of sleep disturbance in our patients were sleep disordered breathing where, 63.5% of which were in the form of hypopneas, and 12.66% had obstructive sleep apnea mostly occurring during REM stage in 60% of patients.

Obstructive sleep apnea (OSA) is a highly prevalent disease characterized by recurrent episodes of upper airway obstruction that result in recurrent arousals and episodic oxyhemoglobin desaturations during sleep. Current understanding of the pathophysiologic basis of the disorder suggests that a balance of anatomically imposed mechanical loads and compensatory neuromuscular responses are important in maintaining upper airway patency during sleep. OSA develops in the presence of both elevated mechanical loads on the upper airway and defects in compensatory neuromuscular responses[13] thus the presence of polyneuropathy could cause or aggravate the condition.

In a study conducted over 23 patients with OSA having peripheral polyneuropathy Dziewas and colleague [14] studied both sural nerves sensory action potential amplitudes pre and post treatment of OSA, there were an increase of the sensory nerve action potential amplitudes by 2.6 mV on average ($p<0.001$) thus the impaired neural function is at least partly reversible with treatment for sleep apneas which points to the importance in detecting sleep apnea in peripheral nerve affection.

It is possible that polyneuropathy and obstructive sleep apnea can lead to each other. A case-control study was performed to evaluate the prevalence of polyneuropathy in obstructive sleep apneas. Out of 24 patients with OSA, 17 (71%) had clinical signs of polyneuropathy versus seven (33%) out of 21 matched controls. The mean amplitude of the sural sensory nerve action potential was smaller in the OSA group than in the control group, indicating axonal nerve damage. The differences were significant and could not be attributed to other known risk factors for polyneuropathy. The severity of axonal damage in patients with OSA correlated with the percentage of the night time with an O_2 saturation below 90%. It is assumed that recurrent intermittent hypoxemia in OSA is an independent risk factor for axonal damage of peripheral nerves.[15]

Moreover, Mayer et al.[16] examined peripheral nerve functions in 17 patients with severe obstructive sleep apnea. Sensory and mixed nerve potential amplitudes and sensory conduction velocity in the median nerve conduction were measured before, during, and after a 30-min period of ischemia. They were lower in OSA patients than in control subjects despite higher supramaximal stimulation. A resistance to ischemic nerve conduction failure (RICF) were detected in seven patients who had a lower mean nocturnal SaO_2 and a higher duration of $SaO_2 < 70\%$ than did other patients. Thus OSA patients had peripheral nerve dysfunction whose severity is partly related to the level of nocturnal hypoxemia. Abnormal preischemic nerve conduction studies suggested axonal lesions in those patients.

In our study no correlation between the severity of peripheral nerve dysfunction and the level of hypopnea were detected but 63.5% of the patients had hypopnea and axonal polyneuropathy patients had a higher hypopnea index (mean =11.3) than patients having demyelination polyneuropathy (mean = 9.1) though it did not reach statistical significance.

More than twenty three percent of our patients had central sleep apnea which was not correlated to the type of neuropathy. Central apnea is characterized by a lack of drive to breathe during sleep, resulting in repetitive periods of insufficient ventilation and compromised gas exchange.[17] During sleep, in normal individuals, alveolar hypoventilation occurs due to a combination of factors: reduction in tidal volume, slowing of metabolism (i.e. reduction of CO_2 production or O_2 consumption), absence of tonic influence from brainstem reticular activating system, decreased chemosensitivity and increased upper airway resistance to airflow. This may be attributed to hypotonia of upper airway dilating muscles and the intercostal muscles, and decreased output from sleep related medullary respiratory neurons, which may be exaggerated in peripheral neuropathy patients.[7]

About forty five percent of our patients had an increase in the leg movement index>15/hour movements were either periodic or nonperiodic which was not related to respiratory events.

Restless legs syndrome (RLS) is characterized by the desire to move the limbs associated with paresthesias of the legs, a motor restlessness, an intensification of symptoms at rest with relief by activity, and a worsening of symptoms in the evening or at night.[18] The prevalence of restless legs in the general adult population is high from 5% up to 10.6% and increasing with age[19], prevalence did not vary significantly by sex in some reports[20], and women were twice as often affected as men in other reports[18]. In our study no correlation could be detected between periodic leg movement index and age or sex of the patients.

The association between RLS and peripheral neuropathy remains controversial. One study[21] found that 15 of 41 patients with RLS had electrophysiologic evidence of polyneuropathy or radiculopathy, although only 7 of the 15 showed clinical signs of neuropathy. Another study[22] involving a consecutive series of patients with polyneuropathy, however, documented only a 5% frequency of RLS. Sural nerve biopsy findings in 7 of 8 patients with primary RLS were consistent with an axonal neuropathy [23] although there may be other explanations. It is not clear why some patients with peripheral neuropathy develop symptoms of RLS whereas others do not.

Polydefkis et al.[24] suggest that two forms of RLS exist; one is triggered by painful dysesthesias associated with small sensory fiber loss, has later onset, and no family history; and one without involvement of small sensory fiber loss, with an earlier onset age, positive family history for RLS, and no pain. In our study no significance was detected between axonal or demyelinating neuropathy regarding LMI.

Twenty five percent of our patient had periodic legs movements of sleep (PLM) which are rhythmic, standard and repetitive contractions of muscles of the extremities during the sleep and present in at least 80% of patients with RLS[19]. Although clinical data is important in detecting PLM they are not sufficiently predictive of its presence to rule in or rule out the diagnosis. Polysomnography is required for establishing the diagnosis of PLM.[25]

In our study PLM was associated with both axonal and demyelinating type of polyneuropathy However, Martinez-Mena and Pastor[26] found none of the nine studied cases of RLS revealed electrophysiological signs of neuropathy; though they have been able to demonstrate the existence of mild alteration of the peripheral nervous system, fundamentally of sensory character.

RLS may be primary or associated with neurological or medical conditions[19]. Wetter et al.[27] reported that PLM index were significantly higher in patients with uremic RLS compared with patients who had idiopathic RLS both groups did not differ in sleep architecture. In this study two out of five patients who had increase in the PLMI were uremic patients.

Sleep disturbances are common among individuals with diabetes. When compared with nondiabetics, patients with diabetes report higher rates of insomnia, excessive daytime sleepiness, and unpleasant sensations in the legs that disturb sleep.[28,29] Patients with diabetes, particularly those with peripheral neuropathy, have restless legs syndrome and periodic limb movements that can cause sleep-onset insomnia[30]. In addition to sleep-disordered breathing which may be improved by continuous positive airway pressure[31].

Our study included seven diabetic polyneuropathy patients (36%) they had increased percentage of sleep stage S1, and reduced percentage of both S2 and SWS, increased limb movement index, increased AHI and most of the events were hypopneas.

This was in accordance to Guilleminault et al.[32], who found obstructive sleep apneas in two and central sleep apneas in one of four type-1 diabetics. And Rees et al.[33] who described sleep apnea syndrome in three of eight diabetics.

Also, Bottini and colleagues[34] found respiratory disturbances in sleep including obstructive sleep apnea in patients with diabetic autonomic neuropathy.

The more recent study was conducted in 2007 by Yale Medical School. The experiment was focused on the correlation between diabetes II and obstructed breathing sleep disorders. 593 people were studied over a six year period. The conclusion was that people with sleep apnea were two and a

half times more likely to develop diabetes than people without sleep apnea.[35]

This study showed that hereditary polyneuropathy patients had the higher number of awakenings during sleep (14.33/hr) compared to other patients (11.14/h), and had a higher LMI and AHI compared to the control group $P<0.05$.

In a series of 44 consecutive patients with Charcot-Marie-Tooth disease (CMT), Gemignani et al.[36] found restless legs syndrome in 10 of 27 CMT type 2 patients (37%) and in none of 17 CMT type 1 patients. In the CMT2 patients, RLS was associated with positive sensory symptoms. In our study out of six patients having hereditary polyneuropathy only one had RLS. Also Akiba and colleagues[37] reported that two of five patients with Charcot Marie Tooth disease showed periodic drop in arterial oxyhemoglobin saturation in relation to episodes of central sleep apnea. Thus, the central sleep apneas are important features of nocturnal disturbed breathing in Charcot Marie Tooth patients.

In conclusion, many sleep abnormalities as well as changes in sleep architecture can occur in polyneuropathy patients particularly diabetic polyneuropathy and mostly during REM stage. And the majority of those patients have evident sleep disturbances even if they are not aware of it. Sleep disordered breathing and periodic limb movement disorder are the most common sleep abnormalities among polyneuropathy patients.

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الملخـص العربــى

الهدف من هذه الدراسة هو تقييم نمط النوم عند مرضى اعتلال الأعصاب الطرفية ومقارنته بالأصحاء من نفس النوع والسن. وقد أجريت الدراسة على مجموعة مرضى تضمنت 20 مريضا باعتلال الأعصاب الطرفية (15من الذكور و 5 من الإناث) ومجموعة ضابطة مكونة من 20 من الأصحاء من نفس النوع و السن وقد تراوحت أعمارهم بين 20 و 70 سنة.

وقد تم تقييم الحالات كالتالى:

- ❖ تقييما إكلينيكيا عن طريق أخذ التاريخ المرضى وتقييم النوم والفحص الإكلينيكى للجهاز العصبى.
- ❖ عمل التحاليل المعملية متضمنة تحليل بول كامل، قياس نسبة السكر فى الدم، صورة دم كاملة، نسبة الصوديوم والبوتاسيوم فى الدم، قياس وظائف الكبد والكلى.
- ❖ رسم العضلات الكهربائى وتوصيل الأعصاب.
- ❖ تقييم النوم باستخدام رسم المخ أثناء النوم في معمل النوم المتعدد.

وقد أظهرت النتائج ما يلى:

- أن عدد مرات الاستيقاظ أثناء النوم كان أعلى بفارق ذى دلالة إحصائية فى المرضى مقارنة بالأصحاء.
- أن هؤلاء المرضى يعانون من زيادة ملحوظة فى النسبة المئوية لمدة المرحلة الأولى للنوم مع انخفاض فى النسبة المئوية لكل من المرحلة الثانية و مرحلة نوم الدلتا من النوم بدون حركات العين السريعة مقارنة بالأصحاء.
- معدل إضطرابات النوم (كنقص أو توقف التنفس أثناء النوم، حركات القدم، حركات القدم الدورية واللاإرادية) كان أكثر فى المرضى مقارنة بالأصحاء بفارق ذى دلالة إحصائية عالية.
- بغض النظر عن سبب أو نوع اعتلال الأعصاب الطرفية، فإضطرابات التنفس التى تحدث أثناء النوم كانت أكثر فى مرحلة النوم ذا حركات العين السريعة عنها فى مراحل النوم بدون حركات العين السريعة.

ولقد استنتج من هذه الدراسة:

أن إضطرابات النوم فى مرض اعتلال الأعصاب الطرفية من الصفات الهامة لهذا المرض. معظمها إضطرابات التنفس التى تحدث أثناء النوم أو حركات القدم الدورية واللاإرادية.