

# Adult Monosymptomatic Nocturnal Enuresis with Obstructive Sleep Apnea Syndrome

Jian-Hua Chen<sup>1</sup>, Rong Huang<sup>2</sup>, Jin-Mei Luo<sup>2</sup>, Yi Xiao<sup>2</sup>, Ying Zhang<sup>3</sup>

<sup>1</sup>Department of Neurology, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences, Beijing 100730, China

<sup>2</sup>Department of Respiratory, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences, Beijing 100730, China

<sup>3</sup>Department of Neurology, Shangqiu First People's Hospital, Shangqiu, Henan 476100, China

To the Editor: Obstructive sleep apnea syndrome (OSAS) is caused by recurrent episodes of complete or partial collapse of the upper airways during sleep and can induce apnea or hypopnea. Recurrent episodes of intermittent hypoxia and higher carbon dioxide levels may lead to frequent interruption of sleep.<sup>[1]</sup> Nocturnal enuresis (NE) is characterized by the involuntary and recurrent voiding of urine during sleep. NE is a symptom of childhood OSAS, while it is rare in adults. Monosymptomatic NE (MNE) is defined as NE without daytime symptoms. We report an adult woman with MNE associated with severe OSAS whose symptoms completely disappeared after nasal continuous positive airway pressure (CPAP) therapy.

A 48-year-old woman presented to the neurology department in November 2014 with a 6-month history of NE. Six months previously, she experienced bed wetting two to three times every night. Enuresis never occurred during the daytime. Her past medical history was normal. She had not received any sedative medication. She did not smoke or drink and had no depression or anxiety. She had snored during sleep for several years.

Her blood pressure was 138/85 mmHg and body mass index was 35.9 kg/m<sup>2</sup>. Blood tests showed normal leukocyte and platelet counts. Hemoglobin, liver and renal function, erythrocyte sedimentation, and thyroid gland function were within normal ranges. Routine urine examination was normal. Head and spinal cord magnetic resonance imaging and head magnetic resonance angiography showed no abnormalities. Carotid artery, abdominal, pelvic cavity, and urinary system ultrasound were normal. Multislice spiral computed tomography urography was done with a normal result. Video electroencephalography (EEG) did not reveal any evidence of epileptiform potentials or persistent delta activity. Overnight recording polysomnography was recommended.

Sleep stages were scored for 30 s intervals according to international standard criteria. Apnea was considered when there was a drop in the flow excursion by  $\geq 90\%$  of the baseline for at least 10 s. Hypopnea was defined when there was a drop by  $\geq 30\%$  of baseline and associated with  $\geq 4\%$  desaturation from pre-event baseline for at least 10 s. Another alternative definition for hypopnea is a drop in the flow excursion by  $\geq 50\%$  of baseline for at least

10 s, associated with  $\geq 3\%$  desaturation from pre-event baseline and/or arousal. Microarousal was scored according to the criteria of cortical EEG arousal  $>3$  s and  $<15$  s; awake was defined as a cortical EEG arousal  $\geq 15$  s.

Polysomnography showed that sleep efficiency was 95.5% and rapid eye movement (REM) latency was 300 min. Sleep stage distributions were N1 8.6%, N2 78.7%, and REM 8.2% with absent slow-wave sleep. The apnea-hypopnea index was 109.6 events/h with the longest apnea time 61.3 s and average apnea time 23.5 s. The overall apnea index was significantly higher during non-REM (103.2 events/h) compared with REM (74.4 events/h) sleep. Mean oxygen saturation was 85.5% and minimal oxygen saturation was 52%. Sleep was fragmented with a total arousal index of 27 events/h. Repeated oxygen desaturations, single snoring, brady- and tachyarrhythmias, and arousals were associated with apneic episodes.

Adult MNE secondary to OSAS was diagnosed. The patient received nasal CPAP treatment with a pressure titrating to 13 cmH<sub>2</sub>O. We followed her for 1 year. NE disappeared during the first night of CPAP therapy, and it has been completely resolved since then.

Few data are available in the literature regarding sleep structures in NE secondary to OSAS. We systematically reviewed the English literature in PubMed up to December 2015. Ten articles with 12 cases were retrieved. Only one case provided percentages of sleep stages. None provided microsleep structure or arousals [Table 1].

In the present case, we found marked prolonged Stage 2 sleep and significant reduction of REM sleep with absent slow-wave sleep, which differed from previously published data showing a marked reduction of Stage 2 and slow-wave sleep. Our findings

**Address for correspondence:** Dr. Rong Huang,

Department of Respiratory, Peking Union Medical College Hospital,  
Chinese Academy of Medical Sciences, Beijing 100730, China

E-Mail: huangrong212@163.com

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**Table 1: Polysomnography in cases with obstructive sleep apnea syndrome with enuresis in literature review**

First author	Number of patients	Gender/age (years)	Published journal year	BMI (kg/m <sup>2</sup> )	Polysomnography parameters						Therapy
					AHI (events/h)	Minimal SaO <sub>2</sub>	Stage N1	Stage N2	Stage N3	Stage REM	
Guilleminault	3	N/R/N/R	Annu Rev Med 1976;27:465-84	N/R	N/R	N/R	N/R	↑	↓↓	Generally normal	N/R
Guilleminault	2	Male/N/R	Arch Intern Med 1977;137:296-300	N/R	N/R	N/R	N/R	N/R	N/R	N/R	N/R
Everaert	1	Male/35	J Urol 1995;153:1236	N/R	36	N/R	N/R	N/R	N/R	N/R	CPAP
Brown	1	Male/26	West J Med 1995;163:478-80	37.33	48.1	46%	N/R	N/R	N/R	N/R	CPAP
Yokoyama	1	Male/53	Urology 1995;45:150-4	34.63	N/R	36%	N/R	↓↓	↓↓	N/R	Imipramine and acetazolamide
Ulfberg	1	Male/68	Scand J Urol Nephrol 1996;30:135-7	N/R	N/R	30%	N/R	N/R	N/R	N/R	CPAP
Arai	1	Female/53	Sleep 1997;20:158-9	34.63	52.6	35%	41.5%	42%	2.5%	14%	Imipramine and dental appliance
Steers	1	Male/60	Lancet 1997;349:1604	36.95	47.5	87.5%	N/R	N/R	N/R	N/R	CPAP
Kramer	5	Male/37.0 ± 3.1	Chest 1998;114:634-7	38.6 ± 2.1	107.4 ± 7.7	73.2 ± 2.7	N/R	N/R	N/R	N/R	CPAP
Arai	1	Female/53	Psychiatry Clin Neurosci 1999;53:319-20	34.63	52.6	35%	N/R	42%	2.5%	14%	Imipramine and acetazolamide
Cao	1	Female/56	Chin Med J 2010;123:3328-61	29.55	70.24	68%	N/R	N/R	N/R	N/R	CPAP
Dushianthan	1	Male/41	Br J Hosp Med (Lond) 2010;71:532-3	32	80.6	N/R	N/R	N/R	N/R	N/R	CPAP

N/R: Not reported; ↑: Elevated; ↓↓: Markedly reduced; REM: Rapid eye movement; BMI: Body mass index; AHI: Apnea-hypopnea index; CPAP: Continuous positive airway pressure.

were coincident with those of Iriarte *et al.* who found that OSAS was accompanied by increased duration of Stages N1 and N2 and decreased duration of Stages N3 sleep.<sup>[2]</sup> Absent of slow-wave sleep and significantly decreased REM sleep with high arousal indices demonstrated the patient's poor sleep quality.

OSAS patients are characterized by a derangement in autonomic cardiovascular regulation.<sup>[3]</sup> Sleep apnea patients often have nocturia, possibly because of increased plasma and urine levels of atrial natriuretic peptide, decreased antidiuretic hormone, and no normal decrease in nocturnal urinary output leading to increased natriuresis and diuresis.<sup>[4]</sup> An increase in intra-abdominal pressure caused by intense inspiratory efforts against a closed upper airway has been implicated in the pathogenesis of enuresis.<sup>[5]</sup>

NE could be a significant problem for the patient. When we encounter obese patients with MNE, especially with a history of habitual snoring, OSAS should be considered. CPAP is considered to be the gold standard for treatment.

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### Conflicts of interest

There are no conflicts of interest.

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