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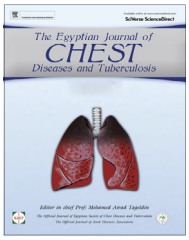

ORIGINAL ARTICLE

# Effect of adenotonsillectomy on nocturnal enuresis in children with OSA

**Helmy A. Heba [a,*], M. Elnabil Lobna [b], Eldemery M.B. Ahmed [a], Shehata Wael [a], Kotb A. Ashraf [a]**

[a] *Faculty of Medicine, October 6 University, Egypt*
[b] *Faculty of Medicine, Ain Shams University, Egypt*

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**KEYWORDS**

OSA;
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ADH;
Urine volume

**Abstract** *Background:* Pediatric obstructive sleep apnea (OSA) is the most severe form of SDB. The most important risk factors for the development of pediatric SDB include adenotonsillar hypertrophy. On the other hand, nocturnal enuresis is common symptom that is associated with (OSA), through influencing the nocturnal secretion of antidiuretic hormone (ADH).

*Objective:* We aimed to study the relation between polysomnography data of children with OSA due to adenotonsillar hypertrophy and levels of ADH, total and night urine volume.

*Subject and method:* The study included 23 Consecutive non obese children with age above 5 years old, complaining of nocturnal enuresis, hypertrophied adenoids and tonsils. A polysomnographic evaluation was done together with serum level of ADH, total and nocturnal urine volume were measured pre and 3 months post adenotonsillectomy.

*Results:* There was a significant negative correlation between ADH and different polysomnographic data including respiratory events (apnea index, total hypopneas, hypopnea index and RDI), oxygen saturation data and the snoring index. There was also a significant negative correlation between night urine volume and desaturation index. comparison between the data means before and after adenoidectomy were done using the paired *t*-test; showed a significant improvement of all the polysomnographic data values, and a significant improvement in serum level of ADH, besides the significant decrease in both the 24 h urine volume and the night urine volume being.

* Corresponding author. Mobile: +20 01222294860.
E-mail address: hebahelmy73@yahoo.com (H.A. Heba).
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*Conclusion:* Children with OSA and nocturnal enuresis, should be considered for early adenotonsillectomy or other treatments to approve normal release of ADH, and subsequent improvement of nocturnal enuresis.



## Introduction

OSA has been defined by the American Thoracic Society [1] is a common but underestimated disease that has been related to increased different morbidity. Pediatric obstructive sleep apnea (OSA) is the most severe form of SDB [2] and has a prevalence of 1–3% in otherwise healthy children [3]. The most important risk factors for the development of pediatric OSA include adenotonsillar hypertrophy, obesity, craniofacial anomalies and neuromuscular disorders [4–8]. There is evidence of an interaction between nasal obstruction and pharyngeal narrowing in the pathophysiology of OSA, OSA leads to sleep fragmentation, which develops as a consequence of repeated arousals, together with intermittent blood gas abnormalities (hypoxia and hypercarbia) that characterize OSA and the unpreserved integrity of both rapid eye movement (REM) sleep and non-REM sleep [9–12].

All these pathological changes may jointly lead to a wide array of morbid consequences, include reduced intelligence and memory, behavioral, deficits, hyperactivity like, aggressiveness, as well as failure to thrive, enuresis and cardiovascular dysfunction [9,12].

As regards nocturnal enuresis, is a bothersome and common symptom in children with OSA [13] through different proposed factors including: decreased arousal response, impaired urodynamics, increased intra-abdominal pressure during obstructive respiratory events increases bladder pressure and altered secretion of hormones that regulate fluid balance as anti-diuretic hormone whose inappropriate levels are 2.7 times more likely to occur in children complain of bedwetting issues [14].

Previous studies discussed the improvement or reversibility of these morbidities by the adequate treatment of OSA based on etiological factors and the further associated improvement of the overall quality of life [15] and reduced healthcare costs [16].

Based on aforementioned considerations, we hypothesized that children who are referred for symptoms of OSA and sleep fragmentation with nocturnal desaturation are more likely to develop nocturnal enuresis due to inappropriate anti-diuretic hormone (ADH) secretion and would be reversible after adenotonsillectomy.

## Subjects and Methods

The study included 23 consecutive non obese children with age above 5 years old, all participants complain of nocturnal enuresis which was considered to be present when responses were in the frequent grade (3–6 times per month) or almost always grade ( > 3 times per week) [17].

The patients were subjected to:

### *Clinical evaluation*

(a) Detailed medical history taking included duration of illness and current therapy.
(b) Thorough clinical and neurological examination to rule out an organic etiology, exclusion criteria were the presence of familial craniofacial or genetic disorders, cerebral palsy, neuromuscular diseases, or any underlying systemic diseases or acute infectious processes.
(c) Anthropometrical parameters were assessed; weight, height, BMI (defined as weight in kilograms divided by height in meters squared) [18], and circumference of the neck measured at the level of the cricothyroid membrane (with the patient in the orthostatic position).
(d) Standardized sleep questionnaire [19].
(e) Ear, nose, and throat (E.N.T) assessment were done by ENT surgeon to demonstrate obvious hypertrophied adenoids and tonsils clinically and confirmed by nasopharyngeal CT scan.

### *Polysomnography*

Polysomnographic (PSG) overnight study was done for the participants enrolled in the study and was studied twice, initially and 3 months postoperatively after undergoing curative adenotonsillectomy. All PSG studies were recorded at sleep laboratory of the neurology department, Ain Shams University Hospital. All patients arrived to the sleep lab, about 2 h before their usual bedtime, which is sufficient for electrode application. No drugs were used to induce sleep. The recordings included four EEG channels (C4-O1, C3-O2, A1-C3, and A1-C4), applied according to the international 10–20 systems of electrode placement, two electro oculogram channels, one chin and one tibialis anterior electromyogram channels, nasal airflow, ventilatory belts, electrocardiogram, oxygen saturation (through pulse oximetry), body position and snoring assessment. Only PSGs $\geqslant$6 h of sleep were considered adequate for testing the sleep pattern. Sleep stage scoring was done visually according to standard criteria of Rechtschaffen & Kales [20], with 30-epochs. Respiratory parameters analysis was done automatically and confirmed manually. An apnea was defined as a cessation of naso-oral airflow lasting $\geqslant$10 s. A hypopnea was scored when there was an obvious reduction usually $\geqslant$20% in airflow and/or effort, lasting $\geqslant$10 s and accompanied by an arousal or a fall in $SaO_2$ of $\geqslant$4%. Obstructive apnea and hypopnea were scored when air flow was absent but respiratory efforts were present. Central apnea and hypopnea were scored in absence of respiratory effort and