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Obstructive sleep apnea and asthma

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Abstract

Both asthma and obstructive sleep apnea (OSA) are common conditions involving the adults and children population, and have significant impact on the healthcare system. For the last few decades a lot of data emerged in terms of the prevalence between asthma and OSA. The prevalence ranges from 38% up to as high as 70%. Based on the current concepts of bidirectional relationship of OSA and asthma, it is sensible to assume that treating one disorder will result in the other's better control and vice versa. This review will look into the pathogenesis of concomitant OSA and asthma and whether the first line OSA therapy will result in better control of asthma in patients with concomitant OSA. There is growing evidence that continuous positive airway pressure (CPAP) in adults and adenotonsillectomy in children which are recommended as first line treatment of OSA can improve their asthma symptoms. However, further confirmation is necessary with larger randomized control trials to further evaluate both conditions and the treatment effects.

Keywords: obstructive sleep apnea, asthma, continuous positive airway pressure, adenotonsillectomy

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Introduction

Global Initiative for Asthma (GINA) defined asthma as heterogeneous disease that is characterized by chronic airway inflammation.[1] The typical symptoms of asthma include wheezing, breathlessness, chest tightness and coughing that vary over time and intensity, together with variable airflow limitation.[1]

It is estimated that about 60 % of asthma is heritable.[2] For example in a recent genome wide association studies, the *ORMDL3/GSDMD* locus on chromosome 17q21 has been reproducibly associated with childhood onset asthma.[3] Other genes, including *IL33* on chromosome 9 and *IL2RB* on chromosome 22, have been variably implicated.[3]

The chronic inflammation of asthma is characterized by infiltration of mast cells, eosinophils and T-helper cell type 2 (Th2) $CD4^+$ T-lymphocytes in the airway wall.[4,5] Inflammatory mediators secreted by these cells are the effectors of the chronic inflammation which results in bronchial hyper-reactivity, mucous production and airway remodeling.[4-6]

Asthma affects more than 330 million people worldwide, with more than 14 % of the world's children and 8.6 % of young adults (aged 18 to 45 years old) experience asthma symptoms.[7] It is more pronounced in parts of Asia, with 107 million sufferers in Southeast Asia and Western Pacific Region.[8] Recent review demonstrated that house dust mite appeared to be strongly associated with asthma in atopic Asians, especially children.[9] The prevalence of asthma in children from many regions of Thailand varies from 6.8-11.9%.[10] The prevalence in Thai adult population is reported to be as high as 12.1% in one study.[11]

Reported risk factors for asthma includes genetic predisposition, family history of atopy, allergic sensitization, caesarean section, tobacco smoke, severe respiratory virus infection, diet, obesity, air pollution and occupational exposures.[2,11,12] Although patients can be subdivided according to several clinical, physiologic, radiographic, and pathologic variables, multiple analyses suggest that adult patients are likely to fall into one of five clusters also known as "asthma phenotypes".[1,2] These phenotypes includes allergic asthma, non-allergic asthma, late onset asthma, asthma with fixed airflow limitation and asthma with obesity.[1]

Obstructive sleep apnea (OSA) is characterized by recurrent collapse of the upper airway during sleep, resulting in substantially reduced or complete cessation of airflow despite ongoing breathing efforts. This leads to intermittent

disturbances in gas exchange and fragmented sleep.[13] The standard diagnostic test for OSA is an overnight polysomnogram (PSG). This study involves several measured physiologic recordings such as electroencephalogram, electrooculogram, electrocardiogram, chin and leg electromyograms, body position, finger pulse oximetry, measurements of airflow, and measurements of thoracic and abdominal respiratory effort.[13] The disease severity is measured using the apnea-hypopnea index (AHI), i.e., the mean number of apneas and hypopneas per hour of sleep. Moreover, respiratory effort-related arousal (RERA) indexes during sleep in combination with apnea-hypopnea index are used to estimate the respiratory disturbance index (RDI).[13]

The diagnosis of OSA requires an AHI or RDI ≥ 5/ hour either with the presence of signs and symptoms of OSA or associated medical or psychiatric disorders such as hypertension, coronary artery disease, atrial fibrillation, stroke, and mood disorders.[13] Alternatively, AHI or RDI ≥ 15/ hour also satisfies the criteria, in the absence of symptoms or co-morbidities.[13] In children, signs and symptoms of OSA (for example snoring, labored/obstructed breathing or sleepiness) are consolidated into one criterion with a polysomnogram evidence of either AHI ≥ 1/ hour of sleep or obstructive hypoventilation coupled with snoring, paradoxical thoracoabdominal movement, or flattening of the nasal airway pressure waveform.[13,14]

The prevalence of OSA defined as an apnea-hypopnea index (AHI) ≥5 was a mean of 22% (range, 9-37%) in men and 17% (range, 4-50%) in women reported in a recent systemic review.[15] The prevalence of OSA in adults in Asia was reported to be 3.7% to 97.3% in another systemic review.[16] The prevalence in Thailand was reported to be 15.4% in men and 6.3% in women.[17] In children, OSA affects about 1.2% to 5.7%, with the peak prevalence between 2 to 8 years old.[18,19] This coincides with the peak age of tonsillar and adenoidal hypertrophy in children.[18] Risk factors for OSA include male gender, age, obesity, smoking, alcohol use, positive family history, increase in neck size (> 17 inches in males and 16 inches in females), craniofacial abnormalities (i.e. micrognathia or retrognathia), narrowing of the upper airway, nasal obstruction, endocrine disorders (i.e hypothyroidism, acromegaly), neurological disorder (i.e. neuromuscular disorders, stroke) and cardiovascular disorder (i.e. hypertension, atrial fibrillation).[15,20] Recently numerous studies had been conducted to explore the relationship of asthma and OSA in terms of epidemiology, pathophysiology or whether the treatment of one disease affect the control of another. This review will delve into the acknowledged connections and the latest diagnosis and therapies.

Epidemiology of asthma and OSA

For the last few decades a lot of data emerged in terms of the prevalence between asthma and OSA. In a Canadian case-control study, they looked into the prevalence and severity of OSA on overnight polysomnography among severe asthmatic group compared with the moderate asthmatic and non-asthmatic control groups.[21] They recruited 26 patients in each group with similar age and body mass index that underwent complete home polysomnography.[21] The prevalence of OSA (defined as AHI ≥15/hour) was 88% in the severe asthma group, 58% in the moderate asthma group, and 31% in the control group ($p<0.01$).[21]

On another note, Alharbi et al tried to look at a reverse relationship between the prevalence of asthma in patients with confirmed diagnosis of OSA.[22] Six-hundred-and-six patients with OSA with a mean age of 40±14.5 years (66.7% males) were included. Asthma was present in 213 OSA patients with a prevalence of 35.1%.[22] In that study, body mass index (>35 kg/ m^2) were the only predictor of asthma.[22]

Guven et al conducted another study in a tertiary care hospital in Turkey that evaluated the presence of OSA in difficult to treat asthma patients.[23] They recruited 47 difficult to treat asthma patients and all of them underwent an overnight polysomnography.[23] In this study, they found that 74.5% (n=35) of the difficult to treat asthma patients had OSA, in which 11 had mild OSA and 24 had moderate to severe OSA.[23]

A recently published retrospective study in Portugal also found similar increase in prevalence of OSA in asthmatic patients.[24] They conducted the study on 47 patients in an outpatient clinic diagnosed to have asthma which subsequently underwent polysomnography (68%) and cardiorespiratory polygraphy (32%).[24] The prevalence of OSA was 57.4%, with 73.3% of them were males.[24]

Most of the studies looking at the prevalence of asthma and OSA were done in adult population. Recently a large multicentric cross sectional study was done in China to investigate the prevalence of asthma and sleep-disordered breathing (SDB) among school-aged children in China.[25] They demonstrated that OSA was significantly associated with asthma after adjusting for confounding factors with a odds ratio (OR) of 1.92 (95% confidence interval: 1.35-1.8).[25] The limitation of this study was that diagnosis of OSA was questionnaire-based as opposed to the "gold-standard" of polysomnography and hence was subjected to measurement bias.

On the whole, there was significantly increase in prevalence OSA in the children and adult population with asthma especially in the severe group. These observations suggested that there might be potential pathophysiologic interactions between asthma and OSA, and warrant further investigations with larger cohorts and to evaluate the clinical implications.

Pathogenesis of asthma and OSA

Various studies had tried to examine the bidirectional interactions of asthma and OSA and these interactions can be classified as direct or indirect effect (**Figure 1**).

Figure 1A : Direct effects in bidirectional interaction

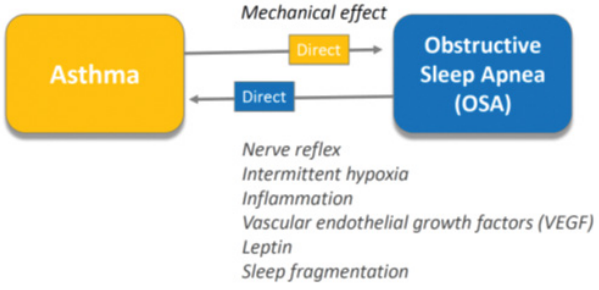

Figure 1B : Indirect effects in bidirectional interaction

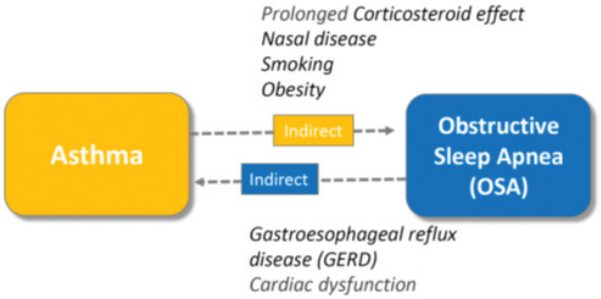

Figure 1 A and B: The bidirectional interactions (overlap) between OSA and asthma.

Effect of Asthma on OSA

Direct effect

a) Mechanical effect

Physiological studies supported the idea that there might be a nocturnal increase in airway resistance in asthmatic as a result of reduction in functional residual capacity and end expiratory lung volume during sleep, especially during REM sleep.[26] This in turn resulted in more upper airway collapse and caused worsening symptoms of snoring and apnea in OSA patients.[26]

Indirect effect

a) Corticosteroid effect

Cortiscosteroid, whether it is inhale or oral, remains the mainstay therapy for asthma control.[1] Yigla et al reported high prevalence of OSA among patients with unstable asthma receiving long-term chronic or frequent burst of oral corticosteroid therapy. The prevalence rate was 95%, and the author concluded that prolonged and especially continuous oral corticosteroid therapy in asthma increases airway collapsibility.[27] A more recent data by Teodorescu et al also did not refute such effects of corticosteroid medications on the upper airway, though the number of patients was slightly smaller.[28] The proposed mechanism of corticosteroid affecting the airway include: inhaled corticosteroids cause fat deposition in and around the upper airway, narrowing the airway cross-section area; glucocorticoids can induce myopathy of the airway dilator muscles, which influences the airway dilation; and glucocorticoids can worsen obesity.[26]

b) Nasal disease

Majority asthmatics have a higher rate of allergic or non-allergic rhinitis and nasal polyposis.[29] Increased nasal resistance results in higher negative oropharyngeal pressure during inspiration and predisposes to airway collapse.[26] In a recent systemic review by Chirakalwasan and Ruxrungtham, showed that nasal congestion due to allergy was associated with an 1.8-fold increase in the risk of developing moderate to severe OSA, although, the degree of nasal obstruction does not directly correlate with the severity of sleep-disordered breathing.[30]

c) Smoking

Smoking is known as an independent risk factor for both asthma and OSA.[26] It induces airway edema, thereby increasing the airway resistance and worsening airway obstruction. The added inflammation could thus increase the upper airway resistance and collapsibility during sleep, with worsening sleep apnea.[26]

d) Obesity

Obesity is considered one of the causal factors for OSA. Peppard et al found that the increase in weight was positively correlated with the AHI; that is, patients who gain 10% of their body weight tend to show an increase of approximately 32% in the AHI, and a 10% reduction in weight resulted in a 26% reduction in the AHI.[31] A 10% increase in body weight increased the chance of developing moderate to severe OSAS by 6 times.[31] Obesity is also a significant risk predictor of asthma. Increasing weight causes more frequent or more severe asthma exacerbations and more difficult-to-control symptoms.[32]

Effect of OSA on asthma

Direct effect

a) Nerve reflex

Studies had shown that repeated snoring could cause damage to the soft tissue surrounding the upper airway and nasal passage due to its vibrating frequencies resulting in airway inflammation.[33] On top of the mechanical trauma, the increase in vagal tone during the apneic episodes in OSA (Muller's maneuvers) would trigger the muscarinic receptors in the central airways and resulting in bronchoconstriction and nocturnal asthma attacks.[26,33]

b) Intermittent hypoxia

In OSA, repeated episodes of partial or complete upper airway obstruction during sleep would lead to intermittent hypoxia and reoxygenation. Subsequently, this would cause complex oxidative stress cascade downstream, inflammation, sympathetic tone surcharges and endothelial dysfunction, resulting in bronchoconstriction.[26] Another postulated trigger for reflex bronchoconstriction is stimulation of the carotid body by the hypoxia that results from obstructive apneas.[34]

c) Inflammation

Studies had shown that OSA could lead to both local and systemic inflammation. As mentioned previously, local inflammation might be due to mechanical stress on the mucosa by snoring.[33] The systemic inflammation that exists in OSA is characterized by the elevation of serum pro-inflammatory cytokines and chemokines such as TNF-α, C- reactive protein (CRP), and interleukin-6 (IL-6) that had been seen in patients with OSA.[33] A recent meta-analyses by Nadeem et al also concurred the same findings and concluded that those with OSAS, on average, had higher levels of systemic inflammatory markers than healthy controls.[35] When the overwhelming inflammation involved the lower airway, it could predispose to asthma and increased the risk of acute or sudden-onset fatal asthma exacerbations.[6]

d) Vascular endothelial growth factors (VEGF)

Some scholars proposed that vascular endothelial growth factors might play a role in the pathogenesis of both asthma and OSA.[26] VEGF is a hypoxia-sensitive glycoprotein, and OSA and asthma can promote its expression.[34] VEGF may contribute to bronchial inflammation, hyper-responsiveness, and vascular remodeling in those patients.[34] Although the relationship between the elevated VEGF levels in OSA and asthma is conceivable, no conclusive data available currently.[26]

e) Leptin

Leptin is a protein produced by adipose tissue that circulates systemically and acts on the hypothalamus to induce satiety and increase metabolism.[34] It has been known to be elevated in OSA patients.[26] Furthermore, evidence of local leptin production in the respiratory compartment, supports the concept that leptin plays an important role in respiration, lung development and the pathogenesis of diverse respiratory diseases.[36] Coupled with the increased levels of serum leptin observed in OSA, the proinflammatory effects of leptin suggest that this hormone might be relevant to asthma exacerbations in OSA.[26] This was supported by the previous evidence that leptin might contribute to airway hyper-responsiveness in a study by Sideleva et al.[37]

f) Sleep fragmentation

It is also postulated by various authors that disturbance in the sleep architecture itself may contribute to the bidirectional interactions of OSA and asthma.[26] Sleep fragmentation and frequent arousals, which are features of OSA, may potentially causes increasing airway resistance and blunting the arousal response to bronchoconstriction.[26] Furthermore, early studies of sleep-disordered breathing demonstrated that patients with asthma were breathing more irregularly (with hypopnea, apnea, and hyperpnea) in REM sleep than those without asthma.[38] This may be related to the increased cholinergic outflow that occurs during REM sleep, which in turn modulates the caliber and reactivity of the lower airways.[38]

Indirect effect

a) Gastroesophageal reflux disease (GERD)

Few authors reported that prevalence of GERD was associated between 58% to 62% in patients with OSA.[26] It is believed that the significant increase in negative intrathoracic pressure caused by upper airway obstruction can predispose to retrograde movement of gastric contents.[39] On the other hand, GERD may induce asthma directly by microaspiration, with respiratory mucosal injury by gastric (acid and pepsin) or duodenal (bile acids and trypsin) contents, and indirectly, via vagally mediated mechanisms or reflex bronchospasm.[26,40] Hence, it is thought that OSA-induced acid reflux may play a role in triggering asthma symptoms.[40] However the association between the three conditions remains complex.[26]

b) Cardiac dysfunction

Various studies had shown the association between OSA and cardiac dysfunction.[41] Since sleep architecture is altered in OSA, the heart loses its normal relaxed function during sleep. The intermittent hypoxia might heightened the sympathetic activity and negative intrathoracic pressure lead to the intermittent increasing after load of left ventricular, causing or worsening heart failure.[26] Congestive heart failure itself has been known to worsen the asthma control by inducing airway hyper-responsiveness.[26,34]

Therapeutic implications

Based on the current concepts of bidirectional relationship of OSA and asthma, it is sensible to assume that treating one disorder will result in the other's better control and vice versa. Sullivan et al described the use of continuous positive airway pressure (CPAP) as a treatment for OSA in the 1980s.[42] It uses pressure to provide a pneumatic splint to maintain airway patency and causes major reduction in respiratory events and their related consequences during sleep.[43] Thus CPAP eliminates or reduces the chronic intermittent hypoxia and sleep fragmentation observed during apneic events.[43] Since then, CPAP had been accepted as the first line treatment for OSA. On the other hand, the role of CPAP in asthma is less clearly defined and at times it is deemed controversial.

For the last decade several clinical studies were done to look into the impact of CPAP on asthmatic patients (**Table 1**).

Table 1. Studies investigating the use of continuous positive airway pressure in treatment of adult asthma and OSA.

First author, year of publication	Study population (N), duration	Primary outcome	Results
Kauppi et al, 2016[44]	OSA patients with asthma (n=153), 3 months	- Severity of asthma (before and after using CPAP) measured by VAS score and ACT score	- Positive findings - VAS scale decreased from 50.8 to 33.6 in women (p<0.001) and 46.8 to 32.9 in men (p<0.001) - ACT score increased from 15.3 to 19.8 in women (p<0.001) and from 17.2 to 19.1 in men (p<0.001)
Lafond et al, 2007[45]	Stable asthma patients with new OSA (n=20), 6 weeks	- QoL questionnaires for asthma and OSA (pre/post CPAP therapy) - Provocative concentration of methacholine causing a 20% fall in forced expiratory volume in one second (FEV1) ≤8 mg/mL (PC_{20})	- Improved only QoL but not bronchial hyperresponsiveness - PC_{20} 2.5 mg/mL (1.4–4.5) compared with baseline PC_{20} 2.2 mg/mL (1.3–3.5) (p=0.3) - QoL improved from 5.0 ±1.2 at baseline to 5.8± 0.9 (p<0.001)
Ciftci et al, 2005[46]	Asthma patients with nocturnal symptoms and with moderate to severe OSA (n=16), 2 months	- Nighttime symptoms based on GINA guidelines - Pulmonary function testing: FEV_1, FVC, FEV1/FVC	- Improved nighttime symptom scores but not in PFT changes - No change in PFT parameters - Nighttime symptom scores from 2.19±1.07 (baseline) to 1.44±1.15 (after CPAP) (p=0.04)

First author, year of publication	Study population (N), duration	Primary outcome	Results
Guilleminault et al, 1988[47]	- 2 groups of patients with frequent nocturnal asthma attacks - Group A = obese adults with mean AHI of 51/hour (n=10) - Group B= adolescents with mean AHI of 8/hour (n=8) - Total 16 patients had CPAP for 4 to 6 months - 2 adolescent patients had surgical interventions	- Frequency of asthma attacks in days	- Reduction in asthma attack in days. - Group A : fewer nocturnal asthma attacks from an average of 1 severe nocturnal asthma attack every 17 days to 0 - Group B : 1 attack every 15 days to 0
Chan et al, 1988[48]	- asthmatic patients with frequent nocturnal attacks (n=9), 2 weeks	- PEFR monitoring (pre and post bronchodilators)	- Improved mean PEFR - Mean (SD) PEFR, expressed as a percentage of the predicted value: from 40 (4) to 50 (4) (p<0.01) prebronchodilator and from 60 (7) to 70 (8)(p<0.001) postbronchodilator

Abbreviation: ACT = asthma control test, AHI=apnea hypopnea index, CPAP = continuous positive airway pressure, FEV_1=forced expiratory volume in 1 second, FVC=forced vital capacity, PEFR=peak expiratory flow rates, PSG = polysomnography, QoL = quality of life, VAS = visual analogue scale

Most recent study by Kauppi et al, used a survey questionnaire to their OSA patients on CPAP. In that study, asthma was defined as self-reported physician diagnosed and on asthma medication, and CPAP was started after the diagnosis was made. The prevalence of asthma among CPAP users was found to be 13%.[44] Self-reported asthma severity decreased significantly from 48.3 (29.6) to 33.1 (27.4) (p< 0.001), and ACT score increased significantly from 15.35 (5.3) to 19.8 (4.6) (p<0.001) without a significant change in the body mass index (BMI).[44] Furthermore, the percentage of patients using rescue medication daily reduced from 36 to 8% with CPAP (p< 0.001).[44]

Another study by Lafond et al, examined the impact of CPAP treatment on the airway responsiveness of asthmatic subjects with OSA. Twenty patients with stable asthma and newly diagnosed OSA by polysomnography were recruited. They had a baseline questionnaire on the quality of life (QoL) and underwent three serial methacholine inhalation challenge prior the recruitment. With the nocturnal CPAP treatment, the AHI dropped from 48.1±23.6/hour to 2.6±2.5/hour (p<0.001). However, there were no significant changes in airway responsiveness after CPAP treatment (provocative concentration causing a 20% fall in forced expiratory volume in one second, FEV1) ; [PC_{20} 2.5 mg/mL (1.4–4.5)] compared with baseline [PC_{20} 2.2 mg/mL (1.3–3.5)] (p=0.3). Intriguingly, the asthma quality of life of the subjects improved significantly from 5.0±1.2 at baseline, to 5.8±0.9 at the end of the study (p<0.001).

An earlier study by Ciftci et al, reported that asthmatic patients with nocturnal symptoms who were diagnosed to have OSA and were given 2 months of CPAP therapy did not have significant improvement in their pulmonary function testing.[46] Conversely, their nocturnal asthma symptoms (nighttime symptoms were quantified as a score according to the frequency of symptoms based on GINA guidelines) improved significantly from a mean (SD) of 2.19 (1.07) to 1.44 (1.15) (p<0.05) after CPAP therapy.[46]

Smaller studies in the past by Guilleminault et al[47] and Chan et al,[48] both investigated asthmatic patients with nocturnal symptoms with concomitant OSA and were treated with CPAP. In the former study, asthmatic patients with nocturnal symptoms were divided into 2 groups; Group A consisted of 10 adults with mean AHI of 51/hour and Group B consisted of 5 adolescents with AHI of 8/hour. Both groups were given CPAP for 4 to 6 months except for 2 adolescent patients that had surgical intervention such as adenotonsillectomy and uvulectomy. In both groups there were significant reduction in terms of nocturnal asthma attacks.[47] In the latter study, 8 adult patients with concomitant asthma and OSA (confirmed by polysomnography), were subjected to use CPAP for 2 weeks. There was marked improvement in mean peak expiratory flow rates, expressed as a percentage of the predicted value from 40 to 50 (p<0.01) prebronchodilator, and from 60 to 70 (p< 0.001) postbronchodilator.[48]

In contrast to adult OSA patients, adenotonsillectomy (AT) is the preferred treatment for OSA in children.[49] For the last 5 years, various authors conducted research to comprehend whether adenotonsillectomy could also improve asthma symptoms in children (**Table 2**).

Bhattacharjee et al, hypothesized that adenotonsillectomy, the first line of therapy for childhood OSA, would be associated with improved asthma outcomes and would then reduce the usage of asthma therapies in children.[50] Children between the ages of 3 to 17 years were included in the study. A total of 13,506 asthmatic children who underwent adenotonsillectomy (AT+ group) were included and 27,012 asthmatic children who did not undergo adenotonsillectomy were in the control group (AT- group). Of note, about 27% of the patients in AT+ group had some form of sleep breathing disorder characterized as sleep apnea, snoring or sleep disturbances.[50] The results showed that in the AT+ group there was a significant reduction in the acute asthma exacerbation by 30.2% (p<0.0001) and reductions in acute status astmathicus by 37.9% (p<0.0001).[50] They also showed

Table 2: Studies investigating the role of adenotonsillectomy in asthmatic children.

First author, year of publication	Study population (N), duration	Primary outcome	Secondary outcome	Results
Bhattacharjee et al, 2014[50]	- Asthma with AT(AT+) (n=13,506), 1 year - Asthma without AT (AT-) (n=27,012), 1 year	- Frequency of asthma exacerbation/status asthmaticus (ARERs and ARHs)	- Acute bronchospasm, wheezing, intubation	-Positive results - Reductions in acute asthma exacerbations (30.2%; 95% CI: 25.6%–34.3%; p<0.0001) - Reductions in acute status asthmaticus (37.9%; 95% CI: 29.2%–45.6%; p<0.0001) - ARERs (25.6%; 95% CI: 16.9%–33.3%; p<0.0001) - ARHs (35.8%; 95% CI: 19.6%–48.7%; p = 0.02) - no significant reductions in these outcomes in children with asthma who did not undergo AT (control AT-)
Kheirandish-Gozal et al, 2011[51]	- Children with poorly controlled asthma (n=92) was subjected to PSG - Children with OSA underwent AT (n=35), 1 year - Children without OSA (n=24), 1 year	- Frequency of acute asthma exacerbation per year	- Acute bronchospasm, wheezing, intubation	- OSA present in 58 patients (63.0%; OR: 40.9, 12.9-144.1, p<0.000001) - AAE in one year for OSA patients with AT decreased from 4.1±1.3/year to 1.8±1.4/year (p<0.0001) - No changes in AAE for non OSA patients

Abbreviation: AT=adenotonsillectomy, AAE=acute asthma exacerbations, ARERs=asthma related emergency room visits, ARHs=asthma related hospitalizations, OSA=obstructive sleep apnea, PSG=polysomnography

that the asthma related emergency room visits and asthma related hospitalizations significantly reduced to 25.6% (p<0.0001) and 35.8% (p=0.02), respectively.[50] However, in the control group (AT-), there was no significant reduction in the outcome.

In an earlier study, Kheirandish-Gozal et al, subjected 92 children between the age of 3 to 10 years old with poorly controlled asthma with a mean of acute asthma exacerbation 3.4±0.4 per year to an overnight polysomnography.[51] OSA (defined as AHI>5 per hour) was present in 58 patients, with a prevalence of 63%.[51] Thirty-five of the OSA patients underwent adenotonsillectomy (AT) and the asthma control assessments before and after 1 year of adenotonsillectomy were analyzed. The frequency of acute asthma exacerbation was reduced significantly in the post AT group from 4.1±1.3/year to 1.8±1.4/year (p<0.0001). However there were no changes in the non-OSA group.[51] The author concluded that treatment of OSA appears to be associated with substantial improvements in the severity of the underlying asthmatic condition in children.

In summary, asthma and OSA are common disorders, and the concomitant presence of both conditions can be detrimental. Due to the bidirectional association of both conditions, we as clinicians should be aware of it. CPAP in adults and adenotonsillectomy in children are recommended as first line treatment of OSA, and there is growing evidence that it can improve their asthma symptoms as well. However, further confirmation is necessary with larger randomized control trials to further evaluate both conditions and the treatment effects.

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