HHS Public Access

Author manuscript

Pediatr Ann. Author manuscript; available in PMC 2019 January 21.

Published in final edited form as:

Pediatr Ann. 2017 September 01; 46(9): e332–e335. doi:10.3928/19382359-20170815-03.

Pediatric Obstructive Sleep Apnea and Asthma: Clinical Implications

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Abstract

Obstructive sleep apnea (OSA) and asthma are common conditions in children with preventable long-term consequences. There is significant overlap in symptomatology and pathophysiology for pediatric OSA and asthma. Recent evidence supports clear associations between the two diseases; however, causality has not been demonstrated. Regardless, it is important to recognize the overlap and evaluate for the other condition when one is present. For example, in patients with severe OSA, clinical evaluation for asthma should be considered, including history for typical asthma symptoms and spirometry. For patients with severe or poorly controlled asthma, OSA should be considered as a complicating condition. Clinical history for OSA as well as pediatric sleep questionnaires may be helpful tools in evaluation of the child with severe asthma. To decrease long-term consequences from asthma and OSA in children, clinicians should consider the relationship between these two diseases. **[*Pediatr Ann*. 2017;46(9):e332-e335.]**

Obstructive sleep apnea (OSA) and asthma are common respiratory conditions in children with overlapping features that may go unrecognized. If left untreated, both pediatric OSA and asthma can lead to long-term, but preventable, consequences. This review provides an

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Disclosure: Evan Bailey discloses an advisory board honoraria received from Vertex Pharmaceuticals. The authors have no relevant financial relationships to disclose.

update on pediatric OSA and describes the intersection between pediatric OSA and childhood asthma, with a focus on relevance in clinical practice.

Pediatric OSA is defined as disordered breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction (ie, obstructive apnea) that disrupts normal ventilation during sleep and normal sleep patterns.[1]

The prevalence of OSA has been inconsistently reported, likely due to wide variations in sample size and populations. However, from level I and II studies (ie, large sample sizes from the general pediatric population), the prevalence rates that are reported range from 1.2% to 5.7%.[2]

Asthma is defined as a chronic inflammatory condition with narrowing, swelling, and additional mucous production in the lower airways. The prevalence rate for childhood asthma is 8%, with nearly 7 million children in the United States currently living with asthma,[3–5] making asthma the most common cause of chronic childhood illness.[6]

The high prevalence of both asthma and OSA in children raises interest in the possible connection between the two. There is, in fact, evidence of a bidirectional relationship between the two disease processes.[7] Wheezing and asthma symptoms often coexist with symptoms of OSA. A common symptom between the two disease processes is nocturnal dyspnea. Ross et al.[8] showed that among 108 children age 4 to 18 years who were recruited from an asthma clinic, 29.6% of children had sleep-disordered breathing. Among those with OSA, there was a 3.6-fold increased odds of having severe asthma at follow up.[8] This suggests that OSA could predict asthma severity.

REVIEW OF PEDIATRIC OBSTRUCTIVE SLEEP APNEA SYNDROME

The symptoms of OSA in pediatric patients usually include a history of snoring, gasping, and pauses in breathing that may result in sudden arousals during sleep. The level of snoring and obstruction may vary depending on the sleep position and stage of sleep. Upper airway obstruction is commonly greatest in the supine sleeping position and during rapid eye movement (REM) sleep.[9] Some children may not snore, in contrast to adults with OSA. Other symptoms of OSA during sleep may include paradoxical movement of the chest and abdomen, a restless sleep pattern, excessive sweating, and nighttime enuresis. In more severe cases, children may sleep in a semi-upright position to help keep the upper airway patent. Parents may be concerned enough to monitor and stimulate the child in severe cases. Daytime symptoms of OSA may include excessive daytime sleepiness, but children are often not sleepy and may be more likely to suffer from poor attention and behavioral problems. Because the cause is often related to adenotonsillar hypertrophy, there may be symptoms of nasal obstruction and mouth breathing and associated adenoid facies.

Physical Findings

The physical findings of children with OSA commonly include enlarged tonsils and adenoids. Other findings may include a high-arched palate, retrognathia/micrognathia, and/or midface hypoplasia. Some children have findings of allergic rhinitis with edematous

turbinates and nasal obstruction. Although children with OSA may have a normal weight, it is not uncommon to see children who may have poor growth and concerns for failure to thrive, or who are overweight and obese.

Consequences

Untreated OSA in childhood may be associated with a number of consequences. Neurobehavioral manifestations are common findings in pediatric OSA. A diagnosis of attention-deficit/hyperactivity disorder is common.[10] Children with OSA may also suffer academically due to associated cognitive deficits.[11] Although cardiopulmonary manifestations may be less common, systemic hypertension, right ventricular dysfunction, and left ventricular dysfunction are potential findings.[12,13] The possibility for cor pulmonale is a rare finding that may be seen in severe OSA, in particular with other comorbid diagnoses such as Down syndrome. This may also manifest as Pickwickian syndrome, also known as obesity hypoventilation syndrome, in severe cases.

Diagnosis

The diagnosis of OSA in childhood commonly involves utilization of polysomnography (PSG). Although the technical component is similar to traditional sleep studies on adults, performing polysomnograms in children is vastly different and more diffiuclt. These challenges make it important for pediatric sleep studies to be performed as an attended in-laboratory PSG rather than an at-home PSG, which is now the common approach for adults with suspected OSA. It also is preferred, when available, that such studies be performed in a child-friendly sleep laboratory with technologists accustomed to working with children and ideally to have the results interpreted by a certified pediatric sleep physician. Guidelines are available on the indications for a PSG.[14,15] The diagnosis of OSA is typically made based on a summary of the number of apneas and hypopneas (the apnea-hypopnea index) and evidence for hypoxemia and hypercapnia. Alternative data that may aid the diagnosis include home audiotapes and videotapes that parents bring to the clinic. Although overnight pulse-oximetry alone may aid in diagnosing OSA, it is less effective at ruling it out.[16] The Pediatric Sleep Questionnaire may be helpful in a clinic setting as an initial screening tool for OSA, with a specific role in predicting neurobehavioral morbidity from OSA as well or better than polysomnogram.[17] When a diagnosis of OSA has been made, further evaluation for tonsil and adenoidal hypertrophy may be indicated. This may be done by X-ray; however, an endoscopic assessment of adenoidal size and airway narrowing can be helpful.

Treatment

The decision to treat OSA in children should be based on a combination of the history, physical examination, PSG results, and the degree of daytime functional impairment. When it comes to treatment for OSA, one size does not fit all. Children with moderate to severe OSA who have functional impairment should be referred to otolaryngology to evaluate for adenotonsillectomy. This is the first-line therapy when these tissues are enlarged.[18] Outcomes of such surgery demonstrate mixed results, including improvement in some parameters such as sleep apnea symptoms, daytime behavior, and quality of life, but appear to be less effective at improving measures of attention and cognitive function.[18]

Continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BIPAP) are alternative options to consider and may be a better alternative in the adolescent patient who is overweight or does not have evidence of adenotonsillar hypertrophy. Choosing between CPAP and BIPAP often depends on the level of severity, comfort, and evidence for hypoventilation in certain populations. A titration study in the sleep laboratory is usually necessary to help determine appropriate pressures and address challenges with implementation. Identifying the mask interface is critical to assure comfort and improve tolerance. Nasal masks are often preferred, but a full-face mask should be considered if the child is a mouth breather. Close follow-up is imperative to address concerns with compliance as well as any problems with mask fit and skin irritation. Humidification of the air may reduce nasal congestion. New masks and tubing are typically supplied every 6 months.

Other alternative therapies should be considered for patients with mild OSA or in situations where surgery or CPAP are either ineffective or not tolerated. Nasal corticosteroids and leukotriene modifiers may help to improve nasal airflow either by improving nasal congestion related to allergies or by reducing adenoid size.[19,20]

Rapid maxillary expansion may be an option for some patients with specific orthodontic airway limitations, including a narrow palate.[21] Improvement in environmental exposures should always be considered, such as tobacco smoke, air pollutants, and allergens that may cause nasal obstruction. Weight loss is recommended in situations where obesity is a complicating factor. Positional therapy may be helpful in circumstances where OSA is greatest in the supine sleeping position. Although oral appliances are often used in adults with OSA, they are typically discouraged in children due to the potential effect such devices may have on a growing mandible.

OVERLAP OF PEDIATRIC OBSTRUCTIVE SLEEP APNEA SYNDROME AND ASTHMA

Nocturnal asthma, a known complication of persistent asthma, results in nighttime hypoxemia and breathing difficulties. Similarly, hypoxia and nocturnal dyspnea are hallmarks of OSA. Thus, a correlation between asthma and OSA has been proposed. Patients with severe asthma have an increased risk of apneas and hypopneas compared to body mass index (BMI)-matched and age-matched controls.[22] A number of biological mechanisms can explain why OSA can exacerbate asthma, or why asthma can exacerbate OSA.

One possible mechanism involves OSA-induced upper airway vibration and collapse of the airways. This vibration and collapse can lead to bronchoconstriction by increasing the airway vagal tone.[23] Increased vagal tone exacerbates nocturnal asthma, and anticholinergics are often used to counter this bronchoconstriction.[24] Another mechanism involves an OSA-induced rise in inflammatory markers. The upper air-way vibration and collapse result in upper airway inflammatory changes that then activate an inflammatory response such as neutrophil recruitment in the lower airways. For example, increase in T-cell lymphocyte infiltration has been seen in the upper airway muscles and mucosa of OSA patients.[25]

Furthermore, OSA increases the proinflammatory cytokines C-reactive protein, tumor necrosis factor-alpha and proinflammatory interleukins.[26–28] In children with OSA, leukotriene concentrations are increased in adenotonsillar tissue. Leukotrienes also play a role in persistent asthma and as a result, leukotriene receptor antagonists are a therapy for certain phenotypes of asthma, as well as for some children with OSA. For example, one study showed that a 16-week course of orally administered leukotriene receptor antagonist was associated with significant improvement in upper airway patency and in severity of OSA compared to a nontreated group.[29] Finally, OSA can increase the chances of gastroesophageal reflux, which has been associated with asthma. Treating OSA can decrease asthma symptoms. Nocturnal asthma attacks are often eliminated and asthma symptoms are improved when CPAP is used to treat OSA.[30,31]

Asthma can exacerbate or result in OSA in several ways. Nocturnal hypoxemia due to asthma can increase the severity of OSA.[32] Asthma itself may lead to OSA after adjustment of confounders such as BMI.[33] It has been proposed[34,35] that asthma can affect the patency of the upper airways through sleep loss and fragmentation and through systemic inflammation-related weakening of respiratory muscles. Consideration of OSA in a patient with severe asthma is important, and the pediatric sleep questionnaire is now validated in children with asthma.[36] Therapies for asthma may also result in OSA. For example, there appears to be a linear dose-dependent association between inhaled corticosteroids and OSA independent of asthma severity.[33]

SUMMARY

Pediatric OSA and asthma are two common conditions with significant long-term consequences. The clear associations between these two conditions warrant the attention of pediatric health care providers as they evaluate these patients.

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