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Review Article

Obesity and Obstructive Sleep Apnoea Syndrome in Children

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Abstract

The prevalence of obesity in children is increasing locally and worldwide. Obesity has been shown to increase the risk of obstructive sleep apnoea syndrome (OSAS) not only in adult but also in children. Obesity and OSAS share similar cardiovascular and metabolic morbidities. OSAS may aggravate the cardiovascular and metabolic morbidities associated with obesity, possibly via potentiation of inflammatory pathways. Obesity also impacts on the management of childhood OSAS, as it lowers the response to adenotonsillectomy, the first-line management. This review will examine the current understanding of the relationship between obesity and OSAS in children, and will discuss the implication of obesity in the management of childhood OSAS.

Keywords: Children, obesity, obstructive sleep apnoea

Introduction

The prevalence of obesity in children has increased worldwide and locally. Obstructive sleep apnoea syndrome (OSAS) is a disorder characterised by recurrent episodes of partial or complete airway obstruction during sleep, resulting in disruption of normal gas exchange and sleep fragmentation.[1] Epidemiological data suggested that OSAS affected 1-4% of children.[2] In adult, obesity and male sex are well established risk factors for OSAS. In children, adenotonsillar hypertrophy is the most important risk factor. However, contrary to the classical description of children with OSAS presented as underweight and having adenotonsillar hypertrophy; the association between childhood obesity and OSAS has been clearly observed in epidemiological studies in the past decade. The incidence of childhood OSAS is anticipated rise with the epidemic of childhood obesity. Both obesity and OSAS are associated with similar adverse cardiovascular, metabolic and neurocognitive outcomes. Obesity also poses substantial impact to the management of childhood OSAS. The aim of this review is to evaluate the current understanding of the relationships between obesity and OSAS in children, and to provide insight on the approach to management of this group of children.

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Obesity as a risk factor for childhood OSAS

Sleep disordered breathing on polysomnography was found in 46% of obese children and adolescent. There was a positive correlation between obesity and apnoea index and an inverse correlation between obesity and oxygen nadir.[3] In a case-control study which included children and adolescents 2 to 18 years of age, showed that obese children are at 4.6- fold risk for sleep apnoea than normal weight children.[4] For each increase of 1 kg/m^2 of BMI above mean, the risk of sleep disordered breathing (SDB) increased by 12%.[4] Similar trends demonstrating obesity increased the risk of OSAS in children have been reported.[3,5]

The relative impact of obesity on OSAS in pre-pubertal children and adolescents remained debatable. MRI study shows that in pre-pubertal children with OSAS the maximum narrowing of upper airway was at the level of the adenoid, tonsils and the soft palate, and is not associated with paraphagyngeal fat.[6] This is consistent with the understanding that adenotonsillar

hypertrophy is the most common risk factor for OSAS among young children. However, OSAS in the adolescents may be more complex. Some evidence suggested that OSAS in the adolescents represent an extension of childhood disease, while others believe that the pathophysiology more resembles adult OSAS with obesity as the most important factor. In a cohort of 8 to 11 years old children, the odds ratio for OSAS given overweight was only 1.3.[7] A follow-up study of same cohort, at ages 13 to 16 years, showed an odds ratio greater than 6.0.[8] However, a recent study showed that the effect of obesity on upper airway narrowing was irrespective of age.[9] The relative contribution adenotonsillar hypertrophy and obesity in OSAS in different age groups, and how the two factors may interact in predisposing to upper airway collapsibility remain to be further examined.

The pathophysiology OSAS attributable to obesity

Fatty infiltration of upper airway structures will result in upper airway narrowing. Concomitantly, increased subcutaneous fat deposits in the anterior neck region will exert force causing pharyngeal collapsibility. However, the relationship between BMI and OSAS severity remained poor, suggesting that fat distribution in the upper airway and possibly other neuromotor and inflammatory mechanism were also important.[9]

Obesity also causes change in pulmonary mechanics, as increased adiposity in the abdominal and thoracic wall increased the overall respiratory load, reduces intrathoracic volume and diaphragm excursion, especially in supine position, which may result in decreased in lung volumes and oxygen reserve, and increases the work of breathing during sleep.[3,10]

Recent evidence has emerged to suggest the potential role of leptin in the link between obesity and sleep apnoea. Leptin is a protein produced by adipose tissue involves in appetite and energy balance. Leptin is a potent respiratory stimulant which acts primarily in hypothalamus and is implicated in hypercapneic responses. Obesity decreases the bioavailability of leptin in the cerebrospinal fluid and increases circulating leptin level, leading to a state of leptin resistance,[11] which results in suppression of overall ventilatory drive and chemoreceptor activity. Leptin resistance has been implicated in the development of obesity hypoventilation syndrome and obstructive apnoea.[12]

Potentiating role of obesity in the morbidities associated with OSAS

Compelling evidence existed showing that paediatrics OSAS is associated with cardiovascular and metabolic consequences such as hypertension and insulin resistance. Much of these morbidities overlap with the consequences of childhood obesity. The cardiovascular and metabolic consequences of obesity were related to cytokines, growth factors and sex steroids secreted by adipose tissue affecting the action of insulin and fatty acids metabolism.[13,14] In OSAS, intermittent hypoxaemia and oxidative stress have been shown to stimulate inflammatory cytokines and potentiate the inflammatory cascade, resulting in changes in endothelial function.[15,16] It is possible that OSAS and obesity interact and augment the severity cardiovascular and metabolic morbidities via the inflammatory cascade. Studies are required to demonstrate the effect of treatment of paediatric OSAS on the reversibility of cardiovascular and metabolic morbidities.

Does OSAS contribute to obesity?

Short sleep duration has been shown to be associated with obesity in children.[17-19] OSAS can cause sleep disruption and sleep deprivation. Hence, OSAS may increase the risk of obesity. On the other hand, obesity increases the risk of OSAS. Sleep deprivation, OSAS and obesity may be interrelated and exacerbate the severity and effects of each condition.

Implication of obesity in treatment of OSAS

Tonsillectomy and adenoidectomy (T&A) is the mainstay of treatment for childhood OSAS. Many of the obese children with OSAS also have adenotonsillar hypertrophy. Surgical removal of the tonsils and adenoids would improve upper airway patency. It is evidenced that the obese children have significant improvement in OSAS after T&A. In a meta-analysis involving 110 obese children with OSAS who underwent T&A, the mean apnea-hypopnea index (AHI) decreased from 29.4 (range 22.2-34.3) to 10.3 (6.0-12.2). However, obese children tend to have lower cure rate. As low as 12% achieved complete cure if a cut-off of AHI <1 was used, and 51% had post-operative AHI in the range of 1-5.[20] This was in contrast to the cure rate after T&A in normal weight children with OSAS, 83-90% achieved an AHI <1

postoperatively.[21,22] The poorer response to T&A can be partly explained by more severe OSAS in obese children preoperatively. As obese children are at risk of residual OSAS, follow-up polysomnography should be considered, especially for those who have persistent snoring. Nonetheless, T&A lead to clinically significant improvement in most obese children with OSAS; it should remain as the first-line treatment.

Obesity increased the postoperative respiratory complications in children with OSAS undergoing tonsillectomy. In particular, they have higher risk of upper airway obstruction in the immediate postoperative period.[23] The American Academy of Pediatrics recommends overnight hospitalisation and monitoring after adenotonsillectomy in this group of children.[24]

Weight gain after adenotonsillectomy should be carefully monitored. Substantial weight gain after T&A has been observed in normal weight and obese children with OSAS.[25,26] Weight gain could be due to reduced energy spent in work of breathing, but has been shown to related to decreased motor activity during sleep and decreased daytime hyperactivity.[25] The rate of weight gain after T&A has been shown to be an independent risk factor for recurrence of OSAS.[27] One should be cautious that by removing the enlarged adnenoids and tonsils, we are not replacing this with another risk factor, namely, obesity, in children with OSAS.

Adult studies have shown weight reduction is associated with improvement in apnoeic episodes, and has become the first line management for OSAS.[28,29] Paediatric data is lacking. Studies have shown weight loss after bariatric surgery[30] and ketogenic diet[31] in morbidly obese adolescents was associated with improvement in OSAS. These studies were limited by small sample size, and the extreme modalities for weight reduction cannot be recommended for most of the obese children with OSAS. Studies are required to demonstrate the effectiveness of weight reduction in the management of paediatric OSAS, particularly with different age groups. Nevertheless, as obesity is associated with less favourable outcome and higher risk of recurrence after T&A, and weight reduction has been shown to improve cardiovascular function,[32] weight reduction should be emphasized as an important treatment for obese children with OSAS.

Conclusion

Obese children are at risk of developing OSAS. Symptoms of sleep disordered breathing should be sought and evaluated in obese children. OSAS may potentiate the morbidities associated with obesity, possibly via the inflammation cascade. Adenotonsillectomy leads to significant improvements in sleep disordered breathing in most obese children, hence should remain the first line of treatment. Residual OSAS is common in obese children, follow-up polysomnography should be considered. Weight reduction should be emphasized an important management to obese children with OSAS.

Further studies are required to delineate the relative contribution of obesity and adenotonsillar hypertrophy in children, and the effectiveness of T&A in obese children among different age groups and different range of weights.

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