Review Article

Obesity and Obstructive Sleep Apnoea Hypopnoea Syndrome in Singapore Children

Jenny PL Tang,[1] *MBBS, MRCP, FAMS*

Abstract

Introduction: Obesity affects about 10% to 15% of our school-going population in Singapore and is a risk factor for development of obstructive sleep apnoea hypopnoea syndrome (OSAHS). This article reviews the prevalence, aetiology, pathophysiology, diagnosis, complications and treatment of obese children with OSAHS with particular reference to children in Singapore. Methods: Review of articles or conference papers reporting data with regards to OSAHS in Singapore children. Results: Prevalence of OSAHS was high in obese children in Singapore and was more common in males with no racial predisposition. Hypersomnolence as a presenting symptom was uncommon. Cognitive function, behaviour, attention and processing speed was affected and improved after intervention. Abnormalities of glucose metabolism were also found with the respiratory disturbance index (RDI) as an independent predictor of insulin resistance. Tonsillectomy and or adenoidectomy was efficacious as treatment and risk of complications was low. No significant increase in weight occurred post intervention in those enrolled in concurrent weight management programmes. Conclusions: Prevalence of OSAHS is high in obese Singapore children and many are 'asymptomatic'. A low threshold for evaluation is necessary for early diagnosis and intervention for prevention of morbidity. Tonsillectomy and/or adenoidectomy is safe and efficacious and remains the first-line treatment in most obese patients.

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Key words: Complications, Diagnosis, Prevalence, Treatment

Introduction

Obesity is becoming a problem of epidemic proportions and is perhaps, the most pervasive medical problem faced by medical providers today. It is a problem affecting about 10% to 15% of our school-going population in Singapore,[1] affecting disease burden in virtually every medical subspecialty. Obesity is a risk factor for development of many diseases including cardiovascular diseases, metabolic and endocrine problems, obstructive sleep apnoea (OSA), cancers, orthopaedic problems, gallbladder disease and psychosocial problems.[2]

In Singapore, overweight/obesity in children is defined as %ideal body weight for height (%IBW) >120% / >140% for gender for children aged 6 to18 years[2] or body mass index (BMI) >85th />95th percentile for age and gender for those <72 months.[3]

Obstructive sleep apnoea hypopnoea syndrome (OSAHS) in children is defined as a disorder of breathing during sleep characterised by prolonged partial upper airway obstruction and/or intermittent complete obstruction that disrupts normal ventilation during sleep and normal sleep patterns.[4]

Although the association of obesity and OSAHS was first noted by Charles Dickens in his description of Joe, an obese, hypersomnolent boy in The Posthumous papers of the Pickwick Club more than 100 years ago, it is only in recent times that the extent of OSAHS in the obese paediatric population has been realised.

This article reviews the prevalence, aetiology, pathophysiology, diagnosis, complications and treatment of obese children with OSAHS with particular reference to children in Singapore.

Methods

Articles or conference papers reporting data with regard to OSAHS in Singapore children were reviewed. A PUBMED search was conducted using the following search parameters: children, Singapore, obstructive sleep apnoea and obese. Conference papers, if any, were also obtained from communication with sleep physicians in the country.

Prevalence

The overall incidence of OSAHS in children is about 0.7% to 3%.[5-7] In obese children, the incidence is much higher, ranging from 13% to 66%.[7-12] The wide range is due

[1] Respiratory Medicine Service, Department of Paediatric Medicine, KK Women's and Children's Hospital, Singapore
Address for Correspondence: Dr Jenny Tang Poh Lin, Department of Paediatric Medicine, KK Women's and Children's Hospital, 100 Bukit Timah Rd, Singapore 229899.
Email: Jenny.Tang.PL@kkh.com.sg

to different cohorts studied, variability in ethnic groups, different inclusion criteria as well as differences in diagnostic criteria for obesity and OSAHS. Chay et al[7] in a study of 3671 obese school children aged 6 to 18 years reported a population prevalence of 0.7% and 13.3% in obese Singapore school children with IBW >140% and IBW >180%, respectively. Tang et al[12] reported a 55% prevalence in 'asymptomatic' morbidly obese Singapore school children aged 6 to 18 years with IBW >160%. Of the children referred to the KK Women's and Children's Hospital (KKH) sleep disorders centre with a possible diagnosis of OSAHS, the prevalence in overweight children with IBW >120% (>6 years) or BMI >85th percentile (< 72 months) was found to be 55% .[13] In the same series, 59% and 64% of obese and morbidly obese (IBW >160%) children, respectively, were found to have OSAHS. These results are similar to that reported by other centres.[8-11]

Aetiology and Pathophysiology

The spectrum of sleep-disordered breathing is a continuum from primary snoring (PS) at one end of the spectrum and OSAHS at the other.[14] Primary snoring is generally believed to be benign although recent studies challenge this.[15,16]

The current understanding of OSAHS supports the existence of a dynamic imbalance in upper airway function resulting from a combination of alterations in structural and anatomical characteristics, protective reflexes and neuromotor function of the upper airway.[17] In obese children, the effects of fat deposition in the pharyngeal muscles and extra-pharyngeal compression from superficial subcutaneous fat further decreases the pharyngeal lumen and increases pharyngeal collapsibility.[18,19] Reduced chest wall compliance and cephalad displacement of the diaphragm by abdominal fat when supine also reduces ventilatory efforts, lung volumes and oxygen reserves.[10] In addition, the reduced lung volumes cause a reflexive decrease in the size of the pharyngeal airway exacerbating the respiratory compromis.[20] Recent evidence has also discussed the potential role of leptin, a potent respiratory stimulant and central chemoreceptor modulator as a link between obesity, OSAHS and metabolic dysfunction.[21]

OSAHS has been reported to have an equal incidence in male and female children.[22] Known risk factors include African American children, children with a family history of OSAHS or with airway inflammatory disorders such as allergic rhinitis and bronchial asthma and a history of prematurity.[23-26] Studies in obese Singapore school children demonstrated no increased risk with different ethnicity comparing between the Chinese, Malay, Indian and other races. These same studies, however, did demonstrate an increased risk with male gender likely due to the higher mean age of children studied.[7,12,13]

Complications

Hypersomnolence: Excessive daytime sleepiness is a common symptom of OSAHS in adults. It has also been reported that obesity without sleep apnoea is associated with daytime sleepiness with subjective reports of sleepiness in up to 57% of obese adults with no OSAHS. This is postulated to be related to a metabolic and/or circadian abnormality.[27] Although overt excessive daytime sleepiness is not very common in children, it is also reported to be more common among obese children and children with more severe OSAHS.[28] Similarly, our studies in children with possible OSAHS demonstrated that hypersomnolence was uncommon by both self-reports of hypersomnolence as well as by objective testing with mean sleep latency tests with prevalence ranging from 11% to 22%.[7,13,29] In children with no apnoea, obese children were found to be significantly more sleepy;whereas in children with sleep apnoea, sleepiness correlated with severity of sleep apnoea.[29]

Neurobehavioural consequences: Behavioural disturbances including restlessness, hyperactivity and aggressive behaviours have been documented as consequences of OSAHS and snoring in children. In addition, these children have been found to have poorer school performance and perform poorly in measures of attention and vigilance as well as that of executive functioning.[30] Improvements in learning and behaviour have been reported following treatment for OSAHS in children.[31] Similarly, assessments of cognitive function, behaviour, attention and processing speed of obese Singapore school children before and after intervention for OSAHS using the Leiter International Performance scale – Revised (LIPS-R), Wide range achievement test 3 (WRAT-3), Inattention Hyperactivity score (CTRS, CPRS), child behaviour checklist (CBCL 4-18) and the Conners Continuous performance Test (CPT) showed a general trend of improvement in attention and a significant improvement in behaviour, cognitive function and processing speed after intervention.[32,33] This is consistent with current evidence suggestive that the neurocognitive and behavioural deficits are at least partially reversible.

Cardiovascular consequences: Obesity has been well documented to increase the risk of cardiovascular disease. Similarly, OSAHS has been associated with an increased cardiovascular morbidity with increased prevalence of systemic hypertension, alterations in blood pressure regulation and changes in cardiac geometry.[34] This is likely to be due to the presence of sustained sympathetic activation during both daytime and nighttime, as well as to increased sympathetic reactivity and endothelial dysfunction. In addition, pulmonary hypertension with cor pulmonale may occur with frequent oxygen desaturations during sleep in severe OSAHS. There is no local data studying the

cardiovascular consequences of OSAHS in obese children. However in our first case reports of 4 children with OSAHS, all were obese and had severe OSAHS with cardiovascular compromise with right ventricular hypertrophy with strain ± cor pulmonale.[35]

Endocrine complications: Obesity is an established risk factor for insulin resistance, type II diabetes mellitus and metabolic syndrome. Similarly, OSAHS has been identified as an important risk factor for metabolic syndrome in adult patients. In children with OSAHS, however, there have been conflicting results with regard to OSAHS as an independent predictor for insulin resistance and metabolic syndrome.[36,37] We studied factors associated with abnormality in glucose metabolism in our cohort of obese Singapore school children and demonstrated that respiratory disturbance index (RDI) was an independent predictor of insulin resistance after correction for IBW.[12]

Growth impairment: Growth impairment in OSAHS has been attributed to various factors including decreased appetite possibly associated with reduced olfaction in children with adenoidal hypertrophy, dysphagia from tonsillar hypertrophy, decreased insulin growth factor (IGF), IGF binding proteins and possibly growth hormone release and increased energy expenditure from increased work of breathing during sleep.[38-40] Failure to thrive with catch up growth, normal weight or obesity with weight gain after adenotonsillectomy has been reported.[39, 41,42] We reported no significant increase in weight in morbidly obese Singapore school children who had concurrent weight management programmes after surgical intervention or CPAP therapy for OSAHS.[43]

Quality of life issues: Both obesity and OSAHS are associated with a poorer quality of life (QoL) when compared to non-obese children or those without OSAHS. In addition, QoL has been shown to improve after intervention for OSAHS.[44-46] Similarly, we demonstrated a poorer QoL in obese children with OSAHS using the Child Health Questionnaire PF-50 (CHQ-PF50) comparable to children with Bronchial Asthma and Juvenile Chronic Arthritis. After intervention for OSAHS and obesity, there was a significant improvement in QoL in global health rating, physical and psychosocial summary with the largest improvements in the domains of General Health (GH) and Mental Health (MH).[32,47]

Treatment

Tonsillectomy and/or adenoidectomy is the mainstay of treatment of paediatric OSAHS with more than 80% of children achieving a 'cure'.[48,49] Many obese children also have adenotonsillar hypertrophy further compromising upper airway patency and one would expect a lesser improvement in disease state. However, most studies evaluating the efficacy of tonsillectomy and/or adenoidectomy of obese children with OSAHS have shown marked improvements in RDI after surgical intervention with the degree of obesity being a major determinant of residual disease.[50-53] Similarly, in a study of morbidly obese Singapore school children with pre and post PSG after surgical intervention, we demonstrated an improvement in RDI from an overall mean of 45.4/h to 7.9/h with improvement in all but 1 patient (96%) and complete resolution of disease in 33% of patients. Initial RDI but not %IBW was found to be a predictor of residual disease, likely because this was a study only in morbidly obese children.[38] Thus, tonsillectomy and/or adenoidectomy can be recommended as first-line treatment in obese patients with adenotonsillar hypertrophy.

Although tonsillectomy and/or adenoidectomy is a low risk procedure, children with obesity and OSAHS show an increased incidence of postoperative complications.[54-57] The American Academy of Paediatrics has recommended overnight hospitalisation and monitoring for this group of high-risk patients.[58] In our own series looking at postoperative complications in morbidly obese patients undergoing tonsillectomy and/or adenoidectomy, the risk of postoperative complications was low at 4.2%.[43] This is likely due to our practice of preoperative polysomnography in almost all patients with possible OSAHS with identification of risk factors and anticipative management in the postoperative period.

Non-invasive ventilation in the form of continuous positive airway pressure (CPAP) as well as bi level positive airway pressure (BiPAP) is used in patients who are not suitable candidates for surgery or as a salvage procedure for residual disease after surgery. In our series of morbidly obese patients, 8% of children had CPAP as first-line therapy and 30% of patients who had surgical intervention eventually required CPAP therapy.[38]

Conclusion

Prevalence of OSAHS is high in obese Singapore children and many are 'asymptomatic'. OSAHS in the obese child increases the risk of morbidity and a low threshold for evaluation is necessary for early diagnosis and intervention to avoid the consequences of delayed treatment. Tonsillectomy and/or adenoidectomy is safe and efficacious and remains the first-line of treatment in most obese patients. Repeat evaluation is recommended following surgical intervention to rule out presence of residual disease. Concurrent weight management programmes are necessary to optimise treatment of OSAHS in this group of patients.

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