Section 10: Obesity hypoventilation in children

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Introduction

Obesity can lead to altered breathing during wakefulness and sleep. Central hypoventilation (related to excessive load on respiratory muscles, decreased function of the ventilatory drive or a combination of both) is addressed in this section, with a specific focus on hypoventilation associated with obesity, also known as obesity hypoventilation syndrome (OHS). OHS may develop either as an isolated disorder associated with obesity or as part of a constellation of genetically related and inherited dysregulation syndromes, such as Prader-Willi syndrome and rapid-onset obesity with hypothalamic dysfunction, hypoventilation, and autonomic dysregulation presenting in childhood (ROHHAD).

The definition of OHS has been broadly described. There is little evidence in children to support any specific criteria; however, based on consensus and expert opinion, the definition of OHS in children is as follows:

1. Obesity (BMI > 30 kg/m²) or BMI > 95th percentile for age and gender or weight > 95th percentile for age
2. Daytime hypercapnia (PaCO₂ > 45 mmHg)
3. Absence of known neurological, cardiac or pulmonary causes of hypoventilation

Literature review: Methodology

A systematic search of the literature from 1946 to March 2014, subsequently updated to August 2015, was conducted in the following databases: Medline, Embase, AMED, PsycINFO, Central, Cinahl. MeSH terms and keywords used captured the concepts “home” and “ventilation” as well as obesity hypoventilation syndrome. We aimed at identifying all studies published in English and French. As well, reference lists from identified publications were hand searched in order to add any missed studies. We also searched the websites of large associations of physicians and health professionals in the fields of respiratory medicine, intensive care, nursing and respiratory therapy for reviews, consensus statements and clinical practice guidelines.

Results

There were no pediatric studies identified that reported specifically on children with OHS. Obese children with sleep-disordered breathing (SDB) have been treated with non-invasive positive pressure ventilation (NIPPV), but whether they represent OHS, SDB or a combination of both, is not clear. Positive airway pressure therapy in obese children has been studied but the subtype of OHS has not been evaluated specifically. In the adult literature, among middle-aged patients, there is evidence to suggest that treatment for OHS is efficacious and improves outcomes.

Discussion

The prevalence of OHS in pediatric patients is not known. The prevalence of OHS in adults with obstructive sleep apnea (OSA) is estimated to be about 10–20%, according to a recent review of more than 3000 patients. Estimates of OSA in children are about 1–4%, with higher rates in children with obesity.

One study has attempted to evaluate OHS in otherwise healthy children. Of the 326 children, 28% were obese, but there was overlap with SDB. Risk factors included ethnicity (African-American race) and adenotonsillar hypertrophy. It is important to note that SDB can result in obstructive hypoventilation in the absence of obesity.

In contrast to adults, the phenotype of OHS in children has not been well-characterized and the clinical presentation of OHS can range from children being asymptomatic to presenting with acute respiratory failure with or without an intercurrent respiratory illness. Other children may present with indolent, yet progressive symptoms (snoring, apneas, headaches, etc.), daytime sleepiness or hypersomnia and associated sequelae such as altered cognitive function, associated left heart failure (shortness of breath on exertion,
activity limitation) or right heart failure (peripheral edema, dyspnea).9 The diagnosis of OHS should be suspected in any morbidly obese child who has symptoms of SDB, is somnolent, or has exercise limitation, especially in the context of daytime hypoxemia and hypercarbia. It is a diagnosis of exclusion, in that other disorders causing hypoventilation need to be ruled out.

The pathogenesis of OHS is not fully understood. Recent evidence in animal models and adults suggests that there may be OHS phenotypes.10,11 OHS may involve any combination of the following: morbid obesity, altered ventilatory response, alteration of chest wall mechanics resulting from obesity, upper airway dysfunction or altered neuromuscular tone, as well as possible contributions from neurohormonal influences of leptin. It is also not clear whether the abnormalities represent a primary defect in the neurological control of breathing, resulting in respiratory control dysfunction or whether it is a result of the habituation of the brainstem centers to hypoxemia and hypercarbia.10,11

There is limited evidence for associated morbidity and sequelae of OHS in children. In adults, OHS subjects have an increased mortality, increased hospitalization, poorer health related quality of life, decreased vigilance and an increased incidence of heart failure, angina and pulmonary hypertension.9 It is expected that children will also experience increased morbidity and mortality with a shortened life expectancy, but there are no studies that have evaluated outcomes or prognosis in children. Quality of life has been reported as improved by both caregiver and child in obese children treated with positive airway pressure therapy.12 However, this study was not designed to specifically evaluate children with OHS.

Evidence for the treatment of OHS is limited in children, in contrast to the adult literature. Treatment in adults consists of weight reduction strategies (exercise, dietary modification, pharmacological approaches and/or bariatric surgery) and positive airway pressure therapy, such as CPAP or noninvasive ventilation (NIV). In rare circumstances, a tracheostomy and invasive mechanical ventilation may be needed to fully correct the degree of hypoxemia and hypoventilation. Notably, of the adults identified with OHS, only a small portion obtains treatment.13 There is overall insufficient evidence for NIV, including methods, outcomes, costs and benefits, and for HMV in children with OHS. At best, any inferences are drawn from adult data about potential benefits or consensus opinion of the group.

Conclusion

OHS is likely an under-recognized cause of morbidity in obese children and is an increasing reason for initiating positive airway pressure therapy in children. The limited evidence, from obese children who are treated for SDB, suggests that positive airway pressure therapy may be effective for children with OHS. There is uncertainty about the timing of initiation of ventilatory support and the outcomes related to the treatment. Many questions remain unanswered, which makes the care of these children challenging.

Research questions

1. What is the optimal treatment choice (noninvasive mode, ventilator mode) for children with OHS?
2. Is weight loss sufficient in children with OHS to fully correct their sleep gas exchange abnormalities?
3. What are the morbidities associated with OHS in children?

Recommendations for obesity hypoventilation in children

1. All children with obesity should receive counseling regarding nutrition, the importance of exercise, the healthcare risks associated with obesity as well as the importance of a healthy lifestyle and body weight. (Consensus)
2. In obese children (as defined above), especially those who are symptomatic with daytime somnolence, evaluation for SDB should be undertaken with a PSG. (Grade 1C)
3. Obesity hypoventilation syndrome is a diagnosis of exclusion and other causes of central hypoventilation need to be excluded. (Grade 1C)
4. In obese children with OHS, NIV should be initiated. (Grade 2C)
5. NIV therapy can be initiated in an outpatient sleep laboratory or on a hospital ward as an inpatient. (Consensus)
6. Annual NIV titrations in a sleep laboratory are recommended. (Consensus)

References


