CASE-CONTROL STUDY OF OBSTRUCTIVE SLEEP APNEA/HYPOPNEA SYNDROME IN OBESE AND NON-OBESE CHINESE CHILDREN

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Background: Obesity is a risk factor for obstructive sleep apnea/hypopnea syndrome (OSAHS) in adults. However, prevalence of OSAHS in children is not clear and relationship between obesity and OSAHS remains controversial. We hypothesized that obese Chinese children were more prone to have OSAHS than the normal population and there was a positive correlation between the degree of obesity and the severity of OSAHS.

Methods: Obese children were recruited from the endocrinology, respiratory and ENT clinics from April 2006 to January 2007. Weight, age and sex matched children were recruited as controls. Standard questionnaires were administered and standardized physical examination was carried out. Weight, height and waist circumference were measured, and weight height ratio (WHtR) was calculated for each child to describe central obesity. Lateral neck roentgenography, overnight sleep polysomnography (PSG), full blood count and arterial blood gas analysis were performed. As body mass index (BMI) in childhood changes substantially with age, BMI Z score was used to define obesity. Children with BMI Z scores >1.96 were considered obese. A/N ratio was calculated as the ratio of adenoidal depths to the nasopharyngeal depths on lateral cephalometric radiographs to measure the upper airway blockage. An adenoidal-nasopharyngeal ratio (A/N) ratio >0.67 was considered adenoid hypertrophy (ATH). OSAHS was defined as an apnea/hypopnea index (AHI) >5 or obstructive apnea index (OAI)>1.

Results: Ninety-nine obese children and 99 controls were recruited. Obese patients had significantly higher AHI and OAI, and lower sleep efficiency and MinSaO₂ than controls. The prevalence of OSAHS was significantly higher in obese children with or without ATH groups than their non-obese counterparts. (Odds ratio: 1.9, 95% CI: 1.21 to 4.7 and 108, 95% CI: 6.2 to 191 respectively). Obesity, tonsillar hypertrophy and adenoid hypertrophy were independent risk factors for OSAHS (P <0.001, 0.042 and 0.004 respectively), while age, sex, allergic rhinitis, and neck circumference were not predictors of childhood OSAHS. For those OSAHS children who were obese, there was a positive correlation between the degree of obesity and AHI (r=0.535, P<0.001), and an inverse correlation between obesity and MinSaO₂ (r=-0.507, P<0.001). A positive correlation was also found between degree of central obesity measured by WHtR and severity of OSAHS expressed by AHI and MinSaO₂ (r=0.341, P<0.001 and r=-0.345, P<0.001 respectively). End tidal CO₂ (ETCO₂), PaO₂, PaCO₂ and bicarbonate was within the normal range, no persistent CO₂ retention was documented.

Conclusions: Obesity is a risk factor for OSAHS and the degree of obesity is positively correlated with the severity of OSAHS. For obese OSAHS patients who had enlargement of adenoid and tonsils, the degree of reduction of AHI should be re-evaluated after adenotonsillectomy.