Obesity-related respiratory problems: a case report

Almen Lai-Na LAM 林麗娜* and Lettie Chuk-Kwan LEUNG 梁竹筠

1Department of Paediatrics and Adolescent Medicine, United Christian Hospital; 2Department of Paediatrics, Kwong Wah Hospital, Hong Kong

Introduction

Obesity is now in an epidemic proportion. By 2013, it was estimated that 1 in 4 children would be obese in Hong Kong. The long term metabolic and cardiovascular complications are well known but the significant respiratory involvement leading to life-threatening condition is not as well known. We presented a case of an obese girl who presented with respiratory failure secondary to various obesity related respiratory complications that mandated intensive care support.

Case

A 17-year-old girl with known mental retardation of unknown aetiology was admitted to the Paediatric unit through the emergency room for management of respiratory distress. She had history of fever, nasal congestions and runny nose, shortness of breathing noted by the mother for one day. The respiratory distress was more severe during sleep with snoring. There was no associated cyanosis. She got past medical history of mental retardation, obesity, obstructive sleep apnoea that was not treated with home continuous positive pressure ventilation because of poor maternal compliance. The mother frequently defaulted the paediatric and dietetic follow-ups. She was studying in a special school with poor attendance resulting in poor progress in her training at school.

Physical examination of the patient showing an obese girl with body weight of 103.8 kg (i.e. about 30 kg greater than 97th centile), body height of 160 cm (i.e. 50-75th centile) and body mass index of 39 kg/m². She got dysmorphic features including frontal bossing, hypertelorism, low set ears, prognathia, small broad hands and Simian crease over right hand. There was no neurocutaneous marker. Acanthosis nigricans was found at posterior neck and axilla. She showed signs of respiratory distress with tachypnoea (respiratory rate of about 30 per minute), use of accessory muscles, tachycardia (heart rate about 120 beats per minute), mild desaturation in room air with oxygen saturation of 90-95% that responded to oxygen supplement. Chest examination showed equal normal breath sound with no added sound. Cardiovascular examination showed normal jugular venous pressure. Apex was difficult to be palpated but there was no abnormal right ventricular impulse. Heart sounds were normal with no heart murmur. There was no ankle oedema.

Initial investigations showed acute respiratory acidosis with first venous blood gas showing pH 7.12, pCO₂ 11.3 kPa, pO₂ 2.3 kPa, HCO₃ 26.6 mmol/L and BE -5.2. Blood for sepsis work-up was unremarkable. Chest X-ray showed cardiomegaly with cardiothoracic ratio of about 0.7 with pulmonary congestion.

In view of Type 2 respiratory failure with CO₂ retention, the patient was transferred to PICU for respiratory support and closed monitoring. She was given bilevel positive pressure (BiPAP) support with inspiratory pressure of 16 cm H₂O and end-expiratory pressure of 6 cm H₂O yielding a tidal volume of 400-500 ml. Respiratory distress and clinical condition gradually improved. In view of fever and respiratory distress, flexible upper airway endoscopy was performed to rule out abscess in the upper airway. The main finding was pharyngomalacia with no evidence of other space-occupying lesion. Artieral blood gas was repeated after stabilization and it showed...

*Author to whom correspondence should be addressed.
Email: almenlln@yahoo.com.hk
compensated respiratory acidosis with mild hypercapnia both during wake and sleep (wake arterial blood gases: pH 7.38, pCO\(_2\) 6.1 kPa, pO\(_2\) 9.9 kPa, HCO\(_3\) 26.9 mmol/L, BE 1.3; and sleep arterial blood gases: pH 7.38, pCO\(_2\) 6.1 kPa, pO\(_2\) 10.9 kPa, HCO\(_3\) 26.5 mmol/L, BE 0.9) hence fulfilling the criteria of obesity hypoventilation syndrome. Diuretics were given for the heart failure. With improvement of heart failure, gradual stepping down of BiPAP support was achieved and only sleep BiPAP was required. However, Cheyne-Stokes breathing pattern was observed with transient desaturation SaO\(_2\) down to 85% during period of cessation of breathing movement. Sleep polysomography was performed that showed mixed apnoea / hypoapnoea with obstructive apnoea-hypopnoea index = 7.0/hour and total desaturation index (DI) = 7.0/hour. Cheyne-Stokes breathing pattern with prolonged circulation time, measured from the end of apnoea to the nadir of the oxygen saturation, was also noted during the sleep study (Figure 1). BiPAP titration showed best results with BiPAP of 10 cmH\(_2\)O on 6 cmH\(_2\)O. Home BiPAP support was discussed and provided. Sleep polysomography was planned to repeat in 1 year time to monitor the progress.

She was noticed to have high blood pressure with systolic blood pressure (SBP) up to 194 mmHg and diastolic blood pressure (DBP) up to 120 mmHg. There was also hypertensive retinopathy with flame-shaped hemorrhage bilaterally. She was found to have congestive heart failure with cardiomegaly and features of pulmonary congestion showed by chest X-ray. Echocardiogram showed left ventricular hypertrophy with impaired contractility with ejection fraction of 35% only. Electrocardiogram was unremarkable with no evidence of cardiomegaly or strain pattern. Cardiac enzymes were also normal.

![Figure 1](image.png)  
Figure 1. Cheyne-Stokes breathing with prolonged circulation time (normal <20 seconds).
24 hour ambulatory blood pressure monitoring showed increased nocturnal DBP load and daytime SBP and DBP load were greater than optimal level (<30-40%). There was also microalbuminuria with albumin/creatinine ratio 6.5 mg/mmolCr. Her blood pressure was well controlled with ramipril of 10 mg daily and amlodipine of 2.5 mg daily.

She also fulfilled the criteria of metabolic syndrome with (i) morbid obesity (BMI 39 kg/m², Waist-hip ratio 1.04 (normal <0.85) body fast percentage assessed by impedance 37.5% (90-97th centile)); (ii) hypertension; (iii) insulin resistance with Fasting Insulin 20.1 mIU/L and fasting glucose 4.7 giving an insulin sensitivity HOMA of 34.4% and insulin resistance of 2.9, although OGTT was unremarkable with normal HbA1C 5.3% ; (iv) and dyslipidemia with low high density cholesterol. There were mild elevated liver enzymes. She was put on a diet of 800 kcal/day after assessment by dietitian with increased activities organized by physiotherapist and occupational therapist daily. Home assessment to ensure safety and optimal rehabilitation after discharged were arranged. We targeted weight reduction at the rate of 0.5-1 kg/week. During her hospital stay, there was gradual weight reduction with BW 100.1 kg on discharge, i.e. weight loss of about 3 kg in 4 weeks time.

Discussion

Obesity is related to a number of respiratory problems, i.e. obstructive sleep apnoea, obesity hypoventilation syndrome. It could be further compounded by co-existing heart failure that result in Cheyne-Stokes breathing.

Obesity sleep apnoea (OSA)

Obstructive sleep apnoea is caused by the complete or partial collapse of pharyngeal wall leading to repetitive interruption of ventilation during sleep. Obese patients are more prone to OSA because of the narrower upper airway resulting from excessive fat deposition in soft palate, tongue, posterior and lateral oropharyngeal areas. The upper airway is also more prone to collapse because of the diminished stretching force on the pharynx as a result of a smaller lung volume secondary to a host of factors, i.e. decreased chest wall and lung compliance; decreased ventilatory muscle strength and endurance; diaphragmatic dysfunction secondary to increased adipose tissue deposition and mechanical disadvantage; respiratory muscle fatigue. There would also be ventilation and perfusion mismatch especially over the lung base in obese patients, which further affect effective ventilation in obese patients.¹

Patients with OSA would have disruptive snoring, witnessed apnoea or gasping, restless sleep, early morning headache, daytime hypersomnolence, nocturnal enuresis, or even behavioral changes. Questionnaires such as Epworth sleepiness scale or Paediatric Sleep Questionnaire enquiring the sleep quality would be helpful in diagnosing and determining the severity of OSA. Other investigation tools for diagnosis of OSA include overnight oximetry, overnight sleep polysomnography. There would be periodic dip in oxygen saturation measured by oximetry, nocturnal hypercapnia and sleep study showing apnoea-hypopnoea index >1.0. It is often associated with significant elevation in BP in children.²

OSA, if left untreated, may leads to severe cardiopulmonary complications, including development of hypertension, especially increased in diastolic blood pressure both during wake and sleep; left ventricular hypertrophy; ventricular dysfunction and heart failure; pulmonary hypertension and cor pulmonale; arrhythmia; coronary artery disease and atherosclerosis.³ Therefore, controlling sleep-related breathing disorder is very important in preventing long term development of severe cardiopulmonary complications. Treatment options included weight reduction, position therapy, mandibular advancement device and usage of positive airway pressure to prevent airway collapsibility. Surgical methods would include uvulopalatopharyngoplasty or tonsillectomy / adenoidectomy in younger patients.
Obesity hypoventilation syndrome

Obesity hypoventilation syndrome is defined as severe obesity BMI >30 kg/m², diurnal PaCO₂ >45 mmHg, FEV1/VC ratio >60%, in the absence of other known cause of hypoventilation. It was proposed to be due to leptin resistance in patient with obesity. Leptin is an adipocyte-derived hormone, besides having a negative feedback by activating specific receptors in hypothalamus associated with appetite suppression and increased in energy expenditure, it may also control breathing by stimulating ventilatory drive in response to an increase in ventilatory load. The suboptimal response to increase in respiratory load in obesity and increased risk of OSA would lead to further hypercapnia and thus aggravating respiratory failure.

Patients with obesity hypoventilation syndrome decompensate with infection or congestive heart failure and would present with acute respiratory failure. It may progress to severe cardiopulmonary compromise that may involve active resuscitation including intubation with ventilatory support, and treatment of aggravating factors like concomitant infections or heart failure Those who presented with less severe cardiorespiratory failure may be stabilized by non-invasive ventilation (NIV). For patients in steady state with hypercapnia but no acidosis, treatment depends on underlying diagnosis and severity of hypercapnia, usually, non-invasive ventilation by continuous positive pressure ventilation (CPAP) or other means of NIV would be given. NIV helped to relieve respiratory distress in hypoventilation by decreasing respiratory load, increasing thoracic compliance, improving nocturnal alveolar hypoventilation. The correction of CO₂ retention would help reset respiratory centre’s response to CO₂. Weight reduction of course is also the mainstay of treatment. Cheyne-Stokes breathing

Cheyne-Stokes breathing usually occurs in patients with heart failure. It is characterized by a crescendo-decrescendo pattern of breathing with central apnoea or hypoapnoea at the nadir of ventilatory effort. This is resulted from a decreased hypercapnia responsiveness combined with a prolonged circulatory time, leading to unstable ventilatory control and thus a pattern of periodic breathing. The circulation time is normally less than 20 seconds, and circulatory time would be increased in cases with increases in central blood volume and/or reductions in cardiac output. This leads to a feedback delay to chemoreceptors in lungs and carotid, leading to the over- and undershooting of ventilation, and thus a periodic breathing pattern. This phenomenon tend to occur later in the night as a result of the increased blood volume with interstitial fluid in the dependent area move to the intravascular compartment after the patient is supine for a long time.

The periodic breathing pattern could be demonstrated by a full night polysomnography, and sometimes, periodic breathing pattern could also be demonstrated in heart failure patient during wakefulness and exercise. Non-invasive ventilation including CPAP support can be used to relieve sleep disordered breathing patterns as well as improving the volume status in patient with congestive heart failure.

Conclusion

Obesity is associated with OSA, hypoventilation and Cheyne-Stokes breathing. The resulting respiratory insufficiency should be managed with reference to contribution from these three diseases.

References