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Case report

Nocturnal hypoventilation-a case report

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Abstract

Nocturnal hypoventilation is commonly seen in disorders affecting the function of the diaphragm or central respiratory drive mechanisms. The consequences of nocturnal hypoventilation such as sleep disturbances, poor gas exchange, day time sleepiness and effect on daily activities are similar irrespective of the precipitating underlying disorder. Here we report a rare case of nocturnal hypoventilation which posed a diagnostic and therapeutic challenge.

Keywords: Nocturnal hypoventilation, respiratory acidosis

Introduction

Nocturnal hypoventilation is a term used to describe insufficient gas exchange during sleep which becomes clinically significant in conditions where either the respiratory muscle pump is compromised or ventilator drive is reduced. The ability to maintain an adequate level of ventilation relies on three main factors: the respiratory muscle strength, respiratory work load and the central respiratory drive. If one or more of these factors become unbalanced, the individual is placed at risk of hypoventilation. When the cause is central, both hypercapnic and hypoxic ventilatory drive is reduced. Here we report a rare case of nocturnal hypoventilation associated with ventilator control abnormality which posed diagnostic and therapeutic challenge.

Case report

A 20 year old male was referred to neurology from a local hospital with recurrent episodes of seizures and respiratory distress. At the time of admission he was conscious but disoriented to time and place. Physical examination was remarkable for tachypnoea (respiratory rate 34/min), low oxygen saturation requiring supplemental oxygen, bilateral rhonchi and crepitations. Examination of other systems was noncontributory. Arterial blood gas (ABG) analysis while receiving supplemental oxygen showed respiratory acidosis. Chest X-ray was normal. He was managed in ICU with Bi-level positive airway pressure mode (BiPAP) with an inspiratory positive airway pressure (IPAP) of 12 cm H_2O, expiratory airway pressure (EPAP) of 6 cm H_2O and 5 L/min of supplemental oxygen. On improvement, he was subsequently shifted to ward with intermittent BiPAP support.

Our next plan was to detect the aetiology of hypoventilation and seizures. Pulmonology opinion was obtained to assess the related causes of respiratory failure. He was born out of consanguinous parentage and childhood was characterised by mild delay in motor development, hyperactivity, poor scholastic performance and recurrent seizures. At the age of 10 years, he had been admitted with generalised tonic, clonic seizures during sleep. He had sustained cardiorespiratory arrest from which he was resuscitated and recovered after ventilatory support. It was documented that he had type 2 respiratory failure. Investigations to assess the cause

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of hypoventilation including tests for diaphragmatic palsy were inconclusive. On clinical improvement, he was discharged on antiepileptics and Home BiPAP with a diagnosis of post-viral demyelination.

During present admission, investigations such as fluoroscopy, electroneuromyogram (ENMG), pulmonary function test (spirometry) and electroencephalogram (EEG) were obtained. EEG revealed epileptiform abnormality arising from left frontotemporal region. ENMG report showed normal phrenic nerve conduction. Pulmonary function test (spirometry) revealed severe restriction with 16% reduction in supine forced vital capacity (FVC) compared to sitting (usually expected - 30-50% reduction in diaphragmatic palsy cases). Fluoroscopy showed normal diaphragmatic movement and no features to suggest palsy.

While in the ward, he had worsening respiratory distress during sleep, with ABG showing severe respiratory acidosis (pH-7.005, PCO_2-135 mm Hg, PO_2 – 203 mm Hg and HCO_3^- of 21.3 mmol/L) and seizures for which he was intubated. On thethird day of intubation, he was extubated. That same night, he had to be reintubated and ventilated due to drowsiness and worsening acidosis. His diagnosis and treatment strategy were reviewed. As he had hypoventilation disorder, which was recurrent during sleep, and was associated with seizures, nocturnal hypoventilation syndrome was suspected. On the second day of reintubation, the patient was extubated, after which a trial of a newer mode of noninvasive ventilation (NIV) targeting alveolar minute ventilation- iVAPS (Intelligent Volume Assured Pressure Support) with maximum pressure support of 25 cm H_2O, minimum pressure support of 8 cm H_2O with 5 L/min oxygen was given. The patient tolerated this well, stabilised and could be discharged with no further recurrence of hypoventilation or seizures on nocturnal iVAPS.

Discussion

Nocturnal hypoventilation refers to deterioration in alveolar ventilation during sleep with consequent changes in respiratory physiology. During sleep, the tonic neural input of respiratory drive to respiratory muscles and chest wall is reduced leading to increase in upper airway resistance. These physiologic changes cause a small degree of hypoventilation with little consequence in normal subjects. However, when it occurs in individuals who already have respiratory muscle weakness such as altered chest wall mechanics, compromised gas exchange or abnormal respiratory drive, this will lead to significant alteration in ventilation and gas exchange. Common disorders associated with nocturnal hypoventilation are given in *Table 1*:

Table 1: Disorders associated with nocturnal hypoventilation

Neuromuscular disorders

Stable or slowly progressive
- Previous poliomyelitis
- High spinal cord injuries
- Spinal muscular atrophy
- Myotonic dystrophy

Rapidly progressive disorders
- Motor neuron disease

Chest wall abnormalities
- Kyphoscoliosis
- Post tuberculosis sequelae
- Obesity hypoventilation syndrome

Lung disorders
- Chronic obstructive pulmonary disease
- Cystic Fibrosis
- Overlap syndrome (lung disease with obstructive sleep apnea)

Ventilatory control abnormalities (to come first followed by others)
- Brain stem injuries: Stroke, infection, tumour
- Congenital central hypoventilation
- Primary alveolar ventilation

Since none of the diagnostic tests for neuromuscular disorders, chest wall abnormalities and lung disorders showed any abnormality in our patient, the possibility of ventilator control abnormality and the presence of neurological problems such as developmental delay, seizure disorder likely to cause nocturnal hypoventilation was considered.[1-3] Major causes of ventilatory control abnormalities are congenital central hypoventilation, primary alveolar hypoventilation and brain stem injury.[4] Congenital central hypoventilation is characterised by alveolar hypoventilation due to disordered respiratory control, anatomic and physiologic autonomic nervous system dysregulation.[5] Individuals with congenital central hypoventilation have reduced tidal volume and monotonous respiratory rates

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during sleep in more severe cases. These patients become more hypoxaemic and hypercarbic but they lack ventilatory and arousal responsiveness to these endogenous ventilatory derangements during sleep as well as the perception of asphyxia during wakefulness at rest and at exercise.

Primary alveolar hypoventilation is characterised by arterial hypercapnia and develops when alveolar ventilation falls below a critical value. This is a rare condition occurring due to diminished responsiveness of respiratory centre itself. Its main causes include intrinsic bronchopulmonary disease, neuromuscular defects and primary respiratory centre damage. This diagnosis can be made only when hypoventilation occurs in the absence of functional abnormality of the lung, chest muscle and motor nerve.[6] Primary alveolar hypoventilation usually occurs during sleep with periods of apnoea.[7,8]

Hence our patient had a rare cause of nocturnal hypoventilation, a ventilator control abnormality which was treated with a newer modality of noninvasive ventilation (NIV) targeting alveolar minute ventilation.

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