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Case Report Open Access

Obesity Hypoventilation Syndrome and Pulmonary Hypertension Case with Excessive Daytime Sleepiness

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Abstract

Obesity and pulmonary hypertension (PH) are two conditions that frequently coexist in clinical practice and also the association between obesity and hypersomnolence has long been recognized. Pulmonary hypertension is more common (50% *vs.* 15%) and more severe in patients with OHS than in patients with OSAS. Here, we present an obese man with very severe excessive daytime sleepiness and very serious pulmonary hypertension, who only gave respond to AVAPS and oxygen treatment.

Keywords: Obesity hypoventilation syndrome; Pulmonary hypertension; Excessive daytime sleepiness

Introduction

There are a rising number of patients with severe pulmonary hypertension (PH) due to alveolar hypoventilation. In obstructive sleep apnea (OSA) and overlap syndrome, PH is rare and pulmonary artery pressure (PAP) is only mildly elevated, however, in alveolar hypoventilation, PH is more frequently observed and of higher severity. Obesity and pulmonary hypertension (PH) are two conditions that frequently coexist in clinical practice and also the association between obesity and hypersomnolence has long been recognized [1]. Sleep related breathing disorders (SRBD) is a large spectrum including the pathologies such as obstructive sleep apnea syndrome (OSAS) and obesity hypoventilation syndrome (OHS). In 2008 PH update; classified SRBD in group 3 as PH due lung diseases and hypoxia [2]. Pulmonary hypertension defined by a mean pulmonary artery pressure exceeding 25 mmHg on right heart catheterization, Is more common (50% *vs.* 15%) and more severe in patients with OHS than in patients with OSAS [3-7].

Case Report

A 29 years old young man admitted to our sleep clinic with excessive daytime sleepiness (EDS). He was a prayer leader of a town mosque in west site of Turkey. He was complaining about sleepiness on duty even on talking and praying in front of people. He was obese with a BMI: 38.5 kg/m^2, dyspneic, orthopneic and he had tendency to sleep, cyanosis in his lips and fingers, flapping tremor in his hands, chemosis in his conjuctivas and bilateral pretibial edema. His arterial blood gases in room air showed hypoxemia and hypercapnia (pH: 7.39, pO_2: 56 mmHg, pCO_2: 46 mmHg, HCO_3: 32 mmol/L, $SatO_2$: 88%) and his pulmonary function tests revealed severe restrictive defect (FVC: 2 ml, 14 ml, 41%, FEV_1: 1, 56, 36%, FEV_1/FVC: 73)

In his polysomnographic (PSG) study severe OSAS (AHI: 36.2/hr.) and very severe nocturnal oxygen desaturations (NOD) that were not related to respiratory events were detected with a min $SATO_2$: 41% and mean $SATO_2$: 69%. In addition to his desaturations also he had serious hypercapnia while sleeping (pH: 7.37, pO_2: 44 mmHg, pCO_2: 62 mmHg HCO_3: 36 mmol/L, $SatO_2$: 77%). In his ECG sinus tachycardia, complete right bundle branch block and in his echocardiography severe PH (systolic PAB: 110 mmHg, mean PAB: 70 mmHg), leftward deviation of the interventricular septum and tricuspid regurgitatiom were detected. We diagnosed him as OHS with all these OHS diagnostic criteria. We applied BPAP-ST with nasal O_2 but no complete response had been achieved and then AVAPS+O_2 therapy had a very good success on his respiratory and sleep problems and pulmonary hypertension. At 1st year control, he lost weight (BMI: 32 kg/m^2), got rid of daytime sleepiness, edema, cyanosis, chemosis and most importantly his pulmonary arterial pressures were decreased (systolic PAB: 62 mmHg, mean PAB: 43 mmHg) with a minimum tricuspid leakage. He is very compliant to his devices and he is very happy now in his work and family life.

Discussion

Multiple studies have shown a higher prevalence of SRBD in patients with pulmonary hypertension as well as an increased prevalence of pulmonary hypertension in patients with SRDB (17% to 53%); and factors such as daytime PO_2, BMI, and AHI are significantly associated with both [3].

The diurnal hypoxemia, hypercapnia, and acidosis associated with OHS are mediators of PH. Secondary contributors to PH in patients with OHS are restrictive lung disease related to severe obesity and the wide intrathoracic pressure shifts in the respiratory cycle due to increased upper airway resistance. Upper airway obstruction results in profound negative intrathoracic pressures during inspiration, up to −70 mmHg. These negative intrathoracic pressures augment RV filling causing a leftward shift of the intraventricular septum which impedes LV filling and thus elevates pulmonary venous pressures and lowers LV stroke volume. This mechanism accounts for the presence of Pulsus paradoxus in patients with OHS and severe lung disease. The pulmonary vascular beds initial response to hypoxemia is vasoconstriction at the pulmonary arteriolar and capillary level. With relief of the hypoxemia this vasoconstriction is reversible. However with chronic hypoxemia, as seen in OHS, pulmonary artery remodeling occurs, and over times the pulmonary arterial

hypertension transitions from a process of vasoconstriction to one of endothelial dysfunction, arterial wall thickening, and fibrosis. At this point the PH becomes much more difficult to reverse [8].

Also patients with OHS was found to be more likely to carry a diagnosis of congestive heart failure (odds ratio 9, 95%, CI 2.3 to 3.5), angina pectoris (odds ratio 9.95%, CI 1.4 to 57.1), and cor pulmonale (odds ratio 9.95%, CI 1.4 to 57.1) [9].

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