Case Report

Hypoxic hepatitis owing to obesity hypoventilation syndrome: case report

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Received January 19, 2016. Accepted February 14, 2016

Abstract

Obesity hypoventilation syndrome (OHS) is an underdiagnosed complication of obesity. It is associated with more morbidity when compared with eucapnic obesity. Here, we are reporting a case of OHS associated with hypoxic hepatitis.

KEY WORDS: Hypoventilation, hypoxic hepatitis, obesity

Introduction

The spectrum of respiratory complications in obese individuals is diverse. They may present normal ventilation in the awake and sleeping state, or sleep-disordered breathing with maintained ventilation during awake state, or awake hypercapnia without other causes for hypoventilation. Hypoxic hepatitis or ischemic hepatitis can be owing to various causes such as cardiac failure, respiratory failure, sepsis, and anemia. We are presenting a patient with obesity hypoventilation syndrome (OHS) and hypoxic hepatitis. There are only scanty reports of similar cases in the literature.[1] OHS is an underdiagnosed entity. Early diagnosis and intervention can contribute to improved outcome of this condition, which if untreated is associated with increased morbidity and mortality.

Case Report

A 42-year-old lady was brought to the casualty with history of altered level of consciousness of acute onset. There was no history of fever, headache, vomiting, decreased appetite, and yellow discoloration of urine. She was not a hypertensive or diabetic patient, not on any drugs, and did not show any addictions. There was history of excessive snoring at night and daytime sleepiness. On examination, her height was 160 cm, and she weighed 110 kg. Her pulse rate of 72 beats/min, blood pressure of 130/70 mm of Hg, and SpO2 of 40% improved to 96% with supplemental oxygen therapy. There was no evidence of pulmonary artery hypertension, focal neurological deficits, or cardiac failure. Investigations showed the following values: hemoglobin: 11.2 g%, total leukocyte count: 13,600/mm^3, erythrocyte sedimentation rate: 8 mm in the first hour, random blood sugar: 115 mg%, serum creatinine: 1.5 mg%, serum sodium: 131 mEq/L, and serum potassium: 4.7 mEq/L.

On day 1 the values were as follows: serum bilirubin total: 2.9 mg%, direct fraction: 1 mg%, serum glutamic-oxaloacetic transaminase (SGOT): 3,093 units/L, serum glutamic-pyruvic transaminase (SGPT): 1,573 units/L, serum alkaline phosphatase (ALP): 85 units/L, serum total protein 6.8 g%, albumin: 3.6 g%, prothrombin time 7 s more than the control, and International Normalized Ratio = 1.55. Anti-HAV, HEV, HBsAg, anti-HBc IgM, anti-HCV, HIV, and ANA were negative. Serum bicarbonate level was 29 mmol/L. Chest X-ray and ECG were normal. After giving supplemental oxygen, her level of consciousness improved, and her repeat liver function tests also started improving. On day 2 the values were as follows: SGOT: 1,390 units/L, SGPT: 930 units/L. On day 3, SGOT: 297 units/L, SGPT: 315 units/L. On day 4, SGOT: 141 units/L, SGPT: 331 units/L.

Because of the persistent hypoxemia (on withdrawing supplemental oxygen, her SpO2 dropped to less than 50%), she was put on noninvasive ventilation. Her transaminases returned to normal level within 10 days. She was sent home on bilevel positive airway pressure support.

Access this article online	
Website: http://www.ijmsph.com	Quick Response Code:
DOI: 10.5455/ijmsph.2016.19012016369	

Discussion

OHS is the combination of obesity [body mass index (BMI) > 30 kg/m^2] with alveolar hypoventilation in the daytime ($paCO_2$ > 45 mm of Hg, paO2 < 70 mm of Hg), in the absence of other causes for hypoventilation.[2] Majority of the patients with OHS present obstructive sleep apnea, while others show sleep hypoventilation which is characterized by an increase in $paCO_2$ by more than 10 mm of Hg when compared with awake state or hypoxia during sleep not explained by obstructive sleep apnea.[3] The incidence of OHS is around 50% in obese people with BMI >50 kg/m^2. In the early stages of hypercapnia, patients are usually asymptomatic. As the hypercapnia worsens, the symptoms include exertional dyspnea, disturbed sleep, daytime increased sleepiness, delirium, myoclonus, and seizures (carbon dioxide narcosis). Pulmonary hypertension and cor pulmonale can develop. OHS may be diagnosed during routine evaluation or it may manifest as an acute on chronic exacerbation as in this case. When compared with obese people who show eucapnia, those with OHS show higher chances of developing systemic hypertension, cardiac failure, and angina. Pulse oximetry reading < 94% and serum bicarbonate value > 27 mmol/L can be used as screening tools to pick up this condition. Serum bicarbonate value >27 mmol/L is highly sensitive (92%) in detecting respiratory acidosis. In this case also, the serum bicarbonate was consistently showing high value. Arterial blood gas analysis could not be done in this case.

Another interesting complication of hypoxia in this case is the associated hypoxic hepatitis as evidenced by the marked rise in transaminases (SGOT: 3,093 units/L, SGPT: 1,573 units/L) and the fall in transaminases by 50% within 72 h. Other causes for the marked rise in transaminases such as viral hepatitis, toxins, and hypotension have been excluded in this case by the relevant investigations. The diagnosis of hypoxic hepatitis is entertained in the typical clinical scenario of cardiogenic or circulatory shock or respiratory failure, with a rise in transaminases at least 20 times the upper limit of normal, fall in transaminases by 50% within 72 h, in the absence of other causes of liver damage. Previously, it was thought that hypotension is a prerequisite for the development of this condition. In fact, hypotension is seen in only half of the cases.[4] The pathogenetic mechanisms of hypoxic hepatitis include passive congestion, ischemia, and arterial hypoxemia. Other laboratory abnormalities in hypoxic hepatitis include rise in lactate dehydrogenase, rise in bilirubin less than four times the upper limit of normal, alkaline phosphatase less than twice the upper limit of normal, and mildly impaired synthetic function of the liver.

Conclusion

OHS is likely to be missed unless there is a high index of suspicion. Early diagnosis is needed to ensure a good outcome.

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How to cite this article: Jacob MG, Rajesh KR, Asokan A, Lekshmi S. Hypoxic hepatitis owing to obesity hypoventilation syndrome: case report. Int J Med Sci Public Health 2016;5: 1979-1980

Source of Support: Nil, **Conflict of Interest:** None declared.