Case Report

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Bariatric Surgery in Morbid Obesity Patient Can Cure Pulmonary Hypertension and Obstructive Apnea: A Case Review

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Abstract

Background: Morbid Obesity can complicate and causing not only metabolic syndrome, but also can cause severe pulmonary hypertension (PH) and obstructive sleep apnea (OSA). Even the OSA itself can also cause pulmonary hypertension and it has been well documented. Series of medical disease can be reversed if we can reduce body mass index (BMI) in our obesity patient.

Method: We present a case of a 41 year old lady with severe OSA secondary to morbid obesity who also was found to have moderate pulmonary hypertension. Her weight was out of control since teenage years. During her initial presentation, her BMI was 57.6 kg/m^2. She often complained of shortness of breath on exertion and failure symptoms. She had a functional classification of NYHA class II. She was given initial treatment of CPAP and dietary restriction. But her BMI was still slow to improve. Subsequently, she underwent bariatric surgery with marked improvement in her BMI.

Findings: Her functional class improved to NYHA class I and her failure medication can be stopped. Her pulmonary hypertension also cured to near normal value. She returns to normal life with no complication from the surgery.

Conclusion: Reduction of BMI in morbid obesity can be dramatically achieved by bariatric surgery which can reverse and established medical complications.

Keywords: Obesity; Pulmonary hypertension; Obstructive apnea

Introduction

According to the National Health and Morbidity Survey, the incidence of obesity in Malaysia has been showing a dramatic upward trend over the last 20 years. In 1996, the average rate of obesity at state level ranged from 3 to 5%, however in 2015 the national rate of obesity had climbed to 33.4% [1]. The World Health Organization (WHO) defines overweight as a body mass index (BMI) of over 25 kg/m^2. A BMI over 30 kg/m^2 is considered obese, and is further subdivided into Obesity class I, Obesity class II, and Obesity class III [2] **(Table 1)**.

Classification	BMI (kg/m^2)
Underweight	≤ 18.49
Normal	18.50-24.99
Overweight	≥ 25.00
Pre-Obese	25.00-29.99
Obese	≥ 30.00
Obesity Class I	30.00-34.99
Obesity Class II	35.00-39.99
Obesity Class III	≥ 40.00

Table 1: Classification of nutritional status based on BMI.

Note: Adapted from Obesity: Preventing and Managing the Global Epidemic (p.8), by the World Health Organization, 2000, Geneva, Switzerland: World Health Organization.

Obstructive sleep apnea is defined as frequent episodes of sleep-related hypopneas or apneas in respiration lasting longer than 10 seconds, corresponding to a partial or complete obstruction of the upper airways despite continuous chest and abdominal respiratory movements [3].

Severity	Apnea-Hypopnea Index
Mild	5-15
Moderate	15-30
Severe	≥ 30

Table 2: Severity of OSA based on AHI.

This condition often leads to daytime somnolence and cardiopulmonary remodeling. The link between OSA and pulmonary hypertension is well documented. The reported prevalence of pulmonary hypertension in patients with OSA

varies from 17% to 70% depending on the method of study, definitions and cut off values for diagnosis [4]. OSA can be categorized by severity based on the Apnea-Hypopnea Index (AHI) (**Table 2**).

Note: Adapted from " Obstructive Sleep Apnea " by Balachandran, Jay S. and Patel, Sanjay R, 2014 Annals of Internal Medicine, 161, ITC1.

Mild OSA: AHI ≥ 5 but <15 events per hour • Moderate OSA: AHI ≥ 15 but <30 events per hour • Severe OSA: AHI ≥ 30 events per hour • The OSA syndrome: AHI ≥ 5 events per hour with daytime sleepiness.

This case report intends to bring attention to the relationship between morbid obesity, OSA and pulmonary hypertension, and how the management of one condition can affect the other.

Case Report

A 41-year-old morbidly obese (BMI 57.6 kg/m^2) Malay lady was referred to the cardiology team for preoperative workup. She had a functional class of NYHA II with complaints of dyspnea on exertion, as well as a background of hypertension, diabetes mellitus, dyslipidemia, and non-alcoholic fatty liver disease.

She had previously been started on subcutaneous liraglutide as an antiobesity agent and initially showed positive results in the first 6 months. Unfortunately, her weight plateaued over the following 18 months. She was then referred for bariatric surgery after failing second-line antiobesity agents. Prior to surgery as is customary, she underwent a preoperative assessment.

Preoperative workup for her involved input from a multidisciplinary team, made up of cardiologists, endocrinologists, anaesthetists and bariatric surgeons.

Investigations

Her baseline ECG showed normal sinus rhythm with no significant abnormalities. However her echocardiogram revealed severe pulmonary hypertension, with a systolic pulmonary arterial pressure (SPAP) of 62 mmHg.

To confirm the diagnosis of pulmonary hypertension, a right heart study was performed which elucidated a mean pulmonary arterial pressure (MPAP) of 45 mmHg, and pulmonary capillary wedge pressure (PCWP) of 22 mmHg. This result is consistent with moderate pulmonary hypertension of post-capillary cause. Coronary angiogram was also performed for her and which showed normal coronary arteries.

She underwent a sleep study which confirmed obstructive sleep apnea with an apnea-hypopnea index (AHI) of 117 (severe OSA). Her pulmonary function test indicated a restrictive pattern of disease.

Treatment

She was referred to otorhinolaryngology and was started on continuous positive airway pressure (CPAP) at home. Otherwise, she remained on her regular medications: liraglutide, furosemide, valsartan and legalon. No specific pulmonary arterial hypertension treatment was commenced as her case of PH is secondary to lung disease and hypoxia.

3 months afterwards during a pre-operative reassessment, an echocardiogram was repeated and showed an improvement in the degree of pulmonary hypertension – the SPAP had reduced to 58 mmHg. She had managed to lose another 6 kg before the operation, and her functional class improved to NYHA I. The team decided to proceed with the surgery and she successfully underwent laparascopic roux-en-Y gastric bypass.

Progress

By 3 months post-operation, her weight was down from 153 kg to 115 kg and she no longer required diuretics for the management of heart failure symptoms. Repeat echocardiography showed significant reduction in her SPAP from 62 mmHg initially to 37 mmHg. 11 months post-operation, her weight had further reduced to 89 kg, and SPAP on echocardiography was 36 mmHg (near normal). A summary of the progress of her weight loss and reduction in SPAP is summarized in **Table 3** below.

	Baseline	Pre operation	1 month Post operation	3 months Post operation	11 months Post operation
Weight	153 kg	147 kg	125 kg	115 kg	89 kg
BMI	57.6 kg/m^2	55.3 kg/m^2	47.0 kg/m^2	43.3 kg/m^2	33.5 kg/m^2
SPAP	62 mmHg	58 mmHg		37 mmHg	36 mmHg

Table 3: Summary of the patient's weight and SPAP following treatment.

Discussion

It is important to first understand the mechanism in which OSA leads to pulmonary hypertension. A pioneer study by the Bologna Sleep Laboratory (University of Bologna) in 1971 showed that pulmonary arterial pressure rises during sleep. This change is universal in all subjects. However, there was no significant change from one sleep stage to the next in normal test subjects. Similarly, despite the fact that average alveolar ventilation falls during sleep, the difference in values from one sleep stage to the next is non-significant. Neither pulmonary arterial pressure nor blood-gas values reached pathological levels during sleep. These results are in keeping with the fact that during sleep the physiologic ventilator response to hypoxia and hypercapnia is reduced.

However, in OSA patients, not only are systemic and pulmonary arterial pressures higher during sleep compared to wakefulness (similar to normal patients), they also increase significantly as they progress into deeper sleep. Blood gas values also indicate that ventilation diminishes progressively in the subsequent stages of slow-wave sleep with a sudden, steeper dip during REM sleep [5].

Cardiac remodeling in OSA patients have been attributed to exposures to hypoxemia, catecholamine excess, elevations in blood pressure, and marked intrathoracic pressure swings which affect the preload and afterload and left heart transmural pressures [6].

The mechanism proposed is thought to be related to the recurrent upper airway obstruction and the subsequent periods of apnea. Alveolar hypoxia leads to pulmonary vasoconstriction, which causes the pulmonary vascular resistance to rise and as a consequence increasing the pre-capillary pulmonary arterial pressure. Ventilatory efforts against a closed upper airway can lead to negative intrathoracic pressures in excess of -80cmHO. This high negative pressure causes an increase in venous return, which leads to an upsurge in the right ventricular preload and therefore stroke volume. These effects combined lead to an overall higher pulmonary blood flow. Higher venous return also causes the interventricular septum to shift to the left and reduces the left ventricular compliance. Furthermore, left ventricular afterload is elevated due to the high negative intrathoracic pressure during inspiration, which further hampers the left ventricular function. This then leads to congestion and contributes to a relative increase in pulmonary venous pressure [4].

Pulmonary vasoconstrictor response to hypoxia appears to vary considerably between individuals. In a study by Sajkov et al., patients with OSA and PH had a more pronounced vascular pressure response to hypoxia than in control subjects (those with OSA but not PH) [7]. This would indicate that some physiological variability between individuals exists which predisposes some to the development of PH secondary to OSA. It would also explain why the correlation between OSA and PH is not 100%. Research in this area is still lacking, however there has been some increase in interest in this field, and more data should become available in the near future.

In our case here, the BMI was reduced dramatically to mild obesity level. This will reduce all physiological effect of obesity to the lungs and subsequently to the pulmonary pressure. This was clearly benefits the patient with improvement of original presentation symptoms.

Conclusion

Morbid Obesity can complicate patient into a lot of other medical diseases. By improving BMI level into near normal level, all the symptoms will be improved and can be objectively seen as well with investigations such as echocardiogram for pulmonary pressure. We recommend bariatric surgery for morbid obesity patient for curing medical complications.

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