

Noninvasive Ventilation Influenced the Survival of Extreme Super-Super Obese with Obesity Hypoventilation Syndrome

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Abstract

Recent studies conclude noninvasive ventilation (NIV) as a standard first-line therapy for any respiratory failure. The efficacy of NIV in obesity is well defined. In this context, the effectiveness of NIV in extreme super-super obese patients with obesity hypoventilation syndrome is not well studied, and the usage of NIV is controversial because of rare incidence. However, identifying the right patient, right timing, right clinical expertise, correct positions, and right appropriate NIV settings can produce positive results. Therefore, we present a unique case of a 32-year-old male weighing 283 kg, with body mass index >92 kg/m² and carbon dioxide retention, whose symptoms were worsening despite adequate conventional treatment. He received continuous NIV for 70 h using a standard protocol with close monitoring. There was improvement in the patient vitals and medical condition with significant reduction in arterial carbon dioxide tension, leading to reduction in the length of intensive care unit stay and prevention of further complications. Long-term research is encouraged to validate NIV effectiveness in this group of patients.

Keywords: Expiratory positive airway pressure, inspiratory positive airway pressure, noninvasive ventilation, obesity hypoventilation syndrome, obstructive sleep apnea

INTRODUCTION

Obesity is a universal health concern in recent years, and it is one of the leading causes of preventable deaths in Saudi Arabia, with an obesity prevalence of 33.7%.^[1] According to the Obesity Medicine Association, obesity is “a chronic, relapsing, multifactorial, neurobehavioral disease, wherein an increase in body fat promotes adipose tissue dysfunction and abnormal fat mass physical forces, resulting in adverse metabolic, biomechanical, and psychosocial health consequences.”^[2] The body mass index (BMI) is a crude and easy tool that is commonly used to measure obesity, and it is the value derived from a person’s weight in kilograms (kg) divided by the square of his/her height in meters (m²). In general, the World Health Organization quotes that a BMI ≥ 30 kg/m² as obese. Obesity is subcategorized into the following grades: Grade 1 (BMI 30 to <35 kg/m²), Grade 2 (BMI 35 to <40 kg/m²), and Grade 3 (BMI > 40 kg/m²).^[3] Table 1 summarizes the Spanish Society for the study of Obesity (SEEDO) classification.^[4] Despite increased awareness over the past decades, obesity, particularly Class III, is continuing to rise.^[5-7] With a rise in obesity, the incidence of obesity hypoventilation

syndrome (OHS) has increased significantly, with almost 50% of people with BMI >50 kg/m² being hospitalized globally.^[7] OHS is a combination of obesity (BMI >30 kg/m²), daytime hypercapnia (arterial carbon dioxide tension >45 mmHg), and sleep-disordered breathing, after exclusion of other disorders that may cause alveolar hypoventilation. Besides, obstructive sleep apnea (OSA) is also associated with hypoventilation in 90% of OHS patients.^[5,6] Currently, OHS has become a significant indication for noninvasive ventilation (NIV).^[7-9] However, there is no report from Saudi Arabia on the use of NIV in extreme super-super obese-associated OHS. Choosing to administer NIV in such patients is unique and challenging. In this case report, we describe the first-ever successful application of NIV in an extreme super-super obese individual and the possibility of its use effectively in the intensive care unit (ICU) in future.

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Table 1: Obesity classification (Spanish Society for the Study of Obesity)

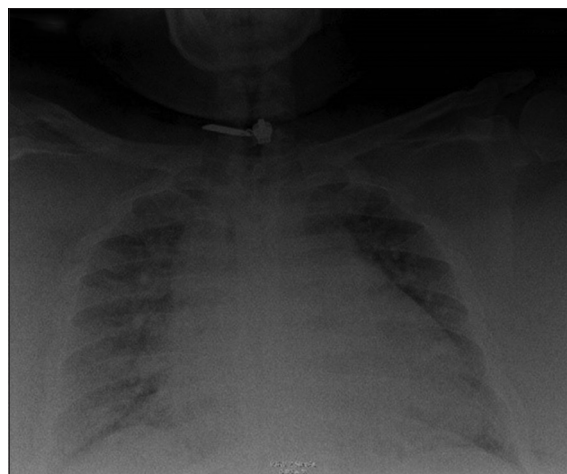
BMI (kg/m ²)	Classification
<18.5	Underweight
18.5-24.9	Normal weight
25.0-26.9	Overweight
27.0-34.9	Class I obese
35.0-39.9	Class II obese
40.0-49.9	Class III obese
50-59.9	Class IV super obese
>60	Class V extreme super-super obese

BMI: Body mass index

CASE REPORT

A 32-year-old Saudi male with no comorbidities and independent in his activities of daily living presented with a history of breathlessness on and off for the past 3 months. It worsened with travel to 2200-m high altitude. In the emergency room, the patient was drowsy with the chief complaints of severe dyspnea, headache, myalgia, and swelling and pain in the lower limb. There was no history of any chronic respiratory diseases. He was afebrile. His heart rate was 115–120 beats/min and regular, respiratory rate was 35–40 breaths/min, blood pressure was 150/90 mmHg, and oxygen saturation was 74% on room air. Physical examination revealed a weight of 283 kg, BMI >92 m², height of 175 cm with a neck circumference of 55 cm, cyanotic nail beds, mild jugular distension, and higher central fat distribution. He was drowsy but easily arousable. Glasgow Coma Scale (GCS) score was 11. In the emergency room, he was treated with O₂ 15 L/min, and the oxygen saturation rose to 90%–91%. His blood biochemistry was within normal limits. His treatment included intravenous fluids (5% dextrose normal saline with 20 mEq potassium chloride added, hydrocortisone 100 mg TID, amoxicillin 1 g TID, inhaled ipratropium bromide 6th hourly, inhaled salbutamol 6th hourly, furosemide 20 mg BID, and unfractionated heparin 7500 SC TID). Arterial blood gas analysis (ABG) was obtained, and sputum for cultures was collected. Anteroposterior chest X-ray revealed bilateral increased bronchovascular markings and cardiomegaly [Figure 1]. Electrocardiogram showed sinus tachycardia. Echocardiography showed dilated right ventricle and mild tricuspid regurgitation. A computed tomography (CT) scan could not be performed to detect pulmonary embolism because the CT table could not accommodate his weight. His initial ABG in the emergency room showed hypoxia and respiratory acidosis (pH 7.26, PCO₂ 86 mmHg, PO₂ 27.9 mmHg, and HCO₃ 29.6 mmol/L).

The diagnosis at admission was OHS with OSA, cor pulmonale, and cellulitis. His condition deteriorated subsequently with desaturation to 76% even with 15 L/min of oxygen. Therefore, the patient was transferred to the ICU for further management. A trial of NIV was given along with other conventional treatment. ABG repeated before NIV showed severe respiratory

**Figure 1:** X-ray admission

acidosis (pH 7.15, PO₂ 65 mmHg, PCO₂ 113 mmHg, and HCO₃ 27.6 mmol/L) [Table 2]. Intubation and invasive ventilation was planned if the respiratory rate increased to >40 breaths/min or if there was any drop in GCS level. To avoid aspiration and to deliver adequate ventilation, high Fowler's position was adopted. The NIV-Bi-level ST Philips Respironics V 60, California, USA) is a single-circuit, pressure-targeted, nonvent mask provided with a calibrated leak. The following settings were used: respiratory rate was set at 25 b/min as backup, inspiratory positive airway pressure (IPAP) of 10 cmH₂O was initiated to overcome upper airway resistance and to reduce the work of breathing (WOB), and expiratory positive airway pressure (EPAP) of 5 cmH₂O was initiated to prevent alveoli collapse at the end of each respiratory cycle. To provide more time for expiration and to reduce WOB, an inspiration and expiration time ratio was set at 1:4. After 2 h of NIV, ABG showed pH 7.12, PCO₂ 136 mmHg, and PO₂ 77.3 mmHg. IPAP was increased in steps of 3 cmH₂O and EPAP in steps of 1 cmH₂O, every 15 min based on the WOB, minute ventilation, respiratory rate, and ABG results. Adequate tidal volume was reached with IPAP 22 cmH₂O and EPAP 10 cmH₂O (as tolerated by the patient). Subsequently, as the patient improved, NIV could be gradually reduced to IPAP of 12 cmH₂O and EPAP of 5 cmH₂O based on ABG and respiratory mechanics. FiO₂ was adjusted between 45% and 80% to keep the SpO₂ 92%. NIV was disconnected only during feeding. Weight reduction also being a potential therapy to improve patients' long-term medical condition, injection liraglutide 3 mg subcutaneous once daily with a plan to escalate dose from 0.6 mg to 3 mg each week from day 2 was considered.

The patient was monitored closely with hourly recording of all vitals; ABG was repeated at 2 h, 4 h, and every 6 h [Table 2]. After 70 h of continuous NIV, the patient's clinical condition improved significantly, along with a reduction in PCO₂ to 42.8 mmHg. The reduction in PCO₂ over 70 h of NIV is shown in Figure 2. The post-NIV ABG result at the 6th h was within normal range. The patient was discharged to the ward on day 8 as shown in Figure 3.

Table 2: Vitals and blood gas parameters before noninvasive ventilation and during and after noninvasive ventilation

Parameter	Before NIV	NIV-70 h													
		At 2 nd	At 4 th	At 10 th	At 16 th	At 22 nd	At 28 nd	At 34 th	At 40 th	At 46 th	At 52 nd	At 58 th	At 64 th	At 70 th	At 6 th post-NIV
HR	120	125	126	120	110	108	106	105	100	98	98	96	95	93	92
BP	150/90	155/99	150/90	148/92	145/90	145/90	143/88	145/88	145/85	140/80	140/80	138/80	135/77	135/75	137/75
RR	40	35	32	30	27	27	25	25	23	25	23	24	22	22	22
ABG (samples)	ABG (1)	ABG (2)	ABG (3)	ABG (4)	ABG (5)	ABG (6)	ABG (7)	VBG (8)	VBG (9)	VBG (10)	VBG (11)	VBG (12)	VBG (13)	ABG (14)	VBG (15)
pH	7.15	7.12	7.17	7.17	7.26	7.31	7.43	7.40	7.44	7.44	7.38	7.38	7.40	7.44	7.44
pCO ₂	113	136	121	102	89.5	84.6	67.6	61.6	57.0	51.9	55.7	53.6	49.1	42.8	48.1
pO ₂	65	77.3	73.8	70.2	68.8	54.0	68.0	40.2	50.2	42.4	57.1	41.6	54.4	87.9	58.2
HCO ₃	27.6	29.6	31.4	27.3	32.1	35.1	40.6	34.2	35.9	28.9	29.5	28.5	28.5	28.5	31.4
SPo ₂	86.5	91	91.3	91	92.4	86.4	95.4	75.3	84.9	75.3	88.8	74.9	86.2	95.6	89

NIV: Noninvasive ventilation, ABG: Arterial blood gas, VBG: Venous blood gas, HR: Heart rate, BP: Blood pressure, RR: Respiratory rate

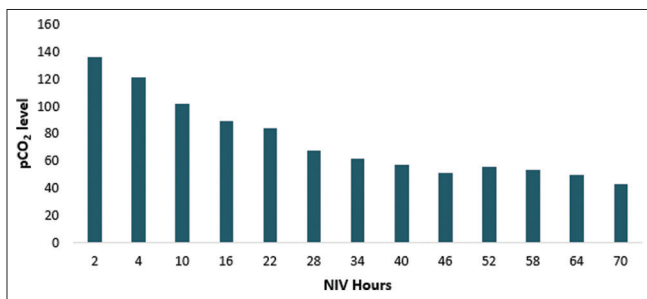


Figure 2: Differential effect of PCO₂ reductions over noninvasive ventilation hours

DISCUSSION

In addition to conventional treatment, NIV was the dominant part of the successful management of this patient. Some obese patients with respiratory etiology are misdiagnosed with chronic obstructive pulmonary disease (COPD) and asthma.^[5] Similarly, this patient was initially diagnosed and treated as exacerbation of COPD although the clinical diagnosis supported OHS. Physicians should be more cautious about the diagnosis of OHS in this background. In comparison with OSA or eucapnic obese patients, the central fat distribution is found to be higher in OHS, and their respiratory muscles are primarily affected by hypoventilation, larger neck circumference, and high waist: hip ratios.^[8] Notably, in this case, the higher central fat distribution reduced the respiratory muscle efficiency, resulting in more significant reduction in lung volume and increased WOB. These features worsened in lying and supine positions due to the cephalic displacement of the diaphragm. Hence, high Fowler’s position was adopted for 70 h of NIV (Bi-level ST). PCO₂ reduction was significant as shown in similar studies demonstrating consistent improvement in awake blood gases and daytime sleepiness with NIV as compared to continuous positive airway pressure (CPAP).^[8,9] This patient required higher positive end-expiratory pressure of 10 cmH₂O and a higher IPAP of 22 cmH₂O for longer duration before reduction of PCO₂ to below 50 mmHg could be achieved. Many comprehensive studies conclude the same



Figure 3: Body mass index >92 kg/m² and excess fat distribution in the region of thorax and neck

result that the NIV is beneficial; it can prevent intubation and associated complications in selected patients. NIV not only enhanced the pulmonary gas exchange but also reduced a load on inspiratory muscles and improved the respiratory mechanics of our patient. In the long term, weight reduction is helpful.^[10,11]

CONCLUSION

The NIV was beneficial in this extreme super-super obese patient with BMI >92 kg/m² with OHA who presented with Type II respiratory failure. NIV prevented intubation and mechanical ventilation complications. Physicians should be aware that each obese patient may not be a candidate for NIV. Awake hypercapnia and hypoxia may persist despite the effective use of NIV. A randomized controlled trial on the use of NIV is recommended in this group of patients.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be

reported in the journal. The patient understands that his names and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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