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### Case report

# A case report of malignant obesity hypoventilation syndrome: A weighty problem in our ICUs



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#### ABSTRACT

*Introduction:* The obesity epidemic is reflected by increasing numbers of morbidly obese patients being admitted to intensive care units (ICUs). These are complicated patients whose care involves many diagnostic and treatment challenges. We are presenting a fatal case of super obesity, hypoventilation, and multi-organ failure known as malignant obesity hypoventilation syndrome (MOHS).

Case presentation: 35 year old African American gentleman with a body mass index (BMI) of 115 kg/m<sup>2</sup> presented to the hospital with respiratory distress. On admission he was noted to have multi-organ dysfunction including respiratory failure, renal failure, cardiac and liver abnormalities. His hospital course was remarkable for recurrent cardiac arrest following extubation, complicated tracheostomy, and progressive organ failure despite medical therapy. After a 30 day hospitalization, patient and family decided on terminal extubation owing to worsening medical condition and lack of therapeutic and disposition options.

Discussion: The super obese present a number of challenges when admitted to the ICU. Patients with respiratory distress are frequently misdiagnosed and treated for asthma and COPD when obesity hypoventilation syndrome (OHS) is more consistent with the clinical picture. OHS in the superobese is often accompanied by multi-system organ dysfunction, a condition with high morbidity and mortality, with limited treatment options. Standard imaging techniques and procedures are made difficult or impossible by body habitus and often require expert intervention. Surgical options have been used in the treatment of the super obese and resulted in rapid weight loss, improvement in respiratory function, as well as improved metabolism and decreased inflammation. The role of surgery in MOHS remains to be elucidated.

*Conclusion:* Physicians should be aware of MOHS as a common condition with high morbidity and mortality. Optimal management remains to be determined.

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#### 1. Introduction

The prevalence of obesity have been rising over the last twenty years currently affecting over 1 in 3 Americans[1]. Over the same time span, class III Obesity (severe or morbid obesity: BMI > 40 kg/  $\rm m^2$ ) in US adults rose by 141% in non-diabetics and 345% in type 2 diabetes mellitus patients with a current prevalence of 6.6% [2,3]. Epidemiological data shows an increased risk of death of 1% for each pound increase between 30 and 42 years of age and 2% between 50 and 62 years [4]. Mortality is higher among hospitalized

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patients less than age 65, with a BMI > 30 kg/m<sup>2</sup> [5]. The cumulative duration of exposure to obesity may be even more important, with longer duration being independently associated with increased risk of hospitalizations and length of stay[6].

As the number of morbidly obese patients admitted to the ICU continues to rise, physicians will increasingly take care of this challenging population. Morbid obesity has been associated with longer ICU length of stay and prolonged mechanical ventilation, with increased overall mortality and ICU mortality currently under debate [7,8]. Factors contributing to ICU mortality in extreme-obesity include increased catheter and blood stream infections [9], limited imaging modalities, increased skin breakdown [10], and diagnostic errors. Additionally, a condition called MOHS was

recently described [11]. MOHS, a disorder characterized by hypoventilation and multi-organ dysfunction due primarily to severe obesity is one of the under recognized causes of ICU mortality. We are presenting a case of severe obesity and MOHS resulting in mortality to alert clinicians to this common, frequently misdiagnosed, condition with high morbidity and mortality.

#### 2. Case

A 35 year old African American gentleman with a past medical history of class III obesity, asthma, depression, hypertension, hyperlipidemia, and type 2 diabetes mellitus who presented to a community hospital with cough and difficulty breathing. He was started on antibiotics and bronchodilators for pneumonia and asthma exacerbation. He appeared lethargic and was intubated when his arterial blood gas (ABG) revealed a PCO2 of 120 mmHg. He was extubated after correction of hypercarbia, but developed hypoxemic respiratory failure, resulting in PEA arrest requiring reintubation. A day later, he was extubated again, this time to BiPAP and transferred to the medical ICU at our tertiary care center.

On arrival, he was hemodynamically stable with mild tachycardia and with a normal O2 saturation. He was 182.8 cm tall, with a weight of 383 kg (kg), BMI 114.5 kg/m $^2$ . On examination he had profound generalized obesity with a crowded oropharynx and large neck. His lungs were clear and heart sounds were regular. Abdomen was immense with no palpable organomegaly. Lower extremity exam revealed 2 + pitting edema. He had multiple pressure ulcers on his buttocks and breast folds.

Bloodwork revealed a creatinine of 2.18 mg/dL with GFR of 44 ml/min/1.73 m<sup>2</sup>, Hemoglobin 8.0 g/dl, white blood count 11.9K, mildly elevated LFTs, albumin of 1.9 g/dl and INR of 1.5. Lactic acid was 1.3 mmol/L, pro BNP 3921 pg/ml, and hemoglobin A1c was 7.3%. ABG showed a pH of 7.23, pCO2 of 91 mmHg and pO2 of 114 mmHg on Bipap 20/10 with 40%FiO2. A blood culture drawn from his right internal jugular central line grew out S. epidermidis. A chest radiograph was non diagnostic (Fig. 1).

In the medical ICU, he completed a course of steroids and antibiotics and was maintained on bronchodilators around the clock. He was noted to have daytime hypercarbia and initially required

continuous BiPAP, which was weaned to nocturnal use only. Pressure ulcers were treated with local wound care and frequent repositioning, requiring ten staff members for mobilization using a 1000lb patient lift system. Nephrology, Cardiology, Infectious Disease, and Endocrine services were consulted and the patient was treated with copious fluids, often 10 L a day or more to maintain a stable blood pressure and adequate urine output. After a period of monitoring and stabilization he was transferred to a regular floor.

Two days after transfer, he was discovered unresponsive, in pulseless electrical activity (PEA) arrest. He was resuscitated and re-intubated for the second time over the course of two weeks. The diagnosis of pulmonary embolism was entertained, but could not be confirmed as the patient exceeded the weight limitations of our CT scanner. Cardiac echocardiogram showed a dilated right ventricle with reduced ejection fraction, as well as dilated left atrium, left ventricular hypertrophy, and LVEF of 60%. Given these findings, anticoagulation was initiated.

Given repeated episodes of respiratory failure and cardiac arrest, it was deemed unsafe to extubate the patient without definitive airway management. Otolaryngology evaluation revealed that a standard tracheostomy would be unsafe due to the excess thoracic adipose tissue (Fig. 2). The patient was taken to the operating room with otolaryngology and plastic surgery and 30 + lbs of lipoid tissue was removed from the chest, prior to tracheostomy placement.

Subsequent hospital course was complicated by worsening renal function and failed attempts to wean the patient off of the ventilator. A bariatric surgery evaluation determined that several hundred pounds weight loss would be required to qualify for surgery. We explored options for discharge to a chronic ventilator facility without success, due to the difficulties posed by managing a super obese patient on a ventilator.

Eventually, the patient expressed a desire to be taken of the ventilator. After extensive discussions with the patient, his family, palliative care, and ethics teams, he was disconnected from the ventilator on day 30 of his hospitalization, and passed away from hypoxic respiratory arrest.

As per the patient's wishes to be cremated, multiple facilities were contacted post mortem without success due to his size and their concern for fire (one facility just had a fire after cremating a

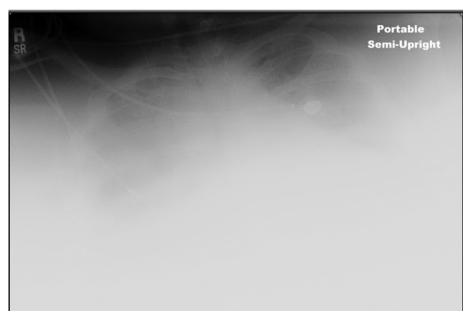


Fig. 1. Admission chest X-ray.

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