ECRONICON OPEN ACCESS

EC PULMONOLOGY AND RESPIRATORY MEDICINE

Review Article

Management of the Super Obese in the Intensive Care Unit

Raghav Gupta*, Deep Patadia, Alyssa Lee Peters, Hassan Patail, Josh Davidson and Dan Buchnea

Department of Pulmonary and Critical Care Medicine, SUNY Downstate Medical Center, USA

***Corresponding Author:** Raghav Gupta, Department of Pulmonary and Critical Care Medicine, SUNY Downstate Medical Center, USA.

Received: September 16, 2019; **Published:** September 25, 2019

Abstract

This article summarizes existing data on the complications and complexities of the management of the super obese patient in an intensive care unit (ICU) setting. We have specifically focused on class III obese critically ill patients (i.e. BMI ≥ 40 kg/m^2, or ≥ 35 kg/m^2 in the presence of comorbidities). It outlines the clinical and pathophysiologic characteristics of obesity hypoventilation syndrome (OHS), acute respiratory failure secondary obesity and post-extubation respiratory failure. It also reviews the literature on outcomes specific to the management of the obese critically ill patient, and complications including tracheostomy, long-term mechanical ventilation, imaging difficulties, vascular access, and nutrition.

Keywords: *Obesity Hypoventilation Syndrome; Intensive Care Unit; Hemodynamics*

Introduction

Obesity is defined as a body mass index (BMI) greater than 30.0. Severe obesity is defined as a BMI ≥ 40 kg/m^2 (or ≥ 35 kg/m^2 in the presence of comorbidities) [1,2]. In 2010 there was an estimated 3.4 million deaths caused by obesity worldwide; studies showing the prevalence of obesity and its pandemic are consistent with an increase in BMI in both men and women from the year 1980 to 2013 [3]. The United States alone has the highest mean BMI among high income countries and obesity affects one in every 3 persons. It was estimated in 2010 that 6.6% of the American population had a BMI over 40 [4].

Management of the super obese patients in the health care setting, aside from their more complex comorbidities is challenging due to inadequacy of standard measuring and lifting devices, imaging and transporting equipment [4]. Obesity and increased BMI specifically has shown to encounter challenges in ICU including prolonged mechanical ventilation, extended length of stay in the ICU, infections, delirium and critical care myopathy [5].

Evidence strongly suggests obesity is a precursor to major coronary heart disease with its associated cardiovascular risk factors including increased blood pressure and lipids, as well as diabetes and thus higher incidence of acute coronary syndromes [6]. Obesity affects cardiopulmonary physiology by its increased inflammatory activity. The proinflammatory state induces accumulation of epicardial fat which can result in myocardiopathy with intra-ventricular asynchrony, thus can lead to heart failure [7]. Most importantly, obesity leads to sleep-disordered breathing including obstructive sleep apnea (OSA) and can cause chronic hypercapnic respiratory failure secondary to obesity hypoventilation syndrome (OHS) [2,8].

Obesity paradox is the positive effect of obesity observed in long term hemodialysis patients associated with reduction in mortality with patients with higher muscle mass [9]. The meta-analysis by Pepper., *et al.* showed various physiologic and biologic hypoth-

eses of obesity paradox which includes increased renin-angiotensin system (RAAS) activity with resultant hemodynamic support needing less vasopressor support; increased adipose tissue and lipoprotein levels which can bind lipopolysaccharide or other bacterial products during sepsis and inactivate them; also adipose tissue can serve as a beneficial source of energy during the catabolic state of sepsis, and has some protective immune functions. Obesity paradox was overall observed in overweight and obese patient and no effect was associated with severely obese patients with BMI more than 40 [10].

Hereby, we review the common difficulties faced during ICU diagnostics and management in the cohort of severe obese patients. With adequate understanding of the complications, we believe a better outcome can be expected when these patients are admitted to ICU.

Obesity hypoventilation syndrome

Epidemiology

It is well known that there is a direct correlation of obesity with OHS. The majority of OHS patients have OSA. Super obese patient constitutes approximately 6% of the general US population [11]. The estimated prevalence of OHS in U.S. is roughly at 0.6% (1 in 160 adults in the US population) [12]. In a recent small study in Northern Greece evaluating the prevalence of OHS and its clinical features amongst patients with reported sleep-disordered breathing patterns, it was discovered that 38 out of the 276 patients (13.8%) had OHS. Within the study population, the OHS subjects were noted to be more obese and somnolent, and were older [13,14]. In another large prospective study done evaluating the prevalence of OHS among men and women with obstructive sleep apnea was 4.5% and 15.6%, respectively, and highest in post-menopausal women [15]. Thirty percent of hospitalized obese patients suffer from OHS, and according to Marick and colleagues, the majority of these patients have been under-diagnosed and perhaps not treated adequately prior to being hospitalized. OHS is accountable for increased morbidity causing a constellation of different systemic disorders attributed to obesity itself. OHS is associated with hypercapnic respiratory failure, left ventricular dysfunction, pulmonary hypertension, chronic renal and liver disease, et cetera [16]. The term malignant obesity hypoventilation has been coined by Marik and colleagues when patients with class III obesity present with severe OHS and multiple associated systemic diseases.

Clinical presentation and diagnosis

OHS is historically referred to as Pickwickian syndrome [17]. It is defined as a combination of obesity (BMI > 30 kg/m^2) and sleep-disordered breathing, mainly from OSA, contributing to chronic awake $PaCO_2$ > 45 mmHg in the absence of any other cause of hypoventilation, including other pulmonary pathology, neuromuscular weakness, or chest wall disorders. Malignant OHS (MOHS) is a syndrome of OHS associated with systemic hypertension, diabetes, left ventricular hypertrophy with diastolic dysfunction, pulmonary hypertension and hepatic dysfunction [18]. A common, but under-recognized presentation of OHS is acute on chronic hypoxemic hypercapnic respiratory failure, often severe, and at times, can be a life-threatening condition necessitating intensive care admission [1,19]. Many patients who have undiagnosed OHS are usually diagnosed incorrectly with obstructive lung diseases including COPD or asthma. OHS remains the diagnosis of exclusion. Forced expiratory volume in one second (FEV1) can be mildly lower in patients with severe obesity, though the FEV1/FVC ratio is normal. Maximum expiratory flow 25 - 75% can be significantly reduced, particularly in male subjects with class III obesity showing small or distal airway obstruction. Expiratory reserve volume (ERV) is markedly reduced with preserved residual volume (RV). The diffusing capacity for carbon monoxide is usually normal or may show a minor increase due to an increase in pulmonary blood volume [20].

Pathogenesis

Obese patients without elevated awake $PaCO_2$, but with raised base excess, have an altered ventilation and oxygenation response especially at night suggesting that several pathophysiological mechanisms have arisen prior to manifestation of OHS with daytime hypercarbia development [2,17].

The diurnal hypercapnia and hypoxia induce pathologic effects that further worsen sleep-related breathing resulting in a slowly progressive worsening of disease. Additionally, resistance to leptin in obesity and OHS likely contributes to blunting of ventilatory drive and inadequate chemoreceptor response to hypercarbia and hypoxemia [17]. It was observed that hypercapnea and hypoxemic drive in OHS patients was respectively one-third and one-sixth that of non-obese patients. Obesity itself causes decrease in the chest wall compliance as a consequence of the restriction by adipose tissue imposed on the expansion of the ribcage and diaphragm. There is a subsequent increase in pulmonary blood volume and at the same time alveolar collapse from small airway obstruction predominantly at the bases. As a result of these anomalies the work of breathing and the ventilatory effort increases. Thus, in patients with BMI > 40 kg/m^2 oxygen consumption (VO_2) at rest can reach up to 16% of total VO_2, though it infrequently surpasses 3% in non-obese patients [20].

Complications

Obesity causes major respiratory complications which increases morbidity and mortality [21]. Manthous., *et al*. identified some preventable therapeutic errors in OHS patients at the time of hospitalization for pulmonary or cardiovascular decompensation. Some of the therapeutic errors include exacerbation of metabolic alkalosis by use of diuretics resulting in worsening of daytime hypoventilation and further hypoxemia, acute hypercarbia secondary to unnecessary supplemental oxygen administration; premature pharmacologic treatment of psychosis leading to exacerbation of sleep-disordered breathing and worsening hypercapnia and quite frequently inaccurately diagnosis of patients as obstructive pulmonary disease with resultant unwarranted and possibly toxic exposure to medications [22].

Here, by we discuss the various challenges of super obese patients being admitted to ICU.

Noninvasive Positive Pressure Ventilation (NIPPV) in Super Obese Patients

Super obesity is a major cause of acute and chronic respiratory failure, and non- invasive ventilation (NIV) is usually implicated to avoid intubation and the numerous complications occurring during the course of prolonged invasive mechanical ventilation. Till present date, the following are the indications for NIV: cardiogenic pulmonary edema, exacerbation of chronic obstructive pulmonary disease (COPD), pulmonary infiltrates in immunocompromised patients, and weaning of previously intubated stable patients with chronic obstructive pulmonary disease [23]. Lemyze., *et al.* evaluated the predictors of NIV success or failure in super obese patients admitted with acute respiratory failure (ARF). They found that severe pneumonia and multiple organ failure often caused early NIV failure in hypoxemic ARF, however, super obese patients with idiopathic hypercapnic ARF did well on NIV, concluding the role of NIV in acute hypercapnic ARF secondary to OHS [24]. Similarly, Ortega., *et al.* found better response to NIV in OHS and COPD as compared to congestive heart failure in patients with acute hypercapneic hypoxemic ARF [25]. The adjunctive role of respiratory stimulant like progesterone for reduction in the $PaCO_2$ in super obese patients is also significant [21]. Further studies are needed to strengthen the role of NIV in acute hypercapnic respiratory failure.

Post-Extubation Non-invasive Positive Pressure Ventilation (NIPPV) in Super Obese Patients

Post extubation use of NIPPV in super obese patients has been found to reduce post-extubation respiratory failure, incidence of extubation failure, duration of intensive care unit (ICU) stay and ICU mortality [26,27]. El-Solh and colleagues matched 62 consecutive critical care patients with severe obesity (BMI ≥ 35 kg/m^2) with historical controls that received conventional therapy and found the NIPPV group had fewer incidents of respiratory failure, ICU days, and total hospital duration [28]. Even in respect to an elective surgery, specifically bariatric surgery it has been demonstrated a benefit of NIPPV in preventing extubation failure in super obese patients [29]. Frat and colleagues (ARCO study group) determined that the ICU course in obese invasively ventilated patients was complicated by stridor and difficult intubations when compared to matched non-obese patients [30]. This may offer one rationale for the benefits of planned NIPPV post-extubation in obese patients to overcome an upper airway obstruction and future RCT are required to solidify the evidence.

Best PEEP for super obese patients

Once super obese patients with OHS requires ventilatory support, high positive end-expiratory pressure (PEEP) is needed to improve lung elastic properties and lung volumes, however, the method to apply the proper PEEP is not clear. Driving pressure (ΔP) is the ratio of tidal volume (VT) to (static) respiratory system compliance (CRS); i.e. ΔP = VT/CRS which can be calculated at bedside as plateau pressure – PEEP. The lowest delta pressure needed to recruit the alveoli is optimal which has also shown to have a mortality benefit [31].

De-recruitment is a high possibility if increasing PEEP continues to cause high plateau pressures. Pirrone., *et al.* noted that alveoli recruitment and better oxygenation is achieved by recruitment maneuvers followed by methods of PEEP titration using either incremental PEEP titration by esophageal pressure which correlates well with pleural pressure to aim for positive transpulmonary pressure or by using decremental PEEP using lowest delta pressure or driving pressure. It was also found that baseline PEEP used before this approach was lower and insufficient to increase lung volumes in super obese patients [32]. Positioning of the super obese patients is as important as giving best PEEP to recruit alveoli. Supine patients tends to develop greater auto PEEP by the mechanism of increased diaphragmatic load causing reduction in expiratory flow, compliance, static and dynamic lung volumes, most importantly functional residual capacity (FRC) leading to increase in intrinsic PEEP [33]. So, it is crucial to do sitting position with PEEP in super obese patients to prevent expiratory flow limitation and auto PEEP [34].

Aspiration risk

Risk of aspiration in the critically ill patients is secondary to sedation which slows down gastric motility and causes cardiovascular collapse further leading to intestinal ischemia. This effect is exaggerated in super obese patients as at times more sedation is needed together with the increase intraabdominal pressures causing high volume of gastric contents which increases risk of aspiration [35]. Difficulty in procedures especially intubation with suboptimal sedation increases risk of aspiration [36]. Grant and colleagues stated that 2 experienced physicians for intubation must be present during securing the airway along with the use of highly curved laryngoscope [37]. Conventional aspiration precautions should be implied.

Early Tracheostomy

Early tracheostomy (ET) which is defined as equal or less than 10 days after tracheal intubation in normal weight patients in ICU decreases the duration of sedation, however, has no effect on mortality, incidence of ventilator associated pneumonia, duration of mechanical ventilation and length of ICU stay [38].

Super obese patients often have high peak airway pressures due to high resistance in airways due to low compliance of the chest wall which leads to prolongation of mechanical ventilation. It is well known that percutaneous dilatational tracheotomy (PDT) is considered to be a safe procedure in super obese patients in ICU [39]. Less known is the proper timing of PDT in super obese patients in ICU. Kaese., *et al.* showed by the retrospective analysis of 23 super obese patients in a single center trial that early PDT on day 2 - 4 of the ventilator in obese and severe respiratory failure patients had better gas exchange by decreasing the dead space and airway resistance together with avoiding side effects of sedation [40]. Future trials are needed to evaluate the benefit of ET over late tracheostomy, which is more than 10 days of tracheal intubation.

Complications of tracheotomy

Morbidly obese patients (BMI > 40 kg/m^2) are at greater risk for complications after surgical procedures including tracheotomies than non-obese patients because of their difficult neck anatomy and decrease respiratory reserve. These complications included aborting the procedure, accidental extubations and para-tracheal insertion. Solh., *et al*. also found complications including extra tracheal tube placement and accidental decannulation in cohort of patients in ICU which is life threatening in the background of minimal respiratory reserve

[35]. Marshal and colleagues found similar complication rates as compared to non-obese patients, however, super obese patients with BMI > 60 were more likely to be dependent on a tracheotomy or ventilator at the time of discharge [41].

Hemodynamic complications

Hemodynamic monitoring is challenging with super obese critically ill patients in the ICU [42]. However, Lagrand., *et al.* stated that hemodynamic monitoring and goals are not so different in non-obese versus obese patients as the interpretation is the same in respect of body surface area [43]. There is an increase in total blood volume and left ventricular dilation with increased stroke volume (SV) and consequently cardiac output with associated decreased left ventricular systolic function. Increased oxygen delivery is provided by the increased cardiac output meeting the metabolic demands of additional adiposity [44]. In super obese patients the non-invasive blood pressure (NIBP) measurement underestimates systolic blood pressure which can affect therapeutic decisions adversely and may negatively impact outcomes [42]. Invasive arterial blood pressure (IABP) measurement should be used in the ICU for proper hemodynamic monitoring; although because of the obscured anatomic landmarks as well as likely peripheral edema cannulation in obese patients may also be difficult [43]. Obesity tends to obscure readings of electrocardiogram including conduction times, electrical axes and the waves voltages especially low QRS-complex voltages. Class III obesity itself is an identified arrhythmogenic factor associated with supraventricular, ventricular arrhythmias and even sudden cardiac death.

TTE is often severely restricted because of poor acoustic windows. Transesophageal echocardiography (TEE) though can provide more information and is quite useful in critically ill patients with unexplained hemodynamic instability to rule out important and reversible pathologies [42].

Another means of continuous but invasive method of hemodynamic monitoring is esophageal doppler technique which measures blood flow velocity in the descending aorta and can approximate SV variations and may guide fluid balance in critically ill patients. It is however very sensitive to probe movement thus is limited to patients who are paralyzed or heavily sedated in ICU [42]. Transthoracic bio-impedance cardiography is another entity based on change in the voltages predicting SV after a constant, low-amplitude (0.5 - 4.0 mA), high-frequency (50 - 100 kHz), and alternating electrical current to the thorax. Its use is still limited in super obese patients despite aggressive work on the mechanics. Endotracheal CO monitoring is another popular method to determine hemodynamics based on bio-impedance cardiography but future studies are required to validate as a routine use [42].

Difficult central lines/catheters and their complications

To date, there has been no specific studies looking at complication rates of vascular access in the morbidly obese [35]. Due to the large neck and increase adipose tissue in the surrounding area may cause an increased challenge placing central lines in the neck or chest. There have been studies done with morbid obese patients in the intensive care units and complications rates were studied with some data including invasive procedures [45]. Subclavian and internal jugular venous cannulation are difficult and sometimes infeasible in super obese patients because of the short broad neck and increased distance from skin to blood vessel, thus increasing risk of insertion complications, which include multiple local skin punctures, thus increased risk of infection and thrombosis, as well as mal-positioned catheters, pneumothorax, or accidental arterial puncture with difficulty in compression [42]. While the trendelenburg position prevents air embolism and enlarges caliber of veins due to increased central venous volume it can lead to acute deterioration of cardiopulmonary status of an super obese patient secondary to elevated diaphragm causing increased intra-thoracic pressure, diminished lung volumes, hence pulmonary reserve [45]. There is an obvious delay in catheter changes further augmenting the infection risk [42]. Femoral vein access may, apart from being less ideal, may be impossible because of severe intertriginous dermatitis. Consequently, peripheral insertion of central lines may be resorted to for routine blood draws, medication administration, and to ensure reliable vascular access, instead of peripheral vascular access and routine venipuncture [44]. Two-dimensional ultrasonography has improved localization, facilitates faster and more successful cannulation of vessels, and is the standard of care during central line placement regardless of BMI [46]. If after many

failed attempts of intravenous access, especially in life-threatening, urgent conditions, intra-osseous access may be an alternative [42]. More studies are needed in the morbidly obese population regarding venous access, specific to central venous catheters.

Infections

It is well known that in super obese patients, low FRC increases the risk of expiratory flow limitation and airway closure with marked reductions in expiratory reserve volume may lead to ventilation perfusion mismatch due to varied distribution of ventilation [47]. This mechanism increases the risk of atelectasis or lung infections leading to the longer ICU stay. It is also noted that increased obesity is associated with increased risk for acute respiratory distress syndrome (ARDS). The proposed mechanism is due to varied tidal volumes which causes ventilator induced lung injury (VILI). In the study, tidal volume in obese patients were low (5 - 6 ml/kg) based on actual body weight, but high (10 - 11 ml/kg) based on predicted body weight, which might have caused VILI by high tidal volumes [48]. Super obese patients have higher rate of candida colonization, ICU-acquired catheter and blood stream infections. The cause is unclear, however, could be secondary to difficulty in obtaining venous access and not changing the lines frequently due to initial difficulty in placement [49]. There are future studies required to determine the independent association of super obesity with infections by removing various confounders affecting the final outcome.

Renal failure

Obesity is an independent risk factor for both chronic kidney disease (CKD) and end stage renal disease (ESRD). Hsu., *et al.* noted that higher body mass index (BMI) was a strong independent risk factor for ESRD even after adjustment for other major risk factors that are associated with ESRD including HTN and DM [48]. Othman., *et al.* found that the annual rate of reduction of glomerular filtration rate (GFR) tended to be greater in obese patients than non-obese patients [49].

There are several mechanisms that may link obesity to renal failure including cytokines, lipotoxicity and or hemodynamic factors [51]. Weisinger., *et al.* showed an association between morbid obese patients and nephrotic range proteinuria with decreased proteinuria associated with decreased BMI [50]. In addition, Serra., *et al.* found that morbid obese patients who were going for bariatric surgery had renal biopsies showing glomerular lesions. These lesions included focal segmental glomerulosclerosis (FSGS), increased mesangial matrix, podocyte hypertrophy, glomerulomegaly and mesangial cell proliferation. It was concluded as BMI, an independent risk factor for glomerular lesions [51]. Increased intraabdominal pressures from visceral fat causing renal vein compression leading to reduced renal perfusion causes decrease in GFR in super obese patients [52]. Arfvidsson., *et al*. further supported the hypothesis as ileo-femoral venous pressures were found to be elevated in super obese patients that positively correlated with increased intraabdominal pressures [53]. Future studies should be looking for early versus late renal replacement therapy on this particular group.

Nutrition

The enteral nutritional in ICU decreases inflammation, infections, length of stay, multi-organ failure, and eventually mortality. A meta-analysis confirmed that early enteral nutrition with 48 hours of ICU admission reduced mortality and nosocomial infections [54]. Taylor and colleagues has drawn attention to the relative state of malnutrition that exists in obese patients [58]. The American Society for Parenteral and Enteral Nutrition (ASPEN) 2016 guidelines, an update of the 2011, had specifically made recommendations for nutrition of obese patients in ICU. It is also believed that super obese patients have an extra nutritional reserve [55]. This false conclusion is bolstered by the obesity paradox shown in an observation study where patients with a BMI between 30 and 40 kg/m^2 admitted to a surgical ICU had lower 60 day and in-hospital mortality [56]. Alternatively, obese patients typically suffer from various metabolic derangements including insulin resistance, lipid metabolism, and erosion of lean body mass at higher rates than the general population that may contribute to overall worse nutritional status [59]. Furthermore, adipose tissue does not merely represent an energy reserve, but is a metabolically active tissue [57].

Caloric requirements

For non-obese patients the accepted caloric requirement for ICU patients (in the absence of indirect calorimetry) is 25 - 30 kcal/kg/day [58]. A 2012 meta-analysis showed that measuring resting energy expenditure (REE) by indirect calorimetry in patients with morbid obesity (BMI ≥ 40 kg/m^2) is useful and the most common REE is 2000 - 3000 kcal/day and even higher in critical care setting. The author's intention was to establish at least the minimum energy requirement to prevent underfeeding [61]. As indirect calorimetric measurements may be difficult, time consuming, and/or infeasible depending on the setting, a predictive equation for energy requirement is often employed by hospital personnel. Frankenfield and colleagues used indirect calorimetry to compare the accuracy of accepted equations in morbidly obese (BMI ≥ 45 kg/m^2) critically ill mechanically ventilated patients and found the (unmodified) Penn State equation was the most accurate estimate of REE, falling within 10% of the calculated value 76% of the time [58]. For obese patients more than 60 years old the modified Penn State equation is preferred.

Protein requirements

Protein requirements in non-severely obese patients are 1.2 - 2.0 g/kg/day and have been shown to decrease 28-day mortality when met appropriately. For severely obese (BMI ≥ 40 kg/m^2) hospitalized patients a hypocaloric high protein diet which is 50 - 70% of caloric need or ≤ 14 kcal/kg/day and ≥ 1.2g/kg of actual weight or 2.0 - 2.5 g/kg of ideal body weight of protein is non-inferior to a eucaloric diet, though some positive outcomes have been observed particularly in patients non-insulin dependent diabetes and trauma patients [59]. Routine measurement of nitrogen balance may not be necessary when administering 2.0 g/kg of ideal body weight in severely obese patients but may be performed if there is concern of protein malnutrition. The ASPEN 2016 guidelines recommended a protein goal of 2.0 - 2.5 g/kg of ideal body weight for patients with BMI ≥ 40 kg/m^2. If this hypocaloric high protein strategy is employed in conjunction with the Penn State estimation (modified if BMI ≥ 30 kg/m^2 and ≥ 60 years old) for REE, then a clinician could use a target of 70% of calculated resting energy expenditure (REE) to accurately provide adequate caloric support to the vast majority of obese patients. This would provide appropriate levels of caloric supplementation for patients with actual REEs up to 40% more than the Penn State Equation would predict, but only risk over feeding in patients in which the actual REE was 30% lower than the calculated REE [59].

Difficulties in imaging

Obtaining imaging on obese patient can be a challenge to many radiologists and technicians. Compared to a normal sized individual there are many limitations and adjustments that need to be taken into consideration when considering radiography for the obese patient. There are numerous medical conditions that obese patients can suffer from, and as a result, physicians rely on diagnostic imaging to facilitate the appropriate care. Unfortunately, there are various imaging modalities that are negatively affected and limited by obesity [59]. Radiography is one of the most cost effective means to obtain imaging and usually is the initial method used. However, the obese patient usually has degraded quality. First, given the extensive body habitus of these patients, a single cassette may not be sufficient to fit the boundaries of the field of interest. Thus, multiple cassettes must be considered by the technician to image an obese patient [60]. Another reason for poor radiographic films is that x-ray beam must penetrate multiple thick layers of adipose tissue and because of the vast thickness, there is increased photon scatter, ultimately resulting in substantially reduced contrast resolution [61]. However, in order to address this issue with obese patients, tight collimation may be instituted, which is a technique used to reduce scatter. Here, a tight beam directed to the region of interest, reducing scatter, resulting in enhanced image quality, a concept known as proper collimation [62]. However, even after this, if there is poor quality in radiography, it leads to additional advanced and expensive imaging modalities to obtain better anatomic visualization. Computed tomography (CT) and magnetic resonance imaging (MRI) has been shown to best visualize intrathoracic and intraabdominal anatomy relative to other imaging techniques with respect to obese patients. However, fluoroscopy, CT and MRI are limited to whether the obese patient can fit on the imaging equipment, given each of these imaging studies has strict weight and diameter restrictions [65]. Unfortunately, many hospitals lack oversized equipment to accommodate obese patients. Of all imaging modalities, it is ultrasonography (USD) that that has been most limited by obesity, particularly abdominal USD [64,65]. The benefit of USD

compared to CT, MRI, and fluoroscopy is that it does not have any limitations on the diameter or weight, however it is the fat attenuation that limits the quality of the image. Thus, it is imperative that ultrasonographers expose an area to where the anatomic target is closest to the body surface in order to reduce sound wave attenuation. Padberg., *et al*. demonstrated approximately two-thirds of obese patients that clinically presented with signs and symptoms suggestive of chronic venous insufficiency did not exhibit anatomic evidence of venous disease via duplex ultrasound [63]. Unfortunately, a diagnosis on the obese patient is delayed if a high-quality image is unable to be obtained. Given limitations in obtaining quality images on obese patients leads to additional radiologic examinations, resulting in increased radiation exposure and health care costs. Yanch., *et al*. described relative effective radiation doses increases exponentially as thickness of excess fat increases [64]. To date, there has been no imaging study particular targeting patients with obesity hypoventilation syndrome or large scale randomized studies regarding the impact particular imaging modalities can have on these patients.

Cardiac ventricular dyssynchrony

Cardiac dyssynchrony is a phenomenon that involves the lack of coordination between contractions of ventricles, resulting in reduced cardiac efficiency. Dyssynchrony is classified as either electrical or mechanical. Electrical dyssynchrony involves compromised electrical conduction and electrical propagation through the myocardial Purkinje network. This results in an abnormal ventricular depolarization, evidenced by a prolonged QRS complex, resulting in early and delayed ventricular contraction, impairing systolic function [65]. Mechanical dyssynchrony is further sub-divided into one of three types: atrio-, inter-, or intra-ventricular processes. Atrioventricular dyssynchrony is the improper coordination between the atria regarding left ventricular filling, defined by a left ventricular diastolic filling time less than 40% of the cardiac cycle. Interventricular dyssynchrony involves a delayed coordination between the contraction of the left and right ventricle. Normally, the left ventricle contracts approximately 10 - 20 milliseconds (ms) after the right ventricle, however if interventricular dyssynchrony is present the difference is more than 40 ms. Finally, intraventricular dyssynchrony involves discordance within the segments of the left ventricle, particularly a late contraction of the lateral regions of the left ventricle compared to the interventricular septum [66,67]. Dyssynchrony is best accessed via a different Doppler and two-dimensional echocardiography measurements, which has been validated on multiple occasions [68,72].

Both obesity and ventricular dyssynchrony have been determined to be independent risk factors for cardiac mortality in patients with heart failure [69]. Obesity leads to hormonal and inflammatory pathways that result in the pathophysiologic accumulation of triglycerides within the myocyte, leading to reactive oxygen species that initiate fibrosis, increased hemodynamic load, hypertrophy, and apoptosis [70]. This mechanism is referred to as cardiac steatosis, which propagates an inflammatory environment due to amassing epicardial fat, thus eventually leading to ventricular remodeling and dysfunction [71]. This process can lead to mechanical dyssynchrony, ultimately indicating worsening of heart-failure. Marfella., *et al*. were one of the first groups to demonstrate an association between interventricular dyssynchrony to that of body fat and secretion of proinflammatory cytokines in obese, premenopausal women. The amount of body fat was directly proportional to the concentration of cytokines, which was in turn directly proportional to the degree of interventricular dyssynchrony. Moreover, after these women lost 10% of their body weight, there was a reduction in the concentration of inflammatory markers and significant improvement in ventricular dyssynchrony, as evidenced by two-dimensional and Doppler echocardiography. This may suggest that cytokines may be responsible for interventricular dyssynchrony in obese females [72]. There is also evidence to suggest that the greater amount of epicardial fat and BMI, the greater the intraventricular asynchrony [73]. However, a limitation of this study had predominantly overweight rather than obese patients, but should initiate interest to investigate how intraventricular dyssynchrony may be a pathophysiological process in the progression of heart failure in a predominantly obese population or those with OHS. There have been many studies exhibiting systolic and diastolic dysfunction in patients with metabolic syndrome, which are independent factors for left ventricular hypertrophy [74]. However, in a study with 190 participants, it was found that obese patients, irrespective of the presence or absence of metabolic syndrome, had higher rates of dyssynchrony compared to non-obese patients [75]. The results of this finding contradict the idea that there can be metabolically healthy obese patients.

Conclusion

With the increased understanding of the pathogenesis of OHS, effective evidence-based therapies are now being adopted to reduce morbidity and mortality of super obese patients in ICU [22]. The optimal management of OHS continues to be ambiguous, though many have been successful. The common aim of treatment in patients with OHS is improvement in alveolar ventilation and oxygenation by using best PEEP with either incremental PEEP titration by esophageal pressure or decremental PEEP using delta pressure. Also important is maintaining a patent upper airway, which is optimally accomplished through timely noninvasive ventilation (NIV) or invasive mechanical ventilation and patient positioning. Early tracheostomy may be considered with the risk of dependence. Hemodynamic monitoring is difficult. Invasive and non-invasive methods are available, although future research should be focused on the latter. Difficulty in imaging and venous catheters is known in super obese patients. Proper positioning, complications from positioning and alternatives to venous access should be known. Obesity paradox has been described, although infection risk is high along with the higher aspiration risk compare to non-super obese patients. Conventional aspiration precautions should be implied. Risk of renal failure is higher due to mechanical compression effect further decreasing GFR and future studies should be looking for early RRT on this particular group. Nutrition in ICU is important and especially in super obese population who are often underfed. Minimal caloric requirements should be met with higher protein diet.

Bibliography

1. Palm A., *et al*. "Gender differences in patients starting long-term home mechanical ventilation due to obesity hypoventilation syndrome". *Respiratory Medicine* 110 (2016): 73-78.

2. Jones SF., *et al*. "Obesity Hypoventilation Syndrome in the Critically Ill". *Critical Care Clinics* 31.3 (2015): 419-434.

3. Ng M., *et al*. "Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013". *Lancet* 384.9945 (2014): 766-781.

4. Sturm R and Hattori A. "Morbid obesity rates continue to rise rapidly in the United States". *International Journal of Obesity* 37.6 (2013): 889-891.

5. Martino JL., *et al*. "Extreme obesity and outcomes in critically ill patients". *Chest* 140.5 (2011): 1198-1206.

6. Landi F., *et al*. "Body mass index and mortality among hospitalized patients". *Archives of Internal Medicine* 160.17 (2000): 2641-2644.

7. Alpert MA. "Obesity Cardiomyopathy: Pathophysiology and Evolution of the Clinical Syndrome". *The American Journal of the Medical Sciences* 321.4 (2001): 225-236.

8. Sequeira TC., *et al*. "Noninvasive Ventilation in the Critically Ill Patient With Obesity Hypoventilation Syndrome: A Review". *Journal of Intensive Care Medicine* (2016).

9. Kalantar-Zadeh K., *et al*. "The obesity paradox and mortality associated with surrogates of body size and muscle mass in patients receiving hemodialysis". *Mayo Clinic Proceedings* 85.11 (2010): 991-1001.

10. Pepper DJ., *et al*. "Increased body mass index and adjusted mortality in ICU patients with sepsis or septic shock: a systematic review and meta-analysis". *Critical Care* 20.1 (2016): 181.

11. Flegal KM., *et al*. "Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010". *The Journal of the American Medical Association* 307.5 (2012): 491.

12. Balachandran JS., *et al*. "Obesity hypoventilation syndrome: epidemiology and diagnosis". *Sleep Medicine Clinics* 9.3 (2014): 341-347.

13. Akashiba T., *et al*. "Clinical characteristics of obesity-hypoventilation syndrome in Japan: a multi-center study". *JAMA Internal Medicine* 45.20 (2006): 1121-1125.

14. Trakada GP., *et al*. "Prevalence and clinical characteristics of obesity hypoventilation syndrome among individuals reporting sleep-related breathing symptoms in northern Greece". *Sleep and Breathing* 14.4 (2010): 381-386.

15. BaHammam AS., *et al*. "Gender differences in patients with obesity hypoventilation syndrome". *Journal of Sleep Research* (2016).

16. Marik PE and Desai H. "Characteristics of patients with the "malignant obesity hypoventilation syndrome" admitted to an ICU". *Journal of Intensive Care Medicine* 28.2 (2013): 124-130.

17. Pierce AM and Brown LK. "Obesity hypoventilation syndrome: current theories of pathogenesis". *Current Opinion in Pulmonary Medicine* 21.6 (2015): 557-562.

18. Esquinas AM and BaHammam AS. "The emergent malignant obesity hypoventilation syndrome: a new critical care syndrome". *Journal of Intensive Care Medicine* 28.3 (2013): 198-199.

19. Tatusov M., *et al*. "A case report of malignant obesity hypoventilation syndrome: A weighty problem in our ICUs". *Respiratory Medicine Case Reports* 20 (2016): 38-41.

20. BaHammam A., *et al*. "Sleep-related breathing disorders in obese patients presenting with acute respiratory failure". *Respiratory Medicine Case Reports* 99.6 (2005): 718-725.

21. Parameswaran K., *et al*. "Altered respiratory physiology in obesity". *Canadian Respiratory Journal* 13.4 (2006): 203-210.

22. Manthous CA and Mokhlesi B. "Avoiding Management Errors in Patients with Obesity Hypoventilation Syndrome". *Annals of the American Thoracic Society* 13.1 (2016): 109-114.

23. Nava S and Hill N. "Non-invasive ventilation in acute respiratory failure". *Lancet* 374.9685 (2009): 250-259.

24. Lemyze M., *et al*. "Determinants of noninvasive ventilation success or failure in morbidly obese patients in acute respiratory failure". *PLOS One* 9.5 (2014): e97563.

25. González ÁO., *et al*. "Evolution of patients with chronic obstructive pulmonary disease, obesity hypoventilation syndrome, or congestive heart failure undergoing noninvasive ventilation in a respiratory monitoring unit". *Archivos de Bronconeumología (English Edition)* 42.9 (2006): 423-429.

26. Girault C., *et al*. "Noninvasive ventilation and weaning in patients with chronic hypercapnic respiratory failure: a randomized multicenter trial". *American Journal of Respiratory and Critical Care Medicine* 184.6 (2011): 672-679.

27. Ferrer M., *et al*. "Non-invasive ventilation after extubation in hypercapnic patients with chronic respiratory disorders: randomised controlled trial". *Lancet* 374.9695 (2009): 1082-1088.

28. El Solh AA., *et al*. "Noninvasive ventilation for prevention of post-extubation respiratory failure in obese patients". *European Respiratory Journal* 28.3 (2006): 588-595.

29. Carron M., *et al*. "Perioperative noninvasive ventilation in obese patients: a qualitative review and meta-analysis". *Surgery for Obesity and Related Diseases* 12.3 (2016): 681-691.

30. Frat JP., *et al*. "Impact of obesity in mechanically ventilated patients: a prospective study". *Intensive Care Medicine* 34.11 (2008): 1991-1998.

31. Amato MB., *et al*. "Driving pressure and survival in the acute respiratory distress syndrome". *The New England Journal of Medicine* 372.8 (2015): 747-755.

32. Pirrone M., *et al*. "Recruitment maneuvers and positive end-expiratory pressure titration in morbidly obese ICU patients". *Critical Care Medicine* 44.2 (2016): 300-307.

33. Pedoto A. "Lung physiology and obesity: anesthetic implications for thoracic procedures". *Anesthesiology Research and Practice* (2012).

34. Lemyze M., *et al*. "Effects of sitting position and applied positive end-expiratory pressure on respiratory mechanics of critically ill obese patients receiving mechanical ventilation". *Critical Care Medicine* 41.11 (2013): 2592-2599.

35. El-Solh AA. "Clinical approach to the critically ill, morbidly obese patient". *American Journal of Respiratory and Critical Care Medicine* 169.5 (2004): 557-561.

36. Drain CB and Vaughan RW. "Anesthetic considerations of morbid obesity". *American Association of Nurse Anesthetists* 47.5 (1979): 556-565.

37. Grant P and Newcombe M. "Emergency management of the morbidly obese". *Emergency Medicine Australasia* 16.4 (2004): 309-317.

38. Meng L., *et al*. "Early vs late tracheostomy in critically ill patients: a systematic review and meta-analysis". *The Clinical Respiratory Journal* (2015).

39. Rosseland LA., *et al*. "Percutaneous dilatational tracheotomy in intensive care unit patients with increased bleeding risk or obesity. A prospective analysis of 1000 procedures". *Acta Anaesthesiologica Scandinavica* 55.7 (2011): 835-841.

40. Kaese S., *et al*. "Successful Use of Early Percutaneous Dilatational Tracheotomy and the No Sedation Concept in Respiratory Failure in Critically Ill Obese Subjects". *Respiratory Care* 61.5 (2016): 615-620.

41. Marshall RV., *et al*. "Tracheotomy Outcomes in Super Obese Patients". *JAMA Otolaryngology-Head and Neck Surgery* (2016).

42. Marik P and Varon J. "The obese patient in the ICU". *Chest* 113.2 (1998): 492-498.

43. Lagrand WK., *et al*. "Haemodynamic monitoring of morbidly obese intensive care unit patients". *The Netherlands Journal of Medicine* 71.5 (2013): 234-242.

44. Stelfox HT., *et al*. "Hemodynamic monitoring in obese patients: the impact of body mass index on cardiac output and stroke volume". *Critical Care Medicine* 34.4 (2006): 1243-1246.

45. El-Solh A., *et al*. "Morbid obesity in the medical ICU". *Chest* 120.6 (2001): 1989-1997.

46. Gilbert TB., *et al*. "Facilitation of internal jugular venous cannulation using an audio-guided Doppler ultrasound vascular access device: results from a prospective, dual-center, randomized, crossover clinical study". *Critical Care Medicine* 23.1 (1995): 60-65.

47. Salome CM., *et al*. "Physiology of obesity and effects on lung function". *Journal of Applied Physiology* 108.1 (2010): 206-211.

48. Hsu CY., *et al*. "Body mass index and risk for end-stage renal disease". *Annals of Internal Medicine* 144.1 (2006): 21-28.

49. Othman M., *et al*. "Influence of obesity on progression of non-diabetic chronic kidney disease: a retrospective cohort study". *Nephron Clinical Practice* 113.1 (2009): c16-c23.

50. WeisingerJR., *et al*. "The nephrotic syndrome: a complication of massive obesity". *Annals of Internal Medicine* 81.4 (1974): 440-447.

51. Darrat I and Yaremchuk K. "Early mortality rate of morbidly obese patients after tracheotomy". *Laryngoscopy* 118.12 (2008): 2125-2128.

52. Sugerman HJ. "Effects of increased intra-abdominal pressure in severe obesity". *Surgical Clinics of North America* 81.5 (2001): 1063-1075.

53. Arfvidsson B., *et al*. "Iliofemoral venous pressure correlates with intraabdominal pressure in morbidly obese patients". *Vascular and Endovascular Surgery* 39.6 (2005): 505-509.

54. McClave SA., *et al*. "Guidelines for the Provision and Assessment of Nutrition Support Therapy in the Adult Critically Ill Patient Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (ASPEN)". *Journal of Parenteral and Enteral Nutrition* 40.2 (2016): 159-211.

55. McClave SA., *et al*. "Nutrition Therapy of the Severely Obese, Critically Ill Patient Summation of Conclusions and Recommendations". *Journal of Parenteral and Enteral Nutrition* 35.5 (2011): 88S-96S.

56. Hutagalung R., *et al*. "The obesity paradox in surgical intensive care unit patients". *Intensive Care Medicine* 37.11 (2011): 1793-1799.

57. Kee AL., *et al*. "Resting energy expenditure of morbidly obese patients using indirect calorimetry: a systematic review". *Obesity Reviews* 13.9 (2012): 753-765.

58. Frankenfield DC., *et al*. "Prediction of resting metabolic rate in critically ill patients at the extremes of body mass index". *Journal of Parenteral and Enteral Nutrition* 37.3 (2013): 361-367.

59. Uppot RN., *et al*. "Effect of Obesity on Image Quality: Fifteen-year Longitudinal Study for Evaluation of Dictated Radiology Reports". *Radiology* 240.2 (2006): 435-439.

60. Uppot RN. "Impact of obesity on radiology". *Radiologic Clinics of North America* 45.2 (2007): 231-246.

61. Fetterly KA and Schueler BA. "Experimental evaluation of fiber-interspaced antiscatter grids for large patient imaging with digital x-ray systems". *Physics in Medicine and Biology* 52.16 (2007): 4863.

62. Barnes GT. "Contrast and scatter in x-ray imaging". *Radiographics* 11.2 (1991): 307-323.

63. Padberg F., *et al*. "Does severe venous insufficiency have a different etiology in the morbidly obese? Is it venous?". *Journal of Vascular Surgery* 37.1 (2003): 79-85.

64. Yanch JC., *et al*. "Increased Radiation Dose to Overweight and Obese Patients from Radiographic Examinations". *Radiology* 252.1 (2009): 128-139.

65. Hawkins NM., *et al*. "Selecting patients for cardiac resynchronization therapy: electrical or mechanical dyssynchrony?" *European Heart Journal* 27.11 (2006): 1270-1281.

66. Brugada J and Vidal B. "Assessing Mechanical Cardiac Asynchrony". *European Journal of Cardiology Practice* 6 (2007): 13-27.

67. Bader H., *et al*. "Intra-left ventricular electromechanical asynchrony: A new independent predictor of severe cardiac events in heart failure patients". *Journal of the American College of Cardiology* 43.2 (2004): 248-256.

68. Gorcsan J., *et al*. "Echocardiography for cardiac resynchronization therapy: recommendations for performance and reporting–a report from the American Society of Echocardiography Dyssynchrony Writing Group endorsed by the Heart Rhythm Society". *Journal of the American Society of Echocardiography* 21.3 (2008): 191-213.

69. Aaronson KD., *et al*. "Development and prospective validation of a clinical index to predict survival in ambulatory patients referred for cardiac transplant evaluation". *Circulation* 95.12(1997): 2660-2667.

70. Lucas E., *et al*. "Obesity-induced cardiac lipid accumulation in adult mice is modulated by G protein-coupled receptor kinase 2 levels". *Cardiovascular Diabetology* 15.1 (2016): 155.

71. Sacks HS and Fain JN. "Human epicardial adipose tissue: a review". *American Heart Journal* 153.6 (2007): 907-917.

72. Marfella R., *et al*. "Effect of weight loss on cardiac synchronization and proinflammatory cytokines in premenopausal obese women". *Diabetes Care* 27.1 (2004): 47-52.

73. Ávila-Vanzzini N., *et al*. "Excessive Weight and Obesity Are Associated to Intra-Ventricular Asynchrony: Pilot Study". *Journal of Cardiovascular Ultrasound* 23.2 (2015): 86-90.

74. Kane GC., *et al*. "Progression of left ventricular diastolic dysfunction and risk of heart failure". *The Journal of the American Medical Association* 306.8 (2011): 856-863.

75. Wang YC., *et al*. "Preclinical Systolic and Diastolic Dysfunction in Metabolically Healthy and Unhealthy Obese Individuals". *Circulation: Heart Failure* 8.5 (2015): 897-904.

Volume 8 Issue 10 October 2019