



CODEN [USA]: IAJPBB

ISSN : 2349-7750

## INDO AMERICAN JOURNAL OF PHARMACEUTICAL SCIENCES

SJIF Impact Factor: 7.187

Available online at: <http://www.iajps.com>

Research Article

### THE CRITICAL CARE OBESITY PARADOX AND IMPLICATIONS FOR NUTRITIONAL SUPPORT.

<sup>1</sup>Dr Wajiha Kiran Awan, <sup>2</sup>Dr Rabia Afroz, <sup>3</sup>Dr Muhammad Ahmad Rauf.<sup>1</sup>MBBS, Nawaz Sharif Medical College, Gujrat., <sup>2</sup>MBBS, King Edward Medical University, Lahore., <sup>3</sup>MBBS, Services Institute of Medical Sciences, Lahore.

Article Received: November 2020 Accepted: December 2020 Published: January 2021

**Abstract:**

*Obesity is one of the root cause of many diseases and problems. Obesity leads to many life threatening disease. It is more common now a days in all group ages either children, adults, elderly. Main cause of obesity is poor healthy eating, consumption of junk food sedentary life style, lack of physical activity. These common life style behavior easily help to achieve obesity which further leads to many fatal diseases like cardiovascular diseases, hypertension, pulmonary disease, liver disorders etc. This article helps us to see the historical views of obesity, the growing obesity epidemic, pathophysiology of obesity, challenges associated with the critically ill obese patient, the obesity paradox that occurs in critical illness, nutrition support in the critically ill obese patient. Obesity is a complex disease involving an excessive amount of body fat. Obesity isn't just a cosmetic concern. It is a medical problem that increases your risk of other diseases and health problems, such as heart disease, diabetes, high blood pressure and certain cancers. Certain populations tend to have higher rates of obesity. Overweight and obesity are often accompanied by other chronic health conditions such as diabetes, heart disease, hypertension, certain cancers, and arthritis. Rural populations with a higher risk of obesity include those who are: Age 60 and above.*

**Corresponding author:**

**Dr. Wajiha Kiran Awan,**  
MBBS, Nawaz Sharif Medical College, Gujrat.

QR code



Please cite this article in press Wajiha Kiran Awan et al, *The Critical Care Obesity Paradox And Implications For Nutritional Support.*, Indo Am. J. P. Sci, 2021; 08(1).

**INTRODUCTION:**

Obesity is the second leading cause of preventable death worldwide. Obesity leads to various life threatening diseases, such as ischemic heart disease, diabetes, and cancer. One third of the population of critically ill patients are obese. [1] This disease process poses significant challenges to the critical care provider. These results suggest that obese critically ill patients could have worse outcomes, as compared to non-obese critically ill patients. Emerging data suggests that critically ill obese patients have improved outcomes, and this phenomenon has been coined as “the obesity paradox”. Therapies like nutrition support for the critically ill obese patients is still challenging. The purpose is to see the pathophysiology of obesity, epidemiology of obesity and outline the historical views, as it describes the challenges associated with the obesity in the intensive care unit, reviewing the critical care outcomes in the obese, define how critical is obesity, and identify the challenges and role of nutrition support in the critically ill obese patient.

**Causes of Obesity:**

- Genetic predisposition.
- Engineered junk foods.
- Food addiction.
- Aggressive marketing.
- Insulin.
- Certain medications.
- Leptin resistance.
- Food availability.

**Historical Views of Obesity:**

The perspective of obesity is associated with a lack of willpower and poor lifestyle choices. Some thoughts are that obesity was therefore the nature’s mechanism for storing nutrient. Some of the population, thought that excess weight was associated with health, strength and affluence. In one study obesity was described as morally reprehensible and undesirable. Also some studies which revealed an association between increased early mortality and excess weight. A panel of psychiatrists of the 1940s described the overweight individual as affective, intensely reactive, and an emotional grown-up child. But by the early 1960s, the perception was that obesity was undesirable. Perception was kept aside and obesity was linked to both mental and physical health problems. [2]

**The Growing Obesity Epidemic:**

World Health Organization and National Institutes of Health defined obesity by using the body mass index (BMI), defined as an individual’s mass divided by the square of the height and which expressed in kilograms

per meters squared ( $\text{kg/m}^2$ ). Obesity is classified, as the classification defines underweight as a body mass index of less than  $18.5 \text{ kg/m}^2$  and class III obesity as a body mass index greater than  $40 \text{ kg/m}^2$  body mass index greater than  $50 \text{ kg/m}^2$  is considered super obesity. Data collected through recent studies suggests that however, if one considers that nearly 70 % of the population is overweight or obese, conclusion is reaching a “saturation point” where the proportion of the population left to become overweight or obese is indeed genetically protected against adiposity. [3,4] Despite of this level, there is a disproportionate rise in class III obesity. Obesity affects some groups more than others. Amongst men, obesity prevalence is similar at all income levels. Higher-income women are less likely to have obesity than low-income women. Overall, there is a trend toward less obesity amongst those with college going population. [5]

**Pathophysiology of Obesity:**

Laws of thermodynamics require caloric intake to be increased over energy expenditure however, it is simple and incorrect to conclude that obesity is a result of increased caloric intake alone. Although obesity is commonly perceived as a lack of willpower and poor lifestyle choices, the pathophysiology is clearly more complex and comprised many factors outside the conscious individual control. The pathophysiology of obesity involves a complex of internal factors environmental factors and genetic factors such as alterations in central nervous system (CNS). Obese environment with the intake of energy-dense foods and reduced physical activity contribute significantly to obesity development. High-fructose corn syrup is found in soft drinks and juices; its consumption has increased more, and its use is a risk factor for hyperlipidemia and obesity. [6,7] Moreover fast food is energy dense with a higher proportion of trans-fatty acids (TFAs), which increase the risk of obesity and heart disease. Second, genetic factors such as monogenic and polygenic alterations can lead to genetic syndromes, which have obesity as a main point. Gene mutations can also cause non-syndromic monogenic forms of obesity, such as that leading to leptin deficiency. Leptin deficiency is the only monogenic form of obesity for which success therapy has been developed. [3] Central nervous system receives signals from several regulatory loops which helps to control energy balance. The central nervous system also receives information about metabolic needs from active tissues such as adipose tissue, liver, stomach, muscle, and bone. Central controls of body weight and appetite involve a complex of adiposity hormones and satiety. When food is consumed, satiety signals such as cholecystokinin (CCK) and glucagon-

like peptide-1 are released to reduce oral intake. Although its signals are dependent on hormones promoting hunger, food intake, are secreted in response to the amount of adipose tissue. Ghrelin is a potent that stimulates food intake. Its signal is a phasic response, while hunger signaling is a tonic response. An incompletely understood interplay exists between hunger hormones like ghrelin and satiety hormones (like insulin, leptin, cholecystokinin), which serves to further phenotypic expression associated with obesity. [3]

### **Challenges Associated With the Critically Ill Obese Patient:**

One third of intensive care unit patients are obese. [8] Obesity poses unique physical and non-physical challenges for the intensive care unit patient's team. Physical challenges include difficulty in breathing and in securing an airway. Compared to the non-obese patient, obese patients have more adipose tissue, resulting in issues with proper ambiguity and positioning. This complicates fundamental including intravenous access, making bag-mask ventilation and placement of an airway. [9,10] Some studies suggest that tracheal intubation and complications between 82 morbidly obese intensive care unit patients (mean  $42 \pm 6$  kg/m<sup>2</sup>) to 124 non-obese patients (mean BMI,  $24 \pm 4$  kg/m<sup>2</sup>) and demonstrated that the morbidly obese had more difficulty during tracheal intubation which were significantly more frequent in obese patients ( $p < 0.05$ ). [11] The functional residual capacity is reduced in obese patients long periods of apnea are not well tolerated which leading to hypoxemia, atelectasis and a need for positive end expiratory pressure (PEEP). Potential consequences of positive end expiratory pressure include reduced cardiac output and venous return. Ventilation may also be more difficult due to baseline chest wall restriction. [10] Additionally, common techniques to gauge volume status, such as bed side ultrasound, which is due to body habitus. Computed tomography Magnetic resonance imaging and computed tomography commonly have weight limits, limiting their use with increasing obesity. Wound healing may be impaired due to immobility and hyperglycemia, which the obese patient is prone to develop. [9] The non-physical challenges include pharmacologic challenges and nutrition support. The pharmacology of common intensive care unit medications is not well studied in obese patients which dosing common intensive care unit medications. The obese patients have greater fat, greater extracellular volume, and greater lean body weight, all of which alter pharmacokinetics. [10] For example, if a larger dose of a lipophilic agent is given, one can anticipate a longer duration of action.

### **The Obesity Paradox That Occurs in Critical Illness:**

When the obese patient becomes critically ill, the intensive care unit team face additional challenges, which puts the obese patient at risk for poor outcomes. Through some studies it is evaluated that the differences in intensive care unit the mortality between extreme obesity body mass index greater than 40 kg/m<sup>2</sup>) and normal-range body mass index using data from a large population survey. Among the studies it is concluded that obesity was not a risk factor for mortality. [12] A hypothesis suggests that anti-inflammatory adipokines favorably modulate the inflammatory response. In contrast with, leptin and pro-inflammatory cytokines, which activate macrophages and induced hepatic TNF- $\alpha$  and IL-6, have inflammatory response. Obesity may result in a form of inflammatory preconditioning it is seen with ischemic preconditioning in the setting of acute or chronic vasculopathy. In obesity, the baseline inflammation is the setting of an acute insult. It is important to consider methodological limitations of studies evaluating obesity outcomes in critical illness. Separating medical and surgical populations may yield different outcomes. A procedure of intervention in the surgical population may predispose the obese trauma patient to unfavorable outcomes. Among trauma patients, multiple studies demonstrate an association between obesity and worse outcomes, including mortality. [13] Studies of intensive care unit patients compared obese body mass index greater than 30 kg/m<sup>2</sup> to non-obese this group included patients who were underweight body mass index lesser than 18.5 kg/m<sup>2</sup>, a known risk factor for poor outcomes. [14,15]

### **Nutrition Support in the Critically Ill Obese Patient:**

Determination of caloric requirements can be challenging in obese patients. Using 25–30 kcal (kg) per actual or ideal body weight (in kg) may lead to overfeeding or underfeeding. Predictive equations such as Harris-Benedict are not accurate in critically ill patients and were not validated in severe obesity. [16] The Ireton Jones equation does take into account obesity but is not validated for mechanically ventilated critical care patients. [17] When available, indirect calorimetry (IC) should be used to determine energy requirements. IC is the most accurate method for determining energy expenditure however its use is limited by cost and required clinical expertise for interpretation. When patients do not meet valid testing criteria or indirect calorimetry is not available. Amongst critically ill obese patients with a body mass index greater than 45 kg/m<sup>2</sup>, the equation was found

to have the highest prediction accuracy at 76 % ( $\pm 10$  %), as compared to other equations. [18,19] In adults older than 60 years with a body mass index greater than 30 kg/m<sup>2</sup>. Even indirect calorimetry has high variability in measuring energy expenditure for intensive care patients with a body mass index greater than 50 kg/m<sup>2</sup>. [20,21]

### CONCLUSION:

Obesity is a chronic disease and places the individual at risk for additional disease state. The prevalence of obesity is increasing, and the pathophysiology is a complex of genetics, obesogenic environment, and neuroendocrine signaling. One third of all critically ill obese patients are obese, and knowledge of the disease process along with a multi-disciplinary approach will be the key to understand the numerous physical and non-physical challenges. The available outcome data of critically ill obese suggests that the obese critically ill patient has improved outcomes as compared to the nonobese critically ill patient, termed the critical care obesity paradox. This should including patient heterogeneity and the drawbacks of body mass index as the classifier of obesity. Nutrition support of the obese intensive care unit patient requires careful assessment of risk factors for malnutrition, assessment of caloric needs, and consideration for hypocaloric nutrition with protein in selected individuals. Additional prospective studies are needed to provide more definitive recommendations for high-protein hypocaloric nutrition.

### REFERENCES:

- Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence and trends in body mass index among US children and adolescents, 1999-2010. *JAMA*. 2012;307(5):483-90.
- Eknoyan G. A history of obesity, or how what was good became ugly and then bad. *Adv Chronic Kidney Dis*. 2006;13(4):421-7.
- Hurt RT, Frazier TH, McClave SA, Kaplan LM. Obesity epidemic: overview, pathophysiology, and the intensive care unit conundrum. *JPEN J Parenter Enteral Nutr*. 2011;35(5 Suppl):4S-13S.
- Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011-2012. *JAMA*. 2014;311(8):806-14.
- Ogden CL, Lamb MM, Carroll MD, Flegal KM. Obesity and socioeconomic status in adults: United States, 2005-2008. *NCHS Data Brief*. 2010;(50):1-8.
- Lowndes J, Sinnett S, Pardo S, et al. The effect of normally consumed amounts of sucrose or high fructose corn syrup on lipid profiles, body composition and related parameters in overweight/ obese subjects. *Nutrients*. 2014;6(3):1128-44.
- Melanson KJ, Zukley L, Lowndes J, Nguyen V, Angelopoulos TJ, Rippe JM. Effects of high-fructose corn syrup and sucrose consumption on circulating glucose, insulin, leptin, and ghrelin and on appetite in normal-weight women. *Nutrition*. 2007;23(2):103-12.
- Finkielman JD, Gajic O, Afessa B. Underweight is independently associated with mortality in post-operative and non-operative patients admitted to the intensive care unit: a retrospective study. *BMC Emerg Med*. 2004;4(1):3.
- Leonard KL, Davies SW, Waibel BH. Perioperative management of obese patients. *Surg Clin North Am*. 2015;95(2):379-90.
- Shashaty MG, Stapleton RD. Physiological and management implications of obesity in critical illness. *Ann Am Thorac Soc*. 2014;11(8):1286-97.
- Frat JP, Gissot V, Ragot S, et al. Impact of obesity in mechanically ventilated patients: a prospective study. *Intensive Care Med*. 2008;34(11):1991-8.
- Diaz Jr JJ, Norris PR, Collier BR, et al. Morbid obesity is not a risk factor for mortality in critically ill trauma patients. *J Trauma*. 2009;66(1):226-31.
- Liu T, Chen JJ, Bai XJ, Zheng GS, Gao W. The effect of obesity on outcomes in trauma patients: a meta-analysis. *Injury*. 2013;44(9):1145-52.
- Lee CK, Tefera E, Colice G. The effect of obesity on outcomes in mechanically ventilated patients in a medical intensive care unit. *Respiration*. 2014;87(3):219-26.
- Ray DE, Matchett SC, Baker K, Wasser T, Young MJ. The effect of body mass index on patient outcomes in a medical ICU. *Chest*. 2005;127(6):2125-31.
- Hurt RT, Frazier TH, McClave SA, Cave MC. Pharmacologic nutrition for the obese, critically ill patient. *JPEN J Parenter Enteral Nutr*. 2011;35(5 Suppl):60S-72S.
- MacDonald A, Hildebrandt L. Comparison of formulaic equations to determine energy expenditure in the critically ill patient. *Nutrition*. 2003;19(3):233-9.
- Frankenfield DC, Coleman A, Alam S, Cooney RN. Analysis of estimation methods for resting metabolic rate in critically ill adults. *JPEN J Parenter Enteral Nutr*. 2009;33(1):27-36.

19. Choban P, Dickerson R, Malone A, Worthington P, Compher C, American Society for Parenteral and Enteral Nutrition. A.S.P.E.N. clinical guidelines: nutrition support of hospitalized adult patients with obesity. JPEN J Parenter Enteral Nutr. 2013;37(6):714–44. Nutrition support guidelines for the hospitalized obese patient.
20. Frankenfield DC, Ashcraft CM, Galvan DA. Prediction of resting metabolic rate in critically ill patients at the extremes of body mass index. JPEN J Parenter Enteral Nutr. 2013;37(3):361–7.
21. McClave SA, Martindale RG, Kiraly L. The use of indirect calorimetry in the intensive care unit. Curr Opin Clin Nutr Metab Care. 2013;16(2):202–8.