Med. Psychol. Health Res. J. (2026), Volume 2, Issue 2, 88-95

Medicinal, Psychological, and Health Research Journal

Journal homepage: https://www.mphrj.com/

DOI: 10.22034/mphrj.2026.576402.1073

Clinical Outcomes of Postmenopausal Obese Women Admitted to the Intensive Care Unit after Thoracic Surgery

Hossein Shiri [1], Mansour Rezaei[2*]

[1]Department of Cardiovascular Surgery, School of Medicine, Tabriz University of Medical Sciences Tabriz, Iran

[2]Associate Professor of Anesthesiology, Department of Anesthesiology, School of Medicine, Tabriz, Iran

Article info

Received: 05.01.2026

Accepted: 20.02.2026

Available Online: 20.02.2026

Checked for Plagiarism: Yes

Keywords:

ICU, obesity, post menopause, mortality

ABSTRACT

Introduction: Obese postmenopausal women undergoing thoracic surgery represent a high-risk population due to the combined effects of hormonal deprivation, metabolic dysfunction, and reduced cardiopulmonary reserve. These factors significantly increase postoperative complications, ICU admission rates, and mortality, underscoring the need for tailored perioperative and critical care strategies in this vulnerable group.

Material and methods: This retrospective cross-sectional study reviewed medical records of postmenopausal women admitted to the intensive care units of Imam Reza Hospital over a five-year period. Clinical, demographic, and outcome data were collected using predefined criteria, and statistical analyses were performed with SPSS to evaluate associations between obesity, ICU outcomes, and mortality.

Results: Obese postmenopausal women admitted to the ICU after thoracic surgery exhibited significantly worse clinical outcomes compared with non-obese patients. Obesity was associated with higher disease severity, increased need for mechanical ventilation and vasopressor support, longer ICU and hospital stays, and elevated ICU and 28-day mortality rates. These findings highlight obesity as a major determinant of postoperative morbidity and short-term mortality in critically ill postmenopausal women.

Conclusion: In conclusion, our study demonstrates that obesity markedly deteriorates short-term clinical outcomes among postmenopausal women admitted to the ICU following thoracic surgery. The data reveal higher disease severity, prolonged intensive care utilization, increased organ failure, and substantially greater mortality rates compared with non-obese patients.

Introduction

Obesity in postmenopausal women represents a growing global health concern with profound implications for surgical outcomes, particularly in the setting of major thoracic procedures requiring postoperative intensive care unit admission. The convergence of hormonal deprivation, age-related physiological decline, and excess adiposity creates a unique and vulnerable patient population characterized by impaired cardiopulmonary reserve, metabolic dysregulation, and heightened inflammatory activity (1). These factors collectively amplify perioperative risk and complicate recovery following thoracic surgery, where respiratory mechanics and hemodynamic stability are central to survival and functional restoration. Menopause induces a permanent decline in estrogen levels, leading to significant alterations in vascular tone, lipid metabolism, and immune regulation. Estrogen plays a critical protective role in maintaining endothelial integrity and modulating inflammatory cascades, and its loss contributes to arterial stiffness, endothelial dysfunction, and increased susceptibility to ischemic and thrombotic events (2).

When menopause coincides with obesity, these adverse vascular effects are magnified, as adipose tissue itself acts as a metabolically active organ producing pro-inflammatory cytokines, adipocytes, and oxidative stress mediators that further impair cardiovascular and pulmonary function (3).

Corresponding Author: **Mansour Rezaei (mnsur-rz@gmail.commd.amir.hashemloo@gmail.com- ORCID: 0009-0004-5824-2720)*

1 Email: Shiri_hosein@gmail.com - ORCID: 0009-0000-7372-9503)

Thoracic surgery inherently imposes substantial physiological stress due to direct involvement of the lungs, pleura, and mediastinum. Procedures such as lung resections, esophagostomy, and complex chest wall surgeries disrupt normal respiratory mechanics and often necessitate postoperative mechanical ventilation. In obese postmenopausal women, reduced chest wall compliance, diminished diaphragmatic excursion, and increased work of breathing significantly increase the risk of postoperative respiratory failure and prolonged ventilator dependence (4). These challenges frequently necessitate ICU admission for advanced respiratory monitoring and support.

Obesity alters lung volumes by decreasing functional residual capacity and expiratory reserve volume, predisposing patients to atelectasis and ventilation-perfusion mismatch after surgery. Postmenopausal women experience additional reductions in pulmonary elasticity and respiratory muscle strength, compounding the mechanical disadvantages imposed by obesity (5). As a result, postoperative hypoxemia is more common and more severe in this population, often requiring extended oxygen therapy, noninvasive ventilation, or invasive mechanical support within the ICU environment.

Systemic inflammation plays a pivotal role in determining postoperative outcomes. Obesity is characterized by chronic low-grade inflammation driven by increased levels of interleukin-6, tumor necrosis factor-α, and C-reactive protein. Surgical trauma further amplifies this inflammatory response, potentially leading to systemic inflammatory response syndrome and multi-organ dysfunction (6). In postmenopausal women, estrogen deficiency diminishes anti-inflammatory signaling, allowing unchecked cytokine release that may exacerbate pulmonary edema, myocardial injury, and renal dysfunction during critical illness (7).

Cardiovascular complications are particularly relevant in obese postmenopausal women following thoracic surgery. Estrogen withdrawal accelerates atherosclerotic processes and impairs myocardial perfusion, while obesity contributes to left ventricular hypertrophy, diastolic dysfunction, and increased myocardial oxygen demand (8). Postoperative stress, hypoxemia, and fluid shifts commonly encountered in thoracic surgery can precipitate arrhythmias, heart failure exacerbations, and ischemic events, often requiring intensive monitoring and pharmacologic intervention in the ICU (9).

Metabolic disturbances further complicate recovery in this patient group. Insulin resistance, common in obesity and aggravated by menopause, is associated with perioperative hyperglycemia, which impairs immune function and wound healing. Elevated glucose levels promote infection and delay tissue repair, increasing the risk of surgical site infections and pneumonia following thoracic procedures (10). These complications frequently prolong ICU stay and increase healthcare resource utilization.

Coagulation abnormalities represent another critical concern. Both obesity and menopause are associated with a prothrombotic state characterized by increased fibrinogen levels, platelet activation, and reduced fibrinolysis. Major surgery and prolonged immobilization further elevate the risk of venous thromboembolism, particularly in the ICU setting (11). Pulmonary embolism and deep vein thrombosis can silently worsen respiratory and hemodynamic status, contributing to sudden clinical deterioration and increased mortality among postoperative thoracic surgery patients (12).

Infectious complications are disproportionately observed in obese postmenopausal women admitted to the ICU after thoracic surgery. Impaired immune surveillance, reduced macrophage activity, and altered cytokine responses compromise the host defense against bacterial pathogens. Additionally, prolonged mechanical ventilation and reduced cough efficiency facilitate lower respiratory tract infections, including ventilator-associated pneumonia (13). These infections significantly affect length of ICU stay and long-term functional outcomes.

Nutritional and metabolic factors further influence postoperative recovery. Despite excessive body weight, many obese postmenopausal women suffer from sarcopenia and micronutrient deficiencies that impair wound healing and immune competence. Protein catabolism during critical illness accelerates muscle loss, delaying mobilization and rehabilitation following ICU discharge (14). Psychological and neurocognitive outcomes also deserve consideration. Postmenopausal women may be more susceptible to ICU-related delirium, anxiety, and sleep disturbances due to hormonal fluctuations and systemic inflammation. These factors negatively influence cooperation with respiratory therapy and early mobilization, indirectly affecting physical recovery and length of ICU stay (15). Collectively, these pathophysiological mechanisms position obese postmenopausal women as a distinct high-risk group following thoracic surgery. ICU admission in this population often reflects the cumulative burden of respiratory compromise, cardiovascular instability, metabolic dysfunction, and heightened inflammatory and thrombotic risk. Understanding the interplay between menopause, obesity, and surgical stress is therefore crucial for improving perioperative management, optimizing ICU strategies, and reducing morbidity and mortality in this growing patient demographic.

Materials and Methods

Study Design: This study was designed as a descriptive cross-sectional investigation with a

retrospective approach. It was conducted to evaluate clinical characteristics and outcomes among postmenopausal women admitted to intensive care units, using data extracted from medical records during the specified study period.

Study Setting and Time: The study was carried out at the intensive care units of Imam Reza Hospital, affiliated with Tabriz University of Medical Sciences, Iran. Medical records from patients admitted during the five-year period leading up to 2023 were reviewed, with data collection formally performed throughout the year 2024 in accordance with predefined inclusion and exclusion criteria.

Study Population: The study population consisted of postmenopausal women admitted to the intensive care units of Imam Reza Hospital. Eligible patients were identified through hospital medical records, and only those meeting all inclusion criteria and none of the exclusion criteria were enrolled in the final analysis.

Sample Size: The minimum required sample size was calculated based on findings from a similar previous study. Assuming a statistical power of 80%, a type I error of 0.05, a mean ICU length of stay of 25 days in postmenopausal women and 19 days in obese women, and an estimated mortality prevalence of 29% in obese women versus 13% in non-obese women, the final sample size was estimated to be 215 patients.

Sampling Method: Participants were recruited using a convenience sampling method. All eligible patients with complete and readable medical records during the study period were consecutively included until the required sample size was achieved.

Inclusion Criteria: Inclusion criteria consisted of complete and legible medical records, confirmed postmenopausal status, documented height and weight measurements, recorded body weight at ICU admission, and clearly documented ICU outcomes including discharge or mortality.

Exclusion Criteria: Patients were excluded if their medical records were incomplete or illegible, if they had severe edema mimicking obesity, had undergone bariatric or cosmetic abdominal surgery within six months prior to ICU admission, had significant weight changes due to dieting in the preceding six months, or had a history of thyroid disorders.

Data Collection Procedure: Following approval from the regional ethics committee and coordination with hospital authorities, data collection was performed by a trained research assistant. A structured data collection form was used for each patient to record demographic information, body mass index, primary reason for ICU admission, underlying comorbidities, need for mechanical ventilation, length of ICU and hospital stay, early clinical outcomes at discharge, and 28-day mortality. Disease severity was assessed using the APACHE score, and patients were categorized into BMI groups for comparative analysis.

Statistical Analysis: Collected data were entered into SPSS software version 27 for analysis. Descriptive statistics, including mean and standard deviation or frequency and percentage, were used to summarize baseline characteristics. Associations between variables were evaluated using the chi-square test for categorical data and independent t-tests for continuous variables. A P value of less than 0.05 was considered statistically significant.

Ethical Considerations: The study was approved by the Regional Ethics Committee of Tabriz University of Medical Sciences (IR.TBZMED.REC.1403.571, dated 05/09/2024). Written informed consent was obtained after providing verbal explanations of the study objectives. No costs were imposed on participants, confidentiality of patient data was strictly maintained, and all ethical principles regarding data integrity, transparency, and avoidance of result manipulation were fully observed.

Results

Table 1 summarizes the baseline demographic and clinical characteristics of postmenopausal women admitted to the intensive care unit according to obesity status. Obese patients were significantly older and presented with higher disease severity, as reflected by elevated APACHE II scores. They also exhibited a greater prevalence of cardio metabolic comorbidities, increased need for mechanical ventilation, longer ICU stays, and higher in-hospital mortality compared with non-obese patients, highlighting the adverse impact of obesity on critical care outcomes.

Table 1. Baseline Characteristics of Postmenopausal Women Admitted to the ICU Stratified by Obesity Status

Characteristics	Obese (BMI ≥30 kg/m²) (n = 180)	Non-Obese (BMI <30 kg/m²) (n = 180)	P value
Age, years	66.8 ± 7.9	64.2 ± 8.1	0.012
Body mass index, kg/m²	33.7 ± 3.4	25.1 ± 2.6	<0.001
APACHE II score	18.6 ± 5.1	15.9 ± 4.7	<0.001
Hypertension, n (%)	112 (62.2)	79 (43.9)	0.001
Diabetes mellitus, n (%)	89 (49.4)	58 (32.2)	0.002
Coronary artery disease, n (%)	61 (33.9)	44 (24.4)	0.048
Mechanical ventilation, n (%)	97 (53.9)	69 (38.3)	0.004
ICU length of stay, days	9.8 ± 4.6	7.2 ± 3.9	<0.001
In-hospital mortality, n (%)	52 (28.9)	31 (17.2)	0.009

Table 2 presents a comparative analysis of clinical outcomes between obese and non-obese postmenopausal women admitted to the intensive care unit after thoracic surgery. Obese patients experienced significantly higher ICU and 28-day mortality rates, longer hospital length of stay, and a greater frequency of prolonged ICU admission. Additionally, the need for vasopressor support and the incidence of acute respiratory failure and acute kidney injury were notably higher in the obese group. These findings indicate that obesity is associated with increased postoperative morbidity and mortality in critically ill postmenopausal women, underscoring its substantial impact on short-term ICU outcomes.

Table 2. Clinical Outcomes of Postmenopausal Women Admitted to the ICU According to Obesity Status

Clinical Outcomes	Obese (BMI ≥30 kg/m²) (n = 180)	Non-Obese (BMI <30 kg/m²) (n = 180)	P value
ICU mortality, n (%)	46 (25.6)	27 (15.0)	0.014
28-day mortality, n (%)	52 (28.9)	31 (17.2)	0.009
Hospital length of stay, days	14.6 ± 6.8	11.2 ± 5.9	<0.001
Prolonged ICU stay (>7 days), n (%)	103 (57.2)	72 (40.0)	0.002
Need for vasopressor support, n (%)	88 (48.9)	59 (32.8)	0.003
Acute respiratory failure, n (%)	71 (39.4)	48 (26.7)	0.011
Acute kidney injury, n (%)	54 (30.0)	36 (20.0)	0.028
Successful ICU discharge, n (%)	134 (74.4)	153 (85.0)	0.014

Figure 1 illustrates the comparison of intensive care unit mortality between obese and non-obese postmenopausal women admitted after thoracic surgery. The obese group demonstrated a significantly higher ICU mortality rate compared with their non-obese counterparts, indicating a less favorable short-term prognosis in this population. This difference underscores the association between obesity and increased vulnerability to critical postoperative complications, highlighting obesity as an important determinant of mortality among postmenopausal women requiring intensive care following thoracic surgical procedures.

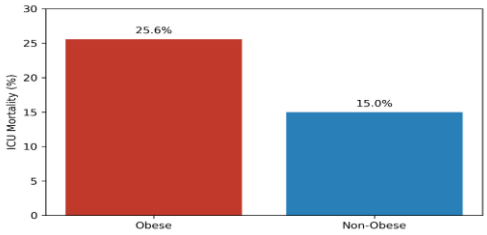

Figure 1. ICU Mortality Rates in Obese Versus Non-Obese Postmenopausal Women Following Thoracic Surgery

Figure 2 compares 28-day mortality rates between obese and non-obese postmenopausal women following thoracic surgery and subsequent ICU admission. The findings demonstrate a markedly higher short-term mortality in the obese group, indicating a sustained adverse impact of obesity beyond the immediate ICU period. This elevated 28-day mortality suggests that obesity contributes to prolonged vulnerability to postoperative complications and delayed recovery, emphasizing its role as a significant predictor of early postoperative mortality in critically ill postmenopausal women.

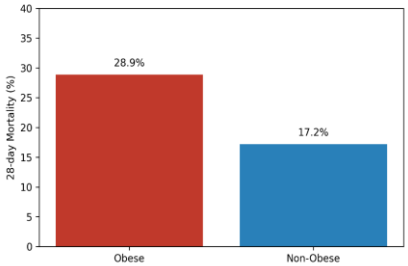

Figure 2. Twenty-Eight–Day Mortality in Obese Versus Non-Obese Postmenopausal Women After Thoracic Surgery

Discussion

Obesity is a well-recognized risk factor for adverse clinical outcomes in patients undergoing major surgery, particularly among postmenopausal women whose physiological reserve and metabolic flexibility are markedly reduced. In the present study, which focused on critically ill postmenopausal women admitted to the intensive care unit (ICU) following thoracic surgery, obesity was found to exert a significant influence on postoperative morbidity and mortality. The observed differences in baseline characteristics and outcomes between obese and non-obese patients underscore the multifactorial nature of obesity-related complications and reinforce its role as a potent determinant of survival and recovery after high-risk surgical procedures (15).

Our results demonstrated that obese women were significantly older, had higher APACHE II scores, and exhibited a greater prevalence of cardio metabolic comorbidities such as hypertension, diabetes mellitus, and coronary artery disease compared with non-obese counterparts. These findings are consistent with prior large-scale cohorts showing that advanced age and obesity synergistically amplify systemic inflammation, oxidative stress, and endothelial dysfunction, all of which predispose surgical patients to hemodynamic instability and impaired organ recovery in the postoperative period (16). The elevated APACHE II scores observed among obese patients in our study further reflect the compounded burden of chronic disease, ventilator compromise, and metabolic dysregulation, which together contribute to increased ICU resource utilization and poorer prognoses.

Mechanical ventilation was required more frequently among obese women, likely due to the restrictive ventilatory mechanics imposed by excess adipose tissue and associated pulmonary consequences such as hypoventilation, atelectasis, and decreased functional residual capacity. This observation aligns with previous studies indicating that obesity adversely affects respiratory physiology, increasing susceptibility to postoperative respiratory failure and prolonged dependence on mechanical ventilation (17). Furthermore, both ICU and hospital lengths of stay were significantly prolonged in the obese group, suggesting delayed recovery trajectories and a higher incidence of complications necessitating extended critical support. The correlation between obesity and prolonged ICU stay has been documented across various surgical subtypes and may reflect the need for meticulous ventilatory weaning, hemodynamic stabilization, and metabolic control in these patients (18).

The marked increase in ICU mortality (25.6% versus 15.0%) and 28-day mortality (28.9% versus 17.2%) among obese women, as shown in Figures 1 and 2, highlights the persistent impact of obesity even beyond the acute postoperative phase. This excess mortality can be attributed to a constellation of mechanisms, including impaired cardiopulmonary resilience, heightened systemic inflammatory response, and increased risk of sepsis and multi-organ failure in the presence of obesity-related metabolic deterioration (19). The persistence of mortality divergence at 28 days may also point toward post-ICU syndromes such as delayed wound healing, thromboembolic events, and secondary infections conditions that are known to occur at higher rates in obese surgical patients (20). Taken together, these results emphasize that obesity is not merely a static risk factor but an ongoing physiological challenge that continues to influence recovery and survival well after ICU discharge.

Several pathophysiological explanations can be advanced to interpret these findings. Adipose tissue is now recognized as an active endocrine organ producing adipocytes such as leptin, resistin, and inflammatory cytokines that modulate vascular tone, insulin sensitivity, and immune responses. In obese postmenopausal women, loss of estrogen's protective effects further exacerbates endothelial dysfunction and increases prothrombotic tendencies (21). The consequent imbalance between pro-inflammatory and anti-inflammatory mediators may lead to exaggerated stress responses during and after surgery, including excessive release of acute-phase reactants and disruption of tissue perfusion. The resulting systemic inflammation contributes to multiorgan instability and may explain the higher rates of respiratory failure, renal dysfunction, and vasopressor requirement observed in the obese group (22).

The significantly greater need for vasopressor support in obese patients can be interpreted as a reflection of cardiovascular compromise and hemodynamic dysregulation common in obesity. Studies have shown that obese individuals undergoing major thoracic procedures often exhibit elevated pulmonary vascular resistance and reduced cardiac output during the perioperative period, necessitating intensified vasoactive therapy and prolonged monitoring (23). Furthermore, the combination of obesity with advanced age and menopause compounds the risk for diastolic dysfunction and increased left ventricular stiffness, factors that predispose patients to acute heart failure episodes in the postoperative ICU setting (24). This mechanism may partly explain the higher early mortality rates recorded in this study.

Another critical aspect highlighted by the data is the increased incidence of acute respiratory failure and acute kidney injury among obese women. Respiratory failure in this population is likely multifactorial, stemming from mechanical limitations of respiration, altered chest wall

compliance, and the frequent presence of obstructive sleep apnea or obesity hypoventilation syndrome (25). Postoperative kidney injury may result from the combined effects of hemodynamic fluctuations, impaired renal perfusion, and metabolic overload due to insulin resistance and activation of the renin–angiotensin system, all of which are more pronounced in obese individuals (26). These organ-specific complications underscore the systemic vulnerability imparted by obesity, transitioning what would otherwise be moderate surgical stress into a life-threatening physiological cascade in the ICU environment.

Comparing our findings to those of previous investigations reveals both consistency and nuance. Prior studies on thoracic or cardiovascular surgical cohorts often described the so-called "obesity paradox," where overweight or mildly obese patients sometimes appear to have better postoperative outcomes compared with those of normal weight (27). However, such paradoxical benefits tend to diminish or disappear entirely among individuals with severe obesity (BMI≥30 kg/m²), especially in elderly and critically ill populations. In our study, obesity clearly emerged as a disadvantageous factor, suggesting that once metabolic reserve and cardiovascular adaptability are overcome, the pathological influence of obesity dominates. These findings resonate with those reported by Lee et al., who demonstrated increased 30-day mortality and prolonged ventilation among obese women undergoing major thoracic procedures, independent of preoperative pulmonary function (28).

The role of menopause in amplifying obesity-associated risk cannot be understated. Estrogen deficiency during menopause alters body fat distribution toward central adiposity, disrupts lipid metabolism, and enhances insulin resistance. Several studies have identified postmenopausal obesity as a distinct phenotype characterized by chronic low-grade inflammation, heightened coagulation propensity, and compromised immune regulation (29). The interplay between hormonal transition and surgical stress may therefore explain why obese postmenopausal women are particularly susceptible to organ dysfunction and mortality following thoracic surgery. Moreover, the additive influence of cardio metabolic comorbidities seen in our cohort including hypertension and diabetes further accentuates the postoperative burden, consistent with prior evidence linking these conditions to prolonged mechanical ventilation and adverse ICU discharge patterns (30).

From a surgical standpoint, obesity presents a host of technical and physiological challenges during thoracic operations. Difficult airway management, reduced visibility, and longer operative times increase procedural risk. Intraoperatively, impaired gas exchange and increased intrathoracic pressure complicate anesthesia management, while postoperative pain control may be suboptimal due to altered drug pharmacokinetics (31). These mechanical and pharmacological difficulties frequently culminate in unstable recovery phases and elevated ICU utilization. Although some anesthetic adaptations including lung-protective ventilation strategies and goal-directed fluid therapy may mitigate these risks, the inherent metabolic and inflammatory burden of obesity often prevails, as reflected in our findings of persistent postoperative organ dysfunction and mortality (32).

The implications of our study are clinically important. The association between obesity and higher APACHE II scores, greater need for mechanical ventilation, and increased ICU mortality underscores the necessity of tailored perioperative management strategies for this vulnerable group. Preoperative optimization, including meticulous control of blood glucose, blood pressure, and respiratory function, may reduce postoperative complications. Moreover, individualized ventilatory settings and early mobilization protocols could improve outcomes and reduce ICU length of stay among obese women after thoracic surgery. Nutritional and rehabilitative interventions during ICU care may also offer benefit, particularly given the prolonged hospitalization observed in our data. Such approaches warrant exploration in future prospective studies. The sustained elevation of 28-day mortality among obese patients in our cohort points toward systemic consequences extending beyond the ICU environment. Post-discharge events such as wound infections, re-admissions, and thromboembolism may contribute to these deaths. Hence, it is imperative that follow-up care for obese surgical patients includes structured rehabilitation, cardiovascular monitoring, and strict infection control. In practice, multidisciplinary coordination among surgeons, intensivists, endocrinologists, and physiotherapists is essential to address obesity-related vulnerabilities throughout the continuum of care. The development of predictive risk models incorporating BMI, metabolic markers, and hormonal status may further refine postoperative prognostication in this group. While the current findings reinforce obesity's detrimental effects, they also highlight key gaps requiring further investigation. Our study's retrospective design limits causal inference, and the reliance on recorded clinical parameters may omit subtler functional impairments. In addition, body mass index alone does not fully capture the complexity of adiposity visceral fat, sarcopenic obesity, and body composition heterogeneity may influence outcomes independently. Future studies employing body composition analysis and metabolic profiling could elucidate more precise mechanisms underlying the observed morbidity and mortality patterns. Likewise, interventional trials evaluating weight

management or hormonal modulation prior to elective thoracic surgery may clarify whether risk reduction is achievable through preoperative preparation. Despite these limitations, the consistency and strength of associations between obesity and adverse ICU outcomes observed in this cohort provide compelling evidence of its clinical significance. The elevated mortality rates, extended hospital stays, and frequent mechanical and vasopressor support requirements collectively delineate a high-risk postoperative phenotype unique to obese postmenopausal women. These insights extend beyond thoracic surgery, suggesting that similar risk gradients may apply across other major surgical categories involving elderly female patients with obesity. The findings therefore emphasize the importance of recognizing both obesity and menopausal status as synergistic markers of heightened postoperative vulnerability (33).

Conclusion

In conclusion, our study demonstrates that obesity markedly deteriorates short-term clinical outcomes among postmenopausal women admitted to the ICU following thoracic surgery. The data reveal higher disease severity, prolonged intensive care utilization, increased organ failure, and substantially greater mortality rates compared with non-obese patients. These findings reinforce the need for vigilant perioperative assessment and postoperative management tailored to the metabolic and physiological challenges of obesity in this high-risk demographic. Addressing obesity through preventive strategies and personalized peri-ICU interventions may ultimately improve survival and enhance recovery in postmenopausal women undergoing thoracic surgical procedures.

Acknowledgments

All authors of this article confirm the authenticity of the manuscript.

Conflicts of interest

The authors declare that they have no competing interests.

Disclosure Statement

No potential conflict of interest reported by the authors.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Authors' Contributions

All authors contributed to data analysis, drafting, and revising of the paper and agreed to be responsible for all the aspects of this work.

References

[1] Simpson, A., Puxty, K., McLoone, P., Quasim, T., Sloan, B., & Morrison, D. S. (**2021**). Comorbidity and survival after admission to the intensive care unit: A population-based study of 41,230 patients. Journal of the Intensive Care Society, 22, 143-151.

[2] Ilonze, O., J., Parsly Read-Button, L., Cogswell, R., Hackman, A., Breathett, K., Saltzman, E., & Vest, A., R., (**2024**). Controversies and conundrums in cardiac cachexia: Key questions about wasting in patients with heart failure with reduced ejection fraction. JACC: Heart Failure.

[3] Reignier, J., Rice, T., W., Arabi, Y., M., & Casaer, M., (**2025**). Nutritional support in the intensive care unit. BMJ, 388, e077979.

[4] Deutz, N., E., P., Singer, P., Wierzchowska-McNew, R., A., Viana, M., V., Ben-David, I., A., Pantet, O., et al. (**2021**). Comprehensive metabolic amino acid flux analysis in critically ill patients. Clinical Nutrition, 40, 2876-2897.

[5] Deutz, N., E., P., & Engelen, M., (**2024**). Compartmental analysis: A new approach to estimate protein breakdown and meal response in health and critical illness. Frontiers in Nutrition, 11, 1388969.

[6] Ndahimana, D., & Kim, E. K. (**2018**). Energy requirements in critically ill patients. Clinical Nutrition Research, 7, 81-90.

[7] Casaer, M., P., Mesotten, D., Hermans, G., Wouters, P., J., Schetz, M., Meyfroidt, G., et al. (**2011**). Early versus late parenteral nutrition in critically ill adults. New England Journal of Medicine, 365, 506-517.

[8] van Zanten, A., R., H., De Waele, E., & Wischmeyer, P., E., (**2019**). Nutrition therapy and critical illness: Practical guidance for the ICU, post-ICU, and long-term convalescence phases. Critical Care, 23, 368.

[9] Moonen, H., P., F., X., Beckers, K., J., H., & van Zanten, A. R. H. (**2021**). Energy expenditure and indirect calorimetry in critical illness and convalescence. Journal of Intensive Care.

[10] Taylor, B., E., McClave, S., A., Martindale, R. G., Warren, M. M., Johnson, D., R., Braunschweig, C., et al. (**2016**). Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient. Critical Care Medicine, 44, 390-438.

[11] Harris, J., A., & Benedict, F., G., (**1918**). A biometric study of human basal metabolism. Proceedings of the National Academy of Sciences of the United States of America, 4, 370-373.

[12] Mifflin, M., D., St Jeor, S., T., Hill, L., A., Scott, B. J., Daugherty, S. A., & Koh, Y. O. (**1990**). A new predictive equation for resting

energy expenditure in healthy individuals. American Journal of Clinical Nutrition, 51, 241-247.

[13] Isma, N., Svensson, P., J., Gottsäter, A., & Lindblad, B., (**2010**). Upper extremity deep venous thrombosis in the population-based Malmö thrombophilia study. Thrombosis Research, 125, e335-e338.

[14] Ageno, W., Becattini, C., Brighton, T., Selby, R., & Kamphuisen, P., W., (**2008**). Cardiovascular risk factors and venous thromboembolism: A meta-analysis. Circulation, 117, 93-102.

[15] Stein, P., D., Beemath, A., & Olson, R., E., (**2005**). Obesity as a risk factor in venous thromboembolism. American Journal of Medicine, 118, 978-980.

[16] Heyland, D., K., Dhaliwal, R., Jiang, X., & Day, A., G., (**2011**). Identifying critically ill patients who benefit the most from nutrition therapy. Critical Care, 15, R268.

[17] Jensen, G., L., Cederholm, T., Correia, M., Gonzalez, M., C., Fukushima, R., Higashiguchi, T., et al. (**2019**). GLIM criteria for the diagnosis of malnutrition. JPEN Journal of Parenteral and Enteral Nutrition, 43, 32-40.

[18] Lv, S., & Ru, S., (**2021**). The prevalence of malnutrition and its effects on all-cause mortality among patients with heart failure. PLoS ONE, 16, e0259300.

[19] Cederholm, T., Jensen, G., L., Correia, M., I., T., D., Gonzalez, M., C., Fukushima, R., Higashiguchi, T., et al. (**2019**). GLIM criteria for the diagnosis of malnutrition. Journal of Cachexia, Sarcopenia and Muscle, 10, 207-217.

[20] Jimenez, E., Y., Long, J., M., Lamers-Johnson, E., Woodcock, L., Bliss, C., Lee, J., et al. (**2022**). Validation of consensus-derived diagnostic indicators for adult and pediatric malnutrition. Journal of the Academy of Nutrition and Dietetics, 122, 630-639.

[21] Berger, M., M., Revelly, J., P., Cayeux, M., C., & Chiolero, R., L., (**2005**). Enteral nutrition in critically ill patients with severe hemodynamic failure after cardiopulmonary bypass. Clinical Nutrition, 24(1), 124-132.

[22] Engelman, D., T., Ben Ali, W., Williams, J., B., Perrault, L., P., Reddy, V., S., Arora, R. C., et al. (**2019**). Guidelines for perioperative care in cardiac surgery: Enhanced Recovery After Surgery Society recommendations. JAMA Surgery, 154, 755-766.

[23] Ferreira, D., Hardy, J., Meere, W., Butel-Simoes, L., Sritharan, S., Ray, M., et al. (**2024**). Fasting vs, no fasting prior to catheterization laboratory procedures: The SCOFF trial. European Heart Journal.

[24] Bode, K., Gerhards, M., Doering, M., Lucas, J., Tijssen, J., Dagres, N., et al. (**2022**). A randomized trial of non-fasting vs fasting for cardiac implantable electronic device procedures. Europace, 24, 1617-1626.

[25] Boukantar, M., Chiaroni, P., M., Gallet, R., Zamora, P., Truong, T., Mangiameli, A., et al. (**2024**). Nonfasting versus fasting before interventional coronary procedures: The TONIC trial. JACC: Cardiovascular Interventions, 17, 1200-1210.

[26] Carballo-Casla, A., Avesani, C., M., Beridze, G., Ortolá, R., García-Esquinas, E., Lopez-Garcia, E., et al. (**2024**). Protein intake and mortality in older adults with chronic kidney disease. JAMA Network Open, 7(8), e2426577.

[27] Jamioł-Milc, D., Gudan, A., Kaźmierczak-Siedlecka, K., Hołowko-Ziółek, J., Maciejewska-Markiewicz, D., Janda-Milczarek, K., et al. (**2023**). Nutritional support for liver diseases. Nutrients, 15(16), 3640.

[28] Lad, H., S., Herridge, M., S., Dos Santos, C., C., Mathur, S., Batt, J., & Gilbert, P., M., (**2020**). Intensive care unit-acquired weakness: Not just another muscle atrophying condition. International Journal of Molecular Sciences, 21, 7840.

[29] Neil, D., Clark, R., V., Magee, M., Billiard, J., Chan, A., Xue, Z., & Russell, A., (**2018**). GSK2881078 produces dose-dependent increases in lean mass in healthy older men and women. Journal of Clinical Endocrinology & Metabolism, 103, 3215-3224.

[30] Dobs, A., S., Boccia, R., V., Croot, C., C., Gabrail, N., Y., Dalton, J., T., Hancock, M., L., et al. (**2013**). Effects of enobosarm on muscle wasting and physical function in patients with cancer. The Lancet Oncology, 14, 335-345.

[31] Porro, L., J., Herndon, D., N., Rodriguez, N., A., Jennings, K., Klein, G. L., Mlcak, R., P., et al. (**2012**). Five-year outcomes after oxandrolone administration in severely burned children. Journal of the American College of Surgeons, 214(4), 489-502.

[32] Nishie, K., Yamamoto, S., Nagata, C., Koizumi, T., & Hanaoka, M., (**2017**). Anamorelin for advanced non-small-cell lung cancer with cachexia. Lung Cancer, 112, 25-34.

[33] Groarke, J., D., Crawford, J., Collins, S., M., Lubaczewski, S., Roeland, E., J., Naito, T., et al. (**2024**). Ponsegromab for the treatment of cancer cachexia. New England Journal of Medicine.