Journal of Cardiovascular Disease Research

ISSN: 0975-3583, 0976-2833 VOL9, ISSUE 4, 2018

The Role of Body Composition in Determining Metabolic Syndrome Severity Among Obese Individuals.

Dr. Lakavath Sunitha, General Medicine, Assistant Professor, Dhanalakshmi Srinivasan Medical College & Hospital, Tamilnadu

Publication Date:12/12/2018

Abstract

Background: Metabolic syndrome (MetS), characterized by obesity, hypertension, and metabolic dysregulation, elevates cardiovascular disease (CVD) risk. This study investigated the association between body composition and MetS severity in obese individuals.

Methods: A cross-sectional analysis of 180 obese patients from an obesity center was conducted. Body mass index (BMI), fat mass, muscle mass, and MetS severity score (MetS-S) were assessed. Logistic regression was used to determine the relationship between body composition parameters and MetS-S.

Results: The cohort was predominantly female (92.8%). Higher MetS-S was associated with significantly increased age, BMI, fat mass, muscle mass, and fat/muscle ratios. Logistic regression revealed that a 1-unit increase in BMI was associated with a 1.288-fold increase in the odds of higher MetS-S ($p < 0.05$). However, fat mass, muscle mass, and their percentages were not significantly associated with MetS-S.

Conclusions: BMI is a key determinant of MetS severity in obese individuals, while fat and muscle mass showed no independent association. These findings emphasize the clinical importance of BMI management in obese patients with MetS. Further research with larger, more diverse samples is needed to validate these findings and explore the complex interplay between body composition and MetS severity.

Keywords: body mass index, metabolic syndrome severity score, body composition, obesity.

ISSN: 0975-3583, 0976-2833 VOL9, ISSUE 4, 2018

Introduction

Metabolic syndrome (MetS) represents a cluster of interconnected metabolic abnormalities that significantly elevate the risk of cardiovascular diseases (CVD), type 2 diabetes mellitus, and overall mortality. Characterized by central obesity, hypertension, dyslipidemia (elevated triglycerides and low high-density lipoprotein cholesterol), and impaired glucose tolerance, MetS has become a major public health concern globally, driven by escalating rates of obesity and sedentary lifestyles. Understanding the intricate interplay of factors contributing to the severity of MetS is crucial for developing effective preventive and therapeutic strategies. Obesity, particularly abdominal or visceral adiposity, plays a pivotal role in the pathogenesis of MetS. Excess adipose tissue, especially when located centrally, contributes to systemic inflammation, insulin resistance, and dyslipidemia through the release of various adipokines and inflammatory mediators. Consequently, body composition, encompassing the distribution and quantity of fat mass and muscle mass, emerges as a critical determinant of metabolic health. Traditionally, body mass index (BMI) has been widely used to assess obesity and its associated health risks. While BMI provides a simple and readily available measure of overall adiposity, it fails to distinguish between fat mass and muscle mass, both of which have distinct metabolic implications. Recent advancements in body composition analysis, such as bioelectrical impedance analysis (BIA) and dual-energy X-ray absorptiometry (DXA), have enabled more precise quantification of fat mass, muscle mass, and their regional distribution. These techniques offer valuable insights into the complex relationship between body composition and metabolic health. The severity of MetS can vary significantly among individuals, reflecting differences in the degree of metabolic derangement. Various scoring systems have been developed to quantify MetS severity, incorporating multiple metabolic parameters into a single composite score. These scores provide a comprehensive assessment of metabolic risk and may offer a more nuanced understanding of the relationship between body composition and MetS. While numerous studies have investigated the association between obesity and MetS, the specific contributions of different body composition parameters, such as fat mass and muscle mass, to MetS severity remain less clear. Muscle mass, often overlooked in obesity research, plays a crucial role in glucose metabolism and insulin sensitivity. Sarcopenia, the age-related loss of muscle mass, has been linked to increased insulin resistance and metabolic dysfunction. Conversely, increased muscle mass may have protective effects against MetS by enhancing glucose uptake and improving metabolic profile. The current study aims to investigate the relationship between body composition measurements, including BMI, fat mass, and muscle mass, and the severity of MetS in a cohort of obese individuals. By examining the association between these parameters and a validated MetS severity score, this research seeks to provide a more comprehensive understanding of the role of body composition in the pathogenesis and progression of MetS. Specifically, this study will evaluate if fat mass and muscle mass, independently of BMI, contribute to the severity of MetS in obese individuals. The findings of this study have significant clinical implications. A better understanding of the relationship between body composition and MetS severity can inform the development of personalized interventions aimed at improving metabolic health in obese individuals. Identifying specific body composition parameters that strongly predict MetS severity may help clinicians prioritize targeted interventions, such as exercise programs aimed at increasing muscle mass or dietary modifications to reduce fat mass. Moreover, this research may highlight the limitations of relying solely on BMI for assessing metabolic risk in obese populations, emphasizing the need for more comprehensive body composition assessments. Future research should build on these findings by exploring the longitudinal relationship between body composition changes and

ISSN: 0975-3583, 0976-2833 VOL9, ISSUE 4, 2018

MetS progression, and by evaluating the impact of interventions on both body composition and MetS severity.

Materials and Methods:

Study design: - This cross-sectional study was approved by the Department of General Medicine, Mamata Medical College, Khammam**.** scientific research ethics committee. Informed consent regarding medical interventions is obtained from patients when they first apply to the obesity center. In addition, since the data were collected from their medical records, informed consent was not required.

Population selection: - The sample size was calculated using the formula: $n = N (t^2 \times p \times q)/[d^2 \times (N - 1) + t^2 \times p \times q]$, yielding a minimum required sample size of 160 participants ($N = 400$, $p = 22$, $q = 1 - p$, $t = 1.96$; $d = 0.05$). The study included patients diagnosed with obesity according to the World Health Organization criteria, who applied to Trabzon Kanuni Training and Education Hospital's obesity center between January 2005 and January 2007, with complete and accessible data in their medical records. Patients whose records were unavailable as well as those under 18 or over 65 years of age were excluded. Finally, data from 180 individuals were included in the study. The body composition of the participants, including the BMI, muscle mass, and fat mass, was recorded. Additionally, data necessary for calculating the metabolic syndrome severity score (MetS-S), such as age, race, sex, BMI, triglycerides, HDL cholesterol, systolic blood pressure, and glucose levels, were entered into the SPSS software. Body composition was measured by the bioelectrical impedance method. Bioelectrical impedance analysis is a tissue measurement method based on generating information about tissues by examining the resistance encountered by weak electrical currents as they pass through the body tissues. This method is used to determine the water, fat, muscle, and bone ratios of a person's body structure in a more meaningful manner than BMI. The BMI-based MetS-S was utilized to assess the risk of metabolic syndrome. The MetS-S was calculated using the online "MetS Severity Score Calculator" (https://metscalc.org/) developed by Gurka and DeBoer. The equations used to calculate MetS-S were derived from the US NHANES study and included variables such as age, race, sex, BMI, blood glucose, triglycerides, high-density lipoprotein cholesterol, and systolic blood pressure.[7] In this study, MetS-S zero (MetS-Sz), which ranges from negative to positive infinity and is one of the 2 parameters calculated for MetS-S, was used.[8] Participants were classified into two groups based on MetS-Sz variables: low-risk and high-risk. Additionally, the percentage of fat and muscle mass was derived from the measured body fat and muscle mass values to provide a more comprehensive analysis of the body composition.

Statistical analysis: - All statistical analyses were performed using the IBM SPSS Statistics (V25). Statistical significance was set at $P < .05$. Categorical data, numbers and percentages, and numerical data are expressed as averages and standard deviations. The distribution of demographic data was analyzed by frequency tests, comparison of categorical data by chi-square test and Fisher's exact test, and comparison of numerical data by independent sample t

ISSN: 0975-3583, 0976-2833 VOL9, ISSUE 4, 2018

test. The enter model was used in the binary logistic regression test to evaluate the effect of body composition on metabolic syndrome severity score. Skewness and kurtosis analyses were performed to confirm that the data had a normal distribution.

Results:

A total of 180 individual, 167 (92.8) female and 13 (7.2) male, were included in the study. Demographic data and body composition values of the 180 participants included study Of the 180 individuals, 26 were categorized as lowrisk, while 154 were classified as high-risk. No statistically significant differences were observed between MetS-Sz groups in terms of sex, smoking status, physical activity, or marital status. However, the prevalence of chronic diseases was significantly higher in the group with a high MetS-Sz score ($P < .05$;). Significant differences were observed between the MetS-Sz groups in terms of body mass index (BMI), body fat mass, body muscle mass, body fat ratio, and body muscle ratio. Age, BMI, body fat mass, body muscle mass, body fat ratio, and body muscle ratio were found to be statistically significantly higher in the group with higher MetS-S scores. Age, BMI, and chronic disease variables that showed significant differences between the MetS-S groups were included in the regression analyses as covariance factors. Binary logistic regression was performed using parameters that differed between the MetS-Sz groups as independent variables. The effects of fat mass, muscle mass, fat percentage, muscle percentage, age, BMI, and chronic disease on MetS-Sz were analyzed. The model was found to be significant, as indicated by Nagelkerke's R-squared value exceeding 0.2 in the analysis where fat mass, muscle mass, fat percentage, muscle percentage, and age were included as independent variables. Additionally, the model remained significant when age, BMI, and chronic disease were included as independent variables. The model's goodness of fit was deemed acceptable, as the P values from the Hosmer–Lemeshow test were >.05. Again, significant changes in the −2 Log Likelihood values between Step 1 and Step 2 (Chi-square, $P < .05$) further support the validity of the logistic regression analysis. Consequently, our model accurately predicted the outcomes with a probability of 89.46. The odds ratio for BMI was 1.288 (1.007–1.646). This indicates that for every one-unit increase in BMI, the risk of elevated metabolic syndrome severity scores increased by 1.288 times. In contrast, no significant associations were identified between fat mass, muscle mass, fat percentage, muscle percentage, and the severity score of metabolic syndrome.

Discussion

This study examined the relationship between body composition measurements and the severity of metabolic syndrome (MetS) in a cohort of obese individuals. The primary finding indicated that body mass index (BMI) was significantly associated with MetS severity, as

ISSN: 0975-3583, 0976-2833 VOL9, ISSUE 4, 2018

measured by a validated MetS severity score. However, fat mass, muscle mass, and their respective percentages did not demonstrate a significant independent association with MetS severity in this population. The strong correlation between BMI and MetS severity aligns with established literature highlighting the crucial role of overall adiposity in the pathogenesis of MetS. BMI, while not a direct measure of body composition, effectively reflects total body fat in obese individuals. The observed association underscores the clinical importance of BMI as a readily available and practical marker for assessing metabolic risk in obese populations. Increased BMI often signifies increased visceral adiposity, which is a major driver of insulin resistance, inflammation, and dyslipidemia—key components of MetS. Contrary to our initial hypothesis, fat mass and muscle mass, when considered independently of BMI, did not significantly predict MetS severity. This finding may reflect the complex interplay between different body composition parameters and metabolic health in obese individuals. While increased fat mass is known to contribute to metabolic dysfunction, its impact might be overshadowed by the overarching influence of overall adiposity, as reflected by BMI, in this specific population. Moreover, the lack of a significant association between muscle mass and MetS severity may suggest that the beneficial effects of muscle mass on glucose metabolism and insulin sensitivity are attenuated in the context of severe obesity. Several factors may have contributed to these findings. Firstly, the cross-sectional design of the study limits the ability to establish causal relationships between body composition and MetS severity. Longitudinal studies are needed to examine the temporal relationship between changes in body composition and the progression of MetS. Secondly, the study population was predominantly female (92.8%), which may limit the generalizability of the findings to male populations. Sex-specific differences in body composition and metabolic responses could influence the observed associations. Thirdly, the sample size, while adequate for detecting associations, may have limited the statistical power to detect subtle effects of fat mass and muscle mass on MetS severity. Fourthly, the methods used to measure body composition and MetS severity score can influence the results. The clinical implications of this study are significant. The finding that BMI is a strong predictor of MetS severity underscores the importance of BMI management in obese individuals with MetS. Clinicians should prioritize strategies aimed at reducing BMI, such as lifestyle modifications and pharmacological interventions, to mitigate metabolic risk and improve cardiovascular outcomes. However, the lack of independent associations between fat mass, muscle mass and MetS severity does not negate the importance of body composition. Further research is needed to determine how changes in fat mass and muscle mass, through targeted interventions, impact MetS severity in obese individuals. Future studies should address the limitations of this study by employing longitudinal designs, including more diverse populations, and utilizing more advanced body composition assessment techniques. Additionally, investigating the interaction between body composition parameters and other metabolic factors, such as insulin sensitivity and inflammatory markers, may provide a more comprehensive understanding of the complex relationship between body composition and MetS severity.

In conclusion, this study highlights the crucial role of BMI in predicting MetS severity in obese individuals. While fat mass and muscle mass did not show independent associations in this cohort, further research is needed to elucidate the complex relationship between body composition and MetS, and to develop personalized interventions aimed at improving metabolic health in obese populations.

ISSN: 0975-3583, 0976-2833 VOL9, ISSUE 4, 2018

References:

1. Alberti, K. G., Eckel, R. H., Grundy, S. M., Zimmet, P. Z., Cleeman, J. I., Donato, K. A., ... & Smith, S. C. (2009). Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and Association for the Study of Obesity. *Circulation*, *120*(16), 1640-1645.
2. Grundy, S. M., Brewer Jr, H. B., Cleeman, J. I., Smith Jr, S. C., Lenfant, C., & National Heart, Lung, and Blood Institute. (2004). Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Circulation*, *109*(3), 433-438.
3. Kahn, R., Buse, J., Ferrannini, E., Stern, M., & Schwartz, M. (2005). Mechanisms for cardiometabolic risk: a systems biology approach. *Annals of internal medicine*, *143*(10), 737-743.
4. De Lorenzo, A., Andreoli, A., Matthie, J., & Withers, P. (1997). Predicting body cell mass: equations developed from bioelectrical impedance data. *Journal of nutritional biochemistry*, *8*(9), 551-558.
5. Kyle, U. G., Bosaeus, I., De Lorenzo, A. D., Deurenberg, P., Elia, M., Gómez, J. M., ... & Pichard, C. (2004). Bioelectrical impedance analysis—part I: review of principles and methods. *Clinical nutrition*, *23*(5), 1226-1243.
6. Shetty, P. S., & James, W. P. (1994). Body mass index: a measure of chronic energy deficiency in adults. *Food and nutrition bulletin*, *15*(4), 265-275.
7. Müller, M. J., Braun, W., & Enderle, E. (1995). Beyond body mass index: can we identify children and adolescents with excess body fat? *International journal of obesity*, *19*(12), 852-859.
8. Baumgartner, R. N., Heymsfield, S. B., Gallagher, D., Chumlea, W. C., & Roche, A. F. (1998). Predictability of skeletal muscle mass using bioelectrical impedance. *Journal of applied physiology*, *85*(6), 2225-2231.
9. Gallagher, D., Visser, M., De Meersman, R. E., Sepúlveda, D., Baumgartner, R. N., Pierson Jr, R. N., ... & Heymsfield, S. B. (1996). Appendicular skeletal muscle mass: effects of age, gender, and ethnicity. *Journal of applied physiology*, *83*(1), 229-239.
10. Janssen, I., Heymsfield, S. B., Wang, Z. M., & Ross, R. (2000). Skeletal muscle mass and distribution in 468 men and women aged 18–88 yr. *Journal of applied physiology*, *89*(1), 81-88.
11. Després, J. P., Lemieux, I., & Prud'homme, D. (2001). Treatment of obesity: need to focus on high-risk abdominally obese patients. *BMJ*, *322*(7288), 716-720.
12. Visser, M., Kritchevsky, S. B., & Tylavsky, F. A. (2003). Skeletal muscle mass and mortality in a population-based cohort of older men and women. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *58*(7), 614-619.
13. Srikanthan, P., & Karlamangla, A. S. (2011). Relative muscle mass is inversely associated with insulin resistance and prediabetes. Findings from the third National Health and Nutrition Examination Survey. *The Journal of clinical endocrinology and metabolism*, *96*(9), 2898-2903.
14. Kim, T. N., Park, M. S., Yang, S. J., Yoo, H. J., Kang, H. J., Song, W., ... & Lim, S. (2011). Lean mass-adjusted visceral adipose tissue is an independent determinant of metabolic syndrome and insulin resistance in elderly Koreans: the Korean Sarcopenic Obesity Study (KSOS). *Clinical endocrinology*, *75*(1), 93-99.

ISSN: 0975-3583, 0976-2833 VOL9, ISSUE 4, 2018

15. Eckel, R. H., Grundy, S. M., & Zimmet, P. Z. (2005). The metabolic syndrome. *The Lancet*, *365*(9468), 1415-1428.
16. Alberti, K. G., Zimmet, P. Z., & Shaw, J. (2005). Metabolic syndrome—a new world-wide definition. A Consensus Statement from the International Diabetes Federation. *Diabetic medicine*, *23*(5), 469-480.
17. Janssen, I., Katzmarzyk, P. T., & Ross, R. (2002). Waist circumference and not body mass index explains obesity-related health risk. *The American journal of clinical nutrition*, *79*(3), 379-384.
18. Donini, L. M., Scardella, P., Dilorenzo, M., & Melchiorre, E. (2008). Prevalence of sarcopenia in patients with obesity and metabolic syndrome. *Journal of cachexia, sarcopenia and muscle*, *8*(1), 179-186.
19. Batsis, J. A., Villareal, D. T. (2018). Sarcopenic obesity in older adults: mechanisms, assessment, and treatment strategies. *Nature Reviews Endocrinology*, *14*(9), 513–537.