Journal of Obesity & Metabolic Syndrome 2025;34:85-87
https://doi.org/10.7570/jomes25032

Editorial

jomes
pISSN 2508-6235
eISSN 2508-7576

Toward Precision in Obesity Diagnosis: Progress, Pitfalls, and the Path Ahead

Myung Jin Kim[1], Chang Hee Jung[2,3,*]

[1]Department of Internal Medicine, Hallym University Sacred Heart Hospital, College of Medicine, Hallym University, Anyang; [2]Department of Internal Medicine, Asan Medical Center, University of Ulsan College of Medicine, Seoul; [3]Asan Diabetes Center, Asan Medical Center, Seoul, Korea

Received April 1, 2025
Reviewed April 3, 2025
Accepted April 3, 2025

*Corresponding author
Chang Hee Jung

https://orcid.org/0000-0003-4043-2396

Department of Internal Medicine, Asan Medical Center, University of Ulsan College of Medicine, 88 Olympic-ro 43-gil, Songpa-gu, Seoul 05505, Korea
Tel: +82-2-3010-1418
E-mail: chjung0204@gmail.com

Obesity is a chronic disease characterized by an abnormal or excessive fat accumulation that affects virtually every system in the body and poses major health risks.[1] Despite its clinical significance and global prevalence, there is still no universal consensus on its definition. The conventional body mass index (BMI)-based diagnosis of obesity has several limitations, as BMI does not reflect body fat distribution or provide information about individual health status. In addition, there are ongoing debates regarding the concept of obesity as a standalone disease entity or only as a risk factor for metabolic disease.

In a recent issue of *The Lancet Diabetes & Endocrinology*,[2] an international, multidisciplinary commission comprising 58 experts developed a novel and updated framework for diagnosing and managing obesity. This report aimed to redefine obesity by establishing objective diagnostic criteria and emphasizing the role of excess adiposity in organ dysfunction. While acknowledging BMI as a useful screening tool, the authors caution against using it as the sole diagnostic measure for obesity. Instead, they recommend confirming excess fat mass through direct body fat measurements whenever possible. If these measurements are unavailable, they suggest supplementing BMI with at least one additional anthropometric indicator, such as waist circumference, waist-to-hip ratio, or waist-to-height ratio, to improve accuracy in assessing adiposity. These measures, which all incorporate waist circumference, have been shown to be superior to BMI for predicting obesity-related health outcomes across various populations, providing sufficient evidence for their use as a universal diagnostic criterion.[3-5]

Despite the various anthropometric measures and body fat assessments proposed to address the limitations of BMI, clinically assessing excess adiposity remains challenging. Although the updated guidelines provided ethnicity-specific BMI and waist circumference thresholds, they could not cover all ethnic groups, and no standards were established for waist-to-hip ratio or waist-to-height ratio. Heymsfield et al.[6] advised against relying on race- and ethnicity-specific BMI cutoffs, as numerous factors beyond racial differences affect body composition and health outcomes. These factors, such as muscle mass, organ mass, and adipose tissue expandability,[7] exhibit significant inter-individual variation, further complicating the evaluation of excess adiposity. Ongoing efforts are needed to establish a validated methodology for assessing excess adiposity to enhance the accuracy and reliability of this updated framework of obesity.

Another key proposal in the commission's report is the distinction between clinical and preclinical obesity. Clinical obesity is defined as a condition of obesity that leads to organ dysfunction,

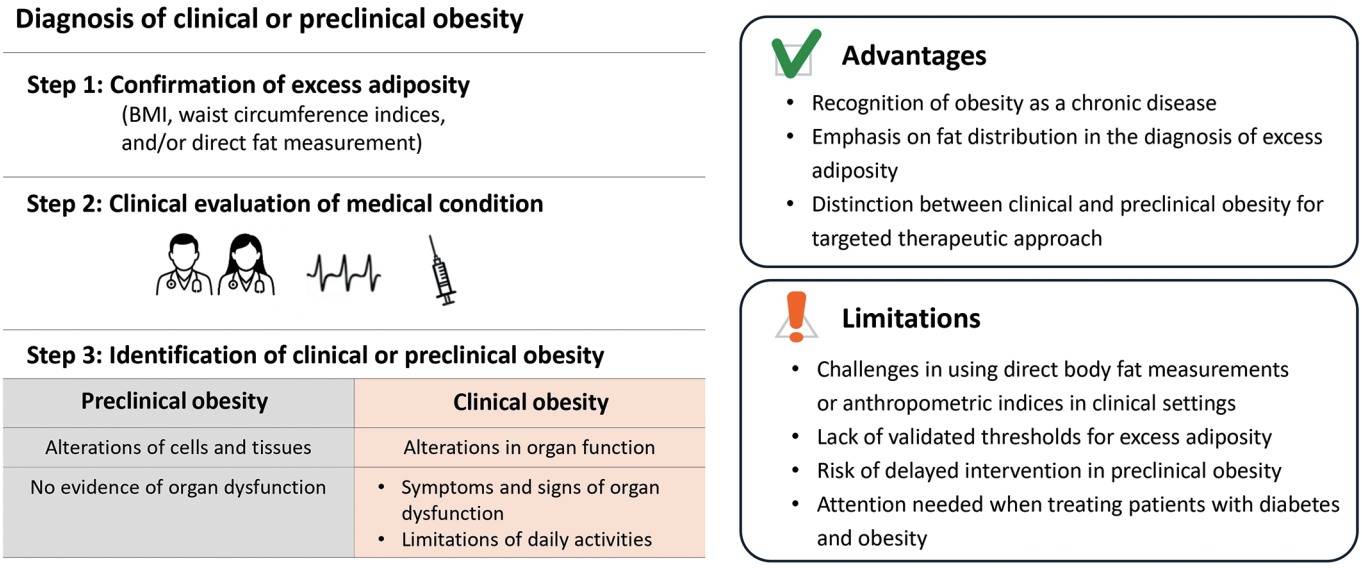

Figure 1. Stepwise diagnostic approach to updated obesity classification. Key advantages and limitations of the new framework are presented. BMI, body mass index.

measurable symptoms, or a significant impairment in daily functioning. As a distinct chronic illness itself, clinical obesity requires timely intervention, with treatment success assessed by improvements in clinical manifestations rather than changes in weight or BMI alone. Preclinical obesity on the other hand refers to excess adiposity without current organ dysfunction but with an increased risk of progressing to clinical obesity and other related diseases. This updated classification reflects the pathophysiological background of obesity, where early adipose tissue expansion may present initially as increased body size alone but will eventually disrupt systemic metabolism and lead to organ damage. For individuals with preclinical obesity, management should focus on overall risk reduction through health counseling, regular monitoring, and, when necessary, active interventions. The revised framework for clinical and preclinical obesity is summarized in Fig. 1.

The reframing of obesity into clinical and preclinical categories represents meaningful progress toward identifying obesity as a distinct disease entity. This approach defines obesity as a disease only when excess adiposity results in measurable dysfunction while excluding individuals with preserved health status from a formal disease label. The central aim of this distinction is to reduce the risk of overdiagnosis and reduce inefficient allocation of public health resources.[8] However, this model also poses concerns about potential underdiagnosis. Individuals with preclinical obesity may experience delays in diagnosis and early intervention—a limitation also raised by the European Association for the Study of Obesity. Labeling this stage as 'preclinical' may obscure the pathological nature of obesity, leading clinicians to postpone appropriate management until overt complications develop. Clinicians should recognize that the intent of this classification is not to delay management but to refine risk stratification and prioritize early interventions based on individual needs.

Careful consideration is required when interpreting the new diagnostic criteria for clinical obesity in cases where obesity coexists with hyperglycemia. In the current guidelines, hyperglycemia is included as part of the metabolic cluster criterion along with abnormal high-density lipoprotein cholesterol and triglyceride levels. The commission decided to exclude type 2 diabetes mellitus from the diagnostic criteria for clinical obesity due to its heterogeneous nature and diverse pathophysiological mechanisms. This decision aimed to avoid misclassifying diabetes subtypes that may not be directly linked to excess adiposity. However, as stated in the consensus statements, obesity is a major pathophysiological driver of diabetes mellitus, and weight management is one of the most important aspects of its treatment.[9,10] There is consistent evidence that weight loss improves glycemia and delays the progression of type 2 diabetes.[11] Therefore, the exclusion of type 2 diabetes mellitus from the definition of clinical obesity should not undermine the critical

role of weight management in diabetes care.

The commission's revised framework for obesity is expected to help clinicians provide personalized, timely, and appropriate care. Additionally, it has the potential to create a positive shift in societal stigma and perceptions of obesity among patients, healthcare professionals, and policymakers. Nonetheless, clinicians need to ensure that treatment is not delayed due to confusion over this new definition. Moreover, factors driving variability in obesity phenotypes remain unclear, particularly with regard to why some individuals with obesity maintain normal organ function over time while others develop early complications despite similar levels of excess adiposity. Further research is needed to better understand these differences and to improve risk stratification in obesity management.

CONFLICTS OF INTEREST

Chang Hee Jung is an associate editor of the journal. But he was not involved in the peer reviewer selection, evaluation, or decision process of this article. No other potential conflicts of interest relevant to this article were reported.

AUTHOR CONTRIBUTIONS

Drafting of the manuscript: MJK; critical revision of the manuscript: CHJ; administrative, technical, or material support: MJK and CHJ; study supervision: CHJ.

REFERENCES

1. Narayan KM, Boyle JP, Thompson TJ, Gregg EW, Williamson DF. Effect of BMI on lifetime risk for diabetes in the U.S. Diabetes Care 2007;30:1562-6.
2. Rubino F, Cummings DE, Eckel RH, Cohen RV, Wilding JPH, Brown WA, et al. Definition and diagnostic criteria of clinical obesity. Lancet Diabetes Endocrinol 2025;13:221-62.
3. Hsieh SD, Yoshinaga H, Muto T. Waist-to-height ratio, a simple and practical index for assessing central fat distribution and metabolic risk in Japanese men and women. Int J Obes Relat Metab Disord 2003;27:610-6.
4. Savva SC, Tornaritis M, Savva ME, Kourides Y, Panagi A, Silikiotou N, et al. Waist circumference and waist-to-height ratio are better predictors of cardiovascular disease risk factors in children than body mass index. Int J Obes Relat Metab Disord 2000;24:1453-8.
5. Hsieh SD, Yoshinaga H. Abdominal fat distribution and coronary heart disease risk factors in men-waist/height ratio as a simple and useful predictor. Int J Obes Relat Metab Disord 1995;19:585-9.
6. Heymsfield SB, Peterson CM, Thomas DM, Heo M, Schuna JM Jr. Why are there race/ethnic differences in adult body mass index-adiposity relationships? A quantitative critical review. Obes Rev 2016;17:262-75.
7. Virtue S, Vidal-Puig A. Adipose tissue expandability, lipotoxicity and the metabolic syndrome: an allostatic perspective. Biochim Biophys Acta 2010;1801:338-49.
8. Jastreboff AM, Kotz CM, Kahan S, Kelly AS, Heymsfield SB. Obesity as a disease: the Obesity Society 2018 Position Statement. Obesity (Silver Spring) 2019;27:7-9.
9. Klein S, Gastaldelli A, Yki-Järvinen H, Scherer PE. Why does obesity cause diabetes? Cell Metab 2022;34:11-20.
10. American Diabetes Association Professional Practice Committee. 8. Obesity and weight management for the prevention and treatment of type 2 diabetes: standards of care in diabetes-2025. Diabetes Care 2025;48(Supplement 1):S167-80.
11. Booth H, Khan O, Prevost T, Reddy M, Dregan A, Charlton J, et al. Incidence of type 2 diabetes after bariatric surgery: population-based matched cohort study. Lancet Diabetes Endocrinol 2014;2:963-8.