



Type 2 diabetes mellitus: Isn't it time to update the terminology?

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Abstract

There is a paradigm shift in the approach to the treatment of type 2 diabetes mellitus (T2DM) based on recent research. "Twin cycle hypothesis" and remission of diabetes proved that diabetes is a disease due to excess fat accumulation above the personal fat threshold. Hence, weight reduction and, whenever possible, achievement of remission has become therapeutic target for the management of T2DM. Increasing evidence suggests that T2DM is a part of the spectrum of diseases caused by excess fat accumulation. Hence, it indeed is the need of the hour to update the terminologies to indicate that T2DM is part of the disease spectrum of excess fat accumulation to improve awareness among healthcare professionals and the public to achieve overall holistic management of the disease spectrum. In this short article, we analyze these factors and propose a new terminology for adaptation worldwide.

Key Words: Diabetes mellitus; Adiposity; Obesity; Personal fat threshold; Personal organ-specific fat threshold; Adiposity induced metabolic dysfunction associated diseases; Metabolic dysfunction associated hyperglycemic disease

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Core Tip: Recent research has led to a paradigm shift in the treatment approach for type 2 diabetes mellitus (T2DM). The "twin cycle hypothesis" and evidence supporting diabetes remission have shown that the condition results from excess fat accumulation above the personal fat threshold. When excess fat deposition leads to organ dysfunction, we term it as adiposity-induced metabolic dysfunction associated disease. T2DM is a part of the spectrum of diseases caused by excess fat accumulation. Hence, it is the need of the hour to update the terminology and we propose a new terminology for T2DM. The proposed terminology is more reflective of the multifaceted nature of the disease, reshapes the management protocols and helps in earlier diagnosis and treatment.

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INTRODUCTION

Diabetes is a common lifestyle disorder characterized by hyperglycemia, which can lead to various complications, including cardiovascular and renal problems[1]. Its prevalence is increasing worldwide. Cardiovascular events and chronic kidney problems are the major causes of morbidity and mortality in people with type 2 diabetes mellitus (T2DM). In addition to that, diabetes mellitus (DM) can lead to retinopathy, neuropathy, fatty liver, sexual dysfunction, *etc.* In short, it is being recognized more as a disease that can affect head to foot, involving major organ systems in the body. The term DM is derived from the Greek word diabetes, which means “passing through” or “large discharge of urine”, and the Latin word mellitus, which indicates “sweet or sugar”[2]. The frequent urination (polyuria) and passage of sugar in the urine (glycosuria) are the classical symptoms of DM, which lead to its nomenclature. The terminology gives the impression that it is only a disease of high blood sugar, with the excretion of sugar in the urine and fails to reflect the recent advances in the understanding of disease pathology and treatment approach and hence it doesn't capture the full spectrum of metabolic disturbances present in T2DM.

WHY TO UPDATE THE TERMINOLOGY

T2DM is a common chronic metabolic condition characterized by predominant insulin resistance associated with hyperglycemia, leading to various acute and chronic complications, including microvascular and macrovascular complications[1]. For many years, treatment aimed to control blood sugar, which is described as glucocentric approach. The concept of glucocentric approach has evolved to cardio-renal risk reduction, overall risk reduction (multi-risk strategy), and organ protective approach over the years, indicating a paradigm shift in the management of T2DM[3,4]. The current aim of treatment of T2DM is not only control of blood glucose but also protection from associated complications and comorbidities of diabetes. However, the terminology gives a false impression that it is only a disease of high blood sugar, and its management is all about blood sugar control. The knowledge we acquired over the years is not reflected in the terminology, leading to a false impression not only among the public but also among health care providers that it is merely a disease of hyperglycemia (Table 1).

Increasing evidence shows that there is a multidirectional interaction between metabolic diseases such as T2DM, cardiovascular disease (CVD), and chronic kidney disease (CKD) leading to the development of cardio-renal-metabolic disease, through various mechanisms like hyperglycemia, insulin resistance, neurohormonal imbalance, oxidative stress, lipotoxicity, endothelial damage, mitochondrial malfunction, chronic inflammation, atherosclerosis, decreased cardiac pumping capacity and fluid retention[5-7]. The new terminology, cardio-renal-metabolic disease clearly indicates the connection between metabolic disease and cardio-renal complications. American Heart Association coined a new term cardiovascular-kidney-metabolic syndrome to emphasis these interconnections among CKD, metabolic risk factors such as obesity and T2DM, and CVD and is categorized into 5 stages from 0 to 4. The current terminology of DM fails to indicate that cardiovascular and renal complications are integral parts of T2DM[8].

T2DM was considered an inevitably progressive disease until recently. Trials like counterpoint, counterbalance, and the diabetes remission clinical trial and metabolic surgery clearly demonstrated that long-term remission of T2DM was achievable through weight reduction[9-13]. Attaining remission of diabetes has become a reality and is a well-described clinical goal in people with T2DM. Remission can be achieved by lifestyle modification and metabolic surgery. But if we closely analyse the mechanism of diabetes remission, it is clear that it is the remission (partial or complete) of excess fat accumulation (obesity) that leads to the improvement of various organ dysfunction. Similarly, in obese individuals, weight reduction is associated with improvement/remission of hypertension, dyslipidemia, metabolic dysfunction associated fatty liver diseases (MAFLD), and other obesity-associated conditions in addition to T2DM[14]. In short, it is the remission of excess fat accumulation that leads to improvement in organ dysfunction, which is variably called remission of diabetes, remission of hypertension, remission of MAFLD *etc.* The root cause of all these conditions and the mechanism of remission are the same, even though we name them differently. In addition to weight loss, bariatric surgery resulted in remission of DM (92%), hypertension (75%), dyslipidemia (76%), and obstructive sleep apnea (96%), *etc*[15]. Newer anti-obesity medications like semaglutide and tirzepatide (diabetes remission 66% to 81%) are also expected to provide similar reports[16-18]. The “twin cycle hypothesis” explains the pathophysiological mechanism of T2DM and according to this hypothesis, chronic calorie excess and the resultant chronic excess fat accumulation leads to hepatic and pancreatic dysfunction, leading to the development of T2DM[19,20]. In short, T2DM is a manifestation of chronic fat accumulation, and remission is possible when this excess fat is mobilized by weight reduction. Weight gain and chronic excess fat accumulation develop because of chronic calorie excess, which is denoted by the term “obesity” for several decades. However, there has been a lot of developments in our understanding and management of obesity in recent years. Rubino *et al*[21] define “obesity” as a “condition characterized by excess adiposity” and coined the new terminology “clinical obesity” to describe “chronic, systemic illness characterized by alterations in the function of tissues, organs, the entire individual, or a combination thereof, due to excess adiposity”. American Association of Clinical

Table 1 Why to update the terminology?

Why to update the terminology
The term diabetes mellitus gives the impression that it is only a disease of high blood sugar, with the excretion of sugar in the urine
Acute and chronic complications, including microvascular and macrovascular complications, are common in T2DM
The glucocentric approach is replaced by overall risk reduction (multi-risk strategy), and the organ protective approach indicates our knowledge about the pathophysiology of the illness
Multidirectional interaction between metabolic diseases such as T2DM, cardiovascular disease, and chronic kidney disease is well established
Cardiovascular and renal complications are integral parts of T2DM
Long-term remission of T2DM was achievable through weight reduction
Weight reduction is associated with improvement/remission of hypertension, dyslipidemia, metabolic dysfunction associated fatty liver disease, and other obesity-associated conditions in addition to T2DM
“Twin cycle hypothesis”: Chronic excess fat accumulation leads to hepatic and pancreatic dysfunction, leading to the development of T2DM
The strong pathophysiological link between diabetes and obesity is well described by the term ‘diabesity’
T2DM in people with normal body weight and even in lean individuals, develops when the “personal fat threshold” is crossed
“Thin fat obesity” indicates that even in people with a normal body mass index can have increased fat deposition in the visceral organs
Term for non-alcoholic fatty liver disease has been updated to metabolic dysfunction associated fatty liver disease/metabolic dysfunction associated steatotic liver diseases
The new terminology reflects the pathophysiological basis of T2DM from other types of diabetes

T2DM: Type 2 diabetes mellitus.

Endocrinologists and the American College of Endocrinology introduced the term adiposity-based chronic disease to replace obesity, which better describes the chronic nature of the disease, its pathophysiologic basis, and the need for a complication-centric approach in the management[22].

We define obesity as an anthropometric term to indicate excess fat deposition in the body based on various measurements, including body mass index, waist-hip ratio, waist-height ratio, or direct measurement of body fat. When excess fat deposition leads to organ dysfunction, we term it as “adiposity-induced metabolic dysfunction associated disease (AMAD)”. Previously, obesity is considered both a risk factor and a disease because of the lack of appropriate terminology to denote both. When excess fat accumulation reaches the stage of the disease, it affects multiple organ systems, producing various manifestations in involved organs, leading to its dysfunction. So, the risk factor has to be differentiated from the disease stage with proper terminology. AMAD is defined as a multisystem disorder with varying manifestations in different organ systems due to excessive fat accumulation above the personal organ-specific fat threshold and resulting pathophysiological changes, with the potential for remission with weight loss (Figure 1). In “remission of AMAD”, there is remission or improvement of all disease spectrum described under AMAD, depending upon the amount of excess fat loss. Rubino *et al*[21] explain this as “remission of clinical obesity”. Studies on remission of diabetes demonstrated that it is associated with loss of intrahepatic and intrapancreatic fat[23]. In simple words, obesity is a condition with body fat excess, and AMAD is a chronic disease due to obesity. The progression of obesity to AMAD is slow and continuous, hence demarcating the end of one condition and the beginning of other is difficult in clinical practice. In simple terms, obesity is a risk factor, whereas AMAD is a chronic disease counterpart of it. AMAD is a better terminology compared to adiposity-based chronic disease, as it incorporates metabolic dysfunction as the major pathophysiological mechanism and also indicates the reversible nature of the disease. In addition to that, it echoes with the established terminology for liver disease [MAFLD/metabolic dysfunction associated steatotic liver diseases (MASLD)], making all the diseases under an umbrella with comparable terminology. The term AMAD reflects the pathophysiological mechanism and reversibility better than the term “clinical obesity”.

The strong pathophysiological link between diabetes and obesity was understood decades before and is well described by the term ‘diabesity’ years back in the 1973 by Sim *et al*[24]. However, T2DM is described in people with normal body weight and even in lean individuals. As it is described, when the “personal fat threshold” is crossed person is prone to develop diabetes[25]. Personal fat threshold is defined as the level at which a person can no longer store subcutaneous fat in the adipose tissue or it is the amount of fat a person can store safely before it starts to spill over into organs like the liver and pancreas. In other words, it shows individual variability in adipose tissue function and fat storage capacity[26, 27]. At this stage, it gets deposited in the visceral organs, leading to various organ dysfunction, including T2DM[28,29]. When we consider AMAD, there is fat threshold for a particular person - “personal fat threshold” and that is different for different individuals. Similarly, there are different thresholds for different organs to manifest the problems of fat accumulation. We term it as “personal organ-specific fat threshold”. Personal organ-specific fat threshold is defined as the level at which a person can no longer store fat in a particular organ without affecting its function (Figure 2). So “personal organ-specific fat threshold” varies from individual to individual and hence it is called personal, it further varies from organ to organ in the same individual and hence it is called organ-specific. Each individual has a threshold for ectopic fat accumu-

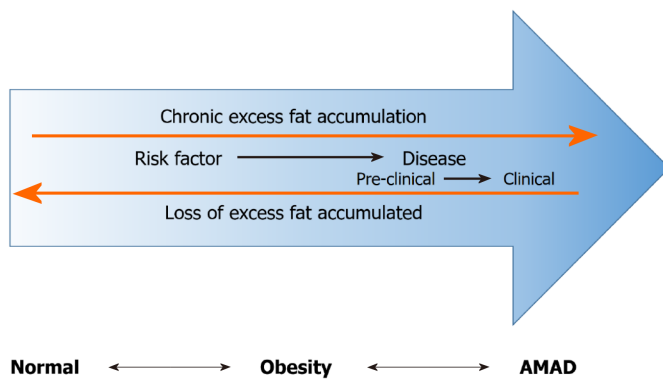


Figure 1 Diagram showing the progression of obesity, i.e., a condition with body fat excess to adiposity-induced metabolic dysfunction associated disease, a chronic disease due to obesity. AMAD: Adiposity-induced metabolic dysfunction associated disease.

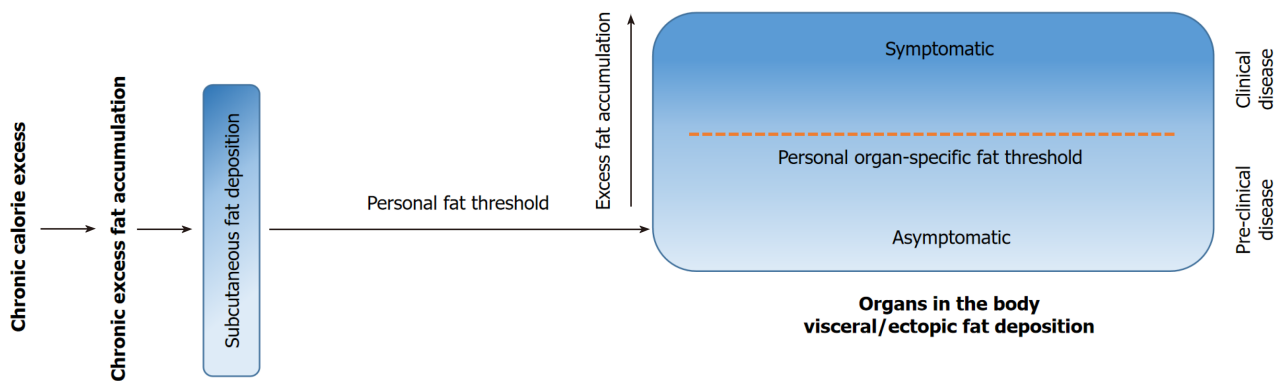


Figure 2 Diagram showing the importance of personal organ-specific fat threshold. When the “personal fat threshold” is crossed, fat gets accumulated in various organs. Initially, this visceral fat accumulation will be asymptomatic, but as more and more fat gets deposited, after a particular level (personal organ-specific fat threshold) it leads to organ dysfunction resulting in clinical disease.

lation in various organs, beyond which organ dysfunction emerges, and this is the personal organ-specific fat threshold. This concept builds on the personal fat threshold hypothesis by adding organ-level specificity. Variable distribution of fat in different organs and different “personal organ-specific fat thresholds” leads to various phenotypical manifestations of AMAD. Rubino *et al*[21] defined a term, pre-clinical obesity, in addition to obesity and clinical obesity, indicating a transition period in between, that is “characterized by excess adiposity and absence of major signs and symptoms of organ dysfunctions due to obesity”. Correlating with our proposed terminology, pre-clinical obesity evolves into clinical obesity when the personal organ-specific fat threshold is crossed. The concept of “thin fat obesity” clearly indicates that even in people with normal body mass index can have increased fat deposition in the visceral organs, and when it exceeds the personal organ-specific fat threshold, they develop organ dysfunction, including diabetes, depending upon the organ fat accumulation status[30]. Thin fat phenotype and normal weight or lean T2DM clearly demonstrate that intra-organ fat accumulation, irrespective of total body weight, is the driving force for various organ dysfunction, including diabetes. In short, T2DM is the disease of “intra-organ fat excess” irrespective of the total body weight.

The fat accumulation in various parts of the body is not uniform. It varies from person to person. Depending upon the predominant organ where excess fat accumulates and the personal organ-specific fat threshold of the individual, disease manifestation varies. Hence, people with chronic excess fat accumulation develop different diseases according to the distribution of fat, independent of total body weight or body fat percentage[31,32]. In addition to that, systemic effects of adiposity (for *e.g.*, chronic inflammation and hormonal changes) also affect remote areas also, away from the organs overloaded with fat. The pathophysiology of adiposity-induced disease spectrum involves one or more underlying mechanisms of fat accumulation (*e.g.*, ectopic fat deposition, insulin resistance, hyperinsulinemia, low-grade inflammation, mitochondrial damage, dysregulation of adipokines, and endothelial dysfunction) and not the absolute amount of fat deposition[21]. Additional pathophysiological mechanisms can also contribute to the development of the disease spectrum. The threshold for fat deposition and fat distribution is influenced by various factors like age, sex, genetics, hormonal status, *etc.*

Depending upon the stage of the organ dysfunction, it can be the preclinical or clinical stage of the disease (Figure 1). Clinical stages can be uncomplicated, complicated, or sequelae of the underlying dysfunction. Remission of organ dysfunction is clearly demonstrable in the early stage of the disease. However, control of organ dysfunction, improvement in the quality of life, and arrest of progression of organ dysfunction is possible at all the stages of the disease, indicating the importance of achieving weight reduction at all the stages of illness. Accumulation of fat in the liver and

subsequent progression of liver disease is a common problem in people with chronic calorie excess, which was initially called non-alcoholic fatty liver disease (NAFLD) and has been updated to MAFLD and to MASLD recently[33,34]. This represents a paradigm shift in the classification of hepatic steatosis. The term NAFLD carries social stigma and is defined by the exclusion of alcohol intake. The new terminology, MAFLD and MASLD, shifts from exclusion to inclusion-based diagnostic criteria, promoting early identification and integrated management of cardiometabolic and hepatic risks. The change in terminology is mainly because the new term reflects the underlying metabolic pathology of the liver disease. Appropriate nomenclature has an overall impact on the disease perception by patients and healthcare professionals. It improves patient awareness and helps to overcome management challenges. Key drawbacks include vague diagnostic criteria resulting in confusion, lack of global consensus, confusion in clinical practice, research discontinuity, risk of underdiagnosing lean individuals, fragmentation in clinical care, and ongoing disagreement among major liver societies [35]. The recent changes in terminology from NAFLD to MAFLD/MASLD is accepted by the academic world as it has implications in the disease outlook and management aspect. Similarly, the terminology update of T2DM is expected to be accepted wholeheartedly by the academic world.

Treatment with sodium-glucose cotransporter-2 inhibitors also causes increased urine output and glucose excretion in urine, again adding confusion to the terminology of DM, which indicates polyuria and glycosuria. Even though sodium-glucose cotransporter-2 inhibitors are used for the treatment of diabetes and are protective, it may give a false impression that it may deteriorate diabetes control by increasing urine output and glucose excretion in urine. Therefore, the nomenclature has to be changed to avoid undue emphasis on polyuria and glycosuria. A terminology that reflects the pathophysiological process of excess fat accumulation and indicating various disorders which is part of the spectrum of a single pathophysiological entity is the need of the hour, in order to emphasize a holistic approach for the treatment of the disease. It also emphasizes that weight reduction is the single most effective way to achieve remission in all these diseases.

PROPOSED TERMINOLOGY

With this background, we propose a new terminology and classification for chronic excess fat (adiposity) accumulation disorders. Chronic fat accumulation results in a spectrum of disease with varying manifestations depending upon the organ loaded with fat. We termed chronic excess fat accumulation as obesity, which has been in use for decades, and organ dysfunction due to chronic excess fat accumulation as AMAD. AMAD includes a spectrum of disease, with varying manifestations depending upon the predominant organ system affected, and shares overlapping features because of other organ and system involvement. Depending upon the major organ system involved, it is further classified as follows: Metabolic dysfunction associated endocrine, cardiovascular, gastrointestinal and hepatobiliary, neurological, genitourinary, circulatory, respiratory, musculoskeletal, immune, and integumentary (soft-tissue) and sexual disease/dysfunction (Table 2). It is common to see more than one metabolic dysfunction-associated disease in a single patient (Figure 3). The common pathophysiology and treatment approach helps to tackle all these abnormalities together rather than treating them as individual diseases. T2DM comes under metabolic dysfunction associated endocrine disease, and we termed it as metabolic dysfunction associated hyperglycemic disease (MAHD), and MAFLD/MASLD comes under metabolic dysfunction associated hepatobiliary disease. Metabolic dysfunction associated endocrine disease includes all the endocrine problems associated with excess fat accumulation, like hypothyroidism, sex hormone imbalance, and growth hormone deficiency in addition to hyperglycemic disease[36].

ADVANTAGES OF THE PROPOSED TERMINOLOGY

The “type 1” and “type 2” DM distinction is oversimplified and does not indicate the underlying pathophysiology. The pathophysiological mechanisms are entirely different in both: Type 1 DM is due to autoimmune pathology, whereas T2DM is a lifestyle disease with complex genetic and environmental factors playing a major role. The new terminology reflects the pathophysiological basis of T2DM from other types of diabetes. Here, we group all the currently considered different diseases with the same pathology into an umbrella of a single disease, which has got same pathophysiological mechanism and similar goal of therapy (like loss of excess fat deposition), helping multidisciplinary collaboration. It also shows that they are all interrelated conditions and not separate diseases, as we are considering now. A person with one condition can have other condition also because they share a common etiopathological mechanism. Weight reduction and improvement in one condition can have benefits in all other conditions and thus, we can manage the whole spectrum of disease holistically. The terminology also implies that a person with one disease can develop another and these diseases are not isolated compartments. It also emphasizes the need for looking into other related diseases in those with adiposity-induced metabolic dysfunction leading to early diagnosis of underlying silent diseases. The terminology of “T2DM” often implies a static condition. But as per the recent evidences, even long-term remission can be achieved[37]. So, it is better to convey a message of hope and empowerment, reinforcing the idea that early intervention can halt or even reverse the course of the disease. Our new proposed terminology serves this purpose. The new terminology denotes the long-term nature of the disease and the need for ongoing care and monitoring even after achieving remission. The central role for insulin resistance in the development of a cluster of cardio metabolic alterations and association of diabetes with cardiovascular and renal complications are also reflected in the new classification and terminology. It also reflects the interconnections between various metabolic disease and CVD, CKD, necessitating early screening for associated problems. Terms “T2DM” may often be linked to weight, lifestyle choices, or personal responsibility, which can lead to

Table 2 Classification of adiposity-induced metabolic dysfunction associated disease

Classification of adiposity-induced metabolic dysfunction associated disease
Metabolic dysfunction associated endocrine diseases
Metabolic dysfunction associated hyperglycemic diseases
Metabolic dysfunction associated cardiovascular diseases
Metabolic dysfunction associated gastrointestinal diseases
Metabolic dysfunction associated hepatobiliary diseases
Metabolic dysfunction associated fatty liver diseases/metabolic dysfunction associated steatotic liver diseases
Metabolic dysfunction associated neurological diseases
Metabolic dysfunction associated genitourinary diseases
Metabolic dysfunction associated circulatory diseases
Metabolic dysfunction associated respiratory diseases
Metabolic dysfunction associated musculoskeletal diseases
Metabolic dysfunction associated immune diseases
Metabolic dysfunction associated sexual diseases/dysfunction
Metabolic dysfunction associated integumentary diseases

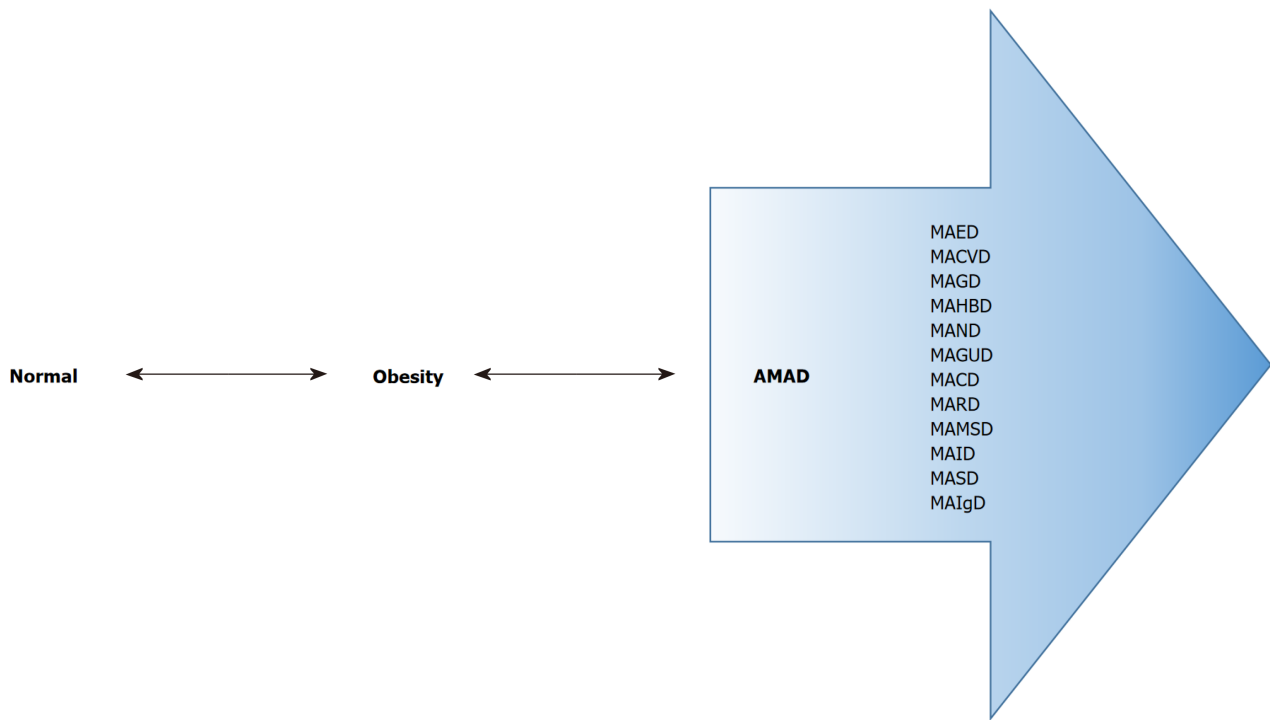


Figure 3 Diagram showing the spectrum of disease coming under adiposity-induced metabolic dysfunction associated disease. AMAD: Adiposity-induced metabolic dysfunction associated disease; MAED: Metabolic dysfunction associated endocrine diseases; MACVD: Metabolic dysfunction associated cardiovascular diseases; MAGD: Metabolic dysfunction associated gastrointestinal diseases; MAHBD: Metabolic dysfunction associated hepatobiliary diseases; MAND: Metabolic dysfunction associated neurological diseases; MAGUD: Metabolic dysfunction associated genitourinary diseases; MACD: Metabolic dysfunction associated circulatory diseases; MARD: Metabolic dysfunction associated respiratory diseases; MAMSD: Metabolic dysfunction associated musculoskeletal diseases; MAID: Metabolic dysfunction associated immune diseases; MASD: Metabolic dysfunction associated sexual diseases/dysfunction; MAIgD: Metabolic dysfunction associated integumentary disease.

Table 3 Advantages and disadvantages of replacing the term “type 2 diabetes mellitus” with “metabolic dysfunction associated hyperglycemic disease”

Advantages of updating to MAHD	Limitations/ arguments
The new terminology reflects the pathophysiological basis of the disease	Terms “associated disease” and “metabolic dysfunction” lack clear and standardized definitions
The new terminology (AMAD and MAHD) echoes with the existing terminology with similar pathophysiological mechanisms. <i>e.g.</i> , Metabolic dysfunction associated fatty liver diseases	
The new terminology is honest and transparent of the major pathophysiological mechanism, thereby providing clarity in care	The new terminology simply exchanges one form of stigma (sugar-related) for another (weight-related)
It groups all the currently considered different diseases with the same pathology into an umbrella of a single disease	
It helps in early diagnosis of other components of AMAD	
It helps multidisciplinary collaboration and holistic care	
It conveys a message of hope and empowerment, reinforcing the idea that early intervention can halt or even reverse the course of the disease	
It reflects the interconnections between various metabolic disease and CVD, CKD, necessitating early screening for associated problems	
It emphasizes the central role of lifestyle medicine in the promotion of overall health from multiple diseases due to excess fat accumulation	
It helps to differentiate other types of diabetes with different pathophysiological mechanisms into different categories	

MAHD: Metabolic dysfunction associated hyperglycemic disease; AMAD: Adiposity-induced metabolic dysfunction associated disease; CVD: Cardiovascular disease; CKD: Chronic kidney disease.

feelings of shame or blame. From the ethical point people can argue that the new term simply exchanges one form of stigma (sugar-related) for another (weight-related), but in fact the new terms show honesty and transparency of the major pathophysiological mechanism, thereby providing clarity in care. The new terminology is more neutral and compassionate and indicate the disease as a multifactorial condition. The new terminology also emphasis the central role of lifestyle medicine in the promotion of overall health from these multiple diseases. In short, the proposed terminology is more reflective of the multifaceted nature of the disease and reshape management protocols and helps in earlier diagnosis and treatment. The new term shows a paradigm shift emphasizing etiology over symptomatology, and our understanding about diabetes over the decades. This etiology-focused nomenclature helps to improve early diagnosis and prevention strategies. Renaming also helps to differentiate other types of diabetes with different pathophysiological mechanisms into different categories (Table 3).

CHALLENGES IN IMPLEMENTING NEW TERMINOLOGY

Uncoordinated change in terminology could lead to documentation errors, misbilling, misclassification in registries, and disruption of continuity of care. Textbooks, continuing medical education content, clinical guidelines (*e.g.*, American Diabetes Association, World Health Organization, European Association for the Study of Diabetes), medical curricula, international classification systems (*e.g.*, International Classification of Diseases, Diagnostic and Statistical Manual of Mental Disorders), PubMed searches, meta-analyses, and clinical trial registries need to be updated. Updating the electronic medical records and coding systems helps to avoid errors in documentation, insurance denials, or data loss in clinical studies. As with any changes in the field of medicine like updating in the terminology, updating the treatment guidelines, there should be extensive educational platforms and patient education activities to familiarize the new advances, which is comparatively easy in the current era of online and offline continuing medical educational activities for the healthcare professionals and various social media and print platforms for the public. Professional and institutional inertia, educational costs, cost for coding system revisions, and electronic health record updates are other challenges to be addressed.

Inclusion and incorporation of the new terminology into clinical guidelines is a challenge. Strategic pathways for integration into clinical guidelines include the educational phase, followed by the use of dual terminology in guidelines MAHD, formerly known as T2DM, and finally the standardization phase to fully replace the terminology “T2DM with MAHD” across protocols and decision trees. Because it brings all the adiposity-induced metabolic disease into an umbrella of a single disease entity, guidelines must integrate this terminology shift with allied areas like obesity management, cardiovascular risk, MAFLD, *etc.* to reflect the overlapping pathophysiological and therapeutic aspects and multi-disciplinary coordination of various specialties like endocrinology, gastroenterology, cardiology *etc.* is required to formulate and implement overall management protocols. Stakeholder engagement for guideline endorsement, adoption of terminology in the upcoming guideline revisions, publication of position statements and editorials supporting the

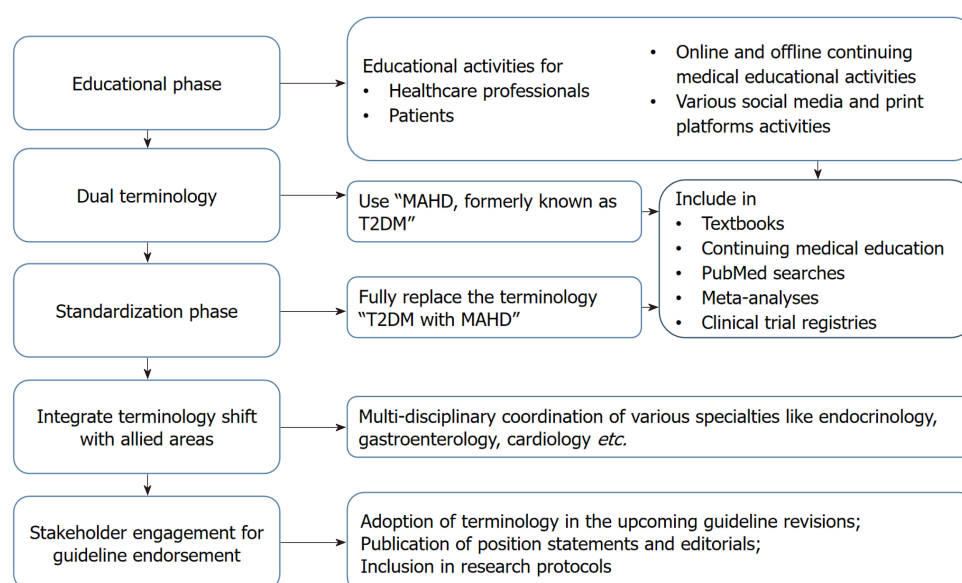


Figure 4 Flowchart illustrating the process for implementing the newly proposed terminology. MAHD: Metabolic dysfunction associated hyperglycemic disease; T2DM: Type 2 diabetes mellitus.

rationale and roadmap, encouraging inclusion in research protocols, and development of pilot guideline updates in academic medical institutions to assess feasibility, helps in smooth implementation (Figure 4).

CONCLUSION

T2DM is a part of the spectrum of disease conditions due to chronic fat accumulation, which can be called AMAD. The new terminology, MAHD clearly indicates the pathophysiological mechanism and inter relation with other components of AMAD. We expect that the academic world would wholeheartedly accept this new terminology, as they did with MAFLD, because of its clinical relevance.

FOOTNOTES

Author contributions: Raveendran AV designed the manuscript, collected the data, wrote and revised the manuscript. The author read and approved the final version of the manuscript to be published.

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