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Type 5 diabetes mellitus: Reemerging evidence for a neglected entity

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Abstract

Type 5 Diabetes Mellitus (T5DM), also known as Malnutrition-Related Diabetes Mellitus (MRDM), is a distinct and underrecognized form of diabetes associated with chronic undernutrition. Although considered rare, it is not uncommon in developing countries like Bangladesh, where malnutrition remains a widespread issue. Diabetes mellitus, a multifactorial endocrine disorder, is associated with both chronic undernutrition and obesity. Malnutrition often involves deficiencies in macro- and micronutrients in prenatal or postnatal life, unhealthy lifestyle behaviors, and low socioeconomic status. Numerous clinical studies have highlighted the connection between malnutrition and diabetes. Diagnostic challenges, overlapping symptoms, and an unclear causal relationship between malnutrition and diabetes hinder its classification and management. Current global diabetes frameworks inadequately address T5DM, contributing to misdiagnosis and delayed treatment. Malnutrition predominantly results in persistent insulin deficiency, glucose intolerance, and insulin resistance, thereby increasing the risk of developing diabetes. Further research is essential to refine the diagnostic criteria, elucidate the underlying pathophysiology, and assess the clinical and public health benefits of formally recognizing T5DM as a distinct subtype of diabetes mellitus. *[J Assoc Clin Endocrinol Diabetol Bangladesh,* July 2025;4(2)*: 72-79]*

Keywords: Type 5 DM, Malnutrition-related diabetes mellitus (MRDM), undernutrition, insulin resistance, glucose intolerance

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Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder marked by persistent hyperglycemia, leading to serious health complications if untreated. While type 1 DM (T1DM), type 2 DM (T2DM), gestational diabetes (GDM), and other specific forms are well documented, other less typical forms, such as type 5 diabetes (T5DM), also known as malnutrition-related diabetes mellitus (MRDM), are often overlooked. T5DM primarily affects individuals in tropical countries and developing regions of the world where long-term undernutrition is prevalent. Chronic malnutrition leads to structural and functional impairment of pancreatic β-cells, inducing oxidative stress and resulting in reduced insulin secretion and compromised insulin action.[1,2] As a result, T5DM presents with insulin dependence and hyperglycemia but, unlike T1DM, is resistant to ketosis due to impaired glucagon response.[3,4] Though first recognized in the mid-20th century, T5DM has received limited attention in global diabetes frameworks. One reason is diagnostic ambiguity—its symptoms often overlap with T1DM and T2DM, and commonly used indicators like low BMI are unreliable markers of malnutrition in many populations.[5] Moreover, whether malnutrition is a root cause or a consequence of diabetes remains uncertain. Some evidence suggests that individuals with T2DM who are underweight or of normal weight exhibit distinct glycemic and hormonal patterns compared to overweight individuals, pointing to the influence of nutritional status on disease behavior.[6]

Proper reclassification of DM and the inclusion of clinically significant dysglycemic conditions are essential to facilitate timely diagnosis and optimize

therapeutic interventions. Misclassification can delay the implementation of appropriate management strategies or lead to delayed insulin therapy. As this form of diabetes is closely linked with poverty, undernutrition, and regional disparities, addressing it requires a life-course approach that integrates nutrition with diabetes care. Further research is needed to refine diagnostic criteria, understand its pathophysiology, development of diagnostic biomarkers and criteria to distinguish T5DM from type 1 and type 2 diabetes., long term outcomes, cost-effective intervention, long-term complications and neurocognitive outcomes, public health research on poverty, food insecurity, and health system barriers, and assess whether recognizing T5DM as a distinct type leads to better outcomes.[7,8] Recognizing and addressing T5DM could have a major impact on diabetes control in resource-limited settings.

Materials and Methods

For this review, a comprehensive literature search was conducted across multiple electronic databases, including PubMed, Embase, Scopus, Google Scholar, and Current Contents, covering the period from January 1990 to June 2025. The search strategy involved combinations of relevant keywords such as “Type 5 diabetes mellitus,” “malnutrition-related diabetes mellitus (MRDM),” and “classification of diabetes mellitus,” along with their synonyms and Medical Subject Headings (MeSH) where applicable. Only manuscripts published in English were considered, including both full-text articles and abstracts. Studies were selected based on relevance to the topic, with emphasis on peer-reviewed literature. Conference proceedings and publications in languages other than English were not specifically searched or included in this review, which may represent a limitation in capturing some region-specific insights or unpublished data.

Overview of the global diabetes scenario

The global prevalence of DM has reached epidemic proportions, primarily driven by the surge in T2DM. As of 2024, an estimated 589 million adults worldwide are affected, with projections rising to 853 million by 2050.9 This alarming increase is attributed to multiple factors, including sedentary lifestyles, obesity, often referred to as "diabesity," as well as underlying nutritional deficiencies. Regional variations in diabetes prevalence are influenced by factors such as ethnicity, genetics, socioeconomic status, and lifestyle changes. In Bangladesh and similar settings, the burden is especially acute due to limited access to preventive care and nutrition. The growing disability-adjusted life years (DALYs) associated with diabetes highlight its rising societal and economic costs.[10] Accurate classification of diabetes is essential for guiding treatment and improving outcomes, particularly in children and adolescent populations.[7] Evidence-based management strategies-using clinical pathways and individualized diabetes care algorithms-further optimize disease control and reduce complications.[11,12] Thus, precise classification forms the foundation of effective diabetes care and public health response.

Classification of diabetes by an officially recognized authorized body

The classification of DM is set by recognized health organizations like the American Diabetes Association (ADA):

1. ***T1DM:*** This form is characterized by an autoimmune destruction of insulin-producing beta-cells in the pancreas, leading to an absolute deficiency of insulin. It includes both idiopathic and autoimmune forms.[13]
2. ***T2DM:*** This type occurs due to a progressive loss of beta-cell insulin secretion, frequently occurring on the background of insulin resistance. T2DM is the most common form and is often associated with obesity, physical inactivity, and diets high in fats and sugars.[14]
3. **GDM:** This type is identified when glucose intolerance is first recognized during pregnancy. It shares many characteristics with T2DM and is influenced by hormonal changes during pregnancy and other risk factors.[15]
4. ***Other specific types:*** This category includes a variety of less common diabetes forms, linked to specific genetic syndromes, drug or chemical exposure, infections, and other endocrinopathies. One example is maturity-onset diabetes of the young (MODY), which is caused by monogenic defects.[16]

Unofficial but clinically important forms of DM

Type 3 DM: Type 3 diabetes is used to describe the hypothesized association between insulin resistance and the pathogenesis of Alzheimer’s disease. Type 3 diabetes emphasizes brain insulin resistance. Insulin regulates not only blood glucose but also critical brain functions, including metabolism and cognition. Impaired insulin

signaling reduces glucose supply to the brain, potentially causing neuronal damage and tau protein buildup-hallmarks of Alzheimer's disease.[17]

Type 3c DM: Type 3c diabetes mellitus (T3cDM), also known as pancreatogenic diabetes, is a condition that arises from diseases affecting the exocrine pancreas. Often misdiagnosed due to its similarity to T1DM and T2DM and limited clinical awareness, T3cDM commonly results from chronic pancreatitis, pancreatic cancer, or other pancreatic conditions that cause inflammation and fibrosis, impairing both exocrine and endocrine pancreatic functions.[18]

Type 4 DM: Type 4 diabetes describes insulin resistance in older, lean adults, distinct from the typical obesity-linked T2DM. It is believed to result from age-related physiological and immune changes, including the accumulation of regulatory T cells (Tregs) in adipose tissue, which contributes to insulin resistance and disease progression in this population.[19]

T5DM: T5DM, also known as MRDM, is a distinct form of diabetes that develops in individuals with a history of chronic undernutrition, predominantly in low- and middle-income countries. It typically affects young, lean individuals who have experienced protein-energy malnutrition and micronutrient deficiency during prenatal, postnatal, and early childhood. The underlying mechanism involves impaired pancreatic beta-cell function and exocrine damage resulting from long-standing malnutrition, which occurs without evidence of autoimmunity.[20]

This also aligns with the recently proposed phenotypic classification of severe insulin-deficient diabetes (SIDD), which is characterized by early onset, low HOMA-β, lower BMI, poor glycemic control, and a high risk of diabetic retinopathy and neuropathy.[33] Another atypical form of diabetes reported in underweight Indian individuals, known as Low BMI Diabetes (LD), closely resembles MRDM. These individuals exhibit marked β-cell deficiency, characterized by significantly lower insulin secretion, compared to lean non-diabetic and T2DM groups. Unlike classical T2DM, they demonstrate enhanced peripheral insulin sensitivity, reduced hepatic glucose production, and minimal adiposity, highlighting a distinct metabolic phenotype likely linked to early-life undernutrition.[34] It is characterized by insulin dependence without ketosis and is often linked to pancreatic calcification and structural damage, especially in Fibrocalculous Pancreatic Diabetes (FCPD).[20] The World Health Organization (WHO) previously classified MRDM as a distinct diabetes category with two subtypes-FCPD and Protein Deficient Pancreatic Diabetes (PDPD).[21] Although excluded from the WHO's classification (1999), MRDM remains clinically significant in tropical regions where malnutrition and infectious diseases are prevalent.[22]

FCPD: primarily affects young adults under 30 from socioeconomically disadvantaged backgrounds with a history of malnutrition. It is characterized by insulin dependence without ketosis, clinical signs of malnutrition, pancreatic exocrine dysfunction, and radiological evidence of pancreatic calcification.[8]

PDPD: also known as Protein-Deficient Diabetes Mellitus (PDDM), occurs in similarly malnourished young individuals but lacks clinical and radiological signs of pancreatic damage. It is associated with low BMI, stunted growth, and relatively higher insulin resistance.[23]

Long-term complications and prognosis of T5DM

Long-term inadequate glycemic control, stemming from malnutrition-induced impairments in insulin secretion and sensitivity, can result in chronic damage to multiple organ systems-including the cardiovascular system, kidneys, eyes, and nervous system-substantially increasing the risk of both microvascular and macrovascular complications.[35] In individuals with early-onset diabetes coexisting with malnutrition, the consequences may be even more severe, including stunted linear growth, delayed pubertal development, impaired cognitive and psychomotor function, and an elevated risk of developing chronic non-communicable diseases in adulthood. Furthermore, emerging evidence suggests that diabetes, particularly when compounded by nutritional deficiencies, can adversely affect skeletal health by reducing bone mineral density and compromising bone microarchitecture, thereby heightening the risk of fragility fractures. From a prognostic perspective, patients with T5DM may be predisposed to more severe long-term outcomes, given the compounded effects of metabolic dysregulation and chronic undernutrition. However, there remains a pressing need for longitudinal studies and prospective cohort analyses to elucidate whether T5DM is associated with a distinct profile of complications and long-term prognostic implications compared to other forms of diabetes.[36]

Epidemiology of T5DM

T5DM is estimated to affect 20 to 30 million people worldwide, primarily in Asia and Africa. This

recognition highlights its impact on lean, malnourished youths in low- and middle-income countries and tropical countries. Historically underreported, cases have been documented in Jamaica, India, Sri Lanka, Bangladesh, Nigeria, Uganda, Ethiopia, Rwanda, and Korea. While some areas have seen declines due to improved nutrition, persistent malnutrition continues to drive new cases. The International Diabetes Federation (IDF) estimates that approximately 25 million people worldwide are affected.[9]

Probability of T5DM among Bangladeshi: Although comprehensive large-scale epidemiological studies are lacking, data from the Bangladesh Institute of Research and Rehabilitation in Diabetes, Endocrine and Metabolic Disorders (BIRDEM) in a previous registry indicate that approximately 30% of all diagnosed diabetes cases may be classified as MRDM.[38] This figure is particularly striking given that T5DM has historically been underrecognized and often misclassified as either type 1 or type 2 diabetes due to overlapping clinical features and the absence of distinct diagnostic criteria. The continued prevalence of childhood undernutrition, micronutrient deficiencies, low body mass index (BMI), and food insecurity in Bangladesh-conditions commonly associated with low and middle-income countries-suggests that T5DM remains a significant but overlooked public health issue. These findings underscore the urgent need for updated classification systems, improved diagnostic tools, and targeted epidemiological studies to assess the true burden of T5DM accurately. Recognizing T5DM as a distinct clinical entity is essential for guiding appropriate management strategies and informing public health interventions.

Historic background of T5DM

First described in Jamaica as "J-type diabetes" in 1955, T5DM occurs mainly in malnourished populations across tropical regions. Predominant in Eastern and Southern India, it has distinct genetic markers (HLA DR3/DQ2 in PDDM; DQ9 in FCPD), differentiating it from T1DM and T2DM. Unlike autoimmune diabetes, T5DM is linked to chronic undernutrition and socioeconomic factors. Diagnosis is challenging due to symptom overlap and the absence of autoimmune indicators.[23,24]

Naming and renaming: MRDM, initially described in 1955 as "J-type diabetes," was formally recognized by the WHO in 1985 as a distinct category of diabetes mellitus, alongside insulin-dependent and non-insulin-dependent forms. Within this classification, MRDM was further subdivided into two clinical entities: FCPD and PDDM (later referred to as Malnutrition-Modulated Diabetes). Due to its predominant occurrence in tropical regions, MRDM has also been colloquially referred to as "tropical diabetes." However, in 1999, the WHO removed MRDM from its classification system due to insufficient epidemiological and clinical evidence to support its distinction as a separate entity. Subsequently, in 2006, FCPD was reclassified under the broader category of diabetes secondary to exocrine pancreatic disorders, a classification reaffirmed in 2019. Over time, MRDM has also been described using various nomenclature, including Ketosis-Resistant Diabetes of the Young and Protein-Deficient Pancreatic Diabetes, underscoring the heterogeneity of its clinical presentations. In early 2025, a panel of experts convened in a national consensus meeting in India and proposed renaming MRDM as "Type 5 Diabetes," reflecting the growing recognition of its distinct pathophysiological and clinical features. At the recent IDF World Diabetes Congress 2025 held in Bangkok, Thailand, the IDF President announced the establishment of a dedicated working group tasked with developing formal diagnostic criteria and therapeutic guidelines for a distinct form of diabetes linked to chronic malnutrition. This condition, now formally recognized as "type 5 diabetes,".

Distinctive criteria from other forms of DM (Figure-1)

T1DM: Autoantibodies and ketoacidosis are characteristics of T1DM. In contrast, T5DM is characterized by low insulin levels, the absence of autoantibodies, and a rare occurrence of ketoacidosis.

T2DM: Characterized by insulin resistance and obesity or metabolic syndrome; absent in Type 5.

MODY: T5DM is a nutritionally acquired diabetes seen mainly in individuals from low socioeconomic backgrounds with chronic undernutrition, whereas MODY is a genetically inherited form affecting well-nourished individuals, often accompanied by family history and organ anomalies.

Type 3c DM (Pancreatogenic): Usually occurs after pancreatitis, pancreatic cancer, or surgery due to pancreatic damage.

Latent autoimmune diabetes in adults & other rare types: Latent autoimmune diabetes in adults (LADA) typically presents after the age of 35 and progresses

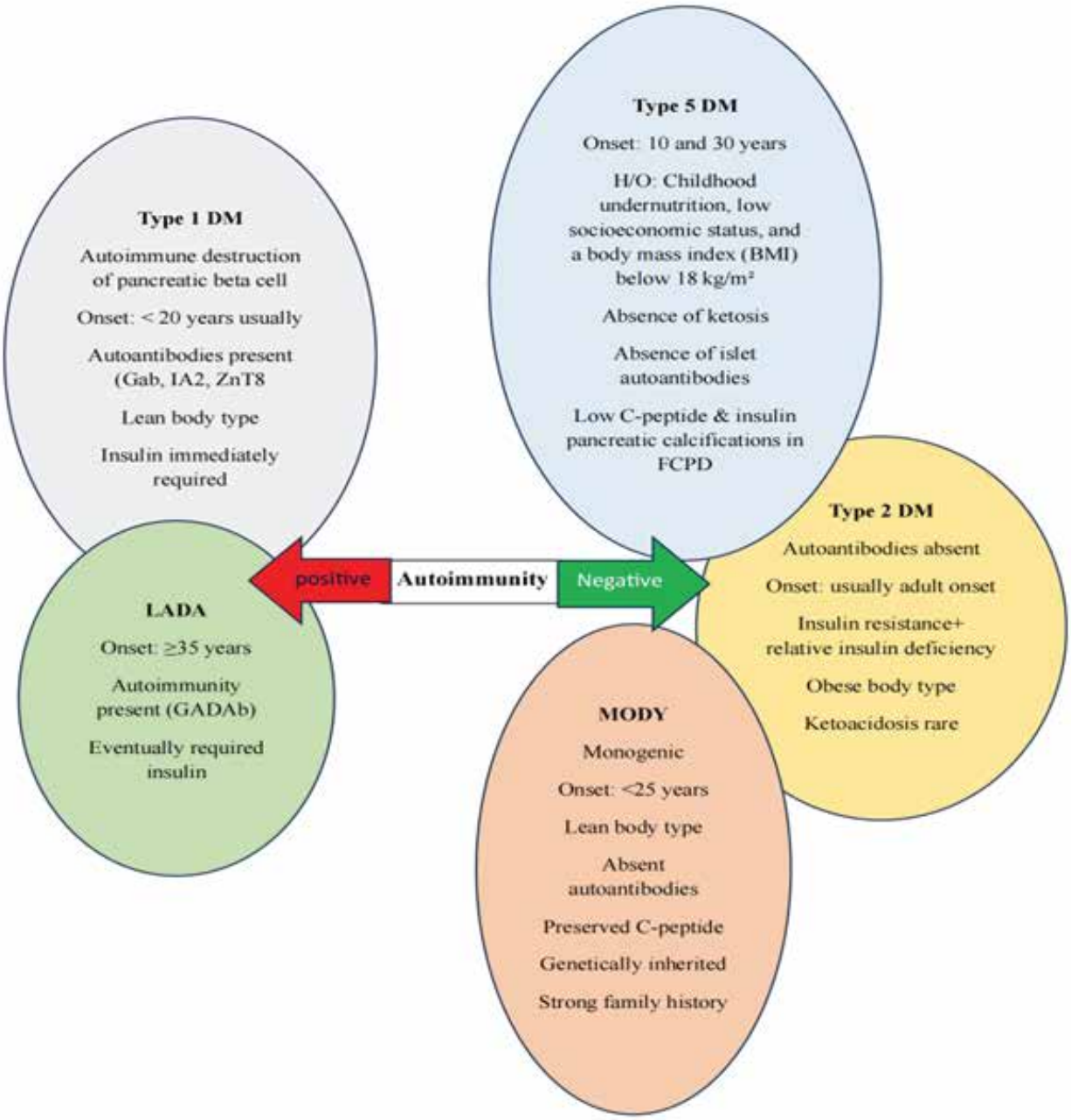

Figure-1: Venn diagram of the difference between different types of DM

more slowly than classical type 1 diabetes, but it remains characterized by the presence of pancreatic autoantibodies, unlike type 5 diabetes, which lacks autoimmune markers and is instead linked to chronic malnutrition.

Pathophysiology/etiology of T5DM

T5DM primarily affects populations with chronic undernutrition, initiating a unique pathophysiological process related to early-life nutritional deficits. The Developmental Origins of Health and Disease (DOHaD) hypothesis posits that parental nutrition and metabolic status during critical periods-such as conception and early embryogenesis-epigenetically program offspring susceptibility to metabolic disorders, including diabetes.[26] Epigenetic modifications, such as DNA methylation and histone modifications, alter gene expression, leading to long-term metabolic dysfunction. Maternal undernutrition, particularly low protein intake during pregnancy and lactation, impairs fetal growth and

disrupts hypothalamic regulation of insulin signaling and glucose metabolism, leading to β-cell dysfunction and an increased risk of diabetes later in life.[27] Micronutrient deficiencies, oxidative stress, and environmental toxins further damage pancreatic β-cells and exocrine function, impairing insulin secretion.[28] Malnutrition can contribute to the development of diabetes by impairing pancreatic development, leading to a reduced β-cell mass and impaired insulin secretion. Chronic protein and micronutrient deficiencies, particularly in early life, damage both exocrine and endocrine pancreatic function. Additionally, intrauterine malnutrition alters glucose metabolism through fetal programming.[37] Socioeconomic factors throughout life influence nutrition and health behaviors, linking early undernutrition to adult metabolic diseases.[29]

Signs and symptoms of T5DM

T5DM: It mainly affects lean males aged 10-30 from low socioeconomic backgrounds, presenting with classic diabetic symptoms and features like neuropathy, delayed puberty, parotid enlargement, steatorrhea, and pancreatic calcification.[8] Both FCPD and PDPD are considered recognized subtypes of T5DM.

FCPD: It is characterized by the triad of abdominal pain, pancreatic calculi, and diabetes, though all three may not always be present. Diagnosis relies on Mohan's criteria, which include evidence of chronic pancreatic disease (e.g., pancreatic calculi or morphological changes), exocrine dysfunction, and exclusion of other causes such as alcoholism or hepatobiliary disorders.

PDPD: PDPD occurs in young, severely malnourished individuals with growth retardation and micronutrient deficiencies. It presents as severe diabetes (fasting glucose >200 mg/dL), leanness (BMI <18 kg/m²), insulin dependence without ketosis, and high insulin needs (>1.5–2 U/kg/day). Unlike FCPD, PDPD lacks pancreatic calcification or ductal changes but exhibits exocrine dysfunction.[8]

Diagnosis

T5DM typically presents between the ages of 10 and 30 years and is often associated with a history of childhood undernutrition, low socioeconomic status, and a BMI below 18 kg/m². Clinical features include random blood glucose levels exceeding 200 mg/dL, the absence of ketosis even after insulin withdrawal, and a requirement for high doses of insulin (typically >1.5 U/kg/day) to achieve glycemic control. Although T5DM shares features with both type 1 and type 2 diabetes, it is distinguished by the absence of islet autoantibodies, a lower risk of ketoacidosis, a predominant male distribution, and a pathophysiology marked primarily by impaired insulin secretion rather than insulin resistance.[30]

Lab & Imaging reveals elevated fasting and postprandial glucose; HbA1c may be falsely interpreted due to anemia or malnutrition, low C-peptide & insulin confirming insulin deficiency, absent autoantibodies, X-ray/CT in FCPD shows pancreatic calcifications

For the diagnosis of MRDM, a defined set of criteria was suggested by Bajaj et.al. (Table-I).[8]

Table-I: Suggested criteria for diagnosis of MRDM[8]

SN	Clinical profile	Point score
1	Age of onset 10-30 years	1
2	BMI <19 Kg/m2	2
3	History of childhood malnutrition	1
4	Childhood stigmata of past or present malnutrition or deficiency state	2
5	Moderate to severe hyperglycemia lack of proneness to ketosis in absence of stress	3
6	Insulin required to achieved metabolic control but no dependence on insulin for prevention	2
7	Pancreatic calcification	3

An aggregated score of more than>10 is suggestive of MRDM

Management

Balanced nutrition and regular physical activity improve insulin sensitivity and preserve ß-cell function. Dietary recommendations include: limiting fat to under 30% of calories, consuming high-protein, energy-dense, and high-fiber foods, and reducing high-glycemic carbohydrates such as sugars and sweetened beverages.[31] Multivitamin supplementation is advised for common deficiencies in vitamins A, C, E, thiamine, B_6, B_{12}, and biotin, especially since metformin use can reduce B_{12} and folic acid absorption.[32] Glycemic control is critical; many patients require high insulin doses. FCPD patients typically need insulin or sulfonylureas, with surgery considered for severe pain. Steatorrhea is managed with pancreatic enzyme replacement and a low-fat diet. PDPD patients are unresponsive to sulfonylureas and depend on insulin, alongside a low-calorie, high-protein, low-fat diet.[8] Regular check-ups for glycemic control, HbA1c, nutritional status, and complication screening are necessary.

Conclusions

The IDF's recognition of T5DM marks a crucial milestone for countries like Bangladesh, where nutritional imbalances significantly impact chronic

diseases such as diabetes. This acknowledgment highlights the pressing need to incorporate comprehensive nutrition strategies into diabetes prevention and management. Evidence links malnutrition-particularly during pregnancy and early life-to impaired glucose tolerance and increased diabetes risk in offspring. Additionally, poor nutrition, unhealthy lifestyles, and socioeconomic challenges contribute to metabolic disorders in adulthood. This review underscores that malnutrition primarily causes persistent insulin deficiency, glucose intolerance, and insulin resistance, thereby elevating diabetes risk in the Bangladeshi population

Conflict of interest

The authors have no conflicts of interest to disclose.

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References

1. Poblete-Aro C, Russell-Guzmán J, Villegas-González B, Cofré-Bola-Dos C, Soto-Muñoz M, Parra P, et al. Exercise and oxidative stress in type 2 diabetes mellitus. Rev Med Chil 2018;146(3):362–72. DOI: 10.4067/s0034-98872018000300362.
2. Wei W, Tu M, Zhong W, Li S, Chen Z, Kang Q, et al. Malnutrition is associated with diabetic retinopathy in patients with type 2 diabetes. J Diabetes Res 2023;2023:1–13. DOI: 10.1155/2023/1613727.
3. Ubani BC, Umoren U, Nwafor NN, Dike FO. Malnutrition-related diabetes mellitus in a destitute male Nigerian. Ibom Med J 2024;17(3):598-601. DOI: 10.61386/imj.v17i3.526.
4. Tripathy BB, Samal KC. Protein deficient diabetes mellitus (PDDM) in India. Int J Diabetes Dev Ctries 1993;13:3-13.
5. Misra A, Joshi S, Mithal A. Malnutrition-related diabetes mellitus: Rushing toward "type 5" amid unresolved questions and limited evidence. Diabetes Metab Syndr 2025;19(5):103250. DOI: 10.1016/j.dsx.2025.103250.
6. Rajamanickam A, Munisankar S, Dolla CK, Thiruvengadam K, Babu S. Impact of malnutrition on systemic immune and metabolic profiles in type 2 diabetes. BMC Endocr Disord 2020;20:1-3. DOI: 10.1186/s12902-020-00649-7.
7. Siller AF, Tosur M, Relan S, Astudillo M, McKay S, Dabelea D, et al. Challenges in the diagnosis of diabetes type in pediatrics. Pediatr Diabetes 2020;21(7):1064-73. DOI: 10.1111/pedi.13070.
8. Balasaheb BJ, Bhirud P. Conceptual study of malnutrition related diabetes mellitus. Journal of Ayurveda and Integrated Medical Sciences. 2019;4(01):89-95. DOI: 10.21760/jaims.v4i01.578.
9. International Diabetes Federation. IDF Diabetes Atlas. 11th ed. Brussels, Belgium: International Diabetes Federation; 2025. Available from: https://diabetesatlas.org/ resources/idf-diabetes-atlas-2025.
10. Kotwas A, Karakiewicz B, Zabielska P, Wieder-Huszla S, Jurczak A. Epidemiological factors for type 2 diabetes mellitus: Evidence from the Global Burden of Disease. Arch Public Health 2021;79(1):110. DOI: 10.1186/s13690-021-00632-1.
11. Ellrodt G, Cook DJ, Lee J, Cho M, Hunt D, Weingarten S et al. Evidence-based disease management. JAMA 1997;278(20):1687-92. DOI:10.1001/jama.1997.03550200063033.
12. Lovic D, Piperidou A, Zografou I, Grassos H, Pittaras A, Manolis A et al. The growing epidemic of diabetes mellitus. Curr Vasc Pharmacol 2020;18(2):104-9. DOI: 10.2174/1570161117666190405165911.
13. Antar SA, Ashour NA, Sharaky M, Khattab M, Ashour NA, Zaid RT, et al. Diabetes mellitus: Classification, mediators, and complications; A gate to identify potential targets for the development of new effective treatments. Biomed Pharmacother 2023;168:115734. DOI: 10.1016/j.biopha.2023.115734.
14. Patil SR, Chavan AB, Patel AM, Chavan PD, Bhopale JV. A review on diabetes mellitus its types, pathophysiology, epidemiology and its global burden. J Res Appl Sci Biotechnol 2023;2(4):73-9.
15. Elliott A, Walters RK, Pirinen M, Kurki M, Junna N, Goldstein JI, et al. Distinct and shared genetic architectures of gestational diabetes mellitus and type 2 diabetes. Nat Genet 2024;56(3):377-82. DOI: 10.1038/s41588-023-01607-4.
16. Li LM, Jiang BG, Sun LL. HNF1A: from monogenic diabetes to type 2 diabetes and gestational diabetes mellitus. Front Endocrinol 2022;13:829565. DOI: 10.3389/fendo.2022.829565.
17. Nguyen TT, Ta QT, Nguyen TK, Nguyen TT, Van Giau V. Type 3 diabetes and its role implications in Alzheimer's disease. Int J Mol Sci 2020;21(9):3165. DOI: 10.3390/ijms21093165.
18. Bhattamisra SK, Siang TC, Rong CY, Annan NC, Sean EH, Xi LW, et al. Type-3c diabetes mellitus, diabetes of exocrine pancreas-an update. Curr Diabetes Rev 2019;15(5):382-94. DOI: 10.2174/1573399815666190115145702.
19. Bapat SP, Myoung Suh J, Fang S, Liu S, Zhang Y, Cheng A, et al. Depletion of fat-resident Treg cells prevents age-associated insulin resistance. Nature 2015;528(7580):137-41. DOI: 10.1038/nature16151.
20. Unnikrishnan R, Mohan V. Fibrocalculous pancreatic diabetes (FCPD). Acta diabetol 2015;52(1):1-9. DOI: 10.1007/s00592-014-0685-9.
21. WHO Study Group on Diabetes Mellitus. Diabetes mellitus: Report of a WHO study group. World Health Organization; 1985.
22. World Health Organization. Definition, diagnosis and classification of diabetes mellitus and its complications: report of a WHO Consultation. Geneva: WHO; 1999.
23. Rao RH, Yajnik C. Malnutrition and diabetes in the tropics. Diabetes Care 1996;19(9):1014-7. DOI: 10.2337/diacare.19.9.1014.
24. Sanjeevi CB, Kanungo A, Samal KC. Immunogenetic studies on malnutrition-modulated Diabetes Mellitus. Ann N Y Acad Sci 2002;958(1):144-7. DOI: 10.1111/j.1749-6632.2002.tb02957.x.
25. Abdulkadir J, Mengesha B, Gabriel ZW, Keen H, Worku Y, Gebre P, et al. The clinical and hormonal (C-peptide and glucagon) profile and liability to ketoacidosis during nutritional rehabilitation in Ethiopian patients with malnutrition-related diabetes mellitus. Diabetologia 1990; 33:222-7.

DOI: 10.1007/BF00404800.
26. Fleming TP, Watkins AJ, Velazquez MA, Mathers JC, Prentice AM, Stephenson J, et al. Origins of lifetime health around the time of conception: causes and consequences. Lancet 2018;391(10132):1842-52. DOI: 10.1016/s0140-6736(18)30312-x.
27. Balcha SA, Phillips DI, Trimble ER. Type 1 diabetes in a resource-poor setting: malnutrition-related, malnutrition modified, or just diabetes?. Curr Diab Rep 2018;18:1-6. DOI: 10.1007/s11892-018-1003-7.
28. Philip G, Thomas PA. Surgery of chronic pancreatitis-a Kerala experience. Proceedings Inaugural Session Indian society of Pancreatology, Medical College, Trivandrum 6th November. 1985.
29. van Abeelen AF, Elias SG, Bossuyt PM, Grobbee DE, van der Schouw YT, Roseboom TJ, et al. Famine exposure in the young and the risk of type 2 diabetes in adulthood. Diabetes 2012;61(9):2255-60. DOI: 10.2337/db11-1559.
30. George AM, Jacob AG, Fogelfeld L. Lean diabetes mellitus: an emerging entity in the era of obesity. World J Diabetes 2015;6(4):613. DOI: 10.4239/wjd.v6.i4.613.
31. Myers EF, Trostler N, Varsha V, Voet H. Insights from the Diabetes in India Nutrition Guidelines Study: adopting innovations using a knowledge transfer model. Top Clin Nutr 2017;32(1):69-86. DOI: 10.1097/TIN.0000000000000089.
32. Raizada N, Jyotsna VP, Sreenivas V, Tandon N. Serum vitamin B12 levels in type 2 diabetes patients on metformin compared to those never on metformin: a cross-sectional study. Indian J Endocrinol Metab 2017;21(3):424-8. DOI: 10.4103/ijem.IJEM_529_16.
33. Deutsch AJ, Ahlqvist E, Udler MS. Phenotypic and genetic classification of diabetes. Diabetologia 2022;65(11):1758-69. DOI: 10.1007/s00125-022-05769-4.
34. Lontchi-Yimagou E, Dasgupta R, Anoop S, Kehlenbrink S, Koppaka S, Goyal A, et al. An atypical form of diabetes among individuals with low BMI. Diabetes Care 2022;45(6):1428-37. DOI: 10.2337/dc21-1957.
35. Li J, Fang H, Wang Z, Ma Y, Nan X. Therapeutic potential of fingolimod in diabetes mellitus and its chronic complications. Diabetes Metab Syndr Obes 2024;17:507–16. DOI: 10.2147/dmso.s385016.
36. Xie YC, Jing XB, Chen X, Chen LZ, Zhang SH, Cai XB et al. Fecal microbiota transplantation treatment for type 1 diabetes mellitus with malnutrition: a case report. Ther Adv Chronic Dis 2022;13:204062232211174. DOI: 10.1177/20406223221117449.
37. Blondeau B, Avril I, Duchene B, Breant B. Endocrine pancreas development is altered in foetuses from rats previously showing intra-uterine growth retardation in response to malnutrition. Diabetologia 2002;45(3):394-401. DOI: 10.1007/s00125-001-0767-4.
38. Rahman T, Rashid HU, Mahtab H, Kabir Y. P-140 Epidemiological and histopathological study of malnutrition-related diabetes mellitus in Bangladeshi subjects. Diabetes Res Clin Pract 2008;79:S106. DOI: 10.1016/S0168-8227(08)70910-0.