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DIABETES MELLITUS: A COMPREHENSIVE OVERVIEW OF CLASSIFICATION, PATHOPHYSIOLOGY, CAUSES, DIAGNOSIS, SYMPTOMS, AND TREATMENT

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ABSTRACT

Diabetes mellitus is a widespread chronic metabolic disorder that includes several types, such as Type 1 (T1DM), Type 2 (T2DM), and gestational diabetes, all marked by elevated blood glucose levels. Type 1 diabetes is an autoimmune disease driven by genetic factors that lead to the destruction of pancreatic beta cells, necessitating insulin therapy. Type 2 diabetes is linked to insulin resistance and metabolic syndrome, influenced by factors like genetics, obesity, lack of physical activity, and ethnicity. Gestational Diabetes Mellitus (GDM) occurs during pregnancy and raises the risk of obesity and Type 2 diabetes in the child. Diagnosis is based on tests such as fasting blood glucose, oral glucose tolerance, HbA1c levels, and specific antibody tests. Long-term complications include atherosclerosis, retinopathy, neuropathy, nephropathy, and cell damage due to osmotic stress, emphasizing the need for proper blood sugar control. Treatment strategies differ: T1DM requires insulin, while T2DM management may involve lifestyle changes, medications, and sometimes insulin. Understanding the nature of diabetes is crucial for effective treatment and improving patients' quality of life.

KEYWORDS: Diabetes Mellitus, Classification, Pathophysiology, Causes, Diagnosis, Treatment, symptoms.

INTRODUCTION

Diabetes mellitus is a disorder of macromolecule metabolism, where the body has impaired endocrine function, leading to difficulty in maintaining proper blood glucose levels.[1] Diabetes can be a chronic condition that develops when the pancreas can no longer produce insulin or when the body becomes unable to effectively use the insulin it produces. Insulin is a hormone made by the pancreas that acts like a key, allowing glucose from the food we eat to move from the bloodstream into the body's cells, where it is either stored or used for energy.[2] In diabetes, the body either doesn't produce enough insulin or can't effectively use the insulin it makes. If high blood sugar levels from diabetes are left untreated, they can damage the nerves, eyes, kidneys, and other organs.[3] Diabetes is one of the most widespread metabolic disorders, affecting over 200 million people worldwide. In this condition, the body either doesn't produce enough insulin or cannot effectively use the insulin it does produce. If left unmanaged, elevated blood sugar levels can harm various organs, including the nerves, eyes, and kidneys. This high global prevalence emphasizes the importance of understanding the disease's causes and the factors that trigger its onset.

Various infectious processes contribute to the development of diabetes, ranging from immune system attacks that destroy the pancreas's beta cells leading to insulin deficiency to abnormalities that result in insulin resistance. These metabolic disturbances contribute to the hallmark symptoms of diabetes, as well as its microvascular and macrovascular complications and increased risk of cardiovascular disease.[4] The pancreas produces insulin, but the insulin that is produced does not function as it should.[5] This condition is known as insulin resistance. To better understand diabetes, it's important to learn how the body turns food into energy a process known as metabolism. The body is made up of countless cells, and for these cells to generate energy, they require nutrients in a basic form.

After you eat, much of your food is broken down into a simple sugar called glucose, which provides the energy your body needs to perform daily activities.[6] If you have very little insulin or your body is resistant to it, excess sugar remains in your bloodstream. People with diabetes have higher-than-normal blood glucose levels.[7] The World Health Organization (WHO) has ranked Pakistan seventh in terms of diabetes prevalence.[8] Recent data shows that over 4.7 million people are affected by diabetes.[9] Common symptoms of diabetes include increased thirst, frequent urination, sweating, blurred vision, rapid weight loss, fatigue, and slow-healing wounds. Most individuals with diabetes experience excessive thirst,

increased hunger, and kidney-related issues.[10] Gestational diabetes occurs only during pregnancy. Hormonal changes during this time can interfere with insulin production, leading to reduced insulin effectiveness. As a result, blood glucose levels rise, which can negatively affect the developing fetus.[11] Prediabetes was found in 7% of individuals. It was also observed that conditions like high blood pressure, high cholesterol, abnormal blood sugar levels, and diabetes were more common among females.[12] In comparison, the WHO report estimates a diabetes prevalence of 8.8% in South America.[13] According to the International Diabetes Federation (IDF) 2016 report, the prevalence of diabetes was estimated at 11.1%, with a significant risk of rising diabetes-related complications partly due to increasing obesity rates. The growing number of people with diabetes and the complexity of managing their care could place a heavy burden on existing healthcare systems. There is a pressing need to apply new research on both personal and societal challenges in diabetes management and to integrate life-saving discoveries into wider clinical practice.[14]

Classification of Diabetes Mellitus

Type 1 Diabetes Mellitus (T1DM)

1. Autoimmune Beta-cell Destruction

This autoimmune response is usually triggered by a combination of genetic predisposition and environmental factors, causing the immune system to target specific antigens present on beta cells (Atkinson et al., 2014).[15]

2. Insulin Deficiency

As the destruction of beta cells continues, insulin production drops to minimal or completely absent levels. Insulin is an essential hormone for regulating glucose metabolism, playing a crucial role in enabling glucose to enter cells, especially in muscle and fat tissues (American Diabetes Association [ADA], 2024).[16]

3. Impaired Glucose Uptake

In the absence of insulin, glucose is unable to enter the cells, causing it to build up in the bloodstream. This leads to sustained high blood sugar levels, or hyperglycemia, which can lead to serious complications such as diabetic ketoacidosis if not properly controlled (Daneman, 2006).[17]

4. Hyperglycemia and Metabolic Dysregulation

When cells are unable to effectively use glucose, blood sugar levels rise, resulting in metabolic imbalances. Over time, this can lead to chronic complications like kidney disease (nephropathy), eye damage (retinopathy), and nerve damage (neuropathy) (ADA, 2024).

Type 2 Diabetes Mellitus (T2DM)

1. Insulin Resistance

In Type 2 diabetes mellitus, the main issue is insulin resistance, meaning that peripheral tissues such as muscle, liver, and fat do not respond properly to insulin in the bloodstream. This condition is commonly linked to factors like obesity, lack of physical activity, and genetic predisposition (Kahn, 2001).[18]

2. Compensatory Hyperinsulinemia

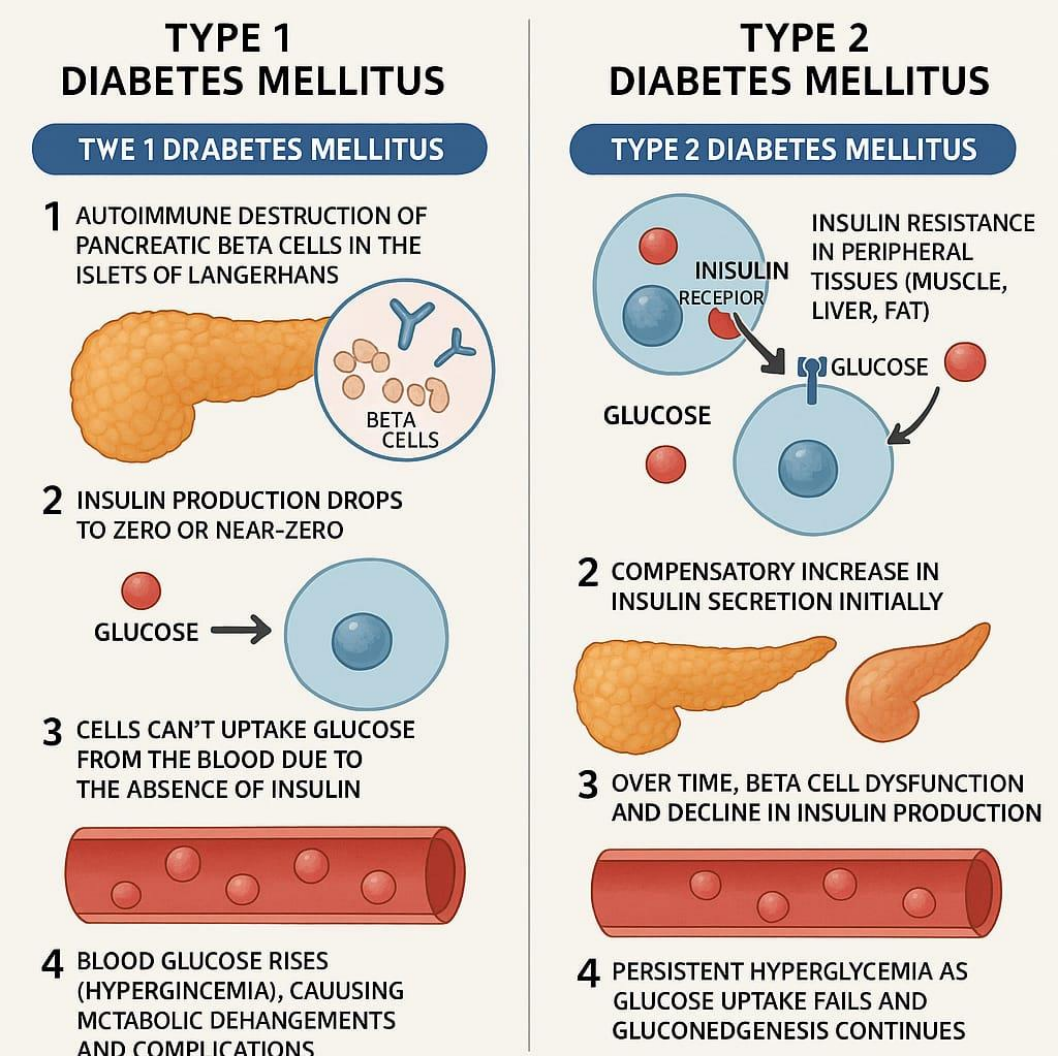
At first, the pancreas responds by producing more insulin to keep blood glucose levels within the normal range. However, this increased insulin output cannot be maintained in the long term (Defronzo, 2004).[19]

3. Beta-Cell Dysfunction

Prolonged insulin demand puts stress on the beta cells, leading to their fatigue and impaired function. Over time, this results in reduced insulin production, further worsening high blood sugar levels (Prentki & Nolan, 2006).[20]

4. Chronic Hyperglycemia

Ongoing insulin resistance, along with reduced insulin production, results in sustained high blood sugar levels. Moreover, the liver continues to produce glucose through gluconeogenesis, even when blood glucose is already elevated, further aggravating the condition (ADA, 2024).



Pathophysiology of Diabetes Mellitus

Diabetes Mellitus (DM) is a long-term metabolic disorder characterized by consistently high blood sugar levels, resulting from impaired insulin secretion, action, or a combination of both. The underlying pathophysiology can be outlined as follows:

1. Pancreatic Dysfunction

Under normal conditions, the pancreas specifically the beta cells within the islets of Langerhans produces insulin, a vital hormone responsible for regulating blood glucose by promoting its uptake into cells.

2. Decreased Insulin Secretion

In Type 2 Diabetes, insulin levels may initially be present, but due to insulin resistance, the effectiveness of insulin is reduced, eventually leading to a decline in its secretion over time.

3. Decreased Glucose Entry into Cells

Insufficient insulin levels or impaired insulin function prevents cells especially in muscle and fat tissue from efficiently absorbing glucose.

4. Hyperglycemia

When glucose uptake by cells is reduced, it causes elevated levels of glucose in the blood, known as hyperglycemia.

5. Chronic Complications

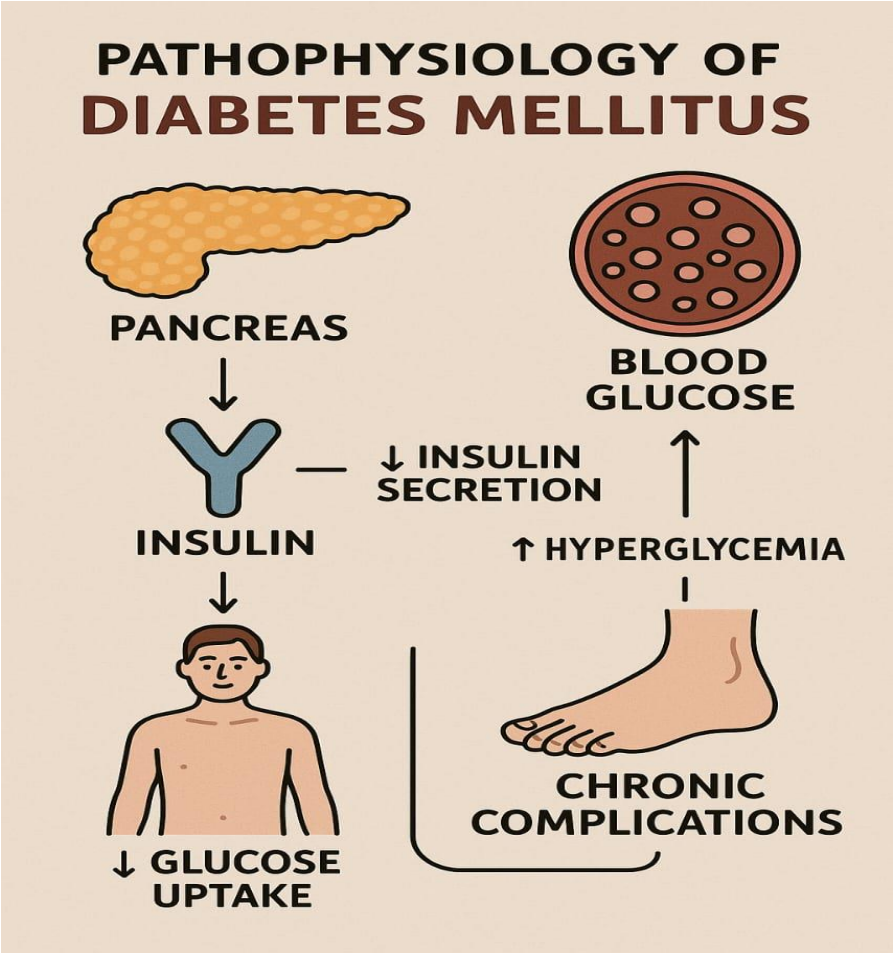
Neuropathy (nerve damage)

Nephropathy (kidney damage)

Retinopathy (eye damage)

Peripheral vascular disease (reduced blood flow, especially to the limbs)

These complications significantly impact the quality of life and increase both the illness and death rates among individuals with diabetes.[21-23]



Causes

Disruptions or abnormalities in B-cell glucose receptors may cause inappropriate responses to elevated glucose levels, or there may be relative B-cell insufficiency. In either case, reduced insulin secretion occurs, which can eventually contribute to β-cell failure.[26] Another theory suggests that microvascular disease leads to cerebral hypoxia, while hyperglycemia directly impairs neuronal metabolism.[27]

In peripheral tissues, decreased insulin sensitivity arises from "down-regulation" and reduced numbers of insulin receptors. Many individuals who are hyperinsulinemic and hypersensitive still maintain normal blood glucose levels, but present with dyslipidemia, hyperuricemia, and central obesity. This indicates relative insulin resistance, particularly in the liver, skeletal muscle, and adipose tissue. Furthermore, hyperinsulinemia has been associated with angiopathy.[28]

Obesity, together with excess glucagon and hyperglycemia, contributes to relative insulin deficiency, causing β-cells to fall behind in compensating. Additionally, abnormalities in nitric oxide metabolism have been linked to impaired perineural blood flow and subsequent nerve damage, as described in two hypotheses.[26]

Diagnosis of diabetes mellitus

Diagnosing diabetes in a patient without symptoms should never rely on just one abnormal blood glucose reading. It's essential for healthcare providers to confirm the diagnosis, as it carries serious, lifelong consequences for the patient. Diagnostic criteria for diabetes mellitus include tests such as urine sugar analysis, blood glucose levels, glucose tolerance tests, evaluation of the renal glucose threshold, impaired glucose tolerance, elevated glucose levels, renal glycosuria, and an extended glucose tolerance curve.[24]

SYMPTOMS

Typical Signs and Symptoms

1. Polyuria – Urinating more often than usual
2. Polydipsia – Feeling extremely thirsty
3. Polyphagia – Experiencing increased hunger
4. Unintended weight loss
5. Constant tiredness or lack of energy
6. Blurry vision

7. Wounds that take a long time to heal
8. Recurrent infections (such as skin or urinary tract infections)
9. Tingling or numbness in the hands or feet (particularly common in Type 2 diabetes)
10. Dry or itchy skin.[25]

Treatment of Diabetes Mellitus

Insulin Therapy

The main objective in managing Type 1 diabetes is to reduce the risk of long-term complications, prevent episodes of hypo- and hyperglycemia, and support normal growth and development. This is achieved through appropriate insulin administration to meet metabolic requirements, regular blood glucose monitoring, proper dietary planning, and adequate physical activity. In newly diagnosed children with Type 1 diabetes, short-term hospitalization is often required for initial management of diabetic ketoacidosis (DKA). During this period, careful monitoring of glucose levels, intravenous administration of insulin and glucose, and correction of hydration and electrolyte imbalances are essential. Additionally, patients and families must receive education about insulin delivery techniques, glucose monitoring, recognition and management of hypo- and hyperglycemia, and basic dietary interventions.[29]

The ultimate goal of insulin therapy is to closely mimic physiological insulin release preventing hypoglycemia between meals and minimizing postprandial hyperglycemia.[30] The method of administration intravenous, intramuscular, or subcutaneous is important for ensuring safe and effective action. Insulin is available in different forms, including human, bovine, and porcine sources. Despite its benefits, insulin therapy may cause side effects, most notably hypoglycemia and weight gain, particularly when dosages are inappropriate or not coordinated with meals.[31,32]

Regular insulin, a short-acting type, begins working within 50–60 minutes. It is a clear, colorless solution administered intramuscularly, subcutaneously, or intravenously.[33] Rapid-acting insulin analogs, such as insulin aspart (Novolog) and insulin lispro (Humalog), act within 15–30 minutes and can be taken immediately before meals, unlike regular insulin which requires earlier dosing. These analogs are also clear solutions and can be delivered via IV, IM, or SC routes, differing from other insulin preparations, which are suspensions.[34]

Hypoglycemic Agents

1. Biguanides (for T2DM management)

Metformin remains the first-line drug for type 2 diabetes management and is FDA-approved. It improves glycemic control by enhancing hepatic insulin sensitivity. Although mostly well tolerated, reported side effects (mainly from case studies) include sleep disturbances such as unusual dreams and, rarely, lactic acidosis.[35] Metformin also activates AMP-activated protein kinase, which regulates hepatic gluconeogenesis.[36] Caution is necessary in elderly patients with kidney impairment due to the increased risk of lactic acidosis. Unlike sulfonylureas, metformin rarely causes hypoglycaemia.[37]

2. Sulfonylureas (for T2DM management)

Sulfonylureas are widely prescribed as second-line agents, particularly for lean patients. Introduced in the 1950s with tolbutamide, they are classified into two groups: first-generation drugs (acetohexamide, tolbutamide, chlorpropamide, tolazamide) and second-generation drugs (glibenclamide, gliclazide, glipizide, gliquidone, and others). The second-generation agents are more potent. These drugs act as insulin secretagogues by stimulating pancreatic β-cells to release insulin, thereby lowering plasma glucose levels.[38-39] Their mechanism involves inhibition of ATP-sensitive potassium channels in islet cells, leading to enhanced insulin secretion.[40]

3. Meglitinide Derivatives (for T2DM management)

Repaglinide and nateglinide are non-sulfonylurea secretagogues that act similarly by promoting insulin release from pancreatic β-cells. They bind to ATP-dependent potassium channels at different sites compared to sulfonylureas.[41] This alters the cell membrane potential, ultimately stimulating insulin secretion.

Glucose regulation in pancreatic β-cells involves glucose transporter 2 (GLUT2), which facilitates the entry of extracellular glucose into the cell. Once inside, glucose is metabolized, generating adenosine triphosphate (ATP) for energy storage and use. The rise in ATP blocks ATP-sensitive potassium channels, leading to depolarization of β-cells. This triggers the opening of calcium channels, allowing calcium influx, which in turn stimulates insulin secretion.[42,43]

4. Alpha-Glucosidase Inhibitors (AGIs) in T2DM

Although not widely used, drugs such as acarbose, voglibose, and miglitol are considered safe and effective for type 2 diabetes management. They are particularly useful in controlling postprandial hyperglycemia but are contraindicated in patients with severe renal impairment. Their clinical application is often limited due to gastrointestinal side effects like flatulence and diarrhea.[65] Studies indicate that voglibose, a newer agent, significantly improves glucose tolerance, enhances normoglycemia rates, and delays disease progression.[44]

5. Dipeptidyl-Peptidase IV (DPP-4) Inhibitors

DPP-4 inhibitors act by blocking dipeptidyl-peptidase-4, an enzyme that rapidly inactivates GLP-1 and GIP. By increasing the active levels of these hormones, they improve islet function and glycemic control in type 2 diabetes.[45] This novel class of drugs is as effective as existing therapies and can be used as monotherapy in patients not controlled with diet and exercise, or as adjunct therapy with insulin, thiazolidinediones, and metformin. DPP-4 inhibitors are weight-neutral, carry a low risk of hypoglycemia, and are generally well tolerated. However, they are relatively expensive, and the long-term effects on β-cell function, morphology, and sustained glycemic control remain uncertain.[46,47]

Herbal Treatment of Diabetes Mellitus

With growing advancements in traditional medicine research, plant-derived therapies have gained attention as eco-friendly and biocompatible alternatives for managing diabetes.

Herbal remedies for diabetes have gradually moved from the fringes of medicine to mainstream research. Numerous authors have reviewed plant-based anti-diabetic agents, but Atta-ur-Rahman's work is considered particularly valuable, as it documents over 300 plant species known for their hypoglycemic activity. His review categorizes these plants according to their botanical names, countries of origin, plant parts used, and types of active compounds. Momordica charantia (bitter melon), belonging to the Cucurbitaceae family, is one well-known example.[48] Globally, the World Health Organization has identified around 21,000 medicinal plants, of which nearly 2,500 are found in India, with about 150 species being extensively used commercially. India is recognized as the world's largest producer of medicinal herbs and is often referred to as the "botanical garden of the world".[49]

CONCLUSION

Type 1 diabetes results from the pancreas failing to produce sufficient insulin, most often due to autoimmune destruction of pancreatic β-cells. Type 2 diabetes, on the other hand, is now understood to arise from autoimmune damage to the pancreas and/or the development of insulin resistance. In type 2 diabetes, the pancreas may still produce normal or even excessive amounts of insulin, but the body's tissues fail to utilize it effectively. The central aim of diabetes management is to restore carbohydrate metabolism to as close to normal as possible. For individuals with complete insulin deficiency, insulin replacement through injections or oral formulations is essential. In contrast, lifestyle modifications such as dietary regulation and physical activity can significantly improve insulin sensitivity in type 2 diabetes. Beyond glycemic control, treatment also focuses on preventing and managing complications associated with both the disease and its therapy. With proper management of blood glucose, individuals with diabetes can adapt to the condition and maintain a fulfilling, healthy life.

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