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REVOLUTIONIZING DIABETES CARE: INSIGHTS INTO NEW THERAPEUTIC APPROACHES AND TECHNOLOGIES –A REVIEW

Podili Niranjan Rao[4]*, Gangavaram Jyothi Reddy[1], Obulam Vijaya Sarathi Reddy[2], Podili Chandrakala[3], Budimepalli Swetha[1] and Vuluchi Srinivasa Rao[4]

[1]Student, S V U College of Pharmaceutical Sciences, SV UNIVERSITY. 517502, Andhra Pradesh, Tirupathi, India.

[2]Principal, S V U College of Pharmaceutical Sciences, SV UNIVERSITY. 517502, Andhra Pradesh, Tirupathi, India.

[3]Associate Professor, Sri Venkateswara Pharmacy College, RVS Nagar, 517127, Andhra Pradesh. India.

[4]*Academic Consulatant, S V U College of Pharmaceutical Sciences, SV UNIVERSITY. 517502, Andhra Pradesh, Tirupathi, India.

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***Corresponding Author**
Podili Niranjan Rao
Academic Consulatant, S V U College of Pharmaceutical Sciences, SV UNIVERSITY. 517502, Andhra Pradesh, Tirupathi, India.

ABSTRACT

Diabetes mellitus, a chronic metabolic disorder characterized by hyperglycemia, continues to pose a major global health challenge. Conventional therapies, though effective in glycemic control, often fall short in preventing long-term complications and ensuring patient compliance. The emergence of next-generation therapeutics offers promising alternatives for more effective, individualized, and sustainable diabetes management. This review explores cutting-edge therapeutic strategies, including incretin-based therapies, SGLT2 inhibitors, dual and triple agonists, gene therapy, stem cell-based approaches, nanotechnology-enabled drug delivery systems, and artificial pancreas technologies. We also delve into digital health innovations such as continuous glucose monitoring (CGM), closed-loop insulin delivery systems, and mobile health applications powered by artificial intelligence (AI). These advanced modalities not only aim to restore metabolic homeostasis but also address the broader aspects of diabetes care, including patient empowerment, reduced treatment

burden, and enhanced quality of life. Despite their potential, these therapies face significant translational challenges including regulatory hurdles, cost-effectiveness, and long-term safety concerns. This review provides a comprehensive overview of the current landscape and future directions in the evolution of diabetes therapeutics, highlighting the need for integrated, multidisciplinary approaches for optimal disease control. Treatment:

KEYWORDS: Diabetes mellitus, novel therapeutics, gene therapy, incretin, SGLT2 inhibitors, artificial pancreas, stem cell therapy, nanotechnology, digital health.

INTRODUCTION

Diabetes mellitus, commonly referred to as diabetes, is a chronic and multifaceted metabolic disorder that has emerged as a significant global public health concern. This condition exerts a profound impact on global morbidity and mortality, affecting hundreds of millions of individuals and placing immense pressure on healthcare systems worldwide.[1] Characterized by persistent hyperglycemia resulting from either insufficient insulin production or impaired insulin function, diabetes can lead to severe complications involving multiple organ systems, substantially diminishing the quality of life of those affected.

As the global burden of diabetes continues to escalate, personalized medicine is gaining recognition as a transformative approach in healthcare. This innovative strategy holds promise in enhancing diabetes care and improving patient outcomes by tailoring treatment to individual patient characteristics. The global concern surrounding diabetes is underscored by alarming statistics: according to the International Diabetes Federation (IDF), an estimated 463 million adults were living with diabetes in 2019, with projections indicating that this number could rise to nearly 700 million by 2045 if urgent action is not taken.[2]

Multiple factors contribute to this growing epidemic, including sedentary lifestyles, unhealthy dietary habits, increasing rates of obesity, and an aging population.[3] Diabetes affects individuals across all ages, races, and socioeconomic statuses. However, its impact is particularly pronounced in low- and middle-income countries, where limited access to healthcare, resources, and education exacerbates the disease burden. This disparity highlights how diabetes contributes to widening global health inequalities.[4]

Conventional approaches to diabetes management, which rely on standardized treatment protocols, have demonstrated limitations in the face of such a heterogeneous disease.

Diabetes exhibits diverse clinical manifestations and variable responses to therapy among individuals. Consequently, uniform treatment strategies may fail to meet the specific needs of each patient, leading to suboptimal glycemic control and poor clinical outcomes. In light of these challenges, personalized medicine—also referred to as precision or individualized medicine—has emerged as a promising alternative.[5]

Personalized medicine represents a paradigm shift in healthcare, aiming to customize treatment plans based on the unique biological, environmental, and lifestyle factors of each patient. By leveraging this individualized approach, clinicians can make more accurate therapeutic decisions, thereby enhancing treatment efficacy and improving health outcomes.[6]

A central component of personalized diabetes care is the understanding of genetic factors that influence disease susceptibility. Genetic screening enables the early identification of individuals at elevated risk for diabetes, facilitating timely preventive strategies and lifestyle interventions.[7] Moreover, genetic insights support the selection of the most appropriate pharmacologic agents for each patient, reducing the likelihood of adverse drug reactions and therapeutic failures.[8]

Advances in technology further bolster the implementation of personalized diabetes care. Tools such as continuous glucose monitors and wearable devices empower patients to take an active role in their disease management. These devices provide real-time data and individualized feedback, aiding patients in making informed decisions regarding their diet, physical activity, and medication adherence.[9] This technology-enhanced, patient-centered approach fosters greater engagement, autonomy, and accountability, ultimately contributing to better long-term health outcomes.[10]

Etiology of Diabetes Mellitus

The etiology of diabetes mellitus is multifactorial, involving complex interactions between genetic, environmental, immunological, and metabolic factors. Based on current understanding, both type 1 and type 2 diabetes develop through distinct yet overlapping pathogenic mechanisms.

- Autoimmune and Viral Factors (Type 1 Diabetes Mellitus): Type 1 diabetes, often referred to as juvenile-onset or insulin-dependent diabetes, is primarily considered an autoimmune disorder. It is characterized by immune-mediated destruction of pancreatic

β-cells, leading to an absolute insulin deficiency. Viral infections such as Coxsackie B virus, rubella (German measles), and mumps (epidemic parotitis) have been implicated in triggering autoimmune responses that result in morphological changes to islet-cell structures and subsequent β-cell destruction.[11]

- Genetic Susceptibility: The genetic basis of diabetes is well-recognized but not completely understood. In genetically predisposed individuals, the pancreas may exhibit increased susceptibility to environmental triggers, including viral infections. This genetic vulnerability may manifest as abnormalities in β-cell glucose sensing or a relative deficiency in insulin secretion.[12]
- Metabolic and Vascular Mechanisms (Type 2 Diabetes Mellitus): Type 2 diabetes is primarily associated with insulin resistance and relative insulin deficiency. A decrease in insulin sensitivity in peripheral tissues (e.g., liver, muscle, and adipose tissue) is often due to "downregulation" of insulin receptors, contributing to hyperinsulinemia. This, in turn, is associated with conditions such as dyslipidemia, hyperuricemia, abdominal obesity, and vascular complications.[13]
- Endocrine and Hormonal Imbalances: Increased glucagon secretion, often seen in obese individuals, exacerbates insulin insufficiency and β-cell dysfunction. Inadequate β-cell compensation may lead to progressive hyperglycemia. Some theories suggest that altered nitric oxide metabolism may impair perineural blood flow, contributing to diabetic neuropathy.
- Monogenic and Secondary Diabetes: Uncommon forms of diabetes include those due to specific genetic mutations (collectively classified as type 3 diabetes). These include conditions such as Maturity-Onset Diabetes of the Young (MODY), diabetes due to pancreatectomy, endocrinopathies, and Gestational Diabetes Mellitus (GDM).[14]
- Receptor and Enzyme Dysregulation: Imbalances in specific receptors and enzymes play critical roles in the pathogenesis of diabetes. Key examples include the GLP-1 receptor, peroxisome proliferator-activated receptor gamma (PPARγ), β3-adrenergic receptor, as well as enzymes such as α-glucosidase and dipeptidyl peptidase-IV (DPP-IV). Disruptions in these systems impair glucose homeostasis and insulin signaling.
- Emerging Molecular Mechanisms: Current research highlights the roles of oxidative stress, advanced glycation end-products (AGEs), protein kinase C activation, and the polyol pathway in the pathophysiology of both microvascular and macrovascular complications in diabetes.[15]

Classification of Diabetes Mellitus

The classification of diabetes mellitus has evolved significantly over the decades to reflect a more nuanced understanding of the disease's etiology and clinical presentation. The earliest widely accepted classification was introduced by the World Health Organization (WHO) in 1980 and later revised in 1985. This system primarily focused on distinguishing between primary (idiopathic) and secondary forms of diabetes.

Primary diabetes, now more commonly referred to as type 1 and type 2 diabetes, represents the most prevalent forms and serves as the focus of most clinical management and research. In contrast, secondary diabetes encompasses hyperglycemia that arises as a consequence of other identifiable pathological conditions.

These Include

- Diseases of the exocrine pancreas (e.g., pancreatitis, pancreatic neoplasms)
- Endocrinopathies (e.g., Cushing's syndrome, acromegaly)
- Surgical interventions involving the pancreas
- Drug-induced diabetes (e.g., glucocorticoids, thiazide diuretics)
- Genetic disorders (e.g., hemochromatosis, monogenic diabetes)

It is increasingly recognized that many individuals do not fit neatly into a single diagnostic category, and the heterogeneous nature of diabetes demands a flexible, multifactorial classification system.[16]

The modern classification, as outlined by the WHO Expert Committee (1999) and adopted in the International Nomenclature of Diseases (1991) and ICD-10 (1992), categorizes diabetes into four principal types[17]:

1. Type 1 Diabetes Mellitus: Previously known as insulin-dependent diabetes mellitus (IDDM), this form results from autoimmune β-cell destruction, leading to absolute insulin deficiency. It typically manifests in childhood or adolescence but can occur at any age.
2. Type 2 Diabetes Mellitus: Formerly called non-insulin-dependent diabetes mellitus (NIDDM), this type is characterized by insulin resistance and a relative insulin secretory defect. It constitutes the majority of diabetes cases worldwide and is frequently associated with obesity, aging, and sedentary lifestyle.
3. Other Specific Types of Diabetes: This category includes diabetes due to identifiable causes such as:

- Genetic defects of β-cell function (e.g., MODY)
- Genetic defects in insulin action
- Diseases of the exocrine pancreas
- Endocrinopathies
- Drug- or chemical-induced diabetes
- Infections
- Uncommon forms of immune-mediated diabetes

4. Gestational Diabetes Mellitus (GDM)

Defined as glucose intolerance that is first recognized during pregnancy, GDM may resolve postpartum but is associated with an increased risk of developing type 2 diabetes later in life.

1. Hormone-Dependent Diabetes (Type 1 Diabetes Mellitus, IDDM)

Type 1 diabetes mellitus, historically referred to as juvenile-onset diabetes or ketosis-prone diabetes, is also known as reaction diabetes due to its autoimmune nature. This form predominantly affects children and young adults and is characterized by the immune-mediated destruction of pancreatic β-islet cells, leading to absolute insulin deficiency.[18] Individuals with type 1 diabetes often present with other autoimmune disorders such as Graves' disease, Hashimoto's thyroiditis, and Addison's disease, highlighting its autoimmune etiology.

Clinically, type 1 diabetes manifests with a rapid onset of symptoms that may be severe and potentially life-threatening if untreated. The presence of autoantibodies—including anti-glutamic acid decarboxylase (GAD), islet cell antibodies, and insulin autoantibodies—serves as markers of the autoimmune process responsible for β-cell destruction.[19] These autoantibodies are detected in approximately 85–90% of patients at diagnosis, supporting the autoimmune hypothesis.[20]

The rate of β-cell destruction varies among individuals, ranging from rapid to more gradual progression, which influences the clinical presentation and timing of insulin dependence. Due to the loss of endogenous insulin production, patients require lifelong exogenous insulin therapy to regulate blood glucose and prevent ketoacidosis and other complications.[21]

Although the exact triggers of the autoimmune response remain unclear, it is widely accepted that type 1 diabetes results from a combination of genetic susceptibility and environmental factors leading to immune-mediated β-cell destruction.

Non-Insulin Dependent Diabetes Mellitus (Type 2 Diabetes Mellitus, NIDDM)

Type 2 diabetes mellitus, also known as ketosis-resistant diabetes, is characterized by a progressive decline in pancreatic β-cell insulin secretion coupled with peripheral insulin resistance [American Diabetes Association, 2014]. Unlike type 1 diabetes, individuals with type 2 diabetes typically retain some endogenous insulin production but exhibit reduced sensitivity to insulin's effects, leading to impaired glucose metabolism.

Patients with type 2 diabetes often demonstrate resistance to oral hypoglycemic agents, complicating glycemic control. Long-term complications associated with this form of diabetes involve damage to the blood vessels (macrovascular and microvascular complications), kidneys (nephropathy), eyes (retinopathy), and nerves (neuropathy), which are major contributors to diabetes-related morbidity and mortality.[22,23]

The etiology of type 2 diabetes is multifactorial, involving an interplay of genetic predisposition and environmental factors. Key risk factors include obesity, sedentary lifestyle, and advancing age, predominantly affecting middle-aged and older adults [Ross and Wilson, 2010]. These factors not only contribute to insulin resistance but also increase the risk of developing serious complications.

Gestational Diabetes Mellitus (GDM)

Gestational diabetes mellitus (GDM) refers to glucose intolerance that is first recognized or diagnosed during pregnancy. This condition includes women who develop type 1 diabetes mellitus during gestation as well as those with previously undiagnosed, asymptomatic type 2 diabetes identified during pregnancy.[24] GDM is characterized by hyperglycemia that arises during pregnancy but does not meet the criteria for overt diabetes.

While gestational diabetes often resolves after delivery, it poses significant risks to both mother and child. Offspring born to mothers with GDM have an increased risk of developing obesity and type 2 diabetes later in life, a consequence believed to stem from intrauterine exposure to maternal hyperglycemia. Therefore, proper diagnosis and management of GDM Alternative Specific Sort (Monogenic Types)

Monogenic forms of diabetes represent a hereditary subset of diabetes mellitus and are most commonly linked to mutations on chromosome 12, affecting the transcription factor known as hepatocyte nuclear factor-1 alpha (HNF-1α). These mutations cause genetic abnormalities in pancreatic beta-cell function. This form of diabetes is typically characterized by an early onset, usually before the age of twenty-five, and is commonly referred to as Maturity Onset Diabetes of the Young (MODY).[25]

Monogenic diabetes includes cases of diabetes in youth that are resistant to ketoacidosis and/or involve defects in insulin action or secretion. It also covers diabetes associated with diseases of the exocrine pancreas, such as pancreatitis or cystic fibrosis, as well as diabetes secondary to other endocrinopathies like acromegaly. Furthermore, diabetes may develop due to pancreatic damage caused by certain medications, chemicals, or infections.[26]

Certain drugs used in the treatment of conditions like HIV/AIDS or following organ transplantation can also induce this form of diabetes. In some families, genetic defects impair the conversion of proinsulin to insulin, and these traits follow an autosomal dominant inheritance pattern. However, monogenic diabetes accounts for less than 1% of all diabetes mellitus cases.[27]

HISTORY

Behavior of Diabetes in Various Regions

The World Health Organization (WHO) global report on diabetes highlights that many regions lack effective policies to promote healthy lifestyles and provide access to quality healthcare. This deficiency results in inadequate prevention and treatment of diabetes, especially for individuals with limited resources (WHO, 2016). According to the International Diabetes Federation (IDF) Diabetes Atlas, approximately 463 million adults aged 20 to 79 years had diabetes in 2019, representing 9.3% of the global population in this age group. This number is projected to rise to 578 million (10.2%) by 2030 and 700 million (10.9%) by 2045 (IDF, 2019).

Regional Insights (IDF, 2019)

- The European region has the highest number of children and adolescents (0-19 years) with type 1 diabetes, totaling 296,500 individuals.
- The Middle East and North Africa region reports the highest age-adjusted prevalence of diabetes (~12%).

- North America and the Caribbean bear 43% of global diabetes-related healthcare costs.
- In South and Central America, 44% of diabetes-related deaths occur in individuals under 60 years.
- In Southeast Asia, 57% of adults with diabetes remain undiagnosed.
- The Western Pacific region recorded over one million diabetes-related deaths in 2019, the highest worldwide.

POPULATION GENETICS

Researchers emphasize the role of population genetics in understanding diabetes progression, focusing on how gene distribution and changes across generations influence disease prevalence.[28] Ethnicity is a significant risk factor; the WHO notes the highest rates of type 2 diabetes among individuals of Asian and African descent, as well as indigenous populations in the Americas and Australian Aboriginal communities (Government of Mexico, 2018).

COMPLICATIONS

Diabetes and Cardiovascular Disease

Cardiovascular disease (CVD) remains the leading cause of death among individuals with diabetes, as reported by health professionals in Uruguay. Recent advances in medicine have shown promising results in reducing cardiovascular events by targeting the shared pathophysiology of diabetes and CVD. Novel medications not only lower blood glucose but also significantly reduce cardiovascular complications.[29,30]

Blood Pressure and Diabetes

Several comorbid conditions worsen target organ damage in primary hypertension, with metabolic syndrome components—central obesity, dyslipidemia, and insulin resistance with hyperinsulinemia—being particularly important.[31,32] Type 2 diabetes frequently coexists with obesity, hypertension, and dyslipidemia. Inflammatory, coagulation, and fibrinolytic abnormalities further increase cardiovascular risk in these patients.[33] Recent research has linked the SGK1 gene with hyperinsulinemia-induced overactivation of the epithelial sodium channel (ENaC) in the kidney's collecting ducts, contributing to hypertension.[34,35]

Diabetes and Obesity

Excess body fat is a major driver of type 2 diabetes, with risk increasing linearly with body mass index (BMI). The global rise in obesity has directly contributed to increased diabetes prevalence. The complex mechanisms include adiposity-induced alterations in beta-cell

function, adipose tissue biology, and multi-organ insulin resistance. Notably, these effects can often be reversed or improved with adequate weight loss.[36,37]

SIGNS AND SYMPTOMS

Because diabetes is a chronic illness, its symptoms are often overlooked or underestimated. Unlike many diseases, hyperglycemia's damaging effects may develop silently over many years before symptoms appear. Early detection is crucial for preventing vascular complications.

Common symptoms include

- Polyuria (frequent urination)
- Polydipsia (excessive thirst)
- Polyphagia (increased hunger)

These are especially evident in type 1 diabetes with rapid onset and in type 2 diabetes when hyperglycemia is severe.

Other signs may include

- Unexplained weight loss (more common in type 1 or advanced type 2)
- Frequent fatigue and irritability
- Recurrent infections (especially genital, urinary tract, skin, and oral infections)
- Delayed wound healing
- Dry mouth
- Burning, pain, or numbness in the feet (neuropathy)
- Itching
- Reactive hypoglycemia
- Acanthosis nigricans (velvety dark patches on the neck, armpits, or groin indicating insulin resistance)
- Decreased vision
- Impotence or erectile dysfunction

Risk Factors in the Development of Diabetes

- **Obesity**
  - Weight exceeding 120% of ideal body weight
  - Body Mass Index (BMI) greater than 27 kg/m²

- **High-Risk Ethnic Background**
  - Asian
  - Pacific Islander
  - American Indian
  - African-American
  - Hispanic
- Hypertension

- **Lipid Abnormalities**
  - HDL (High-Density Lipoprotein) level < 35 mg/dL
  - Triglyceride level > 250 mg/dL

- **Family History**
  - First-degree relative diagnosed with diabetes

- **Birth History and Pregnancy**
  - Infant birth weight > 9 pounds
  - History of gestational diabetes

- **Glucose Abnormalities**
  - Impaired glucose tolerance or impaired fasting glycemia (blood sugar 110–126 mg/dL)

- **Other Medical Conditions**
  - History of vascular disease
  - Polycystic ovarian disease (PCOS)

Pathophysiological Aspects of Diabetes

- Type 2 Diabetes:
  - Characterized by insulin insensitivity, insulin resistance, reduced insulin production, and pancreatic beta-cell failure.
  - These abnormalities lead to decreased glucose uptake in the liver, muscle, and fat cells, resulting in hyperglycemia.
  - Hyperglycemia accelerates fat breakdown, contributing to metabolic imbalance.[40,41]
  - Both insulin resistance and impaired insulin secretion are central to the disease's development.
  - Typically presents in adulthood, often in individuals who are overweight or obese.

- Progressive decline in beta-cell function with aging contributes to disease worsening.

- **Type 1 Diabetes**
- Generally affects younger, non-obese individuals.
- Has a strong genetic component, with a 10-fold increased risk among first-degree relatives.
- Strong associations with specific human leukocyte antigen (HLA) types.
- Environmental triggers, such as viral infections, play a role in disease onset.
- Viral infections can damage pancreatic beta-cells and expose antigens that trigger an autoimmune response.
- Clinical diabetes manifests after >90% of beta-cells are destroyed.
- Insulin deficiency in type 1 diabetes may also impair cognitive functions like memory and learning due to reduced long-term potentiation.

- **Molecular Mechanisms Related to Insulin Resistance and Neurodegeneration**
- Insulin resistance is implicated in tau protein hyperphosphorylation and amyloid-beta (Aβ) plaque formation.
- Hyperinsulinemia leads to competition between insulin and Aβ for the insulin-degrading enzyme, resulting in Aβ accumulation and plaque formation.
- Reduced insulin receptor signaling causes:
  - Tau hyperphosphorylation
  - Inhibition of protein kinase B (Akt)
  - Activation (dephosphorylation) of glycogen synthase kinase-3 beta (GSK-3β)
- These processes link insulin resistance with neurodegenerative changes observed in diabetes.[43,44]

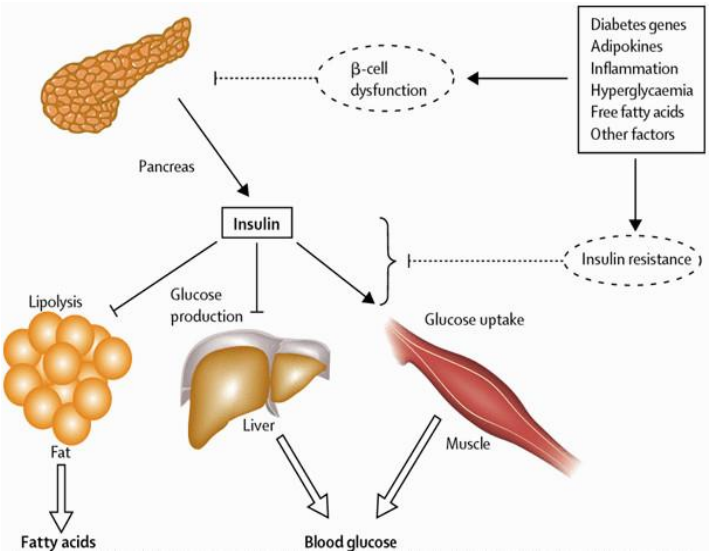

Figure 1: Pathophysiology of hyperglycaemia and increased circulating fatty acids in type 2 diabetes.

Diagnosis and Treatment of Diabetes

Diagnosis

- A single abnormal blood glucose reading should not be used to diagnose diabetes in an asymptomatic patient. Given the significant and long-term consequences of the diagnosis, clinicians must confirm diabetes with reliable testing before diagnosis.[45]

- **Diagnostic tools include**
  - Blood sugar measurement
  - Urine sugar analysis
  - Glucose tolerance tests (oral and intravenous)
  - Renal threshold assessments
  - Cortisone-stressed glucose tolerance test
  - Extended glucose tolerance curves
  - Detection of impaired or increased glucose tolerance
  - Renal glycosuria

Treatment

The primary treatment goal is to address the underlying cause and provide frequent, appropriately dosed insulin, especially in insulin-dependent cases. Once the condition is stabilized, insulin requirements may return to normal.

Goals of Diabetes Care

1. Restore metabolism as close to normal as possible, safely and comfortably.
2. Prevent or slow progression of immediate and long-term complications.
3. Empower patients with education, motivation, and resources to manage their condition effectively.

A. Insulin Therapy: Ultra-Rapid-Acting Insulins

- New ultra-rapid-acting insulins like faster aspart and ultra-rapid lispro (URLi) offer more options for bolus insulin in both type 1 and type 2 diabetes.
- These insulins have:
  - Faster onset of action (e.g., faster aspart acts 5 minutes sooner than standard insulin aspart; URLi acts 10.9 minutes faster in type 1 diabetes and 13 minutes faster in type 2 diabetes).
  - Reduced time to peak insulin concentration compared to traditional short- and rapid-acting insulins.
  - Comparable duration of action to rapid-acting insulins.
- Clinical trials (ONSET 1 and PRONTO-T1D/T2D) show these insulins can be administered at mealtime or shortly after, with better postprandial glucose (PPG) control when given before meals.
- These agents demonstrated non-inferiority in reducing HbA1c, with some showing statistically significant improvements in 1- and 2-hour PPG levels compared to traditional insulins.
- Benefits include improved flexibility in insulin administration timing and potential for better glucose control, especially in patients using continuous glucose monitoring (CGM) and tracking time in range.[46–49]

B. Oral Hypoglycemic (Antidiabetic) Agents

- Biguanides (e.g., phenformin introduced alongside sulfonylureas in 1957) remain foundational therapies.
- Newer classes include:
  - Thiazolidinediones
  - Meglitinide analogues
  - α-Glucosidase inhibitors
  - Dipeptidyl peptidase-4 (DPP-4) inhibitors

- These drugs target various pathophysiological aspects of diabetes and provide alternatives or adjuncts to insulin therapy.

C. Types of Therapy Involved in Diabetes Mellitus

1. Stem Cell Therapy

- Research suggests monocytes and macrophages contribute to insulin resistance and chronic inflammation in type 2 diabetes.
- Stem cell educator therapy is a novel approach aiming to regulate or reverse immune dysfunctions.
- The procedure involves a closed-loop system where the patient's blood is collected, lymphocytes are purified and co-cultured with adherent cord blood-derived multipotent stem cells (CB-SCs) in vitro.
- The "educated" lymphocytes (not the stem cells themselves) are then reinfused into the patient's circulation to modulate immune response.[51]

2. Treatment with Antioxidants

- Various antioxidants—vitamins, plant-derived compounds, supplements, and antioxidant drugs—are used to reduce oxidative stress in T2DM patients.
- Vitamins C, E, and β-carotene are particularly effective in combating oxidative stress and its complications.[52]
- Antioxidants help reduce the risk of diabetes onset and its related complications.

3. Treatment with Anti-Inflammatory Drugs

- Inflammation plays a central role in the pathophysiology and complications of type 2 diabetes.[53,54]
- Key affected tissues include adipose tissue, pancreatic islets, liver, vasculature, and circulating leukocytes.
- Changes include altered cytokine/chemokine levels, leukocyte activation, increased apoptosis, and tissue damage.
- Immunomodulatory drugs targeting these pathways are available to help manage diabetes-associated inflammation.

D. Dietary Management

Patients with or without diabetes should follow these general dietary guidelines:

1. Maintain a balance of proteins, carbohydrates, and fats; in some cases, carbohydrate intake may need to be limited.
2. Diet should resemble a normal, healthy eating pattern as much as possible.
3. Distribute food intake evenly across multiple meals of comparable size throughout the day.
4. Reduce fat and carbohydrate intake to lower overall calorie consumption.
5. Counsel patients to maintain consistent daily eating habits to aid glycemic control.

E. Newer Insulin Delivery Devices

Several advancements have improved insulin administration to achieve better glycemic control with increased convenience and accuracy:

- Insulin syringes
- Insulin pen devices
- Inhaled insulin formulations
- Insulin pumps (including implantable pumps)
- Other novel routes of insulin delivery[55,56]

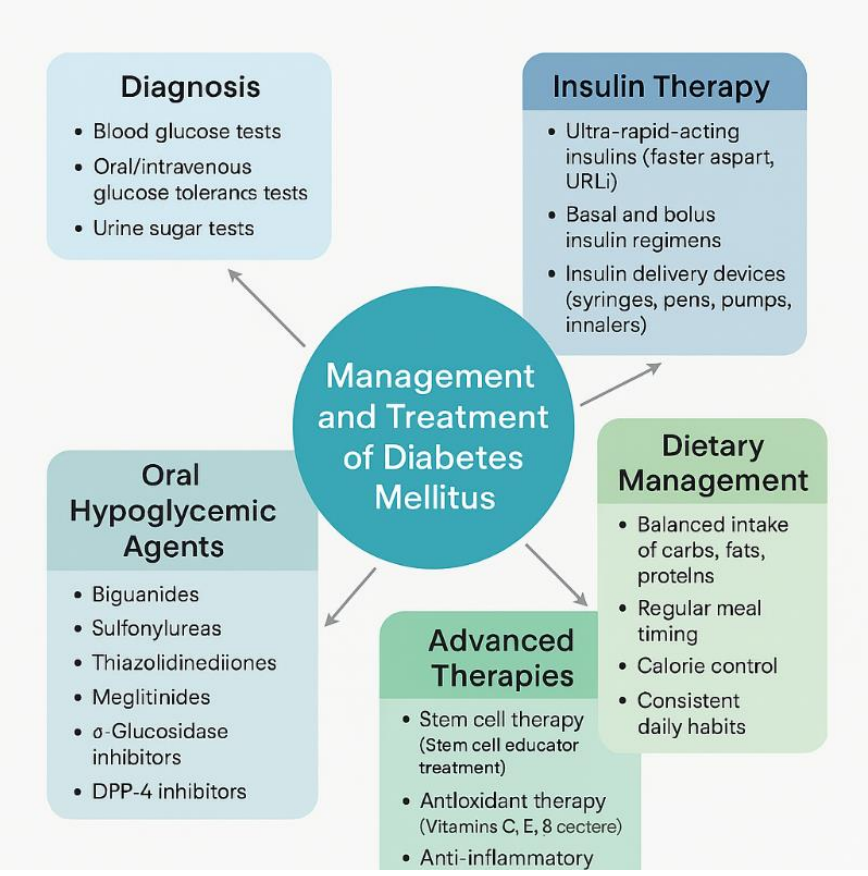

Figure 2: Management and Treatment.

CONCLUSION

The primary goal of diabetes management is to restore glucose homeostasis as close to physiological norms as possible. In T1DM, this necessitates exogenous insulin administration, typically via injections or insulin pumps. For T2DM, lifestyle modifications such as diet control and increased physical activity are critical to enhancing insulin sensitivity. Pharmacological therapies, including metformin and newer agents like SGLT2 inhibitors and GLP-1 receptor agonists, are employed to improve glycemic control and reduce cardiovascular risks.

Beyond glycemic regulation, diabetes management aims to prevent acute and chronic complications, such as cardiovascular disease, nephropathy, retinopathy, and neuropathy, which significantly contribute to morbidity and mortality. Effective blood glucose control has been shown to reduce the risk of these complications by up to 50%, emphasizing the importance of early and sustained intervention. Thus, with comprehensive management, individuals with diabetes can maintain a good quality of life and lower the risk of long-term complications.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest regarding this investigation.

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