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Diabetes Management Strategies: Review of Current Treatment Approaches

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Abstract: *Diabetes mellitus (DM), a chronic metabolic disorder marked by hyperglycaemia, results from defects in insulin secretion, insulin action, or both.Its global burden continues to escalate, affecting over 540 million individuals as of 2024.Different treatment options for the management of DM are evolving rapidly because the usual methods of treatment have not completely tackled the primary causes of the disease and are laden with critical adverse effects.Our findings indicate that substantial progress has been made in DM management with promising results using different treatment regimens, including nanotechnology, gene therapy, stem cell, medical nutrition therapy, and lifestyle modification.Other effective medication includes non-sulfonylurea secretagogues, thiazolidinediones, alpha glucoside inhibitors, and insulin. The review underscores the significance of achieving optimal metabolic control and advocating for public health initiatives that enhance healthcare accessibility and foster patient-centered care. Ultimately, a holistic approach that integrates these emerging technologies and therapies could lead to improved therapy of diabetes and its related complications.*

Keywords: Diabetes mellitus;nanotechnology; stem cell; statin; gene therapy;insulin

I. INTRODUCTION

Diabetes is a condition where blood sugar levels are too high, a condition known as hyperglycemia. This happens when the pancreas doesn't produce enough insulin or the body's cells don't respond to insulin[1, 2].If an elevated glucose level persists, chronic diabetes-related problems that impact numerous organs may occur, such as retinopathy, nephropathy, peripheral neuropathy, peripheral art rial disorders, and cerebrovascular disease [1].According to the World Health Organization, diabetes mellitus is a serious health issue that can lead to significant illness and death[3]. According to recent estimation, 387 million people worldwide are affected from the disease with a prevalence rate of 8.3% and 46.3% still remains undiagnosed[4].

Insulin and glucagon hormones both are secreted by the pancreas. insulin is secreted by the β- cells and glucagon is secreted by the α-cells both are located in the islets of langerhans. Insulin decreases the blood glucose level by the glycogenesis and transport glucose into the muscle, liver and adipose tissue[5]. Diabetes is increasing in incidence and prevalence worldwide, owing to an increase in type 1 diabetes in children and type 2 diabetes due to lifestyle changes, particularly in developing nations. Diabetes is prevalent in sports at all levels of competition, and it is becoming more prevalent due to the growing number of master athletes and the occurrence of type 2 diabetes at younger ages[6]. The disease manifests in several forms, primarily type 1 diabetes mellitus (T1DM), type 2 diabetes mellitus (T2DM), gestational diabetes mellitus (GDM), and rare monogenic diabetes syndromes. Among these, T2DM accounts for more than 90% of global diabetes cases and is frequently associated with obesity, physical inactivity, and insulin resistance [7]. In individuals suffering from type II diabetes mellitus (T2DM), hyperglycemia is not the only characteristic; it also involves multiple complications such as kidney failure, blindness, heart attack, stroke, and amputations of the lower limb[8].

II. EPIDERMINOLOGY

In 2021, the International Diabetes Federation (IDF) reported that global healthcare expenditures for diabetes man agement among individuals aged 20 to 79 reached approximately $966 billion, with projections suggesting they could

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exceed $1054 billion by 2045[9]. It is estimated that 366 million people had DM in 2011; by 2030 this would have risen to 552 million. DM caused 4.6 million deaths in 2011[10]. Furthermore, maximum percentage of 387 million people lives in low and middle income countries andcompriseof40–59agegroupinthepopulation.Population survey by the Indian Council of Medical Research [11].

III. PATHOPHYSIOLOGY

The primary hormone regulating glucose uptake into most body cell such as liver, adipose tissues, muscles is insulin, except in smooth muscle where it acts as the IGF-1 receptor. As a result, insulin insufficiency or receptor insensitivity play a key role in all types of diabetes mellitus[12]. If the amount of insulin available is insufficient, or if cells respond poorly to insulin's effects (insulin resistance), or if the insulin itself is defective, glucose is not absorbed effectively by the body cells that require it, and it is not stored appropriately in the liver and muscles. The end result is elevated blood glucose levels, inadequate protein synthesis, and other metabolites., such as metabolic acidosis in cases of complete insulin deficiency[13].

The pathophysiology of diabetes mellitus varies between its primary forms, Type 1 and Type 2, but both involve disruptions in glucose metabolism due to problems with insulin. In Type 1 diabetes (T1D), an autoimmune process targets and destroys pancreatic beta cells, which are responsible for producing insulin. This destruction leads to an absolute deficiency of insulin, preventing glucose from entering cells and causing elevated blood glucose levels. Genetic predisposition plays a significant role in T1D, with environmental triggers such as viral infections potentially initiating the autoimmune response[14]. Type 2 DM involves insulin resistance, reduced insulin production, and eventual pancreatic beta-cell failure. This affects glucose transport into the liver, muscle cells, and fat cells and increases breakdown,leading to high blood sugar. The involvement of impaired alpha-cell function has recently been recognized in the pathophysiology of type 2 DM[15]. Other forms of diabetes, including gestational diabetes and monogenic diabetes, have distinct pathophysiological mechanisms. Gestational diabetes occurs due to hormonal changes during pregnancy that induce insulin resistance, while monogenic diabetes results from mutations in a single gene affecting insulin production or action.

Table 3.1 tubular comparison of type 1 and type 2 Diabetes:

Aspect	Type 1 diabetes	Type 2 Diabetes
Cause	Autoimmune destruction of bets cells	Insulin resistance,relative insulin deficiency
Insulin Level	Absolute deficiency	Relative dificiency
Onset	Often in youth	Typically in adults
Triggers	Genetic, environmenntal(viral infection	Lifestyle, genetics,obesity

IV. CLASSIFICATION OF DIABETES

4.1.Type I Diabetes mellitus

Type 1 DM, also referred to as reaction diabetes, was historicallydesignated as juvenile-onset or ketosis-prone diabetes. Individuals afflicted with Type I diabetes may exhibit co-morbidities withother autoimmune disorders like Graves' disease, Hashimoto's thyroiditis, and Addison's disease[16]. Type 1 diabetes mellitus, commonly termed insulin-dependent diabetes mellitus, is a diabetic condition predominantly affecting children and young individuals[5].The autoimmune mediated destruction of β-cells results in absolute insulin deficiency, thereby necessitating lifelong exogenous insulin therapy.

The kinetics of β cell destruction demonstrate variability, with rapid deterioration observed in infants and children, whereas a slower degenerative process is evident in adults. Clinical manifestations such as ketoacidosis are prevalent in children and young individuals, while others may present with modest fasting hyperglycemia that can escalate to severe hyperglycemia or ketoacidosis in response to stress or infection[17].

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4.2. Type II Diabetes mellitus

Type 2 DM is also characterized as ketosis-resistant diabetes. DM2 constitutes approximately 80 percent to 90 percent of all DM cases.A significant proportion of individuals diagnosed with Type 2 diabetes exhibit intra-abdominal (visceral) obesity, which is directly correlated with insulin resistance. Type 2 diabetes mellitus represents the most prevalent form of diabetes and is identified ad the fourth leading cause of mortality developed countries. This condition is associated with a twofold increase in excess mortality and two- to fourfold heightened risk of coronary heart disease and stroke [18].

4.3. Gestational Diabetes Mellitus (GDM)

It is defined as any degree of glucose in tolerance resulting in hyperglycaemia of variable severity that is diagnosed during pregnancy[19]. It typically develops in the second or third trimester and is characterized by high blood sugar levels that are not usually present before pregnancy. The condition arises when the placenta produces hormones that interfere with insulin's ability to regulate blood sugar, leading to insulin resistance.

4.4. Monogenic type Diabetes

These types of diabetes are often marked by the beginning of hyperglycaemia at a young age. A few families have been discovered with genetic anomalies that result in the inability to convert proinsulin to insulin, and such features are inherited in an autosomal dominant way. They account for less than 10% of all DM cases[20]. Monogenic diabetes, including maturity-onset diabetes of the young (MODY), results from mutations in single genes affecting β-cell development or function (e.g., HNF1A, GCK)[21].

V. DIAGNOSIS OF DIABETES

Tests for diagnosis of DM are readily available. If a patient is diagnosed with diabetes, the practitioner must be confident that the diagnosis is correct, as the repercussions for the patient are severe and long-term. Urine sugar, blood sugar, glucose tolerance test, renal glucose threshold, diminished glucose tolerance, increased glucose tolerance, renal glycosuria, extended glucose tolerance curve, cortisone stressed glucose tolerance test, intravenous glucose tolerance test, and oral glucose tolerance test are all used to diagnose diabetes mellitus[22].

Traditional diagnostic methods remain widely used; however, recent innovations have introduced more precise, real-time, and personalized tools for glycemic monitoring and diabetes risk stratification. This section outlines both established and emerging diagnostic strategies for diabetes mellitus.

5.1.Conventional Diagnostic Criteria

The conventional diagnosis of diabetes mellitus is primarily based on plasma glucose concentrations and HbA1c levels as outlined by the American Diabetes Association (ADA) and the World Health Organization (WHO).

• Fasting plasma glucose (FPG)

This test checks fasting blood sugar levels. This test is done f irst thing in the morning, before breakfast. Diabetes is diagnosed at fasting blood sugar of greater than or equal to 126 mg/dl.1[22].

• Oralglucose tolerance test (OGTT)

The OGTT is a two-hour test that checks your blood sugar levels before and two hours after you drink a special sweet drink. It tells the doctor how your body processes sugar. Diabetesisdiagnosedat2-hourbloodsugarofgreater than or equal to 200 mg/dl.11[22].

5.2. Novel Biomarkers for Diabetes Detection

In the quest for earlier and more precise diabetes detection, several novel biomarkers are being investigated for their diagnostic and prognostic potential:

• Random Plasma Glucose:

≥200 mg/dL in the presence of classic hyperglycemia symptoms . This test is a blood check at any time of the day if one has severe diabetes symptoms. Diabetes is diagnosed at blood sugar of greater than or equal to 200 mg/dl[22].

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• **C-peptide:**
Reflects endogenous insulin secretion and helps distinguish between T1DM and T2DM.
• **Inflammatory markers:**
C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) are elevated in insulin-resistant states.

5.3 Biosensors and Non-Invasive Glucose Monitoring

The recent development of non-invasive and minimally invasive glucose monitoring techniques represents a significant advance in diabetes diagnostics. These include:
• **Optical Sensors:**
Use near-infrared or Raman spectroscopy to detect glucose through the skin.
• **Electrochemical Biosensors:**
Detect glucose based on enzymatic oxidation reactions on wearable devices.

VI. COMPLICATION

The complications of diabetes can dramatically impair quality of life and cause long-lasting disability. There are a number of promising new therapeutic developments in the management of com plications associated with diabetes. One agent, tolrestat[23]. Another complication associated with neuropathy is the development of foot ulcers. The administration of pentoxifylline in addition to conventional therapy may improve healing in these states. recalling suggestions that some complications of diabetes might in volve a microcirculatory disease affecting the autonomic nervous system [24,25]. Diabetic ketoacidosis(DKA) is a serious and life-threatening condition that should always be treated as a medical emergency. Low insulin levels allow the liver to convert fatty acids to ketone bodies as a source of energy; ketone bodies are intermediate substrates in this metabolic process. Diabetic coma is a medical emergency in which a person with diabetes mellitus becomes unconscious due to Severe diabetic hypoglycaemia, Hypoglycaemia and Hyperglycaemia hyperosmolar state[26,27,28].
Chronic complication of diabetes mellitus such as Microangiopathy. Diabetic nephropathy is kidney damage caused by diabetes that can progress to chronic kidney disease and necessitate renal dialysis. Adult renal failure is most commonly caused by diabetes. Diabetic neuropathy is characterised by aberrant and diminished sensation, which manifests itself in a 'glove and stocking' pattern. Mononeuritis or autonomic neuropathy is another type of diabetic neuropathy. Diabetic amyotrophy is a type of muscle weakening caused by neuropathy in diabetics. Diabetic retinopathy and Diabetic cardiomyopathy alsocause by high blood glucose level[29,30]. Macrovascular disease leads to cardiovascular disease,which is exacerbated by accelerated atherosclerosis.

VII. MANAGEMENT OF DIABETES

There are several modern approaches involved in the management of diabetes. How ever, early diagnosis is central to achieving any targets set in DM management[31]. The first step of conventional therapy is diet and exercise; anti hyperglycemic agents .They are distinguished into various classes, either as monotherapy or, more frequently, in combination with one another:

7.1 Oral Antidiabetic Drugs (OADs)

• **Sulfonylureas** –Tolbutamide, Glipizide, Glimepride, Gliclazide, Glyburide;
• **Biguanides** – Metformin;
• **Meglitinides** – Repaglinide, Nateglinide;
• **Alpha-Glucosidase Inhibitors** – Acarbose,Voglibose,Miglitol;
• **DPP-4 Inhibitors** – Sitsgliptin, Linagliptin,Saxagliptin, Viidagliptin,Alogliptin;
• **GLP-1 Agonists** - Exinatide, Semaglutide, Liraglutide, Albiglutide;
• **SGLT2 Inhibitors** –Canagliflozin, Dapagliflozin, Empagliflozin;
• **Thiazolidinediones (TZDs)** – Pioglitazone, Rosiglitazone;

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7.1.1 Metformin – first-line treatment

Metformin is also notori ous for causing lactic acidosis, especially in patients with kidney disease, liver injury, or other CVS complications that create a low level of oxygen in circulation[32]. Metformin is First-line therapy that decreases hepatic glucose production and improves insulin sensitivity. The combination of SGTL2 inhibitors with metformin may have proved beneficial in curbing hyperglycemia that cannot be con trolled by metformin alone[33]. Metformin Reduces hepatic gluconeogenesis; increases insulin sensitivity, promotes glucose uptakebyphosphorylatingGLUT-enhancerfactor,increases fatty acid oxidation, and lowers glucose absorption from the gastrointestinal system. When compared to sulfonylureas, it has a low rate of hypoglycaemia[34]. It is First-line therapy for type 2 diabetesFirst-line therapy for type 2 diabetes.

7.1.2 Sulfonylureas

Age related reduced renal function, concurrent use of insulin or insulin sensitizers, age above 60 years, recent hospital discharge, alcohol misuse, calorie restriction, multiple drugs, or medications that amplify sulfonylurea effects are all risk factors for hypoglycaemia[35]. Use of long acting sulfonylurea such as glyburide should be avoided in elderly patients with DM and use of short-acting glipizide should be preferred[36].

7.1.3 Meglitinide

Repaglinide and nateglinide are non-sulfonylurea secretagogues which act on the ATP-dependent K-channel in the pancreatic beta cells thereby stimulating the release of insulin from the beta cells, similar to sulfonylurea, though the binding site is different[37]. meglitinides have a quick onset and a brief duration of action (4-6 hours), they reduce the risk of hypoglycaemia[38]. Repaglinide is mostly processed in the liver and excreted in very small amounts through the kidneys, dose adjustments are not required in patients with renal insufficiency, with the exception of those with end-stage renal disease[37].

7.1.4 Thiazolidinediones

Thiazolidinedione is an insulin sensitizer that binds to the peroxisome proliferator activated γ transcription factor[38]. Rosiglitazone and pioglita-zone are used as monotherapy, or in combination with sulfonylurea's, metformin, and insulin. Common side effect is weight gain, especially when the drug is combined with a sulfonylurea or with insulin. The dosage of rosiglitazone is 4-8 mg daily and of pioglitazone 15-45 mg daily. The Thiazolidinediones should not be given to patients if ALT is 2.5 times greater than the upper limit of normal, and liver function tests should be performed once every 2 months for the first year and periodically thereafter[39].

7.1.5 Alpha-Glucosidase Inhibitors

Acarbose, Voglibose and Miglitol have not widely been used to treat type 2 DM individuals but are likely to be safe and effective. Voglibose, which is the newest of the drugs, has been shown in a study to significantly improve glucose tolerance, in terms of delayed disease progression and in the number of patients who achieved normoglycemia[37,40].

7.2 . Insulin Therapy

Insulin remains essential for patients with T1DM and is often required in advanced T2DM. Types of insulin include:

- **Rapid-acting** (e.g., insulin lispro, aspart)
- **Short-acting** (regular insulin)
- **Intermediate-acting (NPH)**
- **Long-acting** (glargine, detemir, degludec)

Insulin is used alone or in combination with oral hypogly caemic medications to control blood sugar levels. If some β-cell function remains, augmentation therapy with basal insulin can help. Insulin is available in four different types of injectables: rapid acting, short acting, intermediate acting, and long acting. When comparedtoshort-actingversions,long-actingformsareless likely to cause hypoglycaemia[41].

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VIII. NEW STRATEGIES FOR TREATING DIABETES MELLITUS

Nanotechnology to treat diabetes mellitus

Recent advances in diabetes research have been leveraged by nanotechnology to develop cutting-edge glucose measurement and insulin delivery techniques with the potential to significantly enhance the well-being of diabetes patients. . This analysis delves into the intersection of nanotechnology and diabetes research, specifically focusing on the developmental of glucose sensors utilizing nanoscale elements like metal nanoparticles and carbon nanostructures[42]. These emerging therapeutic approaches and novel pharmacotherapies are expanding the options for diabetes management, offering more effective, convenient, and personalized care for people living with diabetes[43,44].

The use of nanoparticles for treating diabetes mellitus

The nanoparticle targeted drug delivery approach has enormous benefits which include the improved bioavailabil ity of drugs by targeting specific tissues, organs, and tumors thereby providing the highest dose of drug directly at the targeted site. One of the biggest technological challenges is the scalability of a nanoparticle[45]. Nanoparticles, which are materials with sizes smaller than 100 nm in at least one dimension, have distinct characteristics that change when scaled down to the nanoscale. This enables them to interact with cellular biomolecules in a specific manner. NPs engineered for precise cell delivery carry therapeutic substances [45].

Zinc oxide NPs, . Magnesium NPs, Cerium oxide NPs, . Copper NPs and Selenium NPs are the nanoparticle used to treate diabetes mellitus.

Stem cell Therapy

By altering culture conditions, embryonic stem cells (ESCs) can be transformed into insulin-producing cells. In the laboratory setting, mouse ESCs can be induced to form embryoid bodies through in-vitro differentiation. The selection of nestin-expressing ESCs, these cells were encouraged to mature into a phenotype similar to β-cells[46]. Adding phosphoinositide kinase inhibitors enhanced the differentiation of more ESCs into efficient beta cells[47].

Research into stem cell therapy focuses on regenerating the insulin-producing beta cells of the pancreas. In stem cell therapy involve pancreatic islet transplantation process. This process cause Procedure related risks, immunogenicity[48]. In contrast to type 1 diabetes, which is caused by autoimmune destruction of pancreatic β cells, type 2 diabetes results from irregularities in β cells function together with insulin resistance in peripheral organs[49]. Pluripotent stem (iPS) cells can be generated from type 1 diabetic patients by reprogramming their adult fibroblasts with three transcription factors (OCT4, SOX2, and KLF4). The cells known as diabetes induced pluripotent stem cells; (DiPS) are pluripotent andhave the ability to differentiate into insulin producing cells. Thisisbeneficialintype1disease modeling and cell replacement therapies[50].

Statin techniques

Statins are defined as inhibitors of 3-hydroxy-3-methylglu taryl coenzyme A and inhibit the crucial process of LDL cholesterol in liver, thereby decreasing its level in the blood besides increasing healthy blood vessel lining[51]. Statins work by inhibiting the enzyme HMG-CoA reductase, which is necessary for the liver to make LDL cholesterol. By blocking this enzyme, statins lower LDL cholesterol levels in the blood and improve the health of the blood vessel lining. Because diabetes increases the risk of cardiovascular issues, statins are a crucial treatment option for reducing this risk in individuals with type 2 diabetes[52] .

A recent informa tion published at the meeting of the European association for the study of diabetes in Stockholm suggests that statin treatment is being less explored and applied in patients with type2 diabetes among a large American group of over 100,000 subject[53]. Despite exhibiting good toleration and less adverse effects, statins may cause side effects like myopathies and increase in levels of liver enzymes in type 2 diabetes [54].

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Gene therapy

Gene therapy is a technique that involves remedying the symptoms of an ailment orchestrated by a defective gene via the incorporation of the exogenous normal gene. Its advantage is that a single treatment can be used to cure any type of disease and currently, gene therapy is opening up novel treatment options in different branches of medicine[55].

Gene therapy can also be explained as a method of introduction of a gene or gene manipulation within a cell as a curative regimen in the treatment of disease[56]. T1DM is a widespread epidemic impacting many patients worldwide. Genetic vaccination holds promise for T1DM treatment, offering flexibility in managing T-cell responses. DNA vaccination strategies include plasmid DNA and viral-vector-based vaccinations, both showing positive results in preventing or reversing Type 1 Diabetes Mellitus[57].

Herbal therapy

Herbal remedies are becoming more popular for their ability to reduce blood sugar levels in diabetic individuals with fewer side effects and longer-term use.The many traditional herbal medicines are used to treat the various diseases which are included in the Indian traditional system called the Ayurveda.The many traditional herbal medicines are used to treat the various diseases which are included in the Indian traditional system called the Ayurveda[58]. There are various Indian traditional drugs used in the treatment of the diabetes mellitus are found in Ayurveda. Our Vedic literature like charak Samhita, sushrut Samhita, ashtanghridayam, ashtangsangraham already report the use of plants to treat diabetes. More than 400 plants are incorporated in Vedic literature with various recipes to treat diabetes[59].

In India, Glucobeet from Syzygium cumini has shown promise in treating diabetes. Research indicates that active compounds like glycosides, polysaccharides, and flavonoids play a significant role in regulating blood sugar levels. In Iran, herbal antidiabetic products like B-Glocorex and Galega tablets are available, showing positive results in managing type 2 diabetes[60].

IX. FUTURE PERSPECTIVES

Inhalable insulin, a fast-acting treatment, has received food and drug administration approval for type 1 and 2 diabetes, providing an alternative to injection. It has the advantage of delivery directly into the lungs. Studies have however shown that inhaled insulin is as effective as, but not better than short acting insulin[61].

Personalized medicine approaches are focusing on tailoring treatments based on individual genetic and metabolic profiles, while novel pharmacotherapies and advanced insulin delivery systems aim to improve treatment efficacy and convenience. Additionally, research into metabolic surgery and the development of new lifestyle and behavioural interventions continues to refine strategies for managing diabetes.

In addition to augmenting insulin secretion from islet beta-cells, both in vivo and in vitro studies have produced evidence that GLP-1 action improves islet beta-cell survi val and proliferation[62,63]. Studies have found evidence that GLP-1 exerts multiple beneficial extrapancreatic actions on both tissues positive and negative for GLP-1R expression[62].

Another area of drug research includes designing and use of mucoadhesive microcapsules of various drugs like glipizide to achieve controlled release of the drug and its effective targeting. Mucoadhesion has been a novel approach in drug delivery designing because it causes the slow release of the drug at the action or absorption site thereby enhancing the interaction of the drug with the underlying tissue forms, enhancing the bioavailability of the drugs[64].

X. CONCLUSION

A variety of drugs are being developed for the treatment of type 1 and type 2 diabetes. All of these medications appear to help with glycemic control, but it's unclear whether they'll affect the course of the disease or the micro- and macrovascular implications of uncontrolled diabetes. The management of diabetes mellitus is rapidly evolving, with significant advancements in pharmacological treatments, technology, and personalized care approaches. DMhas become a public clinical challenge that requires urgent attention and the increasing trend in its cases is suggested to continue for more decades. Currently, there is no permanent cure for DM. Many treatment regimens have shown promising results in DMmanagement.

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Future drug therapy of T1DM will depend on the success of ongoing and planned intervention trials. Immunomodulation alone, or possibly combined with immunosuppressive therapy, seems to be promising in reducing the loss of C - peptides after diagnosis.The future of diabetes care lies in a synergistic model that combines modern biomedical technology, individualized patient care, and community-based support systems.To effectively tackle this disease, it is important to focus on public policies that improve access to healthcare, prioritize patientcentered care, and create environments that promote overall health.

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