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REVIEW ARTICLE

A Review on Diabetes Pathophysiology, Pharmacological Treatments, and Lifestyle Interventions

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Abstract: Diabetes mellitus, a condition marked by impaired glucose regulation, encompasses various subtypes, with type 1 and type 2 being the most prevalent. The global incidence of diabetes is rapidly increasing, with projections reaching 552 million by 2030. Insulin resistance, a compromised physiological response of target tissues to insulin stimulation, plays a crucial role in the pathogenesis of diabetes and its associated metabolic abnormalities. Pharmacological interventions, including insulin and hypoglycemic agents, aim to increase insulin secretion, enhance glucose uptake, and reduce gluconeogenesis. However, managing diabetes extends beyond glycemic control, as comorbid mental health conditions, such as dementia, eating disorders, anxiety disorders, and depression, can worsen the progression of diabetes. While no specific pharmacological agent has been approved for treating insulin resistance, various drug classes and lifestyle modifications have shown effectiveness in improving insulin sensitivity. A comprehensive approach combining pharmacotherapy and lifestyle changes is essential for optimal treatment outcomes and reducing the risk of diabetes-related complications. Systematic analysis of the complex mechanisms underlying insulin resistance will facilitate the identification of novel therapeutic targets and improve the management of the closely related metabolic syndrome.

Keywords: Diabetes mellitus; Insulin resistance; Pharmacotherapy; Lifestyle interventions; Glycemic control

1. Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both [1]. It is one of the oldest diseases known to humankind, with descriptions dating back to the 14th century [2]. The absence or reduced production of insulin leads to elevated blood glucose levels, which, if left untreated, can cause permanent damage, dysfunction, and failure of various organs, particularly the eyes, kidneys, nerves, heart, and blood vessels [3].

The global burden of diabetes has reached pandemic proportions, with the International Diabetes Federation (IDF) estimating a worldwide prevalence of 366 million in 2011, projected to rise to 552 million by 2030 [4]. This increasing prevalence is attributed to factors such as population growth, aging, urbanization, obesity, and sedentary lifestyles [5,6]. In addition to the rising incidence, improved medical care has increased the life expectancy of individuals with diabetes, further contributing to the overall prevalence [7]. Diabetes is classified into several subtypes, with type 1 and type 2 being the most common. Type 1 diabetes (T1D) is an autoimmune disorder that leads to the destruction of pancreatic beta cells, resulting in insulin deficiency [8]. Environmental factors and genetic susceptibility play a significant role in the development of T1D, particularly during childhood and adolescence [9]. Without medical intervention, the lack of insulin production leads to metabolic derangements, ketoacidosis, starvation, and death [10]. The incidence of T1D has been increasing worldwide since the 1950s, with an average annual increase of 3-4% over three decades [11].

Type 2 diabetes (T2D), on the other hand, is characterized by insulin resistance and relative insulin deficiency [12]. It is the most prevalent form of diabetes, accounting for more than 90% of all cases [13]. The pathogenesis of T2D involves a progressive decline in insulin secretion by pancreatic beta cells, often in the setting of pre-existing insulin resistance in skeletal muscle, liver, and adipose tissue [14]. T2D is strongly associated with obesity, physical inactivity, and unhealthy dietary habits [15].

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Gestational diabetes mellitus (GDM) is another form of diabetes that occurs during pregnancy. Hormonal changes during pregnancy can lead to insulin resistance, reducing the body's ability to respond to insulin [16]. GDM is associated with significant maternal and fetal morbidity, including macrosomia, neonatal hypoglycemia, and an increased risk of delivery complications [17]. The diagnosis of diabetes is based on glycemic thresholds associated with microvascular complications, particularly retinopathy [18]. The American Diabetes Association (ADA) recommends using either a 2-hour oral glucose tolerance test (OGTT) or glycated hemoglobin (HbA1c) for the diagnosis of diabetes [19]. Prediabetes, a condition that increases an individual's risk of developing diabetes and its complications, is defined by impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or an HbA1c level between 5.7% and 6.4% [20].

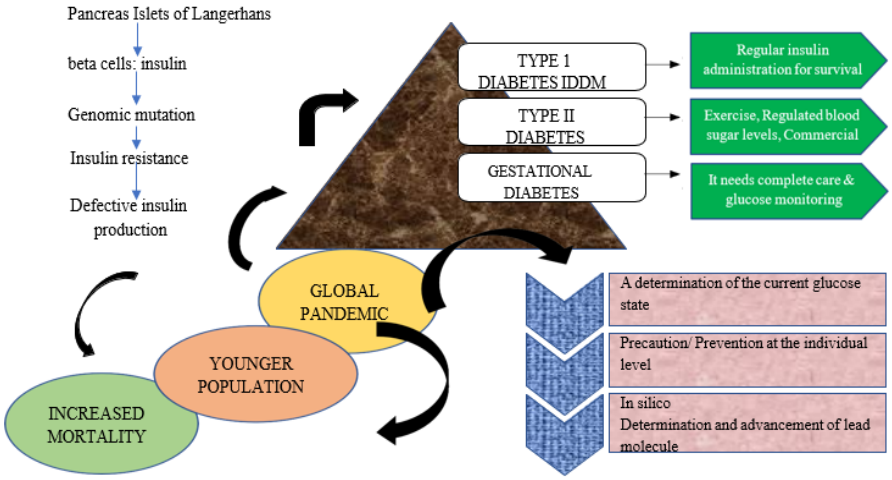

Figure 1. Overview Presentation of Diabetes

Insulin resistance is a central feature in the pathogenesis of diabetes, particularly in T2D [21]. It is characterized by an impaired physiological response of target tissues, such as the liver, muscle, and adipose tissue, to insulin stimulation [22]. Insulin resistance leads to decreased glucose disposal, resulting in compensatory hyperinsulinemia and increased beta-cell insulin production [23]. The metabolic consequences of insulin resistance include hyperglycemia, hypertension, dyslipidemia, visceral obesity, hyperuricemia, increased inflammatory markers, endothelial dysfunction, and a prothrombotic state [24]. Despite advances in medical care, diabetes remains a leading cause of premature mortality, primarily due to associated cardiovascular disease (CVD) [25]. Effective glucose management can reduce the incidence of microvascular complications, such as retinopathy, nephropathy, and neuropathy, but the impact on macrovascular complications, including ischemic heart disease, cerebrovascular disease, and peripheral arteriopathy, appears to be less pronounced [26,27]. The management of diabetes involves a multifaceted approach, including pharmacological interventions, lifestyle modifications, and patient education [28]. Insulin and hypoglycemic agents are the mainstays of pharmacotherapy for diabetes, aiming to increase insulin secretion, enhance glucose uptake, and reduce gluconeogenesis [29]. However, the presence of comorbid mental health conditions, such as dementia, eating disorders, anxiety disorders, and depression, can complicate the management of diabetes and worsen its progression [30]. While no specific pharmacological agent has been approved for the treatment of insulin resistance, various drug classes and lifestyle interventions have shown effectiveness in improving insulin sensitivity [31]. Biguanides (e.g., metformin), thiazolidinediones, and incretin-based therapies have demonstrated beneficial effects on insulin resistance [32]. Lifestyle modifications, including regular physical activity, dietary changes, and weight management, play a crucial role in enhancing insulin sensitivity and overall glycemic control [33].

2. Types of Diabetes Mellitus

2.1. Type I Diabetes

Type 1 diabetes (T1D) is an autoimmune disorder that leads to the destruction of pancreatic beta cells, resulting in insulin deficiency [2]. The development of T1D is influenced by both genetic susceptibility and environmental factors, particularly those encountered during childhood and adolescence [3]. Without medical intervention, the lack of insulin production leads to metabolic derangements, ketoacidosis, starvation, and death [4].

The incidence of T1D has been increasing worldwide since the 1950s, with an average annual increase of 3-4% over three decades [5]. Although T1D accounts for only 5% of all diabetes cases, it remains the most common form of diabetes in children, with an

estimated 100,000 new cases diagnosed annually [6]. Despite advancements in insulin therapy, individuals with T1D continue to face a significant risk of severe complications, including cardiovascular mortality [7,8].

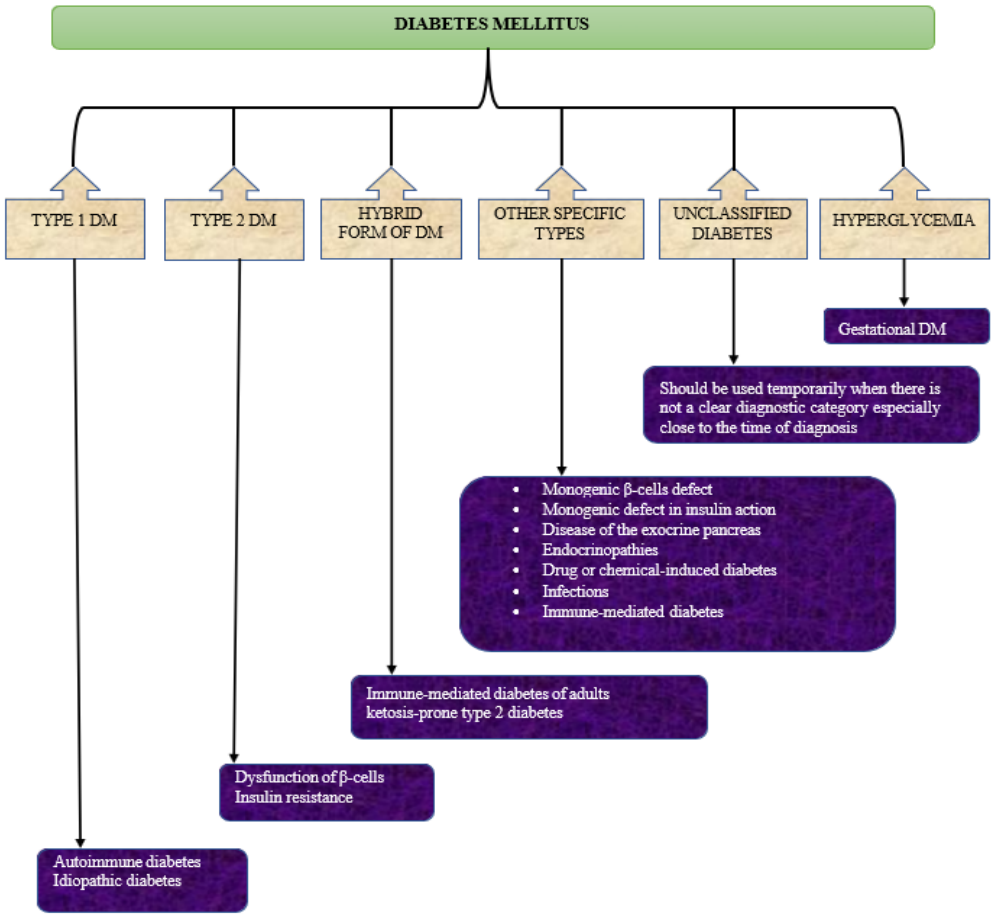

Figure 2. Types of Diabetes Mellitus

2.2. Type II Diabetes

Type 2 diabetes (T2D) is characterized by persistent hyperglycemia or elevated blood glucose levels following a meal containing carbohydrates [9]. In contrast to T1D, which is defined by insulin deficiency, most individuals with T2D have elevated insulin levels (fasting and/or post-glucose ingestion) unless there has been significant beta-cell loss [10,11].

T2D is the most prevalent form of diabetes, accounting for more than 90% of all cases [12]. The pathogenesis of T2D involves a progressive decline in insulin secretion by pancreatic beta cells, often in the setting of pre-existing insulin resistance in skeletal muscle, liver, and adipose tissue [13]. The development of T2D is strongly associated with modifiable risk factors, such as obesity, physical inactivity, and unhealthy dietary habits [14]. Treatment of T2D typically involves oral hypoglycemic agents and lifestyle modifications. Both insulin resistance and decreased insulin production contribute to the progression of the disease [15].

2.3. Gestational Diabetes

Gestational diabetes mellitus (GDM) is a form of diabetes that occurs during pregnancy. Hormonal changes during pregnancy can lead to insulin resistance, reducing the body's ability to respond to insulin [16]. Women who develop diabetes during pregnancy and those who are diagnosed with unrecognized, asymptomatic type 2 diabetes during pregnancy are both classified as having GDM [17]. GDM is associated with significant maternal and fetal morbidity, including fetal macrosomia, neonatal hypoglycemia, and increased risk of delivery complications such as birth trauma, shoulder dystocia, and cesarean delivery [18,19]. Women with GDM are also at an increased risk of developing type 2 diabetes later in life, with 35-60% of women diagnosed with T2D within 20 years after delivery [20,21].

2.4. Other Specific Types

In addition to type 1, type 2, and gestational diabetes, there are other specific types of diabetes that arise from various causes [22]. These include:

- Monogenic beta-cell defects
- Monogenic defects in insulin action
- Diseases of the exocrine pancreas
- Endocrinopathies
- Drug- or chemical-induced diabetes
- Infections
- Immune-mediated diabetes.

2.5. Unclassified Diabetes

Some forms of diabetes do not fit into the typical categories and are classified as "unclassified diabetes" [23]. This term is used temporarily when there is no clear diagnostic category, especially close to the time of diagnosis. Examples include immune-mediated diabetes of adults and ketosis-prone type 2 diabetes.

3. Epidemiology

Diabetes mellitus has become a global health concern, with its prevalence increasing at an alarming rate. According to the International Diabetes Federation (IDF), the worldwide prevalence of diabetes among adults (aged 20-79 years) was estimated to be 463 million in 2019, projected to rise to 700 million by 2045 [1]. The global diabetes prevalence has nearly doubled since 1980, rising from 4.7% to 8.5% in the adult population [2]. The increasing prevalence of diabetes is attributed to various factors, including population growth, aging, urbanization, and the increasing prevalence of obesity and physical inactivity [3]. The rapid rise in the incidence of type 2 diabetes, which accounts for the majority of diabetes cases, is particularly concerning [4].

Diabetes prevalence varies across regions and income groups. Low- and middle-income countries bear a disproportionate burden of diabetes, with approximately 80% of all diabetes cases occurring in these countries [5]. The Western Pacific and Southeast Asia regions have the highest number of individuals with diabetes, accounting for approximately half of the global diabetes population [6]. In addition to the increasing prevalence, diabetes also contributes significantly to morbidity and mortality worldwide. In 2019, diabetes was the ninth leading cause of death globally, with an estimated 4.2 million deaths attributed to the disease [7]. Diabetes and its complications have a substantial economic impact on individuals, families, healthcare systems, and countries [8].

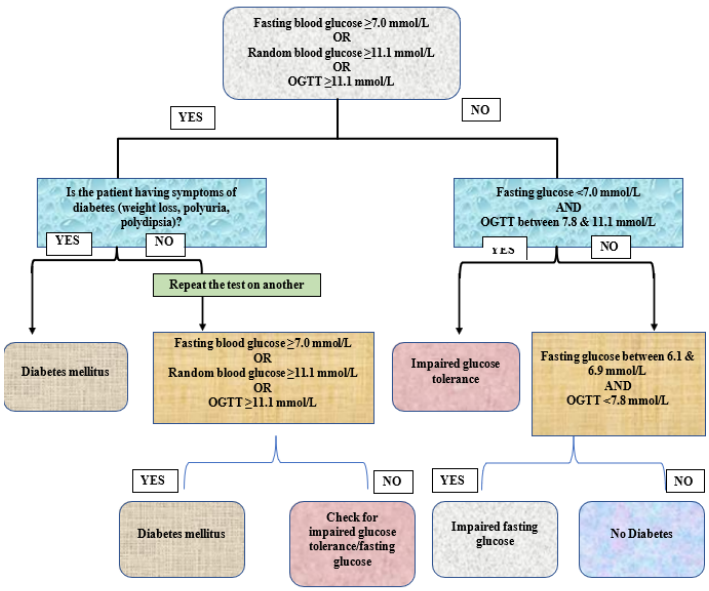

Figure 3. Diagnostic algorithm for diabetes

4. Diabetic Complications

Diabetes is associated with a wide range of complications that can affect multiple organ systems. These complications are broadly classified into microvascular and macrovascular complications [9].

4.1. Microvascular Complications

Microvascular complications of diabetes include retinopathy, nephropathy, and neuropathy [10].

4.1.1. Diabetic Retinopathy

Diabetic retinopathy is a leading cause of blindness in adults. It occurs when high blood glucose levels damage the small blood vessels in the retina, leading to vision loss [11]. Regular eye examinations and early intervention can prevent or delay the progression of diabetic retinopathy [12].

4.1.2. Diabetic Nephropathy

Diabetic nephropathy is a common cause of end-stage renal disease (ESRD) [13]. It develops as a result of damage to the small blood vessels in the kidneys, leading to the loss of protein in the urine and a decline in kidney function [14]. Early detection and management of diabetic nephropathy can slow the progression to ESRD [15].

4.1.3. Diabetic Neuropathy

Diabetic neuropathy is a common complication that affects the nerves throughout the body. It can manifest as peripheral neuropathy, causing pain, numbness, and weakness in the extremities, or as autonomic neuropathy, affecting various organ systems [16]. Diabetic neuropathy increases the risk of foot ulcers, amputations, and cardiovascular complications [17].

4.2. Macrovascular Complications

Macrovascular complications of diabetes include cardiovascular disease, cerebrovascular disease, and peripheral artery disease [18].

4.2.1. Cardiovascular Disease

Individuals with diabetes have a two to four-fold increased risk of developing cardiovascular disease compared to those without diabetes [19]. Cardiovascular complications, such as coronary artery disease, myocardial infarction, and heart failure, are the leading causes of morbidity and mortality in people with diabetes [20].

4.2.2. Cerebrovascular Disease

Diabetes increases the risk of stroke, with individuals with diabetes having a two to four-fold higher risk compared to those without diabetes [21]. Stroke prevention strategies, including blood pressure control, lipid management, and antiplatelet therapy, are essential in reducing the risk of cerebrovascular complications [22].

4.2.3. Peripheral Artery Disease

Peripheral artery disease (PAD) is more common in individuals with diabetes, and its presence is associated with an increased risk of cardiovascular events and lower-extremity amputations [23]. Early detection and management of PAD, including lifestyle modifications and pharmacological interventions, can improve outcomes in people with diabetes [24].

4.3. Other Complications

Diabetes is also associated with various other complications, including:

4.3.1. Diabetic Foot

Diabetic foot complications, such as foot ulcers and Charcot foot, are common and can lead to lower-extremity amputations [25]. Regular foot examinations, proper foot care, and patient education are essential in preventing diabetic foot complications [26].

4.3.2. Dental Complications

Individuals with diabetes have an increased risk of periodontal disease and other dental complications [27]. Regular dental check-ups and maintaining good oral hygiene are important for preventing dental complications in people with diabetes [28].

4.3.3. Genitourinary Complications

Diabetes can lead to genitourinary complications, such as urinary tract infections, sexual dysfunction, and bladder dysfunction [29]. Screening and management of these complications are essential for improving the quality of life of individuals with diabetes [30].

The prevention and management of diabetic complications require a comprehensive approach that includes optimal glycemic control, blood pressure and lipid management, regular screening, and early intervention [31]. Patient education and self-management support are also crucial in reducing the risk of complications and improving outcomes in individuals with diabetes [32].

5. Pathophysiology

The pathophysiology of diabetes mellitus involves a complex interplay of genetic, environmental, and metabolic factors that lead to impaired glucose homeostasis [1]. The two main pathophysiological processes in diabetes are insulin resistance and beta-cell dysfunction [2].

5.1.1. Insulin Resistance

Insulin resistance is a central feature in the pathogenesis of type 2 diabetes [3]. It is characterized by an impaired biological response to insulin in target tissues, such as the liver, skeletal muscle, and adipose tissue [4]. In insulin-resistant states, higher insulin concentrations are required to achieve normal glucose uptake and utilization [5].

The mechanisms underlying insulin resistance are multifaceted and involve various cellular and molecular pathways [6]. These include impaired insulin signaling, reduced glucose transporter translocation, increased inflammatory cytokines, and altered lipid metabolism [7]. Obesity, particularly visceral adiposity, is a major contributor to insulin resistance [8].

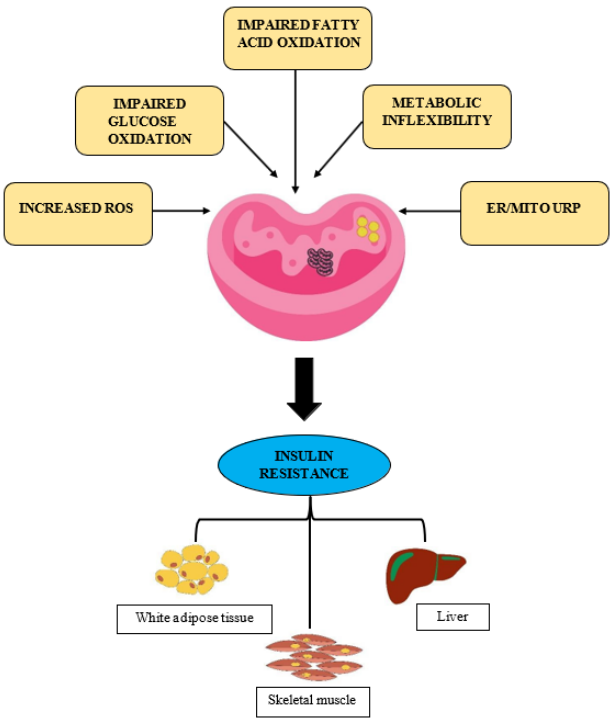

Figure 4. Pathophysiology of Diabetes Mellitus

5.1.2. Beta-cell Dysfunction

Beta-cell dysfunction is another key pathophysiological feature of diabetes [9]. In type 1 diabetes, autoimmune destruction of pancreatic beta cells leads to absolute insulin deficiency [10]. In type 2 diabetes, beta-cell dysfunction is characterized by impaired insulin secretion and a progressive decline in beta-cell mass [11]. The exact mechanisms of beta-cell dysfunction in type 2 diabetes are not fully understood but involve a combination of genetic susceptibility, glucotoxicity, lipotoxicity, and amyloid deposition [12]. Chronic exposure to hyperglycemia and elevated free fatty acids can lead to beta-cell apoptosis and reduced insulin secretory capacity [13].

5.1.3. Glucose Toxicity and Oxidative Stress

Chronic hyperglycemia in diabetes can lead to glucose toxicity and oxidative stress [14]. Excess glucose can cause the formation of advanced glycation end products (AGEs), which contribute to the development of diabetic complications [15]. Hyperglycemia also increases the production of reactive oxygen species (ROS), leading to oxidative stress and cellular damage [16].

5.1.4. Inflammation

Diabetes is associated with a state of chronic low-grade inflammation [17]. Inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), are elevated in individuals with diabetes and contribute to insulin resistance and beta-cell dysfunction [18]. Inflammation also plays a role in the development of diabetic complications, such as cardiovascular disease and nephropathy [19]

6. Pharmacological Treatment

The pharmacological treatment of diabetes aims to achieve and maintain optimal glycemic control, prevent or delay the development of complications, and improve overall quality of life [20]. The choice of pharmacological agents depends on the type of diabetes, individual patient characteristics, and the presence of comorbidities [21].

6.1. Insulin Therapy

Insulin therapy is the mainstay of treatment for type 1 diabetes and is also used in the management of type 2 diabetes when oral hypoglycemic agents fail to achieve adequate glycemic control [22]. Insulin preparations vary in their onset, peak, and duration of action, allowing for individualized treatment regimens [23].

Insulin analogs, such as rapid-acting and long-acting insulins, have been developed to more closely mimic physiological insulin secretion [24]. Insulin delivery methods include subcutaneous injections, insulin pens, and continuous subcutaneous insulin infusion (CSII) pumps [25].

6.2. Oral Hypoglycemic Agents

Oral hypoglycemic agents are the first-line pharmacological treatment for type 2 diabetes [26]. These agents work through various mechanisms to improve glycemic control, including enhancing insulin secretion, improving insulin sensitivity, reducing hepatic glucose production, and increasing glucose uptake in peripheral tissues [27].

6.2.1. Biguanides (e.g., metformin)

Metformin is the most widely used oral hypoglycemic agent and is considered the first-line treatment for type 2 diabetes [28]. It works by reducing hepatic glucose production, increasing peripheral glucose uptake, and improving insulin sensitivity [29].

6.2.2. Sulfonylureas (e.g., glimepiride, glyburide)

Sulfonylureas stimulate insulin secretion from pancreatic beta cells and are effective in lowering blood glucose levels [30]. However, they are associated with a risk of hypoglycemia and weight gain [31].

6.2.3. Thiazolidinediones (e.g., pioglitazone)

Thiazolidinediones improve insulin sensitivity in peripheral tissues and reduce hepatic glucose production [32]. They are associated with weight gain and an increased risk of heart failure [33].

6.2.4. Dipeptidyl peptidase-4 (DPP-4) inhibitors (e.g., sitagliptin, linagliptin)

DPP-4 inhibitors enhance the activity of incretin hormones, leading to increased insulin secretion and reduced glucagon secretion [34]. They have a lower risk of hypoglycemia compared to sulfonylureas [35].

6.2.5. Glucagon-like peptide-1 (GLP-1) receptor agonists (e.g., liraglutide, exenatide)

GLP-1 receptor agonists stimulate insulin secretion, suppress glucagon secretion, delay gastric emptying, and promote satiety [36]. They are associated with weight loss and a low risk of hypoglycemia [37].

6.2.6. Sodium-glucose cotransporter-2 (SGLT-2) inhibitors (e.g., empagliflozin, canagliflozin)

SGLT-2 inhibitors reduce renal glucose reabsorption, leading to increased urinary glucose excretion and lowered blood glucose levels [38]. They are associated with weight loss and a low risk of hypoglycemia [39].

Other pharmacological agents used in the treatment of diabetes include alpha-glucosidase inhibitors, meglitinides, and amylin analogs [40]. The choice of pharmacological treatment should be individualized based on patient characteristics, glycemic targets, and the presence of comorbidities [41].

Table 1. Comparison of pharmacological agents used in the treatment of type 2 diabetes [22-24]

Class	Examples	Mechanism of Action	Advantages	Disadvantages
Biguanides	Metformin	↓ Hepatic glucose production, ↑ Insulin sensitivity	Weight neutral, Low risk of hypoglycemia	Gastrointestinal side effects
Sulfonylureas	Glimepiride, Glyburide	↑ Insulin secretion	Effective glucose lowering	Risk of hypoglycemia, Weight gain
Thiazolidinediones	Pioglitazone	↑ Insulin sensitivity	Effective glucose lowering	Weight gain, Risk of heart failure
DPP-4 inhibitors	Sitagliptin, Linagliptin	↑ Insulin secretion, ↓ Glucagon secretion	Low risk of hypoglycemia	Modest glucose lowering
GLP-1 receptor agonists	Liraglutide, Exenatide	↑ Insulin secretion, ↓ Glucagon secretion, ↑ Satiety	Weight loss, Low risk of hypoglycemia	Gastrointestinal side effects, Injectable
SGLT-2 inhibitors	Empagliflozin, Canagliflozin	↑ Urinary glucose excretion	Weight loss, Low risk of hypoglycemia, CV benefits	Genital mycotic infections, Risk of ketoacidosis

6.3. Combination Therapy

In many cases, a combination of oral hypoglycemic agents or the addition of insulin to oral therapy may be necessary to achieve optimal glycemic control [42]. Combination therapy targets multiple pathophysiological mechanisms and can provide additive or synergistic effects in lowering blood glucose levels [43-45]

7. Lifestyle Interventions

Lifestyle interventions, including dietary modifications, physical activity, and weight management, are cornerstone strategies in the prevention and management of diabetes [1]. These interventions aim to improve insulin sensitivity, reduce cardiovascular risk factors, and promote overall health and well-being [2].

7.1. Dietary Modifications

Dietary modifications are a key component of diabetes management [3]. The primary goals of dietary interventions are to achieve and maintain optimal glycemic control, reduce cardiovascular risk factors, and promote healthy eating habits [4].

The American Diabetes Association (ADA) recommends an individualized approach to medical nutrition therapy (MNT) based on personal and cultural preferences, health literacy, and access to healthful food choices [5]. The ADA does not endorse a single dietary pattern but emphasizes the importance of nutrient-dense foods, such as vegetables, fruits, whole grains, legumes, and lean proteins [6].

Low-carbohydrate diets, such as the Mediterranean diet and the Dietary Approaches to Stop Hypertension (DASH) diet, have been shown to improve glycemic control and reduce cardiovascular risk factors in individuals with diabetes [7,8]. These diets focus on the consumption of whole, minimally processed foods and limit the intake of refined carbohydrates and added sugars [9].

7.2. Physical Activity

Regular physical activity is an essential component of diabetes management [10]. Exercise improves insulin sensitivity, reduces cardiovascular risk factors, and promotes weight loss [11]. The ADA recommends that adults with diabetes engage in at least 150 minutes of moderate-intensity aerobic activity or 75 minutes of vigorous-intensity aerobic activity per week, spread over at least three days [12].

In addition to aerobic exercise, resistance training has been shown to improve glycemic control, increase muscle strength, and reduce the risk of sarcopenia in individuals with diabetes [13]. The ADA recommends that adults with diabetes engage in 2-3 sessions of resistance training per week, targeting all major muscle groups [14].

Table 2. Lifestyle interventions for the prevention and management of diabetes [25-27]

Intervention	Recommendations	Benefits
Dietary Modifications	- Individualized medical nutrition therapy - Nutrient-dense, whole foods - Low-carbohydrate diets (e.g., Mediterranean, DASH)	- Improved glycemic control - Reduced cardiovascular risk factors - Promotion of healthy eating habits
Physical Activity	- ≥150 min/week moderate-intensity aerobic activity - ≥75 min/week vigorous-intensity aerobic activity - 2-3 sessions/week resistance training	- Improved insulin sensitivity - Reduced cardiovascular risk factors - Increased muscle strength - Reduced risk of sarcopenia
Weight Management	- 5-10% weight loss for overweight/obese individuals - Lifestyle interventions (diet, physical activity) - Pharmacological interventions (e.g., GLP-1 agonists, SGLT-2 inhibitors) - Bariatric surgery for severe obesity and type 2 diabetes	- Improved glycemic control - Reduced cardiovascular risk factors - Improved quality of life - Remission of type 2 diabetes in some cases

7.3. Weight Management

Weight management is a critical component of diabetes prevention and treatment, particularly for individuals with type 2 diabetes [15]. Obesity is a major risk factor for the development of insulin resistance and type 2 diabetes [16]. Weight loss of 5-10% of initial body weight has been shown to improve glycemic control, reduce cardiovascular risk factors, and improve quality of life in individuals with diabetes [17].

Lifestyle interventions, including dietary modifications and increased physical activity, are the primary strategies for achieving and maintaining a healthy weight [18]. In some cases, pharmacological interventions, such as glucagon-like peptide-1 (GLP-1) receptor agonists and sodium-glucose cotransporter-2 (SGLT-2) inhibitors, may be used to promote weight loss in individuals with diabetes [19]. Bariatric surgery may be considered for individuals with severe obesity (BMI ≥ 40 kg/m² or BMI ≥ 35 kg/m² with comorbidities) and type 2 diabetes who have not achieved adequate glycemic control with lifestyle interventions and pharmacological treatment [20]. Bariatric surgery has been shown to result in significant and sustained weight loss, improve glycemic control, and lead to the remission of type 2 diabetes in some cases [21]

8. Conclusion

Diabetes mellitus is a complex metabolic disorder with a growing global prevalence. Understanding the different types of diabetes, their epidemiology, pathophysiology, and associated complications is crucial for effective prevention and management strategies. Lifestyle interventions, including dietary modifications, physical activity, and weight management, along with personalized pharmacological treatment, are essential for achieving optimal glycemic control and reducing the risk of complications. Ongoing research efforts continue to provide insights into novel therapeutic approaches and strategies for improving the lives of individuals with diabetes.

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