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# Diabetes mellitus: Understanding the Etiology, Types, Symptoms, Complication, Diagnosis, Treatment and Prevention Strategies

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**ABSTRACT**

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia, resulting from defects in insulin secretion, insulin action, or both. Diabetes mellitus is a leading global health issue affecting approximately 422 million people worldwide. The condition is categorized primarily into Type 1 and Type 2 diabetes, each with distinct etiological and pathophysiological mechanisms. Type 1 diabetes is an autoimmune condition leading to absolute insulin deficiency, whereas Type 2 diabetes involves insulin resistance and often associated with obesity, lifestyle factors and genetic predisposition. Additionally, gestational diabetes and other forms of diabetes contribute to the global burden. Diabetes is associated with severe complications, including cardiovascular disease, kidney failure, and nerve damage, leading to significant morbidity and mortality rates worldwide. This article explores the classification, etiology, complications, diagnosis and treatment approaches for prevention.

**Keywords:** Type 1 DM, Type 2 DM, Gestational DM, Insulin, Blood Glucose, IDDM, NDDM.

## INTRODUCTION

Diabetes mellitus (DM) is one of the most important and largest epidemics public health challenges of the twenty-first century in worldwide. Diabetes mellitus, more simply called diabetes, is a chronic condition that occurs when there are raised levels of glucose in the blood because the body cannot produce any or enough of the hormone insulin or use insulin effectively. It is a metabolic disorder of multiple etiologies distinguished by a failure of glucose homeostasis with disturbances of carbohydrate, fat and protein metabolism as a result of defects in insulin secretion and/or insulin action. Diabetes mellitus as a disease, for example a constellation of symptoms, but not its pathogenesis, has been known by physicians for nearly 3,500 years in ancient Egypt. The term "diabetes" was first coined by Araetus of Cappodocia (81-133AD). Later, the word mellitus (honey sweet) was added by Thomas Willis (Britain) in 1675 after rediscovering the sweetness of urine and blood of patients (first noticed by the ancient Indians). It was only in 1776 that Dobson (Britain) firstly confirmed the presence of excess sugar in urine and blood as a cause of their sweetness. In modern time, the history of diabetes coincided with the emergence of experimental medicine. An important milestone in the history of diabetes is the establishment of the role of the liver in glycogenesis, and the concept that diabetes is due to excess glucose production by Claude Bernard (France) in 1857. The role of the pancreas in pathogenesis of diabetes was discovered by Mering and Minkowski (Austria) in 1889. Later, this discovery constituted the basis of insulin isolation and clinical use by Banting and Best (Canada) in 1921. Trials to prepare an orally administrated hypoglycemic agent ended successfully by first marketing of tolbutamide and carbutamide in 1955[1,2].

Diabetes mellitus (DM) is the most common endocrine disorder and usually occurs when there is deficiency or absence of insulin or rarely, impairment of insulin activity (insulin resistance). The International Diabetes Federation (IDF) estimates the total number of diabetic subjects to be around 40.9 million in India and this is further set to rise to 69.9 million by the year 2025[3,4,5].

Insulin and glucagon hormones both are secreted by the pancreas. Insulin is secreted by the beta (ß) cells and glucagon is secreted by the alpha (α) cells both are located in the islets of Langerhan's. Insulin decreases the blood glucose level by the glycogenesis and transport glucose into the muscles, liver and adipose tissue. Neural tissue and erythrocytes do not required insulin for glucose utilization whereas alpha (α) cells plays an important role in controlling blood glucose by producing the glucagon and it increases the blood glucose level by accelerating the glycogenolysis[6,7].

In addition to increased risk of obesity, metabolic and cardiovascular disorders, and malignancy in future life of fetus after delivery[8]. Type II diabetes mellitus comprises 80% to 90% of all cases of diabetes mellitus. Geographical variation can contribute in the magnitude of the problems and to overall morbidity and mortality[9,10]. Moreover, people with diabetes who undertake moderate amounts of physical activity are at inappreciably lower risk of death than inactive persons[11] It is now well established that a specific genetic constitution is required for such an event to cause[12] The growing burden of diabetes and other noncommunicable diseases is one of the major health challenges to economic developments bedevilling WHO African Region states[13].

## CLASSIFICATION OF DIABETES

The first mostly accepted classification of diabetes mellitus was published by WHO in the year 1980 and, it is modified in the year 1985. The classification encompasses both clinical stages and aetiological types of diabetes mellitus and other categories of hyperglycemia[14,15]. The old and new terms of insulin-dependent (IDDM) or noninsulin- dependent (NIDDM) which were proposed by WHO in1980 and 1985 have disappeared and the terms of new classification system identifies four types of diabetes mellitus:

**1. Type 1 diabetes**

**2. Type 2 diabetes**

**3. Gestational Diabetes Mellitus**

**4. Specific types of diabetes due to other causes**

These were reflected in the subsequent International Nomenclature of Diseases (IND) in 1991and the 10th revision of the International Classification of Diseases (ICD-10) in 1992 [16].

### Insulin Dependent Diabetes Mellitus (IDDM)

This type of diabetes mellitus is also called autoimmune diabetes and previously known as juvenile-onset or ketosis prone diabetes. The individual may also seek with other autoimmune disorders such as Graves' disease, Hashimoto's thyroiditis, and Addison's disease[17]. Type I diabetes mellitus is also known as insulin- dependent diabetes mellitus (IDDM), this occurs mainly in children and young adults; the onset is usually sudden and can be life threaten in [6]. T1DM is thought originating from an immune-mediated disorder, selectively attacking and making destruction of pancreatic β cells following inflammatory infiltration of the islets of Langerhans, pathologic process which was defined as insulitis. Insulitis is pathophysiologically an inflammatory state of the pancreas islets, important parts of the pancreas that are directly responsible for maintaining blood glucose homeostasis[18]. Indeed, approximately 40% of T1DM cases globally occur in people are older than 30 years of age. T1DM results from an absolute deficiency in insulin caused by the failure of secretion by the pancreas, which may be present at the time diabetes is diagnosed[19]. Type 1 is usually characterized by the presence of anti–glutamic acid decarboxylase, islet cell or insulin antibodies which identify the autoimmune processes which leads to beta-cell destruction[20]. The rate of destruction of beta cell is quite variable; it can be occur rapidly in some individuals and slow in others[21]. There is a severe deficiency or absence of insulin secretion due to destruction of ß-islets cells of the pancreas. Treatment with injections of insulin is required[6]. Markers of immune destruction, including islet cell auto-antibodies, and/or auto antibodies to insulin, and auto antibodies to glutamic acid decarboxylase (GAD) are present in 85-90 % of individuals with Type 1 diabetes mellitus when fasting diabetic hyperglycemia is initially detected[22]. The exact cause of diabetes mellitus is remain unknown, although, in most people, there is evidence of an autoimmune mechanism involving auto-antibodies that destroy the beta islet cells[6].

### Noninsulin Dependent Diabetes Mellitus (NDDM)

Type 2 diabetes mellitus is also known as adult-onset diabetes. A key component of type 2 diabetes (T2D) pathogenesis is defective insulin secretion[23] . Among the different forms of diabetes, Type-2 diabetes mellitus (T2DM) accounts for approximately 90% of cases and current estimates indicate that by 2040, approximately 642 million world-wide people will be living with type 2 diabetes. It is a chronic metabolic disorder that is becoming a leading cause of morbidity and mortality[24]. T2DM results from a combination of insufficient insulin secretion from the pancreatic islets and insulin resistance of target cells. Pancreatic beta-cell mass is reduced by 50% in individuals with T2DM compared with non-diabetic subjects[25]. A hypercaloric diet combined with a sedentary lifestyle is a major risk factor for the development of insulin resistance. Standard treatment for T2DM begins with lifestyle modification, and includes oral medications and insulin therapy to compensate for progressive β-cell failure[26]. Although T2DM is historically identified as a condition of increased plasma glucose levels owing to inadequate insulin action, today it is known that along with insulin and glucose a large number of metabolites, hormones, growth factors, neurotransmitters, neuropeptides, cytokines,

behaviours and neuronal signals are up or down-regulated in this disorder. Whether alterations in these signals are causes or consequences of altered insulin signalling and hyperglycemia is not clearly known [27]. Moreover, recent research has also discovered the role of T cells in the development of insulin resistance and various complications in T2DM, including atherosclerosis, nephropathy and neuropathy.

**Gestational Diabetes Mellitus (GDM)**

The glucose intolerance occurring for the first time or diagnosed during pregnancy is referred to as gestational diabetes mellitus (GDM)[4]. Women who develop Type1 diabetes mellitus during pregnancy and women with undiagnosed asymptomatic Type 2 diabetes mellitus that is discovered during pregnancy are classified with Gestational Diabetes Mellitus (GDM)[28]. Gestational Diabetes Mellitus (GDM) is defined as "diabetes diagnosed in the second or third trimester of pregnancy that was not clearly overt diabetes prior to gestation". GDM is one of the most frequent metabolic diseases during pregnancy. The overall incidence of GDM is increasing worldwide and approximately affects 7% (range: 2–18%) of all pregnancies[29]. GDM is associated with considerable risks to both the mother and developing fetus. For the mother, these include a greater likelihood of undergoing a caesarean section, pre-eclampsia and the development of type 2 diabetes mellitus (T2DM); for the baby, macrosomia, shoulder dystocia, and physiological and metabolic abnormalities such as neonatal hypoglycaemia, and obesity with insulin resistance in young adulthood.

**OTHER SPECIFIC TYPES OF DIABETES**

**Genetic defects of the beta-cell**

**Maturity-onset diabetes of the young (MODY):** This type of diabetes is associated with abnormal monogenetic in β-cell function. It typically appears at a young age, usually before 25 years old, and is characterized by reduced insulin secretion with little to no abnormalities in insulin action. The condition is inherited in an autosomal dominant manner, meaning that only one copy of the affected gene from either parent is sufficient to cause the condition. Mutations in several genes, including hepatic transcription factor (HNF)-1, glucokinase, HNF-4, HNF-1α, IPF-1, and NeuroD1, have been identified as causes of MODY[30].

**Genetic defects in insulin action**

**Genes mutations of insulin receptors:** Certain gene mutations of insulin receptors can lead to abnormalities in insulin action. these mutations associated with insulin can cause a variety of metabolic abnormalities, ranging from elevated insulin levels and mild high blood sugar to severe diabetes. In some cases, individuals with these mutations may display additional symptoms such as acanthosis nigricans (skin darkening), virilization (development of male characteristics), and enlarged cystic ovaries in women. Despite the presence of mutant insulin, the impaired binding of these molecules to the insulin receptor can lead to either mild or even normal glucose metabolism[31].

**Diseases of the exocrine pancreas**

Diabetes can arise from several conditions that lead to widespread damage to the pancreas. Such conditions include infection, pancreatitis, pancreatic carcinoma, trauma, and pancreatectomy (surgical removal of the pancreas). Typically, significant damage to the pancreas is necessary for diabetes to develop, but even small, affected portions of the pancreas due to adenocarcinomas can be linked to diabetes. Additionally, certain diseases like cystic fibrosis, hemochromatosis, and fibrocalculous pancreatopathy can also disrupt insulin secretion and harm β-cells[32]. Cystic fibrosis–related diabetes (CFRD) is the most common comorbidity in people with cystic fibrosis, occurring in about 20% of adolescents and 40–50% of adults. Diabetes in this population, compared with individuals with type 1 or type 2 diabetes, is associated with worse nutritional status, more severe inflammatory lung disease, and greater mortality. Insulin insufficiency is the primary defect in CFRD. Genetically determined b-cell function and insulin resistance associated with infection and inflammation may also contribute to the development of CFRD. Milder abnormalities of glucose tolerance are even more common and occur at earlier ages than CFRD[34].

**Endocrinopathies**

Excess amounts of certain hormones can antagonize insulin action and lead to diabetes. Conditions such as acromegaly that is (excess growth hormone), glucagonoma that is (excess glucagon) pheochromocytoma that is (excess epinephrine), and Cushing's syndrome that is (excess cortisol) can cause diabetes, indeed, diabetes can be exacerbated, especially in individuals who have preexisting defects in secretion of insulin. Conditions like somatostatinomas and aldosteronomas can induce hypokalemia, which further contributes to the development and progression of diabetes in affected individuals[31].

**Drug or chemical-induced diabetes**

The only drug which commonly cause diabetes during therapeutic use are the antihypertensive vasodilator diazoxide, and corticosteroids in high doses such as those used to palliate intracranial tumours. Thiazide diuretics have in the past been used in higher doses than necessary to treat hypertension, and the lower doses now used probably carry only a slight risk of inducing diabetes. The risk from beta-blockers is also quite small, but there is some evidence that thiazides combined with beta-blockers may be more likely to cause diabetes than either drug alone. The combination is probably best avoided in patients with a family history of non-insulin-dependent diabetes. The effect of the low-oestrogen combined oral contraceptive pill seems to be slight, and it presents a risk only to women who have had gestational diabetes. Body builders who take enormous doses of anabolic-androgens can develop impaired glucose tolerance. Several drugs, including theophylline, aspirin, isoniazid and nalidixic acid can cause transient hyperglycaemia in overdosage, but only streptozotocin, alloxan and the rodenticide Vacor and intravenous pentamidine are likely to cause permanent diabetes. However, it's important to note that drug-induced reactions leading to diabetes are relatively rare occurrences[30,35].

**Infections**

Some viruses have been linked to the destruction of beta-cells and the emergence of diabetes. These viruses include coxsackievirus B, congenital rubella, mumps, adenovirus, and cytomegalovirus. Their role in inducing diabetes is especially notable in individuals who have genetic predispositions or markers associated with type 1 diabetes. In susceptible individuals, these viruses can trigger an autoimmune response, leading to the destruction of beta-cells and the development of diabetes[32].

**Uncommon Forms Of Immune-Mediated Diabetes**

**Stiff-man syndrome**

This is an autoimmune condition affecting the central nervous system, which is characterized by painful spasms and stiffness in the axial muscles. Individuals with this syndrome typically show elevated levels of glutamic acid decarboxylase (GAD) autoantibodies and are at an elevated risk of developing diabetes[31].

**Anti-insulin receptor antibody-related diabetes**

Antibodies against the insulin receptor can interfere with insulin binding, leading to diabetes. In certain instances, these antibodies can function as insulin agonists, leading to hypoglycemia instead of hyperglycemia. Individuals diagnosed with systemic lupus erythematosus and other autoimmune disorders may show the presence of antibodies that target the insulin receptor. Acanthosis nigricans are often present in individuals with this condition[33].

**Common symptoms of diabetes mellitus**

- Increased thirst and urination (polyuria and polydipsia): This occurs as the body tries to flush out excess glucose.
- Increased hunger (polyphagia): The body's cells are not getting enough glucose for energy, leading to increased appetite.
- Fatigue: Due to the body's inability to use glucose effectively.
- Blurred vision: High glucose levels can affect the shape of the lens in the eye.
- Slow-healing sores or frequent infections: High blood glucose can impair the immune response.
- Unintended weight loss: Often associated with Type 1 diabetes, this happens when the body breaks down fat and muscle for energy due to a lack of insulin[36,37].

In diabetes mellitus, cells fails to metabolized glucose in the normal manner, effectively become starved. The long term effect of diabetes mellitus which includes progressive development of the specific complications of retinopathy with potential blindness, nephropathy that may lead to renal failure, and neuropathy with risk of foot ulcer, Charcot joint and features of autonomic dysfunctions and sexual dysfunction People with diabetes are at increases risk of diseases[4,11].

**Etiology**

The etiology (cause) of diabetes mellitus involves a combination of genetic, environmental, and lifestyle factors. The two main types of diabetes-Type 1 and Type 2-have different etiological mechanisms.

**1. Type 1 Diabetes Mellitus (T1DM):**

**Autoimmune Response:** Type 1 diabetes is primarily caused by an autoimmune reaction where the body's immune system attacks and destroys the insulin-producing beta cells in the pancreas. This leads to absolute insulin deficiency.

**Genetic Factors:** A genetic predisposition plays a role, with certain genes (like those in the HLA region) increasing the risk. However, a family history is less commonly associated with Type 1 diabetes than in Type 2. Environmental Factors: Environmental triggers, such as viral infections (e.g., enteroviruses), may initiate the autoimmune attack in genetically predisposed individuals[38].

**2. Type 2 Diabetes Mellitus (T2DM):**

**Insulin Resistance:** Type 2 diabetes is characterized by insulin resistance, where the body's cells do not respond effectively to insulin. Over time, the pancreas compensates by producing more insulin, but eventually, it fails to keep up with the demand.

**Beta Cell Dysfunction:** As insulin resistance persists, the beta cells in the pancreas begin to function poorly and may eventually stop producing adequate amounts of insulin.

**Genetic Factors:** Type 2 diabetes has a strong genetic component. People with a family history of diabetes are at higher risk. Specific genes related to glucose metabolism, insulin secretion, and fat metabolism may contribute[39].

**Environmental and Lifestyle Factors:**

**Obesity:** Excess fat, especially visceral fat, is a major risk factor for Type 2 diabetes. Fat cells can release substances that impair insulin sensitivity.

**Physical Inactivity:** Lack of exercise is a significant contributing factor.

**Poor Diet:** Diets high in processed foods, sugars, and unhealthy fats can increase the risk.

**Age and Ethnicity:** The risk of Type 2 diabetes increases with age, and certain ethnic groups (e.g., African American, Hispanic, Native American) are at higher risk.

**3. Gestational Diabetes Mellitus (GDM):**

**Hormonal Changes in Pregnancy:** During pregnancy, hormonal changes can lead to insulin resistance. The body may not produce enough insulin to compensate for this resistance, leading to elevated blood glucose levels.

Obesity and family history are also risk factors for gestational diabetes.

**4. Other Forms of Diabetes:**

**Monogenic Diabetes:** Caused by mutations in a single gene affecting insulin production.

**Secondary Diabetes:** Can result from conditions that affect the pancreas (such as pancreatitis) or from the use of certain medications (e.g., corticosteroids)[40,41].

**COMPLICATION OF DIABETES MELLITUS**

Diabetes can affect many different organ systems in the body and, over time, can lead to serious complications. Diabetes complications can be divided into

1. Acute complications

2. Chronic complications

**Acute complications** include hypoglycaemia, diabetic ketoacidosis (DKA), hyperglycaemic hyperosmolar state (HHS), hyperglycaemic diabetic coma, seizures or loss of consciousness and infections.

**Chronic Complications** from diabetes can be classified as microvascular or macrovascular. There is growing evidence that the underlying mechanisms in the pathogenesis of diabetic complications include certain genetic and epigenetic modifications, nutritional factors, and sedentary lifestyle. In addition, diabetes has also been associated with increased rates of cancer, physical and cognitive disability, tuberculosis and depression.

**Microvascular Complications**

Diabetic nephropathy, neuropathy, and retinopathy are the main microvascular complications induced by chronic hyperglycemia via several mechanisms such as the production of advanced glycation end products (AGEs), the creation of a proinflammatory microenvironment, and the induction of oxidative stress.

**Macrovascular Complications**

Macrovascular complications include cardiovascular disease, stroke, and peripheral vascular disease. Peripheral vascular disease may lead to bruises or injuries that do not heal gangrene and ultimately amputation. Coronary artery disease (CAD) leading to angina or myocardial infarction, peripheral artery disease (PAD) contributing to stroke, diabetic encephalopathy and diabetic foot. Atherosclerosis is more common in people with DM than in those without. For example, DM increases the risk for stroke in people aged 20 to 65 years more than 5 times.

**Miscellaneous Complications**

Diabetic cardiomyopathy is a specific complication that develops independently of coronary artery disease or hypertension and it is possible to lead to increased morbidity and mortality[42,43].

**DIAGNOSIS OF DIABETES MELLITUS**

**Table: 1 Diagnosis of diabetes by OGTT**

| PATIENT STATUS | PLASMA GLUCOSE VALUE | DIAGNOSIS |
|---|---|---|
| 1.Fasting value | Below 110 mg/dl (< 6.1 mmol/L) | Normal fasting value |
| 2.Fasting value | 110-126 mg/dl (6.1- 7.0mmol/L) | Impaired fasting glucose(IPG)** |
| 3.Fasting value | 126 mg/dl (7.0mmol/L) | Diabetes mellitus |
| 4.Two-hour after 75g oral glucose load | 140-200mg/dl (7.8-11.1mmol/L) | Impaired fasting glucose(IPG)** |
| 5.Two-hour after 75g oral glucose load | 200mg/dl (11.1mmol/L) or more | Diabetes mellitus |
| 6.Random value | 200mg/dl (11.1mmol/L) or more | Diabetes mellitus |

**Tests for diagnosis of diabetes:**

**1.Urine testing:**

Urine test are cheap and convenient but the diagnosis of diabetes cannot be based on urine testing alone since there may be false-positive and false-negative. Urine is tested for the presence of glucose and ketones.

**a.Glucosuria:** Benedict's qualitative test detects any reducing substance in the urine and is not specific for glucose. More sensitive and glucose specific test is dipstick method based on enzyme-coated paper strip which turn purple when dipped in urine containing glucose.

**b.Ketonuria:** Test for ketone bodies in the urine are required for assecing the severity of diabetes and not for diagnosis of diabetes. However, if both glucosuria and ketonuria are present, diagnosis of diabetes is almost certain. Rotheras test and strip test are conveniently perform for detection of ketonuria.

**2.Single blood sugar estimation:**

For diagnosis of diabetes, blood sugar determinations are absolutely necessary. Folin-Wu method pf measurement of all reducing substances in the blood including glucose is now absolute. Currently used are O-toluidine, somogyi-Nelson and glucose oxidase method. Whole blood or plasma may be used but whole blood values are 15% lower than plasma values.

**3. Oral glucose tolerance test:**

The patient who is scheduled for oral GTT is instructed to eat a high carbohydrate diet foe atleast 3 days prior to the test and come after an overnight fast on the day of the test (for atleast 8 hr). A fasting blood sugar sample is first drawn then 75 gm of glucose dissolved in 300 ml of water is given. Blood and urine specimen are collected at half hourly intervals for atleast 2 hr. Blood or plasma glucose content is measured and urine is tested for glucosuria to determine the approximate renal threshold for glucose. Venous whole blood concentration are 15% lower than plasma glucose value[44].

**Treatment of Diabetes Mellitus**

The treatment is to overcome the precipitating cause and to give high doses of regular insulin. The insulin requirement comes back to normal once the condition has been controlled the aims of management of diabetes mellitus can be achieved by:

1.To restore the disturbed metabolism of the diabetic as nearly to normal as is consistent with comfort and safety.

2.To prevent or delay progression of the short and long term hazards of the disease.

3.To provide the patient with knowledge, motivation and means to undertake this own enlightened care[45].

**A. Types of Therapy Involved In Diabetes Mellitus**

**1. Stem cell therapy**

Researchers have shown that monocytes/ macrophages may be main players which contribute to these chronic inflammations and insulin resistance in T2DM patients[28]. Stem cell educator therapy, a novel technology, is designed to control or reverse immune dysfunctions. The procedure includes: collection of patient's blood circulating through a closed-loop system, purification of lymphocytes from the whole blood, co-culture of them with adherent cord blood-derived multi-potent stem cells (CB-SCs) *in vitro* and administration of the educated lymphocytes (but not the CB-SCs) to the patient's circulation[46,47].

**2. Antioxidant therapy**

A variety of antioxidants, such as vitamins, supplements, plant-derived active substances and drugs with antioxidant effects, have been used for oxidative stress treatment in T2DM patients. Vitamin C, vitamin E and β-carotene are ideal supplements against oxidative stress and its complications. Antioxidant which play an important role in lowering the risk of developing diabetes and its complications[48].

**3. Anti-inflammatory treatment**

The changes indicate that inflammation plays a pivotal role in the pathogenesis of T2DM and its complications[49,50] . In T2DM, especially in adipose tissue, pancreatic islets, the liver, the vasculature and circulating leukocytes, which include altered levels of specific cytokines and chemokines, the number and activation state of different leukocyte populations, increased apoptosis and tissue fibrosis. Immunomodulatory drugs are provided[51,52].

**B. Dietary Management**

Adequate caloric value Dietary management should be taken properly by the both diabetic and non-diabetic patient such as:

1. Balanced in regard to protein, carbohydrate and fats, in all cases it is necessary to restrict carbohydrate intake.

2. Should conform as closely as possible to normal.

3. Food intake should be divided into regularly spaced meals of similar size.

4. Reduce total calorie intake by decreasing both fat and carbohydrate.

5. Patient must be advised to be constant in his dietary habits from day to day.

**C. Newer Insulin Delivery Devices**

A number of innovations have been made to improve ease and accuracy of insulin administration as well as to achieve tight glycaemia control. These are insulin syringes, pen devices, inhaled insulin, insulin pumps, implantable pumps, other routes of insulin delivery.

**D. Oral Hypoglycaemic or Antidiabetic Agents**

Clinically useful biguanide phenformin was produced parallel to sulfonylurea's in 1957. Newer approaches have constantly been explored and have lately yielded thiazolidinediones, meglitinide analogues, α-glucosidase inhibitors, and the latest are dipeptidyl peptidase-4(DPP-4) inhibitors.

**Important Features of Oral Hypoglycaemic Agents**

Diabetes mellitus can be considered a disease of the modern world with a great impact of morbidity, morality and the quality of type of the affected individual. Diabetes mellitus is a frequent complication of cushing syndrome which is caused by chronic exposure to Glucocorticoids by several clinical symptoms such as central obesity, proximal muscles weakness, hirsutism and neurophysiological disturbance, macro-vascular complication autonomic neuropathy, digestive problems, dental problems etc.[11].

**Conclusion**

Diabetes mellitus remains a growing global health challenge affecting hundreds of millions of people. The World Health Organization reports that approximately 422 million people worldwide are living with diabetes, with the majority residing in low and middle income countries. This marks substantial increase in prevalence over the past few decades, largely due to rising diabetes linked to lifestyle factors such as obesity, physical inactivity and poor dietary habits. Additionally, around 1.5 million deaths annually are directly attributed to diabetes. The increase in diabetes prevalence underscores the urgent need for widespread public health interventions. Preventive measures and early detection are critical to managing the disease and reducing the associated complications and mortality.

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