INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: https://www.ijpsjournal.com

OPEN ACCESS

Review Article

Type 2 Diabetes Mellitus

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ARTICLE INFO

Published: 31 May 2026
Keywords:
Type 2 Diabetes Mellitus (T2DM), Insulin Resistance, Hyperglycaemia, Antidiabetic Therapy, Glycaemic Control
DOI:
10.5281/zenodo.20472427

ABSTRACT

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder characterized by hyperglycaemia resulting from impaired insulin secretion, insulin resistance, or both. It is associated with serious long-term complications affecting the eyes, kidneys, nerves, heart, and blood vessels. The prevalence of diabetes is increasing rapidly worldwide, particularly in India, making it a major public health concern. The pathophysiology of T2DM involves β-cell dysfunction and insulin resistance, leading to impaired glucose metabolism and progressive disease development. Common symptoms include polyuria, polydipsia, polyphagia, weight loss, fatigue, blurred vision, and delayed wound healing. Early screening and diagnosis using fasting plasma glucose (FPG), oral glucose tolerance test (OGTT), random plasma glucose, and HbA1c testing are essential for preventing complications and improving disease management. Management of T2DM includes lifestyle modification, regular exercise, diet control, and pharmacological therapy. Metformin remains the first-line drug because of its ability to reduce hepatic glucose production and improve insulin sensitivity. Other commonly used antidiabetic agents include sulfonylureas, thiazolidinediones, DPP-4 inhibitors, GLP-1 receptor agonists, SGLT2 inhibitors, alpha-glucosidase inhibitors, meglitinides, bile acid sequestrants, and insulin therapy. Insulin remains a cornerstone in diabetes management, especially in patients with severe hyperglycaemia or inadequate response to oral medications. Recent advances in diabetes treatment include newer therapies such as dual incretin receptor agonists, AMPK activators, FGF21 analogs, SGLT1/2 inhibitors, and gene-editing approaches, which aim to provide improved glycaemic control and better metabolic outcomes. Exercise therapy, including regular physical activity and micro-exercise, also plays an important role in improving insulin sensitivity, glucose utilization, and overall quality of life. Overall, effective management of T2DM requires a comprehensive approach combining lifestyle changes, pharmacological treatment, and early intervention to reduce complications and improve patient outcomes.

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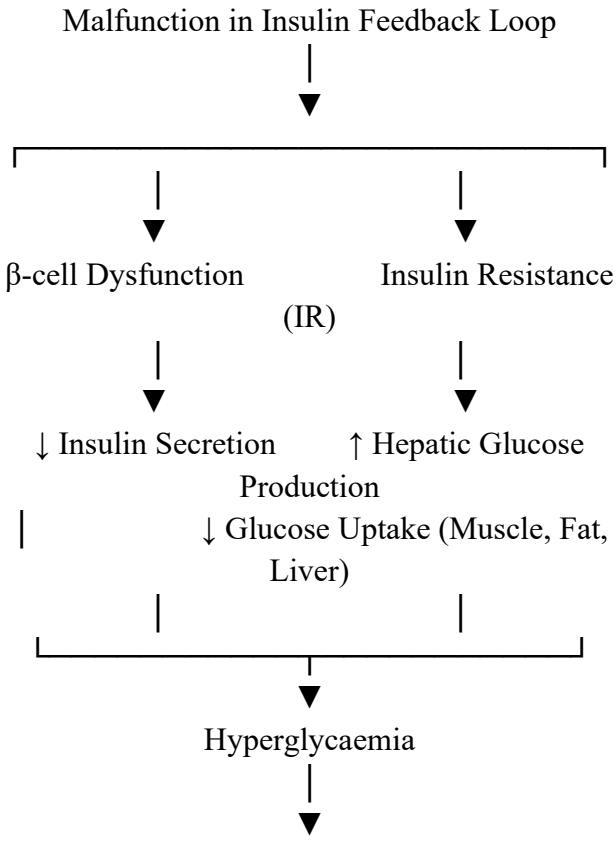
Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

INTRODUCTION

Diabetes mellitus is a metabolic disorder characterized by defects in insulin secretion, insulin action, or both, resulting in chronic hyperglycemia. Over time, this condition can lead to long-term damage, dysfunction, and failure of various organs, particularly the eyes, kidneys, nerves, heart, and blood vessels.(1) Diabetes develops due to multiple pathological mechanisms, including insulin resistance and autoimmune destruction of pancreatic β-cells. These defects impair the metabolism of carbohydrates, fats, and proteins due to inadequate insulin action. Impaired insulin action may result from insufficient insulin secretion, reduced tissue responsiveness, or both. In many individuals, these abnormalities coexist, making it difficult to determine the primary cause of hyperglycemia.(2,3) Diabetes mellitus affects approximately 40.9 million people in India. Insulin and glucagon are hormones secreted by the pancreas. Insulin is produced by the β-cells, while glucagon is produced by the α-cells; both types of cells are located in the islets of Langerhans.Insulin lowers blood glucose levels by promoting glycogenesis (the conversion of glucose to glycogen) and facilitating the transport of glucose into muscle, liver, and adipose tissue. However, certain tissues such as neural tissue and erythrocytes do not require insulin for glucose uptake.On the other hand, α-cells help regulate blood glucose by secreting glucagon, which increases blood glucose levels by stimulating glycogenolysis (the breakdown of glycogen into glucose).Type 2 diabetes mellitus accounts for about 80% to 90% of all diabetes cases. It is associated with an increased risk of obesity, metabolic disorders, cardiovascular diseases, and even malignancies later in life. Additionally, it may pose risks to the fetus during pregnancy and after birth.(4,5)

PATHOPHYSIOLOGY



Progression of Type 2 Diabetes Mellitus (T2DM)

- The disease (T2DM) develops due to a malfunction in feedback loops between insulin secretion and insulin action.
- This malfunction leads to high blood glucose levels (hyperglycaemia).

β-cell dysfunction:

- Causes reduced insulin secretion.
- Limits the body's ability to maintain normal glucose levels.

Insulin resistance (IR):

- Increases glucose production in the liver.
- Decreases glucose uptake in muscle, liver, and adipose tissue.

Combined effects:

- Both β-cell dysfunction and IR occur early in disease development.
- β-cell dysfunction is usually more severe than insulin resistance.
- When both are present, they amplify hyperglycaemia, accelerating the progression of Type 2 Diabetes Mellitus (T2DM).(6,7)

SOME COMMON SIGN AND SYMPTOMS

Diabetes mellitus is a metabolic disorder in which cells fail to properly metabolize glucose, leading to cellular starvation despite the presence of glucose in the blood. This impaired glucose utilization results in a range of symptoms and complications. In the early stages, individuals commonly experience symptoms such as weight loss, polyuria (increased urination), polydipsia (increased thirst), and polyphagia (increased hunger). In addition, other common symptoms include blurred vision, headache, fatigue, slow healing of wounds, itchy skin, and skin rashes. Over time, persistent hyperglycaemia leads to serious long-term complications. These include retinopathy, which can progress to blindness; nephropathy, which may result in renal failure; and neuropathy, which can lead to foot ulcers. Furthermore, diabetes is associated with autoimmune dysfunction and sexual dysfunction, increasing the overall risk of multiple diseases and contributing to progressive deterioration of health if not properly managed.(8,9)

ETIOLOGY OF DIABETES MELLITUS

Etiology is the science of finding causes and origin of arise of disease, it includes:

1. The changes in structure of β-cell of islets of pancreas also cause insulin dependent diabetes.

2. Viruses may also play a role in the etiology of diabetes

3. Thevarious medication side effect and stress condition also produce diabetes

4. The genetic mutation also plays role in the etiology of diabetes is controversial as yet.

CAUSES OF DIABETES MELLITUS

Type 2 diabetes was previously believed to have many different causes, but studies consistently show a strong connection between the disease and overnutrition. The “twin cycle hypothesis” explains that long-term excess calorie intake leads to fat accumulation in the liver and pancreas through two self-reinforcing cycles. This fat buildup gradually impairs insulin function and insulin secretion, ultimately causing type 2 diabetes. The hypothesis also suggested that major weight loss could reverse these changes and restore normal blood glucose levels, allowing researchers to study how the disease develops and how it can be reversed. Over the past 15 years, several studies have confirmed these mechanisms and clarified how environmental and genetic factors interact in the development of diabetes. This understanding has helped create successful national programmes aimed at achieving remission of type 2 diabetes. Overall, current evidence indicates that type 2 diabetes mainly has a common underlying cause related to overnutrition, although it occurs in people with different genetic backgrounds.(10)

SCREENING AND DIAGNOSIS

- Tests for screening and diagnosis of Diabetes Mellitus (DM) are easily available.
- The same tests are used for both screening and diagnosis.
- A positive screening result can indicate:

- Pre-diabetes, or
- Diabetes Mellitus (DM).

- Common tests used include:
- Fasting Plasma Glucose (FPG)
- Oral Glucose Tolerance Test (OGTT)
- Random Plasma Glucose Test
- HbA1c Test (11)

- Early screening helps in the early detection and management of diabetes.
- About 25% of patients with Type 2 Diabetes Mellitus (T2DM) already have microvascular complications at the time of diagnosis.
- These complications may include:
- Eye damage (diabetic retinopathy)
- Kidney damage (diabetic nephropathy)
- Nerve damage (diabetic neuropathy)

- This indicates that T2DM may remain undetected for more than 5 years before diagnosis.
- Type 2 diabetes usually develops gradually with mild or no obvious symptoms.
- Many patients are diagnosed only after complications begin to appear.
- Therefore, regular screening is important for early diagnosis and prevention of complications.(12)

Type 2 Diabetes Mellitus (T2DM) is diagnosed using blood glucose tests or HbA1c values. Diagnosis can be confirmed by any one of the following criteria:

- **Random Plasma Glucose Test:** A random blood glucose level of ≥200 mg/dL in the presence of classic symptoms of hyperglycaemia such as excessive thirst, frequent urination, and unexplained weight loss.

- **Fasting Plasma Glucose (FPG):** A fasting blood glucose level of ≥126 mg/dL after at least 8 hours of fasting.

- **Oral Glucose Tolerance Test (OGTT):** A 2-hour plasma glucose level ≥200 mg/dL after ingestion of 75 g of oral glucose.

- **HbA1c Test:** Glycated haemoglobin (HbA1c) level of ≥ 6.5%.

In most cases, the diagnosis should be confirmed by repeat testing on another day unless the blood glucose level is clearly and unequivocally elevated along with typical symptoms.(13)

- The risk of developing diabetic nephropathy (kidney damage) and distal symmetric peripheral polyneuropathy (nerve damage affecting both sides of the body) increases even at lower levels of hyperglycaemia.
- These glucose levels may be lower than those typically associated with diabetic retinopathy (eye damage).
- This suggests that kidney and nerve complications can begin earlier in diabetes progression, even before noticeable eye complications develop.
- Therefore, early monitoring and control of blood glucose levels are important to prevent diabetic complications.(14)

TREATMENT

The first step of conventional therapy is diet and exercise; antihyperglycemic agents are included (Table 1.). They are distinguished into various

classes, either as monotherapy or, more frequently, in combination with one another:

- **Biguanides** – Metformin;
- **Insulin Secretagogues** - Sulfonylureas, Metglinides;
- **Insulin Sensitizers** - Thiazolidinediones (TZDs);
- **Incretin-Based Therapies** - DPP-4 Inhibitors and GLP-1 Agonists;
- **Renal Glucose Transport Modifiers** - SGLT2 Inhibitors;
- **Carbohydrate Absorption Modifiers** - Alpha-Glucosidase Inhibitors;
- **Other Therapeutic Agents** - Bile Acid Sequestrants;
- **Insulin**

Table 1.(16): Juxtaposition of glucose-lowering agents.

Class of Drug	Drug Examples	Mechanism of Action	Common Adverse Effects
Biguanides	Metformin	Reduce hepatic glucose production and increase insulin sensitivity in peripheral tissues.	Gastrointestinal issues (nausea, diarrhea), Lactic acidosis (rare)
Sulfonylureas	Glimepiride , Glipizide , Glibenclamide (Glyburide)]	Stimulate insulin secretion from pancreatic beta-cells	Hypoglycemia, Weight gain, Gastrointestinal disturbances
Thiazolidinediones (TZDs)	Pioglitazone, Rosiglitazone	Increase insulin sensitivity in peripheral tissues by activating PPAR-gamma receptors.	Weight gain, Edema, Bone fractures, Increased risk of heart failure
Dipeptidyl Peptidase-4 (DPP-4) Inhibitors	Sitagliptin , Saxagliptin , Linagliptin , Alogliptin	Inhibit DPP-4 enzyme, Prolong the action of incretin hormones (GLP-1, GIP), which increase insulin secretion and decrease glucagon release	Nasopharyngitis, Headache, Gastrointestinal issues
Glucagon-Like Peptide-1 (GLP-1) Receptor Agonists	Exenatide , Liraglutide , Dulaglutide, Semaglutide (Ozempic®)	Mimic GLP-1, increasing insulin secretion in response to meals, Inhibit glucagon release, Slow down gastric emptying	Nausea, Vomiting, Diarrhea, Risk of pancreatitis (rare)
Sodium-Glucose Cotransporter 2 (SGLT2) Inhibitors	Canagliflozin, Dapagliflozin, Empagliflozin, Ertugliflozin	Inhibit SGLT2 in the kidneys, Reduce glucose reabsorption, and Increase glucose excretion in urine	Urinary tract infections, Dehydration, Hypotension, Genital fungal infections
Alpha-Glucosidase Inhibitors	Acarbose , Miglitol , Voglibose	Inhibit enzymes in the small intestine that break down carbohydrates and slow glucose absorption.	Flatulence, Diarrhea, Abdominal discomfort

Meglitinides	Repaglinide, Nateglinide, Mitiglinide	Stimulate rapid, short-term insulin secretion from the pancreas, with a quick onset and short duration of action	Hypoglycemia, Weight gain, Gastrointestinal disturbances
Bile Acid Sequestrants	Colestipol (Colestid); Cholestyramine (Locholest, Prevalite, and Questran); Colesevelam (Welchol)	Binds bile acids in the intestines, which may help improve insulin sensitivity, Lower blood glucose	Constipation, Bloating, Gas, Nausea
Insulin	**Rapid-acting insulins** - bolus insulin - Insulin aspart (NovoRapid) Insulin glulisine (Apidra) Insulin lispro (Humalog); **Short-acting insulins** - bolus insulin - Insulin regular (Entuzity, Humulin-R, Novolin ge Toronto); **Intermediate-acting insulins** - basal insulin - Insulin NPH (Humulin-N and Novolin ge NPH); **Long-acting insulins** - basal insulin - Insulin detemir (Levemir) Insulin glargine (Lantus); **Ultra long-acting insulins** - basal insulin - Degludec (Tresiba) Insuline glargine (Toujeo)	Replaces or supplements insulin that is not produced by the pancreas, Promoting glucose uptake into cells	Hypoglycemia, Weight gain, Injection site reactions, Cardiovascular dis

Metformin – first line drugs

- Lifestyle changes such as a healthy diet, regular exercise, and weight control are the first steps in treating Type 2 Diabetes Mellitus (T2DM).
- Along with lifestyle changes, metformin is the most commonly recommended first-line medicine.
- Metformin works in several ways to lower blood sugar levels.

Simple Mechanism of Metformin

- Metformin reduces the amount of glucose (sugar) produced by the liver.
- It activates a protein called AMPK, which helps control energy and sugar balance in the body.
- AMPK blocks a process called gluconeogenesis, through which the liver makes extra glucose.
- As a result, less glucose is released into the blood, especially during fasting.
- Metformin also helps the body respond better to insulin (improves insulin sensitivity).

- It increases the movement of glucose transporters like GLUT4 to the surface of cells.
- This allows more glucose to enter muscle and fat cells, where it is used for energy.
- In addition, metformin decreases the absorption of glucose from the intestine.

Overall Effect

- Decreases blood sugar levels
- Improves insulin action
- Helps control fasting blood glucose
- Supports better management of Type 2 Diabetes Mellitus (T2DM) (17,18)
- Metformin is particularly useful for patients with insulin resistance, where the body does not respond properly to insulin.
- It helps improve the body's sensitivity to insulin and lowers blood glucose levels.
- Studies also suggest that metformin may help reduce cancer-related mortality in people with diabetes.
- This means diabetic patients taking metformin may have a lower risk of death associated with certain cancers.
- Therefore, metformin is widely used as a first-line treatment for Type 2 Diabetes Mellitus (T2DM).(19)

Insulin Therapy- the cornerstone og diabetes management

Insulin is the primary hormone responsible for controlling blood glucose levels and remains one of the most effective treatments for diabetes even after many decades of use. In Type 1 Diabetes Mellitus (T1DM), the body does not produce endogenous insulin, so patients require exogenous insulin therapy for survival. Basal insulin administration is essential for regulating important metabolic processes such as glycogen breakdown, gluconeogenesis, lipolysis, and ketogenesis. In Type 2 Diabetes Mellitus (T2DM), insulin therapy may be required in conditions such as acute illness, surgery, pregnancy, severe hyperglycaemia, or when oral antidiabetic drugs are ineffective or contraindicated. Insulin can be used alone or in combination with oral antidiabetic medications, especially when glycated haemoglobin (HbA1c) levels are ≥7.5%. It becomes particularly important in patients with HbA1c levels ≥10%, especially when diet, exercise, and other antihyperglycaemic treatments fail to adequately control blood glucose levels.(20) nsulins are mainly classified into three groups based on their onset, peak, and duration of action. Fast-acting insulins, such as Insulin Aspart, Insulin Lispro, and Insulin Glulisine, begin to act within 5–15 minutes, reach their peak effect in 1–2 hours, and continue working for about 4–6 hours. These are commonly used to control blood glucose levels around mealtimes. Intermediate-acting insulins, including NPH Human Insulin and Pre-Mixed Insulin, start acting within 1–2 hours, show peak activity after 4–6 hours, and have a duration of action of more than 12 hours. They are often used to provide basal insulin coverage. Long-acting insulins, such as Insulin Glargine and Insulin Detemir, begin working within 1.5–2 hours and provide a relatively constant or flat insulin effect without a pronounced peak, lasting approximately 12–24 hours. These insulins help maintain steady blood glucose control throughout the day and night.(21)

Experimental Diabetes Treatment

- In recent years, many new medicines have been developed for diabetes treatment.

- These medicines help:
  - Lower blood sugar levels
  - Improve insulin action
  - Manage metabolic problems associated with diabetes

New and Emerging Antidiabetic Therapies

- **Activin Type II Receptor Modulators**
  - Example: Bimagrumab
  - Help improve muscle and metabolic function
- **Amylin or Dual Amylin-Calcitonin Receptor Agonists**
  - Example: Pramlintide
  - Help control appetite and blood glucose levels
- **AMPK Activators**
  - Examples: A-769,662, Thienopyridone
  - Improve glucose and energy metabolism
- **FGF21 Analogs**
  - Example: Pegbelfermin
  - Help improve metabolic control and insulin sensitivity
- **Fructose-1,6-Bisphosphatase Inhibitors**
  - Examples: VK0612, MB07803
  - Reduce glucose production in the liver
- **New GLP-1 Receptor Agonists**
  - Examples:
    - Semaglutide
    - Liraglutide
    - Dulaglutide
    - Exenatide
  - Increase insulin secretion
  - Reduce appetite
  - Support weight loss
- **SGLT Inhibitors**
  - Examples:
    - Canagliflozin
    - Dapagliflozin
    - Empagliflozin
  - Remove excess glucose through urine
- **Imeglimin (Glimins)**
  - A newer class of antidiabetic drug
  - Improves energy metabolism and glucose control

Overall Benefits

- Better blood glucose control
- Improved insulin sensitivity

Class of Drug	Drug Examples	Mechanism of Action	Potential Benefits	Side Effects
Dual Incretin Receptor	Tirzepatide (Mounjaro) (24,25)	It combines the GLP-1 receptor agonist and GIP	Significant improvements in glycemic	Gastrointestinal issues (nausea, vomiting),

Agonists (GLP-1/GIP)		(gastric inhibitory polypeptide) receptor agonist actions to enhance insulin secretion, inhibit glucagon release, and promote weight loss.	control and weight reduction.	injection site reactions
GIP Receptor Triagonists	Retatrutide (26)	Stimulates GIP receptors to enhance insulin secretion, reduce glucagon secretion, and improve glucose metabolism.	Potential for better glucose control and weight loss.	Gastrointestinal discomfort, headache, injection site reactions
SGLT1/2 Inhibitors (Dual Inhibition)	Sotagliflozin(27,28)	Dual inhibition of SGLT1 (in the intestines) and SGLT2 (in the kidneys) reduces glucose absorption and enhances renal glucose excretion.	Potential for significant blood glucose lowering and improved weight loss.	Diarrhea, dehydration, hypotension, increased risk of UTIs
Glucagon Receptor Antagonists	Bay 27–9955 [29], LY2409021 30[].	Blocks glucagon receptors, reducing hepatic glucose production and lowering blood sugar levels.	Effective in lowering blood glucose, especially in insulin-resistant patients.	Liver enzyme elevations, gastrointestinal disturbances
GPR119 Agonists	DA-1241(31), ZB-16 (ZB40-0016) [32], JNJ-38431055 [33]	Activates the GPR119 receptor, increasing insulin secretion and improving glucose tolerance.	Potential for better glucose control without weight gain.	Diarrhea, nausea, headache
Fibroblast Growth Factor	Pegylated FGF21 (PEG-cFGF21) [34], Fc-FGF21(RGE) [35], PF05231023 [36], LY2405319 [37]	Mimics the effects of FGF21,	Potential to improve insulin	Fatigue, nausea, headache, possible liver

21 (FGF21) Analog		regulating glucose and lipid metabolism and improving insulin sensitivity.	sensitivity and reduce visceral fat.	enzyme elevation
AMP-activated Protein Kinase (AMPK) Activators	**Indirect:** (metformin) [38], thiazolidinediones (troglitazone, pioglitazone, rosiglitazone) [39], polyphenols (resveratrol, quercetin, genistein, epigallocatechin gallate, berberine, curcumin) [40], triterpene glycosides (ginsenoside, Rb1) [41], α-liponic acid (ALA) [42], cryptotanshinone [43], and DNA-damaging agents (cisplatin or metals, including arsenite, vanadate and cobalt through reactive oxygen species ROS activation) [44]. **Direct:** 5-aminoimidazole-4-carboxamide riboside (AICAR) [45], thienopyridone (A-769662) and benzimidazole (Compound 911) derivatives [46], salicylate (pro-drug of Asprin) [47], AMP mimetics (5-(5-hydroxyl-isoxazole-3-yl)-furan-2-phosphonic acid [48], termed Compound-2 (C-2), and its pro-drug C-13) [49], PT-1 [50], MT 63–78 (Debio0930) [51]	It activates AMPK, promoting glucose uptake and fatty acid oxidation and reducing hepatic glucose production.	Improved insulin sensitivity and glucose metabolism.	Gastrointestinal side effects, fatigue
Bromodomain and Extra-Terminal (BET) Inhibitors	RVX-208 [52,53], ABBV-744 [54]	Inhibits BET proteins that regulate gene expression involved in inflammation and metabolism, improving insulin sensitivity and reducing inflammation.	Potential to reduce insulin resistance and inflammation in T2DM.	Infections, fatigue, gastrointestinal symptoms
Thymosin Beta-4 Analog	TB-500 [55], The actin-sequestering protein thymosin beta-4 (Tβ4) [56]	Regulates glucose homeostasis and enhances pancreatic beta-cell function.	Possible improvement in beta-cell regeneration and glucose control.	Injection site reactions, mild gastrointestinal effects

Monogenic Therapy (Gene Editing)	CRISPR-Cas9 [57], ZFNs [58,59], TALENs [60,61]	Gene-editing technology that aims to correct mutations associated with beta-cell function or insulin resistance.	Potential for long-term, permanent correction of diabetes-related gene defects.	Ethical concerns, potential off-target effects, immune responses
Proprotein Convertase Subtilisin/Kexin Type 9 (PCSK9) Inhibitors	monoclonal antibodies (Evolocumab, Alirocumab, Bococizumab, and Tafolecimab), small interfering RNA (siRNA, Inclisiran), adnectin BMS-962476 [62,63]	Inhibits PCSK9, leading to increased insulin sensitivity and improved lipid metabolism.	Potential for improved glucose control, particularly in diabetic dyslipidemia.	Injection site reactions, muscle pain, fatigue

- Help in weight management
- Reduction in diabetes-related complications
- Better overall diabetes management

Exercise Therapy

Regular exercise

Regular physical exercise is an important part of the comprehensive treatment of Type 2 Diabetes Mellitus (T2DM). Exercise helps muscles take up and use more glucose, improves insulin sensitivity, and lowers blood sugar levels. In addition, regular exercise supports weight loss, improves cardiovascular function, lowers blood pressure and blood lipid levels, and reduces the risk of cardiovascular diseases. It is recommended that patients with T2DM perform at least 150 minutes of moderate-intensity aerobic exercise per week, such as brisk walking, jogging, swimming, or cycling. Strength training exercises like weightlifting and push-ups can also be included to improve muscle strength and glucose utilization.(64)

Micro exercise

In addition to traditional exercise methods, the concept of micro-exercise has gradually emerged. Micro-exercise refers to adding some short-term and mild physical activities in daily life, such as walking up and down stairs, standing while working, and getting up and moving regularly. These seemingly insignificant activities can accumulate to increase energy consumption and promote glucose metabolism. Studies have shown that micro-exercise can improve blood sugar control in T2DM patients and improve their quality of life.(64)

CONCLUSION

Type 2 Diabetes Mellitus (T2DM) is a complex and progressive metabolic disorder characterized by impaired insulin secretion, insulin resistance, and chronic hyperglycaemia. If not properly managed, it can lead to serious complications affecting the eyes, kidneys, nerves, heart, and blood vessels. The increasing prevalence of diabetes, especially in India, highlights the urgent need for early diagnosis, effective treatment, and preventive strategies. Early screening using blood glucose tests and HbA1c estimation plays an important role in identifying the disease before severe complications develop.

Management of T2DM requires a comprehensive approach that includes lifestyle modification, healthy diet, regular exercise, micro-exercise, and pharmacological therapy. Metformin remains the preferred first-line drug because of its effectiveness in improving insulin sensitivity and reducing hepatic glucose production. Various other classes of antidiabetic drugs, including insulin therapy, GLP-1 receptor agonists, DPP-4 inhibitors, SGLT2 inhibitors, and newer emerging therapies, have significantly improved diabetes management and patient outcomes. Recent advances in experimental therapies and gene-based approaches also offer promising future directions for the treatment of diabetes. Overall, proper glycaemic control, early intervention, patient education, and continuous monitoring are essential for reducing complications, improving quality of life, and achieving better long-term outcomes in patients with Type 2 Diabetes Mellitus.

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HOW TO CITE: Gholap Siddhi, Mandlik Rutuja, Madhe Prabhavati, Dr. Pallavi Phalake, Mayuri Mundhe, Type 2 Diabetes Mellitus, Int. J. of Pharm. Sci., 2026, Vol 4, Issue 5, 8268-8283. https://doi.org/10.5281/zenodo.20472427