

A REVIEW OF DIABETES MELLITUS: INSIGHTS FROM RECENT RESEARCH

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Abstract

Diabetes mellitus is also called as simply diabetes, is a group of metabolic disorder in which there are increase blood sugar levels over a prolonged period. This increase blood sugar produces the symptoms of frequent urination, increased thirst, and increased hunger. Untreated, diabetes may cause many complications. Acute complications include diabetic ketoacidosis and nonketotic

hyperosmolar coma. There are 3 main types of diabetes mellitus: Type 1 DM results from the body's failure to produce enough insulin. This form was previously referred to as "insulin-dependent diabetes mellitus" (IDDM). The cause is unknown Type 2 DM begins with insulin resistance, a condition in which cells fail to respond to insulin properly. As the disease progresses a lack of insulin may also develop. This form was previously referred to as "non insulin-dependent diabetes mellitus" (NIDDM) or "adult-onset diabetes". The primary cause is excessive body weight and not enough exercise.

Clinical features similar to diabetes mellitus described 3000 years ago by ancient Egyptians. The term diabetes was first coined by Aretus of Cappadocia. Later word mellitus was added by Thomas Willis in 1675 from patients urine. It was only in 1776 that Dobson firstly found presence of excess sugar in urine. The role of pancreas in insulin isolation and clinical use by Banting and Best in 1921. Then the first oral hypoglycemic agents tolbutamide and carbutamide are discovered in 1955.

Key words: *Diabetes mellitus, causes, side effects, diagnosis and treatment.*

Introduction: Diabetes mellitus is common endocrine disease which affects

more than 100 million people worldwide (6% population). It occurs due to the deficiency or ineffective production of insulin by pancreas which results in increase or decrease in concentrations of glucose in the blood. It is found to damage many of body systems particularly blood vessels, eyes, kidney, heart and nerves.

Diabetes mellitus classified into 2 types i.e. insulin dependent diabetes mellitus (IDDM, Type I) and non-insulin dependent diabetes mellitus (NIDDM, Type II). Type I diabetes is an autoimmune disease characterized by a local inflammatory reaction in and around islets that followed by selective destruction of the insulin secreting cells whereas Type II diabetes is characterized by peripheral insulin resistance and impaired insulin secretion. The presence of diabetes mellitus shows increased risk of many complications such as cardiovascular diseases, peripheral vascular diseases, stroke, neuropathy, kidney failure, retinopathy, loss of vision, etc. Drugs are used primarily to save life and alleviate symptoms. Our body is made up of countless cells. To make energy the cells want food. Once we eat or drink, a lot of your food is countermined into a easy sugar called as aldohexose.

Aldohexose provides the energy our body desires for the daily activities. If you turn

out very little or no endocrine, or square measure endocrine resistant, an excessive amount of sugar remains in our blood. glucose levels square measure more than traditional for people with polygenic disease.

[WHO] World Health Organisation graded Islamic Republic of Pakistan at seventh on polygenic disease prevalence list. Recent read on the prevalence has shown that the regarding quite four. 7 million individuals tormented by polygenic disease. polygenic disease symptoms square measure excessive thirst, frequent voiding, sweating, blurred vision, fulminant weight loss, fatigue and slow healing sores.

Largely patient with polygenic disease suffer from thirst, polyphagia and nephropathy. physiological condition polygenic disease solely occur throughout physiological state. secretion changes influence endocrine that cause the ineffective endocrine production, leading to raised {blood aldohexose blood sugar glucose} level which high glucose level effects embryo. found fast dysglycemia in thirty eight. 95% of a Venezuelan sample, with a prevalence of fourteen.25% for diabetes mellitus and 47% for prediabetes. it had been additionally found that the high blood pressure, hypercholesteremia, dysglycemia and DM were additional current in females.

History:

Diabetes mellitus has been recognized as a disease for about 3,500 years, with some of the earliest mentions found in ancient Egyptian texts. One significant source is the **Ebers Papyrus**, dating back to 1550 BC, which was discovered in Thebes, Egypt, in 1862. This document describes a condition characterized by excessive urination, likely diabetes, and suggests various remedies

like herbal concoctions made from bowes, wheat, and earth.

In ancient India, medical texts known as the **Vedic treatises** discussed diabetes in detail, identifying two types: congenital and late-onset. These texts recognized links between diabetes and factors like heredity, obesity, a sedentary lifestyle, and diet. They recommended remedies such as freshly harvested cereals and special preparations containing certain minerals.

Sushruta, a notable physician from the 5th to 6th century AD, was one of the first to connect the sweet taste of urine with the condition of polyuria, which is excessive urination.

Later, **Aretus of Cappadocia**, who lived from 81 to 138 AD, described diabetes as a wasting disease where the body seems to "melt down." He noted that patients experienced constant thirst and urination, and he coined the term "diabetes," which means "to run through" or "siphon," reflecting these symptoms.

In the 10th to 11th centuries, the Arab physician **Avicenna** provided an accurate description of diabetes, including its complications like nerve damage, gangrene, and erectile dysfunction. He emphasized the sweet taste of urine, which influenced European medical practices for centuries through his influential work, the "Kanon."

Overall, this history highlights how various cultures and physicians have contributed to our understanding of diabetes throughout the ages.

Scientist and their inventions

1. Key Milestones: Two important developments helped us understand diabetes better:

- In the late 1700s, doctors began using chemistry to diagnose diseases, which improved their understanding of diabetes.

- The field of endocrinology emerged, thanks to Claude Bernard and Brown-Sequard. Bernard studied how glands produce hormones, while Brown-Sequard showed that infusing healthy blood could help save lives after adrenal gland removal.

2. Thomas Willis (1675): In 1675, Thomas Willis, a physician in London, discovered that the urine of diabetic patients tasted very sweet, like honey. He called this condition "diabetes mellitus," combining the Greek word for diabetes with the Latin word "mellitus," which means "honey sweet." However, he couldn't explain why it was sweet.

3. Frank's Classification (1679): Four years later, Frank classified diabetes into two types based on how the urine tasted:

- Diabetes Insipidus (tasteless urine)
- Diabetes Vera (sweet urine).

4. Mathew Dobson (1776): In 1776, Mathew Dobson confirmed that sugar was present in the urine and blood of diabetic patients. He realized that the sugar didn't come from the kidneys, as people thought, but was already in the blood. This led him to believe that diabetes was a systemic problem, not just a kidney issue.

5. Identifying Glucose (1815): In 1815, French chemist Michael Chevreul identified the sugar in urine as glucose, clarifying what was happening in diabetic patients.

6. John Rollo's Mistake (1798): John Rollo, a physician, mistakenly thought diabetes was related to the stomach and came from how the body turned plant foods into sugar. He suggested that people cut back on carbohydrates as a treatment.

Banting and Best: dawn of insulin era:

One of the most significant discoveries in medicine was the isolation of insulin in the summer of 1921 by Frederick Banting and Charles Best in Toronto, Canada. They

conducted their research in the laboratory of John Macleod, a professor of physiology who had previously doubted the feasibility of isolating pancreatic secretions involved in carbohydrate metabolism. Despite his skepticism, Macleod allowed Banting to use his well-equipped lab. Best, an undergraduate student of Macleod, assisted with urine and blood tests.

After reviewing the existing literature, Banting and Best recognized the limitations of earlier researchers, notably Von Mering and Minkowski, and sought to build on their work. Their initial experiments yielded an impure pancreatic extract that successfully reduced blood glucose levels in dogs that had undergone pancreatectomy. A key difference in their approach was the involvement of biochemist James Collip, who helped purify the extract for safe human use, culminating in their breakthrough in 1921.

The introduction of insulin marked a new era in diabetes treatment, significantly improving the quality of life for patients and alleviating the dire consequences of starvation and severe complications like ketosis. In December 1921, Banting presented their findings on pancreatic extracts at the American Physiological Society meeting, with Macleod attending since Banting was not a member. Interestingly, Macleod played no role in the research itself. In 1922, Banting and Best published their results in the Canadian Medical Association Journal, notably without mentioning Macleod.

The discovery led to Banting and Macleod receiving the Nobel Prize in Medicine in 1923, although Banting felt overshadowed by Macleod and chose to share his prize money with Best. Macleod shared his prize with Collip, who took over research standardization. The first clinical use of

insulin occurred in January 1922, when a 14-year-old boy named Leonard Thompson received an extract that initially failed but dramatically improved when treated with Collip's method.

Following this, industrial mass production of purified insulin began. By 1936, new formulations such as protamine insulins were introduced, and by the 1950s, insulin zinc suspension became available. Later, Frederick Sanger determined the structural formula of insulin, and in 1960, researchers Nicol and Smith identified the amino acid sequences, revealing the differences between porcine, bovine, and human insulins. This series of advancements solidified insulin's critical role in diabetes management and paved the way for modern treatments.

Risks of polygenic disorder throughout physiological condition

Diabetes during pregnancy can significantly increase risks for both the mother and the baby. For newborns, complications may include metabolic distress, symptoms related to high blood sugar, increased red blood cell production (polycythemia), and thickened blood (hyperviscosity).

If diabetes isn't well managed, especially during the early stages of pregnancy (up to about 10 weeks), it can lead to:

1. Major birth defects
2. Increased chances of miscarriage

Proper care and management are crucial to reduce these risks.

Poor management of polygenic disorders during pregnancy can lead to several serious risks, including:

Fatal Macrosomia: This occurs when a baby is born weighing over 4,000 to 4,500 grams (about 8.8 to 9.9 pounds), which can create challenges during delivery.

Preeclampsia: A condition marked by high blood pressure and potential damage to organs, posing risks to both mother and baby.

Shoulder Dystocia: This is when the baby's shoulder becomes stuck during delivery, which can cause injuries.

Cesarean Delivery: There's an increased likelihood of needing a C-section if complications arise during labor.

Stillbirth: There is a higher risk of the baby being stillborn, meaning the loss of pregnancy after 20 weeks.

Proper management of these conditions is essential to protect the health of both the mother and the baby.

Classification of diabetes

The World Health Organization (WHO) first classified diabetes in 1980, updating it in 1985. The focus is primarily on two main types of diabetes: primary (or main) diabetes and secondary diabetes, which arises from identifiable causes like pancreatic disease, surgery, certain medications, or genetic disorders.

Primary diabetes is recognized as a diverse group of conditions, all characterized by high blood sugar (hyperglycemia). The classification system includes various clinical stages and types of diabetes, acknowledging that individuals may not fit neatly into one category.

In recent classifications, outdated terms like insulin-dependent diabetes mellitus (IDDM) and non-insulin-dependent diabetes mellitus (NIDDM) have been replaced. The current classification identifies four main types of diabetes mellitus: Type 1 (previously IDDM), Type 2 (previously NIDDM), "other specific types," and gestational diabetes. This new framework was incorporated into the

International Classification of Diseases (ICD-10) in 1992.

1.Harmon e dependent diabetes mellitus

Type 1 diabetes, also known as insulin-dependent diabetes (IDDM) or juvenile-onset diabetes, is an autoimmune condition where the body's immune system attacks and destroys the insulin-producing beta cells in the pancreas. This type of diabetes typically develops in children and young adults, and its onset can be sudden and severe, often requiring immediate medical attention.

Individuals with Type 1 diabetes may also experience other autoimmune disorders, such as Graves' disease, Hashimoto's thyroiditis, and Addison's disease. The disease is characterized by the presence of specific autoantibodies, such as anti-glutamic acid decarboxylase (GAD) antibodies, which indicate the immune attack on the pancreas.

The destruction of beta cells leads to a significant or complete deficiency of insulin, the hormone necessary for regulating blood sugar levels. This requires individuals to manage their condition with insulin injections or an insulin pump.

While the exact cause of Type 1 diabetes remains unclear, it is widely believed to involve a complex interplay of genetic and environmental factors that trigger the autoimmune response. The speed at which beta cells are destroyed can vary greatly from person to person, with some experiencing rapid deterioration and others a slower decline. Early detection and management are crucial to prevent complications associated with high blood sugar levels.

2.Non insulin dependent polygenic disorder mellitus

Type 2 diabetes is also known as ketosis-resistant diabetes mellitus. It involves a

gradual decline in the body's ability to produce insulin, combined with insulin resistance, meaning the body doesn't respond effectively to insulin. This type of diabetes is common and often leads to high blood sugar levels.

People with type 2 diabetes frequently experience complications in their blood vessels, kidneys, eyes, and nerves, which can lead to serious health issues and even death. These complications are significant for both types of diabetes.

Several factors can contribute to developing type 2 diabetes, including:

Obesity: Excess weight increases the risk.

Sedentary Lifestyle: Lack of physical activity plays a role.

Age: The risk increases as people get older, particularly in middle-aged and older individuals.

Genetics: Family history can influence susceptibility to the disease.

Patients with type 2 diabetes are at a higher risk of developing both large vessel (macrovascular) and small vessel (microvascular) complications.

3.Gestational diabetes

Gestational diabetes mellitus (GDM) refers to the development of diabetes during pregnancy. This condition can occur in women who previously had undiagnosed type 2 diabetes or can arise for the first time during pregnancy. GDM typically resolves after childbirth, but it poses certain risks for both the mother and the baby. Children born to mothers with GDM are at a higher risk of becoming overweight and developing type 2 diabetes later in life. This is believed to be linked to their exposure to high blood sugar levels in the womb.

In summary, GDM is a temporary form of diabetes that can have lasting effects on both the mother and child, making

monitoring and management important during pregnancy.

4. Alternative Specific Sort

The most common type of heritable polygenic disease involves mutations in the HNF-1 α gene, which plays a critical role in liver function and insulin regulation. This condition is often linked to genetic defects in pancreatic beta cells, leading to diabetes that typically appears before age 25.

This form of diabetes is known as Maturity-Onset Diabetes of the Young (MODY) or ketoacidosis-resistant diabetes in youth. It can also be associated with various other conditions, including diseases of the pancreas, such as pancreatitis or cystic fibrosis, as well as certain endocrine disorders like acromegaly.

Additionally, some medications used in conjunction with HIV/AIDS treatment or after organ transplants may contribute to these conditions. There are rare genetic disorders where individuals cannot convert proinsulin to insulin, which also follow an autosomal dominant inheritance pattern. These genetic issues account for a small percentage of all diabetes cases.

Causes:

Type 1 Diabetes

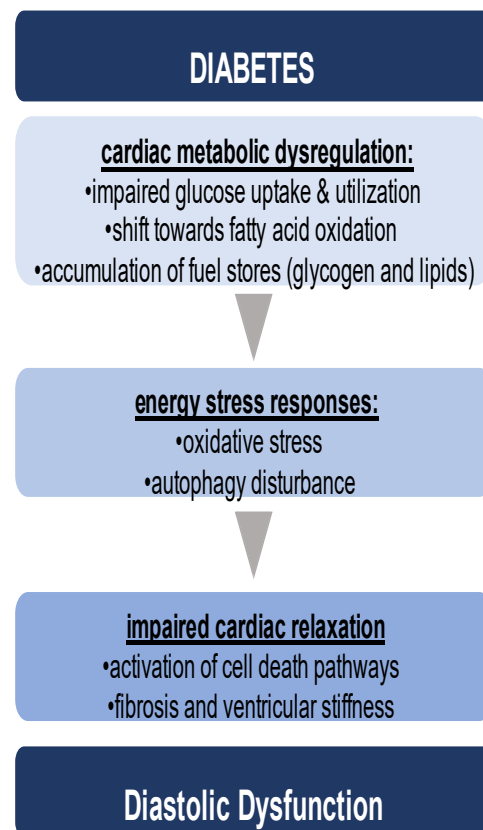
- Immune System Attack: The body mistakenly attacks and destroys the cells in the pancreas that produce insulin.
- Genetics: If you have family members with Type 1 diabetes, you might be at higher risk.
- Environmental Triggers: Certain infections might trigger this immune response.

Type 2 Diabetes

- Insulin Resistance: The body doesn't use insulin properly, so it

needs more insulin to keep blood sugar levels normal.

- Genetics: Having a family history of diabetes increases your risk.
- Lifestyle Choices:
- Being Overweight: Extra body fat, especially around the belly, can lead to insulin resistance.
- Lack of Exercise: Not being active can increase the risk.
- Unhealthy Diet: Eating a lot of processed foods and sugars can contribute to developing Type 2 diabetes.
- Age: The risk goes up as you get older, especially after age 45.



Classification of antidiabetic agent

1. Insulin & its preparation

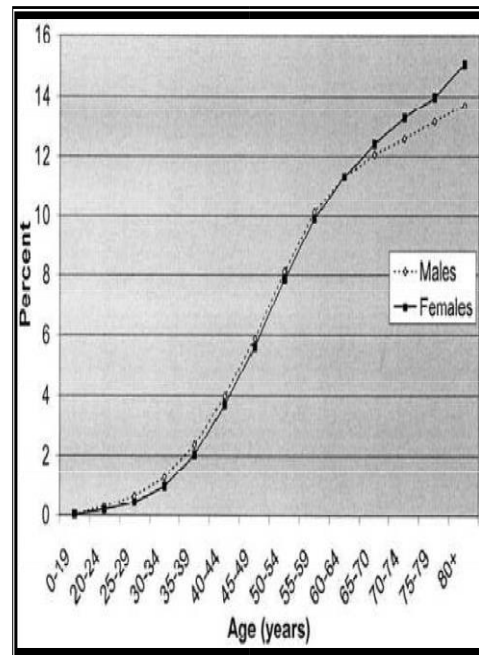
2. Oral hypoglycemic agents

- I. Sulfonyl ureas: tolbutamide, chlorpropamide
- II. Biguanides: metformin, phenformin
- III. Thiazolidinediones: pioglitazone, rosiglitazone
- IV. Meglitinides: repaglinide, nateglinide
- V. Glucosidase inhibitors: acarbose, voglibose

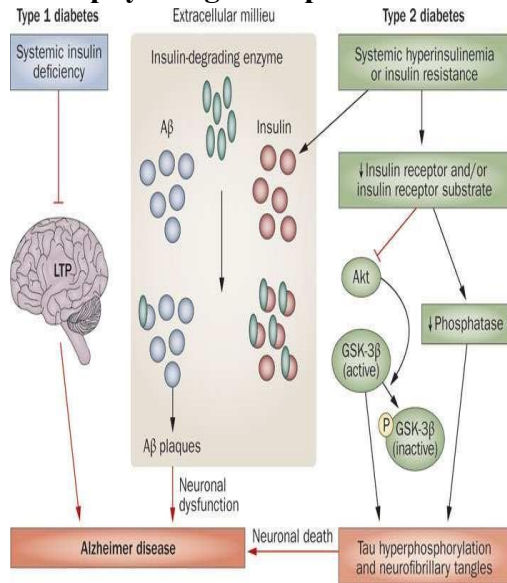
Epidemiology:

In 2011, around 366 millions of people were living with diabetes, and that the number is expected to increase to 552 million by 2030. A significant portion of these individuals—about 80%—are in low- and middle-income countries. In 2011 alone, diabetes was responsible for 4.6 million deaths. By 2030, it's estimated that 439 million people will have type 2 diabetes specifically. The rates of type 2 diabetes vary widely around the world, largely due to different environmental and lifestyle factors. Over the next two decades, the prevalence of diabetes, particularly type 2, is projected to increase, especially in developing countries. Many of those affected are expected to be adults aged between 45 and 64

Fig. 1: Epidemiology of diabetes: A global view



Pathophysiological aspects:



Type 2 diabetes mellitus is primarily characterized by the body's reduced sensitivity to insulin, which leads to insulin resistance. Over time, this resistance results in formation insulin production and, eventually, failure of the pancreatic beta cells. This dysfunction causes a reduction in the transport of glucose into the key tissues like the liver, muscle, and fat cells, while simultaneously increasing the breakdown of fat, leading to elevated blood sugar levels (hyperglycemia). Type 1 diabetes typically develops in younger individuals, often children or adolescents, and these patients are usually not overweight.

There is genetic component, as individuals with the family history have a significantly higher risk. Certain genetic markers, particularly specific histocompatibility antigens are strongly associated with this type. Research involving identical twins indicates that genetic predisposition alone is not enough; environmental factors, such as viral infections, are also required. These infections may damage the pancreatic beta cells and trigger an autoimmune response that progressively destroys more than 90% of these cells before diabetes becomes

clinically evident. In type 1 diabetes, the lack of insulin can impact cognitive functions, potentially affecting learning and memory.

Typical crucial roles in its development. It is more common in adults, particularly those who are obese, and its prevalence increases with age as beta-cell function declines. In patients with type 2 diabetes, insulin resistance is linked to the formation of amyloid beta (Aβ) plaques and hyperphosphorylation of tau proteins, both of which are associated with neurodegenerative diseases. During periods of high insulin levels, insulin competes with Aβ for the insulin-degrading enzyme, which can lead to the accumulation of Aβ and plaque formation. Additionally, reduced signaling through insulin receptors inhibits Akt, which activates glycogen synthase kinase 3 beta (GSK-3β), leading to hyperphosphorylation of tau proteins.

Pathophysiology of type 1 and type 2 diabetes

Sign and Symptoms:

In diabetes mellitus, cells cannot properly metabolize aldohexose (a type of sugar), leading to a state of starvation for the cells. Over time, this can lead to various complications, including:

- Retinopathy: Damage to the retina that can cause vision problems. Also causes loss of vision.
- Kidney Disease: Progressive damage that can result in kidney failure.
- Foot Ulcers: Increased risk of infections and ulcers on the feet due to poor circulation and nerve damage.
- Neuropathy: Nerve damage that can cause pain, tingling, or loss of sensation.
- Sexual Dysfunction: Issues related to sexual health. Can causes sexual problems.

Diagnosis:

The designation of polygenic disease in associate degree symptom less subject ought to never be created on the idea of one abnormal blood sugar value. If a designation of polygenic disease is formed, the practitioner should feel assured that the designation is totally established since the consequences for the individual area unit extensive and long. The designation of polygenic disease mllitus embrace, excretory product sugar, blood sugar, aldohexose tolerance check, urinary organ threshold of aldohexose, diminished aldohexose tolerance, exaggerated aldohexose tolerance, renal symptom, extended aldohexose tolerance curve, cortisone stressed aldohexose tolerance check, endo venous aldohexose tolerance test, oral aldohexose tolerance check.

Treatment:

[1] Selfcare :

[2] Oral hypoglycemic agents

[3] Insulin

[4] Drugs : Metformin, Phenformin, etc

[A] Varieties of medical aid concerned in diabetes mellitus :**[1] Somatic cell medical aid**

Researchers have discovered that the monocytes and macrophages play a important role in chronic inflammation and resistance to hypoglycemic agents in patients with type 2 diabetes (T2DM). It is non insulin dependent diabetes mellitus. To address immune dysfunctions, a novel therapy called somatic cell professional person medical aid has been developed. This approach involves several steps: Follow -

1. Blood Collection: The patient's blood is collected through the controlled system.

2. Lymphocyte Purification: The lymphocytes (a type of white blood cell) are separated from the rest of the blood.

3. Co-culture: These purified lymphocytes are then cultured with multi-potent stem cells derived from cord blood in a lab setting.

4. Administration: Finally, the educated lymphocytes are again introduce into the patient's bloodstream, while the stem cells are not used in the treatment. This method aims is to regulate the immune response and potentially reverse dysfunctions related to chronic inflammation in type 2 diabetes mellitus patients.

[2] Inhibitor medical care

Antioxidants, such as vitamins, supplements, and plant-based compounds, are commonly used to help manage oxidative stress in patients with type 2 diabetes (T2DM). Vitamins C and E, along with carotenoids, are particularly effective in reducing oxidative stress and its related complications. These antioxidants play an

important role in the lowering the risk of developing diabetes and its associated issues.

[3] Anti-inflammatory drug treatment

It seems like you're discussing the role of inflammation in type 2 diabetes mellitus (T2DM) and its complications. In type 2 diabetes mellitus, inflammation is indeed a key player, affecting various tissues such as the pancreas (particularly the islets), liver, blood vessels, and immune cells. Changes in cytokine and chemokine levels, as well as alterations in white blood cell populations and increased cell death, contribute to the disease's pathology.

[B] Dietary Management

Adequate caloric worth Dietary management ought to be taken properly by the each diabetic and non-diabetic patient such as.

1. It's important to maintain the balance between sugars, fats, and other nutrients, and you should limit sugar intake.
2. Your diet should aim to resemble traditional eating habits as much as possible.
3. Meals should be spaced throughout the day and be of similar size.
4. To lower overall calorie intake, reduce both fats and carbohydrates.
5. It's essential for the patient to stick to consistent eating habits every day.

[C] Newer endocrine Delivery Devices

A variety of innovations have been developed to make insulin administration easier and more accurate, helping people manage their blood sugar levels effectively. These include:

1. Insulin syringes: Simple devices for drawing and injecting insulin.
2. Pen devices: Convenient, portable pens that make it easy to deliver precise doses.

3. Inhaled insulin: A form of insulin that can be inhaled, providing an alternative to injections.

4. Insulin pumps: Devices that continuously deliver insulin through a small tube, offering better control.

5. Implantable pumps: Devices surgically placed under the skin to deliver insulin automatically.

6. Other delivery methods: Various new techniques and technologies aimed at improving insulin delivery. These advancements aim to help people with diabetes maintain tighter control over their blood sugar levels.

[D] Oral hypoglycemic or medicament agents

In 1957, the biguanide phenformin was introduced alongside sulfonylureas as a treatment for diabetes. Since then, ongoing research has led to the development of several new classes of medications. Among these are thiazolidinediones, which improve insulin sensitivity; meglitinide analogues, which stimulate insulin secretion; and alpha-glucosidase inhibitors, which slow carbohydrate absorption in the intestines. More recently, dipeptidyl peptidase-4 (DPP-4) inhibitors have emerged, enhancing the body's ability to lower blood sugar by increasing incretin levels. Together, these innovations have expanded treatment options for managing diabetes effectively.

Importance of oral hypoglycemic agents:

Diabetes mellitus is becoming more recognized as a significant health concern today. It greatly affects the health, well-being, and quality of life of those who have it. One common cause of diabetes is Cushing's syndrome, which results from long-term exposure to glucocorticoids, a type of steroid hormone.

Symptoms of Cushing's Syndrome

People with Cushing's syndrome can experience a variety of symptoms, such as:

Central Fat Distribution: Weight gain mainly around the belly and face.

Muscle Weakness: Noticeable weakness, especially in the upper body.

Hirsutism: Increased hair growth in areas typical for men.

Neurological Issues: Mood swings or problems with thinking clearly.

Macrovascular Complications: Issues with blood vessels that can raise the risk of heart disease and stroke.

Digestive Issues: Problems like ulcers or discomfort in the stomach.

Dental Problems: Issues such as gum disease and tooth decay.

Insulin and oral hypoglycemic agents:

Insulin therapy is designed to closely replicate the body's natural insulin production, effectively managing blood sugar levels after meals and preventing low blood sugar (hypoglycemia) between meals. The way insulin is administered—whether through intramuscular or intravenous injections—plays a crucial role in its effectiveness and safety.

There are various types of insulin, including human, beef, and pork insulin, each with its own characteristics. However, insulin therapy isn't without its challenges. The most common side effects include weight gain and hypoglycemia, especially if the dosage isn't correctly matched with food intake.

Weight gain often occurs after starting insulin for uncontrolled diabetes due to increased body fat and muscle mass, as well as decreased calorie loss through urine.

In addition to insulin, there are oral medications for diabetes management, such as sulfonylureas (like glibenclamide and glipizide) and biguanides (like metformin).

These medications help lower blood sugar levels through different mechanisms, offering alternative options for managing diabetes.

Sulfonylureas and biguanides (like metformin) work differently in managing blood sugar levels in type 2 diabetes.

Sulfonylureas stimulate insulin release from the pancreas by binding to specific receptors on pancreatic beta cells. This action closes ATP-sensitive potassium channels, leading to depolarization of the cell membrane, which opens voltage-gated calcium channels. The influx of calcium ions triggers the release of insulin. They can also reduce how quickly insulin is cleared from the liver, increasing overall insulin levels. However, sulfonylureas require a functioning pancreas to effectively lower blood sugar.

Biguanides, particularly metformin, operate differently. They don't stimulate insulin release, so they typically do not cause hypoglycemia, even at high doses. Metformin primarily works by increasing the uptake of glucose in peripheral tissues and decreasing glucose production in the liver by about 20-30%. Some studies also suggest that it may reduce the absorption of glucose from the gut.

In summary, while sulfonylureas directly promote insulin secretion and can lead to low blood sugar, metformin enhances the body's sensitivity to insulin and regulates glucose without causing hypoglycemia.

Herbal treatment of diabetes mellitus

In recent decades, eco-friendly and plant-based medicines have gained significant popularity, moving from niche use to mainstream acceptance, largely due to increased research into traditional medicine. A notable literature review by Atta-ar-Rahman highlights over 300 plant species known for their ability to lower

blood sugar levels. This review categorizes these plants by their botanical names, countries of origin, parts used, and the active compounds they contain.

One of these plants is *Momordica charantia**, commonly known as bitter melon, which belongs to the Cucurbitaceae family. The World Health Organization recognizes around 21,000 plants used for medicinal purposes worldwide, with 2,500 of those found in India. Of these, about 150 species are commercially cultivated on a large scale. India is often referred to as the "botanical garden of the world" because it is the largest producer of medicinal herbs.

Conclusion

Diabetes is very critical and serious complication in today's life. The lifestyle and day today circumstances are playing a major role in occurring this type of serious complications. In this review we get some idea regarding diabetes mellitus.

The main goal of managing diabetes is to normalize carbohydrate metabolism. For those with type 1 diabetes, this means they need insulin replacement therapy, which can be given via injections or pills. People with type 2 diabetes can often improve their condition through dietary changes and regular exercise to reduce insulin resistance.

Additionally, managing diabetes involves preventing or treating complications that can arise from the disease or its treatments. With proper management and control of blood sugar levels, people with diabetes can lead fulfilling lives and enjoy their daily activities.

Reference

1. *Diabetes mellitus* MEDICAL DISORDER WRITTEN BY: The Editors of Encyclopaedia Britannica See Article History

2. *The international diabetes Federation (IDF) is an umbrella organization of over 230 national diabetes associations in 170 countries and territories. The Federation has been leading the global Diabetes community since 1950. 166 Chaussee de La Hulpe, B-1170 Brussels, Belgium, P: +32-2-53855111 F: +32-2-25385114 info@idf.org, last update: 26/03/2020*
3. *American Diabetes Association. Diabetes Basics Last reviewed by a Cleveland Clinic medical professional on 10/02/2018. 9500 Euclid Avenue, Cleveland, Ohio 44195 | 800.223.2273 | 2020 Cleveland Clinic. All Rights Reserved.*
4. *National Diabetes Clearinghouse (NDIC): National Diabetes Statistics (2011) US Department of Health and Human Services*
5. *Atkinson MA, Eisenbarth GS. Type 1 diabetes new perspectives on disease pathogenesis and treatment, Lancet, 2001; 358: 221-229.*
6. *Arora, S., Ojha, S.K., Vohora, D., Characterisation of Streptozotocin induced diabetes mellitus in Swiss Albino mice, Glo J of Pharmacol., 3(2): 81-84 (2009)*
7. *Jothivel, N., Ponnusamy, S.P., Appachi, M., Antidiabetic activities of methanol leaf extract of *Costus pictus* D. Don in alloxan-induced diabetic rats, J of health sci., 53(6): 655-663 (2007)*
8. *Bastaki, S., Review Diabetes mellitus and its treatment, Int J Diabetes & Metabolism, 13: 111-134 (2005)*
9. *K. Huynh, B.C. Bernardo, J.R. McMullen, R.H. Ritchie, Diabetic cardiomyopathy: mechanisms and new treatment strategies targeting antioxidant signaling pathways, Pharmacol. Ther. 142 (2014) 375-415.*
10. *C.S. Lam, Diabetic cardiomyopathy: an expression of stage B heart failure with preserved ejection fraction, Diab. Vasc. Dis. Res. 12 (2015) 234-238.*
11. *H. Bugger, E.D. Abel, Molecular mechanisms of diabetic cardiomyopathy, Diabetologia 57 (2014) 660-671.*
12. *Reecc EA. The history of diabetes mellitus. In: Reece EA. Coustan DR editors. Diabetes Mellitus in Pregnancy. New York, (USA): Churchill Livingstone, 1995. p. 1 - 10.*



13. McGrew RE. *Encyclopedia of Medical history. 1st cd. London. (United Kingdom): McMillam Press; p. 74-297.*
14. Kassender P. *gastric retention (gastroparcsis diabeticorum). Ann Intern Med 1958; 48: 797- 812.*
15. Chaikcn BH, Klein AJV. *Gastric retention and intestinal malabsorption in diabetes mellitus. J Med Soc NJ 1961 : 58:*