

An Overview Of Diabetes:Review

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ABSTRACT

Diabetes mellitus is a fast-growing Global problem with huge health, social and economic consequences. Globally an estimated 463 million adults are living with diabetes, according to the latest 2019 data from the International Diabetes Federation. Diabetes prevalence is increasing rapidly; previous 2017 estimates put the number at 425 million people living with diabetes. The number is projected to almost double by 2030. This chapter introduces the types of diabetes, symptoms, treatment, causes, diagnosis and epidemiology of diabetes.

Keywords: - Diabetes, glucose, insulin, hyperglycemia

I. INTRODUCTION

Diabetes is a disease that occurs when the blood glucose, also called blood sugar, is too high. Blood glucose is the main source of energy and comes from the food we eat. Insulin, a hormone made by the pancreas, helps glucose from food get into our cells to be used for energy. Sometimes our body doesn't make enough—or any—insulin or doesn't use insulin well. Glucose then stays in the blood and doesn't reach the cells. When the amount of glucose in the blood increases, e.g., after a meal, it triggers the release of the hormone insulin from the pancreas. Insulin stimulates muscle and fat cells to remove glucose from the blood and stimulates the liver to metabolize glucose, causing the blood sugar level to decrease to normal levels. In people with diabetes, blood sugar levels remain high. This may be because insulin is not being produced at all, is not made at sufficient levels, or is not as effective as it should be. The most common forms of diabetes are type 1 diabetes (5%), which is an autoimmune disorder, and type 2 diabetes (95%), which is associated with obesity. Gestational diabetes is a form of diabetes that occurs in pregnancy, and other forms of diabetes

are very rare and are caused by a single gene mutation.

CLASSIFICATION

Diabetes is classified by underlying cause. The categories are:

- I.Type 1 diabetes
- II.type 2 diabetes
- III. Gestational diabetes

I. Type 1 diabetes

WHAT IS TYPE 1 DIABETES?

Type 1 diabetes (T1D), previously known as juvenile diabetes, is an autoimmune disease that is a form of diabetes in which very little or no insulin is produced by the islets of Langerhans (containing beta cells) in the pancreas. Insulin is a hormone required for the cells to use blood sugar for energy and it helps regulate normal glucose levels in the bloodstream. Type 1 diabetes is a condition in which our immune system destroys insulin-making cells in your pancreas. These are called beta cells. The condition is usually diagnosed in children and young people, so it used to be called juvenile diabetes.

Both of these are different from type 2 diabetes, in which the body doesn't respond to insulin the way it should.

SYMPTOMS

Signs are often subtle, but they can become severe. They include:

1. Extreme thirst
2. Increased hunger (especially after eating)
3. Dry mouth
4. Upset stomach and vomiting
5. Frequent urination
6. Unexplained weight loss, even though eating and feel hungry
7. Fatigue

8. Blurry vision
9. Heavy, labored breathing
10. Frequent infections of the skin, urinary tract, or vagina
11. Crankiness or mood changes
12. Bedwetting in a child who's been dry at night
13. Signs of an emergency with type 1 diabetes include:
14. Shaking and confusion
15. Rapid breathing
16. Fruity smell of the breath
17. Belly pain
18. Loss of consciousness (rare)

Type 1 Diabetes Causes

Insulin is a hormone that helps move sugar, or glucose, into the body's tissues. The cells use it as fuel.

Damage to beta cells from type 1 diabetes throws the process off. Glucose doesn't move into the cells because insulin isn't there to do the job. Instead, it builds up in the blood, and cells starve. This causes high blood sugar, which can lead to:

Dehydration. When there's extra sugar in the blood, we pee more. That's our body's way of getting rid of it. A large amount of water goes out with that urine, causing our body to dry out.

Weight loss. The glucose that goes out when we pee takes calories with it. That's why many people with high blood sugar lose weight. Dehydration also plays a part.

Diabetic ketoacidosis (DKA). If the body can't get enough glucose for fuel, it breaks down fat cells instead. This creates chemicals called ketones. Our liver releases the sugar it stores to help out. But the body can't use it without insulin, so it builds up in our blood, along with the acidic ketones. This mix of extra glucose, dehydration, and acid buildup is known as ketoacidosis and can be life-threatening if not treated right away. Damage to our body. Over time, high glucose levels in our blood can harm the nerves and small blood vessels in our eyes, kidneys, and heart. They can also make us more likely to get hardened arteries, or atherosclerosis, which can lead to heart attacks and strokes.

We can get type 1 diabetes when something around us, like a virus, tells our immune system to go after our pancreas. Most people with type 1 diabetes have signs of this attack, called autoantibodies.

Type 1 Diabetes Risk Factors

Only about 5% of people with diabetes have type 1. It affects males and females equally. There is high risk of getting it if we:

1. Are younger than 20
2. Are white
3. Have a parent or sibling with type 1

Type 1 Diabetes Diagnosis

Diagnostic tests include:

Diagnosis of diabetes has historically included fasting blood glucose higher than 7 mmol/L (126 mg/dL), any blood glucose of 11.1 mmol/L (200 mg/dL) or higher with symptoms of hyperglycaemia, or an abnormal 2-hour glucose-tolerance test. In 2009, the American Diabetes Association modified their guidelines for diabetes diagnosis to include glycated haemoglobin (HbA_{1c}; a test that averages blood glucose concentrations over 3 months) of 6.5% or higher. Despite efforts to standardise diagnosis of type 1 diagnosis, the causes and typology remain unclear. Particularly among adults, diagnosis of type 1 versus type 2 diabetes can be challenging. Around 5–15% of adults diagnosed with type 2 diabetes might actually have type 1 disease with islet autoantibodies present; if this is the case, perhaps as many as 50% of actual type 1 diabetes cases are misdiagnosed as type 2, meaning that the number of cases of type 1 disease is vastly underestimated. Accurate diagnosis of this disorder is crucial for optimum care and avoiding complications, and correctly noting diabetic ketoacidosis at diagnosis of type 1 disease represents a key window for survival.

II. TYPE 2 DIABETES

Type 2 diabetes is a lifelong disease that keeps the body from using insulin the way it should. It is the most common type of diabetes. People with type 2 diabetes are said to have insulin resistance.

People who are middle-aged or older are most likely to get this kind of diabetes. It used to be called adult-onset diabetes. But type 2 diabetes also affects kids and teens, mainly because of childhood obesity.

Type 2 diabetes are so mild that we cannot notice them. Symptoms include:

1. Being very thirsty
2. Peeing a lot
3. Blurry vision
4. Being cranky

5. Tingling or numbness in the hands or feet
6. Fatigue/feeling worn out
7. Wounds that don't heal
8. Yeast infections that keep coming back
9. Feeling hungry
10. Weight loss without trying
11. Getting more infections
12. Dark rashes around neck and armpits can sometimes be signs that body is becoming resistant to insulin.

Causes of Type 2 Diabetes

The pancreas makes a hormone called insulin. It helps our cells turn glucose, a type of sugar, from the food we eat into energy. People with type 2 diabetes make insulin, but their cells don't use it as well as they should. At first, the pancreas makes more insulin to try to get glucose into our cells. But eventually, it can't keep up, and the glucose builds up in our blood instead.

Usually, a combination of things causes type 2 diabetes. They might include: Genes. Scientists have found different bits of DNA that affect how our body makes insulin. Extra weight. Being overweight or obese can cause insulin resistance, especially if we carry our extra pounds around our middle.

Metabolic syndrome. People with insulin resistance often have a group of conditions including high blood sugar, extra fat around the waist, high blood pressure, and high cholesterol and triglycerides.

Too much glucose from our liver. When the blood sugar is low, our liver makes and sends out glucose. After we eat, our blood sugar goes up, and our liver will usually slow down and store its glucose for later. But some people's livers don't. They keep cranking out sugar.

Bad communication between cells. Sometimes, cells send the wrong signals or don't pick up messages correctly. When these problems affect how our cells make and use insulin or glucose, a chain reaction can lead to diabetes.

Type 2 Diabetes Risk Factors

Certain things make it more likely that we'll get type 2 diabetes. The more of these that apply to us, the higher our chances of getting it are. Some things are related to who we are:

Age. 45 or older

Family. A parent, sister, or brother with diabetes

Risk factors related to our health and medical history include:

1. Prediabetes
2. Heart and blood vessel disease
3. High blood pressure, even if it's treated and under control
4. Low HDL ("good") cholesterol
5. High triglycerides
6. Being overweight or obese
7. Having a baby who weighed more than 9 pounds
8. Gestational diabetes while you were pregnant
9. Polycystic ovary syndrome (PCOS)
10. Depression

Other things that raise risk of diabetes have to do with daily habits and lifestyle. These include:

1. Getting little or no exercise
2. Smoking
3. Stress
4. Sleeping too little or too much

Type 2 Diabetes Diagnosis and Tests

Doctor can test blood for signs of type 2 diabetes. Test on 2 days to confirm the diagnosis. But if blood glucose is very high or the patient has many symptoms, one test may be all they need.

A1c. It's like an average of blood glucose over the past 2 or 3 months.

Fasting plasma glucose. This is also known as a fasting blood sugar test. It measures blood sugar on an empty stomach. The patient won't be able to eat or drink anything except water for 8 hours before the test.

Oral glucose tolerance test (OGTT). This checks blood glucose before and 2 hours after drinking something sweet to see how your body handles the sugar.

III. GESTATIONAL DIABETES

What Is Gestational Diabetes?

Gestational diabetes is a condition in which the blood sugar levels become high during pregnancy. It affects up to 10% of women who are pregnant in the U.S. each year. It affects pregnant women who haven't ever been diagnosed with diabetes.

There are two classes of gestational diabetes. Women with class A1 can manage it through diet and exercise. Those who have class A2 need to take insulin or other medications.

Gestational diabetes goes away after giving birth. But it can affect the baby's health, and it raises the risk of getting type 2 diabetes later in life.

Gestational diabetes GDM is a form of hyperglycemia. In general, hyperglycemia results from an insulin supply that is inadequate to meet tissue demands for normal blood glucose regulation. Studies conducted during late pregnancy, when, as discussed below, insulin requirements are high and differ only slightly between normal and gestational diabetic women, consistently reveal reduced insulin responses to nutrients in women with GDM (17–23). Studies conducted before or after pregnancy, when women with prior GDM are usually more insulin resistant than normal women (also discussed below), often reveal insulin responses that are similar in the 2 groups or reduced only slightly in women with prior GDM (18, 22–26). However, when insulin levels and responses are expressed relative to each individual's degree of insulin resistance, a large defect in pancreatic β cell function is a consistent finding in women with prior GDM (23, 25, 27).

Gestational Diabetes Symptoms

It include

Also symptoms include

- Thirstier than usual
- Hungrier and eat more than usual
- pee more than usual

Gestational Diabetes Causes

When we eat, our pancreas releases insulin, a hormone that helps move a sugar called glucose from your blood to our cells, which use it for energy.

During pregnancy, the placenta makes hormones that cause glucose to build up in our blood. Usually, our pancreas can send out enough insulin to handle it. But if your body can't make enough insulin or stops using insulin as it should, our blood sugar levels rise, and we get gestational diabetes.

Gestational Diabetes Risk Factors

Prone to gestational diabetes include overweight before pregnant.

Having blood sugar levels that are higher than they should be but not high enough to be diabetes (this is called prediabetes)

Have a family member with diabetes

Have had gestational diabetes before

Have polycystic ovary syndrome (PCOS) or another health condition linked to problems with insulin

Have high blood pressure, high cholesterol, heart disease, or other medical complications

Have given birth to a large baby (weighing more than 9 pounds)

Have had a miscarriage

Have given birth to a baby who was stillborn or had certain birth defects

Are older than 25

Gestational Diabetes Tests and Diagnosis

Gestational diabetes usually happens in the second half of pregnancy. Doctor will check for it between weeks 24 and 28, or sooner if we're at high risk.

Doctor will give a glucose tolerance test:

Giving the patient to drink 50 grams of glucose in a sweet drink, which will raise your blood sugar. An hour later, taking a blood glucose test to see how the body handled all that sugar. If the results show that blood sugar is higher than a certain level, a 3-hour oral glucose tolerance test is needed, doctor can also test the patient by making them fast for 12 hours, then giving a 75-gram glucose drink and a 2-hour blood glucose test.

The newly proposed criteria for diagnosing gestational diabetes will result in a gestational diabetes prevalence of 17.8%, doubling the numbers of pregnant women currently diagnosed. These new diagnostic criteria are based primarily on the levels of glucose associated with a 1.75-fold increased risk of giving birth to large-for-gestational age infants (LGA) in the Hyperglycemia Adverse Pregnancy Outcome (HAPO) study; they use a single OGTT. Thus, of 23,316 pregnancies, gestational diabetes would be diagnosed in 4,150 women rather than in 2,448 women if a twofold increased risk of LGA were used. It should be recognised that the majority of women with LGA have normal glucose levels during pregnancy by

these proposed criteria and that maternal obesity is a stronger predictor of LGA. The expected benefit of a diagnosis of gestational diabetes in these 1,702 additional women would be the prevention of 140 cases of LGA, 21 cases of shoulder dystocia and 16 cases of birth injury. The reproducibility of an OGTT for diagnosing mild hyperglycaemia is poor. Given that (1) glucose is a weak predictor of LGA, (2) treating these extra numbers has a modest outcome benefit and (3) the diagnosis may be based on a single raised OGTT value, further debate should occur before resources are allocated to implementing this change.

GESTATIONAL DIABETES TREATMENT

Treatment should be done as soon as possible to keep the mother and the baby healthy during pregnancy and delivery. Doctor's will let the patient to

- Check blood sugar levels four or more times a day.
- check urine for ketones, chemicals
- eating a healthy diet
- making exercise a habit

Diet and Exercise for Gestational Diabetes

Eat a healthy, low-sugar diet. Talk to the doctor to be sure we're getting the nutrition we need. Follow a meal plan made for someone with diabetes:

Trade sugary snacks like cookies, candy, and ice cream for natural sugars like fruits, carrots, and raisins. Add vegetables and whole grains, and watch your portion sizes.

Have three small meals along with two or three snacks about the same times every day.

Get 40% of our daily calories from carbs and 20% from protein. Fifty percent of the carbs should be complex, high-fiber carbs, with fat being between 25% and 30%.

Aim for 20-35 grams of fiber a day. Foods such as whole-grain breads, cereals, and pasta; brown or wild rice; oatmeal; and vegetables and fruits will help get us there.

Limit your total fat to less than 40% of your daily calories. Saturated fat should be less than 10% of all the fat we eat.

Eat a variety of foods to make sure we get enough vitamins and minerals. we may need to take a supplement to cover our bases. Ask your doctor if they think we should take one.

Exercise throughout the pregnancy. Being active is a good way to help manage our blood sugar. Staying fit during pregnancy is also good for our posture and can curb some common problems, like backaches and fatigue.

Get active as soon as possible. Aim for 30 minutes of moderate activity most days of the week. Running, walking, swimming, and biking are good options.

Exercise can lower our blood sugar. So when we work out, always have a form of quick sugar with us, such as glucose tablets or hard candy.

Get the right prenatal care: Not only can our doctor screen us for this condition; they can offer advice on food, activity, and weight loss. They can also point us to other health professionals, like nutritionists, that can help.

If you have morning sickness, eat small snacks. Nibble on crackers, cereal, or pretzels before getting out of bed. As we go through our day, have small meals often and avoid fatty, fried, and greasy foods.

If we take insulin, make sure we got a plan to deal with low blood sugar. Throwing up can make our glucose level drop. Talk doctor if you are not sure what to do.

Safety and Mode of Action of Diabetes Medications in comparison with 5-Aminolev

Diabetes Management-Targeting Pathways

The targeted therapies for patients with type 2 diabetes are established based on various pathways through which glucose control can be achieved.

After food is consumed and digestion begins, glucose levels start to increase, as do other hormones such as glucagon-like peptide (GLP-1) which is released in the intestines. Glucagon-like peptide 1 (GLP-1) is an incretin, which works by triggering insulin production (as insulin acts to decrease glucose levels) and inhibiting glucagon

production (glucagon acts to increase glucose levels). This occurs to counteract the increased glucose, and it induces the feeling of satiety and reduces appetite by sending signals to the brain that one is full. The consumption of food also triggers the release of pancreatic hormones like insulin, amylin, and glucagon. Insulin and amylin both work to decrease glucose levels and inhibit glucagon while glucagon acts on the liver to raise glucose levels.

Diabetes Medications

The American Diabetes Association's Standards of Medical Care in Diabetes Patients specifies the pharmacological management of type 2 diabetes, all of which are discussed below.

Metformin (Type: Biguanide)

Metformin is the most common initial drug prescribed for type 2 diabetes in the world. Metformin acts via an antihyperglycemic pathway through an increased glucose tolerance in patients with type 2 diabetes. Typically, this is measured via blood plasma glucose levels and postprandial plasma levels. As an antihyperglycemic agent, the mode of action entails decreasing hepatic glucose production and the intestinal absorption of glucose and increasing peripheral glucose uptake and utilization. It also helps break down free fatty acids by activating adenosine monophosphate- (AMP-) activated protein kinase in hepatocytes. Additionally, metformin does not affect insulin secretion levels; fasting insulin levels and 24-hour plasma response have been shown to actually decrease. Metformin is secreted without additional metabolism through tubular secretion in the kidney, and because of this, renal issues can prove fatal through lactic acidosis—a rare metabolic complication. This is caused by the inhibition of hepatic gluconeogenesis by inhibiting a mitochondrial isoform of glycerophosphate dehydrogenase (mGPD), preventing glycerol from participating in the gluconeogenic pathway.

SIDE EFFECTS ASSOCIATED WITH USING METFORMIN

Side effects of metformin include:

- physical weakness (asthenia)
- diarrhea
- gas (flatulence)
- symptoms of weakness, muscle pain (myalgia)
- upper respiratory tract infection
- low blood sugar (hypoglycemia)

- abdominal pain (GI complaints), lactic acidosis (rare)
- low blood levels of vitamin B-12
- nausea
- vomiting
- chest discomfort
- chills, dizziness
- bloating/abdominal distention
- constipation
- heartburn

Sulfonylurea (Glipizide, Gliclazide, and Glimepiride)

Sulfonylureas are only beneficial to patients who have retained some degree of residual pancreatic beta cell functionality, as they work by stimulating insulin secretion. This is often difficult due to the death/loss of function of the insulin-producing beta cells in the pancreas which accompanies diabetes. The necessity for beta cells' presence stems from the molecular mechanism of action: on the surface of these cells, sulfonylurea has specific neuronal receptors. When a sulfonylurea-type molecule binds, it causes the cellular membrane to depolarize, leading to the calcium channel opening and resultant calcium influx. Next, this change in the charge of the cell causes actomyosin filaments to contract and, in turn, release insulin from the cell. After a brief period, insulin granule transmission begins, and new insulin granules are formed. Sulfonylureas' target receptor is a complex of the sulfonylurea 1 receptor (SUR1), specifically the K-ATP channel, altering the resting potential. Adding 5-ALA has been shown to aid this process, especially with improved results in patients with consistent insulin resistance by increasing the availability of ATP, which aids with key metabolic processes (like the TCA cycle) within the mitochondria. Sulfonylureas are glucose-level independent, meaning that there is a higher sensitivity to amino acids and, in turn, higher insulin release. An increased sensitivity of beta cells to glucose and non-glucose secretagogues develops; thus, hypoglycemia and weight gain are resultant potential side effects. Finally, an increase in peripheral glucose utilization has been noted with this drug class by both stimulating hepatic gluconeogenesis and increasing the number/sensitivity of insulin receptors

Side Effects of Sulfonylureas Side effects of sulfonylureas may include:

- Signs of low blood sugar, such as sweating, dizziness, confusion, or nervousness

- Hunger
- Weight gain
- Skin reactions
- Upset stomach
- Dark-colored urine

Meglitinides (Repaglinide and Nateglinide)

Meglitinides act on different beta cell receptors, but in a similar fashion to sulfonylureas. They work on the same K-ATP channels and increase insulin secretion. One potential downside is the lower binding affinity present at the surface level of the pancreatic beta cells; combined with the faster dissociation rates, the efficacy of this class of drugs is less than its parallel. Their adverse effects include hypoglycemia and weight gain. Lastly, they are more expensive than sulfonylureas and are commonly used in patients with allergies to the former

Side effects of Meglitinides

Meglitinides are well-tolerated by most people, including elderly people who need help lowering their mealtime blood sugars.

Common

Low blood sugar (hypoglycemia) is the most common side effect of meglitinides. Symptoms of hypoglycemia include sweating, shakiness, lightheadedness, and confusion.

These medications also can cause weight gain.

Severe

Meglitinides are relatively short-acting, which means they're unlikely to cause hypoglycemia. However, if taken without food, these drugs can cause a significant drop in blood sugar.

Someone experiencing hypoglycemia (blood sugar less than 70mg/dL) should consume some form of glucose, such as four ounces of juice. Anyone experiencing signs of diabetic coma, including confusion or loss of consciousness, should seek medical attention immediately.

Thiazolidinediones (Rosiglitazone and Pioglitazone)

Thiazolidinediones (TZDs) effectively attempt to mimic insulin by reducing hyperglycemia even with an impaired insulin tolerance. This leads to substantial reductions in hyperinsulinemia, which is caused by an increase of peripheral glucose consumption and decrease in hepatic glucose levels. There is no change in the secretion levels on insulin, but potential restoration

of pancreatic beta cell insulin reserves has been observed. The exact mode of action has not been specified; however, there are two key effects to be discussed. First, the affinity of TZDs to the binding site known as peroxisome proliferator-activated receptor- (PPAR-) gamma on the adipocyte protein 2 (aP2) molecule, a key gene involved in weight loss efforts, has led to the connection between the TZD hypoglycemic action and the promoter region PPAR-gamma, especially with the PPAR-gamma agonist rosiglitazone. Ultimately, insulin sensitivity is not a direct by-product of this aspect, which raises the second point—TZDs are able to uniquely activate the phosphoinositide 3-kinase (PI3K) pathway with or without PPAR-gamma.

Side effects of Thiazolidinediones

Common side effects associated with TZDs include edema, weight gain, macular edema and heart failure. Moreover, they may cause hypoglycemia when combined with other antidiabetic drugs as well as decrease hematocrit and hemoglobin levels. Increased bone fracture risk is another TZD-related side effect. Thiazolidinediones tend to increase serum low density lipoprotein cholesterol levels, with rosiglitazone having a more pronounced effect compared with pioglitazone

GLP Agonists (Exenatide, Lixisenatide, Liraglutide, Albiglutide, and Dulaglutide)

Glucagon-like peptide (GLP-1), an incretin, is a gastrointestinal peptide involved in the regulation of glucose levels where the hormone is released upon consuming food. It stimulates insulin formation and release, and this occurs upon oral ingestion of food exclusively. GLP binds to receptors present in many tissues including beta cells, gastric mucosa, the kidney, the heart, etc. This hormone is targeted in diabetes because it causes insulin release from the beta cells, as well as slows down gastric emptying and inhibits excess glucagon release after meals. This, in turn, decreases appetite (causing weight loss). GLP agonists, however, are injectable medications that act by enhancing these effects in the body, thereby making it less appealing to some patients.

Side effects of GLP Agonist

Some of the more common side effects include:

- Nausea
- Vomiting
- Diarrhea

DPP4 Inhibitors (Sitagliptin, Saxagliptin, Linagliptin, and Alogliptin)

DPP4 is an enzyme that deactivates the glucose-dependent insulinotropic polypeptide (GIP) and GLP-1. The inhibition of this enzyme causes an increased availability of GLP-1 levels in the body (as seen above). DPP4 inhibitors are a group of drugs that are an oral GLP-1-based therapy; however, they are not as effective at glucose or weight reduction. Their potential side effects are angioedema and pancreatitis, but they have a lower risk of hypoglycemia. This class may be considered in those who are intolerant of or have contraindications to metformin, sulfonyleureas, or thiazolidinediones, such as patients with chronic kidney disease or who are at a high risk of hypoglycemia. They can also be considered an add-on medication; however, this is often cost prohibitive

Side effects of DPP4 Inhibitors Adverse effects of DPP-4 inhibitors include:

- gastrointestinal problems – including nausea, diarrhoea and stomach pain
- flu-like symptoms – headache, runny nose, sore throat
- skin reactions – painful skin followed by a red or purple rash

Sodium Glucose Co-Transporter-2 Inhibitors (Gliflozins)

Sodium glucose co-transporter-2 (SGLT2) works by inhibiting the SGLT2 receptors in the kidneys' proximal convoluted tubule, the site where most glucose is reabsorbed back into the body. These medications prevent the reabsorption of glucose and increase its urinary excretion and can cause polyuria which, in certain cases, can result in postural hypotension. They also contribute to weight loss and some side effects that include urinary and genital infections, as well as diabetic ketoacidosis.

Side effects of Sodium Glucose Co-Transporter-2 Inhibitors (Gliflozins)

Serious side effects of SGLT2 inhibitors include:

- Kidney failure.
- Hyperkalemia (high levels of potassium in the blood)
- Hypotension (low blood pressure)
- Ketoacidosis.
- Increased cholesterol levels.

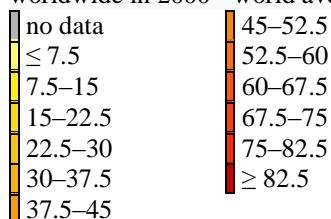
- Serious urinary tract infections.
- Increased bladder cancer risk.
- Serious allergic reactions.

Epidemiology of diabetes

Globally, an estimated 463 million adults are living with diabetes, according to the latest 2019 data from the International Diabetes Federation. Diabetes prevalence is increasing rapidly; previous 2017 estimates put the number at 425 million people living with diabetes. The number is projected to almost double by 2030. Type 2 diabetes makes up about 85-90% of all cases. Increases in the overall diabetes prevalence rates largely reflect an increase in risk factors for type 2, notably greater longevity and being overweight or obese.

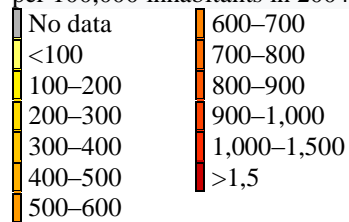
Diabetes mellitus occurs throughout the world, but is more common (especially type 2) in the more developed countries. The greatest increase in prevalence is, however, occurring in low- and middle-income countries including in Asia and Africa, where most patients will probably be found by 2030. The increase in incidence in developing countries follows the trend of urbanization and lifestyle changes, including increasingly sedentary lifestyles, less physically demanding work and the global nutrition transition, marked by increased intake of foods that are high energy-dense but nutrient-poor (often high in sugar and saturated fats, sometimes referred to as the Western pattern diet).

Prevalence (per 1,000 inhabitants) of diabetes worldwide in 2000 - world average was 2.8%.





Disability-adjusted life year for diabetes mellitus per 100,000 inhabitants in 2004



CONCLUSION:

From the above studies, it briefly explains the diabetes and its types. It emphasizes on diagnostic, treatment and medication. Taking medicine has its treatment properties as well as there is side effects.

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