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A REVIEW ON DIABETES MELLITUS AND ITS MANAGEMENT

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ABSTRACT

Diabetes mellitus (DM) is a group of metabolic disorders characterised by hyperglycemia. It is associated with abnormalities in carbohydrate, fat, and protein metabolism and results in chronic complications including microvascular, macrovascular, and neuropathic disorders. Several distinct types of DM exist and are caused by a complex interaction of genetics, environmental factors, and life-style choices. Depending on the etiology of the DM, factors contributing to hyperglycemia may include reduced insulin secretion, decreased glucose utilization, and increased glucose production. The metabolic dysregulation associated with DM causes secondary pathophysiologic changes in multiple organ systems that impose a tremendous burden on the individual with diabetes and on the health care system.

Research and drug development efforts over the past several decades have provided valuable information that applies directly to improving outcomes in patients with DM.

INTRODUCTION:

What is Diabetes?

Diabetes mellitus is a group of metabolic diseases characterized by high blood sugar (glucose) levels, which result from defects in insulin secretion, or action, or both. Diabetes mellitus, commonly referred to as diabetes (and in this article will be referred to as "diabetes"), was first identified as a disease associated with "sweet urine," and excessive muscle loss in the ancient world. Elevated levels of blood glucose (hyperglycemia) lead to spillage of glucose into the urine, hence the term sweet urine. Normally, blood glucose levels are tightly controlled by insulin, a hormone produced by the pancreas. Insulin lowers the blood glucose level. When the blood glucose elevates (for example, after eating food), insulin is released from the pancreas to normalize the glucose level. In patients with diabetes, the absence or insufficient production of insulin causes hyperglycemia [1, 2].

What is impact of diabetes?

Over time, diabetes can lead to blindness, kidney failure, and nerve damage. Diabetes is also an important factor in accelerating the hardening and narrowing of the arteries (atherosclerosis), leading to strokes, coronary heart disease, and other blood vessel diseases. Diabetes affects 15 million people (about 8% of the population) in the United States. In addition, an estimated 12 million people in the United States have diabetes and don't even know it. From an economic perspective, the total annual economic cost of diabetes in 1997 was estimated to be 98 billion dollars in the United States. The per capita cost resulting from diabetes in 1997 amounted to $10,071, while to health care costs for people without diabetes incurred a per capita cost of $2,699. During this same year, 13.9 million days of hospital stay were attributed to diabetes, while 30.3 million physician office visits were diabetes related. Remember, these numbers reflect only the population in the United States. Globally, the statistics are staggering1. Diabetes is the third leading cause of death in the United States after heart disease and cancer [1, 3].

What causes diabetes?

Insufficient production of insulin (either absolutely or relative to the body's needs), production of defective insulin (which is uncommon), or the inability of cells to use insulin properly and efficiently leads to hyperglycemia and diabetes. This latter condition affects mostly the cells of muscle and fat tissues, and results in a condition known as "insulin resistance." This is the primary problem in type 2 diabetes. The absolute lack of insulin, usually secondary to a destructive process affecting the insulin producing beta cells in the pancreas. Glucose is an essential nutrient that provides energy for the proper functioning of the body cells. Carbohydrates are broken down in the small intestine and the glucose in digested food is then absorbed by the intestinal cells into the bloodstream, and is carried by the bloodstream to all the cells in the body where it is utilized. However, glucose cannot enter the cells alone and needs insulin to aid in its transport into the cells. Without insulin, the cells become starved of glucose energy despite the presence of abundant glucose in the bloodstream. In certain types of diabetes, the cells' inability to utilize glucose gives rise to the ironic situation of "starvation in the midst of plenty". The abundant, unutilized glucose is wastefully excreted in the urine.

Insulin is a hormone that is produced by specialized cells (β cells) of the pancreas. (The pancreas is a deep-seated organ in the abdomen located behind the stomach.) In addition to helping glucose enter the cells, insulin is also important in tightly regulating the level of glucose in the blood. After a meal, the blood glucose level rises. In response to the increased glucose level, the pancreas normally releases more insulin into the bloodstream to help glucose enter the cells and lower blood glucose levels after a meal. When the blood glucose levels are lowered, the insulin release from the pancreas is turned down. It is important to note that even in the fasting state there is a low steady release of insulin than fluctuates a bit and helps to maintain a steady blood sugar level during fasting. In normal individuals, such a regulatory system helps to keep blood glucose levels in a tightly controlled range. As outlined above, in patients with diabetes, the insulin is either absent, relatively insufficient for the body's needs, or not used properly by the body. All of these factors cause elevated levels of blood glucose (hyperglycemia) [2, 4, 5].

What are the different types of diabetes?

There are two major types of diabetes, called type 1 and type 2. Type 1 diabetes was also called insulin dependent diabetes mellitus (IDDM), or juvenile onset diabetes mellitus. In type 1 diabetes, the pancreas undergoes an autoimmune attack by the body itself, and is rendered incapable of making insulin. Abnormal antibodies found in the majority of patients with type 1 diabetes. Antibodies are proteins in the blood that are part of the body's immune system. The patient with type 1 diabetes must rely on insulin medication for survival [(4)]. In autoimmune diseases, such as type 1 diabetes, the immune system mistakenly manufactures antibodies and inflammatory cells that are directed against and cause damage to patients' own body tissues. It is believed that the tendency to develop these abnormal antibodies in type 1 diabetes is, in part, genetically inherited, though the details are not fully understood. (Exposure to certain viral infections (mumps and Coxsackie viruses) or other environmental toxins may serve to trigger abnormal antibody responses that cause damage cells where insulin is made. These antibodies can be measured in the majority of patients, and may help determine which individuals are at risk for developing type 1 diabetes. At present, the American Diabetes Association does not recommend general screening of the population for type 1 diabetes, though screening of high risk individuals, such as those with a first degree relative (sibling or parent) with type 1 diabetes should be encouraged. Type 1 diabetes tends to occur in young, lean individuals, usually before 30 years of age, however, older patients do present with this form of diabetes on occasion. This subgroup is referred to as latent autoimmune diabetes in adults (LADA). LADA is a slow, progressive form of type 1 diabetes. Of all the patients with diabetes, only approximately 10% of the patients have type 1 diabetes and the remaining 90% have type 2 diabetes [(3, 5)].

Type 2 diabetes was also referred to as non-insulin dependent diabetes mellitus (NIDDM), or adult onset diabetes mellitus (AODM). In type 2 diabetes, patients can still produce insulin, but do so relatively inadequately for their body's needs, particularly in the face of insulin resistance as discussed above. In many cases this actually means the pancreas produces larger than normal quantities of insulin. A major feature of type 2 diabetes is a lack of sensitivity to insulin by the cells of the body (particularly fat and muscle cells). Thus, larger quantities of insulin are produced as an attempt to get these cells to recognize

that insulin is, in fact, present. In addition to the problems with an increase in insulin resistance, the release of insulin by the pancreas may also be defective and suboptimal. In fact, there is a known steady decline in beta cell production of insulin in type 2 diabetes that contributes to worsening glucose control. (This is a major factor for many patients with type 2 diabetes who ultimately require insulin therapy.) Finally, the liver in these patients continues to produce glucose through a process called gluconeogenesis despite elevated glucose levels. This control of gluconeogenesis becomes compromised.

While it is said that type 2 diabetes occurs mostly in individuals over 30 years old and the incidence increases with age, we are seeing an alarming number patients with type 2 diabetes who are barely in their teen years. In fact, for the first time in the history of humans, type 2 diabetes is now more common than type 1 diabetes in childhood. Most of these cases are a direct result of poor eating habits, higher body weight, and lack of exercise. While there is a strong genetic component to developing this form of diabetes, there are other risk factors - the most significant of which is obesity. There is a direct relationship between the degree of obesity and the risk of developing type 2 diabetes, and this holds true in children as well as adults. It is estimated that the chance to develop diabetes doubles for every 20% increase over desirable body weight [5, 6].

What are diabetes symptoms?

Table 1: Contrasting features of type I diabetes mellitus & type II diabetes mellitus [5].

Sr. No.	FEATURES	TYPE I diabetes mellitus	TYPE II diabetes mellitus
1	Frequency	10-20%	80-90%
2	Age of onset	Early (below 35 yr.)	Late (above 30 yr.)
3	Type of onset	Abrupt and severe	Gradual and insidious
4	Weight	Normal	Obese / non-obese
5	HLA	Linked to HLA DR3, HLA DR4, HLA DQ	No HLA association
6	Family history	Less than 20 %	About 60 %
7	Genetic locus	Unknown	Chromosome 6
8	Pathogenesis	Autoimmune destruction of β- cells	Insulin resistance, impaired insulin secretion.
9	Islet cell antibodies	Yes	No
10	Blood insulin level	Reduced	Normal or increased

11	Islet cell changes	Insulitis, β-cell depletion	No Insulitis, later fibrosis of islets
12	Amylodosis	Infrequent	Common in chronic cases
13	Clinical management	Insulin, diet	Diet, exercise, oral drugs, insulin

The early symptoms of untreated diabetes are related to elevated blood sugar levels, and loss of glucose in the urine. High amounts of glucose in the urine can cause increased urine output and lead to dehydration. Dehydration causes increased thirst and water consumption. The inability to utilize glucose energy eventually leads to weight loss despite an increase in appetite. Some untreated diabetes patients also complain of fatigue, nausea and vomiting. Patients with diabetes are prone to developing infections of the bladder, skin, and vaginal areas. Fluctuations in blood glucose levels can lead to blurred vision. Extremely elevated glucose levels can lead to lethargy and coma (diabetic coma) [5, 8].

How is diabetes diagnosed?

The fasting blood glucose (sugar) test is the preferred way to diagnose diabetes. It is easy to perform and convenient. After the person has fasted overnight (at least 8 hours), a single sample of blood is drawn and sent to the laboratory for analysis. Normal fasting plasma glucose levels are less than 100 milligrams per deciliter (mg/dl). Fasting plasma glucose levels of more than 126 mg/dl on two or more tests on different days indicate diabetes. If the overnight fasting blood glucose is greater than 126 mg/dl on two different tests on different days, the diagnosis of diabetes is made. A random blood glucose test can also be used to diagnose diabetes. Random blood samples (if taken shortly after eating or drinking) may be used to test for diabetes when symptoms are present. A blood glucose level of 200 mg/dl or higher indicates diabetes, but it must be reconfirmed on another day with a fasting plasma6 glucose or an oral glucose tolerance test. When fasting blood glucose stays above 100mg/dl, but in the range of 100-126mg/dl, this is known as impaired fasting glucose (IFG). While patients with IFG do not have the diagnosis of diabetes, this condition carries with it its own risks and concerns, and is addressed elsewhere [2, 5, 8].

The oral glucose tolerance test:

Though not routinely used anymore, the oral glucose tolerance test (OGTT) is a gold standard for making the diagnosis of type 2 diabetes. It is still commonly used for diagnosing gestational diabetes. With an oral glucose tolerance test, the person fasts overnight (at least 8 but not more than 16 hours). Then first, the fasting plasma glucose is tested. After this test, the person receives 75 grams of glucose (100 grams for pregnant women). There are several methods employed by obstetricians to do this test, but the one described here is standard. Usually, the glucose is in a sweet-tasting liquid that the person drinks. Blood samples are taken at specific intervals to measure the blood glucose.

For the test to give reliable results, the person must be in good health (not have any other illnesses, not even a cold). Also, the person should be normally active (not lying down, for example, as an inpatient in a hospital) and should not be taking medicines that could affect the blood glucose. For 3 days before the test, the person should have eaten a diet high in carbohydrates (150- 200 grams per day). The morning of the test, the person should not smoke or drink coffee. The classic oral glucose tolerance test measures blood glucose levels 5 times over a period of 3 hours. Some physicians simply get a baseline blood sample followed by a sample 2 hours after drinking the glucose solution. In a person without diabetes, the glucose levels rise and then fall quickly. In someone with diabetes, glucose levels rise higher than normal and fail to come back down as fast. People with glucose levels between normal and diabetic have impaired glucose tolerance (IGT). People with impaired glucose tolerance do not have diabetes, but are at high risk for progressing to diabetes. Each year, 1-5% of people whose test results show impaired glucose tolerance actually eventually develop diabetes. Weight loss and exercise may help people with impaired glucose tolerance return their glucose levels to normal. In addition, some physicians advocate the use of medications, such as metformin (Glucophage), to help prevent/delay the onset of overt diabetes. Recent studies have shown that impaired glucose tolerance itself may be a risk factor for the development of heart disease. In the medical community, most physicians are now understanding that impaired glucose tolerance is nor simply a precursor of diabetes, but likely its own clinical disease entity that requires treatment and monitoring [4, 7].

Evaluating the results of the oral glucose tolerance test:

Glucose tolerance tests may lead to one of the following diagnoses:

Normal response: A person is said to have a normal response when the 2-hour glucose level is less than 140 mg/dl, and all values between 0 and 2 hours are less than 200 mg/dl.

Impaired glucose tolerance: A person is said to have impaired glucose tolerance when the fasting plasma glucose is less than 126 mg/dl and the 2-hour glucose level is between 140 and 199 mg/dl.

Diabetes: A person has diabetes when two diagnostic tests done on different days show that the blood glucose level is high.

Gestational diabetes: A woman has gestational diabetes when she has any two of the following: a 100g OGTT, a fasting plasma glucose of more than 95 mg/dl, a 1-hour glucose level of more than 180 mg/dl, a 2-hour glucose level of more than 155 mg/dl, or a 3-hour glucose level of more than 140 mg/dl [4, 7].

How is diabetes treated?

The major goal in treating diabetes is controlling elevated blood sugars (glucose) without causing abnormally low levels of blood sugar. Type 1 diabetes is treated with insulin, exercise, and a diabetic diet. Type 2 diabetes is first treated with weight reduction, a diabetic diet, and exercise. When these measures fail to control the elevated blood sugars, oral medications are used. If oral medications are still insufficient, insulin medications are considered. Adherence to a diabetic diet is an important aspect of controlling elevated blood sugar in patients with diabetes. The American Diabetes Association (ADA) has provided guidelines for a diabetic diet. The ADA diet is a balanced, nutritious diet that is low in fat, cholesterol, and simple sugars. The total daily calories are evenly divided into three meals. In the past two years, the ADA has lifted the absolute ban on simple sugars. Small amounts of simple sugars are allowed when consumed with a complex meal. Weight reduction and exercise are important treatments of diabetes. Weight reduction and exercise increase the body's sensitivity to insulin, thus helping to control blood sugar elevations [1, 7, 9].

Medications for type 2 diabetes:

All the information listed below applies to patients who are not pregnant or breastfeeding. At present the only recommended way of controlling diabetes in these situations is by diet,

exercise and insulin therapy. You should refer to your doctor if you are on these medications and are considering becoming pregnant, or if you have become pregnant while taking these medications.

Based on what is known, medications for type 2 diabetes are designed to:

- ✓ Increase the insulin output by the pancreas.
- ✓ Decrease the amount of glucose released from the liver.
- ✓ Increase the sensitivity (response) of cells to insulin.
- ✓ Decrease the absorption of carbohydrates from the intestine.
- ✓ Slow gastric emptying to delay the presentation of carbohydrates for absorption in the small intestine.

When selecting therapy for treatment of type 2 diabetes,consideration should be given to:

The magnitude of change in blood sugar control that each medication will provide. Other coexisting medical conditions (hypertension high cholesterol, etc.) It's important to remember that if a drug can provide more than one benefit (lower blood sugar and have a good effect on cholesterol, for example), it should be preferred. It's also important to bear in mind that the cost of drug therapy is relatively small compared to the cost of managing the long-term complications associated with poorly controlled diabetes [3, 6, 7].

Medications that increase the insulin output by the pancreas:

Historically, increasing the insulin output by the pancreas has been the major area targeted by medications used to treat type 2 diabetes. These medications belong to a class of drugs called sulfonylureas.

Table 2: Oral hypoglycaemic agents [10, 11]

First generation sulfonylureas:

Generic name	Dosage range	Duration of action	Common side effects
Chlorpropamide	100-500 mg	> 48 hr	Prolonged hypoglycaemia, cholestatic jaundice, hypersensitivity, alcohol flush
Tolbutamide	500-3000 mg	6-12 hr	Hypoglycaemia, hypersensitivity
Tolazamide	100-1000 mg	12-24 hr	Hypoglycaemia, hypersensitivity
Acetohexamide	500-1500 mg	12-24 hr	Hypoglycaemia, hypersensitivity

Second generation sulfonylureas:

Generic name	Dosage range	Duration of action	Common side effects
Glipizide	2.5-40 mg	12-24 hr	Hypoglycemia, hypersensitivity
Glyburide	5-20 mg	12-24 hr	Hypoglycemia, hypersensitivity
Glimeperide	1.8 mg	24 hr	Hypoglycemia, hypersensitivity
Glidazide	40-240 mg	12-24 hr	Hypoglycemia, hypersensitivity

Agents enhancing effects of insulin:

Generic name	Dosage range	Duration of action	Common side effects
Metformin (Obimet)	500-2500 mg	6-8 hr	GI upset; diarrhoea; possible resumption of ovulation in premenopausal anovolvutary patients; acidosis (if renal, liver, heart impairment present).
Rosiglitazone	4-8 mg	very long	Liver dysfunction, salt, water retention, edema, congestive heart failure etc.
Proglitazone	15-45 mg	very long	Renal and liver function studies should be done to monitor liver dysfunction, salt and water retention, edema, congestive heart failure.

Other oral agents:

Generic name	Dosage range	Duration of action	Common side effects
Repaglinide (Novonorm)	1.5-16 mg	2-6 hr	Hypoglycemia, arthralgia, leukopenia
Acarbose	25-300 mg	< 4 hr	Diarrhoea, abdominal discomfort, flatulence
Miglitol	25-300 mg	< 4 hr	Diarrhoea, abdominal discomfort, use not recommended when significant renal dysfunction present

New medications that effect glycemic control:

Symlin is the first in a new class of injected antihyperglycemic medications for use in patients with type 2 or type 1 diabetes treated with insulin. Pramlintide, the active ingredient in Symlin, is a synthetic analog of human amylin, a naturally occurring neuroendocrine

hormone synthesized from pancreatic beta cells that contributes to glucose control during the postprandial period. Amylin, similar to insulin, is absent or deficient in patients with diabetes. When used with insulin, this compound can help patients achieve improved glycemic control with additional benefits that cannot be realized with insulin alone. According to published data, Symlin reduces post meal blood sugar peaks, reduces glucose fluctuations throughout the day, enhances satiety (the sensation of fullness) leading to potential weight loss, and lowers mealtime insulin requirements. Studies have shown it improves A1C beyond the effect of insulin alone. Symlin is taken just prior to meals, three times a day. It is given in injection form and is indicated for Type 2 diabetes, as an adjunct treatment in patients who use mealtime insulin therapy and have failed to achieve desired glucose control despite optimal insulin therapy, with or without a concurrent sulfonylurea agent and/or metformin. Symlin is considered a therapy option in patients with insulin-using type 2 or type 1 diabetes that are unable to achieve adequate glycemic control despite individualized insulin management. Insulin-using patients with type 2 diabetes may also be taking concurrent sulfonylurea agent and/or metformin [11, 12].

Byetta (exenatide)**:**

Byetta (exenatide) is a new medication on the market that has it's origins in an interesting place--the Gila monster's saliva. Scientists studying this small lizard noted it could go a long time without eating. They found a substance in it's saliva that slowed stomach emptying, thus making the lizard feel fuller longer. This substance was similar in nature to a gut hormone found in humans known as GLP-1. Thus, the studies began. Ultimately, after modifying this hormone, exenatide (with the trade name Byetta) was developed. Byetta is the first in a new class of drugs for the treatment of type 2 diabetes called incretin mimetics. Byetta has been shown to have many of the same effects on sugar regulation as GLP-1, so it mimics the body's natural physiology for self-regulating blood sugar. Byetta is indicated as adjunctive therapy to improve glycemic control in patients with type 2 diabetes who are taking metformin, a sulfonylurea, or a combination of metformin and a sulfonylurea, but who have not achieved adequate sugar control. It enhances the way the insulin producing beta cells in the pancreas work. Insulin secretion increases only when blood sugars are high

and decreases as blood sugars approach normal. In addition to enhancing the normal physiology of the beta cell, Byetta suppresses glucose release from the liver, slows stomach emptying and the absorption of nutrients including carbohydrate, and reduces the food intake. Just like Symlin, Byetta is given by an injection, but it is given twice a day (usually before breakfast and dinner meals). It comes in a disposable pen form, and is available in 2 doses. The goal is to start with the lower dose for a month or so then move up to the higher dose if needed and if tolerated. Similar to Symlin, the main side effect is nausea, most likely due to its effects on stomach emptying. This medication is temperature sensitive and most is stored at 36-46 degrees F. The risk of hypoglycemia is still a possibility with Byetta especially when used in combination with sulfonylureas. Your physician may choose to decrease the dose of some of your other medications when initially evaluating how you respond to Byetta. Similar to Symlin, weight reduction is seen with Byetta in the majority of patients. This makes it particularly suitable for the typical patient with Type 2 diabetes who is also overweight [13, 15].

Combination medications:

Glyburide/metformin (Glucovance), rosiglitazone/metformin (Avandamet), and glipizide/metformin (Metaglip) are 3 relatively new combination pills that are on the market to treat diabetes. Glucovance combines glyburide with metformin in varying doses. Avandamet is a combination of varying doses of Avandia and metformin. And Metaglip is a combination pill containing glipizide and metformin in varying strengths. The benefit to these agents is fewer pills to take, hopefully leading to better compliance. While they work well, I personally like to give patients individual medications, until I know what doses are working, and then switch to a combination pill once the patient has been stable on the doses of individual medications for a period of time [7, 16, 17].

Treatment of diabetes with insulin:

Insulin is the mainstay of treatment for patients with type 1 diabetes. Insulin is also important in type 2 diabetes when blood glucose levels cannot be controlled by diet, weight loss, exercise, and oral medications. Ideally, insulin medication should be administered in a manner that mimics the natural pattern of insulin secretion by a healthy pancreas. The

complex pattern of insulin secretion by the pancreas is difficult to duplicate. Still, adequate blood glucose control can be achieved with careful attention to diet, regular exercise, home blood glucose monitoring, and multiple insulin injections throughout the day.

Table 3: Insulin preparations [14, 19, 20]

Sr. No.	Types of insulin	Activity		
		Onset	**Peak**	**Duration**
Rapid-acting insulin				
1	Insulin Injection (regular)	30-60 min	2-4 hr	6-8 hr
2	Insulin lispro (insulin analog)	45 min	1 h	3.5-4.5 hr
3	Insulin aspart solution	5-10 min	30-60 min	2-3 hr
Intermediate-acting insulin				
4	Isophane insulin suspension (NPH)	1-2 hr	6-12 hr	18-24 hr
5	Insulin zinc suspension (Lente)	1-2.5 hr	6-12 hr	18-24 hr
Long-acting insulin				
6	Insulin glargine solution	30-60 min	None	8 hr
7	Extended insulin zinc suspension (Ultralente)	4-8 hr	8-20 hr	24-48 hr
Mixed insulin				
8	Isophane insulin suspension and insulin injections (NPH)	30-60 min	2-4 hr	6-8 hr
9	Isophane insulin suspension and insulin injection	30-60 min	2-4 hr	6-8 hr
High-potency insulin				
10	Insulin injection concentrated	-	-	24 hr

The future of pancreas transplantation:

Ultimately, the goal in the management of type 1 diabetes is to provide insulin therapy in a manner that mimics the natural pancreas. Perhaps the closest therapy available at time is a transplant of the pancreas. Several approaches to pancreatic transplantation are currently being studied, including the whole pancreas and isolated islet cells (these groups of cells contain beta cells that are responsible for insulin production). Data available from 1995 indicates that almost 8,000 patients underwent pancreatic transplantation. Most patients

undergo pancreatic transplantation at the time of kidney transplantation for diabetic kidney disease. Transplantation is not without risk. Both the surgery itself and the immunosuppression that must occur afterward pose significant risks to the patient. For these reasons, the kidney and pancreas are usually transplanted at the same time. At present, there is disagreement about whole pancreas transplantation in patients not currently requiring kidney transplantation. The issue of whether the benefits outweigh the risks in these patients is under debate. There is also a chance that diabetes will occur in the transplanted pancreas. Selectively transplanting islet cells is an interesting alternative to whole pancreas transplantation. However, the concern over rejection remains. Attempts to disguise the islet cells in tissues that the body won't reject (for example, by surrounding the islet cells by the patient's own cells and then implanting them) are underway. In addition, researchers are exploring artificial barriers that can surround the islet cells, provide protection against rejection, and still allow insulin to enter the bloodstream [19, 21].

CONCLUSION:

Diabetes is a chronic condition associated with abnormally high levels of sugar (glucose) in the blood. Insulin produced by the pancreas lowers blood glucose. Absence or insufficient production of insulin causes diabetes. The two types of diabetes are referred to as type 1 (insulin dependent) and type 2 (non-insulin dependent). Symptoms of diabetes include increased urine output, thirst and hunger as well as fatigue. Diabetes is diagnosed by blood sugar (glucose) testing. The major complications of diabetes are both acute and chronic. Acutely: dangerously elevated blood sugar, abnormally low blood sugar due to diabetes medications may occur.

Chronically: disease of the blood vessels (both small and large) which can damage the eye, kidneys, nerves, and heart may occur. Diabetes treatment depends on the type and severity of the diabetes. Type 1 diabetes is treated with insulin, exercise, and a diabetic diet. Type 2 diabetes is first treated with weight reduction, a diabetic diet, and exercise. When these measures fail to control the elevated blood sugars, oral medications are used. If oral medications are still insufficient, insulin medications are considered.

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