



ISSN 2231-0541

PHARMANEST

An International Journal of Advances in Pharmaceutical Sciences

Volume 4 | Issue 1 | January-February 2013 | Pages 16-43

Review Article

A DETAILED REVIEW ON DIABETES MELLITUS AND ITS TREATMENT IN ALLOPATHIC AND ALTERNATIVE SYSTEMS

N.V. SATHEESH MADHAV, S.BHATTACHARYA, S.N.UNIYAL, **ANUJ NAUTIYAL***

Faculty of Pharmacy, Dehradun Institute of Technology, Dehradun, Uttarakhand, India

Author for Correspondence: anujnautiyal@rediffmail.com

Received: 28 -01-2013

Revised: 07-02-2013

Accepted: 09-02-2013

Available online: 01-03-2013

ABSTRACT

The current aim of the review is to provide a complete basic to updated knowledge in diabetes mellitus because in India the current status of diabetes was occupying 4.164% of total Indian population and the figure will increase to two-fold by 2030 as per prediction of world diabetes foundation. The review article encompasses epidemiology, etiology, symptoms, method for diagnosis and various system for treating this disease such as allopathic, ayurvedic, homeopathic, acupuncture and yoga. This article will give clear cut idea to reader about the existing therapy that are available for treatment of diabetes mellitus in one stretch platform.

Key Words: Diabetes mellitus, allopathic, ayurvedic, homeopathic, Yoga

INTRODUCTION

Diabetes mellitus (DM) is a group of metabolic disorders of fat, carbohydrate, and protein metabolism that results from defects in insulin secretion, insulin action (sensitivity), or both^[1]. Four major forms of diabetes have been classified: type 1, type 2, gestational (GDM), and other specific types (secondary DM). Type 1 is insulin-dependent diabetes mellitus (IDDM), juvenile-onset or ketone-prone diabetes. It commonly occurs in children, and in adults ≤ 30 years old, but it may also occur at any age, with a fairly abrupt onset; however, newer antibody tests have allowed for the identification of more people with a slower onset adult form of type 1 diabetes mellitus called latent autoimmune diabetes of the adult (LADA). The distinguishing characteristic of a patient with type 1 diabetes is that, insulin production and secretion is destroyed and predisposed to ketoacidosis-accumulation of ketone bodies in body tissues and fluid. Therefore, these patients are dependent on exogenous insulin ^{[2],[3]}.

Type 2 is non-insulin-dependent diabetes mellitus (NIDDM), or adult-on-set diabetes. It is usually diagnosed in adults ≥ 30 years old, but may occur at any age. Now, because of the epidemic of obesity and inactivity in children, type 2 diabetes mellitus is occurring at younger and younger ages, it has been diagnosed in children as young as 2 years of age who have a family history of diabetes. It is characterized by peripheral insulin resistance with an insulin-secretory defect that varies in severity, not prone to ketosis except during

periods of severe physical stress such as infections, trauma or surgery ^{[2],[4]}.

Gestational diabetes mellitus (GDM) is defined as any degree of glucose intolerance with onset or first recognition during pregnancy occurs in approximately 2%-4% of pregnant women, generally during the second or third trimester and the occurrence of GDM increases the risk for developing type 2 diabetes^[2].

Other specific types of diabetes, previously called **secondary diabetes**, are caused by other illnesses or medications. Depending on the primary process involved (e.g., destruction of pancreatic beta cells or development of peripheral insulin resistance), these types of diabetes behave similarly to type 1 or type 2 diabetes. The most common are diseases of the pancreas that destroy the pancreatic beta cells (e.g., hemochromatosis, pancreatitis, cystic fibrosis, pancreatic cancer), hormonal syndromes that interfere with insulin secretion (e.g., pheochromocytoma) or cause peripheral insulin resistance (e.g., acromegaly, Cushing syndrome, pheochromocytoma), and diabetes induced by drugs (e.g., phenytoin, glucocorticoids, estrogens)^[4].

EPIDEMIOLOGY

The world prevalence of diabetes among adults (aged 20-79 years) will be 6.4%, affecting 285 million adults, in 2010, and will increase to 7.7% and 439 million adults by 2030. Between 2010 and 2030, there will be a 69% increase in numbers of adults with diabetes in developing countries and a 20% increase in developed

countries^{[5],[6]}. The prevalence of diabetes in India is estimated to be 1-5%. The prevalence of diabetes in persons 45 to 64 years of age is 7 percent, but the proportion increases significantly in persons 65 years of age or older. Also, the number of diabetics is projected to rise from 15 million in 1995 to 57 million by the year 2025 making it the country with the highest number of diabetics in the world ^[7].

An estimated 285 million people, corresponding to 6.4% of the world's adult population, will live with diabetes in 2010. The number is expected to grow to 438 million by 2030, corresponding to 7.8% of the adult population. 70% of the current cases of diabetes occur in low- and middle income countries. With an estimated 50.8 million people living with diabetes, India has the world's largest diabetes population, followed by China with 43.2 million. The largest age group currently affected by diabetes is between 40-59 years. By 2030 this "record" is expected to move to the 60-79 age group with some 196 million cases. The number of deaths attributable to diabetes in 2010 shows a 5.5% increase over the estimates for the year 2007. This increase is largely due to a 29% increase in the number of deaths due to diabetes in the North America & Caribbean Region, a 12% increase in the South East Asia Region and an 11% increase in the Western Pacific Region. Expressed in International Dollars (ID), which correct for differences in purchasing power, estimated global expenditures on diabetes will be at least ID 418 billion in 2010, and at least ID 561 billion in 2030. An estimated average of ID 878 per person will be spent on diabetes in

2010 globally. The World Health Organization (WHO) predicted net losses in national income from diabetes and cardiovascular disease of ID 557.7 billion in China, ID 303.2 billion in the Russian Federation, ID 336.6 billion in India, ID 49.2 billion in Brazil and ID 2.5 billion in Tanzania (2005 ID), between 2005 and 2015^[8].

ETIOLOGY OF TYPE 1 DM

- **Genetics**-Human leukocyte antigen (HLA) system are considered specific markers of type 1 DM susceptibility,
- Exposure to **cow's milk** and increased risk for IDDM in genetically susceptible individuals
- **Viral infections** (e.g. Coxsackie B virus, rubella, Cytomegalovirus, Epstein-Barr Virus, influenza, hepatitis A, varicella zoster, mumps, measles, rotavirus, polio, and Coxsackie A virus).
- **Autoimmunity** -It is the body's defense system attacks the cells of pancreas that make insulin are destroyed, and therefore they are no longer capable of making insulin ^{[9],[10],[11],[26]}.

ETIOLOGY OF TYPE 2 DM

- **Impaired insulin release** - basal secretion of insulin is often normal, but the rapid release of insulin follows a meal is greatly impaired, resulting in failure of normal handling of a carbohydrate load.
- **The genetic factor** is very strong in type 2 diabetes, with a history of diabetes present in about 50% of first degree relatives.

- **Insulin resistance**-It is a defect in the tissue response to insulin is believed to play a major role. This phenomenon is called insulin resistance and is caused by defective insulin receptors on the target cells. Insulin resistance occurs in association with obesity and pregnancy. In normal individuals who become obese or pregnant, the B cells secrete increased amounts of insulin to compensate. Patients who have genetic susceptibility to diabetes cannot compensate because of their inherent defect in insulin secretion.
- **Inflammation**- It is a defect in the tissue response to excess insulin is believed to play a major role. This phenomenon is caused by the increase in insulin resistance. Macrophages, found in white blood cells in the bone marrow, are key players in the immune response. When these immune cells get into tissues, such as adipose (fat) or liver tissue, they release cytokines, which are chemical messenger molecules used by immune and nerve cells to communicate. These cytokines cause the neighboring liver, muscle or fat cells to become insulin resistant, which in turn can lead to Type 2 diabetes [12].

ETIOLOGY OF SECONDARY DIABETES

- Cushing's syndrome
- Pancreatic disease- hemochromatosis, pancreatitis, cystic fibrosis, pancreatic cancer
- Genetic mutation
- Thiazide diuretics

- Corticosteroids [2],[13]

PATHOPHYSIOLOGY OF DIABETES MELLITUS

In Type 1 diabetes, the disease is caused by the destruction of beta cells in the pancreas. These cells are responsible for producing the body's supply of insulin, the hormone that controls blood sugar levels. People with Type 1 diabetes produce no insulin. This hormone is a chemical signal that stimulates cells to take up sugars from the blood. When no insulin is present, sugars remain concentrated in the blood and cells cannot use the sugar as an energy source.

Type 2 diabetes develops as the result of an abnormal metabolic process called insulin resistance. People with this type of diabetes can make their own supply of insulin, but their bodies do not respond normally to the insulin they make. Cells become insulin resistant, which means they are unable to respond to insulin by taking up sugars from the blood. Insulin resistance does not have a single cause; it is thought that there are several contributing factors, including genetics, poor diet, sedentary lifestyle, and obesity.

With sugar concentrated in the blood as a result of Type 1 or Type 2 diabetes, the body's fluid balance is disrupted. Fluid is drawn away from the tissues and into the blood to offset the high sugar concentration. Urination increases to aid in excreting excess sugar in the urine. Hunger and fatigue increase because the body's cells cannot utilize sugar for energy. The body's stores of fat and protein are used as an alternative energy source, leading to weight loss. In the case of Type 1 diabetes there is a risk of death occurring if the

condition is not treated promptly, as a result of kidney failure and dehydration.

If blood sugar levels are poorly managed, both Type 1 and Type 2 diabetes can increase the risk of a number of diseases and complications. These include atherosclerosis, coronary artery disease, kidney disease, nerve damage, vision loss, sexual dysfunction, high cholesterol, hypertension, and genitourinary infections.

Most of these long term complications develop because chronically high levels of blood sugar are pro-inflammatory. Over time, this inflammation becomes chronic and causes damage to blood vessels and nerves, particularly in the peripheral areas of the body such as legs and feet, and in the delicate blood vessels of the eyes. Complications such as cardiovascular disease are related to cholesterol levels and blood pressure as well as blood sugar levels [14].

PANCREATIC PATHOLOGY [31]

The pathological picture in the pre-diabetic pancreas in type1 diabetes is characterized by:

- Insulinitis (see fig.1) – that is, infiltration of the islets with mononuclear cells containing activated macrophages , helper cytotoxic and suppressor T lymphocytes , natural killer cells and B lymphocytes
- The initial patchiness of this lesion, with , until a very late stage , lobules containing heavily infiltrated islets seen adjacent to unaffected lobules
- The striking beta cell specificity of the destructive process, with the glucagon and other hormone-secreting cells in the islet invariably remaining intact.

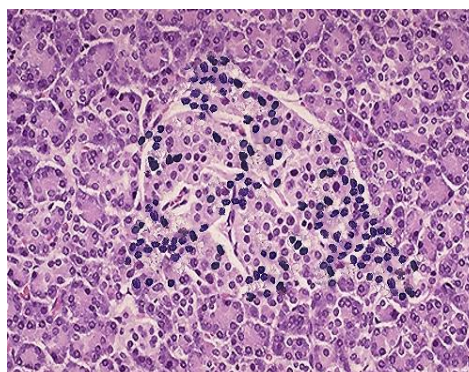
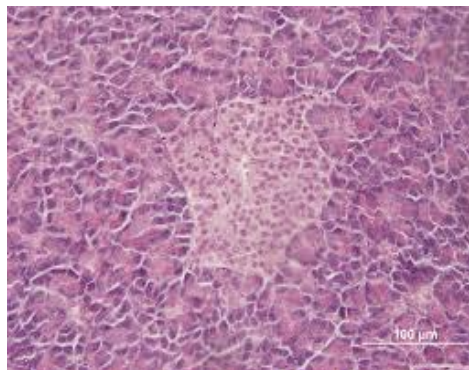


Fig. 1 a) T.s of Normal pancreas

b) Insulinitis, showing chronic inflammatory cell infiltrate in a pancreatic islet.

SYMPTOMS OF DIABETES

Even though the cause of both Type 1 and Type 2 diabetes is high blood sugar, the symptoms of each type are not always the same. In general, both types can cause blurry vision, fatigue, increased thirst and hunger, and increased frequency of urination. However, the patterns of symptoms experienced are slightly different, and symptoms develop at different rates. For Type 1 diabetes, symptoms include fatigue, increased thirst and urination, nausea and vomiting, and weight loss. These symptoms are usually of sudden, rapid onset. Symptoms of Type 2 diabetes are of very gradual onset, often over a period of several

years. In some people, the progress of the disease is so gradual that it may be asymptomatic. The most common symptoms of Type 2 diabetes are fatigue, blurred vision, increased thirst, increased urination, and increased appetite [14].

COMPLICATIONS

People with diabetes may experience many serious, long-term complications. Some of these complications begin within months of the onset of diabetes, although most tend to develop after a few years. Most of the complications are progressive. The more strictly people with diabetes are able to control the levels of sugar in the blood; the less likely it is that these complications will develop or become worse.

Most complications are the result of problems with blood vessels. High sugar levels over a long time cause narrowing of both the small and large blood vessels. The narrowing reduces blood flow to many parts of the body, leading to problems. There are several causes of blood vessel narrowing. Complex sugar-based substances build up in the walls of small blood vessels, causing them to thicken and leak. Poor control of blood sugar levels also tends to cause the levels of fatty substances in the blood to rise, resulting in atherosclerosis and decreased blood flow in the larger blood vessels. Atherosclerosis is between 2 and 6 times more common in people with diabetes than in people who do not have diabetes and tends to occur at younger ages.

Over time, elevated levels of sugar in the blood and poor circulation can harm the heart,

brain, legs, eyes, kidneys, nerves, and skin, resulting in angina, heart failure, strokes, leg cramps on walking (claudication), poor vision, kidney failure, damage to nerves (neuropathy), and skin breakdown. Heart attacks and strokes are more common among people with diabetes.

Poor circulation to the skin can lead to ulcers and infections and causes wounds to heal slowly. People with diabetes are particularly likely to have ulcers and infections of the feet and legs. Too often, these wounds heal slowly or not at all, and amputation of the foot or part of the leg may be needed.

People with diabetes often develop bacterial and fungal infections, typically of the skin. When the levels of sugar in the blood are high, white blood cells cannot effectively fight infections. Any infection that develops tends to be more severe.

Damage to the blood vessels of the eye can cause loss of vision (diabetic retinopathy). Laser surgery can seal the leaking blood vessels of the eye and prevent permanent damage to the retina. Therefore, people with diabetes should have yearly eye examinations to check for damage.

The kidneys can malfunction, resulting in kidney failure that may require dialysis or kidney transplantation.

Damage to nerves can manifest in several ways. If a single nerve malfunctions, an arm or leg may suddenly become weak. If the nerves to the hands, legs, and feet become damaged (diabetic polyneuropathy), sensation may become abnormal, and tingling or burning pain and weakness in the arms and legs may develop. Damage to the nerves of the skin

makes repeated injuries more likely because people cannot sense changes in pressure or temperature.

The Foot in Diabetes

Diabetes causes many changes in the body. The following changes in the feet are common and difficult to treat. Damage to the nerves (neuropathy) affects sensation to the feet, so that pain is not felt. Irritation and other forms of injury may go unnoticed. An injury may wear through the skin before any pain is felt. Changes in sensation alter the way people with diabetes carry weight on their feet, concentrating weight in certain areas so that

calluses form. Calluses (and dry skin) increase the risk of skin breakdown. Diabetes can cause poor circulation in the feet, making ulcers more likely to form when the skin is damaged and making the ulcers slower to heal. Because diabetes can affect the body's ability to fight infections, a foot ulcer, once it forms, easily becomes infected. Because of neuropathy, people may not feel discomfort from the infection until it becomes serious and difficult to treat, leading to gangrene. People with diabetes are more than 30 times more likely to require amputation of a foot or leg than are people without diabetes ^[15].

Table.1. Long-Term Complications of Diabetes ^[15]

Tissue or Organ Affected	Changes occur	Complications
Blood vessels	Fatty material (atherosclerotic plaque) builds up and blocks large or medium-sized arteries in the heart, brain, legs, and penis. The walls of small blood vessels are damaged so that the vessels do not transfer oxygen to tissues normally, and the vessels may leak.	Poor circulation causes wounds to heal poorly and can lead to heart disorders, strokes, gangrene of the feet and hands, erectile dysfunction (impotence), and infections.
Eyes	The small blood vessels of the retina are damaged.	Decreased vision and, ultimately, blindness occur.
Kidney	Blood vessels in the kidney thicken. Protein leaks into urine. Blood is not filtered normally	The kidneys malfunction, ultimately kidney failure occurs.
Nerves	Nerves are damaged because glucose is not metabolized normally and because the blood supply is inadequate.	Legs suddenly or gradually weaken. People have reduced sensation, tingling, and pain in their hands and feet.
Autonomic nervous system	The nerves that control blood pressure and digestive processes are damaged.	Swings in blood pressure occur. Swallowing becomes difficult.

		Digestive function is altered, and sometimes bouts of diarrhea occur. Erectile dysfunction develops.
Skin	Blood flow to the skin is reduced, and sensation is decreased, resulting in repeated injury	Sores and deep infections (diabetic ulcers) develop. Healing is poor.
Blood	White blood cell function is impaired	People become more susceptible to infections, especially of the urinary tract and skin.
Connective tissue	Glucose is not metabolized normally, causing tissues to thicken or contract.	Carpal tunnel syndrome and Dupuytren's contracture [15]

DIAGNOSIS OF DM

Criteria for the Diagnosis of Diabetes

Mellitus and Impaired Glucose

Homeostasis

- Diabetes mellitus--positive findings from any two of the following tests on different days:

Symptoms of diabetes mellitus* plus casual† plasma glucose concentration ≥ 200 mg per dL (11.1 mmol per L)

or

FPG ≥ 126 mg per dL (7.0 mmol per L)

or

2hrPPG ≥ 200 mg per dL

(11.1 mmol per L) after a 75-g glucose load

Or

Glycated hemoglobin (also known as glycohemoglobin, glycosylated hemoglobin or HbA_{1c}) is used to monitor treatment in patients with diabetes mellitus; however, it is not recommended for routine diagnosis of this condition because of a lack of standardization of tests and results.

Glycated hemoglobin (HbA_{1c}) at or above 6.5. An HbA_{1c} of less than 6% is considered normal glucose tolerance (using an A_{1c} assay that has been

standardized to the DCCT normal range of 4-6%). However, an HbA_{1c} of 6-6.4% is neither normal glucose tolerance nor diabetes. Hyperglycemia exists across a continuum in an individual, generally gradually increasing over time. (This criterion was recommended by the American Diabetes Association in 2010; it has yet to be adopted by the WHO).

- **Impaired glucose homeostasis**

Impaired fasting glucose: FPG from 110 to <126 (6.1 to 7.0 mmol per L)

Impaired glucose tolerance: 2hrPPG from 140 to <200 (7.75 to <11.1 mmol per L)

- **Normal**

FPG <110 mg per dL (6.1 mmol per L)
2hrPPG <140 mg per dL (7.75 mmol per L)

†--Casual is defined as any time of day without regard to time since last meal.

*--Symptoms include polyuria, polydipsia or unexplained weight loss.

FPG=fasting plasma glucose; 2hrPPG=two-hour postprandial glucose [16],[17].

Diagnosis of Gestational Diabetes Mellitus

- 1) A screening test is done between 24 and 28 weeks of gestation in all women except those meeting the following criteria: less than 25 years of age, normal body weight, no first-degree relative with DM, and not Hispanic, African-American, Asian or Native American. The screening is done using a 50 g oral glucose load followed by a 1-hr plasma measurement. A value \geq 140 mg/dl leads to a follow-up diagnostic test using a 100 g glucose load over 3 hrs.
- 2) Following the 100 g oral glucose load, diagnosis of GDM may be made if two plasma glucose values equal or exceed the following [2]:

Fasting	105 mg/dl
1-hr	190 mg/dl
2-hr	165 mg/dl
3-hr	145 mg/dl

RECOMMENDATIONS FOR DIABETES SCREENING OF ASYMPTOMATIC PERSONS

Test at age 45; repeat every three years:

Patients 45 years of age or older

Test before age 45; repeat more frequently than every three years if the patient has one or more of the following risk factors:

- Obesity: \geq 120% of desirable body weight or BMI \geq 27 kg per m²

- First-degree relative with diabetes mellitus
 - Member of high risk-ethnic group (black, Hispanic, Native American, Asian)
 - History of gestational diabetes mellitus or delivering a baby weighing more than 4,032 g (9 lb) i.e.,
Hypertensive (\geq 140/90 mm Hg)
 - HDL cholesterol level \leq 35 mg per dL (0.90 mmol per L) and/or triglyceride level \geq 250 mg per dL (2.83 mmol per L)
 - History of IGT or IFG on prior testing
- BMI=body mass index; HDL=high density lipoprotein; IGT=impaired glucose tolerance; IFG=impaired fasting glucose [16],[17].

Differential diagnosis for type 1 and type 2 diabetes mellitus

Islet-cell (IA2), anti-GAD65, and anti-insulin auto antibodies can be present in early type 1, but not type 2 diabetes.

I. Measurements of islet-cell (IA2) autoantibodies within 6 months of diagnosis can help differentiate type 1 and type 2 diabetes. These titers decrease after 6 months.

II. Anti-GAD65 antibodies can be present at diagnosis and are persistently positive over time. These are suggestive of type 1 diabetes [18],[19].

Treatment of Diabetes Mellitus

Treatment of diabetes involves diet, exercise, education, and, for most people, drugs. If people with diabetes strictly control blood sugar levels, complications are less likely to develop. The goal of diabetes treatment, therefore, is to keep blood sugar levels within the normal range as much as

possible. Treatment of high blood pressure and cholesterol levels can prevent some of the complications of diabetes as well. A low dose of aspirin taken daily is also helpful.

People with diabetes benefit greatly from learning about the disorder, understanding how diet and exercise affect their blood sugar levels, and knowing how to avoid complications. A nurse trained in diabetes education can provide information about managing diet, exercising, monitoring blood sugar levels, and taking drugs.

People with diabetes should always carry or wear a medical identification (such as a bracelet or tag) to alert health care practitioners to the presence of diabetes. This information allows health care practitioners to start life-saving treatment quickly, especially in the case of injury or altered mental status.

Diet management is very important in people with both types of diabetes. Doctors recommend a healthy, balanced diet and efforts to maintain a healthy weight. Some people benefit from meeting with a dietitian to develop an optimal eating plan.

People with type 1 diabetes who are able to maintain a healthy weight may be able to avoid the need for large doses of insulin. People with type 2 diabetes may be able to avoid the need for all drugs by achieving and maintaining a healthy weight. Some people who have been unsuccessful in losing weight through diet and exercise may take drugs to help them lose weight or may even undergo stomach reduction surgery.

In general, people with diabetes should not eat much sweet food. They should also try to

eat meals on a regular schedule. Long periods between eating should be avoided. People with diabetes also tend to have high levels of cholesterol in the blood, so limiting the amount of saturated fat in the diet is important. Drugs may also be needed to help control the level of cholesterol in the blood.

Appropriate amounts of exercise can also help people control their weight and maintain blood sugar levels within the normal range. Because blood sugar levels go down during exercise, people must be alert for symptoms of low blood sugar. Some people need to eat a small amount of food with sugar during prolonged exercise, decrease their insulin dose, or both. People with diabetes should stop smoking and consume only moderate amounts of alcohol (up to one drink per day for women and two for men).

Diabetic ketoacidosis is a medical emergency, because it can cause coma and death. Hospitalization, usually in an intensive care unit, is necessary. Large amounts of fluids are given intravenously along with electrolytes, such as sodium, potassium, chloride, and phosphate, to replace those fluids and electrolytes lost through excessive urination. Insulin is generally given intravenously so that it works quickly and the dose can be adjusted frequently. Blood levels of sugar, ketones, and electrolytes are measured every few hours. Doctors also measure the blood's acid level. Sometimes, additional treatments are needed to correct a high acid level. However, controlling the levels of sugar in

the blood and replacing electrolytes usually allow the body to restore the normal acid-base balance.

Nonketotic hyperglycemic-hyperosmolar coma is treated much like diabetic ketoacidosis. Fluids and electrolytes must be replaced. The levels of sugar in the blood must be restored to normal levels gradually to avoid sudden shifts of fluid into the brain. The blood sugar levels tend to be more easily controlled than in diabetic ketoacidosis, and blood acidity problems are not severe.

Insulin Replacement Therapy

People with type 1 diabetes almost always require insulin therapy, and many people with type 2 diabetes require it as well. Insulin is injected. It currently cannot be taken by mouth because insulin is destroyed in the stomach. A nasal spray form of insulin was available but has been discontinued. New forms of insulin, such as forms that can be taken by mouth or applied to the skin, are being tested.

Insulin is injected under the skin into the fat layer, usually in the arm, thigh, or abdominal wall. Small syringes with very thin needles make the injections nearly painless. An air pump device that blows the insulin under the skin can be used for people who cannot tolerate needles. An insulin pen, which contains a cartridge that holds the insulin, is a convenient way for many people to carry insulin, especially for people who take several injections a day outside the home. Another device is an insulin pump, which pumps insulin continuously from a reservoir through a small needle left in the skin. Additional

doses of insulin can be released at programmed times, or release can be triggered as needed. The pump more closely mimics the way the body normally produces insulin. For some people, the pump offers an added degree of control, whereas others find wearing the pump annoying or develop sores at the needle site.

Insulin is available in three basic forms, divided by speed of onset and duration of action:

- Rapid-acting insulin, such as regular insulin, is fast and short acting. Regular insulin reaches its maximum activity in 2 to 4 hours and works for 6 to 8 hours. Lispro, aspart, and glulisine insulins, special types of regular insulin, are the fastest of all, reaching maximum activity in about 1 hour and working for 3 to 5 hours. Rapid-acting insulin is often used by people who take several daily injections and is injected 15 to 20 minutes before meals or just after eating.
- Intermediate-acting insulin (such as insulin zinc suspension, lente, or isophane insulin suspension) starts to work in 1 to 3 hours, reaches its maximum activity in 6 to 10 hours, and works for 18 to 26 hours. This type of insulin may be used in the morning to provide coverage for the first part of the day or in the evening to provide coverage during the night.
- Long-acting insulin (such as extended insulin zinc suspension, ultra-lente, or glargine) has very little effect in the first few hours but provides coverage

for 20 to 36 hours depending on which of these types is used.

Insulin preparations are stable at room temperature for months, allowing them to be carried, brought to work, or taken on a trip. Insulin should not, however, be exposed to extreme temperatures.

The choice of insulin is complex. The following factors are considered before deciding which insulin is best:

How willing and able people are to monitor their blood sugar levels and adjust the insulin dosage

- How varied daily activity is
- How adept people are at learning about and understanding the disorder
- How stable blood sugar levels are during the day and from day to day

The easiest regimen to follow is a single daily injection of intermediate-acting insulin. However, such a regimen provides the least control over blood sugar levels and is, therefore, rarely the best approach. Stricter control may be achieved by combining two insulins—a rapid-acting and an intermediate-acting insulin—in one morning dose. This combination requires more skill, but it offers people greater opportunity to adjust the blood sugar levels. A second injection of one insulin or both may be taken at dinner or at bedtime. Strictest control is usually achieved by injecting a rapid-acting and intermediate-acting insulin in the morning and evening along with several additional injections of rapid-acting insulin during the day. Adjustments can be made as insulin needs

change. Measuring blood sugar levels at various times during the day helps determine the adjustment. Although this regimen requires the most knowledge of the disorder and attention to the details of treatment, it is considered the best option for most people who are treated with insulin, especially people with type 1 diabetes.

Some people, especially older people, take the same amount of insulin every day. Other people adjust the insulin dose daily depending on their diet, exercise, and blood sugar patterns. In addition, insulin needs may change if people gain or lose weight or experience emotional stress or illness, especially infection.

Over time, some people develop resistance to insulin. Because the injected insulin is not exactly like the insulin the body manufactures, the body can produce antibodies to the insulin. Although this is less common with newer insulin preparations, these antibodies may interfere with the insulin's activity, requiring very large doses.

Insulin injections can affect the skin and underlying tissues. An allergic reaction, which occurs rarely, produces pain and burning, followed by redness, itchiness, and swelling around the injection site for several hours. More commonly, the injections either cause fat deposits, making the skin look lumpy, or destroy fat, causing indentation of the skin. Many people rotate the injection sites, for example, using the thigh one day, the stomach another, and an arm the next, to avoid these problems ^[15].

Table.2.Various Insulin Administered Subcutaneously [2],[20]

Type of Insulin	Onset (hrs)	Peak (hrs)	Effective Duration (hrs)
Rapid-acting			
Aspart	15-20 min	1-2	3-4
Lispro	15-30 min	1-2	3-5
Glulisine	15-30 min	1-2	3-5
Short-acting			
Regular	0.5-1.0	2-3	3-6
Intermediate-acting			
NPH	2-4	4-6	10-16
Lente	3-4	6-12	12-18
Long-acting			
Ultralente	6-10	10-16	18-20
Glargine	4-5	-	20-24

Oral Antihyperglycemic Drugs

Oral antihyperglycemic drugs can often lower blood sugar levels adequately in people with type 2 diabetes. However, they are not effective in type 1 diabetes. There are several types. Sulfonylureas (for example, glyburide) and meglitinides (for example, repaglinide) stimulate the pancreas to produce more insulin (insulin secretagogues). Biguanides (for example, metformin) and thiazolidinediones (for example, rosiglitazone) do not affect the release of insulin but increase the body's

response to it (insulin sensitizers). Doctors may prescribe one of these drugs alone or with a sulfonylurea drug. Another class of drug is the glucosidase inhibitors, such as acarbose , which work by delaying absorption of glucose in the intestine.

Oral antihyperglycemic drugs are usually prescribed for people with type 2 diabetes if diet and exercise fail to lower the levels of sugar in the blood adequately. The drugs are sometimes taken only once a day, in the morning, although some people need two or three doses. More than one type of oral drug may be used if one is not adequate. If oral antihyperglycemic drugs cannot control blood sugar levels well enough, insulin

injections alone or in combination with the oral drugs may be needed [15].

Table.3. Various Oral hypoglycemic agents [2],[21]

Drug	Initial daily dose	Maximum dose (mg/day)	Duration of Action
Sulfonylureas			
Acetohexamide	250 mg	1500	Up to 16 hrs
Chlorpropamide	100 mg	500	Up to 72 hrs
Tolazamide	100 mg	1000	Up to 24 hrs
Tolbutamide	250-500 mg	3000	Up to 12 hrs
Glipizide	5 mg	40	Up to 20 hrs
Glyburide	2.5 mg	20	Up to 24 hrs
Glimepiride	1-2 mg	8	24 hrs
Short-acting insulin secretagogues			
Nateglinide	180-480 mg	120 mg t.i.d	Up to 4 hrs
Repaglinide	0.5-2 mg before each meal	16	Up to 4 hrs
Biguanides			
Metformin	500 mg b.i.d	2550	Up to 24 hrs
Thiazolidinediones			
Pioglitazone	15-30 mg	45	24 hrs
Rosiglitazone	4-8 mg	8 mg/day or 4 mg b.i.d	24 hrs
Alpha-glucosidase inhibitors			
Acarbose	NA	25-100 mg t.i.d	1-3 hr
Miglitol	NA	25-100 mg t.i.d	1-3 hr

MONITORING TREATMENT

Monitoring blood sugar levels is an essential part of diabetes care. People with diabetes must adjust their diet, exercise, and take drugs to control blood sugar levels.

Monitoring blood sugar levels provides the information needed to make those adjustments. Waiting until symptoms of low or high blood sugar levels develop is a recipe for disaster.

Many things cause blood sugar levels to change:

- Diet
- Exercise
- Stress
- Illness
- Drug
- Time of day

The blood sugar levels may jump after people eat foods they did not realize were high in carbohydrates. Exercise may cause the levels of sugar in the blood to fall low, requiring that additional sugar be eaten. Emotional stress, an infection, and many drugs tend to increase blood sugar levels. Blood sugar levels increase in many people in the early morning hours because of the normal release of hormones (growth hormone and corticosteroids), a reaction called the dawn phenomenon. And blood sugar may shoot too high if the body releases sugar in response to low blood sugar levels (Somogyi effect).

Blood sugar levels can be measured easily at home or anywhere. Most blood sugar monitoring devices use a drop of blood

obtained by pricking the tip of the finger with a small lancet. The lancet holds a tiny needle that can be jabbed into the finger or placed in a spring-loaded device that easily and quickly pierces the skin. Most people find the pricking nearly painless. Then, a drop of blood is placed on a reagent strip. In response to sugar, the reagent strip undergoes some chemical changes. A machine reads the changes in the test strip and reports the result on a digital display. Most of these machines time the reaction and read the result automatically. Some devices allow the blood sample to be obtained from other sites, such as the palm, forearm, upper arm, thigh, or calf. The machines are smaller than a deck of cards.

A newer device reads blood sugar through the skin without needing a sample of blood. The device is worn like a wristwatch and can measure the level of sugar in the blood every 15 minutes. Alarms on the device can be set to sound when blood sugar levels drop too low or climb too high. Disadvantages of this device are that it must be calibrated periodically with a blood test, it may irritate the skin, and it is somewhat large. Other devices can monitor glucose continuously. However, these devices are not routinely used, as they are expensive and have not been shown to be better than glucose meters. In certain circumstances, these devices are less reliable, such as in severe hypoglycemia.

Most people with diabetes should keep a record of their blood sugar levels and report them to their doctors or nurses for advice in

adjusting the dose of insulin or the oral antihyperglycemic drug. Many people can l

earn to adjust the insulin dose on their own as necessary.

Although urine can also be tested for the presence of sugar, checking urine is not a good way to monitor treatment or adjust therapy. Urine testing can be misleading because the amount of sugar

in the urine may not reflect the current level of sugar in the blood. Blood sugar levels can get very low or reasonably high without any change in the sugar levels in the urine.

Doctors can monitor treatment using a blood test called hemoglobin A_{1C}. When the blood sugar levels are high, changes occur in hemoglobin, the protein that carries oxygen in the blood. These changes are in direct proportion to the blood sugar levels over an extended period. Thus, unlike the blood sugar measurement, which reveals the level at a particular moment, the hemoglobin A_{1C} measurement demonstrates whether the blood sugar levels have been controlled over the previous few months. People with diabetes aim for a hemoglobin A_{1C} level of less than 7%. Achieving this level is difficult, but the lower the hemoglobin A_{1C} level, the less likely people are to have complications. Levels above 9% show poor control, and levels above 12% show very poor control. Most doctors who specialize in diabetes care recommend that hemoglobin

A_{1C} be measured every 3 to 6 months. Fructosamine, an amino acid that has bonded with glucose, is also useful for measuring blood sugar control over a period of a few weeks [15].

AYURVEDIC THERAPY FOR TREATMENT OF DIABETES MELLITUS

WHO has approved the use of traditional medicines as part of health programme. A herbal medicine is defined as a finished, labeled medicinal product that contains active ingredients as aerial or underground parts of plants or other plant material or combinations thereof [22]. There are 1,200 species of plants representing 725 genera in 183 families extending from the marine algae and fungi with anti-diabetic activity. The mechanisms of action of most anti-diabetics herbs are not clear, although a few have been documented [23]. Traditional Medicines derived from medicinal plants are used by about 60% of the world's population. This review focuses on Indian Herbal drugs and plants used in the treatment of diabetes, especially in India [24]. There are many herbal remedies suggested for diabetes and diabetic complications. Medicinal plants form the main ingredients of these formulations [24]. A list of medicinal plants with antidiabetic and related beneficial effects is given in Table 4 [25]. A list of such formulations is given in Table 5 [24]

Table.4. List of medicinal plants with antidiabetic and related beneficial effects

Name of the plant	Reported mechanism of action
<i>Acacia arabica</i> (Lam.) Muhl. ex Willd. Common name: Indian Gum Arabic tree [Family: Leguminosae]	Acts through release of insulin from pancreatic beta cells, which accounts for the hypoglycemic activity
<i>Aegle marmelos</i> (L.) Correa ex Roxb. Common name: Holy Fruit Tree [Family: Rutaceae]	Increases utilization of glucose; either by direct stimulation of glucose uptake or via the mediation of enhanced insulin secretion and also decreases the elevated glucose and glycosylated hemoglobin levels
<i>Allium cepa</i> L. Common name: onion : [Family: Liliaceae]	Lowers blood glucose level and has potent antioxidant activity, which may account for the hypoglycemic potential
<i>Allium sativum</i> L. Common name: garlic [Family: Alliaceae]	Has strong antioxidant activity and rapid reactivity with thiol containing proteins responsible for the hypoglycemic property
<i>Aloe vera</i> (L.) Burm.f. Common name: Aloe [Family: Aloaceae]	Maintains glucose homeostasis by controlling the carbohydrate metabolizing enzymes and stimulates insulin release from pancreatic beta cells
<i>Artemisia pallens</i> Wall. Common Name: Davana [Family: Compositae]	Inhibits glucose re-absorption or increase in peripheral glucose utilization
<i>Annona squamosa</i> L. Common name: Sugar apple [Family: Annonaceae]	Lowers blood glucose level
<i>Andrographis paniculata</i> Nees Common name: King of Bitter [Family: Acanthaceae]	Prevents glucose absorption from gut. Has hypotriglyceridemic effect and antioxidant activity, which may be responsible for beneficial effect in the diabetic state
<i>Azadirachta indica</i> A.Juss. Common name: Neem [Family: Meliaceae]	Inhibits action of epinephrine on glucose metabolism, resulting in increased utilization of peripheral glucose and exhibits hypoglycaemic activity without altering the serum cortisol concentration
<i>Biophytum sensitivum</i> (L.) DC. Common name: Life Plant [Family: Oxalidaceae]	Stimulates pancreatic beta cells to release insulin
<i>Beta vulgaris</i> L. Common name: Garden beet [Family: Chenopodiaceae]	Lowers blood glucose level
<i>Boerhavia diffusa</i> L. Common name: Tar vine [Family: Nyctaginaceae]	Increases plasma insulin levels and improves glucose tolerance, produced significant antioxidant activity

<i>Cassia auriculata</i> L. Common name: Tanner's Cassia [Family: Leguminosae]	Suppresses enhanced gluconeogenesis during diabetes and enhance utilization of glucose through increased glycolysis in addition to pronounced alpha-glucosidase inhibitory actions resulting in a significant and potent lowering of blood glycemic response
<i>Caesalpinia bonducella</i> (L.) Roxb. Common name: Chinese Cinnamon [Family: Caesalpinaceae]	Increases the release of insulin from pancreatic cells
<i>Cajanus cajan</i> (L.) Millsp. Common name: Pigeon pea [Family: Fabaceae]	Lowers plasma glucose level
<i>Citrullus colocynthis</i> (L.) Schrad. Common name: Bitter apple [Family: Cucurbitaceae]	Exerts an insulinotropic effect
<i>Coccinia indica</i> Wight & Arn. Common name: Ivy gourd [Family: Cucurbitaceae]	Suppresses glucose synthesis, through depression of the key gluconeogenic enzymes glucose-6-phosphatase and fructose-1,6-bisphosphatase and enhances glucose oxidation by shunt pathway through activation of its principal enzyme glucose-6-phosphate dehydrogenase. Also has an insulin secretagogue effect and acts like insulin by correcting elevated enzymes in glycolytic pathway and restoring LPL activity in lipolytic pathway with control of hyperglycemia In diabetes.
<i>Casearia esculenta</i> Roxb. Common name: Carilla Fruit [Family: Flacourtiaceae]	Exhibits significant reduction in blood glucose level, a decrease in the activities of glucose-6-phosphatase and fructose-1,6-bisphosphatase and an increase in the activity of liver hexokinase, resulting in potent hypoglycemic activity
<i>Enicostemma littorale</i> Blume [Family: Gentiaceae]	Enhances glucose-induced insulin release from isolated rat pancreatic islets, mediated through K (+)-ATP channel-dependent pathway
<i>Eugenia jambolana</i> Lam. (syn. <i>Syzygium cumini</i> L.) Common name: Indian black berry [Family: Myrtaceae]	May be mediated through an insulin release mechanism or due to alteration in hepatic and skeletal muscle glycogen content and hepatic glucokinase, hexokinase, glucose-6-phosphate and phosphofructokinase levels in diabetic mice. It also enhances serum insulin activity and exhibits normoglycemia and better glucose tolerance
<i>Ficus bengalensis</i> L. Common name: Banyan tree [Family: Moraceae]	Stimulates insulin secretion from beta cells of islets of langerhans and inhibits insulin degradative processes

<i>Ipomoea batatas</i> (L.) Lam. Common name: Sweet potato [Family: Convolvulaceae]	Reduces insulin resistance and possibly acts by maltase inhibition, not by sucrase or glucose transport inhibition at the intestinal membrane
<i>Mangifera indica</i> L. Common name: Mango [Family: Anacardiaceae]	Possibly acts through intestinal reduction of the absorption of glucose as well as pancreatic and extrapancreatic mechanisms
<i>Momordica cymbalaria</i> Fenzlex Naudin [Family: Cucurbitaceae]	May act by increasing hepatic glycogen
<i>Mucuna pruriens</i> (L.) DC. Common name: Velvet bean [Family: Leguminosae]	Possibly acts through stimulation of the release of insulin and/or by a direct insulin-like action due to the presence of trace elements like manganese, zinc, etc.
<i>Morus alba</i> L. Common name: White mulberry [Family: Moraceae]	Acts by increasing glucose uptake
<i>Murraya koenigii</i> (L.) Spreng. Common name: curry-leaf tree [Family: Rutaceae]	Increases glycogenesis and decreases glycogenolysis and gluconeogenesis
<i>Ocimum sanctum</i> L. Common name: Holy Basil [Family: Lamiaceae]	Acts by cortisol inhibiting potency
<i>Punica granatum</i> L. Common name: Pomegranate [Family: Punicaceae]	Inhibits intestinal alpha-glucosidase activity, leading to antihyperglycemic property
<i>Salacia reticulata</i> Wight. Common name: Salacia [Family: Celastaceae]	Inhibits alpha-glucosidase activity
<i>Salacia Oblonga</i> Wall. [Family: Celastaceae]	Acts through inhibition of alpha-glucosidase activity
<i>Swertia chirayita</i> (Roxb. ex Fleming) H. Karst. Common name: Indian Gentian [Family:Gentianaceae]	Stimulates insulin release from islets of Langerhans by depleting aldehyde-fuchsin stained beta-granules and immunostained insulin
<i>Scoparia dulcis</i> L. Common name: Sweet Broomweed [Family: Scrophulariaceae]	Suppresses glucose influx into the polyol pathway leading to increased activities of antioxidant enzymes and plasma insulin and decreases activity of sorbitol dehydrogenase. Also potentiates insulin release from pancreatic islets[24]

Table.5.Commercially available formulations

Drug	Company	Ingredients
Diabecon	Himalaya	<i>Gymnema sylvestre</i> , <i>Pterocarpus marsupium</i> , <i>Glycyrrhiza glabra</i> , <i>Casearia esculenta</i> , <i>Syzygium cumini</i> , <i>Asparagus racemosus</i> , <i>Boerhavia diffusa</i> , <i>Sphaeranthus indicus</i> , <i>Tinospora cordifolia</i> , <i>Swertia chirata</i> , <i>Tribulus terrestris</i> , <i>Phyllanthus amarus</i> , <i>Gmelina arborea</i> , <i>Gossypium herbaceum</i> , <i>Berberis aristata</i> , <i>Aloe vera</i> , <i>Triphala</i> , <i>Commiphora wightii</i> , <i>shilajeet</i> , <i>Momordica charantia</i> , <i>Piper nigrum</i> , <i>Ocimum sanctum</i> , <i>Abutilon indicum</i> , <i>Curcuma longa</i> , <i>Rumex maritimus</i>
Diasulin	Patented formulation	<i>Cassia auriculata</i> , <i>Coccinia indica</i> , <i>Curcuma longa</i> , <i>Embllica officinalis</i> , <i>Gymnema sylvestre</i> , <i>Momordica charantia</i> , <i>Scoparia dulcis</i> , <i>Syzygium cumini</i> , <i>Tinospora cordifolia</i> , <i>Trigonella foenum graecum</i>
Pancreatic tonic 180 cp	ayurvedic herbal supplement	<i>Pterocarpus marsupium</i> , <i>Gymnema sylvestre</i> , <i>Momordica charantia</i> , <i>Syzygium cumini</i> , <i>Trigonella foenum graecum</i> , <i>Azadirachta indica</i> , <i>Ficus racemosa</i> , <i>Aegle marmelos</i> , <i>Cinnamomum tamala</i>
Ayurveda alternative herbal formula to Diabetes:	Chakrapani Ayurveda	Gurmar (<i>Gymnema sylvestre</i>) Karela (<i>Momordica charantia</i>) Pushkarmool (<i>Inula racemosa</i>) Jamun Gutli (<i>Syzygium cumini</i>) Neem (<i>Azadirachta indica</i>) Methika (<i>Trigonella foenum graecum</i>) Guduchi (<i>Tinospora cordifolia</i>)
Bitter gourd Powder	Garry and Sun natural Remedies	Bitter gourd (<i>Momordica charantia</i>)
Dia-care	Admark Herbals Limited	Sanjeevan Mool; Himej, Jambu beej, Kadu, Namejav, Neem chal.
Diabetes-Daily Care	Nature's Health Supply	Alpha Lipoic Acid, Cinnamon 4% Extract, Chromax, Vanadium, Fenugreek 50% extract, <i>Gymnema sylvestre</i> 25% extract <i>Momordica</i> 7% extract, Licorice Root 20% extract
Gurmar powder	Garry and Sun natural Remedies	Gurmar (<i>Gymnema sylvestre</i>)
Epinsulin	Swastik Formulations	vijaysar (<i>Pterocarpus marsupium</i>)
Diabecure	Nature beaute sante	<i>Juglans regia</i> , <i>Berberis vulgaris</i> , <i>Erythrea centaurium</i> , <i>Millefolium</i> , <i>Taraxacum</i>
Diabeta	Ayurvedic cure Ayurvedic Herbal Health Products	<i>Gymnema sylvestre</i> , <i>Vinca rosea</i> (Periwinkle), <i>Curcuma longa</i> (Turmeric), <i>Azadirachta indica</i> (Neem), <i>Pterocarpus marsupium</i> (Kino Tree), <i>Momordica charantia</i> (Bitter Gourd), <i>Syzygiumcumini</i> (Black Plum), <i>Acacia arabica</i> (Black Babhul), <i>Tinospora cordifolia</i> , <i>Zingiber officinale</i> (Ginger)
Syndrex	Plethico Laboretaries	Germinated Fenugreek seed extract ^[25]

HOMEOPATHIC REMEDIES FOR

DIABETES: For example homeopathic remedy is concern they find the correct remedy by looking for symptoms of:

- Main complication
- Physical symptoms
- Mental/emotional/psychological symptoms
- Modalities (when the disease is better and when it is worst)
- Causation (reason behind the disease – root cause)

Homeopathic remedies for diabetes, different peoples are treated with a different medicine with respect to their individual physical, mental, modalities and causation [26].

Remedies that may be helpful include

1. Argentum Metallicum – Frequent urination, esp. at night. Appetite greatly increased, hungry even after a meal. Mouth dry, Weakness, emaciation. Particularly when scrotum, ankles and feet are swollen.
2. Arsenicum Album – Great hunger and unquenchable thirst, especially for small sips. Emaciation and weakness.
3. Helonias – First stages of diabetes. Urine is profuse, clear, containing sugar. Lips dry, stick together. Pain in the lower back.
4. Lactic Acid – Sugar in the urine. Great thirst. High sugar in the urine and passed frequently. Nausea, better by eating. Constipation, debility. Rheumatic pains in the joints.

5. Plumbum – Frequent and copious urination. Violent thirst. Gradual onset of debility. Diminution of sight. Loss of sexual desire, impotence. Dislike of conversation and labor [27].
6. Syzygium jambolanum: It is prepared from jambol seeds. No other remedy causes such a marked degree of diminution and disappearance of sugar in the urine. This remedy acts rapidly and reduces extra sugar immediately. Any patient having classical symptoms of diabetes such as thirst, hunger, weakness, polyuria can take this remedy safely. It can be safely given with any other remedy, whether it be allopathic, ayurvedic or whatsoever. There is no interreaction between them. Use of this remedy is also mentioned in ayurveda. It is to be used in mother tincture form, 5 drops every 6 hrs in very high diabetes and every 8 hrs in mildly high diabetes.
7. Phosphoric acid: Urine is cloudy and can be thick after standing. Profuse urination. Indifference with great debility. This remedy works well in nervous and emaciated patients. These patients have over worked mentally and physically for years together and diabetes in them is the result of some suppressed grief, emotion, shock or tension. These patients also have classical symptoms of diabetes and may also have carbuncles which

usually appear in diabetes. Patient feels extremely weakness and debility. He feels pain in all parts of the body esp. calf muscles. This remedy can also be given in neurosis after amputation or pains after amputation in diabetes. It is to be given in 1x potency, 3ce a day in starting gradually reducing it to 2ce a day.

8. **Aurum metallicum:** This remedy works well in depressed patients. Depression may be the result of their diabetes or diabetes may be the result of depression, it doesn't matter and argentum helps them improve in both the cases. Patient has craving for sugar and nausea with classical signs of diabetes. He has high blood pressure also. This patient may have hidden suicidal tendencies, fear of crowd. He hates his life. Frequent urination with burning. This remedy can be given in 30x, 3times a day in starting and gradually reducing it to twice a day.
9. **Uranium nitricum:** This remedy works wonder in patients suffering ascites with general dropsy as a result of diabetes. Dropsy is due to nephritis which is a major complication in diabetes. All classical symptoms of diabetes are present with swelling of body. There may be burning pain in urination and incontinence of urine. Complete impotency with nocturnal emissions

in male or delayed menses in female patients. Use this remedy in 30x, 3times a day.

10. **Arsenicum bromatum:** This remedy works good in patients who have excessive thirst. 3 to 4 drops of mother ticture, 3 times a day are to be given to the patient till his thirst and hunger return to normal. After that only maintenance dose is required i.e. 3 drops, 2 times a day.
11. **Insulinum:** Administer this remedy in 30x potency at suitable intervals in diabetes mellitus to improve functioning of pancreas^[28].

Other remedies that may be considered are Adrenelinum, Bovista, Chionanthus, Coca, Curare, Gloninum, Heleborus, Iodium, Lachesis, Lycopodium, Morphinum, Moschus, Murex, Natrum Muriaticum, Natrum Sulphuricum, Nitric Acid, Nux Vomica, Opium, Pancreatinum, Phosphorus, Picric Acid, Podophylum, Silica, Silygium Jambol, Sulphur, Tarantula Hispanica, Taraxicum, Terebenthinae ^[27].

These are only a few remedies. Homeopathy has 100s of such wonderful drugs and its proper selection for a patient can be done by a homeopath only.

NOTE: * Regular check up of blood sugar levels is necessary.

- Patient must consume only fresh food.
- Reduce weight.
- Regular exercise or brisk walk or yoga is useful to control sugar levels.

- Protect and cover feet to avoid punctured wounds as a result of poor fitting shoe.
- Do not stop taking drugs prescribed by your physician.
- Patient must be fully aware of hypoglycemia symptoms as low blood sugar can even lead to death in few minutes.
- Above mentioned drugs are for knowledge purpose and can be safely taken after consulting your nearest homeopathic physician [28].

ACUPUNCTURE THERAPY IN DIABETES MELLITUS

Acupuncture literally means 'needle piercing,' the practice of inserting very fine needles into the skin to stimulate specific anatomic points in the body (called acupoints or acupuncture points) for therapeutic purposes. Acupuncture involves stimulation.[29]

Points use to treat diabetes mellitus [30]

1. Jianjing (GB 21)

Location – Midway between 7th cervical vertebra and the acromion.

Puncture – 1.5 cun perpendicularly.



2. Quchi (LI 11)

Location – At the outer end of elbow crease when elbow is semi flexed.

Puncture - 1 to 1.5 cun perpendicularly.



3. Hegu (LI4)

Location – It is situated in the web between the forefinger and thumb on the dorsal aspect of the hand at the top of the 1st dorsal interosseous muscle when thumb is adducted.

Puncture – 0.5 to 1 cun perpendicularly.



4. Zusanli (St 36)

Location – 1 finger breadth lateral to lower end of tibia tuberosity.

Puncture – 1 cun perpendicularly.



5. Taibai (Sp 3)

Location - Posterior and inferior to head of 1st metatarsal bone at border of two colon of skin.>



6. Sanyinjiao (Sp 6)

Location – 3 cun above the medial malleolus, posterior to the border of tibia

Puncture – 1 cun vertical.



7. Taichong (Liv 3)

Location – 2 cun proximal to the margin of the web of the 1st and 2nd toes.

Puncture – 1 cun obliquely in a proximal direction.

**8. Dazhui (Du 14)**

Location – On the back midline between the dorsal spines of the 7th cervical and 1st thoracic vertebra.

Puncture – 1.0 cun perpendicularly.

**9. Pishu (U.B. 20)**

Location – 1.5 cun lateral to spine and 11th thoracic vertebra.

Puncture – Pancreas, Internal secretion, Spleen.

**10. Jiexi (St 41)**

Location- Midway between the malleoli on the anterior ankle crease, between the tendons of extensor digitorum longus and hallucis longus.

Puncture – 0.5 cun perpendicularly.

**11. Waigun (TW 5)**

Location – 2 cun proximal to the midpoint of the dorsal transverse crease of the wrist

between the radius and ulna,

Puncture - 1 cun perpendicularly.

**Mechanism of action of acupuncture therapy for diabetes mellitus**

The effects of acupuncture on diabetes have been observed experimentally and clinically. Animal experiments have shown that acupuncture can activate glucose-6- phosphatase (an important enzyme in carbohydrate metabolism) and affect the hypothalamus.

Acupuncture can act on the pancreas to enhance insulin synthesis, increase the number of receptors on target cells, and accelerate the utilization of glucose, resulting in lowering of blood sugar.

How can acupuncture help diabetic patients?

Scientific studies and clinical tests in international research centers in the past 10 years have shown that acupuncture can help diabetic patients in the following ways:

- lower blood glucose content;

- lower the release of pancreatic glucagons;
- attenuate symptoms of polyphagia (the urge to eat too much), polydipsia (excessive thirst) and polyuria (excessive passage of urine);
- prevent slowing of motor nerve conduction;
- improve microcirculation and myocardial contractility;
- enhance blood outflow and regulate vascular peripheral resistance;
- exert antiatherogenic, antioxidant and immunomodulating effects;
- obliterate atherosclerosis of the legs;
- induce secretion of endogenous beta-endorphin;
- elevate a lowered pain threshold; and
- increase cell proliferation and neuropeptide Y levels [29].

YOGA POSES FOR DIABETES

Yoga is an ancient Vedic practice of various physical postures and breathing techniques to reduce stress and improve health. Yoga has been derived from a Sanskrit word 'yoke' which means union [30]. Yoga refers to be the traditional physical and mental disciplines originating in India. It has the ability to

balance body, mind & spirit. This discipline has curative power for many chronic diseases that include diabetes, by activating glandular systems responsible for the illness. In the case of diabetes, yoga poses give mild and gentle massages to the internal organs such as pancreas, liver, intestine, etc. These organs are responsible for the diabetes cause^[26]. There are various yoga poses which can be effective in warding off diabetes. Sun salutation or Surya Namaskar is a very powerful and helpful yoga exercise for people suffering from diabetes. This exercise tends to improve the blood circulation throughout the body and hence a better level of insulin administration throughout the body. After becoming more familiar with Sun Salutations, one can practice other yoga asanas including Paschimottanasana, Dhanurasana, Ardhamatsyendrasana, Kapalbhati, Anuloma-Viloma, Vajrasana, Shankha Prakshalana and Savasana. Pranayama or the breathing techniques are also very good for controlling and purifying the blood. In addition, practicing meditation can also be helpful in the treatment of diabetes. Regular meditation has actually been known to have a regulative effect on sugar levels. Gentle meditation music can be of great while practicing pranayama as it helps one to concentrate more on the breathing [31].

The yoga poses for are shown in following figures(fig-2 to fig.10):-

Fig.2- Surya Namaskar ^[33]



Fig.3-Paschimothanasana ^[34]



Fig.4-Dhanurasana^[35]



Fig.5-Ardha Matsyendrasana^[36]
Viloma^[38]

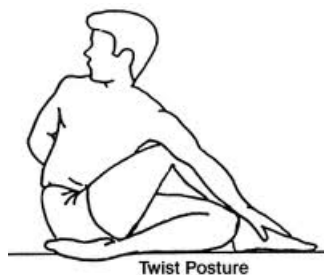


Fig.6-kapalbhati^[37]

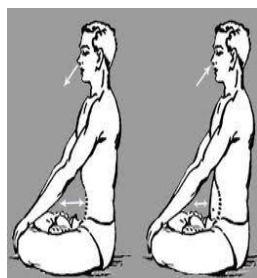


Fig.7-Anuloma-
Viloma^[38]



Fig.8-Vajrasana^[39]

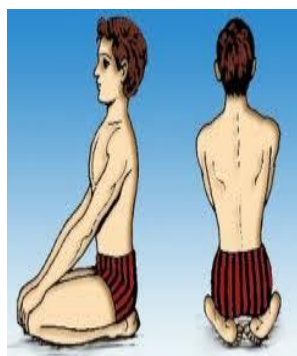


Fig.9-Shankha Prakshalana ^[40]



Fig.10- Savasana^[41]



CONCLUDING REMARKS

The complications and symptoms of diabetes mellitus are treated by allopathic, Ayurvedic, homeopathic and yoga therapy etc. For providing the

synergistic effect the combination of at least two therapeutic systems will give a significant fruitful result. Most of the physician/vaidyas prefer the combination may be allopathic with

yoga, Ayurvedic with yoga, homeopathic with yoga and acupuncture with yoga. And majority physician will advise yoga asans along with medications, because it act as a supplement or catalyst to the therapy, and as the yoga therapy does not have any side effect if it is practiced under supervision of yoga guru.

Extensive research work is going on in allopathic system by eminent scientist and expertise in order to identify a new potent entity or therapy, with devoid of adverse effect, where as in ayurvedic system many researcher are exploring a novel properties of medicinal plant or a potent polyphyto combination to elicit a desired therapeutic effect with devoid of minimal side effect.

REFERENCES

1. Joseph T. Dipiro. Text book of Pharmacotherapy, Diabetes mellitus; 6th edition: 8: 1333.
2. Leon Shargel. Comprehensive Pharmacy Review, Diabetes mellitus; 4th edition: 51:970.
3. Scott R Votey. Diabetes Mellitus, Type 1 - A Review. Available at <http://emedicine.medscape.com/article/766036-overview>.
4. Scott R Votey. Diabetes Mellitus, Type 2 - A Review. Available at <http://emedicine.medscape.com/article/766143-overview>.
5. Shaw JE, Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes Res Clin Pract.* 2010 Jan;87(1):4-14. Epub 2009 Nov 6. Available at <http://www.ncbi.nlm.nih.gov/pubmed/19896746>.
6. Sarah Wild. Global Prevalence of Diabetes. *Diabetes Care* 27:1047-1053, 2004
7. Jain Sarika. Efficacy of standardized herbal extracts in Type 1 Diabetes- An experimental study. *African Journal of Traditional, Complementary and Alternative Medicines* 2006; 3: 23-33.
8. Diabetes fact. Available at www.worlddiabetesfoundation.org/composite-35.htm
9. Theberge chris. Etiology of Type 1 Diabetes. Available at <http://www.nafwa.org/index.php>.
10. Gottlieb MD. Diagnosis and Treatment of Pre-Insulin Dependent Diabetes. *Annual Review of Medicine.* 1998; 49: 397-405.
11. Nepom G.T. Immunogenetics and IDDM. *Diabetes Review.* 1993; 1: 93-103.
12. McCulley DeWayne. The Etiology of Type 2 Diabetes. Available at http://www.deathtodiatetes.com/Death_to_Diabetes.php
13. Jeff Milam. What Is Secondary Diabetes? Available on http://www.ehow.com/facts_5752306_secondary-diabetes_.html.
14. Lloyd Emma. Article on Pathophysiology of Diabetes Mellitus: Jan 25, 2010. Available at <http://www.brighthub.com/science/medical/articles/62282.aspx#ixzz0qAFvkRYg>
15. Diabetes mellitus. Available on the merck manual on line medical library at <http://www.merck.com/mmhe/sec13/ch165/ch165a.html>.
16. Mayfield Jennifer. Diagnosis and Classification of Diabetes Mellitus: Published by the American academy of family physician: October 15, 1998.
17. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care* 1997; 20:1183-97.
18. Scott R Votey. Diabetes Mellitus, Type 1 - A Review: Differential Diagnoses & Workup. Available at <http://emedicine.medscape.com/article/766036-diagnosis>

19. Scott R Votey. Diabetes Mellitus, Type 2 - A Review: Differential Diagnoses & Workup. Available at <http://emedicine.medscape.com/article/766143-diagnosis>
20. Joseph T. Dipiro. Text book of Pharmacotherapy, Diabetes mellitus; 6th edition: 8: 1345.
21. Joseph T. Dipiro. Text book of Pharmacotherapy, Diabetes mellitus; 6th edition: 8: 1348.
22. Patel P.M. Development of HPTLC method for estimation of charantin in herbal formulations. *Pharmacognosy Magazine* Oct-Dec.2006; 2(8): 224.
23. Kolawole O. Matthew. The Effect of *Bridelia ferruginea* and *Senna alata* on Plasma Glucose Concentration in Normoglycemic and Glucose Induced Hyperglycemic Rats. *Ethnobotanical Leaflets* 2006; 10: 209-218.
24. Modak Manisha. Indian Herbs and Herbal Drugs Used for the Treatment of Diabetes. *J Clin Biochem Nutr.* 2007 May; 40(3): 163-173.
25. Pulok K. Mukherjee. Leads from Indian medicinal plants with hypoglycemic potentials. *Journal of Ethnopharmacology* 106 (2006) 1-28. Available online at www.sciencedirect.com.
26. Complementary and alternative medicine treatment for diabetes. Available at <http://hubpages.com/hub/healthy-ojascom>.
27. <http://robertfield3.wordpress.com/homeopathy-tips/homeopathy-tips-for-21709-diabetes>
28. Ameya Pimalgaonkar. Cure the diabetes with the help of homeopathy. Available at www.madeitsimple.com/healthcare.
29. http://www.holisticonline.com/Acupuncture/acp_what_is.htm.
30. N.R.Samdani. Application of Acupuncture therapy in Diabetes mellitus patient. Available at www.pharmainfo.net/reviews.
31. Rajmalhotra. Practice yoga and ward off diabetes. Available at <http://www.indocean.com/practice-yoga-and-ward-off-diabetes/>.
32. Davidson's Principles and Practice of Medicine; Diabetes mellitus; 18th edition: page no. 473-474.
33. Surya Namaskar. Available at <http://eckee.com/surya-namaskar-terminator-of-all-diseases/> assessed on 30/05/2012.
34. Paschimothanasana .Available at www.abc-of-yoga.com ASSESSED ON 30/05/2012.
35. Dhanurasana. Available at www.ceasyoga.com assessed on 30/05/2012.
36. Ardha Matsyendrasana .Available at www.antiagingtreatments.blogspot.com assessed on 30/05/2012.
37. Kapalbhati. Available at www.indianetzone.com assessed on 30/05/2012.
38. Anuloma-Viloma. Available at www.yoga.nuvvo.com assessed on 30/05/2012.
39. Vajrasana. Available at www.openfaves.com assessed on 30/05/2012.
40. Shankha Prakshalana. Available at www.yogapoint.com assessed on 30/05/2012.
41. Savasana. Available at www.loveyourselfnaturally.wordpress.com assessed on 30/05/2012.