

# Role of Homoeopathic Medicines in Type 2 Diabetes Mellitus

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## ABSTRACT

Diabetes Mellitus is metabolic disorder characterized by hyperglycemia in blood circulation mainly resulting from inadequate insulin secretion, insulin action or both and insulin resistance. Homoeopathic remedies have reported beneficial effect for treating in type 2 diabetes mellitus cases.

**KEYWORDS:** Role, homoeopathy medicines, type 2, diabetes

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## INTRODUCTION

Nobody in this world who does not know about the diabetes, commonly called “sugar” in Indian community. Diabetes comprises a group of common metabolic disorder that share the phenotype of hyperglycemia (Increase level of glucose in blood circulation). The dramatic worldwide increase in the prevalence of type 2 diabetes mellitus is posing a massive health problem in both developed and developing countries<sup>1</sup>. Interestingly in developed countries, lower socioeconomic groups are most affected, while in developing countries, the reverse applies. The magnitude of the healthcare issues of type 2 diabetes mellitus results not just from the disease itself but also from its association with obesity and cardiovascular risk factors, particularly dislipidemia and hypertension. Indeed type 2 diabetes mellitus has now been recognized as one manifestation of the “Metabolic Syndrome”, a condition characterized by *insulin resistance* and associated with a range of cardiovascular risk factors<sup>2</sup>.

## Etiology of type 2 diabetes mellitus

It is also called as non insulin depended diabetes mellitus (NIDDM). Type 2 diabetes mellitus is characterized by insulin resistance, impaired insulin secretion, and increased glucose production. Type 2 diabetes mellitus more typically develops with increase in age; it also occurs in children, particularly in obese adults. It does not require insulin therapy<sup>3</sup>.

## Other causes of diabetes mellitus:

Drugs or chemical induced diabetes mellitus: Some drugs such as Nicotinic acid, Glucocorticoids, Thyroid hormones, Diazoxide beta adrenergic agonists, Thiazides,  $\beta$  blockers etc causes diabetes mellitus.

Endocrinal Diseases: This includes Hyperthyroidism, Hypersecretion of Adrenal cortex, Hyperpituitarism, Cushing’s syndrome, Pheochromocytoma, Acromegaly, Somatostatinoma.

Diseases of Pancreas: This includes Pancreatitis, Cystic Fibrosis, Hemochromatosis, Pancreatopathy, Cancer of pancreas, Pancreatectomy.

Other Genetic Syndrome sometime associated with diabetes mellitus like as Down’s syndrome, Klinefelter’s Syndrome, Turner’s syndrome, Huntington’s corea.

## Epidemiology

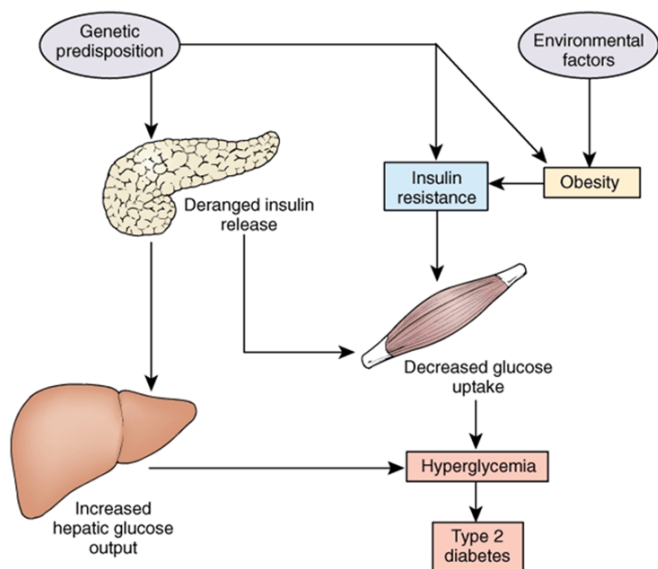
It’s a surprise that India leads the world with largest number of diabetes subjects earning the dubious distinction of being termed the “**Diabetes Capital of World**”. Diabetes Atlas 2006 shows the number of people with diabetes mellitus in India currently around 40.9 million is expected to rise to 69.9 million by 2025. The Asian – Indian community shows the certain unique clinical and biochemical abnormalities which includes, greater abdominal adiposity – higher waist circumference despite lower body mass index, lower

adiponectine and higher high sensitive C – reactive protein levels – leads to *insulin resistance*. Even though the prevalence of micro vascular complications of diabetes mellitus like retinopathy and nephropathy are comparatively lower in Indians but the prevalence of premature coronary artery disease (CAD) is much higher in Indians<sup>4</sup>. The prevalence of diabetes is rapidly increasing day by day over the globe at an alarming rate. In past 30 years the status of diabetes has changed from being considered as a mild disorder of the elderly to one of the major cause of morbidity and mortality affecting the youth and middle aged people. Type 2 diabetes mellitus increase in the prevalence but the more common is type 2. Although both male and female have diabetes but males have slightly higher incidence from females. Among the ethnicity blacks are having three time’s higher incidence than whites.

**PATHOGENESIS**

Type 2 diabetes mellitus is characterized by impaired insulin secretion, insulin resistance, excessive hepatic glucose production and abnormal fat metabolism. Obesity, particularly visceral or central [as evidenced by the hip-waist ratio], is very common in type 2 diabetes mellitus [80% or more are obese]. In the early stages of the disorder, glucose tolerance remains near normal, despite insulin resistance, because the pancreatic beta cell compensate by increasing insulin output. As insulin resistance and compensatory hyperinsulinaemia progress, the pancreatic islets in certain individuals are unable to sustain the hyperinsulinemic state. IGT, characterized by elevations in postprandial glucose, then develops. A further decline in insulin secretion and an increase in hepatic glucose production lead to overt diabetes with fasting hyperglycemia. Ultimately beta cell failure ensues<sup>5</sup>.

**Figure1. Pathophysiology of type 2 diabetes mellitus**



Insulin secretion and sensitivity are interrelated. In type 2 diabetes mellitus, insulin secretion initially increases in response to insulin resistance to maintain normal glucose tolerance. Initially, the insulin secretory defect is mild and selectively involves glucose stimulated insulin secretion. The response to other nonglucose secretagogues, such as arginine is preserved. Abnormalities in proinsulin processing is reflected by increased secretion of proinsulin in type 2 diabetes mellitus. Eventually, the insulin secretory defect

progresses to a state of inadequate insulin secretion. The reason for the decline in insulin secretory capacity in type 2 diabetes mellitus is unclear. The assumption is that a second genetic effect is superimposed upon insulin resistance – leads to beta cell failure. Beta cell mass is decreased by approximately 50% in individuals with longstanding type 2 diabetes. Islet amyloid fibrillar deposit found in the islets of individuals with longstanding type 2 diabetes mellitus. Whether such islet amyloid deposits are primary or secondary event is unknown<sup>6</sup>. The metabolic environment of diabetes may also negatively impact islet function. For example chronic hyperglycaemia paradoxically impairs islet function [glucose toxicity] and leads to worsening of hyperglycaemia. Improvement in glycaemic control is often associated with improved islet function. In addition, elevation of free fatty acid levels [lipotoxicity] and dietary fat may also worsen islet function.

**CLINICAL FEATURES**

Type 2 diabetes mellitus present with Polyuria, polyphagia and polydipsia, but unlike type 1 diabetes, patients are often older [over 40 years] and frequently obese. However, with the increase in obesity and sedentary life style in our society, type 2 diabetes is now seen in children and adolescent with increasing frequency. In some medical attention is sought because of unexplained weakness or weight loss. Most frequently, however, the diagnosis is made after routine blood or urine testing in asymptomatic persons. The infrequency of ketoacidosis and milder presentation in type 2 diabetes is presumably because of higher portal vein insulin levels in these patients than in type 1 diabetics, which prevents unrestricted hepatic fatty acid oxidation and keeps the formation of ketone bodies in check. In the decompensated state, these patients may develop hyperosmolar nonketotic coma due to severe dehydration resulting from sustained osmotic diuresis [particularly in patients who do not drink enough water to compensate for urinary losses from chronic hyperglycaemia]. Typically, the patient is an elderly diabetic who is disabled by stroke or an infection and is unable to maintain adequate water intake. Furthermore, the absence of ketoacidosis and its symptoms [nausea, vomiting, respiratory difficulties] delays the seeking of medical attention until severe dehydration and coma occur<sup>7</sup>.

**Figure 2: Signs & symptoms of type 2 diabetes mellitus**

Signs:	Symptoms:
<ul style="list-style-type: none"> <li>▪ Hyperglycaemia (increased blood glucose level)</li> <li>▪ Glycosuria</li> <li>▪ Dehydration</li> <li>▪ Tachycardia</li> <li>▪ Confusion</li> <li>▪ Coma</li> </ul>	<p><b>Classic Symptoms:</b></p> <ol style="list-style-type: none"> <li>1. Polyuria (increase urination)</li> <li>2. Polyphagia (increased hunger)</li> <li>3. Polydipsia (increased thirst)</li> <li>4. Weight loss</li> </ol> <p><b>Other symptoms-</b></p> <ul style="list-style-type: none"> <li>-blurry vision</li> <li>-headache</li> <li>-fatigue</li> <li>-slow healing of wounds</li> <li>-itchy skin</li> </ul>

**DIAGNOSIS**

New revised criteria for the diagnosis of diabetes mellitus from the expert panel of world health organization and National Diabetes Data Group emphasize the fasting plasma glucose as the most reliable and convenient test for diagnosing diabetes mellitus in asymptomatic individual<sup>8</sup>.

Glucose tolerance is classified in to three categories based on the FPG

- FPG < 110 mg/dl is considered as normal
- FPG ≥ 110 mg/dl but < 126 mg/dl is defined as IFG (Impaired Fasting Glucose)
- FPG ≥ 126 confirm the diagnosis of diabetes mellitus

Fasting plasma glucose is a new diagnostic category analogous to IGT, which is defined as the plasma glucose level between 140mg/dl and 200mg/dl, 2 hour after a 75gm oral glucose load. A random plasma glucose concentration ≥ 200 accompanied by classic symptoms of diabetes mellitus, for example polydipsia (increased thirst), polyuria (increased micturation), polyphagia (increased appetite), weight loss is sufficient for the diagnosis of diabetes mellitus. The two hour plasma glucose commonly called as post parential is still a valid mechanism for diagnosing diabetes mellitus but is not recommended as a part of routine screening.

Glycated hemoglobin (also known as Glycohemoglobin, Glycosylated hemoglobin or HbA1c) 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<sup>9</sup>.

**RISK FACTORS FOR TYPE 2 DIABETES MELLITUS**

- A strong family history
- Obesity
- Age ≥ 45 years

- Previously identified impaired fasting glucose or impaired glucose tolerance
- History of gestational diabetes mellitus
- Hypertension (Blood pressure ≥ 140/90 mmHg)
- High density lipoprotein cholesterol level ≤ 35 mg/dl
- Triglyceride level > 250 mg/dl

**Complications of type 2 diabetes mellitus<sup>10</sup>**

The complication of diabetes mellitus are categorized into two main group i.e. Acute and Chronic complications. The acute complications are due to metabolic disturbances. These include are DKA (Diabetic Ketoacidosis) and Nonketotic Hyperosmolar state.

The chronic complication are also categorized into two broad groups

**Microvascular complications:** These include Ophthalmic Disorders (Retinopathy, Macular edema, Cataract, Glaucoma), Neuropathy (Peripheral neuropathy, Sensory and Motor polyneuropathy), and Nephropathy (ESRD).

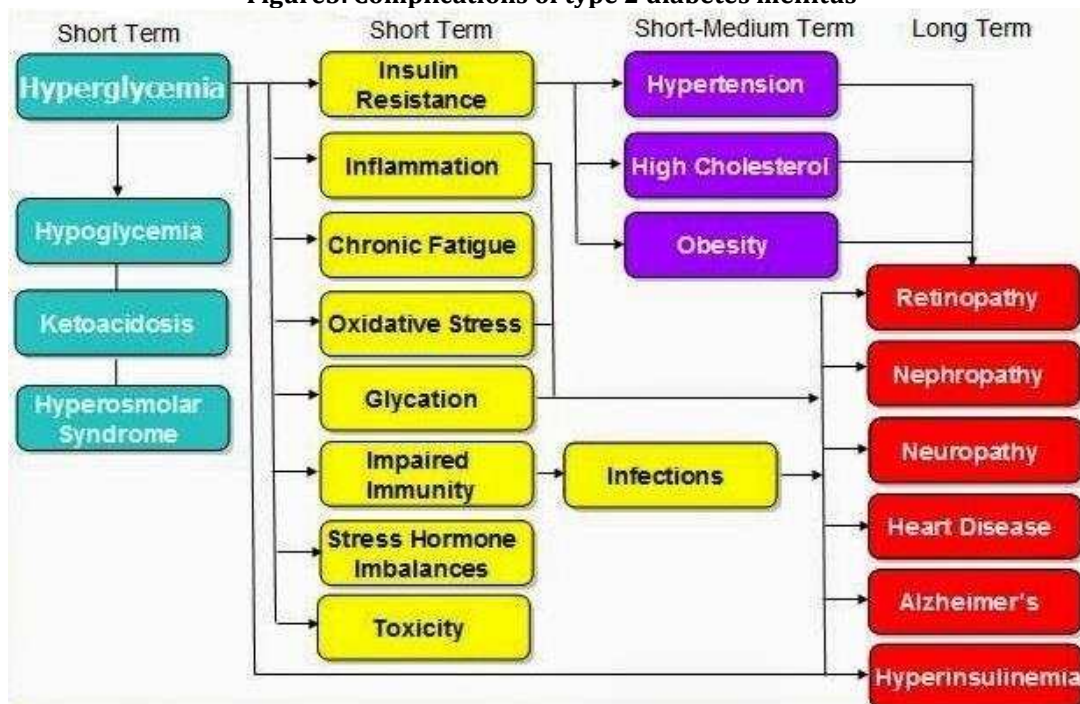
**Macrovascular complications:** These include Coronary Artery Diseases (CAD), peripheral vascular disorders, and cerebrovascular diseases.

**Other complications** include Gastroparasis, Diarrhoea, Uropathy, Sexual dysfunction and Dermatologic complications like eczema, cellulites, and gangrene of distal part of limbs (Diabetic foot). Instead of this mechanism of these complications are not known.

**HYPOGLYCEMIA:**

When the amount of blood glucose falls below the acceptable normal levels, it is called hypoglycemia. It revels always an emergency. It produces a number of symptoms like<sup>11</sup>.

**Figure3. Complications of type 2 diabetes mellitus**



Long standing (or) uncontrolled type 2 diabetes mellitus may leads from acute and chronic complication. In chronic complication are two type microvascular complications (diabetes retinopathy, diabetes renopathy, diabetes neuropathy), macrovasuclar complication (Cardiovascular) and other complications (skin complications).



Diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state are acute complications of diabetes. DKA was formerly considered a hallmark of type 1 diabetes mellitus, but it also occurs in individuals who lack immunologic features of type 1A Diabetes mellitus and who can subsequently be treated with oral glucose lowering agents (these individuals with type 2 diabetes mellitus are often of Hispanic or African American descent). HHS is primarily seen in individuals with type 2 diabetes mellitus. Both disorders are associated with absolute or relative insulin deficiency, volume depletion, and acid base abnormalities. Diabetic ketoacidosis and HHS exist along a continuum of hyperglycemia, with or without ketosis. The metabolic similarities and differences in diabetic ketoacidosis. Both disorders are associated with potentially serious complications if not promptly diagnosed and treated<sup>12</sup>.

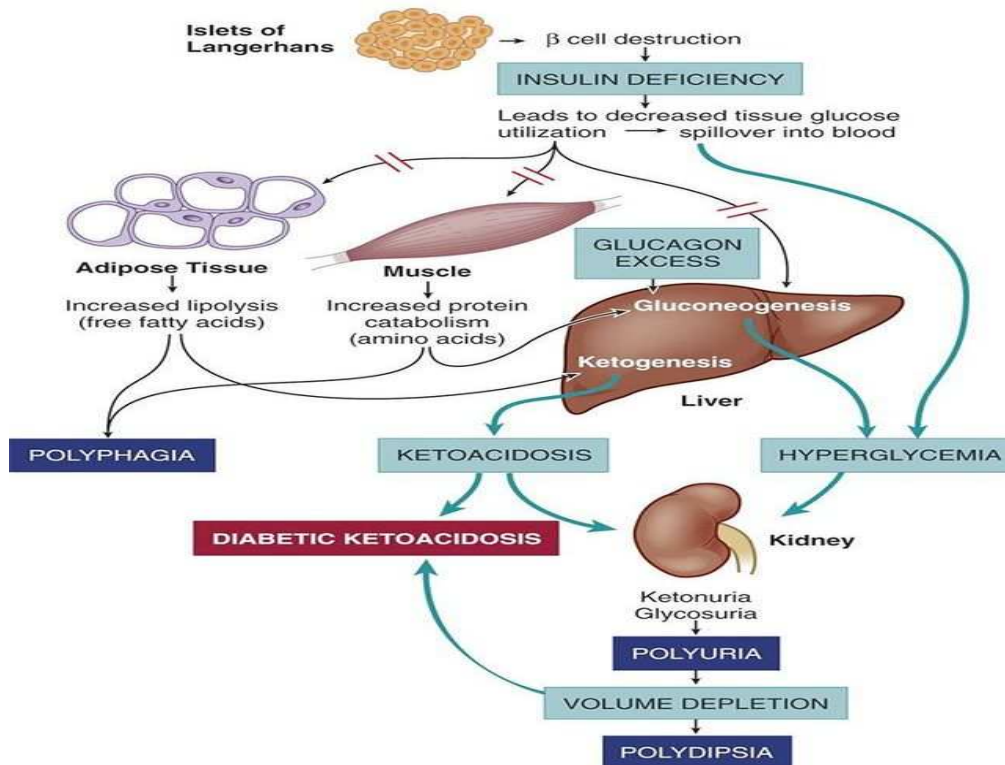
**DIABETIC KETOACIDOSIS (DKA)**

**Clinical Features:**

The symptoms and physical signs of Diabetic ketoacidosis are usually developed over 24 hours. Diabetic ketoacidosis may be the initial symptom complex that leads to a diagnosis of type 1 Diabetes mellitus, but more frequently it occurs in individuals with established diabetes. Nausea and vomiting are often prominent, and their presence in an individual with diabetes warrants laboratory evaluation for Diabetic ketoacidosis. Abdominal pain may be severe and can resemble acute pancreatitis or ruptured viscus. Hyperglycemia leads to glucosuria, volume depletion, and tachycardia<sup>13</sup>. Hypotension can occur because of volume depletion in combination with peripheral vasodilatation. Kussmaul respirations and a fruity odour on the patient’s breath (secondary to metabolic acidosis and increased acetone) are classic signs of the disorder. Lethargy and central nervous system depression may evolve into coma with severe Diabetic ketoacidosis but should also prompt evaluation for other reasons for altered mental status (infection, hypoxia, etc.). Cerebral oedema, an extremely serious complication of Diabetic ketoacidosis, is seen most frequently in children. Signs of infection, which may precipitate Diabetic ketoacidosis, should be sought on physical examination, even in the absence of fever. Tissue ischemia (heart, brain) can also be a precipitating factor.

Although chronic hyperglycemia is an important etiologic factor leading to complications of Diabetes mellitus, the mechanism by which it leads to such diverse cellular and organ dysfunction is unknown. Four prominent theories, which are not mutually exclusive, have been proposed to explain how hyperglycemia might lead to the chronic complications of Diabetes mellitus. One theory is that increased intracellular glucose leads to the formation of advanced glycosylation end products (AGEs) via the nonenzymatic glycosylation of intra and extracellular proteins. Nonenzymatic glycosylation results from the interaction of glucose with amino groups on proteins. AGEs have been shown to cross link proteins (e.g., collagen, extracellular matrix proteins), accelerate atherosclerosis, promote glomerular dysfunction, reduce nitric oxide synthesis, induce endothelial dysfunction, and alter extracellular matrix composition and structure. The serum level of AGEs correlates with the level of glycemia, and these products accumulate as glomerular filtration rate declines.

**Figure4. Diabetes ketoacidosis**



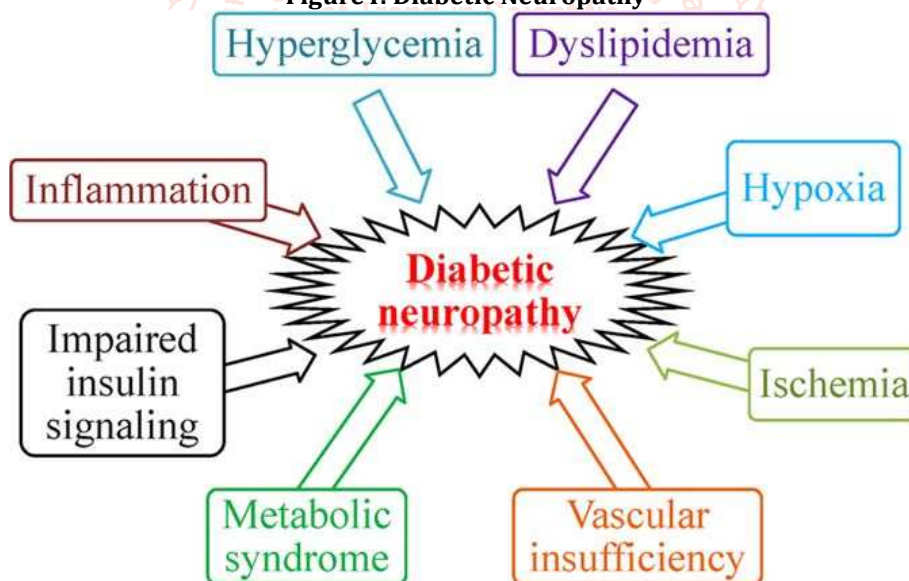
A second theory is based on the observation that hyperglycemia increases glucose metabolism via the sorbitol pathway. Intracellular glucose is predominantly metabolized by phosphorylation and subsequent glycolysis, but when increased, some glucose is converted to sorbitol by the enzyme aldose reductase. Increased sorbitol concentration alters redox potential, increases cellular osmolality, generates reactive oxygen species, and likely leads to other types of cellular dysfunction. However, testing of this theory in humans, using aldose reductase inhibitors, has not demonstrated significant beneficial effects on clinical endpoints of retinopathy, neuropathy, or nephropathy. A third hypothesis proposes that hyperglycemia increases

the formation of diacylglycerol leading to activation of protein kinase C (PKC). Among other actions, PKC alters the transcription of genes for fibronectin, type IV collagen, contractile proteins, and extracellular matrix proteins in endothelial cells and neurons. A fourth theory proposes that hyperglycemia increases the flux through the hexosamine pathway, which generates fructose-6-phosphate, a substrate for O linked glycosylation and proteoglycan production<sup>14</sup>. The hexosamine pathway may alter function by glycosylation of proteins such as endothelial nitric oxide synthase or by changes in gene expression of transforming growth factor (TGF) or plasminogen activator inhibitor-1 (PAI-1).

Growth factors appear to play an important role in Diabetes mellitus related complications, and their production is increased by most of these proposed pathways. Vascular endothelial growth factor (VEGF) is increased locally in diabetic proliferative retinopathy and decreases after laser photocoagulation. TGF is increased in diabetic nephropathy and stimulates basement membrane production of collagen and fibronectin by mesangial cells. Other growth factors, such as platelet derived growth factor, epidermal growth factor, insulin like growth factor I, growth hormone, basic fibroblast growth factor, and even insulin, have been suggested to play a role in Diabetes mellitus related complications. A possible unifying mechanism is that hyperglycemia leads to increased production of reactive oxygen species or superoxide in the mitochondria; these compounds may activate all for of the pathways described above. Although hyperglycemia serves as the initial trigger for complications of diabetes, it is still unknown whether the same pathophysiologic processes are operative in all complications or whether some pathways predominate in certain organs.

Diabetic nephropathy is the leading cause of ESRD in the United States and a leading cause of Diabetes mellitus related morbidity and mortality. Proteinuria in individuals with Diabetes mellitus is associated with markedly reduced survival and increased risk of cardiovascular disease. Individuals with diabetic nephropathy almost always have diabetic retinopathy. Like other microvascular complications, the pathogenesis of diabetic nephropathy is related to chronic hyperglycemia<sup>26</sup>. The mechanisms by which chronic hyperglycemia leads to ESRD, though incompletely defined, involve the effects of soluble factors (growth factors, angiotensin II, endothelin, AGEs), hemodynamic alterations in the renal microcirculation (glomerular hyperfiltration or hyperperfusion, increased glomerular capillary pressure), and structural changes in the glomerulus (increased extracellular matrix, basement membrane thickening, mesangial expansion, fibrosis). Some of these effects may be mediated through angiotensin II receptors<sup>15</sup>. Smoking accelerates the decline in renal function. The natural history of diabetic nephropathy is characterized by a fairly predictable sequence of events that was initially defined for individuals with type 1 Diabetes mellitus but appears to be similar in type 2 Diabetes mellitus. Glomerular hyperperfusion and renal hypertrophy occur in the first years after the onset of Diabetes mellitus and cause an increase of the glomerular filtration rate (GFR).

**Figure4. Diabetic Neuropathy**



Diabetic neuropathy occurs in approximately 50% of individuals with long standing type 1 and type 2 Diabetes mellitus<sup>16</sup>. It may manifest as polyneuropathy, mononeuropathy or autonomic neuropathy. As with other complications of Diabetes mellitus, the development of neuropathy correlates with the duration of diabetes and glycemic control; both myelinated and unmyelinated nerve fibers are lost. Because the clinical features of diabetic neuropathy are similar to those of other neuropathies, the diagnosis of diabetic neuropathy should be made only after other possible etiologies are excluded.

Urinary tract infections (either lower tract or pyelonephritis) are the result of common bacterial agents such as *Escherichia coli*, though several yeast species (*Candida* and *Torulopsis glabrata*) are commonly observed. Complications of urinary tract infections include emphysematous pyelonephritis and emphysematous cystitis. Bacteriuria occurs frequently in individuals with diabetic cystopathy. Susceptibility to furunculosis, superficial candidal infections, and vulvovaginitis are increased. Poor glycemic control is a common denominator in individuals with these infections. Diabetic individuals have an increased rate of colonization of *S. aureus* in the skin folds and nares. Diabetic patients also have a greater risk of postoperative wound infections. Strict glycemic control reduces postoperative infections in diabetic individuals undergoing CABG and should be the goal in all diabetic patients with an infection.

### Miasmatic Background

Diabetes mellitus comprises the pseudopsoric miasm. The pseudopsoric miasm is also known as Tubercular miasm. It is a combination of both Psora and Syphilitic miasm. Tubercular miasm is usually characterized by a "problem child" i.e. slow in comprehension, dull, unable to keep a line of thought, unsocial, morose. He/she getting relief from offensive foot or axillary sweat which when suppressed often induces lung troubles or some other severe disease. The patient always feels better of mental symptoms by an outbreak of an ulcer. The slightest bruise suppurates; the strong tendency is to the formation of pustules. As a general rule, the patient is very intelligent, keen observer and a programmatic planner who wants his life always busy but possesses a sedentary lifestyle.

### Diet and Nutritional Plan:

A diabetic can eat almost any food that other people normally eat provided the food is balanced and within the permissible caloric limits. Proper nutritional management or food plan is essential for better glucose control. This in turn helps to reduce the risk of diabetic complications. The number of factors such as type of diabetic may eat varies on number of factors such as type of diabetics, type of treatment, age of the patient, physical activity etc. The timing and size of the meals would depend on the treatment regimen and your life style. Your physician and dietitian would advice you on these points. Daily consistency regarding the types of food including in the meal, their nutritional information, and the time at which they are consumed will help to normalize the blood glucose levels.

### TREATMENT:

As Homoeopathy is not a science of therapeutics, it is concerned with totality of symptoms or individuality. As regarding the cure of diabetes mellitus by homoeopathic medicine, the individual needs the complete miasmatic and constitutional therapy in the very early stage. In the later stage of type 2 diabetes mellitus especially when the complications arises the therapeutic treatment have more value followed by constitutional treatment.

### Homoeopathic treatment<sup>17</sup>

I found over 40 remedies for diabetes mellitus but when *totality of symptom* agrees every medicine from Materia Medica can be employed. However, only a smaller group is employed most frequently such as:

**Abroma augusta (Olatkambal)** Frequent and profuse urination, dryness of the mouth and great thirst, urination leads exhaustion, Fishy odour of the urine, Diabetes mellitus and insipidus.

**Argentum metallicum (Silver):** Polyuria, frequent urination, urine profuse at night, turbid and sweetish odour, restless sleep, frightful dreams, edematous swollen feet, flatulent distention of abdomen.

**Arsenicum album (Arsenic trioxide):** Urine scanty, burning albuminous, ascites, all prevailing debility, restlessness, burning thirst, drinks often but little at time.

**Codeinum (An Alkaloid from Opium):** Sugar in urine, quantity of urine increased, great thirst, it is said to control disease.

**Cantharis (Spanish fly):** Diabetes complicated with albuminuria, *constant desire to urinate* Membranous scales looking like bran in water. Urine jelly like, sheddy.

**Cephalandra indica (Telakucha):** Diabetes mellitus and insipidus with profuse urination; weakness and exhaustion after urination; sugar in the urine.

**Graphitis (black lead):** Various complications of diabetes where causes are not known.

**Gymnesa sylvestre (Meshasringi or Gurmar):** Is almost specific for DM called as "Sugar Killer" diminishes sugar in urine; Profuse micturition loaded with sugar, extreme weakness after passing large quantities of urine. Polyuria; day and night.

**Helleborus (Snow-rose):** Frequent urging to urinate but small quantities emitted, profuse urination, urine pale and watery, *dropsical swelling*.

**Helonias Chamailirium (Uricorn-root)** Diabetes mellitus and insipidus, urine profuse and clear, phosphatic and albuminous, great thirst, restlessness, profound melancholy, irritable, boring pain across the lumbar region.

**Insulin:** Supposed to be specific and useful in case of carbuncles resulting from diabetes mellitus.

**Lacticum acidum (Lactic acid):** Frequent passing of large quantities of sugar in urine, great thirst, rheumatic pains in joints.

**Murex (Purple Fish):** Frequent urine at night, smells like Valerian, constant urging.

**Natrum phosphoricum:** They are of great value in diabetes. Profuse urination, urine loaded with bile, lithic deposition in urine, sedentary habits especially when there is a succession of boils.

**Natrum sulphuricum (Sulphate of Sodium):** A remedy especially indicated for the so-called hydrogenoid constitution, where the complaints are such as are due to living in damp houses, basements, cellars. Diabetes with nervous origin when due to worry, mental over work and sexual excess.

**Phosphoricum acidum (Phosphoric acid):** Frequent and profuse watery urination, milk-like urine, *great debility*.

**Phosphorus:** Diabetes mellitus in phthisis in impotency, urine contain large amount of salt in the morning and excess of sugar in the evening.

**Plumbum metallicum (Lead):** Urine frequent, scanty, albuminous, low specific gravity.

**Rhus aromatica (Fragrant sumach):** Large quantity of urine, *urine pale*, albuminous, specific gravity low.

**Squilla maritime (Sea-onion):** Great urging *much watery urine*, involuntary spurting when coughing, a slow acting remedy correspondence to ailments requiring several days to reach their maximum.



**Lac defloratum (Skimmed Milk):** Diabetes with faulty nutrition. Albuminuria and other affections of kidney.

**Syzygium Jambolanum (Jambol seeds):** It has a specific action in diminishing and cause to disappear the sugar in urine, great thirst, and weakness, urine in very large quantities, specific gravity high. Ten drops to be taken twice or thrice daily.

**Uranium nitricum (Nitrate of Uranium):** Profuse urination, debility, acid in urine, incontinence, unable to retain urine, excessive thirst, diarrhea of the dyspepticus.

**Terebinthinum (Turpentine):** Profuse, cloudy, smoky, and albuminous urine, sediments like coffee grounds, haematuria..

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