



Homoeopathic Constitutional Medicine in the Management of DM Type 2

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INTRODUCTION

Diabetes is the body's failure to metabolize blood sugar properly. Type 2 diabetes mellitus is the commonest form of diabetes.

Diabetes mellitus affects at least 16 million US residents, ranks 7th as the cause of death in United States, and costs the national economy over \$100 billion yearly.

India has the maximum increase during the last few years. The prevalence of type 2 diabetes mellitus is 2.4% in rural population and 11.6% in urban population.

World health organization predicts that by 2025 India will have the largest number of diabetics in the world. In India there are more than 19.4 million diabetics currently. The number is expected to increase to 57.2 million by 2025 and 80.9 million by 2030. About 32 million people in India are suffering from diabetes and only one-third of them have been diagnosed. Of those diagnosed, only 5-7 million people receive treatments.

It is becoming a great threat to the developing nations like India, as they switch on to the unfavorable modification of life style and dietary habits that are associated with urbanization are believed to be the most important factors for the development of diabetes.

Diabetic patients if undiagnosed or inadequately treated, develop multiple chronic complications leading to irreversible disability and death.

In developed world diabetes is the

Most significant cause of adult blindness in the non-elderly.

Leading cause of non-traumatic amputation in adults.

Diabetic nephropathy is the main illness requiring dialysis.

Modern medicines contribute immensely and saved millions of people but as majority of the patients are mainly from geriatric group and most of the drugs are metabolized in the liver. And as the age advances the ability of the liver to degrade the toxins is diminished which leads to more toxicity. Thus, the side effects of oral hypoglycemic agents and the development of insulin hypersensitivity and resistance must be taken into consideration.

The most common side effects of insulin therapy is low blood sugar, and others like swelling or itching at the site of injection, worsening of diabetic retinopathy lipodystrophy, allergic reactions, sodium retention and general body swelling, a type of chest discomfort, cough and dyspnea.

The side effects of oral hypoglycemic agents are Hypoglycemia, allergic reactions, hyponatraemia, cholestasis, lactic acidosis, anorexia, nausea, vomiting, diarrhea, abdominal fullness, and bloating, transient visual disturbance. Occasional transits elevation of liver enzymes, sinusitis, urinary tract infections, hepatic injury, headache, anemia, and edema, malabsorption of vitamin B12 and folic acid, muscle pain, weakness and weight gain.

Homoeopathy has been said to be having much efficacy in treating type 2 diabetes mellitus because of its systemic analysis and holistic approach of considering body, mind and disease and with the concept of individualization and dynamization. One endeavor's to treat all the symptoms at one time and with one drug. The selection of potency, the repetition time of releasing the remedial force depend upon an accurate assessment of the pathogenesis as well as the pathology and the qualitative assessment of the susceptibility and sensitivity. Thus, all this criteria is considered for an efficient outcome in the management of the case.

Diabetes mellitus is a clinical syndrome characterized by hyperglycemia due to absolute or relative deficiency of insulin. There is disturbance of intermediary metabolism mainly manifesting as chronic hyperglycemia.

WHO recognizes three main forms of diabetes mellitus namely-

1. Type I diabetes mellitus (failure of pancreas to produce insulin)
2. Type II diabetes mellitus (characterized by insulin resistance in target cells)

3. Gestational diabetes

Etiological Classification**I. Type I DM (β -cell destruction, absolute insulin deficiency). Insulin dependent diabetes mellitus (IDDM)**

(a) Autoimmune

(b) Idiopathic

II. Type II DM – Non-insulin dependent (NIDDM)**III. Maturity onset diabetes in young (MODY 1-6)**Genetic defects in β -cell function have the following mutations:a. MODY 1 - Hepatocyte Nuclear Transcription factor (HNF) 4 α

b. MODY 2 - Glucokinase

c. MODY 3 - HNF - 1 α

d. MODY 4 - Insulin promotor factor-1 (IPF1)

e. MODY 5 - INF - 1 β

f. MODY 6 - Neurogenic differentiation1, (Neuro D1)

IV. Secondary causes

Pancreatic disease

Cystic fibrosis

Trauma/pancreatopathy

Hemochromatosis

Pancreatitis

Fibrocalcific pancreatic diabetes (FCPD)

Carcinoma of pancreas

Endocrine disorders

Acromegaly Conn's syn.

Cushing's syn.

Pheochromocytoma

Glucagonoma

Hyperthyroidism

Drug-induced

Glucocorticoids

Phenytoin

Diazoxide Pentamidine

Thiazides α -interferon β -adrenergic agonists Thyroid hormone**4. Infections**

Congenital rubella Coxsackie B virus

Cytomegalovirus

5. Insulin receptor defects

Anti-insulin receptor antibodies (Stiffman syn.)

Lipoatrophic diabetes

6. Other genetic syndromes (sometimes associated with DM)

Down's syndrome

Turner's syndrome

Wolfram's syndrome

Prader-Willi syndrome

Klinefelter's syndrome

Laurence-Moon-Biedl syndrome

Myotonic dystrophy

Friedreich's ataxia

Huntington's chorea

Porphyria

V. Gestational diabetes

VI. Impaired glucose tolerance (Borderline diabetes)

a. Primary: Obese, non-obese

b. Secondary: All conditions mentioned under secondary

DM, cirrhosis of liver, kidney failure, chronic undernutrition, hypokalaemia, stress, e.g. myocardial infarction.

Type 2 diabetes

Pathology

Type 2 diabetes is a diagnosis of exclusion, i.e. it is made when type 1 diabetes and other types of diabetes are ruled out, and is highly heterogeneous. The natural history of typical type 2 diabetes is initially, insulin resistance leads to elevated insulin secretion in order to maintain normal blood glucose levels. However, in susceptible individuals, the pancreatic β cells are unable to sustain the increased demand for insulin and a slowly progressive insulin deficiency develops. Some patients develop diabetes at a young age, usually driven by insulin resistance due to obesity and ethnicity; others, particularly the elderly, develop diabetes despite being non-obese and may have more pronounced β -cell failure. The key feature is a 'relative' insulin deficiency, such that there is insufficient insulin production to overcome the resistance to insulin action. This contrasts with type 1 diabetes, in which there is rapid loss of insulin production and an absolute deficiency, resulting in ketoacidosis and death if the insulin is not replaced.

Insulin resistance

Type 2 diabetes, or its antecedent, impaired glucose tolerance, is one of a cluster of conditions thought to be caused by resistance to insulin action. Thus, patients with type 2 diabetes often have associated disorders including hypertension, dyslipidaemia (characterized by elevated levels of small dense low-density lipoprotein (LDL) cholesterol and triglycerides, and a low level of high-density lipoprotein (HDL) cholesterol), nonalcoholic fatty liver and, in women, polycystic ovarian syndrome. This cluster has been termed the 'insulin resistance syndrome' or 'metabolic syndrome', and is much more common in patients who are obese.

The primary cause of insulin resistance remains unclear; it is likely that there are multiple defects in insulin signalling, affecting several tissues. One theory is centred around the adipocyte; this is particularly appealing, as obesity is a major cause of increased insulin resistance. Intra-abdominal 'central' adipose tissue is metabolically active, and releases large quantities of FFAs, which may induce insulin resistance because they compete with glucose as a fuel supply for oxidation in peripheral tissues such as muscle. In addition, adipose tissue releases a number of hormones (including a variety of peptides, called 'adipokines' because they are structurally similar to immunological 'cytokines') which act on specific receptors to influence sensitivity to insulin in other tissues. Because the venous drainage of visceral adipose tissue is into the portal vein, central obesity may have a particularly potent influence on insulin sensitivity in the liver, and thereby adversely affect gluconeogenesis and hepatic lipid metabolism. Physical activity is another important determinant of insulin sensitivity. Inactivity is associated with downregulation of insulin-sensitive kinases and may promote accumulation of FFAs within skeletal muscle. Sedentary people are therefore more insulin-resistant than active people with the same degree of obesity. Moreover, physical activity allows non-insulin-dependent glucose uptake into muscle, reducing the 'demand' on the pancreatic β cells to produce insulin.

Deposition of fat in the liver is a common association with central obesity and is exacerbated by insulin resistance and/or deficiency. Many patients with type 2 diabetes have evidence of fatty infiltration of the liver (non-alcoholic fatty liver disease (NAFLD)). This condition may improve with effective treatment of the diabetes and dyslipidaemia, but despite this, a few patients progress to non-alcoholic steatohepatitis (NASH, p. 959) and cirrhosis.

Pancreatic β -cell failure

In the early stages of type 2 diabetes, reduction in the total mass of pancreatic islet tissue is modest. At the time of diagnosis, around 50% of β -cell function has been lost and this declines progressively. Some pathological changes are typical of type 2 diabetes, the most consistent of which is deposition of amyloid in the islets. In addition, elevated plasma glucose and FFAs exert toxic effects on pancreatic β cells to impair insulin secretion. However, while β -cell numbers are reduced, β -cell mass is unchanged and glucagon secretion is increased, which may contribute to hyperglycaemia.

Genetic predisposition

Genetic factors are important in type 2 diabetes, as shown by marked differences in susceptibility in different ethnic groups and by studies in monozygotic twins where concordance rates for type 2 diabetes approach 100%. However, many genes are involved and the chance of developing diabetes is also influenced very powerfully by environmental factors

INVESTIGATIONS

Urine testing

Glucose

Testing the urine for glucose with dipsticks is a common screening procedure for detecting diabetes. If possible, testing should be performed on urine passed 1–2 hours after a meal to maximise sensitivity. Glycosuria always warrants further assessment by blood testing. The greatest disadvantage of urinary glucose measurement is the individual variation in renal threshold for glucose. The most frequent cause of glycosuria is a low renal threshold, which is common during pregnancy and in young people; the resulting 'renal glycosuria' is a benign condition unrelated to diabetes. Another disadvantage is that some drugs (such as β -lactam antibiotics, levodopa and salicylates) may interfere with urine glucose tests.

Ketones

Ketone bodies can be identified by the nitroprusside reaction, which measures acetoacetate, using either tablets or dipsticks. Ketonuria may be found in normal people who have been fasting or exercising strenuously for long periods, who have been vomiting repeatedly, or who have been eating a diet high in fat and low in carbohydrate. Ketonuria is therefore not pathognomonic of diabetes but, if associated with glycosuria, the diagnosis of diabetes is highly likely. In diabetic ketoacidosis, ketones can also be detected in plasma using test sticks.

Protein

Standard dipstick testing for albumin detects urinary albumin at concentrations above 300 mg/L, but smaller amounts (microalbuminuria, can only be measured using specific albumin dipsticks or by quantitative biochemical laboratory measurement. Microalbuminuria or proteinuria, in the absence of urinary tract infection, is an important indicator of diabetic nephropathy and/or increased risk of macrovascular disease.

Blood testing

Glucose

Laboratory glucose testing in blood relies upon an enzymatic reaction (glucose oxidase) and is cheap, usually automated and highly reliable. However, blood glucose levels depend on whether the patient has eaten recently, so it is important to consider the circumstances in which the blood sample was taken. Blood glucose can also be measured with colorimetric or other testing sticks, which are often read with a portable electronic meter. These are used for capillary (fingerprick) testing to monitor diabetes treatment. There is some debate as to whether selfmonitoring

In people with type 2 diabetes improves glycaemic control. Many countries now only offer selfmonitoring to people with type 2 diabetes taking insulin therapy. To make the diagnosis of diabetes, the blood glucose concentration should be estimated using an accurate laboratory method rather than a portable technique. Glucose concentrations are lower in venous than arterial or capillary (finger prick) blood. Whole blood glucose concentrations are lower than plasma concentrations because red blood cells contain relatively little glucose. Venous plasma values are usually the most reliable for diagnostic purposes.

Ketones

Blood ketone monitoring is increasingly available. Urinary ketone measurements described above are semi-quantitative, difficult to perform and retrospective (i.e. the urine has accumulated over several hours), and do not measure the major ketone in blood during diabetic ketoacidosis (DKA), beta-hydroxybutyrate (β -OHB). Whole blood ketone monitoring detects β -OHB and is useful in assisting with insulin adjustment during intercurrent illness or sustained hyperglycaemia to prevent or detect DKA. Blood ketone monitoring is also useful in monitoring resolution of DKA in hospitalized patients.

Glycated haemoglobin

Glycated haemoglobin provides an accurate and objective measure of glycaemic control integrated over a period of weeks to months. In diabetes, the slow non-enzymatic covalent attachment of glucose to haemoglobin (glycation) increases the amount in the HbA1 (HbA1c) fraction relative to nonglycated adult haemoglobin (HbA0). These fractions can be separated by chromatography; laboratories may report glycated haemoglobin as total glycated haemoglobin (GHb), HbA1 or HbA1c. In most countries, HbA1c is the preferred measurement. The rate of formation of HbA1c is directly proportional to the ambient blood glucose concentration; a rise of 1% in HbA1c corresponds to an approximate average increase of 2 mmol/L (36 mg/dL) in blood glucose. Although HbA1c concentration reflects the integrated blood glucose control over the lifespan of erythrocytes (120 days), HbA1c is most sensitive to changes in glycaemic control occurring in the month before measurement. Various assay methods are used to measure HbA1c, but most laboratories have been reporting HbA1c values (as %) aligned with the reference range that was used in To allow worldwide comparisons of HbA1c values, the International Federation of Clinical Chemistry and Laboratory Medicine (IFCC) has developed a standard method; IFCC-standardised HbA1c values are reported in mmol/mol. In 2011, many countries adopted the IFCC reference method. HbA1c estimates may be erroneously diminished in anaemia or during pregnancy, and may be difficult to interpret with some assay methods in patients who have uraemia or a haemoglobinopathy.

PRESENTING PROBLEMS IN DIABETES MELLITUS

Newly discovered hyperglycaemia Hyperglycaemia is a very common biochemical abnormality. It is frequently detected on routine biochemical Analysis of asymptomatic patients, following routine dipstick testing of urine showing glycosuria, or during severe illness

HOMOEOPATHIC APPROACH:

Homoeopathy is based on the principles that disease is a total application of mind and body. Moreover, homoeopathy recognizes importance of underlying causes such as genetic and inherited factors as the root of any ailment of the body. Homoeopathic medicine prescribed in such conditions covers those criteria and it is very crucial in management of deep rooted diseases

Concept of disease in homoeopathy is that disease is a total affection of mind and body, the disturbance of the whole organism. Individual organs are not the cause of the illness but the disturbance at internal level (i.e. life-force or vital energy). Therefore, homoeopathy does not believe in giving different medicines for different afflicted parts of body but rather give a constitutional remedy which will cover the disturbance of the whole person.

When we talk about disease like diabetes, we take in consideration in terms of management rather than cure, this is because dietary measures and daily exercises etc are mandatory along with general management, has a vital role in the cure and management of diabetic patients which not only treats the patients superficially but also drives the symptoms away and heals the patient from within. So, undoubtedly one can prove that homoeopathy is the medicine of future.

Miasmatic Background

DM 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.⁸

SAMUEL LILIETHAL recommends the following remedies-

Acetic Acid, Acid Phos, Arg Met, Ars Brom, Berb Vulg, Bovista, Calc Phos, Carb Acid, Cuprum Met, Curare, Hepar, Kali Brom, Kali Mur, Kreos, Lactic Acid, Lac Def, Lith Carb, Lyco, Mag Sulph, Mosch, Nux Vom, Opium, Pic Acid, Sulph Acid, Plumb, Sec Cor, Syz Jamb, Tarent, Tub, Terbenthina, Uran Nit.

WILLIAM BOERICKE recommends the following remedies-

Ars, Aur, Brom, Coca, Codein, Helleb, Syzyg, Phosp, Phlorid, Uran Nit.

E.A. FARRINGTON says

Phosphoric Acid and Lactic Acids are the principal acids for diabetes mellitus. Phosphorus is one of the few remedies that act on the pancreas and can be remembered in diabetes mellitus.¹⁰

T S IYER suggests

For debility, Phos, phos acid, ars, china.

For carbuncles, arnica, phos, ars, sil, hep

For nervous symptoms phos, arg, aur, if cerebral nux vom, sil, phos,

Lactic Acid: An exceedingly good remedy in the gastro hepatic variety of diabetes. ¹⁴

Acid phos: Corresponds diabetes of nervous origin , the urine is increased perhaps milky in colour and containing much sugar. It is unquestionably curative of diabetes in early stages.15

RICHARD HUGHES

Gives much importance to the remedies like acid phos and Uranium and writes further an interesting experience of Dr. Stiegele. He found that syzygium diminished the quantity of sugar in the urine but had no effect on general health, whilst arsenicum improved the general health without diminishing the quantity of sugar. Here therefore combined arsenicum 6 and syzygium 3X as a double medicine and gave it in 6 cases came out with curative results.12

Synthesis repertory 16

In synthesis repertory total 34 rubrics are found in relation with diabetes

GENERALS – DIABETES MELLITUS

During my research work on this topic 30 cases of diabetes mellitus type 2 have been considered by using random sampling technique, age group of 30-70 years, of both sex and religion with all socioeconomic status has been taken. All are prescribed with homeopathic medicine based on miasmatic background, with diet and regimen and accordingly the various outcomes parameters were assessed by different attributes and variables.

Result got after prescription of homoeopathic remedies outcome of 30 cases 23 cases showed improvement, 4 showed partial improvement, and 3 not improved. Overall success rate of treatment after homeopathic medications in diabetes mellitus 2 is 73%.

Conclusion:

There is role of constitutional approach in selection of Homeopathic remedies in cases of diabetes mellitus type 2. Hence Homoeopathic medicine has wide scope and role of constitution in the management of diabetes mellitus type 2.