



Homoeopathic Management of Diabetes Mellitus

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ABSTRACT:

Diabetes Mellitus (DM) is a chronic metabolic disease whose prevalence is constantly increasing worldwide. As a result of this trend, it is fast becoming an epidemic in some countries around the world, with the number of sufferers expected to double in the next decade due to an aging population, adding to the already existing burden on health care providers, especially in less developed countries.

Type 2 diabetes mellitus consists of a series of dysfunctions characterized by hyperglycemia and resulting from a combination of insulin resistance, insufficient insulin secretion, and excessive or inappropriate glucagon secretion.

Type 2 diabetes is asymptomatic with classic symptoms: Polyuria, polydipsia, polyphagia and weight loss and other symptoms with various infections.

Type 2 diabetes is increasingly responsible for hypertension, obesity and other diabetic complications.

The cause of type 2 diabetes is genetic and environmental with this different treatment regimen available. Homeopathy considers the person as a whole and deals primarily with constitutional and organ remedies.

Diabetes was already known in ancient times. Characterized by excessive urine flow and insatiable thirst, the name of this disease was coined by the Greco-Roman physician Aretaeus of Cappadocia (ca. 80-130 AD) and is derived from the Greek word diabainein ("to flow"). The adjective mellitus, which comes from Latin and means "honey-sweet," was added by the German physician Johann Peter Frank (1745-1821) to distinguish diabetes mellitus, or "diabetes," from diabetes insipidus. Johann Peter Frank was also the one who, in 1790, introduced the yeast fermentation test for the quantitative determination of glucose in the urine, freeing the doctors of his time from the need to taste the urine of their patients. Called madhumeha according to Ayurveda, it is a kapha-type metabolic disorder in which reduced agni function leads to a tendency towards high blood sugar.

The World Health Organization (WHO) estimates that more than 180 million people worldwide have diabetes. This number is likely to more than double by 2030. In 2005, an estimated 1.1 million people died of diabetes. Almost 80% of diabetes deaths occur in low- and middle-income countries. Almost half of diabetes deaths occur in people under the age of 70; 55% of diabetes deaths are in women⁸. It is estimated that by 2010 the diabetic population will increase to 221 million from 110 million in 1994. The majority of new cases will be type 2 diabetes and most of these will be in China, the Indian subcontinent and Africa. . From 65 million cases of type 2 diabetes in Asia and Oceania in 1995, the number is estimated to double to 135 million by 2010.

In India, an estimated 19.4 million individuals are currently affected by this deadly disease, which is likely to rise to 57.2 million by 2025. By 2025, India is projected to have the most people with diabetes mellitus in the world. In Kerala, about 8% of the adult population suffers from diabetes; this ranges from 3% in rural areas and 20% in cities. The only exception is coastal fishermen, among whom the prevalence is only 3%. According to the most conservative estimates, there are about 1.5 million diabetics in Kerala.

KEYWORDS: Diabetes Mellitus, Sugar, Homoeopathic Repertory, Homeopathy and Homeopathic Medicine.

INTRODUCTION:

Diabetes known to ancient Indian doctors as "Madhumeh" which means discharge of sweat in urine. It was considered as a kind of 'Prameh' i.e. vital drain. This definition of diabetes is clinically correct even today.

The first moment of writing about diabetes comes from the charakasamhitha written in 400 BC. Charaka wrote that it is a disease of sedentary, obese people whose food intake is very high. Western medicine provides the first literature on diabetes in AD 200. It was written by Dr. Aarits Capaotian. This knowledge may have been transferred from India to Asia Minor at that time, as has been suggested. Charaka's description in his book is very similar to

the symptomatology of Diabetes Mellitus Diabetes Mellitus is a chronic metabolic disorder of protein, fat and carbohydrate metabolism. Today, diabetes mellitus is the leading cause of death. Diabetes mellitus has higher prevalence in urban area than in rural areas due to lifestyle, socio-economic factors, culture, obesity, stress and different way of life and adaptation. Diabetes Mellitus is a silent killer, sometimes diabetes mellitus manifests itself asymptotically, but the patient does not know about the disease.

As of 2014, there are an estimated 387 million people with diabetes worldwide, accounting for about 90% of diabetes cases. This corresponds to about 8.3% of the world's adult population. It is common in both developed and developing countries. The number of people with diabetes is expected to increase to 592 million by 2035. This increase is thought to be primarily due to an aging global population, a decline in exercise and rising obesity rates. The World Health Organization (WHO) considers it a global epidemic.

Now that the disease has become so widespread that it has become a challenge to all medical fraternities, high level of research is going on all around to understand the disease, its clinicopathological correlates and possible solutions in terms of new therapeutic drugs. These drugs have an effect and a side effect and work only to change the effect of the disease but not to change the cause.

The disease continues to progress, and so do they continue to avoid vital organs. In most cases it was seen that the dose of OHA was gradually increased and some of them required insulin to control their blood sugar levels. Due to the current lifestyle of improper diet, lack of exercise, stress and addictions, we get a large number of patients who suffer from diabetes in their early years.

Homeopathy can help improve the overall health of a diabetic. This can be achieved by filing a constitutional remedy. The result will be improved overall well-being, reduced need for medication and improved diabetes control. Therefore, one should try to understand the usefulness of homeopathic medicine exclusively for diabetes.

Hahnemann was the first physician to advocate accurate, unbiased observation as the basis of the scientific clinical investigation of disease. He is aware of the importance of adverse factors, both tangible and intangible. He does not lose sight of the importance of constitutional predisposition in the genesis of the disease.

Studying the individual in a holistic way forms the backbone of homeopathic practice. Similarly, studying Materia Medica with a holistic approach allows us to gain a better and more comprehensive understanding of the simillimum. The study of Materia Medica from source books and clinical experience, when combined with individual study, enables us to develop general guidelines and assists in infusing life with the medicines we study. We must begin to study the individual from his basic instinct and understand how he tries to deal with them; given social circumstances, social values and requirements. We will understand how, thanks to this adaptation, he maintains his own values and remains in society. It can only do so by effectively controlling basic instincts. Adaptation thus requires a certain mobility, control or suppression of these drives. There is a cost in this process, as all of the above movements require some energy expenditure, which creates stress in the individual. Effects at the mind-body level are well known in the form of symptoms and signs.

Under stress, the central adaptation mechanism of the psychoneuro-endocrine axis tries to adapt to the circumstances in its own special way based on individual predisposition and disposition. When the balance is disturbed, it produces clinical manifestations at the mind-body level. Hahnemann's theory of homeopathy helps us determine a theoretical framework for disease that allows us to understand the dynamics of energy patterns in health and disease in the forms of medicine images. The basis of this is the miasmata themselves, which contribute an underlying note. Combined with Hering's law of cure, it allows us a clear picture of how the dynamics of illness and health work.

We are not limited to a diagnosis, but to a clearly defined understanding of the relationship between the picture of symptoms and the picture of treatment. A perfect match between the two, the true simillimum is a clear concept that makes it possible to understand the processes involved in disease and treatment.

There have been many attempts to connect the effect of drugs with homeopathic thinking. From the many attempts to combine them, there is an innate belief that the drugs are related in some special way. This may reflect a deep subjective sense of the relatedness of all things. Disease and health are related because the fluctuating patterns of the vital force rise and fall as it travels through matter.

This is the 21st century, due to modernization and globalization, the world is facing the problem of anxiety, anxiety and various disorders. The destructive tendency is more dominant, indicating a syphilitic miasma.

Total health is still a concern of society today. Many of the Medical College, Govt. and other people with whom they try to overcome the problem. Some have solutions, but a universal conclusion is yet to come.

I am trying to gather information and also want to study these upper respiratory disease problems in detail. I will be the only thread connecting the pearls given by these devotees. Still, I'll try my best to come to an acceptable conclusion.

REVIEW OF LITERATURE:

Health status of India:

The birth rate (births per 1,000 people per year) is 22.22 births/1,000 population (2009 estimate), while the death rate (deaths per 1,000 people per year) is 6.4 deaths/1,000 population. The fertility rate is 2.72 births/woman (NFHS-3, 2008) and the infant mortality rate is 30.15 deaths/1,000 live births (2009

est.). India has the largest illiterate population in the world. India's literacy rate as of 2011 census is 74.04%, with a male literacy rate of 82.14% and a female literacy rate of 65.46%.

India has a huge healthcare burden of managing various epidemics and endemics, which is bound to increase further due to the rapid urbanization and lifestyle changes taking place in the country. India faces several challenges in managing such diseases, including lack of public health awareness, limited number of health facilities and high cost of treatment. There are several barriers related to patients, society and the health care system that must be addressed by government and health care providers. India must take drastic and urgent steps to create an integrated national system for early detection and prevention of the disease.

India, like many other developing countries, faces the dual threat of the ongoing problem of a number of communicable diseases as well as the recent emergence of lifestyle-related non-communicable diseases.

Rapid urbanization and industrialization have brought progress on the social and economic front in developing countries such as India, resulting in dramatic lifestyle changes leading to lifestyle-related diseases. The transition from traditional to modern lifestyles, consumption of liquors and diets rich in fat and calories combined with high levels of mental stress have exacerbated the problem, further leading to the development of many communicable diseases in daily life. One such problem that is troubling the nation a lot is "Diabetes Mellitus".

Diabetes Mellitus:

Diabetes mellitus (DM) includes a group of common metabolic disorders (syndrome) characterized by hyperglycemia. Depending on the etiology of DM, factors contributing to hyperglycemia may include decreased insulin secretion, decreased glucose utilization, and increased glucose production.

The metabolic deregulation associated with DM causes secondary pathophysiological changes in multiple organ systems that pose a tremendous burden to individuals with diabetes and to the health care system. In the United States, DM is the leading cause of end-stage renal disease, non-traumatic lower limb amputations, and adult blindness.

With increasing incidence worldwide, DM is likely to continue to be a major cause of morbidity and mortality in the foreseeable future.

Diabetes has become one of the most common diseases of the current population of human beings on the surface of mother earth. The prevalence of this condition increases as we progress through the development saga. Diabetes in itself is a source of difficulties and psychological burden for the patient, and with this are added problems associated with complications. Although there are many complications in diabetes, the association of dyslipidemia with diabetes is the most problematic, not because of the severity of symptoms, but because of its "silent killer nature." Diabetes can affect lipid and lipoprotein metabolism through several mechanisms.

INSULIN

'Insulin' is a small protein; human insulin has a molecular weight of 5808. It consists of two interconnected amino acid chains. When the two amino acid chains are split, the functional activity of the insulin molecule is lost.

Insulin was first isolated from the pancreas in 1922 by Banting and Best, and the view of a patient with severe diabetes changed from one of rapid decline and death to that of an almost normal person.

Historically, insulin has been associated with "blood sugar" and it is true that insulin has a profound effect on carbohydrate metabolism. Nevertheless, it is precisely the abnormalities of fat metabolism that cause conditions such as acidosis and arteriosclerosis, which are the usual causes of death in the diabetic patient. Also, hospitalized patients with prolonged diabetes, reduced ability to synthesize protein, lead to tissue loss and also many cellular functional disorders. It is therefore clear that insulin affects the metabolism of fats and proteins almost as much as it does the metabolism of carbohydrates.

METABOLIC EFFECTS:

When there are large amounts of energy-giving foods in the diet, especially excessive amounts of carbohydrates and proteins, insulin is secreted in large quantities. This is especially true for an excess of carbohydrates, less so for an excess of protein, but only slightly for fats as well.

Insulin, in turn, plays an important role in storing excess energy substances. In the case of an excess of carbohydrates, it causes their storage as glycogen mainly in the liver and muscles. It causes fat to be stored in adipose tissue. Also, any excess carbohydrates that cannot be stored as glycogen are converted to fat and also stored in adipose tissue. In the case of proteins, insulin has a direct effect on promoting the uptake of amino acids by cells and the conversion of these amino acids into protein. In addition, it inhibits the breakdown of proteins that are already in the cells.

REGULATION OF INSULIN SECRETION:

The main physiological stimulus for insulin secretion is an increase in serum glucose concentration, which can be elegantly demonstrated during in vitro studies with isolated islets of Langerhans.

The time course of the insulin response to glucose has also been studied, and the response to glucose is biphasic with a rapid initial spike followed by a prolonged second phase that continues until the stimuli are removed. The reason for this biphasic response is unclear, but not all insulin secretagogues produce a biphasic release pattern, eg hypoglycaemic sulfonylureas. Many other agents have been shown to affect the rate of insulin secretion in vivo and in vitro. All of them can be grouped as follows:

(a) Primary stimuli- which do not require the presence of glucose to exert their effects, e.g. Glucose, Amino acids (arginine and leucine), Ketones, fatty acids, Glucagon.

(b) Secondary Stimuli/Secretion Potentiator – these require the presence of glucose to exert their effect eg secretin, pancreaticozym, G.I.P. (glucose-dependent insulinotropic peptide), acetylcholine.

(c) Inhibitors- these substances inhibit the secretion of insulin, eg adrenaline, nor-adrenaline, somatostatin.

Primary stimuli of insulin secretion are thought to exert their effects by altering calcium ion (Ca^{++}) fluxes across the B cell membrane to increase the concentration of free intracellular calcium.

In contrast, secondary stimuli or a secretory potentiator may act through changes in intracellular cyclic adenosine monophosphate (c-AMP) concentration.

Regulation of insulin secretion in response to factors such as acetylcholine is the activation of calcium phospholipid-dependent protein kinase (protein kinase C). The response is initiated by stimulating the turnover of inositol lipids in the membrane, leading to the production of glycerol: this in turn leads to the activation of the kinase.

DIABETES MELLITUS:

Introduction:

Diabetes has become a major health problem in India. According to the Diabetes Atlas published by the International Diabetes Federation (IDF), diabetes is increasing alarmingly in India, with more than 65.1 million people estimated to be suffering from the disease, compared to 50.8 million in 2010 (IDF, Diabetes Atlas, 6th edition – 2013). Obesity is threatening to reach epidemic proportions among India's middle-class children and teenagers as young people choose Western fast food over traditional cuisine. Doctors in India put gastric bands on children as young as 13.

The countries with the largest number of diabetics will be India, China and the USA by 2030. It is estimated that one in five people with diabetes will be Indian. Given these numbers alone, the economic burden of diabetes in India is among the highest in the world. However, the real burden of the disease is due to associated complications that lead to increased morbidity and mortality.

A recent report confirmed that increasing obesity in South Asians is primarily due to nutrition, lifestyle and demographic changes, increasingly faulty diets and physical inactivity against a background of genetic predisposition (Misra A, Shrivastava U.; Obesity and dyslipidemia in South Asians; *Živiny* 2013).

Obesity appears to be spreading across India at least in part as a result of the invasion of processed Western foods. India's economic boom has been accompanied by a meteoric rise in the number of people with diabetes - and those at risk. Prevalence rates are as high as 20% in some cities, and recent data have shown surprisingly elevated rates in rural areas. According to a recent report by Standard and Poor's, the fast-food market is worth \$11.3 billion and is set to double in three years, largely due to a surge in market share in smaller cities across the country.

Rapid urbanization and industrialization have brought progress on the social and economic front in developing countries such as India, resulting in dramatic lifestyle changes leading to lifestyle-related diseases. The transition from a traditional to a modern lifestyle, eating a diet rich in fat and calories combined with high levels of mental stress has further exacerbated this problem. There are several studies from different parts of India that reveal an increasing trend in the prevalence of type II diabetes in urban areas. A national urban survey in 2000 found that the prevalence of diabetes in urban India among adults was 12.1 percent. Recent data has illustrated the impact of the socio-economic transformation taking place in rural India. The transition has occurred in the past 15 years, with prevalence rising from 2.4 percent to 6.4 percent.

India as a developing country undergoing epidemiological changes with rapid changes in lifestyle, diet and obesity, diseases like diabetes are no longer considered a disease of the rich. Countries like India, often with limited resources, have to cope with the double burden of infectious diseases and diabetes epidemics.

Definition:

Simply put, Diabetes is a chronic disease that occurs when the pancreas does not produce enough insulin, or when the body cannot effectively use the insulin it does produce. This leads to an increased concentration of glucose in the blood (hyperglycemia).

Risk Factors for Diabetes in Indians:

Risk factors for diabetes in Indians are:

□ Age – Indians develop diabetes at a very young age, at least 10 to 15 years earlier than the western population. Early onset of diabetes provides enough time for the development of chronic complications of diabetes. The incidence of diabetes increases with age. In India, life expectancy has increased; therefore, more and more people are being diagnosed with diabetes.

□ Family history – The prevalence of diabetes increases with a family history of diabetes. The risk of a child developing diabetes with a parental history increases above 50 percent. A high incidence of diabetes is observed in first-degree relatives. Indians have a high genetic risk of diabetes, as observed in Asian Indians who have migrated to other countries. They were found to have higher rates of diabetes compared to the local population.

□ Central obesity – the association of obesity with type 2 diabetes is well known. Even within an acceptable range of body weight, weight gain can increase the risk of diabetes. Excess body fat specially concentrated

in the abdomen has an increased risk of diabetes. The cut-off limits for waist circumference for Indians have been recommended at 90 cm for men and 80 cm for women. Abdominal obesity is defined by a waist circumference above these limits.

□ Physical Inactivity and Sedentary Lifestyle – There is ample evidence to show that physical inactivity is an independent factor in the development of type 2 diabetes. The availability of motorized transport and the shift in employment, combined with an excess of television programs, have reduced physical activity in all population groups.

□ Insulin resistance – Asian Indians have been found to be more insulin resistant compared to the white population. They have higher insulin levels to achieve the same blood glucose control. A cluster of factors consisting of abnormal fats (Dyslipidemia), high blood pressure, obesity and abnormal glucose levels known as metabolic syndrome is highly prevalent in Asian Indians.

□ Urbanization – Developing countries like India are undergoing rapid urbanization. Urbanization is associated with increasing obesity, a decrease in physical activity due to changes in lifestyle, diet and the transition from manual work to less physical occupations.

□ Stress – The impact of stress both physical and psychological along with lifestyle changes has a strong influence on the increasing incidence of type 2 diabetes among people with a strong genetic background.

In a 1998 study, the Diabcare Asia Study, it was observed that of all diabetic patients treated from specialized centers, more than 50% of patients had poor diabetes control according to the American Diabetes Association criteria. The study showed that 4% of patients were on diet therapy alone, 53.9% were on oral antidiabetic drugs, 22% of patients were on insulin, and another 19% were on both insulin and oral medications. This study confirmed that diabetes care in India leaves much to be desired.

CLASSIFICATION:

Although all forms of DM are characterized by hyperglycemia, the pathogenic mechanisms by which hyperglycemia occurs vary widely. Some forms of DM are characterized by absolute insulin deficiency or a genetic defect leading to defective insulin secretion, while other forms share insulin resistance as an underlying etiology. Recent changes in classification reflect efforts to classify DM based on the pathogenic process that leads to hyperglycemia, as opposed to criteria such as age of onset or type of therapy.

The two broad categories of DM are referred to as type 1 and type 2 diabetes. Type 1 DM, also known as insulin-dependent diabetes (IDDM), results from autoimmune destruction of beta cells that usually results in insulin deficiency.

Type 2 DM, also known as non-insulin-dependent diabetes (NIDDM), is a heterogeneous group of disorders usually characterized by varying degrees of insulin resistance, impaired insulin secretion, and increased glucose production. Distinct genetic and metabolic defects in insulin action and/or secretion lead to the common phenotype of hyperglycemia in type 2 DM. The identification of distinct pathogenic processes in type 2 DM has important potential therapeutic implications as pharmacological agents are available that target specific metabolic disturbances.

NIDDM or type 2 is the most common, accounting for about 80% of all diabetics. IDDM or Type 1 makes up most of the remaining 20%. Maturity-onset diabetes of the young (MODY) and secondary diabetes are relatively rare, accounting for less than 1% of cases. However, it is important to identify patients with secondary diabetes so that treatment can be directed at the underlying cause.

Diabetes in association with other generic syndromes is also very rare. In these cases, diabetes is another burden that these people have to bear.

Two features of the current DM classification differ from previous classifications. First, the terms insulin-dependent diabetes mellitus (IDDM) and noninsulin-dependent diabetes mellitus (NIDDM) are outdated. These previous designations reflected the observation that most individuals with type 1 DM (formerly IDDM) have an absolute need for insulin therapy, whereas many individuals with type 2 DM (formerly NIDDM) do not require insulin therapy to prevent ketoacidosis. However, because many individuals with type 2 DM eventually require insulin therapy for glycemic control, the use of the latter term has caused considerable confusion.

The second difference is that in the new classification system age is no longer used as a criterion. Although type 1 DM most often develops before the age of 30, the autoimmune destructive process of beta cells can develop at any age. In fact, it is estimated that 5 to 10% of individuals who develop DM after age 30 have type 1A DM. Similarly, although type 2 DM more typically develops with increasing age, it also occurs in children, especially obese adolescents.

Insulin-dependent diabetes mellitus (IDDM):

Insulin-dependent diabetes mellitus (IDDM) usually has its onset before the age of 30, most often in childhood or adolescence, although it can appear at any age. Clinical onset may be sudden with increased thirst and appetite, excessive urination and weight loss. Occasionally, it may be diagnosed when the patient first presents in ketoacidosis or coma, particularly during intercurrent illness or surgery. Diabetic ketoacidosis is a serious complication that is characteristic of IDDM and is often fatal if left untreated. Patients are usually not obese and may even be emaciated and underweight. These are patients in whom there is an absolute lack of insulin, because its production is extremely low or absent, and whose life can only be maintained by supplementing insulin from external sources.

The pathogenesis of IDDM rests on the triad of (i) genetic susceptibility, (ii) altered immune response, and (iii) environmental trigger(s). Studies of homozygous (identical) twins clearly show the relative importance of genes and environment: the concordance rate for IDDM in such twins is approximately 50%, giving almost equal weight to both. Furthermore, only 10–15% of dizygotic twins, 5–10% of haploidentical siblings, and 3–5% of first-degree relatives with IDDM will develop the disease during life. Finally, it should be kept in mind that despite the apparently strong genetic basis of susceptibility to IDDM, about 80–90% of newly diagnosed subjects have no family history of IDDM.

Genetic susceptibility to IDDM is determined by genes forming the HLA complex, located on the short arm of chromosome 6, although a possible influence of genetic markers outside the HLA region has also been proposed.

It is good to remember that approximately 10% of IDDM patients also have other organ-specific autoimmune disorders such as Grave's disease, Hashimoto's thyroiditis, Addison's disease, and pernicious anemia, suggesting an underlying immunoregulatory disorder.

Epidemiological data provide evidence for the association of environmental triggers with IDDM. Several etiological factors have been proposed. As an environmental trigger, virus-induced diabetes is well recognized in animal models, although the exact role of viruses in the pathogenesis and pathophysiology of IDDM remains uncertain. The most common viruses that can cause diabetes in animals include coxsackievirus, encephalomyocarditis virus (EMC), and rubella virus. Mumps, measles, rubella and Epstein-Barr virus (infectious mononucleosis) have been implicated in humans in addition to coxsackie B virus. Although most of these viruses directly attack pancreatic beta cells, they rarely cause complete and total destruction of these cells.

The likely sequence of events initiated by viral infections includes: (i) partial or mild damage to beta-cells, (ii) immune response against one or more beta-cell antigens, (iii) triggering of an autoimmune process in patients with HLA-associated susceptibility (iv) progressive immune-mediated inflammatory loss of beta cells and (v) clinical onset of IDDM after varying latency in some of these subjects. Such a sequential process is illustrated by the fact that congenital rubella infection can cause IDDM in childhood or adolescence in about 20% of those so infected in utero, and that the resulting diabetic state is immunologically similar to IDDM that occurs in persons without such disease. clinical history.

Although IDDM attributable to coxsackie B4 and cytomegalovirus (CMV) infection in humans has been described, evidence implicating viral infection in immune-mediated beta-cell destruction in large numbers of IDDM subjects remains insufficient.

Recognized environmental factors other than viruses include chemicals and drugs, as well as changes in dietary components. Specific chemicals include alloxan and Vacor (a rodenticide), while streptozotocin and pentamidine are used as drugs. Like viruses, these chemicals and drugs can act directly as beta cytotoxics and can also trigger the immune process. Exposure to nitrosamines (used in meat preservation) in women at the time of conception may increase the risk of IDDM in offspring, especially boys. Diets deficient in certain essential fatty acids or with an increased ratio of n-6/n-3 polyunsaturated fatty acids can alter the immune response, as can changes in dietary protein, and thus play a role in pathogenesis.

New on the list is the ingestion of cow's milk in early childhood and the subsequent development of IDDM. Antibodies are directed against bovine serum albumin (BSA), binding mostly to the ABBOS region of BSA (amino acid sequence 152-168). These antibodies bind to a beta-cell surface protein, p69, which has recently been cloned. The human gene encoding p69 has been mapped to 7p22. Interestingly, this protein was found to be identical to the IDDM-associated islet cell antigen (ICA 69).

The pathophysiology of IDDM is closely related to its natural history. It is now well known that there may be a period of months or even years before any of the metabolic disorders caused by insulin deficiency appear, and that the only indication of the onset of the disease process in this presymptomatic phase may be the presence of circulating islet cell antibodies (ICA) or other evidence the presence of similar immune markers such as antibodies against glutamic acid decarboxylase (GAD). Such a process can then lead to the gradual destruction of beta-cells, ultimately resulting in a reduction of beta-cell mass to about 10-15 percent of normal when clinical signs including hyperglycemia, glycosuria, and weight loss herald the onset of overt IDDM.

The 64K autoantigen was identified as GAD. This enzyme facilitates the biosynthesis of the inhibitory neurotransmitter GABA (gamma-aminobutyric acid). Using the presence of GAD antibodies as a marker for IDDM, it has been suggested that these may have higher sensitivity, specificity, and predictive value for human IDDM than other established markers. The term latent autoimmune diabetes mellitus in adults (LADA) is now increasingly used in relation to a syndrome where clinically diagnosed NIDDM in adults is associated with a significant titer of GAD antibodies, indicating a pathogenetic process similar to that of IDDM, but with a slower rate of progression.

Until the pathogenetic process is finally elucidated, five known facts include (i) the presence of one or more immune markers for a considerable time before clinical onset; (ii) destruction of at least 80 to 90 percent of functional beta cells by the time hyperglycemia first occurs; (iii) demonstrating a lower than normal insulin/C-peptide response to standard glucose challenge for at least some time prior to the manifestation of hyperglycemia and at a time when glucose tolerance is otherwise normal; (iv) evidence of insulinitis as an immune-mediated inflammatory process underlying beta cell destruction and (v) the occurrence of a progressively destructive autoimmune process only when there is a genetic predisposition to IDDM.

Two main questions are the subject of ongoing debate. First, the destructive process affecting beta cells is always relentless and progressive, eventually leading to the clinical onset of IDDM; and second, is the sensitivity associated with class II antigens on the HLA complex uniform in all individuals affected by IDDM?

Although there are studies that suggest that beta cell destruction is progressive and that the decline in their ability to release insulin occurs in a linear fashion over time, there is considerable heterogeneity, suggesting that even in HLA-identical siblings of IDDM patients who show evidence of the presence of immune markers, progression to clinical onset of IDDM does not always occur and, in fact, may not occur at all in most. Thus, it appears that the destructive process affecting beta cells, once initiated, may not always be progressive and that even without interventional therapy there would be a

significant chance of remission of this pathological process. However, it should be clearly understood that remission in this context refers only to the period before the clinical onset of IDDM and should not be confused with the temporary remission that may occur in some patients with IDDM after clinical onset.

The initiating mechanism(s) may be mediated through aberrant expression of specific class II molecules on the surface of beta cells or/and increased expression of class I antigen; such expression can, in the presence of the appropriate genetic background, initiate immune responses leading to an inflammatory response in the islets. This pathological process, called insulinitis, is characterized by the presence of all cell types of the immune system including T and B lymphocytes, macrophages and NK cells, with cytotoxic and helper T lymphocytes acting as effector cells. As part of the immune response, several polypeptides, called cytokines, are released. Some of these cytokines, notably interleukin 1 (IL-1), tumor necrosis factor- α (TNF- α), and interferon γ , produced by macrophages, monocytes, and islet-invading dendritic cells, exhibit toxic effects on beta cells. The final pathway is mediated by the generation of toxic free radicals, both oxygen and nitric oxide, leading to beta-cell destruction. Whether humoral immune mechanisms also play an equally important, if not major, role in the cause of IDDM needs to be considered. Circulating antibodies against beta-cell antigens, including insulin and proinsulin, exhibit binding properties, and such surface-bound antibodies can either initiate cytotoxic mechanisms or directly affect beta-cell function.

Non-insulin-dependent diabetes mellitus (NIDDM):

Non-insulin-dependent diabetes mellitus (NIDDM) can be defined as a metabolic disorder that is characterized by hyperglycemia (high blood sugar) in the context of insulin resistance and relative insulin deficiency. This is in contrast to type 1 diabetes mellitus, in which there is an absolute lack of insulin due to the breakdown of islet cells in the pancreas.

Thus, the main clinical difference between IDDM and NIDDM is the lack of tendency toward ketosis in NIDDM. Such a substantial difference is due to the fact that individuals with IDDM have no or very low residual beta-cell function, as shown by minimal or zero circulating insulin and C-peptide in the fasting state and also in response to glucose challenge. In contrast, circulating insulin and C-peptide levels in NIDDM can be variable, ranging from hyperinsulinemia to normoinsulinemia in most subjects. Even with reduced insulin output, there is generally enough available to prevent ketosis. However, it should be emphasized that patients with NIDDM may develop ketosis under stressful situations, such as fulminant infection, burns, trauma, or surgery, when insulin administration is mandatory.

NIDDM commonly begins in the 45-65 age group, although it can affect relatively younger populations in India and other developing countries. It is further divided into obese and non-obese types, mainly because approximately 80-90% of NIDDM subjects seen in the Western Hemisphere are obese. Clearly, this is not the case in many developing countries including India, where obese NIDDM accounts for less than 60% of such patients.

Although NIDDM is much more common than IDDM, much less is known about its pathogenesis. The main risk factors for NIDDM are genetic susceptibility, obesity, physical inactivity and Western lifestyle including changes in dietary habits involving the quantity and quality of food intake, as well as life stress.

In identical twin studies, the concordance rate for NIDDM is around 90%, as opposed to 50% or less for IDDM. The concordance rate is greater than 40% for siblings and non-identical twins. Thus, genetic factors are much more important in NIDDM, as also shown by the strong familial aggregation in NIDDM. However, genetic studies do not provide any firm data on whether the disease is caused by one or several genes, nor do they provide precise information about the mode of inheritance. An obvious conclusion is the possible clinical and genetic heterogeneity of NIDDM with several well-characterized genetic defects.

ETIOLOGY AND RISK FACTORS OF NIDDM:

□ Predisposing factors and/or secondary circumstances:

o Age: The disease generally develops after the age of 40.

o Gender: Both sexes are affected almost equally.

o Heredity: The disease runs in families. Potential or prediabetics acquire the trait only from a hereditary source. This is also supported by the frequency of diabetes in the affected person's family history.

o Stress and strain: Physical and/or mental stress and strain will accelerate the disease from a latent state.

o Obesity: Obesity has been observed to be associated with diabetes mellitus, but the conventional system of medicine is in the dark to ascertain their causal relationship.

o Infection: Infection played an important role in the development of diabetes. A susceptible constitution induces certain infections such as Coxsackie B4 Virus, cytomegalovirus, adenovirus, which in turn can cause islet cell dysfunction leading to the development of diabetes.

□ Genetic factors:

Evidence for a strong genetic component in the development of NIDDM comes from a number of studies of different designs.

HOMOEOPATHIC APPROACH TO THE TREATMENT OF DIABETES MELLITUS:

According to homeopathic guidelines, the patient is to be treated, not his organs, parts or systems or tissues. So even with Diabetes mellitus, our target of treatment is the patient himself, not the pancreas or the B-cells; therefore, constitutional treatment is the only way to radically cure type 2 diabetes mellitus along with proper diet and regular exercise.

Our goal is not only to lower the blood sugar level, but to achieve improvement in the patient's subjective and objective spheres. In constitutional, antimiasmatic treatment, the totality of symptoms is taken into account, including individualizing personality traits (physical and psychological) of the patient. Undoubtedly, striking, special, rare, special symptoms of the pathogenesis of the disease are taken into account, which give us indications (§ 3) for selection.

For therapeutic purposes, i.e. for the selection of a specific drug for an individual patient suffering from Diabetes mellitus, based on deductive reasoning for the individualization of a specific case of the disease; It involves the study from general to specific, i.e. we are to study on the basis of constant differentiation from the group of drugs that cover and come as similar drugs for a specific disease syndrome consisting of some signs and symptoms persisting throughout the pathology and area. diseases in general. However, in order to select a particular drug from a group of similar drugs, we must work with symptoms and difficulties that are unique and characteristic, i.e. symptoms that belong to the most unusual, peculiar, characteristic and strange symptoms (§153–Organon of Medicine).

From the history of patients suffering from Diabetes mellitus, the only indication will be the summary of symptoms of each case, which should cover the constitution, past and family history of the patients, as well as their miasmatic background.

HOMOEOPATHIC THERAPEUTICS:

There are several remedies which have been symptomatically indicated in Diabetes Mellitus, but if the general symptom agrees, any remedy from the Materia Medica may be used.

Most often, however, only a smaller group is employed, e.g.

Acetic acid: Large quantities of pale urine, unquenchable thirst and great weakness.

Abroma Augusta: Frequent and profuse urination, dry mouth and great thirst, urination leads to exhaustion, Fishy smell of urine, Diabetes mellitus and insipidus.

Argentum Metallicum: Polyuria, frequent urination, copious urine at night, cloudy and sweet smell, restless sleep, frightening dreams, edematous swelling of the legs, flatulence.

Arsenicum Album: Urine thin, burning protein, ascites, prevailing weakness, restlessness, burning thirst, drinks often, but sometimes little.

Codeine: Sugar in urine, increased urine, great thirst, said to control diseases.

Cephalandra Indica: Diabetes with profuse urination; weakness and exhaustion after urination; sugar in the urine.

Gymnesa Sylvestre: It is almost specific for DM called as “Sugar Killer” reduces sugar in urine; Profuse urination loaded with sugar, extreme weakness after urinating a large amount of urine. polyuria; day and night.

Hellebore: Frequent urge to urinate, but a small quantity is passed, profuse urination, urine pale and watery, liquid swelling.

Helonias-Chamailirium: DM and insipidus, urine rich and clear, phosphatic and albuminous, great thirst, restlessness, deep melancholy, irritable, dull pain in lumbar region.

Insulin: It is believed to be specific and useful in case of carbuncles due to DM.

Lacticum Acidum: Frequent excretion of large quantities of sugar in the urine, great thirst, rheumatic pains in the joints.

Natrum Phosphoricum & Natrum Sulphuricum: They are of great value in diabetes. Excessive urination, bilious urine, lithic deposits in the urine, sedentary habits, especially when ulcers alternate.

Phosphoricum Acidum: Frequent and copious watery urination, milky urine, great weakness.

Phosphorus: DM in phthisis with impotence, the urine contains a large amount of salt in the morning and an excess of sugar in the evening.

Plumbum Metallicum: Urine frequent, thin, proteinaceous, low specific gravity.

Rhus Aromatic: Large amount of urine, urine pale, whitish, specific gravity low.

Syzygium Jambolanum: It has a specific effect on the reduction and disappearance of sugar in the urine, great thirst and weakness, urine in very large quantities, high specific gravity. Ten drops to be taken two or three times a day.

Uranium Nitricum: Excessive urination, weakness, acid in urine, incontinence, incontinence, excessive thirst, diarrhea in dyspeptics.

Terebinthinum: Profuse, cloudy, smoky and proteinaceous urine, sediments like coffee grounds, hematuria.

Other valuable medicines are: - Arsenicum iodatum; Aurum metallicum; Boric acid; Bryonia alba; Chamomilla umbellata; Chionanthus virginica; Coca; Crotalus horridus; Curare; Iris versicolor; Creosotum; Morphine; Nux vomica; pancreatin; Silicea terra; Strychninum arsenicosum, etc.

CONCLUSION

DM is one of the most common medical problems affecting individuals in modern times. The increase in incidence may be due to a genetic factor, a modernized lifestyle, obesity, sedentary work and lack of exercise. In addition to these factors, susceptibility plays an important role in causing the disease state if the individual is not predisposed to developing diabetes mellitus II. type, the chances of being affected by this disease state decrease.

Diabetes is a disease that affects a large number of the population and changes the lifestyle of the sufferer. It affects every system of the human body and if left unchecked can lead to irreversible organic changes and serious complications leading to premature death.

There is still no satisfactory treatment in the modern system of medicine. They only offer palliative treatment. For this reason, many patients come to homeopathic doctors with high expectations of a complete cure. Homeopathy as an independent method of therapy has a wide field of application in the treatment of many so-called incurable diseases, as they are referred to by the modern system of medicine. This study was conducted to observe the miasmatic development and effectiveness of homeopathic treatment in patients with diabetes mellitus II. type and understanding of the clinical approach in her management.

An experimental study was conducted with an experimental and a control group of 30 cases suffering from diabetes mellitus II. type. who are already on OHA randomization will be done in two groups, one controlled group and the other experimental group. The drug group will be obtained after repertorization/without repertorization. The drug will be prescribed to the experimental group and the control group will be prescribed a placebo. Monitoring of both groups will take place at an interval of one month.

In each case, from the experimental group, they will have indicated homeopathic medicines, selected on the basis of mental generals and the overall symptom after case analysis. Even the repertorization was also done through computer software. Results were recorded and analyzed using standard statistical methods.

The effectiveness of homeopathic treatment in patients suffering from diabetes mellitus II. type was evaluated according to predetermined parameters. The results were favorable, encouraging and found to be useful for future reference in the experimental group.

A miasmatic splitting was performed, taking all the symptoms in each case and thus determining the miasmatic basis of each. In most cases, Psora was found to be the underlying dominant miasma.

The study concluded that homeopathic treatment with appropriate constitutional antimiasmatic medicine along with proper dietary guidance and physical exercise (if necessary) has a major role in managing patients with diabetes mellitus II.

Undoubtedly, we obtained a favorable and encouraging result, but considering the limitations, it requires further extensive study.

Interpretation:

This study helps us to know the various manifestations of type 2 diabetes mellitus with its associated symptoms, its clinical presentation, complications and the management of such types of cases with the help of homeopathic medicines is beneficial.

SUMMARY:

Stressful life events that played a major factor in patients suffering from Diabetes Mellitus. Stressful events are significantly associated with the development of diabetes mellitus and its progression. It was found that the comparison of FBS before and after treatment showed a statistically significant result. It can also be argued that homeopathy is safe, simple, less expensive and more effective in the treatment of diabetes mellitus.

Homeopathy as a system of treatment has its own philosophy and its therapeutics are based on certain basic principles. From these basic principles, the theory of chronic diseases plays a vital role in the treatment of chronic cases.

To conclude in the words of Hahnemann: "He who has had as much opportunity of observation as I have, . . . he who is driven by the desire for the welfare of his fellow men to think and act for himself, he who, like me, feels hatred for the prejudices and favors of the old or new, or, generally speaking, for any kind of recognition or great name, and one who is eager, like myself, to act and think independently. . . . he will see excellent results in his field, which is the greatest reward an honest physician can expect".

Limited reliability can only be guaranteed for such a chronic disease study with 30 cases over 1 year. A long-term follow-up study will be more reliable. Increasing the sample size can be considered in further studies to provide more statistical evidence. Comparative studies involving other drug systems can also be performed with better results.

The usefulness of the complete repertoire in this study helped me choose the right rubric to find remedies.

REFERENCES/BIBLIOGRAPHY:

1. Harrison TR(2012). *Harrison's Principles of Internal Medicine*. 18th edition. published in New Delhi: Churchill livingstone. Elsevier; 2968 pp.
2. YashPal Munjal (2012) *A.P.I text book of medicine*(2012) published by the association of physicians of India, Mumbai 9th edition 319pp
3. Diabetes Mellitus: homeopathic perspective by Dr. AjitKulkarni, www.hpathy.com
4. Homeopathy Effective in Diabetes treatment: Dr. Lynn, Hardy,NDwww.byregion.net
5. Diabetes Mellitus: defining scope & clinical approach for homeopathic management.: N.L Tiwari&PrashantTamboli
6. Diabetes Mellitus: Defining approach (disease monograph-4) KanjakshaGhoshwww.ijrh.comHarper R.D, Pat, Laliberte R, Petit A. William J. All New Natural Approach to Beating Diabetes. A Practical Guide to Controlling Diabetes. Reprint ed. New Delhi: R D Publications; 2006. p. 98.
7. Lilienthal S. Homoeopathic Therapeutics. 19th ed. New Delhi: B Jain publisher (P) Ltd; 2010. p. 288-94.
8. Boericke W. Pocket manual of Homoeopathic Materia Medica. 9th ed. New Delhi: Mayur Jain Indian Books & Periodicals Publishers; 2006. p.1000.
9. Farrington EA. Clinical Materia Medica. 4th ed revised. New Delhi: B Jain publisher (P) Ltd; 2008. p. 518,568.
10. Iyer TS. Beginners guide to Homoeopathy. Reprint ed. New Delhi: B Jain publisher (P) Ltd; 1999. p. 187.
11. Hughes R. The principle and practice of homoeopathy. Reprint ed. New Delhi; B Jain publisher (P) Ltd; 2004. p. 628.
12. J.G.C.Ledignham,oxford text book of medicine, 3rd Edition, Edited by d. a. warell.
13. Guyton Arthur C., text book of medical physiology, 9th Edition, Harcourt brace & company Asia (p) Ltd., (1998).
14. Sarkar B. K. organon of medicine, 9th Revised Edition, Birla Publications, New Delhi, (2003- 04)
15. Close Stuart, the genius of homoeopathy, Reprint Edition, B. Jain Publishers (Pvt.) Ltd., New Delhi (2003).
16. Allen J. Henry, the chronic miasmas, Volume-I & II, B. Jain publishers (p) Ltd., New Delhi (2004).
17. Banerjee P. N., chronic disease its cause and cure, B. Jain Publishers (p) Ltd., New Delhi
18. Dr. Kent J. T., lectures on homoeopathic philosophy, Indian Books & Periodicals Syndicate, Reprint Edition, (September 2006)
19. Roberts H. A., the principles and art of cure by homoeopathy, B. Jain publishers (p) Ltd., New Delhi (2004).
20. Hughes Richard, the principles and practice of homoeopathy, B. Jain Publishers.
21. Boericke William, homoeopathic materiamedica and repertory, B. Jain Publishers (p) Ltd., New Delhi.
22. Ramakrishnan A. U. & Coulter Catherine R., international journal of diabetis, 2006 Nov; 95, Suppl.1:S215-33., Publisher: Quality Medical Publishing Inc., St Louis, Missouri, USA, 2001. ISBN: 1-57626-155-7
23. American diabetes association, Clinical practice recommendations 2002. Diabetes Care 27:51, 2004
24. Clement s et al, Management of diabetes and hyperglycemia in hospitals. Diabetes Care 27:553, 2004
25. Knowlerw et al, diabetes prevention program research group: Reduction in the incidence of type 2 diabetes with lifestyle Intervention or metformin. N Engl J Med 346:393, 2002
26. Saltielar, kahncr: Insulin signalling and the regulation of glucose and lipid metabolism. Nature 414:799, 2001