

“A STUDY ON THE EFFICACY OF HOMOEOPATHIC MEDICINES IN THE MANAGEMENT OF DIABETES MELLITUS SATISFYING THE CRITERIA OF PSYCHOLOGICAL CONDITION AFFECTING MEDICAL CONDITION OF DSM-IV-TR”

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CHAPTER-1 INTRODUCTION

Diabetes was known even in ancient times. The name of this disease, which is characterized by excessive flow of urine and insatiable thirst, was coined by the Graeco-Roman physician Aretaeus of Cappadocia (approx.80–130 A.D.) and is derived from the Greek word *diabainein* ('to flow through'). The adjective *mellitus*, which comes from Latin and means 'honey-sweet', was added by the German physician Johann Peter Frank (1745–1821) in order to distinguish diabetes mellitus, or 'sugar diabetes', from diabetes insipidus. Johann Peter Frank was also who in 1790, by introducing a yeast fermentation test for the quantitative determination of urinary glucose, relieved the physicians of his time of the need to taste their patients' urine. According to ayurveda, it is called as madhumeha, is a metabolic kapha type of disorder in which diminished functioning of agni leads to a tendency toward high blood sugar.

1.1 DIABETES MELLITUS

Diabetes mellitus (DM) comprises a group of common metabolic disorders that share the phenotype of hyperglycemia^{2,a}. The term diabetes mellitus describes a metabolic disorder of multiple aetiology characterized by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both. It occurs when pancreas does not produce enough insulin or alternatively, when the body cannot effectively use the insulin it produces. Insulin is a hormone that regulates blood sugar. Hyperglycemia, or raised blood sugar, is a common effect of uncontrolled diabetes and over time leads to serious damage to many of the body's systems, especially the nerves and blood vessels.

1.2 EPIDEMIOLOGY

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 from 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 years; 55% of diabetes deaths are in women⁸. Indeed, by 2010 it has been estimated that the diabetic population will increase to 221 million from 110 million in 1994.^{3,4} The majority of the new cases will be those with type 2 diabetes and most of these will be in China, the Indian subcontinent and Africa. It is estimated that from 65 million cases of type 2 diabetes in Asia and Oceania in 1995, the number will double to 135 million by 2010.

In India it is estimated that presently 19.4 million individuals are affected by this deadly disease, which is likely to go up to 57.2 million by the year 2025⁹. By the year 2025, India is predicted to have the most number of people with diabetes mellitus in the world¹. In Kerala about 8% of adult population is diabetic; this ranges from 3% in rural areas and 20% in cities. Only exception is coastal fisher folk among whom prevalence is as low as 3%. By most conservative estimate there are about 1.5 million diabetics' subjects in Kerala^{9b}.

1.3 PSYCHOLOGICAL SYMPTOMS AFFECTING MEDICAL CONDITION

This class belongs under Psychological Factors Affecting General Medical Condition. These symptoms do not meet full criteria for an Axis I³ disorder significantly affect the course or treatment of a general medical condition (the accompanying general medical condition is coded on Axis III⁴)

The Essential feature of Psychological factor affecting Medical condition is the presence of one or more specific psychological or behavioral factors that adversely affect a general medical condition. It is classified under Other Conditions that may be a Focus of Clinical Attention of DSM-IV-R. Psychological Factors play a potential role in the presentation or treatment of almost every medical condition. As per classification is only reserved for situations in which psychological factors have a clinical significant effect on the course or out come of the general medical condition or place the individual at a significantly higher risk for adverse outcome, although it may often not be possible to demonstrate direct causality or the mechanisms underlying the relationship.

1.4 ROLE IN DIABETES MELLITUS

Studies have revealed that when stress occurs it causes the hormone level to shoot up in response to fight or flight response of body. In people with diabetes, stress can alter blood glucose levels. Scientists have studied the effects of stress on glucose levels in animals and people. Diabetic mice under physical or mental stress have elevated glucose levels. The effects in people with type 1 diabetes are more mixed. While most people's glucose levels go up with mental stress, others' glucose levels can go down. In people with type 2 diabetes, mental stress often raises blood glucose levels. Physical stress, such as illness or injury, causes higher blood glucose levels in people with either type of diabetes.⁵. There are findings to demonstrate a significant and consistent association of diabetes complication and depressive symptoms.⁶ Findings also suggest chronic burnout as risk factor for the onset of type 2 diabetes in apparently healthy individuals.⁷. Its evident from epidemiology that Diabetes Mellitus is cause for large scale morbidity and mortality world wide. Its Level is accelerating in coming decades. In India the Projection being 57.2 Million by 2025. Stress and other psychosocial factors has significant part in control on blood glucose level.

CHAPTER-2 AIM AND OBJECTIVE

1. To study the efficacy of Homoeopathic medicines in management of Diabetes Mellitus.
2. Study relation between Diabetes and Psychological factors as specified in Diagnostic and Statistical Manual-IV Revised Text edition¹.
3. To construct a Repertory on Diabetes and its complications.

CHAPTER-3 REVIEW OF LITERATURE 3.1 DIABETES

3.1.1 Definition

The term diabetes mellitus describes a metabolic disorder of multiple aetiology characterized by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both. The effects of diabetes mellitus include long-term damage, dysfunction and failure of various organs. Diabetes mellitus may present with characteristic symptoms such as thirst, polyuria, blurring of vision, and weight loss. In its most severe forms, ketoacidosis or a non-ketotic hyperosmolar state may develop and lead to stupor, coma and, in absence of effective treatment, death.

Often symptoms are not severe, or may be absent, and consequently hyperglycaemia sufficient to cause pathological and functional changes may be present for a long time before the diagnosis is made. The long-term effects of diabetes mellitus include progressive development of the specific complications of retinopathy with potential blindness, nephropathy that may lead to renal failure, and/or neuropathy with risk of foot ulcers, amputation, Charcot joints, and features of autonomic dysfunction, including sexual dysfunction. People with diabetes are at increased risk of cardiovascular, peripheral vascular and cerebrovascular disease.

Several pathogenetic processes are involved in the development of diabetes. These include processes which destroy the beta cells of the pancreas with consequent insulin deficiency, and others that result in resistance to insulin action. The abnormalities of carbohydrate, fat and protein metabolism are due to deficient action of insulin on target tissues resulting from insensitivity or lack of insulin¹¹.

3.1.2. Diagnosis and diagnostic criteria

3.1.2.1 Diagnosis

The requirements for diagnostic confirmation for a person presenting with severe symptoms and gross hyperglycaemia differ from those for the asymptomatic person with blood glucose values found to be just above the diagnostic cut-off value. Severe hyperglycaemia detected under conditions of acute infective, traumatic, circulatory or other stress may be transitory and should not in itself be regarded as diagnostic of diabetes¹². The diagnosis of diabetes in an asymptomatic subject is never be made on the basis of a single abnormal blood glucose value. For the asymptomatic person, at least one additional plasma/blood glucose test result with a value in the diabetic range is essential, either fasting, from a random (casual) sample, or from the oral glucose tolerance test (OGTT). Glycated haemoglobin, reflecting average glycaemia over a period of weeks, was thought to provide such a test. Although in certain cases it gives equal or almost equal sensitivity and specificity to glucose measurement¹³; it is not available in many parts of the world and is not well enough standardized for its use to be recommended.

3.1.2.2 Diabetes in children

Diabetes in children usually presents with severe symptoms, very high blood glucose levels, marked glycosuria, and ketonuria. An OGTT is neither necessary nor appropriate for diagnosis in such circumstances. A small

proportion of children and adolescents, however, present with less severe symptoms and may require fasting blood glucose measurement and/or an OGTT for diagnosis¹⁴.

3.1.3 Diagnostic criteria

The clinical diagnosis of diabetes is often prompted by symptoms such as increased thirst and urine volume, recurrent infections, unexplained weight loss and, in severe cases, drowsiness and coma; high levels of glycosuria are usually present. For clinical purposes, an OGTT to establish diagnostic status need only be considered if casual blood glucose values lie in the uncertain range (i.e. between the levels that establish or exclude diabetes) and fasting blood glucose levels are below those which establish the diagnosis of diabetes.¹⁵ If an OGTT is performed, it is sufficient to measure the blood glucose values while fasting and at 2 hours after a 75 g oral glucose load. For children the oral glucose load is related to body weight: 1.75 g per kg.

The diagnostic criteria for diabetes mellitus in children are the same as for adults. The major change recommended in the diagnostic criteria for diabetes mellitus is the lowering of the diagnostic value of the fasting plasma glucose concentration to 7.0 mmol l⁻¹ (126 mg dl⁻¹) and above, from the former level of 7.8 mmol l⁻¹ (140 mg dl⁻¹) and above.

For whole blood the proposed new level is 6.1 mmol l⁻¹ (110 mg dl⁻¹) and above, from the former 6.7 mmol l⁻¹ (120 mg dl⁻¹).

The new fasting criterion is chosen to represent a value which is at the upper end of the range that corresponds in diagnostic significance in many persons to that of the 2-h post-load concentration, which is not changed.

3.2 CLINICAL STAGING OF DIABETES MELLITUS AND OTHER CATEGORIES OF GLUCOSE TOLERANCE

Table 3.1 Clinical Staging of Diabetes Mellitus and Other Categories of Glucose Tolerance

Types	Stages	Hyperglycaemia			
	Normoglycaemia Normal glucose tolerance	Impaired glucose regulation IGT and/or IFG	Diabetes Mellitus		
			Not insulin requiring	Insulin requiring for control	Insulin requiring for survival
Type 1 • Autoimmune • Idiopathic	←—————→				→
Type 2* • Predominantly insulin resistance • Predominantly insulin secretory defects	←—————→			→	→
Other specific types*	←—————→			→	→
Gestational diabetes*	←—————→			→	→

* In rare instances patients in these categories (e.g. Vacor Toxicity, Type 1 presenting in pregnancy, etc.) may require insulin for survival.

3.2.1 Diabetes

Diabetes mellitus, regardless of underlying cause, is sub-divided into: *Insulin requiring for survival* (corresponding to the former clinical class of "Insulin Dependent Diabetes Mellitus – IDDM"), e.g. C-peptide deficient; *Insulin requiring for control*, i.e. metabolic control, rather than for survival, e.g. some endogenous insulin secretion but insufficient to achieve normoglycaemia without added exogenous insulin; and *Not insulin requiring*, i.e. those who may be controlled satisfactorily by non-pharmacological methods or drugs other than insulin. Together, the latter two sub-divisions constitute the former class of NIDDM.

3.2.2 Impaired glucose regulation

Impaired glucose tolerance and impaired fasting glucose refer to a metabolic state between normal glucose homeostasis and diabetes. It has been suggested that these disorders should be considered as a stage in the natural history of disordered carbohydrate metabolism, indicating risk of future diabetes and/or cardiovascular risk, rather than discrete clinical entities.

3.2.2.1 Impaired glucose tolerance

The importance of this condition is debatable. Controversy has arisen partly because of the high variability in the response to the oral glucose tolerance test. Up to 50 per cent of persons initially classified as having impaired glucose tolerance are reclassified on repeat testing as having normal glucose tolerance. Impaired glucose tolerance is a strong risk factor for the development of type 2 diabetes. It also identifies a group of individuals at higher risk of developing coronary heart disease (CHD).

3.2.2.2 Impaired fasting glucose

More recently, impaired fasting glucose (fasting plasma glucose 6.1 to 6.9mmol/l) has been introduced as a new category of impaired glucose regulation. The exact significance of impaired fasting glucose is yet to be determined, but it would appear to be a stronger predictor of the risk of progression to diabetes than impaired glucose tolerance. A stage of IFG is also recognized because such subjects, like those with IGT, have increased risks of progressing to diabetes and macrovascular disease, although prospective data are sparse and early data suggest a lower risk of progression than IGT¹⁸, although a similar CVD risk factor profile has been shown in IFG and IGT subjects¹⁹. IFG refers to fasting glucose concentrations which are lower than those required to diagnose diabetes mellitus but higher than the "normal". Individuals who meet criteria for IGT or IFG may be euglycaemic in their daily lives as shown by normal or near-normal glycated haemoglobin levels. IGT and IFG are not clinical entities in their own right, but rather risk categories for future diabetes and/or cardiovascular disease^(20, 21).

3.3 NORMOGLYCAEMIA

A fasting venous plasma glucose concentration of less than 6.1 mmol l⁻¹ (110 mg dL⁻¹) has been chosen as "normal." Although this choice is arbitrary, such values are observed in people with proven normal glucose tolerance, although some may have IGT if an OGTT is performed. Values above this are associated with a progressively greater risk of developing micro- and macrovascular complications^(21,23).

The pathological or aetiological processes which often lead to diabetes mellitus begin, and may be recognizable, in some subjects who have normal glucose tolerance. Recognition of the pathological process at an early stage may be useful if progression to more advanced stages can be prevented. Conversely, effective treatments, or occasionally the natural history of some forms of diabetes mellitus, may result in reversion of hyperglycaemia to a state of normoglycaemia. The proposed classification includes a stage of normoglycaemia in which persons who have evidence of the pathological processes which may lead to diabetes mellitus, or in whom a reversal of the hyperglycaemia has occurred, are classified.

3.4 AETIOLOGY

3.4.1 Type 1 diabetes

Type 1 diabetes is considered to be an autoimmune process in which T lymphocytes infiltrate the islets of the pancreas and destroy the insulin-producing beta cell population. Glutamic acid decarboxylase is a strong candidate autoantigen in triggering beta cell specific autoimmunity. The majority of patients with type 1 diabetes have anti-glutamic acid decarboxylase antibodies in their sera at diagnosis. However, both genetic and environmental factors have also been implicated as important factors in the initiation of the autoimmune process. Up to 20 chromosomal regions have been linked with the development of type 1 diabetes. The most important gene loci in defining the risk of type 1 diabetes are located within the human leucocyte antigen (HLA) gene region. HLA-DQ molecules are of primary importance, but HLA-DR may modify the risk conferred by HLA-DQ. The largest contribution from a single locus comes from several genes located in the major histocompatibility complex (MHC) on chromosome 6p21.3 (IDDM 1 Locus). This aggregation accounts for 40 per cent of the familial aggregation of type 1 diabetes. A second locus (IDDM 2) on chromosome 11p15.5 has been confirmed using case control and family based studies. Confirmation of other potential gene loci for type 1 diabetes has been difficult as the results can be variable and difficult to replicate partly due to differences in genetic susceptibility within different ethnic groups. Environmental agents, which interact with genetic factors, have also been implicated in the aetiology of type 1 diabetes. Enteroviruses have been suggested as having the potential to trigger beta cell damage in a significant proportion of patients. Dietary factors such as vitamin D deficiency and early exposure to cow's milk have also been explored as potential triggers of the immune process underlying type 1 diabetes.

3.4.2 Type 2 diabetes

Insulin resistance is coupled with beta cell dysfunction in type 2 diabetes but debate remains as to which is the primary defect. The roles of hyperinsulinaemia and insulin resistance in the pathogenesis of type 2 diabetes are now well recognized. A combination of genetic and environmental factors influences the progression of insulin resistance to diabetes. The finding that non-diabetic relatives of individuals with type 2 diabetes are insulin resistant suggests a strong genetic component. Several epidemiological studies have indicated that a sedentary lifestyle promotes the development of insulin resistance and it is well established that physical exercise can

improve glucose tolerance and insulin sensitivity. The change in insulin sensitivity is independent of obesity and can occur in the absence of any weight loss. As physical fitness improves there is a reduction in hyperinsulinaemia, with serum lipid profile and other metabolic parameters showing some improvement. Excessive food intake is also detrimental to insulin sensitivity and its effects are to some extent independent of fat accumulation.

Intrauterine factors may also be important in that type 2 diabetes is significantly more prevalent in adults who had low birth weight for gestational age. This relationship is independent of adult body weight and social class. The effects of impaired foetal growth may be modified by subsequent growth and the risks of developing type 2 diabetes is highest in those who are small at birth and become overweight. Undernutrition in foetal life may result in permanent physiological changes that would be beneficial if nutrition remained scarce after birth. But, if nutrition becomes plentiful these changes predispose to obesity and impaired glucose tolerance. This has been described as the thrifty phenotype hypothesis.

Table 3.2 type 1 and type 2 diabetes aetiology

Type 1 Diabetes-beta cell destruction	Genetic factors: HLA-DQ molecules - beta cell destruction IDDM 1 Locus (chromosome 6p21.3) IDDM 2 Locus (chromosome 11p15.5) Autoimmunity: antibodies to glutamic acid decarboxylase Environmental: for example, early exposure to cows milk enteroviruses vitamin D deficiency
Type 2 Diabetes- insulin resistance and beta cell dysfunction	Lifestyle: sedentary lifestyle excessive food intake Intrauterine: decreased birth weight thrifty phenotype Genetic: strong familial tendency no candidate genes identified as yet

The aetiological types designate defects, disorders or processes which often result in diabetes mellitus. Diabetes is a chronic disease, characterized by hyperglycaemia. It is caused by deficient insulin production, insensitivity to the action of insulin, or a combination of both of these. Knowledge of the relationship between glucose, insulin and counter-regulatory hormones and glucose homeostasis is important to understand these defects and how they result in abnormal glucose and fat metabolism.

3.4.3 Other specific types

Other specific types are currently less common causes of diabetes mellitus, but are those in which the underlying defect or disease process can be identified in a relatively specific manner. They include, for example, fibrocalculous pancreatopathy, a form of diabetes which was formerly classified as one type of malnutrition-related diabetes mellitus.

Table 3.3 Other specific types of diabetes

Genetic defects of beta-cell function
Chromosome 20, HNF4a (MODY1)
Chromosome 7, glucokinase (MODY2)
Chromosome 12, HNF1a (MODY3)
Chromosome 13, IPF-1/MODY4)
Mitochondrial DNA 3243 mutation
Others
Genetic defects in insulin action
Type A insulin resistance
Leprechaunism
Rabson-Mendenhall syndrome
Lipoatrophic diabetes
Others
Diseases of the exocrine pancreas
Fibrocalculous pancreatopathy
Pancreatitis
Trauma/pancreatectomy
Neoplasia
Cystic fibrosis
Haemochromatosis

Others
Endocrinopathies
Cushing's syndrome
Acromegaly
Phaeochromocytoma
Glucagonoma
Hyperthyroidism
Somatostatinoma
Others

3.5 GESTATIONAL HYPERGLYCAEMIA AND DIABETES

Gestational diabetes is carbohydrate intolerance resulting in hyperglycaemia of variable severity with onset or first recognition during pregnancy. The definition applies irrespective of whether or not insulin is used for treatment or the condition persists after pregnancy. Women who become pregnant and who are known to have diabetes mellitus which antedates pregnancy do not have gestational diabetes but have "diabetes mellitus and pregnancy" and should be treated accordingly before, during, and after the pregnancy.

In the early part of pregnancy (e.g. first trimester and first half of second trimester) fasting and postprandial glucose concentrations are normally lower than in normal, non-pregnant women. Individuals at high risk for gestational diabetes include older women, those with previous history of glucose intolerance, those with a history of large for gestational age babies, women from certain high-risk ethnic groups, and any pregnant woman who has elevated fasting, or casual, blood glucose levels. It may be appropriate to screen pregnant women belonging to high-risk populations during the first trimester of pregnancy in order to detect previously undiagnosed diabetes mellitus. Formal systematic testing for gestational diabetes is usually done between 24 and 28 weeks of gestation.

3.5.1 Diagnosis of gestational diabetes

To determine if gestational diabetes is present in pregnant women, a standard OGTT should be performed after overnight fasting (8–14 hours) by giving 75 g anhydrous glucose in 250–300 ml water. Plasma glucose is measured fasting and after 2 hours. Pregnant women who meet WHO criteria for diabetes mellitus or IGT are classified as having Gestational Diabetes Mellitus (GDM). After the pregnancy ends, the woman should be reclassified as having either diabetes mellitus, or IGT, or normal glucose tolerance based on the results of a 75 g OGTT six weeks or more after delivery. It should be emphasized that such women, regardless of the 6-week post-pregnancy result, are at increased risk of subsequently developing diabetes.

3.6 DESCRIPTION OF AETIOLOGICAL TYPES

3.6.1. Type1 (beta-cell destruction, usually leading to absolute insulin deficiency)

3.6.1.1. Autoimmune Diabetes Mellitus

This form of diabetes previously encompassed by the terms insulin-dependent diabetes, Type 1 diabetes, or juvenile-onset diabetes, results from autoimmune mediated destruction of the beta cells of the pancreas. The rate of destruction is quite variable, being rapid in some individuals and slow in others²⁴. The rapidly progressive form is commonly observed in children, but also may occur in adults²⁵. The slowly progressive form generally occurs in adults and is sometimes referred to as latent autoimmune diabetes in adults (LADA). Some patients, particularly children and adolescents, may present with ketoacidosis as the first manifestation of the disease²⁶. Others have modest fasting hyperglycaemia that can rapidly change to severe hyperglycaemia and/or ketoacidosis in the presence of infection or other stress. Still others, particularly adults, may retain residual beta-cell function, sufficient to prevent ketoacidosis, for many years²⁷. Individuals with this form of Type 1 diabetes often become dependent on insulin for survival eventually and are at risk for ketoacidosis²⁸. At this stage of the disease, there is little or no insulin secretion as manifested by low or undetectable levels of plasma C-peptide²⁹. Markers of immune destruction, including islet cell autoantibodies, and/or autoantibodies to insulin, and autoantibodies to glutamic acid decarboxylase (GAD) are present in 85–90 % of individuals with Type 1 diabetes mellitus when fasting diabetic hyperglycaemia is initially detected³⁰. The peak incidence of this form of Type 1 diabetes occurs in childhood and adolescence, but the onset may occur at any age, ranging from childhood to the ninth decade of life³¹. There is a genetic predisposition to autoimmune destruction of beta cells, and it is also related to environmental factors that are still poorly defined. Although patients are usually not obese when they present with this type of diabetes, the presence of obesity is not incompatible with the diagnosis. These patients may also have other autoimmune disorders such as Graves' disease, Hashimoto's thyroiditis, and Addison's disease.³²

3.6.1.2. Idiopathic

There are some forms of Type 1 diabetes which have no known aetiology. Some of these patients have permanent insulinopenia and are prone to ketoacidosis, but have no evidence of autoimmunity³³. This form of diabetes is more common among individuals of African and Asian origin. In another form found in Africans an absolute requirement for insulin replacement therapy in affected patients may come and go, and patients periodically develop ketoacidosis³⁴.

3.6.2. Type 2 (predominantly insulin resistance with relative insulin deficiency or predominantly an insulin secretory defect with/without insulin resistance)

Diabetes mellitus of this type previously encompassed non-insulin-dependent diabetes, or adult-onset diabetes. It is a term used for individuals who have relative (rather than absolute) insulin deficiency. People with this type of diabetes frequently are resistant to the action of insulin^{35,36}. At least initially, and often throughout their lifetime, these individuals do not need insulin treatment to survive. This form of diabetes is frequently undiagnosed for many years because the hyperglycaemia is often not severe enough to provoke noticeable symptoms of diabetes. Nevertheless, such patients are at increased risk of developing macrovascular and microvascular complications.

The majority of patients with this form of diabetes are obese, and obesity itself causes or aggravates insulin resistance. Many of those who are not obese by traditional weight criteria may have an increased percentage of body fat distributed predominantly in the abdominal region⁴¹. Ketoacidosis is infrequent in this type of diabetes; when seen it usually arises in association with the stress of another illness such as infection^(42,43). Whereas patients with this form of diabetes may have insulin levels that appear normal or elevated, the high blood glucose levels in these diabetic patients would be expected to result in even higher insulin values had their beta-cell function been normal⁽⁴⁴⁾. Thus, insulin secretion is defective and insufficient to compensate for the insulin resistance. On the other hand, some individuals have essentially normal insulin action, but markedly impaired insulin secretion. Insulin sensitivity may be increased by weight reduction, increased physical activity, and/or pharmacological treatment of hyperglycaemia but is not restored to normal^(45,46). The risk of developing Type 2 diabetes increases with age, obesity, and lack of physical activity⁴⁷. It occurs more frequently in women with prior GDM and in individuals with hypertension or dyslipidaemia. Its frequency varies in different racial/ethnic subgroups. It is often associated with strong familial, likely genetic, predisposition. However, the genetics of this form of diabetes are complex and not clearly defined.

3.6.3. Other Specific Types

3.6.3.1. Genetic defects of beta-cell function

Several forms of the diabetic state may be associated with monogenic defects in beta-cell function, frequently characterized by onset of mild hyperglycaemia at an early age (generally before age 25 years). They are usually inherited in an autosomal dominant pattern. Patients with these forms of diabetes, formerly referred to as maturity-onset diabetes of the young (MODY), have impaired insulin secretion with minimal or no defect in insulin action⁴⁸. Abnormalities at three genetic loci on different chromosomes have now been characterized. The most common form is associated with mutations on chromosome 12 in a hepatic nuclear transcription factor referred to as HNF1. A second form is associated with mutations in the glucokinase gene on chromosome 7p⁴⁹. Glucokinase converts glucose to glucose-6-phosphate, the metabolism of which in turn stimulates insulin secretion by the beta cell. Thus, glucokinase serves as the "glucose sensor" for the beta cell. Because of defects in the glucokinase gene, increased levels of glucose are necessary to elicit normal levels of insulin secretion. A third form is associated with a mutation in the HNF4" gene on chromosome 20q. HNF4" is a transcription factor which is involved in the regulation of the expression of HNF1". A fourth variant has recently been ascribed to mutations in another transcription factor gene, IPF-1, which in its homozygous form leads to total pancreatic agenesis. Specific genetic defects in other individuals who have a similar clinical presentation are currently being defined. Point mutations in mitochondrial DNA have been found to be associated with diabetes mellitus and deafness. The most common mutation occurs at position 3243 in the tRNA leucine gene, leading to an A to G substitution. An identical lesion occurs in the MELAS syndrome (Mitochondrial myopathy, Encephalopathy, Lactic Acidosis, and Stroke-like syndrome); however, diabetes is not part of this syndrome, suggesting for unknown reasons different phenotypic expressions of this genetic lesion.

Genetic abnormalities that result in the inability to convert proinsulin to insulin have been identified in a few families. Such traits are usually inherited in an autosomal dominant pattern and the resultant carbohydrate intolerance is mild. Similarly, mutant insulin molecules with impaired receptor binding have been identified in a few families. These are also associated with autosomal inheritance and either normal or only mildly impaired carbohydrate metabolism.

3.6.3.2. Genetic defects in insulin action

There are some unusual causes of diabetes which result from genetically determined abnormalities of insulin action. The metabolic abnormalities associated with mutations of the insulin receptor may range from hyperinsulinaemia and modest hyperglycaemia to symptomatic diabetes. Some individuals with these mutations have acanthosis nigricans. Women may have virilization and have enlarged, cystic ovaries. In the past, this syndrome was termed Type A insulin resistance.

3.6.3.3. Diseases of the exocrine pancreas

Any process that diffusely injures the pancreas can cause diabetes. Acquired processes include pancreatitis, trauma, infection, pancreatic carcinoma, and pancreatectomy. With the exception of cancer, damage to the pancreas must be extensive for diabetes to occur. However, adenocarcinomas that involve only a small portion of the pancreas have been associated with diabetes. This implies a mechanism other than simple reduction in beta-cell mass. If extensive enough, cystic fibrosis and haemochromatosis will also damage beta cells and impair insulin secretion. Fibrocalculous pancreatopathy may be accompanied by abdominal pain radiating to the back and

pancreatic calcification on X-ray and ductal dilatation. Pancreatic fibrosis and calcified stones in the exocrine ducts are found at autopsy.

3.6.3.4. Endocrinopathies

Several hormones (e.g. growth hormone, cortisol, glucagon, epinephrine) antagonize insulin action. Diseases associated with excess secretion of these hormones can cause diabetes (e.g. Acromegaly, Cushing's Syndrome, Glucagonoma and Pheochromocytoma) These forms of hyperglycaemia typically resolve when the hormone excess is removed. Somatostatinoma, and aldosteronoma-induced hypokalaemia, can cause diabetes, at least in part by inhibiting insulin secretion. Hyperglycaemia generally resolves following successful removal of the tumour.

3.6.3.5. Drug- or chemical-induced diabetes

Many drugs can impair insulin secretion. These drugs may not, by themselves, cause diabetes but they may precipitate diabetes in persons with insulin resistance. In such cases, the classification is ambiguous, as the primacy of beta-cell dysfunction or insulin resistance is unknown. Certain toxins such as Vacor (a rat poison) and pentamidine can permanently destroy pancreatic beta cells. Fortunately, such drug reactions are rare. There are also many drugs and hormones which can impair insulin action. Examples include nicotinic acid and glucocorticoids.

3.6.3.6. Infections

Certain viruses have been associated with beta-cell destruction. Diabetes occurs in some patients with congenital rubella. In addition, Coxsackie B, cytomegalovirus and other viruses (e.g. adenovirus and mumps) have been implicated in inducing the disease.

3.6.3.7. Uncommon but specific forms of immune-mediated diabetes mellitus

Diabetes may be associated with several immunological diseases with a pathogenesis or aetiology different from that which leads to the Type 1 diabetes process. Postprandial hyperglycaemia of a severity sufficient to fulfill the criteria for diabetes has been reported in rare individuals who spontaneously develop insulin auto antibodies. However, these individuals generally present with symptoms of hypoglycaemia rather than hyperglycaemia. The "stiff man syndrome" is an autoimmune disorder of the central nervous system, characterized by stiffness of the axial muscles with painful spasms. Affected people usually have high titers of the GAD auto antibodies and approximately one-half will develop diabetes. Patients receiving interferon alpha have been reported to develop diabetes associated with islet cell auto antibodies and, in certain instances, severe insulin deficiency. Anti-insulin receptor antibodies are occasionally found in patients with systemic lupus erythematosus and other autoimmune diseases. As in other states of extreme insulin resistance, patients with anti-insulin receptor antibodies often have acanthosis nigricans. In the past, this syndrome was termed Type B insulin resistance.

3.6.3.8. Other genetic syndromes sometimes associated with diabetes

Many genetic syndromes are accompanied by an increased incidence of diabetes mellitus. These include the chromosomal abnormalities of Down's syndrome, Klinefelter's syndrome and Turner's syndrome. Wolfram's syndrome is an autosomal recessive disorder characterized by insulin-deficient diabetes and the absence of beta cells at autopsy. Additional manifestations include diabetes insipidus, hypogonadism, optic atrophy, and neural deafness.

3.7 COMPLICATION ASSOCIATED WITH DIABETES

Diabetes is a progressive and life-threatening condition with potentially devastating consequences for health. Diabetes increases the risk of atherosclerotic cardiovascular diseases such as CHD, stroke and peripheral vascular disease (macrovascular disease). It is second only to smoking as the major aetiological factor in atherosclerotic vascular disease which can lead to myocardial infarction, stroke and lower limb amputation. Diabetes also causes harm throughout the body by damaging small blood vessels. Initially, these changes to the microcirculation are reversible. In the long term, however, poorly controlled diabetes can lead to a range of serious complications, which include diabetic retinopathy, diabetic nephropathy, and diabetic neuropathy

- 12 % of all cases of blindness are due to diabetes
- 40 % of all diabetics die of cardiovascular complications of the disease
- 50 % of all amputations are performed in people with T2D
- 80 % of all type 2 diabetics have high blood pressure and
- 50 % of all dialysis patients are people with T2D

3.7.1 Chronic Complications of DM

The chronic complications of DM affect many organ systems and are responsible for the majority of morbidity and mortality associated with the disease. Chronic complications can be divided into vascular and nonvascular complications. The vascular complications of DM are further subdivided into microvascular (retinopathy, neuropathy, nephropathy) and macrovascular complications [coronary artery disease (CAD), peripheral arterial disease (PAD), cerebrovascular disease].

Nonvascular complications include problems such as gastroparesis, infections, and skin changes. Long-standing diabetes may be associated with hearing loss. Whether type 2 DM in elderly individuals is associated with impaired mental function is not clear⁵⁰.

Table 3.4 Chronic Complications of Diabetes Mellitus

- Microvascular
- Eye disease
- Retinopathy (nonproliferative/proliferative)
- Macular edema
- Neuropathy
- Sensory and motor (mono- and polyneuropathy)
- Autonomic
- Nephropathy
- Macrovascular
- Coronary artery disease
- Peripheral arterial disease
- Cerebrovascular disease
- Other
- Gastrointestinal (gastroparesis, diarrhea)
- Genitourinary (uropathy/sexual dysfunction)
- Dermatologic
- Infectious
- Cataracts
- Glaucoma
- Periodontal disease

The risk of chronic complications increases as a function of the duration of hyperglycemia; they usually become apparent in the second decade of hyperglycemia. Since type 2 DM often has a long asymptomatic period of hyperglycemia, many individuals with type 2 DM have complications at the time of diagnosis. The microvascular complications of both type 1 and type 2 DM result from chronic hyperglycemia.

3.7.1.1 Diabetic Retinopathy

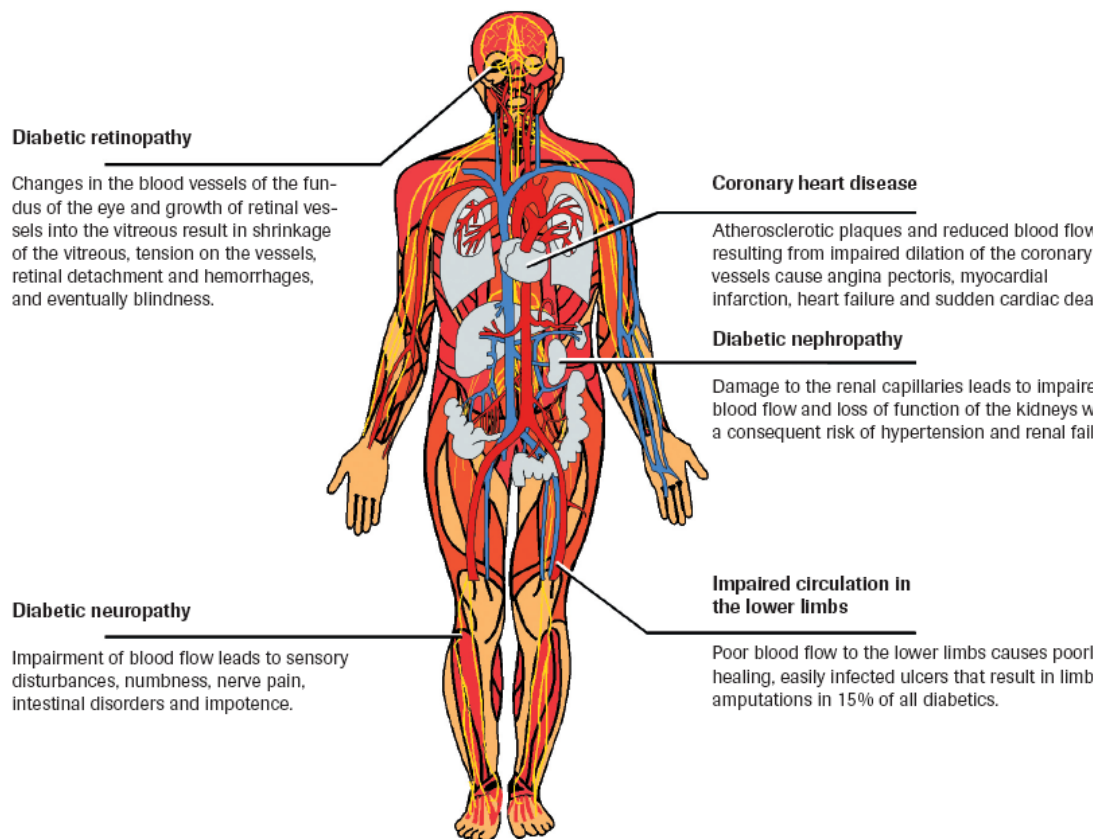
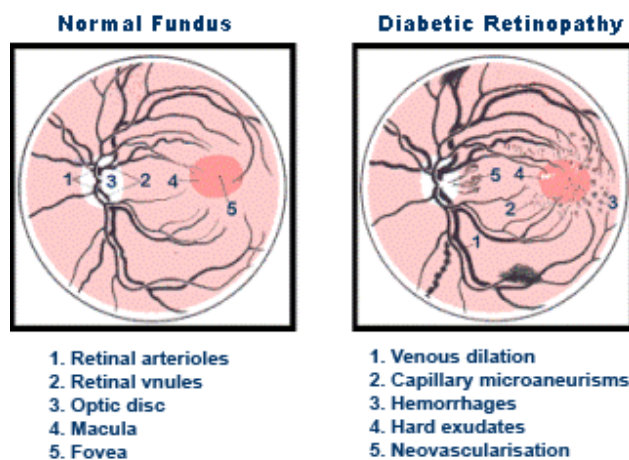


Fig 3.1 Complications of chronic hyperglycemia

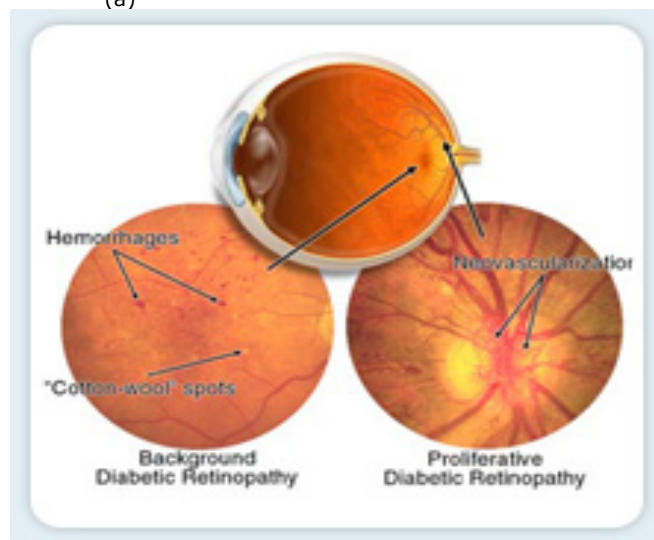
The gravity of this problem is highlighted by the finding that individuals with DM are 25 times more likely to become legally blind than individuals without DM. Blindness is primarily the result of progressive diabetic

retinopathy and clinically significant macular edema. Diabetic retinopathy is classified into two stages: nonproliferative and proliferative. Nonproliferative diabetic retinopathy usually appears late in the first decade or early in the second decade of the disease and is marked by retinal vascular microaneurysms, blot hemorrhages, and cotton wool spots. Mild nonproliferative retinopathy progresses to more extensive disease, characterized by changes in venous vessel caliber, intraretinal microvascular abnormalities, and more numerous microaneurysms and hemorrhages. The pathophysiologic mechanisms invoked in nonproliferative retinopathy include loss of retinal pericytes, increased retinal vascular permeability, alterations in retinal blood flow, and abnormal retinal microvasculature, all of which lead to retinal ischemia.

The appearance of neovascularization in response to retinal hypoxia is the hallmark of proliferative diabetic retinopathy. These newly formed vessels appear near the optic nerve and/or macula and rupture easily, leading to vitreous hemorrhage, fibrosis, and ultimately retinal detachment. Not all individuals with nonproliferative retinopathy develop proliferative retinopathy, but the more severe the nonproliferative disease, the greater the chance of evolution to proliferative retinopathy within 5 years. Clinically significant macular edema can occur when only nonproliferative retinopathy is present. Fluorescein angiography is useful to detect macular edema, which is associated with a 25% chance of moderate visual loss over the next 3 years.



(a)



(b)

Fig 3.2 (a & b) Diabetic retinopathy

Duration of DM and degree of glycemic control are the best predictors of the development of retinopathy; hypertension is also a risk factor. Nonproliferative retinopathy is found in almost all individuals who have had DM for >20 years (25% incidence with 5 years, and 80% incidence with 15 years of type 1 DM). Although there is genetic susceptibility for retinopathy, it confers less influence than either the duration of DM or the degree of glycemic control.

3.7.1.2 Neuropathy and Diabetes Mellitus

Diabetic neuropathy occurs in ~50% of individuals with long-standing type 1 and type 2 DM. It may manifest as polyneuropathy, mononeuropathy, and/or autonomic neuropathy. As with other complications of DM, the development of neuropathy correlates with the duration of diabetes and glycemic control. Additional risk factors are BMI (the greater the BMI, the greater the risk of neuropathy) and smoking. The presence of cardiovascular disease, elevated triglycerides, and hypertension is also associated with diabetic peripheral neuropathy. 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. The ADA recommends screening for distal symmetric neuropathy beginning with the initial diagnosis of diabetes and screening for autonomic neuropathy 5 years after diagnosis of type 1 DM and at the time of diagnosis of type 2 DM. All individuals with diabetes should then be screened annually for both forms of neuropathy.

Polyneuropathy/Mononeuropathy

The most common form of diabetic neuropathy is distal symmetric polyneuropathy. It most frequently presents with distal sensory loss, but up to 50% of patients do not have symptoms of neuropathy. Hyperesthesia, paresthesia, and dysesthesia also may occur. Any combination of these symptoms may develop as neuropathy progresses. Symptoms may include a sensation of numbness, tingling, sharpness, or burning that begins in the feet and spreads proximally. Neuropathic pain develops in some of these individuals, occasionally preceded by improvement in their glycemic control. Pain typically involves the lower extremities, is usually present at rest, and worsens at night. Both an acute (lasting <12 months) and a chronic form of painful diabetic neuropathy have been described. As diabetic neuropathy progresses, the pain subsides and eventually disappears, but a sensory deficit in the lower extremities persists. Physical examination reveals sensory loss, loss of ankle reflexes, and abnormal position sense.

Diabetic polyradiculopathy is a syndrome characterized by severe disabling pain in the distribution of one or more nerve roots. It may be accompanied by motor weakness. Intercostal or truncal radiculopathy causes pain over the thorax or abdomen. Involvement of the lumbar plexus or femoral nerve may cause severe pain in the thigh or hip and may be associated with muscle weakness in the hip flexors or extensors (diabetic amyotrophy). Fortunately, diabetic polyradiculopathies are usually self-limited and resolve over 6–12 months.

Mononeuropathy (dysfunction of isolated cranial or peripheral nerves) is less common than polyneuropathy in DM and presents with pain and motor weakness in the distribution of a single nerve. A vascular etiology has been suggested, but the pathogenesis is unknown. Involvement of the third cranial nerve is most common and is heralded by diplopia. Physical examination reveals ptosis and ophthalmoplegia with normal pupillary constriction to light. Sometimes other cranial nerves IV, VI, or VII (Bell's palsy) are affected. Peripheral mononeuropathies or simultaneous involvement of more than one nerve (mononeuropathy multiplex) may also occur.

Autonomic Neuropathy

Individuals with long-standing type 1 or 2 DM may develop signs of autonomic dysfunction involving the cholinergic, noradrenergic, and peptidergic (peptides such as pancreatic polypeptide, substance P, etc.) systems. DM-related autonomic neuropathy can involve multiple systems, including the cardiovascular, gastrointestinal, genitourinary, sudomotor, and metabolic systems. Autonomic neuropathies affecting the cardiovascular system cause a resting tachycardia and orthostatic hypotension. Reports of sudden death have also been attributed to autonomic neuropathy. Gastroparesis and bladder-emptying abnormalities are often caused by the autonomic neuropathy seen in DM. Hyperhidrosis of the upper extremities and anhidrosis of the lower extremities result from sympathetic nervous system dysfunction. Anhidrosis of the feet can promote dry skin with cracking, which increases the risk of foot ulcers. Autonomic neuropathy may reduce counter regulatory hormone release, leading to an inability to sense hypoglycemia appropriately, thereby subjecting the patient to the risk of severe hypoglycemia and complicating efforts to improve glycemic control.

3.7.1.3 Gastrointestinal/Genitourinary Dysfunction

Long-standing type 1 and 2 DM may affect the motility and function of gastrointestinal (GI) and genitourinary systems. The most prominent GI symptoms are delayed gastric emptying (gastroparesis) and altered small- and large-bowel motility (constipation or diarrhea). Gastroparesis may present with symptoms of anorexia, nausea, vomiting, early satiety, and abdominal bloating. Microvascular complications (retinopathy and neuropathy) are usually present. Though parasympathetic dysfunction secondary to chronic hyperglycemia is important in the development of gastroparesis, hyperglycemia itself also impairs gastric emptying. Nocturnal diarrhea, alternating with constipation, is a feature of DM-related GI autonomic neuropathy. In type 1 DM, these symptoms should also prompt evaluation for celiac sprue because of its increased frequency. Esophageal dysfunction in long-standing DM may occur but is usually asymptomatic.

Diabetic autonomic neuropathy may lead to genitourinary dysfunction including cystopathy, erectile dysfunction, and female sexual dysfunction (reduced sexual desire, dyspareunia, reduced vaginal lubrication). Symptoms of diabetic cystopathy begin with an inability to sense a full bladder and a failure to void completely. As bladder contractility worsens, bladder capacity and the post-void residual increase, leading to symptoms of urinary

hesitancy, decreased voiding frequency, incontinence, and recurrent urinary tract infections. Diagnostic evaluation includes cystometry and urodynamic studies.

Erectile dysfunction and retrograde ejaculation are very common in DM and may be one of the earliest signs of diabetic neuropathy. Erectile dysfunction, which increases in frequency with the age of the patient and the duration of diabetes, may occur in the absence of other signs of diabetic autonomic neuropathy.

Cardiovascular Morbidity and Mortality

The American Heart Association has designated DM as a major risk factor for cardiovascular disease (same category as smoking, hypertension, and hyperlipidemia). Type 2 diabetes patients without a prior MI have a similar risk for coronary artery-related events as nondiabetic individuals who have had a prior MI. Because of the extremely high prevalence of underlying cardiovascular disease in individuals with diabetes (especially in type 2 DM), evidence of atherosclerotic vascular disease (e.g., cardiac stress test) should be sought in an individual with diabetes who has symptoms suggestive of cardiac ischemia, peripheral or carotid arterial disease, a resting electrocardiogram indicative of prior infarction, plans to initiate an exercise program, proteinuria, or two other cardiac risk factors (ADA recommendations). The absence of chest pain ("silent ischemia") is common in individuals with diabetes, and a thorough cardiac evaluation is indicated in individuals undergoing major surgical procedures. The prognosis for individuals with diabetes who have CAD or MI is worse than for nondiabetics. CAD is more likely to involve multiple vessels in individuals with DM.

Risk factors for macrovascular disease in diabetic individuals include dyslipidemia, hypertension, obesity, reduced physical activity, and cigarette smoking. Additional risk factors more prevalent in the diabetic population include microalbuminuria, macroalbuminuria, an elevation of serum creatinine, and abnormal platelet function. Insulin resistance, as reflected by elevated serum insulin levels, is associated with an increased risk of cardiovascular complications in individuals with and without DM. Individuals with insulin resistance and type 2 DM have elevated levels of plasminogen activator inhibitors (especially PAI-1) and fibrinogen, which enhances the coagulation process and impairs fibrinolysis, thus favoring the development of thrombosis. Diabetes is also associated with endothelial, vascular smooth-muscle, and platelet dysfunction. In addition to CAD, cerebrovascular disease is increased in individuals with DM (threefold increase in stroke). Individuals with DM have an increased incidence of CHF.

Hypertension

Hypertension can accelerate other complications of DM, particularly cardiovascular disease and nephropathy. In targeting the goal of BP < 130/80, therapy should first emphasize life-style modifications such as weight loss, exercise, stress management, and sodium restriction.

Because of the high prevalence of atherosclerotic disease in individuals with DM, the possibility of renal vascular hypertension should be considered when the blood pressure is not readily controlled.

3.7.1.4 Lower Extremity Complications

DM is the leading cause of non traumatic lower extremity amputation in the United States. Foot ulcers and infections are also a major source of morbidity in individuals with DM. The reasons for the increased incidence of these disorders in DM involve the interaction of several pathogenic factors: neuropathy, abnormal foot biomechanics, PAD, and poor wound healing. The peripheral sensory neuropathy interferes with normal protective mechanisms and allows the patient to sustain major or repeated minor trauma to the foot, often without knowledge of the injury. Disordered proprioception causes abnormal weight bearing while walking and subsequent formation of callus or ulceration. Motor and sensory neuropathy lead to abnormal foot muscle mechanics and to structural changes in the foot (hammer toe, claw toe deformity, prominent metatarsal heads, Charcot joint). Autonomic neuropathy results in anhidrosis and altered superficial blood flow in the foot, which promote drying of the skin and fissure formation. PAD and poor wound healing impede resolution of minor breaks in the skin, allowing them to enlarge and to become infected.



Fig 3.4 diabetic ulcer

Approximately 15% of individuals with DM develop a foot ulcer (great toe or MTP areas are most common), and a significant subset will ultimately undergo amputation (14–24% risk with that ulcer or subsequent ulceration). Risk factors for foot ulcers or amputation include: male sex, diabetes >10 years' duration, peripheral neuropathy, abnormal structure of foot (bony abnormalities, callus, thickened nails), peripheral arterial disease, smoking, history of previous ulcer or amputation, and poor glycemic control. Large calluses are often precursors to or overlie ulcerations

Infections

Individuals with DM have a greater frequency and severity of infection. The reasons for this include incompletely defined abnormalities in cell-mediated immunity and phagocyte function associated with hyperglycemia, as well as diminished vascularization. Hyperglycemia aids the colonization and growth of a variety of organisms (*Candida* and other fungal species). Many common infections are more frequent and severe in the diabetic population, whereas several rare infections are seen almost exclusively in the diabetic population. Invasive otitis externa is usually secondary to *P. aeruginosa* infection in the soft tissue surrounding the external auditory canal, usually begins with pain and discharge, and may rapidly progress to osteomyelitis and meningitis. These infections should be sought, in particular, in patients presenting with HHS.

Pneumonia, urinary tract infections, and skin and soft tissue infections are all more common in the diabetic population. In general, the organisms that cause pulmonary infections are similar to those found in the nondiabetic population; however, gram-negative organisms, *S. aureus*, and *Mycobacterium tuberculosis* are more frequent pathogens. 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.

Dermatologic Manifestations

The most common skin manifestations of DM are protracted wound healing and skin ulcerations. Diabetic dermopathy, sometimes termed *pigmented pretibial papules*, or "diabetic skin spots," begins as an erythematous area and evolves into an area of circular hyperpigmentation. These lesions result from minor mechanical trauma in the pretibial region and are more common in elderly men with DM. Bullous diseases, *bullosa diabeticorum* (shallow ulcerations or erosions in the pretibial region), are also seen. *Necrobiosis lipoidica diabeticorum* is a rare disorder of DM that predominantly affects young women with type 1 DM, neuropathy, and retinopathy. It usually begins in the pretibial region as an erythematous plaque or papules that gradually enlarge, darken, and develop irregular margins, with atrophic centers and central ulceration. They may be painful. Vitiligo occurs at increased frequency in individuals with type 1 diabetes. *Acanthosis nigricans* (hyperpigmented velvety plaques seen on the neck, axilla, or extensor surfaces) is sometimes a feature of severe insulin resistance and accompanying diabetes. Generalized or localized *granuloma annulare* (erythematous plaques on the extremities or trunk) and *scleredema* (areas of skin thickening on the back or neck at the site of previous superficial infections) are more common in the diabetic population

3.7.2. Acute Complications of Diabetes Mellitus

Hypoglycemia is defined as a blood sugar of <60 mg/dl and is the most common complication of insulin-treated diabetics. Hypoglycemia may cause a sense of hunger initially, followed by irritability, tachycardia, palpitations, cold sweat, decreased mental abilities followed by confusion and coma if not recognized. Treatment requires intake of calories with quickly absorbed sources of glucose (tablets, juice, or candy) if the patient is able to chew/swallow. If the diabetic becomes unconscious or swallowing ability is compromised, then glucagons should be administered as well as intravenous glucose.

Diabetic ketoacidosis (DKA) results from unrecognized or untreated insulin deficiency (type 1 diabetes) or any severe stress in established type 1 diabetics. With the inadequate glucose utilization in this condition, there is unregulated lipolysis with formation of free fatty acids that are converted to ketones. All type 1 diabetics are encouraged to monitor urine ketones when the blood sugar exceeds 240 to prevent the development to DKA. If urine ketones are positive, supplemental insulin is to be given (often by a sliding scale of subcutaneous regular insulin) every two hours and oral fluids increased until the urine ketones are negative. Inability to increase fluid intake, comply with glucose monitoring and supplemental insulin administration, or resolve hyperglycemia/clear urine ketones is an indication for an emergent evaluation by the patient's diabetologist/primary care provider or emergency room physician. DKA can uncommonly occur in a severely stressed type 2 diabetic if there is decreased insulin action and relative decreased secretion.

Symptoms include fever, malaise, headache, dry mouth polyuria, polydipsia, nausea, vomiting, abdominal pain, and lethargy. Treatment requires replacement of what has been lost during the development of DKA: fluids, insulin, and potassium. Thus all type 1 diabetics and infrequently type 2 diabetics with dental infections and hyperglycemia are at increased risk for DKA and need to be monitoring urine ketones and adjusting insulin/fluid intake accordingly. The dental professional needs to recognize the often-increased insulin requirement in this setting and encourage the patient to work with his diabetes management team to optimize control and avoid DKA. Hyperosmolar hyperglycemia nonketotic syndrome is a rare, acute condition of hyperglycemia (usually over 600mg/dl) with the absence of ketones found in type 2 diabetics. This condition is insidious in nature, and patients typically come to medical attention later and sicker. Fluids and insulin therapy accomplish treatment.

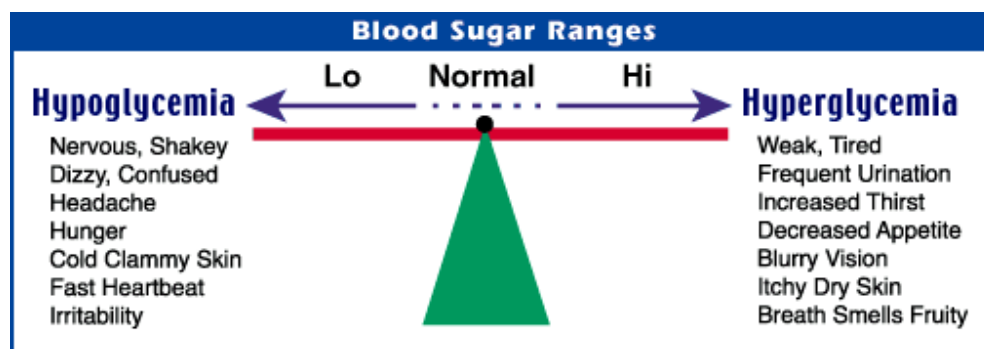


Fig 3.5 acute complication of diabetes mellitus

3.8 PSYCHOLOGICAL FACTORS AND DIABETES MELLITUS

Stress is a part and parcel of modern day life. Stress may be thought of as a) a physiological response to an external stimulus, or b) a psychological response to external stimuli, or c) stressful events themselves, which can be negative or positive or both. The three aspects of stress: stressful events or experiences (sometimes referred to as stressors) and the physiological and psychological/behavioral responses to these. Stressors may be internal and external. Our body responds to stress either as fight or flight response and repeated stress may lead to failing rheostat phenomenon of hypothalamus leading to less efficient hormonal control through feedbacks. This leads to various changes in body functioning at various levels like cellular, organic or systemic and finally leads to various diseases. Diabetes may be an outcome of stress and further sets in a vicious cycle of stress-diabetes relationship

Stress

Stress is defined as a 'stimulus event of sufficient severity to produce disequilibrium in the homeostasis of physiological systems'. Stressor is the threat that exceeds an individual's ability to meet the challenge and thereby evokes a stress response. It is perceived as stressful depending on the meaning that is ascribed to the stimulus, which in turn results in a sensory or metabolic process that is inherently stressful. Therefore, the definition of stress and stressor depends on the person's interpretation of the stimulus as being stressful⁵¹

- (1). Strain is the person's psychological or physiological response(s) to a stress or
- (2). It may take the form of anger, anxiety, frustration or autonomic nervous activation.

Humans are aware of subtle social cues that can affect self-esteem. The balance between stress and resilience determines one's vulnerability to its adverse effects. It also depends on both genetic influences and early life experiences. Genetic polymorphisms, early life and later life events may all activate the stress response inappropriately. Stress has been viewed in three ways:

Stimulus
Response
Process.

Stimulus refers to stress, which can be categorised as emanating from three sources:

Catastrophic events, such as Tornadoes and earthquakes
Major life events
Chronic circumstances, such as living in crowded or noisy conditions.

Response refers to how somebody responds to a particular stress, for example sitting an examination. There are two components:

Physiological, heightened bodily arousal-your heart pounds, mouth goes dry your stomach feels tight and you perspire.
Psychological, involving behaviour, thought patterns, and emotions. Feeling nervous.

Process views stress as a series of interactions and adjustments between the person and the environment. These interactions and adjustments are called transactions. Stress is not seen as a stimulus or a response, but rather as a process. The person suffering stress is seen as an active agent who can influence the impact of a stressor through behavioural, cognitive and emotional strategies.

Life transitions tend to be stressful (Moos and Schaefer, 1986). Changing from one phase to another in life is called a transition; examples include:

Starting school
Moving home
Reaching puberty
Starting college, especially away from home
Starting a career
Getting married
Becoming a parent
Losing a spouse through divorce or death
Retiring.

The timing of a life transition can affect the stress it produces. If a life event occurs at a time when it is not expected then this is stressful. One reason could be that having an event too early or too late could mean that one is deprived of the support of peers. An example of this would be having a baby at the age of 38 or later. Achieving life events late in life could be seen as failing. Some people who graduated late or were promoted late in life feel as though they have failed.

Controllability is another factor that will affect the perception of stress. People tend to appraise uncontrollable events as being more stressful than controllable events (Miller, 1979). There are two types of control:

Behavioural
Cognitive.

Behavioural control means performing some action. For example, being unable to take a tablet for a headache will make experiencing a headache less stressful. In the case of **cognitive** control, we can affect the impact of the events by using some mental strategy, such as distraction or by developing a plan to overcome the problem.

Biological aspects of stress

Walter Cannon (1929) describes the **fight or flight** response of the body after perceiving danger or stress. This response mobilises the organism to respond quickly to danger but the state of higher arousal can be harmful to health if it is prolonged.

3.8.1 General Adaptation Syndrome

Selye (1956) observed in laboratory animals and in human patients the body's reaction to stress. He found that the fight or flight response was only the first in a series of reactions, which he called the general adaptation syndrome (GAS). The GAS consists of three stages:

Alarm reaction
Stage of resistance
Stage of exhaustion.

The **alarm** reaction is like the fight or flight response to an emergency. The body is mobilised. At the beginning of the arousal blood pressure drops below normal for a moment, but then quickly rises to above normal. This arousal is produced by the release of hormones by the endocrine system: the pituitary glands secrete ACTH, which causes a heightened release of adrenaline, noradrenaline, and cortisol by the adrenal glands into the bloodstream. The

body cannot stay in this state for long without serious consequences. Some organisms in a continuous state of alarm have died within hours or days

Stage of resistance. If the reaction continues and is not strong enough to cause death the physiological reaction enters the stage of resistance. The body tries to adapt to the stressor. Physiological arousal declines but remains higher than normal and the body replenishes the hormones released by the adrenal glands. The organism may show few outward signs of stress. However, the body may not be able to resist new stresses. The body becomes increasingly vulnerable to health problems. These health problems include ulcers, high blood pressure, asthma, and illnesses that result from impaired immune function.

Stage of exhaustion. Severe long-term or repeated stress will cause the organism to enter the third stage, the stage of exhaustion. The immune system and the body's energy reserves are weakened until resistance is very limited. If the stress continues, disease and physiological damage become increasingly likely and death may result.

3.8.2 Biochemistry of Stress Response ⁵²

The hypothalamus in the brain produces corticotrophin releasing factor that stimulates the anterior pituitary to secrete corticotrophin or ACTH. ACTH in turn stimulates the adrenal cortex to secrete stress hormones. Arginine vasopressin, a product of the posterior pituitary is synergistic with CRH in stimulating ACTH. Alone however, vasopressin has little secretagogue activity. The autonomic nervous system responds to stress rapidly. The sympathetic and parasympathetic limbs of the autonomic nervous system regulate cardiovascular, respiratory, renal and endocrine systems. The brain ultimately orchestrates the global stress response by fine tuning the secretion of several neurotransmitters: CRH, AVP, opioid peptides, dopamine and norepinephrine, along with prolactin, glucagon, neuropeptide Y and others such as GABA, brain angiotensin II and protein kinase C. The combined stress response involving peripheral and central nervous system, blood cell composition and various activities initiated via a large number of genes, orchestrate the differential activation and suppression of systems for survival and adaptation to changing stimuli.

3.8.3 Physiological Correlates of Stress Response ⁵³

To measure the degree of stress in real life situations, heart rate and respiratory rate are useful surrogates. Fall in heart rate and the respiratory rate correlation coefficient is objective criteria of emotional stress at work. Such cross-correlation coefficients provide integral means to assess emotional stress.

There is increasing evidence that environmental factors cause illness by acting through the central nervous system. Effort is on to tease apart the effect of social environment on the biology of disease, with the aim of preventing disease, rather than trying to cure it. There is complex interaction of social structure, environment, workplace, along with material factors and psychological factors which influence health behavior.

Metabolic syndrome and hypothalamic-pituitary adrenal axis (HPA) (1): Bjorntop postulated that stress could activate the sympathetic nervous system and result in the metabolic syndrome through hormonal dysregulation. Psychosocial stress may trigger the onset of visceral obesity and other components of the metabolic syndrome. Difference in response among individuals to the same stimuli may be responsible for the stress being perceived as 'distress' or 'eustress'. HPA has been shown to be more active in centrally obese men and in the pre-menopausal centrally obese women. Central android obesity and peripheral gynecoid obesity may be associated with differential regulation of HPA, besides being targeted to metabolically important tissues such as liver and visceral fat.

Preferential deposition of fat in the abdomen may be due to activity of enzymes that metabolize glucocorticoids. The enzyme 11 beta HSD exists in two isoforms: type 1 (11 beta HSD1) and type 2 (11beta HSD2). The type 2 isoform irreversibly inactivates cortisol and corticosterone, oxidizing their 11 beta hydroxy groups to metabolites which bind only weakly to hormone receptors. The type 1 isoform catalyzes both the inactivating and activating reactions, which is principally seen in the liver. Stress related metabolic response via glucocorticoids may be modulated by the different isoforms of the enzyme. The beta HSD oxo-reductase activity in subcutaneous abdominal fat tissue was increased in obese individuals, which may activate local glucocorticoid receptors, promoting obesity. There is also evidence that 11 beta HSD activity in the placenta may be responsible for active forms of stress hormones passing through the placenta, resulting in adverse effects in utero. Intrauterine exposure to stress may activate the HPA axis. In populations undergoing health transition, metabolic syndrome and low birth weight may be linked through activation of HPA. The Hoorn Study was a clinical investigation to test the Bjorntop hypothesis. Chronic psychological stress was correlated with prevalence of type 2 diabetes mellitus and with visceral adiposity. More than 2000 adults aged 50-74 years without a history of diabetes mellitus were studied for the number of major stressful events during the preceding five years. An oral glucose tolerance test was given after the history taking. The number of stressful events was positively associated with the prevalence of newly diagnosed diabetes. Anxiety and depression occur in persons with diabetes more frequently than in the general population. In addition other problems are also common including fear of the future, restriction of leisure activities and depression partly as a result of physical disability

3.8.4 Neuroendocrine responses to stress

Stress is a state of threatened homeostasis, in which a stimulus is interpreted as being noxious. A variety of factors can activate the stress response: psychological, biological and physical. Selye described the 'general adaptation syndrome' which results from stressful stimuli. Physiologically the heart pumps more blood, respiration is faster,

blood is preferentially sent to the brain and the muscles. Body systems that are not immediately required to counter the acute stress are slowed down (e.g. growth and reproduction). Increased metabolic activity supplies fuel principally to the brain, heart and muscles. The Hypothalamus in the brain produces corticotrophin-releasing factor that stimulates the anterior pituitary to secrete corticotrophin or ACTH. ACTH in turn stimulates the adrenal cortex to secrete stress hormones. Arginine vasopressin, a product of the posterior pituitary is synergistic with CRH in stimulating ACTH. Alone however, vasopressin has little secretagogic activity. The autonomic nervous system responds rapidly to stress. The sympathetic and parasympathetic limbs of the autonomic nervous system regulate cardiovascular, respiratory, renal and endocrine systems. The brain ultimately orchestrates the global stress response by fine tuning the secretion of several neurotransmitters: CRH, AVP, opioid peptides, dopamine and norepinephrine, along with prolactin, glucagon, neuropeptide Y and others.⁵⁴

3.8.5 Emotions and psychological stress

Unlike lower animals, the human brain which is more developed, is sensitive to subtle social cues that can affect self-esteem. Threats to self-esteem and fear of losing control over one's environment can elicit a stress response. The balance between stress and resilience determines an individual's vulnerability to stress. This vulnerability depends on both genetic and early life influences. Genetic polymorphisms can affect genes that regulate stress system. Both early and late life events may activate the stress response inappropriately. Metabolic syndrome and hypothalamic-pituitary-adrenal axis. In obesity circulating levels of the adrenal cortical hormone cortisol may be either normal or low. Environmental stress can result in obesity. Bjorntop postulated that stress could activate the sympathetic nervous system and result in the metabolic syndrome through hormonal dysregulation. Difference in response among individuals to the same stimuli may be responsible for the same stress being perceived as 'distress' or 'eustress'. Hypothalamic-pituitary-adrenal axis (HPA) was more active in centrally obese men and in the pre-menopausal centrally obese women. Central android obesity and peripheral gynecoid obesity may be associated with differential regulation of HPA. Preferential deposition of fat in the abdomen may be due to activity of enzymes that metabolize glucocorticoids.

The enzyme beta HSD exists in two isoforms: type 1 (11 beta HSD1) and type 2 (beta HSD2). The type 2 isoform irreversibly inactivates cortisol and corticosterone, oxidizing their beta hydroxyl groups to metabolites which bind only weakly to hormone receptors. The type 1 isoform principally found in the liver catalyses both the inactivating and activating reactions. Stress-related metabolic response may be modulated by the different enzyme isoforms. The beta HSD oxo reductase activity in subcutaneous abdominal fat tissue was high in obesity, which may activate local glucocorticoid receptors, further promoting obesity. beta HSD activity in the placenta may also be responsible for adverse effects on the fetus, by allowing stress hormones to pass through the placenta. Intrauterine exposure to stress may activate the HPA axis. In populations undergoing health transition metabolic syndrome and low birth weight may be linked through activation of HPA. The Hoorn Study tested the Bjorntop hypothesis. Chronic psychological stress was correlated with prevalence of type 2 diabetes mellitus and with visceral adiposity. Over 2000 adults aged 50–74 years without known diabetes mellitus were evaluated for the number of major stressful events during the preceding five years. Oral glucose tolerance test was given after the history was elicited. It was found that the number of stressful events was positively associated with the prevalence of newly diagnosed diabetes⁵⁵.

3.8.6. Stress and Diabetes

The term STRESS needs to be clarified because it can be used in different ways. Hereditary and family history are important for the onset of diabetes; however, sudden onset is often associated with emotional stress, which disturbs the homeostatic balance in persons who are predisposed to the disorder. Psychological factors that seem significant are those provoking feelings of frustration, loneliness and dejection. Patients with diabetes must usually maintain some dietary control over their diabetes. When they are depressed and dejected, they often overeat or over drink self-destructively and cause their diabetes to get out of control. This reaction is especially common in patients with juvenile or type 1 diabetes. In known diabetes patient, ketoacidosis can produce some violence and confusion. More commonly, hypoglycemia (often occurring when a diabetic patient drinks alcohol) can produce severe anxiety stress, confusion and disturbed behaviour.⁵⁶

3.8.7. Role of Stress in the Onset of Diabetes

Stressful experiences have been implicated in the onset of diabetes in individuals already predisposed to developing the disease. As early as the beginning of the 17th century, the onset of diabetes was linked to "prolonged sorrow" by an English physician. Since then, a number of research studies have identified stressors such as family losses and workplace stress as factors triggering the onset of diabetes, both type 1 and type 2. For example, Thernlund et al.² suggested that negative stressful experiences in the first 2 years of life may increase the risk of developing type 1 diabetes in children. Other factors, such as high family chaos and behavioral problems, were also implicated. Other research has also supported the hypothesis that stressful experiences can lead to increased risk for developing type 1 or type 2 diabetes. In a large population-based survey of glucose intolerance, Mooyetal.⁶ demonstrated an association between stressful experiences and the diagnosis of type 2 diabetes. Although this was a cross-sectional study, the authors investigated stress levels in people with previously undetected diabetes in order to rule out the possibility that the disease itself influenced reports of stressful experiences. They also took other factors into account, such as alcohol consumption, physical activity level, and education. Bjorntop has attempted to explain the physiological links between stressful experiences and the onset of

diabetes. He argues that the psychological reaction to stressors of defeatism or helplessness leads to the activation of the hypothalamo-pituitary-adrenal (HPA) axis, leading in turn to various endocrine abnormalities, such as high cortisol and low sex steroid levels, that antagonize the actions of insulin. At the same time, an increase in visceral adiposity (increased girth) is seen, which plays an important role in diabetes by contributing to insulin resistance.⁸ Increased visceral adiposity can be measured by waist-to-hip ratio. In the Mooy et al. study of type 2 diabetes, there was only a weak association between stressful experiences and waist-to-hip ratio, suggesting that other factors, so far unidentified, may play a mediating role. Where stressful experiences have been implicated in the onset of type 1 diabetes, researchers have often attempted to explain this in terms of the effects of stress on the autoimmune system. Bottazo et al. hypothesized that environmental factors (e.g., viruses or toxic agents) trigger the autoimmune destruction of the β -cells in genetically predisposed individuals.⁵⁷

3.8.8. Stress and Diabetes Control

In recent years, some researchers have turned their attention to the possibilities of stressful experiences influencing diabetes control. A number of laboratory studies have been conducted to demonstrate the effects of specific stressful situations (for example, arithmetic problem solving, unpleasant interviews) on blood glucose levels. Many of these studies have demonstrated that these types of stressors can destabilize blood glucose levels, at least for hours at a time. World and have attempted to measure naturally occurring stress. These later studies are not without problems however, such as, the myriad possibilities for measurement and/or observation, which makes cross-study comparisons difficult. Stress may take the form of day-to-day hassles, and it may be that major life events (death of a close relative, losing a job) are an added layer of complexity, along with long-term chronic difficulties (e.g., providing long-term care for a relative or long-term unemployment).

In an attempt to overcome some of the previous methodological limitations, a prospective in-depth investigation into the relationship between glycemic control over time was designed. Individuals with type 1 diabetes were interviewed using an in depth interview schedule and then followed up quarterly for a year with measures of diabetes control (hemoglobin A1c [A1C]). Unlike previous studies, the participants were asked about both negative and positive stressors in their lives. The results showed that those whose glycemic control deteriorated over time were more likely to report negative stress, whereas those whose control improved over the follow-up period reported positive stress. Negative stressors included interpersonal conflicts, death of a close tie, and disturbed behavior of someone close, whereas positive stressors were events such as engagement to be married, birth of a child, or a desired change in employment.

Studies such as the one reported above have their limitations. For example, not all individuals perceive stressors in the same way; what is a negative stressor in one person's life might actually be a positive one in another's, so the context in which stress occurs is also important. Some people react to stressful events in a way that makes them psychologically vulnerable, for example, they may experience feelings of hopelessness or anxiety, particularly in the context of social isolation or poverty. Others may respond to stress in positive terms or as a "challenge," or they may feel better able to cope with the stress because they have several social supports or the support of a loving family. It is easier to categorize major stressors into those that are positive and those that are negative; it is more difficult for more minor stress or "hassles." Long-term chronic difficulties are also important, but may change in perceived level of severity or negativity over time.

One study found that children judged to have a "Type A" personality structure had an increased blood sugar elevation in response to stress. Children with a calmer disposition had a smaller glucose rise when stressed. (Stabler et al. 1987) A 1997 study suggested that Type I patients with a history of a psychiatric illness might be at increased risk for developing diabetic retinopathy. Those patients with a psychiatric history were found to have a higher average glycosylated hemoglobin. (a measure of long term diabetic control) (Cohen et al. 1997) Children whose relatives made more critical comments had significantly poorer glucose control. Interestingly enough, emotional over involvement between family members was not correlated with poor diabetic control. (Koenigsberg et al. 1993) Diabetic adolescents had a higher incidence of suicidal ideation than expected. Those with suicidal ideation took poorer care of themselves. Not living in a two-parent home was associated with poorer long-term diabetic control. (Goldston, et al. 1997)⁵⁸

Recent studies have suggested that effective treatment of depression can improve diabetic control. In a study by Lustman and colleagues, glucose levels were shown to improve as depression lifted. The better the improvement, the better the diabetic control. (Lustman et al. 1997a)

Diabetics with major depression have a very high rate of recurrent depressive episodes within the following five years. (Lustman et al 1977) A depressed person may not have the energy or motivation to maintain good diabetic management. Depression is frequently associated with unhealthy appetite changes. The suicidal diabetic adolescent has constant access to potentially lethal doses of insulin.

The impact of stressful experiences on diabetes is clearly varied and may depend on other psychosocial factors. One of these is social support, and research has shown this may provide a buffering effect in times of stress. Psychological support is also important. In a recent meta-analysis of randomized controlled trials, Ismail et al.⁵⁹ concluded that people with type 2 diabetes who received behavioral based diabetes education or psychological

interventions were likely to show improvements in both glycemic control and psychological distress. The relationship between stressful experiences and metabolic control is thought to differ greatly among individuals in terms of both the strength and the direction of the relationship, and these differences have serious implications for the design of effective interventions to reduce the impact of stressors. Precisely how stressors affect glycemic control remains controversial, and there may be both physiological and behavioral pathways between stressors and health status. The mechanisms through which this may take place may be direct (through physiological effects on the neuroendocrine system) or indirect (through alterations in health care practices in times of stress).

3.8.9. Behavior

The behavioral mechanisms through which stressful experiences might affect diabetes control are varied and often complex. There are, of course, many different types of stress, there are shorter- and longer-term stressors, and people may respond to these very differently. Difficulties in measurement such as those mentioned above also apply to measuring behavior. At the same time, differences in resources such as social supports, ability to cope, and other psychosocial variables will all affect both the response to and the behavior resulting from stressful experiences. Reactions to external stressors, for example, feelings of anxiety or depression, may lead to difficulties with self-care manifested through less physical activity, poorer diet, or difficulties with medication taking. Experiences of stress may lead to other unhealthy behaviors, such as smoking, which in turn are linked to poor blood glucose control but also to a greater risk of developing diabetes complications. Data from Smith's study indicated a range of behavior described as occurring in response to stress.

These behaviors ranged from unhealthy lifestyle patterns associated with alcohol and tobacco consumption to increased physical activity and relaxation, such as walking, yoga, swimming, meditation, and hypnotherapy. One particular type of stress was investigated in a Swedish study. Agardh et al. demonstrated that work stress, as indicated by low decision latitude (or fewer opportunities for decision making), along with a low sense of coherence, significantly increased risk for type 2 diabetes. Low sense of coherence is thought to negatively affect people's ability to cope with stressors and also to be linked to unhealthy lifestyle patterns that could lead to poor health. Research also supports the behavioral link. Peyrot et al. found that stress and coping affected glycemic control by interfering with self-care practices. Coping behavior was also shown to affect glycemic control in a study of type 1 and type 2 diabetes that used sophisticated statistical techniques to demonstrate a "network" of interlinked variables in relation to the achievement of treatment goals.

For example, active coping behavior was associated with higher self-efficacy and greater satisfaction with doctor-patient relationships. The researchers suggested that their findings have clinical implications for diabetes care because coping behavior (a key factor in their analysis) was linked to self-care but could also be influenced by the health care professionals involved in that care. However, little work has been carried out to try to implement the findings of coping research into clinical practice. Changes in clinical practice have usually involved behavioral interventions (task-oriented) rather than cognitive ones or have only included coping implicitly rather than explicitly. Diabetes-related distress may also affect self-care behavior as demonstrated when the Problem Areas in Diabetes (PAID) scale was developed. The scale covers negative emotions related to living with diabetes, for example, "feeling alone with diabetes" and "worrying about the future and the possibility of serious complications." Although often associated with depression, diabetes-related distress has been found to be predictive of diabetes self-care behavior as well as blood glucose control.⁶⁰

Depression and diabetes-related distress can occur together and can have serious implications for the management of diabetes, because those affected may feel unable or unmotivated to carry out self-care behaviors such as blood glucose testing or healthy eating. A different group of researchers in the United States has identified both diabetes-related stress and other stressors as important in predicting self-care behavior. If study participants reported, "My life is out of control because of my diabetes" or "I have other problems more serious than diabetes," they were less likely to report attention to diet or exercise as part of their diabetes self-care.

In summary, studies strongly suggest that stressful experiences have an impact on diabetes self-care behavior; however, there are many different factors that may mediate this relationship. Before turning to the implications for clinical practice, we consider the physiological mechanisms behind stress and its impact on diabetes.

3.8.10. Physiological Mechanisms Behind Stressful Experiences

Any stressful event might be judged by people in different ways, based on factors such as previous experience, psychological factors, and social influences. An event that is seen by one individual as particularly threatening might be seen as totally harmless by another individual. However, when a situation is regarded as threatening, that is, seen as having the potential to cause harm to the individual, a specific pattern of physiological responses is elicited, known as the stress response or "fight/flight" response. This pattern of responses has developed as a result of human evolution and is aimed at priming the individual for action, so that the situation can be dealt with by either fighting or fleeing the threat. The actions initiated by the central nervous system in response to a threat affect the entire body and are associated with three different bodily systems: the autonomic nervous system, the neuroendocrine system, and the immune system⁶¹. The autonomic nervous system is concerned with the regulation of smooth muscle, cardiac muscle, and glands and regulates the functions over which there is no conscious control, such as cardiovascular function, digestion, and metabolism. It consists of two distinct branches: the

parasympathetic and the sympathetic nervous system, the latter being the most dominant in times of stress. The sympathetic system is involved with the preparation of the body for action. It increases oxygen and nutrient supplies to the muscles by increasing the blood flow to the skeletal muscles and freeing glucose and lipids from its stores. It also prepares the immune system to deal with possible injury. With regard to the effects of stress on the neuroendocrine system, the HPA axis is of considerable importance. Upon encountering a threat or a stressor, the hypothalamus secretes corticotropin-releasing factor, which causes the release of adrenocorticotropin. This in turn travels to the adrenal cortex, where it leads to the secretion of glucocorticoid hormones, in particular cortisol.

Cortisol exerts considerable influence over bodily functions, both when the body is at rest and during stress. In normal circumstances, it is secreted according to a circadian (daily) rhythm, with cortisol levels highest in the morning and lowest in the evening. However, exposures to stress stimulate the HPA axis to release additional amounts of cortisol to maintain homeostasis and reduce the effects of stress. Cortisol influences a wide range of processes, including the breakdown of carbohydrates, lipids, and proteins to provide the body with energy. It also has an effect on bone and cell growth and may modulate salt and water balance.

Cortisol has an immunosuppressive effect and therefore plays a role in the regulation of immune and inflammatory processes. That the central nervous system communicates with and exerts an influence on the immune system is now well established; brain lesions can alter a variety of immune measures, and both the autonomic and the neuroendocrine system have been shown to influence the state of the immune system. Because both the neuroendocrine and the autonomic system are influenced by psychosocial factors, it follows that the immune system is also affected by such factors, although the precise nature of these complex interactions remains to be determined. Although there is still much unknown about the effects of acute stress on the immune system and studies have been limited in the number of immune parameters studied, one review revealed that stress influences both circulating cell numbers and the function of immune cells. The cells generating the immune defense are generally known as white blood cells and consist of several subgroups including the lymphocytes (T-cells, B-cells, and natural killer cells), which have received much attention in stress research. Although research into the effects of stress continues, it seems clear that there is a range of responses to stressful experiences, both physiological and behavioral/emotional. The final section of this article focuses on the implications of stress research for practice in the care of individuals with diabetes.⁶²

3.8.11. The Deadly Combination of Diabetes and Depression

A study published in the June 2005 issue of the ADA journal, *Diabetes Care*, examined the long-term effects of diabetes and depression on mortality. More than 10,000 adults were studied. The study participants fell into one of four groups, those who had: 1) neither diabetes nor depression, 2) depression but no diabetes, 3) diabetes but no depression, or 4) both diabetes and depression. After 8 years, the researchers examined how many people in each of the four groups had died. The results showed that, whether you had diabetes or not, depression raised the risk of mortality over the 8-year period. Diabetes, whether you were depressed or not, also raised the risk of mortality. Most importantly, it was the combination of depression and diabetes that raised the risk the most. People who had diabetes and were significantly depressed were 2½ times more likely to die over the 8-year period compared to people who had neither condition. Comparing people with diabetes who were depressed vs. people with diabetes who were not depressed, the researchers reported that depression increased the risk of mortality by 30%. *BDI Connection e-Newsletter*, Number 1, September 2005⁶³

3.8.12. Coping with Stress

In general one can cope with stress by focusing either on the emotional effects of stress or solving the problems of stress, or both. In emotion focused coping, cognitive changes are affected that lead to viewing stressful situations as less stressful; i.e. the situations themselves are unchanged, only the emotional response is sought to be altered. Denial or defense mechanisms are commonly employed methods and they may be useful for a certain period. Ultimately it often becomes necessary and effective to confront and change the stressors directly. In problem focused coping, one acquires problem solving skills to directly deal with the stressor and change it. The latter may not always be possible, e.g. emotion focused coping may be required in situations such as end-stage renal disease or other conditions.⁶⁴

3.8.13. Resources for Coping

Positive Beliefs: A positive self-image and a positive attitude are especially significant coping resources. Such positive belief can come from a belief in oneself or in others. People who feel they have an internal locus of control, i.e. a feeling they have significant control over the events in their lives, tend to cope better than those who feel they have no control. Social skills: The better one's social skills, the less the stress. Social skills also help in communicating one's need, to enlist help and to decrease hostility. Social support is an important coping resource, be it from families, friends or social organizations such as diabetes self care groups. Support groups help not only by providing other people for relief, but also because it is possible to learn techniques for coping from others in similar situations. Material resources are invaluable in coping with stress. In facing minor or major stresses, people with money who can use it effectively generally fare better and experience less stress than those without money.

3.9 Psychosomatic Disorders

The PSYCHOSOMATIC concept

Psychosomatic literally means "mind-body". It is used to describe the effect of mind on body and body on mind. The term psychosomatic is derived from the Greek words, psyche and soma. Psyche in ancient times meant soul or mind and more recently has come to mean behavior. Soma typically refers to the physical organism-the body. Therefore it indicates relationships between psychological processes or behavior on one hand and somatic structures or body organs on the other.⁶⁵

Historical perspectives

In the primitive society (10,000 BC) disease was considered to be caused by spiritual powers and must be fought by spiritual means. After hundred years of religious dominance, interest in natural disease and cures or diseases returned. During the renaissance (1440-1650), the study of material world renewed itself. Descartes (1596-1650) propounded that soul and body interact at pineal gland. Morgagni (1682-1771) showed that disturbed organ could cause disease and death. He correlated disease symptoms with disturbances of organs and so proved that an imbalance of body fluids could not be cause of illness. On the other hand, Descartes' idea of the reality of subjective viwere to the extreme was carried by Berkeley (1685-1753) and other idealist concluded that only the self exists and all reality is subjective. The materialists came to opposing conclusion, that only matter exists that that it generates all that is called spirit.

La Metterie believed that all thought was the result of the mechanical action of brain. Cannabis concluded that just as liver secretes bile, the brain secretes thoughts. Cobb took the mind to be the functioning of the brain, though he did not consider it a strictly materialistic opinion, Leibnitz believed that the irreducible entity of monad was itself neither material nor immaterial but that the material and immaterial systems of a human being run parallel courses that did not interact.

Modern concepts

In the nineteenth century, the mind-body schism spread to its farthest division. It was Freud (1856-1939) who brought psyche and soma together. He demonstrated the importance of emotions in producing both mental disturbances and somatic disorders. His early psychoanalytic formulations detailed the role of psychic determinism in somatic conversion reactions. Freud re-dignified the study of the emotions as separate study and pointed to their relationship between the soma in the new field of psychiatry.

Physicians have always been concerned with the relevance of the body-mind relationship to clinical practice and have been aware of the far reaching effects of disturbance of the one on the other.

Etiological concepts were expanded when it was recognized that the disturbed function might be the cause of altered structure. Many chronic disturbances are not caused primarily by external mechanical or chemical factors or bacteria but by the continuous functional stress arising during the every day life of the individual in his struggle for existence. Fear, aggression, guilt and sexual tension, if repressed, result in permanent chronic emotional tensions which disturb the functions of the vegetative organs. For example, the inhibited rage seems to have a specific relationship to the cardiovascular system. Thus the psychosomatic approach brings internal physiological processes into synthesis with the individual's relations to the social environment. The term psychosomatic designates merely a method of approach both in research and in therapy which is aimed toward the simultaneous and coordinated use of somatic and psychological methods and concepts.

The term psychosomatic was coined by Heinroth in 1818 to emphasize that both mind and body are important in medicine, but was popularized after the First World War. Many pioneers of the psychosomatic movement left Europe in 1930 to work in the USA where they developed their ideas with American psychiatrists such as Flanders, Dunbar. After second world war Britain became an important center for psychosomatic research and practice. Later psychiatrists in all parts of the world were influenced by the new knowledge, in the 1930s there was much discussion on the nature and classification of psychologically determined physical disturbances and particularly on the differentiation between the symptoms of conversion hysteria and somatic manifestations of emotional distress. At that time, the term psychosomatic disorder was introduced in relation to a small number of physical diseases where emotional conflict appeared to be aetiologically important.

All illnesses can be considered to be psychosomatic. That is, they inevitably involve the mind's reaction (psyche) to a physical (soma) illness. However, in some illnesses, psychological factors seem to play a particularly important part. They can influence not only the cause of the illness, but can also worsen the symptoms and affect the course of the disorder. It is these illnesses that are termed psychosomatic disorders. PSYCHOSOMATIC MEDICINE is based on the observation that psychological factors and social cultural stresses may play a role in the predisposition, onset, course, and response to treatment of some physiological changes and biomedical disorders.

There is well established that both the mind and body may have significant influences on one another, and somato-psyche (bodily effects on mental functions) are at least as prominent and should receive equal attention as psychosomatic factors. Psychological and Socio-cultural factors may play a predisposing, initiating, or sustaining

role in many disorders. Therefore, if a broad definition of psychosomatic is used, terming specific illness as psychosomatic and others as not is not wise, since the effect of psychosocial factors on biomedical illness is primarily a matter of degrees. There are three factors which must be present simultaneously for a person to develop a psychosomatic disorder, if the term is utilized in a more focused and specific sense.

1. The individual must have a biological predisposition to the particular biomedical disorder. The diathesis may be genetically determined, secondary to trauma or other pathologic processes or due to environmental insults or damaging personal habits.
2. The individual must have a personality vulnerability-there must be a type or degree of stress that individual's psychological defense mechanisms and personality structure cannot manage or contain.
3. The individual must experience a significant psychosocial stress in his susceptible personality area.

3.9.1. Classification

The revised fourth edition of Diagnostic and statistical manual of mental diseases (DSM-IV-TR) does not use the term psychosomatic. Instead it describes psychological factors affecting medical conditions as one or more psychological or behavioural problems that adversely and significantly effect the course or outcome of a general medical condition, or that significantly increase a person's risk of an adverse outcome". Criteria in 10th revision of International classification of diseases and related health problems (ICD-10) are more general. This is classed under Diagnostic criteria for psychological and Behavioral factors associated with Disorders or Diseases classified else where. DSM-IV-R Diagnostic criteria for 316 ...[Specified Psychological Factor] Affecting...[Indicate the General Medical Condition]

A. A general medical condition (coded on **Axis III**) is present.

B. Psychological factors adversely affect the general medical condition in one of the following ways:

(1) The factors have influenced the course of the general medical condition as shown by a close temporal association between the psychological factors and the development or exacerbation of, or delayed recovery from, the general medical condition

(2) The factors interfere with the treatment of the general medical condition

(3) The factors constitute additional health risks for the individual

(4) Stress-related physiological responses precipitate or exacerbate symptoms of the general medical condition

Choose name based on the nature of the psychological factors (if more than one factor is present, indicate the most prominent):

Mental Disorder Affecting...[Indicate the General Medical Condition] (e.g., an Axis I disorder such as Major Depressive Disorder delaying recovery from a myocardial infarction)

Psychological Symptoms Affecting...[Indicate the General Medical Condition] (e.g., depressive symptoms delaying recovery from surgery; anxiety exacerbating asthma)

Personality Traits or Coping Style Affecting...[Indicate the General Medical Condition] (e.g., pathological denial of the need for surgery in a patient with cancer; hostile, pressured behavior contributing to cardiovascular disease)

Maladaptive Health Behaviors Affecting...[Indicate the General Medical Condition] (e.g., overeating; lack of exercise; unsafe sex)

Stress-Related Physiological Response Affecting...[Indicate the General Medical Condition] (e.g., stress-related exacerbations of ulcer, hypertension, arrhythmia, or tension headache)

Other or Unspecified Psychological Factors Affecting...[Indicate the General Medical Condition] (e.g., interpersonal, cultural, or religious factors)⁶⁶

HOMOEOPATHIC CONCEPT OF PSYCHOLOGICAL FACTORS AFFECTING MEDICAL CONDITION

Homoeopathy, as well as modern medical thought, supports of view that psychological factors are important in all disease. Whether the role is initiation, progression, aggravation, or exacerbation of a disease is open to debate and varies from disorder to disorder.

In 6th edition of Organon Hahnemann, shows the influence and the repercussion of the psychic on the physical in his note to the paragraph 17 where he writes:

"Most severe disease may be produced by sufficient disturbance of the vital force through the imagination and also cured by the same means."

"A warning dream, a superstitious fancy, or a solemn prediction that death would occur at certain day or at a certain hour, has not unfrequently produced all signs of commencing and increasing disease, of approaching death and death itself at the hour announced, which could not happen without the simultaneous production of the inward change (corresponding to the state observed externally); and hence in such cases all the morbid signs indicative of approaching death have frequently been dissipated by an identical cause, by some cunning deception or persuasion to belief in the contrary, and health suddenly, restored, which could not have happened without removal, by means of this moral remedy, of the internal and external morbid change that threatened death."

In the APHORISM 215 he makes the following observations:

Almost all the so - called mental and emotional diseases are nothing more than corporeal diseases in which the symptom of the derangement of the mind and disposition peculiar to each of them is increased, whilst the corporeal symptoms decline (more or less rapidly), till it at length attains the most striking one - sidedness, almost as though it were a local disease in the invisible subtle organ of the mind or disposition. It clearly concedes the psychosomatic ailments of the modern authors. In 1810 Hahnemann was already insisting on the essential relationship between physical and the psychic condition of the patient. In the paragraph 210 of the organon he writes:

"..... They do not, however, constitute a class of disease sharply separated from all others, since in all other so - called corporeal diseases the condition of the disposition and mind is always altered...."

And in paragraph 213:

"We shall, therefore, never be able to cure conformably to nature - that is to say, homoeopathically - if we do not, in every case of disease, even in such as are acute, observe, along with the other symptoms, those relating to the changes in the state of the mind and disposition, and if we do not select, for the patient's relief, from among the medicines a disease - force which, in addition to the similarity of its other symptoms to those of the disease, is also capable of producing a similar state of the disposition"⁷⁹.

4.1 MIASMATIC BACKGROUND OF DIABETES

Predisposition to beta cell destruction is the main causative factor in primary Idiopathic diabetes mellitus. The presence of this predisposition corroborates with the basic conception in homeopathy about the nature and origin of all Chronic diseases. We must keep in mind Dr. Kent's 7th observation in this context. He arrived at the observation by treating several such incurable patients. Homoeopathically, Non-insulin dependent Diabetes Mellitus is either due to Psora or Sycosis or both. *Psora leads to functional deficiency, Sycosis leads to incoordination* and a combination of the two leads to simultaneous presence of both the conditions. These cases do not generally require exogenous insulin but if persist for a long time, being maltreated or non treated, cellular destruction may finally take place and non-Insulin dependent cases may turn to be Insulin dependent. Until then, the prognosis of these cases is better and the patients may be cured as the condition is *reversible*. But the medicinal treatment must be *supplemented by proper dietetic management and physical exercise* to reduce obesity. Diet control and adequate physical exercise alone may help to control the progress of these conditions. If obesity is reduced, Insulin receptor becomes much more effective and the Insulin resistant state steadily improves resulting in normalcy of blood sugar level. But unless the predisposition is corrected by anti-miasmatic constitutional treatment the patient will not be cured and slightest error in diet and regimen will lead to a relapse of the condition⁶⁷.

4.1.1 Secondary Diabetes Mellitus

Secondary Diabetes Mellitus is *associated with* pancreatic diseases. Adrenal tumors, Thyrotoxicosis, Cushing's syndrome, administration of drug like cortisone, Genet disorders, defects of Insulin receptor and many other chronic degenerative disorders. In fact, secondary Diabetes Mellitus should not at all be considered as Diabetes but one of the *resultant effects of some primary degenerative condition elsewhere*. In homoeopathy we believe these degenerative conditions to be the resultant effects of a maltreated, untreated or suppressed miasmatic state,—the Psora, Sycosis, Syphilis or a combination of two or three of them. *Suppression of Skin diseases, Rheumatic affections, superficial ulcers*, repeated acute diseases in childhood, various early manifestations of miasmatic chronic diseases may finally result in destruction or *degenerative changes anywhere in the system*, being considered as different nosological entities. But they are nothing but the different manifestations of a single or complex miasmatic state. Naturally in secondary Diabetes Mellitus we observe a *relative deficiency of Insulin* in most cases and very rarely an absolute deficiency when the degenerative process reaches its acme, causing complete destruction of Beta cells. The diagnosis of the miasmatic background responsible for such degeneration depends on a careful anamnesis of the family history, past history, personal history and the evolution of symptomatology of the patients. Unless the patients advance to the state of absolute deficiency, they may be completely cured, if the primary miasmatic dyscrasia is eradicated by constitutional anti miasmatic treatment.

4.2. HOMOEOPATHIC MANAGEMENT OF DIABETES

Kent, the master homoeopath, said a century ago what modern medicine claims to have discovered recently. He said: "The person who says diabetes disease is insane in medicine, talk of a species of diabetes." Each patient has a specific constitution. By this, we mean that each person has a specific individual body, mind and disease. That is why different

people get diabetes at different times, of differing severity, with different complications, and with varying response to the same treatment. In Homoeopathy, we try to find a medicine to suit the mental disposition, the physical attributes, as well as the various complications of the patient. It has been repeatedly verified that stress is a primary contributor to the diabetic process. If stress is removed, there is a false perception of reality, that is what we call disease. Each diabetic has his own individual disease state that is aggravating his diabetes, besides other things.

The homoeopath aims to identify the disease state and treat it with the similar remedy. When the disease state is removed, the diabetes loses its grip like a creeper without a stick. Now let us see what should be our approach as homoeopaths in treating a case of Diabetes Mellitus. If a case of Diabetes Mellitus comes to us for treatment, our first duty is to decide whether the case is *curable or not*. This may generally be assessed from the aetio-pathology of the condition, the family history, the age of onset, the severity of the present condition, the assessment of renal function and the presence of complications. But the final prognosis of course depends on observing the effect of the well-indicated medicine administered. Suppose a patient who has been suffering from *Insulin dependent Diabetes Mellitus* (being all along under allopathic treatment) comes to us for consultation. From what has been discussed so far, it is probably clear that this case is incurable and requires regular doses of Insulin for survival. Naturally, we are to clearly explain the patient as to why his/her condition is incurable and that the scope of medicine is very much limited here. Still the patient may avail the benefit of constitutional treatment along with insulin therapy in order to avoid serious complications and for a better future. If the Beta cells are not completely destroyed and at least some healthy cells are there to function, then constitutional treatment may check further destruction of cells and the remaining healthy cells may start functioning much more efficiently secreting more Insulin to compensate. Naturally there is every possibility that the dose of Insulin may be reduced in some cases. But this must be done in consultation with the attending allopathic physician and that also gradually.

In *Non-Insulin Dependent Diabetes Mellitus* the situation is altogether different. In such cases oral hypoglycaemic drugs may be stopped from the very beginning of homoeopathic treatment. Because, homoeopathic constitutional medicines may perform the function of oral hypoglycaemic drugs by correcting the miasmatic dyscrasia, checking destruction of cells as also by *stimulating cells to act much more efficiently*. This, at the same time, may help in reducing obesity *supported by adequate exercise and dietetic control*. Thus the Insulin resistant state or the defect in Insulin receptor may be corrected and the patient may be completely cured of Diabetes Mellitus. In these cases we should not be afraid of withdrawing allopathic medicines straightway.

Our next duty is to consider about the diet of the patient suffering from Diabetes Mellitus. Constitutional treatment may fail to produce any desired result *unless proper dietetic measures are strictly followed*. As homoeopaths our aim should not be to bring down the blood sugar level. Potentised homoeopathic medicines act dynamically and not physiologically. Hence, any attempt to lower down the blood sugar level by potentised medicines is bound to be futile. Medicines given in mother tinctures like *Sygzium jambo.*, *Cephalandra Indica* etc. may lower down the blood sugar temporarily because they act partly physiologically. But their administration without any symptom similarly is not the principal of homoeopathy and the quantity in which they are used is not sufficiently physiological to produce any lasting and tangible effect in the system. Our principle is to select constitutional medicine covering the totality of symptoms of the patient *including the miasmatic background*. We are *to take care of the fundamental cause and the disease process* and not the ultimates of the disease *The environmental causes* must at the same time be *taken proper care of*⁶⁸.

4.2.1. Dietary management

In all chronic patients, besides the actual medicines of all kinds, the diet and regimen that is to be avoided are explained in detail in aphorisms 259,260 and 261.

Dose

The dose of a medicine whose selection has been accurately homoeopathic must be reduced to the degree of minuteness appropriate for a gentle remedial effect. According to Hahnemann, pure experiment, careful observation and accurate experience can alone determine the degree of minuteness necessary to affect the best cure

in	a	given	case.
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Repetition Of Doses-

The only axiom for repetition is to repeat when the original symptoms reappear or when improvement ceases. Potency- In general it may be stated that any curable diseases may be cured by any potency, when the indicated remedy is administered, but that the cure may be much accelerated by selecting the potency appropriate to the individual

Diagnosis

Diabetes mellitus is usually based on the basis of clinical history and lab examination of plasma glucose concentration. FPG ≥ 126 mg/dl (7.0 mmol/l). Fasting is defined as no caloric intake for at least 8 h. is identified as Diagnostic.

4.2.2. General management

It is true that high carbohydrate diet does not produce Diabetes Mellitus, but there cannot be any doubt that the *state of hyper glycaemia will be aggravated further by high carbohydrate diet* with the consequent effects and complications of hyperglycaemia including coma and sudden death. The basic principle of diet is *low caloric and high residual diet*. The total caloric requirement is to be decided first and then the percentage of protein, fat and carbohydrate. The standard caloric requirement is 30 K. Cal. per Kg of body weight with slight variations depending on the individual physical activity of the patient.

The protein constituent should consist of 15 to 20% of the total requirement, the fat 25 to 30% and the carbohydrate 50 to 80%. As regards the articles of diet, the economic condition of the patient, the food habit of the patient and the availability of the diet recommended must be considered in all cases. A routine diet chart for all classes of patients may not be of any help. Green vegetables like carrot, spinach, peas, tomatoes etc. are to be recommended in all cases specially in the form of salads to facilitate high residual but low caloric diet. In recommending diet we must not forget that our aim should be *to stop weight loss in patients who are progressively emaciating* and to *encourage weight loss in patients who are becoming progressively obese*. Next to diet most important factor is exercise. *Adequate physical exercise in the form of jogging, walking, yogasanas etc.* are essential in obese Diabetes Mellitus. On the other hand *exercise and over exertion are contraindicated in progressively emaciating patients*. In maturity onset Non-Insulin dependent Diabetes Mellitus obesity is a common associated feature and unless the patient takes part in active exercise to reduce obesity, medicines, even well selected, may not be of much help to the patient.

The other factors involving the general management consist of the following;

- a) Personal cleanliness:** to avoid complications, personal hygiene is to be well cared for. Due to hyperglycaemia, tissues are liable to easy infection and the patient frequently suffers from pruritus, boils carbuncle etc. which may be avoided to a great extent by observing cleanliness.
- b) To avoid mental worries and anxieties** as far as possible. Stress and strain aggravate hyperglycaemia by harmonic stimulation. As such, these are to be avoided.
- c) To avoid alcohol:** alcohol increases blood sugar as also it affects the liver. Hence it is to be avoided to prevent complication.
- d) Foot care:** Any injury to foot in a diabetic patient may lead to ulceration or even gangrene or there may be great delay in healing even a minor abrasion or wound due to sugar retention in tissues and consequent pyogenic infection especially staphylococcal. Hence; the patient should not walk bare-footed; must maintain cleanliness of feet and must always try to avoid trauma to feet as far as possible.
- e) Moderation in all spheres of life:** Night watching, excessive smoking, sexual excesses, attending invitation party etc. are all harmful to diabetic patients. As such the patient must be advised to adopt moderation in all spheres.
- f) Adoptability:** A diabetic patient must know how to live a more or less normal life with his diabetes even if persisting all through his/her life. In fact the patient should be a physician to himself/herself. The patient must not be panicky or despair- red of recovery of the illness, but at the same time he/she must not be over confident or careless about his/her illness.
- g) Regular Check-up of blood sugar:** the patient should check his/her post prandial blood sugar (2 hours after meal) regularly for a few years even if it is normal after few months of treatment. This will help a homoeopath to understand whether the miasmatic dyscrasia has been completely eradicated and if continuation of constitutional treatment is still necessary
- h) Regular Urine examination** is necessary to assess renal function and early detection of complication if any.
- i) Occasional blood examination** for cholesterol is necessary for early detection of the possibility of atherosclerosis.
- j) Occasional visits to Ophthalmologist and Cardiologist** for early detection of eye and cardio-vascular complications and to take necessary care there of.

4.3 HOMOEOPATHIC THERAPUTICS

Abroma augusta It is an excellent remedy where the quantity of sugar is excessive and the urine is loaded with high specific gravity: Patient passes large quantity of clear urine at night, excessive thirst, insomnia and prostration are other marked features. Patient is averse to do any physical or mental labor. (Homoeopathic Treatment of Diabetes Mellitus-Kansal Kamal⁶⁹)

Acetic acid

Abundant sugar in urine, increased and light-colored, great thirst, but cold drink lies heavy on stomach; ascites and hydrothorax, oedema pedum; gangrenous ulcers; pale, waxen skin; extreme prostration; decomposition of animal matter. (S.LILIETHAL, Homoeopathic Therapeutics)⁷²

Argentum met

Profuse, turbid, sweetish urine; aggravation at night, sometimes like whey, it distresses him at night, has to rise so often; emaciation and great weakness; face pale and sallow, scrotum and feet oedematous and itching; foetid taste in mouth; disposition to gangrene. (S.LILIETHAL, Homoeopathic Therapeutics)

Argentum nitricum Diabetes of nervous and gastric origin in patients who are mentally and physically exhausted. Dried emaciated persons due to long continued mental work. Craving for sweets. Nervous, impulsive, want to do things in a hurry. Intolerance of heat. Dyspepsia, belching accompanies all gastric complaints. Profuse urination, impotence, irregular blotches over the skin. (Homoeopathic Treatment of Diabetes Mellitus-Kansal Kamal)

Arnica montana Arnica montana causes dryness in the mouth, with considerable thirst; a frequent desire to urinate, with a copious emission of pale urine; deglutition is prevented from an inordinate dryness of the mouth. Diabetes has been known to follow a blow on the liver. The Arnica, in such case, would be a suitable remedy. (W.Morgan-Diabetes⁷⁰)

Arsenicum album Hepatic origin of diabetes due to chronic alcoholism. Horrible thirst, emaciation and exhaustion with hallucination, tendency to boils, pruritus vulvae skin dry, emaciation and great mental and physical restlessness. Diabetic gangrene. Neuropathy – The patient complains of burning pains especially in the palms and soles which are better by heat. Neuralgias and multiple neuritis. Also useful in carbuncles and gangrene with cadaverous odor and burning pains which are better by heat. Ulcers on toes and soles with wooden feeling in soles. Dry rough scaly skin. Chilly patients. Dry tongue and mouth with intense thirst but can only drink a little at a time. Increased quantity of urine with great burning while passing urine. Urine contains albumin. (PAVRI KERI R.S,Essentials of Diabetes Mellitus and Its Treatment by Homoeopathy⁷¹)

Arsenicum bromatum Dr Anshutz recommends it in both Diabetes mellitus and Diabetes insipidus, with loss of weight, burning thirst. Mixed diet with meat is recommended.(Mathur)

Berberis vulg

Constant urging, with pain in neck of bladder, urine very slow to flow, with pain in lumbar and renal region, amelioration by rest; after urinating sensation in bladder as if one must go again soon or as if some urine remained behind; pale-yellow urine, with a gelatinous sediment; weakness of sexual organs; pale, sallow face, sunken cheeks; sickly expression; dryness and sticky feeling in mouth and fauces; sticky, frothy saliva, like cotton; increased thirst and appetite, amelioration by eating; pulse slow and weak; paralyzed, bruised sensation in back, aggravation from slight exertion; skin sticky and scaling off; intense coldness of knees. (S.LILIETHAL)

Bovista

Frequent desire to urinate, even immediately after urination, with emission of a few drops; urine bright-red or yellowish-green, becomes turbid; bright yellow, with slowly forming cloud; turbid, like loam-water, with violet sediment; general languor and enervation, particularly in joints; visible palpitation after exertion, as if the heart were working in water; backache, with stiffness after stooping; urticaria. (S.LILIETHAL⁷²)

Bryonia alba No other remedy has such dryness of the lips as a symptom of hepatic disorders as Bryonia has, and this is often one of the first symptoms of diabetes. There is a persistent bitter taste in mouth. Patient is languid, morose, dispirited. Patient emaciates and may lose strength due to inability to eat. (Homoeopathic Treatment of Diabetes Mellitus-Kansal Kamal)

Calcarea phos

Glycosuria when lungs are implicated, diminishing the quantity of urine and lowering its specific gravity; sore aching in bladder, aggravation after urinating, involuntary sighing; chronic cough of consumptives, who suffer with cold feet; profuse sweat in phthisis. (S.LILIETHAL)

Carbolic acid

Short, dry, hacking cough; excessive urination, the urine containing sugar; copious flow of limpid, colorless urine; diarrhoea or torpor of intestines; unusual appetite and thirst for stimulants; languor and profound prostration; cold skin, horripilations; obesity or tendency to it. (S.LILIETHAL)

Cuprum met

Urine acid, straw-colored, turbid after standing, a reddish, thin sediment adhered to vessel, viscous, offensive, bloody, scanty or suppressed; great and slowly progressing emaciation; suppurating tuberculosis of lungs and evident signs of depression of brain; very great thirst; increased hunger; sweetish taste of mouth; increased urination, especially at night, dry, very infrequent stool; decrease of sexual desire. (S.LILIETHAL)

Cantharis vesicatoria The primary effect of Cantharis is to cause strangury; its secondary effect is that of a paralytic inability to retain the urine. This physiological fact suggests the use of Cantharides in both incontinence of urine and even Diabetes: it causes frequent and profuse micturition of a pale color, and white flocculent sediment; hence Cantharis is suitable in cases of Diabetes complicated with Albuminuria, etc. (W.Morgan)

Carbo vegetabilis Both Pulte and Laurie record this substance as a remedy in Diabetes. Hahnemann Gives the following pathogenetic effects: Great dryness in the mouth early when waking; excessive thirst and hunger; pain in the liver, as if bruised; frequent desire to urinate, with anxiety day and night; Diabetes; general physical depression; great lassitude and languor after a short walk, with attacks of sudden weakness and fainting. The moral symptoms are striking: there is an indescribable anguish; oppression of the chest; general uneasiness, impatience, and great irritability.(W.Morgan)

Carbolicum acidum Carbolic acid suits those patients who are broken down, prostrated. Septic conditions especially in persons who are somnolent, who easily fall asleep. Frequent urination at night more so in old patients. Urine contains albumin and ketones. Intense halitosis with dyspepsia or constipation. Great desire for stimulants. PAVRI KERI R.S⁷⁶

Causticum Diabetic neuropathy with paralysis and numbness. Cracking in knees. Stiff muscles with sensation as if the tendons were shortened. Paralysis ameliorated by cold applications even by cold drinks. Emaciation. Cataract(PAVRI KERI R.S)

Ceanothus americanus Diabetes in persons with splenic troubles, enlargement and pain in the left side of the abdomen which is worse lying on the left side. Green frothy urine which contains sugar. Diabetes complicated with hypertension (PAVRI KERI R.S)

Cephalandra indica It is a great homoeopathic drug for Diabetes mellitus, associated with biliousness, abscess, boils and carbuncles, profuse urination making the patient weak with dryness of mouth and considerable thirst, often worse after urination. The whole body "burns like fire", relieved by cold bathing.(Homoeopathic Treatment of Diabetes Mellitus-Kansal Kamal)

Chionanthus virginica Diabetes with liver disorder. (Dr George Royal⁷³)

Coca Diabetes mellitus with impotence. No appetite except for sweets. (Homoeopathic Treatment of Diabetes Mellitus-Kansal Kamal I)

Cuprum arsenicosum Diabetes with cramps in extremities, at night and Amel by heat. The profuse amount of urine suddenly becoming very scanty, drowsiness with tendency to coma. Varicose ulcers with tendency to become gangrenous. (G. Royal)

Curare Diabetes-Glycosuria with motor paralysis. (Boericke)⁷⁵ Ptosis of right side. Very distressing dyspnoea. Weakness of hands and fingers in pianists Reflexes diminished or abolished. (Diabetes Mellitus:Its Diagnosis and treatment :KN Mathur⁷⁴)

Fluoricum acidum This remedy is suited to chronic cases which demonstrate a syphilitic taint either in the past or the family history. Complaints of old age or of those who are prematurely old. Alcoholics. Hot patients whose discharge tend to be thin, foul and acid. This remedy is found more useful when the patient consults you with some full-down complication of Diabetes like ulcers (which have red edges). Redness of the palms. Diabetes with circulatory troubles of the lower extremities due to atony of the veins and the capillaries. (PAVRI KERI R.S)

Hepar sulphur The slightest contradiction makes him break out into the greatest violence, he could kill somebody without hesitation; sight gets dim when reading; heaviness and pressure in stomach after a moderate meal, unusual hunger, much thirst; desire for acids and wine; sexual desire increased, erections feeble; urine acid, burning, making the inner surface of the prepuce or of the pudenda sore and ulcerated; emission of much pale urine, with pressure on bladder; emission of pale, clear urine, which on standing becomes turbid, thick, and deposits a white sediment. (S.LILIETHAL)

Helleborus niger The symptomatology of this remedy corresponds to one of the acute medical emergencies – Diabetic Pre-coma. The patient is in a low state, he sees and hears imperfectly with generalised muscular weakness. This is most commonly due to hypoglycaemia either due to inadequate oral intake or an overdose of insulin or oral hypoglycaemic agents. The patient picks at his lips and clothes. There is a constant chewing motion of the jaw. Sighing respiration with slow, small, soft pulse. The patient shrieks and shouts; he cannot be fully aroused. Another characteristic symptom is that the patient greedily swallows water even when unconscious.(PAVRI KERI R.S)

Iris versicolor Diabetes in patients with a tendency to sick headaches which are associated with visual disturbances. Greasy taste in the mouth, with burning of the whole alimentary tract. Profuse clear urine with burning in the urethra after urination. Iris has a marked action on the pancreas. (PAVRI KERI R.S)

Glycerinum

Diabetes with profuse and frequent urination, increases specific gravity and sugar in urine, with emaciation and mental and physical debility. Influenzal pneumonia in diabetics. Irritating coryza. (Boericke and Anshutz) (Diabetes Mellitus:Its Diagnosis and treatment :KN Mathur)

Gymnema sylvestre It is confidently accepted as a sovereign remedy for diabetes by experienced homeopaths. The features are: urine is loaded with sugar, after passing urine patient feels extremely weak. Profuse urination, passes several times a day and in copious quantity. There is a burning all over the body, boils and carbuncle appearing anywhere on body. Drinks water often in large quantity. Sexual power gone or lost. Profuse urination after sexual intercourse. (Homoeopathic Treatment of Diabetes Mellitus-Kansal Kamal)

Helonias dioica Dull gloomy and irritable; melancholy; impotence; pain and lameness in the back; numb feet. 'Patient is better when kept busy'; Urine albuminous, phosphatic, profuse and clear, contains sugar. Used in tincture or 3rd potency. Constant aching and tenderness over kidneys.(Homoeopathic Treatment of Diabetes Mellitus-Kansal Kamal) Diabetes, first stages ; urine profuse, clear, contains sugar. Lips dry, stick together: great thirst, restlessness ; emaciation; irritable and melancholy. Diabetes with rheumatic symptoms. Albuminuria with great weakness(Mathur K.N Diabetes mellitus diagnosis and treatment)

Hepar

The slightest contradiction makes him break out into the greatest violence, he could kill somebody without hesitation; sight gets dim when reading; heaviness and pressure in stomach after a moderate meal, unusual hunger, much thirst; desire for acids and wine; sexual desire increased, erections feeble; urine acid, burning, making the inner surface of the prepuce or of the pudenda sore and ulcerated; emission of much pale urine, with pressure on bladder; emission of pale, clear urine, which on standing becomes turbid, thick, and deposits a white sediment. (S.LILIETHAL)

Insulin

Long before the discovery of Insulin Dr.Pierre Jousset of Paris prepared a pancreatic juice on a glycerine basis which he administered to diabetic patients in doses of 10 or 20 drops a day in water and had results sufficiently good to consider pancreatic juice, orally administered, as a remedy of great value in diabetes. Dr. Cartier, his practical successor, praised it insisted on smaller doses given by mouth as larger doses and hypodermic injections of it had no effect in ordinary diabetes. Baker advises the homoeopathic strengths of Insulin 3d to 30th and reports happy results therefrom. Great care must be taken not to overdose. Boericke says that it maintains the blood sugar at a normal level and the urine remains free of sugar. Epileptic convulsions and mental derangements have been produced by hypodermic use of this hormone.(W,DEWEY⁷⁷)

Iodium It shows almost all the classical symptoms of Diabetes, unquenchable thirst, voracious appetite with steadily increasing emaciation, hepatic and gastric troubles, increased urination, tendency to eruption and boils, great debility, slightest effort induces perspiration. Diabetes due to pancreatic diseases. Urine acid, thick, with cuticle on the surface. (Homoeopathic Treatment of Diabetes Mellitus-Kansal Kamal)

Kali brom

Emaciation, paleness, skin cold and dry, pulse rapid and feeble, tongue red and tender, gums spongy and bleeding; thirst excessive; appetite voracious; bowels constipated; urine pale, frequent, of great density, and loaded with sugar; liver tumid and tender. (S.LILIETHAL)

Kali mur

Excessive and sugary urine; itching in urethra; stomach and liver deranged; dry and light-colored stools; pain in kidneys; great weakness and somnolence. (S.LILIETHAL)

Kali phos

Nervous weakness; breath peculiar, or haylike odor; thirst, voracious hunger, emaciation; hepatic troubles. (S.LILIETHAL)

Kreosotum

Perfect depression of the trophic nervous system. Heaviness all over, with drowsiness; depressing of spirits; head feels confused and dull; dim-sightedness; flat, bitter taste; appetite, with sensation of fulness; intermittent, hard, dry stool; frequent and copious emission of hot, clear urine; bruised sensation in chest and all along the back; physical exhaustion, worse from rest; great itching of genitals during and after micturition. (S.LILIETHAL)

Lacticum acidum Diabetes due to gastro-hepatic disorders, copious urination, urine light yellow, thirst, nausea, weakness, voracious appetite and constipation, dry skin and tongue, also occasional gastralgia are the commonly presenting symptoms.(Kansal) .Diabetes with rheumatic symptoms. Tongue dry parched, voracious hunger Nausea better from eating. Rheumatic pains in joints, shoulders, wrist and knees with much weakness. (Boericke) (Diabetes Mellitus:Its Diagnosis and treatment :KN Mathur)

Lachesis

Despondency and peevishness; dimness of eyes; livid-gray complexion; readily bleeding gums; sweetish taste; constipation; violent urging to urinate, with copious discharge; impotence; difficult suffocative breathing; laming pain and weakness in back and extremities; gangrene; emaciation with muscular relaxation. (S.LILIETHAL)

Lacticum acidum An exceedingly good remedy in the gastro-hepatic variety of diabetes and good results often follow its use. It has a fine clinical record. The symptoms are : urinates copiously and freely, urine light yellow and saccharine, thirst, nausea, debility, voracious appetite and costive bowels. Dry skin, dry tongue, gastralgia (W.DEWEY)

Lac vaccinum defloratum

Excessive aching of back; enormous quantities of urine voided daily, with excessive lassitude and prostration; intense throbbing headache, especially in forehead, with nausea, vomiting and most obstinate constipation. (S.LILIENTHAL)

Lecithinum Urine contains phosphates, sugar and albumin. Brain fag, neurasthenia and insomnia, forgetful. Tired weak, short breath and loss of flesh. Male power enfeebled. (Boericke) (Diabetes Mellitus:Its Diagnosis and treatment :KN Mathur)

Lithium carb

Very frequent urination, disturbing sleep; turbid urine, with much mucous deposit; dark reddish-brown deposit in urine. (S.LILIENTHAL)

Lycopodium

Peevish and depressed in mind; thirst and hunger constant, but worse at night; flatulence; faeces small in quantity; want of natural warmth; sexual desire and power gone; lithic acid gravel; pulmonary phthisis, pituitosa and purulenta, with hectic; great emaciation; mental, nervous and bodily exhaustion; gouty lithaemia. Diabetes due to gastric disorder and hepatic diseases. Polyuria during night, Pruritus vulvae, numbness in limbs; hands and feet go numb with tearing pain in limbs especially while at rest (intermittent claudications). Skin prone to ulceration. (Homoeopathic Treatment of Diabetes Mellitus-Kansal Kamal)

Lycopus Virg

Diabetes mellitus and insipidus from some derangement of the central nervous system or sympathetic; morbus Basedowii; copious flow of clear urine of great density, containing sugar; intense thirst; great emaciation, etc. increased bronchial irritation, with sighing respiration; cardiac depression. (S.LILIENTHAL)

Magnesia sulph

Gloominess, especially mornings, and disinclination for work; mouth and throat very dry, as if numb, with a sweetish-bitter taste, in the morning, disappearing after breakfast; aversion to all food; slight thirst which can be resisted; urine copious, light-yellow, soon becomes turbid and deposits copious red sediment; erections without desire for an embrace; exhaustion and prostration, amelioration by rest momentarily. (S.LILIENTHAL)

Medorrhinum Clarke mentions Diabetes in the clinical symptomatology of this remedy. A family history of gonorrhoea, aggravation of all symptoms from sunrise to sunset, amelioration at the sea-shore, obstinate rheumatism (and sequelae of rheumatism), albuminuria, glandular enlargements, difficulty in mental concentration, impatience are the guiding symptoms to the use of this remedy in Diabetes. The patient cannot speak without crying, which ameliorates. Patient craves salt, sweets, ice, green fruits. Great thirst especially for alcoholic beverages. Itching of the body worse when thinking of it. Neuropathy, burning of the palms and the feet, which though cold to touch, are better when uncovered and fanned.(PAVRI KERI R.S)

Morphinum A most useful remedy for diabetic neuropathy. Intensely painful neuralgias better by hot applications. Multiple neuritis. Diabetic Pre-coma and Coma with very dry mouth and great thirst. Difficult swallowing from paralysis of the pharynx; better hot drinks, worse solids. Incessant, deathly nausea with vomiting. Diarrhoea or constipation with horrible tenesmus. Alternate tachycardia and bradycardia. Diaphragmatic paralysis. Melancholic delirium. Neuralgic pains cause twitching and jerking of limbs.(PAVRI KERI R.S)

Moschus

Unquenchable thirst; great emaciation; costiveness; impotence; frequent passage of large quantities of saccharine urine; paralytic condition of the brain; dimness of sight; earthy complexion; great dryness of the mouth and putrid taste; great thirst for stimulants and aversion to food; prickling in the skin; general exhaustion, with coldness all over. (S.LILIENTHAL)

Natrum sulph

Depressed, irritable, taciturn, tired of life; dulness in head and weakness of sight; dryness and burning in the eyes; nosebleed; dryness of mouth and throat; great thirst for very cold drinks; voracious appetite, with a boring pain; disgust while eating; foetid flatus; increased urination, especially at night; pains in small of back, with burning urine; aemoptoe; cough, with purulent expectoration. (S.LILIENTHAL)

Nitricum acidum Nitricum Acidum diminishes the secretion of urine, and moderates the thirst and heat in Diabetes. (W.Morgan-Diabetes)

Nux vomica

Good livers and sedentary habits. Acidity, with dyspeptic troubles; constriction of the throat; dry cough; pains in the back; numbness; parietic condition of the lower extremities; after ineffectual desire to urinate, frequent and more copious urination than could be expected from the quantity of liquid taken; sexual desire strong; spinal lesions exciting cause. (S.LILIETHAL)

Opium

After mental shocks or injuries. Dulness, sadness, weak memory; vision obscured as by a fog; face bloated, congested or sunken and pale; tongue thickly coated, dry; mouth and oesophagus dry; frothy sputa; ravenous hunger and unquenchable thirst; constipation more than diarrhoea; great pain and difficulty in expelling urine; no passage of urine or faeces; urine turbid, brown, with an iridescent film, scanty; weariness and numbness all over. (S.LILIETHAL)

Pancreatinum Diabetes with lientric diarrhea: Thin diabetics. Panereatic diabetes. (Dr Jousset)(Diabetes Mellitus:Its Diagnosis and treatment :KN Mathur)

Phosphoric acid

Neurogenic glycosuria. Debility from loss of animal fluids; bad effects from grief, anguish, sorrow and care; all the joints feel bruised; very sensitive to fresh air; lassitude and heaviness; weakness of mind; falling out of the hair; dimness of eyes; excessive thirst; eructations from acids; pressure in stomach; hard, difficult stool; shortness of breathing; urine thick, like milk (chyluria) or lime-water, with whitish curds, with stringy, bloody lumps, or clear, limpid, and containing much sugar; pain in back and kidneys; dull pressure in bladder; great weakness and emaciation; furunculosis. (S.LILIETHAL)

Phosphorus

Useful in diabetes and pancreatic diseases, especially in those of a tuberculous or gouty diathesis. The pancreatic involvement will call attention to Phosphorus Glycosuria, with phthisis; urine profuse, pale, watery; or turbid, whitish, like curdled milk, with brickdust sediment and variegated cuticle on surface; gouty diathesis; cerebral disease; cheesy degeneration of lungs. It should be remembered in Diabetes Mellitus, when it has been preceded or is accompanied by disease of the pancreas.(Farrington)

Picric acid

Cortex of brain congested; urine contains sugar and albumen, dark red, of high specific gravity; great indifference, lack of will power to do anything; eyes feel dry, as if full of sand, sight dim and confused; saliva white, frothy and stringy; disgust for food; very great thirst for cold water; great sexual desire with emissions; excessive languor and prostration, it seemed difficult to move the limbs; feet cold, chilly, cannot get warm, followed by clammy sweat; chilly all over, except head and spine; throbbing, jerking of muscles with great pains between hips. The urine has an abnormally high specific gravity and contains sugar; it is also albuminous. (Farrington E.A., Lesser Writings (with therapeutic Hints and some clinical cases)⁷⁸)

Plumbum

Lowness of spirits, anguish and melancholy; diminution of sight; dryness of mouth; dry, cracked tongue; feeling of contraction and constriction in throat; fever with unquenchable thirst; dingy color of skin; gangrene; constipation; hectic fever with dry, hacking cough from suppuration of lungs; great exhaustion; impotence; excessive emaciation; great hunger; obstinate belching and vomiting. Chronic lead-poisoning produces a perfect picture of glycosuria and of morbus Brightii, and Hering considered it one of the most important drugs in this form of disease. (S.LILIETHAL)

Podophyllum

Chalky stools; profuse and frequent micturition immediately after drinking; excessive hepatic action; hot, sour flatus. (S.LILIETHAL)

Ratanhia

Considerable emaciation and weakness; limbs sore and aching; great appetite; insatiable thirst and constant dryness of the mouth; gums livid and swollen; soreness in the kidneys; severe pains in small of back, improved by motion; hard stool, with straining; frequent urging to urinate, with scanty discharge, or passes large quantities of light-colored urine. (S.LILIETHAL)

Secale corn

Great general lassitude; heaviness of limbs; loss of strength; emaciation; gangrene; skin dry and withered; furuncles; petechiae; fever, with unquenchable thirst; diminished power of the senses; dryness of the mouth; morbidly great appetite; cardialgia; costiveness; diarrhoea; watery urine; increased quantity of urine. (S.LILIETHAL)

Senna Urine contains sugar, acetone, oxalates and phosphates. Prostration, fainting, constipation with colic and flatulence. Liver enlarged and tender. (Mathur)

Syzygium jambolanum It is capable of reducing the amount of sugar in the urine, especially when used in tincture form or very low trituration, great thirst, weakness, emaciation. Patient passes large quantity of urine of high specific gravity, old ulcers of skin: diabetic ulceration. (Homoeopathic Treatment of Diabetes Mellitus-Kansal Kamal)

Tarentula hispanica Copious urine with sugar. Diabetes with grief, anxiety, restlessness and weakness, with emaciation. Patients impulsive, violent, irritable, hysterical, feign sickness. Patients with increased sexual passion, intractable constipation. Patients, who feel sore all over the body, feel better from music, rubbing. Consolation causes weeping. (Kent). (Diabetes Mellitus:Its Diagnosis and treatment :KN Mathur)

Terebinthinae oleum Inability to concentrate the mind; dull, languid mind, relieved by frequent micturition; despondency; wearied of life; obscuration of sight; sunken features; lips cracked and slightly bleeding; epistaxis; spongy gums; tongue dry and red; foul breath; hunger and thirst, with debility; aversion to meat; rancid or acrid eructations; burning in stomach and hypochondria; tympanitis; albuminuria, with frequent micturition; sugar is noticed in urine after large doses of Ol Tereb (S.LILIETHAL)

Thuja occidentalis Glycosuria after a long-suppressed gonorrhoea, frequent desire to urinate day and night; craving alternates with want of appetite; longs for cold food and drink; urine contains sugar, foams, deposits a brown mucus; debility aggr. mornings. (S.LILIETHAL)

Uranium nitricum This remedy is praised highly by Hughes and others in diabetes originating in dyspepsia. It has polyuria, polydipsia, dryness of the mouth and skin. It causes sugar in the urine. Dr. Laning said that no remedy gives such universally good results; it lessens the sugar and quantity of the urine; he recommended the 3x trituration. It is when the disease is due to assimilative derangements that Uranium is the remedy, and symptoms such as defective digestion, languor, debility and much sugar in the urine, enormous appetite and thirst, yet the patient continues to emaciate. (W.DEWEY-Practical Homoeopathic Therapeutics) The patient, inspite of good appetite and thirst, emaciates. For all the complications of diabetes, such as diabetic nephropathy, degeneration of liver, high blood pressure and dropsy: unable to retain the urine without pain. Impotence. (Homoeopathic Treatment of Diabetes Mellitus-Kansal Kamal)

4.4 THERAPUTIC HINTS

Diabetes occurring in various types of constitutions:- Tubercular - Phos., Iod., Tuber. Leucophlegmatic - Calc. carb., Graph Psoric - Sulphur, Graph Sycotic - Thuja., Nat. Sulph. Syphilitic - Kali iod. Scrophulous, Lymphatic - Hepar sulph., Iod. Exciting or causative factors:- Due to digestive derangement - Uran. nit. Due to Gastro-Hepatic disease - Ars. Iod., Bry., Calc., Lact. acid, Lyco, Nux. vom., Uran. nit. Due to Nervous strain and stress - Ign., Phos. acid Due to Pancreatic Diseases - Iris v., Insulin, Pancreatin, Phos., Due to Injury or fall - Arnica Due to Syphilis - Kali. iod. Diabetes associated with:- associated with Debility - Acetic acid, China, Op., Phos. Acid, Phos., Pic. acid associated with Gangrene, Boils, Carbuncles - Ars. alb. associated with Diarrhea - Ars. alb. associated with Gouty symptoms - Lac. Acid, Nat. Sulph., Colch., Nux vom., Kali iod. associated with Impotency - Coca, Mosch. associated with Sexual and Urinary symptoms - Ars., Acon., Canth., Kali bich. Tereb., associated with Rheumatic symptoms - Lactic acid. associated with Melancholia - Helon. associated with Emaciation - Helon. associated with Thirst and restlessness - Hel. associated with Motor paralysis - Curare. associated with Rapid progress - Cur., Morph. associated with Ulceration - Syzyg. associated with Skin boils and furuncles - Arnica, Ars, Sili, Hepar associated with Restlessness and irritability of skin - Codeinum. associated with Swollen ankles - Arg. met. associated with Diabetic coma - Opium associated with Hepatic symptoms - Digit, Kali. bich, Merc., Iod., Hepar sulph. associated with Pulse affections - Phos., Ars. iod., Hepar.

Symptoms of diabetes (clinical):- a Polyuria - Acon., Alumina, Apis, Berb. vulg., Canth., chimaph., Equis., Helon., Kreosote, Lili. tig., Lyco., Nux.v. , Phos. acid., Sarsa., Scilla, Sepia, Sul., Uva.u. b. Nocturia - Aur. mur., Caust., Con., Ferr. Pic., Kali c., Kreo, Phos. acid, Puls., Sul. c. Thirst (Polydipsia) - Acetic acid, Acon., Bry., Lact. acid, Op., Phos., Sul. d. Appetite increased (Polyphagia) - Abrot., AlfaI.,Anac.,Iod.,Lyco.,Nat.m. ,Nux v., Phos.,Sulph.,Thy.,U. nit. - Yet loses flesh-abortion., - Acetic acid, Iod., Nat. Mur., Sul. e. Desire (Craving) for sweets - Arg. nit., Cina, Coca, Kali.C. , Lyco., Sul. f. Constipation - Bryonia, Cheli, Hydrastis, Nux vomica, Silicea, Calc. carb., Sulphur. Symptoms of complications:- a. Boils - Arnica, Anthracinum, Bellis,Calc. pic., Mer. Sol., Phos. acid., Sili., Sul., Tar.c. , Myristica seb. b. Gangrene - Ars., Carbo veg., Echinacea, Lach., Plumb., Secale, Sul. acid. c. Emaciation with increased appetite -Abrot, Iod., Nat. mur., Plumb. d. Artherosclerosis - All. sat., Ars. iod., Bar.c. , Glon., Kali iod., Plumb iod. e. Cataract - Caust., Cinararia, Silicea, Phos., Calc. flour. f. Paraesthesia (Numbness) - Gels., Sang. can. (in hands and feet) g. Diabetic nephropathy - Eel. serum. (K.W. Syndrome). If above medicines fails -Syzyg., Silicea. **Repertory of remedies** (Diabetes Mellitus:Its Diagnosis and treatment :KN Mathur)

Kent Sugar in urine : BOV, HELO N, LYCO, PHOS AC, PHOS, PLB, TARENT C, TEREB, URAN N. Acet ac, Arg m, Ars a, Benz ac, Cal c, Cal p, Carb ac, Carb v, Chel, Chin, Chin ars, Colch, Cup ars, Curare, Elaps, Ferr m, Hep s, Iris v,

Kali chlor, Kali phos, Kreos, Lac def, Lach Lac ac, Lesithin, Lycopus v, Lyss, Med, Nat s, Nit ac. Op, Petrol, Pic ac, Podo, Rat, Secal cor, Sil, Sulphur, Thuja, Zinc.

Boericke Diabetes- Acet ac, Adren, Arg m, Ars brom, Ars alb, Aur, Bor ac, Bov, Bryo, Capsicum, Cham, Chel, Chimaph, Chionanthus, Coca, Crot t, Cup ars, Cur Ferr iod, Fluor ac, Glon. Glycer, Helleb, Helon, Iod, Iris Kali acet, Kali brom, Kreos, Lach, Lact ac, Lecithin, Lyco, Lyssin, Morph, Moschus, Murex, Nat m, Nat sulph, nit ac; Nux v, Op, Pancreat, Phaseol. Phos ac, Phos, Phlorid, Picr ac. Plum Iod, Plumb, Pod, Rhus atomat, Sea cor, Si1, Sizyg J, Strych ars, Sulphur, Tarent h, Tarax, Terebenth, Uran n, Urea, Vanad.

Causes and Miasms Nervous origin-Ars, Aur mur, Calc c, Ign, Phos ac, Strych ars. Gastro-hepatic origin-Ars iod, Ars a, Bryo, Cal c, Cham, Chel, Kreos, Lact ac, Lept, Lyco, Nux vom, Uran n. Pancreatic origin-Iris, Pancreatin, Phos. Tubercular origin-Calc c, Cal p, Dros, Phos, Sep, Tub. Septic origin-Anthax, Cal s, Hep s, Kali s, Pyrog, Staphylococcin Streptococcin. Allergic origin-Ars a, Apis, Bov, Cal C, Carbo v, Graph, Lyco, Merc, Mez, Petrol, Psor, Rhus t, Sulphur. Endocrinic origin-Thyroid, Pituitary, Adrenal, Insulin

Concomitants - Anxious- Acon, Arg nit, Ars alb, Cal c, Caust, Kali ars, Kali c, Kali p, Lyco, Nat c, Nit ac, Psor, Sec cor, Sulph. Impatient-Acon, Cham, Ign, Sep, Sulph, Tarent H. Irritable-Ant cr, Apis, Ars a, Aur, Colo, Hepar, Lyco, Nat c, Nat m, Nit ac, Nux v, Phos, Sulph. Suicidal-Ant cr, Ars a, Aur, Ign, Nats, Nux v, Psor, Ver a. Nervous-Arg n, Ars a, Aur m, Gels, Helon, Ign, Kali phos, Lecithin, Nux v, Phos, Sep, Sil, Tar h. Restlessness-Acon, Ars a, Aur m, Cham, Ign, Iod, Med, Nat m, Nux v, Phos, Psor Pyrog, Rhus t, Sil, Tar h. Debility-Acet ac, Ars a, Arg n, Cal c, Cal p, Carb ac, Carbo v. Chin ars, Curare, Gels, Hellon, Iod, Kali p, Merc, Mur ac, Nat c, Nit ac, Phos ac, phos, picr ac, Stych ars, Zinc ars, Zinc picr. Pruritus-Agar, Alum, Ars a, Cal c, Conth, Carb ac, Crot t, Graph, Lyco, Kreo, Merc, Mez, Petrol, Psor, Rhus t, Sep, Sulph, Syzgium, Tar c. Impotence-Arg n, Graph, Lyco, Phos, Picr ac, Sele, Stych. Sterility-Aur m Nat, Aur m, Borax, Con, Graph. Hellon, Iod, Med, Nat c, Nat mur, Phos, Thyroid. Cramps-Colo, Cup, Cup ars, Kali p, Mag p, Nux v, Sulph.

4.5 REPERTORIAL SYMPTOMS RELATED TO DIABETES MELLITUS AND ITS COMPLICATION

Table 4.1. Common manifestation of diabetes

SYMPTOMS	CHAPTER WITH RUBRICS AND SUB-RUBRICS
Polydypsia	Stomach,Thirst,small quanitie, often
Polyphagia	Stomach,Appetite,ravenous,canine
Polyuria	Bladder,Urination,Frequent
Loss of Weight	Generalities,Emaciation

Table 4.2. Manifestations Of Diabetic Keto-Acidosis

SYMPTOMS	CHAPTER WITH RUBRICS AND SUB-RUBRICS
Anorexia	Stomach,Appetite,Wanting
Nausea	Stomach,Nausea
Vomiting	Stomach,Vomiting
Increased Urine formation	Urine,Copius
Altered Level of consciousness	Mind,confusion,intoxication,as if
Coma	Generalities,Faintness
Air hunger	Respiration,Gasping
Sweet odour from mouth	Mouth,Odour,Sweet
Lowering body temperature	Skin,coldness

Table 4.3. Manifestations Of Hyperosmolar Non-Ketotic Diabetic Coma

Unable to drink sufficient water	Stomach,Thirstless.desire to drink with
Altered Level of consciousness	Mind,Confusion,intoxication,as if
Convulsions	Generalities,convulsion
Transient Hemiplegia	Generalities,Paralysis,one sided

Table 4.4. Manifestation Of Hypoglycemia

Sweating	Perspiration, Profuse
Tremor	Gene,Trmbling,externally
Tachycardia	Generalities,Pulse ,frequent,Accelerated
Anxiety	Mind,Anxiety
Hunger	Stomach,Appetite,increased
Dizziness	Head,Intoxication,As from
Headache	Head,Pain,headache in general
Clouding of vision	Vision,Dim Vision,foggy

Blurred mental activity	Mind,Stupefaction
Confusion	Mind,Confusion
Abnormal Behaviour	Mind,Delusion,Hallucination,Imagination
Convulsions	Generalities,Convulsion
Loss of Consciousness	Generalities,Faintness

Table 4.5. Manifestations Of Lactic Acidosis

SYMPTOMS	CHAPTER
Nausea	Stomach,nausea
Vomiting	Stomach,Vomiting
Restlessness	Mind,Restlessness
Air hunger	Respiration,Gasping
Altered level of consciousness	Mind,confusion,intoxication as if
Stupour	Mind,unconsciousness
Coma	Generalities ,faintness

Table 4.6. Manifestations Of Diabetic Neuropathy

Numbness	Extremities,numbness
Tingling	Extremities,tingling,pricking
Pin and needle sensation	Extremities,pain,stiching foot and sole
Burning sensation	Burning,sole
Wrist drop	Paralysis,wrist
Foot drop	Paralysis,foot
Hyperesthesia	Extremities,sensitive,foot
Dysphagia	Throat,pain ,swallowing,on
Diarrhea after eating	Reactum,diarrhea,eating after
Rectal incontinence	Rectum ,invouluntary stool
Nocturnal sweating	Perspiration,profuse,night
Dependent part oedema	Extremities,swelling,lower limbs,dropsical
Small size of pupil	Eyes,pupils,contracted

Table 4.7. Manifestations Of Nephropathy

Nephromegaly	Kidney,swelling
Increased glomerular filtration	Urine,copious
Microproteinuria	Urine,cloudy
Macroproteinuria	Urine,albuminous

Table 4.8. Manifestations Of Retinopathy

Micro-aneurism	Eye,injected
Dilated veins	Eye,redness
Haemorrhage	Eye,ecchymosis
Cotton wool spots	Eye,spots,on cornea
Hard exudates	Eye,Discharges of mucous or pus
Retinal detachment	Eye,Detachment o,of retina
Corneal ulceration	Eye,ulceration,cornea

Table 4.9. Manifestations Of Dermatological Manifestations

Carbuncles(repeated tendency)	Skin,Eruptions,carbuncles
Furuncles(repeated tendency)	Skin,eruptions,boils
Delayed wound healing	Generalities,Wound,heal,slow to
Easy suppuration	Skin,Unhealthy Skin,Ulcers,suppurating

Table 4.10. Manifestations of Diabetic Foot

Moist gangrene	Skin,gangrene Extremities,Gangrene,Foot
Offensive discharge in gangrene	Skin,Ulcer,discharge,offensive
Easy Ulceration	Skin,Ulcers Unhealthy
Delayed wound healing	Generalities,Wound,heal,slow to
Easy suppuration	Skin,Unhealthy Skin,Ulcers,suppurating

CHAPTER-5

5.1 REPERTORY ON DIABETES MELLITUS AND ITS COMPLICATION

Diabetes is being a essentially a nosological diagnosis, is not always directly represented in the most repertories except in latest repertories. For eg; Kent's Repertory does not give a direct rubric Urine, Copious Urine, Sugar, Urine, Albuminous Urine, Specific gravity increased, Stomach Thirst Increased, Extremities Burning sensation for neuropathic symptoms etc.

Plan and construction

In this repertory the chapters are arranged in 27 chapters. The References are included from various repertories.

GRADATION OF MEDICINES

Three gradation are given

First grade - Roman letters EG:- Acet ac
Second grade - *Italics* EG:- *Uran nit.*
Third grade - BOLD CAPITAL EG: SULPH.

MIND:

Alcoholism - diabetes; with - med. nux-v.
Anxiety - Diabetes in: Nat s. phos.
Anxiety - Diabetes in - makes diabetes worse: Cod.
Coma - diabetes; in - alum. ars. carb-v. carb-n-o. cur. op.
Delusion - someone else; doing, by his side does all he is - diabetes in: Ars.
Dullness - diabetes, in - *Helon. Op.* ph-ac. sul-ac.
Dullness, sluggishness, difficulty in thinking and comprehending - in
diabetes: Acet ac. Helon. nat s. op. phos ac. phos. sulph ac.
Fear - Diabetes in: Cod. Nat s.
Fear - sudden - followed by - diabetes mellitus - op.
Gloomy - diabetes in: Helon.
Grief - diabetes; with - aur. aur-m-n. ign. mag-m. nat-s. ph-ac. tarent.
Irritability - diabetes, in - *Helon. Lycps-v. nux-v.*
Laziness - diabetes in: Lac ac.
Melancholy - diabetes in: Helon.
Memory - weakness of memory - diabetes; in - kali-br. lyc. nux-m. nux-v.
ph-ac.
Memory - impaired with - dryness of mouth - diabetes in: Kali br.
Prostration of mind, mental exhaustion, brain fag - diabetes in: Nat s.
Restlessness - diabetic - helon.
Restlessness - night at - Diabetes in: Lac ac.
Sadness - diabetes during - *Helon. Nat s. op.*
Shock - causes - diabetes: *Op.*
Temper - irritable - diabetes in: Nux v.
Thoughts - difficult - diabetes in: Nat s.

VERTIGO:

Fainting - ears - roaring - diabetes in : Uran n.

HEAD:

Brain - depression, cerebral (chlorosis) - diabetes in: Cupr m.

EYES

Circumorbital - circles - dark - diabetes in: *Uran n.*
Inflammation - Retina - diabetic - sec.
Retinitis - diabetes in: Crot h. phos. sec.

VISION

Sight - dim - diabetes in: Tarent.
Sight - dim - dull - diabetes in: Sul ac.
Sunken - eyes - diabetes in: *Uran n.*

HEARING

Illusion - roaring - fainting spells with - diabetes in: *Uran n.*

FACE:

Earthy - diabetes in: Arg n.

Emaciation – cheek bones prominent – diabetes in: *Uran n.*
Pale – face – diabetes in: *Arg m. Uran n.*
Red – deep – diabetes in: *Uran n.*
Sunken – Collapsed, Hippocratic, hollow – diabetes in: *Nat s.*
Swelling – bloated, icteric – diabetes in: *Chel.*
Dry – lips – diabetes in: *Ars*
Suppuration – of left – parotid gland – diabetes with: *Con.*
Suppuration – of left – parotid gland – with profuse sweat, disturbing
sleep - diabetes with: *Con.*

MOUTH

Bleeding –gums – diabetes in: *Kali br.*
Cold – tongue – diabetes in: *Uran nit.*
Clammy – mouth – diabetes in: *Uran nit.*
Dry–parched and sticky–tongue–diabetes in: *Lac-ac*
Dryness – mouth of – in diabetes: *Cur. rat.*
Dryness – mouth of – thirst with - in diabetes: *Uran nit.*
Dryness – mouth of – in diabetes - weak memory with: *Kali br.*
Dryness – mouth – bread, could not moisten least bit of – diabetes in: *Ars.*
Dryness – tongue – diabetes in: *Helon. lac ac.*
Red – bright too - tongue – diabetes in: *Nat s.*
Red – too – tongue – diabetes in: *Kali br. ran n.*
Retracted – gums – diabetes in: *Nat s.*
Spongy – gums – diabetes in: *Kali br.*
Swelling – gums – diabetes in: *Rat.*
Sensitive – tender tongue – diabetes in: *Kali br.*
White – tongue – diabetes in: *Helon. uran n.*

TEETH & GUMS:

Caries, decayed, hollow – diabetes in: *Sulph ac.*

STOMACH

Acidity – diabetes in: *Uran nit.*
Appetite – ravenous, canine, excessive – with emaciation – diabetes during:
Am c. coloc.
Aversion – meat – diabetes in: *Tarent.*
Burning – epigstrium – diabetes in: *Uran nit.*
Cramp like pain – epigastrium– diabetes in: *Uran nit.*
Derangement of stomach – diabetes in: *Nux v.*
Desire – effervescing for, liquids – diabetes in: *Ph ac.*
Desire – tea – diabetes in: *Uran nit.*
Distension – epigastrium – diabetes in: *Nat s.*
Drinking – uneasiness – stomach in – diabetes: *Lac ac.*
Dyspepsia – acid – diabetes in: *Uran nit.*
Emptiness – feeling of in stomach, sinking – diabetes in: *Lac ac.*
Faintness – epigastrium – diabetes in: *Uran nit.*
Gastralgia – diabetes in: *Sec.*
Hunger – diabetes in: *Cupr.*
Hunger – diabetes, with canine hunger: *Iod. Kali br. ac ac. rat.*
Nausea – diabetes in: *Lac ac.*
Oppression – epigastrium – diabetes in: *Arg m.*
Retching – eating after – diabetes in : *Lac ac.*
Sensitive touch to – epigastrium – diabetes in: *nat s.*
Shooting – in stomach – diabetes in: *Cur.*
Thirst – constant – diabetes in: *Uran nit.*
Thirst – excessive– diabetes in: *ACET AC Ars alb. Coloc. cupr m.cur. lac*
ac.lyc. ph ac. phos. pic ac.rat. ter. uran nit.
Thirst – excessive – evening and night – diabetes in: *cur.*
Thirst – excessive – fever with – diabetes in: *Sec.*
Thirst – excessive – coldest water, nothing but, would satisfy – diabetes in:
Lycop.
Thirst – unquenchable – in diabetes: *Sec.*
Vomiting – bile of – diabetes in: *Op.*
Vomiting – mucous of – diabetes in: *Op.*
Water brash – eating after – diabetes in: *Lac ac.*

ABDOMEN

Ascites – vehement thirst in ascites – diabetes: Acet ac.
 Atrophy of pancreas which causes diabetes: Merc.
 Liver – enlarged – diabetes in: Nat s.
 Liver – sensitiveness – tender – diabetes in: *Kali br.*
 Liver – sharp pain – diabetes in: Sulph ac.
 Liver – stitches – diabetes in: Sulph ac.
 Pale – livid – diabetes in: *Rat.*
 Pancreas – kidney disease of, preceding or accompanying diabetes: Phos

RECTUM

Constipation – diabetes in: *Uran nit.*
 Constipation – diabetes in–which relieves: *Cupr. kali br. kali m. ac ac.*
 lac d. mosch. op. tarent.
 Hemorrhoids – diabetes in: *Uran nit.*

STOOL

Stool – chalky – diabetes in: Podo.
 Stool – dry – diabetes in: *Cupr. uran nit.*
 Stool – gray – diabetes in: Nat s.
 Stool – light in colour – diabetes in: *Uran nit.*
 Stool – odourless – in relieved diabetes: *Uran nit.*

BLADDER

Desire – frequent to urinate – turbid, sweetish, profuse urine at night:
 Arg met.
 Dull, pressing – sensation – bladder in - diabetes in: *Phos ac.*
 Shooting – bladder – diabetes in: Op.
 Urination – constant desire – diabetes in: *Uran nit.*
 Urination – difficult – diabetes in: Op.
 Urination – frequent – diabetes in: Cop. podo.
 Urination – frequent–foamy, strong smelling dark–diabetes in: Lac ac.
 Urination – frequent – sleep during – diabetes in: Arg m.
 Urination – night – diabetes in: Cupr. nat s.
 Urination - Urge to – sleep during: Nat s.

KIDNEY:

Complaints of kidneys - accompanied by – diabetes - saroth.
 Kidney – aching – urinating – worse before and better after: LYC.
 Kidney – dull pain – pressure on– diabetes in: Nat s.
 Kidney – pain – diabetes in: Phos ac. phos.
 Kidney – pain – sore – diabetes in: rat.
 Kidney – weak – diabetes with: Phos.

URINE:

Urine – acid – diabetes in: Nat s.
 Urine – clear – diabetes in: *Ph ac.*
 Urine – decreases, scanty – diabetes in: RAT.
 Urine– foaming, on being passed–diabetes in: Nat s.
 Urine – increased – copious, polyuria – diabetes in: Acet ac,op.LAC AC.
 nat m. nat s. phos. podo. sec. tarax.
 Urine – increased – drunk, greater than quantity of water – diabetes in:
 Coloc.
 Urine – increased – frequent – diabetes in: tarax.
 Urine – increased – night – diabetes in: Arg m.
 Urine – increased – pain violent over lumbar region – diabetes in: Tarent.
 Urine – increased – paralysis of legs – diabetes with: Tarent.
 Urine–milk like (chyluria) – diabetes in: Coloc. *ph ac.*
 Urine – odorless – diabetes in: Camph.
 Urine – odor: sweetish diabetes in: Arg m.
 Urine – pale – diabetes in: Camph. nat s. *rat. tarax.*
 Urine - Specific gravity–increased: Arn. cahin. colch.p

Urine - sugar

Acet-ac. Adren. alf. all-s. alumn. am-a.am-c. aml-ns. ant-c. ant-t. anthraco. arg-met.arg-n.arist-m.arn. ars. Ars-br. ars-i. aur. aur-m.bar-c. bell.benz-ac. berb. Bor-ac.bov. bry. calc. calc-p. calc-sil. camph.caps.Carb-ac. Carb-v.

carc. cean. *Cham. Chel. Chim. Chin.* chinin-ar. *Chion.* chir-fl. chlol. *Coca₁ Cod.* coff. coff-t. *Colch. Coloc.* con. conv. cop. crat. *Crot-h.* cupr. cupr-ar. *Cur. Laps.* eup-pur. fel. ferr. ferr-i. *Ferr-m.* ferr-p. fl-ac. glon. glyc. grin. *Hell. helo.* HELON. *Hep. Hydr.* ign. ins. iod. *Iris.* kali-act. kali-bi. kali-br. kali-c. *Kali-chl.* kali-m. kali-n. *Kali-p. Kreos. Lac-ac. Lac-d. Lach.* lat-m. *Lec. led.* lith-c. LYC. lycpr. *Lycps-v.* lyss. mag-c. mag-s. *Med.* meph. merc. morg-p. morph. mosch. mur-ac. murx. nat-m. *Nat-s. Nit-ac. Nux-v.* op. ourl. *Pancr.* petr. PH-AC. phase. *Phlor.* PHOS. *Pic-ac.* PLB. plb-i. *Podo.* ran-b. *Rat. Rhus-a.* sal-ac. sec. sep. *Sil.* squil. stry-ar. *Sul-ac. Sulph.* SYZYG. tarax. TARENT. TER. *Thuj.* uran-met. URAN-N. urea. vanad. vinc-r. vince. zinc. zinc-p.

Urine – sugar loaded with: Kali br.

Urine – thick – coagulates on standing: Coloc.

Urine – turbid – diabetes in: Arg m.

Urine – visid – diabetes in: Nat s.

Urine – watery – diabetes in: Sec.

Urine – whey like - Diabetes in: Arg m.

GENITALIA: MALE:

Erection – troublesome – incomplete - diabetes in: Coca. mosch. Ph ac.

Erection – wanting - impotency - diabetes in: Coca. cupr. Helon. mosch.
Ph ac.

Oedema of scrotum and feet – diabetes in: Arg met.

Pain – shooting – diabetes in: Op.

Sexual desire – diminished - diabetes in: Coca. cupr.

Sexual power – loss of – cold after a – preceding diabetes: Mosch.

Swelling of scrotum – diabetes in: Arg m.

GENITALIA: FEMALE

Amenorrhoea – diabetes in: *Uran n.*

Eruption – severe itching of vulva, labia swollen, with humid eruption with
diabetes: Sep.

Menses – Disturbances of – diabetes in: Uran nit.

Menses - suppressed menses - diabetes; in - uran-n.

RESPIRATION:

Dyspnoea – diabetes in: Arg m. nat s.

COUGH

Cough – aggravation - diabetes in: sec.

CHEST:

Eruption – erythema covered with – chest – diabetes in: Nat s.

Flat – chest – diabetes in: Nat s.

Lungs; complaints of the - accompanied by – diabetes - calc-p.

Phthisis pulmonalis - accompanied by – diabetes - phos.

BACK

Heat – lumbar region – renal region in – Addison's disease , morbus
brightii, diabetes in: Nat m.

Pain – diabetes in: *Ph ac.*

Pain – lame, feeling with – diabetes in: Helon.

Pain – lumbar region – motion better by, severe – diabetes in: *Rat.*

Pain – lumbar region – polyuria, with violent – diabetes in: Tarent.

Spinal cord – inflammation – diabetes with: *Ph ac.*

EXTREMITIES:

Aching – joints – diabetes in: *Rat.*

Crawling – limbs – diabetes in: *Uran nit.*

Formication – limbs – diabetes in: *Uran nit.*

Gangrene – diabetic - carb-ac. con. lach. lyc. sec. solid.

Gangrene - Feet – diabetic - lyc.

Gouty symptoms – diabetes with: Lac ac. nat s.

Heaviness – legs – diabetes in: Sec.

Heaviness – feet – diabetes in: Nat s.

Numbness – legs – diabetes in: *Nux v.*

Pain - Lower limbs - Sciatic nerve - accompanied by -diabetes mellitus-
kreos.

Pain – ankle – diabetes in: Nat s

Pain – legs – aching – preventing sleep – diabetes in: Lac ac.
 Paralysis – legs – diabetes in: Nux v.
 Paralysis – partial – legs – diabetes in: Atro. nux v.
 Peculiar sensation – in diabetes: Nux v.
 Rheumatic pain - diabetes in: Lac ac.c
 Restlessness – limbs – diabetes in: Nux v.
 Soreness – limbs – diabetes in: Rat.
 Swelling – ankle - diabetes in: Arg m
 Swelling – feet – diabetes in: Arg m
 Swelling – legs – diabetes in: Uran nit.
 Tired feeling – diabetes in: lac ac.
 Tired feeling – legs – diabetes in: Uran nit.
 Walking – difficult, weak – diabetes in: Nat s
 Walking – inability – diabetes in: Uran nit

SLEEP:

Awaking – sweat – falling asleep, five minutes after, most profuse on head and upper portion of body – diabetes with parotitis: Con.
 Sleeplessness – diabetes in: Carc. coca. *uran nit.*
 Sleepiness, obstinate – diabetes in: Uran n.

CHILL:

Chill – diabetes, after chill: Nat s.
 Chilliness – limbs – diabetes in: Lac ac.

FEVER:

Rheumatic – wet getting from – diabetes in: Nat s.
 Sweat – night at – diabetes in: Uran nit.
 Sweat – fetid - sweetish – diabetes in (relieved): Uran n.
 Typhoid – including typhus – diabetes in: Sulph ac.

PERSPIRATION:

Diabetes – Peculiar sweet smell about the patient as if in diabetes: Pyrog.

SKIN:

Blackness of external parts – diabetes in: Ars. con. *kreos.* lach. sec. solid.
 Clammy – diabetes in: Uran nit.
 Coldness – diabetes in: Sulph ac.
 Dry skin – diabetes in: *kali br.* lac ac. *uran nit.*
 Eruption – Petechiae – diabetes in: Sec.
 Eruption – miliary – diabetes in: Tarent.
 Flabby – relaxed – Skin – diabetes in: Nat s.
 Gangrene – diabetes in: Carb ac. con. lach. sec. solid.
 Gangrenous inflammation – diabetes in: nat pyru. sec.
 Itching – diabetes in: Mang. *sulph ac.* *Cephd-i.*
 Induration – diabetes in: Uran nit.
 Rough – Harsh and dry, no sweat – Diabetes: Lac ac.
 Suppression – furuncles – diabetes in: *Ph ac.* sec. tarent.
 Ulcers – diabetes in: Asaf. syzyg.

GENERALS:

Diabetes Mellitus

abrom-a, acet-ac, adren, aether, alf, all-s, allox, aloe, alum, am-act, anthraco, apoc, arg-met, arg-n, arist-m, ars, *Ars-br.* asc-c, aspar, aur, aur-m-n, bar-m, *Bor-ac.* bov, brid-fr, calc, calc-p, calc-sil, canth, carb-ac, carb-v, carc, card-m, *Carl.* caust, cean, cephd-I, chel, chim, *Chion.* chlol, chlorpr, clem, coca, cod, coff, coloc, con, cop, cortico, cortiso, cub, cupr, cupr-ar, cur, eup-pur, ferr-I, ferr-m, ferr-p, fl-ac, flor-p, friedr, gal-ac, galeg, glyc, *Gymne.* hed, helon, hydrang, hygroph-s, indgf-a, ins, *Inul.* iod, iris kali-act, kali-br, kali-chl, kali-I, kali-p, kiss, *kreos.* *Lac-ac.* lac-d, lach, led, lept, lith-c, lyc, lycps-v, mag-act, mag-o, mag-p, mag-s, mang-act. med. meny. merc. merc-d. moni. morind-l morind-m. morph. mosch. mur-ac. murx. nat-ch. nat-lac. nat-m. nat-p. NAT-S. nauc-l.nep. nit-ac. nux-v. *Op.* orthos-s.oxyg. pancr.peps. perh.ph-ac. *Phase.* phlor.phos.pic-ac. pilo.plan. plb. podo. rad-br. rad-met. ran-b. rat. *Rhus-a.* rhus-r. rhus-t. sacch-l. sal-ac. sanic. sarcol-ac. saroth. sep. *Ser-ang.* sil. spong. *Squil.* stict.stront-c. stry-ar. sul-ac. sulfonam. sulph. syph. SYZYG. tarent. TER. *Terebe.* term-a. thuj. thyr. *uran-m.* *Uran-n.* *Urea.* vanad. vichy-g. vinc-r. vince.
 Diabetes mellitus - accompanied by -Abdomen - distention of; tympani tic-uran-n.
 Diabetes mellitus - accompanied by – Acne - ars-br.

Diabetes mellitus - accompanied by - Ankles; swelling of the - arg-met.
 Diabetes mellitus - accompanied by - Apoplexy - con.
 Diabetes mellitus - accompanied by - Diarrhea - ars. gal-ac. kali-act. pancr.
 Diabetes mellitus - accompanied by - Feet; numbness of - helon.
 Diabetes mellitus - accompanied by - Tongue - cracked - bor-ac.
 Diabetes mellitus - accompanied by - Tongue - red discoloration of the tongue - bor-ac.
 Diabetes mellitus - accompanied by - Tongue - red discoloration of the tongue - bright - *Nat-s*
 Diabetes mellitus - accompanied by - Tongue - white discoloration of the tongue - helon. uran-n.
 Diabetes mellitus - accompanied by - Tongue - dry - bor-ac.
 Diabetes mellitus - accompanied by - indigestion - nux-v. podo. uran-n.
 Diabetes mellitus - accompanied by - Liver; complaints of - chion. kali-br. kali-c. mag-c. nat-p. podo.
 Diabetes mellitus - Pancreas; from complaints of - iris. pancr. phos.
 Diabetes mellitus - accompanied by - constipation
 carl. graph. kreos. lac-d. nat-s.
 Diabetes mellitus - accompanied by - respiration; asthmatic - nat-s.
 Diabetes mellitus - Diabetes mellitus - accompanied by - leucorrhea - abrom-a.
 Diabetes mellitus - accompanied by - rheumatic pains
 helon. lac-ac. *Led. Sarcol-ac.* syph.
 Diabetes mellitus - accompanied by - boils
 Anthraci. Anthraco. arn. ars. cephd-i.
 chlorpr. graph. ins. iod. led. nat-p. ph-ac.
 Diabetes mellitus - accompanied by - carbuncles
 abrom-a. ars. cephd-i. *Crot-h.* graph. gymne.
 ins. kreos. *Lach.* led. ph-ac.
 Diabetes mellitus - accompanied by - psoriasis - mang-act.
 Diabetes mellitus - accompanied by - gangrene- *Ars. con. cupr-ar.* kreos.
 lach. merc. *Sec. solid.*
 Diabetes mellitus - accompanied by - Skin; itching of the - con. graph. sul-ac.
 Diabetes mellitus - accompanied by - ulcers - sec. syzyg.
 Diabetes mellitus - accompanied by - abscesses - ars.
 Diabetes mellitus - accompanied by - arteriosclerosis - aur. chlorpr. plb. syzyg.
 Diabetes mellitus - accompanied by - blackness of external parts - *Ars. Kreos. kres. Sec.*
 Diabetes mellitus - accompanied by - hypertension - sec.
 Diabetes mellitus - accompanied by - hyperthyroidism - kali-i.
 Diabetes mellitus - accompanied by - Urinary tract; inflammation of- canth. helon. rhus-a.
 Diabetes mellitus - accompanied by - Vagina; coldness of - bor-ac.
 Diabetes mellitus - accompanied by - Vulva; itching of the - pic-ac. sep.
 Anaemia - diabetes in: Podo.
 Emaciation - diabetes in: ARG M. ars alb. ars-br. cupr. cur. dig. graph.
 helon .kali-br. lac-d. merc. nat-s. pancr. ph-ac. rat.
 tarent. uran-met. Uran n.
 Emaciation - body of - with good appetite - diabetes in: *Coloc.*
 Emaciation - diabetes - large quantity of sugar in urine, with Emaciation, thirst, restlessness and melancholia: Helon.
 Emaciation - progressive - slowly - diabetes in: Cupr met.
 Family history - diabetes of: Carc. sacch. thuj.
 Gastro - hepatic origin - diabetes: *Ars. ars i. bry. calc.cham.chel.kreos. lac-ac.lept. lyc. nux v. phos. uran n.*
 Inflammation - gangrenous- diabetics; in - ars. nat-pyru. sec.
 Nervous origin - diabetes: *Ars. aur m. calc. ign. ph ac. phos.*
 Neurological complaints - accompanied by - diabetes - helon.
 Paralysis, motor - diabetes with: cur.phos.
 Pulse - accelerated - diabetes in: *Uran nit.*
 Pulse - slow - diabetes in: Op.
 Pulse - small - diabetes in: *Uran nit.*
 Pulse - weak - diabetes in: *Kali br.*
 Rapid course, with - diabetes: Cur._M, morph.

Shock - followed by - diabetes mellitus - op.
 Swelling, edema – diabetes with: Lac ac.
 Torpid - chlorosis with inactive (Diabetes): Nat m. sulph ac.
 Weakness - diabetes in: ACET AC. arg m. ars.carb ac. carc. coca. lac ac.
 nat s. op. ph ac. phos. sec. sulph ac.
 Weakness – morning in – diabetes in: Nat s.
 Weakness - diabetes mellitus, in-acet-ac. *Arg-met. Ars. carb-v. carc. con.*
 graph. kali-c. *Lac-ac.* op. phos.

**CHAPTER-6
 MATERIALS AND METHODS**

- 1)Prospective study was conducted by studying the patients attending out-patient and in-patient sections of Dept.of Case taking and Repertorization, Govt. Homoeopathic Medical College, Kozhikode, showing signs and symptoms of Diabetes Mellitus with positive findings having met required standards as per DSM-IV-TR obtained from case history. Period of study is 1 year.
- 2) All cases were treated according to principles of homoeopathy .Each case was repertorised. Repetition, change of potency and remedy was done according to Homoeopathic philosophy.
- 3) Each case was reviewed at 2 weeks, or 4 weeks interval.
- 4) Effectiveness of treatment was assessed by reduction or disappearance of distressing symptoms along with blood glucose levels-fasting and or post prandial at regular intervals
- 5) Final evaluation of data will be done by using the statistical technique -Paired "t" test. The initial and final blood sugar values are compared using the above test and critical ratio for the same is determined. Then its level of significance was assessed by noting the p value.

6.1 DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS

Fasting blood Glucose ≥ 126 mg/dl (7.0 mmol/l). Fasting is defined as no caloric intake for at least 8 h.

6.2 INCLUSION CRITERIA

- 1)During the history taking the case was identified as per diagnostic criteria of Diabetes mellitus.
 - 2)Study was be made on male and females. The Diabetes positive cases was screened on basis on DSM-IV -TR satisfying the criteria for psychological symptoms affecting medical conditions¹⁶
- A)A general Medical condition is present.(In this case Diabetes Mellitus)
 B)Psychological factors adversely affect the general medical conditions in one of following ways
- (1) the factors have influenced the course of the general medical condition as shown by a close temporal association between the psychological factors and the development or exacerbation of, or delayed recovery from, the general medical condition
 - (2) the factors interfere with the treatment of the general medical condition

**CHAPTER-7
 STATISTICAL ANALYSIS & RESULTS**

Statistical analysis of change in Fasting blood sugar level
 To analyze the difference between pre-treatment and post-treatment observations paired't' test is used.
 Let X_1 be the value before treatment and X_2 after the treatment
 Let the hypothesis be H_0 : no difference before and after treatment
 $H: X_2 < X_1$

Table 7.1 Statistical analysis of change in Fasting blood sugar level

Sl. No.	X_1	X_2	d (X_1-X_2)	d^2
1	160	80	80	6400
2	150	175	-25	625

3	180	276	-96	9216
4	220	124	80	6400
5	140	80	60	3600
6	145	85	55	3025
7	280	160	120	14400
8	160	80	80	6400
9	170	90	80	6400
10	170	210	-40	1600
11	220	150	70	4900
12	200	210	-10	100
13	164	180	-16	256
14	188	90	98	9604
15	220	240	-20	400
16	160	82	78	6084
17	160	180	-20	400
18	163	85	78	3969
19	240	145	95	100
20	180	110	70	4900
21	210	210	0	0
22	255	225	25	625
23	170	100	70	4900
24	173	210	-37	1369
25	200	116	84	7056
26	192	124	68	4624
27	240	124	116	13456
28	245	175	70	4900
29	255	140	115	13225
30	190	240	-50	2500
Σ d= 1278				Σ d² = 138434

$$\Sigma d = 1278$$

$$\Sigma d^2 = 138434$$

$$\bar{d} = \frac{\sum d}{n} = \frac{1278}{30} = 42.6$$

$$SD_{(d)} = \sqrt{\frac{\sum d^2 - \frac{(\sum d)^2}{n}}{n-1}}$$

By substituting the values, S D = 10.335

$$\text{Paired } t = \frac{|\bar{d}|}{SD_{(d)} / \sqrt{n}} = \frac{|\bar{d}| \times \sqrt{n}}{SD_{(d)}}$$

$$t_{29} = 2.622$$

From the table paired t value is t_{29} 1.699 at 5% significance and t_{29} 2.462 at 1% level of significance.

$$\text{So here } t > t_{\alpha}$$

The calculated value of t is greater than table value. So we reject the null hypothesis and accept the alternate hypothesis. That is the mode of treatment is effective in controlling diabetes.

CHAPTER-9 CONCLUSION

Stressful life events that played a major factor in patients suffering from Diabetes Mellitus. Significantly the stress events are associated with the onset of diabetes mellitus and its progress. It is found that the comparison of the FBS before and after treatment showed statistically significant result. It can also be claimed that Homoeopathy is safe, simple, less expensive and more effective in treating diabetes mellitus.

Homoeopathy as a system of medical treatment has a philosophy of its own and its therapeutics is based on certain fundamental principles. Out of these fundamental principles theory of chronic disease play a vital role in treating chronic cases.

To conclude in Hahnemann's words "*He, who has had as many opportunities as I to make observations,... he, who is induced by his desire for the welfare of his fellow beings to think and act for himself, he, who like myself feels hatred for the prejudices and preferences for old or new, or, generally speaking, for any kind of recognition or great name, and he, who eagerly endeavours, as I myself have done, to act and to think independently.... he will see excellent results for his industry which is the greatest reward that an honest physician can expect*".

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

BIBLIOGRAPHY

- [1] P.731-733 DSM-IV-TR fourth edition pub. by American Psychiatric Association
- [2] a. p.2152 Harrison's principles of Internal Medicine 16th edition.
b. WHO (1980) Techn.Rep.Ser. No.646 Parks Textbook of Preventive and social medicine 16th edition
- [3] Axis I disorders. Clinical Disorders p.28 DSM-IV-TR fourth edition
- [4] Axis III disorders General Medical Conditions Appendix G DSM-IV-TR fourth edition
- [5] How Stress Affects Diabetes .American Diabetic Association .www.diabetes.org
- [6] Association of Depression and Diabetes Complications: A Meta-Analysis Psychosomatic medicine vol63 p.619-630(2001)
- [7] Burnout and Risk of Type 2 Diabetes :A prospective Study of Apparently Healthy Employed Persons Psychosomatic Med vol68j p.863-869
- [8] World Health Organization. Fact sheet N^o312.Septemper 2006. www.who.int
- [9] Epidemiology of diabetes in India--current perspective and future projections . [J Indian Med Assoc.](#) 2002 Mar;100(3):144-8
b.Health Action by people-NGO organization in Kerala. www.kerala.gov.in
- [10] p.832 Synopsis of Psychiatry .Kaplan&Sadock's. pub-Lippincott williams&Wilkins
- [11] Definition and diagnostic criteria for diabetes mellitus and other categories of glucose intolerance,2.1,p.2 :WHO/NCD/NCS/99.2
- [12] 2.2 Diagnosis and diagnostic criteria,3-4; WHO/NCD/NCS/99.2
- [13] McCance DR, Hanson RL, Charles MA, Jacobsson LTH, Pettitt DJ, Bennett PH et al. Comparison of tests for glycated haemoglobin and fasting and two hour plasma glucose concentrations as diagnostic methods for diabetes. BMJ 1994; 308: 1323-28.
- [14] 2.2.2 Diabetes in children,P.4; WHO/NCD/NCS/99.2
- [15] 2.3 Diagnostic criteriaP.5, ; WHO/NCD/NCS/99.2
- [16].
- [17]. Tripathy BB, Samal KC. Overview and consensus statement on diabetes in tropical areas. Diabetes Metab Rev 1997; 13: 63-76.
- [18] Shaw JE, Zimmet PZ, de Courten M, Dowse GK,Gareeboo H, Hemraj F et al. IFG or IGT: what best predicts future diabetes? A view of the new ADA recommendations. Diabetes Care 1999 (In press)
- [19] Larsson H, Berglund G, Lindgärde F, Ahrén B. Comparison of ADA and WHO criteria for diagnosis of diabetes and glucose intolerance. Diabetologia 1998; 41: 1124-1125.
- [20] Fuller JH, Shipley MJ, Rose G, Jarrett RJ, Keen H.Coronary heart disease risk and impaired glucose tolerance: the Whitehall Study. Lancet 1980; i: 1373-76
- [21] Alberti KGMM. The clinical implications of Impaired Glucose Tolerance. Diabet Med 1996; 13: 927-37.
- [22] Reaven GM. Role of insulin resistance in human disease. Diabetes 1988; 37: 1595-607.
- [23] McCance DR, Hanson RL, Pettitt DJ, Bennett PH, Hadden DR, Knowler WC. Diagnosing diabetes mellitus - do we need new criteria? Diabetologia 1997; 40: 247-55.
- [24] Zimmet PZ, Tuomi T, Mackay R, Rowley MJ, KnowlesW, Cohen M et al. Latent autoimmune diabetes mellitus in adults (LADA): the role of antibodies to glutamic acid decarboxylase in diagnosis and prediction of insulin dependency. Diabetic Med 1994;11: 299-303.
- [25] Humphrey ARG, McCarty DJ, Mackay IR, Rowley MJ, Dwyer T, Zimmet P. Autoantibodies to glutamic acid decarboxylase and phenotypic features associated with early insulin treatment in individuals with adult- onset diabetes mellitus. Diabetic Med 1998; 15: 113-19
- [26]. Japan and Pittsburgh Childhood Diabetes Research Groups. Coma at onset of young insulin-dependent diabetes in Japan: the result of a nationwide survey. Diabetes 1985; 34: 1241-46.
- [27]. Zimmet PZ. The pathogenesis and prevention of diabetes in adults. Diabetes Care 1995; 18: 1050-64.
- [28]. Willis JA, Scott RS, Brown LJ, Forbes LV, Schmidli RS, Zimmet PZ et al. Islet cell antibodies and antibodies against glutamic acid decarboxylase in newly diagnosed adult-onset diabetes mellitus. Diabetes ResClin Pract 1996; 33: 89-97.
- [29] Hother-Nielsen O, Faber O, Sørensen NS, Beck- Nielsen H. Classification of newly diagnosed diabetic patients as insulin-requiring or non-insulin-requiring based on clinical and biochemical variables. Diabetes Care 1988; 11: 531-37.
- [30] Verge CF, Gianani R, Kawasaki E, Yu L, Pietropaolo M, Jackson RA et al. Predicting type I diabetes in first-degree relatives using a combination of insulin, GAD, and ICA512bdc/IA-2 autoantibodies. Diabetes 1996;45: 926-33.

- [31] Mølbak AG, Christau B, Marner B, Borch-Johnsen K, Nerup J. Incidence of insulin-dependent diabetes mellitus in age groups over 30 years in Denmark. *Diabet Med* 1994; 11: 650-55.
- [32] Betterle C, Zanette F, Pedini B, Presotto F, Rapp LB, Monciotti CM et al. Clinical and subclinical organ-specific autoimmune manifestations in type 1 (insulin-dependent) diabetic patients and their first-degree relatives. *Diabetologia* 1983; 26: 431-36.
- [33] McLarty DG, Athaide I, Bottazzo GF, Swai ABM, Alberti KGMM. Islet cell antibodies are not specifically associated with insulin-dependent diabetes in rural Tanzanian Africans. *Diabetes Res Clin Pract* 1990; 9: 219-24.
- [34] Ahrén B, Corrigan CB. Intermittent need for insulin in a subgroup of diabetic patients in Tanzania. *Diabet Med* 1984; 2: 262-64.
- [35] DeFronzo RA, Bonadonna RC, Ferrannini E. Pathogenesis of NIDDM. In: Alberti KGMM, Zimmet P, DeFronzo RA, eds. *International Textbook of Diabetes Mellitus*. 2nd edn. Chichester: John Wiley, 1997: pp 635-712.
- [36] Lillioja S, Mott DM, Spraul M, Ferraro R, Foley JE, Ravussin E et al. Insulin resistance and insulin secretory dysfunction as precursors of non-insulin-dependent diabetes. Prospective Study of Pima Indians. *N Engl J Med* 1993; 329: 1988-92.
- [37] Mooy JM, Grootenhuys PA, de Vries H, Valkenburg HA, Bouter LM, Kostense PJ et al. Prevalence and determinants of glucose intolerance in a Dutch population. The Hoorn Study. *Diabetes Care* 1995; 18: 1270-73.
- [38] Harris MI. Undiagnosed NIDDM; clinical and public health issues. *Diabetes Care* 1993; 16: 642-52.
- [39] Campbell PJ, Carlson MG. Impact of obesity on insulin action in NIDDM. *Diabetes* 1993; 42: 405-10.
- [40] Bogardus C, Lillioja S, Mott DM, Hollenbeck C, Reaven G. Relationship between degree of obesity and in vivo insulin action in man. *Am J Physiol* 1985; 248: E286-E291.
- [41] Kissebah AH, Vydellingum N, Murray R, Evans DF, Hartz AJ, Kalkhoff RK et al. Relationship of body fat distribution to metabolic complications of obesity. *J Clin Endocrinol Metab* 1982; 54: 254-60.
- [42] Banerji MA, Chaiken RI, Huey H, Tuomi T, Norin AJ, MacKay IR et al. GAD antibody negative NIDDM in adult black subjects with diabetic ketoacidosis and increased frequency of human leukocyte antigen DR3 and DR4: flatbush diabetes. *Diabetes* 1994; 43: 741-45.
- [43] Umpierrez GE, Casals MMC, Gebhardt SSP, Mixon PS, Clark WS, Phillips LS. Diabetic ketoacidosis in obese African-Americans. *Diabetes* 1995; 44: 790-95.
- [44] Polonsky KS, Sturis J, Bell GI. Non-insulin-dependent diabetes mellitus: a genetically programmed failure of the beta cell to compensate for insulin resistance. *N Engl J Med* 1996; 334: 777-84.
- [45] Simonson DC, Ferrannini E, Bevilacqua S, Smith D, Barrett E, Carlson R et al. Mechanism of improvement in glucose metabolism after chronic glyburide therapy. *Diabetes* 1984; 33: 838-45.
- [46] Wing RR, Blair EH, Bononi P, Marcus MD, Watanabe R, Bergman RN. Caloric restriction per se is a significant factor in improvements in glycemic control and insulin sensitivity during weight loss in obese NIDDM patients. *Diabetes Care* 1994; 17: 30-36.
- [47] Zimmet PZ. Kelly West Lecture 1991: challenges in diabetes epidemiology: from West to the rest. *Diabetes Care* 1992; 15: 232-52.
- [48]. Byrne MM, Sturis J, Menzel S, Yamagata K, Fajans SS, Dronsfield MJ et al. Altered insulin secretory response to glucose in diabetic and nondiabetic subjects with mutations in the diabetes susceptibility gene MODY 3 on chromosome 20. *Diabetes* 1996; 45: 1503-10.
- [49] Froguel P, Vaxillaire M, Sun F, Velho G, Zouali H, Butel MO et al. Close linkage of glucokinase locus on chromosome 7p to early-onset non-insulin-dependent diabetes. *Nature* 1992; 356: 162-64.
- [50] Harrison 19th edition
- [51] Fieldmann , *Introduction to Psychology, Stress*
- [52] *How Stress Affects Diabetes* .American Diabetic Association .
- [53] Association of Depression and Diabetes Complications: A Meta-Analysis *Psychosomatic medicine* vol63 p.619-630(2001)
- [54] Burnout and Risk of Type 2 Diabetes :A prospective Study of Apparently Healthy Employed Persons *Psychosomatic Med* vol68] p.863-869
- [55] p.832 Synopsis of Psychiatry .Kaplan&Sadock's. pub-Lippincott williams&Wilkins
- [56].Psychological Stress and the onset of IDDM In children. *Diabetes Care* vol18 p.1323-1329 (1995)
- [57].Body fat Distribution, Insulin resistance and metabolic disorders. *Nutrition* vol.13
- [58].Effect of Acute Psychotic Stress in Nondiabetic Subjects onBeta-cell Function and Insulin Sensitivity .*Diabetes care* vol26:1462-1467,2003
- [59] P. BARGLOW, MD et al, Stress and Metabolic Control in Diabetes: Psychosomatic Evidence and Evaluation of; *Psychosomatic Medicine* Vol. 46, No. 2 (March/April 1984) Methods
- [60] Depression-Related Hyperglycemia in Type 1 Diabetes: A Mediatonal Approach; ***Psychosomatic Medicine* 67:195-199 (2005)**PATRICK J. LUSTMAN, PHD
- [61] 290 diabetes care, volume 28, number 2, february 2005
- [62] diabetes care, volume 30, number 3, march 2007 555
- [63] 2856 diabetes care, volume 27, number 12, december 2004

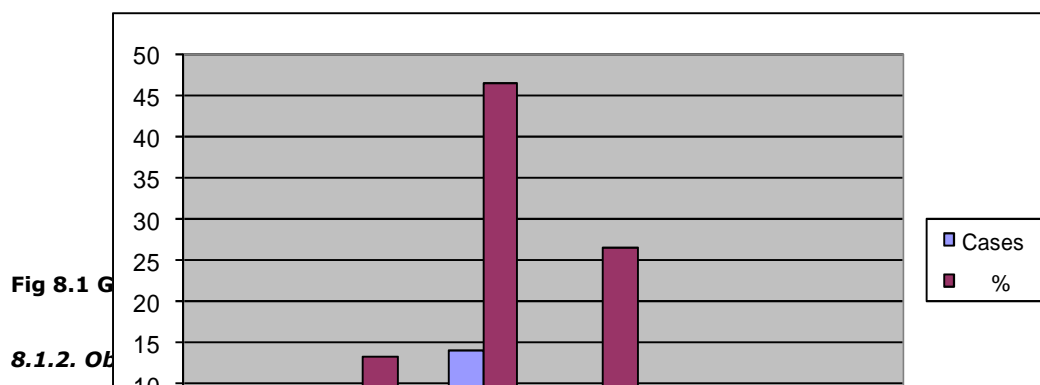
[64] G. R. Sridhar and K. Madhu, Psychosocial and cultural issues in diabetes mellitus, , CURRENT SCIENCE, VOL. 83, NO. 12, 25 DECEMBER 2002
 [65] www.nlm.nih.gov/hmd/emotions
 [66] p.734 DSM-IV-R Diagnostic criteria for 316 .DSM-IV-TR fourth edition pub. by American Psychiatric Association
 [67] S.P Dey, The Scope of Homoeopathy In Diabetes Mellitus
 [68] S.P Dey, The Scope of Homoeopathy In Diabetes Mellitus
 [69] -Kansal Kamal ,Homoeopathic Treatment of Diabetes Mellitus P.
 [70] W.Morgan-Diabetes
 [71] PAVRI KERI R.S,Essentials of Diabetes Mellitus and Its Treatment by Homoeopathy
 [72] S.LILIENTHAL, Homoeopathic Therapeutics
 [73] Dr George Royal-Text Book of Homoeopathic Materia Medica
 [74] Diabetes Mellitus:Its Diagnosis and treatment :KN Mathur
 [75] Boericke
 [79] Organon of Medicine 6th Edition
 [80] Repertory of the Homoeopathic Materia Medica by J T KENT
 [81] Synthesis Repertorium Homoeopathicum Syntheticum- Frederick Schroyens
 [82] Roger Van Zandervoot – Complete Repertory 4.5
 [83] Homoeopathic Medical Repertory - Robin Murphy
 [84] Boenninghausen’s Therapeutic Pocket Book
 [85] Boenninghausen’s characteristics and repertory- Boger
 [86] Clinical Repertory – J H Clarke
 [87] A Concise Repertory on Homoeopathic Materia medica – Phatak
 [88] Repertory to the more Characteristic symptoms of the -Materia Medica. - Constantine Lippe
 [89] Synoptic Key - C.M Boger
 [90] The Concordance Repertory of the characteristic symptom of Materia medica. - Genry William D
 [91] Repertory to Herring guiding symptoms - Knerr Calvin B

**CHAPTER-8
 OBSERVATION AND DISCUSSION
 8.1 OBSERVATIONS**

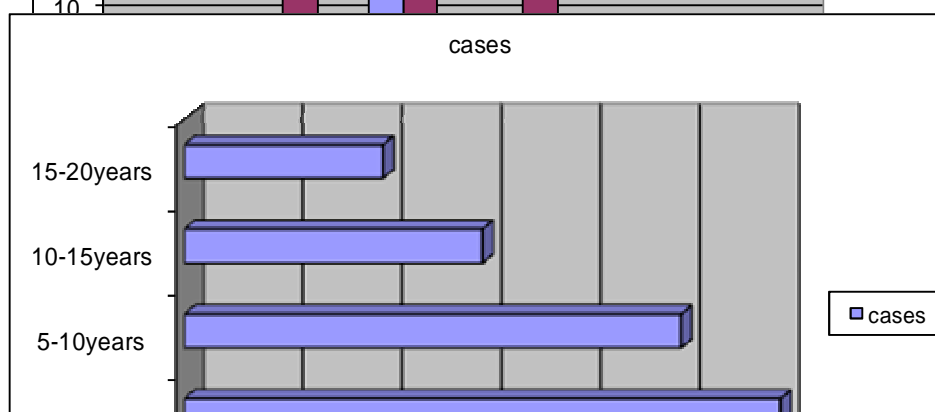
8.1.1 Age Distribution

30 patients belonging to the age group 30-80 were selected for study. Among the 30 patients maximum prevalence of Diabetes Mellitus was noted between 40-49 years of age(46%).The second highest prevalence was found in age group 50-59 () and next age group (%).The distribution is presented in table below, also the diagrammatic representation is furnished.

Age Group	30-39	40-49	50-59	60-69	70-79
Patients	4	18	8	2	2
Percentage	13.3%	46.6%	26.6%	6.6%	6.6%



8.1.2. Ob



Representation of patients according to the Domicile

Fig 8.2 Graphical Representation of patients according to the Duration of illness

8.1.3. Observations in Patients According to Domicile

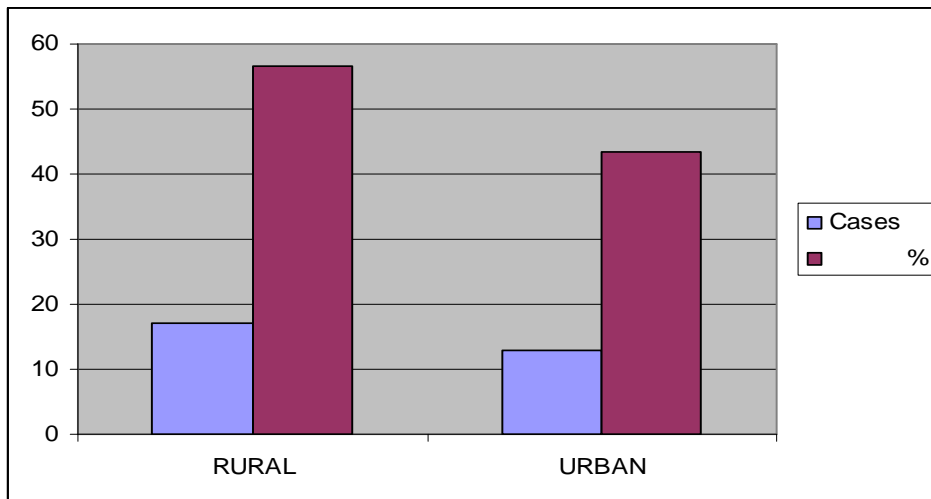


Fig 8.3 Representation of patients according to Domicile

Table 8.1 Representation of patients according to Domicile

Domicile	Cases	Percentage
RURAL	17	56.6%
URBAN	13	43.3%

8.1.4 Socio-Economic Distribution

Among 30 persons included in the study, highest prevalence was in per class .Next frequency was in middle class.

Table 8.2 Representation of patients according to the Socio - Economic Status

Socio Economic Class	No. of cases	Percentage
Poor class	20	66.7
Middle class	6	20.0
High class	4	13.3

8.1.5. Observations in Patients According to the Socio - Economic Status

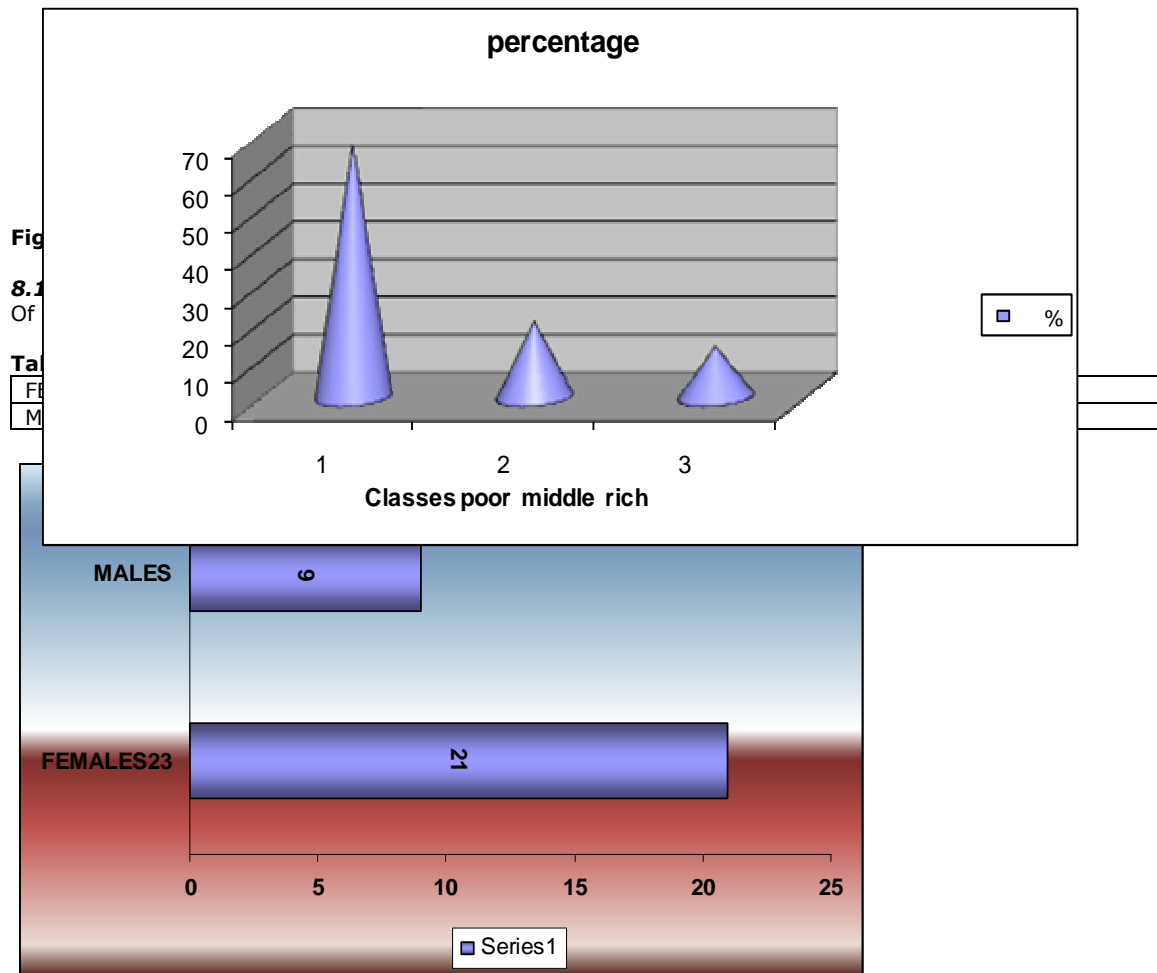


Fig 8.5 Graphical distribution of Gender Distribution

8.1.7. Distribution of Family History

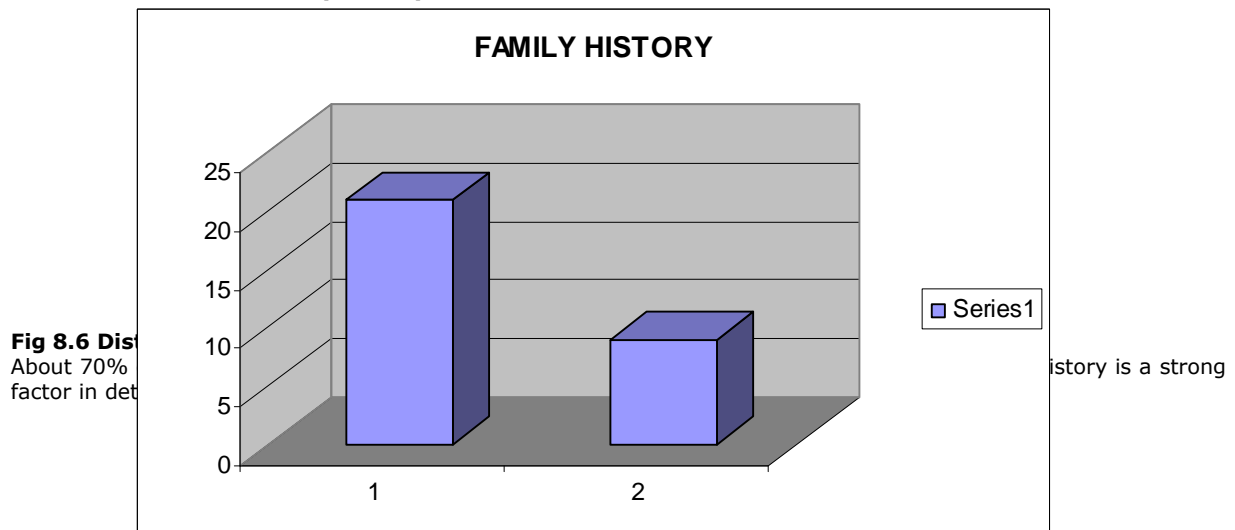


Fig 8.6 Dis
About 70%
factor in det

istory is a strong

8.1.8. Observation of Symptomalogical Presentation of Diabetes Mellitus Cases

Table 8.4 Changes in presenting Clinical Features after treatment

Symptom	Cases	>	%	<	%	N	%	D	%
Weakness	26	12	46.1	3	11.5	8	30.7	3	11.5
Loss of weight	6	2	33.3	0	0	3	50	1	16.6
Increased thirst	18	11	61.1	1	5.5	4	22.2	2	11.1
Increased hunger	11	7	63.6	1	9	2	18.1	1	9
Increased Urinary urgency	9	3	33.3	0	0	3	33.3	3	33.3
Blurred vision	5	1	20	1	20	3	60	0	0
Frequent infections of the skin	4	1	25	0	0	2	50	1	25
Itching of skin and/or genitals) case	17	4	23.5	3	17.6	7	41.1	3	17.6
Slow healing of cuts and bruises	9	4	44.4	0	0	2	22.2	3	33.3
Recurring skin, gum or urinary tract infections	13	6	46.1	1	7.6	4	30.7	2	15.3
Burning Sensation ,	15	7	46.6	3	20	3	20	2	13.3
Dryness of mouth	4	2	50	0	0	1	25	1	25
Tingling or numbness in the legs, feet or fingers	16	5	31.2	1	6.2	8	50	2	12.5

Case-No Patients
<-Aggravation

N-No change
>-amelioration

D-Disappearance

Distribution of Presenting Complaint

Of the 30 cases presented, 26 cases had Weakness as a prominent symptom, 6 cases of complaint of significant loss of weight, increased thirst or Polydypsia was presented by 18 cases. Increased hunger was presenting feature in 11 cases. Increased Urinary urgency and frequency was reported in 9 cases.

Other Associated symptoms Tingling or numbness in the legs, feet or fingers (16 cases), Blurred vision(5 cases), Frequent infections of the skin (4 cases)., Irritability and mood changes(7 cases), Itching of skin and/or genitals)17 cases), Slow healing of cuts and bruises(9 cases), Recurring skin, gum or urinary tract infections(13 cases), Burning Sensation ,Dryness of mouth was presented in 4 cases and Drowsiness(2 cases)

Table 8.5 Distribution of Presenting Complaint

Sl no	Presenting Complaint	Number	Percentage
1	Weakness	26	86.6
2	Loss of weight	6	20
3	Increased thirst	18	60
4	Increased hunger	11	36.6
5	Increased Urinary urgency and frequency.	9	30
6	Blurred vision	5	16.6
7	Frequent infections of the skin	4	13.3
8	Itching of skin and/or genitals) case	17	56.6
9	Slow healing of cuts and bruises	9	30
10	Recurring skin, gum or urinary tract infections	13	43.3
11	Burning Sensation ,	15	50
12	Dryness of mouth	4	13.3
13	Tingling or numbness in the legs, feet or fingers	16	53.3

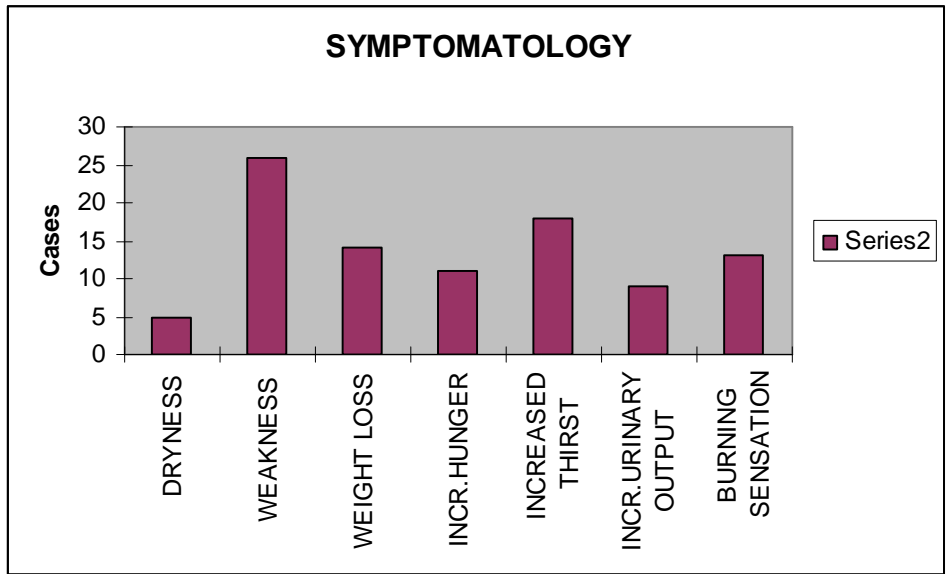


Fig 8.7 Distribution Of Symptomalogical Presentation Of Diabetes Mellitus Cases

8.1.9. Distribution According to Miasms

In all the 30 cases included in the study the Miasmatic Symptomatology Psora was found to be the first dominant 53%, miasm. Sycosis was the second dominant miasm, 28% and Syphillis the third. with 19%.

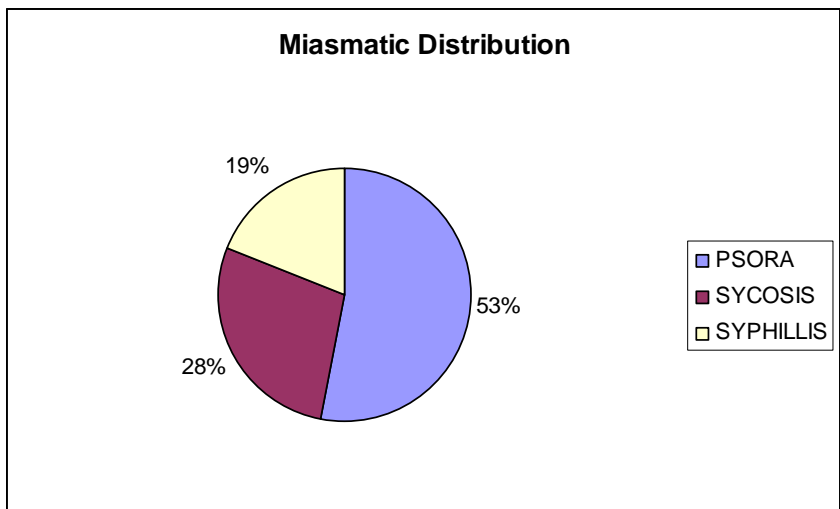


Fig 8.8 Distribution According to Miasms

8.1.10. Comparison of Fbs Values Before and After Treatment

Table 8.4 Comparison of FBS values before and after Treatment

FBS Before Treatment	FBS After Treatment
160	80
150	175
180	276
220	140

140	80
145	85
280	200
160	80
170	90
170	210
220	180
200	220
164	180
188	90
220	240
160	82
160	180
163	100
240	250
180	110
210	210
255	240
170	100
173	240
200	116
192	124
240	124
245	220
255	240
190	240

Fig 8
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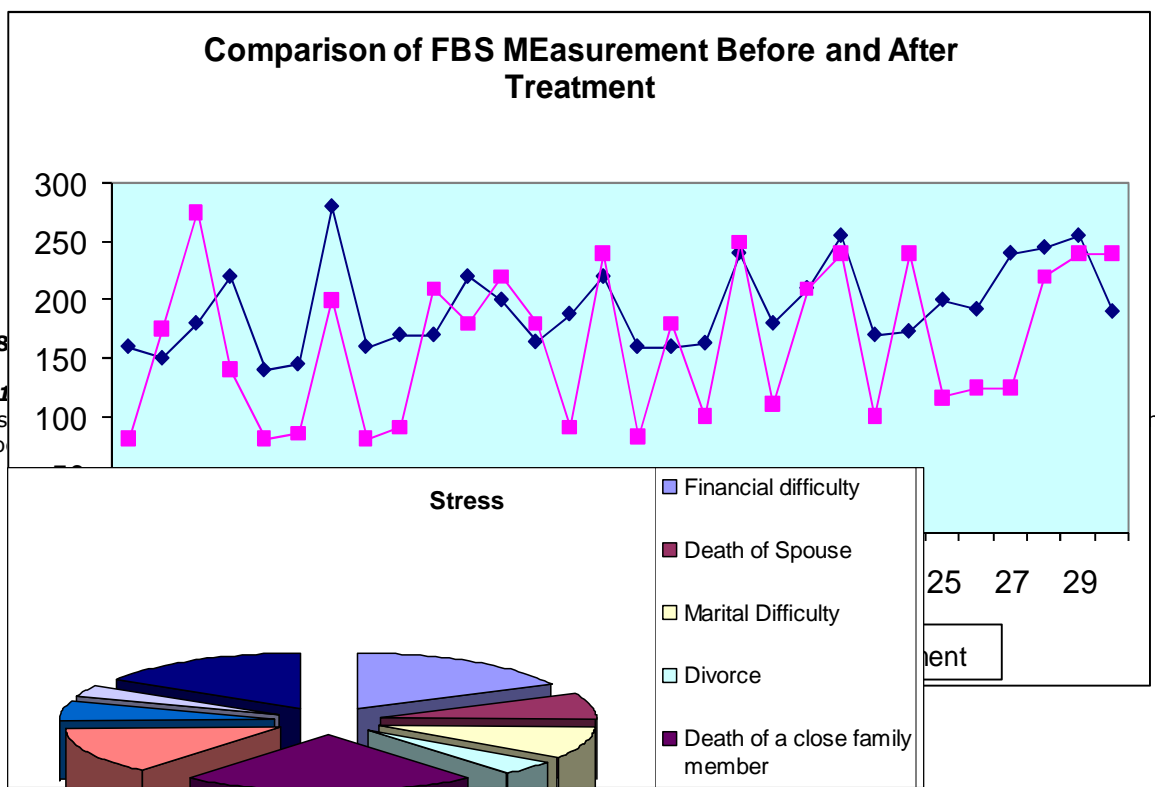


Fig 8.10 Stressful Life Events associated with Cases of Diabetes

8.1.12 Observation of Remedies Used

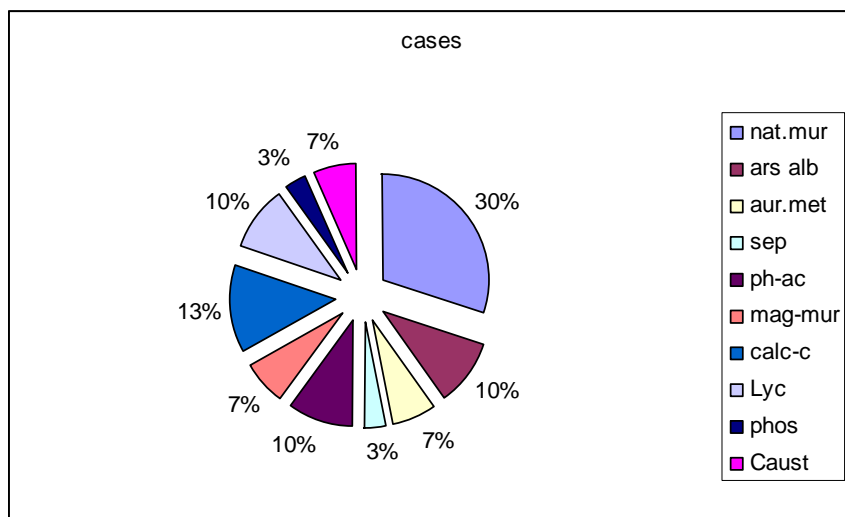


Fig 8.11 Representation of Remedies Used

Medicines were selected on basis on constitutional similarity also taking into consideration of stress factors. Among 30 cases medicine indicated most of times is Nat.mur- 23%(9 cases). Then Calc.carb 10%, in 4 cases followed by Phos.acid, Ars.alb, and, Lyco, 10%, in 3 cases each Aur.met, Caust, Mag.mur 7%ie, 2 cases each. Phos & Sep 3%ie, 1 This shows the effectiveness of Constitutional drugs having in the treatment of Diabetes Mellitus. The most effective was Nat.mur, Calc.carb and Phos.acid. followed by other drugs.

8.2 DISCUSSION

To arrive at a valid conclusion, I am indebted to discuss some of the findings that have evolved out of this study. The result is exclusively based on the observation and result presented in former section.

1. Age incidence: The incidence was maximum in the age group 40-50 i.e 46.6%, The next greater prevalence was in age group 30-40. i.e 26.6%.
2. Sex Predominance : In study Female out numbered males.
2. Domicile: Rural population amount to 56.6% % and urban population 43.3 %.

3. Distribution of patients according to socio economic class:-In this study conducted, Diabetes mellitus is found more among poor class (66.7%) followed by middle class.It showing life situation and environment plays a significant role.
4. Duration of Illness of Majority was 1-5 years followed 5-10 years.
5. The Family History of Diabetes was noted in 21 cases i.e 70% cases compared with %30 having no family history of Diabetes. Showing strong Genetic Trait.
6. Distribution of clinical features: Among the symptoms given Weakness was dominating feature, Weight loss, Burning Sensation followed by Increased thirst,and Increased hunger.
7. The Miasmatic Dominance noted was Psora(53%),followed by Sycosis 28% and Syphilis 19%.Diabetes Mellitus has involvement of all 3 miasms.,in varied proportions.
8. Evaluation of change in disease criteria: The comparison of the FBS measurement before and after treatment showed statistically significant result.
9. Stressful life events associated in a major number cases were Death of closed loved ones, resulting in significant period of grief, which was followed by Stressful periods due to financial difficulties.
10. Medicines used: Medicines were selected on basis on constitutional similarity also taking into consideration of stress factors. Among 30 cases medicine indicated most of times is Nat.mur- 23%. Then Calc.carb 10%, followed by Phos.acid, Ars.alb, and, Lyco, 10%, Aur.met, Caust, Mag.mur 7%,Phos &Sep 3% This shows the effectiveness of Constitutional drugs in the treatment of Diabetes Mellitus.

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