REVIEW ARTICLE

REVIEW ON IMMUNE MEDIATED (TYPE I DIABETES MELLITUS, INSULIN DEPENDENT DIABETES MELLITUS (IDDM))

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Abstract:
Diabetes mellitus describes a metabolic disorder of multiple aetiology. It is characterized by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, action or both. Several pathogenetic processes are involved in the development of diabetes. These include processes which destroy the beta cells of the pancreas with consequent insulin efficiency, 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 tissues resulting from insensitivity or lack of insulin. Type I diabetes mellitus results from autoimmune destruction of the insulin-producing cells of the pancreas. It indicates the processes of beta cells destruction that may ultimately lead to diabetes mellitus in which insulin is required for survival as it keeps the blood sugar level under control. High levels of glucose are responsible for the symptoms and complications of the disease.

Key Words: Type 1 diabetes mellitus, causes, Management, clinical and metabolic features Type 1 diabetes mellitus.

Introduction
The term diabetes mellitus describes a metabolic disorder of multiple aetiology. It is characterized by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, action or both (WHO, 1999). It has also been defined by the World health organization (WHO) on the bases of laboratory findings; as a fasting venous plasma glucose concentration greater than 7.8 mmol/L or greater than 11.1 mmol/L 2 hours after a carbohydrate intake or oral ingestion of 75mg of glucose even if the fasting concentration is normal (WHO). 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 the absence of effective treatment, death. Most at times, symptoms are not severe or may be absent, and consequently hyperglycemia sufficient to cause pathological and functional changes may be present for a long time before 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 ulcer, amputation, charcot joint, 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 efficiency, 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 tissues resulting from insensitivity or lack of insulin (Stoffers et al., 1997).
CLASSIFICATION OF DIABETES MELLITUS:
The first widely accepted classification of diabetes mellitus was published by WHO in 1980 and 1985 (WHO 1985). The 1980 and 1985 classifications of diabetes mellitus and allied (categories of glucose intolerance include clinical classes and two statistical risk classes. The 1980 expert committee proposed two major classes of diabetes mellitus and named them IDDM or type I, and NIDDM or type 2. In the 1985 study group report, the terms type I and 2 -were omitted, but the classes IDDM and NIDDM were retained. In both the 1980 and 1985 reports other classes of diabetes were included such as impaired glucose tolerance (IGT) as well as gestational diabetes mellitus (GDM). These were reflected in the subsequent international nomenclature of disease (IND) in 1991 and the tenth revision of the international classification of disease in 1992 the 1985 classification was widely accepted and is used internationally. It represents a comprise between clinical and aetiological classification and allowed classification of individual subjects and patients in a clinically useful manner even when the specific cause or etiology was unknown. The recommended classification includes both staging of diabetes mellitus based on clinical descriptive criteria and a complementary aetiological classification.

Revised classification encompasses both clinical states and aetiological types of diabetes mellitus and other categories of hyperglycaemia, as suggested. Moreover, individual subjects may move from stage to stage in either direction. Persons who have or develop diabetes mellitus can be categorized by stage according to the clinical characteristics, even in the absence of information concerning the underlying aetiology-the classification by aetiological type results from improved understanding of the cause of diabetes mellitus. The new classification contains stages, which reflect the various degrees of hyperglycaemia in individual subjects with any of the disease processes which may lead to diabetes mellitus. All subjects with diabetes mellitus can be categorized according to clinical stages and this is achievable in all circumstances. The stage of glycaemia may change over time depending on the extent of the underlying disease processes. The disease process may be present but may not have progressed far enough to cause hyperglycaemia. The aetiological classification reflects the fact that the defect or process which may lead to diabetes may be identifiable at any stage in the development of diabetes.

TYPE I DIABETES
This encompasses the majority of cases which are primarily due to pancreatic islet beta - cell destruction and are prone to keto acidosis. Type I diabetes mellitus results from autoimmune destruction of the insulin-producing cells of the pancreas. It indicates the processes of beta cells destruction that may ultimately lead to diabetes mellitus in which insulin is required for survival as it keeps the blood sugar level under control. High levels of glucose are responsible for the symptoms and complications of the disease. However, most of the insulin-producing cells are destroyed before the patient develops symptoms of the disease. It has therefore been reported that most individuals with type I diabetes may be metabolically normal before the disease clinically manifests, but the process of beta cell destruction can be detected.

Type I is usually characterized by the presence of anti-gulatonic acid decarboxylase, Islet cell or insulin anti-bodies which identify the auto-immune processes that lead to betacell destruction. It has equally been reported that subjects with this clinical form of diabetes, particularly non-caucasians, show no evidence of an autoimmune disorder is demonstrable and these are classified as type I idiopathic. Aetiological classification may be possible in some circumstances and not in others. Thus, the aetiological type I process can be identified and sub-categorized if appropriate antibody determination are performed. The rate of beta cell destruction of the pancreas as stated earlier is quite variable, being rapid in some individuals and slow in others (Zimmet et al., 1994). The rapidly' progressive form is commonly observed in children, but also may occur in adults (Humphrey et al., 1998). The slowly progressive form generally occurs in adults and is sometimes referred to as Latent Autoimmune Diabetes in adults. Some patients particularly children and adolescent, may present with keto acidosis as the first manifestation of the disease. Others have modest fasting hyperglycaemia and or keto acidosis in the presence of steers or infection or other stress. Still others, particularly, adults, may retain residual beta cell function, sufficient to prevent keto acidosis for many years (Zimmet, 1995). Individuals with this form of type I diabetes often oecome dependent on insulin for survival eventually and are at risk of keto acidosis (Willis et al., 1995). This stage of the disease, there is little or no insulin secretion as manifested by low or undetectable levels of plasma c-peptide.

Makers of immune destruction, including Islet of cell autoantibodies, and/or auto antibodies to insulin and autoantibodies to glutamic acid decarboxylase are present in 85-90% of individuals with type I diabetes mellitus when fasting diabetic hyperglycaemia is initially detected (Verge et al.,1996).

The peak incidence of this type of type I diabetes occurs in childhood and adolescence but the onset may occur at any age, ranging from childhood to the ninth decade of life (Molbak et al., 1994). There is a genetic predisposition of autoimmune destruction of beta cells, and it is also related to environmental factors that are still poorly defined. Although patients are not usually 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.

**Epidemiology of Type I Diabetes Mellitus:**
Type I diabetes mellitus accounts for about 10% of all diabetes, affecting approximately 1.4 million people in the U.S and 10 - 20 million globally (Rewers 1991., Libman et al., 1993). About 40 percent of persons with type I diabetes develops the disease before 20 years of age thus making it one of the most common severe chronic diseases of childhood. In the U.S where 30,000 new cases occur each year, type I diabetes affects 1: 300 children and as many as 1: 100 adult during the life span.

Type I diabetes is the leading cause of renal disease, blindness, amputation, and a major cause of cardiovascular disease and premature death in the general population (Chase et al., 2004). The prevalence of type I diabetes in children aged less than 15 years ranges from 0.05 to 0.03% in most European and North American population (Libman et al.,1993).

In the 1990's, the number of type I diabetic patients 0-9 years of age in the United States was estimated at approximately 123,000 individuals.

However, in 2000, with the growth of this segment of the US population to over 80 million, there were approximately 160,000 children with type I diabetes. One of the most striking characteristics of type I the large geographic variability in the incidence (Karvonen et al., 2002).

For instance, Scandinavia and the Mediterranean of Sardinia have the highest incidence rates in the world while oriental population has the lowest rates. A child in Finland is 400 times more likely to develop diabetes than one in China. While there is a strong south-north gradient in the incidence, the geographic and ethnic variations in type I diabetes reflect the prevalence of susceptibility genes or that of causal environmental factors or both. In the general population, the prevalence of B cell autoimmunity appears to be roughly proportional to the incidence of type I diabetes in the populations. In contrast, the prevalence of B cell autoimmunity in first degree relatives of type I diabetic persons does not differ dramatically between high and low risk countries. The incidence of B cell autoimmunity is higher in relatives younger than 5 years compared to those 5-9 years old (Adojaan et al., 1996).

The clinical picture of the disease is similar in low and high risk areas making it unlikely that the inter-population difference is due to misclassification of different types of diabetes. However, in many populations, type 2 diabetes is an increasing or already the predominant form of diabetes in children making correct diagnosis and treatment increasingly difficult. Type I diabetes incidence peaks at the ages of 2, 4-6 and 10 - 14 years, perhaps due to alterations in the pattern of infections or increase in insulin resistance. Recently published data suggest that in many populations, the highest rate of incidence is observed in the 10-4 age group, while the highest annual increase in the 0-4 years age children. The age distribution of onset of type I diabetes similar across geographic areas and ethnic groups. There have only been a few studies that incidence of type I diabetes in adults, mainly because of the difficulty of distinguishing type I from insulin requiring type 2 diabetes in older individuals. The incidence decreases in the third decade of life.

It has been speculated that the incidence of type I diabetes increases again in the fifth through seventh decades of life though there is no hard evidence of such increase, and it is not known whether there are etiologic differences between childhood and adult onset type I diabetes. The incidence data from early 1990 suggested a significant racial difference in type I diabetes risk in multiracial populations, although not of the same magnitudes as the geographic differences. In the US, non-Hispanic whites were about one and a half times as likely to develop type I diabetes as African Americans (Dabelea, 2005) or Hispanics (Kostraba et al., 1992). This was similar to the differences reported from Montreal, where children of British decent had about one and a half the risk of type I diabetes in children of French decent.

Recent data from such study have shown a rising trend in type I diabetes not only in non-Hispanic white population, but also among Hispanic and African American Children (Dabelea, 2005).

In fact however, the number of new type I diabetes patients of African-American origin aged 10-14 years has risen significantly during the last 10-15 years and the incidence of type I diabetes in this age group is almost equal in the white and African American population, it is still almost two fold difference between non - Hispanic white and African - American children in the incidence of type I type in young children 0 - 9 years (Rewers et al., 2004).

It was suspected that the marked increase in incidence in the African American population may be in part due to misclassification of cases actually having type 2 diabetes, as there is an epidemic of type 2 diabetes in children in the US, which largely affects African - American children over the age of 10 years and many cases of type 2 diabetes require treatment with insulin at the time of diagnosis.

Similarly to this observation, the high annual increase in the incidence of type I diabetes has been recently reported among the children of south Asian immigrants (Indian, Pakistani, Bangladesh in UK) (Dabelea, 2005). Children
AETIOLOGY

Genetic Factors

In family history of type 1 diabetes, in moderate type 1 diabetes areas, such as the United States, the risk of type 1 diabetes by the age of 20 years is approximately 1:300. The risk is increased to about 1:50 in offspring of type 1 diabetes mothers and 1:15 in offspring of type 1 diabetes fathers, the reason for this parental gender is not known. The risk of siblings of type 1 diabetes probands ranges from 1:12 to 1:35 (Alien et al., 1999) and is further increased, in HLA-identical siblings (Redondo et al., 1999).

The primary loci of genetic susceptibility to type 1 diabetes have been mapped to the HLA-DR, DQ and recently also to the OP region (Cruz et al., 2004). While 50% of non Hispanic whites in the United States have HLA OR3 or OR4 allele, at least one of these allele is present in 95% of patients with type 1 diabetes (Rewers and Zimmet, 2003). The estimated risk for general population of children who have the HLA-OR3/4 genotype is approximately 1:15, (Rewers et al., 1996). Only 2.4% of the general population carries this genotype, compared to 30-40% of type 1 diabetes patients. No particular HLA type seems to be associated with B-cell autoimmunity, although associations between different patterns of insulin auto antibodies and HLA OR phenotypes have been reported, (Barker et al., 2004; Kukko et al., 2004).

Environmental Factors: Twin and family studies indicate that genetic factors alone cannot explain the aetiology of type 1 diabetes. Seasonally, increase and epidemics of type 1 diabetes as well as numerous ecological, cross-sectional and retrospective studies suggest that certain viruses and components of early childhood diet may cause type 1 diabetes.

An increased incidence of type I diabetes in patients with congenital rubella syndrome (CRS) is particularly interesting. While CRS is responsible for a minute proportion of type 1 diabetes and there is little evidence that postnatal rubella exposure to the wild strain causes type I diabetes, CRS provides an example of viral persistence leading to type I diabetes. The incubation period of type I diabetes in CRS patients is 5-20 years and persistent rubella virus infection of the pancreas has been demonstrated in some cases. While CRS is not associated with
particular HLA-DR alleles, the distribution of the HLA-DR3 and 4 alleles among patients with CRS and diabetes resembles that in non-CRS type 1 diabetes patients.

Finally, a molecular mimicry has been reported between a rubella virus protein and a 52kD B-cell autoantigen. The evidence is strongest for picornaviruses, which include human (enteroviruses and retroviruses) and animal pathogens. Enteroviruses have been most strongly linked to human type I diabetes, but convincing proof of causality remains elusive, epidemics of type I diabetes associated with concurrent epidemics of enteroviruses and multiple cross-sectional seroepidemiological studies have been suggestive, but not entirely convincing. At least 90% of type I diabetes patients demonstrate prolonged period of B-cell autoimmunity that is hardly compatible with an acute cytolytic enteroviral infection being a major cause. Enteroviral infection could, however, nitrate B-cell autoimmunity through molecular mimicry or a persistent B-cell infection with impairment of insulin secretion and expression of self-antigens.

Cross-sectional studies of anti-Coxsackie antibodies in B-cell autoimmunity have been weak and inconclusive and have been recently replaced by studies based on detection of picornaviral RNA in bodily fluids using polymerase chain reaction PCR.

Prospective studies of non-diabetic relatives and general population children found a strong relation between enteroviral infections, defined by PCR, and development of islet autoantibodies in Finland have suggested uterine infections can lead to type 1 diabetes in a significant proportion of the cases (Lonrot et al., 2000). A meta-analysis found a 50% increase in type 1 diabetes risk associated with a breastfeeding duration of less than 3 months, and exposure to breast-milk substitutes prior to 3 months of age, but a subsequent meta-analysis reported much lower risk estimates. Breasfteeding may be viewed as a surrogate for the delay in the introduction of diabetogenic substances present in formula or early childhood diet. The reports that newly diagnosed diabetic children, compared with age-matched controls, have higher levels of serum antibodies against cow's milk and betaactoglobulin as well as against bovine serum albumin have been difficult to reproduce. More recent cohort studies failed to find an association between mfant diet exposures and beta cell autoimmunity.

Interestingly, a study from Finland suggested that current cow's milk consumption was more closely linked to pre-diabetic 'autoimmunity and diabetes than infant exposure (Couper et al., 1999).

It has been recently discovered by two prospective large studies (DAISY and German BABYDIAB) that early ingestion of cereals between ages 0 and 3 months contribute to increased development of anti-islet auto-antibodies in offspring of type 1 diabetic mothers and children (relatives of TIDM subjects and from general population) with high risk HLA-DR3/4, genotypes. There is increasing evidence that vitamin D might contribute to pathogenesis and prevention of type 1 diabetes. Active vitamin D prevents type 1 diabetes in animal models, modifies T-cell differentiation, modulates T cell action and modulates cytokine secretion, shifting the balance to regulatory T cell. Maternal intake of vitamin D in food during pregnancy was significantly associated with a decreased risk of islet autoimmunity appearance in offspring. Recent evidence from the large European studies suggests that vitamin D deficiency in pregnancy increases the incidence of type 1 diabetes in offsprings and that high dose vitamin D supplementation early in life have a protective effect eicosapentaenoic acid and docosahexaenoic acid, during the first year of life were found to significantly reduce risk for disease. It seems possible that birth seasonality in children and/or the presence of seasonal pattern as diagnosis of type 1 diabetes could be explained by variation in endogenous vitamin D production during different year season. The monthly averages of maximal daily temperature and daily hours of sunshine were inversely related to the number of new patients per month in Belgium.

Chemical Compounds (streptozotocin or dietary nitrates and nitroazines induce B-cell autoimmunity in animal models. Circumstantial evidence suggests a connection between type 1 diabetes and consumption of foods and water containing nitrates, nitrates or nitroazines. Multiple hits of dietary B-cell toxins may render genetically resistant individuals susceptible to diabetogenic virus leading to type 1 diabetes.

It has recently hypothesized that excess weight gain and increase in insulin resistance in early childhood is a trigger event, which initiates the autoimmunity leading to B-cell destruction and type 1 diabetes development. The rising blood glucose (glucotoxicity) accelerates beta-cell apoptosis directly or by inducing beta-cell immunogens in genetically predisposed subjects. This so called Accelerator Hypothesis seems to be supported by several epidemiology case-control and population based cohort studies. Study from Norway found almost linear correlation between incidence rate of type 1 diabetes and birth weight. The risk of type 1 diabetes was higher for more than twofold in children with birth weight 4500g in comparison to newborns with the lowest birth date -and sex matched controls (In this nationwide case-control study ten percent increment in relative weight was associated with a 50-60% increase in the risk of type 1 diabetes before 3 years of age and a 20-40% increase from 3 to 10 years of age. The most recent epidemiological observations suggest that high birth weight could possibly result from a moderating effect on intrauterine growth of HLA genotypes conferring a high risk of diabetes.
The wide variation in childhood type 1 diabetes incidence rate within the different populations could also be partially explained by indicators of national and individual prosperity. These indicators could reflect differences in environmental risk factors such as nutrition or lifestyle that are important in determining the risk of type 1 diabetes. The EURODIAB study has shown a positive association of the incidence rates with the value of gross domestic product.

Type 1 diabetes is likely caused by an interactive effect of genetic and environmental factors within a limited age-window. While both the susceptibility genes and the candidate environmental exposures appear to be quite common, the disease is still uncommon, raising a possibility of low penetrance. In mice, the host's genes restrict the diabetogenic effect of picornaviruses in a manner compatible with recessive trait not -related to the major histocompatibility complex. In humans, on the other hand, susceptibility of diabetogenic enteroviruses appears to be genetically restricted by HLA - DR and DQ alleles viral type and epidemicity. In general, the HLA-DR3 allele, present in most patients with type 1 diabetes, is associated with viral persistence. Stress may progress the development of type 1 diabetes by stimulating the secretion of counter-regulatory hormones and possibly by modulating immune system.

CLINICAL AND METABOLIC FEATURES OF IDDM
Most of the metabolic changes in IDDm are caused by insulin deficiency.

Usually there is hyperglycaemia where the plasma glucose concentration exceeds 11mmol/L, there will be glucosuria which produces an osmotic diuresis which causes polyuria. Cerebral cellular dehydration due to hyperosmolality, secondary to hyperglycaemia causes thirst polydipsia. There is also abnormalities in lipid metabolism. Lyposis is enhanced and plasma free fatty acid concentration rises. The rate of cholesterol synthesis may be increased and increased breakdown of protein may cause muscle wasting.

Coronary artery disease is the main cause of death in persons with Type I diabetes and accounts for a large proportion of premature morbidity and mortality in the general population (Costacou et al., 2003). Diabetes retinopathy is the most common cause of new cases of blindness as reported in the US, about 80% of type 1 diabetic patient develop diabetic retinopathy and 25% develop vision threatening proliferative diabetic retinopathy. This is usually caused by alterations in renal blood flow and loss of retinal pericytes.

Kidney disease is associated with several abnormalities, including proteinuria and progressive renal failure. The presence of small amounts of glycation of haemoglobin and plasma protein may occur. Infections are common in diabetic patients and may aggravate renal and peripheral vascular disease. Infants born to women with poorly controlled diabetes tend to be large at birth and to have an increased incidence of fetal abnormalities.

Diabetic ketoacidosis is a more severe form of the metabolic events outlined above. It may be precipitated by infection or by vomiting. In the absence of insulin, there is increased lipid and protein breakdown, enhanced hepatic gluconeogenesis and impaired glucose entry into the cells. Numerous previous studies have demonstrated that increased systolic or diastolic blood pressure is a powerful predictor of macro vascular complications (Chase et al., 2001) coronary artery disease is the main cause of death in these persons.

Anaemia is a common complication. It is often more severe and occurs at an earlier stage in patients with diabetic nephropathy. Numerous studies have addressed the interaction between diabetes and renal failure in its pathogenesis. The anaemia associated with nephropathy results from EPO deficiency, which seems to develop in patients with type 1 diabetes who have relatively normal levels of serum creatinine. Early EPO deficiency anaemia occurs in both types 1 and 2 diabetes with the prevalence higher in type 1 diabetes (Bosman et al., 2001). There is also a greater prevalence of EPO deficiency anaemia associated with the presence of autonomic neuropathy in diabetic patients in most studies to date, the predominant risk factor for the development of anaemia in a diabetic population has been found to be the presence of renal disease impaired renal function or albuminuria (Tomas et al., 2004). One of the most potent causes of suboptimal red cell response to erythropoietin EPO is chronic and overt inflammation associated with an increased production of cytokines such as tumor necrosis factor inter leukin 1 or interferon which might suppress erythrocyte stem cell proliferation.

DIAGNOSIS OF IDDMs
The diagnosis of diabetes mellitus can be made with a fair degree of confidence in individuals with the classical symptoms of the disease and elevated fasting or postprandial levels of blood glucose and glucosuria. The blood insulin level may also be used in the diagnostic workup of individuals who are suspected of diabetes.

The most commonly used screening tests are the determination of the fasting blood sugar level or the two hour post prandial blood glucose level, while the most common diagnostic one is the oral glucose tolerance test (OGTT). The normal levels for fasting blood glucose vary between 7.8mmol/L to 11.1mmoI/L of blood for both
fasting and two hour postprandial test. Levels in excess of 11.1 mmol/L are indicative of diabetes mellitus, while values between 7.8 mmol/L to 11.1 mmol/L.

**Urine Testing**
Testing the urine for glucosuria is another method of diagnosis. Four kinds of urine can be chosen for testing namely: 24 hour specimen, fractional specimen (24 hour collected 4 times), the first voided specimen or the secondvoided specimen. 24 hour reveals how much glucose is lost in a 24 hour period. This is important because, it reveals the overall control. Fractionating; can pinpoint glucose spill. The first voided is a reasonable compromise for home use, it represents a fractional urine collected in the bladder. The second voided gives information about the blood glucose level only for the moment in time and tells nothing about the situation in the previous ones. Methods used in testing can be clinitest, paper strip and some other methods.

**MANAGEMENT OF IDDM**
The goals of the treatment of IDDM are to prevent excessive postprandial hyperglycemia and, therefore, the symptoms of glucose wastage. To prevent hypoglycemia if the patient is using insulin or oral agent, achieve and maintain ideal body weight in adults and normal growth and development in children; return serum triglycerides and cholesterol to normal levels and prevent or delay large and small blood vessel disease. Depending on the needs of the patient, the goals of therapy may be achieved by diet and insulin or oral hyperglycemic agents. The insulin dependent diabetic will need a diet appropriate to maintain ideal body weight status and growth combined with daily insulin injections.

**DIET THERAPY**
The diet prescription for a patient with diabetes mellitus must be translated in a diet pattern acceptable to the patient. The diet must be nutritionally adequate, maintain normal blood glucose level as far as possible throughout 24 hours and promote desirable weight status in the growth and development of children and adolescent. The American Diabetes Association recommends that 40 percent of carbohydrate derived from cereal grains be recommended for diabetics 15 to 20 calories from protein and 30 percent of calories from FAT.

**Exercise**
Is also important in the management of diabetes as it helps the body burn off some of the excess glucose as energy. Proper care of the foot is necessary since the disease can cut the blood supply to the feet and reduce feelings. Care of the skin or other parts of the body is also very important. Diabetics are less able to resist injury and infections. Counseling the patient by experts equally helps in the management.

**CONCLUSION**
Diabetes mellitus describes a metabolic disorder of multiple aetiology. It is characterized by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, action or both. Several pathogenetic processes are involved in the development of diabetes. This encompasses the majority of cases which are primarily due to pancreatic islet beta - cell destruction and are prone to keto acidosis. Type I diabetes mellitus results from autoimmune destruction of the insulin-producing cells of the pancreas. It indicates the processes of beta cells destruction that may ultimately lead to diabetes mellitus in which insulin is required for survival as it keeps the blood sugar level under control. High levels of glucose are responsible for the symptoms and complications of the disease. However, most of the insulin-producing cells are destroyed before the patient develops symptoms of the disease. It has therefore been reported that most individuals with type I diabetes may be metabolically normal before the disease clinically manifests, but the process of beta cell destruction can be detected. Type I is usually characterized by the presence of anti-gultamic acid decorboxylase, Islet cell or insulin anti-bodies which identify the auto-immune processes that lead to betacell destruction.

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