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REVIEW ARTICLE

A REVIEW OF DIABETES MALLITUS (*Prameha)*

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ABSTRACT

Introduction: Developing country most probably face the problem of lifestyle disorder disease in this situation India is second rank in diabetes Mellitus. Than review of Diabetes Mellitus to understand this disease in present study.

Materials and Methods: References have been collected and relevant matter is compiled from available literature. Available commentaries of present era are also reviewed. All Compiled matter is reorganized and critically analyzed for the discussion and attempt has been made to draw some fruitful conclusion.

Result and Discussion: In the present study Diabetes Mellitus is rapidly growing disease and a review on this disease to understand the causes, classification, Management this are understanding than help to prevent the disease.

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INTRODUCTION

Diabetes is fast gaining the status of a potential epidemic in India with more than 62 million diabetic individuals currently diagnosed with the DISEASE (Joshi and Parikh, 2007; Kumar *et al*., 2013) In 2000, India (31.7 million) topped the world with the highest number of people with diabetes mellitus followed by China (20.8 million) with the United States (17.7 million) in second and third place respectively. According to Wild *et al*. (2004) the prevalence of diabetes is predicted to double globally from 171 million in 2000 to 366 million in 2030 with a maximum increase in India. It is predicted that by 2030 diabetes mellitus may afflict up to 79.4 million individuals in India, while China (42.3 million) and the United States (30.3 million) will also see significant increases in those affected by the disease. (Wild *et al*., 2004; Whiting *et al*., 2011) India currently faces an uncertain future in relation to the potential burden that diabetes may impose upon the country. Many influences affect the prevalence of disease throughout a country, and identification of those factors is necessary to facilitate change when facing health challenges. So what is the factors currently affecting diabetes in India that are making this problem so extreme? The aetiology of diabetes in India is multifactorial and includes genetic factors coupled with environmental influences such as obesity associated with rising living standards, steady urban migration, and lifestyle changes. Yet despite the incidence of diabetes within India, there are no nationwide and few multi-centric studies conducted on the prevalence of diabetes and its complications. The studies that have been undertaken are also prone to potential error as the heterogeneity of the Indian population with respect to culture, ethnicity, and socio- economic conditions; mean that the extrapolation of regional results may give inaccurate estimates for the whole country. According to the Lancet study, China, India and USA are among the top three countries with a high number of diabetic populations. (Source: Think stock Images) Keeping it as a view a permanent Logo was designed that 14th November is the world Diabetes day. The actual aim was to raise public awareness, to highlight the extent of its epidemic cause - symptoms treatment – complication & prevention. It was designated by UN in 1991 as it coincides with the birth anniversary of Frederick Banting who along with Charles Best was responsible for the discovery of Insulin in Oct. 1921. With a little of thinking one can ask that why it kept on 14th Nov. The children's day. Perhaps it is to keep the society disease (DM) free, by maintaining the health of mother and child, by eradicating I in childhood than next generation is healthy. In this review articles we have focus in the major burden in India in face of Diabetes mellitus.

Aims

A review of Diabetes Mellitus in literature

Objectives

The objectives of this research project

1) To study Diabetes mellitus in available literature
2) To study Diabetes Mellitus their symptoms, classification and preventation

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MATERIALS AND METHODS

References have been collected and relevant matter is compiled from available literature. Available commentaries of present era are also reviewed. All Compiled matter is reorganized and critically analyzed for the discussion and attempt has been made to draw some fruitful conclusion.

OBSERVATIONS AND DISCUSSION

Diabetes Mellitus means

The word Diabetes is originated from the French word named "Jiyabatis" which means *punctured pitcher* or a pitcher with a leak, so that the water sprinkles out of it. (The Diabetes – Purshuram Shastry) The word diabetes is derived from the diobos a fountain meaning similar to that of the fountain. The term Diabetes mellitus contains two words i.e. 'Diabetes' and 'Mellitus'. In Greek language the 'Diabetes' means 'to run through a siphon' and the term 'Mellitus' means honey.

Defination

a variable disorder of carbohydrate metabolism caused by a combination of hereditary and environmental factors and usually characterized by inadequate secretion or utilization of insulin, by excessive urine production, by excessive amounts of sugar in the blood and urine, and by thirst, hunger, and loss of weight — compare type 1 diabetes, type 2 diabetes. https:// www.merriam-webster.com/dictionary/diabetes%20mellitus.
Diabetes mellitus is one of the world's major diseases. It currently affects an estimated 143 million people worldwide and the number is growing rapidly. In India, about 5 per cent population suffers from diabetes. Medical health experts assert that regular check-ups and timely detection plays a vital role in controlling and managing the problem. Ironically, due to patient resistance and feeling of disbelief that 'I can have diabetes too', most patients tend to defer on detection and treatment that often leads to complications. Practitioners feel that patient adherence to medication and lifestyle modifications play an important role in diabetes management and this can help them lead a normal life. Un-monitored prevalence of diabetes also results in increased risk of vascular complications like cardiovascular, renal, neural and visual disorders which are related to the duration of the disease.

Table 1. Classification of D.M. in Country-wise

S. No.	Country	Persons with DM (Millions)
1	China	98.4
2	India	65.1
3	USA	24.4
4	Brazil	11.9
5	Russia	10.9

Epidemic of diabetes is spreading from metro cities to smaller towns and even to villages. More than 50 per cent of diabetic subjects are undiagnosed. Nearly 20 per cent of adult patients in a general medical OPD will have diabetes but more than half of these patients are unaware of their disease.

Clinical features

DM and its complications produce a wide range of symptoms and signs; those secondary to acute hyperglycemia may occur at any stage of the disease, whereas those related to chronic complications begin to appear during the second decade of hyperglycemia. Individuals with previously undetected type 2 DM may present with chronic complications of DM at the time of diagnosis. The history and physical examination should assess for symptoms or signs of acute hyperglycemia and should screen for the chronic complications and conditions associated with DM. History A complete medical history should be obtained with special emphasis on DM-relevant aspects such as weight, family history of DM and its complications, risk factors for cardiovascular disease, prior medical conditions, exercise, smoking, and ethanol use. Symptoms of hyperglycemia include polyuria, polydipsia, weight loss, fatigue, weakness, blurry vision, frequent superficial infections (vaginitis, fungal skin infections), and slow healing of skin lesions after minor trauma. Metabolic derangements relate mostly to hyperglycemia (osmotic diuresis, reduced glucose entry into muscle) and to the catabolic state of the patient (urinary loss of glucose and calories, muscle breakdown due to protein degradation and decreased protein synthesis). Blurred vision results from changes in the water content of the lens and resolves as the hyperglycemia is controlled. In a patient with established DM, the initial assessment should also include special emphasis on prior diabetes care, including the type of therapy, prior HbA1c levels, self-monitoring blood glucose results, frequency of hypoglycemia, presence of DM-specific complications, and assessment of the patient's knowledge about diabetes. The chronic complications may afflict several organ systems, and an individual patient may exhibit some, all, or none of the symptoms related to the complications of DM (see above). In addition, the presence of DM-related comorbidities should be sought (cardiovascular disease, hypertension, dyslipidemia).

Type I Diabetes Mellitus : It usually begins before age 40 years. This type of Diabetes is characterized by a rapid onset, with symptoms such as Polydypsia, Polyuria, Polyphagia, loss of weight and strength. In the fulminating case, the most striking features are those of salt and water depletion i.e. loose dry skin, furred tongue, cracked lips, tachycardia, hypotension and reduced intraocular pressure. Breathing may be deep and sighing due to acidosis, the breath is usually fetid and the sickly sweet smell of acetone may be apparent. Once the symptoms develop, Insulin therapy is required. Occasionally an initial episode of Ketoacidosis is followed by a symptom free interval (the "honeymoon" period), during which no treatment is required.

Type II Diabetes Mellitus : It may remain asymptomatic as long as complication can occur. It usually begins in middle life or later. The typical patient is overweight. Symptoms begin gradually. Pruritis vulvae or balanitis is a common presenting symptoms since the external genitalia are especially prone to infection by fungi (candida) which flourish on skin and mucous membranes contaminated by glucose. Blurred or decreased vision due to retinopathy is found. Depression or loss of tendon reflexes at the ankles and impaired perception of vibration sensation distally in the legs indicate neuropathy. Hypertension and signs of atherosclerosis are common and may include diminished or impalpable pulses in the feet, bruits over the carotid or femoral arteries and gangrene of the feet. Signs of water and salt depletion with associated mental changes may be seen in cases with severe hyperglycemia. Diabetes Mellitus is defined as the state of chronic hyperglycemia due to impairment of insulin secretion or its

action. Diabetes mellitus (DM) comprises a group of common metabolic disorders that share the phenotype of hyperglycemia. Several distinct types of DM exist and are caused by a complex interaction of genetics, environmental factors, and life-style choices. Depending on the aetiology of the DM, factors contributing to hyperglycemia may include reduced insulin secretion, decreased glucose usage, and increased glucose production. The metabolic deregulations associated with DM causes secondary path physiologic changes in multiple organ systems that impose a tremendous burden on the individual with diabetes and on the health care system. In the United States, DM is the leading cause of end-stage renal disease, non traumatic lower extremity amputations, and adult blindness. With an increasing incidence worldwide, DM will likely continue to be a leading cause of morbidity and mortality for the foreseeable future.

Classification

Recent advances in the understanding of the aetiology and pathogenesis of diabetes have led to a revised classification. Presently the disease is classified on the basis of the physiological status.

I. Type 1 diabetes (-cell destruction, usually leading to absolute insulin deficiency)

A. Immune-mediated
B. Idiopathic

II. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretary defect with insulin resistance)

III. Other specific types of diabetes

A. Genetic defects of -cell function characterized by mutations in:

1. Hepatocyte nuclear transcription factor (HNF) 4 (MODY 1)
2. Glucokinase (MODY 2)
3. HNF-1 (MODY 3)
4. Insulin promoter factor (IPF) 1 (MODY 4)
5. HNF-1 (MODY 5)
6. Mitochondrial DNA
7. Proinsulin or insulin conversion

B. Genetic defects in insulin action

1. Type A insulin resistance
2. Leprechaunism
3. Rabson-Mendenhall syndrome
4. Lipoatrophic diabetes

C. Diseases of the exocrine pancreas, pancreatitis, pancreatectomy, neoplasia, cystic fibrosis, hemochromatosis, fibrocalculous pancreatopathy

D. Endocrinopathiesacromegaly, Cushing's syndrome, glucagonoma, pheochromocytoma, hyperthyroidism, somatostatinoma, aldosteronoma

E. Drug- or chemical-inducedVacor, pentamidine, nicotinic acid, glucocorticoids, thyroid hormone, diazoxide, adrenergic agonists, thiazides, phenytoin, -interferon, protease inhibitors, clozapine, beta blockers

F. Infections congenital rubella, cytomegalovirus, coxsackie

G. Uncommon forms of immune-mediated diabetes “stiff-man" syndrome, ant insulin receptor antibodies.

H. Other genetic syndromes sometimes associated with diabetes Down’s syndrome, Klinefelter's syndrome, Turner's syndrome, Wolfram's syndrome, Friedreich's ataxia, Huntington's chorea, Laurence-Moon-Biedl syndrome, myotonic dystrophy, porphyria, Prader-Willi syndrome

IV. Gestational diabetes mellitus (GDM)

Differential diagnosis

The differential diagnosis of the Diabetes mellitus can be done with following conditions :

1) Hyperglycemia due to Tissue insensitivity to Insulin

- Hormonal tumors – Acromegaly, Pheochromocytoma etc.
- Liver diseases – Cirrhosis, Hemochromatosis etc.
- Muscle disorder – Myotonic disorders
- Pharmacologic agents – Sympathmimetic drugs etc
- Insulin receptor disorders

2) Hyperglycemia due to reduced Insulin secretion

- Hormonal tumors
- Pharmacological agents

Insulin biosynthesis, secretion, and action biosynthesis

Insulin is produced in the beta cells of the pancreatic islets. It is initially synthesized as a single-chain 86-amino-acid precursor polypeptide, reproinsulin. Subsequent proteolytic processing removes the aminoterminal signal peptide, giving rise to proinsulin. Proinsulin is structurally related to insulin-like growth factors I and II, which bind weakly to the insulin receptor. Cleavage of an internal 31-residue fragment from proinsulin generates the C peptide and the A (21 amino acids) and B (30 amino acids) chains of insulin, which are connected by disulfide bonds. The mature insulin molecule and C peptide are stored together and consecrated from secretary granules in the beta cells. Because the C peptide is less susceptible than insulin to hepatic degradation, it is a useful a marker of insulin secretion and allows is crimination of endogenous and exogenous sources of insulin in the valuation of hypoglycaemia. Human insulin is now produced by recombinant DNA technology; structural alterations at one or more residues are useful for modifying its physical and pharmacologic characteristics (see below).

Secretion

Glucose is the key regulator of insulin secretion by the pancreatic beta cell, although amino acids, ketones, various nutrients, gastrointestinal peptides, and neurotransmitters also influence insulin secretion. Glucose levels 3.9 mmol/L (70 mg/dL) stimulate insulin synthesis, primarily by enhancing protein translation and processing, as well as inducing insulin secretion. Glucose stimulates insulin secretion through a series of regulatory steps that begin with transport into the beta cell by the GLUT2 glucose transporter. Glucose phosphorylation

by glucokinase is the rate-limiting step that controls glucose-regulated insulin secretion. Further metabolism of glucose-6-phosphate via glycolysis generates ATP, which inhibits the activity of an ATP-sensitive K+ channel. This channel is a complex of two separate proteins, one of which is the receptor for certain oral hypoglycaemic (e.g., sulfonylureas, meglitinides); the other subunit is an inwardly rectifying K channel protein. Inhibition of this K+ channel induces beta cell membrane depolarization, opening of voltage-dependent calcium channels (leading to an influx of calcium), and stimulation of insulin secretion. Careful studies of insulin secretory profiles reveal pulsatile pattern of hormone release, with small secretory bursts occurring about every 10 min, superimposed upon greater amplitude oscillations of about 80 to 150 min. Meals or other major stimuli of insulin secretion induce large (four- to fivefold increase versus baseline) bursts of insulin secretion that usually last for 2 to 3 h before returning to baseline. Derangements in these normal secretory patterns are one of the earliest signs of beta cell dysfunction in DM (see below).

Action

Once insulin is secreted into the portal vein, ~50% is removed and degraded by the liver. Unrestricted insulin enters the systemic circulation and binds to its Receptor in target sites. The insulin receptor belongs to the tyrosine kinas' class of membrane-bound receptors. Insulin binding to the receptor stimulates intrinsic tyrosine kinas' activity, leading to receptor utophosphorylation and the recruitment of intracellular signaling molecules, such as insulin receptor substrates (IRS) 1 and 2. These and other adaptor proteins initiate a complex cascade of phosphorylation and dephosphorylation reactions, ultimately resulting in the widespread metabolic and mitogenic effects of insulin. As an example, activation of the phosphatidylinositol-3-kinase (PI-3 kinase) pathway stimulates translocation of glucose transporters (e.g., GLUT4) to the cell surface, an event that is crucial for glucose uptake by skeletal muscle and fat. Activation of other insulin receptor signaling pathways induces glycogen synthesis, protein synthesis, lipogenesis, and regulation of various genes in insulin-responsive cells. Glucose homeostasis reflects a precise balance between hepatic glucose production and peripheral glucose uptake and utilization. Insulin is the most important regulator of this metabolic equilibrium, but the effects of other pathways including neural input, metabolic signals, and hormones (e.g., glucagon) result in integrated control of glucose supply and utilization. In the fasting state, low insulin levels promote hepatic luconeogenesis and glycogenolysis to prevent hypoglycemia. Low insulin levels decrease glycogen synthesis, reduce glucose uptake in insulin-sensitive tissues, and promote mobilization of stored precursors. Reduced insulin levels are also permissive in allowing glucagon to stimulate glycogenolysis and gluconeogenesis by the liver and renal medulla. These processes are of critical importance to ensure an adequate glucose supply for the brain. Postprandially, a large glucose load elicits a rise in insulin and fall in lucagon, leading to a reversal of these processes. The major portion of postprandial glucose is utilized by skeletal muscle. Other tissues, most notably the brain, utilize glucose in an insulin-independent fashion.

Diagnosis

- Diabetes may be diagnosed on the basis of one abnormal plasma glucose (random ≥11.1 mmol/L or fasting ≥7 mmol/L) in the presence of diabetic symptoms such as thirst, increased urination, recurrent infections, weight loss, drowsiness and coma.
- In asymptomatic people with an abnormal random plasma glucose, two fasting venous plasma glucose samples in the abnormal range (≥7 mmol/L) are recommended for diagnosis.
- Two-hour venous plasma glucose concentration ≥11.1 mmol/L two hours after 75 g anhydrous glucose in an oral glucose tolerance test (OGTT).
- The World Health Organization (WHO) now recommends that glycated haemoglobin (HbA1c) can be used as a diagnostic test for diabetes. An HbA1c of 48 mmol/mol (6.5%) is recommended as the cut-off point for diagnosing diabetes. A value less than 48 mmol/mol does not exclude diabetes diagnosed using glucose tests. (Arora *et al*., 2010)

Management

The management plan for a person with diabetes includes: (Zargar *et al*., 2000)

- Diabetes education: structured education and self-management (at diagnosis and regularly reviewed and reinforced) to promote awareness.
- Diet and lifestyle: healthy diet, weight loss if the person is overweight, smoking cessation, regular physical exercise.
- Maximising glucose control while minimising adverse effects of treatment, such as hypoglycaemia.
- Reduction of other risk factors for complications of diabetes, including the early detection and management of hypertension, drug treatment to modify lipid levels and consideration of ant platelet therapy with aspirin.
- Monitoring and early intervention for complications of diabetes, including cardiovascular disease, feet problems, eye problems, kidney problems and neuropathy.

Conclusion

The goals of therapy for type 1 or type 2 DM are to: (1) eliminate symptoms related to hyperglycemia, (2) reduce or eliminate the long-term microvascular and macrovascular complications of DM, and (3) allow the patient to achieve as normal a life-style as possible. To reach these goals, the physician should identify a target level of glycemic control for each patient, provide the patient with the educational and pharmacologic resources necessary to reach this level, and monitor/treat DM-related complications. Symptoms of diabetes usually resolve when the plasma glucose is 11.1 mmol/L (200 mg/dL), and thus most DM treatment focuses on achieving the second and third goals.

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