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# In Shortly About Diabetes Mellitus

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**Abstract**

**Diabetes mellitus is a chronic, incurable systemic disorder characterized by permanently elevated blood sugar. It is caused by hereditary factors, and is due to a decreased secretion or reduced biological action of the hormone insulin, or a combination of these two factors. This deficiency interferes with the exchange of carbohydrates, fats and proteins in the body, which is manifested by typical problems, and after a long time effects on the structure and function of blood vessels, nerves and other vital organs and organ systems. Diabetes is one of the most common endocrinological diseases today. It is steadily increasing, especially in developed countries of the world, as a result of modern lifestyles and an increase in external etiological factors, among which obesity is particularly prominent. Diabetes mellitus most commonly occurs in older life as a result of general degenerative and sclerotic changes in the body.**

**Keywords:** ***Diabetes Mellitus; Type 1; Type 2; Insulin***

## 1. Introduction

Diabetes mellitus is defined as a metabolic disorder of multiple aetiology characterized by chronic hyperglycemia with disturbances of carbohydrate, protein and fat metabolism resulting from deficiency in insulin secretion, insulin action, or both [1]. The clinical diagnosis of diabetes is often indicated by the presence of symptoms such as polyuria, polydipsia and unexplained weight loss, and is confirmed by documented hyperglycemia.

The clinical presentation ranges from asymptomatic type 2 diabetes to the dramatic life-threatening conditions of diabetic ketoacidosis (DKA) or hyperosmolar non-ketotic coma (HONK)/hyperosmolar hyperglycemic state (HHS). The principal determinants of the presentation are the degrees of insulin deficiency and insulin resistance, although additional factors may also be important. In addition, pathological hyperglycemia sustained over several years may produce functional and structural changes within certain tissues. Patients may present with macro-vascular complications that include ischaemic heart disease (IHD), stroke and peripheral vascular disease (PAD), whereas the specific micro-vascular complications of diabetes include retinopathy, nephropathy, and neuropathy.

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## 2. Diagnosis

Assigning a type of diabetes to an individual often depends on the circumstances present at the time of diagnosis, and many diabetic individuals do not easily fit into a single specific type [1]. An example is a person who has acquired diabetes because of large doses of exogenous steroids and who becomes normoglycaemic once the glucocorticoids are discontinued. In addition, some patients may present with major metabolic decompensation yet can subsequently be treated successfully with oral hyperglycemic agents. Thus, for the clinician and patient, it is less important to label the particular type of diabetes than it is to understand the pathogenesis of the hyperglycemia and to treat it effectively.

Patients under the physical stress associated with surgical trauma, acute myocardial infarction, acute pulmonary oedema or stroke may have transient increases of plasma glucose that often settle rapidly without specific anti-diabetic therapy. However, such clinical situations are also liable to unmask asymptomatic pre-existing diabetes or to precipitate diabetes in predisposed individuals. Such increases, which may have short- and longer-term prognostic importance, should not be dismissed. As a minimum, appropriate follow-up and retesting is indicated following resolution of the acute illness. If there is doubt about the significance of hyperglycemia, the blood glucose level should be rechecked 30-60 min later and the urine tested for ketones.

If hyperglycemia is sustained, treatment with insulin may be indicated. More marked degrees of hyperglycemia, particularly with ketonuria, demand vigorous treatment in an acutely ill patient.

### 2.1 Type 1

The clinical presentation of type 1 diabetes in younger patients is usually acute with classical osmotic symptoms [1]. In general, symptoms will have been present for only a few weeks with osmotic symptoms; weight loss predominates and gradually increases in intensity. Weight loss reflects:

- catabolism of protein and fat resulting from profound insulin deficiency
- dehydration if hyperglycemia is marked

Associated symptoms, particularly blurred vision, are not uncommon although generally less prominent. Although the islet β-cell destruction of type 1 diabetes is a process that occurs gradually over many years, it was very uncommon to detect type 1 diabetes during the early asymptomatic stages of the condition. In some patients, once symptoms appear, diagnosis may sometimes be expedited by awareness of symptoms in other family members with diabetes. Despite the presence of significant osmotic symptoms, some patients do not seek medical advice and a significant proportion (approximately 5%-10%) of patients with type 1 diabetes continue to present in diabetic ketoacidosis (DKA).

Type 1 diabetes is due to destruction of β-cell in the pancreatic islets of Langerhans with resulting loss of insulin production [2]. A combination of environmental and genetic factors that trigger an autoimmune attack on the β-cell is responsible, occurring in genetically susceptible individuals. Thus, among monozygotic identical twins only about one-third of the pairs are concordant for diabetes in contrast to the situation in Type 2 diabetes where almost all pairs are concordant. The process of islet destruction probably begins very early in life and is known to start several years before the clinical onset of diabetes.

Islet cell antibodies are present at diagnosis in most Type 1 diabetic patients and gradually decline and disappear during the following years. Antibodies to specific proteins have more recently been identified: these include antibodies to glutamic acid decarboxylase (GAD, a 64-kDa antigen); and even closer association is found in the presence of antibodies to tyrosine phosphatase (37 kDa, IA-2). The presence in a non-diabetic individual of three or more antibodies (islet cell antibodies, anti-GAD antibodies, anti-IA-2 antibodies, anti-insulin autoantibodies) indicates an 88% chance of developing diabetes within 10 years.

The presence of insulinitis at the onset of Type 1 diabetes represents the role of inflammatory cells (for example, cytotoxic T cells and macrophages) in β-cell destruction. Macrophages also produce cytokines leading to activation of lymphocytes known to be present at the onset of Type 1 diabetes.

### 2.2 Type 2

The majority of patients with type 2 diabetes are diagnosed at a relatively late stage of a long, pathological process that has its origins in the patient's genotype (or perhaps intrauterine experience), and develops and progresses over many years [1].

The presenting clinical features of type 2diabetes range from none at all to those associated with the dramatic and life-threatening, hyperglycemic emergency of the hyperosmolar non-ketotic syndrome (HONK)/hyperosmolar hyperglycemic state (HHS). In many patients with lesser degrees of hyperglycemia, symptoms may go unnoticed or unrecognized for many years; however, such undiagnosed diabetes carries the risk of insidious tissue damage. It has been estimated that patients with type 2 diabetes have often had pathological degrees of hyperglycemia for several years before the diagnosis is made.

This form of diabetes, which accounts for about 90–95% of those with diabetes, previously referred to as non-insulin-dependent diabetes, type II diabetes or adult-onset diabetes, encompasses individuals who have insulin resistance and usually have relative (rather than absolute) insulin deficiency. At least initially, and often throughout their lifetime, these individuals do not need insulin treatment to survive.

There are numerous causes of Type 2 diabetes, which is now known to include a wide range of disorders with differing progression and outlook [2]. The underlying mechanism is due either to diminished insulin secretion—that is, an islet deficiency, associated with increased peripheral resistance to the action of insulin resulting in decreased peripheral glucose uptake, or increased hepatic glucose output. Probably as many as 98% of Type 2 diabetic patients are "idiopathic"—that is, no specific causative defect has been identified. Whether decreasing insulin secretion or increasing insulin resistance occurs first is still uncertain, but the sequence of events may vary in different individuals. Obesity is the commonest cause of insulin resistance.

Some adults (especially those not overweight) over 25 years of age who appear to present with Type 2 diabetes may have latent autoimmune diabetes of adulthood (LADA) and become insulin dependent. Autoantibodies are often present in this group of patients. Type 2 diabetes is a slowly progressive disease: insulin secretion declines over several decades, resulting in an insidious deterioration of glycaemic control which becomes increasingly difficult to achieve.

### 2.3 Insulin

Insulin is responsible for the movement of glucose transport proteins onto the surface of muscle cells, allowing the importation of glucose from the circulation into the cytoplasm where it can be used in energy production [3]. In addition, insulin stimulates the conversion of hepatic glucose into glycogen while suppressing the conversion of glycogen into glucose. Insulin also plays an important role in stimulating liver lipid synthesis. This enables the liver to "store" excess calories in a more efficient and osmotically inactive form that ultimately is deposited as visceral fat contained in adipocytes. Insulin also promotes entry of amino acids into the cells and stimulates protein synthesis. Insulin stimulates endothelial cells to produce nitric oxide (eNOS) a potent vasodilator increasing blood flow to skeletal muscle. The main stimulus for insulin release is elevation of the level of glucose in the blood, as occurs after a meal. In diabetes mellitus, glucose is absorbed normally.

Most young diabetics require insulin injections and so do about 10%-15% of older patients [4]. The majority of elderly diabetics are controlled by diet and drugs. As a consequence, they are sometimes considered to be only 'mild diabetics'. However, this is misleading when one considers the increased health risks to which they are exposed. Whatever the nature of diabetes, its treatment or its duration, it is a serious health problem.

Many elderly diabetics who require insulin are unable to manage their own injections. Impaired vision (due to diabetic eye changes) and a lack of dexterity (sometimes as a consequence of diabetic nerve damage) are the main hindrances. In these cases, they must either depend on a relative or a district nurse to assist in drawing up the insulin and administering the injection. The process can be made easier by using fixed syringes and by storing preprepared syringes in the refrigerator. The situation is further complicated when it is impossible to control the blood sugar by a single daily injection. Sometimes, precision of control is sacrificed for the convenience of once daily injections-the patient must be made aware of the disadvantages as well as the benefits of such a dosage regime. Long acting insulins and mixtures of insulins can be dangerous to use in elderly patients, because of the risk of night time hypoglycemia (low blood sugar levels).

### 2.4 Risks

Persons with diabetes mellitus and lesser abnormalities of glucose tolerance are at risk of developing cardiovascular disease and the metabolic syndrome, a constellation of metabolic and vascular abnormalities, including central obesity, insulin resistance, hyperinsulinemia, glucose intolerance, hypertension, dyslipidemia, hypercoagulability, and increased risk of coronary and cerebral vascular disease [5]. Individuals with type 2 diabetes mellitus have a 2- to 4-fold increased risk of coronary heart disease (CHD) and a 4-fold increase in mortality from CHD. In addition, there is an increased risk of cardiovascular mortality before the development of type 2 diabetes. It is thought that the increase in cardiovascular disease and fatal coronary heart disease is due to hypertension, hyperglycemia, hyperinsulinemia, dyslipidemia, inflammation, and the prothrombotic state.

Studies in apparently healthy persons have also emphasized the role of inflammatory and atherothrombotic mechanisms that may have relevance to persons with diabetes mellitus. Because most diabetes-related deaths are due to cardiovascular disease, management strategies must involve both reducing coronary heart disease risk factors and improving traditional diabetic risk factors, such as glycemic control, and non-traditional risk factors, such as inflammatory and atherothrombotic aspects of diabetes.

### 2.5 Nutrition

Before the discovery of insulin, in 1921, the recommendations for the appropriate diet for diabetes used to suggest a strict, monotonous and rigid diet, with very high percentages of fat and protein and very low percentages of carbohydrates [6]. This made it very difficult for diabetic subjects to adhere to these recommendations. Over the years, these recommendations have been adjusted, mainly because of the availability of insulin and new medications, as well as because prolongation of the life of the diabetic person is accompanied by an increase in cardiovascular disease. Nowadays, the diet for diabetes is synonymous with a healthy diet for the general population, with a wider variety of nutritional options and more complex (or slowly absorbed) carbohydrate-rich foods in the daily dietary plan.

Food and nutrition advice has ranged from "starvation diets" to high- or low-carbohydrate or low-fat diets to nutritional supplements that will provide a cure [7].

Over the years, various diabetes organizations have published nutrition recommendations on the basis of available research and clinical observations. In recent years, the goal in the development of diabetes nutrition therapy recommendations has been to have the recommendations be based on evidence rather than theories. For example, it was longstanding advice that people with diabetes should not eat sugar or foods containing sugars. This information was based on the assumption that because sugars were small molecules, they would be absorbed rapidly, causing blood glucose levels to increase at a greater rate than starches (which are larger molecules). When research first revealed that total amounts of carbohydrate were more important than the source, the public, and many health professionals, were surprised. However, almost all diabetes nutrition recommendations now acknowledge that sugary foods can be substituted for starchy foods.

The primary goals of diabetes medical nutrition therapy (MNT) are to support the achievement and maintenance of as normal blood glucose levels as safely possible, a lipid profile that reduces the risk for cardiovascular disease, blood pressure in an ideal range, and improved or continued quality of life. Important questions then become, what is the evidence that diabetes MNT can achieve these goals and what types of MNT interventions are effective? It is important that clinicians, regardless of their field of practice, know expected outcomes from their interventions, when to evaluate such outcomes, and what interventions contribute to successful outcomes.

## 3. Treatment

The present day life style contributes to the alarming rise in the occurrence of diabetes and the ailments associated with it [8]. Lifestyle modifications inclusive of dietary alterations, weight reduction and regular physical activity are indicated for prevention of diabetes. Accordingly, a holistic health care approach (instead of a conventional drug-based approach alone) for the treatment is highly warranted. Chronic diseases such as diabetes are associated with diminished quality of life and psychological depression and anxiety. Mind-body therapies have behavioural and psychological effects that may help patients cope with disease and improve mood and quality of life. The MNT is a component of diabetes management and of diabetes self-management education. The MNT is the preferred term and should replace other terms, such as dietary management and diet therapy. The MNT for people with diabetes should be individualized with consideration given to each individual's metabolic profile, usual food and eating habits, treatment goals, and prospected outcomes. Monitoring of

metabolic parameters such as HbA1c, glucose, blood pressure, lipids, body weight, and renal function, as well as quality of life is essential to assess the need for changes in therapy and ensure successful outcomes.

## 4. Health Care

Taking a comprehensive approach to diabetes prevention and care implies that polices and activities are put in place to address primary prevention, early diagnosis (including screening if appropriate), management of diabetes and its complications, and rehabilitation for those affected by complications [9]. A comprehensive approach will include policies and activities outside the formal health sector, particularly for the primary prevention of T2DM. For example, promoting healthier diets and greater physical activity could involve policies on food production, marketing and taxation, and policies on design of local environments and public transport. The WHO' s strategy on diet and physical activity provides a framework for developing national and international policy that is relevant to countries at all levels of development. The best indication of a comprehensive approach is a national government - led strategy that covers primary prevention through to rehabilitation, with the caveat that the presence of a strategy does not guarantee that it has been implemented. A recent survey carried out through IDF member organizations found that of the 98 countries from which responses were received, just over 70% claimed that their country had implemented a national diabetes program. The region with the lowest proportion of countries (30%) with a national diabetes program was sub-Saharan Africa, but even in richer regions, such as Europe, over 20% of responding countries did not have a national diabetes program.

Integrated care for people with diabetes refers to the need to provide care for conditions coexisting with diabetes within the same primary health care service. Within most high income countries, primary health care has been developed to provide a range of services covering most of the needs with people with diabetes, and indeed with other chronic conditions. In low and middle income countries, however, integration of care is often a challenge, as donor funding is most often given to specific disease programs such as HIV/AIDS, tuberculosis or malaria, or large-scale government funding is allocated for specific vertical programs, such as HIV/AIDS.

These four core principles -

i) continuity of care,
ii) access to care,
iii) coordination between different levels of care and
iv) multidisciplinary team work

- really belong together, as they concern providing good quality health care for managing diabetes and preventing its complications.

A particular challenge in low resource settings is that of moving away from a focus on episodic curative care. High patient numbers, with acute infectious illness and low numbers of trained professionals have nurtured this approach, which will tend to wait for the patient to present with gangrene of the foot rather than invest energy in identifying the patients at risk. Likewise, there is more focus on treating the problem than empowering the person. For example, a patient with elevated blood glucose levels is more likely to receive a change in prescription than a useful exchange of information about diet or exercise and advice about overcoming barriers to adherence.

## 5. Conclusion

Some patients have marked and frequent fluctuations in glycemia for no clear reason. Severe hypoglycemia and hyperglycemia occur, typically requiring emergency care and hospitalization. Labile plasma glucose levels are more common in type 1 due to complete lack of endogenous insulin, but can occur in all forms. Known causes are hidden infections, gastroparesis and endocrine diseases. In such patients, the implementation of therapeutic measures, such as insulin preparation and injection, and glycemic control, should be evaluated first and foremost. A higher frequency of self-control reveals unnoticed deviations and provides the patient with useful feedback. A comprehensive history of diet and meal schedules can reveal factors that contribute to poor control. Responsible pathology should be ruled out by physical examination and targeted laboratory tests. Some patients are assisted with the transition to more intensive insulin delivery, which allows for more frequent adjustments based on self-control.

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