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Diabetes mellitus and its intraoral presentations: A review

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Abstract

Diabetes Mellitus has become a global epidemic and presents many complications, usually proportional to the degree and duration of hyperglycemia. Individuals with diabetes are at increased risk for periodontal disease, salivary gland dysfunction, dental caries, mucosal abnormalities, and oral burning, all of which can negatively impact patient quality of life. The aim of this review is to highlight the different oral manifestations, diagnosis and dental treatment considerations for diabetic patients.

Keywords: Diabetes Mellitus, oral cavity, metabolic disease, insulin

Introduction

Diabetes mellitus represents a group of metabolic diseases that are characterized by hyperglycemia due to a total or relative lack of insulin secretion and insulin resistance or both. Diabetes' means 'polyuria' and 'mellitus' means 'honey'. The name 'diabetes mellitus' was coined by Thomas Willis, in 1675. Diabetes mellitus classified in to Type I and Type II. [1, 2]

Type I diabetes mellitus

- Due to deficiency of insulin (destruction of β-cells in islets of Langerhans).
- Occur at any age, usually occurs before 40 years of age.
- This requires insulin injection.
- So it is also called insulin-dependent diabetes mellitus (IDDM).
- When it develops at infancy or childhood, it is called juvenile diabetes.
- Develops rapidly and progresses at a rapid phase.
- Not associated with obesity, but may be associated with acidosis or ketosis.

Etiology of type I diabetes mellitus

- Degeneration of β-cells in the islets of Langerhans of pancreas.
- Destruction of β-cells by viral infection.
- Congenital disorder of β-cells.
- Destruction of β-cells during autoimmune diseases.

Other forms of type 1 diabetes mellitus?

1. **Latent autoimmune diabetes in adults (LADA):**

- Slow onset diabetes.
- Slow progress than IDDM and it occurs in later life after 35 years.
- It is difficult to distinguish LADA from type II diabetes mellitus, since pancreas takes longer period to stop secreting insulin.

2. **Maturity onset diabetes in young individuals (MODY):**

- Rare inherited form occurs before 25 years.
- Due to hereditary defects in insulin secretion.[3,4,5]

Type II Diabetes Mellitus

- Type II diabetes mellitus is due to insulin resistance (failure of insulin receptors to give response to insulin). So, the body is unable to use insulin. About 90% of patients have type II diabetes mellitus.
- Occurs after 40 years.
- Only some forms require insulin.
- It can be controlled by oral hypoglycaemic drugs. So it is also called non insulin dependent diabetes mellitus (NIDDM).
- Type II diabetes mellitus may or may not be associated with ketosis. Often it is associated with obesity.[6]

Etiology of type II diabetes mellitus

In this, structure and function of β-cells and blood level of insulin are normal. But insulin receptors may be less, absent or abnormal, resulting in insulin resistance. Common causes of insulin resistance are:

1. Genetic disorders (significant factors causing type II diabetes mellitus)
2. Lifestyle changes such as bad eating habits and physical inactivity leading to obesity.
3. Stress.

Other forms of type II diabetes mellitus?

1. **Gestational diabetes**

- It occurs during pregnancy.
- Due to many factors such as hormones secreted during pregnancy, obesity and lifestyle before and during pregnancy.
- Usually disappears after delivery of the child but woman has high risk of development of type II diabetes later.

2. Pre-diabetes

- Also called chemical, subclinical, latent or borderline diabetes.
- It is the stage between normal condition and diabetes.
- Person does not show overt (observable) symptoms of diabetes but there is an increase in blood glucose level.
- Though it is reversible, the affected persons are at a high risk of developing type II diabetes mellitus.[7,8]

Features)	Type I (IDDM)	Type I I(NIDDM)
Age of onset	Usually before 40 year	Usually after 40 year
Major cause	Lack of insulin	Lack of insulin receptor
Insulin deficiency	Yes	Partial deficiency
Immune destruction of β-cells	Yes	No
Involvement of other endocrine disorders	No	Yes
Hereditary cause	Yes	May or may not be
Need for insulin	Always	Not in initial stage May require in later stage
Insulin resistance	No	Yes
Control by oral hypoglycemic agents	No	Yes
Symptoms appear	Rapidly	Slowly
Body weight	Usually thin	Usually overweight
Stress-induced obesity	No	Yes
Ketosis	Yes	May or may not be

Signs and symptoms of Diabetes Mellitus (DM)

Manifestations of DM develop because of three major setbacks of insulin deficiency:

1. Increased blood glucose level (300 to 400 mg/dL) due to reduced utilization by tissue.
2. Mobilization of fats from adipose tissue for energy, leading to elevated fatty acid content in blood. This causes deposition of fat on the wall of arteries and development of atherosclerosis.
3. Depletion of proteins from the tissues.

Signs and symptoms of diabetes mellitus

- Glucosuria
- Osmotic diuresis
- Polyuria
- Polydipsia
- Polyphagia
- Asthenia
- Acidosis
- Acetone breathing
- Kussmaul breathing (increase in rate and depth of respiration)
- Circulatory shock
- Coma

Oral manifestations of diabetes mellitus

- Gingivitis.
- Periodontitis.
- Dental caries.
- Tooth loss.
- Oral candidacies.
- Oral mucosal lesions such as traumatic ulcers and irritation.
- Fibroma.
- Impaired wound healing.
- Xerostomia.
- Salivary gland hypo function.
- Sialosis.
- Burning mouth sensation.
- Impairment of taste.[9,10,11]

Path physiology of oral manifestations in diabetes

Following mechanism is involved:

- The polyol pathway converts glucose into the enzyme sorbitol by aldose reductive that causes tissue damage and numerous other diabetic complications.

- Formation of advanced glycosylation end products (AGE), due to binding of glucose to proteins, lipids and nucleic acids, results in the alteration of structures and functions, in addition to its deposition in specific organs causes various complications.
- Atheroma deposits are formed and accumulate in the basal membrane and lumen causing decreased cellular defence capacity and impaired polymorph nuclear leukocyte response.

Diabetes and periodontal tissues

Advanced periodontal disease occurs due to number of structural and functional hyperglycaemia-related alterations:

- Thickening of basement membranes of blood vessels (microangiopathy) which leads to deterioration of microcirculation in periodontal tissue and decreased supply of oxygen and nutrients to tissues and accumulation of harmful metabolites.
- Impaired functions of polymorph nuclear leukocytes leading to abnormalities of adherence, phagocytises and chemo taxis.
- Impaired gingival fibroblast proliferation and collagen synthesis.
- Enhanced collagen's activity.
- Formation of AGEs that bind to monocyte receptors and induce production of inflammatory mediators like TNF, prostaglandin E-2 and interleukin-1.

All these mechanisms may lead to impaired host resistance and accelerated inflammatory host response and result in loss of periodontal fibres, loss of the alveolar supporting bone, and eventually loss of teeth.

Dental caries in diabetes

Poor metabolic control, salivary gland hypo function, and high salivary glucose concentrations promotes growth of Streptococcus mutans and Lactobacillus. So diabetic patients are more at high risk for dental caries. [12, 13, 14]

Diabetes and wound healing

Impaired wound healing in diabetes is due to:

- Reduced host response to inflammation due to impairment of polymorph nuclear leukocyte functions leading to abnormalities of adherence, phagocytises, chemo taxis, and intracellular killing.
- Synthesis of collagen in extracellular matrix gets decreased, the degradation of newly synthesised collagen is accelerated and collagen's activity is increased.
- Formation of AGEs may lead to increased thickness of basement membranes of blood vessels, which impairs the supply of oxygen and nutrients to tissues.

Diabetes-related changes in salivary gland function

- Dehydration, as the result of prolonged hyperglycaemia and consequently polyuria, is major cause of xerostomia and salivary gland hypo function in diabetics.
- Gradual degeneration of salivary gland tissue can lead to salivary hypo function and altered salivary composition.

Diabetic sialosis

- 10-25% of type 1 or type 2 diabetics may develop a bilateral asymptomatic enlargement of the parotid glands and, more rarely, of the sub mandibular glands, known as diabetic sialosis.
- Enlarged parotid and sub mandibular glands are characterized by fatty infiltration, fibrous tissue, enlargement of acinar cell and reduction in acinar tissue, but without signs of inflammation.

Oral mucosal disorders in diabetes mellitus

Candidal colonization increases in diabetic patient due to:

- Poor metabolic control.
- High concentrations of glucose in the blood and saliva.
- Reduced salivary flow rates, low salivary pH.
- Reduction in antimicrobial substances in the saliva.

Oral infection with Candida may clinically present as median rhomboid glossaries, atrophic glossaries, denture steatites, pseudo membranous candidacies and angular cheilitis.

Diabetic neuropathy (DN)

Diabetic neuropathy (DN) is a common disorder and is defined as signs and symptoms of peripheral nerve dysfunction in a patient with diabetes mellitus (DM) in whom other causes of peripheral nerve dysfunction have been excluded. Hyperglycaemia induces:

- Rheological changes, which increases endothelial vascular resistance and reduces nerve blood flow.
- Depletion of nerve myoinositol through a competitive uptake mechanism.
- Activation of polyol pathway in the nerve through enzyme aldose reductive leads to

accumulation of sorbitol and fructose in the nerve and induces no enzymatic glycosylation of structural nerve proteins.
- Oxidative stress.
- Activation of protein kinas C, also linked to vascular damage in DN.

These changes result in abnormal neuronal, axonal, and Schwann cell metabolism, which results in impaired axonal transport. [15, 16]

Diagnostic criterion for diabetes

Glucose Tolerance Test

- Patient scheduled for oral GTT is instructed to eat a high carbohydrate diet for at least 3 days prior to the test and come after an overnight fast on the day of the test (for at least 8 hours).
- A fasting blood sugar sample is first drawn.
- Then 75 gm of glucose dissolved in 300 ml of water is given.
- Blood and urine specimen are collected at half-hourly intervals for at least 2 hours (four samples).
- Blood or plasma glucose content is measured and urine is tested for glucosuria to determine the approximate renal threshold for glucose.

Revised criteria for diagnosis of diabetes by oral GTT (as per American Diabetes Association, 2007)

Fasting (for > 8 hours) value

- Below 100 mg/dl (< 5.6 mmol/L): Normal fasting value
- 100-125 mg/dl (5.6-6.9 mmol/L): Impaired fasting glucose (IFG)
- 126 mg/dl (7.0 mmol/L) or more: Diabetes mellitus

Two-hour after 75 gm oral glucose load

- < 140 mg/dl (< 7.8 mmol/L): Normal post-prandial GTT
- 140-199 mg/dl (7.8-11.1 mmol/L): Impaired post-prandial glucose tolerance (IGT)
- 200 mg/dl (11.1 mmol/L) or more: Diabetes mellitus

Random value

- 200 mg/dl (11.1mmol/L) or more in a symptomatic patient: Diabetes mellitus

Glycosylated haemoglobin test

- It determines the blood glucose status over the 30 to 90 days prior to collection of the blood sample.
- In circulation glucose attaches to a portion of the hemoglobin on red blood cells.
- The higher the plasma glucose levels over time, the greater is the percentage of haemoglobin that becomes glycated.

There are two different glycated haemoglobin assays:

- Haemoglobin A1 (HbA1):<8%(>10%- poorer glycolic control)
- Haemoglobin A1C (HbA1c): 6.0-6.5%.

Fructosamine test

- Determines the blood glucose status over 2-4 weeks prior to test
- Helps in managing women with gestational diabetes.
- The normal range for fructosamine is 2.0 to 2.8 mmol/L.

Self–blood glucose monitoring (SBGM)

- Small handheld gluco meters are used.
- Gluco meters use a small drop of capillary blood from a finger stick sample to assess glucose levels within seconds.

Other tests

Extended GTT

- The oral GTT is extended to 3-4 hours for appearance of symptoms of hyperglycaemia.
- Useful test in cases of reactive hypoglycaemia of early diabetes.

Intravenous GTT

- Performed in persons who have intestinal malabsorption or in postgastrectomy cases.

Cortisone-primed GTT

- Useful investigative aid in cases of potential diabetics.

Insulin assay

- Plasma insulin can be measured by radioimmunoassay and ELISA technique.

Benedict's qualitative test

- Detects any reducing substance in the urine and is not specific for glucose.
- More sensitive and glucose specific test is dipstick method based on enzyme-coated paper strip which turns purple when dipped in urine containing glucose.

Dental treatment considerations for diabetic patients

A. Medical history

- To assess the glycemic control.

- Recent blood glucose levels.
- Frequency of hypoglycemic episodes.
- Antidiabetic drugs being used:

1. Dosage.
2. Tim e of administration.
3. Frequency.

- Drugs which alter the glucose levels through interference with insulin or carbohydrate metanolism.

Drugs with hyperglycaemic effects:

1. Corticosteroids.
2. Thiazides
3. Phenytoin
4. Epinephrine
5. Oral contraceptives
6. Thyroid products
7. Calcium channel blockers.

Drugs which potentiate the hypoglycaemic effects of sulfonylureas:

1. Salicylates
2. Sulphonamides
3. AC Einhibitors
4. MAO inhibitors
5. Beta adrenergic blockers.

B. Scheduling the visit

- Morning appointments are advisable because endogenous cortical levels are higher at that time.
- For patient taking insulin, appointment should be scheduled so that they do not coincide with the peaks of insulin activity.

C. Diet

- Make sure that patient has eaten normally and taken medications as usual.
- If the patient skips breakfast but takes normal dose of insulin, risk of hypoglycaemic shock is there.

D. Before starting treatment

- Blood glucose levels should be measured.
- If sugar level <70mg/dl, oral glucose to be given before treatment to decrease the risk of hypoglycaemic shock.

E. During treatment

- Most common complication in dental office is hypoglycaemic shock.
- When insulin or other ant diabetic drugs exceed physiological needs there may be severe decline in blood sugar levels.
- Maximum risk is during peak insulin activities.

F. After treatment

- Patients with poorly controlled DM are at greater risk of developing infections and delayed wound healing.
- Acute infections adversely affect the insulin resistance and glycemic control which in turn may further affect the body's capacity to heal.
- Therefore antibiotic coverage is must.
- If the dentist anticipates that normal dietary intakes will be affected after treatment, insulin or oral ant diabetic drugs and their doses may need to be adjusted in consultation with the patient's physician.
- Salicylates potentiate the hypoglycaemic effect of sulfonylureas and produces hypoglycaemia. Therefore, aspirin and aspirin containing compounds should not be given to diabetic patients.
- Patients with severe periodontitis, adjunctive use of tetracycline antibiotics in conjunction with mechanical periodontal therapy may have beneficial effects on glycolic control as well as on periodontal status.[17,18,19]

Conclusion

Although dental care providers have traditionally played a primary role in the examination and diagnosis of the specific disorders of these tissues, other health care providers who are responsible for diagnosing and managing patients with diabetes and pregnant patients can also easily screen for these oral abnormalities. Changes in oral soft tissues, in addition to periodontal tissues, can be helpful in the diagnosis of diabetes in undiagnosed patients and may serve as aids in monitoring the care of patients with known diabetes.

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