

# Diabetic Patients in Dental Clinics: A Review

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Medical emergencies can and do happen anywhere at any time, and that includes in dental offices. Medical emergencies are a very stressful, chaotic event for all in the office. Under these types of conditions, anxiety and confusion can exist, as well as the inability to recall proper treatment protocols; many facilities have an emergency drug kit but are not familiar with its contents. The time to become familiar with the emergency drug kit is not during a crisis, but through continuing education and mock emergency drills. The dentist and the entire team need to be proficient in handling these emergencies until EMS arrives. Adverse outcomes and death may result even if an emergency is handled correctly. However, education and preparation will optimize the chances of a favourable result.

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**D**iabetes mellitus (DM) is a metabolic disorder characterized by impaired insulin secretion, varying degrees of insulin resistance, or both resulting in hyperglycaemia. Hyperglycemia is the most clinically important metabolic aberration in Diabetes mellitus (DM) and the basis for its diagnosis.

Apart from the obvious impact of impaired glucose metabolism, DM and chronic hyperglycemia are associated with important ophthalmic, renal, cardiovascular, cerebrovascular, and peripheral neurological disorders.

Management of the diabetic dental patient must take into consideration the impacts of diabetes on dental disease and dental treatment, as well as a clear appreciation for the comorbidities that accompany long-standing diabetes mellitus.

**Prevalence of Diabetes**

Approximately 200 million individuals worldwide and 15.7 million Americans suffer from diabetes mellitus, representing a 50% increase in the incidence of diabetes within the past two decades. Although 10.3 million of these Americans have been diagnosed with diabetes, 5.4 million individuals are unaware that they have the disease. Approximately 798,000 Americans are diagnosed with diabetes annually.

**Classification**

Most cases of DM can be classified as type-I (formerly, insulin-dependent diabetes) and type-II (formerly, non-insulin-dependent diabetes).

Blood glucose elevation that does not satisfy the definition of type-I or type-II Diabetes mellitus (DM) is classified as impaired glucose tolerance or impaired fasting glucose.

Type-I Diabetes mellitus is characterized by an absolute insulin deficiency brought about by the autoimmune destruction or accelerated disappearance of pancreatic beta cells. However, some patients have no evidence of an autoimmune mechanism. Such patients are said to have type-1B Diabetes mellitus.

Mononuclear lymphocytic infiltrates, principally T lymphocytes, have been identified in pancreatic islets in individuals with type-I Diabetes mellitus. Insulin administration is essential in a typical patient with type I DM. If patients do not receive insulin, they develop dehydration resulting from severe hyperglycemia and ketoacidosis, both of which when not treated can lead to coma and death rapidly.

Type II Diabetes mellitus: Insulin resistance frequently precedes and it is characterized by a decreased response of the target tissues to the normal levels of circulating insulin. In contrast to type-I Diabetes mellitus, hyperglycemia in type-2 DM is principally a result of insulin resistance. The eventual loss of the ability of the pancreas to increase insulin output, in the setting of insulin resistance, creates a relative insulin deficiency and progression to established type-2 DM.

Gestational diabetes mellitus: It is characterized by glucose intolerance that is first diagnosed during pregnancy in a woman who has not had DM. True gestational diabetes mellitus resolves during the postpartum period. However, as many as 50 percent of women who had GDM remain at risk of developing type 2 diabetes mellitus, so gestational diabetes mellitus is thought to be a harbinger of DM in later life.

The pathophysiology of gestational diabetes mellitus is identical to that of type 2 diabetes mellitus with pancreatic beta-cell dysfunction that is unable to meet the increased demands associated with insulin resistance during pregnancy. A minority of patients may develop type I diabetes mellitus for the first time during pregnancy, which emphasizes the connection between pregnancy and autoimmune disease. Recognition of gestational diabetes mellitus is important because it provides an opportunity to initiate interventional strategies to prevent the development of type II diabetes mellitus and to prevent foetal abnormalities by helping the patient maintain tight glycemic control during pregnancy.

**Diagnosis**

The diagnosis Diabetes mellitus is based on the measurement of fasting plasma glucose (FPG) or plasma glucose 2 hours following a 75-gm oral glucose challenge [oral glucose tolerance test (OGTT)]. According to the American Diabetic Association (ADA) normal plasma glucose defined by as a fasting plasma glucose  $\leq 100$  mg/dL. The diagnosis of Diabetes mellitus is straight forward in the patient who presents with classic symptoms of polyuria, thirst, weight loss, fatigue, visual blurring, and a fasting plasma glucose  $\geq 126$  mg/dL, or a random value of at least 200 mg/dL.

The diagnosis of DM should be confirmed by plasma glucose evaluation on a subsequent day. In the absence of these classic symptoms, glucose intolerance may exist as impaired fasting glucose when the fasting plasma glucose is between 100 and 125 mg/dL. Similarly, plasma glucose of 140 to 199 mg/dL following

oral glucose challenge test defines impaired glucose tolerance (IGT). The classification of IFG and impaired glucose tolerance is important because individuals with IFG and IGT are at greater risk of developing diabetes and atherosclerotic cardiovascular disease even if they do not develop DM.

**Diabetic emergencies**

If hypoglycemia occurs, glucose should be given by mouth as tablets, syrup, or a sugary drink, if the patient can cooperate. For those patients who are not able to cooperate, glucose is also available as an oral gel in a dispenser (GlucoGel). If these measures are impossible or ineffective, for example in an uncooperative, semi-conscious patient, the usual treatment of first choice is glucagon (1 mg/ml injection) 1 mg by intramuscular or subcutaneous injection. Patients, who do not respond to glucagon, for some time may have exhausted their supplies of liver glycogen, will require up to 50 ml of intravenous glucose solution. Clearly, patients who have reached this stage should be managed under medical supervision and are unlikely to be seen in dental practice. There may be uncharacteristic aggression, drowsiness and a moist skin. Pulse may be rapid and full and blood sugar will be low.

**Treatment of Diabetes Mellitus**

Medical nutrition therapy (also known as dietary therapy) and lifestyle modification form the center piece of the management of diabetes mellitus (DM), irrespective of modality of therapy chosen.

The goals of therapy are to prevent complications of diabetes mellitus (DM). Tight blood glucose control prevents microvascular complications in both type I and type II DM. Although glycemic control may not be as effective in reducing macrovascular complications, aggressive therapies aimed at blood pressure levels, lipid levels and smoking cessation are effective in preventing macrovascular complications.

**Insulin Therapy**

Insulin therapy is the mainstay for patients with type I DM, and, in most patients, frequent multiple dosing (basal and bolus) plans are common. Continuous insulin delivery via pumps also is a fairly common practice. All of these methods typically involve subcutaneous injection, and a variety of insulin preparations can be used that allow the physician and patient to select the best method on the basis of cost and flexibility. Insulin therapy should mimic the physiological release of insulin, which is characterized by a continuous basal secretion, to

prevent fasting hyperglycemia, as well as prandial insulin release to prevent postprandial hyperglycemia.

During fasting, long-acting basal insulin, which has a flat profile without a peak, is used, and at mealtime, a bolus injection of fast-acting insulin is administered to produce a peak coinciding with absorption of ingested carbohydrates.

In the past, insulin was derived from porcine and bovine sources. These sources have been replaced by recombinant human insulin. Many types of insulin have been developed to produce varying levels of onset of action, ranging from rapid-acting (example, analoginsulins such as aspart, lispro and glulisine) to intermediate acting (example, neutral protamine Hagedorn, commonly referred to as NPH) to long-acting (example, glargine and detemir).

Types of insulin and their profiles.		
TYPE OF INSULIN	CHARACTERISTIC	ACTION
Rapid- Acting Insulin Insulin lispro Insulin aspart Insulin glulisine	Analog insulin Altered amino acid sequence-promoted insulin monomers that are absorbed rapidly Injected shortly before meals Minimize hypoglycemia Used for continuous subcutaneous insulin infusion and conventional subcutaneous injection therapy	Onset of action: 0.25 to 0.50 hour Peak action : 1 to 2 hours Duration of action: 4 to 5 hours
Short- Acting Insulin Regular	Soluble human insulin Injected 30 to 60 minutes before meals for optimal action; failing to do results in postprandial hyperglycemia Less convenient than rapid-acting analogs	Onset of action: 0.50 to 1 hour Peak action: 2 to 4 hours Duration of action: 6 to 8 hours
Intermediate- Acting Insulin Neutral protamine Hagedorn, commonly referred to as NPH (isophane suspension)	Formed by adding protamine to human insulin Acts as both basal and bolus insulin because of its peak at 4 to 6 hours Hypoglycemia is a problem because of these peaks	Onset of action: 2 to 3 hours Peak action: 4 to 6 hours Duration of action: 6 to 8 hours
Long – Acting Insulin Glargine Detemir	Insulin analogs Glargine: Provides consistent level in plasma for a long duration and is peakless Detemir: Binds to albumin via fatty acid chain, hence slower absorption and consistent levels	Onset of action: 1 to 2 hours Peak action: none Duration of action: up to 24 hours for glargine and 14 to 24 hours for detemir

**Table .Types of Insulin and their profile**

**Oral Hypoglycemic Agents**

These are the first-line agents used to treat patients with type II DM, and they either increase pancreatic insulin secretion or improve insulin action (the term “sensitizer” is used to describe them). Although debate continues about the merits of one kind over another, each class of OHA generally is as effective as the other. At first approximation, most OHAs lead to an average 1.0 to 1.2 percent decrease in glycosylated haemoglobin (HbA1c). These OHAs can be used alone or in combination with one another and with insulin. Regimens should complement each other and not produce the same effects; for example, combining a sulfonylurea with a meglitinide may not be effective because both act on the sulfonylurea receptors to release insulin. On the other hand, either of these can be combined with any of the insulin sensitizers or the incretin therapies. Use of combination therapies is commonplace for the control of DM.

Oral hypoglycemic agent characteristics.		
AGENT	MODE OF ACTION	ADVERSE EFFECT
Insulin Secretagogues		
Sulfonylureas [currently third generation (glipizide, glimepiride, etc.)]	Bind to sulfonylurea receptors on the beta cells triggering release of insulin Duration of action and daily doses vary by agent	Hypoglycemia Weight gain
Meglitinides (repaglinide, nateglinide)	Bind to sulfonylurea receptors Short duration of action, quick onset of action , taken 15 minutes before meals to target postprandial hyperglycemia	Generally none, but possible hypoglycemia
Insulin Sensitizers		
Biguanides (metformin)	Decrease hepatic gluconeogenesis and increase peripheral glucose uptake Contraindicated in renal insufficiency and heart failure Promote weight loss and low risk of developing hypoglycemia when used alone	Diarrhea, abdominal pains Risk of lactic acidosis
Thiazolidinediones (rosiglitazone, pioglitazone)	Activate peroxisome proliferator-activated receptor to affect glucose and lipid metabolism Improve peripheral glucose uptake in skeletal muscle and fat Take as long as 6 to 12 weeks to attain optimal therapeutic effect	Weight gain Water retention May precipitate congestive heart failure in susceptible people Possible increase in risk of experiencing bone loss
Glucosidase Inhibitors		
Acarbose/Miglitol	Inhibit a-glucosidase in the gut and thus, prevent breakdown of some complex carbohydrates into simple sugars that then cannot be absorbed Prevent postprandial glucose excursions	Bloating, diarrhea and flatulence due to action of colonic bacteria on undigested carbohydrates

**Table .Oral hypoglycaemic agent**

**The management of diabetic patients undergoing surgery**

In well-controlled diabetics requiring local anesthesia, all that is required is to ensure that these patients are treated promptly, which usually means placing them first on an operating list. They should also have had their normal diet and diabetic medication.

Management becomes more complicated when the patients have to be fasted and outlines of management for patients with type I and type II diabetes are following:

Preoperative management in Type I Diabetes mellitus – fasted patient

- The patient should be first on the list of patients
- All long-acting insulin should be stopped the night before surgery
- Intravenous access should be obtained at an early stage
- If surgery is in the morning, all subcutaneous morning insulin should be stopped
- If surgery is in the afternoon, the usual short-acting insulin should be given in the morning at breakfast but no medium or long-acting insulin
- The urea and electrolytes should be checked on the morning of surgery and an intravenous infusion of 1 litre of 5% dextrose with 20 mmol/l potassium chloride over 8 hours should continue until the patient is eating normally. Dextrose may need constant infusion to maintain the

- blood glucose
- 50 units of short-acting insulin should be added to 50 ml 0.9% saline, which can be given by an infusion pump, and is given according to a sliding scale that can be adjusted dependent on the blood glucose measurements
- The blood glucose measurements should be checked hourly aiming at a level of 7–11 mmol/l
- Postoperatively the intravenous insulin and dextrose, potassium chloride and sliding scale should be continued until the patient is eating
- Finger-prick glucose should be checked every 2 hours.

**Preoperative management in Type II Diabetes mellitus – fasted patient**

- These patients may be managed by attention to diet or more commonly, use of oral hypoglycaemic. A fasting blood glucose of >10 mmol/l may require management along the lines of a type I diabetic.
- Patients taking a long-acting sulfonylurea should have the dose halved the day before surgery and the tablet should be omitted altogether on the day of surgery.
- The fasting blood glucose level should be checked on the morning of surgery and treatment is only needed if the level is more than 15 mmol/L.
- The blood glucose level should be monitored in any event using a finger-prick blood sample If the blood glucose level is more than 15 mmol/L, insulin should be used.

**Dental Consideration**

Managing the care of patients with DM in the dental office should not pose a significant challenge.

Hypoglycemia is the major issue that usually confronts dental practitioners when they are treating patients with DM, especially if patients are asked to fast before undergoing a procedure.

- Staff members should be trained to recognize and treat patients who have hypoglycemia.
- Patients who have DM and exhibit unusual behavior should raise suspicion among staff members, and a glucometer should be used to test their blood glucose levels.
- Every dental office should have a protocol for treating hypoglycemia in conscious and unconscious patients. It is prudent to have snack foods or oral glucose gels or tablets available for such emergencies, especially in offices in which a large number of minor surgical procedures are performed.
- Glucose gels are particularly helpful in treating children or adults who are uncooperative because the glucose begins to be absorbed when it is exposed to a mucosal surface.
- Patients taking insulin are advised to carry their own glucometers with them, so asking them to check their own blood glucose levels can be a simple remedy. Patients who are at risk of developing hypoglycaemia are those who have received insulin therapy for a while, and screening for patients who report taking insulin should alert staff members to this.
- Although patients who take OHAs are at a lower risk of developing hypoglycemia than are those receiving insulin, the risk is increased when the patient has renal or hepatic disease.
- Although no level of hyperglycemia is completely safe, there are no specific guidelines regarding high blood glucose levels and how they should be managed before or during a procedure.
- If blood glucose levels are elevated to the point that the patient has altered sensorium, it is prudent to avoid performing any procedures in that patient. Having well-controlled blood glucose levels important for infection prevention and proper healing; however, a scheduled procedure probably does not need to be postponed as long as the patient is conscious and able to follow instructions.
- Post operative instructions should emphasize the importance of blood glucose level control during the healing phase, and the patient's primary care physician should be kept informed to help the patient maintain adequate glycemic control.

**Complications:**

Macro and micro vascular complication of diabetes mellitus are of many type

Microvascular and macrovascular complications of diabetes mellitus.		
COMPLICATION	FEATURES	PREVENTION
Retinopathy	Hemorrhages, exudates, retinal detachment, macular edema	Annual Screening for early diagnosis and treatment Intensive glucose control
Nephropathy	Most common cause of end-stage renal disease in United States Earliest sign is microalbuminuria	Intensive glucose control
Neuropathy	SEither peripheral or autonomic Peripheral: pain, tingling, numbness and predisposition to foot ulcers Autonomic: affects cardiovascular, gastrointestinal and genitourinary systems and awareness of hypoglycaemia	Examination of feet at every visit to the health care provider Intensive glucose control
Cardiovascular Disease	Diabetes mellitus is considered a coronary artery disease equivalent Risk is increased when associated with hypertension, microalbuminuria or retinopathy Risk of experiencing a silent myocardial infarction	Aggressive control and treatment of hypertension and hyperlipidemia

**Table. Macro and micro vascular complication of diabetes mellitus**

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