Diabetes Mellitus: The Disease, Drugs and the Dental Patient

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Learning Objectives
1. Discuss the epidemiology of Diabetes
2. Define Diabetes and differentiate between each type
3. Describe the signs and symptoms of Diabetes mellitus.
4. Identify risk factors and systemic complications of Diabetes.
5. Discuss the oral complications and management considerations for the dental patient with diabetes
6. Discuss oral pathologies involved in Diabetes
7. Discuss the potential consequences of uncontrolled diabetes on oral health
8. Recognize signs and symptoms of hypoglycemia and discuss guidelines for management of this condition

• The most common complication of Diabetes mellitus therapy in the Dental Clinic is ___________:  

A. Hyperglycemia  
B. Ketoacidosis  
C. Hypoglycemia
• What is the best time to schedule a dental visit for your Diabetic patient?

A. Mid afternoon  
B. Late evening  
C. Morning appointment

• The hypoglycemic properties of Sulfonylureas/ Biguanides/ Meglitinides Thiazolidinediones may be potentiated by:
  • Salicylates  
  • Ibuprofen  
  • Diclofenac sodium  
  • Célécoxib  
  • Epinephrine  
  • ACE-inhibitors  
  • Corticosteroids  
  • Calcium channel blockers  
  • Tetracyclines  
  • Quinolones

• Diabetic patients may have an increased incidence of ______ (True/ False)
  1. Salivary gland dysfunction: True /False
  2. Periodontal disease: True/ False
  3. Fungal infection: True/ False
  4. Oral burning and Taste disturbance: True/ False
  5. Traumatic ulcers and Irritational Fibromas: True/ False
Diabetes mellitus

Complications of Diabetes

Progression of Foot Ulcers
Oral Manifestations of Diabetes

Acute pseudomembranous Candidiasis

Oral Manifestations of Diabetes

Oral candidiasis is associated with an increase in:

• salivary glucose
• decreased saliva
• impaired immune response
• Opportunistic infections
• Poor wound healing

Oral Manifestations of Diabetes

Lichen planus
'In the past half-century, we have come to recognize that the mouth is a mirror of the body, it is a sentinel of disease, and it is critical to overall health and well-being. The challenge facing us today – to help all Americans achieve oral health – demands the best efforts of public and private agencies as well as individuals'
STANDARDS OF MEDICAL CARE IN DIABETES

Diabetes facts
• 347 Million people worldwide have diabetes
• 29.1 Million in the United States
• Estimated 2.0 million deaths (2016)
• 1/3rd of diabetics unaware of their disease
• 90% have Type 2 diabetes: Symptoms?
• Leading cause of blindness in adults
• Twice as likely to develop heart disease
• Diabetes = Obesity / Obesity = Diabetes?
• Diabetes cost $174 billion annually

World Health Organization WHO
Are we doing a good job with treatment?

- Only 52.5% of diabetics have a HBA1C < 7%
- Only 51.1% of diabetics have a BP < 130/80
- Only 56.2% of diabetics have an LDL cholesterol < 100 mg/dl.
- Only 18.8% of diabetics are meeting all 3 goals!

Why do we do such a poor job?

- Patient: Cost, side effects, fear of side effects, fear of injections, denial of disease
- Physician: Clinical inertia/ not enough time
- DHCP: Physician's responsibility
- Poor resources
- Concern about cost and pill burden
- Care directed at acute problems
- Lack of knowledge of goals

Cardiovascular risk factors

- Advancing age
- Diabetes and other high blood glucose conditions
- Dyslipidaemia
- Genetic background
- High alcohol consumption
- Hypertension
- Insulin resistance
- Left ventricular hypertrophy
- Male gender
- Menopause
- Obesity
- Sedentary lifestyle
- Smoking

Bold text: modifiable risk factor
Oral manifestations of Diabetes mellitus
• Candidiasis
• Gingival inflammation
• Suppuration
• Tooth mobility
• Recurrent, acute/chronic gingival and periodontal infections
• Dental abscesses
• Xerostomia
• Increased salivary viscosity
• Angular cheilosis
• Parotid enlargement
• Burning mouth
• High caries rate in uncontrolled or poorly controlled diabetes

What is Diabetes?

Disease or Syndrome?
› METABOLIC SYNDROME characterized by high levels of blood glucose resulting from:
1. Defect in insulin production/secretion
2. Defect in insulin action/Glucose uptake
3. Both
› Chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism
› Concerns: Long-term damage, system dysfunction and failure of various organs.
Diabetes: Characteristic Symptomatology

A. Polyuria
B. Polyphagia
C. Polydipsia
D. Blurring of vision
E. Weight loss.

- **Severe form**: Ketoacidosis or a Nonketotic hyperosmolar state
- Disorientation in time/ space / person
- Stupor
- Coma
- Death
- Often ASYMPTOMATIC!

Diabetes Long-term Effects

- Progressive **Retinopathy** with potential blindness
- Chronic **Nephropathy** that may lead to ARF (Acute renal failure)
- Peripheral **Neuropathy** with risk of foot ulcers, Charcot joints, autonomic dysfunction including sexual dysfunction, amputation.

**CVS:**
- Cardiovascular, peripheral vascular and cerebrovascular disease.
- Thromboembolism
- Inflammatory disorders: Periodontitis

Inflammation and Diabetes

[Diagram showing the relationship between genes and environment, adipose tissue aging, adipocyte, macrophage, oxidative stress, DNA damage, telomere dysfunction, and various inflammatory and metabolic processes related to diabetes.]
Diabetes and Periodontal disease?

Proposed mechanism of Diabetes-Periodontitis association

- Altered host response
- Alterations in connective tissue
- Microangiopathy
- Alterations in gingival crevicular fluid
- Altered subgingival microflora
- Hereditary predisposition
Bacteria
Dental biofilm
Gingivitis
Periodontitis

Inflammatory reactions

RISK FACTORS
• Diabetes mellitus
• Smoking
• Poor oral hygiene
• Male gender
• Race/ethnicity
• Low socio-economic status

DENTAL EFFECTS
• Connective tissue degradation
• Alveolar bone resorption
• Tooth mobility
• Abscess
• Tooth loss

Metalloproteinases and prostaglandins
Types of Diabetes

- Type 1 Diabetes Mellitus
- Type 2 Diabetes Mellitus
- Gestational Diabetes
- Other types:
  - LADA (Latent Autoimmune Diabetes in Adults)
  - MODY (Maturity onset diabetes of youth)
  - Secondary Diabetes Mellitus

Type 1 Diabetes Mellitus (T1DM)

- Previously Insulin-dependent diabetes mellitus (IDDM) or juvenile-onset diabetes.
- Innate immune system destroys pancreatic beta cells (Insulin deficit / Glucagon excess)
- Usually strikes children and young adults, although disease onset can occur at any age.
- Type 1 diabetes may account for 5% to 10% of all diagnosed cases of diabetes.
- Risk factors: Autoimmune, genetic, environmental
Diabetes mellitus type-1

- Also called insulin dependent diabetes or Juvenile diabetes.
- Autoimmune destruction of insulin-producing (beta cells) of the pancreas
- Results in total insulin deficiency
- Affects 1 in 300 children/adults

The Pancreas

Alpha Cells: Glucagon
Beta Cells: Insulin
Delta Cells: Somatostatin
F/G Cells: Gastrin/other pancreatic hormones

What is Insulin? What does it do?
Acidosis: Ketones build up in blood pH.
Ketouria: Ketones in urine
End artery/ end-organ damage: Macular degeneration of the retina

Type 2 Diabetes mellitus

- Non-insulin-dependent diabetes mellitus (NIDDM)
- Adult-onset diabetes.
- 90% to 95% of all diagnosed diabetes
- Begins as insulin resistance, a disorder in which the cells do not use insulin properly
- As the need for insulin rises, the pancreas gradually loses its ability to produce insulin.
Type 2 Diabetes

- Older age, obesity, family history, history of gestational diabetes, impaired glucose metabolism, physical inactivity, race/ethnicity
- African Americans, Hispanic/Latino Americans, American Indians, Asian Americans and Native Hawaiians
- Type 2 diabetes is increasingly being diagnosed in children and adolescents.
What is β-cell dysfunction?

- Major defect in individuals with type 2 diabetes
- Reduced ability of β-cells to secrete insulin in response to hyperglycemia

Insulin resistance and β-cell dysfunction are core defects of type 2 diabetes

- Genetic susceptibility, obesity, Western lifestyle
- Insulin resistance
- β-cell dysfunction
- Type 2 diabetes

Why does the β-cell fail?

- Chronic hyperglycemia
- Glucotoxicity
- Lipotoxicity
- Pancreas
- High circulating free fatty acids
- β-cell dysfunction
- Oversecretion of insulin to compensate for insulin resistance

- 1, 2
- 3
More than 80% of patients progressing to type 2 diabetes are insulin resistant.

Insulin resistant; low insulin secretion (16%)

Insulin resistant; good insulin secretion (1%)

83%

Insulin resistant; low insulin secretion (54%)

Insulin sensitive; good insulin secretion (29%)

Insulin sensitive; low insulin secretion (16%)

Insulin resistance – reduced response to circulating insulin

Insulin resistance

Liver

Muscle

Adipose tissue

Glucose output

Glucose uptake

Glucose uptake

Hyperglycemia

Insulin resistance is as strong a risk factor for cardiovascular disease as smoking

Odds ratio for incident CHD

Age  Smoking  Total cholesterol HDL cholesterol  Insulin resistance

0.6  1.0  1.4  1.8
Insulin resistance is closely linked to cardiovascular disease

- Present in > 80% of people with type 2 diabetes
- Approximately doubles the risk of a cardiac event
- Implicated in almost half of CHD events in individuals with type 2 diabetes

Insulin resistance is linked to a range of cardiovascular risk factors

- Hyperglycemia
- Dyslipidemia
- Hypertension
- Damage to blood vessels
- Clotting abnormalities
- Inflammation

Metabolic Syndrome

Loss of β-cell function occurs before diagnosis

- Up to 50% loss
Gestational diabetes
- A form of glucose intolerance during pregnancy.
- More frequent among African Americans, Hispanic/Latino Americans, American Indians.
- Obese women with a family history of diabetes.
- During pregnancy, gestational diabetes requires treatment to normalize maternal blood glucose levels to avoid complications in the infant.
- After pregnancy, 5-10% of women with gestational diabetes have type 2 diabetes.
- Women with GD have a 20% to 50% chance of developing diabetes in the next 5-10 years.

LGA Babies: #1 Cause?
Other types of DM

- Other specific types of diabetes result from specific genetic conditions
- Maturity-onset diabetes of youth
- Surgery
- Drugs
- Malnutrition
- Infections
- Such types of diabetes may account for 1% to 5% of all diagnosed cases of diabetes.

LADA

- Latent Autoimmune Diabetes in Adults (LADA) is a form of autoimmune (type 1 diabetes) which is diagnosed in individuals who are older than the usual age of onset of type 1 diabetes.
- Alternate terms that have been used for “LADA” include Late-onset Autoimmune Diabetes of Adulthood, “Slow Onset Type 1” diabetes
- Often, patients with LADA are mistakenly thought to have type 2 diabetes, based on their age at the time of diagnosis.

MODY (Maturity Onset Diabetes of the Young)

- Monogenic Autosomal dominant form of Diabetes
  - Mutations in transcription factors or in Glucokinase enzyme lead to insufficient insulin release from pancreatic β-cells
  - Originally, diagnosis of MODY was based on presence of nonketotic hyperglycemia in adolescents or young adults in conjunction with a family history of diabetes.
  - Genetic testing has shown that MODY can occur at any age and that a family history of diabetes is not always obvious.
Drug induced Diabetes Mellitus

- Atypical Antipsychotics - Alter Insulin receptor binding
- Beta-blockers - Inhibit insulin secretion, Glucocorticoid
- Calcium Channel Blockers - Inhibit secretion of insulin
- Corticosteroids - Peripheral insulin resistance and Gluconeogenesis
- Fluoroquinolones - Block ATP sensitive potassium channels.
- Naicin - Increased free fatty acid mobilization
- Phenothiazines (Typical) - Inhibit insulin secretion
- Protease Inhibitors - Inhibit the conversion of proinsulin to insulin.
- Thiazide Diuretics - Inhibit insulin secretion due to hypokalemia
- Furosemide- Increased insulin resistance, FFA mobilization
- Lithium/ Alcohol/ Opioids/ Rodenticides

Diagnosis of Diabetes Mellitus

Values for Diagnosis of Diabetes Mellitus

<table>
<thead>
<tr>
<th>Glucose Concentration, mmol/L (mg/dL)</th>
<th>Venous</th>
<th>Capillary</th>
<th>Venous</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diabetes Mellitus:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting</td>
<td>≥ 6.1 (110)</td>
<td>≥ 6.1 (110)</td>
<td>≥ 7.0 (126)</td>
</tr>
<tr>
<td>or</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-hour post-glucose load or both</td>
<td>≥ 10.0 (180)</td>
<td>≥ 11.1 (200)</td>
<td>≥ 11.1 (200)</td>
</tr>
<tr>
<td>Impaired Glucose Tolerance (IGT):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting (if measured)</td>
<td>&lt; 6.1 (&lt; 110)</td>
<td>&lt; 6.1 (&lt; 110)</td>
<td>&lt; 7.0 (&lt; 126)</td>
</tr>
<tr>
<td>and</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-hour post-glucose load</td>
<td>≥ 6.7 (120) &amp; &lt; 10.0 (&lt; 180)</td>
<td>≥ 7.8 (140) &amp; &lt; 11.1 (&lt; 200)</td>
<td>≥ 7.8 (140) &amp; &lt; 11.1 (&lt; 200)</td>
</tr>
<tr>
<td>Impaired Fasting Glycaemia (IFG):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting (if measured)</td>
<td>≥ 5.6 (100) &amp; &lt; 6.1 (&lt; 110)</td>
<td>≥ 6.6 (120) &amp; &lt; 6.1 (&lt; 110)</td>
<td>≥ 6.1 (120) &amp; &lt; 7.0 (&lt; 126)</td>
</tr>
<tr>
<td>and</td>
<td></td>
<td></td>
<td></td>
</tr>
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<td>&lt; 6.7 (&lt; 120)</td>
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<td>&lt; 7.8 (&lt; 140)</td>
</tr>
</tbody>
</table>
Management of Diabetes Mellitus

The major components of the treatment of diabetes are:

A. Diet

- Diet and Exercise
- Oral hypoglycaemic therapy
- Insulin Therapy

Diet is a basic part of management in every case. Treatment cannot be effective unless adequate attention is given to ensuring appropriate nutrition.

Dietary treatment should aim at:
- Ensuring weight control
- Providing nutritional requirements
- Allowing good glycemic control with blood glucose levels as close to normal as possible
- Correcting any associated blood lipid abnormalities
- Monitoring daily intake
- Decreasing salt intake
B. Oral Anti-Diabetic Agents

- Five classes of oral anti-diabetic agents:
  
i. Biguanides
  ii. Insulin Secretagogues – Sulphonylureas
  iii. Insulin Secretagogues – Non-sulphonylureas
  iv. α-glucosidase inhibitors
  v. Thiazolidinediones (TZDs)

B.1 Oral Agent Monotherapy

*If glycemic control is not achieved*

- HbA1c > 6.5%
- FPG > 7.0 mmol/L or RPG > 11.0 mmol/L with lifestyle modification within 1–3 months,
- ORAL ANTI-DIABETIC AGENT should be initiated.

*In the presence of marked hyperglycemia in newly diagnosed symptomatic type 2 diabetes*

- HbA1c > 8%
- FPG > 11.1 mmol/L or RPG > 14 mmol/L
- Oral anti-diabetic agents can be considered at the outset

Criteria for the Diagnosis of Diabetes

- A1C ≥ 6.5%
  OR
  - Fasting plasma glucose (FPG) ≥ 126 mg/dL (7.0 mmol/L)
    OR
    - 2-h plasma glucose ≥ 200 mg/dL (11.1 mmol/L) during an OGTT
      OR
      - A random plasma glucose ≥ 200 mg/dL (11.1 mmol/L)
B.1 Oral Agent Monotherapy

As first line therapy:
- Obese type 2 patients: Metformin, Acarbose or TZD.
- Non-obese type 2 patients: Metformin or insulin secretagogues

Metformin is the drug of choice in overweight/obese patients. TZDs and Acarbose are acceptable alternatives in those who are intolerant to metformin.

If monotherapy fails, a combination of TZDs, Acarbose and Metformin is recommended.

If targets are still not achieved, insulin secretagogues may be added.

B.2 Combination Oral Agents

Combination oral agents is indicated in:
- Newly diagnosed symptomatic patients with HbA1c >10
- Patients who are not reaching targets after 3 months on monotherapy
- Consider intermediate-acting / long-acting insulin
- Insulin dose can be increased until target FPG is achieved.

Primary sites of action of oral antidiabetic agents

- Glucosidase inhibitors
- Sulfonylureas/ meglitinides
- Biguanides
- Thiazolidinediones
- Carbohydrate breakdown/ absorption
- Insulin secretion
- Glucose output
- Insulin resistance
- Insulin resistance
The dual action of thiazolidinediones reduces HbA1c.
General Guidelines for Oral Anti-Diabetic Agents

- Oral agents NOT recommended for diabetes in pregnancy
- Insulin therapy is recommended for diabetes diagnosed during stress such as infections.
- In patients with co-morbidities, targets are individualized
- Start with minimal dose oral anti-diabetic agent, while reemphasizing diet and physical activity.
- 2-16 weeks should be given to allow achievement of steady state blood glucose control
- In elderly non-obese patients, long acting Sulphonylureas are to be avoided. Renal function should be monitored.

C. Insulin Therapy: When to use?

Short-term use:
- Acute illness, surgery, stress and emergencies
- Pregnancy
- Breast-feeding
- Insulin may be used as initial therapy in type 2 diabetes
- In emergency hyperglycaemia
- Severe metabolic decompensation (diabetic ketoacidosis, hyperosmolar nonketotic coma, lactic acidosis, severe hypertriglyceridaemia)

<table>
<thead>
<tr>
<th>Types of insulins</th>
<th>Brand names (generic name in brackets)</th>
<th>Basal/short-acting</th>
<th>Dosing schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid-acting (clear)</td>
<td>Humalog® (lispro insulin) NonRapid® (insulin aspart)</td>
<td>Bolus</td>
<td>Usually taken right before eating or to lower high blood glucose</td>
</tr>
<tr>
<td>Short-acting (clear)</td>
<td>Humulin®-R (insulin regular)</td>
<td>Bolus</td>
<td>Taken about 30 minutes before eating or to lower high blood glucose</td>
</tr>
<tr>
<td>Long-acting (clear)</td>
<td>Humulin®-P (insulin protamine zinc)</td>
<td>Basal</td>
<td>Often taken at bedtime, or twice a day (morning and bedtime)</td>
</tr>
<tr>
<td>Intermediate-acting (clear)</td>
<td>Lantus® (insulin detemir) Levemir® (insulin detemir)</td>
<td>Basal</td>
<td>Usually taken once or twice a day</td>
</tr>
</tbody>
</table>

Premixed (clear):
A single vial contains a fixed ratio of insulins (the numbers refer to the ratio of rapid- or short-acting to intermediate-acting insulin in the mix):
- Humalog® Mix 25: Humulin® (10/40, 30/70) NovoRapid® (10/100, 20/80, 30/70, 40/60, 50/50)
Combination of basal and bolus insulins Depends on the condition
Dental Recommendations

- DHCP may detect undiagnosed cases of diabetes and refer patients to physicians for further evaluation.
- Team: physician, nutritionist, dental hygienist and dentist can maintain the patient's oral health and possibly improve the patient’s metabolic control
- Reduce comorbidity factors by supporting patients in tobacco-use cessation programs.
- Prolonged and severe hyperglycemia is associated with systemic and oral complications.

Dental Recommendations

- Management Plan: Consider the following.
- age
- school or work schedule and conditions
- physical activity
- medications (insulin or oral hypoglycemic)
- diet and eating patterns
- social situation and personality
- cultural factors
- the presence of complications (systemic/oral)
- any other medical conditions.
### Dental Complications

- The oral complications of uncontrolled diabetes mellitus are devastating.
- gingivitis and periodontal disease
- xerostomia and salivary gland dysfunction
- susceptibility to bacterial, viral and fungal (that is, oral candidiasis) infections
- caries; periapical abscesses; loss of teeth;
- impaired ability to wear dental prostheses
- taste impairment
- lichen planus
- burning mouth syndrome

### Gingivitis and periodontal disease

- "sixth complication of diabetes mellitus"
- most common oral complication of diabetes.
- starts with gingivitis and with poor glycemic control, progresses to periodontal disease.
- In one study, the prevalence of periodontal disease was 9.8 % in 263 patients with type 1 diabetes, compared with 1.7 % in nondiabetes.
- Patients with type 1 diabetes and retinopathy tend to exhibit more loss of periodontal attachment by the 4th-5th decades of life.
- Good oral hygiene and frequent dental checks

### Gingivitis and periodontal disease

- Periodontitis appears to be related to several pathological events including plaque
- Periodontal flora similar in diabetics and non-diabetics
- Differences in the host response to periodontal pathogens cause increased tissue destruction
- Impairment in cell-mediated immunity such as neutrophil (PMN) chemotaxis and macrophage function and vascular disease.
- History of chronic periodontal disease can disrupt control of diabetes, periodontal infections may have systemic repercussions.
Radiographic appearance of diabetic patient with severe periodontal destruction and bone loss.

Clinical photograph showing periodontal abscess in poorly controlled diabetic patient.

Diabetic patient with poor oral hygiene with dental carries and tooth loss.
**Gingivitis and periodontal disease**
- Altered response to infection
- Microvascular changes
- Increased glucose concentrations in the saliva
- Salivary hyperglycemia results in additional bacterial substrate and plaque formation.
- Increased gingival crevicular fluid glucose may diminish the ability of periodontal fibroblasts to contribute to periodontal healing.
- Preventive periodontal therapy must be included in the comprehensive care
- Explicit oral hygiene instruction and frequent periodic dental examinations and prophylaxis.

**Salivary gland dysfunction and xerostomia**
- Xerostomia and salivary hypofunction:
  - Polyuria
  - Underlying metabolic or endocrine problem
  - Dry, atrophic and cracking oral mucosa is the eventual complication
  - Accompanying mucositis, ulcers and desquamation
  - Inflamed, depapillated tongue. Difficulty in lubricating, masticating, tasting and swallowing are among the most devastating complications from salivary dysfunction and may contribute to impaired nutritional intake.

**Salivary gland dysfunction and xerostomia**
- An increase in the rate of dental caries has been reported in young patients with diabetes
- 1 Study showed patients with diabetes did not have a higher coronal or root-surface caries rate than patients without diabetes
- Association existed between older adults with diabetes and active caries and tooth loss; this was even more significant in patients with diabetes having poor glycemic control.
- The dentist can offer topical treatments such as fluoride-containing mouth rinses and salivary substitutes to help prevent caries and minimize discomfort
Oral Candidiasis

- Opportunistic fungal infection: hyperglycemia?
- Oral lesions: median rhomboid glossitis (central papillary atrophy), atrophic glossitis, denture stomatitis, pseudomembranous candidiasis (thrush) and angular cheilitis.
- Candida albicans is a constituent of the normal oral microflora that rarely colonizes and infects the oral mucosa without predisposing factors. These include immunologically compromised conditions (for example, AIDS, cancer or diabetes), the wearing of dentures in conjunction with poor oral hygiene and the long-term use of broad-spectrum antibiotics.

Oral Candidiasis

- Salivary dysfunction
- Compromised immune function
- Salivary hyperglycemia
- Provide a potential substrate for fungal growth

Burning Mouth Syndrome

- Symptoms of pain and burning are intense.
- In uncontrolled diabetes etiologic factors include salivary dysfunction, candidiasis and neurological abnormalities such as depression.
- Autonomic and sensory-motor neuropathies increase prevalence of oral neuropathy
- Neuropathy may lead to oral symptoms of paresthesias and tingling, numbness, burning or pain caused by pathological changes involving the nerves in the oral
- Improvement in glycemic control has allayed symptoms associated with burning mouth
### Lichen planus

- Common, chronic mucocutaneous disease
- Etiology: immunologically mediated process
- Hypersensitivity reaction characterized by an intense T lymphocytic infiltrate (CD4+ and especially CD8+ cells) located at the epithelial–connective tissue interface.
- Other immune-regulating cells (macrophages, dendritic cells, Langerhans' cells) are seen in increased numbers in lesions of lichen planus.
- A study of 40 patients with lichen planus found that 11 patients (28%) had overt or latent diabetes which may have initiated immunopathogenesis of lichen planus.

### Acute Infections of the Oral Cavity

- Recurrent bouts of herpes simplex virus
- Periodontal abscess
- Palatal ulcers. Case reports of life-threatening deep neck infection from periodontal abscess
- Ulcers were not superficial, but represented deep granulomatous disease.
- Pathogenic mechanisms associated with the increased susceptibility to periodontal infections (impaired wound healing, diminished chemotaxis and PMN function) may play a role in the greater likelihood of developing acute oral infections.

### SUMMARY OF GENERAL MANAGEMENT

- Assess Glycemic control
- Refer patients with signs of undiagnosed diabetes to a physician for diagnosis and treatment
- Obtain a consult with the patient’s physician if systemic complications are present
- Assess the use of medications for oral complications
- Use a glucometer to avert dental chair emergencies
- Aggressively treat acute oral infections
- Schedule patients for frequent recall visits to monitor and treat oral complications
- Maintain optimal oral hygiene and diet
- Support and follow up patients in smoking-cessation programs
### Dental Management

- **Candidiasis**: Clotrimazole troches contain relatively high levels of sugar.
- Nystatin vaginal suppositories have been useful along with lozenges (sugar containing)
- Antifungal creams: Clotrimazole 1%, Miconazole 2%, Ketokonazole 2% may contain Hydrocortisone-Iodoquinol combinations
- Clotrimazole-betamethasone
- Nystatin-Triamcinolone
- Angular chelitis: Antifungal-steroid-anti-inflammatory creams

### Management of HSV

- Recurrent orofacial HSV infection
- Treatment initiated as early as possible in the prodromal stage to reduce duration/symptoms of the lesion.
- Oral acyclovir, prophylactically and therapeutically, may be considered
- Frequent recurrent herpetic episodes interfere with daily function and nutrition.
- In the patient with diabetes and renal insufficiency or renal failure, acyclovir should be avoided because of its potential for nephrotoxicity

### Management of Burning Mouth Syndrome

- In diabetes, xerostomia and candidiasis may increase symptoms of burning mouth.
- Low dosage benzodiazepines (Clonazepam/Midazolam)
- Tricyclic antidepressants (amitriptyline, nortriptyline)
- Anticonvulsants (Carbamazepine and gabapentin)
- Potential side effects include xerostomia.
- Consultation with the patient's physician is necessary because of the potential of these drugs for addiction and dependence.
Surgical considerations

- Periodontal surgical procedures may be performed although it is important to maintain a normal diet during the postsurgical phase to avoid hypoglycemia and ensure effective repair.
- DHCP must review any previous history of complications, assess the patient’s glycemic control.
- Consult: Physician and nutritionist.
- Supportive periodontal therapy at close intervals (two to three months).

Surgical Considerations

- Periodontal infections may complicate the severity of diabetes mellitus and the degree of metabolic control.
- Well-controlled diabetic generally does not require post-surgical antibiotics.
- However, the administration of post-surgical antibiotics is appropriate if there is significant infection, pain and stress.
- The selection of antibiotics is predicated on sensitivity and specificity results, spread of infection and past control.

Dental Recommendations

- The mainstay of periodontal therapy for patients with diabetes is nonsurgical.
- Combination of nonsurgical debridement and tetracycline antibiotic therapy in diabetics with advanced periodontitis may have a potential positive influence on glycemic control.
- Multiple studies: The use of Tetracyclines in the treatment of periodontal disease was associated with an improvement in glycemic control as assessed by HbA1c assays.
Surgical Considerations

Additional therapeutic benefit of Tetracyclines:
• Inhibitors of connective tissue–degrading enzymes matrix metalloproteinases
• Low-dose Doxycycline has been shown to inhibit human gingival crevicular fluid collagenase at doses that are not antimicrobial
• Eliminates risk of bacterial resistance.
• Tetracyclines can function as inhibitors of bone resorption or bone loss
• This is independent of it's antimicrobial use

Dental Vignette 4: Barry Ateric

• Morbidly obese 50 year old male
• Presents with severe 3rd molar pain x 1 week
• Pain is unbearable. Anxious but comfortable.
• Last visit to the dentist > 4 years ago
• On several different medications
• He states that he took his medication this morning, but he did not eat breakfast because it was painful to chew. He states he will eat after his appointment.

Dental Vignette 4: Barry Ateric

Rx:
• Glucophage XR (Metformin): Biguanide 500 BID
• Avandia (Rosiglitazone): Thiazolidinedione 2 OD
• Diabeta (Glibenclamide): Sulfonylurea 1.25 BID
• Avapro (Irbesartan): ARB 150 OD
• Apo-Hydro (Hydrochlorothiazide HCTZ): 12.5 OD
• Zocor (Simvastatin): HMG CoA Reductase Inhibitor 20 BID
• Ecotrin (ASA): 81 OD
• Advil (Ibuprofen): 600 PRN
• Centrum Adult: OD
Dental Vignette 4: Barry Ateric

- Once in the chair Barry starts profusely sweating
- You notice he is trembling and twitching
- Appears to be agitated
- Starts speaking in high pitched/incoherent voice
- Treatment is stopped
- Patient tries to stand up, stumbles and falls
- He collapses in the dental chair and seems to have lost consciousness.
- Pulse is 98/min, BP: 160/110mmHg
- Next steps?

Hypoglycemia

Low blood sugar symptoms include:
- Headache
- Shaking
- Feeling tired
- Weakness
- Hunger

Consequences of Hypoglycemia

- Cognitive, psychological changes (Confusion, irritability)
- Falls
- Recurrent episodes of hypoglycemia
- Uncontrolled diabetes
- Dementia (elderly)
- CV events
  - Cardiac autonomic neuropathy
  - Cardiac ischemia
  - Angina
  - Fatal arrhythmia
Symptoms of Hypoglycemia

<table>
<thead>
<tr>
<th>Classification</th>
<th>Blood Glucose Level (mg/dL)</th>
<th>Typical Signs and Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild hypoglycemia</td>
<td>~50-70</td>
<td>• Neurogenic: palpitations, tremor, hunger, sweating, anxiety, paresthesia</td>
</tr>
<tr>
<td>Moderate hypoglycemia</td>
<td>~50-70</td>
<td>• Neuroglycopenic: behavioral changes, emotional lability, difficulty thinking, confusion</td>
</tr>
<tr>
<td>Severe hypoglycemia</td>
<td>&lt;50*</td>
<td>• Central:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Severe confusion, unconsciousness, seizure, coma, death</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Requires help from another individual</td>
</tr>
</tbody>
</table>

*Severe hypoglycemia symptoms should be treated regardless of blood glucose level.

Treatment of Hypoglycemia

- Hypoglycemia symptoms (BG <70 mg/dL)
  - Patient conscious and alert
    - Consume glucose-containing foods (fruit juice, soft drink, crackers, milk, glucose tablets); avoid foods also containing fat
    - Repeat glucose intake if SMBG result remains low after 15 minutes
    - Consume meal or snack after SMBG has returned to normal to avoid recurrence
  - Patient severely confused or unconscious (requires help)
    - Glucagon injection, delivered by another person
    - Patient should be taken to hospital for evaluation and treatment after any severe episode

Critical Questions in the Dental Office

a. What type of diabetes do you have, and when was it diagnosed?
b. Have you been experiencing any health problems over the last few days, weeks, or months?
c. Are you taking all of the medications that have been prescribed for you? If not, which one(s) don't you take and why?
d. What is your A1C level? When was the last A1C taken?
Critical Questions in the Dental Office

e. How often do you check your blood glucose level, and what was the most recent value?
f. Do you watch your carbohydrate intake and follow an exercise regime? What time did you last eat? What did you consume?
g. Who helps you manage your diabetes? Do you see your physician, nurse, or dietitian on a regular basis? When was your last visit?

Critical Questions in the Dental Office

h. Do you experience low blood sugar levels? If so, how often? What are your symptoms? When was your last event?
i. Do you smoke or use any tobacco products? If so, how much?
j. Do you drink alcoholic beverages? If so, how often and how much do you drink on a weekly basis?
k. Have you taken your diabetes medication today?

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