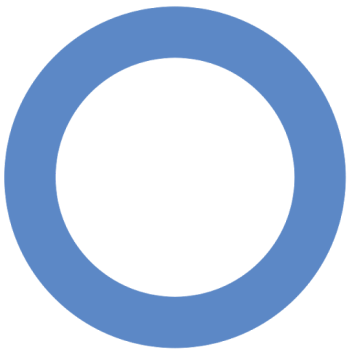
DIABETES



world diabetes day

14 November

Objectives:

- The student should have an understanding of the pathogenesis and major histopathological changes seen in diabetes mellitus type 1 and type 2.
- The student should recognize the major complications of diabetes mellitus.

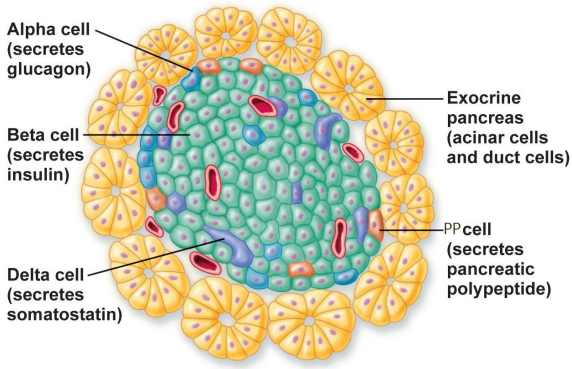
Important note: **Please check out this link before viewing the file to know if there are any additions or changes. The same link will be used for all of our work: Pathology Edit**

Red: Important
Dr. Rikaby notes
Grey: Extra notes

Introduction.

Islets of Langerhans (endocrine pancreas) contain 4 major and 2 minor cell types.

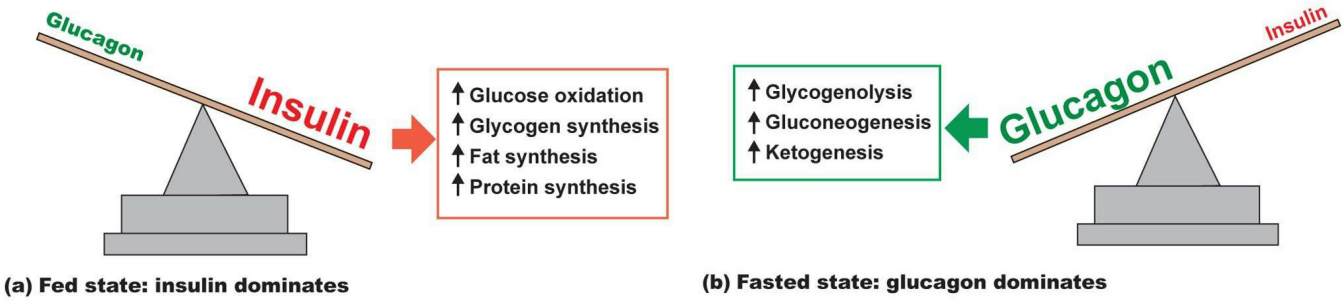
- **Major cell types:**
  1. **β** cell produces insulin.
  2. **α** cell secretes glucagon.
  3. **δ** cells contain somatostatin, which suppresses both insulin and glucagon release.
  4. **PP** cells contain a unique pancreatic polypeptide.
- **Minor cell types:**
  - D1 cells.
  - Enterochromaffin cells.



Glucose homeostasis:

<table>
<tr><th colspan="3">Postprandial[1] state</th><th>Fasting State</th></tr>
<tr><td colspan="3">High blood glucose level → secretion of insulin from beta cells → insulin binds to its receptors on peripheral tissue (liver, muscles, adipose tissue) and exert its <u>anabolic effects:</u></td><td rowspan="2">low blood glucose level → secretion of glucagon from alpha cells → glucagon exert its <u>glycogenic effect</u> on the liver and causes

- ↑ Gluconeogenesis
- ↑ glycogenolysis
- ↑ ketogenesis</td></tr>
<tr><th>Liver</th><th>Muscles</th><th>Adipocytes</th></tr>
<tr><td>↓ Gluconeogenesis
↑ Glycogen synthesis
↑ Lipogenesis</td><td>↑ Glycogen synthesis
↑ Protein synthesis</td><td>↑ Glucose uptake
↑ Lipogenesis
↓ Lipolysis</td></tr>
</table>



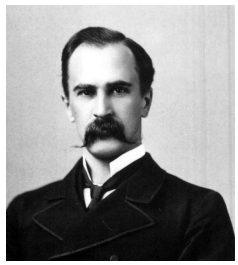
(a) Fed state: insulin dominates

(b) Fasted state: glucagon dominates

★ In a normal person, the extracellular concentration of glucose in fed and fasting states is maintained in a tightly limited range.

[1] after eating food.

Diabetes Mellitus.



Sir William Osler defined diabetes mellitus as: a syndrome due to a disturbance in carbohydrate metabolism from various causes, in which sugar appears in the urine, associated with thirst, polyuria, wasting and imperfect oxidation of fats.

Diabetes mellitus is: An inadequate release of insulin, or insulin resistant.

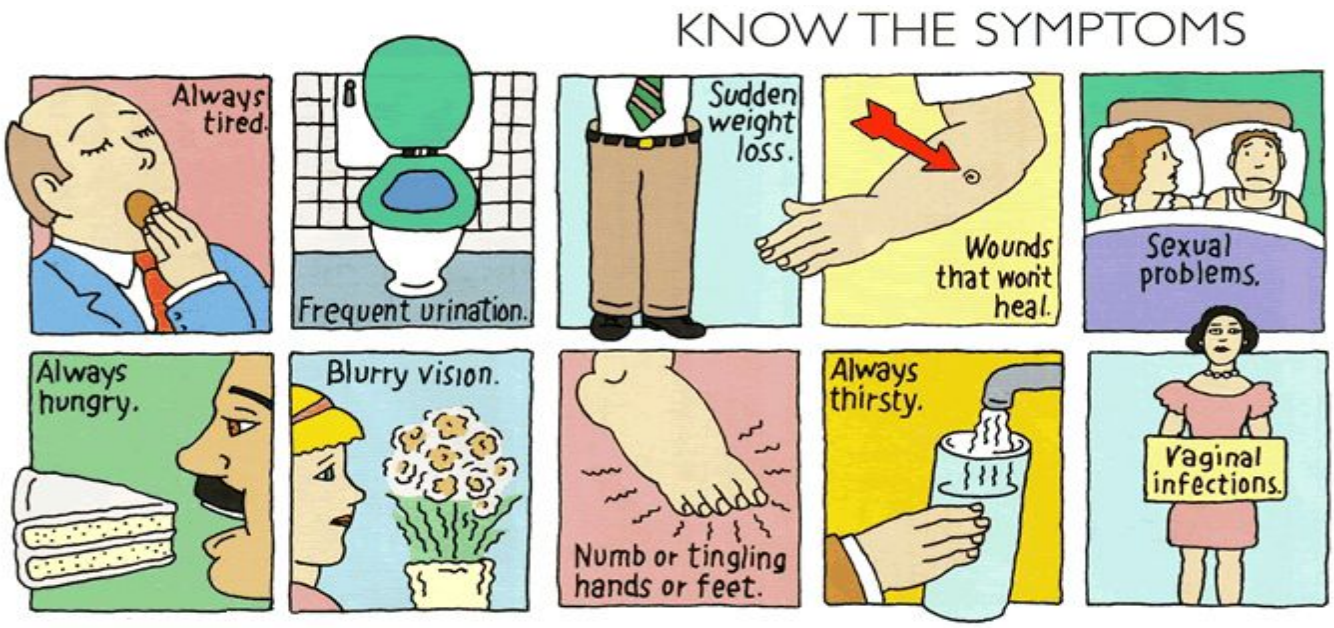
- Not a single disease entity but rather a group of metabolic disorders sharing the common underlying feature of hyperglycemia.
- The **chronic hyperglycemia** and attendant **metabolic deregulation** of diabetes mellitus may be associated with secondary damage in multiple organ systems, especially the **kidneys, eyes, nerves, and blood vessels.**

EPIDEMIOLOGY.

- Very common metabolic disorder, and it is increasing in prevalence throughout the world, diabetes in KSA and in the Middle East is more common than other countries. 16-20% of population in gulf countries have diabetes either clinical or subclinical (they don't know).
- According to the American Diabetes Association, diabetes affects over 20 million children and adults, or 7% of the population, in the United States.
- Approximately 1.5 million new cases of diabetes are diagnosed each year in the United States.

Types of diabetes.	
Type 1 DM ***(10%)***	● Formerly known as **insulin-dependent (IDDM)** or **juvenile-onset diabetes.** ● Caused by **autoimmune destruction** of the insulin-producing Beta-cells.
Type 2 DM ***(80-90%)***	● Formerly known as **non–insulin-dependent (NIDDM)** or **maturity-onset diabetes.** ● Typically associated with genetic & obesity results **resistance** to the metabolic action of insulin in its target tissues and **inadequate secretion** of insulin from the pancreas.
Maturity-onset diabetes of the young (MODY) (T3DM)	● Resembles type 2 DM, but occurs in young age group. ● Rare *autosomal dominant* form of inherited diabetes, ● It has very strong family history & very common in KSA. ● Associated with a variety of **gene defects** that affect β-cell function, including **glucokinase** (an important sensor for glucose metabolism within the β-cell) and several mutations in genes that control the development and function of the β-cells. Mutations in these genes, however, do not account for the typical prevalent forms of T2DM.
Gestational diabetes	Develops in a few percent of healthy pregnant women, owing to the **insulin resistance** of pregnancy **combined with a β-cell defect,** but almost always abates following parturition[2]. ❑ May Put both Mother and Fetus at risk. ❑ These women highly susceptible to **overt T2DM later in life.** ❑ Pathogenesis: unknown but it is believed that there are some hormones produced by the placenta (estrogen, cortisol, and human placental lactogen) can have a blocking effect on insulin.
Secondary to other endocrine conditions	● **Secondary** to other **endocrine conditions** (Cushing's syndrome, pheochromocytoma, chronic pancreatitis or pancreatic cancer) or **drug therapy** (ex: Beta-blockers, glucocorticoids) ● Also known as T4DM.

[2] ولادة



Symptoms of diabetes:

1. Polydipsia [3].
2. Polyuria[4].
3. Glycosuria (this is due to high glucose in blood → high osmolarity → high glucose infiltration to kidney → high glucose in urine)
4. Loss of weight and muscle weakness.
5. Polyphagia[5] (high glucagon → lipolysis, gluconeogenesis and glycogenolysis)
6. Malaise.
7. Infection.

Diagnosis of DM: (REMEMBER: Normal blood glucose level 70 - 120 mg/dL)

Diagnosis of DM is made if any one of the following three criteria

1. **Random glucose concentration** greater than 200 mg/dL (with classical signs & symptoms)
2. **Fasting glucose concentration** greater than 126 mg/dL on more than one occasion.
3. **Postprandial glucose concentration** [oral glucose tolerance test (OGTT)], in which glucose concentration is greater than 200 mg/dL 2 hours after a standard carbohydrate load.
4. **HbA1c:** Glycated hemoglobin is a form of hemoglobin that is measured primarily to identify the three month average (approximate life span of RBCs) plasma glucose concentration, normal result is 5.6% or less, means controlled DM, while more than 6.4% means uncontrolled DM.

Management:

- **Type 1** : **Insulin** absolutely required.
- **Type 2** : lifestyle modification; **diet**, **exercise**, **oral drugs**, often insulin supplement need.

[3] increased thirst
[4] increase in urine frequency
[5] increase in appetite

Type 1 Diabetes Mellitus

It is an autoimmune destruction of the B cells in the islets of Langerhans.

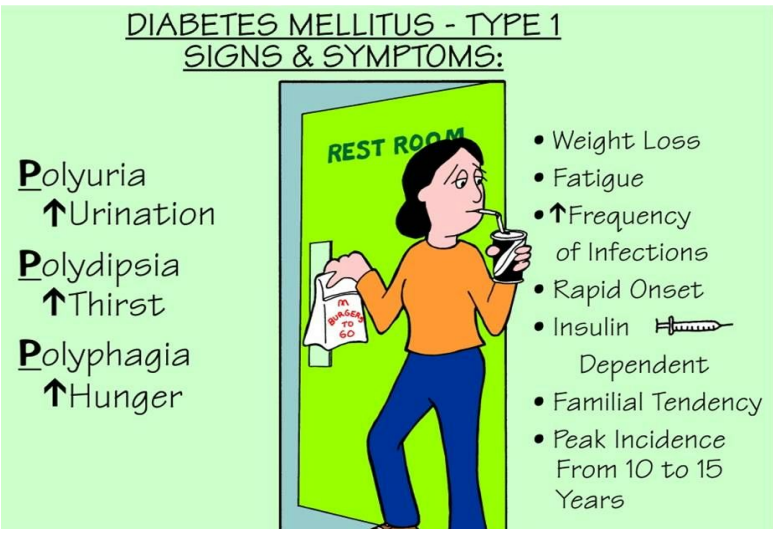
The disease is characterized by:

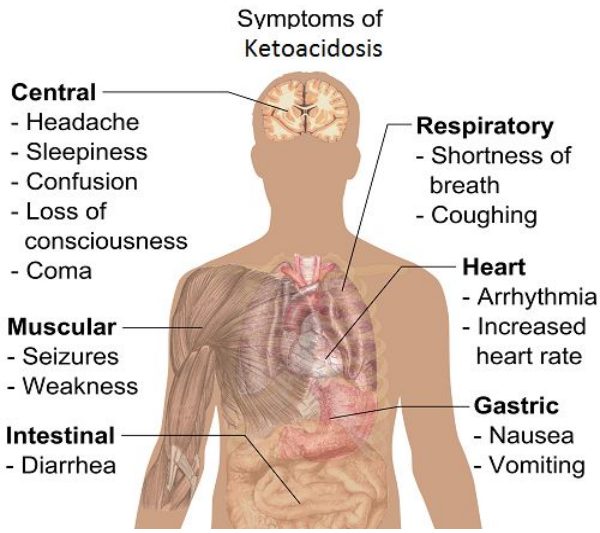
- ❑ **Few or no functional B cells in the islets of Langerhans** → Extremely limited or nonexistent insulin secretion.
- ❑ **Hyperglycemia** results from unsuppressed hepatic glucose output and reduced glucose disposal in skeletal muscle and adipose tissue which leads to **glycosuria** and **dehydration** from loss of body water into the urine.
- ❑ As a result, body **fat** rather than glucose is preferentially metabolized as a **source of energy via** oxidation of fat → **ketone bodies** (acetoacetic acid, B-hydroxybutyric acid and acetone) which are released into the blood from the liver and lead to **metabolic ketoacidosis**.

(If uncorrected, the progressive acidosis and dehydration ultimately lead to coma and death)

EPIDEMIOLOGY

- T1DM is most common among northern Europeans and their descendants and is not seen as frequently among Asians, African-Americans, or Native Americans.
- Affects mainly children but it can develop at any age, the peak age is at **puberty**.
- Children of fathers with T1DM are 3 time more likely to develop the disease than children of diabetic mothers.
- Some older patients may present with **autoimmune β-cell destruction** that has developed slowly over many years.
- An increased incidence in late fall and early winter has been documented in many geographical areas. Some viruses have antigens similar to those in beta cells of pancreas. In winter, some people get infected by those viruses. The viruses then will target an autoimmune reaction against the beta cells of the pancreas in patients who have genetic susceptibility.
- <20 have a parent or sibling with the disease.
- Monozygotic twins: 50%
- Children with Fathers with T1DM are three times more likely to **develop** the disease than are children of diabetic mothers.

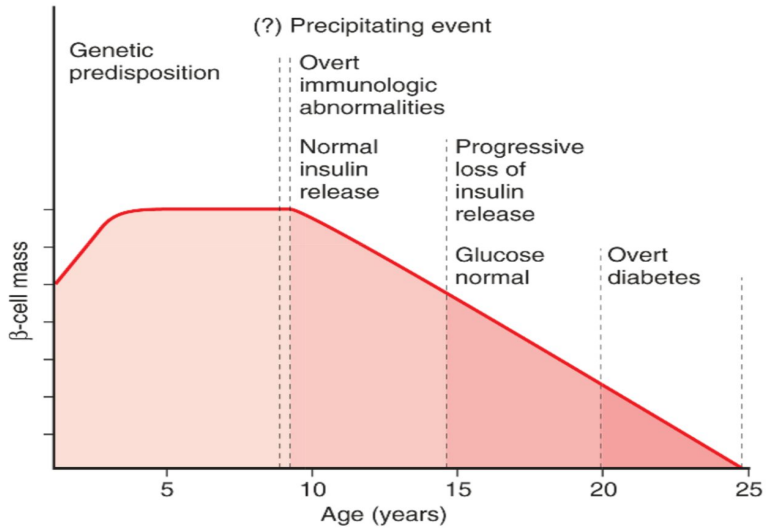




PATHOGENESIS:

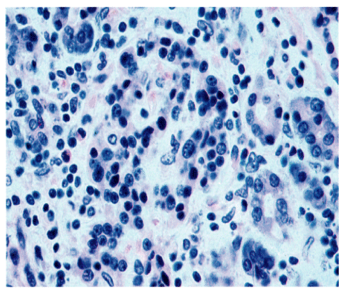
1. **Genetic susceptibility** associated with class II MHC molecules on **6p21** (HLA-D). Mostly with **HLA-DR3** and **HLA-DR4** + **Environmental factors** such as CMV, Mumps and group B **Coxsackie** , Rubella viruses → Autoimmunity.
2. **Autoimmunity:** Cell-mediated immune mechanisms، CD8+T lymphocytes predominate, although some CD4+cells are also present.(Direct injury)
3. The infiltrating inflammatory(indirect) cells also elaborate cytokines, for example, IL-1, IL-6, interferon-alpha, and nitric oxide, lead to → to B cell injury(generally develops slowly → years)
4. Many patients develop **islet cell antibodies** months or years before insulin production decreases and clinical symptoms appear.

An autoimmune origin for T1DM was initially suggested by the demonstration of circulating antibodies against components of the B cells (including insulin itself) in most newly diagnosed children with diabetes.



Detection of serum antibodies to islet cells and certain islet antigens remains a useful clinical tool for differentiating between type 1 and type 2 diabetes.

Morphology.



- **Insulitis:** Lymphocytic infiltrate in the islets, sometimes accompanied by a few macrophages and neutrophils.
- The exocrine pancreas in chronic T1DM often exhibits diffuse interlobular and intra- acinar **fibrosis**, accompanied by atrophy of the acinar cells.
- As the disease becomes **chronic**, Beta cells are progressively depleted.
- Fibrosis of the islets is uncommon.
- There is **NO** deposition of ***amyloid*** in the islets.

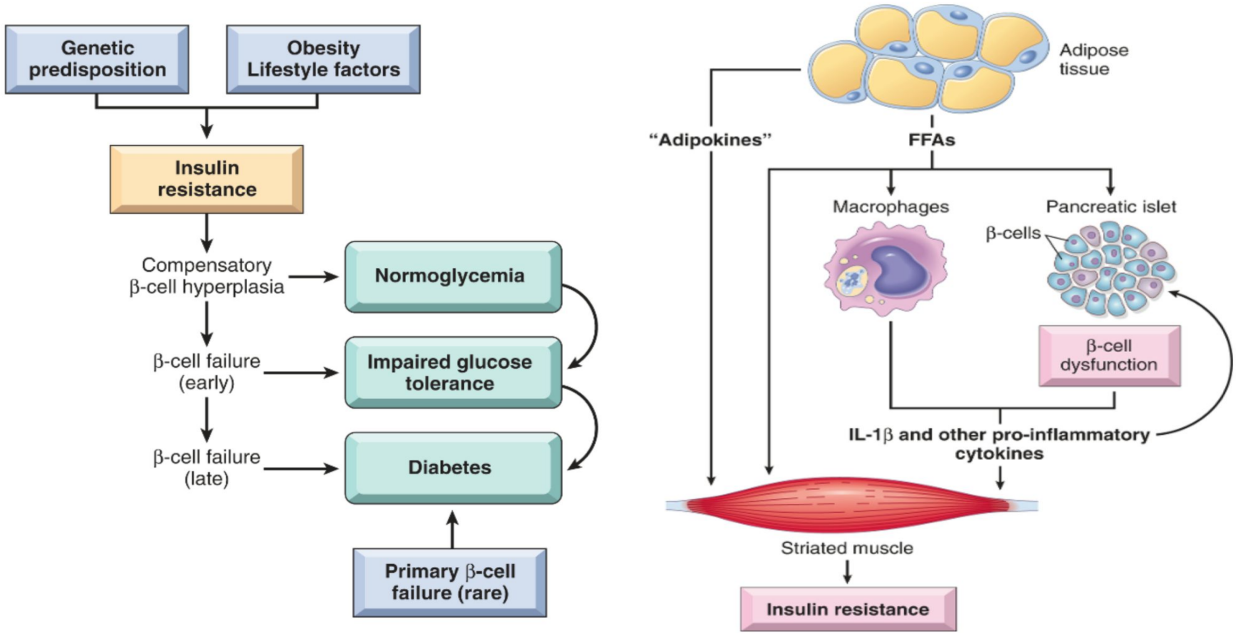
Type 2 Diabetes Mellitus

Insulin resistance is defined as the failure of target tissues to respond normally to insulin. It can be caused by variety of factors, mainly genetic predisposition, sedentary lifestyle, hypertension & obesity (which itself has strong genetic determinants).

PATHOGENESIS:

It is a heterogeneous disorder characterized by a combination of:

1. Reduced tissue sensitivity to insulin.
2. Inadequate secretion of insulin from the pancreas (failure of the B-cells increased demand for insulin).



It could be caused by two mechanisms:

A) Genetic predisposition and environmental factors such as obesity:

1. The large amount of fat in circulation deposit in organs (liver & muscles) → inhibit insulin signaling by influencing insulin receptors (either numbural or structural) → organs become resistance to insulin.
2. There will be increase in demand of insulin in the body → beta cells of pancreas will secrete large amount of insulin → hyperinsulinemic state will compensate for peripheral resistance and keep the glucose level normal for several years.
3. After several years, beta cells of pancreas will become exhausted (it will lose its mass) *or* sometimes it can be affected by the chronically elevated plasma levels of FFA that occur in obese persons (induce the secretion of pro-inflammatory cytokines that cause more beta cell dysfunction) → impaired glucose tolerance → diabetes.

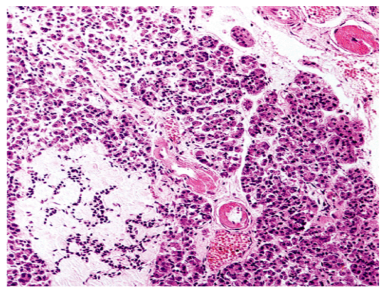
B) Rarely primary beta cell failure → lead directly to type 2 diabetes without an intervening state of insulin resistance.

- Beta cells functional abnormality is <u>specific for glucose</u>, since the B-cells retain the ability to respond to other stimulants, such as amino acids

EPIDEMIOLOGY.

- The disease usually develops in adults, with an increased prevalence in **obese** persons *(80% of patients overweight)* & in the **elderly** *(10% of persons older than 65 years old).*
- Recently, T2DM has been appearing in increasing numbers in younger adults and adolescents, due to **obesity** and **lack of exercise**.
- Strongly genetically associated (more than DM1) among monozygotic twins → both are almost always affected (90%).
- **No association** with genes of the major histocompatibility complex (**MHC**), as seen in T1DM.

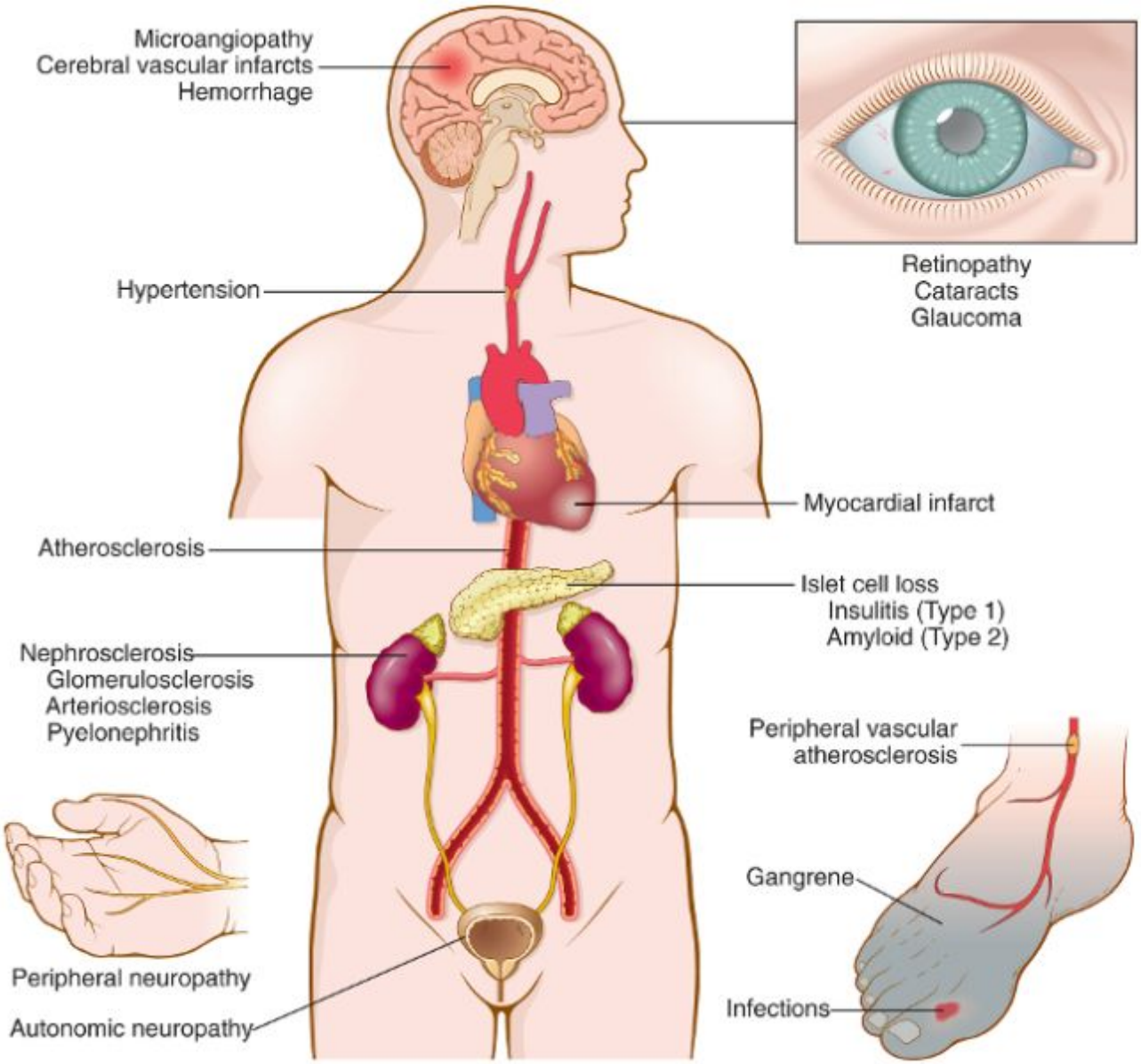
Histological Findings:



- No consistent reduction in the number of B-cells reduction is most often is seen in type 1 diabetes.
- No morphologic lesions of B- cells.
- In some islets, fibrous tissue accumulates, sometimes to such a degree that they are obliterated.
- **Islet amyloid** is often present particularly in patients over 60 years of age. appearing as deposition of pink, amorphous.
- Amyloid is stained by **Congo Red** stain.

	Type 1 Diabetes Mellitus	Type 2 Diabetes Mellitus
onset	Usually before 20	Usually after 30
Speed of onset	**Abrupt**; **symptomatic** (polyuria, polydipsia, dehydration); often sever with **ketoacidosis**	**Gradual**; usually subtle hyperosmolarity
weight	Normal weight; recent weight loss is common	**Overweight**
genetics	Genetics < 20%	> **60%**
concordant in Monozygotic Twins	Monozygotic Twins 50% concordant	90% concordant
HLA Association	ABS to islet cell AG +	Negative
Histopathology	. Early—inflammation . Late—atrophy and fibrosis	Late-Fibrosis, amyloid
B-cell mass	**Markedly reduced**	**Normal or slightly reduced**
Insulin levels	**Markedly reduced**	**Elevated (early); normal or decreased (late)**

Complications of Diabetes.



1- Diabetic Microvascular Disease: Due to non-enzymatic glycosylation of proteins that deposit in basement membrane of small blood vessels→ hyaline *arteriolosclerosis* results in:

- ❏ Renal Failure and Blindness.
- ❏ Frequent occurrence of hypertension → development of arteriolar lesions.
- ❏ Aggregation of platelets in smaller blood vessels and impaired fibrinolytic mechanisms.

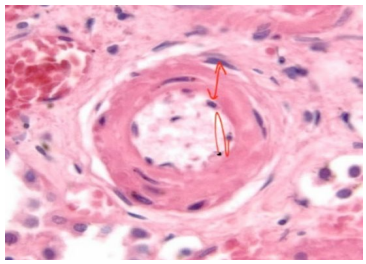
2- Diabetic Macrovascular Disease: Due to non-enzymatic glycosylation of proteins that deposit in basement membrane of medium-sized blood vessels → atherosclerosis. such as :

- ❏ Reduce blood flow to the heart → coronary atherosclerosis.
- ❏ healing of chronic ulcers that develop from trauma and infection of the feet in diabetic patients is commonly defective

3- Osmotic damage: Some cells uptake glucose without the need of insulin, such as schwann cells, pericyte of retinal blood vessels and lens → hyperglycemia → more glucose → converted into sorbitol by *aldose reductase* which requires NADPH. NADPH is an important factor in Glutathione system (antioxidant) → depletion of NADPH → increase cellular oxidative stress → cellular damage.

- ❏ Diabetic neuropathy.
- ❏ Blindness and cataract.

Diabetic Nephropathy.



Pathogenesis:

- hyaline Arteriolosclerosis of efferent arterioles → hyperfiltration → glomerulosclerosis (kimmelstiel-Wilson disease) → **nephrotic syndrome**
- hyaline Arteriolosclerosis of afferent arterioles → low blood supply in glomeruli → diffuse sclerosis → **chronic renal failure**

Epidemiology

- 30% to 40% of T1DM ultimately → renal failure, while up to 20% of T2DM .
- Diabetic nephropathy accounts for one third of all new cases of renal failure.
- The prevalence increases with the severity and duration of the hyperglycemia.
- Kidney disease due to diabetes is the most common reason for renal transplantation in adults.

Diabetic Retinopathy.

Pathogenesis: can be seen in two conditions:

- **Blindness due to:**
  1. High glucose in pericyte of retinal blood vessels → High sorbitol → increase the osmotic pressure of cells → damage of cells → aneurysm.
  2. Microvascular hyalinosis → ischemic infarction.
- **Cataract** High glucose in lens → High sorbitol → increase the osmotic pressure → damage of lens.

Epidemiology

- The risk is higher in T1DM than in T2DM, but more common T1DM.
- The most important cause of blindness in the Unites States in persons under the age of 60 years.

Diabetic Neuropathy

Pathogenesis:

- **Microvascular hyalinosis** involving the small blood vessels of nerves contributes to the disorder. The small blood vessels supplying the nerves is occluded → ischemic infarction or by metabolic factor or osmotic toxicity (damage) secondary to hyperglycemia.
- High glucose in schwann cells → High sorbitol → increase the osmotic → damage of cells → defect in melanin of PNS → diabetic neuropathy.

Characterized by

- The most ***common*** and distressing complications of diabetes.
- **burning pain and abnormal sensations in the extremities.**
- Affects Sensory and Autonomic Innervations,
- Changes in the nerves are complex, and abnormalities in axons, the myelin sheath, and Schwann cells have all been found.
- Peripheral neuropathy can leads → **foot ulcers.**
- Plays a role in the **painless destructive joint disease** that occasionally occur.

Infections.

Pathogenesis: Poorly Controlled DM → inhibition of phagocytosis and leukocyte mobilization by hyperglycemia Because of vascular compromise and other factors, infections may be more difficult to control and may be more severe.

For Example :

- **Renal papillary necrosis** may be a devastating complication of bladder infection.
- **Mucormycosis**: A dangerous infectious complication of poorly controlled diabetes is often fatal fungal infection tends to originate in the nasopharynx or paranasal sinuses and spreads rapidly to the orbit and brain.

Epidemiology

- infections cause about 5% of diabetes-related deaths.
- In a person with diabetic neuropathy, a trivial infection in a toe may be the first event in a long succession of complications (gangrene, bacteremia, pneumonia) that may ultimately lead to death.
- patient present with a lot of fungal infection → do blood sugar test may be the first sign of diabetes.

Other complications.

- **Sudden myocardial infarction** due to a sudden rupture of accelerated atheroma plaques in coronary arteries (most common cause of death in diabetic patients).
- **Ischemia in toes or legs** → causes dry gangrene → amputation. (caused by neuropathy and atheroma[6])
- **Cellulitis**: inflammation of subcutaneous tissue → may develop septicemia as complication
- **Intermittent claudication**[7] in patient with severe atheroma

e.g.: the patient will walk normally until he has to go upstairs → his muscles need more blood supply so he feels pain "crampy leg pain" and disappears when at rest, this is history of (major vascular occlusion in the leg) due to diabetes or atheroma.

[6] degeneration of the walls of the arteries caused by accumulated fatty deposits and scar tissue, and leading to restriction of the circulation and a risk of thrombosis.

[7] " العرج المتقطع

Summary.

COMPARISON BETWEEN T1DM AND T2DM		
Characteristic	**TYPE 1**	**TYPE 2**
Prevalence	5%–10%	90%–95%
Age at onset	<30 years	>40 years
Speed of onset	Rapid	Insidious
Body habitus	Usually thin	Usually obese (80% of cases)
Genetics	Family history uncommon Environmental factors required for expression HLA-DR3 and HLA-DR4	Family history common No HLA association Increased in Native Americans and in blacks.
Associations	Other autoimmune diseases: Graves disease, Hashimoto thyroiditis, pernicious anemia, Addison disease.	No autoimmune associations
Pathogenesis	Lack of insulin Pancreas devoid of β-islet cells Insulitis: T-cell cytokine destruction (type IV HSR) and autoantibodies against β-islet cells (>80%) and insulin (>50%) (type II HSR) Triggers for destruction and atrophy—e.g., viruses	Relative deficiency of insulin. Early stages have hyperinsulinemia Insulin resistance related to receptor and postreceptor problems Decreased insulin receptors: downregulation by increased adipose. Postreceptor defects: most important factor; examples—tyrosine kinase defects (normally is activated when insulin attaches to its receptor [muscle, adipose]), GLUT-4 abnormalities (normally moves from the cytosol to the cell membrane to attach to glucose and bring glucose into the cytosol) Fibrotic β-islet cells contain amyloid (No autoantibodies).
Clinical findings	Polyuria (osmotic diuresis from glucosuria), polydipsia, polyphagia, weight loss	Insidious onset of symptoms Recurrent blurry vision: alteration in lens refraction from sorbitol Recurrent infections: bacterial, Candida Target organ disease: nephropathy, retinopathy, neuropathy, coronary artery disease Reactive hypoglycemia: too much insulin is released for a glucose load (early finding) Increased risk for Alzheimer disease
	Ketoacidosis (hyperglycemia, coma; production of ketone bodies); lactic acidosis from shock (losing sodium by osmotic diuresis from glucosuria)	HNKC: enough insulin to prevent ketoacidosis but not enough to prevent hyperglycemia Lactic acidosis may occur from shock (losing sodium by osmotic diuresis from glucosuria)
Treatment	Insulin	Weight loss: upregulates insulin receptor synthesis Oral hypoglycemic agents; may require insulin

Secondary and other causes of DM

Cause	Example
1) Pancreatic disease	cystic fibrosis (CF), chronic pancreatitis
2) Drugs	glucocorticoids, pentamidine, thiazides, α-interferon
3) Endocrine disease	pheochromocytoma, glucagonoma, Cushing syndrome
4) Genetic disease	hemochromatosis, metabolic syndrome, maturity onset diabetes of the young (MODY)
5) Insulin-receptor deficiency	Acanthosis nigricans is a phenotypic marker
6) Infections	mumps, cytomegalovirus (AIDS patients)
7) Impaired glucose tolerance (IGT)	
8) Gestational diabetes mellitus (GDM)	
9) Maturity onset diabetes of the young (MODY) * it has very strong family history & very common here. **a.Autosomal dominant (AD) inheritance** (1) Various subtypes (2) Mutations of transcription factor genes (e.g., glucokinase gene) **b.Patients <25 years old and are not obese.** **c.Mild to severe hyperglycemia** **•Impaired glucose-induced secretion of insulin release** **d.Resistance to ketosis** **e.May progress into type 2 DM** **f.Treatment varies with regard to oral hypoglycemic agents or insulin.**	
10) Insulin resistance syndrome **a.Genetic defect causes insulin resistance that is exacerbated by obesity.** **b.Commonly associated with polycystic ovary syndrome in women** **c.May be associated with acanthosis nigricans** **d. Increased risk for developing Alzheimer disease**	

Diagnosis of DM

(blood glucose levels normally 70 to 120 mg/dL)

1	A random glucose concentration greater than 200 mg/dL, with classical signs and symptoms .
2	A fasting glucose concentration greater than 126 mg/dL on more than one occasion.
3	An abnormal oral glucose tolerance test (OGTT), in which the glucose concentration is greater than 200 mg/dL 2 hours after a standard carbohydrate load.
4	HbA1c : Glycated hemoglobin is a form of hemoglobin that is measured primarily to identify the three month average plasma glucose concentration , 5.6% or less is normal .

Complications of Diabetes Mellitus	
Atherosclerotic disease	● Increased incidence of strokes, CAD, and peripheral vascular disease ● Acute MI is the most common cause of death ● Gangrene of the lower extremities; diabetes is the most common cause of nontraumatic amputation of the lower extremity
Renal disorders	● Renal failure due to nodular glomerulosclerosis ● Renal papillary necrosis
Ocular disorders	● Increased risk for cataracts and glaucoma. ● Retinopathy (15%): ● Nonproliferative: microaneurysm formation, flame hemorrhages, exudates ● Proliferative: formation of new vessels (neovascularization), increased risk for retinal detachment and blindness, annual ophthalmologic examination is mandatory (photocoagulate microaneurysms).
Peripheral nerve disorders	● Diabetes mellitus is the most common cause of peripheral neuropathy in the United States; occurs in 70%–80% of cases ● Sensory: paresthesias, patients complain of burning feet, ↓pinprick sensation, ↓proprioception (ataxia) ● Motor dysfunction: muscle weakness, ↓deep tendon reflexes ● Neuropathy is the most important risk factor for neuropathic ulcers on the bottom of the feet (patient cannot feel pain) ● Treatment for neuropathy: duloxetine (selective serotonin and norepinephrine reuptake inhibitor), topical capsaicin, amitriptyline
Autonomic nervous system disorders	● Autonomic neuropathy: gastroparesis (delayed emptying of stomach), impotence, neurogenic bladder, orthostatic hypotension ● Treatment for gastroparesis: prokinetic agents (e.g., metoclopramide)
Cranial nerve (CN) disorders	● Diabetes is the most common cause of multiple cranial nerve palsies ● Cranial nerves most often involved: CN III, IV, and VI
Infectious disorders	● Urinary tract infections ● Candida infections: e.g., vulvovaginitis ● Malignant external otitis due to Pseudomonas aeruginosa ● Rhinocerebral mucormycosis: Mucor extends from the frontal sinuses to the frontal lobes, producing infarction (vessel invader) and abscesses ● Cutaneous infections: usually Staphylococcus aureus abscesses
Skin disorders	● Necrobiosis lipoidica diabeticorum: well-demarcated yellow plaques over the anterior surface of the legs/dorsum of ankles ● Lipoatrophy: atrophy at insulin injection sites due to impure insulin ● Lipohypertrophy: increased fat synthesis at insulin injection sites.
Joint disorders	● Neuropathic joint: related to lack of sensation; bone or joint deformity from repeated trauma

MCQ's.

1- The risk factors for type 2 diabetes mellitus include:

a- All of the options listed are correct.
b- family history
c- inactivity
d- being overweight

2- Type 1 diabetes mellitus was previously called:

a- diabetes insipidus
b- non-insulin-dependent diabetes
c- adult-onset diabetes
d- juvenile-onset diabetes

3- Type 2 diabetes mellitus was previously called:

a- diabetes insipidus
b- non-insulin-dependent diabetes
c- insulin-dependent diabetes
d- juvenile-onset diabetes

4- When a pregnant woman develops diabetes mellitus it is called:

a- gestational diabetes
b- diabetes insipidus
c- juvenile-onset diabetes
d- prediabetes

5- During Mid Morning, blood glucose levels fall and stimulate the secretion of which hormone?

a- PTH
b- Insulin
c- Glycogen
d- Glucagon

6- The risk factors for type 1 diabetes mellitus include:

a- All of the options listed are correct.
b- family history
c- viral infection
d- being overweight

7- Which is the most common form of diabetes?

a- type 2 diabetes mellitus
b- diabetes insipidus
c- They are all about the same frequency.
d- type 1 diabetes mellitus

8- Ahmad is a 45 year old academic professor who came to your clinic for routine checkup. He said that 4 of his siblings have developed DM over the last 6 years so he is afraid to be diabetic he also added that he tried to educate himself about the disease and lost some weight. The results of the test was as shown :

- fast blood glucose was 85 mg/dl 70-100 mg/dl
- Hb A1c
- K+ 4.7 mEq/L 3.7 to 5.2 mEq/L
- postprandial sample. 160 mg/dl <140 mg\dl

from your knowledge what do you expect to see at the level of the endocrine cells of the pancreas:

a- β Necrosis and atrophy
b- diffuse Fibrosis and amyloidosis
c- Lymphocytes infiltration and macrophages with active inflammation
d- β cells hyperplasia

9- What is primary cause of diabetic complication including : retinopathy , nephropathy and peripheral neuropathy:

a- Low immunity
b- Microangiopathy
c- Atheroma
d- Systemic disturbances

10- A 35 year old patient comes to your clinic with newly diagnosed diabetes. Lab tests reveal no C-peptide in her blood. She has lost a lot of weight recently, despite the fact that she has been eating a lot. This patient has:

a- adult-onset diabetes
b- type 2 diabetes
c- type 1 diabetes
d- need more info

Explination: c peptide is a biomarker for the presence of active endogenous secretion of insulin . thus lack of c peptide indicates lack of insulin secretion

Answers: 1-A 2-D 3-B 4-A 5-D 6-C 7-A 8-D 9-B 10-C

For any suggestions or questions please don't hesitate to contact us on: Pathology434@gmail.com
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