



Round : Internal medicine

Group no : A4

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DIABETES MELLITUS

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- B. TYPE II DIABETES MELLITUS

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- C. POLYDIPSIA

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I. PATHOPHYSIOLOGY

A. Type I Diabetes Mellitus

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1. Pathophysiology:

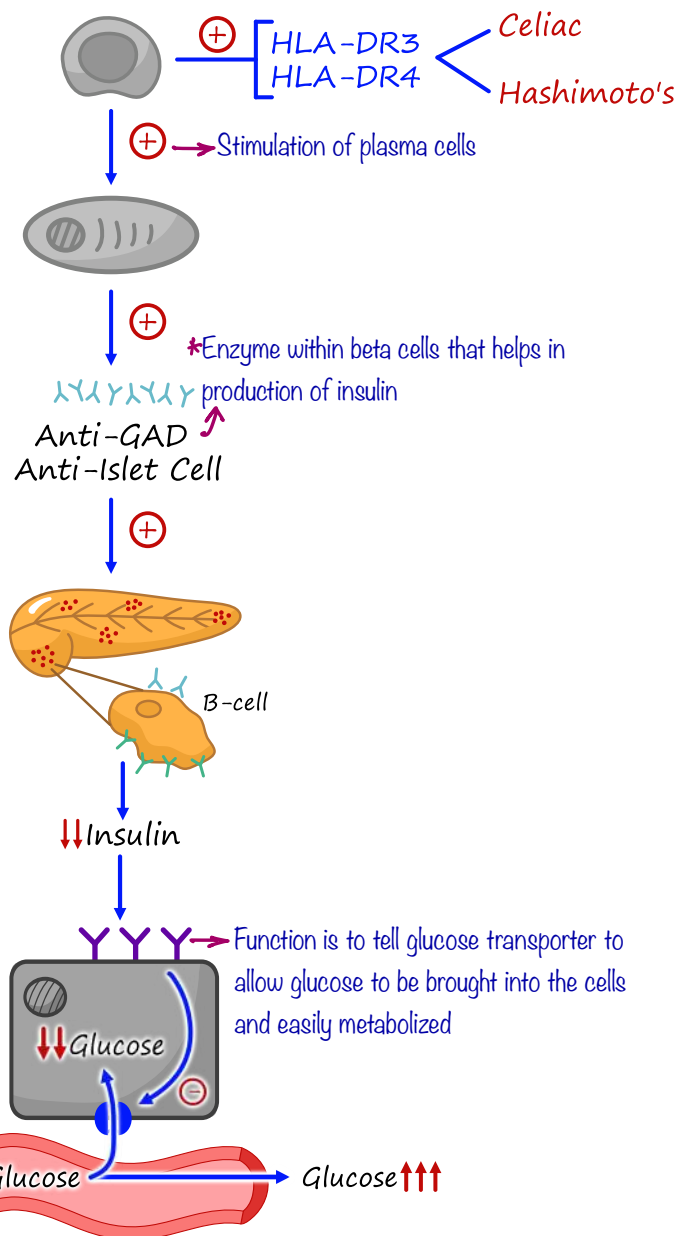
- Autoimmune diseases $\uparrow$ Risk of autoantibody formation $\rightarrow$ Plasma cells produce $\uparrow$ **Anti-GAD and Anti-islet antibodies** $\rightarrow$ $\uparrow$ B-cell destruction $\rightarrow$ $\downarrow$ Insulin production $\rightarrow$ $\downarrow$ **Insulin levels** $\rightarrow$ Inability to uptake glucose $\rightarrow$ **Hyperglycemia**

2. Causes of Type I Diabetes Mellitus:

Autoimmune Disease

- HLA-DR3 and HLA-DR4 mutations $\rightarrow$ Abnormal production of autoantibodies by plasma cells
 - Assess for patients $< 30y$ with a family history of type I DM, or a personal history of celiac disease and Hashimoto's as they all share the above HLA mutations

*Reduction of insulin $\gg$ it won't be able to bond to its appropriate receptors $\gg$ insulin receptors won't be activated as much $\gg$ glucose won't be able to enter cells and will remain in the bloodstream causing hyperglycemia



GLUT Type	Tissues	Insulin Dependent?	Key Features
GLUT-1	Brain (BBB), RBCs, cornea, placenta	No	High-affinity, ensures basal glucose uptake
GLUT-2	Liver, pancreas (β -cells), kidney, small intestine	No	Low-affinity, bidirectional (glucose sensor in pancreas)
GLUT-3	Neurons, placenta	No	High-affinity, steady glucose supply to neurons
GLUT-4	Skeletal muscle, cardiac muscle, adipose tissue	Yes	Insulin-dependent, stored in vesicles, translocates to membrane with insulin
GLUT-5	Small intestine, sperm	No	Fructose transporter



B. Type II Diabetes Mellitus

F: >50 / M: >40 >150 >130/85 >100

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1. Pathophysiology:

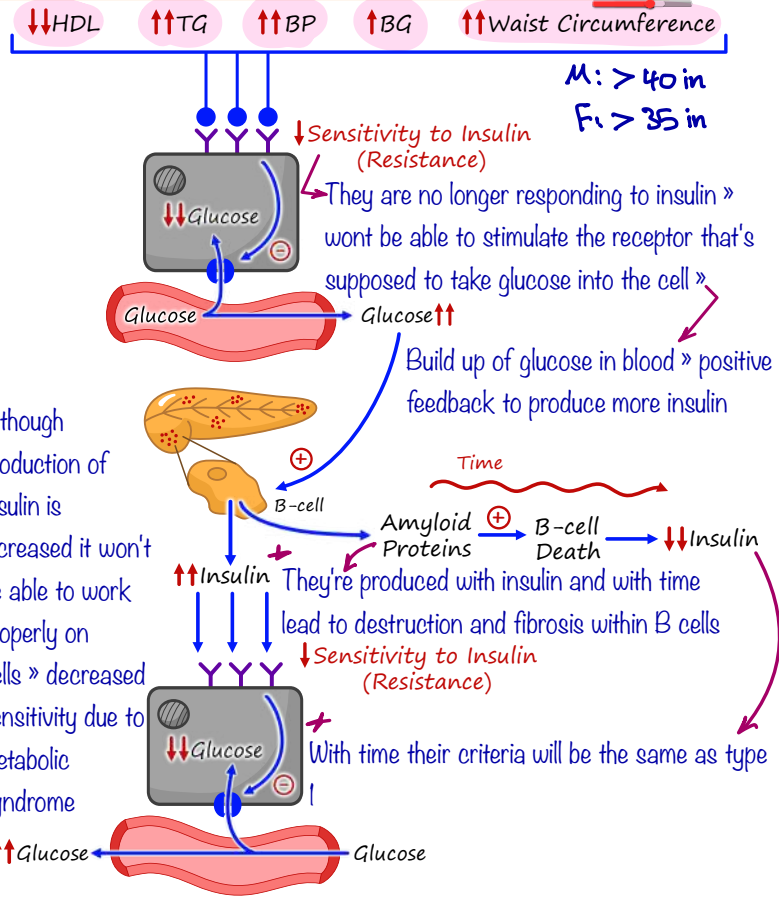
- Obesity/metabolic syndrome → ↑Adipose tissue formation → ↑Release of adipokines → ↑Adipokines → ↑Insulin resistance → ↓Glucose uptake → **Hyperglycemia** → ↑B-cell production of insulin to compensate for hyperglycemia → ↑**Insulin levels** → Overtime from excessive Beta cell activity leads to amyloid protein production → ↑**Beta cell death occurs over time** → This leads to an eventual **reduction of Insulin levels**

2. Causes of Type II Diabetes Mellitus:

Obesity/Metabolic Syndrome

- Assess for patients **> 40** with features of metabolic syndrome or a family history of Type II DM:
 - Obesity → ↑ Adipokines →
↑ RAAS and SNS activity → **↑ BP (hypertension)**
 - Obesity → ↑ Adipokines →
↑ VLDL production by the liver → **↑ Triglycerides**
 - Obesity → ↑ Adipokines →
↓ HDL levels in the bloodstream
 - Obesity → ↑ Adipokines →
Favors fat deposition in the central portion of the body →
↑ Waist circumference

*Although production of insulin is increased it won't be able to work properly on cells » decreased sensitivity due to metabolic syndrome



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II. CLASSIC FINDINGS OF DIABETES MELLITUS

A. POLYURIA

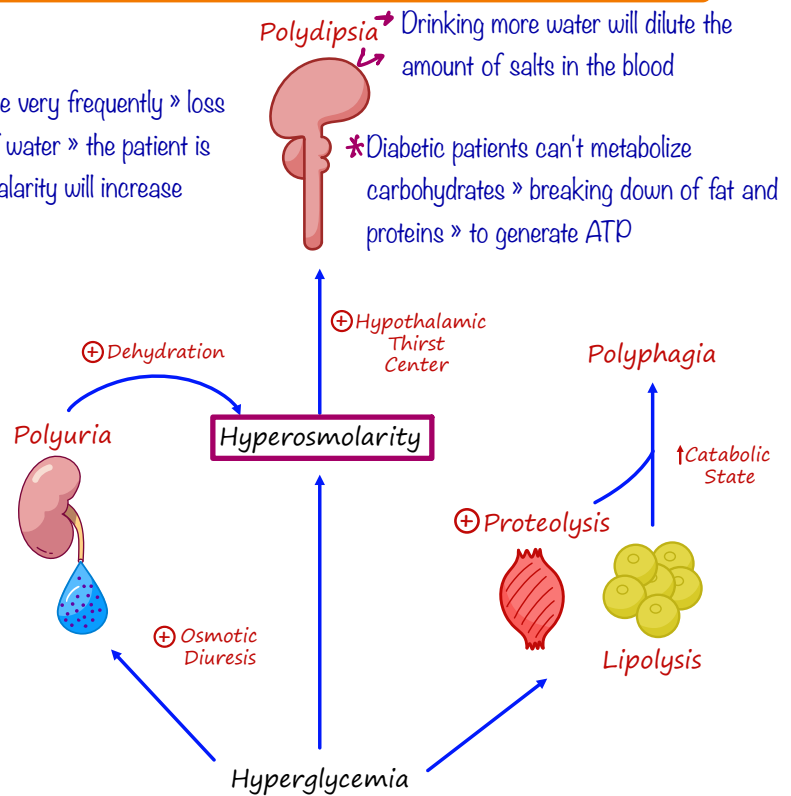
- Hyperglycemia → ↑Glycosuria → Pulls water into the urine via osmotic diuresis → Large volume of water in urine
- They will have to pee very frequently » loss of large amounts of water » the patient is more thirsty & osmolarity will increase

B. POLYPHAGIA

- Hyperglycemia → Triggers catabolism of fats and proteins for fuel since glucose is unable to be taken up in the cells → ↑Catabolic state → ↑Hunger

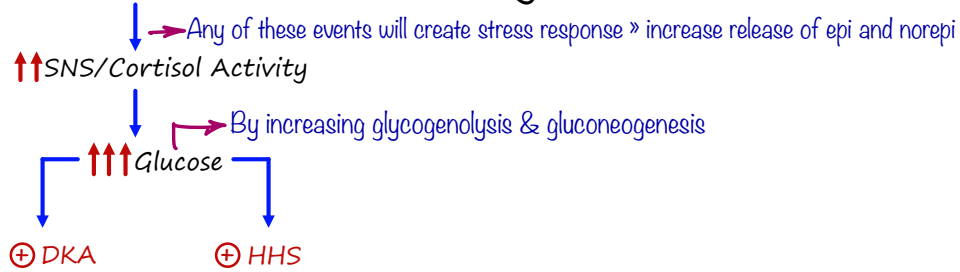
C. POLYDIPSIA

- Hyperglycemia → ↑Glycosuria → Pulls water into the urine via osmotic diuresis → Large volume of water in urine → ↓Water in bloodstream → **Hyperosmolarity of the blood** → **Triggers the hypothalamic thirst center** in attempts to ↑water intake and bring the osmolality down



III. COMPLICATIONS OF DIABETES MELLITUS

Infection, Infarction, Inflammation, Iatrogenic ^{Any surgical procedure}



A. DIABETIC KETOACIDOSIS (DKA)

• Pathophysiology:

- ↑ Stress/Trigger on the body in a patient with DM (usually type I DM) → ↑ Sympathetic nervous system (SNS) activity → ↑ Gluconeogenesis/Glycogenolysis → Severe Hyperglycemia

• Presentation:

1. Kussmaul Breathing and Fruity Breath Odor

- Type I DM → ↓ Insulin production → ↑ Lipolysis by adipose tissue and liver cells → ↑ Ketogenesis → ↑ Ketone bodies → ↓ pH → Triggers activation of peripheral chemoreceptors → ↑ RR and depth → Kussmaul breathing
- ↑ Ketone bodies such as acetone are exhaled and give off a fruity odor

2. Anion Gap Metabolic Acidosis (AGMA)

- Type I DM → ↓ Insulin production → ↑ Lipolysis by adipose tissue and liver cells → ↑ Ketogenesis → ↑ Ketone bodies → ↓ pH and ↑ Anion gap (> 12) ^{Because they release a lot of protons}

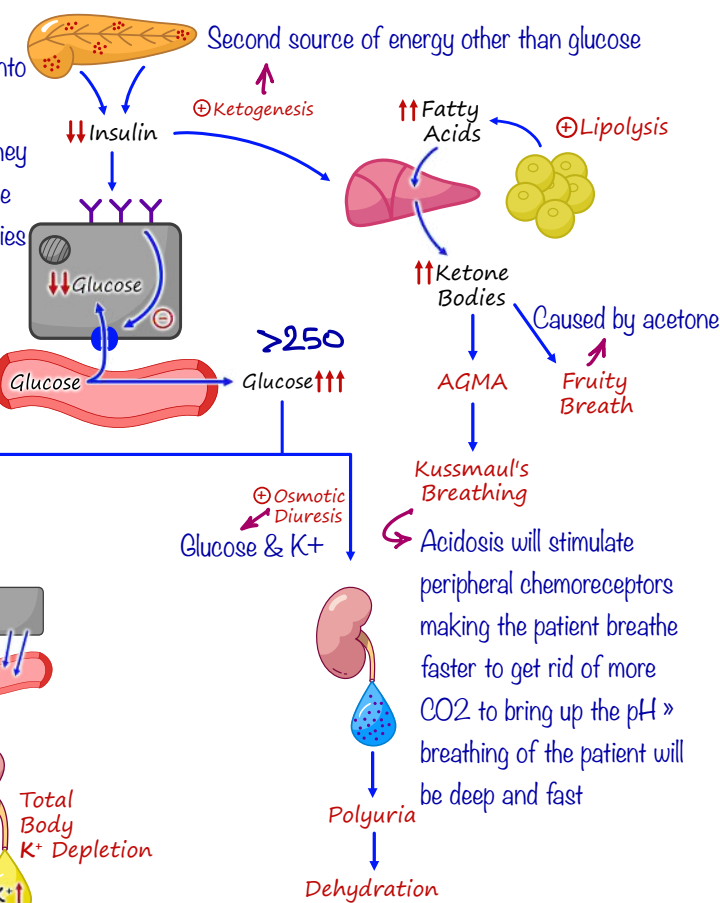
3. Dehydration/Hypovolemia

- Hyperglycemia → ↑ Glycosuria → Pulls water into the urine via osmotic diuresis → Large volume of water in urine → Intravascular volume depletion → Hypovolemia
- ↓ Skin turgor and dry mucous membranes
- Flat Jugular vein
- Hypotension and tachycardia
- Acute kidney injury (↑ Creatinine)

4. Total Body K⁺ Depletion

- Hyperglycemia → ↑ K⁺ drag out of tissue cells → Depletes K⁺ intracellularly
- This needs to be monitored during insulin administration as this will drastically lower K⁺

Pancreatic B-cells



- D: Diabetic – glucose > 250 mg/dL
- K: Ketones – positive serum/urine ketones
- A: Acidosis – pH < 7.3, HCO₃⁻ < 18, AG > 12



C. HYPERGLYCEMIC HYPEROSMOLAR SYNDROME (HHS)

● Pathophysiology:

- ↑Stress/Trigger on the body in a patient with DM
 Early stage (usually type II DM) → ↑Sympathetic nervous system (SNS) activity → ↑Gluconeogenesis/Glycogenolysis → Severe Hyperglycemia
- **No ketogenesis** as insulin is still being produced in type II DM
 → No AGMA or associated Kussmaul breathing

● Presentation:

○ Dehydration/Hypovolemia

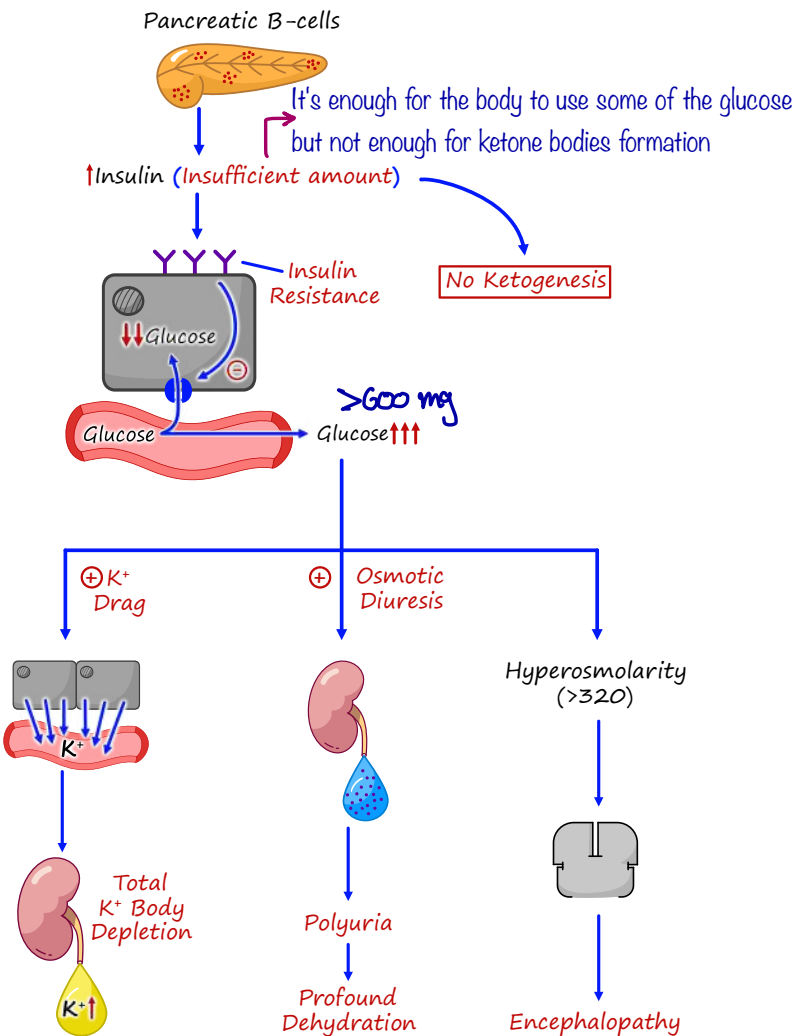
- Hyperglycemia → ↑Glycosuria → Pulls water into the urine via osmotic diuresis → Large volume of water in urine → Intravascular volume depletion → **Profound Hypovolemia**
 - ↓Skin turgor and dry mucus membranes
 - Flat Jugular vein
 - Hypotension and tachycardia
 - Acute kidney injury (↑Creatinine)

○ Total Body K⁺ Depletion

- Hyperglycemia → ↑K⁺ drag out of tissue cells → **Depletes K⁺ intracellularly** → ↑K⁺ loss in the urine
 - This needs to be monitored during insulin administration as this will drastically lower K⁺

○ Encephalopathy

- Hyperglycemia → ↑Glycosuria → Pulls water into the urine via osmotic diuresis → Large volume of water in urine → ↓Water in the bloodstream → **Hyperosmolarity of the blood (> 320)** → Confusion and altered mental status



D. ATHEROSCLEROTIC CARDIOVASCULAR DISEASE (ASCVD)

● Pathophysiology:

- Hyperglycemia → ↑ Nonenzymatic glycation of proteins → Hyaline arteriosclerosis and basement membrane thickening → ↑ Atherosclerosis of larger vessels supplying CNS, Heart, and Extremities

● Presentation:

○ TIA or CVA

■ Neurological deficits

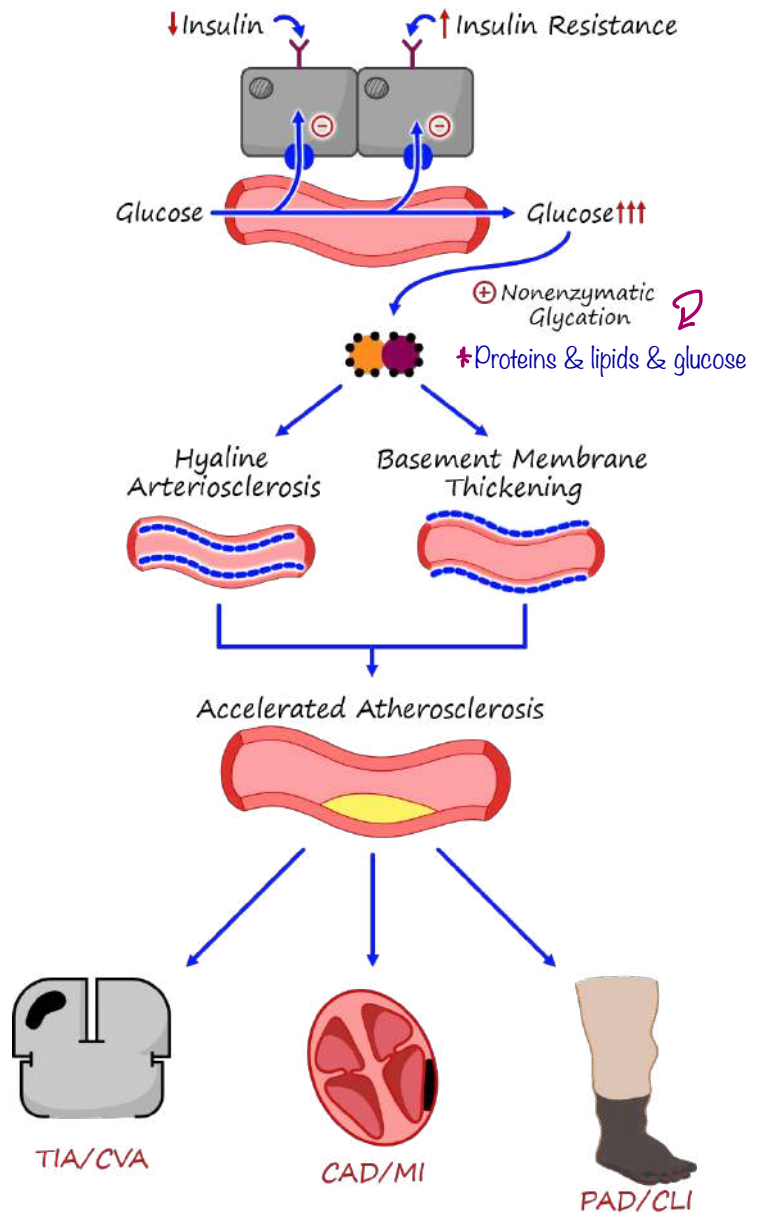
- The type of deficits depends on the vascular territory that is occluded by atherosclerotic plaque

○ CAD or MI

- Angina can be seen in stable CAD or MI
- Troponin and ST changes (e.g. ST elevation or depression) suggest MI

○ PAD or CLI

- Claudication intermittently suggests stable PAD
- Claudication at rest, Ulcers, and Gangrene suggest CLI



Macrovascular

Stage	Description	GFR (mL/min/1.73 m ²)	Clinical Notes
Stage 1	Kidney damage with normal or ↑ GFR	≥ 90	Proteinuria, microalbuminuria, but normal GFR
Stage 2	Mild ↓ in GFR	60–89	Early kidney damage, often asymptomatic
Stage 3a	Mild to moderate ↓ in GFR	45–59	May begin to see complications (e.g., anemia, bone disease)
Stage 3b	Moderate to severe ↓ in GFR	30–44	More evident symptoms, management intensifies
Stage 4	Severe ↓ in GFR	15–29	Preparation for dialysis or transplant begins
Stage 5	Kidney failure (ESRD)	< 15 or on dialysis	End-stage renal disease; requires dialysis/transplant



E. DIABETIC NEPHROPATHY

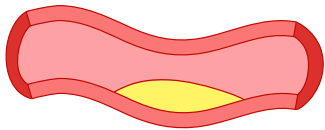
● Pathophysiology:

- Hyperglycemia → ↑Nonenzymatic glycation of proteins → Hyaline atherosclerosis and basement membrane thickening → ↑Atherosclerosis of efferent arterioles → ↑GFR → ↑Glomerulosclerosis from chronic hyperfiltration → Loss of podocyte function → ↑Proteinuria

● Presentation:

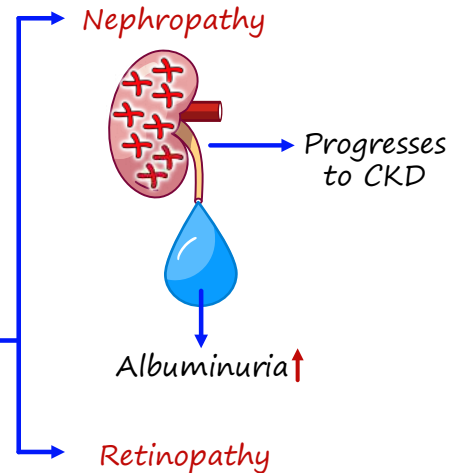
- ↑Risk of CKD
 - ↓GFR (<60) and ↑Creatinine
 - ↑Albuminuria
 - 30-300 mg/day

Accelerated Atherosclerosis



*Damage is done to the glomerulus » glomerular sclerosis » kidney can't excrete waste products & loss of a lot of albumin

Microvascular



F. DIABETIC RETINOPATHY

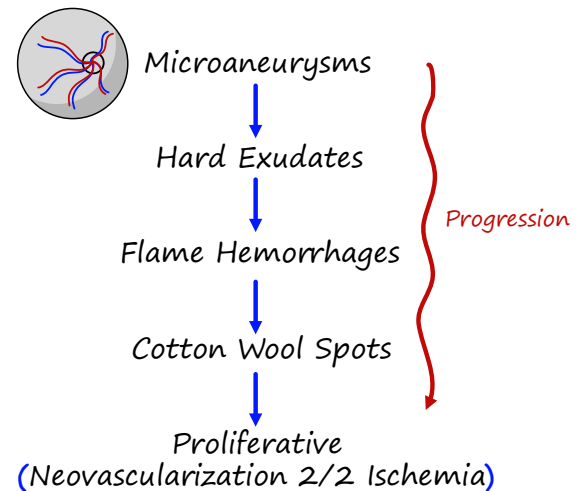
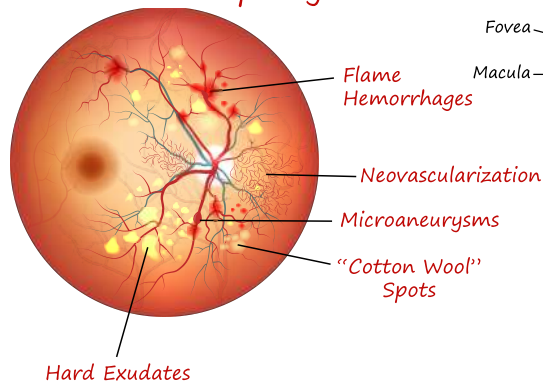
● Pathophysiology:

- Hyperglycemia → ↑Nonenzymatic glycation of proteins → Hyaline atherosclerosis and basement membrane thickening → ↑Atherosclerosis of retinal arteries
- Nonproliferative Retinopathy
 - Microaneurysms → Hard exudates → Flame hemorrhages → Cotton wool spots
- Proliferative Retinopathy
 - Retinal ischemia → Formation of new retinal vessels to supply ischemic retina → New vessels can bleed easily, and vitreous hemorrhage

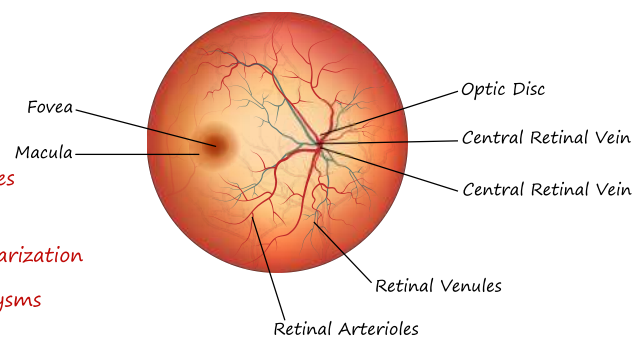
● Presentation:

- Progressive Vision loss
- Abnormal Fundoscopic exam
 - Microaneurysms
 - Tiny red dots on the retina
 - Hard exudates
 - Yellow deposits on the retina
 - Flame hemorrhages
 - Red flame-shaped structures on the retina
 - Cotton wool spots
 - Fluffy white patches on the retina
 - Neovascularization
 - New abnormal vessels in the retina, +/- vitreous hemorrhage

Diabetic Retinopathy



Normal Retina



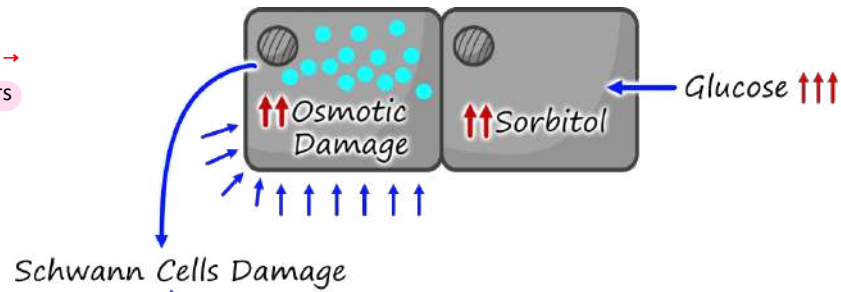
G. CN III Palsy

• Pathophysiology:

- Hyperglycemia → ↑Nonenzymatic glycation of proteins → Hyaline atherosclerosis and basement membrane thickening → ↑Atherosclerosis of vasa nervosum supplying 3rd nerve fibers → **Loss of somatic function of 3rd nerve**
 - Since parasympathetic fibers are intact → There is **pupil-sparing**
 - This means a normal size pupil and reactive to light

• Presentation:

- **Ptosis**
 - Due to the weakness of levator palpebrae superioris
- **Down and out configuration of the eye**
 - Due to the unopposed function of the lateral rectus



H. DIABETIC NEUROPATHY

• Pathophysiology:

- Hyperglycemia → ↑Sorbitol formation in cells → ↑Osmotic shifting into cells → ↑Osmotic damage → ↑Schwann cell damage → ↑Neuron dysfunction

• Presentation:

○ Peripheral Neuropathy

- Symmetric distal sensory dysfunction (**stocking-glove distribution**)
 - Loss of sensation of LEs
 - Paresthesia or burning sensation of LE's

○ Gastroparesis

- Vagus nerve dysfunction → **Delayed gastric emptying** → Gastric distention
 - Nausea and vomiting
 - Early satiety
 - Abdominal pain

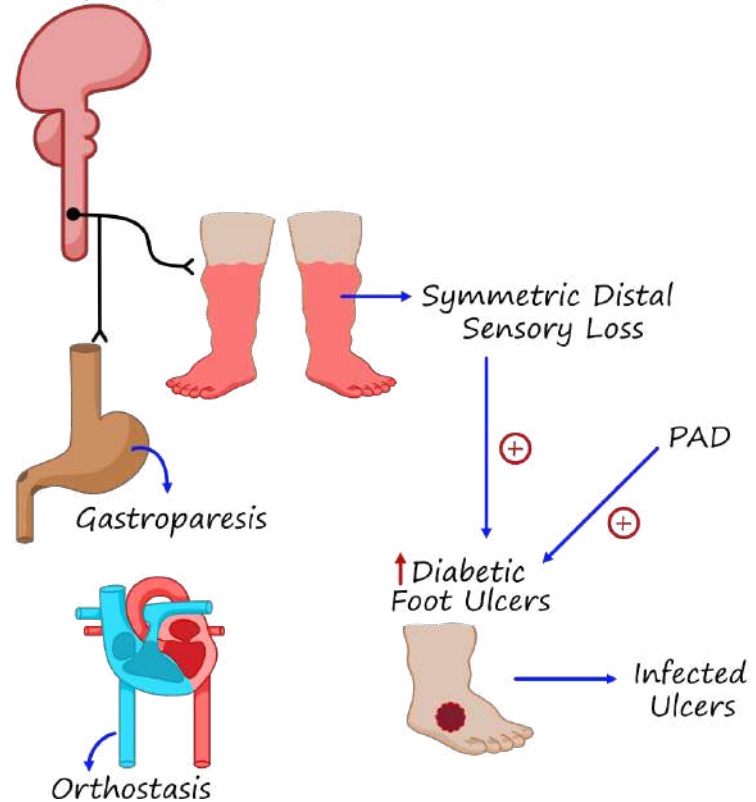
○ Orthostasis

- Autonomic nerve dysfunction → **Loss of vasoconstriction of veins** → ↓Venous return → ↓Preload → ↓SV/CO → ↓BP in changes in position (e.g. sitting → Standing)
 - Pre-syncope or Syncope with postural changes

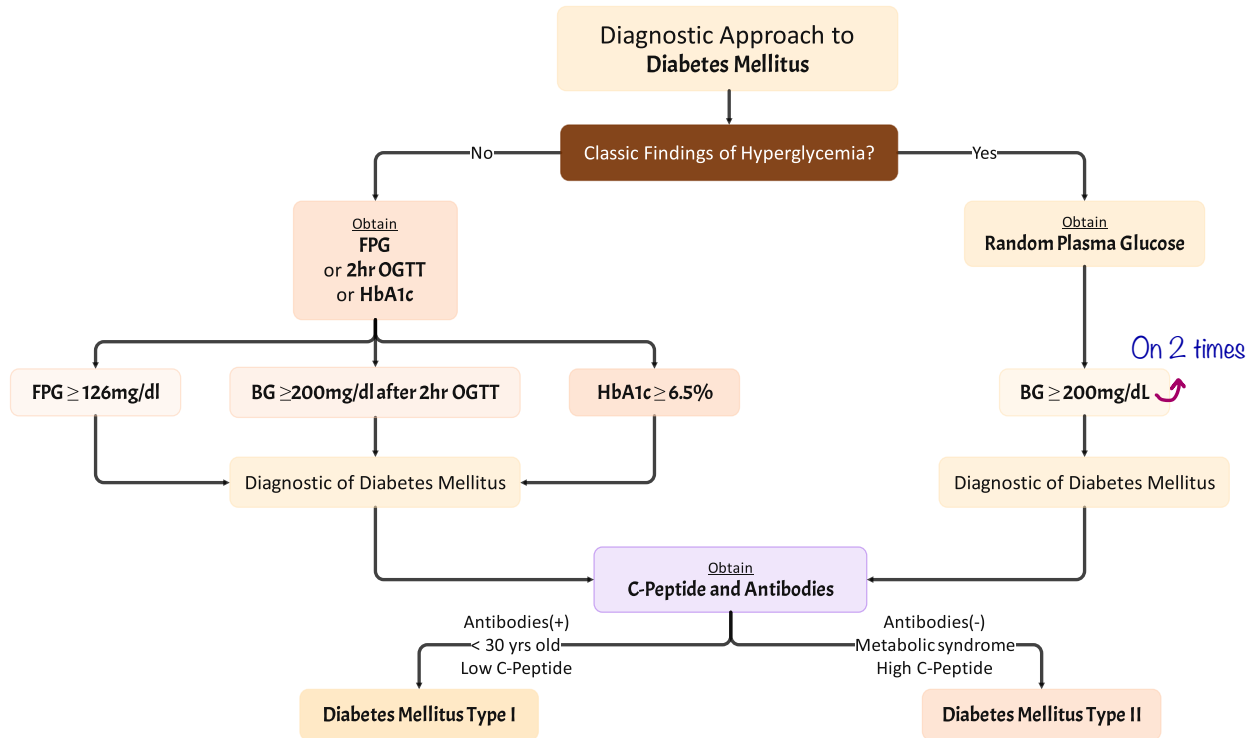
○ Diabetic Foot Ulcers

- Neuropathy → Loss of LE sensation → Unable to feel if LE injury forms and additionally DM → ↑Risk of PAD → **Arterial ulcers** can form which impair wound healing
 - The combination of both mechanisms leads to foot ulcers that can become infected leading to **cellulitis, osteomyelitis, and sepsis** in a worst-case scenario

Neuropathy



IV. DIAGNOSTIC APPROACH TO DIABETES MELLITUS



ASSESS FOR THE PRESENCE OF DIABETES

1. Obtain **Fasting Plasma Glucose (FPG)**

- **Indications:**
 - Assess for the presence of hyperglycemia in a fasted state (at least 8hrs)
- **Abnormal Findings:**
 - FPG ≥ 126mg/dl on two separate occasions → Diagnostic of DM

2. Obtain **2hr OGTT**

- **Indications:**
 - Inconclusive FPG test
 - Suspect gestational diabetes
- **Abnormal Findings:**
 - Glucose ≥ 200mg/dl 2hrs after 75g of glucose → Diagnostic of DM

3. Obtain **HbA1c**

- **Indications:**
 - Assess for the presence of diabetes by checking average glucose level over the past three months
- **Abnormal Findings:**
 - HbA1c ≥ 6.5% → Diagnostic of DM

4. Obtain **Random Blood Glucose**

- **Indications:**
 - Symptoms of hyperglycemia (polyuria, polyphagia, and polydipsia)
 - Suspect DKA or HHS
- **Abnormal Findings:**
 - Random glucose ≥ 200mg/dl + symptoms → Diagnostic of DM

5. Obtain **Antibodies and C-peptide**

- **Indications:**
 - Differentiate type I from type II
- **Abnormal Findings:**
 - **Autoantibody testing**
 - (+) Anti-GAD and Anti-islet antibodies → Suggest type I DM
 - **C-peptide levels (made with insulin by beta cells)**
 - ↑C-peptide → Suggests Type II DM (early/mid stage)
 - ↓C-peptide → Suggests Type I DM



A. INSULIN THERAPY

• Therapies:○ **Rapid Acting Insulin**

- Lispro
- Aspart

○ **Short-acting insulin**

- Regular insulin

○ **Long-acting Insulin**

- Glargine
- Detemir

• Indications:

- Type I DM
- Type II DM refractory to 3 or more antidiabetic medications with HbA1c > 7%
- Type II DM with HbA1c > 9.5%

• Purpose:○ **Rapid Acting Insulin**

- Used in a basal-bolus regimen
 - Given as **prandial insulin** subcutaneously (e.g. prior to meals)

○ **Short-acting insulin**

- Used in **hyperglycemic crisis** (e.g. DKA and HHS)
 - Used as a continuous infusion

○ **Long-acting Insulin**

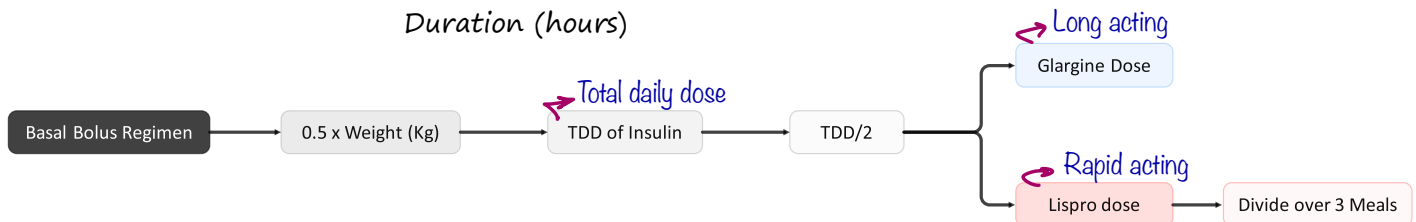
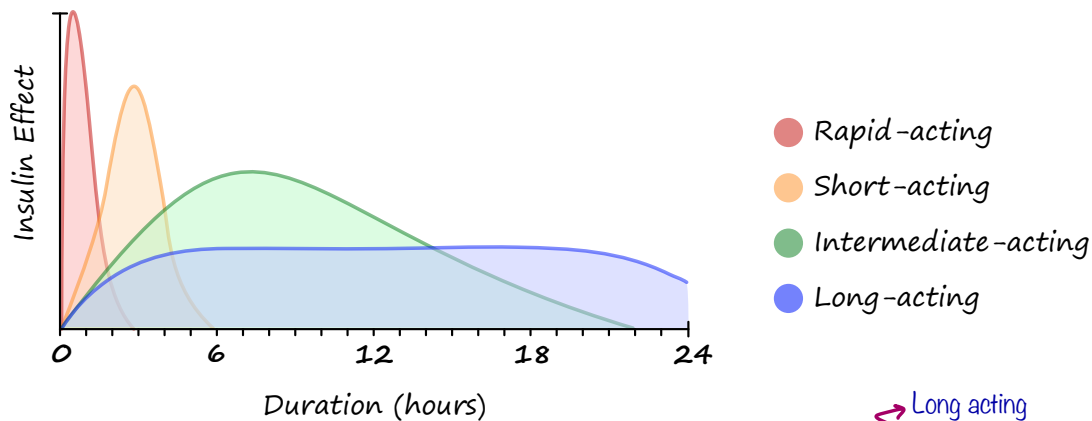
- Used in a basal-bolus regimen
 - Given as **basal insulin** subcutaneously (e.g. given at noon)

• Monitoring:○ **Basal bolus regimen** (Long-acting daily PLUS rapid-acting 3 times daily)

- Monitor blood sugar 3 times daily with meals and at night before bed
- Aim for blood sugar of 90-180mg/dl

○ **Short-acting insulin infusion**

- Monitor blood sugar every hour to ensure proper titration
- Aim for blood sugar of 90-180mg/dl



Type of Insulin	Peak and Duration of Action	Purpose
<u>Rapid Acting</u> - Lispro - Aspart	Peak ~ 1 hour Duration ~ 3 - 4 hours	- Prandial insulin for basal bolus scheme
<u>Short Acting</u> - Regular	Peak ~ 2 - 3 hours Duration ~ 4 - 6 hours	- Insulin infusion for hyperglycemic crisis
<u>Intermediate Acting</u> - NPH	Peak ~ 6 - 10 hours Duration ~ 10 - 16 hours	- Basal insulin for basal bolus scheme
<u>Long Acting</u> - Glargine - Detemir	No peak Duration ~ 24 hours	- Basal insulin for basal bolus scheme



B. ANTIDIABETIC MEDICATIONS

Therapies:

- **Biguanides**
 - Metformin
- **GLP-1 Agonists**
 - Liraglutide
 - Dulaglutide
- **DPP-4 Inhibitors**
 - Sitagliptin
 - Saxagliptin
- **SGLT2-Inhibitors**
 - Empagliflozin
 - Canagliflozin
- **Sulfonylureas**
 - Glyburide
 - Glipizide
- **Thiazolidinediones**
 - Pioglitazone
 - Rosiglitazone

Indications:

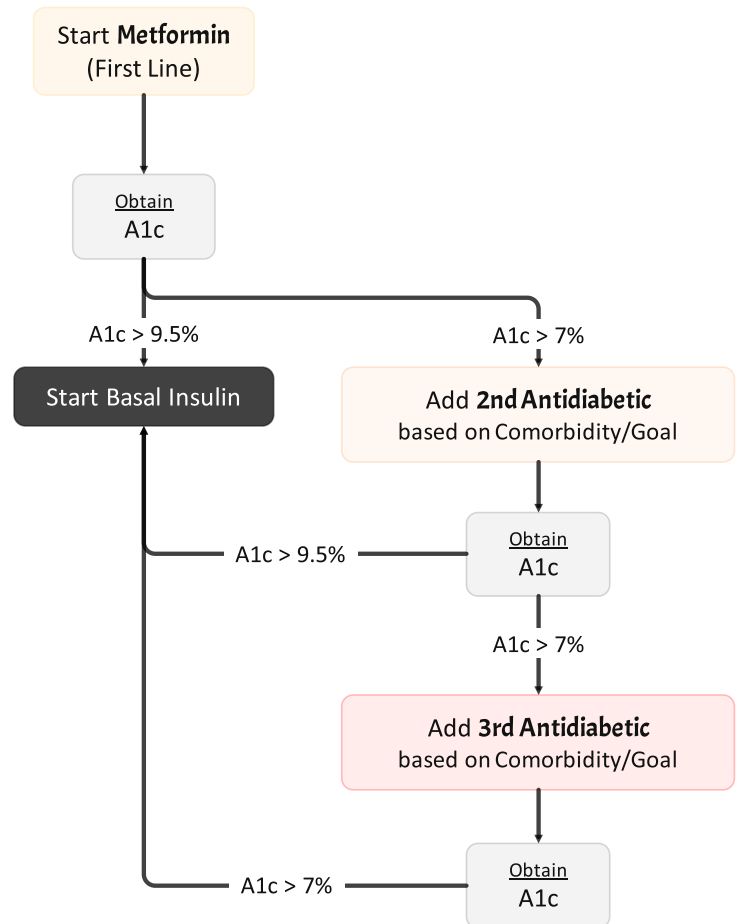
- **Biguanides**
 - First-line medication in DM-II
- **GLP-1 Agonists**
 - **Second-line medication in DM-II** with the following comorbidities:
 - **ASCVD**
 - Reduces ASCVD
 - **CKD**
 - Renal protective
 - **Obesity**
 - Induces weight loss
- **DPP-4 Inhibitors**
 - **Second-line medication in DM-II** with the following comorbidities:
 - **Obesity**
 - Induces weight loss
- **SGLT2-Inhibitors**
 - **Second-line medication in DM-II** with the following comorbidities:
 - **ASCVD**
 - Reduces ASCVD
 - **CKD**
 - Renal protective
 - **CHF**
 - Cardioprotective
 - **Obesity**
 - Induces weight loss
- **Sulfonylureas**
 - Second-line medication in DM-II
- **Thiazolidinediones**
 - Second-line medication in DM-II

Purpose:

- Antidiabetic medications → Reduce the HbA1c to < 7% → Reduce DM complications

Monitoring:

- Monitor blood sugar for hypoglycemia
 - Common with sulfonylureas
- Monitor HbA1c every 3-6 months to assess for improvement in HbA1c
 - If A1c > 7% on metformin → **Initiate 2nd agent** →
 - If A1c remains > 7% despite 2 agents, then **add 3rd agent** →
 - If A1c remains > 7% despite 3 agents → **Add insulin**
- Monitor for worsening CHF with Thiazolidinediones



Comorbidity/Goal	Treatment
ASCVD	- GLP-1 Agonists - SGLT2-Inhibitors
CHF	- SGLT2-Inhibitors - Avoid Thiazolidinediones
CKD	- GLP-1 Agonists - SGLT2-Inhibitors
Weight Loss	- GLP-1 Agonists - DPP-4 Inhibitors - SGLT2-Inhibitors
Less Hypoglycemia	- GLP-1 Agonists - DPP-4 Inhibitors - SGLT2-Inhibitors - Thiazolidinediones - Avoid Sulfonylureas



C. MANAGEMENT OF DKA

Therapies:

- IV fluids (e.g. ½ NS or LR)
- Potassium replacement
- Insulin infusion (e.g. regular insulin)
- Bicarbonate
- Dextrose infusion (e.g. D5 ½ NS or D5 LR)

Indications:

- DKA based on ↑ Blood glucose (> 250), Ketoacidosis, ↑ Anion gap, and history of DM

Purpose:

IV Fluids

- Used to replete intravascular volume in dehydration/hypovolemia, given glycosuria causes massive volume loss via osmotic diuresis

Potassium Replacement

- Used to replete K⁺ levels given hyperglycemia can deplete total K⁺ levels and insulin infusions will further reduce extracellular K⁺ via shifting it into cells

Insulin Infusion (e.g. regular insulin)

- Used to treat ketoacidosis as the decrease in insulin impairs glucose uptake, leading to increased fatty acid metabolism and excessive ketone body production. Administering insulin reduces ketogenesis and improves glucose utilization

Bicarbonate

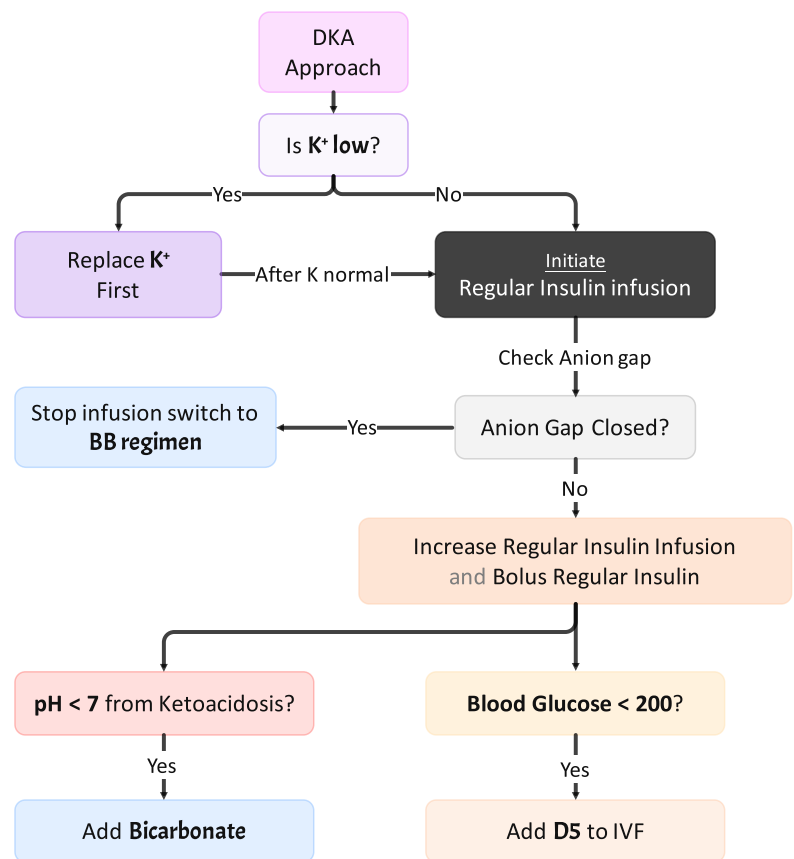
- Used to treat severe ketoacidosis when pH is < 7 which can alter CNS and cardiovascular activity despite the use of insulin infusion designed to reduce ketogenesis

Dextrose Infusion

- Used when blood glucose is < 200 and need to keep using insulin and in fact, need to use higher levels of insulin to normalize the anion gap

Monitoring:

- Monitor BMP every 2 hours to assess if the anion gap is normalizing/closing with insulin infusion
- Monitor BMP every 2 hours to assess if hypokalemia is present to ensure adequate K⁺ repletion
- Monitor pH to assess if pH < 7 to indicate use of bicarbonate
- Monitor blood glucose every hour while on infusion to assess if need for addition of D5 to IVF to allow continued use of insulin if anion gap is still elevated
- Monitor HR, BP, and Urine output while on IVF to ensure adequate hydration



DKA	- IV Fluids for dehydration (LR or 1/2 NS) - Potassium replacement while receiving insulin - Insulin infusion for ketoacidosis until anion gap closes - Bicarbonate for severe acidosis (pH < 7) - Add dextrose if glucose < 200 to allow continued insulin
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D. MANAGEMENT OF HHS

- **Therapies:**
 - IV fluids (e.g. ½ NS or LR)
 - Potassium replacement
 - Insulin infusion (e.g. regular insulin)
- **Indications:**
 - HHS based on **↑↑↑ Blood glucose (> 600)**, **dehydration** and **serum osmolality > 320** and a **history of DM**
- **Purpose:**
 - **IV Fluids**
 - **Used to replete intravascular volume** in dehydration/hypovolemia, given glycosuria causes massive volume loss via osmotic diuresis
 - **Potassium Replacement**
 - **Used to replete K⁺ levels** given hyperglycemia can deplete total K⁺ levels and insulin infusions will further reduce extracellular K⁺ via shifting it into cells
 - **Insulin Infusion (e.g. regular insulin)**
 - Used to treat hyperglycemia and hyperosmolality
- **Monitoring:**
 - Monitor **BMP** every 4-6 hours to assess if hypokalemia is present to ensure adequate K⁺ repletion
 - Monitor **serum osmolality** every 4-6 hours to assess if it's decreasing with insulin infusion and adequate hydration
 - Monitor **blood glucose** every hour while on infusion to assess if glucose is reducing with insulin infusion
 - Monitor **HR, BP, and Urine output** while on IVF to ensure adequate hydration

E. MANAGEMENT OF DIABETIC RETINOPATHY

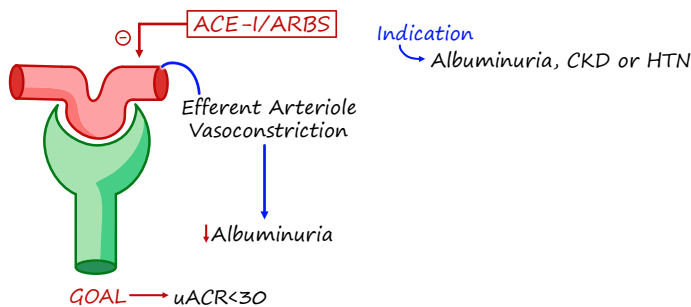
- **Therapies:**
 - Annual eye exam
 - Laser Photocoagulation
 - VEGF-inhibitors
 - Bevacizumab
- **Indications:**
 - In all patients with DM with concern for DM retinopathy
- **Purpose:**
 - **Annual eye exam**
 - Identify the presence of nonproliferative vs proliferative retinopathy and treat it before vision loss develops
 - **Laser Photocoagulation**
 - Used in proliferative retinopathy
 - **Inhibits neovascularization** in proliferative retinopathy preventing progressive vision loss
 - **VEGF-Inhibitors**
 - **Used as first-line therapy** in nonproliferative with macular edema and proliferative retinopathy
 - **Inhibits fluid leakage in macular edema** in nonproliferative retinopathy preventing progressive vision loss
 - **Inhibits neovascularization** in proliferative retinopathy preventing progressive vision loss
- **Monitoring:**
 - Monitor annually under fundoscopy for improvement or worsening of retinopathy

HHS	- IV Fluids for dehydration - Insulin infusion for hyperglycemia / hyperosmolality - Potassium replacement while receiving insulin
Retinopathy	- Annual eye exams for screening - Laser photocoagulation and VEGF-I for proliferative type



F. MANAGEMENT OF DIABETIC NEPHROPATHY

- **Therapies:**
 - Annual UACR and BMP
 - ACE-I or ARBs
- **Indications:**
 - **Annual uACR and BMP**
 - Monitor for development of CKD based on albuminuria and GFR, raising concerns for DM nephropathy
 - **ACE-I or ARBs**
 - Albuminuria (> 30mg/day) in patient with DM
- **Purpose:**
 - **Annual uACR and BMP**
 - **Monitor BMP for worsening renal function (e.g. ↓GFR)** which will allow the identification of DM nephropathy and ensure treatment early
 - **Monitor Urine albumin for worsening albuminuria (30-300mg/day)** which will allow the identification of DM nephropathy and ensure treatment early
 - **ACE-I or ARBs**
 - **Reduce Albuminuria** in patients with DM by inducing efferent arteriole vasodilation which reduces glomerular pressure and pushing of proteins into Bowman's capsule



- **Monitoring:**
 - Monitor BMP for any hyperkalemia or worsening of GFR/creatinine with ACE-I or ARB use

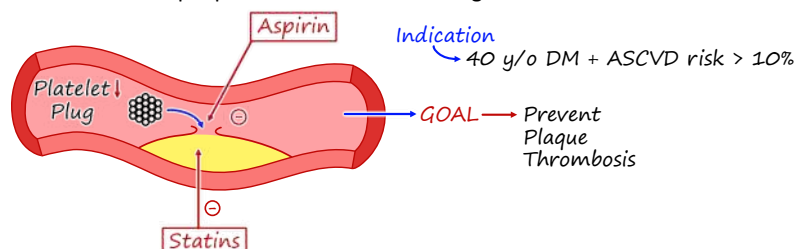
Nephropathy	- Annual UACR and BMP for screening - ACE-I or ARBs for proteinuria and/or hypertension
Neuropathy	- Annual Foot exams for screening - Gabapentin/Pregabalin vs TCAs vs SNRIs
Atherosclerosis	- Annual Lipid panel for screening - Statin therapy and ASA for primary prevention

G. MANAGEMENT OF DIABETIC NEUROPATHY

- **Therapies:**
 - Annual foot exams
 - Neuropathic medications
 - Gabapentin
 - Pregabalin
 - Amitriptyline
 - Duloxetine or Venlafaxine
- **Indications:**
 - **Annual foot exams**
 - Diagnosis of DM and monitoring for foot ulcers
 - **Neuropathic medications**
 - **Pain and Paresthesia** associated with DM neuropathy
- **Purpose:**
 - **Neuropathic medications**
 - Reduce **Pain and Paresthesia** associated with DM neuropathy
- **Monitoring:**
 - Monitor for improvement or worsening of pain/paresthesia and adjust the dose of medications as needed

H. MANAGEMENT OF ATHEROSCLEROTIC CARDIOVASCULAR DISEASE

- **Therapies:**
 - Annual lipid panel screening
 - Statin therapy
 - Aspirin
- **Indications:**
 - **Annual Lipid Panel Screening**
 - Assess for hyperlipidemia which ↑Risk of ASCVD
 - **Statin Therapy**
 - > 40yrs old + DM + ASCVD risk > 10%
 - **Aspirin**
 - > 40yrs old + DM + ASCVD risk > 10%
 - **Clinical ASCVD** (e.g. PAD, CAD, CVA)
- **Purpose:**
 - **Annual Lipid Panel Screening**
 - Assess for hyperlipidemia which increases the risk of ASCVD
 - **Statin Therapy**
 - Prevent plaque formation → Reducing events of ASCVD
 - **Aspirin**
 - Prevent plaque thrombosis → Reducing events of ASCVD



- **Monitoring:**
 - Monitor lipid panel annually to assess if LDL is decreasing

