Diabetic Complications

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Objectives:

- **Not given yet**

References:
Doctor notes - Black
Important - Red
Step up / davidson - Blue
Master of the board- Green
Extra explanation - Grey

Optional:

Chapter 21

Diabetes complications

Diabetes complications	
Acute complication	**Chronic complication**
1-Hypoglycemia	1-Microvascular complications • Neuropathy • Retinopathy • Nephropathy
2-Hyperglycemia: • Diabetic ketoacidosis (DKA) • Hyperosmolar Hyperglycemic nonketotic syndrome (HHNS)	2-Macrovascular complications • Vasculopathy

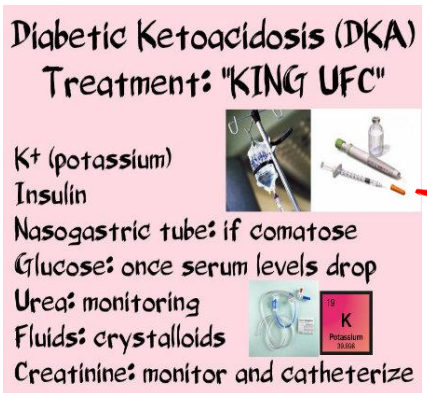
Prevalence increases as we go from down to up , but the opposite with mortality which increases as we go from up to down.

A- Acute complications

1-Hypoglycemia : The most common complication of DM and the most common associated with mortality in DM due consequences of it such as: car accidents.

2-Diabetic ketoacidosis : is an acute, life-threatening medical emergency that could occur in both Type1 DM (usually) and Type 2 DM (rare).	
Pathophysiology	DKA arises secondary to insulinopenia and excess of glucagon . cells are unable to use glucose in order to form ATP ,so they are used fats instead of glucose to form ATP which leads to produce very toxic waste products (especially to brain) is called ketones (B-hydroxybutyrate and acetoacetate). Brain is considered the only tissue that does not produce ketones because brain doesn't need insulin to take the glucose. Brain has its own receptors for glucose transport that is independent of insulin
Predisposing factors of DKA	1-Any type of stress illness : (Infections, Mi, Stroke , Recent surgery , Trauma , GI bleeding and sepsis 2-Inadequate administration of insulin

Consequences of events in DKA	**Severe hyperglycemia → Ketonemia and ketonuria → metabolic acidosis → volume depletion**
Clinical features of DKA : All symptoms develop rapidly less than 24 hours	• Nausea and vomiting • **Kussmaul respiration** • **Abdominal pain** (more common in children) mimics the acute abdomen and usually with guarding and rigidity • Fruity (acetone) breath odor • Marked dehydration → orthostatic hypotension , Tachycardia and volume depletion • Polydipsia , polyuria, polyphagia and weakness • Altered consciousness , drowsiness and frank coma may occur if not treated
Diagnosis of DKA	• **Severe Hyperglycemia** (serum glucose level > 450 mg/dL and < 850 mg/dl • **Metabolic acidosis with hlgh anion gap**: blood PH < 7.3 and serum bicarbonate < 15 mEq/L • **Positive ketonemia and ketonuria** (false negative at circulatory collapse) • Hyperosmlarity • Hyponatremia (Increase serum glucose level leads to fluid shift from intracellular to extracellular space) • Hyperkalemia because of acidosis • Phosphate and magnsium level may be low
Treatment of DKA	1. **Insulin** (immediately) 2. **IV fluid [normal saline]** (immediately) and adds 5% glucose once blood glucose level reaches to 250 mg/dl to prevent hypoglycemia. 3. **Potassium: 1-2 hours after administration of insulin** because insulin causes shift of potassium into cells → hypokalemia 4. Serum bicarbonate : not in all cases
Complications of treatment of DKA	• **Cerebral edema :** if glucose level decrease rapidly • **Hyperchloremic non gap metabolic acidosis** : due rapid infusion of normal saline



Easy mnemonic!

3-Hyperosmolar hyperglycemic nonketotic acidosis (HHNS) : A state of severe hyperglycemia , hyperosmolar and dehydration typically occurs in elderly with type 2 DM (viscosity of blood very high)	
Pathophysiology	• low insulin level leads to severe hyperglycemia → osmotic diuresis → dehydration • **ketogenesis is minimal because small amount of insulin is released from pancreas**
Predisposing factors	1. **Severe stress illness** 2. **Inadequate administration of medications**
Clinical features of HHNS	• Polyuria and thirst • Signs of severe dehydration and volume depletion → Hypotension and tachycardia) • **CNS and focal neurologic manifestations (such as: seizures) due hyperosmolar** • **Lethargy and confusion may develop leads convulsion and coma**
Diagnosis of HHNS	• **Severe hyperglycemia : serum glucose level > 600 mg/dl and frequently > 900 mg/dl** • **Hyperosmolarity** : serum osmolarity > 320 mOsm/L • **Serum PH > 7.3 and Serum bicarbonate > 15 mEq/L** • **Negative for ketonemia nd ketonuria** • BUN is elevated , Pre-renal azotemia is common
Treatment of of HHNS	1. **IV fluid** (normal saline) : the most important therapy. Switch to Half saline if patient stabilize. and adds 5% glucose once blood glucose reaches 250 mg/dl to prevent hypoglycemia 2. **Insulin**

Notes :

1-The most accurate measure of severity of DKA is serum bicarbonate

2-HHNS is associated with mortality more than DKA

B-Chronic complications

In page 2, the first schedule

1-Diabetic neuropathy: the most common chronic complication of DM	
Peripheral neuropathy Most likely affects sensory nerves in stocking or glove pattern and usually starts at the longest nerve (feet)	• Pathophysiology : Myelin sheath is produced by schwann cells ,unfortunately these cells are insulin dependent. in diabetic patients, schwann cells use sorbitol instead of glucose → deposition of sorbitol in schwann cells → swelling of schwann cells → increase pressure to the peripheral nerves • Clinical presentation : tingling, paresthesia and numbness. • Loss of sensation + subsequent ischemia of pressure points (charcot joints) → ulcer formation • Painful diabetic neuropathy : hypersensitivity to touch , with severe burning pain especially at night. treated by gentamicin, pregabalin or tricyclic antidepressant
Cranial nerves complications secondary to nerve infarction	• Most often involves CN 3,4 and 6. • Presentation of Diabetic third nerve palsy : Diplopia ,ptosis, eye pain. Inability to adduct the eyes and spread the pupil.
Mononeuropathies secondary to nerve infarction	• Diabetic lumbosacral plexopathy : Severe deep pain in the thigh with atrophy and weakness of thigh and hip muscles. recovery takes weeks to months • Diabetic truncal neuropathy : pain in the distribution of one of intercostal nerves • Median nerve neuropathy, ulnar nerve neuropathy and common peroneal neuropathy
Multineurrits multiplex	Patient presents with either drop hand or foot
Autonomic neuropathy occurs in severe diabetes	• Impotence (most common presentation in men) • Neurogenic bladder → retention and incontinence • Gastroparesis • Constipations and diarrhea (alternating) • Postural hypotension • Gastroparesis : DM leads to reduces the ability of gut to sense stretch of the wall of the bowel. the stretch is the main stimulant to gastric motility. Gastroparesis is an immobility of bowels that leads to bloating, constipation and early satiety , vomiting and abdominal discomfort. Treated by metoclopramide and erythromycin, which increases gastric motility.

Notes :
1-Blood supply of nerves is called vaso-nervosa
2-Blood supply of vessels is called vaso-vasorum
3-The most active cells in the body are rods and cons (insulin dependent)

2-Diabetic retinopathy:	
Proliferative disorders	• Retina responds to low level insulin → proliferation of new vessels (neovascularization) which these vessels are fragile. with minimal trauma → retinal hemorrhage and vitreous hemorrhage → clots → fibrous bands → retinal detachment → blindness. • Treatment by laser photocoagulation (that is prevent bleeding)
Non-proliferative disorders	• Funduscopic examination shows hemorrhages, exudates, microaneurysms and venous dilatation • Patients are asymptomatic until retinal edema or ischemia that involves central macula → blindness • Hypertension leads to exacerbate this condition.
Others	Diabetes leads to increased risk of glaucoma and cataracts

3-Diabetic nephropathy: 1-The most common cause of ESRD 2- leads to glomerulosclerosis	
Pathophysiology	Increase Pressure state by dilatation of afferent constriction of efferent → -Increased GFR → glomerulosclerosis (kimmelstiel wilson disease) → Microalbuminuria → Proteinuria + nephron ischemia → CKD → ESRD
Microalbuminuria and proteinuria	Microalbuminuria : Albumin > 30 mg/dl a ACR : 3-30 mg/ mmol Proteinuria: Albumin > 200 mg/dl ACR : > 30 mg/dl
Treatment	• HTN usually develops between Microalbuminuria and proteinuria ,so control HTN helps to slow progress to ESRD • ACEI → slow progress of microalbuminuria to proteinuria and slow decline of GFR • Protein restriction diets are recommended

4-Vasculopathy: The most common chronic complication associated with mortality	
Coronary artery disease	• The most common form vasculopathy and the most killer • 80% will be 3 vessels disease because of autonomic neuropathy that causes silent MI
Cerebrovascular disease	Stroke or TIA
Peripheral artery disease	The most common presentation of PAD is intermittent claudication and critical limb ischemia (diabetic foot)

Others chronic complications of DM

- **Deafness and inner ear disease**
- **Infections due impaired WBC function , reduced blood supply and neuropathy**
- **Periodontal disease → also increases insulin resistance**
- **Fatty liver because of hypoinsulinemia**
- **Diabetic dermopathy : tinea pedis and tinea cruris**
- **Medications complications : Metformin → gastritis and photosensitivity**

Common infections in patient with DM:

- Foot ulcers and osteomyelitis
- Pneumonia
- Staphylococcus aureus infections
- Group B streptococcal infections
- Mucocutaneous candidiasis
- Urinary tract infections

Rare (life-threatening) infections in patient with DM:

- Rhinocerebral mucormycosis
- Malignant otitis externa
- Emphysematous pyelonephritis

MCQ's

Q1: A 55-year-old man comes to the physician after being told at a health feir that he has type 2 diabetes mellitus. He is asymptomatic and, besides being a "big eater" , he lias no other bad habits. He does not smoke cigarettes, and he drinks only two or three glasses of wine week. In 5 Years he is most likely to exhibit which of the following?
(A) Fatty liver
(B) Intermittent claudication
(C) Diabetic foot
(D) Retinopathy
(E) 3+ proteinuria

Q2: A 49-year-old woman presents to her physician's office with a long-standing history of polydipsia, polyuria, central obesity, and hyperlipidemia. She is currently taking metformin, a sulfonylurea, and an angiotensin-converting enzyme (ACE) inhibitor. ACE inhibitors are most beneficial in preventing or slowing the progression of which of the following diabetic complications?
(A) Diabetic ketoacidosis
(B) Diabetic nephropathy
(C) Diabetic neuropathy
(D) Diabetic retinopathy
(E) Peripheral vascular disease

Q3: A 75-year-old woman is brought to the emergency department after being found unconscious by a neighbor. The woman has a history of type 2 diabetes. A stat blood draw demonstrates a plasma glucose of 975 mg/dL. which of the following additional findings would be most consistent with the patient's probable diagnosis?
(A) Blood urea nitrogen 5 mg/dL
(B) Plasma strongly positive for ketones
(C) Serum creatinine 0.3 mg/dL
(D) Serum osmolality 380 mOsmol/kg
(E) Serum sodium 132 mEq/L

Answers: 1.D, 2.B, 3.D

Thank you

If you have any question please contact with us at:
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