Diabetes Mellitus

Part I (Introduction)

تحذير هام

هذا الفيديو و جميع الرسوم التوضيحية المتاحة فى هذا الموقع هم تحت حماية قانون الملكية الفكرية

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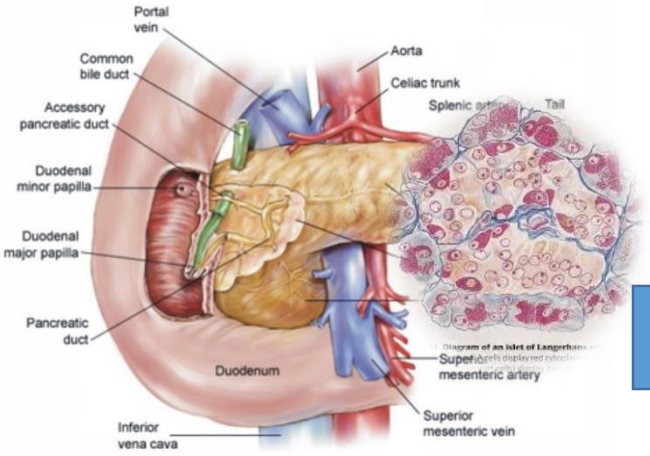
Diabetes Mellitus

Definition

Diabetes Mellitus is a chronic metabolic syndrome characterized by hyperglycemia and other metabolic derangements. The problem is caused by deficiency or diminished action of Insulin Hormone.

❶ Failure of β cells of the pancreas to produce Insulin (Type 1 DM) ❷ Insulin receptor defect (Type 2 DM) ❸ Increase in Anti-Insulin hormones ❹ Damage of the pancreas such as in Hemochromatosis, pancreatic cancer, and pancreatic removal.

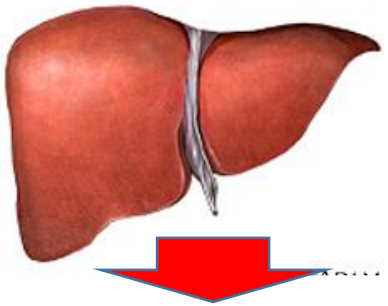


Pancreatic damage

Insulin

Type I Diabetes

1- Acromegaly
2- Pheochromocytoma
3-Cushing Syndrome



Glucose

Type 2 Diabetes

Type I

1. This type of DM typically occurs in young age
2. It is due to absolute insulin deficiency due to destruction of β cells of the pancreas
3. Because Insulin levels are very low in this condition, the patient is more liable to the development of DKA
4. The condition is most likely caused by an environmental factor (e.g. virus) that causes injury to β cells. The damage of β cells causes release of its antigens which stimulate the production of autoantibodies that ultimately destroys all β cells.
5. Anti-Insulin Antibodies are usually present

Chromosome 11

HLA

Environmental Factor

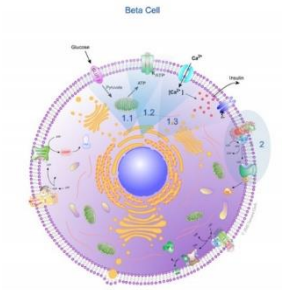
1- Viruses (e.g. Mumps- Coxsackie Virus (24 amino acids mimic GAD of pancreatic beta cells)
2- Vacor
3-Hydrogen Peroxide

Ir

DQ w7 (3.1): PROTECTS
DQw8 (3.2): MAXIMUM RISK

Humeral Immunity

Cell mediated immunity



1- Leak of β cell proteins leads to antibody formation (most against GAD)
2- Molecular mimicry with the virus
3-Expression of class II HLA (considered Non-Self)

Islet cell antibodies

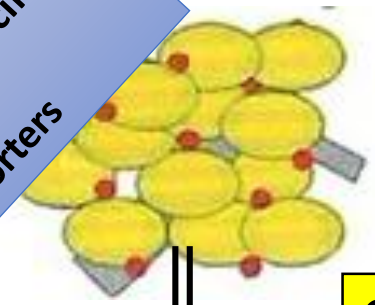
Type 2

1. This type of DM typically occurs in old age
2. The primary defect in this problem is at the receptor level (Post-Receptor Defect)
3. The defect impairs insulin action and results in Hyperglycemia.
   The pancreas reacts to this by increasing its insulin production as an attempt to lower the blood glucose levels (for this reason Insulin levels are usually high –or normal in type 2 DM)
4. Because insulin levels (unlike type 1 DM) are NOT very low (the patient usually does not develop DKA).
5. Obesity increases the Post-Receptor defect and thus aggravates the condition (Most patients are not obese)

Genetic?!

Impair insulin response to Glucose

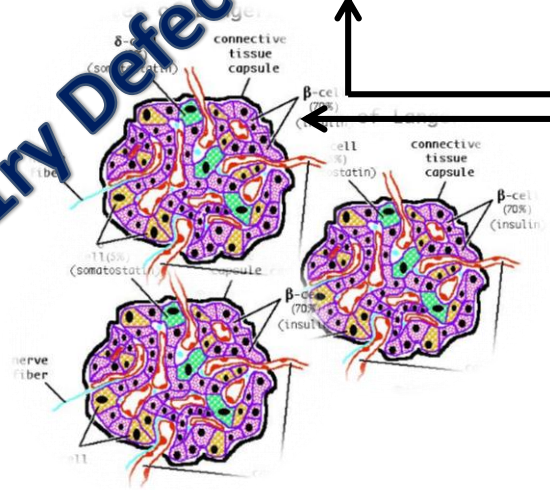
Decrease specific glucose transporters



Obesity

Desensitization

1ry Defect



Glucose

Post-Receptor Defect

Down regulation of insulin receptors

Type 1 DM

Anti-Insulin Antibodies

Insulin

ttt by Insulin

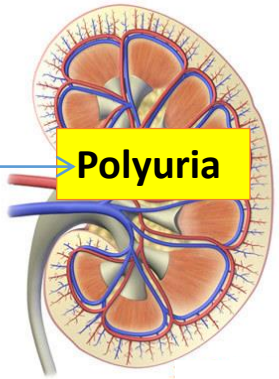
Type 2 DM

Insulin resistance

Insulin

Diet Plus Oral Hypoglycemic drugs

Clinical picture: Polyuria-Polydipsia- Polyphagia- Weakness- Loss of weight.



Glucose (in blood)

Polyuria

Increase Plasma Osmolality

Thirst Center

Polydipsia

Insulin

Glucose (in cells)

Loss of electrolytes (Na, K, Mg)

Polyphagia

weakness

Increased Lipolysis

Increase Protein Catabolism

Loss of Weight

Diagnostic Chriteria of DM

	Normal	IGT	DM
①FPG (mg/dL)	< 100	100-125	≥ 126
②2hs after glucose load (mg/dL)	< 140	≥ 140-199	≥ 200
③HbA1c%	< 5.7	5.7-6.4	≥ 6.5

A fasting plasma glucose > 126 mg/dl or HbA1c of > 6.5% is diagnostic of DM if confirmed by repeated testing

Diabetes Mellitus

Part II (Complications)

تحذير هام

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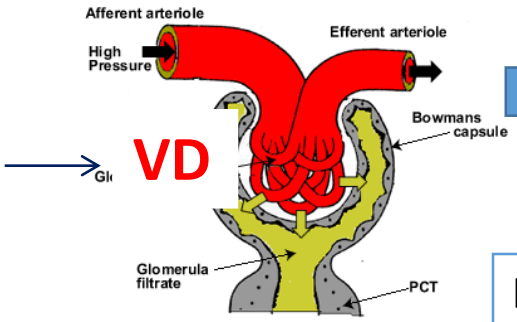
Pathogenesis of DM

1. Decrease in contractile ability of meningeal cells
2. Immune Dysfunction
3. Non enzymatic glycosylation of proteins
4. Exceeded the capacity of glycolysis

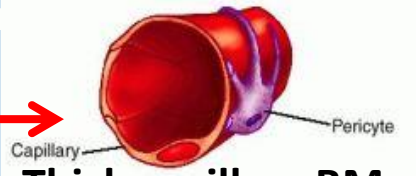


VD → ↑GFR

Infections

AGEs

- Impair interaction between lamina propria and proteoglycans → Increase capillary permeability → Proteinuria
- Irreversible cross linkage to LDL → Atherosclerosis
- Irreversible cross linkage to Albumin → Thick capillary BM



Attached to collagen in vessel wall

Interact with specific receptors on Macrophages

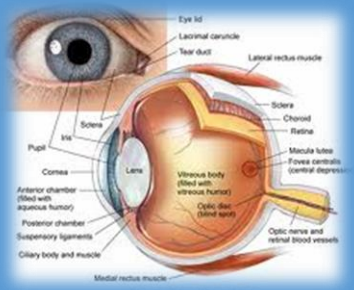
IL1
TNF

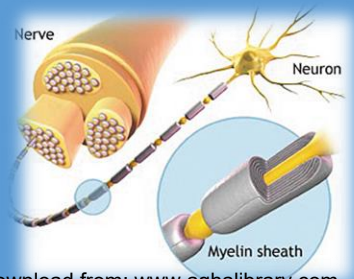
Microangiopathy

- Retinopathy
- Neuropathy

Polyol pathway → Sorbitol → Eye damage

The presence of microangiopathy, neuropathy, and metabolic derangements in diabetic patients makes them liable to develop many complications.





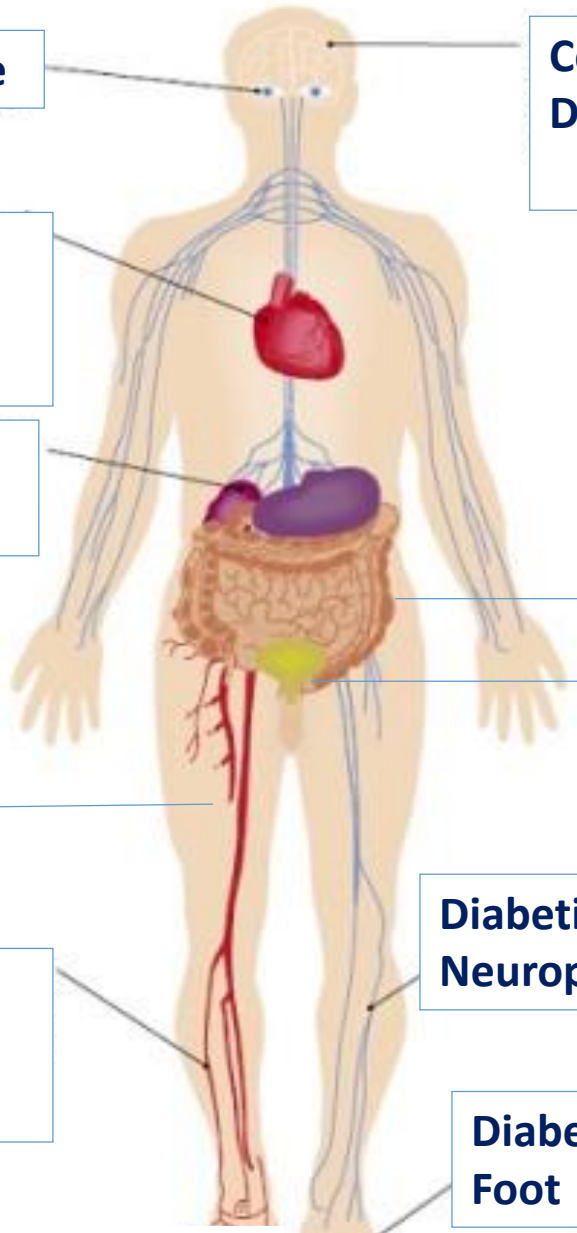
The most prominent complications of DM are:

1-Diabetic eye disease

2-Diabetic kidney disease (nephropathy)

3-Diabetic neuropathy

Complications of DM



Eye disease

Cerebro -Vascular Disease(Stroke and Coma)

Ischemic Heart Disease

Diabetic Kidney

Diarrhea

Sexual Dysfunction

Skin infections

Diabetic Neuropathy

Diabetic vascular disease

Diabetic Foot

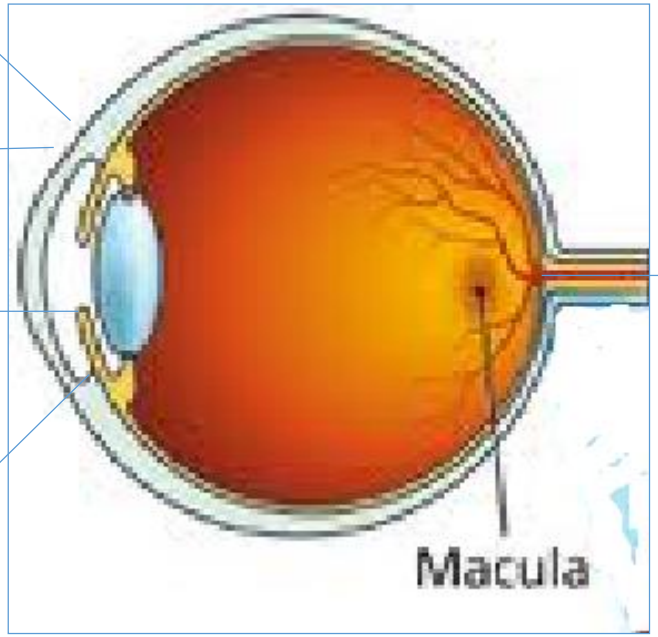
Diabetic Eye Disease

Recurrent sties

Xanthlasma

Cataract

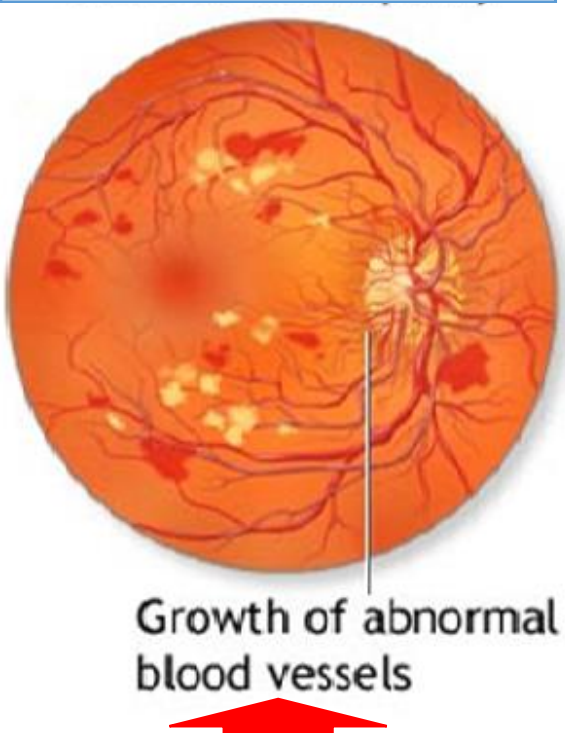
Glaucoma



Retinopathy

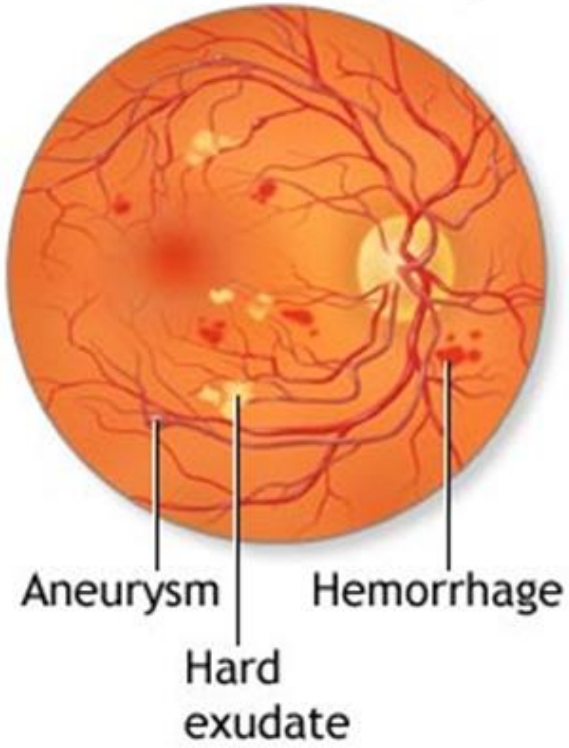
Diabetic Retinopathy

Proliferative retinopathy



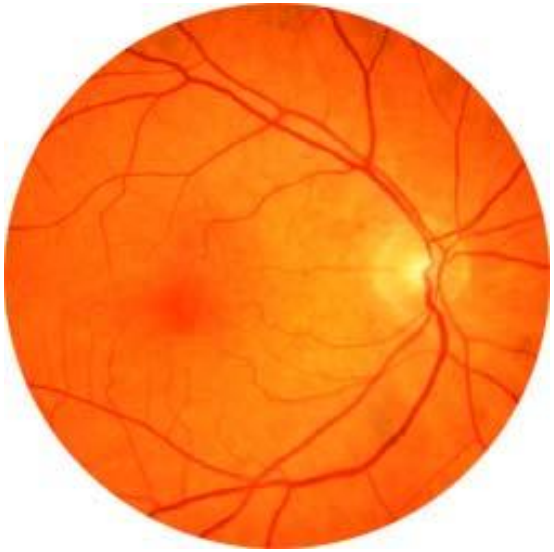
Early photocoagulation can prevent BLINDNESS!!

Exudative



1- **Hard exudates:** Leak of Plasma
2- **Cotton Wool exudates:** due to ischemia (ominous sign)

Normal Retina



Diabetic Kidney Disease

❶ Many factors play a role in the pathogenesis of Diabetic nephropathy.

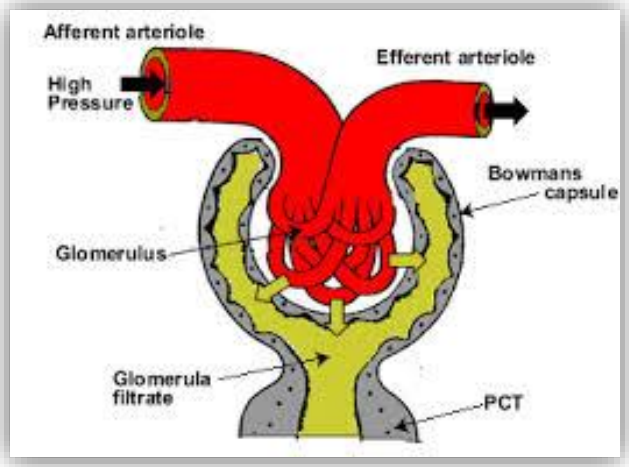
These include:

Hyper-filtration injury (due to ↑ GFR)- Atherosclerosis- Infections- VUR (due to autonomic neuropathy)- and others.

❷ The condition can range from mild proteinuria to End Stage Renal Failure (ESRF).

(**Proteinuria** is considered to be the main feature of this condition)

❸ Early proteinuria could be diminished by the use of **ACE-I**

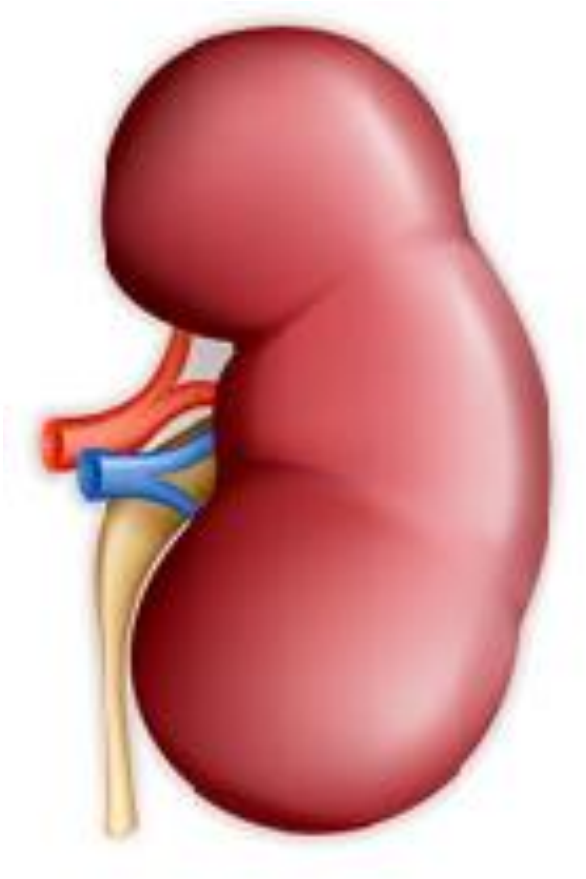


Stage I: Increase GFR-increase kidney size-micro albuminuria more than 15ug/minute BUT less than 100 ug/minute-NO increase in incidence of Hypertension.

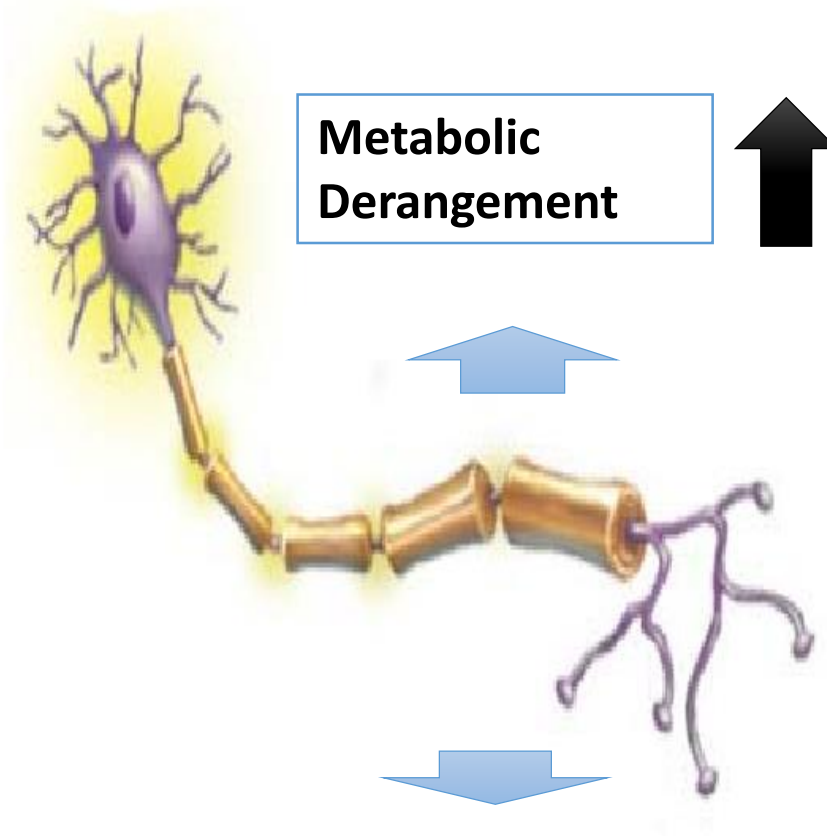
Stage II: Declining GFR-increasing proteinuria that may reach nephrotic range within years (Hypertension and Edema?!)

Stage III: Rapid decrease in GFR that leads to Azotemia- Hypertension and oedema worsens…Finally, Hypoproteinemia and widespread microangiopathy.

Stage IV: ESRF



Diabetic Neuropathy



Metabolic Derangement

Decrease Myoinositol uptake in Nerve Cells

Sorbitol

Nerve dysfunction

Diminished Na-K ATPase activity

PROGRESSIVE

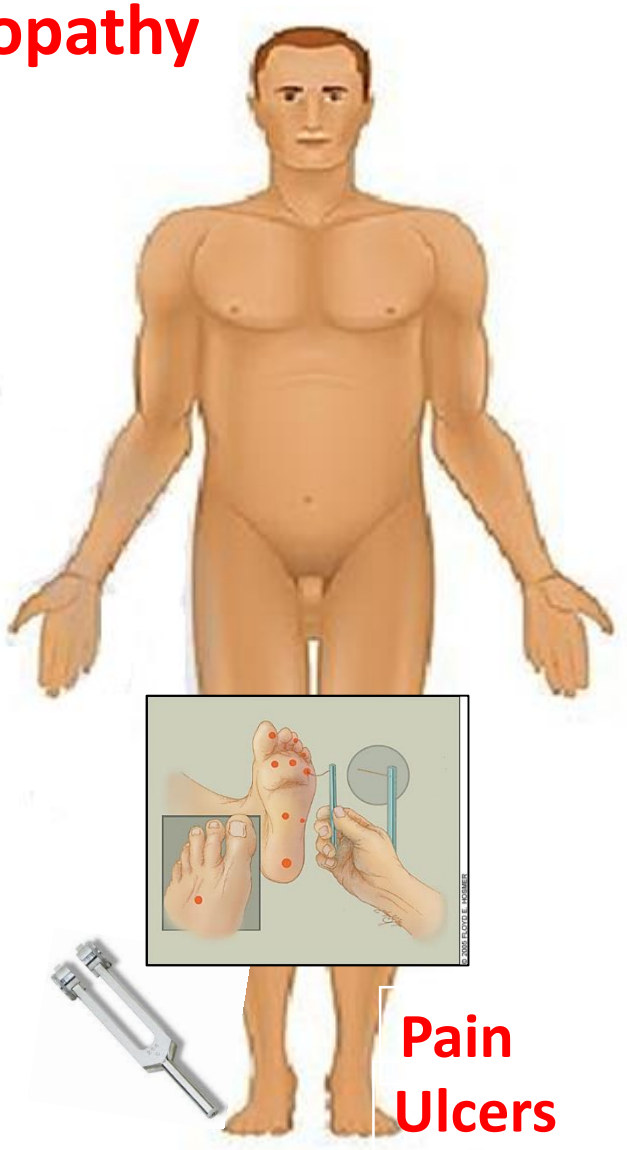
Segmental Demyelination

Due to obstruction of vasa nervosa?!

REVERSIBLE

1 Peripheral neuropathy

1. The Most common type
2. A generalized sensorimotor polyneuropathy affecting legs earlier than hands
3. Early loss of tendon reflexes/vibration sense
4. Muscle weakness
5. May lead to deformities and leg ulcers
6. Early detection by Neurothesiometer -for pain threshold
7. If severe pain treatment by Amitriptyline/topical capsaicin (depletes substance P) also ?! Mexiletine



Pain
Ulcers

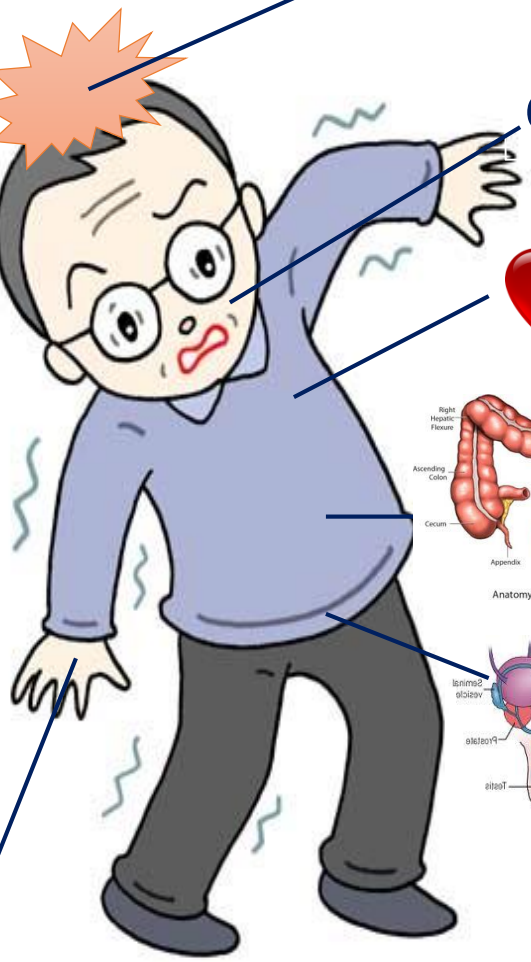
Early loss of vibration sense

2 Autonomic Neuropathy

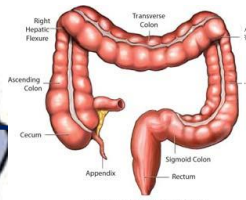
Postural Hypotension

(ttt) by (Stop precipitating drugs- elevate head of the bed-stocking-high Na Intake Fludrocortisone)

Gustatory sweating



Resting Tachycardia

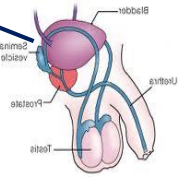


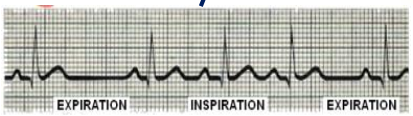
Anatomy of Large Intestine

Constipation/ Diarrhea

Retention of urine….(infections)

Impotence





Loss of RSA

3 Mononeuropathy (or Mononeuropathy Multiplex)

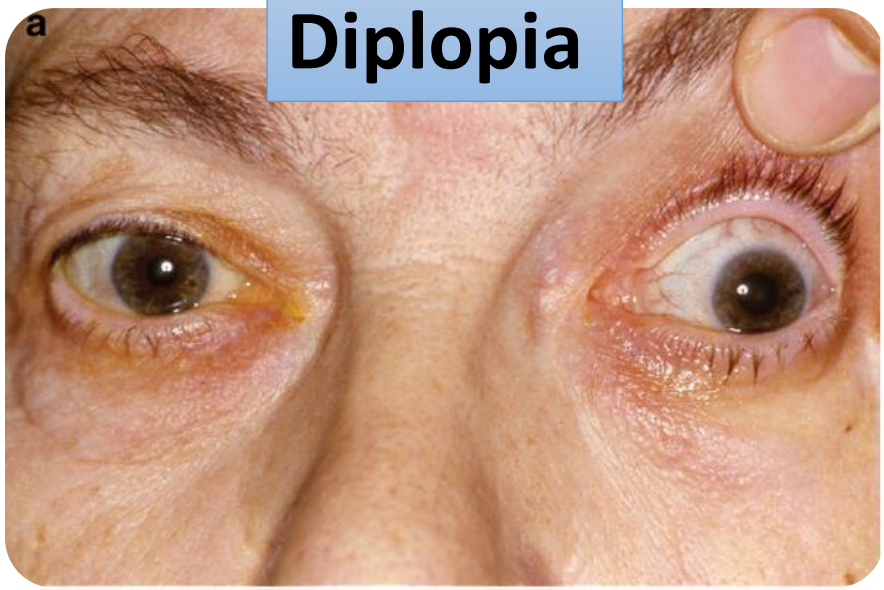
1-?!Acute thrombosis or Ischemia of vasa nervosa

2-Acute onset neuropathy

3-Reversible within 1-3 MONTHES

4-The commonest site is the 3ed cranial nerve

5-If+++++++ pain insomnia and weigh loss



Reversible

Diabetes Mellitus

Part III (Treatment)

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Treatment of DM

Diet

The following are general recommendations for diabetic patients

Ideal body weight especially by decreasing caloric intake.

Carbohydrate (50%): better complex Carbohydrates.

Fat (35%) with saturated fat <1/3

Protein (15%) (substitute poultry, veal, and fish for red meat)

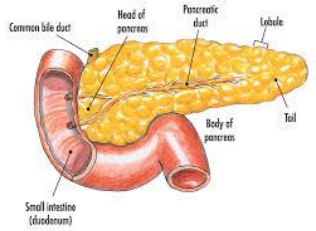
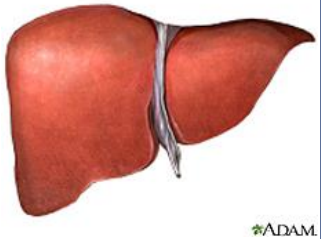
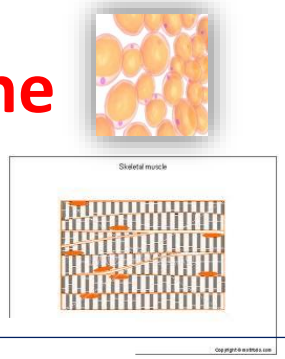
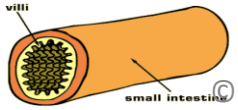
Increase soluble fiber content e.g. beans-oat meals-apple skin……as they retard nutrient absorption rates thus improve the glycemic control.

Artificial sweeteners

Moderate regular exercise and increase Carbohydrates before exercise

Treatment of DM

Oral Hypoglycemic Drugs

Sulfonylureas		Increase Insulin secretion by beta cells of the pancreas	**Hypoglycemia**
Biguanides		Decrease Hepatic glucose production (useful in Obese patients)	**Lactic acidosis**
Thiazolidinedione		Increase Muscle and adipose tissue glucose uptake	**Liver damage** (Monitor the Liver)
Alpha glycosidase inhibitors		Slow carbohydrate absorption	**Flatulence**

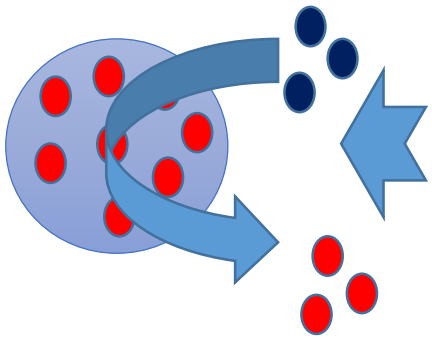
Treatment of DM

Drug interactions of Sulfonylureas [1]

Treatment of DM

Drug interactions of Sulfonylureas [2]

With oral Anticoagulants



Sulfonylureas causes displacement of oral Anticoagulants which can lead to bleeding

Treatment of DM

Insulin

Indications for using Insulin

1. **IDDM (Type 1)**
2. **NIDDM (Type 2) after failure of diet and oral hypoglycemics (i.e. failure to bring FPG below 180 mg/dl)**
3. **Pregnancy**
4. **Emergency conditions such as Infections-Operations-DKA**

Treatment of DM

Types of Insulin

	Onset	Peak	Duration
Rapidly acting (Regular Insulin)	1H	6h	14h
Intermediatelly acting (NPH)	1h	10h	24h
Long acting (protamine Zink)	7h	16h	36h

Treatment of DM

How to use Insulin

①Since the response to insulin is individual so we should use "Trial and error" approach

②Initial dose is usually 15-20 U (if the patient is obese 25-30 U due to insulin resistance)…then gradually increase the dose by 5-10 U per step

③Rotate injection sites within the same area to avoid lipoatrophy at injection sites But AVOID random rotations between different areas to avoid day to day variability.

④If the patient needs to only increase soluble insulin (Normally: NPH 2/3 + Sol 1/3) consider mixing problem and shift to separate injections.

Trial and error

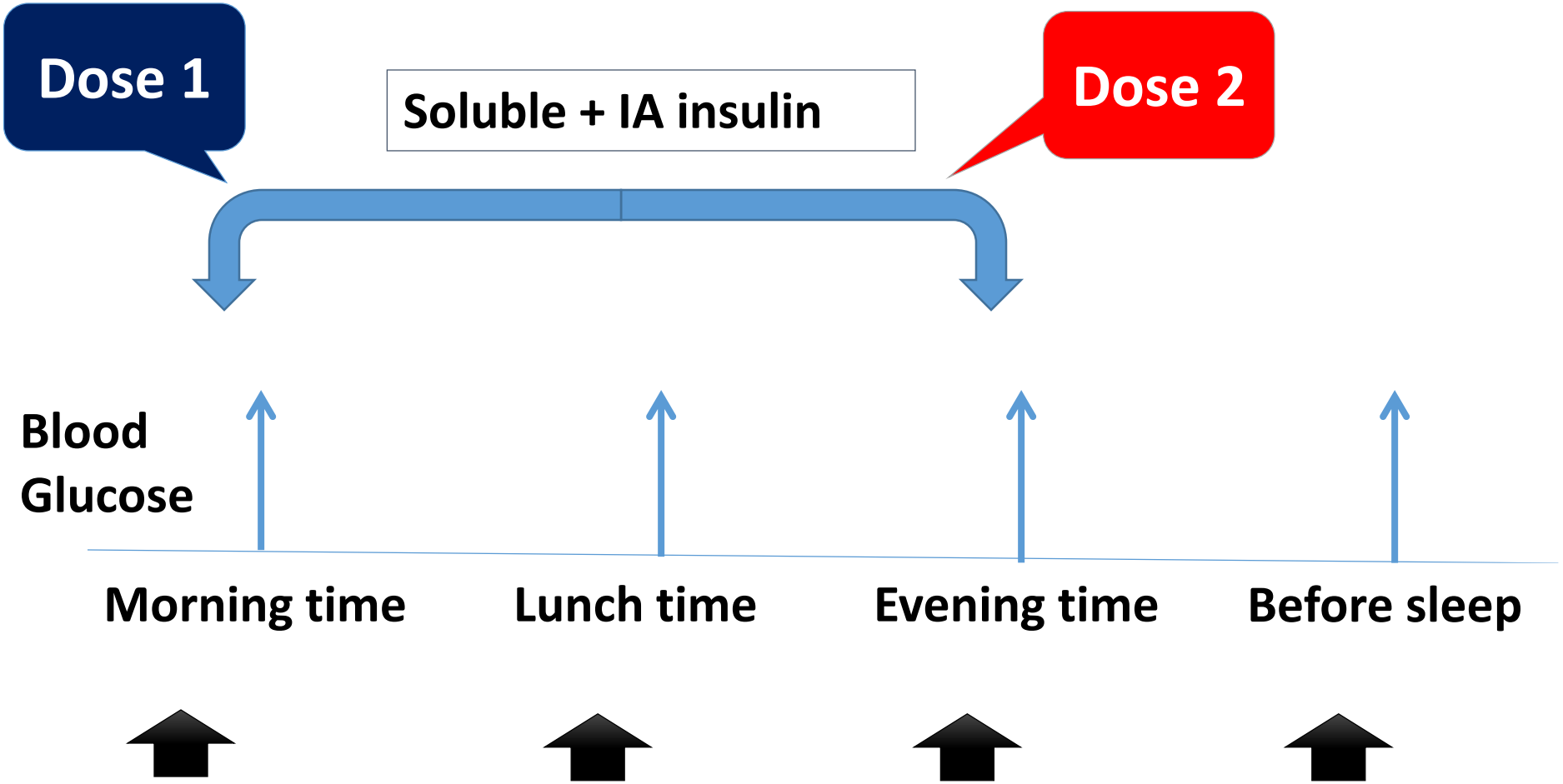
Small initial dose 15-20 U

Rotate injections

Treatment of DM

How to adjust the Insulin dose

❶Most people use two doses of mixed insulin per day (Sol plus Med acting) (one injection before breakfast and one before dinner)
❷This gives easier adjustment for blood glucose
❸Insulin is given 30 minutes before meals so that by the time it is absorbed it matches the absorption of food
❹Initially you need to measure the patient's blood glucose 4 times a day (before breakfast-before lunch-before dinner- and before bedtime)
❺When blood glucose is high at any time you need to increase the Insulin dose that controls it e.g. if blood glucose is high in the morning, you need to increase the insulin dose that controls this time of the day (which is by increasing the evening Intermediate acting insulin as its peak of action is after 10 hours i.e. covers the morning blood glucose) [see the illustrations in the next page]



Diabetes Mellitus

Part IV (DKA)

تحذير هام

هذا الفيديو و جميع الرسوم التوضيحية المتاحة فى هذا الموقع هم تحت حماية قانون الملكية الفكرية

هذا الفيديو و جميع الرسوم التوضيحية المتاحة فى هذا الموقع هم تحت حماية قانون الملكية الفكرية و لا يحق لأحد إستخدامه أو إستنساخه أو إعادة طبعه أو طبع أجزاء منه بدون إذن مسبق من الدكتور طارق عبد الحميد (أو من ينوب عنه قانونياً). و من يتعدى على الملكية الفكرية المذكورة فسوف يتعرض للمسائلة القضائية عن جريمة إنتهاك حقوق الملكية الفكرية. و إستخدام الفيديوهات و المادة العلمية المذكورة مكفول فقط لمشترى واحد فقط و لأخذ تصريح إستخدام لأكثر من شخص برجاء الإتصال بالدكتور طارق عبد الحميد

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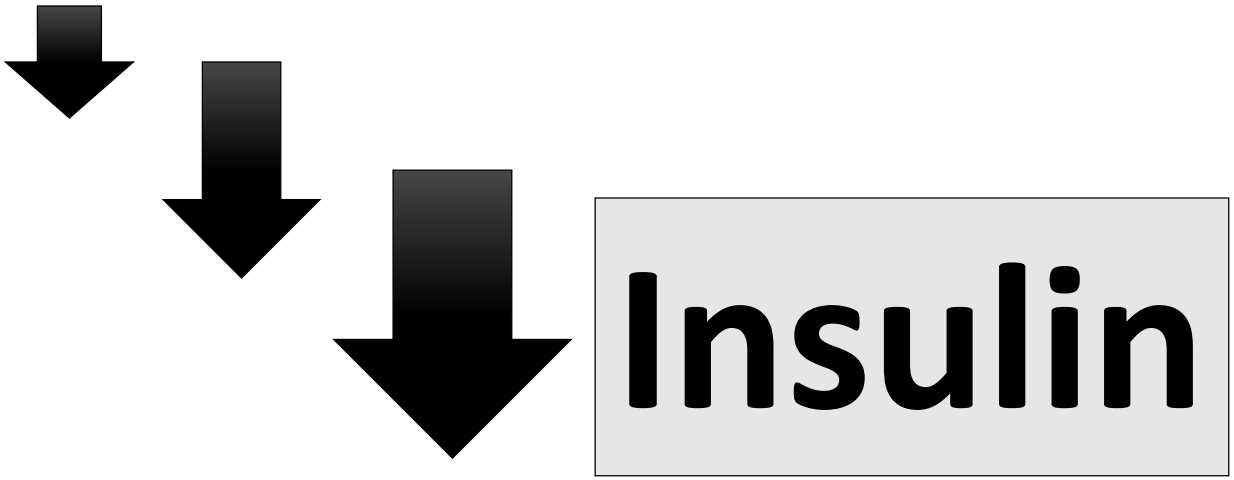
Diabetic Keto Acidosis DKA

Definition

DKA is a life threatening condition that occurs secondary to severe insulin deficiency.



Diabetic Keto Acidosis DKA

Aetiology

1. **Decrease insulin dose**
2. **Severe infections** (due to increase anti-insulin antibodies as part of immune reaction against the infection)
3. **Type 1 DM**
4. **Stress** e.g. AMI-Pancreatitis-Uremia-accidents- and Surgical Operations (due to increase anti-stress hormones which interfere with insulin action).

Pathogenesis of DKA

Insulin

Glucose

Glycosuria

Polyuria

Hematocrit value

Loss of electrolytes Na, K (Total)

Hypovolemic

RBF

Shock

Adrenocortical activity

Serum osmolality

BUN& Creatinine

Leukocytosis

CNS depression

Lipolysis

Ketone bodies

Acidosis

ANV

Deep Rapid Respiration (Kusmols breathing)

Mental Confusion

Cardiac Contractility

(Hyperkalemia)..... k

Clinical Manifestations of DKA

Weakness-Weight Loss

Neck Veins do not fill to ½ in supine position

Confusion coma

Blurred vision

Polydipsia
Polyphagia
Smell of acetone

Deep rapid respiration (Kussmauls breathing)

Anorexia-Nausea-Vomiting
Abdominal Pains (due to dry peritoneum)

Polyuria-Glycosuria

Postural Hypotension

Poor skin turgor (due to dehydration)

K

Special investigations for DKA

1-High blood glucose level (More than 250mg/dl)

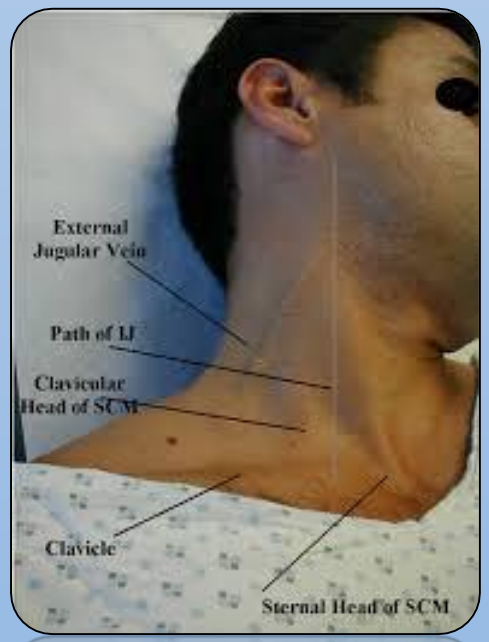
2-Acidosis

3-Low serum bicarbonate level

4-Positive ketone bodies in blood

Note1
Hypovolemic in DKA can present as:

Orthostatic Hypotension
Dry mouth
Abdominal pain
Poor Skin turgor
Failure to fill the jugular veins to its 1/2 while the patient is supine

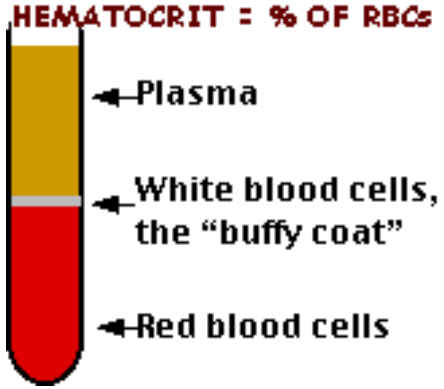


Note 2

Since **Hematocrit values is typically high is DKA** (due to loss of water in polyuria), accordingly the presence of NORMAL Hematocrit in patients with DKA may indicate the presence of underlying anemia.

Normal Hematocrit in DKA

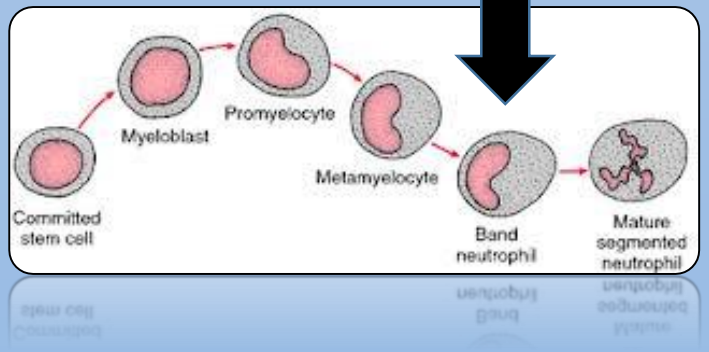
= ?!Anemia



Note 3

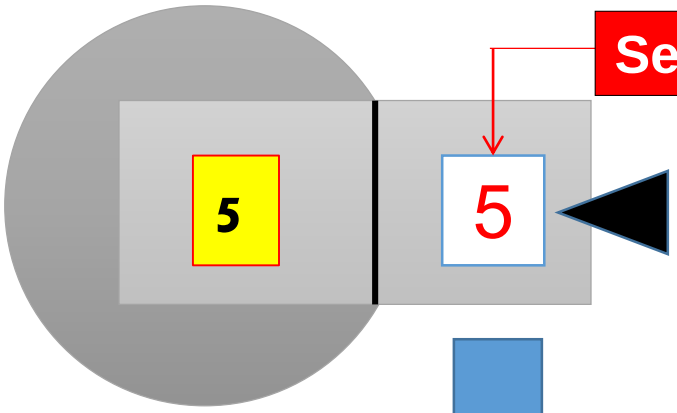
Leukocytosis associated with DKA is typically associated with : **No increase in band neutrophils.** If Band Neutrophils were increased....You should suspect infection.

Infection Increases Band Neutrophils.



The dilemma of K in DKA

k

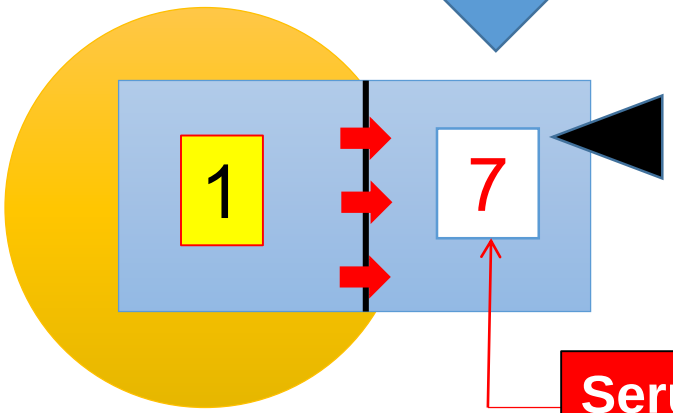


Serum K is 5 units

Normal situation

Total K is 10 Unites

DKA



In DKA the total K is low but serum K is high

Serum K is 7 units

Total K is 8 Unites
(due to loss in urine)

Treatment of DKA

1-Soluble Insulin IV to correct the hyperglycemia
2-Saline Infusion to correct the hypovolemic
3-K infusion to correct the total K deficit (but take care to give it ONLY with or after Insulin to avoid marked hyperkalemia (as serum K is already high due to the presence of acidosis)
4-Correction of any predisposing factors e.g. ttt of associated infection by antibiotics
5-ttt of complications e.g. Acidosis- Low PO4- others

Insulin | **Saline** | **K ONLY after Insulin** | **ttt of associated infection** | **ttt of acidosis (if needed**

Note:

Bicarbonate is used ONLY if PH is less than 7.1

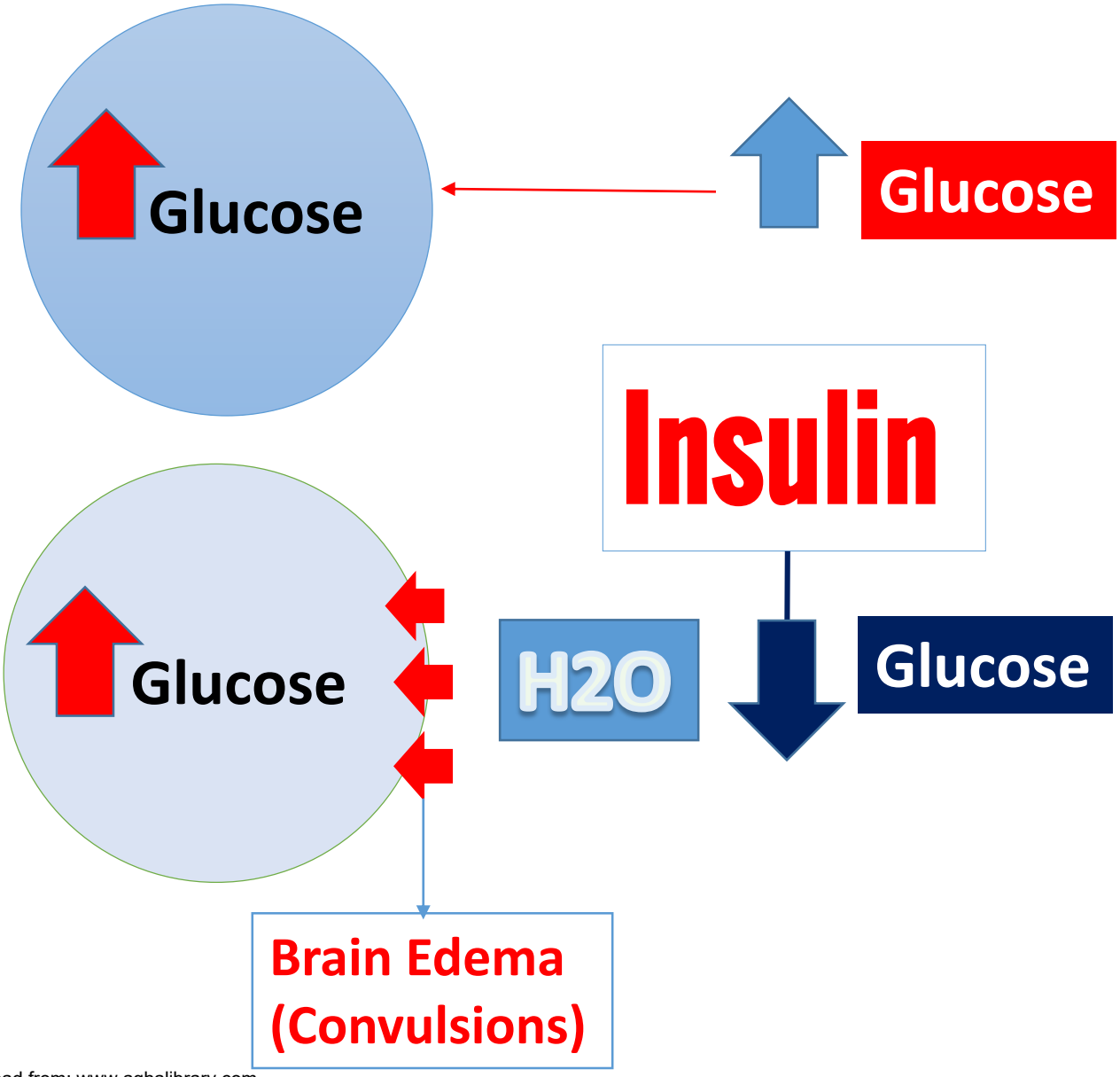
Mild acidosis is usually corrected after giving insulin as the acid radicals are metabolized.

Saline infusion

1. 0.9% Saline infusion 1L/h for the first 2 hours then ½ L/h.

2. When blood glucose level is lowered to 250 mg/dl change the fluid to 5% glucose containing solution to prevent Hypoglycemia and brain edema (due to disequliberation syndrome.

Disequliberation syndrome



Insulin

Start by 0.15 unit/Kg as an IV bolus to prime the tissues insulin receptors then give 0.1 unit/kg/h as continuous infusion or IM injection

K

If the patient is not uremic and has an adequate urinary output KCl 10-30 mEq/h is given 2-3 hours AFTER Insulin

Tomato Juice

Banana

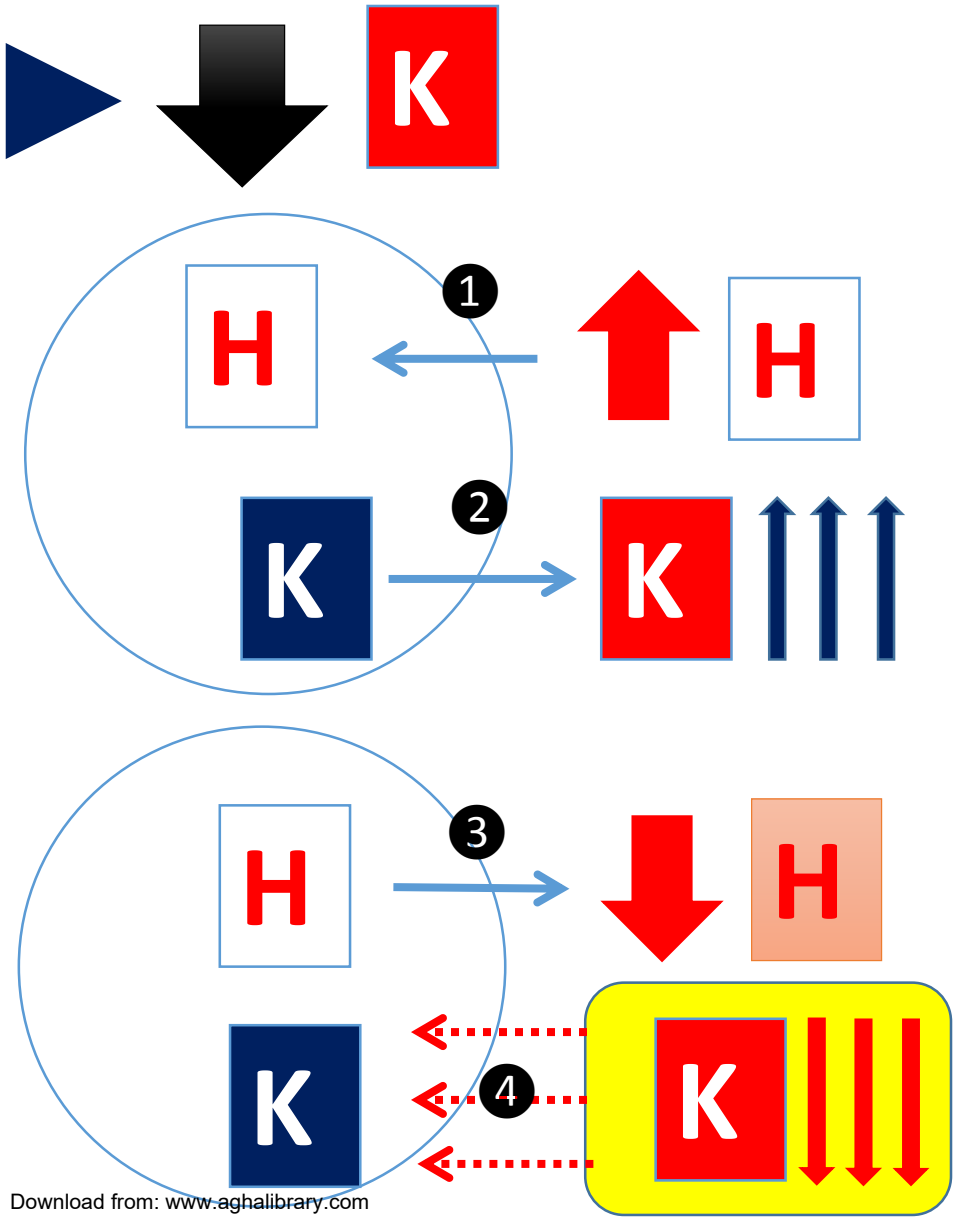
Bicarbonate

Only if PH less than 7.0

One or two ampules of Na Bicarbonate should be added to 1L of 0.45% saline.

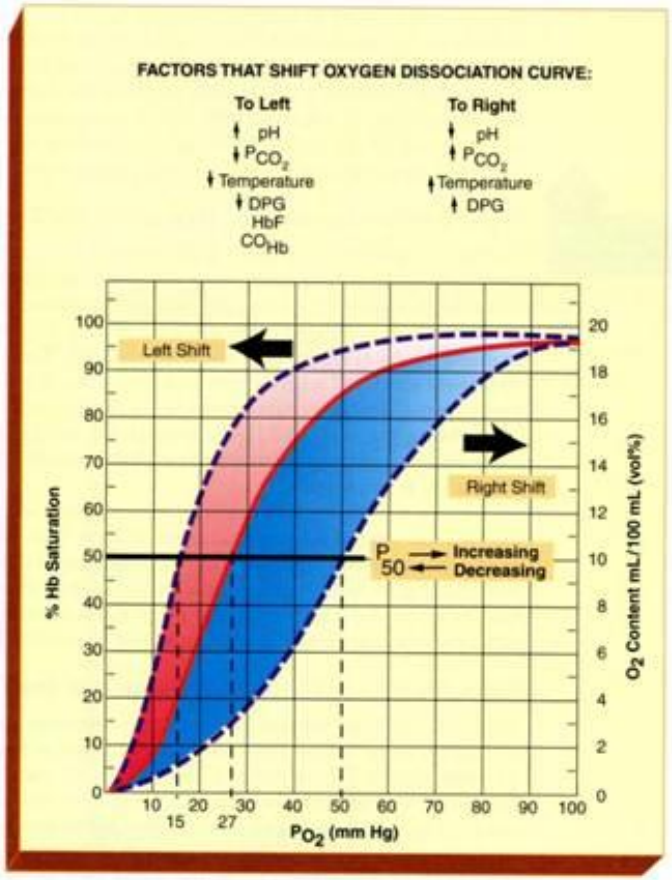
Avoid adding Na Bicarbonate to 0.9% saline as this will lead to the creation of markedly hypertonic solution which will aggravate the hyperosmolar state.

Complications of Bicarbonate therapy [1]



Complications of Bicarbonate therapy [2]

Tissue Hypoxia



Shift to the Left

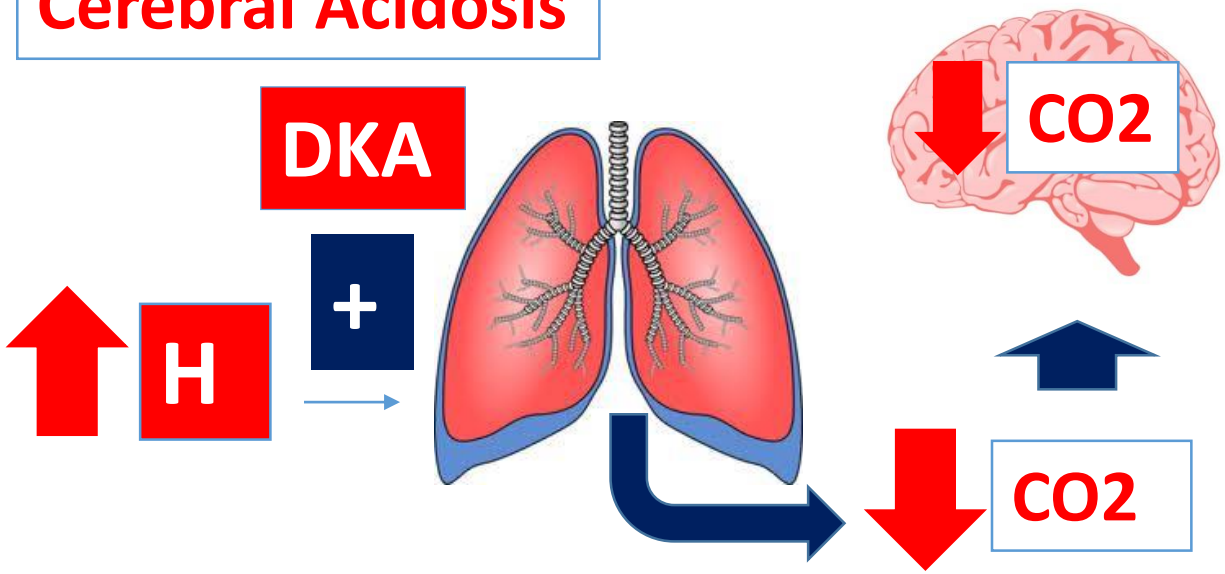
Impair Oxygen delivery to tissues

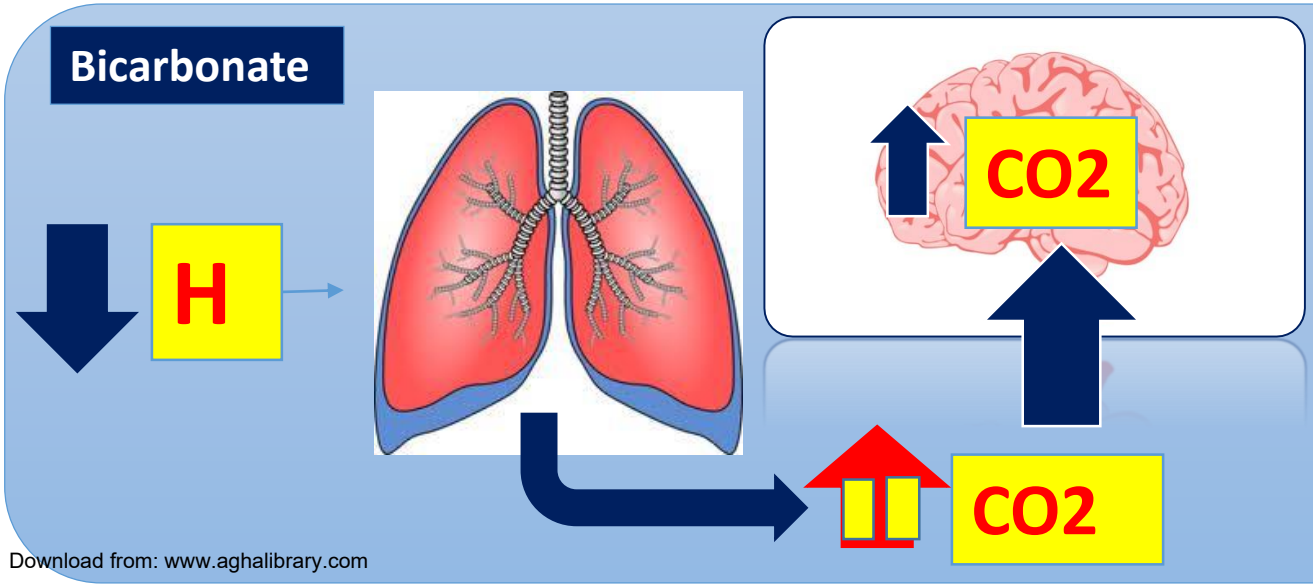
+

Shock

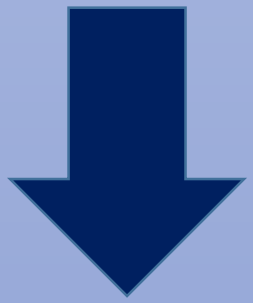
Complications of Bicarbonate therapy [3]

Cerebral Acidosis

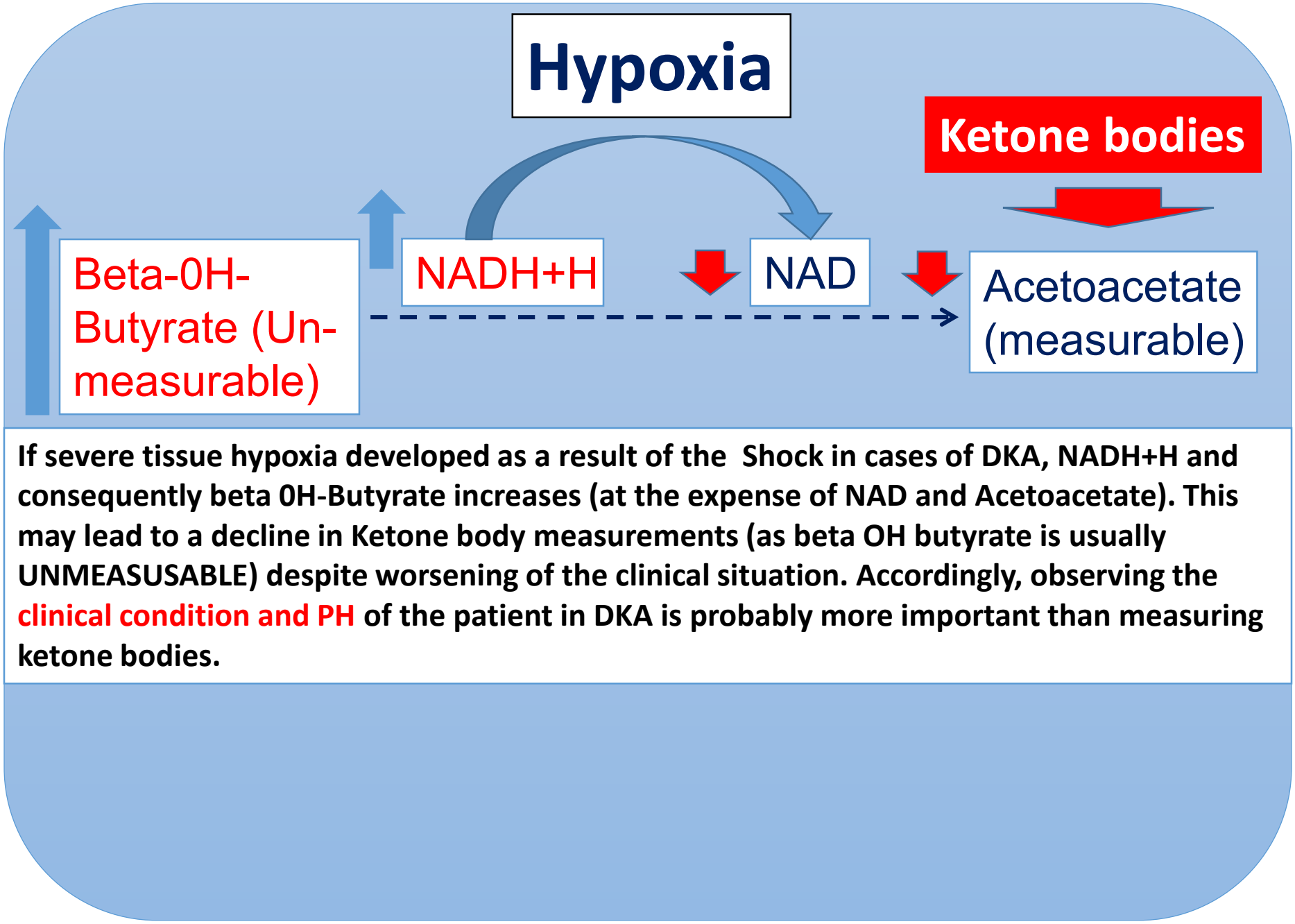




After insulin therapy the body tries to build its own denatured proteins...this utilizes PO4 and may cause Hypophosphatemia that manifests as severe **Muscle Weakness** which may need treatment by PO4.



PO4



If severe tissue hypoxia developed as a result of the Shock in cases of DKA, NADH+H and consequently beta 0H-Butyrate increases (at the expense of NAD and Acetoacetate). This may lead to a decline in Ketone body measurements (as beta OH butyrate is usually UNMEASUSABLE) despite worsening of the clinical situation. Accordingly, observing the clinical condition and PH of the patient in DKA is probably more important than measuring ketone bodies.

Diabetes Mellitus

Part V (Hypoglycemia, HONKS, Others)

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Hypoglycemia

Definition

A Metabolic Syndrome caused by severe by hypoglycemia in diabetic patients

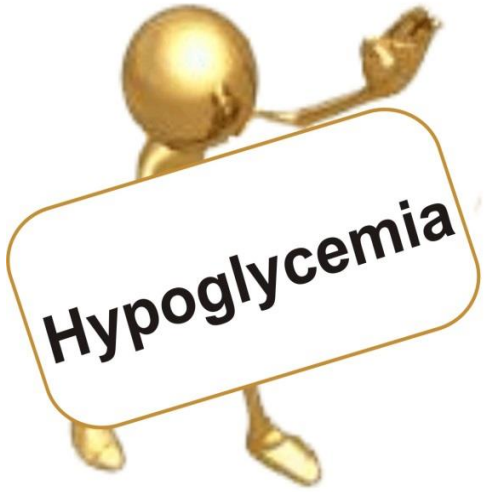
Aetiology

Predisposing factors for the development of hypoglycemia in a diabetic patient include:

1- **Overdose of Insulin or Sulfonylurea**

2- Lacking a Meal after insulin injection

3- Severe exercise after insulin injection (as this may increase the rate of insulin absorption from the injection site)

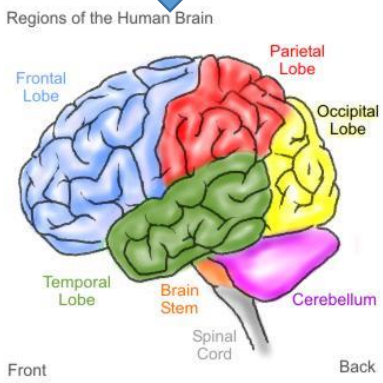


GLUCOSE

Low serum glucose level (usually less than 60 mg/dl)

Adrenalin

Sympathetic Nervous system



- Sweating
- Palpitation
- HR
- Tremors
- Irritability
- Nervousness
- Hunger pains

- Visual disturbance
- Confusion
- Headache
- Abnormal behavioral
- Convulsions
- Coma with Hyperreflexia (due to dysfunction of the pyramidal tract)

Treatment of Hypoglycemia

IV 50% 50 CC Glucose

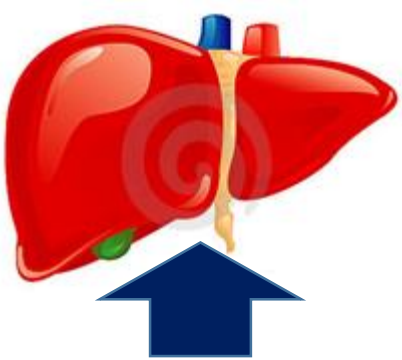
or/

IM Glucagon 1mg IM

or/

Oral Glucose (if the patient is conscious)

Note

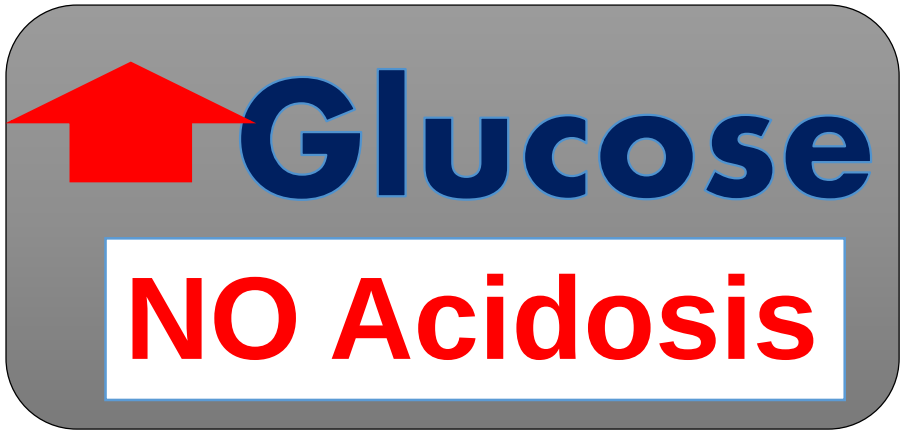


The treatment should be followed by an ORAL sank to replenish hepatic glucagon and thus prevent recurrence of Hypoglycemia.

Hyperosmolar Non-Ketotic Syndrome

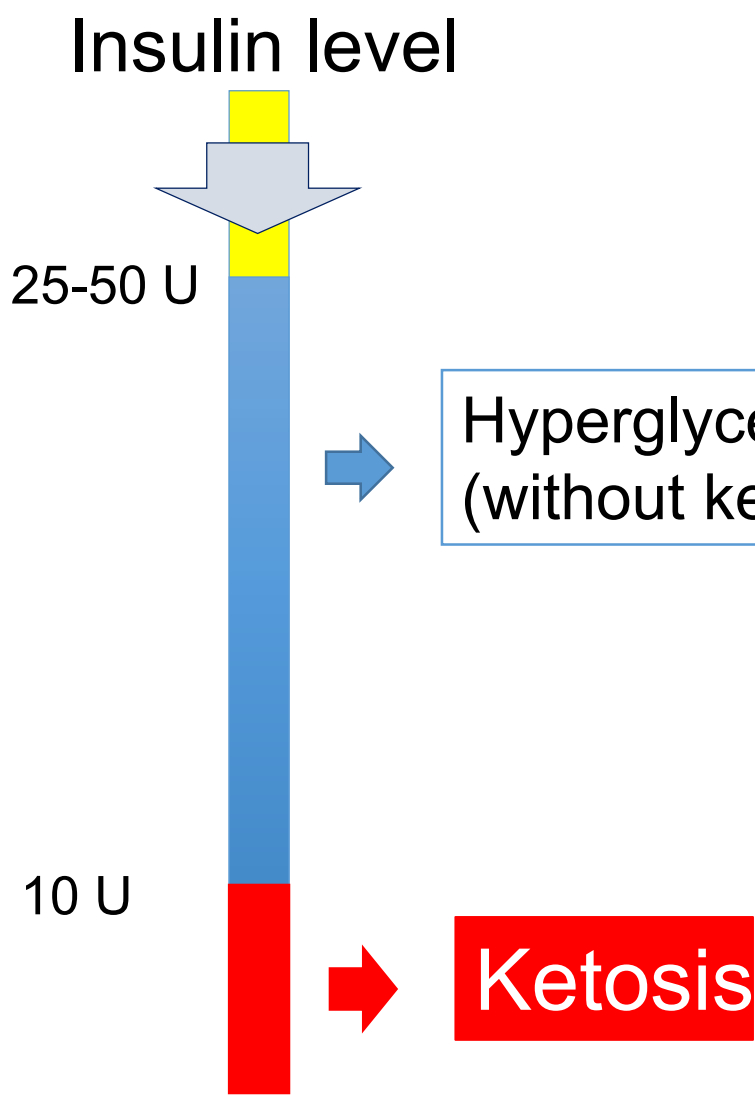
Definition

Marked increase in serum osmolality in a diabetic patient WITHOUT associated acidosis.



Aetiology of HONKS

Insulin deficiency



Hyperglycemia (without ketones)

Hyperglycemic hyperosmolar Non-Ketotic Syndrome (HONKS)

Ketosis

Pathogenesis of HONKS

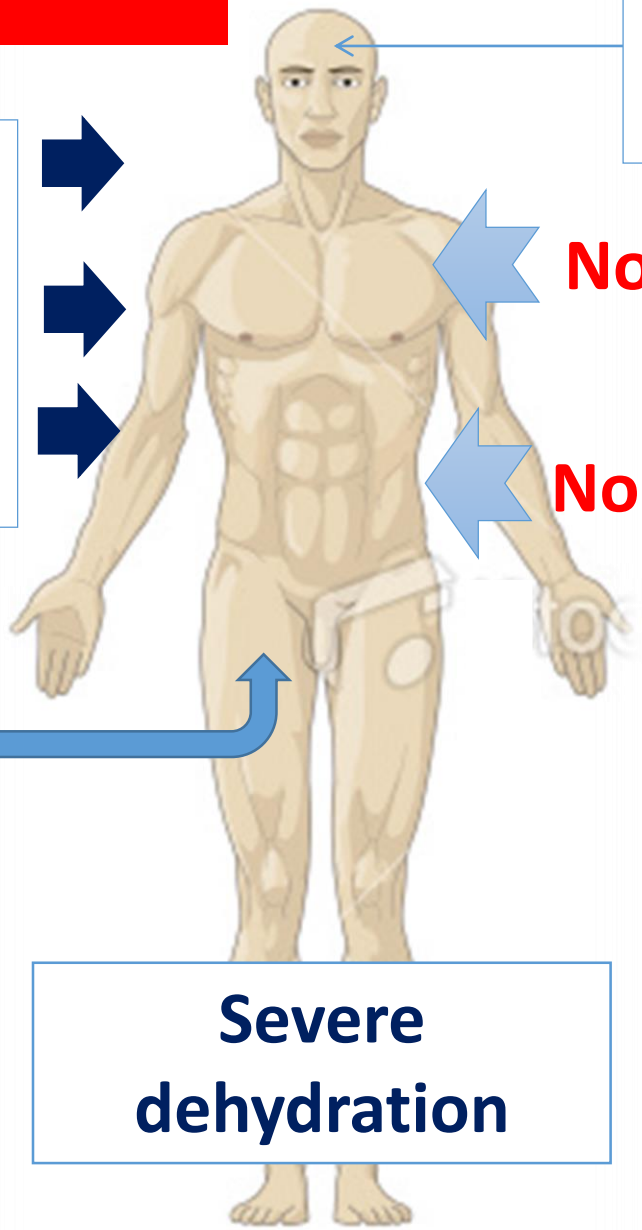
1-Same pathophysiology as DKA except that serum insulin levels are not low enough to cause ketosis.
2-Since ketosis is absent, anorexia and vomiting are NOT typically present
3-The lack of vomiting makes the patient late in seeking medical advice….this delay causes:

1. **Higher glucose concentration on initial measurements**
2. **Higher serum osmolality: this can cause more focal neurological signs than DKA**
3. **More severe dehydration**

The last 3 points explain to us why and how HONKS have higher mortality rate than DKA!

Clinical picture

Preceded by polyuria and polydipsia secondary to insulin deficiency



Focal neurological signs

No Kussmols breathing

No anorexia or Vomiting

Muscle weakness

Severe dehydration

Special investigations for HONKS

1. Hyperglycemia: More than 600 mg/dl
2. High serum osmolality (More than 310 mosm/kg)
3. No acidosis (except if complicated by shock as the latter may cause hypoxia which may result in Lactic acidosis)

Treatment of HONKS

Saline	Insulin	K

Management of complications of DM (1)

Severe vomiting	→	Check for DKA
Diabetic diarrhea	→	Empirical antibiotic therapy e.g. tetracycline cephalosporin
Erectile dysfunction	→	Sildenafil (NEVER use it with nitrates as this can cause fatal hypotension)

Management of complications of DM (2)

Slaw gastric empting	→	Metoclopramide (Note: my cause extrapyramidal manifestations due to its anti dopaminergic action)
Painful neuropathy	→	Amitriptyline
Retinopathy	→	Photocoagulation

Management of complications of DM (3)

Orthostatic Hypotension →
1. Compressive garments
2. NaCl
3. Fludrocortisone

Nephropathy and High BP → **ACEI (as it decreases micro albuminuria)**