

# DIABETIC KETOACIDOSIS

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# Introduction

**Diabetes Mellitus:** It is a chronic disease characterized by disturbance in carbohydrate metabolism due to inadequacy of insulin use and secretion.

- Prevalence, 2.8% in the year 2000, 4.4% in 2030
- One million new case in US
- 171 million diabetic patients in the whole world
- Turkey prevalence 7.2%

# Introduction

- Prevalence increased 33% from the year 1990 to today
  - Increase in obesity
  - Increased life expectancy and elderly population (>65 y)
- Diabetic emergencies are very frequent and cause fatal results
  - DKA
  - HONC
  - Hypoglycemia
- In the developed countries it is the 5th most cause of death

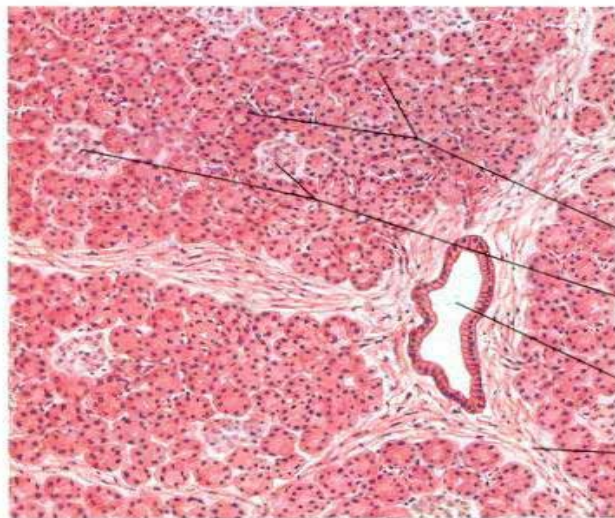
# Anatomy

## ■ Pancreas

- Behind stomach, between spleen and duodenum

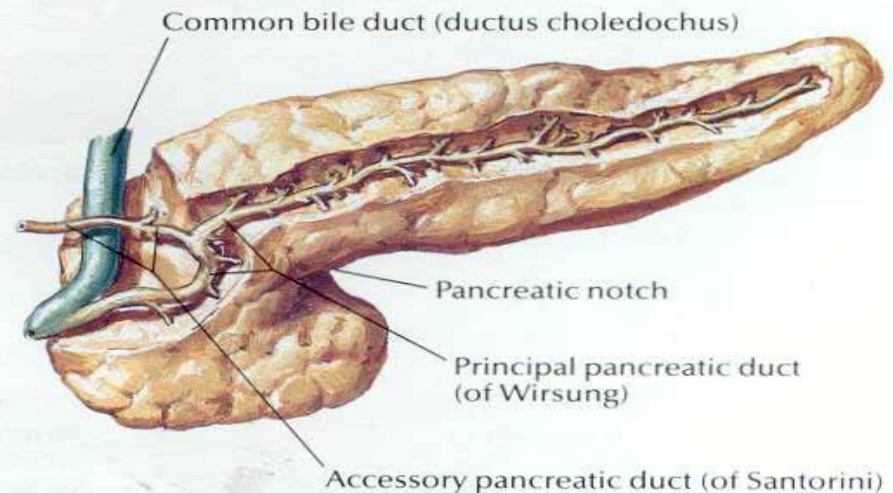
## ■ Islands of Langerhans

- $\alpha$  cells - glucagon
- $\beta$  cells - insulin, pro-insulin ve C-peptide
- $\Delta$  cells - somatostatin/gastrin



Low-power  
section  
of pancreas

Alveoli  
Islands (of  
Langerhans)  
Interlobular duct  
Interlobular septum



# DM

- Diabetes, causing macrovascular complications like atherosclerotic cardiovascular, cerebrovascular and peripheral vascular diseases results in atherogenesis and hypertension
- Additionally may cause complications like DKA, HONC and hypoglycemia requiring emergency care
- Early diagnosis and treatment are essential to prevent end stage complications

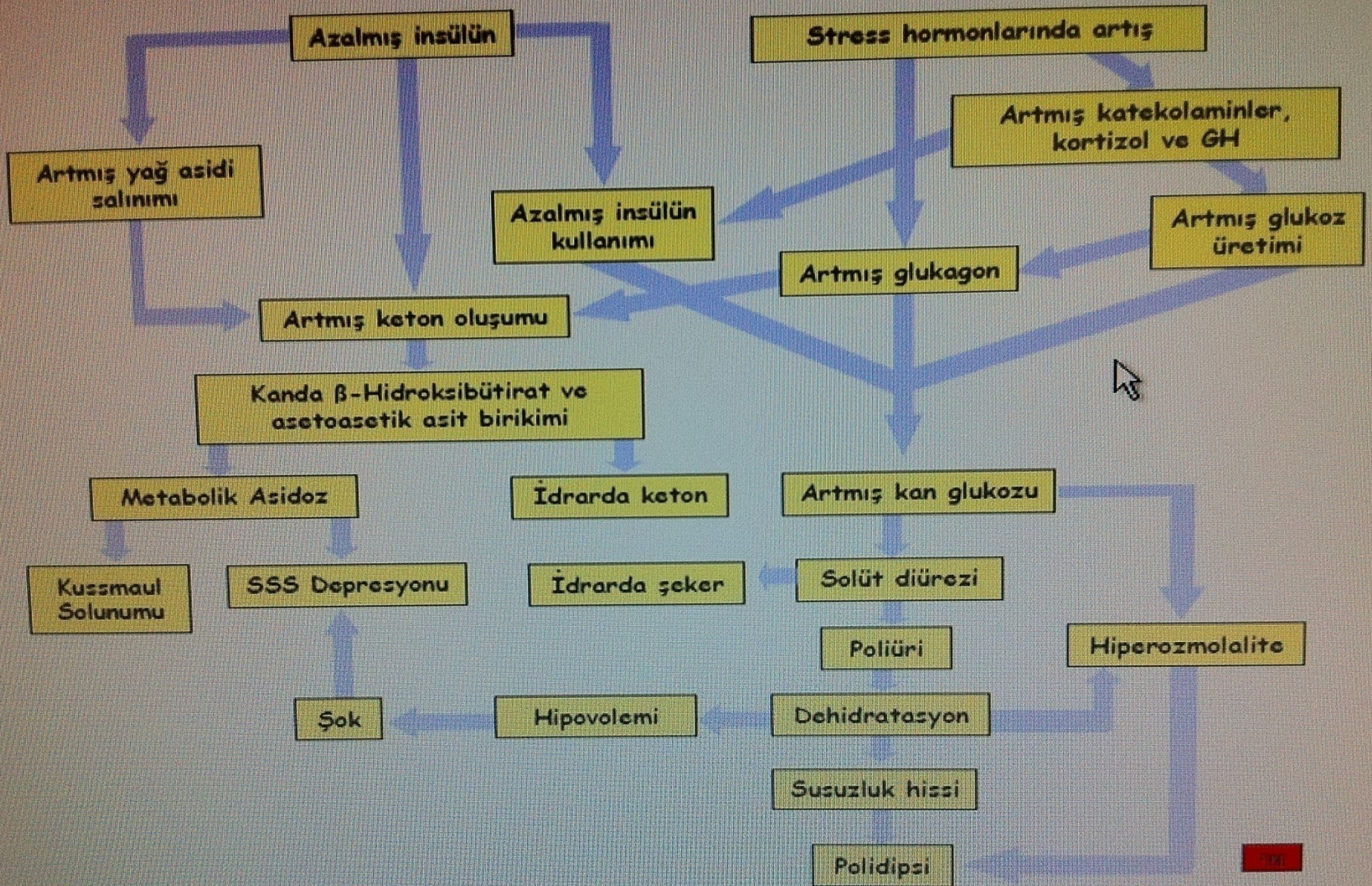
# DKA Definition

- In DKA, there is absolute or relative insulin deficiency. Hyperglycemia, dehydration and acidosis follow this condition
- There will be also disturbances in intermediate metabolisms

# DKA

- This absolute or relative deficiency in insulin level causes an increase in counter-insulin hormones like cortisol, glucagon, growth hormone and catecholamines
- This deficiency in insulin level, primary anabolic hormone, cause difficulty in muscle, adipose tissue and liver to take glucose
- Glucose production and lipid mobilization in liver increase, peripheral glucose usage decreases and ketone production is stimulated







# DKA Etiology

## Absolute insulin deficiency

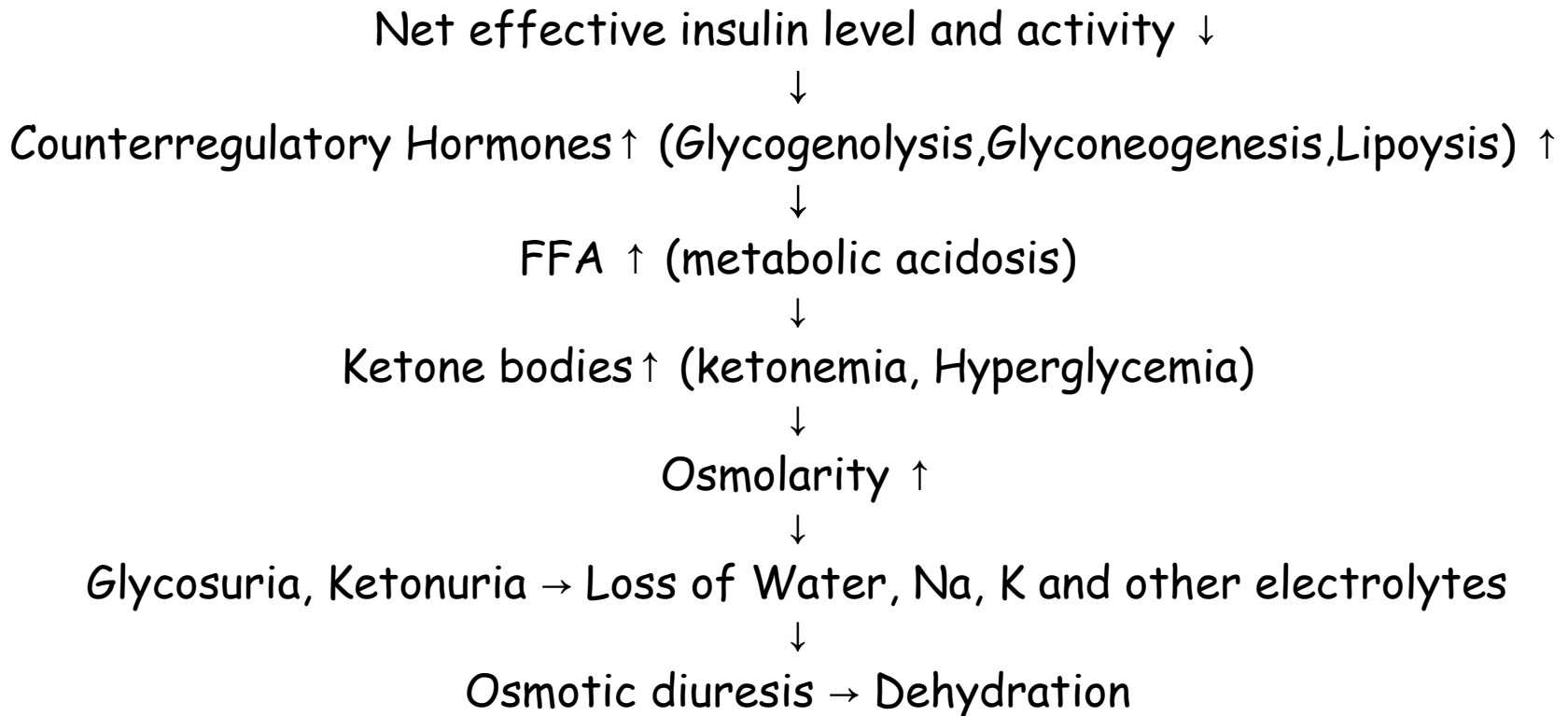
- Incompliance to insulin treatment, skipping the dose
- New onset Type 1 DM

# DKA Etiology

## Relative insulin deficiency

- Infection
- Myocardial Infarction
- Trauma
- Cerebrovascular disease
- Excessive exercise
- Fatigue
- Alcohol
- Steroids
- Adrenergic agonists
- Other stress sources

# Pathophysiology





# DKA

## Classical Triad

- Hyperglycemia (Usually  $>250$  mg/dL)
- $\text{HCO}_3^-$   $\downarrow$  ( $<15$  mEq/L)
- Ketonemia, ketonuria, acidosis ( $\text{pH} < 7.3$ )

# Signs

- Polyuria, dehydration
- Sodium, Phosphorus, Magnesium deficiency
- Deep hypokalemia may be seen

# Signs and Symptoms

- Thirsty, polydipsia, polyuria, nocturia  
(Classical Hyperglycemic symptoms)
- Kussmaul breathing
- Generalized weakness
- SSS depression (Letargy)
- Ketonuria/ketonemia
- Anoreksia (sometimes increased appetite)
- Nausea/vomiting
- Abdominal pain



# Differential

- Fasting ketosis
- Alcoholic ketoacidosis
- Acute appendicitis
- HONC
- Hypokalemia
- Hyponatremia
- Lactic acidosis
- Metabolic acidosis
- Myocardial infarction
- Pneumonia, immune-compramised
- Septic shock
- Salycylate poisoning
- Urinary infection

# Physical Examination

## General signs

- Sick appearance
- Dry skin
- Dry mucous membranes
- Decreased skin turgor
- Decreased reflexes
- Sensorial blunting
- Coma

## Vital signs

- Tachycardia
- Filiform pulse
- Hypotension
- Tachypnea
- Hypothermia
- Fever (if infection +)

# Physical Examination

## Specific signs

- Scent of ketone in the mouth (rotten fruit)
- Conscious, lethargic or comatous
- Abdominal tenderness (acute abdomen??)
- Hyperventilation secondary to acidosis  
(kussmaul)



# Laboratory

- Glucose:**  $>250$  mg/dL, Finger tip measurement must be made
- Na:** Every 100 mg/dL increase of glucose over 100 mg/dL, cause a decrease of 1.6 mEq/L in serum Na level
- K loss:** (5 mEq/kg) It may be masked as hyperkalemia due to acidosis !! ECG can be used to detect cardiac effects of potassium
- In the acidosis,  $H^+$  moves to intracellular space,  $K^+$  moves to extracellular space and paradoxally  $K^+ \uparrow$  seen
- As a result of rehydration and insulin treatment,  $K^+ \downarrow$  seen

# Laboratory

**Bicarbonate:** ↓ To evaluate acidosis, it is used together with anion gap

**Phosphorus:** Hypophosphatemia risk (poor feeding, chronic alcoholism) investigated

**Hyperamilasemia:** Can be seen without pancreatitis

**BUN:** ↑

# Laboratory

- **Anion gap:**  $\uparrow$  ( $\text{Na} - (\text{Cl} + \text{HCO}_3)$ ) (Due to bicarbonate decrease)
- **PA Lung X-ray:** To differentiate pulmonary infection
- **Brain CT:** Especially in children DKA can cause brain edema
- **ECG:** DKA can be seen due to a cardiac event or it may cause cardiac complications

# Laboratory

- CBC: WBC** > 15.000 or if there is a shift to left, think of bacterial infection!!!!
- Arterial or venous blood gas analysis:** Generally pH <7.3. Venous pH is <0,015 than arterial pH

# Laboratory

- Serum ve Urinary ketone:** (Only acetone and acetoacetic acid can be measured) Complete urine analysis
- Osmolality:** Osmolality diabetic coma patients is typically  $>330$  mOsm/kg water. If less than this value, think of another cause of coma!!!!  
$$2(\text{Na}+\text{K}) + (\text{Glucose}/18) + (\text{BUN}/2.8)$$

# Treatment-Prehospital

## Isotonic Sodium Chloride:

%0.9 NaCl 1 L bolus infusion (In the first hr)  
(With respect to vital signs and hypovolemia,  
more can be given)

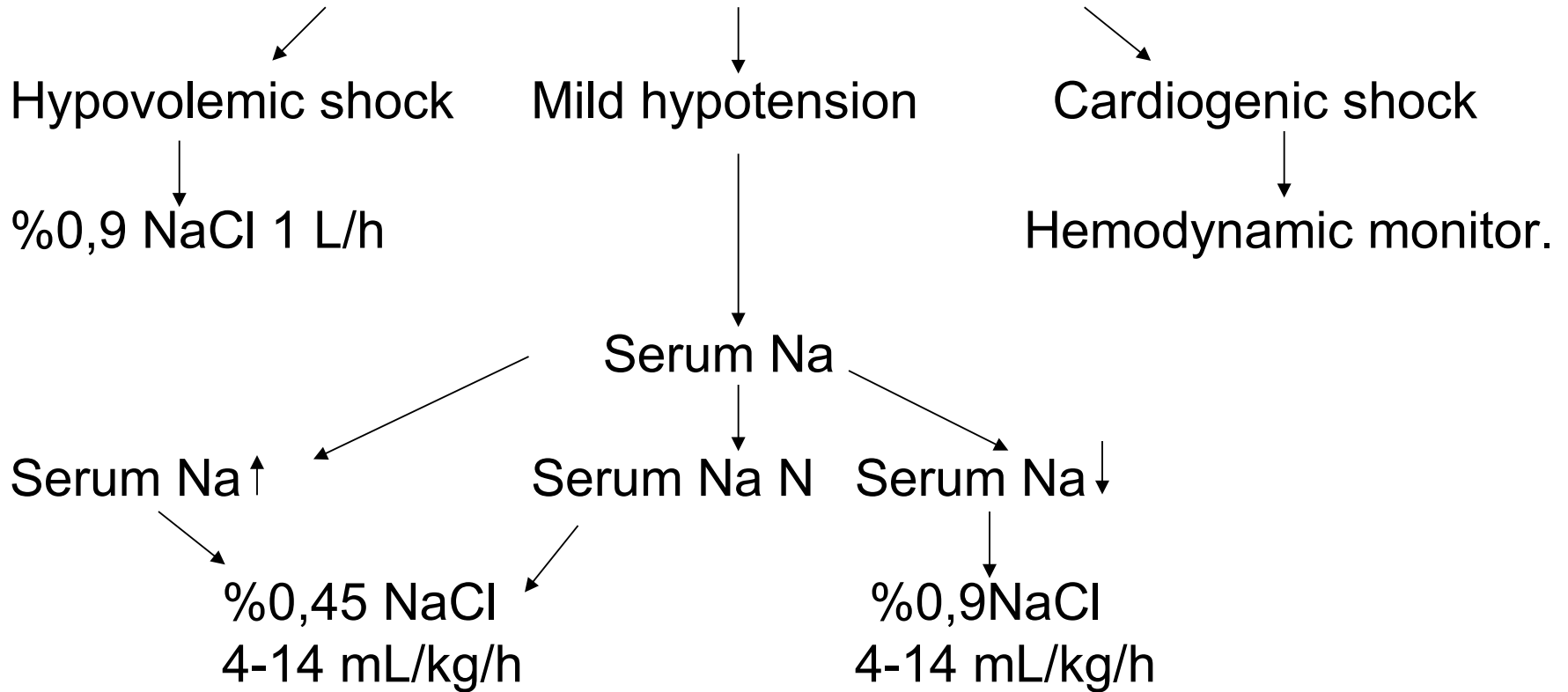


# Emergency Department Management

- ABC
- Monitorization
- Improve tissue perfusion and dehydration
- Normalise glucose and osmolality
- Improve acidosis and ketosis
- Replace electrolyte and volume loss
- Detect and treat comorbid conditions (infection...)

# DKA (Treatment, IV Fluids)

First 1-2 liter %0.9 NaCl (For hemodynamic stability)



# Treatment: Insulin

**IV acces**



**Reguler Ins.**



**0.1 U/Kg bolus**



**0.1 U/Kg/h inf.**



**SC/IM**



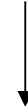
**Reguler Ins.**



**0.4 U/Kg**



**0.1 U/Kg/h**



**If serum glucose level doesn't decrease 50-70 mg/dL in the first hour, begin double insulin /hour**

# Treatment

↓ Serum Glucose to 250 mg/dL,  
**Insulin** 0.05-0.1 U/Kg/hr infusion

or

5-10 U sc/2 hr

**%5 Dextrose** 150-250 mL/hr

(**Iatrogenic hypoglycemia!!!!!!**)

Keep serum glucose between 150-200 mg/dL  
untill maintenance of metabolic control

# Treatment

- By an effective treatment we expect to correct acidosis in 2-3 hours, ketone bodies in 4-6 hours
- No insulin preparation is used other than regular insulin
- American Diabetes Association (ADA) may recommend IM Insulin treatment, but absorption problem can be seen due to peripheral collapse

# Treatment-Potassium

**Total K deficiency: 40-100 meq/L**

If  $K^+ > 6$  meq/L, don't give

If patient is oliguric, don't give

If  $K^+$  5-6 meq/L, give 10 meq/hr

If  $K^+$  3-5 meq/L, give 20 meq/hr

If  $K^+ < 3$  meq/L, give 30 meq/hr

In the first 36 hours, totally 300 meq/hr  $K^+$  is enough

**Maximum infusion rate 40 meq/L/hr**



# Treatment-Bicarbonate

-If  $\text{pH} < 6.9$

88 mEq/0.5-1 hr  $\text{NaHCO}_3$  infusion

-If  $\text{pH} = 6.9-7.0$

44 mEq/0.5-1 hr  $\text{NaHCO}_3$  infusion

-If  $\text{pH} > 7.0$

$\text{HCO}_3^-$  - Don't give!

-Untill  $\text{pH}$  becoming  $> 7.0$  give  $\text{HCO}_3^-$  every 2 hour

-Monitorize serum  $\text{K}^+$

**\*Be alert for brain edema in children!**

# Complications

- Hypoglycemia
- Hypokalemia
- Hyperchloremia (Temporary)
- Cerebral edema (Children)

# Consultation

- DKA patients must be hospitalized
- Close monitorization of the treatment
- Intensive care unit

# Mortality

- Before 1922 (Invention of insulin): %100
- Today: %2



**Thank You**