Endocrine Emergencies

Diabetic Ketoacidosis and Hyperosmolar non-ketotic hyperglycemia

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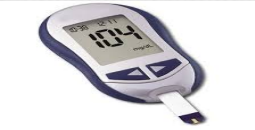
no need to memorize the normal ranges of the electrolyte
Na: 136-146
K: 3.5-5.3

18 years old diabetic patient was found to be in coma

- What questions need to be asked ?
- Differentiating hypo from hyperglycemia ?
- Symptoms suggestive of infection / is the patient taking insulin
- Other symptoms (abdominal pain) . Signs.
- Clinical and lab.
- Easiest way to make diagnosis .
- Check urine for ketones and GLUCOCHECK for blood glucose

Suggest DKA, more common in young than adults, no body know why it happens



- History:
- Is she taking insulin regularly?
- Did she have her meal? Hypoglycemia
- History of infection? Infection doesn't have to be so severe, only URTI or cold can trigger DKA
- Physical signs of DKA:
- Acetone smell
- Dehydrated, reduced skin turgor, hypotension, postural hypotension, dry axilla, in very young children: sunken eyes
- During hypoglycemia, skin is wet, increased discharge of the parasympathetic tone, they get shivering, diaphoresis, hunger
- When blood sugar is extremely high, the Glucometer can't read the result, it will show ?600+, also if very low, it will show "very low".
- Urine dipstick in DKA, 4+ acetone, 4+ glucose
- Ketone bodies are acetoacetic acid, beta-hydroxybuteric acid and …

Lab. Results

- Serum glucose
- ABG
- Urea and electrolytes
- CBC
- ECG
- CXR
- Cultures Because infection can be a trigger for DKA
- Urine

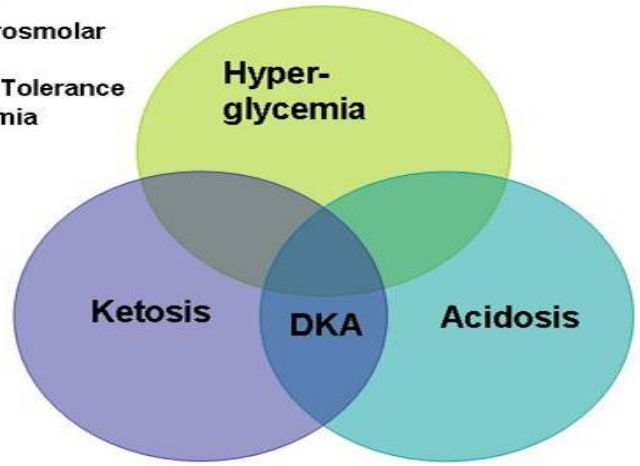
DIABETIC KETOACIDOSIS

- **Criteria :** 4 of them has to be present to diagnose DKA
- Blood sugar above 14 mmol/l
- Arterial Ph below 7.3
- Bicarbonate concentration below 15 mEq/l.
- Presence of ketonurea or ketonemia.

Metbolic acidosis types:
High anion gap >12, eg: lactic acidosis, in shock
Normal anion gap <12, eg: normal anion gap, in renal tubular acidosis
How to calculate anion gap? Cationa (Na) – Aniona (Cl+HCO3)

Diabetic Ketoacidosis

Other Hyperglycemic States
Diabetes Mellitus
Non-Ketotic Hyperosmolar Coma
Impaired Glucose Tolerance
Stress Hypoglycemia



Other Ketotic States:
Ketotic Hypoglycemia
Alcoholic Ketosis
Starvation Ketosis

Other Metabolic Acidotic States
Lactic Acidosis
Hyperchloremic Acidosis
Salicysm Aspirin toxicity
Uremic Acidosis
Drug-Induced Acidosis

High anion gap

DKA (precipitating factors)

- 1. Infection 30%
- 2. Stopping insulin 30%
- 3. First presentation of type 1 DM 30%
- 4. No obvious cause : psychological factors 10%

Stress releases hormones that oppose the action of insulin such as: growth hormone, glucagon, cortisol

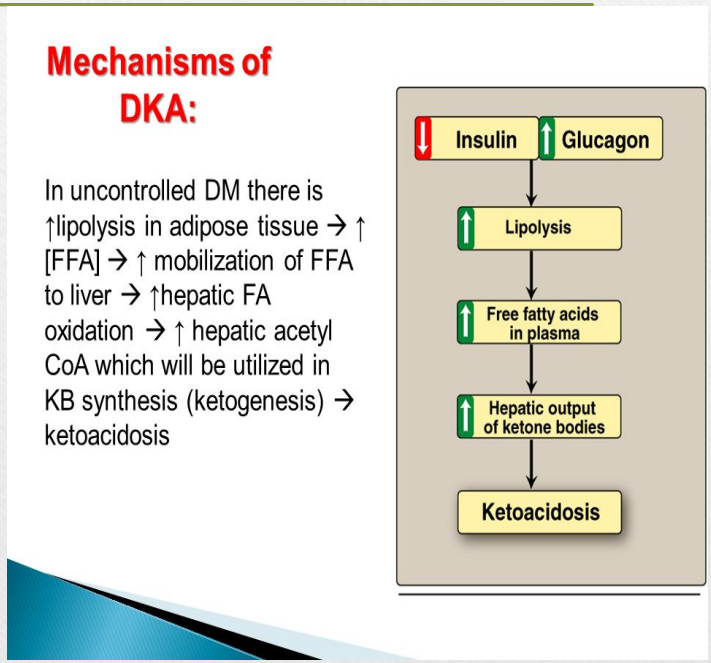
Pathogenesis

1.Ketogenesis:

When there's no insulin, body can't utilize the glucose

Due to insulin deficiency & increased concentration of counter regulatory hormones esp. epinephrine

TG → **FFA** → LIVER → **KETONE BODIES .**



2.Hyperglycemia :

A : gluconeogenesis

B: accelerated glycogenlysis

C : impaired glucose utilization

Diabetic Ketoacidosis

Kussmal breathing, airhunger or hyperventilation. Cuz there is metabolic acidosis and the patient trys to correct it with respiratory alkalosis, by hyperventilating to wash the CO2 out.

- **Any type 1 diabetic patient with nausea , vomiting , abdominal pain , CNS depression , shortness of breath ,fever , signs of infection is a candidate for DKA.**
- **Check urine for ketones. Check blood glucose by meter.**
- **Look for signs of dehydration : skin turger , hypotension, tachycardia , Kussmaul breathing.**
- **Acetone smell.**
- **Send blood for : glucose , urea and electrolytes CBC,ABG , ECG , CXR .**

LAB.

- Glucose :> 17 mmol/l
- PH < 7.3
- Bicarbonate : < 15 mEq / l
- Ketonemia and ketonurea
- High anion gap (Na – Cl + bicarb) > 14 m Eq / l

TREATMENT

If we get a patient with hyperkalemia, K=6.9, it can lead to cardiac arrest.
One of the ways to reduce K level is by injecting insulin and infusing glucose at the same time, 50% glucose and 5 units on insulin.
Both will shift K form extracellular to intracellular compartment.
So in managing DKA we have to check K level and correct it when it goes down

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- Admit patient to ICU. Cuz we need to monitor the patient each hour for glucose level And every 2 hours for K level
- Monitor the following : blood (finger stick) or plasma glucose every 1-2 hours.
- Plasma K every 2-4 hours (important) .
- Other electrolytes every 4 hours.
- ABG as needed until PH is >7.1
- Plasma phosphate , Mg , Ca , on admission : if low repeat every 4 hours.
- Urine for ketones every voiding.

TREATMENT

MONITORING

USE A FLOW SHEET

ESSENTIAL ELEMENTS OF TREATMENT

- 1. Insulin : 0.1 u /kg , followed by iv infusion of regular insulin If she is 60 KG, we give her 6 units as bolus, then we infuse her with another 6 units every hour.

0.1u/kg/hr. Plasma glucose should fall by 4 – 5.5

mmol / l every hour. If no response by 4 hrs double

the dose.

Fluids in Treatment Of DKA

If you give the patient fluid more than required, the patient will go into cerebral edema

- Start with normal saline (15-20 ml /kg) first 2 hours :

  1 litre in first hour

  1 litre in second hour

Then assess : if patient was initially hypotensive , give a third litre in the next 2 hours. DON'T EXCEED MORE THAN 50 ML/KG IN THE FIRST 4 HOURS.

When blood glucose reaches 14 mmol , give 0.45 % saline infusion + 5 % glucose to run at 150 – 300 ml / hour .

To prevent the patient from going to hypoglycemia and cuz lowering glucose quickly, can cause cerebral edema.

Potassium

Before managing DKA, the K level might be normal, but once you give a touch in insulin it will drop

- Always deficient (UP TO 200 meq)
- Initial level could be high because of acidosis
- Replace as KCL & 1/3 as KPO4
- Usually 20 – 30 meq /hr is needed . Replace if K+ if
- LESS THAN 5.3 Meq/l
- ECG monitoring

BICARBONATE

- **Bicarbonate : only if PH IS ≤ 6.9** OR BICARBONATE IS < 5 .

MCQ

WHY ?

1. WORSENING OF HYPOKALEMIA

2. PARADOXICAL CNS ACIDOSIS

Give one ampoule of 7.5 % sod bicarb. (50 mmol) + 250 ML sterile water . Add 15 meq of K CL for each ampoule (if K is ≤ 5.5 meq/l) .

3 loss of drive for hyperventilation causing high PCO2 AND CEREBRAL ACIDEMIA

Aspirin causes of cerebral edema in DKA:
High fluid
High HCO3
Lowering glucose too rapidly

Criteria For Resolution

- Blood glucose < 200 mg /dl (11.1 mmol
- Serum bicarbonate > 18 meq /l
- PH > 7.3
- Calculated anion gap < 12

Starting Subcutaneous Insulin

- When patient is able to eat
- Allow overlap between insulin infusion and subcutaneous insulin
- If patient is newly diagnosed , the initial total insulin dose should be 0.6 u/kg/day.

Complications Of Therapy

1.hypoglycemia

2. Hypokalemia

3.Cerebral edema : occurs in pediatric patients . May occur when blood sugar drops quickly to <14 mmol/l

4. ARDS : rare

PREVENTION

- 1. EDUCATION .

  Never ever stop the insulin, if the child does't want to eat, at least give him snack like crakcers and so

- 2. SICK DAYS MANAGEMENT :

  HYDRATION

  TREATMENT OF INFECTION

  MONITORING FOR GLUCOSE & KETONES

  USE OF SHORT ACTING INSULIN

HYPERGLYCEMIC HYPEROSMOLAR STATE

Different from DKA by absence of ketosis and presence of higher plasma glucose. Glucose is usually > 33 mmol and osmolality > 320 mosM.

Patient is typically a type 2 DM Elderly, pH is not acidic

SERUM PH MORE THAN 7.3

SERUM BICARBONATE IS HIGHER THAN 18

Pathogenesis

- Hyperglycemia
  Very high eg; 900, 1000 ... Etc.
  osmolality will be high too >300
  Normal osmolality 285
- **Ketogenesis : not operating (some insulin is still available)**
  So there is no acidemia
- Dehydration is more severe
  Renal threshold in blood glucose: 200 mg/dl
  So if blood glucose is above 200, glucose will appear in urine
  But in this case the elderly patient is usually dehydrated, and is accumulating glucose in blood
  Cuz there is decreased urine production due to dehydration
- Hyperosmolar state

DKA vs HHS

Diagnostic Criteria	
• Hyperglycemia : Blood glucose ≥14 mmol/L • Acidosis : pH < 7.3, HCO_3 <15 mmol/L • Ketonaemia or ketonuria	• Plasma glucose level of ≥ 33 mmol/L • Arterial pH > 7.3, serum bicarbonate > 15 mmol/L • Absence of severe ketonaemia or ketonuria • Serum total osmolality >330 mmol/L

Management

- **Fluids : 0.9% saline in first hour and 2nd hour . Then give 0.45% saline at about 500 ml / hr or less . Watch cardiac status carefully esp. in cardiac patients. Add 5% glucose when blood glucose reaches 14 - 16 mmol /1 .**

Management is the same in DKA and HHS with 2 exception:
1- be careful with fluid because patients in HHS are elderly with heart diseases and fragile
2- very sensitive to insulin in HHS

Mortality is higher in HHS than DKA

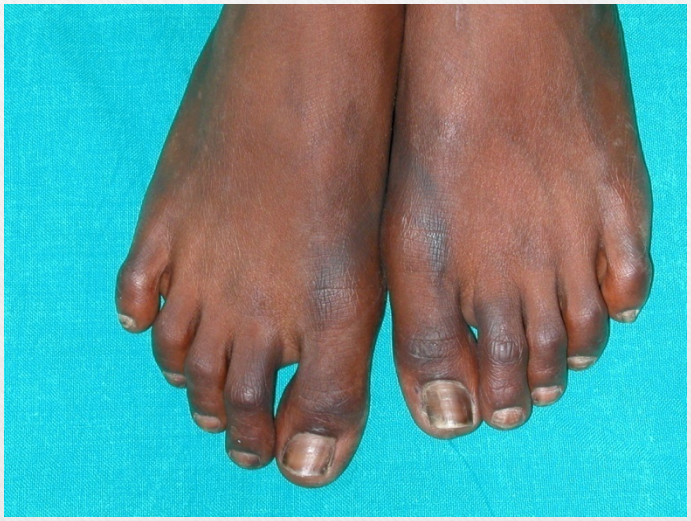
Insulin

- **5 – 10 units regular insulin bolus .**
- **0.1 u/kg /hr infusion**
- **When blood glucose reaches 14 – 16 mmol/l give 1 - 2 u /hr + saline / glucose infusion .**

CASE

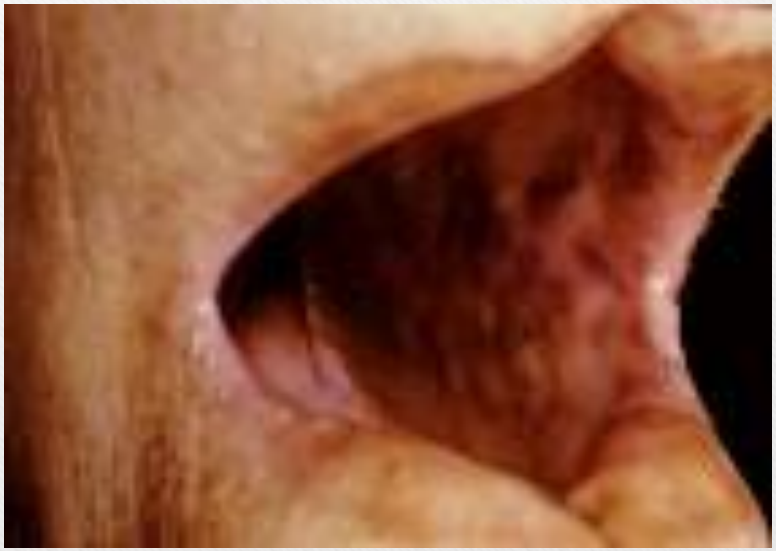
45 YEARS OLD Saudi lady

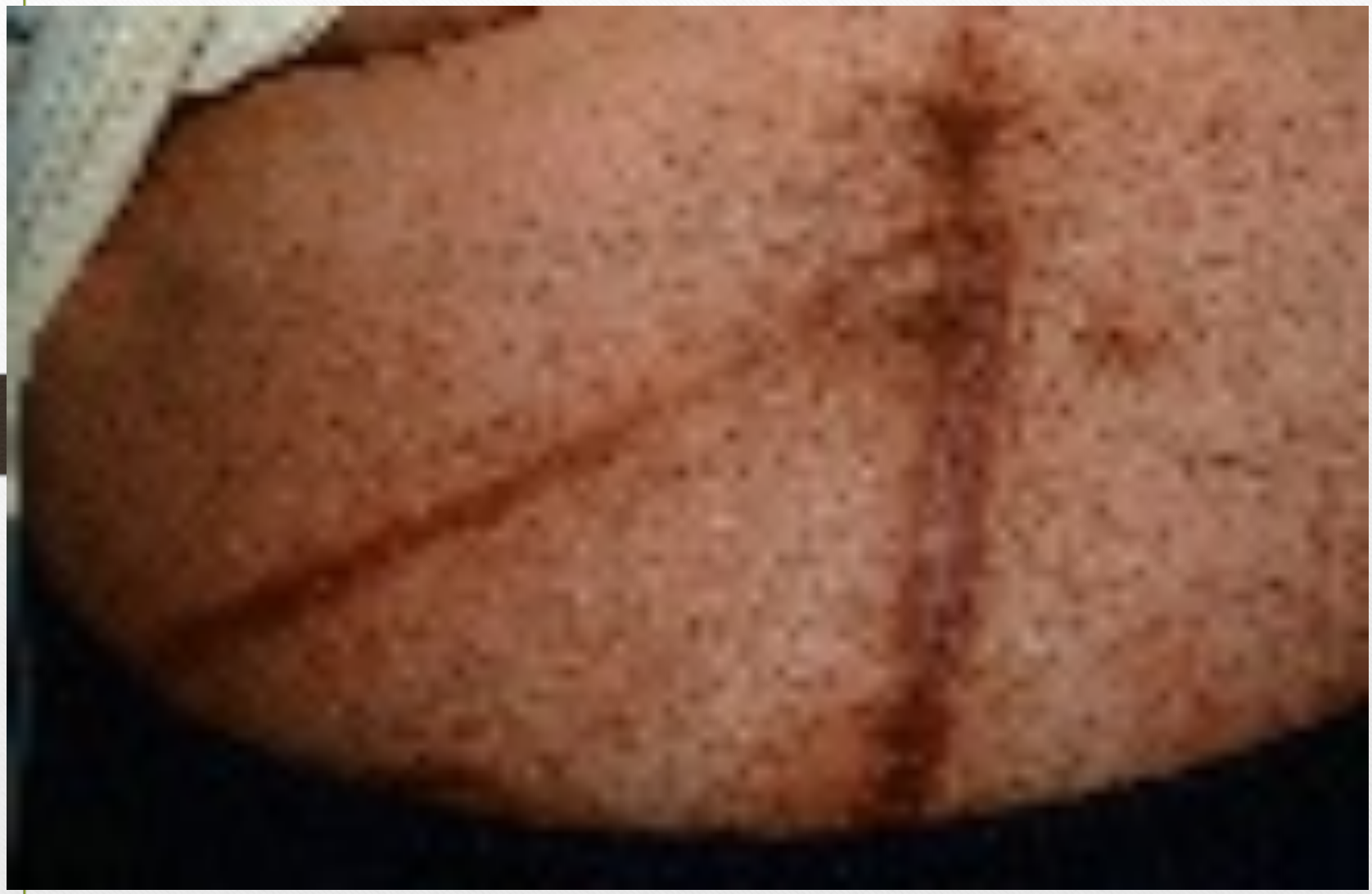
- **Fatigue**
- **Tiredness**
- **Increased skin pigmentation**



proopiomelanocortin, a prohormone that is cleaved into the biologically active hormones ACTH, MSH, and others

Hyperpigmentation is seen in buccal mucosa, areola, knuckles, new scars, axilla, perineum





Typical electrolytes

Electrolyte can come in OSCE
Normal levels will be provided

- Na 126
- K 5.5
- Cl 89
- Bicarb 20

Hyponatremia and hyperkalemia due to loss of aldosterone

HYPONATREMIA AND HYPERKALEMIA ARE NOT SEEN IN SECONDARY HYPOADRENALISM (pituitary problem)

Because aldosterone is not under the effect of ACTH, it's under control of RAAS and K
ACTH controls zona fasciculata (gives cortisone) and zona reticularis (gives androgen)
Loss of androgen in females present as loss of pubic and axillary hair.

What about glucose ! ?

Causes of fasting hypoglycemia

- EX
- P
- L
- A
- I
- N

Fasting Hypoglycemia

- Exogenous insulin or OHA
- Pituitary dis
- Liver dis gluconeogenesis
- Addison's dis
- Insulinoma
- Neoplasm

CBC

Classic finding in Addison

- NEUTROPENIA
- RELATIVE LYMPHOCYTOSIS
- EOSINOPHILIA

TREATMENT

- IV SALINE +GLUCOSE
- IV STEROIDS Measure cortisone level before giving steroids

PREVENTION

- Extra steroids at times of stress And infections and surgeries
- Double dose for URTI
- Higher doses for more severe illnesses

35 years old lady with hyperthyroidism (post surgery) , developed the following :

- **Fever : 40c**
- **Agitation**
- **Tachycardia : 140 /min**
- Upon taking further history , the patient was NOT euthyroid before surgery

Never ever expose a hyperthyroid patient to surgery unless the patient is euthyroid
Because under severe stress the thyroid will release even more extra hormone and the patient will develop thyroid storm

MANAGEMENT OF THYROID STORM

- IODIDE Big amounts will stop the release of thyroid hormone from the gland
- ANTI THYROID DRUGS
- BETA BLOCKERS
- STEROIDS
- ANTI PYRETICS
- SUPPORTIVE CARE

CASE

81 YEARS OLD LADY

- FOUND IN COMA
- TEMPERATURE : VERY LOW
- PULSE : 45 /MIN

OTHER CLINICAL FINDINGS THAT COULD BE FOUND IN MYXEDEMA COME

- **Dry skin**
- **Loss of eyebrows**
- **Big tongue**
- **Look for a thyroidectomy scar**

DO NOT FORGET BREATHING

- HYPOVENTILATION IS A BIG PROBLEM
- TYPICAL ABG'S :

PCO2 : HIGH

PO2 : LOW

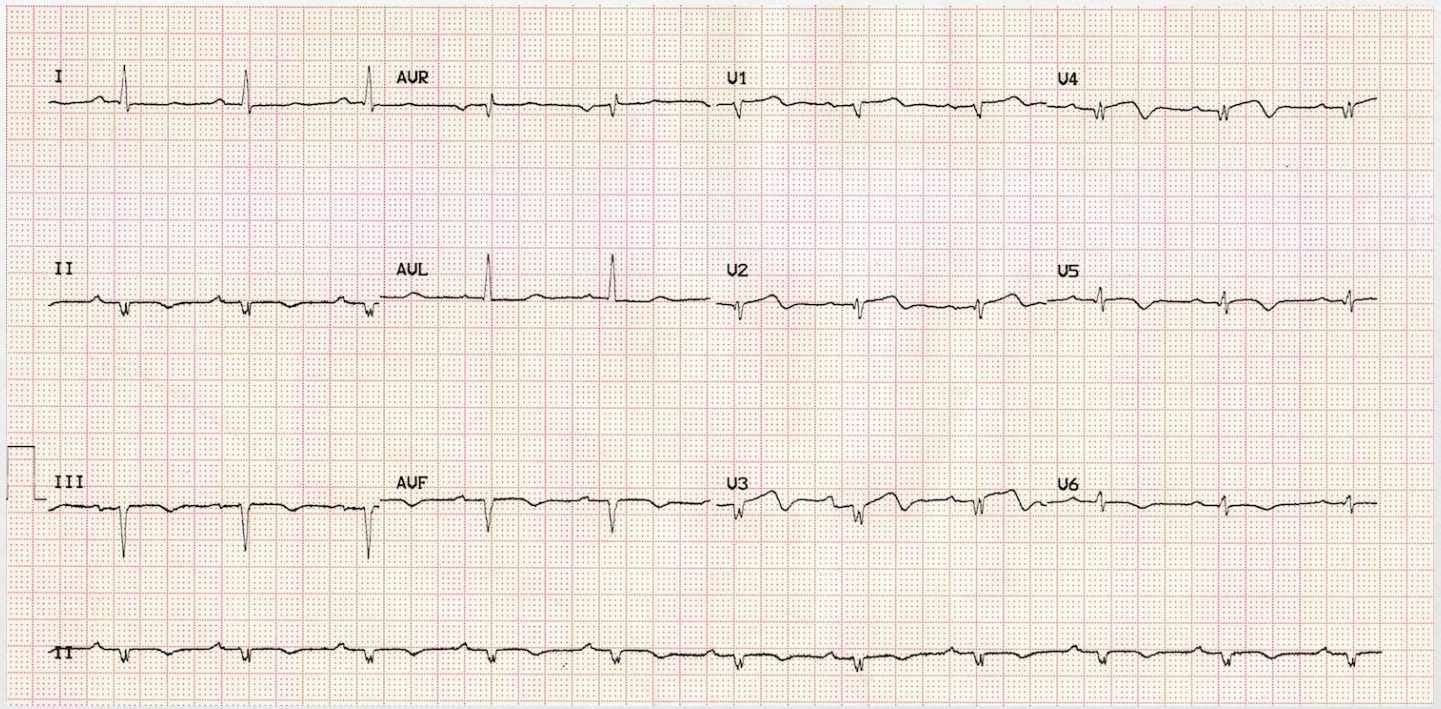
PATIENTS MAY NEED ASSISSTED VENTILATION

respiratory failure
type 1: hypoxia normal CO_2
type 2: hypoxia and hypercapnia

sodium

- Look for hyponatremia
- High cholesterol
- High CPK From muscles and the heart

ECG : LOW VOLTAGE & BRADYCARDIA



TREATMENT OF MYXEDEMA COMA

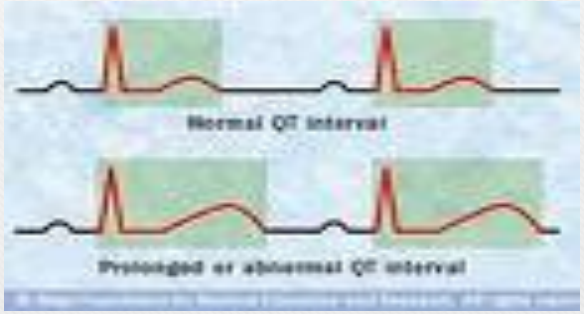
- VENTILATION
- IV THYROXINE
- IV STEROIDS Because we are giving thyroxine, the metabolic rate will increase, so we aloso give steroid to follow the sudden increase in metabolic rate
- WARM PATIENT

SEVERE HYPOCALCEMIA

Post-thyroidectomy and post parathyroidectomy

- TETANY
  Adduction of the thumb , hyperextension of the fingers
- CONVULSIONS ESP. IN THE YOUNG
- ECG CHANGES (QT INTERVAL)
- **Trousseau's sign** Inflating the cuff above the systolic blood pressure will result in carpopedal spasm
- **Chvostek's sign** Upon tapping over the zygomatic arch

PROLONGED QT INTERVAL



TREATMENT OF SEVERE HYPOCALCEMIA

- IV CALCIUM GLUCONATE SLOWLY AND UNDER ECG MONITORING

Things that are never given as bolus:
Calcium
Potassium

SEVERE HYPERCALCEMIA

- A 60 years old man presented with confusion. Serum calcium found to be 3.7 mmol/l (N 2.1-2.5)

  Sever hypercalcemia when >3.5 mmol/L

Causes of Hypercalcemia:
Chronic renal failure through tertiary hyperparathyroidism
Primary hyperparathyroidism
Thiazide diuretic
Vitamin D intoxication
Squamous cell carcinoma
Breast cancer
Multiple myeloma
Sarcoidosis through granuloma production of 1,25-dihydroxy VitD

Causes of hypercalcemia can be categorized as parathyroid related and parathyroid unrelated, if we find the parathyroid hormone is high in blood then it's due to parathyroid related cause and vice versa

Causes of severe hypercalcemia

- Primary hyperparathyroidism
- Malignancy induced hypercalcemia
- Iatrogenic (high doses of calcium and vitamin D)

Treatment of severe hypercalcemia

- **Saline hydration** To induce clciuresis, we flush the calcium in urine
- **Calcitonin** has an anti-parathyroid effect
- **Bisphosphonates** inhibit osteoclastic activity
- **Steroids : only useful in sarcoidosis , multiple myeloma and lymphoma**
- **Dialysis : in renal failure or CHF**