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Diabetic Ketoacidosis (DKA)

Diabetic emergencies (Acute complications)

1. Diabetic Ketoacidosis (DKA)
2. Hyperosmolar hyperglycaemic state (HHS)= Hypperosmolar non-ketotic acidosis (HONK)
3. Hypoglycemia

} Hyperglycemia

Ketone Bodies

1. Acetone: not metabolically active, contributes to the smell of acetone in patients with DKA so help in diagnosis only
2. Acetoacetate : metabolically active
3. β-Hydroxybutyrate : metabolically active

} They are acid in nature so can decrease PH (normal range: 7.35-7.45)

- They are produced by the liver (ketogenesis) and utilized for energy production by peripheral tissues (Ketolysis) for example the brain in prolong starvation
- Because the liver lacks enzymes that can degrade ketones , it can't use the Ketone bodies to produce energy

1st Diabetic Ketoacidosis (DKA):

✓ **Triad of symptoms:**

1- Hyperglycemia
2- High anion gap metabolic acidosis (anion gap : gap b/w anion cl, HCO3 & cation K, Na in mmol)
3- Ketonemia
(it's difficult to have urine simple to find this pt. has ketonuria either b/c he maybe comatose or in semiconscious and dehydrated)

✓ Characteristically associated with T1DM !!
✓ It has become increasingly common in T2DM
✓ DKA may be the first presentation of T1DM!! like the case=)

Precipitating factors for DKA

1. Infection (30-40%) e.g. sore throat ,UTI
2. Inadequate insulin treatment or non-compliance (20%)
3. Severe illness e.g., Myocardial infarction → Hypoxia → depend on anaerobic → lactic acid production ↑ → acidosis → more stress on diabetic body
4. Trauma →due to high Anti-insulin
5. Drugs: e.g., steroids

2nd Hyperosmolar hyperglycaemic state (HHS)= Hypperosmolar non-ketotic acidosis (HONK):

- This is a state characterized by the presence of hyperosmolar blood due to high blood sugar with insufficient levels of insulin to uptake glucose inside the cells & can't inhibit gluconogenesis & glycogenlysis BUT the amount is just enough to prevent lipolysis and ketogenesis, which means there will be little or no ketone bodies in the blood

- ✓ Little or no accumulation of ketone bodies **!!**
- ✓ Serum [glucose] is often >50 mmol/L
- ✓ Plasma osmolality may reach 380 mosmol/Kg (normal 275-295)
  -Due to hyperglycemia (osmotic pressure of glucose→ increase blood osmolality)
  -Hyperosmolality cause cell dehydration &shrink
- ✓ Neurological abnormalities are frequently present
  -Because of disturbance in electrolyte balance due to increase K level – remember : the insulin help the cell to uptake the K =)
- ✓ Usually occurs in elderly patients with T2DM
  - Why in type 2? Because in type 1 there is no insulin
- ✓ Has a substantially higher mortality than DKA (up to 15%)

  -Because usually HHS occur in old pt has uncontrolled diabetes, diabetic neuropathy, nephropathy, retinopathy, microvascular disease, heart disease So has multi organ impairment.

3rd Hypoglycemia:

- ✓ Common complication of treatment with insulin or oral hypoglycaemics**!!**

  - Diabetic patients do not have the capacity of maintaining normal blood levels as you know because insulin is not functioning well and as a result of that glucagon does not work well to keep the blood sugar level in normal range; so a high dose of insulin will cause hypoglycemia.
  - This can happen either by having high dose of TTT or doing excessive exercise, or not to eat after taking the dose.

✓ More common in patients with T1DM

-Because these patients take insulin as treatment in daily basic

✓ Due to impaired protective responses to hypoglycemia:

- *Insulin is supplied exogenously and its release cannot be turned off*

- so no regulatory mechanism like in normal situation hyperglycemia → ↑insulin ,

if hypoglycemia → ↑glucagon & ↓insulin

- *Glucagon & adrenaline* (anti insulin) *response to hypoglycemia becomes impaired later in the course of DM*

-glucagon impaired due to the auto immune destruction of pancreas with time the whole of pancreas will effected (alph cell)

- peripheral neuropathy cause Adrenaline impairment.

✓ Clinical presentation:

- **Symptoms of sympathetic overactivity** *(when plasma [glucose] is <3.6 mmol/L, **abrupt fall!!**) with anxiety, tremors, sweating and palpitation*

-Hypoglycemia stimulate sympathetic system =)

- **Symptoms of neuroglycopenia** *(when plasma [glucose] is <2.6 mmol/L, **gradual fall!!**) with headache, confusion, drowziness*
- *ultimately loss of consciousness or seizures (when plasma [glucose] is <1.5 mmol/L)*

A CASE of DKA!!

A 14-year-old girl was admitted to a children's hospital in coma. Her mother stated that the girl had been in good health until approximately 2 weeks previously, when she developed a sore throat and moderate fever. She subsequently lost her appetite and generally did not feel well. Several days before admission she began to complain of undue thirst and also started to get up several times during the night to urinate. However, on the day of admission the girl had started to vomit, had become drowsy and difficult to arouse, and accordingly had been brought to the emergency department.

Hints:

-Young age(T1DM)

-Infection (precipitating factor)

-Polyuria, nocturia , thirst(polydipsia)

-Vomiting due to high level of keton bodies(ketoacidosis) which stimulate chemoreceptor

- coma

On examination:

- She was dehydrated
- Her skin was cold
- She was breathing in a deep sighing manner (Kussmaul respiration)
  → respiratory compensation to get more oxygen inside the body to buffer the acidosis
- Her breath had a fruity odor. → due to the acetone accumulation in the blood
- Her blood pressure was 90/60 mmHg (N: 120/80)
- Her pulse rate 115/min. → this is reflex tachycardia (due to hypovolemia)
- She could not be aroused

-A provisional diagnosis of T1DM with complicating ketoacidosis and coma (DKA) was made by the intern on duty.

-The admitting diagnosis was confirmed by the laboratory findings

Laboratory findings: blood results

Plasma analyte	Patient's results	Normal levels
Glucose (mmol/L)	50	4.2-6.1
Ketoacids	++++ *Due to ketoacidosis*	(trace)
Bicarbonate (mmol/L)	6 * Low because its being used to try and compensate for the acidity of the blood*	22-30
Arterial blood pH	7.07 * Acidosis Because of the ketone bodies*	7.35-7.45
Na^+ (mmol/L)	136 N	136-146
Cl^- (mmol/L)	100 N	102-109
PCO_2 (kPa)	2.7 *Due to deep breaths* Hyperventilation* increasing the oxygen level in the body * respiratory alkalosis to compensate metabolic acidosis*(mechanism of compensation)	4.3-6.0
***Anion gap (mmol/L)**	35.5* High anion gap indicates acidosis*	7-16
K^+ (mmol/L)	5.5 * no insulin so the K can't enter cell &normally K enter cell when H+ come out from cell but H+ is high in blood so can't go out*	3.5-5.0
Urea nitrogen (mmol/L)	15 (it increase → sign of Dehydration) renal impaired caused by Hypovolumia & hypotension decrease renal profusion	2.5-7.1
Creatinine (µmol/L)	200 (increased due to impaired renal function → lead to increase GFR)	44-80
Albumin (g/L)	50	41-53
Osmolality (mOsm/kg serum water)	325	275-295
Hematocrit	0.500* due to dehydration(Hypovolumia) hemoconcentration	0.354-0.444

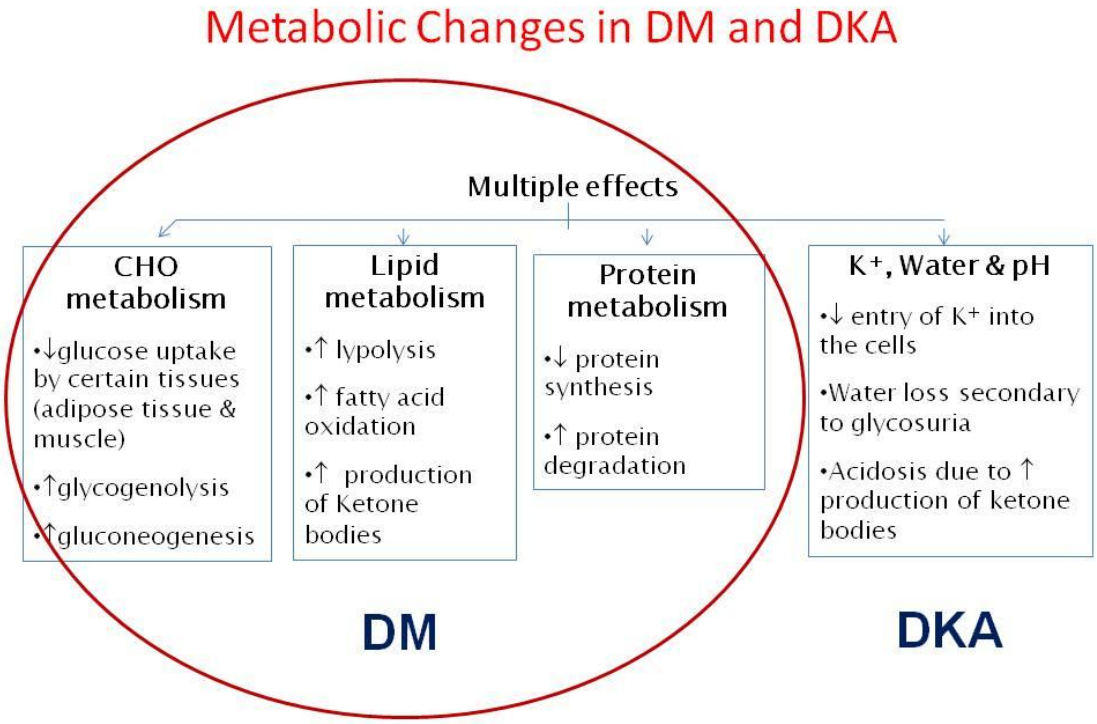
***Anion gap (A^-)= (Na^+ + K^+)– (HCO_3^- + Cl^-)**

Urine results:

Urine analyte	Patient's results	Normal level
Glucose	++++	-
Ketoacids	++++	-

Interpretation of Laboratory findings:

Results	Interpretation
Hyperglycemia	Confirm the diagnosis of DKA
Glucosuria	
Ketonemia	
Ketonuria	
↓ pH	Severe metabolic acidosis due to ↑ production of ketone bodies
↓ bicarbonate and PCO_2	Metabolic acidosis with partial respiratory compensation (the hyperventilation)
↑ anion gap	Due to ↑ ketone bodies in the blood
↑ urea & creatinine	1- Renal impairment (dehydration → ↓ blood volume →↓ renal perfusion) 2- Dehydration 3- Degradation of protein (for urea) →No insulin→↑protein catabolism
↑K^+	↓ Uptake of potassium by cells in the absence of insulin
↑ Plasma osmolality	Due to hyperglycemia and fluid loss



Quick Revision

- DKA Usually associated with which type of diabetes?

  Type 1 But can come in T2DM

- -Hyperglycemia, Ketonemia, High anion gap trid of which Acute complications of DM?

  DKA

- Ketogenesis acquired in liver But Ketolysis acquired in peripheral tissue why?-

  Because the liver lacks enzymes that can degrade ketones , it can't use the Keton bodies to produce energy

- -The main characteristics of HHS are?

  1- no accumulation of keton bodies 2- Hyperglycemia 3-hyperosomality 4-Neurological symptoms 5- Old pt 6- T2DM 7- high mortality than DKA

- -Hypoglycemia commonly caused by?

  As complication of treatment with insulin or oral hypoglycaemics.

- When the pt. with hypoglycemia will develop Symptoms of sympathetic overactivity?

  Glucose <3.6 mmol/L, abrupt fall!!

- When the pt. with hypoglycemia will develop Symptoms of neuroglycopenia?

  Glucose <2.6 mmol/L, gradual fall!!

- When the pt. with hypoglycemia will loss of consciousness or has seizures?

  Glucose <1.5 mmol/L)

- Hypoglycemia usually associated with which type of Diabetes ?

  T1DM because these patients take insulin as treatment in daily basic

- -The most common Precipitating factors for DKA is?

  Infections

- - bicarbonate and PCO2 in DKA decrease why?

  Metabolic acidosis with partial respiratory compensation

- Hyperkelmeia in DKA due to?

  ↓ Uptake of potassium by cells in the absence of insulin

- ↑ urea & creatinine In DKA due to?

  1- Renal impairment (dehydration → ↓ blood volume →↓ renal perfusion)
  2- Dehydration
  3- Degradation of protein (for urea)

  Metabolic changes In DKA?

  K+ decrease entering to cell , Water loss due to glycosuria ,Acidosis due to high Ketone bodies.

- The main difference B/w DKA & HHS?

  - DKA mainly acquired in T1DM but can happen in T2DM ,, HHS with T2DM
  - Ketonemia & ketonuria seen in DKA,, HHS little or no accumulation of ketone bodies
  - DKA may be the first presentation of T1DM