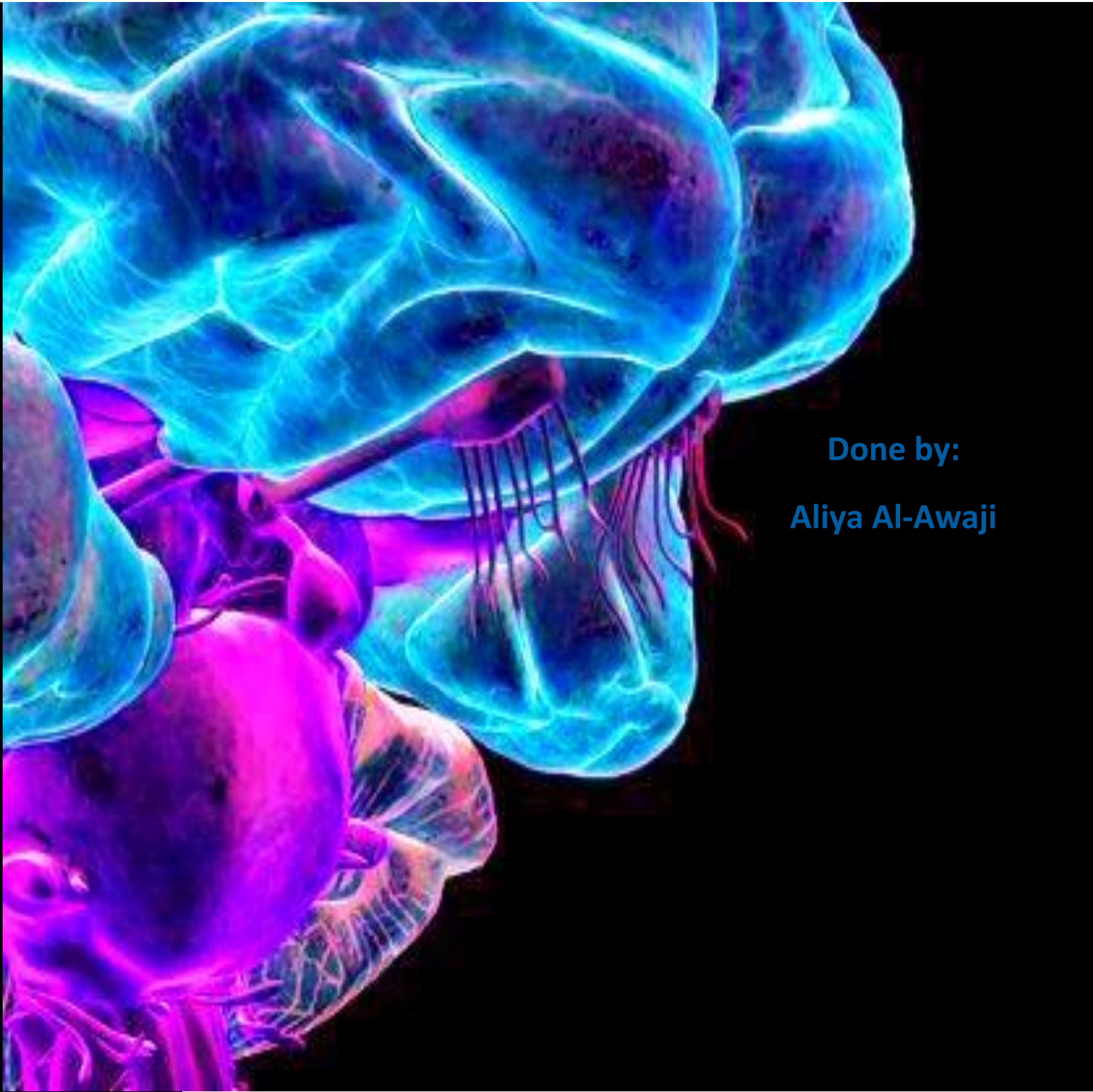


Diabetic Ketoacidosis (DKA)

Done by:

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Diabetic emergencies: any significant increase or decrease in blood glucose level considered an emergency.

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

- The first two conditions are types of Hyperglycemia.

A. Diabetic Ketoacidosis (DKA):

- Triad of:

1. Hyperglycemia
2. High anion gap metabolic acidosis

The **anion gap** is the difference in the measured **cations** and the measured **anions** in **serum, plasma, or urine**. The magnitude of this difference (**i.e. "gap"**) in the serum is often calculated in medicine when attempting to identify the cause of **metabolic acidosis**.

3. Ketonemia = ketone bodies in the blood

- Characteristically associated with T1DM (type 1 diabetes mellitus)
- It has become increasingly common in T2DM (type 2 diabetes mellitus)
- DKA may be the first presentation of T1DM.

Ketone Bodies:

1. Acetone → neutral in nature (i.e. doesn't change pH level). We can detect it in the breath of diabetic patients.
2. Acetoacetate → acidic in nature.
3. β -Hydroxybutyrate → acidic in nature.

Has a metabolic function, hence detectable in the urine

- They are produced by the liver (ketogenesis) and utilized for energy production by peripheral tissues (Ketolysis).

B. Hyperosmolar hyperglycaemic state (HHS) = Hyperosmolar non-ketotic acidosis (HONK): usually affect T2DM and elderly patients. And it's difficult to control.

- a. Little or no accumulation of ketone bodies

- b. Serum [glucose] is often >50 mmol/L (normally it's from 4.4 to 6.1 mmol/L).
- c. Plasma osmolality may reach 380 mosmol/Kg (normal 275-295)
- d. Neurological abnormalities are frequently present
- e. Insulin levels are insufficient (but not absent like in DKA) to allow appropriate glucose utilization but are adequate to prevent lipolysis and subsequent ketogenesis.
- f. Usually occurs in elderly patients with T2DM
- g. Has a substantially higher mortality than DKA (up to 15%)

C. Hypoglycemia:

- a. Common complication of treatment with insulin or oral hypoglycaemics or even changing lifestyle without dosage adjustment (e.g. exercising).
- b. More common in patients with T1DM (insulin-dependent).
- c. Due to impaired protective responses to hypoglycemia:
 - i. Insulin is supplied exogenously and its release CAN'T be turned off
 - ii. Glucagon & adrenaline response to hypoglycemia becomes impaired later in the course of DM (both α and β cells of the pancreas will be defected).
- d. Clinical presentation:
 - i. Symptoms of sympathetic overactivity (when plasma [glucose] is <3.6 mmol/L, abrupt fall): anxiety, tremors, sweating and palpitation.
 - ii. Symptoms of neuroglycopenia (when plasma [glucose] is <2.6 mmol/L, gradual fall): headache, confusion, drowsiness and ultimately loss of consciousness or seizures or even coma (when plasma [glucose] is <1.5 mmol/L)

Precipitating factors for DKA:

- Infection (30-40%) → like common cold or any viral infection which is considered as a stressful situation that cause the release of stress hormones, which opposes the action of insulin.
- Inadequate insulin treatment or non-compliance (i.e. describes the degree to which a patient not following the medical advice correctly) (20%)
- Severe illness e.g., Myocardial infarction.
- Trauma → type of stressful situation.
- Drugs: e.g., steroids, which is diabetogenic by itself.

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 before, 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.
- On examination:
- She was dehydrated
- Her skin was cold
- She was breathing in a deep sighing manner (Kussmaul respiration)
- Her breath had a fruity odor
- Her blood pressure was 90/60 mmHg (Normal: 120/80)
- Her pulse rate 115/min. → sympathetic overactivity
- She could not be aroused
- A provisional diagnosis of T1DM with complicating ketoacidosis and coma (DKA) was made by the intern on duty

Laboratory findings: blood results:

The admitting diagnosis was confirmed by the laboratory findings shown below:

Plasma analytes	Patient's results	Normal levels
Glucose (mmol/L)	50 (DKA not HHS)	4.2-6.1
Ketoacids	++++(↑↑↑)	(trace)
Bicarbonate (mmol/L)	6 (acidosis)	22-30
Arterial blood pH	7.07 (↓)	7.35-7.45
Na ⁺ (mmol/L)	136 (normal)	136-146

Cl ⁻ (mmol/L)	100 (lower normal)	102-109
PCO ₂ (kPa)	2.7 (↓)	4.3-6.0
*Anion gap (mmol/L)	35.5 (↑↑) metabolic acidosis	7-16
K ⁺ (mmol/L)	5.5 (↑)	3.5-5.0
Urea nitrogen (mmol/L) for kidney function	15 (↑↑) renal impairment	2.5-7.1
Creatinine (μmol/L) for kidney function	200 (↑↑) renal impairment	44-80
Albumin (g/L)	50 (normal)	41-53
Osmolality (mOsm/kg serum water)	325 (↑↑) due ↑ to glucose	275-295
Hematocrit for RBCs volume	0.500 (↑) due to dehydration	0.354-0.444

*Anion gap (A⁻) = (Na⁺ + K⁺) - (HCO₃⁻ + Cl⁻)

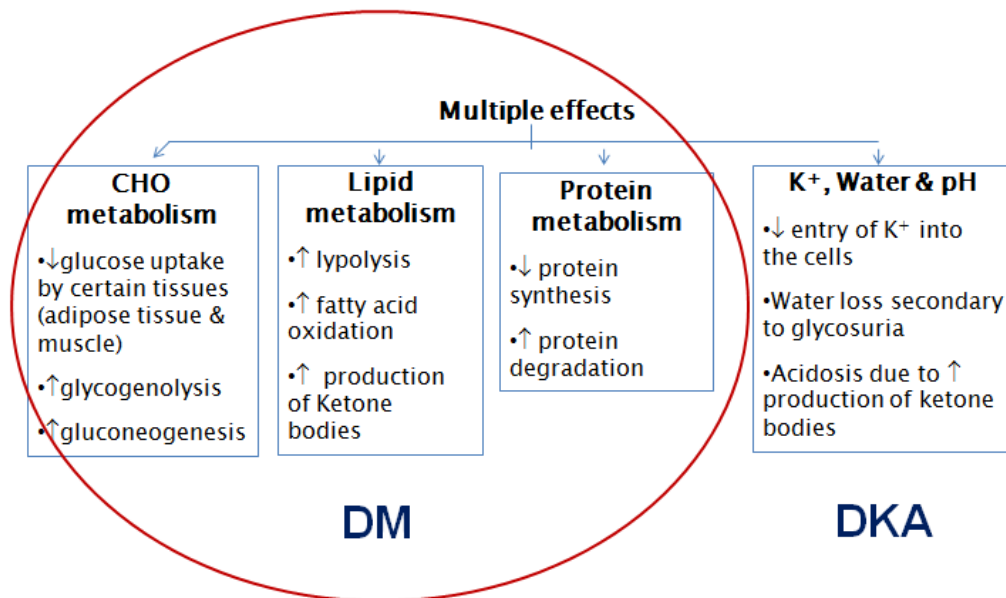
Laboratory findings: Urine results:

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	<ol style="list-style-type: none"> 1. Renal impairment (dehydration → ↓ blood volume → ↓ renal perfusion) 2. Dehydration 3. Degradation of protein (for urea)
↑ K ⁺	↓ Uptake of potassium by cells in the absence of insulin
↑ Plasma osmolality	Due to hyperglycemia and fluid loss

Metabolic Changes in DM and DKA:



Summary:

