ΕΛΛΗΝΙΚΗ ΔΗΜΟΚΡΑΤΙΑ
Εθνικόν και Καποδιστριακόν
Πανεπιστήμιον Αθηνών

Case presentation

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Case Presentation

An 18 year old female with Type 1 diabetes melitus presents with:

- Drowsiness
- Respiratory Distress
- Progressive anorexia with weight loss (~ 6kg)
- Polyuria, Polydipsia since a week ago

- **Has stopped taking her insulin**

Physical Examination

- BP = 100/60 mmHg → 85/40 mmHg while standing
- Pulse = 120 bpm
- Deep, rapid and labored breaths
- RR= 40
- Cold extremities
- Prolonged capillary refill time

Laboratory Data

	Serum Result	Known Previous Values	Reference Range
Hematocrit	**48%**	38%	37-47
Na^+	**128 mEq/L**		135-145
K^+	**5.6 mEq/L**		3.5-5.0
Urea	**111 mg/dL**		10-35
Creatinine	**1.8 mg/dL**	0.8	0.8-1.0
Glucose	**900 mg/dL**		60-100
Cl^-	**89 meq/l**		98-107
pH	**7.1**		7.35-7.45
pCO2	**20 mmHg**		40
HCO_3^-	**7 meq/L**		24
Serum Ketones	**Positive 2+**		Negative

What two acid-base disorders could be responsible for a low PCO_2, if the pH was not known?

1) Metabolic acidosis (secondary respiratory compensation).
2) Respiratory alkalosis.

Based on the data given, which one of these acid-base disorders is most likely, and why?

Metabolic acidosis. Due to:

1. Her blood pH being acidic and
2. her medical history the patient is at high risk for this acid-base disorder.

Is this a single acid-base disorder?

Respiratory compensation for metabolic acidosis is a decrease in PCO_2 of 1.2 mm Hg for every 1.0 meq/l decrease in serum total HCO_3.

ΔHCO_3= 24-7=17 meq/L

Normal respiratory compensation= 17 x 1.2= ~ 20 mm Hg.

Thus, metabolic acidosis with normal respiratory compensation.

Calculate the anion gap. How is the anion gap helpful in metabolic acidosis, and what is its significance in this patient?

AG= Na – (Cl + HCO_3)

AG = 128 – (89 + 7) = 32 mEq/L

The anion gap is helpful in establishing the etiology for metabolic acidosis.

In this patient, the generated ketoacids (acetoacetate and beta hydroxybutyric acid) are buffered by $NaHCO_3$. The increased anion gap is due to the presence of the sodium salts of these acids in the serum.

What is the pathogenesis of diabetic ketoacidosis?

- Insulin deficiency prevents glucose from entering cells and being metabolized.
- In response to the body's need for energy, proteins and lipids are metabolized in excessive quantities.
- This process generates excessive amounts of ketoacids.
- Under normal circumstances, ketoacids are metabolized to H_2O and CO_2 under the influence of insulin.
- In the absence of insulin, ketoacids accumulate in the serum, thus causing metabolic acidosis.

What therapeutic intervention will interrupt the formation of additional ketoacids?

Insulin administration will restore carbohydrate metabolism providing energy that will no longer need to be derived from the metabolism of fat and protein.

After the administration of insulin the acidosis significantly improved without the administration of bicarbonate. Why did this improvement occur?

After the restoration of normal carbohydrate metabolism, the salts of the ketoacids, which had not been lost in the urine, were completely metabolized to bicarbonate.

Thank you!

For Your Attention!