ABR / Questions and case studies

Answer the questions and evaluate the answers by pressing the submit button In the Case Studies section, click on the blue "show" button in the green box and cheerfully check

Questions

1 The concentration of hydrogen ions (H^+) in body fluids is in the order of:

- ☐ A - µmol / l
- ☐ B - nmol / l
- ☐ C - mmol / l
- ☐ D - pmol / l
- ☐ A - 200 mmol
- ☐ B - 40 µmol
- ☐ C - 70 mmol
- ☐ D - 7 mmol / l

2 Serum buffer bases are given by:

- ☐ A - [HCO_3^-] + [proteinate]
- ☐ B - [Cl^-] + [HCO_3^-] + [HPO_4^{2-}] + [lactate]
- ☐ C - [HCO_3^-] + [CO_3^{2-}]

3 Recognition of metabolic acidosis (MAC) in a mixed ABR disorder allows:

- ☐ A - finding increased values of "anion gap"
- ☐ B - reduction of pO_2 in arterial or central venous blood
- ☐ C - low hemoglobin in the blood
- ☐ D - increased lactate in the blood

4 Significant hypoproteinemia with a decrease in residual anions leads to: '

- ☐ A - MAL
- ☐ B - MAC
- ☐ C - RAL + MAC
- ☐ D - MAC + RAC

Submit

Case reports

Patient with scoliosis and heart defect

A 17-year-old patient was admitted to hospital with a congestive heart defect and scoliosis. Upon admission, the laboratory was examined and again 24 hours later.

Power:

- protein 1 g
- pH = 6
- 3-6 hyaline cylinders
- other findings normal

+ Laboratory results

-		*'reception'*	*'in 24 hours'*	-	pH	7.2	7.46	-	pCO_2	14.0 kPa	5.3 kPa	-	HCO_3^-	40 mmol / l	29 mmol / l	-	BE	5.0 mmol / l	5.0 mmol / l	-	pO_2	17.6 kPa	17.6 kPa	-	saturation O_2	97.90%	99%	- Na^+ || 146 mmol / l || 139 mmol / l	-	K^+	5.0 mmol / l	3.3 mmol / l	-	Cl^-	94 mmol / l	96 mmol / l	-

'Questions:'

1. *'What was the ABR failure on reception?'*
2. *'What was the ABR disorder after 24 hours?'*
3. *'What caused the decrease in serum K^+ in 24 hours?'*
4. *'Does the clinical condition match the laboratory findings?'*

Answers -

1. RAC partially compensated (HCO_3^- rise and BE)
2. MAL, apparently chloride-responsive after hypercapnia.
3. Because pH directly affects K^+ movement, a change in pH leads to a change in K^+ in ECT unless K^+ is therapeutically delivered. An increase in pH by 0.1 leads to a decrease in K^+ by about 0.6 mmol / l, ie an increase in pH by 0.26 → a decrease in K^+, by 2.6 × 0.6 = 1.56, in our case a decrease in K^+ by 1.70 mmol /l# A similar situation occurs with multiple syndromes. Scoliosis makes deep breathing difficult, and the reduction in minute volume seems to have worked together. The subsequent decrease in HCO_3^- can be explained by a congestive heart defect (hypoxic lactic acidosis). However, the retention of CO_2 was probably of a different origin (difficulty breathing).

Patient in acute respiratory distress

A 51-year-old man was admitted to hospital with acute respiratory distress syndrome (Acute Respiratory Distress Syndrome). The patient smokes 3 packs of cigarettes a day.

+ Laboratory results on admission

- pH || 7.41	-	pCO_2	5.4 kPa	-	HCO_3^-	26.0 mmol / l	-	pO_2	17.6 kPa	- saturation O_2 || 76%	-	carbonylHb	11,50%	-	venous saturation	54%	-	P_{50}	4.33 kPa	- 2,3-bisphosphoglycerate || 5.3 (standard 3.3-5.3)	-	Hb	201 g / l

'Questions:'

1. *'How do you rate ABR at the time of admission?'*
2. *'Is the oxyhemoglobin dissociation curve shifted?'*
3. *'What is the significance of the other results?'*

Answers -

1. ABR is normal.
2. Yes, the curve is shifted to the right. Normally, P_{50} is 3.4-3.6 kPa; Thus, P_{50} is higher with virtually no increase in 2,3-bisphosphoglycerate. Thus, the affinity of Hb for oxygen is significantly reduced.
3. Hemoglobin is elevated, this secondary polycythemia is not common in chronic respiratory diseases. Although there is no high saturation of arterial blood (probably due to lung damage), which does not allow quality replacement, the tissues receive quite a lot of oxygen due to the arteriovenous difference.

Patient in diabetic coma

A 15-year-old girl is brought in a coma to ARO. She is a diabetic for 7 years, she took insulin. She already had several seizures hypoglycemia and ketoacidosis. She's been learning a lot at school lately and may have neglected some insulin injections.

+ Laboratory results on admission

-		*'blood'*	*'urine'*		-	pH	7.11	ketonuria	3	-	pCO_2	2,7 kPa	glycosuria	3	-	HCO_3^-	8 mmol / l			-	pO_2	12.7 kPa			-	saturation O_2	97.90%			-	glycemia	58.3 mmol / l			-	Na^+	148 mmol / l			-	K^+	5.8 mmol / l		

'Questions:'

1. What is the diagnosis?
2. Calculate the anion gap (AG), what is the cause of high AG?
3. What is the significance of increased osmolality?
4. Why are chlorides and HCO_3^- reduced, what is the meaning of "normal" Na^+ and increased K^+ ?

[redacted] -

1. Diabetic ketoacidosis (hyperglycemia, acidosis, ketonuria, glycosuria).
2. AG = Na^+ + K^+ - HCO_3^- - Cl^- = 58.8 mmol / l; the cause is ketoacidosis (other causes are lactic acidosis, uremia, intoxication ...).
3. Increased serum osmolality is due to water loss by osmotic diuresis and hyperglycemia. This is common in ketoacidosis.
4. Diabetic ketoacidosis is a form of MAC that leads to a decrease in HCO_3^- . Patients breathe rapidly to compensate for acidosis and this leads to a reduction in pCO_2 (pO_2 is usually normal). Hypochloridemia occurs due to osmotic diuresis. High Na^+ is a relative effect of water loss. The patient is thirsty, but usually drinks fluids without enough Na + and other electrolytes. Plasma K^+ levels should be evaluated carefully. Of the total body K^+ , only about 2-3% are in the ECT. Hyperkalaemia is caused by severe acidosis and partly also osmotic diuresis. (K^+ stores in a diabetic coma are always greatly reduced and K^+ is required to correct the condition.

Patient with pulmonary insufficiency and hypokalemia

Patient 55 years old, with chronic pulmonary obstructive disease who has been treated with thiazide drugs for a long time (such as diuretic).

Laboratory results on admission

pH || 7.42

-	pCO_2	11,6 kPa	-	HCO_3^-	55 mmol / l	-	pO_2	8,4 kPa	-	K^+	2.6 mmol / l

'Questions:'

1. *'What ABR fault is this?'*

[redacted] -

1. Chronic lung disease leads to reduced gas exchange: CO retention $_2$ → respiratory acidosis, O_2 deficiency → hypoxia → hypoxic lactic acidosis. Long-term use of thiazide preparations leads to loss of K^+ in the urine → hypokalaemic metabolic alkalosis.

Links

Related Articles

- ABR
- Acid-base imbalances
- Diabetic ketoacidosis
- Oxygen transport by blood

Template:Pathobiochemistry of metabolic pathways (Masopust)

Source

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Source

- ws:ABR/Otázky a kazuistiky