L15: Acid base balance

Medicine433@yahoo.com

MEDICINE 433

objectives

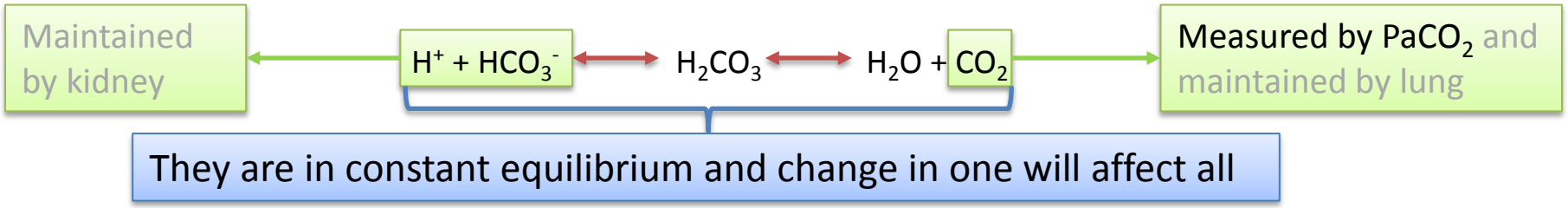
1. State the normal value for PH,PCO2,HCO3
2. Understand the basic mechanism of acid base disturbance
3. Interpret basic acid base disturbance
4. List common differential diagnosis for different acid base disorder

Color index: Step up to medicine , slide , Doctor's note , Kumar , Extra Explanation

Physiology overview

❖Blood pH refers to the level of H^+ ions and maintained by several buffering systems.

❖Most importantly the bicarbonate-carbonic acid buffer pair that depends on the balance between bicarbonate ions and carbonic acid:



❖HCO_3^- is also an important anion involved in the electrolyte balance.

❖To easily calculate the levels of the "other anions" we use the anion gap (AG):

$AG = (Na^+) - (Cl^- + HCO_3^-)$ "the normal value AG is 8-12"

❖This normal value represents all uncounted anions (e.g. proteins, phosphate, organic acids, sulfate) and the neglected K^+ .

❖An increased anion gap suggests the presence of uncounted anions (endogenous or exogenous).

•Plasma anion gapis the difference between positively and negatively charges
•Plasma anion gap is used to determine the cause of Metabolic Acidosis

Important to know

- ***Normal arterial blood pH*** = 7.35 – 7.45
- ***$PaCO_2$*** = 35-45
- ***Serum HCO_3^-***= 22-26
- ***Anion gap*** = 8-12

A ***decrease*** in blood pH is called ***acidaemia*** and is caused by ***acidosis***
An ***increase*** in blood pH is called ***alkalaemia*** and is caused by ***alkalosis***

Steps in Acid-Base Analysis

➢Step 1: Acidemic or Alkalemic?

- The pH of the arterial blood gas measurement identifies the disorder as alkalemic or acidemic.
- Normal arterial blood pH = 7.35 – 7.45
- Acidemic: pH < 7.35

➢Step 2: Is the primary disturbance respiratory or metabolic?

- To determine whether the disturbance affects primarily
  The arterial PaCO2 or The serum HCO3-.
- Respiratory disturbances alter the arterial PaCO2 (normal value 35-45)
- Metabolic disturbances alter the serum HCO3- (normal value 22-26)

➢Step 3: Is the respiratory disturbance acute or chronic?

Acute respiratory acidosis:

HCO_3^- **increase by 1 mEq/l for every 10 mmHg increase in** PaCO2

Chronic respiratory acidosis:

HCO_3^- **increase by 3-3.5 mEq/l for every 10 mmHg increase in** PaCO2

Acute respiratory alkalosis:

HCO_3^- **decrease by 2 mEq/l for every 10 mmHg decrease in** PaCO2

Chronic respiratory alkalosis:

HCO_3^- **decrease by 4-5 mEq/l for every 10 mmHg decrease in** PaCO2

Steps in Acid-Base Analysis

➢Step 4: For a metabolic acidosis, is there an increased anion gap?

- Anion gap = [Sodium] - ([Chloride] + [Bicarbonate]) Or
  AG = $[Na^+]$ - ($[Cl^-]$ + $[HCO_3^-]$).
- Normal AG 8-16
- Serum Osmolality = (2 x (Na + K)) + (BUN) + (glucose)

➢Step 5: Are there other metabolic processes present in a patient with an increased anion gap metabolic acidosis?

➢Step 6: Is the respiratory system compensating adequately for a metabolic disturbance

Metabolic acidosis:

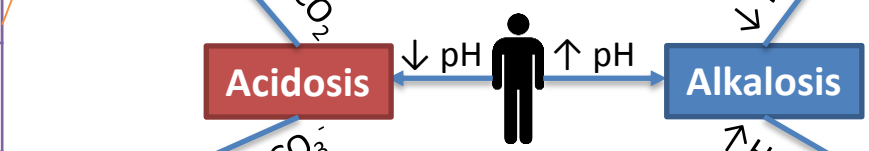
PCO2 decreases by 1 mmHg for every 1 mEq/l decrease in HCO3

Metabolic alkalosis:

- PCO2 increases by 0.6 mmHg for every 1 mEq/l increases in HCO3

Going through the next slides, will answer these steps

Overview



Acute:
Airway obstruction
sedatives
Head trauma
Chronic:
COPD
Neuromuscular disease
Extreme obesity
Chest wall deformity

Hyperventilation:
Anxiety
pain
Pregnancy
fever
Thyrotoxicosis
Drugs
Restrictive lung disease

Renal failure
Lactic acidosis
Ethanol ingestion
Aspirin intake
DKA
starvation
Ethylene glycol ingestion

RTA
Diarrhea

Saline responsive:
Vomiting
Diuretic intake

Saline resistant:
Hypertensive (hyperaldestronism)
Barrter's syndrome
Gitelman's syndrome

AG= Anion gap

U_{Cl}= urine chloride

RTA=renal tubular acidosis

Barrter's syndrome= a rare inherited defect in the loop of Henle

Gitelman's syndrome= an inherited defect in the Na-Cl symporter

Overview

Effects of acidosis and alkalosis	
Acidosis	Right shift in oxygen-hemoglobin dissociation curve diminishes the affinity of hemoglobin for oxygen (increasing tissue oxygen delivery)
	CNS depression
	Decreased pulmonary blood flow
	Arrhythmias and Impaired myocardial
	Hyperkalemia
Alkalosis	Decreased cerebral blood flow
	Left shift in oxygen-hemoglobin dissociation curve increases the affinity of hemoglobin for oxygen (decreasing oxygen delivery to tissues)
	Arrhythmias
	Tetany and seizures

Respiratory Acidosis

- Defined as reduced blood pH and increased $PaCO_2$
- Pathophysiology: **alveolar hypoventilation** that's an arise from problems in:

CNS: (depression of respiratory centers)
Brainstem problems: tumors, trauma
Drug induced: morphine, anesthetics, sedatives and narcotics

Peripheral nerve problems: (denervation of respiratory muscles)
Guillain-Barre syndrome (ascending paralysis) (next slide)

Neuromuscular junction (NMJ) diseases:
Myasthenia gravis

Chest wall problems: (abnormal respiration)
Muscle problems: Duchenne's dystrophy
Deformities: kyphosis

Bronchial tree problems:
COPD
Airway obstruction

Also divided to

Acute:
Respiratory pathophysiology - airway obstruction, severe pneumonia, chest trauma/pneumothorax
Acute drug intoxication (narcotics, sedatives)
Residual neuromuscular blockade
CNS disease (head trauma)

Chronic: (pH might be normal because of compensation by kidney within 24 hours)
Chronic lung disease (COPD)
Neuromuscular disease
Extreme obesity
Chest wall deformity

In COPD :Constriction of bronchial tree leads to Co2 accumulation in alveoli and causes hypercapnia and hypoxia as a result hypoventilation will develop (it's not directly causes hypoventilation)
(not all COPD develop hypoventilation ,only those with irreversible constriction and sever disease

Respiratory Acidosis

Clinical features:

- ***Somnolence, confusion, myoclonus with asterixis*** (quick, involuntary muscle jerks)
- They also might have signs of acute CO_2 retention: ***headaches, confusion,*** *and* ***papilledema***. (because increased $PaCO_2$ causes increased cerebral blood flow which increases CSF pressure, as well as increased free calcium due to displacement from albumin by H^+

Treatment:

- Verify patency of airways.
- Treat underlying cause.
- If PaO_2 is low (<60 mmHg) give supplemental oxygen (not in COPD patients as it can exacerbate symptoms)
- Intubation and mechanical ventilation might be required for:
  - Severe acidosis
  - $PaCO_2$ > 60 or inability to increase PaO_2
  - Mental deterioration

Guillain–Barré syndrome is Demyelinating diseases causingrapid-onset muscle weakness as a result of damage to the peripheral nervous systemso, it can cause hypoventilation by affecting respiratory muscles

Respiratory alkalosis

- Increased blood pH and decreased $PaCO_2$
- Pathophysiology: alveolar hyperventilation. That arises in:
  - Anxiety (panic attack)
  - Pulmonary embolism, pneumonia and asthma
  - Pregnancy
  - Pain
  - Sepsis
  - Liver cirrhosis and hepatic failure
  - Drugs (salicylates toxicity)
  - Restrictive lung disease
  - Overaggressive mechanical ventilation

Treatment:
Breath into paper bag to recycle the exhaled CO_2

Treatment:
Treat the underlying cause
No need for treatment in case of pregnancy

Clinical features:

Lightheadedness, ***dizziness***, ***anxiety***, ***paresthesias***, and ***perioral numbness***.(related to decreased cerebral blood flow because of vasoconstriction, as well as decreased free calcium due to increased binding with albumin because of decreased H^+)

Tetany and Arrhythmias

Renal compensation

When there is considerable change in the levels of $PaCO_2$ the kidney will compensate by excreting HCO_3^- this process needs hours to reach normal pH. The levels of compensation are:

- Acute respiratory acidosis:

HCO_3^- increase by ***1*** mEq/l for every 10 mmHg increase in $PaCO_2$

- Chronic respiratory acidosis:

HCO_3^- increase by ***3-3.5*** mEq/l for every 10 mmHg increase in $PaCO_2$

- Acute respiratory alkalosis:

HCO_3^- decrease by ***2*** mEq/l for every 10 mmHg decrease in $PaCO_2$

- Chronic respiratory alkalosis:

HCO_3^- decrease by ***4-5*** mEq/l for every 10 mmHg decrease in $PaCO_2$

Metabolic Acidosis

- Characterized by a decreased blood pH and decreased HCO_3^-
- Very important to calculate the anion gap (AG) (see slide 3)

Mnemonic for causes of metabolic acidosis is ***DR KARMEL***

Diarrhea — *normal AG*
Renal tubular acidosis

Ketoacidosis — *high AG*
Aspirin
Renal failure
Methanol
Ethanol, Ethylene glycol
Lactic acidosis

Normal AG metabolic acidosis: (decrease in HCO_3^- coincides with increased Cl^- so the AG remains normal)

Mechanisms

- Diarrhea
- Pancreatic and intestinal fistulas

→ HCO_3^- loss from GIT

- Ureterosigmoidostomy

→ Colon exchanges HCO_3^- for Cl^-

- Renal tubular acidosis (proximal and distal)
- (causes: multiple myeloma, cystinosis, wilson's disease SLE, sjogren's syndrome, amphotericin B intake…etc)
- Drugs: acetazolamide (carbonic anhydrase inhibition)

→ HCO_3^- loss from kidney

•In Metabolic acidosis you're losing HCO3 (diarrhea) OR gaining acid (exogenous :intoxication by alcohol or endogenous : renal failure , ketoacidosis , lactic acidosis)

Metabolic Acidosis

High AG metabolic acidosis:

Ketoacidosis:

- Diabetic ketoacidosis
- Starvation
- Alcohol abuse

Lactic acidosis:

- Low tissue perfusion (ischemia, shock)
- Short bowel resection (colonic flora will digest glucose to D-lactate)
- Excessive energy expenditure (seizures)
- Malignancies
- Drugs (metformin)

Renal failure: (accumulation of organic anions)

Ingestion:

- Methanol, ethanol
- Ethylene glycol
- Salicylates (aspirin)
- 5-oxoproline (metabolite of acetaminophen)

Metabolic Acidosis

Clinical presentation:

1. Hyperventilation (for compensation)
2. Decreased cardiac output and decreased tissue perfusion which can cause lactic acid production and worsening acidosis.

Both are seen more in severe acidosis (pH< 7.2)

Treatment:

Treat the underlying cause.
Sodium bicarbonate is sometimes used in severe acidosis (esp. in normal AG acidosis)
Mechanical ventilation might be needed (esp. in DKA)

Metabolic Alkalosis

Characterized by increased pH and increased HCO_3^-

It is useful to distinguish between the following:

- ***Metabolic alkalosis with volume contraction (saline responsive) is due to fluid loss***
  1. Vomiting (HCl loss)
  2. Diuretics (relative increase in HCO_3^- level)
  3. Villous adenoma
- ***Metabolic alkalosis with volume expansion (saline resistant) is usually due to pathology of adrenal gland or kidney***
  1. Hyperaldosteronism (secondary hypertension)
  2. Barrter's syndrome
  3. Gitelman's syndrome

This distinction can be made by history or by measuring urine chloride (U_{Cl}). A results of less then 20 suggests fluid loss (saline responsive metabolic alkalosis), while a result of more then 20 suggests saline resistant metabolic alkalosis.

Note: hypokalemia and metabolic alkalosis <u>are related</u> (one happening will cause the other) because decreased serum H^+ will cause intracellular H^+ cause to efflux out of cells in exchange for K^+ and vice versa as well as the increase in kidney K^+ excretion seen in hyperaldosteronism, and diuretics intake

Metabolic Alkalosis

Treatment:

1. Treat the underlying cause.
2. Give normal saline plus potassium in saline responsive
3. Spironolactone (K^+ sparing diuretic) might be considered in saline resistant

Respiratory compensation

When there is change in the levels of HCO_3^- the lung will compensate by changing the respiratory rate which will change the $PaCO_2$ levels. The levels of change are:

Metabolic acidosis:

$PaCO_2$ decreases by **1** mmHg for every 1 mEq/l decrease in ***HCO_3^-***

Metabolic alkalosis:

$PaCO_2$ increases by **0.6** mmHg for every 1 mEq/l increases in ***HCO_3^-***

Case studies

pH =7.2, pCO2 = 60, HCO2 = 24.

What it is the primary problem? Acute respiratory acidosis
Compensation?
Differential diagnosis? Myasthenia graves – sever COPD.. etc
Treatment ? Treat the underling cause, if he still has problem "intubation"

What do you expect the ABG in the following patients to be:

24 years old male with acute SOB, and wheezes for 2days.

Past hx: Bronchial asthma

expect to one of these

1- hyperventilation respiratory alkalosis (initially)
2- normalization (sine of getting fatigue)
3- hypoventilation respiratory acidosis

67 years old women, HTN,DMII, COPD presenting with cough and SOB

Acute respiratory acidosis

Or acute in tope of chronic (because she may had chronically retaining CO2 so her pCo2 is abnormal (e.g. 60 mmHg) and when she present in acute pCo2 is further increased (e.g. it becomes 80 mmHg)

Case studies

pH: 7.25
$[HCO_3^-]$: 20 mEq/L
$PaCO_2$: 52 mmHg

What it is the primary problem ? Respiratory acidosis
Compensation? No
Secondary problem ? Metabolic acidosis
Differential diagnosis ? Pt has DM and bronchial asthma and he just started DKA

32 y/o male present w/ 2d Hx of intractable vomiting. ; pH 7.51, pCO_2 41
Na132, Cl 90 32 K3.4 HCo2= 33 creatinine1.6
What is the predominant acid-base disorder?
Alkalosis (Metabolic)
Treatment ? Saline

A 58- year old man presents to the Emergency Department with abdominal pain and hypotension. And he is diabetic and atherosclerotic Investigation reveal the following:
Na 140 K 4 Cl 90
HCO3 = 5 PH 6.8 PCO2 36 PO2 7
Analyze the acid-base disorder(s) seen in the patient.
Metabolic Acidosis
Next step is anion gap = 45(high)
Differential diagnosis :
Renal failure
Lactic acidosis
Ketoacidosis
Diagnosis
Because he is atherosclerotic he developed ischemia which causes lactic acidosis)
Treatment
Raise the bloods pressure

Case studies

pH: 7.32
$[HCO_3^-]$: 19 mEq/L
$PaCO_2$: 55 mmHg

What it is the primary problem? Metabolic acidosis
Compensation?
Differential diagnosis? DKA , Lactic acidosis
What other investigation you want to do? Anion gap

Quick hit

	pH	pCO2	HCO3	Interpretation
1	7.41	40	24	**normal**
2	7.5	42	35	**metabolic alkalosis**
3	6.72	40	5	**metabolic acidosis**
4	7.26	63	25	**respiratory acidosis**
5	7.52	18	25	**respiratory alkalosis**

56 yo M with Hx of COPD is admitted with 1-wk Hx of dyspnea, productive cough and diarrhea
(Na) 125, (Cl) 103 , (BUN) 42, (Glucose) 100, (K) 3.5, (HCO3-) 10, (Creat) 1.4
ABG 7.14 pCO_2 30 pO_2 50
What is the predominant acid base disorder ?
Metabolic acidosis
What pCO2 is expected with normal respiratory compensation ? 40 – (1.2 * (24-10)) = 23.2, this is not full compensation b/c pCO2 is 30 – indicates an underlying primary respiratory acidosis, suggested by the Hx of COPD, dyspnea, and productive cough (lungs not able to appropriately compensate)
What is the Anion Gap ?
125 – (103+10) = 12 – normal AG ∴ etiology is either diarrhea or RTA – most likely diarrhea b/c of the history

MCQs

1- 70-year-old is found to be hypotensive in his home. He appears volume-depleted. Initial blood gases show a *pH of 7.2*, *PCO2 (mmHg) 35*. Other electrolytes are Na+ 136 meq/L Cl− 114 K+ 4.0 *HCO3 14* The primary acid-base disorder in this patient is:

a. Respiratory acidosis

b. Metabolic acidosis

c. Respiratory alkalosis

d. Metabolic alkalosis with compensation

2-The most likely cause of this acid-base disorder is:

a. Renal failure b. Diarrhea c. Diabetic ketoacidosis d. Alcohol ketoacidosis

3- 25-year-old diabetic male is brought to the emergency room in coma with BP of 140/70 and P of 90. Blood sugar measurement by finger stick is elevated to 400. Blood gases show a *pH of 7.2*, *HCO3 − of 8 meq/L*, *PCO2 of 33*, BUN of 12 mg/dL, creatinine of 1.2 mg/dL. Anion gap is 18. The most likely cause of this metabolic acidosis is:

a. Lactic acid

b. Acetoacetate and betahydroxybutyrate

c. Bicarbonate loss

d. Retained sulfate and phosphate anions

MCQs

4- A young man presents to the emergency room with cyanosis and disorientation. Blood gases show *pH of 7.2*, *PCO2 of 68 mm Hg, HCO3 of 25 meq/L*. There are multiple needle tracks in the antecubital fossa. The rest of the examination is normal. The most likely acid-base problem is:

a. Acute respiratory acidosis
b. Acute metabolic acidosis
c. Chronic respiratory acidosis
d. Chronic metabolic alkalosis with respiratory compensation

5- A 30-year-old female on birth control pills is seen in the emergency room with tachyphemia(rapid and irregular speech). She complains of parathesias. The *pH is 7.5, HCO3 – is 19 meq/L, PCO2 is 25 mm Hg*. Which of the following is correct?

a. The patient is having an anxiety Attack
b. The most likely diagnosis is pulmonary embolism
c. The acid-base disorder is an acute respiratory alkalosis
d. The most likely cause of the alkalosis is vomiting

MCQs

6- A 60-year-old male with severe chronic obstructive lung disease was admitted for respiratory failure with a *pH of 7.3, HCO3 of 40 meq/L, PCO2 of 80 mm Hg*. The patient required intubation and mechanical ventilation. On the ventilator his PCO2 is now 40 mm Hg. The acid-base disorder that this patient is most likely to have is

a. Mild respiratory acidosis

b. Metabolic acidosis secondary to Lactate

c. Metabolic alkalosis

d. There is not likely to be an acid-base disorder

7- A 65-year-old male with severe chronic bronchitis and CO2 retention develops acute pneumococcal pneumonia and sepsis. Blood gases on admission show a pH of 7.0, PCO2 of 65 mm Hg, HCO3 of 8 meq/L. Serum Na+ is 135, K+ is 4.0, Cl is 110. The acid-base disorder is best described as :

a. Pure respiratory acidosis

b. Mixed metabolic and respiratory Acidosis

c. Pure metabolic acidosis

d. Mixed respiratory acidosis and metabolic alkalosis

Answers :1-B 2-B 3-B 4-A 5-C 6-C 7-B

Explanation of some answers

Q3:The patient has a metabolic acidosis with an anion gap. Since he is a diabetic with an elevated blood sugar, diabetic ketoacidosis is the most likely cause for this anion gap acidosis. Retained anions are acetoacetate and betahydroxybutyrate. Some lactic acid accumulation is also possible, but the patient's blood pressure is stable, and this process would be much less likely. Since the patient is not in renal failure, retained sulfate and phosphate anions should not be responsible for the metabolic acidosis in this patient. There is no evidenc given for bicarbonate loss—such as would occur with diarrhea.

Q6 : Prior to intubation, the patient had a chronic respiratory acidosis with a compensatory metabolic alkalosis. Once ventilated, the respiratory acidosis resolved,BUT THE HCO3 is still elevated leaving what would certainly be a significant metabolic alkalosis.

Q7: The patient has a severe acidosis, with both an elevated PCO2 and a very low bicarbonate. This is caused by hypoventilation and sepsis. The patient is most likely to have a lactic acidosis associated with the infectious process. It is an anion gap acidosis.

DONE BY

Faroq Abdulfattah	Raneem AlOtaibi
Hassan Almalak	Areej Alwahaib
Fahad Al-Ghtani	

Medicine is a science of uncertainty and an art of probability