Acid Base Problems

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• The cases in this will be the substance of the exercise. Answer initially to the best of your ability. Then discuss with your neighbor and I will ask again.
• If you get how to do and understand these cases you will have learned how to do moderately difficult acid base problems.
• My “lecture” will focus on how to solve these case by case.
• Be prepared! (is the boy scout’s marching song)

Objectives (slide 1)

- Upon completion of this section, the student will be able to:
  - Describe the following components of arterial acid-base balance:
    - the respiratory component, in which pulmonary ventilation results in carbon dioxide elimination and controls the arterial pCO2,
    - the important buffers,
    - the metabolic component, which is affected by tissue metabolism and the gastrointestinal system, and which is regulated by the kidneys.
    - Describe the appropriate use of arterial blood gases (ABGs) and how the values are derived.

Objectives (slide 2)

- Recognize the following conditions and list their common causes:
  - metabolic acidosis, non-anion gap
  - metabolic acidosis, anion gap
  - metabolic alkalosis
  - respiratory acidosis
  - respiratory alkalosis
  - Recognize the importance of urine Cl- measurement and urine pH in the evaluation of metabolic alkalosis and metabolic acidosis, respectively.
- Recognize mixed acid-base conditions.
  - Recognize appropriate and inappropriate respiratory compensation for a given acute or chronic metabolic abnormality.
Terms 1
- Acidemia — An arterial pH below the normal range (less than 7.36).
- Alkalemia — An arterial pH above the normal range (greater than 7.44).
- Acidosis — A process that tends to lower the extracellular fluid pH (hydrogen ion concentration increases). This can be caused by a fall in the serum bicarbonate (HCO₃⁻) concentration and/or an elevation in PCO₂.
- Alkalosis — A process that tends to raise the extracellular fluid pH (hydrogen ion concentration decreases). This can be caused by an elevation in the serum HCO₃⁻ concentration and/or a fall in PCO₂.

Terms 2
- Metabolic acidosis — A disorder that causes reductions in the serum HCO₃⁻ concentration and pH.
- Metabolic alkalosis — A disorder that causes elevations in the serum HCO₃⁻ concentration and pH.
- Respiratory acidosis — A disorder that causes an elevation in arterial PCO₂ and a reduction in pH.
- Respiratory alkalosis — A disorder that causes a reduction in arterial PCO₂ and an increase in pH.

Terms 3
- Simple acid-base disorder — The presence of one of the above four disorders with the appropriate respiratory or renal compensation for that disorder.
- Mixed acid-base disorder — The simultaneous presence of more than one acid-base disorder. Mixed acid-base disorders can be suspected from the patient's history, from a lesser- or greater-than-expected compensatory respiratory or renal response, and from analysis of the serum electrolytes and anion gap. As an example, a patient with severe vomiting would be expected to develop a metabolic alkalosis due to the loss of acidic gastric fluid. If, however, the patient developed hypovolemic shock from the fluid loss, the ensuing lactic acidosis would lower the elevated serum HCO₃⁻ possibly to below normal values, resulting in acidemia.

Primary Acid Base Disorders
- Respiratory Acidosis
  - Decreased pH, increased pCO₂
- Respiratory Alkalosis
  - Increased pH, decreased pCO₂
- Metabolic Acidosis
  - Decreased pH, decreased HCO₃⁻, may have increased anion gap, may have increased Cl⁻
- Metabolic Alkalosis
  - Increased pH, increased HCO₃⁻, may have decreased Cl⁻
A basic approach to a suspected acid/base disorder (partial list) slide 1

- **History** (ingestion, underlying metabolic disease, predisposing factors for hypovolemia, diarrhea, vomiting, NG suction, diuresis, respiratory failure, altitude, suspected medication abuse or malingering)
- **Physical exam** exhaustation, tachycardia, tachypnea, skin turgor, signs of pulmonary disease, trauma, drug use)
- **ABG** (pH, PCO2, pO2, HCO3)
- **Electrolytes** Na⁺, K⁺, CO₂, Cl⁻, BUN, Creat, Blood sugar, acetone
  If metabolic alkalosis or chronic acidosis check urine electrolytes

A basic approach to a suspected acid/base disorder (partial list) slide 2

- Determine presence or absence of acidemia (CO₂ (HCO₃) or the pH is low) or alkalemia
- Calculate anion gap
- If HCO₃ or CO₂ is low or anion gap is present, calculate expected CO₂ (Winter’s rule)

Mixed Acid Base Disorders

- Respiratory Acidosis with Metabolic Acidosis
  - Metabolic acidosis with resp depression or failure
- Respiratory Alkalosis with Metabolic Acidosis
  - <HCO₃, >anion gap, pH near normal eg. Sepsis w/ primary hyperventilation, salicylate overdose
- Metabolic acidosis with metabolic alkalosis
  - Anion gap is > but HCO₃ and pH are near normal
- Respiratory alkalosis with Metabolic acidosis and metabolic alkalosis
- Respiratory acidosis with metabolic acidosis and metabolic alkalosis
- Metabolic acidosis (non anion gap) with metabolic acidosis

Equations and axioms

- Anion gap = [Na⁺] - ([Cl⁻] + [HCO₃⁻])
- [H⁺] = 24 / pCO₂ x [HCO₃⁻] (derived from Henderson Hasselbach equation)
- Acidemia is present when the CO₂ (HCO₃) or the pH is low
- Winter’s rule (for measuring respiratory compensation in metabolic acidosis): HCO₃⁻ x 1.5 + 8 +/- 2
- Compensation never returns pH entirely to normal (but a mixed disorder may have normal pH in setting of acidosis or alkalosis)
- A metabolic acidosis is present when anion gap is increased even in the absence of acidemia
- Urine Anion Gap= ([Na⁺]u + [K⁺]u) - [Cl⁻]u
  - A negative urine AG reflects high [NH₄] excretion
  - Delta/delta if delta HCO₃/delta gap =1, then prim met acidosis of anion gap type only. If not =1 possibly mixed acidosis or resp. alkalosis + met acidosis
  - If delta anion gap is increased, (anion gap-normal anion gap) + [HCO₃]s > 30, then an underlying metabolic alkalosis is present
- pH=6.10 + LOG [HCO₃⁻]/(0.3×pCO₂)
Normal Values
(room air, sea level)

• pH 7.37-7.43
• [H+] 37-43 nmol/liter
• pCO2 36-44 mmHg
• [HCO3] 22-26 meq/Liter
• K+ 3.5-5.2 meq/dl

Introductory case

• Anna was a 54 year old family medicine patient who came in with a chief complaint of severe shortness of breath for 4 hours. She had a history of pulmonary emphysema, cigarette smoking 2 ppd for many years, was a recovering heroin addict on methadone and had been having some joint pain, probably due to osteoarthritis, in her neck.
Introductory case

- Her physical exam was remarkable for an agitated tremulous patient working very hard to breathe. Her ENT was wnl. Chest had bilateral coarse breath sounds without focal rales.
- CBC had Hct of 38, WBC 8.3 with 60 polys 6 bands, 29 lymphs, and 5 monos on differential.
- ABG was ordered and an admission in the ICU was arranged.

Introductory case

- My initial diagnosis was exacrerbation of COPD, probably due to infection.

Case #1

- A 37 year old male patient has been involved in a motor vehicle accident and was transferred by paramedics to the ER. The patient complains of abdominal pain and dizziness, and has ecchymosis over his LUQ abdomen.
- BP 80/40 Pulse 150 RR 26 Afebrile
- pH 7.3 Na 140
- HCO3 16 K 5.8
- pCO2 29 Cl 100
- pO2 90 CO2 15
- O2 sat 94%

What is the primary acid base disorder?

- 1. Respiratory acidosis
- 2. Respiratory alkalosis
- 3. Metabolic acidosis
- 4. Metabolic alkalosis
- 5. no primary acid/base disorder present
Case #1

• Is the patient acidemic or alkalemic?
  a. acidemic
  b. Alkalemic
  c. normal

• What is the anion gap?
  a. 12
  b. 11
  c. 25
  d. 24

Case #1 answers

• Basic acid/base disorder? Metabolic acidosis.
• What is the anion gap? 25
• Is compensation adequate? 30-35 would be pCO2 with optimal compensation,
• So slight degree of hyperventilation (ddx, pain, fear, respiratory alkalosis due to hyperventilation)
• Lactic acidosis due to shock most likely
Back to introductory case

- Anna’s ABG results were:
  - pO₂ 80
  - pCO₂ 46
  - HCO₃ 20
  - pH 7.2
- Additional questions to be asked? Tests to be run?

Case #2

- A 27 year old woman is admitted to the ER with complaints of weakness, a tingling sensation in feet and hands and “unsteadiness.” She works as a model and has recently been in a competition. She denies drug ingestion but admits to working on rapid weight loss to meet the expectations of the people judging her.
  - Labs: pH 7.55  Na⁺ 132  BUN 20
  - pCO₂ 50  K⁺ 3.5  Creat 0.8
  - pO₂ 90  Cl⁻ 90
  - HCO₃ 45
  - Do you want to order anything else?

Case #2 answers

- Urine electrolytes: Urine Cl 30 mml/d, Urine K 20 mmmol/d
- What is the diagnosis?

From “Metabolic Alkalosis” Galla, JH and Luke, RG Best Practice of Medicine 2001
Case 2 what is the diagnosis?

a. Diuretic use most likely  
b. Bulimia and vomiting most likely  
c. Chronic hyperaldosteronism most likely

Case #2

• Basic disorder? Metabolic alkalosis (pH up, bicarb up, pCO2 elevated which should, if anything cause a respiratory acidosis.)
• Cause? Vomiting, diuretic use in differential. Vomiting could be due to bulimia, possibly pregnancy likely candidates BUT with a high urine chloride, and a moderately increased urine K+, diuretic use is most likely

Case #3

• A 50 year old man with a history of alcohol abuse and liver disease on follow up visit after being placed on diuretics for increasing weight and edema.
• LABS: pH 7.43 Na+132
• pCO2 39 K+3.7
• HCO3 25 Cl-84

Define the acid base status

• 1. Respiratory acidosis
• 2. Respiratory alkalosis
• 3. Metabolic acidosis
• 4. Metabolic alkalosis
• 5. no primary acid/base disorder present
Case #3

- Mixed metabolic acidosis and hypochloremic volume contraction alkalosis. pH is normalized by the combination. This patient is probably third spacing fluid from ascites, and has hypovolemia made worse by diuretics.

The Introductory case again

- Anna was seen by my colleague, the ICU doctor who looked at the ABG and asked:
  - Have you taken any meds?
  - The patient said “only aspirin.”

The Introductory case again

- ABGs again:
  
  - pO2 80, pCO2 46
  - HCO3 20, pH 7.2

Case #4

- A 54 year old woman status post dental cleaning with end stage renal disease on hemodialysis. Admitted to ER with chief complaint of change in mental status. Found with a BP of 90/60 and a heart rate of 125 and a temperature of 103.
  - Labs pH 7.44 Na+136 K+5.5
  - pCO2 12 Cl- 106
  - HCO3 8
What is the acid base diagnosis?

1. metabolic acidosis, non-anion gap
2. metabolic acidosis, anion gap
3. metabolic alkalosis
4. respiratory acidosis
5. respiratory alkalosis

Case #4

- Mixed metabolic acidosis with respiratory alkalosis that goes beyond compensation (expected pCO2 with HCO3 of 8 would be 20. Differential diagnosis is sepsis and neurological disorders, both are possible with endocarditis

Case #5

- A 23 year old male with no prior medical history has had 2 months of 20 lb. inadvertent weight loss, accompanied by increase in appetite and general fatigue and weakness. In the last two days he has developed shortness of breath, abdominal pain and general malaise.
- General appearance acutely ill male in considerable distress. A musty odor is noted.
- VS T 101 P 115 R 18 BP 90/60
- Chest clear Cor RRR Abd diffusely tender without rebound

CBC
Lytes Na 140 K 6.0 CO2 12 Cl 105 Glu 658
ABG pH 7.15 pCO2 26 pO2 90 HCO3 13

What is the primary acid base disorder?

1. Respiratory acidosis
2. Respiratory alkalosis
3. Metabolic acidosis
4. Metabolic alkalosis
5. no primary acid/base disorder present
What is his anion gap?
- 1. normal
- 2. less than 6
- 3. 20-25
- 4. 25-30
- 5. >30

What additional tests might you order?
- 1. Serum cyanide level
- 2. Carboxyhemoglobin
- 3. serum ketone
- 4. whole body MRI for neoplasia
- 5. HIV

What treatment will you begin with?
What is the diagnosis?

Oh no, not the introductory case again!
- The salicylate level was 80mg/dl
- (top of therapeutic range =30mg/dl)
Case #6

- A 35 year old man, admitted secondary to suicide attempt with Tylenol OD., on the ventilator.
- pH 7.69   Na⁺ 135
- pCO₂ 30   K⁺ 4.0
- HCO₃ 35   Cl⁻ 84

Define his acid base status.

- 1. Respiratory acidosis
- 2. Respiratory alkalosis
- 3. Metabolic acidosis
- 4. Metabolic alkalosis
- 5. Metabolic acidosis with respiratory alkalosis

answer

- Patient has metabolic alkalosis due to volume contracture, and is being over ventilated which gives him a respiratory alkalosis on top of that.

Case #7

- A 47 year old, non diabetic, obese male, admitted with confusion, slurred speech and lethargy.
- pH 7.10   Na⁺ 144
- pCO₂ 60   K⁺ 4.8   BUN 24
- pO₂ 75   Cl⁻ 92   Creat 1.4
- TCO₂ 53   HCO₃ 46   glucose 118
Define the acid base status.

- 1. Metabolic acidosis
- 2. Respiratory acidosis
- 3. Metabolic alkalosis
- 4. Respiratory alkalosis

**answer**

- Metabolic (non anion gap) and respiratory acidosis with metabolic alkalosis vs. chronic resp acidosis compensation

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**CARBON DIOXIDE (TOTAL), serum**

- Synonyms: CO₂, TCO₂, ECO₂
- Reference Ranges:
  - Newborn - 15 days: 13-22 mmol/L.
  - 15 days - 15 years: 20-28 mmol/L.
  - 15 years - adult: 24-31 mmol/L.
- Critical Values: <10 mmol/L or > 40 mmol/L.
- Test Method: Enzymatic Multi Point Rate

- **Increased in:**
  - Respiratory acidosis
  - Empyema
  - Hypokalemia
  - Cystic Fibrosis
  - Severe, prolonged vomiting
  - Pulmonary Edema
  - Metabolic alkalosis
  - Pneumonia
  - Excessive intake of antacids
  - Cushing's Syndrome
  - Congestive Heart Failure
  - Primary aldosteronism

- **Equations and axioms**
  - Anion gap = [Na⁺] - ([Cl⁻] + [HCO₃⁻])
  - [H⁺] = 24 x pCO₂/[HCO₃⁻] (derived from Henderson Hasselbach equation)
  - Acidemia is present when the CO₂ or the pH is low
  - Winter’s rule (for measuring respiratory compensation in metabolic acidosis): HCO₃⁻ x 1.5 + 8 = (+/- 2) expected pCO₂
  - Compensation never returns pH entirely to normal
  - A metabolic acidosis is present when anion gap is increased even in the absence of acidemia
  - A negative urine AG reflects high [NH₄⁺] excretion
Case #8

- A 67 year old man with an 114 pack year smoking history and diagnosed emphysema and chronic bronchitis has had 3 days of bloody diarrhea with abdominal pain and fever to 101.2. His vs include a T of 101.0, R of 34, P 52, BP 82/45, pulseO2sat 93%.
- Physical exam significant for a markedly dyspneic man breathing with marked accessory muscle use. Chest is marked by rhonchi but no rales are present. Heart sounds are distant and no murmurs or gallops are appreciated. Abd. Has hypoactive bowel sounds, has a low lying liver edge with no enlargement to percussion, no guarding or rebound.
- WBC is 3.8 with 40% bands. ABGs:
  - pO2 79 pCO2 40  pH 7.12  HCO3 12
  - What is the cause of his acidemia?

What is his primary acid base disorder?

- 1. Respiratory acidosis
- 2. Respiratory alkalosis
- 3. Metabolic acidosis
- 4. Metabolic alkalosis
- 5. no primary acid/base disorder present

Is respiratory compensation as expected?

- Yes.
- No, he has an excessive level of hyperventilation
- No, he has inadequate respiratory compensation

Case #8 answer

- Metabolic and respiratory acidosis (the latter due to inability to compensate
- For metabolic acidosis due to decreased pulmonary function).
- If you use Winter’s rule you can see that the pCO2 is way above where it should be for
- Degree of met. Acidosis.
Case #8

• What management will he require?
• How come he is not more hypoxic?

Case #9

• A 21 y.o. woman with IDDM, had a set of values as follows for more than 2 months. She denied drug ingestion or diarrhea. Is this renal or non-renal acidosis? What is the anion gap? The osmolal gap? Is the renal response “normal”?

<table>
<thead>
<tr>
<th>Blood</th>
<th>Urine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na+</td>
<td>136</td>
</tr>
<tr>
<td>K+</td>
<td>2.9</td>
</tr>
<tr>
<td>Cl-</td>
<td>103</td>
</tr>
<tr>
<td>CO2</td>
<td>19</td>
</tr>
<tr>
<td>Glucose</td>
<td>190</td>
</tr>
<tr>
<td>BUN</td>
<td>11</td>
</tr>
<tr>
<td>Creatinine</td>
<td>.9</td>
</tr>
<tr>
<td>pH</td>
<td>7.35</td>
</tr>
<tr>
<td>Osmolality</td>
<td>293</td>
</tr>
</tbody>
</table>
Osmolal Gap is the difference between measured serum osmolality and calculated serum osmolality. It is typically calculated as:

\[ OG = \text{measured serum osmolality} - (2 \times \text{serum sodium} + \text{serum glucose} + \text{serum urea}) \]

Where:
- \(2 \times \text{serum sodium} + \text{serum glucose} + \text{serum urea} = \text{the calculated serum osmolality and all measures are in mmol/L.}\)
- \(OG = \text{osmolal gap}\)
- In US customary units the calculated osmolarity is: \( (2 \times \text{sodium} ) + \text{glucose/18 + BUN/2.8} \).

A normal osmolal gap is < 12 mmol/L. Causes of an elevated osmolal gap are numerous.

Several causes are:
- Ethanol intoxication
- Methanol ingestion
- Isopropanol ingestion
- Ethylene glycol ingestion

Define the AB disturbance.

Why is the sodium not lower if she is volume contracted?

Why is she hypokalemic?

What is the cause?

Renal Tubular Acidosis

Apart from the causes of increased acidity, there are four types of metabolic acidosis caused by the inability of the kidney to excrete acid. These conditions, termed renal tubular acidosis themselves have a number of potential (including hereditary) causes.

Type 1 (distal) RTA: decreased acid secretion in the collecting ducts. The urine is relatively alkaline (pH greater than 5.5).

Type 2 (proximal) RTA: bicarbonate in prourine is poorly reabsorbed in the proximal tubules. It is usually mild, with bicarbonate levels between 14-20.

It can be isolated, or part of a more generalized disorder with associated glycosuria, aminoaciduria and phosphaturia, termed as the Fanconi syndrome.

Type 3 RTA: occurs in children.

Type 4 RTA: this form occurs in deficiency of aldosterone, the principal mineralocorticoid. Aldosterone is required for the secretion of potassium and hydrogen in the distal tubules, as well as retention of sodium. In this type of RTA there is mild hyperkalemia and metabolic acidosis due to acid retention.

Plasma and Urine Osmolal Gaps

- Plasma osmolal gap
- Plasma osmolality measured-calculated: Plasma osmolality (calc) = 2 x [Na+] + [Glu]/18 + BUN/2.8
- > plasma osmol. Gap reflects unmeasured unionized compounds (alcohol, mannitol, isopropanol, ethylene glycol)

Urine osmolality gap = measured urine osmolality-(2 x [Na+]u +[K+]u) + [glucose]u/18-UUN/2.8
Renal Tubular Acidoses

- Proximal (type II) low serum K+, blood pH will achieve new set point, urine pH<5.5 Fanconi’s (genetic), myeloma, lead, Sjogren’s syndrome, amyloid, etc.
- Distal type I urine pH > 5.5, K+ wasting drug (amphotericin, genetic, nephrocalcinosis, analgesics, licorice, <Mg++, sickle cell, etc.)
- Generalized distal type IV loss of mineralocorticoid, hyperkalemia, <ammoniagenesis. Drug ACE inhibitors
- Buffer deficiency- <ammoniagenesis renal failure similar to type IV but K+ can be normal