Complex Acid Base Disturbances

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Medical Intensive Care Unit
Objectives

• Utilize a systematic approach to evaluate acid-base disturbances
• Identify simple and complex acid-base disturbances (mixed, secondary, tertiary)
• Apply rules of compensation
• Identify/recognize the etiology of acid base disorders
Case Study – Mr. L

- 43 yo male transferred to MICU from a referring facility with hypotension, diarrhea, anemia and a presumed GI bleed. Prior to transport, pt. received 1L NS, 1u PRBC and 1gm ceftriaxone
- PMH: HTN, ETOH Cirrhosis, Bilateral foot ulcerations.
- Home medications: furosemide, lactulose, lisinopril, fluoxetine, temazepam and a recent antibiotic
- Social Hx: Unemployed computer technician, lives with elderly mother. 1 PPD smoker, denies illicits, last ETOH 12/2013
Case Study cont. - Physical Exam

GENERAL: Ill appearing male, poor hygiene, lethargic, confused.

NEURO: RASS 0, CAM +. Non-focal, PERL. Following commands

HEENT: mucous membranes dry, oropharyngeal inflammation, sclera icteric, severe dental caries, missing teeth

PULMONARY: normal respiratory rate, clear breath sounds. Deep respirations. 02 sat 100% on 3L

CV: Sinus tachycardia. 2+ BLE edema.

GI: hypoactive bowel sounds, abdomen soft, non-distended. Mild abdominal tenderness LLQ.


SKIN: Jaundiced. Ulcerations outer aspect bilateral feet. Heels macerated L>R. Stage II pressure ulcer sacrum.
Case Study cont.

Labs:
WBC 3.4, PCV 18, Plt 46
Na 144, K 2.2, Cl 127, CO2 8, BUN 27, Cr. 0.63, Gluc 26, AG 9, Ammonia 48
Lactate 6.5, Troponin 1.7, CPK 1063, BNP 700
Alb. 0.9, T. Bil 3.9, AlkP. 46, AST 113, ALT 33, LDH 285
INR 5.2
UDS + benzodiazepines and acetaminophen
ABG pH 7.50, PaCO2 18, HC03 14

Chest xray with evidence of volume overload
Stepwise Analysis

• Start with a thorough History and Physical
  Look for acid-base clues

• Verify that the lab values are consistent.
  \[ \text{HC03 (ABG)} = \text{C02 (venous electrolytes)} \pm 2 \]

• Is pH low, normal or high?
  Acidemia < 7.35
  Normal 7.35 – 7.45
  Alkalemia > 7.45

• Is the primary disturbance metabolic or respiratory? If both exist, pick the one that seems most severe at first glance.
Stepwise Analysis Cont.

• Is it simple or mixed?
• Is the disturbance acute or chronic?
• Have compensatory changes occurred?
• For metabolic acidosis, calculate the Anion Gap
  \[ AG = (Na^+) - (HC03^- + Cl^-) \]
  (This will help develop your list of differential diagnoses)
• Is there a secondary or tertiary disturbance present?
• Determine the etiology of the disturbance (s)
<table>
<thead>
<tr>
<th>Metabolic Acidosis</th>
<th>Metabolic Alkalosis</th>
<th>Respiratory Acidosis</th>
<th>Respiratory Alkalosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of Diabetes</td>
<td>Vomiting</td>
<td>+ Tobacco use</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
<td>NG suction</td>
<td>COPD</td>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Loop or Thiazide Diuretics</td>
<td>Ingestions- Sedatives, Narcotics</td>
<td>Pulmonary edema</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>Massive diuresis</td>
<td>Severe obesity</td>
<td>Chronic liver dx</td>
</tr>
<tr>
<td>Starvation</td>
<td>Hypokalemia</td>
<td>Neuromuscular disorders</td>
<td>Stroke</td>
</tr>
<tr>
<td>Ingestions- Alcohols, Acetaminophen, ASA</td>
<td></td>
<td></td>
<td>Head Injury</td>
</tr>
<tr>
<td>Binge drinking</td>
<td></td>
<td></td>
<td>Aspirin toxicity</td>
</tr>
</tbody>
</table>
Mr. L’s Clues

- Hypotension
- Diarrhea
- Poor nutritional status
- Ingestion??
- Loop diuretics
- Hypokalemia
- Tobacco use
- Chronic liver disease
- Pulmonary edema
- Elevated lactate
Acidemia

Low HC03
High PC02

Low Hc03
Low PC02

High HC03
Low PC02

High HC03
High PC02

Mixed metabolic acidosis and respiratory acidosis

Predominant Metabolic Acidosis

Predominant Respiratory Acidosis

Not possible; likely lab error

Mixed metabolic acidosis and respiratory alkalosis

Simple metabolic acidosis

Simple respiratory acidosis

Mixed respiratory acidosis and metabolic acidosis

Mixed respiratory acidosis and metabolic alkalosis

Simple respiratory acidosis

Apply Compensation Rules

Measured PC02 too high
Measured PC02 too low
Measured PC02 is appropriate

Measured HC03 is too low
Measured HC03 is too high
Measured HC03 is appropriate
Alkalemia

- High HC03 Low PC02: Mixed metabolic alkalosis and respiratory alkalosis
- High Hc03 High PC02: Predominant Metabolic Alkalosis
- Low HC03 Low PC02: Measured HC03 is too low
- Low HC03 High PC02: Measured HC03 is too high

Apply Compensation Rules

- Measured PC02 too high: Mixed metabolic alkalosis and respiratory acidosis
- Measured PC02 too low: Mixed metabolic alkalosis and respiratory alkalosis
- Measured PC02 is appropriate: Simple metabolic alkalosis
- Measured HC03 is too low: Simple respiratory alkalosis
- Measured HC03 is too high: Mixed respiratory alkalosis and metabolic acidosis
- Measured HC03 is appropriate: Simple respiratory alkalosis
Metabolic Acidosis

pH < 7.35 with normal or ↓ HC03

• Caused by one of four mechanisms
  – Decreased H⁺ excretion - Distal RTA
  • Diminished NH4⁺ production – Renal failure, Hypoaldosteronism (Type IV RTA)
  – Increased H⁺ load – lactate, DKA, toxins, Rhabdo
  – Bicarbonate loss – Diarrhea, Pancreatic Fistula, Ureteral Diversion. Renal bicarb loss (Proximal RTA)
  – Bicarbonate dilution

Metabolic Acidosis

• Threats to life
  – Hemodynamic Instability (↓myocardial contractility, ↓intravascular volume, ↓PVR)
  – Cardiac arrhythmias (hyper or hypokalemia)
  – Ventilatory failure
  – Presence of toxins
  – Nutritional deficiencies (B vitamins, Wernicke’s)

Anion Gap

- AG = (Na⁺) – (HC0₃⁻ + Cl⁻)
- Three clinical applications
  1) Presence or absence of an AG assists in determining the cause of a metabolic acidosis.
  2) The AG is useful in determining the presence of a mixed acid/base disturbance by calculating the Delta Ratio
  3) The AG can be useful in detecting selected disorders that occur with a low, not high AG. (Lithium toxicity, Multiple Myeloma with production of cationic paraproteins)

Anion Gap

A. NORMAL ION DISTRIBUTION
B. METABOLIC ACIDOSIS due to acid accumulation; decreased $\text{HCO}_3^-$, increased anion gap
C. METABOLIC ACIDOSIS due to $\text{HCO}_3^-$ loss; decreased $\text{HCO}_3^-$, normal anion gap, increased $\text{Cl}^-$

http://www.flickr.com/photos/dokidok/2369729114/in/photostream/
• Hypo-albuminemia can mask an increased concentration of gap ions and lower the value of the AG.

• Adjusted AG =

\[ \text{AG} + 2.5 \times (\text{normal albumin g/dL} - \text{albumin g/dL}) \]

Example: Mr. L’s albumin = 0.9 g/dL and AG 9

\[ 9 + 2.5 \times (4 - 0.9) = 9 + 7 \]

Adjusted AG = 16
Etiology: Anion Gap Metabolic Acidosis

- Excess endogenous or exogenous acid
- MUDPILES
  - M: Methanol
  - U: Uremia
  - D: Diabetic Ketoacidosis
  - P: Propofol (Paraldehyde)
  - I: Ischemia, INH
  - L: Lactic acidosis
  - E: ETOH ketoacidosis / Ethylene Glycol
  - S: Salicylates / starvation ketoacidosis

- Massive Rhabdomyolysis – release of intracellular phosphate and sulfate
<table>
<thead>
<tr>
<th>Letter</th>
<th>Substance</th>
</tr>
</thead>
<tbody>
<tr>
<td>G</td>
<td>Glycols – Ethylene glycol and methanol</td>
</tr>
<tr>
<td>O</td>
<td>Oxoproline (most commonly associated with chronic acetaminophen ingestion)</td>
</tr>
<tr>
<td>L</td>
<td>L-Lactate (Increased anaerobic metabolism due to tissue hypoperfusion)</td>
</tr>
<tr>
<td>D</td>
<td>D-Lactate (Unusual form of lactic acidosis that can occur in patients with short gut syndrome or in DKA)</td>
</tr>
<tr>
<td>M</td>
<td>Methanol</td>
</tr>
<tr>
<td>A</td>
<td>Aspirin</td>
</tr>
<tr>
<td>R</td>
<td>Renal Failure</td>
</tr>
<tr>
<td>K</td>
<td>Ketoacidosis</td>
</tr>
</tbody>
</table>
Serum Osmolar Gap

- To effectively manage critically ill patients, it is vital to account for the reason a patient has an elevated anion gap.
- Six common poisonings/ingestions elevate the AG including salicylates, methanol, ethanol, ethylene glycol, cyanide and carbon monoxide.
- The osmolar gap is the difference between the calculated and measured osmolality.
- Measurement of the serum osmolar gap is useful in patients with a high AG metabolic acidosis, particularly when methanol or ethylene glycol poisoning is suspected.

Serum Osmolar Gap

- The osmolar gap is less sensitive in late presentations of these poisonings, since most of the parent alcohol may have already been metabolized and the osmolar gap may not be sufficiently sensitive to exclude a small ingestion.
- The increase in serum osmolar gap is usually less pronounced in ketoacidosis, lactic acidosis, and advanced chronic kidney disease and therefore the serum osmolarity and osmolar gap calculations are usually not done in these populations.

Sivilotti, Marco L A and Winchester, James F  Methanol and Ethylene Glycol Poisoning. UpToDate.  
(August 5, 2014)
Serum Osmolar Gap

- Calculated osmolarity:
  \[(1.86 \text{ [Na]} + \text{BUN}/2.8 + \text{Glucose}/18 + \text{Ethanol}/4.6)\]

- The osmolar gap is the *difference* between the calculated and measured osmolarity.
- Osmolar gap greater than 10 mOsm: ethanol, ethylene glycol, isopropanol and methanol are the most likely causes.

Non-Gap Metabolic Acidosis

- GI Losses of HC03\(^-\) (Diarrhea, fistulas)
- Renal HC03\(^-\) loss (Type II RTA)
- Renal dysfunction (Hypoaldosteronism, Type IV RTA)
- Ingestions – ammonium chloride, hyperalimentation fluids
- Dilutional metabolic acidosis
  - Usually due to administration of large volume saline
A Word About NaHCO3

• Direct treatment of acute metabolic acidosis with NaHCO3 is **not** indicated, **unless**:
  – pH < 7.10
  – Overt physiologic compromise is present
  – Excessive work of breathing is required to maintain pH > 7.2

• NaHCO3 is not indicated in DKA, even with pH < 7.10 due to the risks of developing rebound alkalosis or exacerbating hyperosmolality.
Metabolic Alkalosis

- pH > 7.45 with normal or ↑HC03
  1) Anion loss (chloride)
  2) HC03 gain
- GI Losses (vomiting, NG suction)
- Urinary losses
- Contraction alkalosis
- Correction of long-standing respiratory acidosis with invasive or non-invasive mechanical ventilation
- Rarely, due to regulation of K⁺ by exchanging H⁺ ions for K⁺ in tissues and kidneys resulting in H⁺ wasting (Cushing's, steroid therapy)
- Hypokalemia is often present and contributes to the cause and maintenance of the metabolic alkalosis

Respiratory Acidosis

- pH ↓ 7.35 with normal or ↑PaCO2
- Decreased ventilation
  - Acute
    - Exacerbation of severe asthma
    - PNA
    - Pulmonary edema
    - Post cardiac-arrest
    - Drug overdose
    - Administration of O2 in setting of chronic hypercapnia.
  - Chronic:
    - Chronic obstructive lung diseases
    - Neuromuscular diseases
    - Compensated – elevated PaCO2 with near normal pH
Symptoms of Respiratory Acidosis

- Somnolence
- Headache
- Restlessness
- Anxiety
- Tremors
- Delirium
Respiratory Alkalosis

- pH ↑ 7.45 with normal or ↓ PaCO2
- Increased ventilation
  - CNS Disturbances (Head injury, CVA)
  - Pain
  - Inappropriate mechanical ventilation
  - Drugs (salicylates, catecholamines, cocaine, meth, pcp, theophylline)
  - Hypoxemia
  - Liver Disease
  - Pulmonary receptor stimulation / ↓ lung compliance (PNA, pulmonary embolism, asthma, pulmonary fibrosis, pulmonary edema)
  - Pregnancy
Compensation Basics

- Compensatory changes are dependent upon normal function of the compensating system.
- Most compensation is incomplete.
- Lack of adequate compensation may indicate the presence of a co-existing secondary disorder.
- Different compensatory formulas are applied, depending upon the primary disturbance.
- Humans have good buffering systems for acidosis but a reduced ability to buffer alkalosis.
Respiratory Compensation

- PC02 changes in the same direction as the serum HC03
  - PC02 ↓ in metabolic acidosis
  - PC02 ↑ in metabolic alkalosis

- Respiratory compensation decreases the change in ratio of HC03:PC02, and therefore in the pH.

- Rapid onset of response, within 30 minutes
- Complete within 12-24 hours
- Respiratory compensation can occur to a physiologic max of PaC02 50 to 55; higher PaC02 in this situation indicates a concomitant Respiratory Acidosis
Renal Compensation

• HC03 changes in the same direction as the PC02
  – HC03 ↑ in respiratory acidosis due to increased H⁺ secretion
  – HCO3 ↓ in respiratory alkalosis due to decreased H⁺ secretion and urinary HC03 loss

• Slower onset of compensation, within hours
• Completion takes days to weeks for maximal effect
• Expected findings differ in acute (little or no renal compensation) and chronic (full renal compensation) respiratory acid-base disorders.

Acid-Base Nomogram
## Expected Compensation

<table>
<thead>
<tr>
<th>Primary Disorder</th>
<th>Primary Change</th>
<th>Compensatory Change</th>
<th>Expected Compensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic Acidosis</td>
<td>↓ HC03⁻</td>
<td>↓ PaC02</td>
<td>ΔPaC02 = 1.2 Δ HC03⁻</td>
</tr>
<tr>
<td>Metabolic Alkalosis</td>
<td>↑ HC03⁻</td>
<td>↑ PaC02</td>
<td>ΔPaC02 = 0.9 Δ HC03⁻</td>
</tr>
<tr>
<td>Respiratory Acidosis Acute</td>
<td>↑ PaC02</td>
<td>↑ HC03⁻</td>
<td>ΔHC03⁻ = 0.10 Δ PaC02&lt;br&gt;ΔHC03⁻ = 0.35 Δ PaC02</td>
</tr>
<tr>
<td>Chronic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiratory Alkalosis Acute</td>
<td>↓ PaC02</td>
<td>↓ HC03⁻</td>
<td>ΔHC03⁻ = 0.2 Δ PaC02&lt;br&gt;ΔHC03⁻ = 0.5 Δ PaC02</td>
</tr>
<tr>
<td>Chronic</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

# Metabolic Acidosis

<table>
<thead>
<tr>
<th>Primary Change</th>
<th>Compensatory Change</th>
<th>Compensation Formulas</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓ HC03⁻</td>
<td>↓ PaC02</td>
<td>ΔPaC02 = 1.2 Δ HC03⁻</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PaC02 = (1.5 x HC03) + 8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>*Winters Formula, estimates the expected PaC02 ±2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PaC02 = the last two digits of the pH</td>
</tr>
<tr>
<td></td>
<td></td>
<td>pH 7.25 → PaC02 25</td>
</tr>
</tbody>
</table>
## Metabolic Alkalosis

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<tr>
<td>↑HC03⁻</td>
<td>↑PaC02</td>
<td>ΔPaC02 = 0.9 Δ HC03⁻</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PaC02 = (0.9 x HC03) + 15</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PaC02 = 40 + .7(measured - normal HC03)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PaC02 ↑ 0.7mmHg for every 1mEq elevation in the serum HC03</td>
</tr>
</tbody>
</table>
# Respiratory Acidosis

<table>
<thead>
<tr>
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<th>Primary Change</th>
<th>Compensatory Change</th>
<th>Compensation Formulas</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ACUTE</strong></td>
<td>↑PaC02</td>
<td>↑HC03⁻</td>
<td>( \Delta HC03^- = 0.10 \Delta PaC02 )</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>( HC03 = \frac{(PC02 - 40)}{10} + 24 )</td>
</tr>
<tr>
<td><strong>CHRONIC</strong></td>
<td>↑PaC02</td>
<td>↑HC03⁻</td>
<td>( \Delta HC03^- = 0.35 \Delta PaC02 )</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>( HC03 = \frac{(PC02 - 40)}{5} + 24 )</td>
</tr>
</tbody>
</table>

- \( HC03 \uparrow \) 1 / 10mm Hg increase in PaC02
## Respiratory Alkalosis

<table>
<thead>
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<th>Primary Change</th>
<th>Compensatory Change</th>
<th>Compensation Formulas</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ACUTE</strong></td>
<td>↓ PaC02</td>
<td>↓ HC03⁻</td>
<td>ΔHC03⁻ = 0.2 Δ PaC02</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>HC03 = 24 - (40 – PaC02 / 5)</td>
</tr>
<tr>
<td><strong>CHRONIC</strong></td>
<td>↓ PaC02</td>
<td>↓ HC03⁻</td>
<td>ΔHC03⁻ = 0.5 Δ PaC02</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>HC03 = 24 - (40 – PaC02 /2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>HC03 ↓ 5 / 10 mmHg decrease in PC02</td>
</tr>
</tbody>
</table>

**Decrease in PC02**
Respiratory Compensation in Metabolic Acidosis

- Winter’s formula is used to evaluate respiratory compensation.
- If the PaC02 is < expected, then a concomitant respiratory alkalosis is also present.
- If the PaC02 is > expected, then a concomitant respiratory acidosis is also present.
- Normal or elevated PaC02 in the presence of severe metabolic acidosis may signal impending respiratory failure.
Winter’s Formula

\[(1.5 \times HC03) + 8 = PaC02 \pm 2\]

ABG: 7.22 / 30 / 10
Na 139, K 4.0, Cl 90, C02 10
\[(1.5 \times 10) + 8 = 23\]

Measured PaC02 30, expected PaC02 23
Conclusion – Concomitant respiratory acidosis is present
Acute Respiratory Alkalosis

\[ \text{HC03} = 24 - (40 - \text{PaCO2} / 5) \]

Mr. L ABG: 7.50 / 18 / 14

\[ 24 - (40 - 18 / 5) \]
\[ 24 - 22/5 \]
\[ 24 - 4.4 = 19.6 \]

Expected HC03 19.6, actual bicarb 14

Conclusion – Not compensated
# Finding Mixed Acid-Base Disturbances

<table>
<thead>
<tr>
<th>Method</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>At a glance: PC02 <strong>and</strong> HC03 both add to the pH change</td>
<td>Mixed acid-base disorder is present</td>
</tr>
<tr>
<td>Calculate the corrected HC03 Corrected HC03 = HC03 + ΔAG</td>
<td>Corrected HC03 &gt; 28 → Alkalosis Corrected HC03 &lt; 20 → Acidosis</td>
</tr>
<tr>
<td><strong>Calculate the Delta Ratio (Δ / Δ) = Δ Anion Gap/ Δ HC03⁻</strong></td>
<td>Δ / Δ = 1-2 → uncomplicated high AG metabolic acidosis Δ / Δ &lt; 1 → combined high AG metabolic acidosis and normal AG metabolic acidosis Δ / Δ &gt; 2 → combined high AG metabolic acidosis and concurrent metabolic alkalosis</td>
</tr>
</tbody>
</table>
Delta / Delta
ΔAG / ΔHC03⁻

Mr. L
ABG 7.50 / 18 / 14
BMP: Na  144, Cl 127, C02  8, AG =  9
    Adjusted AG = 16

16 – 10 / 24 – 14 =  6/10 = 0.6

Δ / Δ < 1  → combined high AG metabolic acidosis and normal AG metabolic acidosis
Delta / Delta
ΔAG / ΔHC03⁻

1) ABG: 7.26 / 15 / 14
   BMP: Na 130, Cl 90, C02 15, AG = 25
   \(25 - 10 / 24-14 = 15/10 = 1.5\)
   Δ/Δ 1-2 : Uncomplicated high AG metabolic Acidosis

2) ABG: 7.22 / 30 / 10
   BMP: Na 139, Cl 110, C02 10, AG = 19
   \(19 – 10 / 24 – 10 = 9/14 = 0.64\)
   Δ/Δ : Combined high AG metabolic acidosis and normal AG metabolic acidosis
Putting It All Together

• History and Physical:  Mr. L’s Clues
Hypotension, diarrhea, poor nutritional status, possible ingestion, loop diuretics, hypokalemia, tobacco use, chronic liver disease, pulmonary edema, elevated lactate

• Verify consistency of lab values
$\text{HC03 (ABG)} = \text{C02(venous electrolytes)} \pm 2 \text{ HC03 14, C02 on BMP 8 (Approx. 4hrs later)}$
• Is pH low, normal or high? 7.5, High → Alkalemia
• If both the Is the primary disturbance metabolic or respiratory?
  At first glance, respiratory is most severe.
• Is the disturbance acute or chronic? Likely Acute on Chronic
• Have compensatory changes occurred?
Acute Respiratory Alkalosis

$\text{HC03} = 24 - (40 - \text{PaCO2} / 5)$

Mr. L ABG: 7.50 / 18 / 14

$24 - (40 - 18 / 5)$

$24 - 22 / 5$

$24 - 4.4 = 19.6$

Expected HC03 19.6, actual bicarb 14

Conclusion – Not completely compensated

• Is the disturbance simple or mixed?
Alkalemia

High HC03
Low PC02

Mixed metabolic alkalosis and respiratory alkalosis

High Hc03
High PC02

Predominant Metabolic Alkalosis

Low HC03
Low PC02

Predominant Respiratory Alkalosis

Low HC03
High PC02

Not possible; likely lab error

Apply Compensation Rules

Measured PC02 too high
Mixed metabolic alkalosis and respiratory acidosis

Measured PC02 too low
Mixed metabolic alkalosis and respiratory alkalosis

Measured PC02 is appropriate
Simple metabolic alkalosis

Measured HC03 is too low
Mixed respiratory alkalosis and metabolic acidosis

Measured HC03 is too high
Mixed respiratory alkalosis and metabolic alkalosis

Measured HC03 is appropriate
Simple respiratory alkalosis
• Mr. L has a mixed Respiratory Alkalosis and Metabolic Acidosis

• Calculate the Anion Gap

\[ AG = (Na^+) - (HC0_3^- + Cl^-) = 144 - (8 + 127) = 9 \]

• Adjust the AG for hypoalbuminemia

\[ AG + 2.5 \times (\text{normal albumin g/dL} - \text{measured albumin g/dL}) \]

• Adjusted AG = 16

• Is there a secondary or tertiary disturbance present?

Calculate the Delta / Delta ratio
ΔAG / ΔHC03⁻

Mr. L
ABG 7.50 / 18 / 14
BMP: Na 144, Cl 127, CO₂ 8, AG = 9

Adjusted AG = 16

16 – 10 / 24 – 14 = 6/10 = 0.6

Δ / Δ < 1 → combined high AG metabolic acidosis and non-gap metabolic acidosis
• Mr. L has a Respiratory Alkalosis, a high AG Metabolic Acidosis and a Non-gap Metabolic Acidosis

• Likely Etiologies
  – Respiratory Alkalosis (Chronic liver failure, pulmonary edema)
  – High AG Metabolic Acidosis
    • Can you account for the AG? Does the osmolar gap need to be calculated?
    • Lactic acidosis 2/2 shock, malnutrition ketoacidosis
  – Non-gap Metabolic Acidosis (Diarrhea)
Summary of Key Points

• Systematic approach, use H&P clues
• Pick the most severe-appearing abnormality and start there
• The flow charts are your friends
• Account for the anion gap
• Apply compensation formulas – find the formulas that are easiest for you
• Use Delta / Delta to detect mixed disturbances
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