Chapter 36
Blood Gas I

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A 36-year-old woman presents to the emergency room with severe abdominal pain, nausea, vomiting, anorexia, and somnolence.

ABG: pH 7.20, PCO_2 35 mmHg, pO_2 68 mmHg on room air
Laboratory values: Na 130 mEq/L, Cl 80 mEq/L, HCO_3 10 mEq/L

1. How do you diagnose a simple acid–base disorder?
2. What blood gas abnormality does this patient have?
3. How do you calculate anion gap and corrected anion gap?
4. How do you diagnose a mixed acid–base disorder and does this patient have mixed acid–base disorder?
5. What is Winter's formula?
6. Is there any compensation in this blood gas value?
7. What are the possible causes of metabolic acidosis?
8. What are the possible causes of respiratory acidosis?

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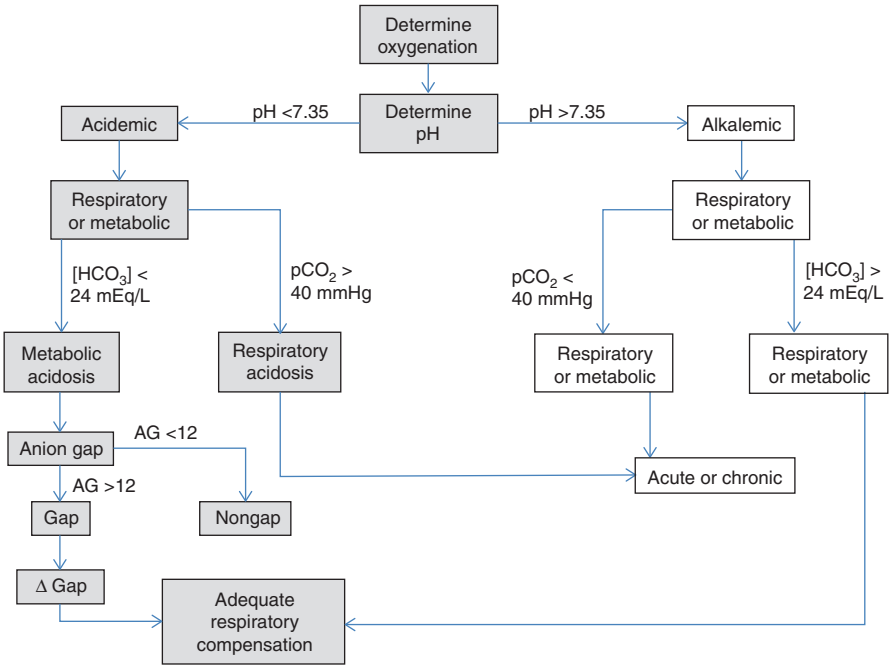

Fig. 36.1 Diagnosis of acid–base disorders (This figure was published in Miller textbook, Chap. 21 in 2011. Permission obtained from Elsevier to reproduce the image.)

Answers

1. Initially the pH is used to determine acidosis or alkalosis, and then the value of $PaCO_2/HCO_3$ is used to determine if the derangement is metabolic or respiratory. If it is of respiratory origin, then we will have to determine whether the process is acute or chronic. If it is due to a metabolic component, then respiratory compensation should be calculated using the appropriate formula.

2. Our patient has a pH less than 7.4, which signifies acidosis. The HCO_3 is less than 24 mEq/L; therefore the primary abnormality in this patient is metabolic acidosis. This chart (Fig. 36.1) shows the steps to follow in order to diagnose an acid–base disorder [1].

3. Anion gap (AG) = Na − (Cl + HCO_3)

   (a) AG is the difference in the 'routinely measured' **cations** (Na) and 'routinely measured' **anions** (Cl and HCO_3) in the blood and depends on serum phosphate and albumin concentrations [2]. Determination of AG is useful in determining the cause of acidosis [3, 4]. The normal value for serum AG is usually 8–12 mEq/L. In our patient, AG = 130 − (80 + 10) = 40 mEq/L. So, this patient has a high AG, most likely due to starvation or diabetic ketoacidosis.

(b) In a normal healthy patient, negatively charged albumin is the single largest contributor to the AG [5]. Hypoalbuminemia causes a decrease in AG; hence AG is corrected to albumin level using the equation of Figge as follows: corrected AG = AG + [0.25 × (44 – Albumin)] [6].

- If corrected AG >16, there is high AG acidosis.
- If corrected AG <16, non-AG acidosis.

4. Delta gap formula can be used to assess mixed acid–base disorder.

(a) Δ gap = AG − 12 + HCO_3 (12 is normal serum AG value)

- If Δ gap <22 mEq/L, then concurrent non-gap metabolic acidosis exists.
- If Δ gap >26 mEq/L, then concurrent metabolic alkalosis exists.

(b) In our patient, Δ gap = 40 − 12 + 10 = 38 mEq/L. So, there is a concurrent metabolic alkalosis probably from vomiting in addition to high AG metabolic acidosis in this patient.

So, there is a concurrent metabolic alkalosis probably from vomiting in addition to high AG metabolic acidosis in this patient.

5. Winter's formula is used to determine whether there is an appropriate respiratory compensation during metabolic acidosis [1].

(a) Winter's formula: $PCO_2 = (1.5 \times HCO_3) + 8$

- If measured PCO_2 > calculated PCO_2, then concurrent respiratory acidosis is present.
- If measured PCO_2 < calculated PCO_2, then concurrent respiratory alkalosis is present.

6. In our patient, calculated $PCO_2 = (1.5 \times 10) + 8 = 23$ mmHg according to Winter's formula.

Our measured PCO_2 of 35 mmHg is higher than the calculated PCO_2 of 23 mmHg, so our patient also has concurrent respiratory acidosis. Usually, metabolic acidosis is compensated by respiratory alkalosis, but due to somnolence in this patient, concurrent respiratory acidosis exists.

7. Causes of anion gap metabolic acidosis are easily remembered by pneumonic MUDPILES [1].

M: methanol
U: uremia
D: diabetic ketoacidosis
P: paraldehyde
I: infection, INH therapy
L: lactic acidosis
E: ethanol, ethylene glycol
S: salicylates (aspirin)

Causes of non-gap metabolic acidosis:

- Excessive administration of 0.9% normal saline
- GI losses: diarrhea, ileostomy, neobladder, pancreatic fistula
- Renal losses: renal tubular acidosis
- Drugs: acetazolamide

8. Respiratory acidosis which is from increased CO_2 is due either to increased production or decreased elimination [2].

   (a) Increased production of CO_2:

   - Malignant hyperthermia
   - Hyperthyroidism
   - Sepsis
   - Overfeeding

   (b) Decreased elimination of CO_2:

   - Intrinsic pulmonary disease (pneumonia, ARDS, fibrosis, edema)
   - Upper airway obstruction (laryngospasm, foreign body, OSA)
   - Lower airway obstruction (asthma, COPD)
   - Chest wall restriction (obesity, scoliosis, burns)
   - CNS depression (anesthetics, opioids, CNS lesions)
   - Decreased skeletal muscle strength (myopathy, neuropathy, residual effects of neuromuscular blocking drugs)
   - Rarely, an exhausted soda–lime or incompetent one-way valve in an anesthesia delivery system can contribute to respiratory acidosis.

References

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