BLOOD GAS ANALYSIS

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Abstract

Blood gas analysis is a diagnostic tool commonly used to evaluate the partial pressure of blood gases and acid-base content. Based on the source of the blood sample, there are three types of blood gas analysis: arterial blood gas analysis, venous blood gas analysis, and capillary blood gas analysis. The results of blood gas analysis of arterial and capillary blood samples have correlations. The values of pH, $HCO^{3,}$ and pCO_2 are interrelated, they can be substituted for one another. The clinical process causes the blood to become acidic or alkali. Acidosis is characterized by a decrease in HCO3- or an increase in pCO2, leading to an increase in H ions and a decrease in pH, and alkalotic condition characterized by an increase in HCO3- or a decrease in pCO2, leading to a decrease in H+ ions and an increase in pH.

Keywords: blood gas analysis, acidosis, alkalosis

INTRODUCTION

Blood gas analysis is a diagnostic tool and commonly used to evaluate the partial pressure of blood gases.[1,2] The most commonly measured and calculated variables are $PaCO_2$, PaO_2, and pH.[3,4] In addition, blood gas machines also calculate HCO^{3-} and base excess (BE).[5] Comprehension of blood gas analysis enables practitioners to assess the state of respiration, blood circulation, and metabolic disorders.[1,2] Blood gas analysis can be performed on blood obtained from anywhere in the circulatory blood system (arteries, veins, or capillaries).[2]

Based on the source of the blood sample, there are three types of blood gas analysis: arterial blood gas analysis, venous blood gas analysis, and capillary blood gas analysis.[7] Arterial blood gas analysis is the most accurate and is the gold standard for measuring paO_2, $paCO_2$, and pH.[8]

Taking blood samples through arteries or capillaries reveals the amount of gas present. The results of the assessment are expressed as partial pressures of oxygen (PO_2) and carbon dioxide (PCO_2) in kiloPascals (kPa) where the assessment corresponds to the concentration of these gases in the blood sample. The results of the assessment are also expressed as millimeters of mercury (mmHg) where 1 kPa = 7.5mmHg).[9]

Carbon dioxide (CO_2) and oxygen (O_2) are blood gases that generally bind to hemoglobin and affect the pH of blood including plasma. The pH of the body plays an important role in providing an optimal environment for body chemicals to work. Therefore, pH balance plays an important role in the continuity of metabolic processes so that the body must maintain its balance. The pH of a solution is expressed as the level of acidity or alkalinity depending on the concentration of H^+ ions. Plasma is slightly alkaline with a pH value of 7.32-7.4 for neonates and 7.35-7.45 for children.[9]

Body fluids contain a variety of buffers, which are the first line of defense against changes in pH. Based on their location in the body compartment, buffers are divided into two groups, namely extracellular buffers and intracellular buffers.[10]

Bicarbonic acid is the main buffer in the extracellular compartment of the body. The body can regulate bicarbonate levels through the kidneys and CO_2-levels through the lungs. Carbonic acid is a weak acid and easily dissociates to produce CO_2 which can be transported in dissolved form and expelled from the body through the lungs when breathing. The relationship between pH, HCO^3 and CO_2 in this system is described by the Henderson-Hasselbalch equation and the equation: $H^+ + HCO^3 \leftrightarrow H^2CO^3\ H_2O + CO_2$. Bicarbonate provides about 40% of the body's buffering capacity for metabolic acids with the remainder of the buffering capacity coming from intracellular buffers.[11] In premature infants, serum bicarbonate levels are relatively low, making them more susceptible to acidosis.[12]

Although buffers represent the first line of defense against changes in pH, they alone cannot maintain long-term acid-alkaline balance or accompanied by significant and acute changes in H^+ ion production. Therefore, other mechanisms are needed, namely renal and respiratory compensation. In primary metabolic disorders, the respiratory system provides compensation, while in primary respiratory disorders, the compensation is carried out by the renal system.[11]

The respiratory response occurs more rapidly (minutes-hours) than the renal mechanism, which takes about 3-4 days, with renal alkaline excretion more rapid than acid excretion.[11]

The kidneys prevent the loss of HCO^{3-} in the urine and maintain plasma

levels by excreting acid and producing new bicarbonate. Thus they can respond to acid-alkaline disturbances by acidifying or alkalizing the urine.[13]

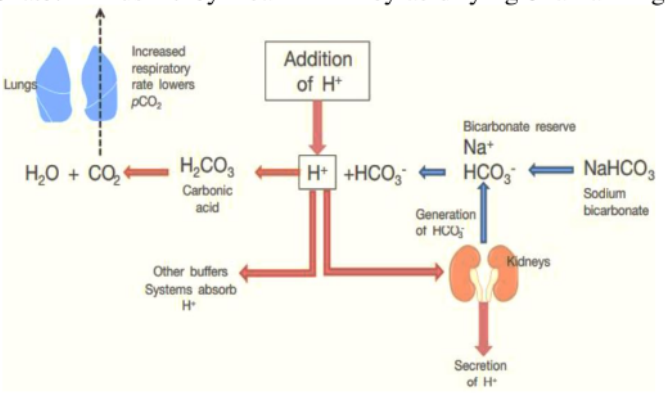

Figure 1. Human body's compensation system

Arterial blood gas analysis is the gold standard for assessing acid-alkaline and ventilation status, even though it causes discomfort and may lead to complications chance up to 11.3%.[7] In neonates it is safer to choose umbilical artery catheterization, although thrombosis and bleeding may occur.[14]

A sampling of venous blood gas for acid-alkaline assessment and gas exchange can avoid complications that may occur in arterial blood gas (ABG) examinations, besides that venous blood is more often available via venipuncture or insertion of intravascular access.[7,15]

VBG is not the main substitute for ABG, but can still be used when a venous catheter has been installed and blood gas analysis is needed as soon as possible to reduce mortality and morbidity, especially in conditions of septic shock or patients undergoing major surgery.[6,15] Therefore, VBG can be used as an initial assessment in emergency cases such as septic shock because it allows faster monitoring of outcomes.[16,17]

The results of blood gas analysis of arterial and capillary blood samples have correlations, and the values of pH, HCO^3, and pCO_2 are interrelated so that they can be substituted for one another, except for the arterial pO2 value which has a poor correlation with the capillary pO_2 value.[18]

DISCUSSION

The clinical processes that cause the blood to become acidic or alkalic are called acidosis or alkalosis. Acidosis and alkalosis refer to physiological processes that lower or increase pH, while acidemia and alkalemia only describe an abnormal state of blood pH.[19] Acidosis is caused by conditions that result in a decrease in HCO^{3-} or an increase in pCO_2, leading to an increase in H ions and a decrease in pH. Alkalosis is caused when the primary disturbance causes an increase in HCO^{3-} or a decrease in pCO_2, leading

to a decrease in H^+ ions and an increase in pH.[13]

1. Metabolic acidosis

Metabolic acidosis occurs due to an increase in the amount of exogenous acid entering the body, excess production of endogenous H^+ ions, inadequate H^+ excretion, or excessive loss of bicarbonate in the urine or feces.[13] Characterized by BE 5 mmol/L, PCO2=35– 45 mmHg, and pH<7.35. The anion gap is important for evaluating the cause of metabolic acidosis, namely the difference in the amount (gap) of the most abundant serum cations (Na+) and the amount of the two most abundant serum anions (HCO^{3-} and $Cl^{-)}$ in the serum.[12]

The increase of anion gap indicates an unmeasured increase in anions which can be caused by excess acid production or under acid excretion. Normal anion gap acidosis results from loss of bicarbonate. Cl^- reabsorption increases and becomes the main anion that accompanies Na so that the number of anions in the plasma remains normal. Therefore, normal anion gap acidosis is also known as hyperchloremic metabolic acidosis.[13]

In metabolic acidosis with a normal serum anion gap, hyperchloremia compensates for the loss of bicarbonate by the gastrointestinal tract (diarrhea, fistula or external drainage, short bowel syndrome) or by the kidneys (lack of acidification of urine by the renal tubules). Hyperchloremic metabolic acidosis with a normal serum anion gap can also be induced by large volumes of normal saline infusion as in low birth weight infants after inadvertent chloride administration via parenteral nutrition.[20]

Table 1. Differential diagnosis of metabolic acidosis

	No	Mnemonic	Causes
High anion gap metabolic acidosis	1	M	Methanol
	2	U	Uraemia (renal failure)
	3	D	Diabetic, alcoholic, starvation ketoacidosis
	4	P	Paracetamol, propylene glycol, paregoric
	5	I	Inborn errors of metabolism, iron, ibuprofen, isoniazide
	6	L	Lactic acid
	7	E	Ethylene glycol
	8	S	Salicylates (aspirin)
Normal anion gap metabolic acidosis	1	D	Diarrhea
	2	R	Renal tubular acidosis type I, II, IV (hypoaldosteronism), or medication-induced
	3	C	Chloride excess

Increased serum anion gap metabolic acidosis indicates an unmeasured increase in serum anions, either endogenous (lactate, ketone bodies, organic acids) or exogenous. The most common causes during the neonatal period are lactic acidosis due to perinatal hypoxia-ischemia, hemodynamic compromise during adaptation, septic shock, severe

respiratory distress syndrome, hypovolemia, or severe anemia.

2. Metabolic alkalosis

Blood gas analysis for metabolic alkalosis was determined by BE>5 mmol/l and pH>7.45, whereas PCO2 depended on the effectiveness of respiratory compensation. This is due to increased bicarbonate and/or excessive loss of H^+ ions. This is rare in neonates.[13,19]

The body responds to metabolic alkalosis through respiratory compensation where the respiratory rate decreases that causes the increase of PCO2. However, respiratory compensation is limited by severe hypoxia, so the respiratory compensatory response cannot normalize pH. The kidneys also respond to metabolic alkalosis by increasing alkaline excretion so that the urine pH increases to 8.5-9.0. Hypochloremia and hypokalemia usually occur due to increased urine loss. Alkalosis can worsen if there is a contraction of the ECF (extracellular fluid) and hypokalemia because it will increase bicarbonate reabsorption. Metabolic alkalosis can be treated by treating the underlying etiology. Measurement of urine chloride can help differentiate the cause of metabolic alkalosis. If the urine chloride level is less than 10 mEq/liter,

These cases can be treated with the administration of sodium chloride.[13] This disorder often results from excessive renal hydrogen ion loss due to prolonged use of diuretics (furosemide), which is usually associated with hypokalemia.[13,19]

If the metabolic alkalosis is caused by mineralocorticoid overactivity or potassium depletion, urine chloride levels may be increasing more than 20 mEq/liter and cause resistance to sodium chloride treatment.[13]

Another cause of metabolic alkalosis with hypokalemia and hypochloremia is loss of gastric fluid from vomiting or diarrhea. In the absence of these two etiologies, further investigation is needed. Bartter syndrome should be suspected when associated with severe metabolic alkalosis, dehydration, premature infants, and polyhydramnios[19]

3. Respiratory Acidosis

Blood gas parameters will show PCO2>45 mmHg and pH<7.35, while BE depends on the ability of renal compensation. Respiratory acidosis is the result of an increase in PCO_2 levels which causes an increase in H^2CO^3 and H ions followed by a decrease in pH. An increase in H^+ ions can be associated with an increase in CO_2 production or a decrease in CO_2 elimination through the respiratory system.[13,20]

Respiratory acidosis occurs either in an acute or chronic form affecting the respiratory or neurological systems. The rise in pCO_2 is initially buffered by non-bicarbonate, protein & phosphate buffers. If this increase continues, as in premature infants with chronic lung disease, the kidneys will respond by excreting H^+ ions while producing & reabsorbing bicarbonate. This causes the elevation of plasma bicarbonate

level but pH level returns to normal. This is the compensatory phase of respiratory acidosis that only begins to occur after a few hours to days.[13]

Increased CO_2 production can be caused by several causes. The main cause in children is an increase in cellular metabolic activity, which can be seen in infection or fever. Increased CO_2 production can also be associated with the body's carbohydrate load. This disorder is often caused by iatrogenic causes such as excessive parenteral nutrition above carbohydrates relative to body requirements. Given the body's ability to remove CO_2 and increase the respiratory rate to compensate for the increased CO_2 production. The increased excess CO2 production alone is not sufficient to cause respiratory acidosis unless the body's compensation is limited as in paralyzed patients.[20]

Respiratory acidosis often results from an inability to eliminate CO_2 from the bloodstream based on 3 body components: a neurological component (to recognizing the need to remove CO2), a musculoskeletal component (to physically moving the chest while ventilation), and an alveolar component (allows for the diffusion of CO2 out of the bloodstream). Failure of any of these components can lead to respiratory acidosis. Neurological causes include injury (eg. traumatic or stroke), seizures, narcotics, and other pharmacological agents that cause neurological depression. Musculoskeletal failure may result from an acute cause such as flail chest or chest wall edema or an underlying musculoskeletal disorder such as myasthenia gravis or muscular dystrophy.[20]

4. Respiratory alkalosis

Analysis of blood gases in respiratory alkalosis marked by PCO2 values <35 mmHg and pH>7.45, while BE depends on the effectiveness of renal compensation. This occurs with excessive lung CO_2 loss and results in a decrease in pCO_2 leading to an increase in pH.[13,20]

Respiratory alkalosis results from hyperventilation of any cause. Hyperventilation is the mechanism responsible for the reduction in arterial pCO2 in all cases of respiratory alkalosis.[19]

Respiratory alkalosis is much less common than other acid-alkaline disorders. The causes of primary respiratory alkalosis usually involve an increased respiratory rate due to toxins or a primary central nervous system event. Salicylate intoxication and hyperammonemia can cause hyperventilation. Anxiety and stress can also cause hyperventilation.[20] It is also often iatrogenic, associated with mechanical ventilation. Rapid pCO_2 reduction has been associated with periventricular leukomalacia and intraventricular hemorrhage, so timely intervention is essential. With a decrease in pCO_2, pH rises and rapid buffering occurs by the release of H ions to lower plasma bicarbonate. The kidneys also respond by increasing bicarbonate excretion which causes a decrease in plasma bicarbonate and a movement of pH values toward normal. Final correction is achieved with treatment of the underlying disorder.[13]

Table 2. Differential diagnosis of respiratory alkalosis

Mnemonic	Causes
A	Ammonia (urea cycle defect, hepatic encephalopathy, anxiety)
M	Medication (progesterone, salicylates)
I	Increased intracranial pressure
S	Sepsis
H	Hypoxemia, hyperthermia

5. Mixed Disorder

Under certain conditions, more than one primary disturbance of the acid-alkaline balance may occur simultaneously. It should be suspected if a compensatory response is found under the expected range of acid-alkaline assessment. For example, in respiratory distress syndrome or pneumonia with sepsis, respiratory acidosis (due to ventilation failure) and metabolic acidosis (due to lactic acidosis) may coexist. The respiratory disease prevents a compensatory decrease in pCO_2 and the metabolic component prevents a compensatory increase in plasma bicarbonate, resulting in a greater fall in pH. Similarly, in chronic lung disease with loop diuretics treatment, respiratory acidosis and metabolic alkalosis may occur. Thus plasma bicarbonate and pH were higher than expected. Thus plasma bicarbonate and pH were higher than expected. Patients with liver failure may develop metabolic acidosis and respiratory alkalosis, with a decrease in plasma bicarbonate & pCO2 and a slight change in pH.[13]

CONCLUSION

Blood gas analysis is a diagnostic tool to evaluate the partial pressure of gases in the blood and acid-alkaline content which is carried out based on history taking, physical examination, and laboratory parameters. Invasive blood gas assessment is divided into 3 types based on the source of the blood sample: arterial blood gas (ABG) analysis as the gold standard, and venous blood gas (VBG) analysis, and capillary blood gas (CBG) analysis. Its results include the values of pH, pCO2, pO2, HCO3, and base excess.

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