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ACID-BASE STATUS IN CAMELS-A COMPREHENSIVE REVIEW AND ANALYSIS

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ABSTRACT

The study was aimed to analyse healthy and diseased camels' acid-base and electrolyte profiles to understand how these variations can lead to metabolic, systemic and respiratory disorders. The normal blood pH of camels ranges between 7.35 and 7.45 (7.44 ± 1.04). If this range is exceeded or decreased, it may cause metabolic and respiratory acidosis alkalosis, which may hinder the normal physiological functions of the body organs. Acid-base disorders have been observed in association with severe diseases such as myocardial infarction, trypanosomiasis, tick paralysis, tick-borne diseases, helminthic infections, bent neck syndrome, barter syndrome, Gitelman syndrome, Liddle syndrome, Glucocorticoid-remediable aldosteronism, pneumonia, bronchitis, left ventricular contractility, asthma, syncope, interstitial lungs disease and Peripheral and circumoral paresthesia. It was concluded that these imbalances can impact the immune system function in camels and increase susceptibility to infections, while fluctuation in essential ion levels may cause paralysis by affecting muscle contractions and relaxation processes.

Key words: Acid-base stauts, camel

The term "acid-base balance" describes the acidity and alkalinity of blood needed to sustain biological functioning (Zhang *et al,* 2022).

Camels show their physiological adaptations for arid environments and maintain their acid-base homeostasis by keeping the blood pH within narrow limits, ranging from 7.35 to 7.45 (Zouari *et al,* 2020). Compared to other animal species, camels have well-developed respiratory and renal systems to conserve water and maintain electrolyte balance (Wilson, 2012). The camel's kidneys excrete excess acids or bases in urine, contributing to acid-base equilibrium. The diet, primarily consisting of dry vegetation, can influence their acid-base status. However, they are adapted to efficiently utilise these components, ensuring their acid-base balance remains within normal limits under typical feeding conditions (Fesseha and Desta, 2020).

Body function depends on maintaining the proper acid-base balance and the arterial blood pH and pCO_2 greatly impact the pH levels of intracellular and interstitial fluid (Asopa *et al,* 2021).

An artery's blood sample assesses the pH level, or acid-base balance, as it carries oxygen-rich blood from the lungs into the body. The effectiveness of the lungs' ability to take in air and convert it into blood is measured by an arterial blood gas (ABG) test.

The initial value to be studied in assessing acid-base disorders is pH from ABG. It is followed by defining a main disturbance, figuring out the serum anion gap and assessing compensation. Metabolic acidosis, respiratory acidosis, metabolic alkalosis and respiratory alkalosis are the four basic acid-base diseases (Stegeman *et al,* 2020).

In the normal physiological state, the blood pH of healthy dromedary camels is about 7.44 ± 1.04. Clinically, metabolic acidosis is characterised by a pH of less than 7.35 and a low HCO^{3-} level. The anion gap aids in identifying the root cause of metabolic acidosis (Asopa *et al,* 2021). Strong ion difference (SID) was used to assess acid-base status in healthy camels and for the diagnosis of metabolic acidosis (Elkhair and Hartmann, 2010).

Uremia, diabetic ketoacidosis and salicylate poisoning can all result in an increased anion gap metabolic acidosis. It mainly happens in working camels as a result of concentrated overfeeding. Over the past few years, this ailment has become one of camels' most prevalent digestive issues. Acidotic camels display tympany, doughy rumen, anorexia, dehydration and recumbency (McCaffrey and Allinson, 2021).

Metabolic alkalosis in camels can be brought on by increased serum HCO^{3-} concentrations

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(overabundance of feed high in alkali), prolonged vomiting, or respiratory disorders, which impacts their acid-base equilibrium and lower arterial blood pH and shift it into the alkaline range (Elkhair *et al,* 2018).

When there is excess CO_2 in the blood, the blood pH drops, leading to respiratory acidosis in camels. Several environmental factors, including heat, dehydration, a high-fibre diet, exercise and heat stress, may contribute to respiratory acidosis in camels. Serum sodium, creatinine, urea and plasma hormone levels are significantly altered by dehydration, which affects the levels of ABGs (Mohamed *et al,* 2021).

Although camels' special digestive system allows them to absorb nutrients effectively, the fermentation of fibre can release volatile fatty acids and CO_2 that can enter the circulation. Respiratory acidosis can result from increased CO_2 generated by intense physical activity (Tharwat, 2021).

Respiratory alkalosis in camels occurs when the partial pressure of CO_2 decreases, leading to increased blood pH. It can be caused by panting, stress, hyperventilation at high altitudes, or pain or anxiety. Camels are adapted to hot and arid environments and their rapid breathing can result in excessive CO_2 loss (Elsayed, 2020).

This review study was aimed to examine healthy and diseased camels' acid-base and electrolyte profiles and to investigate how variations can contribute to metabolic, systemic and respiratory disorders.

Literature Review

Studies examining blood gases, acid-base and electrolyte profiles have demonstrated that the acid-base state in healthy dromedary camels varies with age. In comparison to older calves (11–21 days), younger calves (1-4 days) have lower potassium (K^+), higher sodium (Na^+) and higher chloride (Cl^-) concentrations. Older calves also have lower haemoglobin oxygen saturation (sO_2) levels and greater partial pressure of carbon dioxide (pCO_2). These results indicate an age-related influence on these parameters in healthy dromedary calves' first three weeks of life (Osman *et al,* 2023).

Subsequent investigations highlight the significance of trace elements for camel productivity and health, including copper, zinc, iron and selenium. Due to the potential effects of trace mineral toxicities or deficiencies on camel growth, metabolism and productivity, it is important to assess and maintain these (Abdelrahman, 2022).

Studies have indicated that blood pH in dehydrated or exercised camels stays within the normal range despite these stressors, demonstrating the critical function the acid-base balance plays in camel health. Camel survival in the face of stressors such as exercise and dehydration depends on maintaining the acid-base balance.

The blood pH remains within the usual range even in camels devoid of water, demonstrating the adaptability of camels to such circumstances. This equilibrium is essential to camels' overall physiological performance and homeostasis, enabling them to adjust to external stresses and preserve their health despite difficult conditions (Abdoun *et al,* 2012; Okab *et al,* 2012).

In camels, acid-base imbalances can result in Myocardial infarction (MI). Numerous factors that alter acid-base homeostasis in camels may give rise to MI. Studies indicated that modifications in strong electrolytes like sodium, potassium and chloride, as well as weak acids like proteins and phosphate, may be the cause of disruptions in the acid-base state of camels, including hyperchloraemic acidosis and hypoproteinaemic alkalosis (Elkhair and Hartmann, 2010).

The hallmark of metabolic acidosis is a drop in blood pH, which can bring several abnormalities. Furthermore, camel dehydration has been demonstrated to affect their acid-base balance, underscoring the complex connection between water deprivation and the physiological difficulties of these animals (Abdoun *et al,* 2012).

Acid-base imbalances can bring on several dangerous disorders. Conditions including uremia, diabetic ketoacidosis and significant loss of bicarbonate ions through the gastrointestinal system owing to diarrhoea can result in metabolic acidosis, which is defined by a lack of HCO_3^-, leading the blood to be too acidic (Seifter and Chang, 2017).

Conversely, disorders such as Cushing's disease and consuming large amounts of HCO_3^- or antacids can result in metabolic alkalosis, an excess of HCO_3^- that makes the blood excessively alkaline. These acid-base abnormalities can majorly impact health and may be a factor in the onset or aggravation of certain diseases (Seifter and Chang, 2017).

An important factor in camel trypanosomiasis is acid-base imbalances. Studies on *Trypanosoma evansi*-infected camels have demonstrated that these animals can have severe parasitemia, which can cause physiological alterations that impact acid-base

balance (Ahmadi-hamedani *et al,* 2014). Furthermore, research has demonstrated the effect of Trypanosoma infection on the oxidative status in camel blood, demonstrating notable alterations in antioxidant levels between infected and uninfected animals (Darwish *et al,* 2023).

Additionally, studies have shown that normocytic and normochromic anaemia, lymphocytosis and changes in acute-phase proteins like alpha-1 acid glycoprotein can occur in camels infected with trypanosomiasis. These findings highlight the intricate interactions between the disease and haematological parameters that affect the acid-base status of infected camels.

An acid-base imbalance and altered ABGs levels can exacerbate urinary tract infections (UTIs) and other urinary disorders, including cystitis, urine retention, hydronephrosis, red urine, renal masses, ruptured bladder and ruptured urethra in camels. Studies reveal that oxidative stress—associated with an acid-base imbalance—contributes to UTIs in dromedary camels.

A disruption in the normal redox state of cells caused by an acid-base imbalance can have harmful effects by producing reactive oxygen species (ROS), which can cause tissue damage and malfunction. The UTIs in camels may partly be caused by this oxidative stress-related disruption of the redox state (El-Deeb and Buczinski, 2015; Tharwat, 2023).

Acidosis and alkalosis in camels can result from electrolyte abnormalities through various pathways. Strong ion acidosis in the event of acidosis can be caused by certain electrolyte imbalances, such as hyponatremia (reduction in strong cation concentration) or increases in strong anions, such as L-lactate, D-lactate and ketoacids. High protein concentrations such as phosphate, albumin and globulin can also cause nonvolatile buffer ion acidosis. These abnormalities may cause metabolic acidosis in camels, detrimental to their acid-base equilibrium.

In camel calves, hyper D-lactatemia may result in metabolic alkalosis without dehydration. In addition, diseases such as pneumonia, severe pulmonary emphysema, depression of the respiratory center and left-sided heart failure can cause respiratory alkalosis. Alkalosis can result from these electrolyte imbalances and disruptions because they can upset the camels' acid-base balance (Ali *et al,* 2012; Constable *et al,* 2017).

Electrolyte abnormalities in camels are frequently caused by diseases and treatments that disrupt the body's normal fluid balance. The most prevalent electrolyte imbalance, hyponatremia, can be caused by several diseases that result in elevated antidiuretic hormone (ADH) levels and decreased circulating blood volume, such as hepatic cirrhosis and congestive heart failure. Conversely, hypernatremia is usually brought on by an excess of hypertonic saline, certain drugs such as lithium and excess fluid loss through the skin and gastrointestinal tract (Faye *et al,* 2018).

Methodology

Search Study

This review and literature search considered the most current publications and online abstracts on acid-base status in healthy and ill camels. Google Scholar, Springer, PubMed, Medscape, Medline and Science Direct were used in order of priority. The terms were carefully chosen from the research published between 2010 and 2024.

These keywords included were "Acid-base imbalance in camels," "Metabolic and respiratory acidosis in camels," "Metabolic and respiratory alkalosis in camels," "Electrolyte imbalance," and "ABGs and pH level in camels".

Selection criteria

Studies of camels who underwent acidosis and alkalosis due to acid-base, ABGs and electrolyte level imbalance included the identification of haemostatic parameters that are associated with acidic and basic conditions of the body and to reduce the rate of systemic and metabolic acidosis/ alkalosis, caused by environmental elements fluctuation. These studies were selected by following the inclusion and exclusion selection criteria.

Inclusion criteria

The study assessed relevance by including peer-reviewed articles from 2010-2024, reviews on metabolic acidosis and alkalosis and studies on hemostatic strategies for maintaining acid-base levels in camels to enhance robustness and replicability.

Exclusion criteria

The study excluded articles published in languages other than English due to unclear reporting of relevant information and ambiguity in its abstract.

Metabolic Acidosis

Hepatic Lipidosis

Metabolic acidosis in camels, particularly impacting pregnant and lactating camels, can result

in hepatic lipidosis, a disorder characterised by the buildup of hepatic fat in the liver and has been commonly seen in llamas and alpacas due to other metabolic abnormalities (Anderson *et al,* 1994; O'Conor Dowd, 2014).

Metabolic acidosis in camels leads to high fatty acid mobilisation, hepatic triglyceride synthesis and low-density lipoprotein secretion, causing hyperlipemia. Symptoms include lethargy, hypercholesterolemia, recumbency, ketonuria, weight loss and azotemia, causing liver dysfunction, muscle damage and anorexia (Foreman, 2019; Saun, 2023).

Helminth Infections

Metabolic acidosis can increase helminth infections by affecting the host's immune response and metabolic homeostasis, potentially impairing the host's ability to fight them. However, if it interferes with this immune response, it can create a favourable environment for helminth survival and growth (Wiria *et al,* 2014 and Kokova *et al,* 2021).

Tick paralysis

Metabolic acidosis has been observed in association with tick paralysis, in which ticks generate neurotoxins during feeding, gradually paralysing the host. Metabolic acidosis can exacerbate the neurological symptoms of tick paralysis by compromising the host's immune system and physiological homeostatic mechanisms (Cope, 2018).

A camel's physiological environment can drastically change by metabolic acidosis, impacting intracellular calcium levels and potassium channels. Muscle contraction and neuromuscular transmission are intimately related to variations in intracellular calcium concentrations. Tick neurotoxins alter potassium channels, influencing intracellular calcium levels and causing an imbalance that obstructs regular cellular functions. Increased intracellular calcium levels from abnormal potassium channel function can interfere with synaptic transmission, weaken muscles and ultimately cause the paralysis that tick-infested hosts experience (El-Aly *et al,* 2024; Chand *et al,* 2016).

Bent neck syndrome

Acidosis inhibits muscle function and disrupts the electrolyte balance, which can result in weakening and unusual spasms of the muscles. These muscular anomalies might show up as lateral deviation of the neck in bent-neck syndrome due to muscle imbalances and weakening brought on by metabolic disruptions (Al-Sobayil and Mousa, 2009).

Trypanosomiasis

Changes in pH resulting from metabolic acidosis could affect the production of cytokines and immune cell activity. These two factors are critical for building an effective response against infections. A weakened immune system may result from metabolic disorders and nutritional changes due to acidosis, leaving the host more vulnerable to parasite diseases like trypanosomosis; this causes the haematocrit count, haemoglobin and red blood cell count to decrease and leads to anaemia (Baldissera *et al,* 2015; de Aquino *et al,* 2021; Tharwat, 2021).

Metabolic alkalosis

Bartter syndrome

Bartter syndrome can lead to metabolic alkalosis in camels, a rare kidney disease characterised by potassium wasting, salt-wasting nephropathy and abnormal electrolyte levels that compromise renal tubular function and acid-base hemostasis, which is typified by elevated plasma HCO_3^- concentration and systemic pH (Heilberg *et al,* 2015; Mabillard and Sayer, 2018).

Hyperplasia of biliary epithelium

In camels, metabolic alkalosis can impair liver function, which may impact the control of bile secretion and production. Biliary epithelial cells may proliferate and multiply more readily in the liver due to cellular reactions brought on by metabolic alkalosis. Metabolic alkalosis-induced pH imbalances can affect gene expression and cellular communication pathways in the liver, possibly resulting in biliary epithelial hyperplasia. The biliary epithelium may thicken and expand due to this aberrant cellular response (Tharwat, 2020).

Gitelman syndrome

Electrolyte abnormalities like those seen in Gitelman syndrome in camels may be brought on by metabolic alkalosis. Metabolic alkalosis-induced pH imbalances can impact the kidneys' ability to reabsorb electrolytes like magnesium and potassium and decreased excretion of calcium. In camels with metabolic alkalosis, these electrolyte imbalances can lead to hypokalemia and metabolic alkalosis, which are important aspects of Gitelman syndrome (Mabillard and Sayer, 2018).

Liddle syndrome

Because of a mutation in epithelial sodium channels (ENaC) caused by metabolic alkalosis, the channel is always present on the apical membrane

of renal tubular cells, preventing its destruction by the ubiquitin-proteasome system. Raised ENaC levels on the membrane cause increased water retention, salt resorption and a condition similar to hyperaldosteronism. Increased salt reabsorption contributes to hypertension and abnormal electrolyte levels associated with Liddle syndrome (Rodby, 2023).

Glucocorticoid-remediable aldosteronism (GRA)

Metabolic alkalosis can upset electrolyte balance and acid-base homeostasis, which may impact aldosterone synthesis. Under the control of ACTH, ectopic aldosterone synthase activity in the adrenal cortex results from a chimeric gene duplication in GRA. Increased aldosterone production is a result of this dysregulation, which also contributes to the hypertension and electrolyte abnormalities typical with GRA (Halperin and Dluhy, 2014).

Respiratory acidosis

Pneumonia and Bronchitis

Decreased lung function caused by respiratory acidosis makes it more difficult for the camel to clear infections from its respiratory system. Increased body fluid acidity can also impede an ineffective defense against disease. Due to the combination of decreased immunity, compromised lung function and favorable conditions for bacterial proliferation caused by respiratory acidosis, the risk of pneumonia in camels has significantly increased (Nahed *et al,* 2016).

Reduced left ventricular contractility

Reduced actin-myosin interactions are necessary for the heart muscle to contract efficiently. Decrease calcium binding to troponin C, a crucial regulatory protein in muscle contraction, is linked to decreased left ventricular contractility during respiratory acidosis. Reduced left ventricular contractility results from the heart's diminished capacity to contract strongly due to these problems with calcium management and contractile mechanisms.

Additionally, despite the lower left ventricular contractility, respiratory acidosis causes hemodynamic alterations, such as increased venous return (which equals cardiac output). This compensatory mechanism maintains cardiac output despite the detrimental effect on contractility by raising heart rate and lowering systemic vascular resistance (Tharwat *et al,* 2014).

Asthma

Asthma exacerbations resulting in respiratory acidosis can cause hypercapnia, which can be made worse by therapeutic oxygen treatment. It can also alter the acid-base balance and elevate transcutaneous pCO_2 levels.

Respiratory acidosis, a common symptom of severe asthma, can lead to hypoxemia, circulatory compromise and lactic acidosis. Chronic hypocapnia can exacerbate acid-base imbalances, causing non-anion gap acidosis. Severe asthma exacerbations can also result in metabolic acid-base abnormalities, such as high anion gap or non-anion gap metabolic acidosis (Vasileiadis *et al,* 2019).

Respiratory alkalosis

Interstitial lungs disease

Respiratory alkalosis, a disorder causing excessive breathing and low blood carbon dioxide levels in camels can lead to interstitial lung disease due to its impact on the lungs. It can cause changes in blood pH, electrolyte levels and oxygen and carbon dioxide levels, potentially affecting lung tissue integrity and contributing to interstitial lung disease. It can also cause vasodilation and damage to fragile interstitial tissue (Brinkman and Sharma, 2018).

Syncope

In respiratory alkalosis, a drop in $paCO_2$ and an increase in pH levels that follow might impact cerebral blood flow and cause cerebral vasoconstriction; however, it is a rare condition reported in camels. Brain blood flow changes can cause syncope, dizziness and mental disorientation, among other neurological symptoms. Reduced cerebral blood flow from the lower $paCO_2$ levels may result in syncope or a brief loss of consciousness (Kohli *et al,* 2021).

Peripheral and circumoral paresthesia

Blood-ionised calcium levels can be impacted by respiratory alkalosis, characterised by a drop in $paCO_2$ and a rise in the following pH levels. Reduced levels of ionised calcium (Ca^{++}) in the extracellular fluid can result from alkalosis due to enhanced protein binding. This decrease in ionised calcium can have an impact on nerve excitability and function, resulting in symptoms such as circumoral paresthesia (tingling around the mouth) and peripheral paresthesia (tingling or numbness in the limbs), a very common symptom reported in respiratory alkalosis (James and Evans, 2023).

Discussion

Animals maintain homeostasis through physiological adjustments, including maintaining

acid-base balance. Blood function relies on maintaining this balance, with arterial blood pH and pCO_2 significantly influencing intracellular and interstitial fluid pH levels.Anion gap analysis can be used to detect metabolic acidosis, which is defined by a pH below 7.35 and low HCO_3^- levels. Healthy camels have a normal blood pH of 7.44 ± 1.04. Increased serum HCO_3^- concentrations, prolonged vomiting, or respiratory conditions cause metabolic alkalosis in camels, which affects their acid-base equilibrium and lowers arterial blood pH.

Heat, dehydration, a high-fibre diet, exercise, heat stress and respiratory disorders can all affect camels' blood levels of CO_2, which can change their plasma hormone levels, creatinine, urea and salt levels. It can result in respiratory acidosis in camels. Because of their high blood pH, quick breathing and reduced pCO_2, camels suffer respiratory alkalosis, which leads to significant CO_2 loss in hot and dry conditions.

Camel health depends on the proper acid-base balance, which maintains normal blood pH levels in the face of stresses like exercise and dehydration and ensures the animals' survival. Acid-base imbalances in camels, such as hypoproteinaemic alkalosis and hyperchloraemic acidosis, can result in alterations in strong electrolytes and weak acids, which can cause myocardial infarction.

Illnesses and treatments frequently disrupt fluid balance, leading to electrolyte imbalances in camels. Hypernatremia is brought on by excessive saline, lithium and fluid loss, whereas hyponatremia is typically caused by high ADH levels and decreased blood volume.

Strong ion acidosis brought on by hyponatremia or elevated anions and nonvolatile buffer ion acidosis brought on by high protein concentrations are two examples of electrolyte imbalances that can result in acidosis and alkalosis in camels. Reactive oxygen species (ROS) are produced when an acid-base imbalance damages and malfunctions tissue. The UTIs in camels may be caused by this disturbance linked to oxidative stress.

Metabolic acidosis in camels, especially in pregnant and lactating mothers, can lead to hepatic lipidosis, a liver fat buildup disorder triggered by high fatty acid mobilisation, elevated triglyceride synthesis and low-density lipoprotein secretion. It can impair the host's physiological homeostatic mechanisms and immune system; symptoms may arise; therefore, it increases the susceptibility to various pathogenic infections such as helminthic infections, tick-borne diseases, trypanosomosis and tick paralysis, a condition in which the host becomes paralysed as a result of neurotoxins that ticks release while eating.

In camels, metabolic alkalosis can affect bile secretion and production and the rare kidney disease Bartter syndrome. Biliary epithelial hyperplasia may cause the liver to thicken and enlarge. Moreover, metabolic alkalosis can contribute to electrolyte abnormalities like those seen in Gitelman syndrome by impairing the kidneys' capacity to reabsorb electrolytes like potassium and magnesium, resulting in hypokalemia and metabolic alkalosis.

Camels with respiratory acidosis suffer from impaired lung function, which may compromise their immunity to infections and increase their risk of pneumonia. Additionally, pneumonia is exacerbated by decreased left ventricular contractility and calcium binding to troponin C. Hypercapnia can result from asthma flare-ups and can get worse when oxygen therapy is administered. Acid-base abnormalities, hypoxemia and metabolic acid-base imbalances can also be caused by severe asthma.

Interstitial pulmonary diseases (IPDs) can result from respiratory alkalosis, a condition that causes camels to breathe excessively and have low blood carbon dioxide levels. The IPDs affect blood pH, electrolyte levels, O_2 and CO_2 levels, potentially affecting lung tissue integrity. Additionally, IPDs may affect cerebral blood flow, leading to syncope, vertigo and confusion. The IPDs also alters the levels of blood-ionised calcium, which excites nerves and produces symptoms including peripheral and circumoral paresthesia.

The acid-base status of camels is a complex issue due to the lack of published studies and the inability to accurately evaluate and compare findings.

Conclusion

This review study comprehensively analysed the acid-base status in healthy and diseased camels, revealing a complex interaction between ABGs, electrolytes, acidity and alkalinity. These interactions can lead to or exacerbate serious disorders in camels, such as metabolic and respiratory acidosis and alkalosis. Furthermore, these imbalances can impact the immune system, potentially increasing susceptibility to pathogenic infections. Additionally, fluctuating levels of essential ions may contribute to paralysis by affecting the smooth and skeletal muscle contractions and relaxation processes.

Recommendations

Comparative studies should analyse acid-base state variations in healthy camels and those with respiratory or renal dysfunction diseases. Long-term research should observe acid-base balance during physiological phases, using metabolic profiling to identify biomarkers and examining breed-specific variances and genetic variants.

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Conflict of Interest

Author declares no conflict of interest.

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