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Prevalence and Severity of Serum Bicarbonate Deficiency in Canine Acute and Chronic Kidney Disease: A Retrospective Study

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ABSTRACT

A lack of serum bicarbonate frequently occurs in people suffering from acute (AKI) or chronic (CKD) kidney disease as a result of disrupted renal synthesis and reabsorption processes. Although alkali therapy is commonly used in both human and animal CKD treatment, information about how often such imbalances appear in canine AKI and CKD remains limited. This study aimed to determine how prevalent and how severe bicarbonate depletion is among dogs diagnosed with AKI, acute-on-chronic kidney disease (ACKD), or CKD, and to explore possible links with IRIS classification and calcium–phosphate metabolism changes. Medical records from dogs evaluated at the Nephrology and Urology Service of the Veterinary Teaching Hospital, University of Pisa, between January 2014 and January 2022 were retrospectively reviewed. Serum bicarbonate levels below 22 mmol/L were considered deficient, with moderate cases defined as 18–22 mmol/L and severe ones as <18 mmol/L. Among 521 dogs, 397 (76%) had reduced bicarbonate, including 142 (36%) with moderate and 255 (64%) with severe deficiency. The AKI and ACKD groups showed significantly greater occurrence ($p = 0.004$) and severity ($p = 0.02$) compared with CKD dogs. In these groups, serum bicarbonate had an inverse relationship with creatinine, urea, and phosphate levels. The frequency of deficiency rose in advanced stages in AKI ($p = 0.01$), ACKD ($p = 0.0003$), and CKD ($p = 0.009$). Dogs with calcium–phosphate product (CaxP) ≥70 mg^2/dL^2 were more often and more severely affected ($p = 0.01$ for both) than those with lower CaxP values. These results suggest that bicarbonate shortage is highly common in AKI, ACKD, and CKD, worsening with disease progression. The stronger impact seen in AKI and ACKD may stem from rapid renal decline or non-renal factors. The observed association with CaxP disturbances indicates a likely link between metabolic acidosis and mineral–bone imbalance.

Keywords: Bicarbonate, Dog, AKI, CKD, ACKD, Renal failure

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Introduction

In normal conditions, kidneys are essential for keeping acid–base homeostasis by reclaiming and producing bicarbonate within renal tubules. With chronic kidney disease (CKD), reduced functioning tissue leads to a gradual onset of metabolic acidosis.

Although proper diagnosis includes evaluating both blood pH and bicarbonate, such analyses are not always feasible in routine practice. Because serum bicarbonate measurement is typically part of biochemical testing for renal disorders, it can act as a substitute marker for acid–base balance. Concentrations below 22 mmol/L are usually interpreted as indicative of metabolic acidosis. This imbalance is known to be frequent in both people and animals with CKD, especially in advanced stages [1, 2]. Among uremic patients, metabolic acidosis contributes to muscle wasting and bone loss [3].

In human CKD, bicarbonate below 22 mmol/L has been linked with faster disease progression and higher death risk [4]. Therefore, alkali treatment is frequently applied in veterinary and human settings to limit the harmful consequences of acidosis on muscle and skeletal systems [1, 5]. However, there is still minimal information regarding how commonly bicarbonate deficiency occurs in dogs with AKI and CKD, especially in early phases.
The current study's purpose was to determine how often and how severely bicarbonate deficiency occurs in dogs with AKI, ACKD, and CKD, and to assess its association with IRIS grade/stage and calcium–phosphate irregularities.

Materials and Methods

A retrospective analysis was performed using records of dogs diagnosed with AKI, ACKD, or CKD that were admitted to the Nephrology and Urology Department of the "Mario Modenato" Veterinary Teaching Hospital, University of Pisa, from January 2014 to January 2022. Case information was obtained from the hospital's digital archive (OCIROE).
Dogs were allocated to AKI, ACKD, or CKD categories based on historical data, physical findings, lab results, and ultrasound imaging.

- AKI was assigned to cases fulfilling the following: (1) a sudden appearance of clinical signs consistent with AKI (such as anorexia, vomiting, or lethargy); (2) no sonographic evidence of long-term renal disease (e.g., reduced cortico-medullary definition, decreased kidney size, renal asymmetry, cysts, or cortical echogenicity increase); and (3) absence of previous azotemia (serum creatinine <1.4 mg/dL or SDMA <18 µg/dL).
- CKD classification was applied to dogs with historical, lab, or imaging features indicating chronic kidney impairment without an acute decline during the preceding three months.
- ACKD diagnosis required: (1) acute development of AKI-compatible symptoms, and (2) either pre-existing azotemia (serum creatinine >1.4 mg/dL or SDMA ≥18 µg/dL) and/or imaging findings typical of chronic renal changes.

Serum profiles used in the study included creatinine, urea, SDMA, total and ionized calcium, phosphate, calcium–phosphate product (CaxP), and bicarbonate. Blood was obtained from the jugular, cephalic, or saphenous veins using 2.5 mL methacrylate tubes, centrifuged within 15 minutes, and analyzed using a SAT 450 automated chemistry analyzer (Assel, Rome, Italy).
Samples were excluded if: (1) any biochemical parameter was missing, (2) they were collected post-dialysis, or (3) dogs were already receiving oral or IV sodium bicarbonate therapy. Information retrieved from each included record covered diagnosis (AKI, ACKD, CKD), age, breed, body weight, diet, and administration of phosphate binders or bicarbonate supplements.
CKD cases were stratified according to the IRIS staging system (stages 1–4) using serum creatinine and/or SDMA. AKI and ACKD were graded by IRIS grades 1–5 [6]. Serum bicarbonate <22 mmol/L was considered decreased; 18–22 mmol/L indicated moderate deficiency, and <18 mmol/L indicated severe deficiency. Abnormal CaxP values were defined as ≥70 mg^2/dL^2.

Statistical analysis
The distribution of continuous variables was evaluated using the Kolmogorov–Smirnov test. Because several parameters deviated from normality, continuous data were expressed as medians with minimum and maximum values. Comparisons among AKI, ACKD, and CKD groups were conducted with the Kruskal–Wallis test. The Fisher's exact test assessed differences in the rate and severity of bicarbonate deficiency across diagnostic categories, IRIS grades/stages, and between dogs with normal versus high CaxP levels. Median serum bicarbonate concentrations between normal and abnormal CaxP groups were compared with the Mann–Whitney U test. Associations between serum bicarbonate and creatinine, urea, phosphate, total calcium, ionized calcium, and CaxP were determined using Spearman's rank correlation. All analyses were performed with GraphPad Prism®.

Results and Discussion

A total of 610 case records were identified for dogs diagnosed with AKI (n = 135), ACKD (n = 191), and CKD (n = 284). Exclusions were made for 85 post-dialysis samples, 10 records with missing data, and nine dogs already receiving intravenous sodium bicarbonate, resulting in 521 cases for final analysis.
Cases were divided as follows: AKI (n = 100), ACKD (n = 144), and CKD (n = 277).

- AKI group: intact females (n = 17), spayed females (n = 18), intact males (n = 61), neutered males (n = 4); median body weight 15 kg (4.6–40 kg) and median age 5 years (1–17 years).
- ACKD group: intact females (n = 32), spayed females (n = 55), intact males (n = 50), neutered males (n = 7); median body weight 21 kg (2.9–34 kg) and median age 8 years (1–20 years).
- CKD group: intact females (n = 63), spayed females (n = 64), intact males (n = 129), neutered males (n = 21); median body weight 16 kg (2.5–30.6 kg) and median age 10 years (1–19 years).

The pattern of bicarbonate deficiency in AKI, ACKD, and CKD dogs across various IRIS grades and stages is displayed in **Figure 1**.

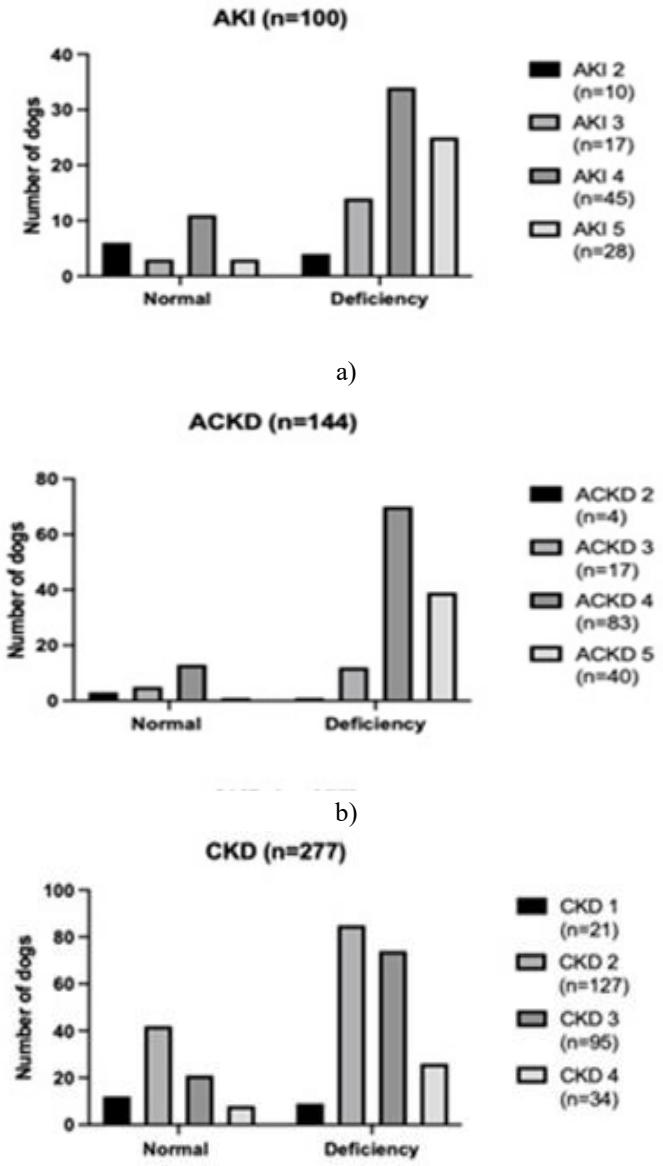

Figure 1. Overview of bicarbonate profile distribution in canines diagnosed with AKI, ACKD, and CKD across distinct clinical grades and disease stages.

A renal-specific therapeutic diet was implemented for 62 out of 100 dogs with AKI (62%), 110 out of 144 with ACKD (76%), and 260 out of 277 with CKD (94%). The remaining 38/100 (38%) AKI, 34/144 (24%) ACKD, and 17/277 (6%) CKD dogs received a low-fat gastrointestinal diet.

Comparative evaluation of median serum concentrations of creatinine, urea, bicarbonate, total calcium, ionized calcium, phosphate, and the calcium–phosphate product (CaxP) using the Kruskal–Wallis test is presented in **Table 1**.

Table 1. Median serum indices for creatinine (Cr), bicarbonate (Bicarb), total calcium (tCa), ionized calcium (iCa), phosphate (Phos), and CaxP in AKI, ACKD, and CKD groups were assessed through the Kruskal–Wallis test.

Parameter	Reference Range	AKI (n = 100)	ACKD (n = 144)	CKD (n = 262)	p-Value
Creatinine (Cr, mg/dL)	0.6–1.5	7.3[a] (2.0–22.9)	7.6[a] (2.0–23.8)	2.7[b] (1.2–10.2)	<0.0001
Urea (mg/dL)	15–55	263[a] (86–652)	337[b] (88–643)	136[c] (20–658)	<0.0001
Bicarbonate (Bicarb, mmol/L)	14–28	16.5[a] (3–39)	16[a] (3–44)	19[b] (4–38)	<0.0001
Total Calcium (tCa, mg/dL)	8.7–11.8	10.2[a] (6.9–16.6)	10.3[a] (4.2–15)	11[b] (4.5–17.3)	<0.0001
Ionized Calcium (iCa, mmol/L)	1.17–1.48	1.2[a] (0.64–1.91)	1.1[a] (0.21–1.53)	1.32[b] (0.62–2.43)	<0.0001
Phosphorus (Phos, mg/dL)	2.5–5.0	11.7[a] (2–22.7)	12.3[a] (3–29.3)	5.4[b] (2.6–23.3)	<0.0001
Calcium × Phosphorus Product (CaxP, mg²/dL²)	<70	117.5[a] (17.4–258.5)	126[a] (30.7–237)	59.8[b] (26.3–181.3)	<0.0001

Significance values from the Kruskal–Wallis and Dunn's post-hoc analyses are denoted by p-values and superscript annotations, respectively. Statistical relevance was determined at $p < 0.05$.

Bicarbonate concentrations were obtained for all 521 dogs enrolled. A total of 397/521 (76%) exhibited bicarbonate deficiency; 142/397 (36%) showed a moderate, and 255/397 (64%) a severe reduction. The deficiency occurred in 77/100 (77%) AKI, 123/144 (85%) ACKD, and 197/277 (71%) CKD subjects.

Within the AKI group, affected dogs were classified as grade 2 (n=4), grade 3 (n=14), grade 4 (n=34), and grade 5 (n=25). The ACKD group showed grade 2 (n=1), grade 3 (n=13), grade 4 (n=70), and grade 5 (n=39). CKD dogs were categorized into stage 1 (n=9), stage 2 (n=85), stage 3 (n=74), and stage 4 (n=26).

Regarding intensity, moderate deficiency appeared in 24/77 (31%) AKI, 35/123 (28%) ACKD, and 83/197 (42%) CKD cases; while severe forms affected 53/77 (69%), 88/123 (72%), and 114/197 (58%) dogs, respectively. A detailed comparison of frequency and severity among the disease categories is provided in **Table 2**.

Table 2. (A, B) Fisher's statistical comparison of bicarbonate deficiency prevalence and severity among AKI, ACKD, and CKD dogs (2A), and between dogs with CaxP values <70 and ≥70 mg²/dL² (2B).

(A) Serum Bicarbonate Levels among Study Groups				
Category	**AKI (n = 100)**	**ACKD (n = 144)**	**CKD (n = 277)**	**p-value**
Within normal range	23 (23%)	21 (15%)	80 (29%)	0.004
Below normal (deficit)	77 (77%)	123 (85%)	197 (71%)	
• Mild to moderate	24 (31%)	35 (28%)	83 (42%)	
• Marked (severe)	53 (69%)	88 (72%)	114 (58%)	0.02

Distribution by IRIS grade/stage

IRIS Grade / Stage	AKI (n = 100)				ACKD (n = 144)				CKD (n = 277)			
	2 (n = 10)	3 (n = 17)	4 (n = 45)	5 (n = 28)	2 (n = 4)	3 (n = 17)	4 (n = 83)	5 (n = 40)	1 (n = 21)	2 (n = 127)	3 (n = 95)	4 (n = 34)
Normal range	6 (60%)	3 (18%)	11 (24%)	3 (11%)	3 (75%)	5 (29%)	13 (16%)	1 (2%)	12 (57%)	42 (33%)	21 (22%)	8 (24%)

Deficient	4 (40%)	14 (82%)	34 (76%)	25 (89%)	1 (25%)	12 (71%)	70 (84%)	39 (98%)	9 (43%)	85 (67%)	74 (78%)	26 (76%)
p-value	**0.01**		**0.0003**	**0.009**								

(B) Serum Bicarbonate and Calcium–Phosphate Product (CaxP)

Parameter	CaxP < 70 mg²/dL² (n = 197)	CaxP ≥ 70 mg²/dL² (n = 319)
Normal (n = 129)	61 (47%)	68 (53%)
Deficient (n = 387)	136 (69%)	251 (79%)
p-value	**0.01**	
Severity of deficiency		
• Moderate (n = 132)	58 (44%)	74 (56%)
• Severe (n = 255)	78 (30%)	177 (70%)
p-value	**0.01**	

Deficiency was defined as serum bicarbonate <22 mmol/L, while a normal concentration was ≥22 mmol/L. Moderate deficiency was between 18–22 mmol/L, and severe deficiency was <18 mmol/L. Statistical cut-off was set at $p < 0.05$.

Serum CaxP values were measured in 516 dogs, revealing abnormalities in 319/516 (62%) and normal findings in 197/516 (38%). Among those with abnormal CaxP, 251/319 (79%) were bicarbonate-deficient, versus 136/197 (69%) in the normal CaxP group.

In the abnormal CaxP subset, 74/251 (29%) showed moderate and 177/251 (71%) severe deficiency, while in the normal CaxP subset, 58/136 (43%) had moderate and 78/136 (57%) severe deficiency.

Median bicarbonate values were markedly lower in dogs with abnormal CaxP (16 mmol/L; range 3–44) than in those with normal CaxP (19 mmol/L; range 3–24), reaching statistical significance ($p < 0.0001$).

Spearman's correlation outcomes between serum bicarbonate and biochemical parameters (creatinine, urea, phosphate, total calcium, ionized calcium, and CaxP) in the three renal disease groups are detailed in **Table 3**.

Table 3. Spearman correlation coefficients showing relationships between serum bicarbonate (mmol/L) and serum creatinine (mg/dL), urea (mg/dL), phosphate (mg/dL), total calcium (mg/dL), ionized calcium (mmol/L), and CaxP (mg²/dL²) among AKI, ACKD, and CKD dogs.

Bicarbonate vs.	AKI (n = 100)		ACKD (n = 144)		CKD (n = 277)	
	Spearman r	p Value	Spearman r	p Value	Spearman r	p Value
Creatinine	−0.31	0.001	−0.17	0.03	−0.10	0.07
Urea	−0.29	0.003	0.20	0.01	−0.08	0.14
Phosphate	−0.24	0.01	−0.20	0.01	−0.12	0.05
Total calcium	0.18	0.08	−0.04	0.58	0.01	0.83
Ionized calcium	−0.06	0.50	0.18	0.02	−0.05	0.39
CaxP	−0.16	0.11	−0.19	0.02	−0.10	0.09

Statistical significance is considered at $p < 0.05$.

A notable reduction in serum bicarbonate levels was highly prevalent across all three examined groups, with an overall rate of 76%. This outcome was expected since both bicarbonate depletion and metabolic acidosis are well-documented consequences of renal dysfunction in human and veterinary contexts [1]. Under normal physiological circumstances, the kidneys maintain acid–base equilibrium by generating and reabsorbing bicarbonate [1, 7]. Roughly 80% of urinary bicarbonate is reabsorbed at a consistent rate within the proximal tubule, meaning that increased tubular bicarbonate reuptake is not typically the major compensatory response during acidosis. Instead, the production of new bicarbonate mainly occurs in the proximal tubule through ammonia synthesis [2]. In this process, glutamine serves as the key precursor for ammoniagenesis, while bicarbonate reabsorption depends on a specific membrane transporter, the electrogenic sodium bicarbonate cotransporter isoform 1A (NBCe-1A) [8]. These transporters, located exclusively in the proximal tubule, are vital for bicarbonate recovery. Experiments involving NBCe-1A knockout mice demonstrated spontaneous metabolic acidosis, reduced ammonia excretion,

and a sharp decline in serum bicarbonate levels [9]. Consequently, injuries to the proximal tubule observed in AKI, ACKD, or CKD are likely to play a significant role in the onset of metabolic acidosis.

Within the CKD group, bicarbonate deficiency was detected in 71% of dogs, showing a progressive rise in frequency with advancing IRIS stage. This pattern mirrors findings in human medicine and likely reflects the gradual deterioration of residual kidney function [1, 10]. As renal function worsens, the capacity to preserve acid–base equilibrium declines. During CKD progression, total renal acid excretion drops, even though single nephron acid secretion may increase. Despite this compensatory effort, the number of remaining nephrons becomes inadequate to sustain acid–base homeostasis. The fall in ammoniagenesis has been associated with impaired glutamine uptake at the proximal tubule and is considered a main driver of metabolic acidosis in patients with moderate CKD [1]. In human cases where GFR falls below 15 mL/min/1.73 m², the ability to eliminate titratable acids declines markedly, intensifying metabolic acidosis [11]. Thus, the finding that most dogs with advanced CKD exhibited bicarbonate deficiency aligns with previous human studies, which also report rising prevalence at CKD stages 4 and 5 [2]. However, the absence of a clear correlation between serum bicarbonate and creatinine or urea among CKD dogs implies that factors beyond the loss of renal function—such as dietary acid intake and extra-renal compensatory mechanisms—may influence acid–base status [12].

Interestingly, even though most IRIS stage 1 dogs displayed normal bicarbonate levels, 43% still exhibited some degree of deficiency. This unexpected observation suggests that bicarbonate reduction might be more common than typically assumed. Earlier investigations in CKD-affected cats reported metabolic acidosis mainly in later disease stages, implying it was more a result of CKD progression than a contributing cause [10]. The appearance of bicarbonate deficiency in early CKD (stage 1) may therefore stem from increased non-renal acid production, rather than decreased renal function. Net acid load plays an essential role in acid–base regulation and is influenced by dietary composition. In humans with CKD, consumption of highly acidogenic diets has been implicated in metabolic acidosis development [13]. Since dogs at IRIS stage 1 had not yet been introduced to renal prescription diets, their greater acid load could explain this pattern. Furthermore, other defects in tubular acid excretion cannot be excluded, as seen in human CKD cases with obstructive nephropathy, sickle cell nephropathy, or diabetic nephropathy [13].

The incidence of bicarbonate deficiency was markedly greater among dogs with AKI and ACKD compared to those with CKD, likely reflecting a faster and more extensive decline in renal tissue in acute forms. This may arise from an abrupt drop in tubular ammoniagenesis and a simultaneous elevation in filtered acid load, ultimately reducing serum bicarbonate. Beyond renal loss, nonrenal mechanisms can also play a part in this imbalance. Hyperkalemia, which often develops in AKI and ACKD dogs because of poor urine output, is a known contributor. Elevated potassium can induce reversible metabolic acidosis through suppression of ammonia excretion. Animal experiments in mice have demonstrated that hyperkalemia alone can trigger metabolic acidosis even in the absence of other contributing conditions, including renal mass reduction, adrenal failure, or drug effects [14]. It is also noteworthy that a larger proportion of CKD dogs were maintained on renal prescription diets than those with AKI or ACKD. In AKI, bicarbonate deficiency commonly results from multiple concurrent factors [15]. Among extra-renal influences, gastrointestinal bicarbonate loss can be important, as digestive disturbances are frequently observed in AKI cases. Diarrhea, for instance, has been reported in 41% of dogs with AKI, being notably more frequent in those that did not survive. In AKI, diarrhea may stem directly from uremic toxin irritation or indirectly from secondary conditions such as overhydration or pancreatitis [16]. Accelerated intestinal transit during diarrhea also diminishes bicarbonate absorption.

A further reason for bicarbonate depletion in AKI and ACKD dogs could be marked hyperphosphatemia. In this study, serum phosphate concentrations were significantly higher in AKI and ACKD dogs than in those with CKD, and a negative linear correlation existed between bicarbonate and phosphate levels in both groups, indicating worsening acidosis with phosphate elevation. This aligns with findings in humans where pronounced hyperphosphatemia fosters acid generation and bicarbonate neutralization [17]. Additionally, the severity of bicarbonate loss appeared to parallel the rise in serum creatinine and urea, suggesting that acidosis intensifies as functional renal capacity decreases.

Dogs with AKI and ACKD also demonstrated a higher incidence of severe bicarbonate deficiency compared to CKD cases. This disparity may reflect disease dynamics: while CKD generally progresses slowly and variably depending on multiple factors [18], many dogs in this cohort with CKD were in early IRIS stages 1 or 2, when renal function remains relatively stable and uremic crises are less common [19]. Consequently, severe acidosis was less likely in these individuals. Conversely, AKI and ACKD groups primarily included animals at advanced

disease stages, where the rapid fall in GFR and renal mass, coupled with conditions such as hyperphosphatemia or gastrointestinal losses, intensify bicarbonate depletion. Acute pancreatitis may further contribute, given its established association with AKI [20] and poorer outcomes in dogs undergoing hemodialysis [21]. Normally, pancreatic bicarbonate secretion prevents premature enzyme activation, but acidosis may lower pH and activate proteases. In turn, pancreatitis exacerbates bicarbonate loss both directly—through pancreatic fluid depletion—and indirectly—via lactic acidosis resulting from shock, sepsis, or GI bleeding [22].

The occurrence of metabolic acidosis is linked to a variety of clinical consequences and unfavorable prognoses. When acidosis develops, adaptive mechanisms such as single-nephron ammonium production can become harmful, encouraging fibrotic processes in renal tissue [2]. It has also been recognized that metabolic acidosis independently elevates the risk of death among individuals with CKD. Patients who exhibit low serum bicarbonate levels tend to lose kidney function more rapidly and are more prone to progress to terminal renal failure [2]. Among the possible complications associated with bicarbonate depletion, disruptions in calcium–phosphate balance appear particularly significant, since bone acts as a key buffer in maintaining acid–base homeostasis [2]. In the present analysis, dogs exhibiting abnormal calcium–phosphate (CaxP) product values had lower bicarbonate concentrations than those with normal CaxP. Furthermore, both the occurrence and severity of bicarbonate deficiency were increased in animals showing mineral metabolism abnormalities, implying a connection between disturbed calcium–phosphate regulation and acidosis.

Bicarbonate depletion can accelerate bone mineral loss through several biological routes. In human CKD cases, low bicarbonate has been directly associated with decreased bone density, supporting the idea that acidosis perpetuates mineral imbalances. In dogs with AKI and ACKD, serum bicarbonate was negatively correlated with phosphate levels, and in ACKD dogs, an additional inverse relationship was observed with CaxP. These results suggest that bones contribute to buffering during acidosis by releasing calcium carbonate and phosphate to neutralize excess hydrogen ions [2]. Beyond this buffering response, metabolic acidosis stimulates bone resorption by activating osteoclasts and reducing osteoblast function, leading to progressive bone weakening [2]. Such findings align with observations in human medicine, where AKI frequently results in mineral dysregulation, including low calcium, high phosphate, and secondary hyperparathyroidism [23].

Conversely, in the CKD dog group, no significant link emerged between bicarbonate concentration and calcium–phosphate indicators. This contrasts with human CKD, where chronic acidosis often coexists with overactive parathyroid function and both processes contribute to bone loss. In people with CKD, acidosis heightens the sensitivity of osteoblast-like cells to parathyroid hormone (PTH), thereby intensifying bone breakdown [3]. The difference seen between AKI/ACKD and CKD dogs could be attributed to milder mineral disturbances in the CKD population. Dogs with CKD had notably lower phosphate and CaxP values but higher bicarbonate and ionized calcium compared with AKI and ACKD animals. Such trends may reflect that many CKD dogs were in early disease stages, where metabolic and mineral imbalances are less pronounced. Additionally, those at more advanced stages were often fed prescription renal diets, which are known to help stabilize calcium–phosphate homeostasis.

This study has a few constraints. Detecting initial stages of AKI or ACKD in clinical conditions remains challenging; hence, the actual prevalence of bicarbonate deficiency might be underestimated. Because venous blood gas data were unavailable for most animals, parameters such as pH, lactate, and anion gap could not be evaluated—factors that would have clarified the specific form of metabolic acidosis. Moreover, the analysis focused solely on serum bicarbonate at the first hospital visit, and follow-up data were incomplete since most dogs continued care with their referring veterinarians. Another possible technical limitation involves the handling of blood samples. Underfilled vacutainer tubes can yield falsely low bicarbonate measurements, regardless of whether they are tested immediately or after several minutes [24]. Although proper filling procedures were followed for all samples in this study, a minor influence on the final readings cannot be completely dismissed.

Conclusion

Bicarbonate deficiency was a widespread finding in both acute and chronic kidney diseases, with frequency rising alongside IRIS stage or grade. Detection of deficiency even in stage 1 CKD implies that metabolic acidosis may begin early in disease progression. Its severity was greatest in AKI and ACKD, likely reflecting abrupt renal decline and extrarenal influences. Moreover, abnormalities in calcium–phosphate metabolism were associated with higher occurrence and intensity of bicarbonate depletion, reinforcing a possible link between acid–base and

bone mineral disorders. Considering its prevalence and clinical significance, serum bicarbonate assessment should become a standard diagnostic parameter in all dogs affected by acute or chronic kidney disease, regardless of disease stage.

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