STUDY OF RENAL PATHOLOGIES AND THE IMPLICATIONS OF RENAL FUNCTION IN THE HOMEOSTASIS OF BIRDS

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Abstract

Renal pathologies in birds are an area of major interest in avian medicine, with direct implications for health and productive performance. The anatomical and physiological characteristics of the excretory system make it difficult to assess renal function, as urine sampling is not feasible in practice. Thus, blood biochemical analyses are the main method of ante-mortem diagnosis, being used to identify hyperuricemia, gout, nitrogen metabolism disorders, and other renal pathologies, but also to monitor physiological status on poultry farms. However, most kidney diseases are detected late, most often post-mortem, through morphohistological examination. Undetected kidney diseases cause significant disturbances in nitrogen metabolism, with a direct impact on homeostasis, and on morbidity and mortality at the herd level. In this context, phytotherapy emerges as a valuable adjuvant strategy, helping to support the growing organism and optimize hydration. This paper provides a summary of the latest data on renal physiology in birds and associated pathologies, with a focus on species of economic interest, such as the ROSS 308 broiler. The role of serum biomarkers in assessing renal function, current prevention strategies, and the research directions needed to implement solutions adapted to intensive farming systems are highlighted.

Key words: *broilers, renal pathologies, nitrogen metabolism, phytotherapy, serum biomarkers*

INTRODUCTION

Kidney diseases in birds can be classified as prerenal (decreased blood supply through the renal artery), renal (damage to the renal parenchyma), postrenal (obstruction of the excretory tract), and mixed, with acute or chronic progression. The presence of kidney disease is characterized by structural and functional changes in the kidneys and a decrease in glomerular filtration rate (or the body's physiological response to dehydration) or the onset of glomerulopathies. Renal function contributes to the optimal growth of broilers by maintaining water homeostasis, as the kidneys play a role in fluid reabsorption and nitrogenous waste excretion, thus dictating the necessary regulation of electrolyte balance [1].

In birds, the main product of nitrogen excretion is uric acid, and its concentration in the blood is influenced by the state of hydration. Under conditions of dehydration, uric acid excretion decreases, thus increasing the concentration of uric acid in the blood. Yet, birds have a unique ability to maintain renal blood flow under conditions of hemodynamic changes (e.g., hemorrhage) due to the particularities of port-renal circulation [2]. According to Yang and Nishimura (2021), birds are the only vertebrates capable of producing concentrated urine.

An increase in uric acid concentration (prerenal disease) is caused by dehydration, decreased circulating blood volume, and congestive heart failure, which lead to reduced renal perfusion without structural

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damage to the kidney. Urea plays a minor role in nitrogen metabolism, and its serum concentration is not a reliable indicator of renal function. The main parameter for assessing water status and renal function is plasma uric acid [4].

The accumulation of uric acid in the blood (hyperuricemia) occurs when the integrity of the urinary tract and the functional capacity of the proximal tubules are severely impaired, indicating an actual renal (intrarenal) cause. The process of water and electrolyte osmoregulation in birds involves postrenal processing of urine in the terminal segments of the cloaca, particularly in the coprodeum and colon, where water and ion reabsorption occurs [5]. Urine in birds is like a suspension of urate crystals and mucopolysaccharide microsphere complexes, in which uric acid is present in concentric layers and its consistency varies depending on the species [6].

MATERIAL AND METHOD

The current study is a narrative update analysis of kidney disease in birds, based on a critical review of recent scientific literature published between 2018 and 2025. Both review studies, experimental and case studies addressing physiological, biochemical, pathological, and therapeutic aspects related to avian kidney function were selected and analyzed.

The bibliographic research was conducted in the main international scientific databases, using keywords such as avian kidney disease, hyperuricemia, uric acid metabolism, avian gout, renal function in birds, and avian nephropathy. Sources were identified and selected from established platforms such as Web of Science, Elsevier (ScienceDirect), Frontiers, SpringerLink, Wiley Online Library, Taylor & Francis, MDPI, and PubMed, as well as from specialized veterinary journals (e.g., Indian Journal of Animal Sciences, Avian Pathology, Poultry Science, Journal of Veterinary Science).

The data extracted from these sources were comparatively analyzed and synthesized in order to identify current trends, proposed biomarkers, and emerging therapeutic strategies. The results were organized thematically according to the type of disease (hyperuricemia, acute tubular necrosis, toxic, infectious, metabolic, or deficiency nephropathies) and the pathophysiological mechanisms involved.

RESULTS AND DISCUSSIONS

Hyperuricemia is an increase in the level of uric acid in the blood above the normal physiological limit for the species. The reference value for uric acid in chickens is 4.5 mg/dL. Hyperuricemia is a metabolic disease characterized by excessive accumulation of uric acid in serum, and excess serum uric acid causes oxidative stress and inflammatory response in the kidneys, therefore contributing to the deterioration of renal function. Increased reabsorption and decreased excretion of uric acid cause hyperuricemia [7,8]. According to the literature, hyperuricemia accelerates glomerular hypertension, causes proteinuria, and leads to renal failure [9].

In one of the most recent experimental studies conducted on 80 broiler chickens, the authors concluded that uric acid can be used as a useful biomarker in predicting all degrees of acute tubular necrosis lesions, while creatinine and blood urea nitrogen (BUN) are only useful in predicting severe forms of acute tubular necrosis. The authors also emphasize that biochemical parameters can be influenced by nutritional and metabolic factors, which must be taken into account when interpreting the results [10]. In renal failure, intestinal elimination of uric acid is accentuated, concomitant with reduced renal excretion. Therefore, there are complementary mechanisms of cooperation between the intestine and the

kidneys to maintain balance. Significant amounts of uric acid in saliva and gastrointestinal fluids support the physiological role of the intestine in uric acid elimination [11].

Gout is a chronic metabolic disease characterized by localized inflammation and tissue damage due to the deposition of monosodium urate crystals. Clinically, it manifests as hyperuricemia, acute arthritis, uric acid kidney stones, and in severe cases, kidney dysfunction, joint deformities, and death. Gout occurs through inflammation caused by the formation and accumulation of urate crystals, with hyperuricemia being the early stage of gout [9, 12].

Acute kidney injury occurs following unexpected disruption of kidney function, accompanied by intoxication (symptoms of toxicity) or ischemia.

Table 1. Major kidney diseases in birds and the recent findings.

Kidney diseases	*Discovered mechanisms*	*References*
Hyperuricemia and visceral gout	The most frequent renal metabolic disorder in birds. Urate crystal deposition causes inflammation, oxidative stress, and tubular necrosis. Experimental models show that a high-protein diet and water restrictions induce stable hyperuricemia.	[13, 14, 15]
Acute tubular necrosis (ATN)	Acute kidney injury associated with urate accumulation; uric acid is a sensitive biomarker of severity of injury. In broiler chicken studies, levels > 5 mg/dl correlate with moderate-to-severe injury.	[10, 16]
Acute uric acid nephropathy	Obstruction of the renal tubules by urate crystals causes inflammation and cell death. Activation of the NLRP3 inflammatory pathway and increased expression of IL-1β and TNF-α in the kidneys.	[16, 17]
Articular gout	Deposition of urate in joints and periarticular tissues. Associated with chronic hyperuricemia and metabolic unbalance. From a pathophysiological point of view, it is similar to human gout.	[13, 14, 18]
Chronic kidney disease (CKD)	It occurs following repeated or persistent damage (toxicity, infections, unbalanced nutrition). Renal fibrosis, tubular degeneration, and urate retention are observed.	[19]

Acute kidney injury in birds is often induced by elevated blood uric acid concentrations, which cause urate deposits in the renal tubules and trigger oxidative stress, inflammation, and cell apoptosis [16]. Therapy aims to reduce uric acid by increasing renal excretion of urates. Uric acid concentration is influenced by purine metabolism disorders and renal dysfunction [7]. Mitochondria are involved in the production of reactive oxygen species, and excess production can damage DNA, RNA, carbohydrates, proteins, and other molecules. Podocytes are cells arranged around the capillaries in the glomerulus and line the Bowman's capsule, forming the third (epithelial) layer through which blood is filtered [20]. Mitochondrial dysfunction contributes to the development of kidney disease through an imbalance between the production and elimination of reactive oxygen species, and increased oxidative activity in the renal mitochondria makes the organ vulnerable to damage [21].

Recent review studies on hyperuricemia and avian gout have

highlighted major advances and challenges in the field.

According to Wang (2024), the most stable method of inducing hyperuricemia in birds combines a purine-rich diet with water restriction, which ensures a higher success rate and greater stability of the experimental model. Avian models reproduce the pathophysiological mechanisms found in mammals but produce less renal damage, yet are useful due to their durability and reproducibility. The authors pointed out that the evaluation of these models is based on a wide range of indices-biochemical (uric acid, enzymes, electrolytes), histopathological, and anatomical-but there is still no standardized protocol among published studies. The deposition of urate crystals in the renal tubules produces not only mechanical effects but also activates inflammatory and oxidative stress pathways, contributing to the progression of tubular necrosis and renal interstitial inflammation [14].

Regarding therapeutic interventions, Lee (2023) highlighted the testing of hypouricemic agents (allopurinol, benzbromarone) in avian models, with favorable effects on renal function and histology. Chen (2024) also reported promising results in the use of plant extracts, such as *Smilax china*, which reduced serum uric acid levels and attenuated renal damage.

An emerging line of research, mentioned by Wang (2024), concerns the role of gut microbiota in purine metabolism. Some bacterial species isolated from the gut of birds can degrade urate, which could offer a probiotic approach to controlling hyperuricemia. Avian hyperuricemia is favored by diets with excess protein and calcium, nutritional imbalances, and genetic differences between bird breeds and lines (meat hybrids), which explains the variability in physiological response between studies [15].

Renal function regulates serum uric acid concentration in order to maintain blood uric acid homeostasis, which depends on the interaction between uric acid reabsorption and excretion in the renal tubules [24]. Uric acid affects renal function by depositing monosodium urate crystals in the joints, kidneys, and other tissues. It has the ability to stimulate intracellular oxidation, induce endothelial dysfunction, and induce renal fibrosis and glomerulosclerosis, accompanied by tubulointerstitial damage [21]. Renal function can be impaired by blocking angiotensin receptors and inhibiting angiotensin-converting enzymes under the action of nutritional factors, such as a reduced protein concentration in food and the absence of nitrogen in the diet causes protein malnutrition in chickens [21, 25]. A balanced diet results in low levels of uric nitrogen in the blood, implicitly reducing protein catabolism. Total and efficient use of nitrogen results in a decrease in urea synthesis. An animal's amino acid requirements are dynamic and are influenced by genetic profile, age, and physiological status, and uric acid circulating in poultry blood is a metabolic indicator of amino acid imbalances and deficiencies [26].

Acute tubular necrosis is one of the main causes of acute renal failure of renal origin [27]. Uric acid crystalopathies: acute urate nephropathy and various forms of urate-associated urolithiasis. Urate crystallization causes necrosis of renal tubular epithelial cells and inflammation. Mitochondrial dysfunction contributes to the development of kidney disease through an imbalance between the production and elimination of reactive oxygen species, and increased oxidative activity in the renal mitochondria causes the organ to be vulnerable to damage [22, 28].

In the liver, the enzyme adenosine deaminase catalyzes the deamination of adenosine to produce inosine. The enzyme xanthine oxidase is involved in the conversion of hypoxanthine and xanthine to uric acid. Xanthine dehydrogenase is the

final enzyme in purine metabolism that contributes to the production of uric acid. Hepatic xanthine oxidase and adenosine deaminase catalyze hypoxanthine and adenine into uric acid and oxygen [29]. Deficiencies or enzymatic changes in hepatic xanthine oxidase (XOD) cause an increase in uric acid concentration. XOD regulates uric acid generation through the continuous oxidation of hypoxanthine and xanthine, and increased XOD activity is the main factor affecting purine metabolism [9]. For the most part, uric acid is actively secreted by the proximal renal tubules, and the rest of the uric acid (approximately 10%) is filtered by the glomerulus [30]. In kidney disease, when there is a disruption of the integrity of the urinary tract or an obstruction of urine flow, there is an increased concentration of uric acid in the blood. Uric acid is the main nitrogenous end product of protein catabolism, and the plasma uric acid value varies depending on a metabolic imbalance caused by a deficiency or excess of amino acids (failure to meet the bioequivalence relationship of AA) [31]. Uric acid is filtered and reabsorbed freely through active transport, and the mechanism of renal urate transport involves the participation of integral transport proteins of the plasma membrane. The mechanism of uric nitrogen recycling in chickens consists of urine reflux from the cloaca into the cecum [32]. Nitrogen digestion in birds and fluid transport is facilitated by reverse intestinal peristalsis, and cloacal reflux through the colon selectively delivers fluids, including urine, to the cecum [32]. In birds (*Gallus gallus domesticus*), uric acid and urea are also broken down by the cecal microbiota, producing ammonia. Ammonia will either pass rapidly into the cecal mesenteric vein or be converted into amino acids and proteins by the microbial flora. Thus, nitrogen can become available to the host following degradation, contributing to the formation of amino acids synthesized by the microbiota [33].

Urine, which contains uric acid, is excreted into the colon and, through antiperistalsis movements, enters the cecum, where it is used as a source of nitrogen for the growth of microbial flora. In chickens, it has been shown that microbial protein and amino acids can be absorbed directly from the cecum, thus providing a new source of protein for the host [34].

Birds use approximately 30-50% of the nitrogen ingested during their metabolism, and the excreted portion is the source of ammonia emissions [35,36]. Nitrogen catabolism in birds results in uric acid, 55-80% of excreted nitrogen, present in urine as a colloidal precipitate, which is then converted to ammonia (5-25% of excreted nitrogen) and eliminated along with other compounds, urea, and creatinine. Free amino acids contribute very little to total nitrogen excretion [37]. The nitrogen balance represents the equilibrium between nitrogen intake (represented by proteins in food) and nitrogen excretion through urine and feces. The total nitrogen content in excrement increases with age and thus reflects nitrogen intake [36]. Nitrogen excretion from the avian body involves mechanisms of increased water intake, and polyuria, often having a nutritional cause, leads to increased moisture in the birds' litter.

The significance of indicators for renal function and the interpretation of results obtained through biochemical analyses are important for identifying renal diseases, which influence increased mortality and morbidity in a flock of birds.

CONCLUSIONS

Hyperuricemia and uric acid nephropathy are the most common kidney disorders in birds, influenced by nutritional (high-protein and/or calcium-rich diet), toxic, and physiological factors. Recent studies have shown that uric acid accumulation causes oxidative stress,

inflammation, and acute tubular necrosis, and monitoring serum uric acid can serve as an early biomarker of kidney damage.

We recommend the implementation of integrated strategies for the prevention and monitoring of renal function in birds, based on balancing protein and mineral rations, ensuring adequate hydration, controlling toxic factors, and using serum biomarkers such as uric acid, followed by inorganic phosphorus and, in some cases, the uric acid/creatinine ratio for early diagnosis of disease.

It is also necessary to standardize experimental models and methods for evaluating kidney disease to facilitate comparison between studies and the application of results in veterinary practice and the poultry industry.

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