

Comprehensive Review on Visceral Gout in Commercial Broilers

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ABSTRACT

Gout in chickens arises due to their uricotelic mechanism for nitrogenous waste excretion and the absence of the enzyme uricase, which converts uric acid into less harmful compounds. Although uric acid is non-toxic, its precipitated crystals cause mechanical damage to tissues, including the kidneys, heart, lungs, and intestines (visceral gout), as well as joints (articular gout). This multifactorial metabolic disorder is influenced by infectious agents, nutritional imbalances, and management practices. Key infectious contributors include Nephropathic Infectious Bronchitis Virus (IBV), Avian Nephritis Virus (ANV), and Chicken Astrovirus (CAstV). Nutritional factors such as high dietary calcium (>2%), excessive crude protein (>30%), hypovitaminosis A, hypervitaminosis D3, dehydration, and mycotoxin-contaminated feed can precipitate renal failure and gout. Poor management practices, including high brooding temperatures reducing water intake, alongside toxicity from antibiotics, anticoccidials, and pesticides, further exacerbate the condition. Clinical signs comprise reduced feed and water intake, lethargy, ruffled feathers, weight loss, and abnormal droppings. Hematological findings include elevated TEC, TLC, Hb, and PCV, alongside decreased ALT, AST, ALP, serum proteins, albumin, and GSH. Gross lesions reveal chalky white urate deposits on serosal surfaces of organs, while histopathology demonstrates urate crystal deposition with inflammatory cell infiltration. Preventive measures include low-protein diets, supplemented water, and B-complex and electrolyte supplementation to mitigate gout incidence.

Keywords: Gout; Poultry; Pathological lesions; Treatment

Introduction

Gout is a prevalent metabolic disorder in domestic birds, characterized by the excessive buildup of urates in tissues (Ali [1]). In affected birds, uric acid levels can rise dramatically, reaching up to 44 mg/100 ml, compared to the normal range of 5–7 mg/100 ml. The kidneys play a crucial role in maintaining fluid and electrolyte balance, eliminating metabolic waste, and producing essential hormones. When kidney function is impaired, uric acid accumulates in the bloodstream and deposits in various tissues, including the kidneys, liver, heart, and joints [2]. Hyperuricemia, a key factor in gout development, is linked to purine metabolism dysfunction and environmental influences [3]. Elevated uric acid forms sharp crystals that damage tissues, triggering

inflammation. Unlike mammals such as rats and rabbits, which produce uricase to convert urate into soluble allantoin, birds lack this enzyme, making them more susceptible to gout [4]. Renal damage in poultry often goes unnoticed until significant kidney function is lost, as birds may remain productive until two-thirds of kidney tissue is compromised. Gout arises from multiple causes, including infections (e.g., IBV, CAstV, ANV), nutritional imbalances (such as excessive dietary calcium), toxins, and poor management practices. Mineral imbalances, particularly between calcium and phosphorus, contribute to renal failure and gout, leading to economic losses in poultry farming. Despite being a known issue, gout remains a diagnostic challenge, as affected birds often show minimal symptoms until shortly before death [2].

Incidence of Gout

Seasonal Variation

The prevalence of gout in poultry is highest during colder months, followed by summer, with the lowest incidence occurring in the rainy season [5-9]. Cold temperatures contribute to increased uric acid formation and reduced dissolution, leading to cold stress and elevated mortality. The higher occurrence of gout during the rainy season compared to summer may be attributed to poor hygiene and management conditions [10].

Age-Related Susceptibility

Young chicks exhibit higher susceptibility and mortality due to visceral gout compared to adult birds [6-13]. As pullets reach sexual maturity, they are transitioned to high-calcium diets to support egg production. If pre-existing kidney damage is present, the increased calcium excretion can overwhelm renal function, leading to gout [2].

Causes of Gout

Gout was initially considered a nutritional disorder, but advancements in diagnostics have identified viral associations [9]. It is a multifactorial metabolic disease resulting from kidney damage due to various causes [2]. Hyperuricemia elevated blood uric acid levels can arise from overproduction, impaired enzymatic degradation, or reduced renal excretion due to factors such as drug toxicity (e.g., aspirin, mercuric salts) [1].

Nutritional Causes

Dietary Calcium: Excessive calcium (>2%) in broiler diets disrupts kidney function, leading to urate deposition on visceral surfaces and high flock mortality [14,15]. The Bureau of Indian Standards (BIS, 1992) recommends a maximum of 1.2% calcium, while [16] suggest 1% as optimal.

Protein Levels: High dietary protein increases glomerular filtration rate (GFR), causing glomerular injury and eventual glomerulosclerosis [7,14,17-19]. recommends 22% and 20% crude protein for broiler starter and finisher diets, respectively. While healthy kidneys tolerate high protein, pre-existing renal damage exacerbates uric acid accumulation, especially with protein adulterants like urea [2].

Mineral Imbalances

Calcium-Phosphorus Ratio: A disrupted Ca: P ratio (ideally 2:1) leads to calcium-sodium-urate crystal formation [1,2]. Excess calcium in immature pullets causes kidney damage, while low phosphorus reduces urinary acidification, increasing gout risk [20,21].

Sodium: High sodium intake strains kidney function. Sodium bicarbonate, used for heat stress management or eggshell quality, alkalinizes urine, promoting kidney stone formation [2,18,22].

Sulphates: Excessive calcium excretion due to reduced resorption increases gout risk.

Dehydration

Water deprivation leads to urate deposition [23]. Poor brooding temperatures, insufficient water access, prolonged hatchery holding, and transportation without water exacerbate renal failure [2].

Vitamin Imbalances

Vitamin D₃: Excess increases calcium absorption, promoting urate crystal formation.

Vitamin A Deficiency: Prolonged deficiency causes tubular epithelial sloughing, obstructing urate excretion [2].

Infectious Causes

Several viral pathogens contribute to gout in poultry, including Avian Nephritis Virus (ANV), Infectious Bronchitis Virus (IBV), and Chicken Astrovirus (CAstV) [24]. Among these, IBV—particularly the Massachusetts strain—induces severe nephropathy, leading to visceral gout and high mortality in young broilers [1,9,25]. Vertical transmission of these viruses can impair kidney function in progeny, predisposing chicks to gout [2].

Miscellaneous Causes

Mycotoxins: Nephrotoxic mycotoxins such as ochratoxin A, citrinin, oosporein, and deoxynivalenol (DON) damage renal and hepatic tissues, reducing uric acid excretion [2,24-26].

Metabolic Disorders: Hypoxia-induced ascites elevates uric acid production, mimicking gout symptoms in early stages [2].

Nephrotoxic Agents: Antibiotics (e.g., gentamicin, sulfonamides, nitrofurans) impair renal function, particularly in young chicks. Disinfectants (phenol, cresol) and chemicals (copper sulfate) induce dehydration and water refusal. Pesticides, anticommodials, and industrial chemicals may also precipitate gout [7].

Types of Gout

Visceral Gout

Acute condition characterized by urate deposition on serosal surfaces (liver, kidneys, pericardium, air sacs). Predominantly affects young broilers (2–3 days old) and layers (>14 weeks). Kidney dysfunction leads to hyperuricemia, causing urate accumulation in tissues [27].

Articular Gout

Chronic form with urate deposits in joints (legs, wings, synovial membranes), inciting granulomatous inflammation. Periarticular gout involves monosodium urate (MSU) crystal deposition around joints. In which Swollen, painful joints, reluctance to perch, greenish

diarrhea, and dehydration. Rarely systemic; kidneys may appear normal unless dehydration occurs [1].

Pathophysiology of Gout

Hyperuricemia results from impaired renal excretion, leading to urate crystal deposition in tissues [28-30]. Kidney damage manifests as atrophy, gritty urate deposits, and dilated ureters, with compensatory hypertrophy in functional nephrons [31-33]. Birds lack uricase, preventing conversion of uric acid to soluble allantoin, unlike ureotelic mammals [1].

Inflammatory Mechanism in Articular Gout

Crystal-Induced Inflammation: Monosodium urate (MSU) crystals trigger complement activation (C3a, C5a), recruiting neutrophils and macrophages. Phagocytosis releases free radicals, leukotrienes (e.g., LTB₄), and lysosomal enzymes, damaging joint tissues.

Cytokine Release: Macrophages secrete pro-inflammatory cytokines (IL-1, IL-6, IL-8, TNF- α), exacerbating synovitis and cartilage degradation.

Chronic Joint Damage: Persistent hyperuricemia sustains crystal deposition, leading to progressive arthritis (Vegad, cited in [1]).

Therapeutic Implications

Lowering serum urate levels is critical to dissolve crystals and prevent acute flares or chronic joint damage [1].

Clinical Symptoms

Affected birds exhibit dullness, dehydration, ruffled feathers, and segregatory behavior. Additional signs include restlessness, moist vent with whitish pasty droppings, difficulty in movement, and painful joints, often causing a shuffling gait or inability to perch. Wing involvement may impair flight, while swollen, reddened feet may progress to blisters and sores due to urate deposition. Postmortem findings include emaciation, dehydration, and enlarged hock/phalangeal joints. Other manifestations are reduced appetite, lethargy, weight loss, feather plucking, abnormal droppings (chalky urates), and behavioral changes ([1,34,35].

Clinical Pathology

Serum Markers

Hyperuricemia (>5–7 mg/dL) and elevated creatinine (>0.7–22 mg/dL) indicate renal dysfunction, likely due to impaired uric acid excretion or increased nucleic acid degradation (Chandra et al., 1985). NSAID overuse exacerbates renal damage, worsening hyperuricemia [36]. High-calcium/protein diets elevate uric acid [21,37]. Creatinine, a muscle-derived metabolite, reflects glomerular filtration rate (GFR) decline [38].

Hematological Changes

Increased PCV, Hb, and TEC suggest dehydration [6-8]. Leukocytosis (lymphocytosis) indicates immune activation against infection or bone marrow stimulation [39,40]. Elevated AST/ALT signals hepatic injury, particularly in high-protein diets or diclofenac toxicity [18,21]. Reduced glutathione (GSH) and increased TBARS reflect oxidative stress in renal/hepatic tissues [35,36]. GSH depletion impairs detoxification, exacerbating free radical damage [41,42]. TBARS/MDA levels indicate lipid peroxidation and cell membrane damage [43,44].

Protein and Lipid Metabolism

Hyperproteinemia may arise from ammonia/CO₂ accumulation, reducing water intake and renal flushing (Phatak [45]). Hypcholesterolemia and reduced bilirubin correlate with MSU crystal-induced renal injury [18].

Postmortem Lesions

Urate Deposits

White chalky urate deposits were observed on serosal surfaces of the pericardium, air sacs, peritoneum, liver, kidneys, and ureters. Visceral congestion was attributed to emaciation and dehydration [46].

Kidneys

Enlargement and congestion, often bulging from the renal fossa. Frosted appearance due to urate crystal accumulation, with pinpoint hemorrhages. Ureters: Distended with semi-solid urates, appearing cord-like [8].

Liver

Enlarged and friable, with urate deposits on the capsular surface. Sinusoidal congestion, hemorrhages, fatty changes, and necrotic foci [1,15].

Heart

Pericardial urate deposition (uric acid pericarditis) with myocardial congestion and hemorrhages [47].

Lungs

Urate deposition in parenchyma and air capillaries, leading to atelectasis and emphysema [19].

Spleen

Splenitis and subcapsular hemorrhages with urate deposits [19].

Musculoskeletal System

Articular Gout: White tophi in joints (shoulder, elbow, carpus, hip, knee, toe) with periarticular necrosis and edema [19].

Microscopic Lesions

Kidneys

Glomerular atrophy, tubular degeneration (vacuolar, necrotic), and urate crystals (needle-shaped, pink amorphous deposits) surrounded by inflammatory cells [24,48].

Liver

Fibrous proliferation in Glisson's capsule, necrotic hepatocytes, and urate crystals [49].

Proventriculus & Bursa

Edema, hemorrhage, and lymphocytic depletion [19].

Histochemical Staining

Urate crystals stained black with De Galantha's stain [34,50].

Prevention and Control

Nutritional Management

Balanced Ca: P ratio, adequate vitamins (A, D₃), and optimal sodium levels. Avoid excessive sodium bicarbonate (>2 kg/ton). Use conventional protein sources; test feed for mycotoxins and apply binders if contaminated.

Medication & Water Quality

Limit nephrotoxic drugs (antibiotics, sulfonamides, anticoccidials). Ensure fresh, potable water; avoid copper sulfate unless prescribed.

Husbandry Practices

Monitor ventilation to prevent ammonia/CO₂ buildup. Prevent dehydration through proper brooding and water access.

Treatment of Gout in Poultry

Hydration and Nutritional Management

Ensure unrestricted access to clean water with sufficient drinker space. Implement a low-protein diet (3-5 days) adjusted for age/breed requirements to reduce uric acid production. Broken maize and jaggery (5g/L water for 3-5 days) may help mitigate mortality [50-67].

Vaccination and Biosecurity

Infectious Bronchitis (IB) Control: In endemic areas, administer nephrotropic IB vaccines (e.g., day 4 or beak dip at day 1 for broilers).

Urinary Acidification

Vinegar: 1-2 mL/L water for ≤24 hrs. Potassium chloride: 1 g/L water for ≤24 hrs. Ammonium chloride/sulfate: 2.5 kg/ton feed for 7 days. Allopurinol (10-40 mg/L water, BID) until mortality ceases. Me-

thionine hydroxyl analogue (0.6%) + 3% dietary calcium to support renal function [26].

Supportive Care

Electrolytes to combat dehydration. Vitamin supplementation (A, D₃, K, B-complex) to address deficiencies [68-72].

Conclusion

Gout remains a significant economic burden in poultry due to high mortality rates. Key etiological factors include: Infectious triggers (e.g., nephropathic IBV). Nutritional imbalances (Ca: P ratio, hyperproteinemia). Mycotoxins, dehydration, and electrolyte disturbances.

Preventive Measures

Feed analysis to ensure optimal mineral/vitamin levels. Urine acidification to dissolve/prevent uroliths. Integrated management (breeder, hatchery, farm practices).

Conflict of Interest

The authors declare no competing interests.

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