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Critical evaluation of pathogenesis and treatment of gout including urolithiasis in poultry

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Abstract

Gout and urolithiasis are important metabolic and renal disorders in poultry that significantly impact productivity, health, and economic returns in the poultry industry. These conditions are primarily associated with impaired kidney function, leading to accumulation of uric acid in the bloodstream (hyperuricemia) and subsequent deposition of urate crystals in visceral organs and joints. Urolithiasis, characterized by the formation of kidney stones and ureteral obstruction, often coexists with gout and further exacerbates renal damage. The etiology is multifactorial, involving nutritional, infectious, toxic, genetic, and environmental factors. High dietary protein, excess calcium, imbalanced calcium-phosphorus ratio, vitamin A deficiency, and high sodium intake contribute significantly to disease development. Additionally, nephrotoxic agents such as mycotoxins and certain antibiotics, along with viral infections like infectious bronchitis virus and avian nephritis virus, play a critical role in kidney dysfunction.

Pathogenesis involves disruption of purine metabolism, increased uric acid production, and decreased excretion due to renal impairment, resulting in urate crystal precipitation in tissues. Environmental stressors, particularly dehydration caused by inadequate water intake or extreme temperatures, further intensify disease occurrence. Clinically, affected birds exhibit poor growth, reduced egg production, lameness, and high mortality rates.

Currently, there is no specific treatment for gout in poultry; therefore, management primarily focuses on preventive strategies. These include providing balanced nutrition, maintaining optimal calcium-phosphorus ratios, ensuring adequate hydration, controlling mycotoxin contamination, and minimizing exposure to nephrotoxic substances. Early diagnosis and improved management practices are essential to reduce disease incidence and economic losses.

Keywords: Avian gout, urolithiasis, poultry kidney disorders, hyperuricemia, urate deposition

Introduction

Gout, together with urolithiasis are economically important diseases in poultry industry due to increased incidence causing production loss, mortality and non-availability of specific treatment (Dhara *et al.*, 2010) ^[44] resulting in up to 40% mortality in broilers (Barathiraja and Mohapatra, 2024) ^[17]. In poultry, close to 29% of all disease conditions had abnormal pathology associated with renal disorders (Siller, 1981) ^[144]. Urolithiasis is an acquired and degenerative disease characterized by severe atrophy of one or both kidneys, distended ureters containing uroliths, have varying degrees of renal and visceral urate deposition and primarily affecting laying hens and male broiler breeders (Moyle JR *et al.*, 2011) ^[92] with increased mortality and decreased egg production (Wideman *et al.*, 1983; Mallinson *et al.*, 1984) ^[166, 85], weight loss and enlargement of the toes, which may be accompanied by depression and dehydration (Fitz-coy SH *et al.*, 1988; Sid H *et al.*, 2011 and Crespo R, 2020) ^[50, 142-143, 38, 121]. Laying hens affected by urolithiasis show decrease in moderate egg production due to atrophy of ovarian follicles (Wideman RF *et al.*, 1983; Sid H *et al.*, 2011 and Crespo R, 2020) ^[166, 142-143, 38, 121]. Among the genetic lines, Leghorns and Cornish crossed chickens have higher predisposition to developing gout (The Poultry Pro, 2025) ^[159]. Urolithiasis is an acquired degenerative kidney disease characterized by focal mineralization of the kidneys and progressive obstruction of the ureters by kidney stones (Wideman Jr RF, 2015) ^[172].

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Gout in chickens is also a clinical manifestation of severe kidney dysfunction characterized by high levels of uric acid in the blood and deposition of urates on internal organs, surfaces and joints. Death from gout is usually due to kidney failure.

The kidney is one of the vital organs that carries out diverse metabolic and excretory functions: maintaining the chemical composition of body fluids, removal of metabolic waste and toxic products, regulation of blood pressure and conservation of fluids and electrolytes. These functions are affected by certain diseases from bacterial, viral, parasitic (Cojean *et al.*, 2020) [34] or physiological or metabolic disorders. One such metabolic disorder associated with kidney damage is gout (Alice Mitchell, 2015) [10]. Gout poses a great challenge within broiler industry because of significant morbidity and mortality (Yadav *et al.*, 2020) [176].

Gout in chicken has multifactorial origin: infectious, nutritional, toxins and combination of factors that damage kidney and impair renal functions (Narsapur, 2011) [96]. A prevalent metabolic disorder in poultry, avian gout is characterized by the excessive buildup of sodium urate crystals (Dalbeth *et al.*, 2016) [39] in tissues due to an arthritic form of hyperuricemia (Yang *et al.*, 2020) [157], and this condition occurs in two distinct forms - visceral and articular gout (Guo *et al.*, 2005; Hong *et al.*, 2020) [56, 59]. Gout is not a disease condition, but a clinical sign of severe kidney dysfunction (Vegad, 2015) [163]. Necropsy findings are sufficient to diagnose (Lierz, 2003; McGavin and Zachary, 2007) [77, 88].

An increase substrate load on the kidney that leads to dysfunction of this organ with precipitation of insoluble products within the kidney or other organs cause urolithiasis or urate deposition (Rocio, 2020) [121]. When kidney functions impaired, uric acid accumulates in the bloodstream or body fluids and deposit and precipitate as calcium sodium urate crystals (Dalbeth *et al.*, 2019) [40] in kidneys, intestine, liver, heart, air sac and joints (Alice Mitchell, 2015) [10]. Hyperuricemia is linked to abnormal purine metabolism, environmental influences (Dalbeth *et al.*, 2016) [39] and chronic renal diseases (Sun LM *et al.*, 2024) [154]. Dietary factors like higher levels of protein and calcium intake, play a crucial role in gout development (Huang KH, 2006) [60]. Bird's lack of the uric acid oxidase and glutamine synthetase enzyme failed ammonia to be converted to urea through the guanine cycle exacerbates the accumulation of uric acid and subsequent tissue damage, leading to hyperuricemia and gout (Li XM *et al.*, 1998; Zhang *et al.*, 2018) [76, 179]. In birds' uric acid is produced from kidneys, intestine, muscles, endothelial cells, however, mainly synthesized in liver and excreted through kidneys (Chaudhary K *et al.*, 2013) [32]. Understanding the mechanisms of gout pathogenesis is crucial to effective management and prevention strategies (Bulbule *et al.*, 2014) [25]. Factors that are associated with poultry gout includes high protein content in feed in order to improve egg production and growth in broilers, excessive salt, calcium-phosphorus imbalance, vitamin deficiency, genetics, diseases and water deprivation (Bulbule *et al.*, 2014) [25]. Gout involves a variety of mechanisms: increased synthesis and decreased metabolism of uric acid caused by abnormal purine metabolism and astrovirus infection (Shao *et al.*, 2017; Zhang *et al.*, 2018) [139, 179].

In gout, the blood levels of uric acid can be as high as 44 mg/100 ml against the normal range of 5-7 mg/100 ml.

Low protein diets, jaggery, vitamin B complex and electrolytes in water help control gout (Sandhyarani *et al.*, 2022) [127], immunoglobulin treatment is another potential

therapy (Divya *et al.*, 2023) [46] in broilers against the visceral gout.

Incidence

Cold stress during winter months results in water refusal. In these dehydrated birds there is excess formation and decreased dissolution of uric acids which results in high mortality due to gout (Srivastava *et al.*, 2002; Jana *et al.*, 2008; Panigrahi *et al.*, 2017; Lakkawar *et al.*, 2018 and Yadav *et al.*, 2020) [151, 64, 110, 176, 64]. Highest prevalence of gout morbidity and mortality was recorded during colder months (Panigrahi *et al.*, 2021) [104] probably due to decrease water consumption being the predisposing factor. The findings of this study are similar to those of Karasawa *et al.* (1991) [70] and Singh N *et al.* (2013) [145].

Summer and rainy season has lower prevalence. Gout during rainy season than summer could be ascribed to poor hygienic conditions (Sayed, 2001) [134] or the highly humid climate that favors rapid mycotoxin growth leading to decreased feed and water intake (Udaya Sai *et al.*, 2023) [162]. But in recent past, higher incidence of gout in chicken has been reported in summer and winter (Wideman *et al.*, 1994; Banday *et al.*, 2009; Singh *et al.*, 2013) [171, 16, 145] in different geographic regions of India causing heavy mortality (Amaravathi *et al.*, 2015; Vishwakarma *et al.*, 2015) [11, 165].

Young chicks less than 10 days old to 3 weeks are more susceptible (Mallinson *et al.*, 1984; Wideman *et al.*, 1994; Olsen *et al.*, 2012; Panigrahi *et al.*, 2021) [85, 171, 100, 104]. Higher brooder temperature can cause dehydration leading to gout in birds.

When pullets sexually matured, to meet demand for eggs production, they are placed on high calcium diets. If the kidneys were previously damaged and no longer function normally with high level of calcium excretion, the result is the gout (Alice Mitchell, 2015) [10]. In laying hens, urolithiasis is a consequence of events occurring during the pullet growing period (Wideman *et al.*, 1985) [167].

Causes and Predisposing factors

Initially considered a nutritional disorder, advancements in diagnostics have identified viral association of gout in poultry (Panigrahi, 2017) [110]. This metabolic disorder has management, infectious and noninfectious cause or their combinations. Factors that can precipitate gout are high calcium and vitamin D₃ with low phosphorus in diet, excess sodium bicarbonate, urea adulteration, water deprivation, vitamin A deficiency (Shane SM, 2005) [137], mycotoxins, nephrotoxic antibiotics such as sulfonamides and aminoglycosides (Schmidt *et al.*, 2003; Sahu *et al.*, 2017) [135, 124]. Prolonged dehydration trigger gout and urolithiasis in poultry (Onderka DK *et al.*, 1987) [101]. Certain viral diseases of poultry like infectious bronchitis virus, avian nephritis virus, infectious bursal disease virus and chicken astro virus can cause gout (Bayry *et al.*, 2005; Bulbule *et al.*, 2014; Gogoi *et al.*, 2017; Panigrahi *et al.*, 2019) [18, 25, 53, 103]. The highest prevalence of gout is recorded during colder months in birds with a definite kidney damage (Yadav *et al.*, 2020) [176]. Turkeys are particularly susceptible due to their genetic predisposition and heritage breeds like Narragansetts and Slate being rather prone to gout.

Nutritional causes

Leeson and Summers (2005) [74] mentioned 1% calcium level as appropriate in chick diets. High calcium in broiler diets lead to some disorders in the physiologic process of kidneys

resulting in urates deposition on serous surfaces of viscera (Pater *et al.*, 2007; Feizi *et al.*, 2011) ^[109, 49]. The optimum crude protein levels as per BIS 2007 is 22 and 20% in broiler starter and finisher rations, respectively. An increase in dietary protein that are rich in nucleoprotein and purine based, including animal viscera, fish meal, soybean (Konstantinov, 1970; Loeb, 1972) ^[72, 81] can cause an increase in kidney size and glomerular filtrate rate with subsequent glomerular injury, accumulation of mesangial deposits leading to glomerulosclerosis (Khan and Alden, 2001; Pater *et al.*, 2007; Singh N *et al.*, 2013; Hong *et al.*, 2020) ^[71, 109, 59, 145]. Nucleoprotein led to more progressive urea formation in the body (Xi *et al.*, 2019) ^[174]. Adulteration of protein supplements with urea increases the nitrogenous component, further enhancing uric acid production which couple with damaged kidney led to gout (Li XM *et al.*, 1998; Alice Mitchell, 2015) ^[76, 10]. Extreme dietary protein (Singh and Ghosh, 2014) ^[146], 30-40% can produce gout in experimental birds.

High fat diet may lead to increase adenosine deaminase and xanthine oxidase activity, that activate purine metabolism and significantly increase serum uric acid levels (Lin *et al.*, 2009) ^[79]. Also, there is increased activity of lipoprotein lipase, hepatic lipase and total esterase causing abnormal lipid metabolism.

Calcium Phosphorus ratio: Excess dietary calcium with low available phosphorus results in precipitation of calcium sodium urate crystals. Immature pullets fed on such diets over a long period of time have damage kidneys. Phosphorus partially protects the kidney against calcium-induced damage and, as a urinary acidifier, prevents stone formation in the kidney. When calcium phosphorus ratio is maintained at 2:1 the kidneys perform its normal physiological functions including secretions, excretions and resorption (Ali and Sultana, 2012) ^[7-8]. Higher levels of proteins cause uric acid production and nephropathy while high calcium with low phosphorus increased retention and decrease uric acid excretion (Mir *et al.*, 2005) ^[89]. Feeding growing pullets, a layer ration high in protein and calcium developed visceral gout (Guo *et al.*, 2005; McGavin *et al.*, 2007; Saif, 2008) ^[56, 88, 123]. Low phosphorus, imbalance between calcium and phosphorus levels (Ansar *et al.*, 2004) ^[12] in another factor.

Sodium intoxication (Sodhi *et al.*, 2008) ^[150], hard water with higher salt content puts stress on kidneys. Sodium bicarbonate used to improve egg shell strength or combat heat stress, if used with high calcium level contribute to gout by making urine more alkaline (alkalosis) thus help formation of kidney stones (Ejaz *et al.*, 2005; Ali *et al.*, 2018) ^[47, 9].

Vitamin A deficiency causes sloughing of tubular epithelium and subsequent blockage resulting in urates accumulation, together, higher dietary vitamin D₃ increase calcium absorption from the gut thereby aggravating deposition of urate crystals (Alice Mitchell, 2015) ^[10].

Water deprivation on account of too high or too low brooding temperature, inadequate number of nipples and faulty water height induced dehydration, cause visceral deposition of urates (Onderka, 1987) ^[101]. Low water pH leads to water rejection by poultry because of epithelial irritability. Chicks held for extended time in hatchery or transportation without water leads to renal failure and gout (Alice Mitchell, 2015) ^[10]. Dehydration is frequently seen in ready to lay pullets after transfer if they do not adapt to drinkers in the production house (Rafael Lera, 2023) ^[114]. Severe or persistent dehydration increase resorption of water causing reduction in urine flow, promoting urate precipitation in renal tubules and

ureters leading to impaction and renal failure (Lierz, 2003; Pollock, 2006; Agim, 2015) ^[77, 113, 3]. But research has not demonstrated a direct role in gout formation due to water.

The mycotoxin poisoning is more harmful. The role of mycotoxins and phytotoxins in the causation of kidney damage and gout in poultry is well documented (Sathiyaseelan M *et al.*, 2018) ^[133]. Aflatoxin being hepatotoxic and a potent nephrotoxic, under high dietary sodium chloride concentration produce degenerative changes in kidney tubules with collection of mononuclear cells along with heterophils (Hamilton, 1971; Rao *et al.*, 1988; Mohmand *et al.*, 2010) ^[57, 118, 90]. Citrinin, ochratoxin A, oosporin and deoxynivalenol (DON) are other hepatotoxic or nephrotoxic mycotoxins that damage kidney and liver thereby reducing uric acid excretion (Guo *et al.*, 2005; Gaba A *et al.*, 2010; Sid and Fettah, 2011) ^[56, 51, 142-143]. The combination of ochratoxin and aflatoxin is found to be more dangerous (Banday *et al.*, 2009) ^[16]. Copper sulphate in water is associated with water refusal, it could lead to gout (Banday *et al.*, 2009; Yadav *et al.*, 2020) ^[16, 176]. Poisoning of feed by disinfectants like cresol and phenol cause hyperuricemia and gout (Chaudhary *et al.*, 2013; Dalbeth *et al.*, 2021) ^[32, 41].

Infectious causes of poultry gout

All nephropathic microorganisms can damage renal function or block urate excretion. The common viruses involved in the pathogenesis of gout are nephropathic strains of infectious bronchitis: Mass and 793B types are most common in India (Elankumaran *et al.*, 1999) ^[48], infectious bursal disease, chicken astro virus and Marek's disease virus (Bayry *et al.*, 2005; Xu *et al.*, 2019) ^[18, 175]. Infectious bronchitis and chicken astrovirus infections have the greatest impact on the poultry industry, usually causing visceral gout in chicken, ducklings and gosling (Reynolds and Schultz-Cherry, 2008; Bulbule *et al.*, 2014; Sanjiv *et al.*, 2023) ^[119, 25, 130] through vertical transmission and high mortality in broilers (Gaba *et al.*, 2010; Panigrahi, 2017) ^[51, 110]. They can also affect reproductive and urinary system due to chronic viral persistence in kidneys. Avian nephritis virus affects kidneys and also causes enteritis in broilers with stunting syndrome and nephropathy (Takashe *et al.*, 1994) ^[155]. A number of factors are responsible for the severity of nephritis associated with IBV: age and breed of the birds, strain of IBV, environment and management, geographical location and immune status of the affected flocks.

Avian astroviruses, including chicken astrovirus, avian nephritis virus and goose astrovirus are associated with enteric disorders, mainly infected through the fecal oral route and cause visceral gout in chicken, duckling and gosling (Linlin Li *et al.*, 2022) ^[78] due to severe kidney damage.

In acute IBDV infection, kidneys appear swollen and mottled (Ley *et al.*, 1979; Ingrao *et al.*, 2013) ^[75, 63] containing urate deposition on account of decreased fluid intake, diarrhea and polyurea in affected chicks (Bulbule *et al.*, 2014) ^[25].

Avian mycoplasmas, pullorum disease, staphylococcus, aspergillus, *Eimeria* and histo-trichomoniasis are of lesser importance in inducing gout in chicken (Pegram *et al.*, 1981; Slemons *et al.*, 1990; Trampel *et al.*, 2000) ^[111, 147, 161].

Cold and wet environment help develop gout by promoting crystallization of urate. When uric acid level is high in poultry, cold temperature and humidity will cause local microvascular contraction and slow down blood flow thus promoting deposition of urates in joints (Chen W *et al.*, 2022) ^[33]. In very cold winter water consumption decreased and ultimately predisposing chicks to gout with mortality

(Karasawa *et al.*, 1991; Singh *et al.*, 2013)^[70, 145] specially in birds under 3 weeks of age (Olsen *et al.*, 2012)^[100].

Urolithiasis is a degenerative kidney lesion primarily seen in laying flocks, however, immature pullet *al.so* experiences outbreaks with mortality exceeding 2-50% over several months (Broadfoot D *et al.*, 1954; Blaxland JD *et al.*, 1980; Cowen BS *et al.*, 1987)^[22, 21, 37] and it is characterized by severe atrophy of one or both kidneys, distended ureters often containing uroliths with varying degrees of renal and visceral urate deposits. Excess calcium fed during growing period are linked to this condition. The effect of excess calcium is accelerated if the diet contains low available phosphorus (Shane *et al.*, 1968; Wideman *et al.*, 1985)^[138, 167], although, other causes include: Chicks or pullets' exposure to nephropathic IB virus like the Gray strain (Niznik *et al.*, 1985; Glahn *et al.*, 1989)^[98, 52] and renal cryptosporidiosis (Trampel DW *et al.*, 2000)^[161], water deprivation, metabolic alkalosis induced by elevated dietary sodium and potassium over chloride ratios (Siller, 1981; Julian, 1982)^[144, 68]. Vitamin A deficiency (Siller W, 1981)^[144], treatment with sodium bicarbonate (Davison S *et al.*, 1992)^[42] and mycotoxins such as Oosporein (Pegram R *et al.*, 1981)^[111] are some of the noninfectious factors that induced urolithiasis. Diclofenac sodium results in uric acid crystal deposition, necrosis of hepatocytes and monocytes, Leucocyte infiltration of the liver and heart parenchyma and visceral gout in broiler chickens (Sandhyarani K *et al.*, 2019)^[126].

Pathogenesis

Avian gout spreads the world over with mortality over 37% (Rahul, 2025)^[115]. Overproduction and underexcretion of urates because of decreased kidney function led to hyperuricemia and monosodium crystal deposition on serosal surfaces, often start at the heart then to kidney, liver and air sacs (Saif, 2008; Sandhyarani *et al.*, 2019; Liu C *et al.*, 2022)^[123, 126], gradually to other organs. The precipitation of urates on surface of different visceral organs might be attributed to the kidney damage leading to hyperuricemia (Siller, 1981)^[144].

Visceral gout is the acute form of the disease, associated with huge mortality and its ultimate pathogenesis not completely understood. Any injury that causes damage to kidney interferes with the elimination of uric acid, which then accumulate in the blood (hyperuricemia), leading to visceral gout (Aruna, 2023)^[13]. The damaged kidneys appear atrophied with whitish gritty materials and enlarged ureters. Compensatory enlargement of other parts of normal kidneys observed to maintain adequate renal function (Siller *et al.*, 1981; Goryo *et al.*, 1984; Jones *et al.*, 1998; Banday *et al.*, 2009)^[144, 55, 6, 7, 16].

Birds are more prone to gout because they are uricotelic - the end product of protein metabolism is mainly uric acid as they lack the enzyme uricase which converts uric acid to allantoin. Uric acid is water insoluble, can form urate deposition with cations such as sodium, potassium and calcium under certain pH conditions (Kanbara *et al.*, 2012)^[69]. Sometimes precipitated monosodium crystals trigger a chain of events with progressive hyperuricemia and precipitation of urate at predilection sites, terminating into death or that end in joint injury (Hong *et al.*, 2020)^[59]. Those areas where the solubility of sodium urate is lower than other areas constitute the predilection site. Lower temperature reduces urate solubility and favor precipitation, with time form tophi (Sanjiv *et al.*, 2023)^[130]. Once crystals are deposited into a joint, inflammatory reactions activate complement to generate

C3a and C5a which induce accumulation of neutrophils and macrophages in the joints, synovial membranes and cartilage to release proteases to develop acute gouty arthritis (Ali and Sultana, 2012; Vegad, 2015)^[7-8, 163] which further exacerbate renal excretory dysfunction (Xi *et al.*, 2019)^[174].

In poultry, kidney is the only organ, depending on its structure and function, is responsible for normal excretion and reabsorption of uric acid (Shiderman *et al.*, 1981)^[140]. The physiologic impact of the kidney damage was the result of reduced renal mass, rather than inappropriate renal handling of minerals and electrolytes (Wideman *et al.*, 1992) as the number of glomeruli reduced significantly in birds affected by urolithiasis (Niznik *et al.*, 1985)^[98].

If secretion by renal tubules is reduced, obstruction of uric acid excretion led to hyperuricemia. New Hampshire chickens are genetically prone to articular gout due to a defective gene associated with uric acid transport mechanism in renal tubules (Cole *et al.*, 1980)^[36]. These hyperuricemia susceptible strains of poultry produced more alkaline urine and had higher proportion of juxtamedullary nephrons (Wideman *et al.*, 1992)^[170].

High calcium diet and long-term vitamin A or D₃ deficiency causes gout in poultry (Guo *et al.*, 2005)^[56] by increasing calcium ion concentration and deposition in renal tubular epithelial cells, leading to the formation of kidney stones and chronic renal insufficiency eventually causing gout (Marcocci *et al.*, 2011; Ding *et al.*, 2019)^[36, 45]. In such situations the ratio of calcium to phosphorus is unbalanced and low phosphorus promote gout (Alagawany *et al.*, 2021)^[6].

Vitamin A deficiency can led to keratinization of renal tubules and ureteral epithelium, a condition not conducive to the excretion of uric acid and phosphorus, ultimately renal failure (Chandra *et al.*, 1984). Lack of vitamin D₃ lead to mineral metabolism disorder, proportion imbalance and excretion obstacles. However, excessive inclusion of vitamin D supplementation cause hyperuricemia and gout.

Uroliths are composed of compact masses of microcrystalline to fine pleomorphic crystals of calcium sodium urate, with random substitute of magnesium and potassium for the calcium and sodium, respectively (Oldroyd N, 1986). The physiologic impact of kidney damage was the result of reduced renal mass rather than inappropriate renal handling of minerals and electrolytes as is evident by a significant reduction of glomeruli numbers in urolithiasis affected birds (Niznik RA *et al.*, 1989). The uroliths may cause sudden death by blockage of ureters, probably occurring secondary to kidney damage (Mallinson *et al.*, 1984)^[85].

Chicken astrovirus, avian nephritis virus, aspergillus nephritis and nephropathic infectious bronchitis virus acts on renal tissues, causing organic lesions increasing uric acid production from purine metabolism eventually leading to gout (Jin *et al.*, 2018; Wu *et al.*, 2020)^[66, 173]. Chicken astrovirus has been isolated from gout affected chicks in commercial broilers in India (Bulbule *et al.*, 2013)^[24].

Increased ammonia and carbon-monoxide in sheds affect normal ventilation, reduced water intake leading to nonavailability of adequate water to flush out the urinary system and clogging (Phatak, 2001)^[112].

Symptoms

Gout affected birds are dull, dehydrated, ruffled feathers, showed segregator and restlessness behavior, have moist vent with whitish droppings. They exhibit difficult movement and standing on legs, shift weight from one foot to other. Birds have swollen feet, blisters and sores, joint pain and

immobility (Aktar and Sarkar, 2015. Sandhyarani, 2022) ^[5, 127]. Mortality in young chicks is high.

Other signs include decreased appetite, debility, weight loss, chalky urates in stool and change in behavior (Ali and Sultan, 2012; Patel NJ *et al.*, 2014; Aktar and Sarkar, 2015) ^[7-8].

Chickens affected with urolithiasis die suddenly, in good condition and in full lay, have reduced muscle mass, small pale comb, also white pasting on peri cloacal feathers (Blaxland JD *et al.*, 1980; Brown TP *et al.*, 1987) ^[21, 23].

Pathological chemistry

Chicken affected with gout have a significantly increased serum uric acid levels reaching up to 44 mg/ml; more than 5-7 mg/100 ml, indicating renal dysfunction due to impaired uric acid excretion or increased nucleic acid degradation and serum creatinine, over 0.7-22 mg/dl due to the failure of excretory function or degenerative changes in organs (Chandra *et al.*, 1985) ^[29] reflecting a decline glomerular filtration rate (Benjamin. 1978) ^[20]. As per Narsapur VS (2011) ^[96], blood uric acid level of 10-20 mg/dl is enough to appear gout symptoms and when this rises to 25-42 mg/dl there is severe gout mortality. High levels of uric acid in blood precipitates monosodium/calcium urate crystals in tissues or organs and cause physical damage and inflammatory reactions. Broilers that suffered from gout in early age could recover completely, however, greater is the loss due to lower weight gain and higher feed conversion ratio. Death from gout is usually due to kidney failure.

Non-steroidal anti-inflammatory drugs (NSAID) repeated usage over a prolonged period induced renal dysfunction (Shafi *et al.*, 2015) ^[136]. High calcium high protein diets increase uric acid in the birds (Chandra *et al.*, 1984; Guo *et al.*, 2005) ^[56]. Hematological changes such as increased PCV, TEC, Hb values suggest dehydration due to diarrhea (Jana *et al.*, 2008; Singh *et al.*, 2013; Yadav *et al.*, 2020) ^[176, 64, 145]. Leukocytosis suggests the onset of cellular immunological response to infections (Thanagari *et al.*, 2012) ^[158] or bone marrow stimulation (Singh *et al.*, 2013; Shafi *et al.*, 2015 and Salam, 2017) ^[145, 136]. Antioxidant profiling reveals decreased glutathione stimulating hormone (GSH) which induce oxidative stress and promote pathogenesis of many diseases (Sandhyarani *et al.*, 2019) ^[126]. Glutathione participates in many cellular reactions to scavenge free radicals (Lu, 1999) and detoxify and eliminate xenobiotics. Increased Thio barbituric acid reactive substances (TBARS) in kidney and liver tissues antioxidant profile better predict oxidative damage (Morrow, 2000). The raised superoxide dismutase level and malondialdehyde (MDA) in renal tissues suggest cell damage in kidney (Gokcimen *et al.*, 2001) ^[54].

Types of Gout

Visceral, articular and renal gout are the three major forms which are differentiated based on the sites of uric acid deposition that consist needle shaped crystals called tophi.



Visceral Gout: It is an acute form of the metabolic disease (Zhang *et al.*, 2018) ^[179] causing huge mortality, characterized by urate deposition on serosal surfaces of multiple visceral organs mainly liver, kidneys, pericardium, air sacs as well as peritoneal cavity and the thoracic walls (Rexhepi *et al.* 2015; Avantika S, 2024) ^[120, 15]. Uric acid itself is not toxic to poultry but precipitated crystals can cause severe damage to tissues (Majid S *et al.*, 2025). The condition predominantly affects young broilers of 1-3 days old and layers less than 1-4 weeks (Wideman *et al.*, 1988) ^[169]. Pullets above 14 weeks are more likely affected. Kidney dysfunction leads to hyperuricemia causing urate accumulation in tissues and tubules are distended with urate crystals (Banday *et al.*, 2009) ^[16]. A sudden onset and complete obstruction of one ureter led to rapid atrophy of the corresponding kidney and this might be responsible for pressure atrophy of the kidney upstream from the blockage and loss of renal parenchyma (Wideman, 1987) ^[168]. Both males and females are susceptible and the general cause of visceral gout is renal failure to excrete urates (Rocio, 2020) ^[121].

In chicken infected with avian astrovirus, histopathological studies reveal urate deposits in kidneys, necrosis and degeneration of the epithelial shells of the proximal convoluted tubules with infiltration of granulocytes, lymphatic infiltrations and interstitial nephritis cyst like formation in crypt of the duodenum (Abdullahi Raji and Omar AR, 2022) ^[1]. Severe congestion in the heart, necrotic foci and degenerative changes and urate deposition around the central vein in liver and mild to moderate interstitial nephrosis in kidneys were found in dead chicken embryos (Panigrahi *et al.*, 2019). The increased permeability of kidney epithelial cells induced by astrovirus certainly explain the development of gout (Bulbule *et al.*, 2013) ^[24]. Visceral gout occurs if the rate of urate formation is greater than the excretion capacity of urinary organs (Tang *et al.*, 2018) ^[156].

In 2011-2012, India reported a 40% mortality rate in broilers with visceral and articular gout and the pathogen being India subgroup Biii CAStV (Bulbule NR *et al.*, 2013; Smyth VJ *et al.*, 2017; Panigrahi S *et al.*, 2019) ^[24, 149].

The two species of astrovirus infecting chicken are Avian Nephritis virus (ANV) and Chicken Astrovirus (CAStV). ANV infects only the chicken, most susceptible in day old to 4 weeks of age (Imada and Kawamura, 1979) ^[61] specially among the immunocompromised chicks (Narita *et al.*, 1990) and are associated with severe interstitial nephritis of varying pathogenicity and tissue tropism (Decaessteck *et al.*, 1989) ^[43]. Runting stunting syndrome, underperformance and growth checks in chicken have been under the responsibility of CAStV strains causing severe kidney damage leading to mortality of about 65% in broilers against 100% in specified pathogen free chicken (Imada *et al.*, 2000; Patel *et al.*, 2017) ^[62]. Vaccination of broiler breeders against CAStV could protect the chicken during the first few days of life, and thus preventing the disease (Abdullahi Raji and Omar AR, 2022) ^[1].



Articular Gout: It is a chronic form of the disease, a sporadic individual problem of less economic importance, affecting birds above 4-5 months of age. High protein diet and genetics cause a defect in tubular secretion of uric acid (Austic and Cole, 1972) ^[14]. The genetic link to spontaneous articular gout in New Hampshire breed is the consequence of selection anomalies in the transport of uric acid in renal tubules (Cole and Austic, 1980) ^[36]. Deposition of urate crystals are mainly observed on synovial surface of toes, wing joints, hip joints and inciting granulomatous inflammation (Lumeij JT, 1994) ^[83]. Urate deposition has predilection for peripheral articulations (Banday *et al.*, 2009) ^[16]. The joints become swollen, red, tender and warm to touch. The most commonly affected wing joint in birds is the wrist (Ali and Sultana, 2012) ^[7-8]. Soft tissues around the joints, especially feet are involved, however, very rarely comb, wattle and trachea may also be affected (Shivaprasad *et al.*, 1998) ^[141]. It is also stated that factors like synovial pH, water and electrolyte level or components in the synovium such as proteoglycans and collagen affect uric acid solubility in joints (Aruna, 2023) ^[13]. Rarely systemic in nature, kidneys appear normal unless dehydration occurs (Ali and Sultana, 2012) ^[7-8].

Birds with articular gout have prolonged duration of the disease, looks depressed, dehydrated, have moist vent and greenish diarrhea but without internal organ damage. In the early stages, the swelling is soft and painful that gradually harden to form bean sized nodules that are either mobile or immobile (Roman, 2023) ^[122]. They prefer to sit on flat surface than walk because of the pains. If force to walk, the birds become noisy due to discomfort. When swollen joints are cut open, white milky urates flow from the joint cavity.



Renal gout: Urate crystals are deposited in renal parenchyma. Kidney tubules distended with calcium urate calculi. Kidney lobules are enlarged to maintain renal function. Chicken fed high cholesterol diets are more prone to renal disease. Renal problems caused by IBV often follow an initial respiratory infection in broilers and kidneys are pale, swollen, blotchy and distended tubules are white (Cook J, 2009) ^[35].

Postmortem Findings

Rapid screening of birds reveals emaciated and dehydrated carcasses with chalky white urate deposits on subcutaneous tissues over various organs including the pericardium, liver, kidneys, ureters, lungs, spleen, surfaces of the breast muscle, serosal surfaces of the gastrointestinal tract and air sacs (Sandhyarani *et al.*, 2019; Yadav *et al.*, 2020) ^[126, 176].

Visceral congestion could be attributable to emaciation and dehydration of the birds leading to hemoconcentration (Satalkar A, 2007) ^[132]. Gross lesions reveal chalky white urate deposits on serosal surfaces of organs while histopathology demonstrates urate crystal deposition with inflammatory cell infiltration (Adnan Y *et al.*, 2025) ^[2].

Kidneys shows consistent nephropathy either unilateral or bilateral often characterized by enlargement with moderate to severe congestion and bulging out of the renal fossa. Frosted appearance of kidneys is due to the accumulation of urate crystals with pin point hemorrhages on the serosal surface. Ureters of either side distended with retained semi fluid to semi solid chalky urates, giving them a cord-like appearance. The ureters were markedly dilated and filled with mucus or molded with yellow -grey uroliths (Lopes MC *et al.*, 2021) or due to blockage by uroliths with varying degrees of associated kidney degeneration (Lakkawar *et al.*, 2018) ^[79]. Urolithiasis characterized by blockage of one or both ureters by urate concretions are other major lesions seen particularly in caged laying hens (Charlton *et al.*, 2006) ^[31].

Microscopically, there is marked congestion and hemorrhages involving glomeruli, cortical and medullary tubules, collecting ducts and medullary tracts with, focal to diffuse degenerative changes in tubular epithelium, such as vacuolar degeneration, necrosed epithelial cells and varying degrees of fibrosis (Blaxland J *et al.*, 1980) ^[21]. Some tubules have degenerated heterophils or proteinaceous cast in the lumen which may get ruptured due to crystalline formation with multinucleated giant cells, Tophi formation (Agim R *et al.*, 2015) ^[3]. Uric acid aggregates to form needle shaped crystals as blue or pink radiating amorphous material surrounded by inflammatory cells in kidney parenchyma (Muhammed *et al.*, 2012; Bulbule *et al.*, 2013) ^[24] and the extend of kidney damage is directly correlated to the degree of urate deposition (Chandra and Balwant, 1980; Chandra R *et al.*, 1998) ^[27, 30]. Much of the urate deposits are lost when tissues are processed for histopathology, but evidence of its presence is seen as amorphous material under microscope. Hyaline cast were present in some tubular lumen with glomerular changes such as atrophy, distortion and segmentation (Mir *et al.*, 2005; Feizi *et al.*, 2011) ^[49]. Feathery crystals or basophilic spherical masses may be seen within tissues under the microscope in few cases (Rocio Crespo, 2020) ^[38, 121].

Liver is enlarged and friable with chalky white urate deposition on the capsular surface. The liver had sections of individual hepatocyte necrosis, severe sinusoidal congestion with fibrin thrombi evident multifocally, hemorrhages in parenchyma and fatty changes (Feizi *et al.*, 2011; Sultana *et al.*, 2012; Nitin and Ghose, 2014; Patel *et al.*, 2014; Amaravathi *et al.*, 2015; Agim R *et al.*, 2015; Behtari and Feizi, 2015; Ramzan *et al.*, 2015; Sandhyarani *et al.*, 2019) ^[49, 152, 97, 101, 11, 3, 19, 116, 126, 117]. The subcapsular hepatocytes flattened with elongated nucleus and intense cytoplasmic basophilia along with proliferation of fibrous connective tissues in Glisson's capsule (Mir *et al.*, 2005; Mudasir *et al.*, 2017) ^[93].

Pericardium firmly adhered to the heart displaying varying degrees of urate deposits on its serosal surface and crystals deposits on the epicardium - uric acid pericarditis with myocardial congestion due to severe blood vessel engorgement and focal to diffuse hemorrhages between muscle fibers (Ramzan *et al.*, 2012; Mudasir *et al.*, 2017) ^[193, 16].

The lungs showed urate deposition in parenchyma, air capillaries and parabronchi. Edematous fluids present in the

air capillaries and atria leading to emphysema and atelectasis (Mir *et al.*, 2005) ^[89].

Spleen showed splenitis, subcapsular hemorrhages and urate deposition in the subcapsular region and parenchyma (Harr *et al.*, 2002; Mir *et al.*, 2005) ^[58, 89]. There was depletion of lymphocytes along with the presence of needle like urate crystals (Mudasir *et al.*, 2017; Panigrahi *et al.*, 2021).

Chicken with urolithiasis show moderate drop in egg production (Crespo R, 2020) ^[38, 121], accompanied by depression, dehydration (Fitz-coy SF *et al.*, 1988) ^[50] weight loss and enlargement of toes. The ureters are distended by uroliths (Sid H *et al.*, 2011) ^[142-143].

Diagnosis

Complete history, signs and lesions could help diagnosis of avian gout. Microscopically, dark colored sharp acicular crystals seen in the smear of tophi with De Glantha stain and by determination of uric acid concentration through HPCL, biosensor method, phosphotungstic acid methods and uricase method (Sandhyarani *et al.*, 2022) ^[127]. Blood test to assess serum uric acid and creatinine levels and X rays or ultrasound image detect crystal build up in joints (Rahul S, 2025) ^[115]. RT-PCR or quantitative real time PCR are used for initial screening and confirmation of CAstV virus isolates (Tang *et al.*, 2005; Todd *et al.*, 2009; Smyth *et al.*, 2010) ^[157, 160, 148] from kidney samples of gout affected chicks and molecular characterization has been done by nucleotide sequencing of the ORF1a and ORF1b regions (Pantin-Jackwood *et al.*, 2006; Bulbule *et al.*, 2013) ^[24]. Murexidase test (Narsapur VS, 2011) ^[96] on the deposits recovered from dead birds confirm urate deposits.

Treatment and Prevention.

The counter measures to treat gout are limited and often inadequate. Some important aspects in deciding therapeutic regimen are:

- Lowering of the uric acid level and normalization of renal function, the easiest and most vital being boosting water consumption else medication can be futile.
- As per requirements antiviral or mycotoxin treatment.

The use of xanthine oxidase inhibitors such as allopurinol and febuxostat is gaining importance in poultry gout management (Pavani Veldi *et al.*, 2023; Sanjiv K *et al.*, 2023) ^[110, 130].

Allopurinol at 10-15 mg per Kg body weight administered orally or intramuscular injection once a day. Allopurinol is a xanthine oxidase inhibitor drug that decrease formation of uric acid and also help in the dissolution of uric acid crystals thus prevent formation of chronic arthritis (Sun *et al.*, 2022) ^[153]. Long term allopurinol use is linked to liver damage. However, relevant experiments showed that Piper-betle L is more efficacious than allopurinol in the treatment of avian gout (Chakravarthi *et al.*, 2021) ^[26].

Febuxostat, 80-120 mg daily reduce uric acid by inhibiting xanthine oxidase (Park *et al.*, 2022) ^[106, 131] has better ameliorative potential than allopurinol in birds. Extended usage leads to fibrosis and chronic hepatotoxicity (Sanjiv *et al.*, 2019) ^[128].

Dexamethasone, a hormone class by increasing enzyme activity and metabolism in the body reduce uric acid deposition is currently the most effective in the treatment of avian gout with success rate as high as 92% (Chen Wei *et al.*, 2022) ^[33].

Birds undergo a phenomenal debility during gout. Sorbitol and propylene glycol act as ready source of energy to help maintain feeding behavior and encourage performance.

Probenecid is of great significance in the treatment of chronic avian gout as it reduces urate concentration by inhibiting urate reabsorption and accelerate uric acid dissolution.

A recent study by Vikrama *et al.* (2022) ^[164] concludes that pepper extracts alleviate gout symptoms by increasing antioxidant capacity in broilers.

Low protein feed balanced with important amino acids, that meets nutritional needs without overloading the system, provision for adequate water and vitamin A supplementation help in checking the progression of the disease but not reversal of tissue damage. Addition of oats, barley or alfalfa meal reduce protein level but also provide essential vitamins and minerals. Urine acidification with ammonium chloride or ammonium sulphate @ 2.5-7.5 kg/ton feed for 7-10 days enhanced elimination of urates.

Sodium bicarbonate and Potassium chloride @ 1 gm per liter water due to its urine alkalization make uric acid less likely accumulate and crystallize in urine that greatly reduce mortality. During dehydration, the minerals potassium, sodium and chloride play essential roles in maintaining osmotic balance, acid-base balance and integrity of mechanisms that regulate the transport of substances across the cell membrane. Ensure the feed's appropriate level of sodium, chloride and other ions. However, it is better to avoid use of sodium bicarbonate more than 2 kg/ton feed.

High level of methionine in feed is helpful. DL Methionine or methionine hydroxy analogue can be used @ 6 kg/ton feed to dissolve kidney stones (Banday, 2009) ^[16]. Increasing calcium level to almost 3% is another consideration as calcium mitigate uric acid production. Ensure adequate levels of vitamin A, D, K, B complex and minerals in diets along with ammonium sulphate or ammonium chloride at correct levels will minimize load on kidneys. Raw materials used in poultry diets should be of good quality and adding toxin binders when needed will limit the impact of mycotoxins (Rafael Lara, 2023) ^[114].

Detection and isolation of infectious bronchitis associated with gout, CAstV induced gout in broilers could be prevented by vaccination in breeders (Reynolds DL *et al.*, 2008) ^[119]. Chicks infected with CAstV are extremely susceptible to dehydration, therefore, fresh water and electrolytes should be made available whenever possible giving sufficient drinking space. Jaggery @ 5-7 gm per liter water for 3 days reduce mortality (Ahmed, 2003; Singh N *et al.*, 2013; Adnan *et al.*, 2025) ^[4, 145, 2]. Periodically check the water quality to avoid developing waterborne diseases.

In infectious bronchitis endemic areas, use of nephrogenic strain IB live spray vaccine at hatchery or beak dip on arrival or at least within 4 days is proven to be beneficial in commercials. Vaccination of astrovirus inclusive of ANV and CAstV to breeders prevent vertically transmitted virus to their progeny.

Farm and hatchery could play a pivotal role in prevention of acute or chronic gout. Optimum brooding temperature, adequate ventilation and humidity at farm and hatchery ensuring that chicks from eggs stored and handled optimally are not dehydrated during long distance transport (Alice Mitchell, 2015) ^[10].

In chickens the most common immunoglobulin is IgY, which is present in the blood and neutralizes invading pathogens by triggering complement proteins and gets eliminated thus give a robust immune system against illnesses (Jia M, 2021; Zymanczyk-Duba *et al.*, 2022) ^[65, 180]. IgY treated chicks showed significant improvement in body mass, gamma-glutamyl transferase and uric acid levels and mortality over 6

weeks similar to the control group confirming the effectiveness of immunoglobulin treatment (Divya B *et al.*, 2023) ^[46].

Conflict of Interest

Not available.

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