

Gout in poultry

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Avian gout is a metabolic disorder in which high level of uric acid in the blood (hyperuricaemia) subsequently precipitates as monosodium/calcium urate crystals in a variety of locations, particularly in the kidneys and on the serous membranes of various internal organs (liver, heart, air sacs) or various joints. These crystals, being insoluble in tissue fluids and having sharp ends, cause physical damage to the tissues leading to a cascading inflammatory reaction. (Sandhyarani K *et al.*, 2022). Avian gout is responsible for a great deal of morbidity (sickness) and mortality (deaths) in both broilers and layers.

Pathophysiology:

Uric acid is the primary catabolic product of protein, non-protein nitrogen and purines in birds. Birds excrete uric acid as primary nitrogen metabolite which comprises of 70-80% of excreted nitrogen in urine. The birds lack the enzyme uricase, which converts uric acid into allantoin. It is quite evident in poultry that kidney dysfunction can play vital role in the development of gout unlike mammals which availability of enzyme hypoxanthine guanine phosphoribosyl transferase (HGPRT) for conducting salvage pathway to recycle purine break down product.

Etiology:

Gout is a multifactorial metabolic disease mainly due to damaged kidneys (nephropathy) from any number of potential causes which includes nutritional or metabolic causes like excess dietary calcium, high protein diet, excess salt, low phosphorus, imbalance between Ca-P levels, adulteration of feed with urea and deficiencies of vitamin A and D. Water deprivation followed by over dosages of certain drugs like sulphas and the antibiotic aminoglycosides often causes damage. Mycotoxins phytotoxins in feed, Infectious causes like IB (Infectious Bronchitis), Reo viral

infection, Avian Nephritis, Chicken astro virus, Infectious Bursal disease and Inclusion body hepatitis. In addition, some managemental stress factors including high brooding temperature and higher level of ammonia concentration in the shed can also cause high mortality due to gout. (Sanjiv Kumar *et al.*, 2023)

Types of Gout:

Based on the location of urate crystals deposition, Avian gout is classified as visceral gout and articular gout.

- Visceral gout, also called 'avian urolithiasis', it is characterized by deposition of urate in the thoracic and abdominal cavity and on the surface of the viscera such as the kidney, heart, liver, mesentery and peritoneum, often accompanied by renal failure.
- Articular gout is characterized by urate deposition in the joint capsule, articular cartilage and surrounding tissue.

Pathology:

Visceral gout is a common clinical disease in poultry. Typical clinical symptoms include anorexia, depression, strong desire to drink, diarrhea and staining of the feathers around the cloaca with white faeces. The disease is often caused by serious failure of multiple





organs. Grossly, dry patches of white chalky urate deposits were observed on the serosal surfaces of pericardium, air sacs, peritoneum, liver, kidneys and ureters (Fig.1).

Nephropathy, with unilateral to bilateral enlargement and snowflake pattern on their surface and parenchyma with moderate to severe congestion of kidneys (Fig.2). Ureters thicken and harden due to Urate obstruction. Microscopically, marked congestion, hameorrhages and focal to diffuse degenerative changes in tubules and glomeruli. Hyaline casts were present in few tubular lumens.

Liver is enlarged, friable with white chalky urate deposition on the surface of the capsule. Microscopically severe sinusoidal congestion, haemorrhages in parenchyma, fatty change and foci of necrotic hepatocytes and proliferation of fibrous connective tissue in glisson's capsule.

In heart, the pericardium is covered with urate deposits on its serosal surface i.e uric acid pericarditis. Pericardium is firmly adhered to heart. Myocardium showed urate deposition along with destruction of myocardial cells and infiltration of inflammatory cells, myocardial congestion and focal to diffuse haemorrhages between muscle fibers.



Fig 1 Chalky white deposits on pericardial sac (Y Ravikumar *et al.*,2018)

Lungs showed deposition of urates in the parenchyma, air capillaries and parabronchi. The air capillaries near tophi are collapsed and the remaining which are away from urate deposition showed emphysema. Oedematous fluid was present in air capillaries and atria.

Microscopically parenchyma visceral organs exhibits aggregates of uric acid crystals characterized by needle shaped urate crystals as pink radiating amorphous material surrounded by a narrow zone of inflammatory cells (Fig.3) .These uric acid crystals were observed in black color by De Galantha's stain (Fig.4). Birds with articular gout have a prolonged duration (Chronic form) of disease without internal organ damage. Deposits develop on synovial membranes in the toes (Fig.5) and wing joints and incite a chronic granulomatous reaction to urate crystals (Tophi). In the early stage, the swelling is soft and painful with no obvious boundaries; the mid-swollen parts gradually become hard, forming pea or broad bean sized nodules that are either slightly mobile or immobile. swollen joints are cut open, white, milky urate flows from the joint cavity (Fig.6) (Linlin wang et al., 2024)



Fig 2 Urate deposition in kidneys and ureters with enlargement (Y Ravikumar *et al.*,2018)





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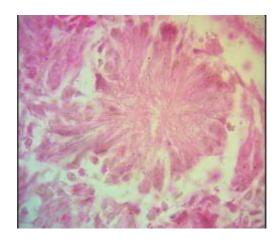


Fig.3 Section of kidneys showing radiations of needle like urate crystals (Lakshmi Namratha *et al.*, 2019)

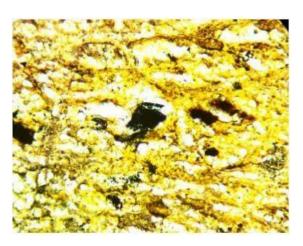


Fig.4 Photo micrograph of kidney showing black colored urate crystals Degalantha's stain X 400 (Sandhyarani K *et al.*, 2022)



Fig.5 Deposits develop on synovial membranes in the toes-granulomatous reaction to urate crystals (Tophi).



Fig.6 Cut section of swollen joints white, milky urate flows from the joint cavity

Prevention And Treatment

The following methods can be employed for treatment and prevention of gout.

- Minimize dehydration, get free and ample access to drinking water
- Ensure correct level of calcium and phosphorous levels in feed.
- Use toxin binders and liver tonics to keep minimum levels of fungal toxins in feed. Avoid excessive protein levels in diet over suggested.
- Use recommended levels of aluminium-free Sodium bicarbonate (baking soda) in feed

during high temperatures. Use of Jaggery 2 to 5 grams per liter. of

- water or electrolyte at recommended doses in water.
- Review Infectious Bronchitis and infectious bursal disease vaccine programme in Breeders and broiler farms
- Some of the drugs like Dexamethasone, Allopurinol, , Probenecid, Colchicines, Piperbetle-l (Chakravarthi PV et al., 2021)) are also effective against gout.





• High level of Methionine in feed also found helpful to some extent.

References

- Chakravarthi PV et al., 2021. Therapeutic antigout and antioxidant activity of piper betle l. in gout-induced broilers. British Poultry Science 19: 1-8.
- Linlin wang, Jialin, Bo wang, Xianglin yin, Jinfeng wei, and Hongbin qiu (2025). Progress in modeling avian hyperuricemia and gout (Review), BIOMEDICAL REPORTS 22: 1, 2025
- Sandhyarani K, Madhuri D, Ravikumar Y (2022). Review gout in

- chicken. Adv. Anim.Vet. Sci. 10(3): 702-711.
- Sanjiv Kumar, Sudhanshu Kumar and Ajeet Kumar (2023).

 Understanding the mechanism of gout in poultry and its management International Journal of Veterinary Sciences and Animal Husbandry; SP-8(1): 28-33
- Y Ravikumar, Sawale GK, G Ramesh, B Mahesh, D Madhuri and M Lakshman (2018), Visceral gout in ducks, The Pharma Innovation Journal 2019; 8(1): 256-257

