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Gout in poultry

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Abstract
Poultry industry is facing a huge challenge to reduce the economic losses due to gout particularly when it affects poultry. Uric acid is produced in the liver and end product of nitrogen metabolism in birds. Significantly high serum uric acid level (hyperuricemia) in a super saturated state may get deposited in kidney or joints or visceral organs. Function of kidneys restore by way of providing vitamin A supplementation, change of feed formulations, use of toxin binders and agents which could regulate renal physiology i.e. nephroprotective agents. The use of xanthine oxidase inhibitors *viz*. allopurinol and febuxostat is increasingly gaining importance in poultry gout management.

Keywords: Poultry, hyperuricemia, kidneys

Introduction
Gout is a common metabolic disorder that results in abnormal accumulation of urates in poultry. Uric acid production Nitrogenous base, Purine i.e. adenine and guanine are the precursor of uric acid and the source of purines may be endogenous as a result of cell death or it may be exogenously acquired through feeds, particularly animal protein feed. Uric acid in birds is produced from the different body organs like kidneys, intestine, muscles, endothelial cells, however, mainly synthesized in liver and excreted via kidneys [1].

Etiology: Gout in poultry may be due to infections, nutritional imbalance, toxicity or impaired renal functions that it is of multifactorial origin. As uric acid is excreted through kidneys, 70% of the kidney dysfunction is attributed to formation of hyperuricemia and gout. Deficiency of vitamins and minerals. Viral infection causing nephropathy like avian nephritis, astrovirus, Infectious Bursal Disease are among the important causes. Injudicious use of antibiotics like gentamycin, nitrofurosones etc. or poisoning of feed by disinfectants like cresol and phenol, can all be other causes of hyperuricemia and thus gout [2, 3].

Pathogenesis of gout: Birds are more prone to gout because they are uricotelic, the waste product of protein metabolism is mainly in the form of uricacid. In birds are lack uricase enzyme which converts uricacid into allontoin. Uricacid is water soluble, any injury or damage to birds kidney, whatever cause it interferes with elimination of uricacid, it accumulates in the blood and it leads to visceral gout. Deposition of urate crystals on the surfaces of internal organs. The crystals deposited in joints its leads to articular gout.

Types, Clinical Signs and lesions: Based on location of urate deposition, gout in poultry has been classified as: [a] Visceral gout (urate deposits are seen on one or multiple visceral organs as well as on the peritoneal surface or thoracic wall), [b] Renal gout (urate crystals are mainly deposited in renal parenchyma) and [c] Articular gout (deposition of urate crystals are observed in synovial surface of tarso-metatarsal joint as well as in hip joint). In visceral gout, kidneys tubules are distended with urate crystals shows brittle white staghorn calcium urate calculi. Urate deposits are seen on one or multiple visceral organs as well as on the peritoneal surface or thoracic wall. In articular form, urate deposition develops on synovial membrane of tarso-metatarsal joint as well as in hip joint and wing joint and incite a granulomatous reaction [4].

It is also being stated that factors like synovial pH, water and electrolytes level or some other components in the synovium such as proteoglycans and collagen may affect the solubility of uric acid in the joint [5].

Gross lesions: Articular gout characterized by tophi, the white chalky crystals deposit around joints, particularly feet region, the joints are enlarged. In visceral form, the parietal surface of pericardium is covered by a thin layer of garish granular material that may have a metalic sheen.

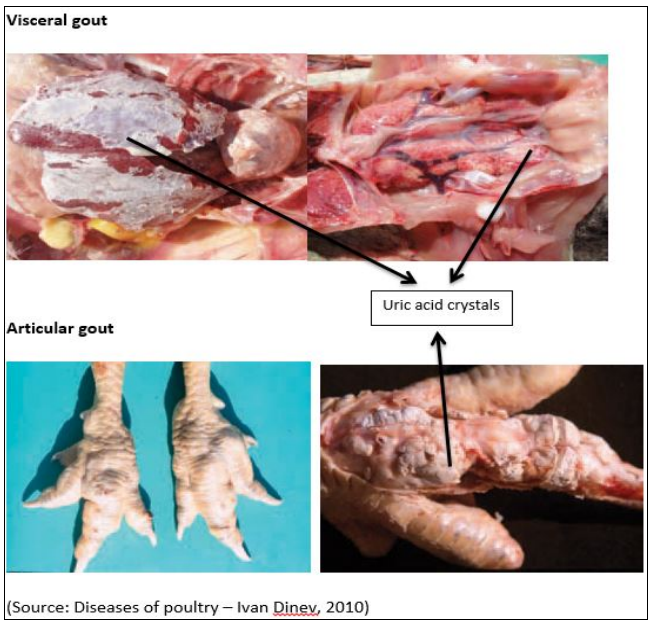

Fig 1: Precipitation of urates in the visceral organs.

Microscopic lesions: The clusters of urate crystals appear as pale, elongated, needle shaped structures in tissue sections. The crystals are surrounded by an inflammatory reaction of macrophages, Lymphocytes, fibroblasts and foreign body giant cells. Presence of urates or inflammatory reaction occur in renal tubules and ureter.

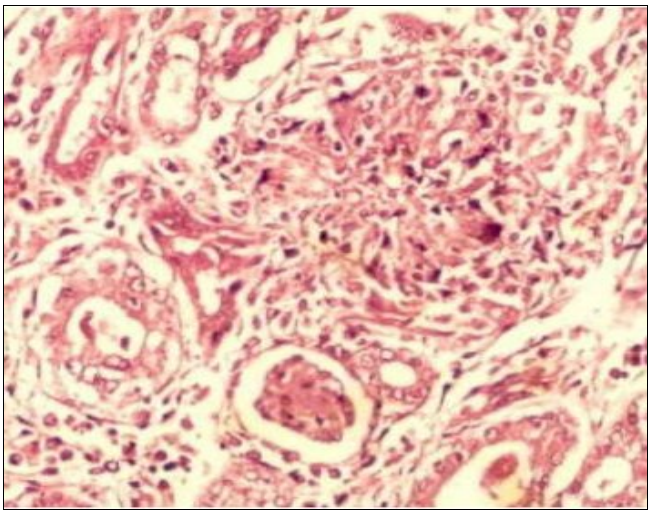

Fig 2: Kidneys: urates deposition

Diagnosis: Based on symptoms of dullness, dehydration, soiled vent with whitish pasty droppings, reluctant to move, swollen and painful joints are all indicative of gout in birds. Urate deposits are white and semisolid and must be differentiated from yellow fibrinous or purulent inflammatory exudates. Macroscopically, the aspirated material from articular gout looks like toothpaste. Microscopically, dark coloured acicular crystals can be seen in smears of tophi, when special staining is done using De Glantha stain. It helps to demonstrate urate crystals, which appears as sharp needle shaped tophi. A polarizing microscope is helpful in identification. Determination of uric acid concentration includes HPLC, biosensor methods, phosphotungistic acid methods and uricase methods [6].

Treatment of gout: Should be given plenty of water, feed contains recommended level of protein, use of urine acidifiers. Ensure adequate levels of vitamin supplements such as vitamin A, D, K, B complex and minerals. Use allopurinol 10-40 mg in drinking water, twice daily.

Prevention and control: Maintain the proper nutrition diet such as proper calcium and phosphorous ratio, vitamins and minerals. Required level of sodium chloride, conventional source of protein should be used.

References

1. Chaudhary K, Malhotra K, Sowers J, Aroor A. Uric acid - key ingredient in the recipe for cardio-renal metabolic syndrome. Cardio renal Med. 2013;3:208-220.
2. Dalbeth N, Gosling AL, Gaffo A, Abhishek A. Gout. Lancet. 2021;397:1843-1855.
3. Xi Y, Huang Y, Li Y Huang Y, Yan J, Shi Z. The effects of dietary protein and fiber levels on growth performance, gout occurrence, intestinal microbial communities, and immunoregulation in the gut-kidney axis of goslings., Poult. Sci. 2022;101:101780.
4. Lumeij JT. Gastroenterology. In: Avian Medicine: Principles and Application (eds BW Ritchie, GJ Harrison, LR Harrison); c1994. p. 482-521.
5. Mandal AK, Mount DB. The molecular physiology of uric acid homeostasis. Annu Rev Physiol. 2015;77:323-45.
6. Sandhyarani K, Madhuri D, Ravikumar Y. Review-gout in chicken. Adv. Anim. Vet. Sci. 2022;10(3):702-711.