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Pathology and management of gout in Giriraja breeder chicks

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Abstract
An outbreak of gout was recorded during October to November, 2015 in a flock of Giriraja breeder chicks maintained under intensive management system that resulted in mortality of 55 birds in a flock of 2000. Both visceral and articular gout were observed simultaneously. Grossly, dry platery patches of white chalky deposits were observed on the serosal surfaces of pericardium, liver, kidneys and ureters. Microscopically, kidneys showed varying degree of urate crystal deposition amidst an intense granulomatous inflammatory reaction. In addition, pericarditis and hepatitis were the other predominant lesions. The findings of positive murexide test, demonstration of needle shaped urate crystal under phase contrast microscopy and urate crystals by De Galantha staining method on tissue sections of affected organs were confirmatory of gout. Exclusion of concentrate feed and provision of additional drinkers, incorporation of low protein diet, jaggery mixed water, supplementation of A, D_3, K, B complex and electrolytes helped in control of gout and mortality of chicks.

Keywords: Gout, pathology, management, Giriraja chicks

Introduction
In the present days of high demand for meat and eggs, the poultry has been genetically engineered for higher productivity. Selection and rearing of broiler as well as layer birds is aimed with higher production. In the process to achieve the target, the birds are subjected to stress and health of the vital organs is compromised. This has resulted in increased incidence of metabolic and production related disorders. The kidney is a vital organ of the bird with diverse metabolic and excretory function viz. maintaining the chemical composition of body fluids, removal of metabolic waste and toxic products, regulation of blood pressure and blood volume and conservation of fluids and electrolytes.

In general, avian kidneys comprise 1 to 2.6% of body weight compared to an average of 0.5% of body weight in mammals [27, 17]. Kidney mass also is relatively larger in those birds with active salt glands. The kidneys are long, paired and located in depressions in the pelvis bone in the abdominal cavity. The kidneys are normally reddish-brown in color and have 3 distinct lobes or divisions [9, 17].

The primary function of the kidneys is to maintain the chemical composition of body fluids (blood). The kidney serves a variety of other functions in the body; removal of metabolic waste and toxic products, conserving fluids and vital electrolytes, regulation of blood volume, and production of hormones that regulate blood pressure and production of red blood cells. When renal function stops, uric acid, normally excreted by the kidney in the urine, are then deposited any place that blood is circulated. A bird with no renal function is likely die within 36 hours [9, 7, 8].

Avian urolithiasis, or known as gout, is resultant to kidney damage from any of a number of potential cause, namely infectious or nutritional disease, ingestion of toxins, or a combination of factors [22]. Both visceral (deposition of white chalk-like material on the surface of various abdominal organs as well as the pericardial sac) and articular (white chalk-like deposits in joints of the limbs) gouts have been reported in pheasants, Japanese quail, ducks, aviary birds and chickens. Seasonal variation, with higher incidence of gout in summer and winter than in autumn has also been reported in chicken [15, 40, 3, 30]. In recent past an increase in the incidence of gout has been reported in different geographical parts of India causing heavy mortality in chicks [30, 4, 36, 2].

The present paper describes an investigation into outbreak of gout and associated renal

changes in Giriraja breeder chicks of an organized poultry farm in Pondicherry, Southern part of India.

Materials and Methods

Morbidity followed by mortality associated with renal damage and gout was recorded in 55 of 2000 Giriraja breeder chicks of 3-4 weeks age during October to November, 2015, reared under intensive management system (deep litter) in an organised poultry farm in Pondicherry, India. The reported clinical signs were decreased feed and water intake, lethargy, poor weight gain, weight loss and abnormal droppings. The dead birds were subjected to detailed necropsy examination. Representative tissue samples from affected organs were collected in 10% neutral buffered formalin for histopathological studies. 4-5 µ thick sections were cut and stained with routine Haematoxylin and eosin technique. Duplicate sections were cut at 8 µ thickness and stained by De Galantha method for demonstration of urate crystals [18]. Amorphous material from hepatic, renal and joint surfaces was collected for confirmation of uric acid with murexide test. The collected solid samples were first treated with conc. nitric acid, which was slowly evaporated away; subsequent addition of ammonia solution (NH4OH) gave a purple color, due to formation of murexide and to confirm the presence of uric acid. The white chalky deposits on various visceral organs were also subjected to microscopic examination. Depending on the severity of lesions, kidneys were evaluated from mild to severe tissue changes.

Results

The reported ante-mortem clinical manifestation of affected chicks was dullness, dehydration, ruffled feathers, restlessness, difficulty in movement and standing on legs, painful joints and moist vent with whitish pasty droppings. At postmortem, all the carcasses were emaciated and dehydrated with moderate enlargement of hock and phalangeal joints. Grossly, dry platery patches of white chalky urate deposits were observed on the serosal surfaces of pericardium, liver, kidneys and ureters. Nephropathy, observed consistently, was characterized by unilateral to bilateral enlargement and moderate to severe congestion of kidneys, most prominently of the cranial lobe, which bulged out of the bony depression (Fig. 1). The kidney lobes of the contralateral side were atrophied, especially the caudal lobes. Occasionally, the cranial lobes of both the kidneys were enlarged. Ureters of either side were found to be distended with retained semifluid to semisolid chalky white urates, giving cord-like appearance to ureters (Fig. 2, 3, 4). The ureters were markedly enlarged due to blockages by uroliths and the entire obstructed kidney showed varying degree of degeneration.

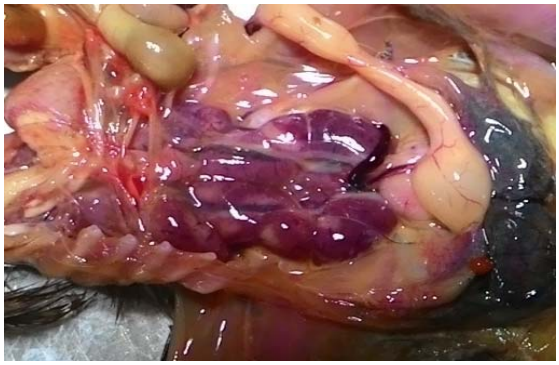

Fig 1: Enlarged and congested kidneys.

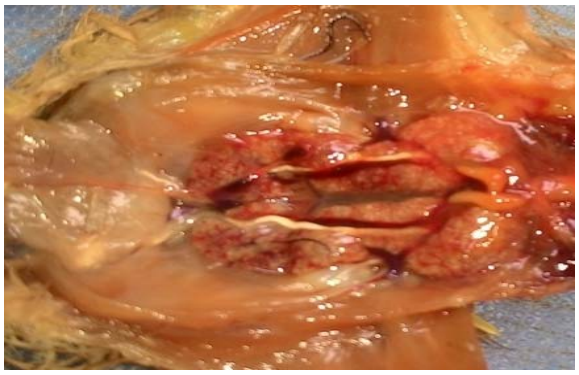

Fig 2: Discolored kidneys with dilated ureters filled with chalky white material.

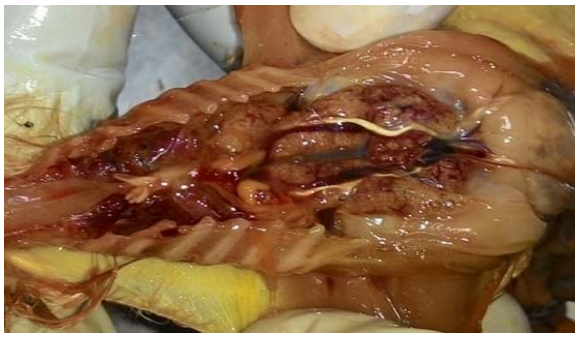

Fig 3: Enlarged, discolored and gouty kidneys with dilated ureters.

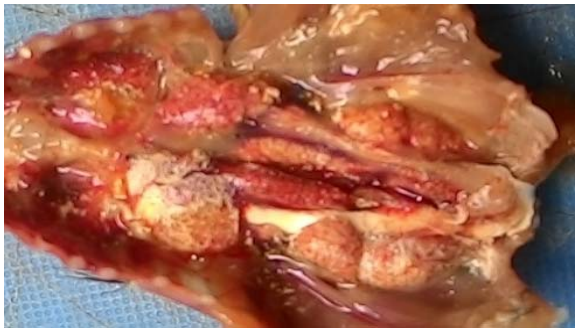

Fig 4: Diffuse chalky white deposits on Kidneys

Viscid thick masses of white urates were also observed in most of the joints (articular surfaces) and over the tendon sheath (Fig. 5). The hock joints were most consistently and severely affected (100% cases), the deposits were also observed on the articular surface of the bones. Lesions were most severe in kidneys followed by heart, liver and joints.

Fig 5: Chalky white urate deposition in the hock joint

The Murexide test (a colorimetric based technique) employed to identify the presence of urate in samples collected from

deposits on visceral organs from the carcasses revealed appearance of light purple color indicated the presence of urates. The white chalky deposits collected from the pericardium, liver, kidneys and ureters subjected to Phase Contrast Microscopy showed numerous needle shaped crystals arranged individually or as aggregation to form clumps, indicative of urate crystals (Fig. 6).

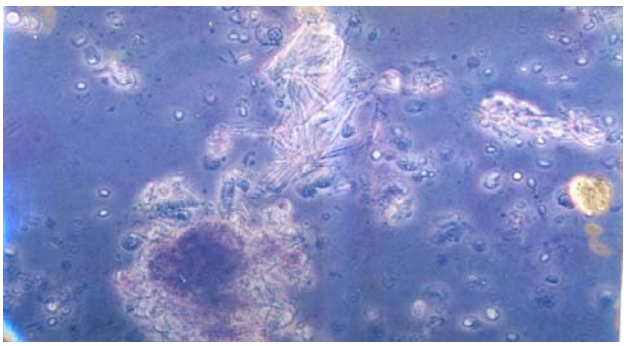

Fig 6: Phase Contrast Microscopy showing numerous needle shaped urate crystals.

Histopathological examination revealed varying degree of degenerative changes and inflammatory reaction in the pericardium, liver and kidneys. The renal parenchyma was atrophied and revealed degenerative and necrotic changes associated with haemorrhages involving glomeruli, cortical and medullary tubules, collecting ducts and medullary tracts. Glomerular changes included atrophy, distortion and segmentation. The tubules showed dilatation, degeneration and desquamation. Large deposits of radiating fine needle shaped crystals replacing parenchyma was surrounded by intense granulomatous inflammatory reaction (Fig 7, 8). Collecting ducts and medullary tracts also revealed presence of urate crystals and numerous heterophils. These crystals appeared black against yellow background on De Galantha staining confirming them to be urates (Fig. 9)

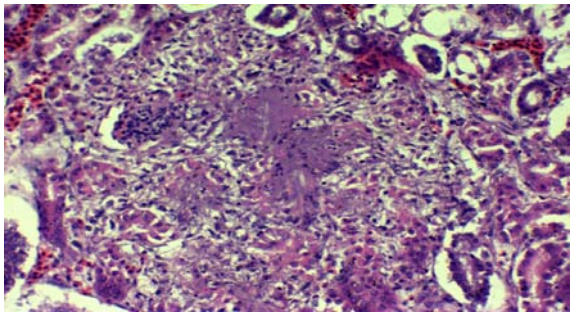

Fig 7: Kidney showing granulomatous reaction H & E x 400

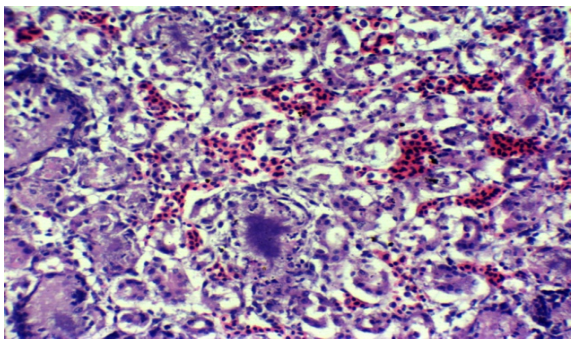

Fig 8: Kidney showing congestion and urate crystals H & E x 200

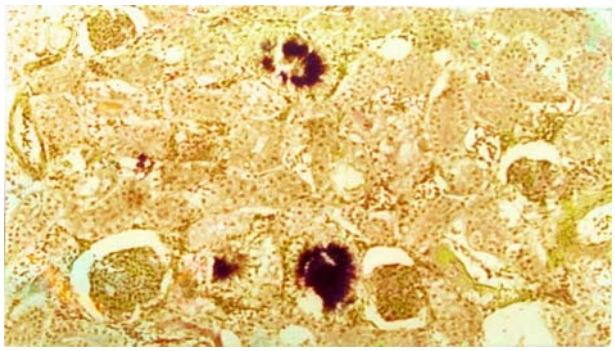

Fig 9: Kidney showing black urate crystals deposits De Galantha x 400

Discussion

Gout in poultry should not be considered as a disease entity but as a clinical manifestation of severe renal dysfunction that causes hyperuricemia and urolithiasis causing tubular damage thus resulting in renal failure. Mortality among young chicks is high in comparison to adult birds [38, 39, 19]. The cause of gout is often difficult to determine and is mostly multi-factorial. Higher levels of dietary proteins cause excess uric acid production and nephropathy while higher dietary levels of calcium and low levels of phosphorus lead to increased retention and decreased excretion of uric acid [20]. Dehydration due to water deprivation was also a common cause for visceral deposition of urates [23]. Infectious agent like infectious bronchitis virus also induces gout [1]. In addition, products used on a routine basis and can result into toxicity includes antibiotics, anticoccidials, minerals, vitamins, manufactured chemicals, and pesticides can also induce gout in poultry. There are several mycotoxins that are nephrotoxic and/or hepatotoxic in laying hens, including ochratoxin A, oosporein, and deoxynivalenol (DON). The kidney and liver damage caused by ingesting these mycotoxins can cause gout among other clinical signs [12, 4, 28]. Chicken is more prone to gout due to uricotelic mechanism for excretion of nitrogenous waste and lack the enzyme uricase in the system [40, 8, 35, 11]. In case of gout, the blood levels of uric acid can be as high as 44 mg/100ml as compared to 5-7mg/100ml in a normal bird. Gout occurs due to decrease in kidney function to the point where uric acid (a nitrogenous waste) accumulates in the blood and body fluids. The uric acid subsequently precipitates as calcium sodium urate crystals in a various locations, particularly in the kidneys and on the serous membranes of the liver, heart, air sacs, and joints. Uric acid is not toxic but precipitated crystals can cause mechanical damage to tissues *viz.* kidneys, heart, lungs, intestines and also in various joints. The damaged kidneys appear atrophied or missing portions of kidney lobes, whitish gritty material in kidneys and enlarged ureters. Compensatory enlargement of remaining part of normal kidney can be observed in an attempt to maintain adequate renal function [29, 10, 13, 3]. Obstruction of ureters and its branches might be responsible for pressure atrophy of the kidney upstream from the blockage and loss of renal parenchyma in the present study [38]. A sudden and complete obstruction of one ureter leads to rapid atrophy of the corresponding kidney and then reduces size to almost vanishing point [13]. The organs affected in the present study were serosal surfaces of pericardium, liver, kidneys and ureters. The precipitation of the urates on surface of different visceral organs might be attributed to the kidney damage leading to hyperuricemia [29].

Although the hock joints were found to be severely affected in the present study, but other sites, such as the joints of the wings and vertebrae can also be affected [10, 6, 16]. Concurrent involvement of viscera and joints was a consistent feature in the present investigation corresponded with the findings of earlier report [20]. This is contrary to the observations of Sonmez (1992) [32], who reported that not all the organs were affected in all the cases and joints were involved only in 5-8.2% cases.

Although gout has been recognized as a cause of mortality in chicken, it continues to be a diagnostic and therapeutic challenge. Clinical manifestations and postmortem findings are generally sufficient to diagnose gout in poultry under field conditions. Murexide test on the deposits recovered from dead birds, confirms urate deposits. This test can be used to confirm the presence of urates in deposits on the surfaces of visceral organs [32, 25].

Blood uric acid estimation of the affected flock is also confirmatory. However, in the present study uric acid levels were not estimated due to paucity of resources in the laboratory. Additionally, paraffin embedded sections from the kidneys can be stained with De Galantha staining method to demonstrate and visualize the urate crystals [14, 34, 16, 12]. The gross and histopathological changes were suggestive of concurrent visceral and articular gout, which was further confirmed by positive murexide test, visualization of needle shaped urate crystal under phase contrast microscopy and demonstration of urate crystals by De Galantha staining method on tissue sections of affected organs.

In the present study, a significant reduction in mortality of chicks was observed on advice of the following measures:

1. Provision of plenty of water and additional drinkers in poultry shed.
2. Change of feed to low protein diet from 22% CP to 20% CP for 7 days.
3. Provision of jaggery mixed water @ 5g/litre for 7 days.
4. External supplementation of A, D3, K and B complex vitamins through water.
5. Supplementation of electrolytes through water.

The investigation concluded that the Giriraja chicken is prone to gout, management practices and nutritional requirements of bird's *viz-a-viz* water, protein and vitamin requirement for optimum health need to be studied during the young age. Gout, when recognized as a problem in field cases, is likely to be a multi-factorial problem and identifying a specific cause is often difficult. Another reason for diagnostic difficulty is that kidney damage occurring during the growing period often has no apparent signs until the birds later come into egg production and are fed high calcium diets. Field cases and research have demonstrated the importance of interactions between 2 or more contributing factors, especially nutrition and management in the control and prevention of gout.

Conclusion

The presence of gout associated mortality in poultry indicates some form of kidney damage in the early stage of maturity. There exists a strong association of infectious and calcium-phosphorus dietary imbalance in the causation of gout related mortality among the birds of different age groups. In addition, other factors such as electrolyte imbalance, mycotoxins, seasonal variation and water deprivation need to be recognized as possible contributory reasons for mortality due to gout. The study concluded that gout, when recognized as a problem in field cases, is likely to be a multi-factorial problem and identifying a specific cause is often difficult. Field case study and research have demonstrated the importance of interactions between 2 or more contributing factors, especially nutrition and management in the control and prevention of gout.

Conflict of Interest

All authors declare no conflicts of interest. All authors participated and approved the manuscript for publication.

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