

Original Research Article

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Pathology of Gout in Commercial Broiler Chicken

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ABSTRACT

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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. The present study reports pathomorphology of visceral Gout in commercial broiler chicken from Puducherry and Tamil Nadu region in India. A total of 120 kidney samples were collected for gross and histopathological examination. 24 (20%) samples showed gross changes of visceral gout. Microscopically, gout was recorded in 28 (23.33%) cases. The renal lesions characterized by varying degree of tubular degeneration and necrosis. In addition, radiating fine needle shaped crystals were noticed in the lumen and interstitium of renal parenchyma. De Galantha staining method on tissue sections of affected organs confirmed the cases as gout.

Introduction

Renal diseases have been shown to be fairly common in avian species. In poultry, as much as 29.6% of all disease conditions had abnormal pathology associated with or attributable to renal disorders (Siller, 1981). The cause for the development of kidney lesions is usually multi factorial and may be attributed to nutritional deficiencies, managemental problems, toxins and infectious agents. In addition, the urinary system of poultry is so unique that the blood can be directed from the lower limbs straight into the renal parenchyma. This may increase the

effect of nephrotoxic drugs and/or enhance elimination by taking the compound directly to the kidneys (<http://avianmedicine.net/content/uploads/kidneys.pdf>).

In poultry, renal diseases may lead to gout, which can further damage the kidneys or additional body systems. Gout is more a sign of renal damage than a distinct disease. Several outbreaks of gout were reported in commercial broilers in India during 2011 and 2012, causing up to 40% mortality in the birds (Bulbule *et al.*, 2013). It is a metabolic disorder resulting in hyperuricemia and the deposition of uric acid or urates in tissues. The

nutritional or metabolic causes of gout include 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 kidney damage. 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 (Bulbule *et al.*, 2014). The role of mycotoxins and phytotoxins in the causation of kidney damage and gout in poultry is well documented. Primary infectious nephritis due to bacterial agents does not appear to be common in poultry.

The present paper describes the occurrence and pathology of gout and associated renal changes in commercial broiler chicken.

Materials and Methods

The study was carried out on broiler kidney samples collected from various farms located at Udumalpet, Coimbatore, Salem and Puducherry, Southern part of India. The age of the birds varied from day-old to six weeks. A total of 240 kidney samples (both left and right) from 120 broiler birds were collected for examination. Kidney samples with lesions were included for the study.

Representative tissue samples fixed in 10% NBF and in absolute alcohol were processed by paraffin embedding technique for microtomy and 4-5 µm thick sections were prepared and stained by routine Haematoxylin and Eosin (H&E) for detailed histopathological studies as described by Luna (1968). Parallel sections from tissue samples fixed in absolute alcohol were also stained by DeGalantha method for gout.

Results and Discussion

Renal diseases have been shown to be fairly common in avian species. Pathological lesions in the kidneys are marked but often overlooked in routine post-mortem examination in spite of the fact that they can greatly help in reaching a conclusive diagnosis. Hence, the present study was aimed to investigate the occurrence and pathological characterization of gout in broiler chickens from slaughtered and necropsied cases.

Gout is a metabolic disorder resulting in hyperuricemia and the deposition of uric acid or urates in tissues. Grossly, gout was recorded in 24/120 (20%) cases and it was characterized by swollen pale kidneys with urate deposits and dilated ureters with chalky white material (Fig. 1). The other noticeable findings observed was presence of chalky white masses surrounding the heart, kidneys, liver and other organs (Fig. 2). Microscopically, gout was recorded in 28/120 (23.33%) cases on both the kidneys. The kidneys showed severe lesions characterized by moderate to severe tubular dilation, necrosis and large deposits of radiating fine needle shaped crystals in the tubular lumen (Fig. 3) and in the interstitium and also in myocardium (Fig. 4). These deposits were surrounded by a zone of granulomatous inflammation (Fig. 5). The crystals appeared black against a yellow background on DeGalantha staining confirming theme to be of urates and the cases of gout (Fig. 6). The gross and histopathological features observed in the present study concur with the observations of earlier workers (Ansar *et al.*, 2004; Feizi *et al.*, 2011; Auda, 2013).

Gout is multifactorial in origin. It is rather the result of kidney damage from any of a number of potential causes, which can be infectious, nutritional, toxic, poor management or possibly a combination of factors.

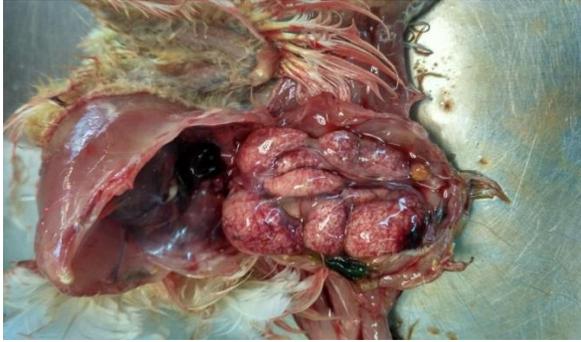


Fig.1 Swollen kidneys with chalky white deposits



Fig.2 Chalky white deposits noticed in the pericardial sac and surface of liver.

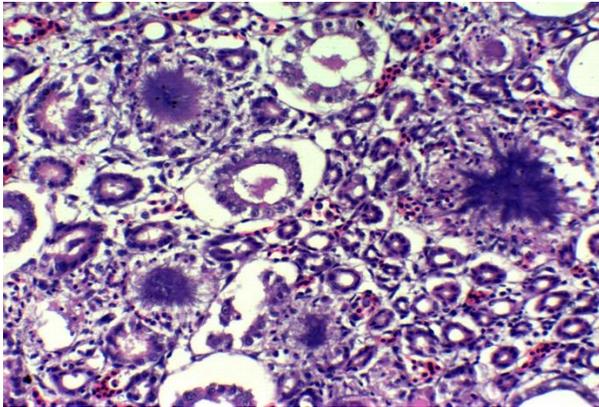


Fig.3 Kidney-Presence of fine needle shaped urate crystals in tubules. H&E x400

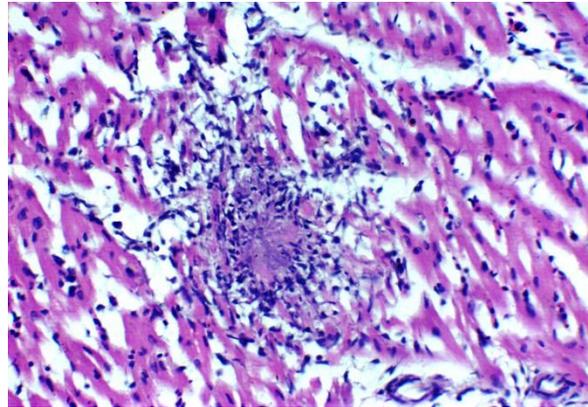


Fig.4 Heart- Presence of fine needle shaped urate crystals. H&E x400

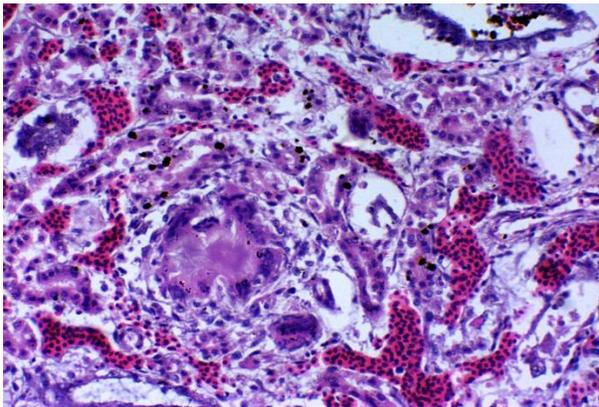


Fig.5 Kidney-Urate granuloma surrounded by zone of hyperemia. H&E x400

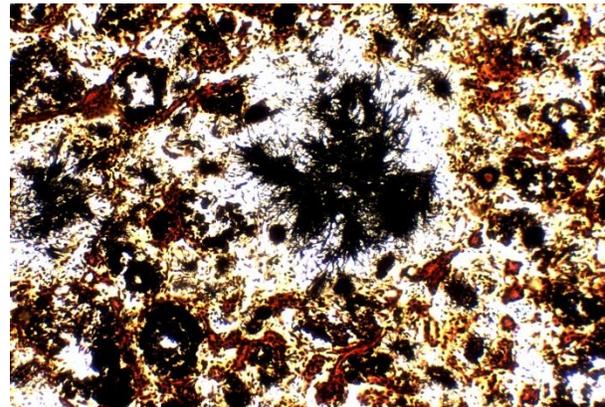


Fig.6 Kidney-Fine needle shaped black urate deposits. De Galantha x200

The nutritional or metabolic causes of gout include excess of dietary calcium (Feizi *et al.*, 2011), high protein diet (Singh and Ghosh, 2014), excess salt (Sodhi *et al.*, 2008), low phosphorus, imbalance between Ca-P levels (Ansar *et al.*, 2004), adulteration of feed with urea and vitamin A and D deficiency. Water deprivation followed by over dosages of certain drugs like sulphas and the antibiotic aminoglycosides (Gentamicin) often causes gout (Javed *et al.*, 2013). In addition, it is mentioned that managemental stress factors including high brooding temperature and higher level of ammonia concentration in the shed cause high mortality due to gout (Bulbule *et al.*, 2014). The authors have further said that primary infectious nephritis due to bacterial agents does not appear to be common in poultry. *E. coli* and *Salmonella* have occasionally been isolated from cases of pyelonephritis, but the infection is likely secondary. The role of mycotoxins in the causation of gout is well documented and has been discussed under tubular changes.

In addition to the above mentioned etiological agents, the specific viral diseases often reported to be associated with kidney damage are Infectious bronchitis virus, avian nephritis virus and disease caused by chicken astro virus. According to Bulbule *et al.*, (2013), several outbreaks of gout were reported in commercial broilers in India during 2011 and 2012, causing up to 40% mortality in the birds. According to the authors, the managemental and nutritional causes were not involved in the causation of gout. The infectious agents such as chicken astrovirus, avian nephritis virus and infectious bronchitis virus were detected in gout cases. In the present study, ANV was detected in two gout cases. Similarly, Eswaran *et al.*, (2014) from Tamil Nadu and Ghodasara *et al.*, (2015) also detected ANV in clinical cases of gout from Raipur, Chhattisgarh.

In the present study, the occurrence of gout was more in birds less than 3 weeks of age than 3-6 weeks of age. Similar observations were also made by Singh *et al.*, (2013). In addition the authors have recorded more cases of gout during colder months

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