

Pathomorphological Study of Visceral Gout in Desi Fowl

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ABSTRACT

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Ten birds from local poultry farm were brought to Division of Veterinary Pathology, SKUAST-J for necropsy examination. Postmortem examination revealed a white chalky material deposit on pericardial sac, liver, spleen and kidneys. Moreover enlargement of kidneys with necrotic foci was also evident, along with swelling of ureter in some birds. Tissue samples were collected in absolute alcohol from affected organs for histopathological examination and it was diagnosed as visceral gout from gross findings and presence of bluish coloured crystal on histopathological examination.

Introduction

Gout is a common metabolic disorder that results in abnormal accumulation of urates in domestic birds¹. It occurs as two distinct forms, namely, visceral and articular gout (Amaravathi *et al.*, 2015; Feizi *et al.*, 2012; Feizi *et al.*, 2011). Visceral gout has been reported in various caged and aviary birds from different parts of the world. It is among the most commonly diagnosed causes of mortality in poultry with symptoms include anorexia and emaciation (Riddell, 1987). Gout is mostly associated with kidney damage in birds.

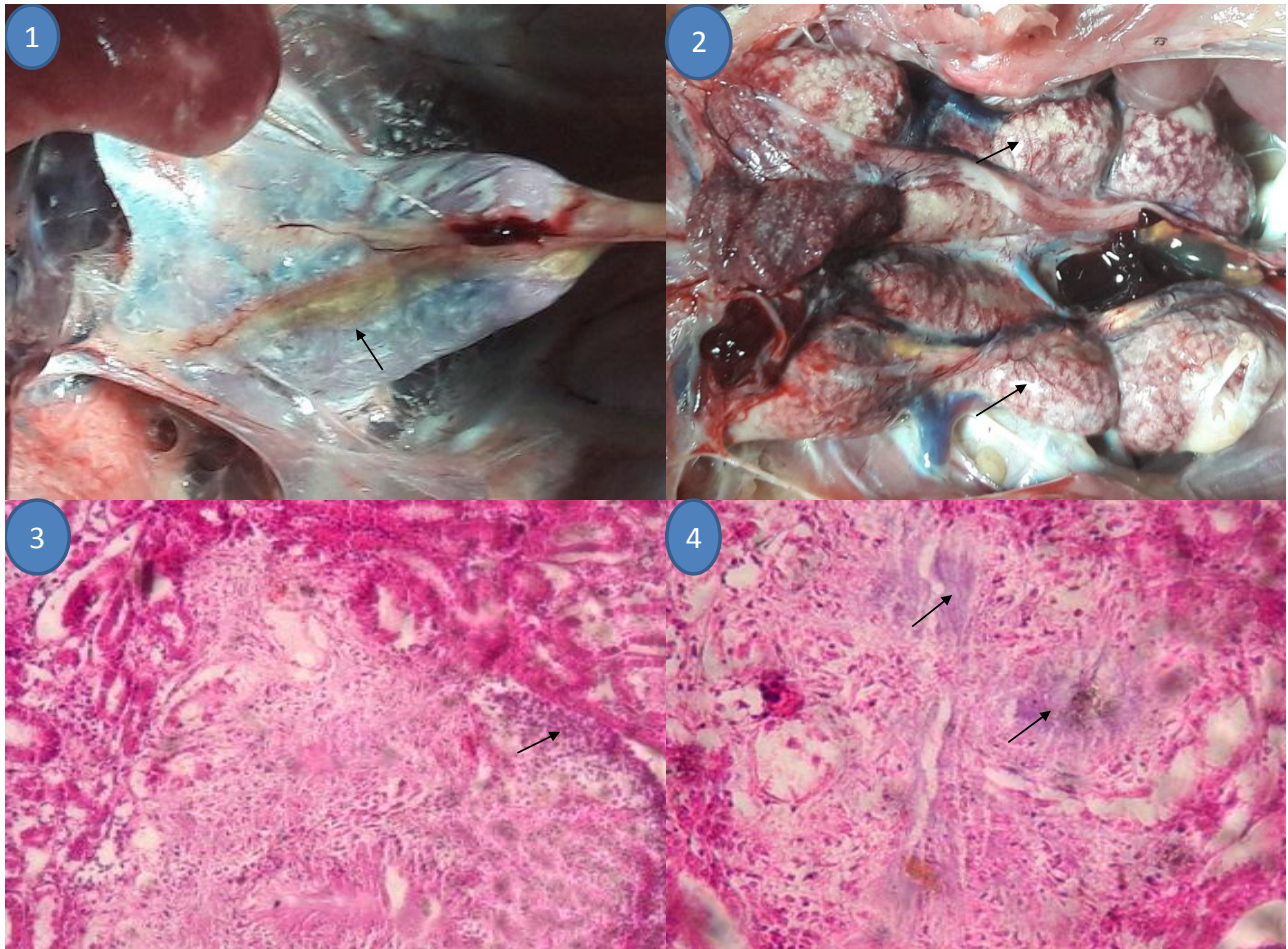
The causes of gout are many, as kidney damage occurs due to multi-etiological factors which can be broadly categorised as nutritional, metabolic, infectious and other causes like mycotoxins. Higher levels of dietary proteins cause excess uric acid production (Li *et al.*, 1998). Dietary protein plays an important role in the development and progression of kidney injury. An increase in dietary protein can cause an increase in kidney size and glomerular filtrate rate (GFR), with subsequent glomerular injury, accumulation of mesangial deposits, and

eventually glomerulosclerosis (Khan *et al.*, 2001). Birds are uricotelic and lack uricase; the enzyme which converts uric acid to less harmful substances. Uric acid itself is not toxic but precipitated crystals can cause severe mechanical damage to tissues like kidneys, heart, lungs, intestine (visceral gout) and also in the joints (articular gout). Visceral and articular gout have been reported in pheasants, Japanese quail, ducks, aviary birds and chickens (Fitz-Coy *et al.*, 1988; Nayak *et al.*, 1988; Rao *et al.*, 1993). The present paper presents a rare case of visceral gout in hill fowl.

Materials and Methods

Ten birds from local poultry farm were brought to Department of Veterinary Pathology, SKUAST-J for postmortem examination. After post mortem examination, tissue samples were collected and fixed in absolute alcohol from affected organs prior to processing. After overnight washing in running water and dehydration in ascending grades of alcohol, the tissues were embedded in paraffin and 5 μ thick sections were cut and stained with haematoxylin and eosin (H&E) (Luna, 1968).

Fig.1 Showing deposition of urate crystals on the pericardium. **Fig.2** Showing urate crystals deposited in kidney tubules. **Fig.3** Showing infiltration of inflammatory cells in kidney parenchyma and destruction of renal tubules. **Fig.4** Showing presence of bluish coloured urate deposits (tophi) in kidney parenchyma



Results and Discussion

On gross examination, white chalky material on pericardial sac (Fig 1), liver, spleen and enlargement of kidneys with necrotic foci (Fig 2) were found. Swelling of ureter was also found in some birds. Histopathologically, severe damage of tubules characterized by moderate to severe tubular dilation and necrosis and central bluish gouty depositions surrounded by radiations of needle like urate crystals followed by granulomatous reaction of lymphocytes, macrophages and fibroblasts with haemorrhages was noticed. The collecting renal tubules, urinephric ducts and ureters were also filled by gouty deposits with epithelial hyperplastic changes and chronic inflammatory cellular reaction in their wall.

The interstitial tissue showed edema, congestion, hemorrhage and lymphomononuclear cell infiltrations. Glomerular changes included thickening of Bowman's capsule and proliferative glomerulonephritis. These lesions were in consistence with the earlier study,⁸ who reported white chalky masses in the liver, kidney, visceral organs and joints. Histological changes of kidney comprised of cell infiltration, destruction of both glomeruli and tubules with urate crystal deposits. The remainder of the kidney parenchyma was degenerative, necrotic and haemorrhagic. Microscopic changes of gout induced in laying pullets by sodium bicarbonate toxicity which revealed significant urate deposits associated with tubular necrosis (Mubarak and Sharkawy 1999). In addition to kidney, other visceral organs are commonly affected by gout. Outbreak of gout attributed to disproportionate Ca: P ratio (3.5: 1) and 16.39% crude protein has been reported (Mir *et al.*, 2005). Grossly, dry platery patches of white chalky urate deposits were observed on the breast muscles and on serosal surfaces of

pericardium, peritoneum, mesentery, proventriculus, gizzard, testes, ovaries, over the abdomen and chest wall.

Needle shaped urate crystals (arrows) were also found in liver, spleen and heart on histopathological examination (Fig 3, 4).

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