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PATHOLOGY OF GOUT IN GROWING LAYERS ATTRIBUTED TO HIGH CALCIUM AND PROTEIN DIET

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Abstract

An outbreak of gout was investigated in a growing layers farm of 45000 birds located in Tripoli, Libya. Mortality was commenced at 39 days-old reaching to a total of 489 birds within 10 days. Post mortem was conducted and kidney tissue samples were fixed in 10% neutral formalin prior to processing. Feed samples were sent to the Animal Nutrition Department for analysis which revealed 23.47% crude protein and 3.5% calcium. Gross lesions comprised of deposition of chalky white material covering the pericardium and enlarged kidneys with necrotic foci, hemorrhage and nephrosis. Most of birds showed swollen ureter of one or both sides. Histopathologically, severe damage of tubules characterized by moderate to severe tubular dilation and necrosis and central bluish gouty deposition surrounded by radiations of needle like urate crystals followed by granulomatous reaction of lymphocytes, macrophages and fibroblasts. 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 walls. The interstitial tissue showed edema, congestion, hemorrhage and lymphomononuclear cell infiltration. This gout outbreak in growing layers is attributed to high concentration of crude protein and calcium in diet.

Keywords: **Gout, Layers, Calcium, Protein**

Introduction

Gout is one of the important disorders associated with kidney damage in birds. The causes of gout are many as kidney damage occurs due to multi-etiological factors which can broadly categorized as nutritional and metabolic causes, infectious causes 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 and Alden 2001). Birds are uricotelic and lack uricase; the enzyme which converts uric acid to less harmful substances. Impairment of kidney function results in excessive accumulation of uric acid in tissues. 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 gouts 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). Experimentally, the combined effects of high dietary calcium and protein concentrations on the induction of visceral gout in growing birds of a layer strain was

investigated (Guo *et al*., 2005) and a typical visceral gout was induced by the high calcium and protein diet.

The present paper describes pathological investigation into an outbreak of gout in growing layer chickens.

Materials and Methods

Mortality was recorded in growing layers farm of 45000 birds located in Tripoli, Libya. Mortality associated with gout was commenced at 39 days-old reaching to a total of 489 birds within 10 days. Dead chickens were brought to the Department of Poultry and Fish Diseases, Faculty of Veterinary Medicine, Tripoli, Libya for diagnosis. Post mortem was conducted and tissue pieces from affected kidneys were fixed in 10% neutral formalin 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) as per the method of Luna (1968). Feed samples were sent to the Animal Nutrition Department, Faculty of Veterinary Medicine, Tripoli, Libya for analysis.

Results and Discussion

The total mortality of approximately 1.1% (489 out of 45000) within 10 days observed in present outbreak was almost similar to the general incidence of 2-15% in layers associated with gout (Sonmez 1992). Mortality of 18.77% was reported in Kashmir favorella poultry over a period of 6 months which was attributed to 16.39% crude protein in poultry feed (Mir *et al*., 2005). In another report, 181 of 300 broilers died over a 29 day period due to high protein diet (Nayak *et al*., 1988).

In present study, the gross and histopathological changes were suggestive of visceral gout due to high protein feed which was confirmed by 23.47% crude protein in feed and high calcium 3.5% (Table 1). Gross lesions comprised of deposition of chalky white materials on pericardial sac and enlargement of kidneys with necrotic foci and hemorrhage (Figure 1). Most of birds showed swelling of ureter of one or both sides (Figure 2). Histopathologically, severe damage of tubules characterized by moderate to severe tubular dilation and necrosis and central bluish gouty depositions (Figure 3) surrounded by radiations of needle like urate crystals (Figure 4) followed by granulomatous reaction of lymphocytes, macrophages and fibroblasts with haemorrhages (Figure 5). 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 walls (Figure 6). The interstitial tissue showed edema, congestion, hemorrhage and lymphomononuclear cell infiltration (Figure 7). Glomerular changes included thickening of Bowman's capsule and proliferative glomerulonephritis (Figure 8).

These lesions were in consistence with the earlier study by Nayak *et al*. (1988) 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. Mubaraka and Sharkawy (1999) described microscopic changes of gout induced in laying pullets by sodium bicarbonate toxicity which revealed significant urate deposits associated with tubular necrosis.

In addition to kidney, other visceral organs are commonly affected by gout. Mir *et al*. (2005) reported an outbreak of gout attributed to disproportionate Ca : P ratio (3.5 : 1) and 16.39% crude protein. Grossly, dry platery patches of white chalky urate deposits were observed on the breast muscles, neck and on serosal surfaces of pericardium, peritoneum, mesentery, proventriculus, gizzard, testes, ovaries, over the abdomen and chest wall. The

urates were observed on kidneys, in the pericardial sac and over the pericardium, on liver, spleen, lungs and air sacs. Nephropathy, observed consistently, was characterized by unilateral enlargement of kidneys, most prominently of the cranial lobe, which bulged out of the bony depression. 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. Histopathological examination revealed urate deposits in the kidneys, liver, spleen, lungs and joints resulting in severe damage. Kidney 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 degeneration and desquamation. Uric acid deposits replacing parenchyma was surrounded by inflammatory cells including heterophils initially, followed by lymphocytes, macrophages and giant cells along with fibroplasias. Collecting ducts and medullary tracts also revealed presence of urate crystals and numerous heterophils.

In a recent study, Al-Hamadani and Mohammed (2009) reported lesions of kidney in layers that may be caused by multiple agents. These lesions were variable from visceral gout that characterized grossly by precipitation of urate salts to enlargement of kidney with congestion and hemorrhage or paleness. Histological examination of kidney showed swelling and degeneration of epithelial cell of renal tubules (Nephrosis) that caused stenosis of blood vessels and infiltration of mononuclear inflammatory cells, in addition to desquamation of epithelial cell of renal tubules with dilation of some renal tubules and hemorrhage in the interstitial tissue of kidney. Other cases showed necrosis of renal tubules epithelium, necrosis of glomerular renal tuft and dilation of bowman's space. There were precipitation of urate salts in the lumen of renal tubules and these cases observed mostly in peak of production.

Although most of poultry feed producers are following the requirements suggested by NRC (1994), these requirements have been considered low nowadays due to continued increase in growth rate of birds and the demand for increasing the daily recommended intake of vitamin requirements for the consumers (Lesson 2007). But re-evaluation of dietary protein and vitamin specifications has to be worked out taking into consideration the difference in requirements between poultry breeds.

It is concluded that growing layers are susceptible to high protein and calcium diets. The study stressed the need to evaluate the concentrations of all feed components in poultry feed.

Table 1. Result of feed sample analyses*

Fiber %	Fat %	Protein %	Calcium %	Ash %	Moisture %
4.43	2.82	23.47	3.5	6.58	10.23

*Average of different feed samples taken from the same feed source of the affected farm.

Figure legend

Fig. 1. Deposition of chalky white materials on pericardial sac (arrow) and enlargement of kidneys with necrotic foci (arrow)

Fig. 2. Swelling of ureter of one side (arrow) and enlargement of kidneys

Fig. 3. Section of kidney showing severe damage of tubules characterized by moderate to severe tubular dilation and necrosis and central bluish gouty depositions (arrows). H&E ×40

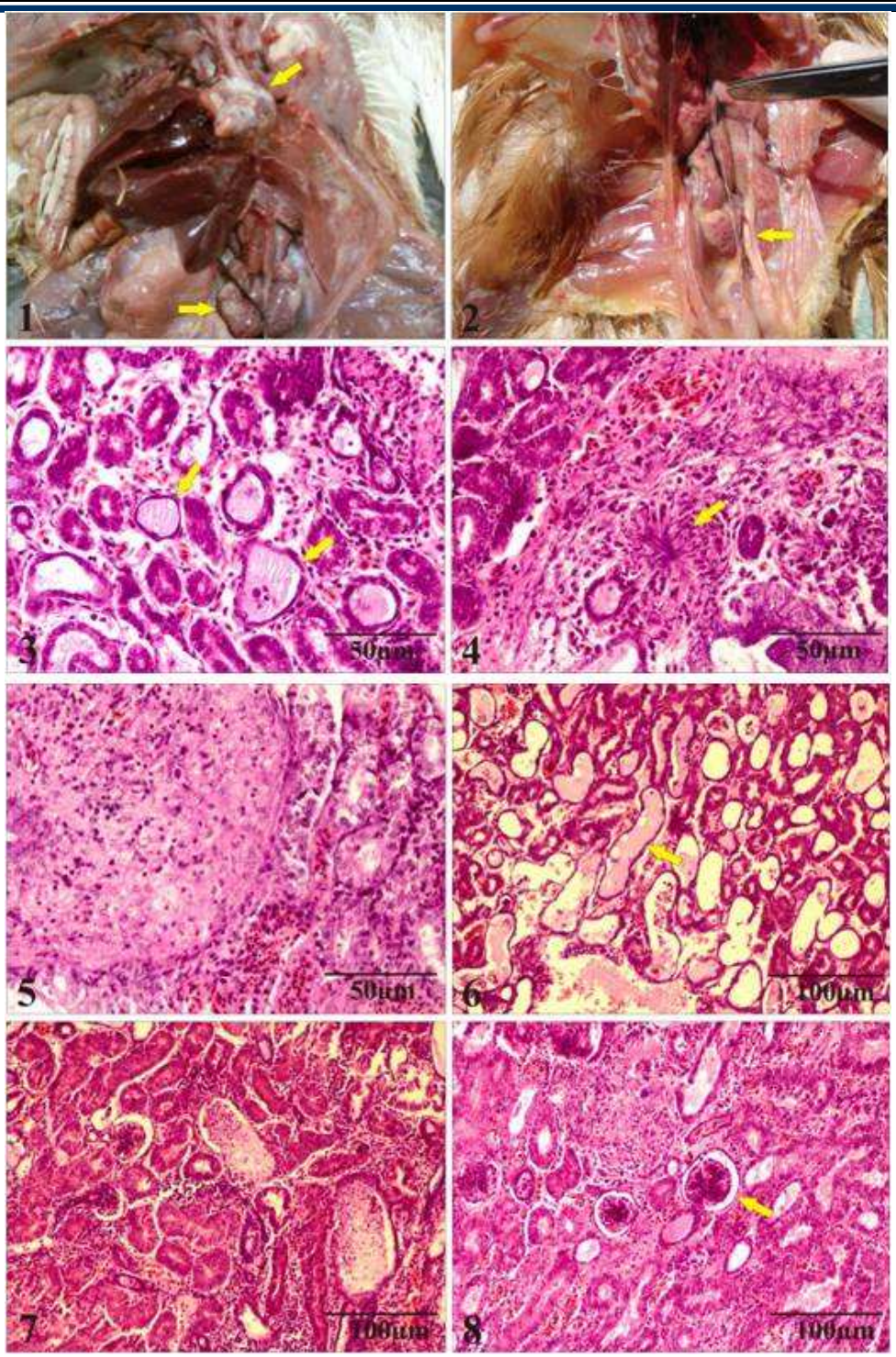
Fig. 4. Section of kidney showing interstitial radiations of needle like urate crystals (arrow). H&E ×40

Fig. 5. Section of kidney showing granulomatous reaction of lymphocytes, macrophages and fibroblasts with haemorrhages. H&E ×40

Fig. 6. Section of kidney showing accumulation of gouty deposits in the collecting renal tubules, urinephric ducts and ureters with epithelial hyperplastic changes and chronic inflammatory cellular reaction their walls (arrow). H&E ×20.

Fig. 7. Section of kidney showing edema, congestion, hemorrhage and lymphomononuclear cell infiltration in the interstitial tissue. H&E ×20

Fig. 8. Section of kidney showing thickening of Bowman's capsule and proliferative glomerulonephritis (arrow). H&E ×20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