NUTRITIONAL AND METABOLIC DISEASES ASSOCIATED WITH 
HEPATO - RENAL PATHOLOGY IN CHICKEN

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ABSTRACT

Hepato-renal pathology associated with nutritional and metabolic diseases in chickens was studied using samples brought for autopsy to the Centre of Excellence in Pathology. Among these, fatty liver syndrome was observed in 41 chickens. The liver showed moderate to severe enlargement with yellowish to yellowish brown discoloration and hepatocytes were distended with fat vacuoles. Gout was observed in 18 chickens showing deposition of white chalky crystals on the visceral organs and joints. Microscopically the liver and kidneys showed urate deposition. Ascites syndrome was observed in 13 chickens with accumulation of clear yellowish fluid in the abdominal cavity. Histology of liver revealed multifocal random areas of necrosis and disorganization of hepatic cords. Kidney showed intertubular hemorrhages and degeneration.

Key words: Hepato-renal pathology, Nutritional diseases, Metabolic diseases, Chickens.

INTRODUCTION

Nutritional and metabolic diseases are of great importance in poultry due to decline in egg production and hatchability. Nutritional and metabolic diseases are seen associated with the deficiency of some of the nutrients or failure in one of the body hormone or enzyme system or the failure or reduced activity of some metabolic function. Rahman and Samad (2004) observed that among 31.64% birds affected with different systemic and miscellaneous disorders, nutritional and metabolic diseases accounts for about 2.4% in a one year study conducted at BRAC Poultry Diagnostic Centre, Gazipur. The present study was conducted to understand the gross and histopathological changes occur in the liver and kidney of chicken during various nutritional and metabolic diseases.

MATERIALS AND METHODS

A total of 200 lesions bearing liver and kidney samples were collected from chicken brought for autopsy to the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Thrissur, Kerala during 2008-2010. For histopathological study, tissue pieces were collected in 10 % neutral buffered formalin and these were processed and embedded in paraffin. Sections were cut at 4-5 micron thickness and stained with routine Haematoxylin and Eosin stain.

RESULTS AND DISCUSSION

Fatty liver syndrome that includes fatty liver hemorrhagic syndrome (FLHS) and fatty liver kidney syndrome (FLKS) was observed in 34 and 7 chickens, respectively. The liver of chickens suffering from FLHS was seen with moderate to severe enlargement and yellow to yellowish brown discoloration. Blood clots were present on the surface of the liver and heart.

The changes observed in the liver of FLKS affected chickens were almost similar to those of FLHS except blood clots on the surface of the liver and heart of the later group. The kidneys were slightly enlarged with pale or yellowish discoloration and fragile in consistency. The hepatocytes were distended with fat vacuoles and normal architecture of the liver was disturbed by distorted and disrupted hepatic cords containing large lipid droplets (Fig.1). Focal or diffuse areas of hemorrhages were also observed. The tubular epithelium of the kidneys showed mild degenerative changes in cases of
FLHS. The lining of the epithelial cells of tubules were distended with fat globules displacing the nucleus to one side and the glomerulus also contained fat vacuoles in cases of FLKS. These observations were in accordance with Lonkar and Prasad (1988), Jordan and Pattison, (1996), Karadas et al. (1999) and Sathyanarayan (2007).

Excessive energy in diet induces FLHS regardless of the source. Excessive consumption of high energy diets in birds having restricted movement in cages is considered to result in a positive energy balance and excessive fat deposition. Excess fat may disrupt the architecture of the liver resulting in the weakening of the reticular framework and blood vessels in the liver. A pathogenic relationship between hepatic steatosis and hemorrhage has been suggested (Crespo and Shivaprasad, 2008). FLKS is a biotin deficiency related metabolic disease in chicken resulting in impaired hepatic gluconeogenesis and increased fat deposition. The problem is caused by low activity of biotin dependent enzyme-pyruvate carboxylase. Birds die from hypoglycaemia and the clinical signs and death are imputed to hypoglycaemia (Julian, 2005).

Gout was observed in 18 chickens. Deposition of white chalky urate crystals on the surface of the liver, kidney, other visceral organs and joints were observed. Kidneys were also found to be enlarged with white chalky crystals. Ureters were found to be distended with urates. Microscopically liver showed urate deposition, focal hepatitis, areas of focal hepatic necrosis, dissociation of hepatic cords and fatty vacuolation of few hepatocytes. In the kidneys, uric acid deposits were found surrounded by inflammatory cells. The kidney parenchyma was atrophied and degenerative, and necrotic changes were observed associated with intertubular hemorrhages. Varying degrees of fibrosis were also evident in the kidney parenchyma. Masson’s trichrome staining was done to demonstrate fibrous tissue proliferation, where in collagen fibres took blue color. (Fig.2) These gross and histological findings agreed with observations of Coy et al (1988), Ahmed et al. (2003), Mir et al (2005) and Crespo and Shivaprasad (2008). In liver and kidney sections stained with De Galantha’s, urates stained characteristically black, showing cotton ball appearance with radiating crystals at the periphery (Fig. 3), which was in agreement with observations of Mir et al. (2005).

Gout should not be considered as a disease entity but as a clinical sign of severe renal dysfunction that causes hyperuricemia. There are many causes for gout. 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. (Mir et al. 2005). Dehydration due to water deprivation was also a common cause for visceral deposition of urates (Onderka et al. 1987). Infectious agent like infectious bronchitis virus also induces gout (Ahmed et al. 2003).

Ascites syndrome was observed in 13 chickens. The affected chickens were seen with marked abdominal distension and accumulation of
FIG. 4: Ascites syndrome: accumulation of clear yellowish fluid with fibrin clots in the abdominal cavity and pale areas on the surface of the liver.

Ascites syndrome is a metabolic disease of multifactorial etiology causing significant mortality in chickens worldwide. Several factors including atmospheric hypoxia, housing environment, respiratory diseases, rapid growth rates, high-energy rations, toxins, nutritional aspects and feed additives are known to influence the incidence of ascites in broiler chickens. Ascites syndrome is prevalent in broiler farm all over Kerala irrespective of high and low altitude areas. However, the incidence in the flock appears to be higher in farms located in high altitudes (Jacob, 1996).

REFERENCES


