

A RARE OCCURRENCE OF FATTY LIVER KIDNEY SYNDROME (FLKS) IN AN ADULT CROSSBRED CHICKEN– A CASE STUDY

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Abstract: A dead layer crossbred chicken of 28 weeks of age was presented for post mortem examination with a history of sudden drop in egg production, anorexia, dullness, depression and abdominal swelling for a period of 1 week followed by sudden death. Post-mortem examination of the carcass revealed paleness, enlarged abdomen filled with sero-sanguineous fluid, enlarged liver, kidneys and spleen. Histopathological examination revealed mild haemorrhage, diffuse periportal hepatocellular hypertrophy with sinusoidal narrowing and intracytoplasmic macrovesicular steatosis in liver, diffuse glomerulonephritis with vacuolar degeneration in the kidneys and moderate myocardial degeneration with mild vacuolation in heart. However, scoring of fatty liver in the present case was 2, revealing that 50% or more hepatocytes contained vacoules of variable size. Also, scoring in kidney revealed 1 (mild). The scoring of heart and skeletal muscle revealed as score-0 (ie. no to very rare vacuolization). Thus present study showed the features of FLKS in both gross and histopathology, in a 28-week-old crossbred chicken. With significant increase in popularity of keeping backyard chickens and with increasing frequency of mortality due to FLKS, it is necessary that proper veterinary and nutritional recommendations to be made to owners for prevention.

Keywords: FLKS, glomerulonephritis, layer chicks, steatosis, vacuolations.

Fatty Liver and Kidney syndrome (FLKS) occasionally referred to as fatty liver and Kidney disease or pink disease, would affect both broiler and layer-type chicks usually when they were 10- 30 days old, and/ or chicks from young breeding stock. Genetic factors may also influence susceptibility. When the birds were subjected to mild stress caused by high or low temperatures, and/ or short term fasting, liver glycogen reserves become rapidly depleted and a progressive hypoglycaemia develops that ultimately proved fatal (Whitehead, 1979). However, field outbreaks were seen associated with wheat-based diets containing low levels of protein/ fat and also in the commercial diets which were subsequently found to be low in biotin, or with specially formulated low- biotin diets. In the absence of an adequate supply of biotin, the hepatic activity of pyruvate carboxylase, a biotin-dependent enzyme, becomes so low that gluconeogenesis in the liver via pyruvate would become negligible. There was

evidence of increased lipogenesis causing an increase of triacylglycerols in the liver lipids and an increased production of saturated fatty acids, particularly palmitic acid (Butler, 1976).

A 28-weeks-old female crossbred layer chicken was presented for post-mortem examination, with a history of anorexia, dullness, depression, sudden drop in egg production, abdominal swelling for a period of one week, followed by sudden death. In the present case, the bird was reported to be fed with broken wheat, rice, broken rice, ragi and commercial feed (at times). Postmortem examination of bird revealed paleness of comb, along with enlarged abdomen filled with sero-sanguinous ascitic fluid (Fig. 1).

Grossly, liver was tan to yellowish in colour, friable, enlarged approximately of 12x6 cm in size and covered the abdominal cavity entirely (Fig. 1). On incision, cut surface revealed corrugation with severe congestion and haemorrhage. The spleen and kidney were enlarged and pale (Fig. 2). Other organs such as proventriculus, gizzard and intestines were devoid of food particles and the mucosa was congested. In the thoracic cavity, the heart revealed paleness (Fig. 3) and enlarged in size while the lungs were found normal.

The representative tissue samples were collected and fixed in 10% neutral buffered formalin, processed for paraffin embedding, sectioned and stained with H&E for histopathological evaluation (Bancroft and Gamble, 2008).

Histopathological examination of liver revealed mild haemorrhage, diffuse periportal hepatocellular hypertrophy with sinusoidal narrowing and intracytoplasmic macrovesicular steatosis (Fig. 4 and Fig. 5). Kidney revealed diffuse glomerulonephritis with vacuolar degeneration (Fig. 6) and multifocal cellular degeneration was observed in spleen. Heart revealed moderate myocardial degeneration with mild vacuolation. Diffuse congestion was observed throughout the digestive tract. The gross and histopathological features of the present case were diagnosed as Fatty Liver and Kidney syndrome, which was in accordance with Butler (1976), where he reported that FLKS outbreaks had also been noticed in older flocks, as seen in our study. However, there were no inflammatory reactions in liver and kidney, which was in accordance with Wight and Siller (1975).

The degree of hepatocellular vacuolization characterized by single or multiple clear, well-demarcated cytoplasmic vacuoles was graded on the following criteria as viewed on H& E stained sections: (0) no to very rare vacuolization, (1) less than 50% of hepatocytes containing vacuoles of any size, (2) 50% or greater hepatocytes containing variably sized vacuoles or diffuse vacuolization with small vacuoles, and (3) diffuse vacuolization with large or medium to large vacuoles (Trott *et al.*, 2014).

The scoring was performed on H& E sections from two different areas of liver (from the present case). A random of 10 fields were selected and scoring of hepatocellular vacuolization was carried out (100 x magnification). The histopathological scoring revealed Score – 2; ie., 50% or more hepatocytes revealed vacuolization. However, assessment of degree of vacuolization in kidney (Score – 1) and heart & skeletal muscle (Score -0) revealed no to very rare vacuolization.

The involvement of various nutritional, toxicological, environmental and genetic factors had been suggested but, eventually, formulation of a diet that regularly reproduced high levels of mortality (20- 30%) showed that FLKS was basically a nutritional disorder. However, FLKS occurs in birds with little or no hepatic gluconeogenic capacity via pyruvate carboxylase (biotin dependent enzyme) as a result of a dietary insufficiency of biotin but that the initiation of the syndrome is probably associated with the inhibition of other pathways of gluconeogenesis (Whitehead *et al.*, 1978).

The role of biotin in preventing FLKS is thus explained in terms of its ability to maintain gluconeogenesis via pyruvate carboxylase and thus preventing hypoglycemia from occurring (Pearce and Balnave, 1978).

However, kidney gluconeogenic activity was significantly higher in FLKS affected birds (hence enlarged kidneys), suggesting an attempt by this organ to offset the reduced hepatic capability (Bannister *et al.*, 1975).

FLKS is thus a fascinating metabolic disorder in which nutritional factors render the bird susceptible to the condition and environmental factors initiate it. With significant increase in popularity of keeping backyard chickens and the high frequency of mortality due to FLKS, it is necessary that proper veterinary and nutritional recommendations to be made to owners for prevention. Educating the owners of backyard chickens regarding the nutritional recommendations should aid in decreasing the incidence of FLKS.

References

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Figures:



Fig. 1. Showing enlarged liver covering the whole peritoneal cavity, along with sero-sanguinous fluid (arrow mark).



Fig. 2. Showing enlarged and pale kidneys

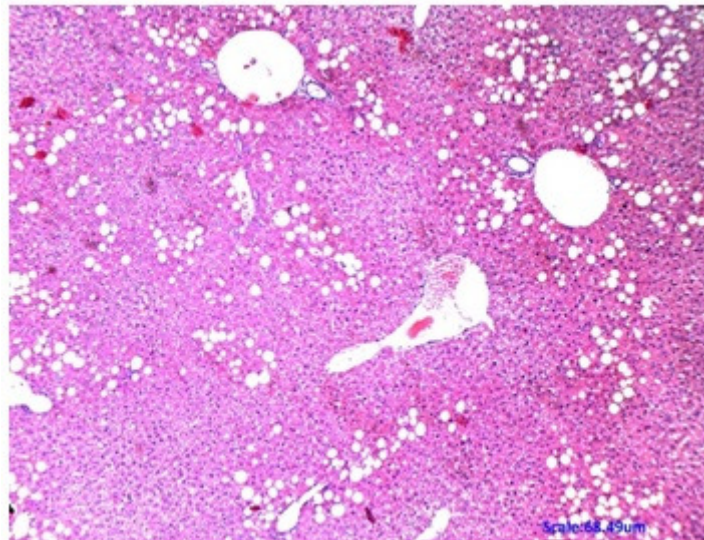


Fig. 3. Liver showing diffuse vacuolar degeneration and intraparenchymal haemorrhage.
H&E x 100.

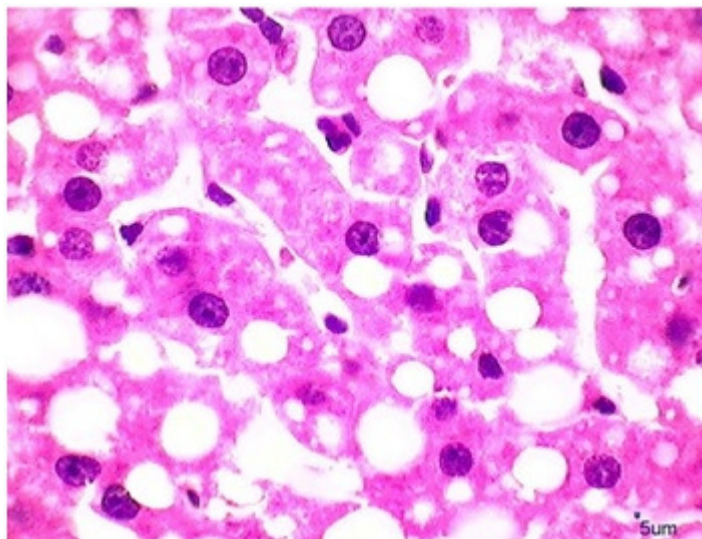


Fig. 4. Liver showing multifocal macrosteatosis within hypertrophic hepatocytes. H&E x 400.

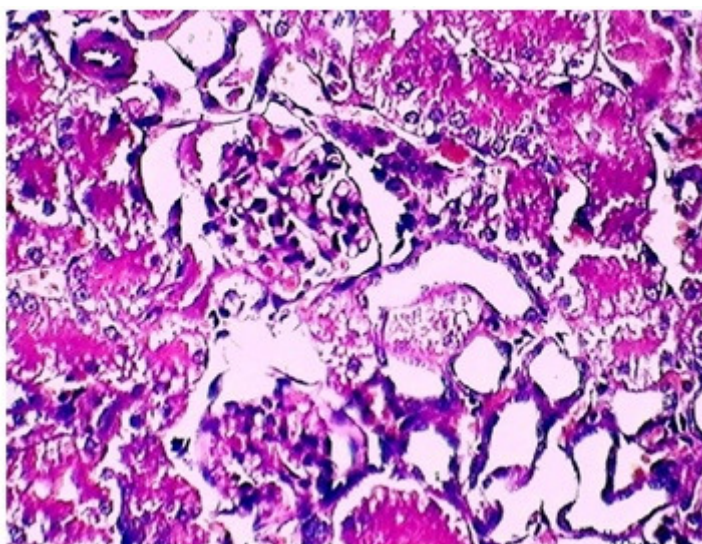


Fig. 5. Kidney showing diffuse glomerulonephritis and vacuolar degeneration. H&E x 400.