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A Case Report of Aflatoxicosis in a Country Chicken Unit with Retarded Egg Production

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A B S T R A C T

A backyard country chicken unit with 200 numbers of 8 month old birds was reported to have the production of poor quality eggs *viz.*, small sized, yolkless and watery albumin. Three of the birds were also showed continuous oozing of blood in between digits. The supplementation with mineral mixture and multivitamins yielded negative results. Three birds with the production of inferior quality eggs and retarded growth were humanely slaughtered and carried out post mortem examination. Post-Mortem examination showed enlarged, pale coloured liver, the cut surface bulged out with sticky oily substance. Microscopically, the liver showed vacuolar degeneration of hepatocytes, interlobular bile duct proliferation and infiltration of mononuclear cells in the intercellular area. The Oviduct was very pale and small. The mucosa of the magnal and shell gland portions was dry and less folded. The ovary revealed atretic and haemorrhagic follicles.

Introduction

Aflatoxins (AFs) B1, B2, G1 and G2 are mycotoxins that may be produced by 3 fungi of the *Aspergillus* species, *A. flavus, A. parasiticus* and *A. nomius*. Among the different types of AFs produced, AFB1 is the most prevalent and potent, often found in high concentrations in cereal grains, peanut meal, maize, groundnuts and their products (Gowda *et al.*, 2005; Cortes *et al.*, 2010). Aflatoxin toxins can cause tissue necrosis, hepatic cirrhosis and liver cancer (Abedi and Talebi, 2015). Aflatoxins influence the metabolism of poultry and the main manifestations of chronic aflatoxicosis in layers are reduced and retarded egg production (Siloto *et al.*, 2011).

The aflatoxins have nutritional and toxicological effects on reproduction in male and female chicken (Kovacs, 2004).

The different categories of the ovarian follicles in the domestic hens may differently get affected by noxious impacts of the AFB1.

Aflatoxin even affects the blood coagulation mechanism in chicken due to the loss of one active component of tissue thromboplastin.

Factors VII, V, X and even Prothrombin, were primarily affected during this coagulopathy (Doerr *et al.*, 1976).

Materials and Methods

Three numbers of 8-month-old, 800-g country chicken with history of poor quality and quantity of egg production and continuous bleeding from a wound in between toes, below the left shank region was reported to Veterinary University Training and Research Centre, Ramanathapuram, Tamilnadu,. TANUVAS Mineral mixture supplementation at the rate of 5g/ bird/ day for a period of 2 months, in addition to the layer ration of feed mixture and Ethamcip (10 mg/kg orally; Cipla limited, Mumbai) for 1 month were carried out. Finally, the ailing birds (3 Nos) were sacrificed and autopsy were conducted on the carcass. The feed samples for toxic analysis was collected and analysed for the presence of fungal toxins.

Results and Discussion

The reported bird was a country chicken of 7month old. The eggs laid were of varying sizes from peanut to quail eggs and were of irregular in shape without egg yolk. The albumin present was also watery in nature (Figure 1). Some of the eggs laid were leathery. The ailing bird had an open wound with oozing of blood in between the toes in the left leg.

The wound was locally treated with povidone iodine solution. The oozing of blood stopped in about a month, but the production was continued to be the same with deteriorated quality. The shape and other qualities of the eggs were of the same and were unresponsive to the treatment. Post-mortem examination revealed visible deposits of sub-cutaneous fat in peritoneum and all visceral organs (Figure 2). The liver was pale, yellow and enlarged. Cut surface bulges out with sticky oily materials. The mucus membrane from the ventricles did not peel off easily. Intestine was filled with the segments of cestodes and the mucosa was thick and severely congested. Ovary showed varying sizes of ruptured and atretic follicles. Toxicological analysis of the feed sample showed the presence of Aflatoxin (B1 158ppb).

Microscopic study of liver showed interlobular bile duct proliferation, lymphocyte infiltration and mild vacuolar degeneration in hepatocytes. Ovary showed the presence of atretic follicles and the uterine portion of oviduct had atrophic changes in the shell glands.

The reduction in production might be due to the drop of nutritive status of the birds due to the parasitic infestation and the damage caused to the liver by the toxin. The reduction in the quality might have been contributed by the effect of the toxin on the shell gland and also on the hepatic parenchyma and is in accordance with the report of Aravind *et al.,* (2001).

Rosmaninho *et al.,* (2001) reported that, the main manifestations of chronic aflatoxicosis in layers are production drop and enhanced hepatic fat levels. Aflatoxicosis caused poor egg production and decreased egg weight (Huff *et al.,* 1975). The influence of aflatoxicosis on blood coagulation was reported by Bababunmi and Bassir (1982). The effect of aflatoxin on reproductive performance of poultry breeding stock has been identified and assessed by the early workers (Hafez *et al.,* 1982; Pandey and Chauhan, 2007; Aly and Anwer, 2009).

The toxicity (158ppb) of the source of contaminant (aflatoxin B1) in feedstuffs (GNC), produced significant liver damage and coagulation responses in the bird, probably the tissue thromboplastin was lost during aflatoxicosis and Prothrombin the most sensitive factor was reduced in this case might be the cause for delayed blood coagulation.

Fig.1 (A): Abnormal size and shape of eggs. (B): Cut open eggs showing under developed albumen without egg yolk. (C): Leathery nature of albumen. (D): Cut open egg without albumen and egg yolk

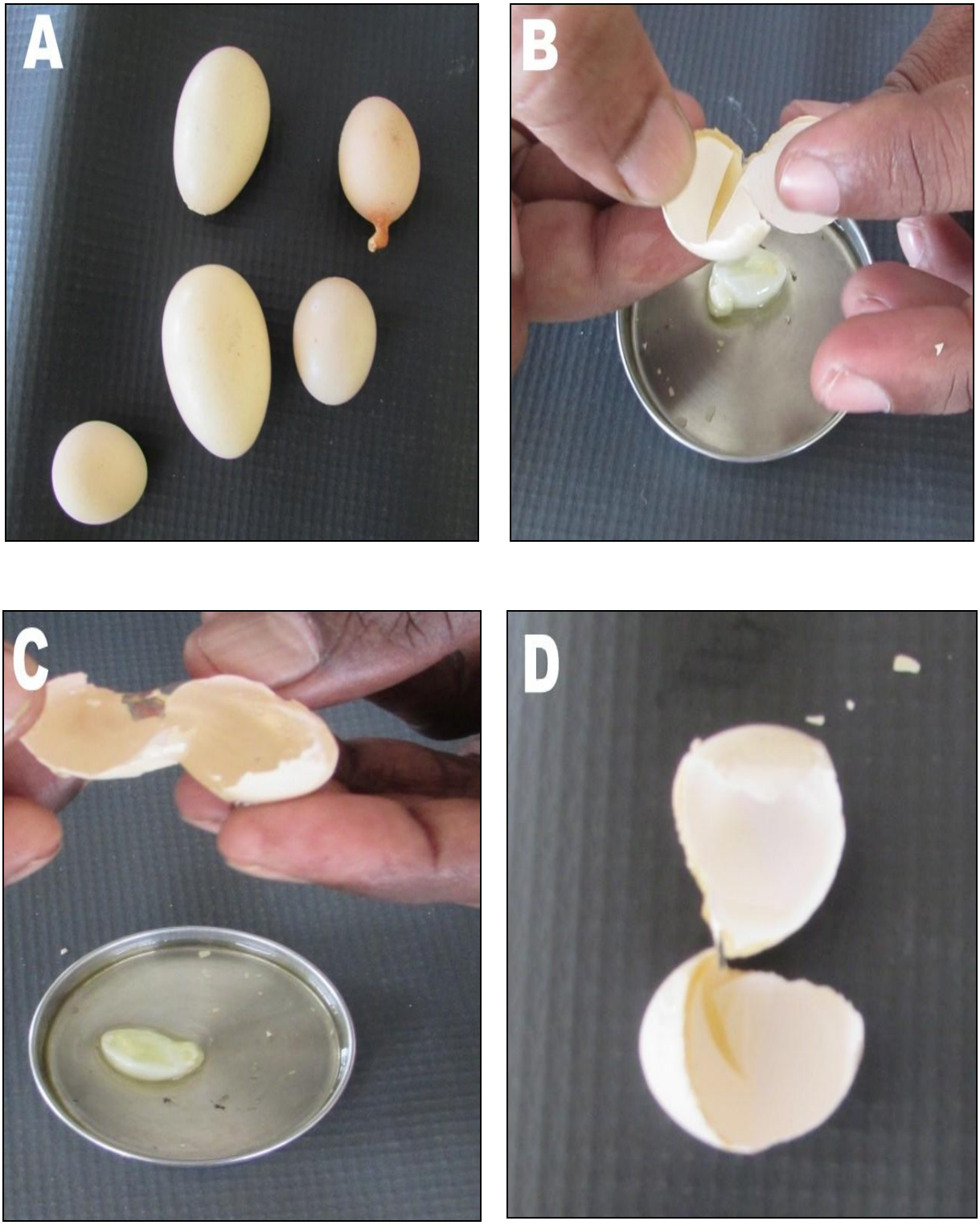
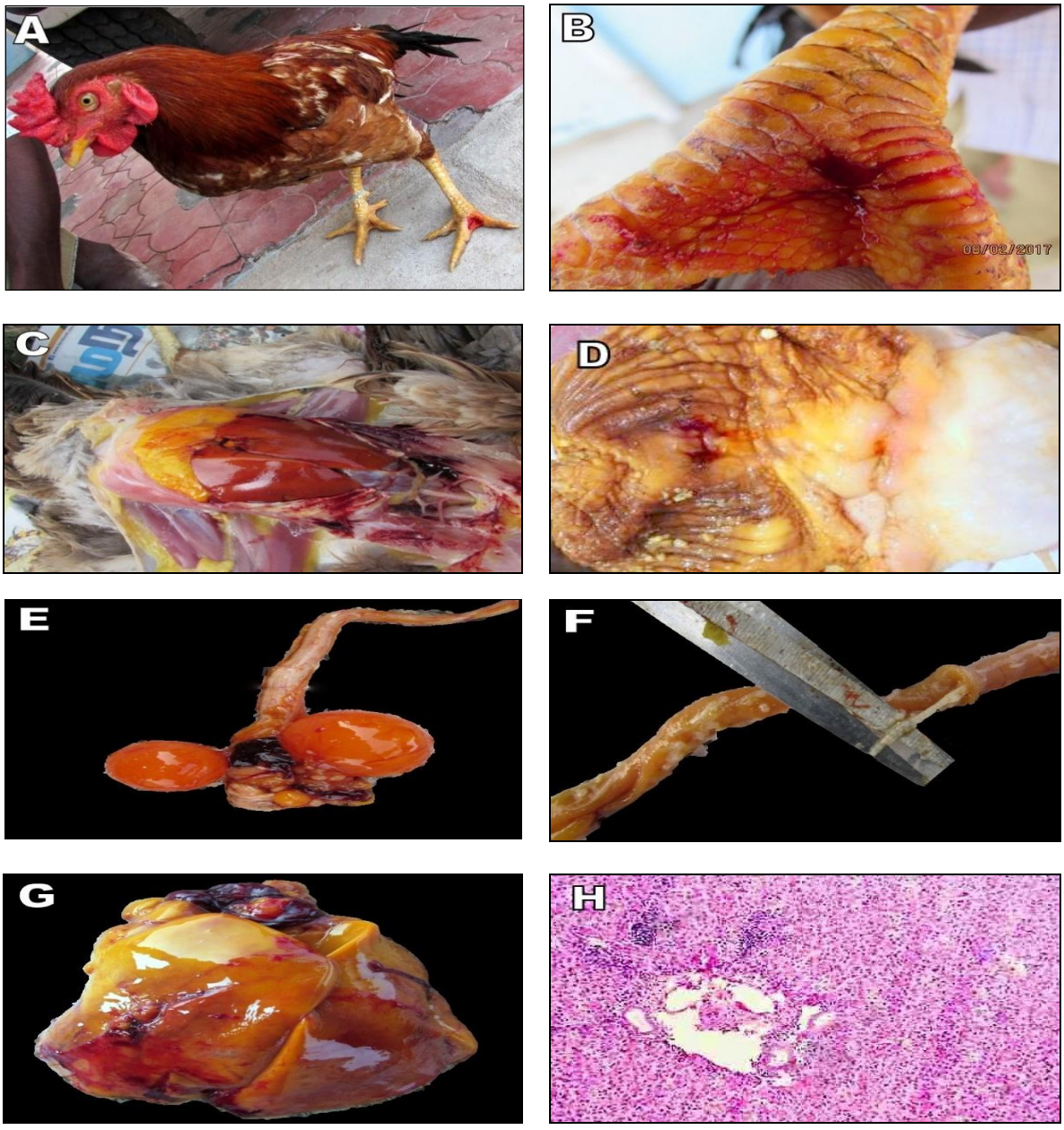


Fig.2 (A): Self-mutilated wound in between toes, below the left shank region (B): Continuous oozing out of uncoagulated blood (C): Subcutaneous fat deposition in peritoneum and visceral organs (D): Proventriculus exhibiting toxic deposition (E): Atretic and immature ovarian follicle. (F): Cestodiasis in small intestine. (G): Fatty and Cirrhotic liver (H): Liver lesion with interlobular bile duct proliferation, lymphocyte infiltration and mild vacuolar degeneration in hepatocytes



The accumulation of chronic toxicity was well evinced during post-mortem lesions predominant in liver (fatty liver and cirrhosis) and further by interlobular bile duct proliferation, lymphocyte infiltration and mild vacuolar degeneration in hepatocytes. The mucosa of Proventriculus did not peel off easily showed the chronic accumulation of

toxicity in the case studied. The cestode infection in the present case was being a concomitant infection attributed due to deprived immune response due to aflatoxicosis. The present report was in confirmation to the study of hemorrhagic episodes and high incidence of bruising in poultry during aflatoxicosis prompted an investigation of the effect of aflatoxin on parameters relating to hemostatic competence in chickens (Bababunmi and Bassir, 1982) stated tissue thromboplastin and Prothrombin, the most sensitive factor were affected drastically, suggest that severe impairment of extrinsic and common clotting pathway functions occurs during aflatoxicosis in the chicken and that prothrombin was primarily affected during this coagulopathy.

Effect of aflatoxin contaminated diet on performance of laying hen in the study showed reduced egg production and laid small eggs with abnormal shapes and the size varied from peanut to quail egg sized. Some of the eggs laid were leathery and the eggs laid with shell were devoid of albumin and egg yolk, opined to the study (Aly and Anwer, 2009), stated a significant decrease in egg production and egg weights of laying hen. Aflatoxin incorporated into the feed of laying hens may cause relevant lesions in liver and in kidneys, heart and ovaries.

The ovaries showed follicular atresia which has a detrimental effect on egg production (Hafez *et al.,* 1982; Pandey and Chauhan, 2007), similar to the lesions of atretic follicles in female reproductive tract. The variation of aflatoxin residue in the eggs confirm that only small quantities of the aflatoxins are likely to be deposited, while the majority of aflatoxins detoxified and /or stored in liver and other poultry tissues such as ovary, kidney, crops, breast muscles, thigh and excreted in excreta (Trucksess *et al.,* 1983; Bintvihok *et al.,* 2002).

The case report described the condition of aflatoxicosis and the unawareness of poultry owner feeding the flocks with contaminated feedstuff (GNC) and visible symptoms of bleeding from wound as a result of delayed blood clotting mechanism and poor quality of egg produced.

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