Effect of T-2 toxin on growth, performance and haematobiochemical alterations in broilers

V V Pande, N V Kurkure* & A G Bhandarkar
Department of Pathology, Nagpur Veterinary College, Maharashtra Animal and Fishery Sciences University, Nagpur 440 006 India

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Administration of dietary T-2 toxin in 120 days old broiler chicks led to significant lower body weights and increase in feed conversion ratio from 2nd week of age. There was significant reduction in haemoglobin and packed cell volume in T-2 toxicated birds at 4 ppm level only. The other hematological parameters like TEC, TLC and absolute leucocyte count did not showed any variation due to T-2 toxin in feed. Significant reduction in serum total protein and cholesterol levels and rise in serum uric acid and LDH levels of broilers were observed due to dietary T-2 toxin. The result suggests that T-2 toxin is toxic to broilers even at very low concentrations.

Keywords: Broilers, Haemato-biochemical, Poultry, T-2 toxin

Mycotoxins are worldwide contaminants of food and are secondary metabolic by-product of various toxigenic fungi growing on suitable substrate. Mycotoxins such as aflatoxins and ochratoxins are produced optimally at 24°-28° C but T-2 toxins are produced maximally at 15°C1. T-2 toxin is the most important toxin causing emerging threats in poultry industry. T-2 toxin, a secondary toxic metabolite is produced mainly by fusarium spp. i.e. F. sporotrichoides, F. tricinctum, F. eusetti, F. culmorum, F. avenaceum, F. roseum, F. nivale, F. poae and by others species like Cephalosporium spp, Myrothecium spp, Stachybotrys spp and Trichoderma spp. There is a paucity of scientific literature regarding its pathogenicity. Hence, in the present study an attempt has been made to study the effect of T-2 toxin on growth and performance and haematobiochemical changes in the broilers.

A known culture of Fusarium sporotrichoides MTCC1894 (Institute of Microbial Technology, Chandigarh, India) was used for the production of T-2 toxin. The culture of F. sporotrichoides inoculated on Sabouraud’s dextrose agar (SDA) was incubated for 7-14 days at room temperature. The conidia were suspended in Sabourad’s dextrose broth (SDB) by gently scrapping the agar surface with a wire loop and used for toxin production with slight modification in the suggested method2. Toxin was quantified at Animal Feed and Quality Control Laboratory (AFAQCL), Namakkal, Tamilnadu employing standard method3. The experiment was conducted at poultry farm of Nagpur Veterinary College, Nagpur. One-day-old commercial broiler chicks (Vencobb)(120) obtained from Venkteshwara Hatcheries Ltd, Hyderabad were divided randomly into 4 groups of 30 birds each at day old stage. Feed and fresh water were available ad libitum to all chicks from 1st to 42nd day and they were reared under standard mangmental condition. Feed free of toxin binder and mycotoxins was procured from local feed manufacturer.

Dietary levels of T-2 toxin in experimental diets were adjusted by adding calculated amount of mouldy maize to the diet. The experimental diets containing graded concentration of T-2 toxin were fed at 1, 2 and 4 ppm in group B, C and D from 1st to 42nd day of age respectively. Group A birds did not receive T-2 toxin and served as control.

To study the effect of T-2 toxin on growth, experimental chicks were weighed individually every week and weekly feed intake was also recorded to calculate feed conversion ratio (FCR) of each treatment group. For haematological and serum biochemical investigations, blood samples were collected from jugular vein using EDTA anticoagulant from six birds of each group at 21st and 42nd day of age4. Serum was separated and stored at 4°C. Individual serum sample was analyzed for serum total protein, cholesterol, uric acid, lactic dehydrogenase (LDH) using commercial kits (Transasia Pvt. Co. Ltd. Daman) on auto analyzer (Systorms clinical chemistry analyzer 171) within 24 hr of collection. All data were subjected to one-way ANOVA5.

Growth and performance — Significant (P< 0.05) reduction in body weight was observed in group D since first week; group C from second week, group B fourth week onward as compare to group A (Table 1). The highest FCR was recorded in group D whereas,
lowest FCR was recorded in group A. FCR of groups B and C was same but more than group A. The reduction in body weight of chicks in the present study due to T-2 induced toxicosis was dose dependent, which was similar as described by Wyatt et al. and Natraja et al. However, there was depressed body weight gain at the toxin levels above 4 ppm only. Raju and Devegowda also reported reduced body weight and poor feed conversion efficiency of broilers due to T-2 toxin @ 3 mg/kg of feed. Reduction in body weight of T-2 toxicated chicks may due to inflammation, contact erosion and irritation of alimentary tract resulting into decrease in feed consumption and consequently decrease in body weight of toxicated birds.

Haematobiochemical parameters— At 21st day of age the mean haemoglobin (Hb) values of chicks from all T-2 toxin fed groups were significantly lower as compared to control. The effect was predominantly seen in group D wherein Hb value was significantly lowest as compared to group B and C. At 42nd day of age, numerically lowest value of Hb was recorded in group D (7.00 ± 0.50) and highest in group A (8.40 ± 0.60). However the difference in Hb concentration among experimental groups was not significant. On comparison of packed cell volume (PCV) of the experimental groups, it was observed that PCV was significantly reduced in group D as compared to other groups (A, B, and C) at 21st day of age. On 42nd day of age, there was significant rise in serum uric acid of all T-2 toxicated groups as compared to control group. There was significant effect of T-2 toxin on the serum LDH value of chicks at 21st day (P<0.05) and 42nd day (P<0.01) of age. At the both period of observations there was rise in serum LDH of chicks fed 4 ppm dietary T-2 toxin (group D) alone as compared to groups A, B, and C which was similar to the findings in birds fed T-2 toxin at various dose levels. The observed reduction in total serum total protein and cholesterol might be due to hepatotoxic effect of T-2 toxin. The rise in uric acid level might be the result of poor utilization of protein for tissue protein synthesis rather than decreased renal function as postulated earlier by Coffin and Combs. The alterations recorded in the LDH values of experimental chicks could be attributed to the lesions caused by T-2 toxin in vital organs of chicks.

References
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