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EFFECT OF MONENSIN, AN IONOPHORE ANTIBIOTIC ON SERUM BIO-CHEMICAL PROFILE IN BROILER CHICKEN*

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Use of monensin, a Na+ selective monovalent ionophore, as a coccidiostat feed additive in poultry is now on increase because of its high efficacy and slow development of resistant strains to it, compared to other anticoccidial agents. Though, monensin is safe at approved level (121 ppm), toxicosis can occur even with slightly higher levels because of its narrow safety margin. Further, difficulty of ensuring an even distribution of the drug throughout the feed aggravates this problem (Dowling, 1992). Data on serum bio-chemical profile during monensin toxicosis are scanty. Therefore, the present investigation was undertaken to study the serum biochemical profile in experi- mentally induced monensin toxicosis in broiler chicken.

Materials and Methods

A total of one hundred and twenty, Cob broiler chicks of one day age from the same genetic stock were randomly assigned to four groups each consisting 30 chicks. Chicks in group-I served as control and received standard broiler diet. Chicks in groups II,III and IV were fed with standard broiler diet containing monensin (Coban-100[1] containing 10% monensin sodium) at 121, 363 and 726 ppm, respectively, for 8 weeks. All the birds in four groups were provided with 24 hours of light and free access to water and feed. They were also protected against common poultry diseases by preventive vaccination. Blood samples were collected by venesection from 10 randomly selected birds in each group at the end of 4th and 8th week of the experimental period. Care was taken to prevent haemolysis of the blood samples. Serum was seperated from the blood samples and aspartate transaminase (AST) (Reitman and Frankel's method), alanine transaminase (ALT) (Reitman and Frankel's method), lactate dehydrogenase (LDH) (King's method) and creatine phosphokinase (CPK) (Modified method of Hughes) were determined by using commercially available ready to use kits[2]. The data were analysed by using t-test as per Snedecor and Cochran (1967).

Results and Discussion

Mean values corresponding to the activity of serum aspartate transaminase, alanine transaminase, lactate dehydrogenase and creatine phosphokinase in all the groups at the end of 4th and 8th week of the experimental period are presented in Table 1.

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1. *Venky's India Pvt. Ltd., Pune, India*
2. *Span Diagnostics Pvt. Ltd., Surat, India*

Table 1 Effect of dietary monensin on serum enzymatic activity (Mean IU/L ± S.E.) of broilers

Group (Monensin ppm)	Age (Weeks)	Serum aspartate transaminase (AST)	Serum alanine transaminase(ALT)	Serum lactate dehydrogenase(LDH)	Serum creatine phosphokinase(CPK)
I (0)	4	$109.4^{A} \pm 1.31$	$15.0^{A} \pm 0.46$	$170.00^{A} \pm 0.82$	$457.00^{A} \pm 0.29$
	8	$107.0^{A} \pm 0.59$	$10.0^{A} \pm 0.52$	$154.00^{A} \pm 0.84$	$454.00^{A} \pm 0.93$
II (121)	4	$141.0^{B} \pm 1.06$	$22.0^{B} \pm 0.72$	$243.00^{B} \pm 1.00$	$693.00^{B} \pm 6.60$
	8	$168.0^{b} \pm 1.06$	$28.2^{b} \pm 0.70$	$221.00^{b} \pm 1.08$	$560.00^{C} \pm 1.90$
III (363)	4	$352.0^{C} \pm 1.34$	$52.0^{C} \pm 1.66$	$959.00^{C} \pm 10.06$	$1991.00^{C} \pm 140.17$
	8	$298.0^{c} \pm 1.05$	$35.0^{c} \pm 0.77$	$623.00^{c} \pm 16.70$	$761.00^{c} \pm 1.75$
IV (726)	4	$427.0^{D} \pm 1.37$	$64.0^{D} \pm 0.77$	$1050.00^{D} \pm 21.68$	$3097.00^{D} \pm 252.56$
	8	$321.0^{d} \pm 1.56$	$38.33^{d} \pm 0.50$	$891.66^{d} \pm 33.77$	$1217.00^{d} \pm 87.21$

While the serum AST activity was unaltered in group-I, there was a significant ($P<0.05$) increase in all the monensin-fed groups. Elevated serum AST activity is in concurrence with the reports of Horovitz et al.(1988). Increase in the serum AST activity can be attributed to damage to the hepatic cells and myocardium by monensin. Muylle et al.(1981) stated that serum AST activity was increased because of injury to muscles and hepatocytes since monensin was stored and metabolized in liver.

Serum of birds in all the monensin-treated groups revealed significantly ($P<0.05$) elevated activity of ALT while there was no change in the control group birds. Similar reports were also made by Horovitz et al. (1988). However, Sawant *et al.* (1990) did not observe any consistent alterations in the activity of serum ALT in monensin fed quails.

Birds among groups II, III and IV showed significantly ($P<0.01$) higher activity of serum LDH when compared to that of control group. Such findings were also reported by Horovitz et al. (1988). Increase in the activity of serum LDH in monensin-fed birds is suggestive of myocardial damage during monensin toxicosis (Langston *et al.*, 1985).

Greatly elevated activity of serum CPK was observed in all the monensin-fed groups. This elevation in the serum CPK activity was significant ($P<0.01$) when compared to that of control group birds. This effect of monensin on serum CPK activity is in close agreement with the findings of Horovitz et al. (1988). Emaciation and damage to the skeletal muscles might be the cause for elevated serum CPK activity since the enzyme CPK is relatively specific to striated muscles (Freedland and Kramer, 1970).

Significantly elevated activities of various serum enzymes viz., aspartate transaminase, alanine transaminase, lactate dehydrogenase and creatine phosphokinase in monensin-fed birds

in the present study might be directly related to the cell necrosis and cell death. Langston et al. (1985) stated that monensin carries Na+ into the cells to be exchanged for a proton, thus causing acidosis and loss of K+ from the cells. Increase in Na+ concentration of the cell increases Ca++ influx and increased Ca++ concentration produce mitochondrial swelling early in the disease process. As the cell become saturated with Ca++ cellular phospholipases and proteolytic enzymes get activated which finally affects the cellular membrane integrity.

Summary

Monensin, an ionophore antibiotic fed to broiler chicken at the rate of 121 (recommended use level), 363 and 726 ppm produced a significant and dose dependent increase in the activity of serum aspartate transaminase (AST), alanine transaminase (ALT), lactate dehydrogenase (LDH) and creatine phosphokinase (CPK) indicating hepatic, cardiac and skeletal muscle damaging potentials of the compound.

References

Dowling, L. (1992). Ionophore toxicity in chickens: a review of pathology and diagnosis. Avian Diseases, 21: 355-368

Freedland, R.A. and Kramer, J.W. (1970). Use of serum enzymes as aids to diagnosis. Advances in Veterinary Science and Comparative Medicine. 14: 61-103

Horovitz, C.T., Avidar, Y., Bogin, E., Schlosbert, A., Shkap, I., Weisman, Y. and Egyed, M.N. (1988). Enzyme profile in blood and tissues of chickens fed various levels of monensin. J. Vet. Med. 35: 473-480

Langston, V.C., Graley, F., Lovell, R. and Buck, W.B. (1985). Toxicity and therapeutics of monensin: A review. Vet. Med. 10: 75-84

Muylle, E., Vandenhende, C., Dyaert, W., Thoonen, H. and Vlaeminck, K. (1981). Delayed monensin sodium toxicosis in horses. Equine Vet. J. 13: 107-108

Sawant, S.G., Terse, P.S. and Dalvi, R.R. (1990). Toxicity of dietary monensin in quail. Avian Diseases. 34: 571-574

Sendecor, G.W. and Cochran, W.G. (1967). Statistical methods. 6th Ed. Oxford and IBH Publishing Company, Calcutta, Bombay and New Delhi.