TOXICITY OF DICLOFENAC SODIUM IN

TOXICITY OF DICLOFENAC SODIUM IN BROILER BIRDS

R. K. Sharma, K. Shrman, Varsha Sharma and N.V. Bhamre Dept of Veterinary Pharmacology,

College of Veterinary Science and Animal Husbandry, Jabalpur (M.P.). 482001, India Received 14-10-2011 Accepted 14-2-2012

ABSTRACT

The present research work was conducted to study the effect of diclofenac sodium on broilers birds. The study was carried out on 40 broiler birds equally divided in two group i. e. treatment and control. In treatment group diclofenac sodium was administered @5mg/kg b.w. /day for 21 days. In treatment group mortality, reduction in weight gain and lameness was observed. However, except haemoglobin and heterophil count, most of other haematological parameters did not show any significant changes. Significantly higher levels of uric acid, creatinine and urea were found in treatment group, denoting renal damage. Necropsy examination of dead bird show deposition of uric acid crystals on internal organs.

KEY WORDS: Diclofenac sodium, gout, broiler bird, uric acid crystal, nephrotoxicity

INTRODUCTION

Diclofenac sodium a non-steroidal anti-inflammatory drug (NSAID) belongs to the family phenyl acetic acid is a potent analgesic drug. This drug is being frequently used in management of inflammatory and painful conditions in human and veterinary practice. It was used as an economical analgesic to ease lameness in broiler and in beak trimming in layer birds. NSAIDs, inhibits enzyme cyclo-oxygenase (COX-1 and COX-2), involve in the formation of prostaglandins and has been implicated as a cause of its renal side effects. In conditions of severe renal dysfunction there is an increase in blood uric acid concentration resulted in visceral gout (Gilbert, *et al.* 2003). Considering the common use of NSAID's and their detrimental effects on avian species, this study was planned in broiler birds.

MATERIALS AND METHODS

The present research work was carried out on 40 day old chicks kept under standard housing and management conditions. All the birds were offered balance diet fulfilling all nutritional requirements but free from coccidiostats and antibiotics. Feed and water were provided *ad-libitum* to the birds. These birds were divided in to two groups A and B, 20 birds in each group. Group A, served as untreated control and group B treated with diclofenac sodium @ 5 mg / kg b.w. /day mixed with feed for 21 days. Birds were observed twice daily and subjectively evaluated for clinical signs and behavioural alterations.

Two ml blood was collected in heparinized vial from wing vein weekly for 3 weeks. Blood samples were analysed for estimation of haematological and biochemical parameters by standard methods in use. Plasma was separated immediately and stored at -20°C until further use. Post mortem examinations of dead birds were carried out. The birds were sacrificed at the end of the study i.e. on 21st day; and liver and kidney samples were collected for gross and histo-pathological examination. These organs were fixed in 10% buffered formalin and processed for histo-pathological examination using routine paraffin embedding method. Data were analyzed using unpaired t-test between control and treatment groups.

RESULTS AND DISCUSSION

The effect of Diclofenac sodium administration on Haemato-Biochemical alterations is presented in Table.

broiler birds				
Parameter	0	Time interval		
	Group	1 st week	2 nd week	3 rd week
Average weekly weight gain (g/bird)	Control	100.00±0.01 ^a	225.15±0.03 ^a	330.80±0.01 ^a
	Treatment	85.15±0.02 ^b	150.25±0.02 ^b	245.35±0.03 ^b
Erythrocyte count(million/mm ³)	Control	3.98±0.01	4.12±0.02	4.13±0.02
	Treatment	4.34±0.02	4.53±0.01	4.42±0.01
Total leukocyte count (thousand /mm ³)	Control	19.96±0.01 ^a	22.51±0.02 ^a	18.73±0.01 ^a
	Treatment	16.01±0.02 ^b	16.23±0.02 ^b	16.12±0.01 ^b
Lymphocyte (%)	Control	50.65±0.01	58.35±0.01	58.21±0.02
	Treatment	58.09±0.02	57.01±0.02	55.11±0.02
Heterophils (%)	Control	18.06±0.01 ^a	18.10±0.29 ^a	20.20±0.02 ^a
	Treatment	47.90±0.03 ^b	49.70±0.01 ^b	51.34±0.03 ^b
Monocyte (%)	Control	9.35±0.01	9.21±0.03	9.10±0.02
	Treatment	8.09±0.02	8.01±0.01	8.76±0.01
Eosinophils (%)	Control	4.65±0.02	5.50±0.01	4.75±0.01
	Treatment	4.04±0.02	4.15±0.01	4.01±0.02
Basophils (%)	Control	1.05±0.01	1.01±0.02	0.99±0.01
	Treatment	0.99±0.02	0.95±0.01	0.90±0.03
PCV(%)	Control	31.08±0.01	39.35±0.02	42.50±0.02
	Treatment	29.05±0.01	30.76±0.02	32.21±0.01
Haemoglobin (g/dl)	Control	11.09±0.01 ^a	12.01±0.02 ^a	12.08±0.02 ^a
	Treatment	8.53±0.03 ^b	6.91±0.02 ^b	4.49±0.01 ^b
Uric acid (mg/ dl)	Control	6.15±0.02 ^a	6.38±0.01 ^a	6.47±0.01 ^a
	Treatment	9.44±0.03 ^b	17.56±0.02 ^b	33.19±0.01 ^b
Urea (mg/dl)	Control	0.42±0.02 ^a	0.67±0.01 ^a	0.53±0.03 ^a
	Treatment	2.79±0.02 ^b	2.93±0.01 ^b	3.50±0.01 ^b
Creatinine (mg/dl)	Control	2.07±0.01 ^a	2.01±0.02 ^a	2.09±0.02 ^a
	Treatment	3.43±0.01 ^b	3.59±0.03 ^b	4.65±0.02 ^b

Table : Effect of Diclofenac administration on Haemato-Biochemical parameters in				
broiler birds				

Values mean ±SE; n=20. Mean with different superscript differ significantly (P<0.05)

36

2012) TOXICITY OF DICLOFENAC SODIUM IN

Clinically the birds were dull and depressed showing apathy, ruffled feather and reluctant to move. Lamness was developed six days after administration. Patel (2005) also reported similar findings in diclofenac sodium treated birds. At the end of 3rd week 40 % mortality was observed. Similar findings were reported by Agrawal and Prajapati (2008) in Cobb 100 chicks and Reddy *et al.* (2006) in Vanaraja and PB1 birds within 12 days after administration of diclofenac sodium at same dose rate by oral route.

From perusal of the result, it appears that administration of diclofenac sodium causes a significant decrease in weight gain in all the three groups. Our findings corroborate with the reports of Hussain *et al.* (2008)

There was a significant alteration in Hemato-biochemical paramenters in birds administered with diclofenac sodium. Except erythrocyte and lymphocyte (which are increased non significantly) other blood components such as monocyte, eosinophil, basophil and PCV decreased non significantly, but there was a significant increase in heterophil lelvel and at the same time haemoglobin level and leukocyte level was decreased significantly which may be due to reduced feed intake and increased level of stress in birds. Increase in heterphil count indicate tissue damage. On the other hand biochemical parameters uric acid, urea and creatinine increased significantly in all the treatment groups as compared to control group birds. Diclofenac induces necrosis of proximal convoluted tubules leads to reduction in uric acid, and creatinine excretion causing rapid elevation of uric acid and creatinine in blood. Increased uric acid, creatinine and urea in the present study corroborates with the reports of Uma *et al.* (1999), Jain *et al.* (2011) and Hussain *et al.* (2008) respectively.

PM findings: Post mortem examination of dead and sacrificed bird revealed typical sign of visceral gout with deposition of urate crystal on pericardium, gizzard, intestine, thigh muscle and subcutaneous tissues. Liver was found to be covered with urate deposits, forming white crystalline layer on its surface. Kidneys were pale with white chalky urate deposits on the capsule.

REFERENCES

Agarwal, S. and Prajapati, K. S. (2008). Indian Journal of Veterinary Pathology 32(1):34-37.

Gilbert, M. L., Oaks, M. Z., Viran R. T., Watsaon S., Ahmed M. I., Chaudhary M., Arshad S., Mahmood A., Khattak R. M., and Khan A. A. (2004). The status and decline of vultures in the provinces of Punjab and Sind, Pakistan: a 2003 update. Maybyrg

Hussain I., Khan M. Z., Khan A., Javed I., and Saleemi M. K. (2008). Avian Pathology **37**(3):315-21.

Jain T., Koley K. M., Vadlamudi V. P., Ghosh R. C., Roy S., Tiwari S. and Sahu U. (2011)., Indian Journal of Pharmacology **41**:237-41.

Patel A. K. (2005). Experimental studies on etiology of gout in broiler chicks. M.V.Sc.Thesis, Anand Agriculture University, Gujrat.

Reddy N. C., Anjaneyulu Y., Sivasankari B. and Rao K. A. (2006). Environmental Toxicology and Pharmacology **22**:142-47.

Uma C. A., Vijayasarathi S. K., Nalini T. S., Satyanarayana M. L. and Rao, S. (1999). Indian Journal of Veterinary Pathology **23**: 94-95.

37