Pathomorphological alterations in oxytetracycline toxicosis in goats*

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This paper deals with the pathomorphological alterations in different organs of goats receiving high doses of long-acting oxytetracycline. This could be a model study, the findings of which may find applicability in ruminants as a whole under field conditions in respect of proper planning of treatment schedule with adequate dose as well as early detection of toxicosis.

Healthy indigenous goats (10), 2 to 3 years old, weighing 10-15 kg and maintained under identical feeding and managemental conditions, were used. Toxicity was induced in 5 goats of group A by 3 i/m injections of long-acting oxytetracycline @ 50 mg/kg body weight on alternate days. The animals were allowed to recover spontaneously or die. Group B of 5 goats did not receive antibiotic treatment and served as healthy control.

For pathomorphological studies, all the 5 dead animals of group A, and 2 animals from group B which were sacrificed on day 32 were thoroughly necropsied and gross changes were recorded. Representative pieces of tissues (3-5 cm in thickness) from kidneys, liver, intestine, brain, lung and heart were fixed in 10% formal saline and paraffin sections (4-5 μ) were stained with haematoxylin and eosin for histopathological studies (Lillee and Fulmer 1976).

The clinical signs of oxytetracycline toxicity in group A goats began to appear from day 4. Symptoms were depression and kyphosis initially followed by anorexia, profound weakness, oculo-nasal discharge, diarrhoea, dehydration, salivation and dyspnoeic respiration leading to coma and death of all the animals by day 7 (1 animal each died on days 4, 5 and 6, and the rest 2 on day 7). At the time of death, a marked increase in serum urea nitrogen (86.82±3.48 mg/dl against day 0 value of 17.19±0.94 mg/dl) and serum creatinine (5.61±0.38 mg/dl against day 0 value of 1.11±0.04 mg/dl) was observed suggesting severe azotaemia as reported elsewhere along with other blood biochemical alterations. The control (group B) animals remained clinically healthy with normal serum urea nitrogen and creatinine levels (Kumar 1991).

The pathomorphological changes in different organs were discussed.

Gross lesions

In group A animals, about 1 litre of straw-coloured fluid containing fibrin clots was present in the peritoneal cavity. Kidneys were pale and swollen showing diffuse greyish areas on its surface. Liver was swollen and hard, and had diffuse greyish-white foci on its surface. Epicardium and intestinal mucosa showed congestion, brain, lungs, spleen, urinary bladder and adrenal glands had mild congestion in few animals.
October 1994] PATHOMORPHOLOGICAL CHANGES DUE TO OXYTETRACYCLINE TOXICOSIS

Histopathological lesions

Group A: Marked tubular degenerative changes, particularly in proximal convoluted tubules of kidney leading to vacuolar degeneration (Fig. 1), were the consistent observations. Engorgement of blood vessels in medullary region and at places, areas of haemorrhages were also evident. Glomeruli occasionally showed granular eosinophilic mass.

Liver showed mild to moderate granular and vacuolar degeneration in hepatic cells in peripheral portion of hepatic globules (Fig. 2). Superficial coagulative necrosis of epithelium and elongation of villi were the main lesions seen in lungs. Heart showed

engorgement of blood vessels and focal areas of haemorrhages in between muscle fibres. In brain, focal of gliosis and satellitosis were recorded.

**Group B:** Two animals sacrificed at the end of the experiment did not reveal any gross or histological changes of pathological significance.

The varying degrees of congestion and marked degeneration of the renal tubules, particularly proximal convoluted tubules as observed in group A animals are in conformity with the earlier reports related with clinical abuse of oxytetracycline (Lairmore et al. 1984). Renal excretion is the principal route of elimination for parenterally administered tetracyclines. Nephrotoxic properties of oxytetracycline are attributable in part to the inhibitory effect of the drug on the oxidative enzymes of renal tubular cells (Fox et al. 1976). Further, it would have been most appropriate to correlate the blood concentration of oxytetracycline with the induced tissue alterations. Blood values of drug were not estimated.

In this study such a high dose of antibiotic was selected on the basis of personal knowledge of its use, sometimes in the field by veterinarians in a bid to have quick responses though the information is not usually documented. However, hepatic and renal damages have been reported with high doses (33 mg/kg b wt) of oxytetracycline in the therapy of respiratory diseases in feedlot cattle (Griffin et al. 1979). The present study also showed mild hepatic degeneration in addition to renal tubular damage which could be attributed to the ability of drug to diffuse with highest concentration in the kidneys and liver (Huber 1982). Hepatocellular degeneration was also reported in man treated with tetracycline products (Combes et al. 1972) and calves treated with tetracycline containing degradation products (Teuscher et al. 1982). However, Lairmore et al. (1984) did not find any evidence of hepatic damage in oxytetracycline associated nephrotoxicosis in feedlot calves. The lesions observed in other vital organs could be attributed to secondary effects of toxæmia and uremia caused by severe renal tubular insufficiency.

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**REFERENCES**


