PREVALENCE AND PATHOLOGY OF POLIOENCEPHALOMALACIA IN GOATS

By

N. DIVAKARAN NAIR

THESIS
Submitted in partial fulfilment of the requirement for the degree

Doctor of Philosophy
Faculty of Veterinary and Animal Sciences
Kerala Agricultural University

CENTRE OF EXCELLENCE IN PATHOLOGY
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
MANNUTHY, THRISSUR
KERALA.

1999
CERTIFICATE

Certified that the thesis entitled "PREVALENCE AND PATHOLOGY OF POLIOENCEPHALOMALACIA IN GOATS" is a record of research work done independently by Shri. N. Divakaran Nair, under my guidance and supervision and that it has not previously formed the basis for the award of any degree, diploma, fellowship or associateship to him.

Mannuthy
6 - 10 - 1999

Dr. A. Rajan
(Chairman, Advisory Committee)
Dean (Retired)
College of Veterinary & Animal Sciences, Mannuthy
ABSTRACT

Polioencephalomalacia (PEM) is a significant emerging diseases problem in goats. Although, PEM in goats was recognized and reported as early as 1956, its etiology is poorly understood and symptomatology, pathological features and therapeutic approach have not been well defined and documented.

Hence an investigation was undertaken to assess the prevalence of the disease based on the data available from 76 Veterinary hospitals of the state for a period from 1991 to 1994. This data documented revealed an increasing trend in the occurrence of the disease and significantly high incidence was recorded in the first five months of which the peak incidence was observed in the month of April.

Spontaneous cases of the disease were studied in detail and experiments were conducted taking goat as a model using selected incriminating agents such as Amprolium (350 mg/kg body weight), Amprolium and rice gruel (350 mg/kg, and ad libitum rice gruel), rice gruel (ad libitum), sodium sulphate (150 mg/kg body weight followed by 500 mg/kg body weight on the 10th day) BHC (2.5 mg/kg followed by 5 mg/kg body weight on the 10th day) and Ficus tsiela Roxb. The experiment was for a period of 45 days.
Symptomatology, weight of the animals at fortnightly intervals, weight of the brain, CSF protein concentration, brain autofluorescence, gross and histopathological alterations of the brain and ultrastructural pathology were the markers utilized for evaluating the disease processes.

The sodium sulphate, BHC and Amprolium and rice gruel treated group showed symptoms and lesions more or less similar to the spontaneous cases. Only few animals in each group developed the disease such as two in amprolium and rice gruel treated group, four in BHC treated group and three in sodium sulphate group. This showed that individual idiosyncrasy plays an important role in the manifestation of the disease.

The symptoms developed at different latent periods were not progressive as compared to the spontaneous cases where the symptoms were progressive. The symptoms included lethargy, depression, knuckling at the fetlock, frequent tremors, opisthotonos and loss of eye preservation reflex. Blindness was seen in one of the natural cases.

The histological lesions of the brain in all the cases were comparable in different segments of the brain. Mostly it was characterized by diffuse laminar cortical degeneration and necrosis, occasional neuronal swelling,
The glial cell reaction and white matter vacuolation. Vascular changes predominated in the sodium sulphate group and also in the natural cases. There was glial cell response in the form of nodules in sodium sulphate group and natural cases. A predominant perivascular and neuropil accumulation of lymphocytes, gitter cells and monocytes were seen in the natural cases. These were considered as secondary deposition following a toxic degenerative neuropathy. The necrotic focus could well be delineated in few of these cases by the bluish or creamy autofluorescence of the affected brain, but was not found to be of any primary diagnostic value as all the affected brain did not show fluorescence.

Ultrastructural investigation revealed the basic reaction of the brain tissue to be similar in both the experimental and natural cases except for their intensity. Ultrastructural lesions were characterized by neuronal swelling, membrane lysis, segregation of the filamentous and granular component of nucleolus, cytoplasmic organellar damage such as fragmentation of RER, partial degranulation of ribosomes, mitochondrial swelling, cristolysis and complete disappearance of organelle. Neuropil spongiosis and splitting of myelin at the intraperiod line and formation of multiple vacuolations of the white matter were characteristic. From this observations it was clearly
delineated that the primary insult was a biochemical one which caused much damage to the volume control mechanism of the cell and subsequent cellular damage.

The CSF protein evaluation revealed high protein level in the spontaneous cases whereas in the experimental cases, the concentration remained within the normal range indicating that it has no diagnostic value.

In this investigation it has not been possible to induce PEM with Amprolium even at a dose rate of 350 mg/kg body weight and it was proved that amprolium is not a cause for PEM. Rice gruel ad libitum was found to be tolerated by the animal except one which showed dullness and abdominal distension towards the end of the experiment. Diffuse neuronal degeneration was observed in the brain of this animal. Based on this observation it was concluded that rice gruel consumption every day as a component of the concentrate feed in goats might not cause any detrimental effects and the problem comes only when it is fed in large quantities on a single day.

*Ficus tsiela* Roxb. though produced vascular damage and diffuse neuronal degeneration in one of the experimental animals, goats were found to be highly resistant to *Ficus tsiela* Roxb. toxicity and the variation in the
susceptibility of different species to this toxicity was brought to light.

From this investigation it was also clarified that sodium sulphate, BHC, rice, and amprolium could be initiating agents of PEM under certain circumstances and no single cause seems to be responsible for PEM.