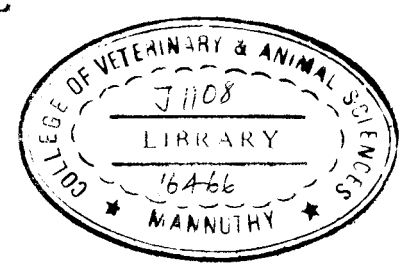
CLINICAL PROPAEDEUTICS AND RUMEN FLUID CHANGES IN POLIOENCEPHALOMALACIA OF GOATS

By

PAME T. MALIEKAL



THESIS

Submitted in partial fulfilment of the requirement for the degree of

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences
Kerala Agricultural University

Department of Clinical Medicine
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
MANNUTHY, THRISSUR - 680651
KERALA, INDIA

2000

DECLARATION

I hereby declare that the thesis entitled **"CLINICAL PROPAEDEUTICS AND RUMEN FLUID CHANGES IN POLIOENCEPHALOMALACIA OF GOATS"** is a bonafide record of research work done by me during the course of research and that this thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title, of any other University or Society.

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CERTIFICATE

Certified that this thesis, entitled **"CLINICAL PROPAEDEUTICS AND RUMEN FLUID CHANGES IN POLIOENCEPHALOMALACIA OF GOATS"** is a record of research work done independently by **Smt. Pame T. Maliekal**, under my guidance and supervision and that it has not previously formed the basis for the award of any degree, diploma, associateship or fellowship to her.

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ACKNOWLEDGEMENTS

I wish to express my deep sense of gratitude to ***Dr. P.C. Alex****, Associate professor and Head, Department of Clinical Medicine for his scholarly advice, never ending enthusiasm, valuable suggestions, constructive criticism and long suffering in the pursuit of this work as the chairman of my Advisory Committee. It has been a privilege to work under his priceless guidance.*

I am greatly indebted to ***Dr. P.G. Baby****, Associate Professor, Department of Clinical Medicine for his expert advice , incessant encouragement, creative suggestions and continued interest shown during the study.*

I express my profound gratitude to ***Dr. S. Ajithkumar****, Assistant Professor, Department of Clinical Medicine and* ***Dr. A.D. Mercy****, Associate Professor, Department of Nutrition for their scrupulous navigation, enduring interest and valuable discussion during the execution of this work.*

I am indebted to ***Dr. (Mrs). Usha Narayana Pillai*** *for her constant support throughout the work.*

I owe my thanks to ***Dr. K. M. Jayakumar****, Associate Professor,* ***Dr. R. Vijayan*** *and* ***Dr. (Mrs) Premni Aleyas****, Assistant Professor, Department of Clinical Medicine for their constructive suggestions and intimate help.*

I gratefully acknowledge ***Smt. Narayanikutty****, Professor and Head, Department of Statistics,* ***Smt. Mercy*** *and* ***Mr. K.V. Prasdan****, Technical Assistants, Department of statistics for their help in analysis of the data.*

I am thankful to ***Mr. K.L. Jose****, Farm Supervisor,* ***Mr. Chandran, K.R.****, Farm Assistant and* ***Mr. M. Gangadharan****, Department of Pathology.*

The timely help extended by ***Ramani, Mohanan, Sobhana*** *and* ***Ratnam*** *are gratefully acknowledged.*

I am grateful to ***Dr. Karthiyani,*** *and* ***Shibu, K.V.****, Department of Physiology for their help and assistance.*

I am thankful to the ***Dean****, College of Veterinary and Animal Sciences, Mannuthy for the facilities provided for this research work.*

With great fondness, I express my heartfelt thanks to my colleagues ***Dr. Anil J. Thachil, Dr. Jayakrishnan, T.N., Dr. Vinu David, Dr. Arun Raphel., Dr. Raveendran.P*** *and* ***Dr. Venkitesh kumaar*** *for their help and valuable assistance.*

I sincerely thank other ***staff members*** *of the Department of Clinical Medicine for their whole hearted co-operation and sincere help throughout the work.*

I am grateful to the Kerala Agricultural University for awarding me the fellowship for the postgraduate study.

I extend my gratefulness to ***Dr. C.V. Sreeranjitkumar****, Research Associate, Department of Microbiology for his timely help.*

Last but not least, I am indebted to the Almighty for his blessings, my father who insisted me to take up this task, my mother who motivated me throughout the period of study and my husband who encouraged me throughout the work.

Pame T. Maliekal.

DEDICATED TO

MY PARENTS, BROTHER AND

HUSBAND

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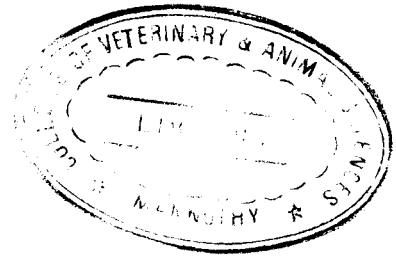

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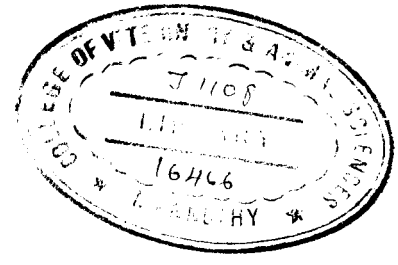

LIST OF ABBREVIATIONS

PEM	-	Polioencephalomalacia
CCN	-	Cerebrocortical necrosis
Hb	-	Haemoglobin
ESR	-	Erythrocyte sedimentation rate
PCV	-	Packed cell volume
TEC	-	Total erythrocyte count
TLC	-	Total leukocyte count
DLC	-	Differential leukocyte count
SAT	-	Sedimentation activity time
MBRT	-	Methylene blue reduction time
TVFA	-	Total volatile fatty acid
min	-	minute

INTRODUCTION

1. INTRODUCTION

The livestock sector contributes significantly to the economy of our country. Goat rearing is an important source of income particularly to marginal and small farmers as well as landless agricultural labourers. Goats are popularly known as "poorman's Cow".

In India, goat population increased from 47.08 million in 1951 to 110.21 million in 1987 (Sastri,1995). This accounts for twenty percent of the total world population and stands first in the world. Nearly thirty five percent of the total meat production in India is from goats (Chopra, 1992).

In Kerala, there are 1.58 million goats as per the livestock census 1987. As 90 percent of the population in Kerala are non-vegetarian, there is tremendous scope for goat rearing for meat purpose.

The goats too suffer from many infectious and non-infectious diseases. Nutritional deficiency / insufficiency disorders constitute a significant

proportion of non-infectious diseases. Intensive domestication has made the goat more vulnerable to deficiency diseases. Polioencephalomalacia (PEM) is a very common disease among goats in Kerala and majority of the cases are thiamine responsive.

The term Polioencephalomalacia literally means, softening (malacia) of the grey matter (polio) of the brain (encephalo). It is a condition characterized clinically by head pressing, opisthotonus, nystagmus, strabismus and convulsive seizures. Since the condition affects cerebral cortex the condition will become irreversible, if not treated in the early stages. Early stage of the disease generally responds to thiamine therapy.

As studies on clinical cases of Polioencephalomalacia are very few, there is only limited information about the clinico-pathological changes in PEM cases. A thorough knowledge about the epidemiology, clinical signs and pathogenesis are very much essential for the successful therapeutic management and control of the condition. Therefore a study on thiamine responsive PEM was proposed with the following objectives.

1. To study the occurrence, epidemiology and clinical signs.
2. To study the changes in the rumen fluid
3. To study the haematology and biochemical changes in blood.

REVIEW OF LITERATURE

2. REVIEW OF LITERATURE

Polioencephalomalacia occurred in young cattle, sheep, goat, deer etc. and was characterised by sudden onset of blindness, head pressing, opisthotonus and convulsions, a rapid response to thiamin therapy in early stages and pathologically by acute cerebral oedema and laminar necrosis of the cerebral cortex (Radostits *et al.*, 1994).

2.1 Etiology

Strains of *Clostridium sporogenes*, which produced thiaminase type I, were isolated from animals affected with PEM. Alimentary thiaminases might be produced by either *Clostridia* or members of the genus *Bacillus* (Morgan and Lawson, 1974).

According to Brent (1976), rumen acidosis established rumen conditions conducive for the development of cerebrocortical necrosis due to the decreased pH which was optimum for thiaminase activity or by proliferation of thiaminase producing bacteria.

High thiaminase activity in the rumen of CCN affected animals could degrade all the thiamin usually ingested and synthesized (Boyd and Walton, 1977).

In ruminants, both organic and inorganic Sulphur compounds were reduced to sulfides by rumen bacteria. Ruminants did not require vitamin B_1 in their diet as it was synthesized in the rumen by the microbes. Inadequate microbial synthesis, impaired absorption and utilization of vitamin B_1, presence of vitamin B_1 antimetabolites, increased demand for vitamin B_1 or increased rate of B_1 excretion, all these could lead to thiamin inadequacy. Diets high in sulphur decreased the amount of vitamin B_1, entering the duodenum probably due to reduced synthesis of vitamin B_1. (Gooneratne *et al.*, 1989).

Thiamin deficiencies were recorded in sheep exported live by sea. The high sheep density, competitive feeding condition and high carbohydrate diet caused proliferation of thiaminase producing bacteria (Thomas *et al.*, 1990).

Amprolium was found to antagonise thiamin at absorption level in intestine by interfering with

phosphorelation of thiamin and amprolium was used to induce PEM (Lonkar and Prasad , 1992b)

Polioencephalomalacia was reported in cattle consuming water with elevated sodium sulphate levels (Hamlen *et al.*, 1993)

Polioencephalomalacia associated with ingestion of ammonium sulfate was recorded in sheep and cattle (Jeffrey *et al.*, 1994).

2.2 Epidemiology

2.2.1 Species

Polioencephalomalacia was reported in sheep and cattle (Jensen *et al.*, 1956 and Terlecki and Markson, 1958), horses (Cymbaluk *et al.*, 1978), goats (Smith,1979), white tailed deer (Wobester and Runge,1979), antelope (Blood *et al.*, 1979) and dogs (Breud and Vendevelda,1979).

2.2.2. Incidence

Morbidity rate in sheep and cattle were generally less than 10 per cent and mortality rate in affected animals approached 100 per cent (Spence *et al.*, 1961).

PEM in goats was first reported in India by Tanwar (1987) from the arid zone of Rajasthan. Subsequently the condition was reproduced experimentally in goats (Fakhruddin *et al.*, 1987a).

Nair (1999) conducted a detailed investigation on PEM of goats in Kerala and reported the following. The disease occurred throughout the year with maximum incidence in the month of April followed by May, March and February. Reduced incidence in January, August, September and October.

District-wise distribution indicated maximum incidence in Malappuram (14%) followed by Ernakulam (13%), Kottayam (10%) and Kozhikode (10%).

2.2.3. Age

In cattle, animals of 9-12 months were mostly affected (Blood *et al.*, 1979). The age group commonly affected were 2 months to 2.5 years, in goats 3 weeks to 5 years in sheep and 3 weeks to 8 years in cattle (Smith, 1979). In a study conducted by Lonkar *et al.* (1993) the incidence was mostly in adult goats as against the findings of Fakhruddin *et al.* (1987a).

2.2.4.Sex

Incidence of PEM in sheep was 38 per cent in males and 62 per cent in females (Jensen *et al.*, 1956). Most of the affected animals were females, but this could be taken as a reflection of a sex bias, because male goats were either disposed off or slaughtered at an early age and females were retained for breeding and milk production (Sobhanan,1981; Lonkar *et al.*, 1993).

2.2.5. Diet

In Kerala, rice (a carbohydrate rich diet) formed the main component of concentrate ration. It would lead to lowering of rumen p^H and possibly creation of a favourable environment for increased thiaminase activity (Sobhanan, 1981). Lowering of pH increased the activity of potential thiaminase producers and histamine release, which could act as a co-substrate for thiaminase activity (Smith,1979).

2.3 Pathogenesis

Thiamin pyrophosphate is a co-enzyme for the decarboxylation of not only pyruvate but also glyoxylate, 2-oxobutyrate, 2-oxoisovalerate, 2-oxoisocaproate, 2-oxo β-methylvalerate, 2-hydroxy

pyruvate, 2-oxoglutarate and 2-oxoadipate (Edwin and Jackman, 1981).

Thiamin is necessary for the production of thiamin diphosphate, a coenzyme which played a role in the activation of the transketolase. Transketolase found in the glial cells and erythrocytes, is an important enzyme involved in the glucose metabolism. Since brain is glucose dependent, glial cell transketolase played an important role in brain metabolism (Hamlen *et al.*, 1993).

Thiamin status of an animal would be dependent on the dietary thiamin intake, thiamin synthesis, presence of thiaminase in the rumen and the effects of antimetabolites (Radostits *et al.*, 1994).

Deficiency of thiamin resulted in increased blood concentration of pyruvate, reduction in lactate to pyruvate ratio and depression of erythrocyte transketolase. Brain has a greater dependence on the pentose phosphate pathway for glucose metabolism, in which transketolase was a rate limiting enzyme. Oedema of intracellular compartment principally involving astrocytes and satellite cells, has been suggested to

be due to a reduction in the ATP production following a defect in carbohydrate metabolism in the astrocyte. Such changes decreased the activities of ATP-dependent sodium and water transport in the neurons and resulted in intra neuronal swelling (Radostits *et al.*, 1994).

The toxic effect of excess sulphur was unique in ruminants. The brain was the target organ. Sulphite derived radicals could cause lipid peroxidaton and damage to brain tissues. It has been shown that a deficit of thiamin increased the intensity of lipid peroxidation. Free thiamin was thought to protect the cell by scavenging potentially toxic intermediates generated by the myeloperoxidase hydrogen peroxide halide system (Olkowski *et al.*, 1992).

Sulfide inhibited cellular respiration leading to hypoxia and it might be sufficient to create neuronal necrosis in polioencephalomalacia (Radostits *et al.*, 1994).

2.4. Clinical signs

Polioencephalomalacia was a degenerative disease of brain, seen in feedlot or housed cattle and sheep

and to a lesser extent in pastured animals (Markson *et al.*, 1974).

Staggering gait, excessive salivation and normal rumen motility and consistency were reported in an affected dairy cow (Colontino and Bulmer , 1977).

Bradycardia, dropped heart beats, ataxia, muscular fasciculation, periodic hypothermia, blindness, diarrhoea and weight loss were noted in amprolium induced thiamin deficiency in horses (Cymbaluk *et al.*, 1978).

Champing of jaws, frothy salivation, head pressing, irritation signs, vertical or horizontal nystagmus and opisthotonus were reported by Blood *et al.* (1979).Depression, ruminal atony, anorexia, nystagmus, lateral or sternal recumbency were reported in range cattle (Dickie *et al.*, 1979). Wobester and Runge (1979) reported clinical signs similar to PEM in ruminants, like wild deer also. According to Smith (1979) early signs included excitability and elevation of head which might be interpreted as increased nervousness.

Blindness, ophisthotonus, extensor rigidity and nystagmus were noted later in goats (Maxwell, 1980) and sheep (Chick *et al.*, 1981).

Excitement, bleating followed by low carriage of head, drooping of ears, leaning on to walls or obstacles, aimless wandering, anorexia, weakness, ataxia, horizontal nystagmus, increased respiratory rates, tonic-clonic convulsions, opisthotonus, star-gazing posture and self inflicted injuries to the eye and superficial organs during convulsions were reported (Sobhanan, 1981).

Tonic- clonic spasms, opisthotonus, nystagmus and grinding of teeth were observed in lambs of one to ten weeks of age (Mc Donald, 1982). Champing of jaws, lateral recumbency, paddling movements and bleating were reported in affected goats (Tanwar *et al.*, 1983).

Affected sheep after a brief period of dullness wandered aimlessly in wide circles. They became ataxic and collapsed with intermittent convulsions. Hyperaesthesia, nystagmus, opisthotonus and trismus were evident with coma and death shortly afterwards. These clinical signs were fairly typical of CCN but

were not pathognomonic and in some cases resembled lead poisoning, bacterial meningitis, encephalic listeriosis, coenurosis, aflatoxicosis and hypomagnesemia (Jackman, 1985; Rammell and Hill, 1986).

The neck, forelegs and hindlegs were stiff. Lateral recumbency with marked opisthotonus and sometimes locked jaw condition were noted in the affected animals. Grinding of teeth, frothy salivation, twitching of ears and eyelids and convulsions were noted. Hyperaesthesia and bilateral nystagmus were also observed (Tanwar, 1987). Dragging gait, reluctance to move, absence of menace responses, dyspnoea, laboured breathing and normal pupillary response were noted (Fakhruddin *et al.*, 1987a).

Sudden falling on ground and when raised the goats stood keeping hind legs wide apart for maintenance of balance of body. Drooling of saliva, retention of faeces and urine, slight diarrhoea, opisthotonus, paddling and convulsions were noted. Convulsing animals shifted the site in circles on ground in lying down position. Bulging of eyes, temporary blindness, nystagmus, absence of menace reflex, presence of

pupillary reflex, cyanosis, dyspnoea and laboured breathing were recorded(Gauri and Vashistha, 1988).

The clinical signs reported in sheep were blindness and a tendency to press head against solid objects or obstacles and cessation of feeding (Jubb and Huxtable, 1991).

Anorexia, depression, head pressing, blindness, twitching of facial muscles, convulsions, stiffness of body, paddling, grinding, dyspnoea and death were noted. Clinical course lasted for 4 to 10 days. In later stages, bilateral corneal opacity with cutaneous injury around the eyes was noted (Lonkar and Prasad, 1992a).

Since amprolium was found in the brain tissue in the experimentally induced disease it was called as "amprolium poisoning encephalopathy" (Radostits *et al.*, 1994).

Tanwar and Malik (1995) reported the following clinical signs in amprolium induced PEM in buffalo calves. Lacrimation, depression, dullness, anorexia, rigid stance, raising of head, standing motionless for

several hours, muscle tremors and abnormal posture and gait. Rumination was present inspite of anorexia. Abnormalities of posture and gait were characterized by ataxia, kyphosis and abduction of limbs. Muscular weakness, blindness, lateral recumbency and tonic-clonic convulsions were noted. Frequent irritation, absence of menace reflex and presence of pupillary light reflex were noted. Circling movements, aimless wandering and torticollis in some calves.

Pulse and respiratory rates were normal except in some cases with laboured breathing (Tanwar *et al.*, 1983; Tanwar, 1987; Fakhruddin *et al.*, 1987a). Pulse was weak and irregular (Sobhanan, 1981). Temperature was usually normal but elevated in extreme convulsive phase only (Sobhanan, 1981; Fakhruddin *et al.*, 1987a). Temperature of 38 to 40^0 C was reported by Tanwar *et al.* (1983). Visible mucous membranes were congested (Sobhanan, 1981; Tanwar, 1987).

2.5. Rumen fluid

2.5.1. Physical characters

2.5.1.1. pH

pH of rumen fluid in healthy goats was found to be 6.63 ± 0.08 (Pillai, 1988), 7.08 ± 0.06 (Lal *et al.*,

1990) 6.88 ± 0.065 (Bask *et al.*, 1993a) and 6.32 ± 0.03 (Rai and Pandey,1980). A mean pH value of 5.90 ± 0.12 and lower value of 5.68 were observed at two hours after feeding by Nanjappan and Govinda Rao (1984). A pH value of 6.5 to 7.5 by Choudhari *et al.* (1985), 6.4 to 7.4 (Pradhan *et al.*, 1988) 6 to 7 (Mohan *et al.*, 1992) and 6.93 ± 0.27 to 7.45 ± 0.15 (Shihabudheen, 1998) were also reported in normal goats.

Tanwar (1987) and Lonkar and Prasad (1994) reported that the pH of rumen liquor did not show any significant difference between control and PEM induced group.

2.5.1.2. Colour

Normal colour of rumen liquor in goats varied from grey and olive to brownish green (Dirksen, 1979), greenish yellow (Pillai, 1988) and olive green (Shihabudheen, 1998).

The colour of rumen liquor in healthy sheep was green to greenish brown (Pradhan *et al.*, 1988) and brown to green (Mohan *et al.*, 1992).

Gray discolouration of ruminal fluid was reported by McAllister *et al.* (1997) in sulphur induced PEM.

2.5.1.3. Consistency

Thick consistency was reported by Pillai (1988) and Shihabudheen (1998), semiliquid (Pradhan *et al.*, 1988) and viscus (Mohan *et al.*, 1992) in normal goats.

2.5.1.4. Odour

Rumen fluid from healthy animals had an aromatic odour and this depended on the nature of rumen contents (Sankaranarayan and Nambiar, 1972; Dirksen, 1979; Pillai, 1988; Mohan *et al.*, 1992; Shihabudheen, 1998). Aromatic and goatish odour was recorded by Pradhan *et al.* (1988).

2.5.2. Microbial activity

2.5.2.1. Protozoal activity

The average protozoan activity ranged from moderate (++) to vigorous (+++) depending on the interval between the time of rumen fluid collection and the last feeding (Misra *et al.*, 1972). Dirksen (1979) reported abundant protozoa (+++) in normal rumen

liquor. Protozoal activity of (+++) was recorded by Shihabudheen (1998) in healthy goats.

2.5.2.2. Iodophilic activity

Iodophilic activity of protozoa could be recognised by black colouration of starch contents in the protozoa by the addition of Lugol's iodine. It could be graded as o, +, ++, +++ depending on quantity of starch contained (Srinivasan and Gnanaprakasam, 1990).

2.5.2.3. Protozoal count.

Protozoal concentration of 25.66 ± 1.75 x 10^4/ ml strained rumen liquor (SRL) was reported by Rai *et al.*(1972) in goats. A range of 1.66 x 10^5/ ml to 2.56 x 10^5/ ml were noted in healthy steers (Sankaranarayanan and Nambiar, 1972). Total protozoal count of 13.84 ± 0.77 x 10^5 per ml rumen fluid was recorded by Nanjappan and Govinda Rao (1984). Choudhari *et al.* (1985) reported the protozoal count to range from 1.11 to 7.2 x 10^5/ ml of rumen liquor in healthy goats.

2.5.2.4. Sedimentation activity time (SAT)

Nicholas and Penn (1958) reported that determination of sedimentation activity time (SAT) was useful to measure the rumen microbial activity. Sedimentation activity time of 8 to 18 minutes with an average of 12.8 minutes were noted in Indian cattle (Misra *et al.,* 1972). Dirksen (1979) observed SAT in cattle as 4 to 8 minutes. In goats, up to 22.16 ± 3.76 minutes were recorded (Sen, 1982). Pillai (1988) recorded it as 12.5 ± 0.61 minutes and 13.83 ± 0.75 minutes by Shihabudheen (1998) in goats.

2.5.2.5 Methylene blue reduction time (MBRT)

Methylene blue reduction time in healthy cattle was found to be 3 to 6 minutes by Dirksen (1979). Pradhan *et al.* (1988) recorded 10.25 minutes. Bask *et al.* (1993a) found that MBRT was 11.48 ± 0.45 minutes in healthy goats. A range of 3.37 ± 0.21 was noted by Shihabudheen (1998).

2.5.3. Total Volatile Fatty Acids (TVFA)

Sankaranaryanan and Nambiar (1972) observed a range of 60.70 mM/l to 99.08 mM/l in healthy Steers. Rai *et al.* (1972) reported daily mean and peak TVFA values of 72.08 ± 1.8 and 91.96 ± 7.44 mEq/l rumen liquor respectively in goats. The peak concentration observed at two hours after feeding was due to ample availability of nutrients and maximum fermentation activity during this period.

Total volatile fatty acid levels in different feed groups were detected as follows: Range concentrate group 71.37 ± 3.79 mEq/l, stall fed group 90.86 ± 1.907 mEq/l and range group 52.06 ± 2.025 mEq/l. (Rai and Pandey, 1980). Mean rumen TVFA of 108.30 ± 6.06 mM/l and a peak concentration of 130 mM/l, two hours after feeding were observed (Nanjappan and Govinda Rao, 1984).

2.5.4. Total acidity

In cattle, total acidity of rumen fluid was normally 8 to 25 units. It may reach 70 units in hyperacidity (Dirksen, 1979).

2.5.5. Lactate

Lactate level of rumen liquor in normal goats ranged from 3 to 4.5 mg per cent (Verma *et al.*, 1975). Lactate level of 21.2 ± 2.08 mg per cent was reported in rumen liquor in simple indigestion in goats (Vihan and Rai, 1985). Increased level of lactate was recorded in nutritionally induced PEM in calves by Sager *et al.* (1990).

2.5.6. Ammonia

Rai *et al.* (1972) found the daily mean and peak value as 31.64 ± 1.87 and 60.12 ± 2.25 mg/100ml rumen liquor respectively in goats. The change in the concentration due to time of sampling was also significant; the peak was observed at two hours after concentrate feeding and the concentrations thereafter declined and by sixth hour they were lowest.

Prasad *et al.* (1972) reported decreased rumen NH_3-N levels in cattle and buffaloes suffering from indigestion.

Rai and Pandey (1980) revealed that the rumen NH_3-N values in goats ranged from 22.50 ± 0.905, 18.52 ±

1.628 and 12.98 ± 0.563 mg precent respectively in range concentrate, stall fed and range groups.

2.6. Haematology

2.6.1. Haemoglobin

The haemoglobin level of apparently healthy goats was reported to be 10.09 g/dl (Bhargava, 1980), 9.98 ± 0.56 g/dl (Pyne *et al.*, 1982), 8 to 14 g/dl (Benjamin, 1985), 8.76 ± 0.32 g/ dl (Bask *et al.*, 1993b), 8 to 12 mg/ dl by Radostits *et al.* (1994), and 12.77 ± 0.54 g/dl (Shihabudheen, 1998) and 8.9 ± 0.23 g/dl (Gauri and Vashistha, 1988).

Haemoglobin level was reported to be normal (Pass, 1968) in an outbreak of PEM in calves. Normal haematological values were noted in PEM of goats (Tanwar, 1987). Fakhruddin *et al.* (1987a) noted a decrease in haemoglobin content from 8.5 ± 0.55 to 7.4 ± 0.90 g per cent in goats. Almost normal haemogram was reported by Fakhruddin *et al.* (1987b) in PEM of cattle. Haemoglobin level of 9.6 ± 0.98 g per cent was reported by Gauri and Vashistha (1988) in thiamin deficiency CCN in goats. A sudden increase in haemoglobin level from 6.7 g per cent to 10.5 g per cent was noted at the

onset of clinical symptoms of PEM (Lonkar and Prasad, 1992a).

2.6.2. Haematocrit (Packed cell volume-PCV)

Normal haematocrit in healthy goats was reported as 24 to 48 per cent (Benjamin, 1985), 34.55 ±0.565 (Lal *et al.*, 1990), 34.01 ± 0.68 per cent (Das and Misra, 1991), 22 to 38 per cent (Radostits *et al.*, 1994) and 29.83 ± 0.95 per cent (Shihabudheen, 1998).

A normal haematocrit value was recorded by Pass (1968) in an outbreak of PEM in calves. An increase in PCV was noted in sheep and cattle suffering from PEM (Spence *et al.*, 1961). Packed cell volume of 24 to 32 per cent was reported by Tanwar (1987). A decrease in PCV from 31.57 ± 1.47 to 25.85 ± 1.79 per cent was noted (Fakhruddin *et al.*, 1987a). PCV of 33.6 ± 4.14 per cent was recorded in clinical cases of PEM in goats (Gauri and Vashistha, 1988). A range of 15.7 per cent to 21.6 per cent was reported by Lonkar and Prasad (1992a) in induced CCN in goats.

2.6.3. Erythrocyte sedimentation rate (ESR)

Benjamin (1985) reported normal ESR in goats as 2 to 2.5 mm in 24 hours. Shihabudheen (1998) recorded normal ESR in goats as 5.2 ± 0.40 to 8.00 ± 1.70 mm in 24 hours.

2.6.4. Total erythrocyte count

Erythrocyte count in normal goat was reported as 10.96 x 10^6/ mm^3 (Sharma *et al.*, 1973), 10.12 x 10^6/ µl (Bhargava, 1980), 12.35 ± 0.35 x 10^6/ µl (Bask *et al.*, 1993b) and 8 to 18.0 x 10^6/ µl (Radostits *et al.*, 1994) and 14.70 ± 0.44 x 10^6/µl (Shihabudheen, 1998).

A decrease in the number of erythrocyte from initial mean value of 13.62 ±1.06 to 10.84 ± 0.98 million/ mm^3 was reported by Fakhruddin *et al.* (1987a) in amprolium induced PEM of goats. Almost normal values were reported by Fakhruddin *et al.* (1987b) in PEM of cattle. Total erythrocyte count of 14.1 ± 0.87 million / cmm was recorded by Gauri and Vashistha (1988).

2.6.5. Total leukocyte count (TLC)

Total leukocyte count in healthy goat was reported to be 12.5 x 10^3/ µl (Sharma *et al.*,1973), 10.09x10^3/ µl

(Bhargava, 1980), 10.18 ± 0.24 x 10^3/ µl (Pyne *et al.*, 1982), 4 to 13 x 10^3/ µl (Radostits *et al.*, 1994) and 13.51 ± 0.55 x 10^3/ µl (Shihabudheen, 1998).

A TLC of 12.20 to 17.25 thousands/ mm^3 was reported by Tanwar (1987) and a slight increase in leukocyte count from 10,139.57 ± 495.91 to 11,453.57 ± 698.84 /cmm was detected by Fakhruddin *et al.* (1987a) in PEM affected goats. Gauri and Vashistha (1988) reported TLC of 11683 ± 1044 / cmm in a clinical study of CCN of goats. In amprolium induced PEM of goats total leukocyte count of 11,900/ cmm to 13,200/ cmm was reported by Lonkar and Prasad (1992a).

2.6.6. Differential leukocyte count. (DLC)

Pyne *et al.* (1982) reported DLC in healthy goat as neutrophils 34.61 ± 0.65 per cent, lymphocytes 60.25 ± 0.14 per cent, monocytes 3.06 ± 0.16 per cent, eosinophils 2.04 ± 0.14 per cent and basophils 0.04 ± 0.02 per cent. According to Benjamin (1985) the values were 30-48 per cent, 50 - 70 per cent, 1-4 per cent, 3-8 per cent and 0 - 2 per cent respectively. According to Shihabudheen (1998) neutrophils formed 35.67 ± 1.82

per cent, lymphocytes 59.83 ± 1.87 per cent, eosinophils 2.50 ± 0.56 per cent, monocytes 0.83 ± 0.31 per cent and basophils 0.33 ± 0.21 per cent of the total leukocytes.

A definite neutrophilia was recorded in CCN of sheep and cattle (Spence *et al.*, 1961). A normal DLC was reported by Pass (1968) in an outbreak of PEM in calves. Neutrophils 21-40 per cent, lymphocytes 57- 73 per cent, monocytes 1- 3 per cent, eosinophils 0 -2 per cent were recorded by Tanwar (1987) in goats with PEM. An increase in neutrophils from 42.0 ± 1.69 to 51.55 ± 2.29 per cent in PEM of goats from onset of disease till recovery was recorded by Fakhruddin *et al.* (1987a). Gauri and Vashistha (1988) reported a DLC with lymphocytes- 41.9 ± 3.98 per cent, monocytes - 0.6 ± 0.80 per cent, neutrophils- 56.4 ± 3.81 per cent and eosinophils - 0.7 ± 0.99 per cent in PEM affected goats.

2.7 Serum biochemistry

2.7.1 Glucose

Varma (1967) reported normal blood glucose level in healthy goats as 54.66 ± 2.70 mg per cent. Blood

glucose level in healthy goats was reported to be 52.39 ± 0.92 mg/ dl (Pillai, 1988), 59.66 ± 0.98 mg/dl (Lal *et al.*, 1990), 48.24 ± 1.7 mg/ dl (Das and Misra, 1991), 30 to 65 mg /dl (Radostits *et al.*, 1994) and 47.76 ± 10.23 mg/ dl (Shihabudheen, 1998).

A rise in blood glucose level to 127.5 mg/100 ml from the normal level of 50 to 80 mg per 100 ml was observed by Pass (1968) in an outbreak of PEM in calves. Elevation of blood glucose level was noted only when clinical signs were present in sheep (Spicer and Horton, 1981). A steep rise in the blood glucose level from 45.9 to 79.0 mg/ dl at the onset of clinical symptoms and subsequent decline to the pre-experimental (50 mg/ dl) level after recovery was recorded by Lonkar and Prasad (1993) in induced CCN in goats. Syamasundar and Malik (1993) recorded, significant elevation in blood glucose level at the onset of clinical signs (125. 95 ± 11.32 mg/ dl) in amprolium induced PEM in buffalo calves after 45 days of induction.

2.7.2Calcium

Serum calcium levels reported in healthy goats were 9.70 mg/ dl (Boss and Wanner, 1977), 9.05 ± 0.12

mg/ dl (Pyne *et al.*, 1982) and 9.15 ± 0.27mg/ dl (Shihabudheen, 1998). Normal calcium levels in sheep were reported as 5 to 7 mEq/l (Benjamin, 1985) and 11.5 to 13 mg/ dl (Radostits *et al.*, 1994).

In an outbreak of PEM in calves, normal calcium levels were reported by Spence *et al.* (1961) and Pass (1968). A similar finding was reported by Thornber *et al.* (1981) in induced thiamin deficiency in lambs. Plasma calcium concentrations remained within normal limits in all sheep throughout the course of sulphur induced PEM (Gooneratne *et al.*, 1989). Calcium levels were within normal levels (9.99± 0.69 mg/ dl) in amprolium induced PEM in buffalo calves (Syamasundar and Malik, 1993). Tanwar *et al.* (1994) reported no significant changes in serum calcium levels in induced PEM in buffalo calves.

2.7.3 Phosphorus

Phosphorus level in the serum of healthy goats were 6.91 mg/dl (Boss and Wanner, 1977) 4.53 ± 0.18 mg/dl (Pyne *et al.*, 1982) and 5.72 ± 0.44 mg/ dl (Shihabudheen, 1998).

Serum levels of phosphorus were normal in CCN of sheep and cattle (Spence *et al.*, 1961). Normal serum phosphorus levels were reported (5.24 ± 0.64 mg/dl) in amprolium induced PEM in buffalo calves (Syamasunder and Malik, 1993; Tanwar *et al.*, 1994).

2.7.4 Magnesium

Serum magnesium level in healthy goats was found to be 2.33 mg/dl (Boss and Wanner, 1977) and 2.13 ± 0.06 mg/dl (Pyne *et al.*, 1982), 2.33 ± 0.18 mg/ dl (Shihabudheen, 1998).

Normal magnesium level was recorded in an outbreak of PEM in calves (Pass, 1968), in sheep (Spence *et al.*, 1961) in lambs (Thornber *et al.*, 1981) and in buffalo (Syamasundar and Malik, 1993; Tanwar *et al.*, 1994). Plasma magnesium concentration of non-vitamin B_1 supplemented sheep was elevated at the 2.5 to 3 week and 6 week samplings, but they declined significantly to normal levels thereafter. Magnesium levels of vitamin B_1 supplemented sheep were low at the beginning, but progressively increased during the course of the experiment (Gooneratne *et al.*, 1989).

2.7.5 Total protein and Albumin

Normal total serum protein in goats ranged from 6.40 to 7.90 g per cent with albumin concentration of 2.7 to 3.90 g per cent and Albumin to Globulin ratio of 0.63 to 1.26 (Benjamin, 1985). The average total protein concentrtion in the plasma was found to be 7.78 ± 0.26g per cent (Varma, 1967). Pillai (1988) recorded total serum protein as 6.43 ± 0.062 g/ 100 ml, albumin as 3.20 ± 0.03 g/100 ml and A:G ratio as 1.014 ± 0.011 in healthy goats.

An insignificant fall in serum protein concentration from (7.37 ± 0.18 g/dl (before induction) to 6.20 ± 0.27 g/dl (after induction)) was recorded in amprolium induced PEM in buffalo calves (Syamasundar and Malik, 1993). An increase in the levels of total serum protein from 5.4 g / dl to 6.2 g / dl at the onset of clinical symptoms was reported by Lonkar and Prasad (1992a) in induced CCN in goats.

2.7.6 Lactate and pyruvate

The lactate concentration in the blood of healthy goat was reported to be 13.02 ± 1.02 mg per cent (Pillai , 1988), 10.72 ± 0.84 mg per cent (Das and

Misra, 1991), 9.27 ± 0.392 mg per cent (Lal *et al.*, 1990), 9.8 mg per cent (Lonkar and Prasad, 1993) and 16.78 ± 0.78 mg/ dl (Shihabudheen, 1998).

Blood pyruvic acid content in healthy goat was recorded to be 1.1 mg per cent (Lonkar and Prasad, 1993) and 2.12 ± 0.05 mg/ dl (Shihabudheen, 1998).

Lactate to pyruvate ratio of 6.17 ± 0.42 was recorded by Shihabudheen (1998) in healthy goats.

Pyruvate and lactate levels showed marked variations and pyruvate levels were elevated in some cases after the appearance of clinical signs but showed wide variations from sheep to sheep. Lactate values showed marked variations throughout the trial (Spicer and Horton, 1981). Blood lactate level ranged from 5.25 to 11.06 mmole /litre and the blood pyruvate level from 0.08 to 0.26 mmole / litre (Chick *et al.*, 1981). In amprolium induced PEM in buffalo calves, significant elevation in blood lactate and blood pyruvate levels were noted. Blood lactate levels of 47.75 ± 5.60 mg/dl, blood pyruvate level of 2.56 ± 0.12 mg/dl and a lactate to pyruvate ratio of 20.53 ± 1.36 were recorded by

Syamasundar and Malik (1993). An increase in the lactate (9.8 to 28.1 mg per cent) and pyruvic acid levels (1.1 to 3.5 mg per cent) and decrease in lactate / pyruvate ratio (9.1 to 8.5) were observed at the onset of clinical symptoms in induced CCN in goats. The levels of lactate (14.4 mg per cent) and pyruvic acid (1.2 mg per cent) became normal on recovery (Lonkar and Prasad, 1993)

MATERIALS AND METHODS

3. MATERIALS AND METHODS

Ten clinical cases with symptoms suggestive of Polioencephalomalacia which were brought to the Veterinary College Hospital, Mannuthy were selected. The cases were selected after detailed clinical examination. Characteristic clinical signs of PEM were present in all the selected cases. All the selected cases were treated with thiamine hydrochloride at a dose rate of 10 mg per Kg body weight, five injections intravenously at three hours intervals. From second day onwards thiamine was given at a dose rate of 10 mg/Kg body weight half of the dose given intravenously and half intramuscularly, once daily. This was continued till complete recovery/ death of the animal. Only six cases that responded to thiamine therapy were included in this study.

3.1. Parameters studied:

A complete history was collected from the owners of each case. The present and past history, feeding practices, management practices, patient data,

duration of the disease, predisposing causes etc. were recorded as per the Proforma given in Appendix.

Data on the occurrence of the disease during 1998 and 1999 were collected from the records maintained in the College Hospital.

The samples of blood and rumen fluid were collected before starting the therapy. Rumen fluid and blood samples were collected and analysed for certain selected parameters after the course of treatment to evaluate the changes in clinical pathology). Samples collected from six apparently healthy goats maintained under similar conditions served as controls.

3.1.1. Rumen fluid

Rumen fluid was collected with the help of rumen fluid extraction apparatus. The collected rumen liquor was strained through a four folded cheese cloth and all the selected parameters were estimated immediately.

3.1.1a Physical characters

pH of the rumen fluid was recorded immediately after collection with strips of wide range pH paper.

Physical characters of rumen fluid like colour, odour and consistency were assessed as per the method of Misra and Tripathy (1963).

3.1.1b Microbial activity

Assessment of protozoal activity was done as per the method described by Misra *et al.* (1972). Sedimentation activity time was detected soon after collection of rumen fluid as per the method of Nicholas and Penn (1958). Methylene blue reduction test was done as per the method of Dirksen (1979). Iodophilic activity was determined as per Alonso (1979).

Total protozoal count was done as described by Sankaranarayanan and Nambiar (1972). Five millilitre of the strained rumen fluid was taken and the volume was made upto twenty five ml by adding ten percent formal saline (10% formaldehyde v/v in 0.35% sodium chloride). Formalin checked further bacterial and protozoal activity. Ten ml was taken from the above and stained by adding 10 drops of two percent eosin. It was allowed to set for 5 to 10 minutes for the protozoa to take up the stain. A drop of the fluid was charged in haemocytometer with Neubauer ruling and the

total number of protozoa in eight chambers were counted.

Total volatile fatty acid level was estimated as per the method of Barnett and Reid (1957). One ml of the ruminal fluid was taken in Markham apparatus followed by one ml of oxalic acid- potassium oxalate buffer (0.5 ml of 10% potassium oxalate and 0.5 ml of 5% oxalic acid). The mixture was steam distilled. About 80 ml of the distillate was collected and titrated against 0.01N sodium hydroxide using phenolphthalein indicator. The value obtained was multiplied by a factor 10 to get the total volatile fatty acids in mEq/l of the sample.

Total acidity was detected as per Dirksen (1979). Lactate content was detected as per the method of Noll (1974) using supernatent of rumen liquor after centrifuging at 3000 rpm. Ammonia levels were estimated as per the method of Conway (1957).

3.1.2 Blood

Approximately five ml of blood was collected from jugular vein in a dry glass vial, with EDTA (1-2 mg/ml) as anticoagulant for haematology. Ten

millilitre was collected separately in a glass stoppered centrifuge tube for separation of serum.

Haematology was done as per the procedure described by Schalm *et al.* (1975).

3.1.3 Serum

Glucose was estimated as per Trinder (1969), phosphorus as per Zilversmit *et al.* (1950), calcium as per Sarkar and Chauhan (1967) and magnesium was estimated using atomic absorption spectrophotometer (Perkin-Elmer model, 3380).

Total protein level was detected using biuret method described by Weichselbaum (1946) and albumin by the method explained by Doumas *et al.* (1971).

Lactate was estimated as per the method of Noll (1974) and pyruvate level detected as per Czhok and Lamprecht (1974).

3.2 Statistical analysis.

Statistical analysis was conducted according to the method described by Snedecor and Cochran (1980).

RESULTS

4. RESULTS

Ten clinical cases which exhibited signs suggestive of PEM were subjected to detailed clinical examination. Four animals were in an advanced stage of the disease and did not respond to thiamin treatment. Animals that responded very well to the treatment were only included in this study (Plates 1-7). Six apparently healthy goats served as control.

4.1. Epidemiology

The data on the occurrence of thiamin responsive PEM was collected from the Veterinary College Hospital, Mannuthy. The data for the years 1998 and 1999 revealed that 21.3 per cent of the recorded cases were goat cases and out of this 1.12 per cent were PEM cases. (Table 1).

The history revealed that none of these animals were previously affected with a similar disease. History did not suggest any possible predisposing cause for the development of the disease.

Most of them were regularly fed with rice-gruel and ground nut cake in small quantities.

Semi-intensive type of rearing was commonly practiced by most of the farmers. The animals were sent out for browsing in the afternoon. Most of them were fed with jack leaves.

Most of the owners reared the goats for milk purpose and the male kids were usually disposed off for meat purpose at an early age.

The study revealed maximum occurrence in the month of August (1.86%), followed by 1.62 per cent in the month of December, April (1.39%), June (1.36%), July (1.30%) and January (1.29%). The least occurrence was noted in the month of February and May (0.43%) (Table 1, Figure 1).

Females were found to be more prone to this condition. Out of 35 thiamin responsive PEM cases presented to the Veterinary Hospital, Mannuthy, 14.28 per cent (5) were males and the rest (30) were females (Figure 2).

The age group of the affected animals ranged from six months to five years. Most of the owners brought

their animals to the hospital in a state of lateral recumbency.

4.2. Clinical findings

On clinical observation, the rate of respiration was recorded as 27.17 ± 0.70 / minute before treatment. In the healthy control the respiratory rate was 20.50 ± 1.26 / min. Statistically significant ($p \leq 0.05$) increase was noted in the respiratory rate in the diseased group when compared to the control group (Table 2, Figure 3).

The pulse rate was 83.00 ± 1.97/minute in diseased group and 81.67 ± 1.98 per min in healthy control. No statistically significant difference was noted in the pulse rate between the two groups (Table 2, Figure 3).

The body temperature was found to be 102.80 ± 0.24°F before treatment. In the control group temperature was recorded as 102.60 ± 0.22°F. There was no statistically significant difference between the two groups of animals (Table 2, Figure 3).

In the early stages of the disease the following symptoms were reported by the owners. All the animals were dull and depressed. Excitement with bleating followed by low carriage of head, leaning on the walls, aimless wandering and anorexia, ataxia and rolling of eye balls were reported. The goats were presented to the hospital in a state of lateral recumbency. Hyperaesthesia noted on slight provocation such as, handling of the animal. Tonic- clonic convulsions, opisthotonus and star-gazing posture were noted. The affected animals often had injuries on the eyelids due to convulsions and rubbing on the ground. The animals lie down only on one side. On turning to the opposite side the animals struggled violently and shifted to the previous position by itself. Champing of jaws and frothy salivation were noted only in one case. Most of the animals used to eat green leaves even when they were in lateral recumbency. Grinding of teeth and paddling movements were noted in affected animals. The recumbent animals shifted site in circles. Corneal opacity developed in two animals due to prolonged recumbency and self inflicted injuries to the eye. In one case bed sores developed due to prolonged recumbency on cement floor.

4.3. Rumen fluid

4.3.1. Physical characters.

The pH was found to range from six to eight with a mean value of 7.00 ± 0.26 in diseased goats. In the control group the pH was recorded as 7.08 ± 0.30 (Table 3).

The colour of the rumen liquor was found to be olive green / greenish yellow. There was no change in the colour of the rumen liquor in the diseased animals compared to the control group of animals (Table 3).

The consistency was thick in both the groups. Aromatic odour was noted in the PEM affected goats and in the healthy control group (Table 3).

No statistically significant difference was noted in the physical characters of the rumen liquor between the two groups.

4.3.2. Microbial activity

The protozoal motility ranged from moderate (++) to vigorous (+++) in the diseased group. In healthy control group protozoal motility was (+++) vigorous (Table 4).

Iodophilic activity ranged from (++) to (+++) in the diseased goats and in the control group iodophilic activity was obtained as (+++) (Table 4).

Protozoal count of 4.53 ± 0.89 x 10^5 / ml was found in diseased animals and in the control group the protozoal count was 4.03 ± 0.51 x 10^5/ ml. No significant difference was noted in protozoal count between the two groups (Table 5).

The mean sedimentation acivity time (SAT) was found to be 12.83 ± 0.79 minutes in diseased animals. In the normal healthy goats, SAT was recorded as 12.00 ± 0.58 min. No statistically significant difference was noted between diseased and control groups (Table 5).

Mean value of methylene blue reduction time (MBRT) was 4.66 ± 0.98 minutes and 3.50 ± 0.43 minutes respectively in diseased and control groups. No statistically significant difference was noted between the two groups of animals (Table 5).

4.3.3. Total volatile fatty acids (TVFA)

Total volatile fatty acids concentration in the rumen liquor was 67.00 ± 2.80 mEq/l, in PEM affected animals and 70.66 ± 2.28 in healthy control. No statistically significant difference was noted between diseased group and control group of goats (Table 5).

4.3.4. Total acidity (TA)

Mean value of total acidity (TA) was 15.33 ± 1.33 units in diseased animals and 13.16 ± 0.83 units in control group. No statistically significant difference was noted in total acidity between the two groups of goats (Table 5).

4.3.5. Lactate

The mean lactate content of rumen liquor was 64.22 ± 16.29 mg/dl before treatment. Statistically significant increase ($p \leq 0.01$) was noted in diseased animals when compared to the normal healthy animals (20.18 ± 1.91 mg/dl) (Table 5 and Figure 4). After treatment and recovery the mean lactate level was found to be 20.90 ± 2.91 mg/ dl.

4.3.6. Ammonia

The mean ammonia level of the rumen liquor was 34.55 ± 6.91 mg/dl in diseased and 32.76 ± 2.58 mg/dl in the healthy control. No statistically significant difference was noted between the diseased animals and control group of animals (Table 5).

4.4. Haematology

Mean haematological values were presented in the Table 6.

4.4.1. Haemoglobin

The mean haemoglobin value was 10.93 ± 1.38 g per cent in diseased animals and 12.33 ± 0.54 g per cent in healthy control. No statistically significant difference was noted in the mean haemoglobin values between the diseased and control groups of animals.

4.4.2. Haematocrit (Packed cell volume- PCV)

Mean haematocrit values in diseased goats were 31.16 ± 3.41 per cent and in healthy control animals the haematocrit was recorded as 29.50 ± 1.59 per cent. No statistically significant difference was noted in

the mean haematocrit value between the two groups of animals.

4.4.3. Erythrocyte sedimentation rate (ESR)

The mean value of ESR was 5.40 ± 0.25 mm per 24 hours in diseased animals. In healthy control goats the ESR value was 5.23 ± 0.26 mm per 24 hours. No statistically significant difference was observed between the diseased and control groups of animals.

4.4.4. Total erythrocyte count (TEC)

The mean total erythrocyte count was 12.93 ± 0.87 x 10^6/ mm^3 in diseased goats. In the control group of goats the value was recorded as 14.13 ± 0.57 x 10^6/ mm^3. No statistically significant difference was noted between diseased and control groups of animals.

4.4.5. Total leukocyte count (TLC)

The mean total leukocyte count was found to be 14.61 ± 0.49 x 10^3 / mm^3 in diseased and in the healthy control animals TLC was recorded as 13.28 ± 0.53 x 10^3 / mm^3. No statistically significant

difference was noted in the TLC between the two groups.

4.4.6. Differential leukocyte count (DLC)

The mean values of neutrophils were found to be 67.33 ± 3.05 per cent in diseased animals. In the normal healthy animals it was recorded as 36.66 ± 2.16 per cent. Statistically significant increase was noted in diseased animals when compared to the control group (Figure 5). After treatment it was recorded as 35.16 ± 2.36 per cent.

The mean value of lymphocytes were 31.33 ± 3.08 per cent in diseased goats. In the healthy control group the lymphocyte percentage was 59.80 ± 1.87 per cent. Statistically significant difference ($p \leq 0.01$) was noted between diseased and control group of goats (Figure 5). After treatment it was 64.16 ± 2.50 per cent.

The mean value of eosinophils were recorded as 1.00 ± 0.00 per cent in diseased animals. In the healthy control group it was recorded as 1.50 ± 0.56 per cent. No statistically significant difference was

noted between diseased goats and control groups of goats.

Mean value of monocytes were recorded as 0.83 ± 0.00 per cent in diseased goats. In healthy control group it was recorded as 0.83 ± 0.40 per cent. No statistically significant difference was noted between these two groups of animals.

Mean value of basophils were recorded as 0.17 ± 0.18 per cent in diseased goats and in healthy control group it was recorded as 0.35 ± 0.20 per cent. No statistically significant difference was noted between the two groups.

4.5. Serum biochemistry

Mean biochemical values were presented in the Table 7.

4.5.1. Glucose.

The mean glucose level was 98.50 ± 8.83 mg/ dl in diseased animals. Statistically significant increase ($p \leq 0.01$) was noted (Fig. 6) in the glucose level in diseased goats when compared to healthy control group

(52.83 ± 2.76 mg/dl). After recovery the glucose level came to a level of 54.17 ± 5.51 mg/ dl.

4.5.2. Calcium

The mean calcium level was found to be 9.37 ± 0.24 mg/dl in the diseased group. In healthy control group of animals the calcium level was noted as 9.27 ± 0.39 mg/ dl. No statistically significant difference was noted between these values.

4.5.3. Phosphorus

Mean phosphorus value was found to be 4.92 ± 0.17 mg/dl in the diseased group. In normal healthy goats the phosphorus level was recorded as 5.38 ± 0.26 mg/dl. No statistically significant difference was noted between before treatment and control group of goats.

4.5.4. Magnesium

The mean value of magnesium levels was found to be 1.08 ± 0.15 mg/dl before treatment. In healthy control group Magnesium level was noted as 1.23 ± 0.21 mg/ dl. No statistically significant difference was noted between diseased and control group of goats. The

mean post treatment value was 1.00 ± 0.00 mg/dl and was not statistically different from the pretreatment value.

4.5.5. Total protein and Albumin

Mean total protein value was found to be 7.72 ± 0.15 g/dl in diseased goats. In the control group the total protein was recorded as 6.72 ± 0.26 g/dl. No statistically significant difference was noted in the total protein level between diseased and control groups of goats.

Albumin level was recorded as 3.75 ± 0.13 g/dl in diseased animals and in normal healthy goats albumin level was recorded as 3.61 ± 0.23 g/dl. No statistically significant difference was noted between diseased and control groups of animals.

4.5.6. Lactate and Pyruvate

The mean lactate level in the diseased group was 72.06 ± 5.42 mg/dl. Marked variation was noted in the lactate level from animal to animal and it ranged from 59.3 to 97.3 mg /dl. In healthy control group the lactate value was recorded as 16.73 ± 0.48 mg/dl.

Statistically significant difference ($p \leq 0.01$) was noted between the control and diseased group (Figure 7). The mean lactate level was found to be 21.93 ± 3.32 mg/ dl after the treatment, which was found to be significantly different from the pre-treatment values.

A mean pyruvate value of 5.07 ± 0.34 mg/dl was found before treatment. In healthy control group the pyruvate level was recorded as 2.32 ± 0.07 mg/dl. Statistically significant ($p \leq 0.01$) difference was noted between before treatment and control group of animals (Figure 8). The mean post-treatment value was 2.72 ± 0.23 mg/dl after treatment and was significantly different from the pretreatment value.

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Table 1. Data on the occurrence of PEM for the year 1998 and 1999

(Veterinary Hospital, Mannuthy)

Month	Total number of cases	Number of goat cases	Number of PEM cases	PEM cases in percentage
January	1141	310	4	1.29
February	1140	235	1	0.43
March	1303	284	2	0.70
April	1081	216	3	1.39
May	1072	231	1	0.43
June	1114	220	3	1.36
July	1348	307	4	1.30
August	1335	269	5	1.86
September	1240	301	4	1.33
October	1279	266	2	0.75
November	1280	250	2	0.80
December	1389	247	4	1.62
Total	14722	3136	35	1.12

Total goat cases = 21.30 %
PEM cases among goat cases = 1.12%

Table 2. Mean clinical data of healthy control and PEM affected goats.

Parameter	Control group Mean ± SE	Diseased group Mean ± SE
Resp. (per minute)	20.50 ± 1.26	27.17 ± 0.70*
Pulse (per minute)	81.67 ± 1.98	83.00 ± 1.97
Temp (° F)	102.60 ± 0.22	102.80 ± 0.24

*- Significant statistically ($p \leq 0.05$)

Table 3. Rumen liquor - Physical characters in healthy control and PEM affected goats.

Parameter	Healthy control Mean ± SE	Diseased group Mean ± SE
pH	7.08 ± 0.30	7.00 ± 0.26
Colour	Olive green	Olive green
Odour	Aromatic	Aromatic
Consistency	Thick	Thick

Table 4. Rumen liquor - protozoal activity and iodophilic activity in healthy control and PEM affected goats.

Protozoal activity		Iodophilic activity	
Control group	Diseased group	Control group	Diseased group
+++	++	+++	++
+++	++	+++	++
+++	+++	+++	+++
+++	++	+++	+++
+++	+++	+++	++
+++	++	+++	++

Table 5. Rumen Liquor- parameters studied in healthy control and PEM affected goats.

Parameters	Control group Mean ± SE	Diseased group Mean ± SE
SAT (minutes)	12.00 ± 0.58	12.83 ± 0.79
MBRT (minutes)	3.50 ± 0.43	4.66 ± 0.98
Protozoal count ($x10^5$/ml)	4.03 ± 0.51	4.53 ± 0.89
TVFA (mEq/l)	70.66 ± 2.28	67.00 ± 2.80
Total acidity (TA) (units)	13.16 ± 0.83	15.33 ± 1.33
Lactate (mg/dl)	20.18 ± 1.91	64.22 ± 16.29**
Ammonia (mg/dl)	32.76 ± 2.58	34.55 ± 6.91

**- Statistically significant ($p \leq 0.01$)

Table 6. Mean haematological values of healthy control and PEM affected goats.

No.	Parameter	Control group Mean ± SE	Diseased group Mean ± SE
1	Haemoglobin (g%)	12.33 ± 0.54	10.93 ± 1.38
2	PCV (%)	29.50 ± 1.59	31.16 ± 3.41
3	TEC (x 10^6/ mm^3)	14.13 ± 0.57	12.93 ± 0.87
4	TLC (x10^3/mm^3)	13.28 ± 0.53	14.61 ± 0.49
5	ESR (mm/24hrs)	5.23 ± 0.26	5.40 ± 0.25
6	DLC (%)		
	Neutrophils	36.66 ± 2.16	67.33 ± 3.05**
	Lymphocytes	59.80 ± 1.87	31.33 ± 3.08**
	Eosinophils	1.50 ± 0.56	1.00 ± 0.00
	Monocytes	0.83 ± 0.40	0.83 ± 0.00
	Basophils	0.35 ± 0.20	0.17 ± 0.18

**- Statistically significant $p<0.01$

Table 7. Mean serum biochemical values of healthy control and PEM affected goats.

No.	Parameter	Control group Mean ± SE	Diseased group Mean ± SE
1.	Glucose (mg/dl)	52.83 ± 2.76	98.50 ± 8.83**
2.	Total protein (g/dl)	6.72 ± 0.26	7.72 ± 0.15
3.	Albumin (g/dl)	3.61 ± 0.23	3.75 ± 0.13
4.	Lactate (mg/dl)	16.73 ± 0.48	72.06 ± 5.42**
5.	Pyruvate (mg/dl)	2.32 ± 0.07	5.07 ± 0.34*
6.	Calcium (mg/dl)	9.27 ± 0.39	9.37 ± 0.24
7.	Phosphorus (mg/dl)	5.38 ± 0.26	4.92 ± 0.17
8.	Magnesium (mg/dl)	1.23 ± 0.21	1.08 ± 0.15

**- Statistically significant $p \leq 0.01$
*- Statistically significant $p \leq 0.05$

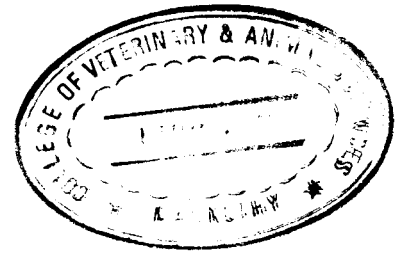

Fig. 1. Month-wise occurrence of PEM among goats

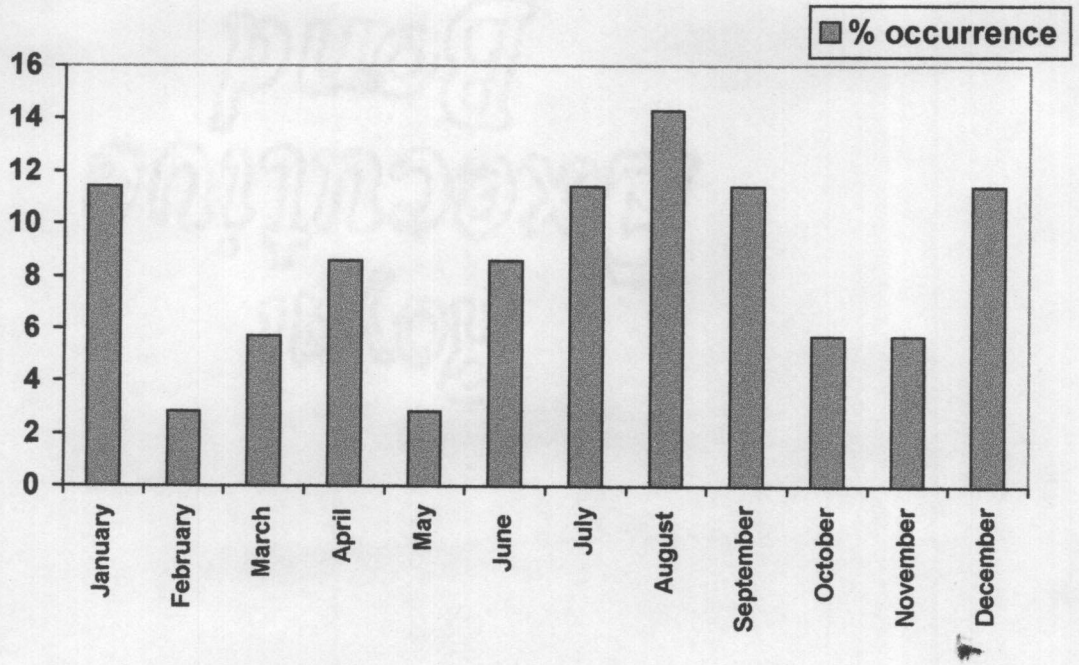

Fig. 2. Sex-wise frequency of PEM among goats

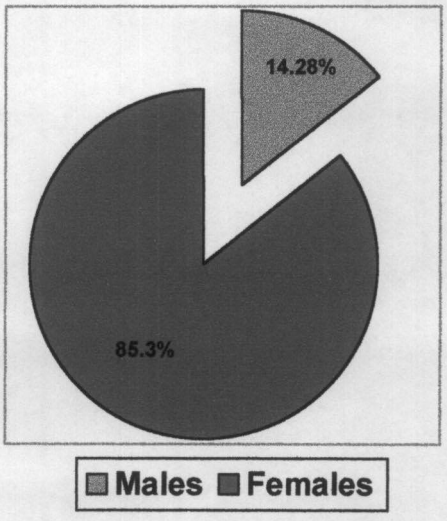

Fig. 3. Clinical data- in control and diseased goats

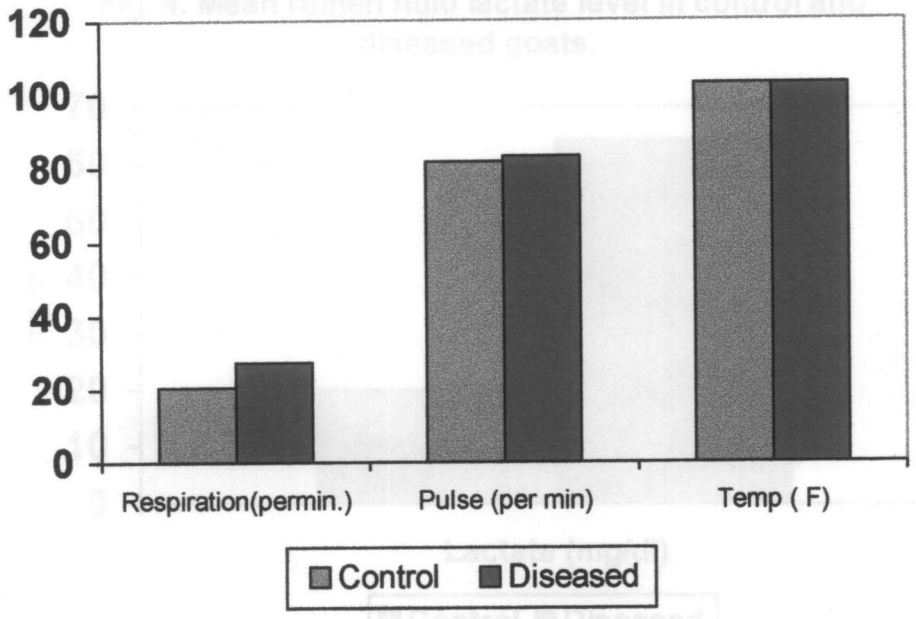

Fig. 4. Mean rumen fluid lactate level in control and diseased goats.

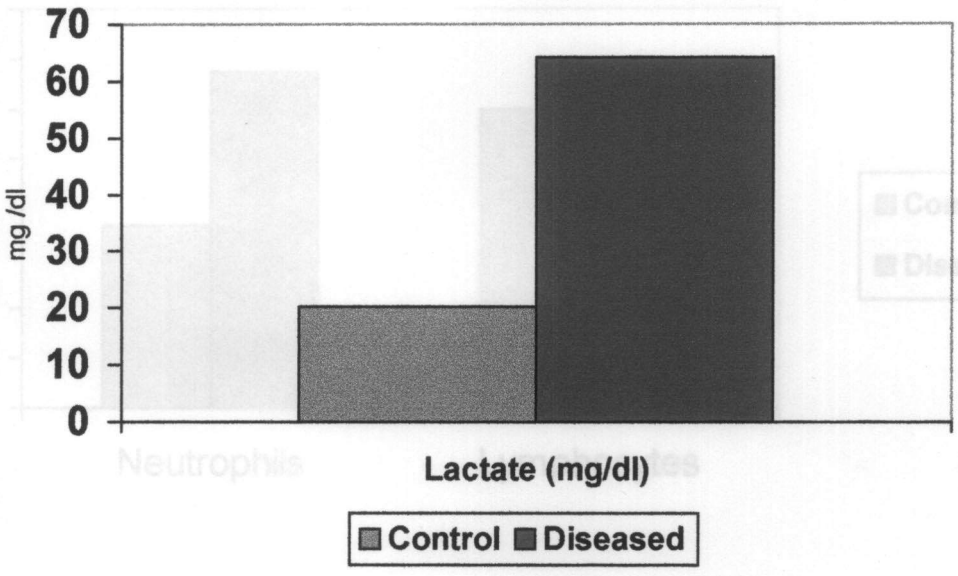

Fig. 5. Differential leukocyte count in control and diseased groups (%)

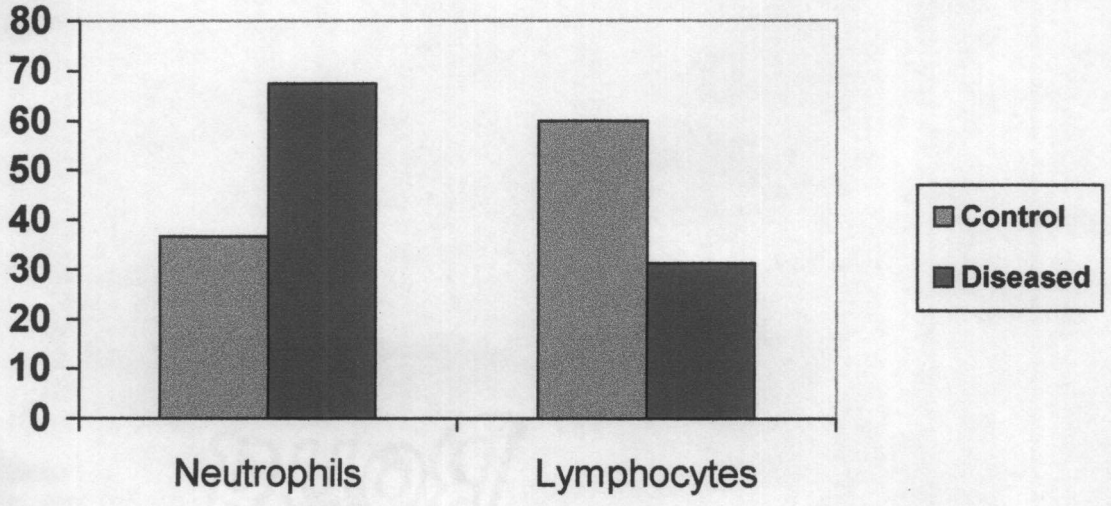

Fig. 6. Mean serum glucose level in the control group and PEM affected goats.

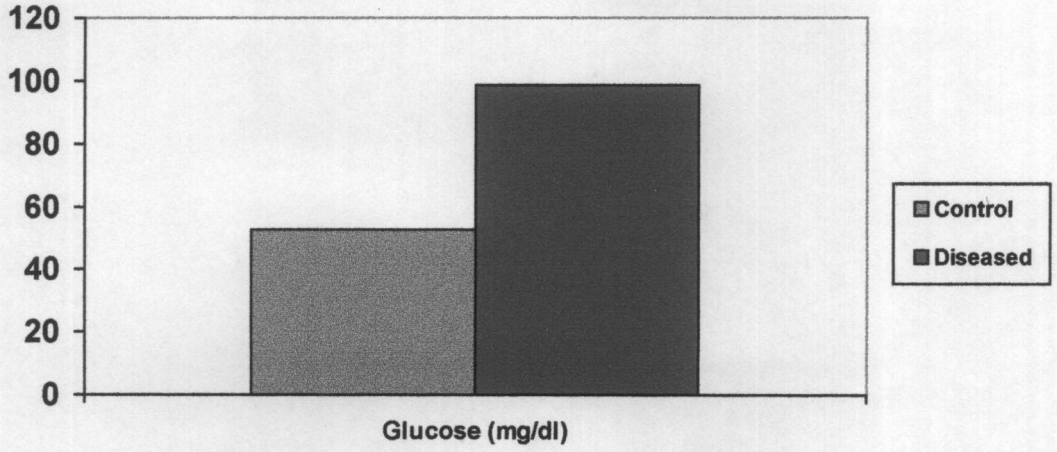

Fig. 7. Mean serum lactate level in the control group and PEM affected goats.

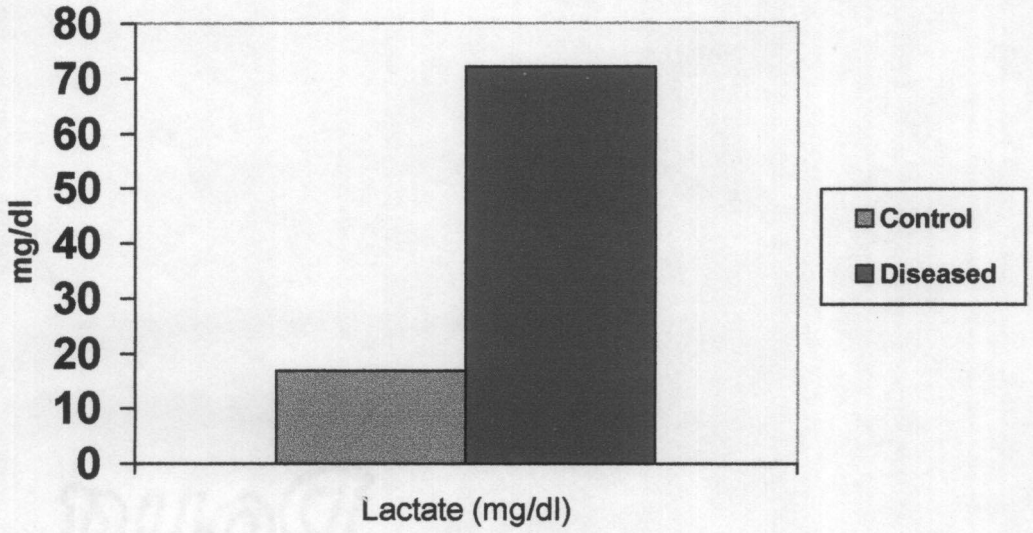

Fig. 8. Mean serum pyruvate level in the control group and PEM affected goats

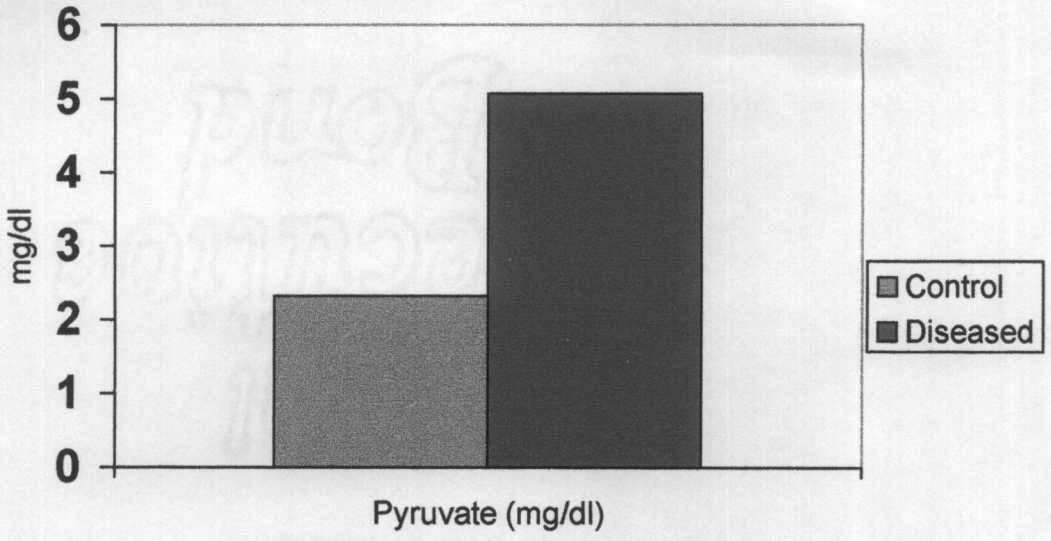

Plate 1. Clinical signs in PEM of goats.

Plate 2. Goat I affected with PEM- before treatment.

Plate 3. Goat I affected with PEM- during treatment

Plate 4. Goat I affected with PEM- after treatment.

Plate 5. Goat II affected with PEM- before treatment.

Plate 6. Goat II affected with PEM- during treatment

Plate 7. Goat II affected with PEM- after treatment.

DISCUSSION

5.DISCUSSION

Data on the occurrence of the disease collected from the University Hospital, Mannuthy for the year 1998 and 1999 revealed that 21.30 per cent of the cases presented to the hospital were goat cases and 1.12 per cent of these cases exhibited symptoms suggestive of polioencephalomalacia (Table 1). An incidence rate of 1.43 per cent was reported by Sobhanan (1981) and 2.44 per cent by Lonkar *et al.* (1993). The slight difference in the percentage of occurrence might be due to the small population considered for this study.

Although the disease occurred throughout the year, maximum occurrence was during the monsoon and post-monsoon period (Aug- January) and the least occurrence during the later part of the summer (February - May) season (Fig 1). Jensen *et al.* (1956) and Tanwar (1987) observed that CCN occurred when animals were shifted to lush pasture. Some vegetation might contain thiaminase mimicking substances causing destruction of thiamin in the rumen (Dickie and Berryman, 1979). According to Tanwar (1987) there was

no correlation between the occurrence of PEM and temperature, humidity and rainfall. Little and Sorensen (1969) could not observe any seasonal influence on the occurrence. Lonkar *et al.* (1993) observed the disease throughout the year.

This study revealed that the disease was more prevalent in females and this could be due to more number of females in the population (Fig. 2). Male goats are either disposed off or slaughtered at an early age, whereas females are retained for breeding and milk production. Lonkar *et al.* (1993) also reported similar findings.

The age group of the animals affected generally ranged from six months to five years. Th disease was recorded in young goats of two months to one year and the oldest affected was 2.5 years according to Sobhanan (1981). Similar observations were made by Jubb and Huxtable (1991) and Smith (1979). Rumen becomes functional by three months of age in the case of goats (Ranjhan, 1993). Goats like other ruminants meet their thiamin requirement through intraruminal microbial synthesis (Ewan, 1996) so any alteration in the microbial flora may cause thiamin deficiency.

In Kerala, rice (a carbohydrate rich diet), form the main component of concentrate ration. The irregular or excess feeding of rice often accounts for majority of ruminal dysfunctions with lowering of pH (Aleyas and Vijayan, 1981) and possibly creation of a favourable environment for increased thiaminase activity (Sobhanan, 1981). Sapienza (1981) suggested that sudden changes from roughage to concentrate diet resulted in the rapid multiplication of gram-positive bacteria and *Clostridium sp.* which inturn produced enzymes which reacted with thiamin in the rumen and caused CNS disorders.

Jack leaves are commonly fed to goats in Kerala. Goats are sent out for browsing in semi-intensive type of rearing that is usually practiced in Kerala. Common roughage eaten by goats needs screening for the presence of substances simulating thiaminase.

5.2 Clinical findings

The basic clinical data collected from the diseased animals did not show statistically

significant difference from the control animals except for the rate of respiration.

The rate of respiration was 27.17 ± 0.70 per minute in diseased animals and 20.50 ± 1.26 per minute in healthy control group (Table 2, Fig.3). A non-significant increase was also noted in the pulse rate in the diseased goats (83.00 ± 1.97 per minute). The significant increase in the respiratory rate and non-significant increase in pulse rate could be due to the excitement and occasional convulsions. These findings were in accordance with Smith (1979) and Fakhruddin *et al.* (1987a).

Similar to the findings of Little and Sorensen (1969) the disease syndrome started without any premonitary signs. In the initial stages of the disease, excitement, frequent bleating, followed by low carriage of head, leaning on to the wall, aimless wandering and upward turning of head were reported by owners. All the cases were presented to the hospital in a state of lateral recumbency. Even in lateral recumbency they consumed concentrates and leaves in small quantities. The signs observed agreed with those

reported by Markson *et al.* (1974) and Pierson and Jensen (1975). Nystagmus (vertical nystagmus in four cases and horizontal nystagmus in two cases) a constant finding in the present study was also reported by Jensen *et al.*(1956), Terlecki and Markson (1958), Little and Sorensen (1969), Blood *et al.* (1979), Smith (1979), Sobhanan (1981), Fakhruddin *et al.* (1987a) and Lonkar and Prasad (1992a). Hyperaesthesia and paddling movements were noted. Grinding of teeth and salivation were noted in one case. Similar findings were recorded by Fakhruddin *et al.* (1987a), Tanwar (1987) and Gauri and Vashistha (1988).

Corneal opacity that developed in two of the animals was traumatic in origin due to frequent shaking and rubbing of the head on cement floor of the shed. Since the animals were lying on right side, the eye on that side only developed opacity. Similar findings were recorded by Lonkar and Prasad (1992a).

On the third day of treatment with thiamin, the goats were able to stand up although the posture and gait was unsteady. After five days of the treatment the animals were able to walk normally and started

normal feeding. The two animals which were brought to the hospital with less severe clinical signs recovered completely within three days of treatment.

Another characteristic finding noted in this study was that the affected animals preferred to lie only on one side and on turning to the other side, the animals struggled severely and returned to the previous position by itself. Thornber *et al.* (1981) reported mild localised brain lesions in thiamin deprived lambs. The manifestation of clinical signs may not be an effect of necrotic lesions but rather an effect of severe brain oedema which is a prominent feature in severe cases of PEM. Blindness, opisthotonus, nystagmus and extensor rigidity indicated central nervous system disturbances (Maxwell, 1980). The only visible lesions were those in the grey matter of the cerebral cortex where multiple foci of necrosis of the neurons occurred and many of the cerebral gyri were swollen (Rammell and Hill, 1986). Alteration in the behaviour and posture occurred probably due to brain lesions which led to loss of integrity of cranial nerves (Singh *et al.* 2000).

The major clinical signs manifested such as nystagmus, lateral deviation of head, circling and lying only on one side suggested that the lesions were predominantly localised/ asymmetrical. Increased intracranial pressure due to diffuse cerebral oedema might be the reason for symptoms like opisthotonus, nystagmus and muscle tremor (Radostits, *et al.*1994). Animals had inability to rise during the later stages of the disease and it may be due to the involvement of the brain-stem as suggested by Rodostits *et al.* (1994).

5.3 Rumen fluid

5.3.1 Physical characters

No statistically significant difference was noted in the pH of rumen liquor between the two groups. The colour, consistency and odour were same in both the diseased and control groups (Table 3) of goats. In the light of the observations the hypothesis of chronic ruminal acidosis as one of the predisposing factors for the development of polioencephalomalacia requires further proof.

5.3.2 Microbial activity

5.3.2.1 Protozoal activity

The protozoal activity in the healthy control was in agreement with that of Pillai (1988). Protozoal activity in the diseased goats ranged from moderate (++) to vigorous (+++) but in the control group all animals had vigorous (+++) protozoal activity (Table 4).

5.3.2.2 Iodophilic activity

In the diseased goats iodophilic activity ranged from ++ to +++ and in the healthy control group it was +++ (Table 4).

5.3.2.3 Protozoal count

No statistically significant difference was noted in the protozoal count between diseased and control groups of animals (Table 5). The values obtained in the present study were in the normal range (1.11 to 7.2×10^5 per ml) as reported by Choudhari *et al.* (1985).

5.3.2.4 Sedimentation activity time (SAT)

No statistically significant difference was noted in the sedimentation activity time between diseased and control group of goats (Table 5). The SAT obtained

in the healthy control was in agreement with that of Pillai (1988).

5.3.2.5 Methylene blue reduction time (MBRT)

No statistically significant difference was noted in the MBRT value between the two groups of goats (Table 5). The MBRT was found to be within the reference range reported by Shihabudheen (1998) in healthy goats.

5.3.3 Total volatile fatty acids (TVFA)

No statistically significant difference was noted in TVFA values between diseased (67.00 ± 2.80 mEq/l) and control (70.66 ± 2.28 mEq/ l) groups of goats (Table 5). The TVFA values obtained in the healthy control group were found to be similar to the findings of Pillai (1988). According to Rai *et al.* (1972) the peak concentration of TVFA was observed two hours after feeding and there could be some difference due to the interval between feeding time and collection time.

5.3.4 Total acidity (TA)

No statistically significant difference was noted in total acidity between diseased (15.33 ± 1.33) and control groups (13.16 ± 0.83) of goats (Table 5). In cattle, total acidity was recorded as 8 to 25 units (Dirksen, 1979).

5.3.5 Lactate

The mean lactate content of rumen liquor was 64.22 ± 16.29 mg/dl, 20.90 ± 2.91 mg/ dl and 20.18 ± 1.91 mg/ dl in diseased, after treatment and control group of animals respectively (Table 5 and Figure 4). Statistically significant difference ($p<0.05$) was noted in the diseased animals. An increase in the lactate content of rumen liquor in diseased goats was in agreement with that of Sager *et al.* (1990) in nutritionally induced PEM in calves. Although the lactate level was high it was not enough to induce marked lowering of the rumen pH. But it might be possible that this change could induce microbial changes in the rumen.

5.3.6 Ammonia

No statistically significant difference was noted in the level of ammonia between diseased and control groups of goats (Table 5). Values obtained in the control group were in agreement with the report of Rai *et al.* (1972).

The overall findings in the rumen fluid suggested that there were no major changes in the rumen environment in cases of PEM. Before making a final opinion detailed microbiological and cultural studies are required.

5.4 Haematology

Mean haematological values were given in Table 6.

5.4.1 Haemoglobin

No statistically significant difference was noted in the haemoglobin level between the control and diseased animals. Normal haemoglobin level was recorded by Tanwar (1987), Fakhruddin *et al* (1987a) and Gauri and Vashistha (1988) in PEM of goats. Similar finding was reported in calves by Singh *et al.* (2000).

5.4.2 Haematocrit (Packed cell volume)

No statistically significant difference was noted in PCV between the two groups of goats. The control group values obtained were similar to the reports of Shihabudheen (1998) in healthy goats.

Similar findings were reported by Pass (1968) and Tanwar (1987) in PEM of goats. The values obtained in the present study were within the normal range.

5.5.3 Erythrocyte sedimentation rate. (ESR)

The mean value of ESR in the control group was within the normal range reported by Shihabudheen (1998). Difference in the ESR values between the healthy goats and PEM affected goats was not significant.

5.4.4 Total erythrocyte count (TEC).

No statistically significant difference was noted between the two groups. Mean values of total erythrocyte count in healthy control group in the present study were comparable to the reports of Radostits *et al.* (1994) and Shihabudheen (1998).

Fakhruddin *et al.* (1987a) and Singh *et al.* (2000) reported slight decrease in TEC values in amprolium induced PEM of goats and explained that it could be due to the suppressive effect of amprolium on erythropoiesis.

5.4.5 Total leukocyte count (TLC)

Mean value of total leukocyte count in healthy control goats obtained in the present study was comparable to the values reported by Shihabudheen (1998).

Slight increase in TLC in the diseased group was non-significant and it could be due to stress as reported by Fakhruddin *et al.* (1987a). Singh *et al.* (2000) also reported slight increase in TLC in PEM induced calves. Tanwar (1987) reported a TLC of 12.20 to 17.25 thousands/ mm^3 in PEM cases.

5.5.6 Differential leukocyte count (DLC)

A statistically significant increase ($p \leq 0.01$) was noted in the neutrophil count in diseased goats (Figure 5). After treatment and recovery the values became normal. The control values obtained for

neutrophil count were similar to the observations of Benjamin (1985) and Shihabudheen (1998).

Increased percentage of neutrophils in the present study was in accordance with Spence *et al.* (1961), Fakhruddin *et al.* (1987a) and Gauri and Vashistha (1988). This increase might be due to the stress response in diseased goats. Neutrophilia, lymphopaenia and eosinopaenia were reported in stress (Benjamin, 1985).

A significant lymphopaenia and a nonsignificant eosinopaenia were noted in diseased goats in the present study. Non significant difference was noted in the monocyte and basophil count between the two groups of goats.

5.5 Serum biochemistry

5.5.1 Glucose

The mean glucose levels were 98.50 ± 8.83 mg/dl and 52.83 ± 2.76 mg/dl in diseased and control groups of goats respectively. Statistically significant increase was noted in the level of glucose in diseased

goats when compared to the control group (Table 7 and Fig. 6).

The values obtained for the control group were similar to the observations of Varma (1967) in healthy goats. Significant increase in the glucose level observed in this study was found to be in agreement with that of Pass (1968), Lilja (1973), Spicer and Horton (1981), Lonkar and Prasad (1993) and Syamsundar and Malik (1993) in PEM affected goats.

The hyperglycemia observed might be due to the combined effects of stress, convulsions, tetany and cerebral damage which occurred in PEM. The elevations in glucose levels represent an adrenergic response since trauma has been shown to cause increase in glucagon leading to hyperglycemia (Brockman and Many, 1976).

5.5.2 Calcium

No statistically significant difference was noted for calcium level between the two groups of goats. The value obtained for the control group was in agreement with that of Shihabudheen (1998).

The values obtained were in the normal range and it was in agreement with the findings of Spence *et al.* (1961) and Syamasundar and Malik (1993). The finding confirmed that serum calcium level was not having any role in the manifestation of clinical signs associated with PEM.

5.5.3 Phosphorus

The phosphorus levels obtained for the control group was in the normal range as reported by Pyne *et al.* (1982). No statistically significant difference was noted between diseased and control groups of goats.

The phosphorus levels were found to be within the normal range and was in agreement with the findings of Spence *et al.* (1961) and Syamasundar and Malik (1993) in PEM affected goats.

5.5.4 Magnesium

No statistically significant difference was noted for magnesium levels between the two groups.

Normal magnesium level was recorded in outbreaks of PEM in sheep and cattle (Spence *et al.*, 1961), in

calves (Pass, 1968), in lambs (Thornber *et al.*, 1981) and in buffalo calves (Syamasundar and Malik, 1993, Tanwar *et al.*, 1994).

5.5.5 Total protein and albumin

Total protein and albumin levels in control group were found to be similar to the observations of Pillai (1988) in normal goats. No statistically significant difference was noted between the two groups of goats. A nonsignificant fall was recorded by Syamasundar and Malik (1993) in amprolium induced PEM in buffalo calves. The normal protein level suggested normal liver function in PEM affected goats.

5.5.6 Lactate and Pyruvate

Statistically significant increase ($p \leq 0.01$) was noted for the lactate level in diseased goats (72.06 ± 5.42mg/dl) compared to the control group of goats (Table 7 and Figure 7). The mean value (16.73 ± 0.48 mg/dl) obtained in the control group was found to be similar to the observations of Pillai (1988) in healthy goats.

The significant rise in the lactate content was in agreement with the findings of Syamasunder and Malik (1993) and Lonkar and Prasad (1993). Increase in the lactate level at the onset of clinical symptoms was reported in CCN or thiamin deficiency suggesting a link among CCN, thiamin deficiency and lactate levels (Mueller and Asplund, 1981). Peters (1967) suggested that pyruvate and lactate accumulated in the blood because tissues were unable to oxidise pyruvate properly in thiamin deficiency. Markson *et al.* (1974) and Loew *et al.* (1970) stipulated that pyruvate metabolism was TPP dependent. Hence in thiamin deficiency there is rise in blood pyruvate level. Lactate is readily formed from pyruvate by lactate dehydrogenase. Increased absorption of lactate from the rumen of affected animals might also have contributed to the serum lactate level.

The pyruvate level (5.07 ± 0.34 mg/dl) in diseased goats showed statistically significant increase ($p \leq 0.05$) when compared to the control group of goats (2.32. ± 0.07 mg/dl) (Table 7, Figure 8).

The pyruvate level recorded in the control group in the present study was in agreement with that of Shihabudheen (1998).

The significant increase in pyruvate level in the present study was in agreement with that of Syamasundar and Malik (1993) and Lonkar and Prasad (1993). The mean pyruvate and lactate level came down significantly after the course of treatment.

CONCLUSION

The present study revealed that Polioencephalomalacia is a disease condition with characteristic nervous signs that occurred throughout the year. The feeding practices (rice gruel) in Kerala might predispose to PEM in goats. Characteristic clinical signs such as nystagmus, staggering gait, lateral deviation of head, circling movement and lying only on one side were observed. The physical characters and microbial activity of rumen fluid were apparently normal. The increased lactate level in the rumen could induce microbial changes in the rumen. Detailed microbiological studies are needed in this aspect. Marked lymphopaenia and neutrophilia suggested

stress in affected goats. Increased blood lactate and pyruvate levels indicated thiamin deficiency. The hyperglycemia observed in the present study might be due to the combined effect of stress, convulsions and cerebral damage. The common roughage consumed by the goats need screening for the presence of thiaminase mimicking substances. Further studies on the following lines are suggested.

1. Cultural studies on rumen microflora of diseased goats.
2. Role of sulphur in the etiopathogenesis of PEM
   a. Whether excess sulphur induces subclinical acidosis in rumen.
   b. Whether thiaminase producing organisms are selected or encouraged periodically by high intake of sulphur.
   c. Relation between high intake of sulphur and urinary B_1 excretion

SUMMARY

SUMMARY

The present study analysed the epidemilogy, clinical signs, changes in rumen liquor, haematology and serum biochemical alterations in goats affected with Polioencephalomalacia.

Six goats affected with thiamine responsive symptoms suggestive of Polioencephalomalacia were utilised for the study. The required clinical materials were collected before starting the treatment. Six apparently healthy goats maintained under identical conditions served as the healthy control. Detailed history of the affected animals were collected from the owners, using a questionnaire (Appendix). Samples of rumen liquor, whole blood and serum were collected. Studies on rumen fluid included physical characters, microbial activity, total volatile fatty acids, total acidity, lactic acid and ammonia level. Haematological parameters like Hb, PCV, ESR, TEC, TLC and DLC were studied. Biochemical parameters like glucose, calcium, phosphorus, magnesium, total protein, albumin, lactic acid and pyruvate, levels in the serum samples were estimated.

Epidemiological data revealed that the disease occurred throughout the year with maximum incidence in the monsoon and post-monsoon period. The disease was more prevalent in females and the age group affected ranged from six months to five years. The carbohydrate rich diet (rice gruel) was found to be one of the predisposing causes for the development of Polioencephalomalacia of goats in Kerala.

The characteristic clinical symptoms were nystagmus, lateral deviation of head, circling and lying only on one side. In the initial stages of the disease excitement, frequent bleating followed by low carriage of head, leaning on to the wall, wandering and upward turning of head were reported by the owners. The body temperature and pulse rate were within the physiological limits. The rate of respiration showed significant increase in diseased animals.

A highly significant increase in lactic acid content of rumen fluid was noted in affected goats. Haematology revealed significant neutrophilia and lymphopaenia indicative of stress. Biochemical changes

in the serum revealed significant increase in glucose, lactate and pyruvate levels.

The increase in the lactate level in the rumen liquor was not enough to induce marked lowering of the rumen pH, but this change might be sufficient to induce microbial changes in the rumen. Detailed microbiological studies were needed to detect the changes in the microbial flora. The hyperglycemia might be due to the combined effect of stress, convulsions and cerebral damage. Increased blood lactate and pyruvate level suggested a stage of thiamine deficiency. The normal protein level suggested normal liver function in PEM affected goats. Normal serum calcium and magnesium level confirmed that these electrolytes were not involved in the etiopathogenesis of PEM.

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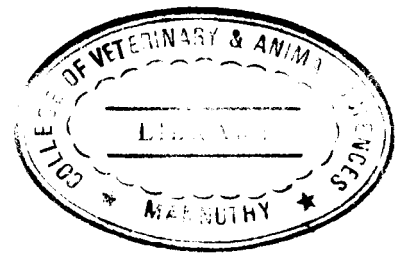

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CLINICAL PROPAEDEUTICS AND RUMEN FLUID CHANGES IN POLIOENCEPHALOMALACIA OF GOATS

By

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ABSTRACT OF A THESIS

Submitted in partial fulfilment of the requirement for the degree of

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ABSTRACT

A study on Polioencephalomalacia was conducted in the Department of Clinical Medicine, College of Veterinary and Animal Sciences, Mannuthy, for a period of two years (1998 and 1999). The epidemiology, clinical findings, changes in rumen fluid, haematology and serum biochemical changes were studied in PEM affected goats.

Six goats affected with Polioencephalomalacia and that responded to thiamine therapy were utilised for this study. Detailed history was collected. Samples of rumen fluid and whole blood from the healthy and diseased animals were collected and analysed. Rumen liquor parameters, viz., physical characters, microbial activity, total volatile fatty acids, total acidity, lactate and ammonia levels were recorded. Haematological parameters like Hb, PCV, ESR, TEC, TLC and DLC were recorded. Glucose, total protein, albumin, lactate, pyruvate, calcium, phosphorus and magnesium levels in the serum were estimated.

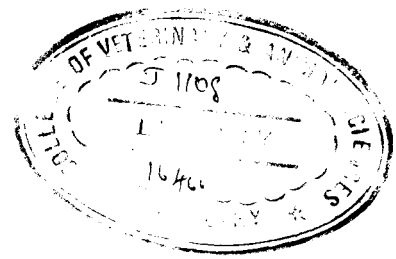
The disease occurred throughout the year with maximum occurrence in the monsoon and post-monsoon

period. Occurrence was more in females and in the age group of six months to five years. History suggested carbohydrate rich diet as one of the predisposing factor for the development of Polioencephalomalacia in Kerala.

The clinical symptoms noted were nystagmus, lateral deviation of head, circling and lying only on one side.

A highly significant increase in the lactate content of rumen fluid, was noted in goats affected with Polioencephalomalacia. Haematology revealed significant neutrophilia and lymphopaenia indicating stress condition. Biochemical changes in the serum revealed significant increase in glucose, lactate and pyruvate levels.

The increase in lactate level in the rumen fluid was not enough to induce marked lowering of the rumen pH. Detailed cultural studies are needed to detect the changes in rumen microbial flora in diseased goats. Increased blood lactate and pyruvate levels indicated thiamine insufficiency.



APPENDIX

PROFORMA FOR THE COLLECTION OF EPIDEMIOLOGICAL DATA

1. Serial No. :

2. Name and Address of the owner :

3. Patient data :
   a) Species :
   b) Age :
   c) Sex :
   d) Breed :
   e) Colour :

4. Purpose for which the animals are maintained

5. Present history :

6. Past history :

7. Feeding practices :
   a) Type of feed given :
   b) Quantity fed :
   c) Frequency of feeding :

8. Management practices :

9. Breeding history :

10. Duration of disease :

11 Predisposing causes, if any :

12. Any other information :