METABOLIC PROFILE OF KETOTIC CROSSBRED DAIRY COWS

By

MANOJ JOHNSON

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

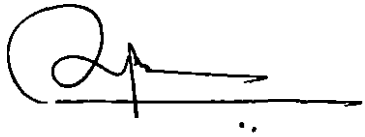
MANNUTHY, THRISSUR - 680 651

KERALA, INDIA

1999

DECLARATION

I hereby declare that this thesis entitled "**METABOLIC PROFILE OF KETOTIC CROSSBRED DAIRY COWS**" 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.



Mannuthy
12.07.1999

MANOJ JOHNSON

CERTIFICATE

Certified that this thesis entitled "**METABOLIC PROFILE OF KETOTIC CROSSBRED DAIRY COWS**" is a record of research work done independently by Sri. Manoj Johnson, under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to him.

Dr. V.S. Balakrishnan
(Chairman, Advisory Committee)
Professor (RC)
College of Veterinary &
Animal Sciences, Mannuthy

Mannuthy
12.07.1999

CERTIFICATE

We, the undersigned members of the Advisory Committee of **Sri. Manoj Johnson,** a candidate for the degree of Master of Veterinary Science in Clinical Medicine, agree that this thesis entitled "**METABOLIC PROFILE OF KETOTIC CROSSBRED DAIRY COWS**" may be submitted by Sri. Manoj Johnson, in partial fulfilment of the requirement for the degree.

Dr. V.S. Balakrishnan
Professor (RC)
(Chairman, Advisory Committee)
College of Veterinary & Animal Sciences

Dr. P.C. Alex
Associate Professor and Head
Department of Clinical Medicine
(Member)

Dr. P.G. Baby
Associate Professor
Department of Clinical Medicine
(Member)

Mr. M. Nandakumaran
Professor
Department of Animal Nutrition
(Member)

External Examiner

ACKNOWLEDGEMENT

I wish to express my profound thanks and deep sense of gratitude to the Chairman of my Advisory committee and Major Advisor Dr. V.S. Balakrishnan, Professor (RC), College of Veterinary and Animal Sciences, Mannuthy for the valuable guidance and constant encouragement throughout this programme of study.

I wish to pay my gratefulness to Dr. P.C. Alex, Associate Professor and Head, Department of Clinical Medicine, Dr. P.G. Baby, Associate Professor, Department of Clinical Medicine and Mr. M. Nandakumaran, Professor, Department of Animal Nutrition for their advice, constructive criticism and valuable suggestions as members of the Advisory Committee.

I am deeply indebted to Dr. N.M. Aleyas, Professor and Head (Retd.) for the valuable help rendered throughout the various stages of my work, collection of materials and preparation of the thesis.

I wish to thank Dr. K.M. Alikutty, Professor and Head (Retd.), Department of Clinical Medicine for the valuable guidances for this study.

Grateful acknowledgements are extended to Dr. S. Ajitkumar, Dr. (Mrs) Usha Narayana Pillai, Dr. R. Vijayan, Dr. K.M. Jayakumar, Dr. Premni Aleyas and other staff of the Department of Clinical Medicine for their constant help and co-operation during the study.

My sincere thanks are extended to Dr. A. Rajan, Dean (Retd.) College of Veterinary and Animal Sciences and Dr. S. Sulochana, Dean (i/c), College of

Veterinary and Animal Sciences for providing necessary facilities for conducting this research work.

I am thankful to the staff of the Department of Statistics for their help in statistical analysis.

I am thankful to Dr. C.N. Dinesh for his constant moral support and inspiration for the successful completion of the study.

My sincere gratitude to my colleagues, Dr. Madhu Rajan Mathews, Dr. P.K. Shihabudheen and Dr. S. Sivaraman for their kind deeds in helping my research work.

I also wish to place on record my sincere thanks to all other friends for their support, encouragement and help throughout the long period of study.

MANOJ JOHNSON

CONTENTS

LIST OF TABLES AND FIGURE

Introduction

INTRODUCTION

Kerala aims to produce 30 lakhs tonnes of milk by the year 2000 AD to improve the per capita availability of milk. Milk production in Kerala is mainly through small farmers who own two or three cows.

The data collected from the field veterinary institutions indicated the prevalence of metabolic diseases among crossbred cattle (Anon, 1994). The common metabolic diseases encountered in the field are milk fever, ketosis and downer cow syndrome. Occasionally hypomagnesaemia and hypophosphataemia are also associated with the above said disease conditions.

In the early part of lactation there is extremely high turnover of fluids, salts and soluble organic materials in dairy cows. The imbalance of any of these metabolites, can result in metabolic disease among bovines.

All the cows in the early post-partum period are in a state of negative energy balance and any stress at this time can make the animal more vulnerable to ketosis. It is characterised by hypoglycaemia, ketonaemia, ketonuria and low levels of liver glycogen which adversely affect the milk yield in early lactation. A delay in the treatment of such cases or improper diagnosis can result in irrepairable loss and

recovered animals may not attain the previous production levels (Choudhuri, 1990).

Ketosis is recognised as one of the major diseases of economic importance as it results in production loss. Published data on the incidence, epidemiology and clinical signs of ketosis in crossbred cattle in India are meagre, especially in Kerala. An understanding of the disease among our cattle and the gravity of economic loss to the farmers will help to suggest managemental measures to reduce the loss in milk production.

Compton metabolic profile test is based on the concept that the laboratory measurement of the components of the blood will reflect the nutritional status of the animal, with or without the presence of clinical abnormalities. It assesses the input-output (nutrient-productivity) relationship which is an attractive tool for the veterinarian engaged in animal health services (Radostits *et al.*, 1994).

Hence the study was undertaken with the following objectives.

1. To study the epidemiology and clinical features of ketotic cows.

2. To evaluate the changes in haematological and biochemical parameters in ketosis using Compton Metabolic Profile Test.

3. To assess the economic loss and suggest suitable managemental practices.

Review of Literature

REVIEW OF LITERATURE

Ketosis in dairy cows was described as early as 1849 (Udall, 1943). It is a disease caused by impaired metabolism of carbohydrates and volatile fatty acids and biochemically characterised by ketonemia, ketonuria, hypoglycaemia and low level of hepatic glycogen (Radostits *et al.*, 1994).

Epidemiology

Ketosis was reported from many countries, in USA and UK (Schultz, 1974, West 1990), Norway (Overby *et al.*, 1974, Simensen *et al.*, 1987), Germany (Schafer and Bethe, 1976), USA, USSR, Sweeden and Bulgaria (Simenov, 1978), Yugoslavia (Nenadovic *et al.* 1978; Fatur, 1990), Checkoslovakia (Lebeda *et al.*, 1982), Japan (Hamana *et al.*, 1985), Serbia (Krdzalic *et al.*, 1988, Fatur, 1990), Switzerland (Gitzwiller, 1989), Denmark (Willadsen *et al.*, 1993) and India (Ziauddin *et al.* 1992, Bhuin *et al.*, 1993, Venkateshwarulu and Rao, 1993).

Sjolemma and Van Der Zande (1923) are thought to be the first to give the details of occurrence of ketosis. They reported that fatty, high producing cows were more susceptible and that the condition usually occurred seven to ten days post partum.

Jazbec (1967) stated that most of the cases of ketosis occurred during first six weeks of parturition and the cows which developed ketosis were heavy yielders and pure German Friesians were the commonly affected one than the German Friesian x Jersey crosses.

Emery *et al.* (1968) reported that the incidence of ketosis with clinical signs occurred widely and about ten per cent of the cases were within the first one month and nearly all the cases occurred within six weeks of calving.

Fox (1971) noted that the cases of ketosis occurred mostly during the peak production years (average 3-6 years of age) and the disease frequency was maximum in the first three to four months after calving.

Baird *et al.* (1974) reported that almost all cases of ketosis occurred between three to six weeks after calving. In another study Baird (1982) stated that the condition occurred spontaneously in high yielders between second and seventh week of lactation and the condition was not common in primpara cows.

Lebeda *et al.* (1982) noted that majority of the cases of ketosis occurred during first three months of lactation and the winter season. Both the degree and frequency of ketonuria decreased as the lactation advanced through out the year.

Dohoo *et al.* (1983) reported an incidence rate of 4.4 per cent of clinical ketosis and 9.6 per cent of subclinical ketosis in Canadian Holstein Friesian cows.

Zharov and Kondrakhin (1983) recorded that in USSR 80 per cent of the cows were affected with ketosis during the winter housing.

Kauppinen (1983) observed a prevalence of 13 per cent for clinical ketosis and 34 per cent for subclinical ketosis in Finnish Ayrshire and Holstein cattle. He also studied the prevalence of bovine ketosis in relation to number and stage of lactation and reported that 3,7,20,22 and 13 per cent incidence at first, second, third, fourth and fifth calving respectively.

Riemann *et al.* (1985) reviewed the epidemiological association of ketosis in Norwegian dairy cows. According to them the highest rates were observed in herds with low standard management and care.

Bjarnason (1986) studied the incidence of ketosis among cows in 37 farms in Iceland and stated that incidence of ketosis increased towards the end of the long (8 months) period of winter housing and appeared to be associated with feeding of concentrates without adequate roughage.

Reddy *et al.* (1986) noted an incidence of 41.37 per cent, when the urine was randomly tested with Rothera's reagent in corssbred cattle. They also noted that the incidence was more during the first and second month of lactation.

Simensen *et al.* (1987) reported that risk of ketosis was higher with increased number of lactation and milk yield. The incidence increased with inclement weather.

In Serbia, Krdzalic *et al.* (1988) noted that most cases of ketosis occurred during winter (50%) than in summer or autumn (32%). He also observed that out of 469 cases studied 27 per cent occurred in the first five days after calving and 42 per cent between six and ten days after calving. The breed most affected was Holsteins (55.9 per cent) than East Friesians (41.5%) or Simmentals (26.6%).

According to Singh and Kasaralikar (1988) occurrence of ketosis was at the maximum between zero to one month after calving (42.86%) followed by two to three months (28.57%), one to two months (21.42%) and three to four months (7.14%).

Yao *et al.* (1988) recorded that although early ketonemia occurred during 7 to 8 months of gestation, the middle and advanced conditions occurred mainly during a period of one to eight weeks after parturition and the highest incidence took place within one week after parturition.

Rautmare *et al.* (1989) stated that the prevalence of clinical ketosis among buffaloes were 41.17 per cent, 29.41 per cent, 23.53 per cent and 5.88 per cent in fourth, third, second and fifth lactation respectively.

Singh and Kasaralikar (1990) observed the common occurrence of clinical ketosis in November (37.71%), in eight to nine year old buffaloes (57.14%) and in fourth lactation. According to them the disease occurred with greater frequency in the first month of lactation.

West (1990) reported that average interval from calving to the diagnosis of ketosis was 16.4 ± 2.0 days.

Ziauddin *et al.* (1992) noted that the breed most commonly affected were Holstein Friesians and most of the cases occurred in the September and lowest in June. The age groups most affected were between five to seven years.

Anantwar and Singh (1993) conducted detailed studies on ketosis in buffaloes and observed that the highest incidence occurred from September to December (55.65%).

Bhuin *et al.* (1993) recorded that out of the 504 dairy cows examined, the breed wise incidence of ketosis were, seven (4.5%), five (4.2%), three (2.5%) and one (0.9%) in Jersey crossbreds, Holstein Friesian crossbred, other crossbreds and

non-descript cows respectively. They also noted that 50 per cent of the clinical ketosis cases occurred during September to December, 31.2 per cent in May to August and 18.75 per cent in January to April.

In a study on 1431 high yielding cows, Fatur *et al.* (1993) noted an incidence of 24.7 per cent of subclinical and 1.4 per cent of clinical ketosis.

Willadsen *et al.* (1993) stated that ketosis was common during the winter months and the incidence was increased with number of lactation and the prevalence was more during the third post partum week.

Venkateshwarulu and Rao (1993) observed 11.1 per cent, 24.7 per cent, 16.66 per cent and 3.33 per cent incidence of ketosis in age groups <3, three to six, six to nine and above nine years respectively in dairy cows. According to them breed did not have any influence. The incidence was highest in the first month of lactation (23.52%) decreasing to 20 per cent in the second month, 10.25 per cent in the third month and 2.85 thereafter.

Radostits *et al.* (1994) recorded that ketosis occurred commonly during the first month of lactation and then the second month and very rarely during late pregnancy and cows of any age may be affected but the disease increased from a low

prevalence at the first calving to the peak at fourth. They also stated that its occurrence depended so much on management, nutrition and climate.

Clinical signs

Sjollema and Van Der Zande (1923) were the first to present the data showing abnormally high levels of acetone bodies in the blood and urine of cows exhibiting symptoms of ketosis. They observed rapid loss of body weight, lack of appetite, dry faeces, rapid decrease in milk yield and in many cases nervous disturbances. They also reported that liver of those cows that died due to ketosis was yellow and exhibited fatty degeneration.

Schultz (1968) recorded the symptoms of ketosis as loss of appetite, refusal of concentrates, depression, constipation and urine showed positive response in Rothera's colour test. In nervous form of ketosis dullness and characteristic signs like salivation, excitation, inco-ordination of the hind legs were noted. In all the cases acetone like odour in breath was also reported.

Kronfeld and Emery (1970) described ketosis as a disease with anorexia, hypolactia and ketonuria as usual signs, They also observed some behavioural changes such as lack of

allertness, blank expression and non-responsiveness to external stimuli.

According to Fox (1971) the usual signs of ketosis were diminished appetite, decreased milk yield, nervousness, profuse salivation, unusual gait, licking and sometimes unmanageably excited. The rumen was hard to touch and faeces was hard and firm.

Schultz (1974) noted that out of all cases he studied 10 per cent were nervous type of ketosis with behavioural changes like licking or sucking of inanimate objects, incoordination, depression, dullness and acetone smell in breath.

King (1979) observed ketosis with the following symptoms decreased dry matter intake and selective forage intake particularly hay in preference to concentrates. Generally temperature, respiration and heart rates were within normal range. Ruminal contraction rates were depressed and ruminal contractions were weak and incomplete. A ruminal "ping" was detected on some occasions and faeces was often found dry and scanty.

Sarode *et al.* (1981) reported that symptoms of ketosis were characterised by ketonemia, hypoglycaemia and ketonuria and occurred due to impaired carbohydrate and fat metabolisms. They evaluated the clinical findings in respect of blood

ketone and glucose levels and co-related the same with ketonuria under the stress and lactation.

Baird (1982) observed that typical ketosis occurred spontaneously in susceptible high yielders and signs include loss of appetite particularly for concentrate feeds and decreased milk production and rapid loss of body condition. Body temperature was normal and milk was positive for Rothera's test.

Dohoo and Martin (1984) indicated the production loss due to ketosis.

Rautmare *et al.* (1992) described symptoms of ketosis in buffaloes as reduced milk yield, reduced appetite for concentrates, false chewing movements, salivation and licking the body continuously. There was anorexia and ceased rumination and urine was positive for Rotheras test.

Anantwar and Singh (1993) during their work on ketosis in buffaloes noted that 87.6 per cent of the cases were wasting type and only 12.39 per cent were nervous type. They described the usual symptoms as refusal to concentrates, licking of inanimate objects, reduction in milk yield and emaciation. Urine and milk from such animals showed colour reaction with Rothera's test.

Radostits *et al.* (1994) described the signs of ketosis under two categories as wasting form and nervous form. The wasting form is characterised by decrease in appetite to concentrate but takes hay. Milk yield is reduced, weight loss and some times deprived appetite is seen. The faeces may be dry and firm. A typical description as "woody" appearance due to apparent wasting and loss of cutaneous elasticity is also described. The typical cases of nervous forms occurs suddenly and usually is bizarre. The syndrome is suggestive of delirum, circling, straddling or crossing of legs, head pushing or leaning into the stanchion, apparent blindness, wandering, aimless movements, vigorous licking, chewing movements and salivation.

Hemoglobin (Hb) and Packed cell volume (PCV)

Steger *et al.* (1972) did not notice any significant changes in the Hb concentration in ketotic cows although there were changes in glucose and ketone body concentration.

Aleyas and Alikutty (1973) reported that the mean values of haemoglobin and haematocrit value of healthy Australian Jersey and crossbred (Jersy x Sindhi) cows were 10.26 and 9.10 g% and 36.00 and 28.58 g% respectively.

According to Schalm (1975) the mean haemoglobin value in normal cattle ranged from 8.0 to 15.0 g/dl with a mean of 11.0 g/dl.

Kuusksalu (1987) observed that in ketosis of dairy cows there was reduction in haemoglobin and haematocrit.

Prasad *et al.* (1987) reported that the mean value of PCV in clinically normal crossbred dairy cows of five to eight years of age in advanced pregnancy and early lactation were 29.71 ± 1.60 and 30.71 ± 0.81 per cent, respectively.

Fatur and Jazbec (1990) did studies on post parturient ketosis in cows and observed that 6 per cent of the diseased cows showed anaemia.

Salim and Joshi (1992) stated that the mean value of PCV in apparently healthy cattle aged one to six years was 30.75 ± 4.68 per cent.

Blood Glucose

Hupka (1928) first demonstrated the hypoglycaemia associated with ketosis in cows and noted that sugar administration was beneficial for the recovery.

Shaw (1943) noted changes in blood glucose and acetone prior to, during and after the development of ketosis. The symptoms were not seen until the 9th day post-partum, although the blood glucose was low from the third day onwards. There was a gradual increase in blood acetone bodies to a high level by the ninth day, when the cow exhibited the first signs of inappetance.

Shaw (1956) conducted extensive studies on various biochemical aspects of bovine ketosis and recorded low concentration of blood glucose and high level of blood ketone bodies in ketotic cows.

Kronfeld *et al.* (1960) analysed liver glycogen levels in non-lactating, lactating and ketotic cows and reported that the plasma glucose concentration in all the cows were directly related to the liver glycogen concentration and inversely to the plasma total ketone body concentration.

Radloff and Schultz (1967) observed increased fatty acids and ketone bodies and decreased serum sugar and triglycerides in ketosis.

Sastry (1968) observed a value of 25 mg% of blood glucose in ketotic cows against the 50 mg% of healthy lactating cows.

Payne *et al.* (1970) reported that the mean blood glucose concentration among dairy animals varied from herd to herd and it ranged from 36.7 to 54.1 mg/100 ml with a mean of 45.4 mg/100 ml.

Panduranga *et al.* (1975) conducted studies on lactating and dry cows with anorexia of one to 15 days duration and found that anorectic animals without ketosis had a mean blood glucose level of 54.4 mg% as against 40.5 mg% in animals with ketosis, whereas the control cows had 60.5 mg%.

Rossow *et al.* (1976) found an increase in blood ketone bodies and a decrease in blood glucose levels with increase in milk yield. They conducted the study on large dairy units and noted that ketone body concentration was highest between eight to 14 days of lactation, while blood glucose was at its lowest level.

Reynaert *et al.* (1977) collected blood samples from ketotic and normal lactating cows and observed lowest plasma glucose and serum growth hormone levels and high serum free fatty acid levels in ketotic cows.

Horber *et al.* (1980) reported lower levels of blood glucose and elevated levels of plasma free fatty acids and ketone bodies in ketotic animals.

Kaneko (1980) recorded the blood glucose concentration in healthy adult cow as 45 to 75 mg% with a mean of 57 ± 7 mg%.

Wiener and Russell (1980) reported that the mean plasma glucose level in healthy Ayrshire female cattle aged six month to six years was 53 ± 0.6 mg%.

Sarode *et al.* (1981) observed hypoglycaemia, ketonaemia and ketonuria in clinical cases of ketosis in Sahiwal cows.

Baird (1982) remarked that the precipitating cause of ketosis was probably due to imbalance between glucose supply and requirement leading to decreased carbohydrate status and decreased insulin secretion associated with fat mobilisation and increased hepatic ketogenesis resulting in excess accumulation of ketone bodies in blood.

Grohn *et al.* (1983) carried out studies on various blood components and fatty infiltration of liver to ascertain, healthy, mildly ketotic and severly ketotic cows. They observed a positive correlation between fatty infiltration of liver and blood ketone bodies and liver specific enzymes and a negative correlation with blood glucose.

Aslan and Nimazlioglu (1985) studied the post-parturient status of blood glucose in cows and noted that blood glucose falls to 37.5 mg% in first 10 days after calving and 30.4 mg%

in 20 days against 37.5 mg% 10 days before calving. After 30 days of calving and after feeding sugar pulp with molasses the mean blood glucose value rose to 45.8 mg%.

Normal value of glucose in whole blood of healthy cattle was reported as 35-55 mg/dl (Benjamin, 1985).

Bauer *et al.* (1991) investigated the blood glucose in post-parturient cows and noted that there are three phases of blood glucose concentrations in cows within 30 days of calving. He suggested a first fall in glucose content from an initial high level at calving to a minimum at day seven. The second was a steady rise until day 26 after which a constant concentration was maintained.

Rautmare *et al.* (1992) investigated ketosis in buffaloes and observed that there was a marked hypoglycaemia (44.82 mg%) in ketotic animals.

Anantwar and Singh (1993) observed significant decrease ($P<0.01$) in blood glucose (51.95 ± 3.83 mg%) and attributed it to the rapid utilization of glucose by the mammary gland to make lactose coupled with inadequate food intake to replenish glucose supply.

Hayatgheybi and Singh (1993) studied the biochemical status of healthy and ketotic buffaloes and noted

hypoglycaemia (48.03 ± 0.69 mg%) ketonemia (15.53 ± 0. 64 mg%) and hypocalcaemia (7.23 ± 0.14 mg%) as the constant findings in ketotic animals.

Nakajima *et al.* (1993) noted two levels of glucose content in blood in correlation with ketone content in urine. When the urine content of ketone was about 10 mg%, the blood glucose was 42.0 ± 10.4 mg% and when the urine content of ketone was less than 10 mg%, the mean blood glucose was 60.6 ± 8.1 mg%.

Radostits *et al.* (1994) stated that blood glucose are reduced from the normal of 50 mg% to 20-40 mg% in cattle with ketosis. They noted that the severity and rate of onset of signs were correlated with the blood glucose level than the ketone body levels in blood.

Venkateshwarulu *et al.* (1994) noted a significant increase in blood glucose and decrease in blood ketone body level after the ketotic cows were treated with glucose and glycerin.

Anantwar *et al.* (1995) conducted treatment trials for ketosis on buffaloes and noted a pretreatment blood glucose level of 51.68 ± 2.36 mg% and a post treatment level of 65.58 ± 2.54 mg%.

Hamakawa *et al.* (1995) noted a decrease in blood glucose starting at day five post-partum. In clinically normal control cows the blood glucose level recovered by 14th day, but in those animals which had ketosis the glucose remained as reduced even after the 14th day post-partum.

Blood urea nitrogen

Payne *et al.* (1970) recorded the serum urea nitrogen level ranging from 9.5 to 20.5 mg% with the mean value of 14.9 mg% in healthy cows.

Blowey (1975) in his study indicated that when cows had low blood glucose level there was chance for high values of plasma urea nitrogen, indicating a high level of metabolic nitrogen circulating within the body.

Kondrakhin (1976) recorded a positive correlation between BUN and milk urea in cows with ketosis.

Zhabolenko (1976) noted that in ketosis along with blood glucose and albumin, BUN was also reduced, but ketone bodies and serum lipids were increased.

Kaneko (1980) reported that the nromal value of serum urea nitrogen in domestic animals in general ranged from 10-30 mg%.

According to Benjamin (1998) the normal value of blood urea nitrogen in cattle ranged from 10-30 mg per cent.

Ketone bodies in blood

Sjollema and Van Der Zande (1923) were the first persons to give reports of acetone bodies in blood and urine of cows exhibiting symptoms of ketosis.

Henry (1969) reported spectrophotometric methods of estimation of acetone and acetoacetic acid from blood. Preformed acetone and acetone derived from aceto acetic acid is isothermally distilled into alkaline vanillin, where they react to form red vanillal acetone or divanillal acetone. According to him the normal blood ketone body level is 0.5 to 3 mg%.

Rajan and Ganapathy (1973) in their study noted that average blood ketone body levels in healthy cows three days to six months after parturition was 1.3 to 1.9 mg% with a mean of 1.5 mg%. But in ketotoic cows, 15 days to nine months after calving with complete anorexia and suspended rumination, the range was 7.2 to 26 mg% with a mean of 12.4 mg%.

Vikharev (1975) reported that normal level of ketone bodies in blood of cows was less than 7 mg%.

Rossow *et al.* (1976) reported that in cows with yield of 30 kg per day blood glucose was significantly lower and ketone body levels significantly higher than in cows with lower yields and noted that ketone body concentration was highest between the 8th and 14th day of lactation, while blood glucose was at its lowest level.

Schafer and Bethe (1976) carried out works on 25 herds of cattle and noted that subclinical ketosis started occurring at serum concentrations of 5 mg% of ketone bodies.

Schwalm and Schultz (1976) reported that cows with ketosis showed depressed milk yield and serum insulin and elevated levels of blood acetone, plasma free fatty acid and cholesterol.

Huhold and Thiemann (1979) observed by microdistillation technique that the normal value of serum ketone bodies in medium yielding cows (3000 to 4000 kg/year), two to six weeks postpartum is 5.92 ± 2.47 mg/100 ml and later than six weeks postpartum is 4.35 ± 0.94 mg/100 ml. In high yielders (over 4000 kg/year) corresponding values were 5.87 ± 2.17 and 5.20 ± 2.22 respectively.

Horber *et al.* (1980) estimated concentration of ketone bodies, acetoacetic acid and beta-hydroxy butyric acid in blood at different stages of lactation and found that they

were significantly higher for cows with primary ketosis than for the healthy cows examined. They also noted that during the period of highest milk yield ketone body concentration tended to increase even in healthy cows, an effect that is considered to be of physiological origin.

Margolles *et al.* (1988) studied biochemical characteristics of ketosis in Holstein cows and noted the changes in ketone bodies, glucose, calcium, magnesium, inorganic phosphorous, cholesterol and total lipids after 40 days of postpartum. Out of the animals examined 19.47% showed hyper acetonemia (more than 5.75mg %) and 15.67 per cent showed hypoglycemia (less than 24.06 mg%).

In ketotic buffaloes, Singh and Kasraliker (1990) found ketone body levels of 15.53 ± 0.64 mg% in blood.

Anantwar and Singh (1993) recorded that there was a decrease in ketone bodies in serum from 14.56 ± 1.5 mg% to 1.96 ± 0.15 mg% when the ketotic buffaloes recovered from the disease after the treatment.

Hayatgheybi and Singh (1993) in a study on ketotic buffaloes noted that hypoglycemia and ketonemia are consistent and the mean values of serum ketone bodies were 15.53 ± 0.64 mg%, against 2.0 ±0.11 mg% in healthy ones.

Nakajima *et al.* (1993) noted that, the blood ketone body level vary from 6.1 ± 6.7 to 38.3 ± 8.6 mg % 15-20 days after parturition and after 40-60 after parturition, it was reduced to 2.3 ± 3.1 to 11.1 ± 11.8 mg % in the same group.

Radostits *et al.* (1994) noted that hypoglycemia, ketonemia and ketonuria are characteristic of ketosis and the blood ketone body levels were elevated from a normal of less than 10mg% to 10 mg to 100 mg%.

Venkateshwarulu *et al.* (1994) noted by clinical treatment trials on ketosis in dairy cows that after recovery there was increased glucose and decreased ketone body levels in blood.

Calcium

Robertson *et al.* (1956) explained that hypocalcemia in normal cows at parturition was caused by drain of calcium into milk.

Halse and Valle (1958) reported low blood calcium levels in ketotic cows but the significance was not recorded.

The normal serum calcium level in cattle ranged between 8.0 to 12.0 mg per cent out of which 3.6 to 7.7 mg per cent existed in the diffusible ionised form and the remaining in non-diffusible protein bound form (Moodie, 1960).

Mylrea and Bayfield (1968) reported the serum calcium concentration in apparently healthy dairy cattle as 10.2 ± 0.56 mg per cent.

Cote *et al.* (1969) noted in bovine ketosis that although ketone body levels were raised, plasma glucose and serum calcium levels were depressed.

Green (1975) recorded in his study that hypercalcaemia was noted in ketosis. But he concluded that ketosis had little effect on the serum calcium levels in animals.

Yoshida (1978) noted in his study on ketosis in bovine animals that there was a decrease in mean serum calcium levels from 2.08 to 1.97 m mol/lit.

Kaneko (1980) stated that the normal serum calcium level in normal adult animal averaged 10 mg per cent and ranged between 9.0 to 12.0 mg per cent.

Margolles *et al.* (1988) observed hypocalcaemia in case of hyperacetonaemia in Holstein cows within forty days of parturition.

Goncharova *et al.* (1990) observed low levels of calcium in serum of ketotic cows.

Nikalaenka and Syamenau (1990) observed hypocalcaemia in cows during stall fed period in Russia and noted a recovery from subclinical ketosis when those animals were treated with calcium chloride combined with methionine.

Singh and Kasaralikar (1990) observed in buffaloes that there was slight hypocalcaemia (7.23 ± 0.14 mg%) occurring in ketosis along with ketonaemia and hypoglycaemia.

Lean *et al.* (1992) correlated fatty infiltration of liver with hypocalcemia.

Rautmare and Anantwar (1993) reported slight hypocalcaemia in buffaloes with ketosis coupled with hypoglycaemia and a significant change in inorganic phosphorous.

Fatur and Trenti (1994) investigated serum mineral status in ketotic dairy cows and recorded a calcium level of 2.42 ± 0.18 millimols/lit.

Radostits *et al.* (1994) noted a small but a significant fall in serum calcium levels down to about 9 mg per cent in ketosis.

Phosphorus

Shaw (1956) noticed hypophosphataemia in ketotic cows and explained that it was associated with poor nutritional condition of the animals.

Cote *et al.* (1969) could not detect any changes in serum phosphorus value in ketotic cows although plasma glucose and serum calcium were depressed.

Payne *et al.* (1970) reported that the mean serum inorganic phosphorus level in dairy cattle was 5.42 mg/dl and its range varied from 3.6 to 7.2 mg/dl.

Caple *et al.* (1977) reported hyperphosphataemia in ketotic cows.

Rashid (1977) noted a mean inorganic phoshorus level of 5.7 mg per cent of serum in Ayrshire dairy cows during his one year study in several farms.

Yoshida (1978) observed a decrease in inorganic phosphorus level in serum from 2.23 mmol/lit to 1.60 mmol/lit during ketosis in cows.

Kaneko (1980) observed that serum inorganic phosphorus level in normal adult cattle varied from 4.0 to 7.0 mg% (1.3 to 2.3 mmol/lit).

Prasad *et al.* (1987) reported that the mean serum inorganic phosphorus values in normal crossbred dairy cows aged five to eight years during advanced pregnancy and early lactation were 6.50 ± 0.41 and 5.47 ± 0.27 mg/dl respectively.

Margolles *et al.* (1988) recorded a hyperphosphataemic state in 17.85 per cent of the 180 ketotic cows studied.

Fatur and Jazbec (1990) did a metabolic profile study in ketotic cows and noted hyperphosphataemia in 25 per cent of cases studied.

Singh and Kasaralikar (1990) observed that there was no significant changes in plasma inorganic phosphorus in recently calved ketotic buffaloes although there were hypoglycaemia, ketonaemia and hypocalcaemia.

Swenson and Reece (1993) and Reece (1997) noted a normal level of serum phosphorus as 2 to 6 mg/dl in normal cows.

Fatur and Trenti (1994) recorded a level of 1.93 ± 0.44 mmol/lit of phosphorus in ketotic dairy cow.

Magnesium

Duncan *et al.* (1939) could not detect any changes in plasma magnesium in ketotic cows when compared with normal cows.

Breirem *et al.* (1948) reported hypomagnesaemia associated with ketosis.

Cote *et al.* (1969) found no change in serum magnesium concentration in ketotic cows although plasma glucose and serum calcium were depressed.

Payne *et al.* (1970) reported that the mean value of serum inorganic phosphorus in normal dairy cows was 2.58 mg/dl which ranged from 2.0 to 3.1 mg/dl.

Rayssiguier (1977) correlated elivated fatty acid levels in serum associated with fatty infiltration of liver to hypomagnesaemia in ketosis.

Magnesium concentration was low (0.75 ± 0.95 mmol/lit) throughout the year in cows which frequented mid summer ketosis (Yoshida, 1978).

The mean serum magnesium level in dairy cows 14 to 21 and 38 to 45 days post partum were 2.24 ± 0.08 and 2.23 ± 0.70 mg/dl respectively (Larson *et al.*, 1980).

Hypomagnesemia was detected in 9.67 per cent of hyperacetonaemic cases in a study done in Cuba (Margolles, 1988).

Goncharova *et al.* (1990) reported a normal level of magnesium in cows during lactation.

Swenson and Reece (1993) and Reece (1997) reported that normal cows had serum megnesium value of 1.5 to 2.5 mg/dl.

Sodium

The normal serum sodium in apparently healthy dairy cows ranged from 131 to 152 mEq/lit and the mean value was 141 ± 5.2 mEq/lit (Mylrea and Bayfield, 1968).

Payne *et al.* (1970) reported the normal serum sodium level in dairy cows ranged from 135.00 to 143.10 mEq/lit and the mean value was 139.00 mEq/lit.

Rashid (1977) recorded a mean value of sodium in dairy cows in 314.9 mg% and stated that there is a positive correlation between serum sodium and ketone bodies.

Fatur and Jazbec (1990) reported a reduced value for serum sodium in 49 per cent of ketotic cases be studied.

Salim and Joshi (1992) reported that the serum sodium level in apparently healthy cows was 142.68 ± 3.51 mEq/lit.

Potassium

Carlstrom (1950) reported low levels of potassium compared to normal in cases of ketosis in bovines.

Saarinen and Shaw (1950) noted that there were no significant alteration in plasma potassium, inorganic phosphorus or blood chlorides in bovine ketosis.

Mylrea and Bayfield (1968) reported that the normal serum potassium levels in apparently healthy dairy cattle was 4.7 ± 0.48 and it ranged from 3.7 to 5.7 mEq/lit.

Fatur and Trenti (1994) stated that the value of serum Ca, Mg, P, Na and K in ketosis were different from normal cows and the value of K in ketosis was 5.00 ± 0.64 mmol/lit.

Serum potassium values in normal healthy cows varied from 3.9 to 5.8 mEq/lit (Kaneko, 1980; Benjamin, 1998 and Radostits *et al.*, 1994).

Total protein

Mylrea and Healey (1968) reported that the mean serum protein level in apparently healthy dairy heifers and cows

were 6.90 ± 0.66 and 7.90 ± 0.64 g/dl with value ranging from 5.5 to 8.2 and 6.6 to 9.2 g/dl respectively.32

Steger *et al.* (1972) recorded that they could not detect any change in the serum total protein concentration in ketotic cows, when compared with the normal controls.

Schalm *et al.* (1975) observed that the mean serum total protein in healthy cattle was 6.6 to 8.0 g/dl.

In a study Savoiskii and Fedorov (1976) recorded reduction in total blood protein in ketosis. The average value in ketotic cases was 8.16 g% against normal values of 7.62 to 9.35 g% in normal.

Zhabolenko (1976) noted a higher level of total protein, lipids and ketone bodies in the serum of ketotic cows.

Reece (1997) reported a normal value of 5 to 8 g/dl of total protein in serum of normal cows.

Albumin

Mylrea and Healy (1968) reported that the normal serum albumin value in apparently healthy cows was 8.2 ± 0.43 g/100 ml.

The normal serum albumin levels in Sahiwal and crossbred cattle during pre and post parttum, seventh week of lactation and non-lactating periods were 3.40 ± 0.07, 3.21 ± 0.08, 3.17 ± 0.06 and 3.25 ± 0.06 g/100 ml respectively (Singh and Choudhary, 1988).

Jovanovic *et al.* (1990) noted a condition of hypoalbuminaemia in 91 per cent of the ketosis cases he dealt with.

The normal range of serum albumin value of 2.1 to 3.6 g/100 ml in cattle was reported by Radostits *et al.* (1994).

Asmare *et al.* (1997) recorded that although plasma ketone bodies were increased in clinical ketosis, there was a reduction in plasma albumin level.

Globulin

McLennan and Willoughby (1973) observed that there was a increase in the serum globulin during the post calving period.

Singh and Choudhary (1988) noted gradual increase in the serum globulin concentrations in postparturient cows till 7th week and then it showed decrease.

According to Swenson and Reece (1993) reported that the value of serum globulin in normal cows was 3.6 to 4.4 g/dl.

Radostits et al. (1994) suggested that a normal value of serum globulin as 3.9 to 5.6 g/dl in cows.

Rajora and Pachauri (1994) recorded that although albumin concentrations did not vary between pre and post-parturient periods, there was an increase in the globulin during post parturient period resulting in an increase in total protein values and a decrease in A:G ratio. They observed a mean value of serum globulin in post parturient cows as 4.142 ± 1.962 g%.

Ketone bodies in urine

Sastry (1968) observed the normal values of urinary ketones in healthy cattle as 0-15 mg%. He reported that in cases of ketosis the values might go upto 500 mg per cent or above.

Schultz (1968) noted pine-purple colour developing when acetone or acetoacetic acid react with sodium nitroprusside and gave the opinion that it could be used to detect ketones in samples. He further reported that Rothera's test did not measure beta hydroxybutyrate in milk and urine.

Kaneko and Cornelius (1970) emphasised the significance of hypoglycaemia and ketonaemia in the detection of ketosis

and commented that measurement of ketone bodies in blood can be replaced by detection of it in urine and milk.

Fox (1971) did trials with Rothera's test in urine for the detection of clinical ketosis. He, after allowing the solution to stand for 15 minutes, read the test on the basis of colour intensity developed, ie no colour - negative, slight lavender(+), deep lavender - (++), beet red or purple - (+++) and deep beet red - (++++).

Kronfeld (1972) observed that the nitroprusside test (Rothera's test) gave a purple colour when it reacted with acetoacetate and acetone and not with betahydroxybutyrate. He later noted that the test was sensitive to acetoacetate levels of 1 mg% and acetone levels of 2 mg%.

Rossow *et al.* (1976) recommended that for proper early diagnosis of ketosis ketone body and blood glucose levels in blood be measured in the second week after parturition and the detection of urinary acetone could only be used as a diagnostic test in herds where ketosis is not suspected and it cannot be used to determine the distribution of sub-clinical ketosis in a herd.

Radostists *et al.* (1994) suggested that quantitative estimation of urinary ketones may be unsatisfactory because of the wide variation that may occur depending on the concentration of the urine. They observed that in clinically normal cattle urinary ketone levels may go up to 70 mg%; although usually they are lower than 10 mg%.

Ketone bodies in milk

Emery *et al.* (1964) observed that Rothera's test could indicate ketone bodies up to 2 mg% levels in milk.

Kronfeld (1972) observed a close correlation between blood ketone body concentration and acetone content in milk. They noted that when blood ketone body concentration was less than 5 mg%, acetone content of milk was less than 0.3 mg per cent, but when blood concentration exceeds 5 - 10 mg per cent, the milk acetone concentration was raised from 0.2 - more than 6 mg per cent and when blood ketone levels exceeds 10 mg%, the acetone in milk was also increased.

Shultz (1974) noted that Rothera's test on milk and urine are indicators of ketosis in animals. He also stated that milk test gave more accurate results and became positive when blood ketone body levels became 10-15 mg%.

Piatkowski *et al.* (1974) drew the conclusion that examination of milk ketone bodies by Rothera's test gave a more reliable, but a technically simple and more convenient means of diagnosing ketosis than examination of blood samples.

Mironov (1975) stated that in severe cases of ketosis the ketone body concentration in milk may rise upto or above 50 mg%.

According to Kondrakhin (1976) the levels of ketone bodies in blood and milk were increased in ketosis but the level of urea decreased slightly and concluded that ketone bodies and urea measurement in milk is to be adopted instead of blood measurement for the routine checking of ketosis.

Andersson and Emanuelson (1985) reported milk acetone concentration of above 0.40 millimols/lit be conclusive of hyperketonemia.

Heydrych and Wieckowski (1991) improvised a colour test for the detection of ketone bodies in milk for the diagnosis of ketosis. Acetone concentration in milk was evaluated using 1 ml of milk and 3 ml of KOH and concentrated salicilaldehyde. Development of light orange and dark orange colour corresponds with 149, 370 and 1169 micromols/ml of acetone respectively.

Nielen *et al.* (1994) stated that when blood betahydroxybutarate concentration exceeds 1. 4m Mols/lit, the milk test has a sensitivity of 90 per cent and specificity of 96 per cent. But at the same instance urine test lacked specificity (less than 67%), but the sensitvity was 100 percentage. They also stated that the milk and urine are useful in detecting ketosis, milk test is preferred due to its easy obtainability combined with overall better test characteristics.

Radostits *et al.* (1994) reported that ketone levels in milk was rather less variable, ranging from a normal of 3 mg% to an average level of 40 mg% in ketosis.

Materials and Methods

MATERIALS AND METHODS

The study was conducted at College of Veterinary and Animal Sciences, Mannuthy over the period of three semesters during the year 1995-97.

Epidemiology

Data on the occurrence of ketosis among dairy cows for the last six years was collected from the Veterinary College Hospital, Mannuthy.

Detailed anamnesis of the clinical cases studied were recorded using a proforma.

Details of age, breed, parity, stage of lactation, ration, milk yield etc. were also noted.

Experimental design

Twenty dairy cows affected with primary ketosis which attended the out-patient unit of the Veterinary College Hospital, Mannuthy and field cases randomly selected formed the experimental group. They were subjected to metabolic profile test which consisted of estimation of Haemoglobin(Hb), Packed cell volume (PCV), blood glucose, Blood Urea Nitrogen (BUN), total serum proteins, albumin and globulin, serum

Calcium (Ca), Phosphorus (P) and Potassium (K). Estimation of ketone bodies in blood and detection of ketone bodies in urine and milk were also done.

Twelve healthy dairy cows maintainted under identical field conditions during the vulnerable period for ketosis i.e., within two months of calving selected at random constituted the control group. They were also subjected to Compton metabolic profile test and the results were compared with the experimental group.

Parameters Studied

Clinical signs

Clinical signs shown by the animal at the time of clinical examination and that obtained from the history were also recorded.

Haemoglobin and Packed cell volume

Five millilitre of blood was collected in clean vial with EDTA as anticoagulant and estimated haemoglobin and Packed Cell Volume as per the method of Schalm (1975).

Blood biochemistry

Fifteen millilitre of blood was collected in a screw capped test tube for the separation of serum and three millilitre of blood was collected in another vial with sodium fluoride as anti coagulant for blood glucose estimation.

The following estimations were done

Blood glucose was estimated by the method of Folin and Wu (1920).

Blood urea nitrogen estimation was carried out Diacetyl monoxine method (Wybenga, 1971).

Estimation of blood ketone bodies was done according to Henry (1969).

Total serum protein and albumin were estimated by Biuret and Bromocresol green dye binding methods (Doumas, 1978) and globulin was calculated by difference.

Serum calcium and phosphorus were estimated by O-cresolphthalein (Barnet, 1973) and modified metol methods (Daly, 1972) respectively.

Estimations of sodium, potassium and magnesium were done by Atomic Absorption Spectrophotometry (AOAC, 1992).

Urinanalysis

Ketone body level in urine was detected using Rothara's test and expressed as traces (+), moderate (++), severe (+++) and very severe (++++).

Milk

Milk samples were analysed for ketone bodies using Rothera's test.

Treatment

All the cases studied were given replacement therapy consisting of Dextrose 25% and Beplex forte given intravenously and oral antacids containing sodium bicarbonate (100 g), Pulvis Nuxvomica (30 g), Magnesium carbonate (100 g) and Pulvis Chiretta (60 g) for one to two days.

Assessment of milk production and economic loss

Economic loss was calculated by adding together the actual loss in milk during the disease, and the cost of treatment expressed in rupees.

Statistical analysis

The data collected were analysed as per Snedecor and Cochran (1980).

Results

RESULTS

Epidemiology

Data on the occurrence of bovine ketosis was collected from the records of the Veterinary College Hospital, Mannuthy during the period 1993-98. Out of the total numberof 16175 bovine animals presented, 227 (1.4%) were found to be suffering from primary ketosis. The year and monthwise occurrence recorded are presented in Table 1. Detailed epidemiological study conducted on 20 animals during the period indicated that the incidence was more during the month of March to May. The animals affected were in the age groups of three to seven years and were 9-60 days postpartum. Though crossbred Jersey, crossbred brown Swiss and crossbred Holstein Friesian cows were found affected, it was more in the crossbred Jersey cows. Parity study indicated that animals in the second or third lactation were more affected. All the animals studied were reported to be in good health condition before the onset of the disease. The details are presented in Table 2.

The animals of the experimental group were maintained on various types of feed ingredients namely rice gruel, groundnut

cake, coconut cake, rice or wheat bran etc. without observing any standard recommendations and forage was provided *ad libitum*. The control group animals were fed with compounded concentrates at the rate of approximately 400 g/kg milk produced, mineral suppliments and fodder *ad libitum* were also given.

Clinical signs

The clinical data of healthy and ketotic cows are presented in Table 3. The respiration, pulse and rectal temperature ranged between 68-98/min, 36-48/min and 100.4 to 102.6°F respectively.

The conjunctival mucous membrane was pale to pale roseate. In most of the cases the frequency of rumination was reduced. Ruminal contractions were weak and ranged from three to five/five min. Body wasting was a consistant finding. There was significant reduction in appetite and milk yield. Though most of the animals continued to consume roughages in variable quantities there was total refusal of concentrates by all the animals. The dung was hard, scanty, coated with mucus and passed with difficulty. While in the byre the animals exhibited nervous signs like head pushing, leaning on to wall, manger, supporting crossbars of manger and frequently falling were exhibited by severe cases. Champing of jaws with

salivation and biting the crossbars of the manger, chain, rope etc. were also noticed. Some cows were dull, depressed, disinclined to move and non responsive to external stimuli. But some were hyperaesthetic and unmanageably excited. Majority of the animals had a typical acetone smell in their breath.

Analysis of blood

Haemoglobin

The values of haemaglobin observed in healthy and diseased cows are presented in Tables 4 and 5 respectively. The values obtained in healthy animals varied between 7.0 and 13.0g% and that of diseased group between 6.2 and 10.8g%. The mean value of haemoglobin obtained in healthy normal cows was 9.37 ± 0.16 g per cent and that of diseased cows was 8.66 ± 0.24 g per cent. There was no significant difference between the mean values of healthy and diseased animals.

Packed cell volume (PCV)

Packed cell volume values observed in healthy and diseased cows are presented in Tables 4 and 5 respectively. The PCV values in healthy animals ranged between 22 and 45 per cent with a mean of 31.83 ± 2.50. In the diseased animals the values were in the range of 22 to 40 per cent with a mean of

32.35 ± 1.03. There was no significant difference in the mean values between the two groups.

Blood glucose

Blood glucose concentrations noticed in healthy and diseased cows are presented in Tables 4 and 5 respectively. The blood glucose level varied between 42.5 and 69.8 mg per cent with a mean of 54.51 ± 2.48 mg% in healthy cows. The values were between 25.2 and 43.0 mg% with a mean of 33.80 ± 0.99 mg% in ketotic cows. The difference in values was found to be statistically significant ($P<0.05$).

Blood urea nitrogen

The range of values of blood urea nitrogen observed in healthy and diseased cows are presented in Tables 4 and 5respectively. The minimum and maximum values observed in healthy and the experimental group were 17.54 and 24.28 mg% that in healthy group were 16.19 and 40.74 mg% respectively. The mean values obtained in healthy and diseased cows were 20.64 ± 0.62 mg% and 28.37 ± 1.89 mg% respectively. The differences in values between the two groups were statistically significant ($P<0.05$).

Calcium

Serum calcium levels obtained in healthy and diseased cows are presented in Tables 4 and 5 respectively. The values obtained in both healthy and diseased group ranged between 7.0 to 11.5 and 5.59 to 10.37 mg% respectively with a mean values of 10.01 ± 0.40 mg% and 9.13 ± 0.26 mg% respectively. The difference in values was statistically insignificant.

Phosphorus

Serum phosphorus values in healthy controls and diseased cows are presented in Tables 4 and 5 respectively. The values varied from 3.79 to 6.80 and 2.83 to 5.43 mg% in healthy and diseased cows respectively. Their mean values were 5.40 ± 0.29 mg% and 3.96 ± 0.13 mg% respectively. The difference in values was statistically significant ($P<0.05$).

Magnesium

Magnesium levels in serum of healthy cows are presented in Tables 4 and that in the diseased cows are presented in Table 5. The values were in the range of 1.95 to 3.7 mg% in healthy cows and 1.20 to 2.85 mg% in ketotic cows respectively. The mean value of serum magnesium in healthy cows was 2.58 ± 0.13 mg% and that in the diseased cows was

1.90 ± 0.13 mg%. The difference in value was statistically significant (P<0.05).

Sodium

Serum sodium contents in healthy and diseased cows are presented in Tables 4 and 5 respectively. The minimum and maximum values obtained in healthy group were 130.11 and 167.29 mmols/lit and that in the diseased group were 119.35 and 167.29 mmols/lit respectively. The mean values of serum sodium in healthy cows were 147.11 ± 3.12 mmol/lit and that in diseased cows were 142.99 ± 3.01 mmol/lit. The difference in values was statistically not significant.

Potassium

Potassium contents in the serum of healthy and diseaed cows are presented in Tables 4 and 5 respectively. The concentrations varied between 3.82 and 5.4 with a mean of 4.62 ± 0.15 mmol/lit in healthy cows and 3.62 and 6.53 with a mean of 4.50 ± 0.20 mmol/lit in ketotic cows respectively. The difference in values was statistically not significant.

Total protein

Serum total protein values of healthy cows are presented in Table 4 and the values of diseased cows are presented in

Table 5. The minimum and maximum values noted in healthy cows were 5.70 and 8.93 g% and that in the diseased group were 4.10 and 7.73 g% respectively. The mean values of total protein of healthy and diseased cows were 7.47 ± 0.29 g% and 5.62 ± 0.24 g% respectively. The difference in values was statistically significant ($P<0.05$).

Albumin

Serum albumin concentrations of healthy cows are presnted in Table 4 and that of diseased cows in Table 5. The values were in the ranges from 2.8 to 4.7 g% in healthy cows with a mean of 3.56 ± 0.16 g%. In diseased cows the values were in the range from 1.76 to 4.65 g% with a mean of 2.93 ± 0.18 g%. The difference in values was statistically significant ($P<0.05$).

Globulin

Globulin concentrations in serum of healthy and ketotic cows are presented in Tables 4 and 5 respectively. The values ranged between 1.0 and 4.81 with a mean of 3.91 ± 0.32 g% in healthy cows and 1.96 and 3.57 with a mean of 2.71 ± 0.11 g% in ketotic cows respectively. The difference in values was statistically significant ($P<0.05$).

Ketone bodies in blood

Blood ketone body concentration of healthy and keotic cows are presented in Tables 4 and 5 respectively. The minimum and maximum values of healthy blood ketone bodies in cows were 2.15 and 7.63 mg% and that in the diseased cows were 16.1 and 33.2 mg% respectively. The mean blood ketone body concentration of healthy cows was 4.17 ± 0.51 mg% and that in the diseased group was 26.13 ± 1.09 mg% and found that the difference in values was statistically significant ($P<0.05$) and there was negative correlation (-0.776) with blood glucose concentration.

The results of analysis of blood are compared and presented in Table 6 and Fig.1.

Urine ketone bodies

All the cases were positive for Rothera's test for varying degrees (Table 7). Out of the twenty cases studied 17 were having very severe (++++) reaction and three had severe (+++) reaction.

Milk ketone bodies

All the cases studied were positive for ketone bodies by Rothera's test (Table 7).

Milk production and economic loss

Average cost of treatment, including cost of medicines, conveyance charges, service charges etc. came to around Rs.300/-. Average milk yield of the affected animal before the onset of the disease was 8.5 l/day. The course of the illness was found to be on an average of 5.75 days. Milk yield due to the disease reduced by an average of 2.3 l/day.

There was an average loss of 13.23 l of milk due to ketosis per animal. As the local sale price of milk is Rs.11/l the economic loss due to actual loss of milk production is found to be (11 x 2.3 x 5.75) = 145.5 = Rs.146. Hence the minimum economic loss arising from the loss of milk production and cost of treatment comes to about 146 + 300 = Rs.446/- (Table 8).

Table 1. Incidence of ketosis recorded at Veterinary, College Hospital, Mannuthy during the period from 1993-1998

	January		February		March		April		May		June		July		August		September		October		November		December	
Year	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B
1993	2	243	3	237	4	251	5	192	2	272	1	181	3	217	2	240	3	226	1	225	2	202	3	200
1994	2	233	3	211	6	209	5	198	3	184	3	148	4	210	4	242	3	186	3	223	3	184	2	195
1995	1	217	3	193	5	200	3	186	6	188	4	174	3	225	3	221	3	208	2	245	2	226	1	216
1996	1	214	4	206	3	195	3	179	2	196	1	230	4	234	4	247	3	223	2	224	1	167	2	249
1997	3	234	2	186	5	225	5	198	3	183	2	207	3	251	3	225	4	269	2	204	5	272	4	250
1998	2	267	4	249	7	316	4	241	6	228	3	213	4	311	4	301	2	234	4	287	5	282	4	420

A = Number of ketotic cases/month
B = Number of bovines attended the clinics
Total cases = 16175
Total ketotic cases = 227 (1.4%)

Table 2. Epidemiological details of ketotic cows

Case No.	Age	Parity	Breed	Days of lactation
1	6	2	CBJ	60
2	6	2	CBBS	9
3	7	3	CBHF	28
4	5	2	CBBS	30
5	3	1	CBHF	17
6	4	2	CBJ	45
7	5	2	CBJ	24
8	4	1	CBJ	29
9	4	1	CBJ	21
10	7	3	CBHF	35
11	7	4	CBJ	33
12	6	3	CBBS	41
13	3	1	CBBS	12
14	3	1	CBHF	14
15	7	3	CBBS	16
16	7	3	CBBS	28
17	5	2	CBJ	30
18	7	3	CBHF	35
19	8	4	CBJ	19
20	6	2	CBJ	14

CBJ - Crossbred Jersey
CBBS - Crossbred Brown Swiss
CBHF - Crossbred Holstein Friesian

Table 3. Clinical data of healthy and ketotic cows

Healthy cows					Ketotic cows				
Case No.	Temperature (°F)	Pulse rate (/min)	Respiration rate (/min)	Mucous membrane	Case No.	Temperature (°F)	Pulse rate (/min)	Respiration rate (/min)	Mucous membrane
1	101.2	72	32	Pale roseate	1	102.0	88	46	Pale roseate
2	100.8	78	28	Pale	2	102.4	78	40	Pale roseate
3	101.8	82	28	Pale roseate	3	10.20	76	38	Pale roseate
4	102.0	88	36	Pale roseate	4	100.8	86	42	Pale roseate
5	101.6	76	30	Pale roseate	5	101.0	74	46	Pale roseate
6	102.4	70	26	Pale roseate	6	101.8	84	38	Pale roseate
7	101.0	74	24	Pale roseate	7	100.8	68	36	Pale roseate
8	102.8	72	26	Pale roseate	8	100.4	76	40	Pale roseate
9	100.6	78	32	Pale roseate	9	102.0	72	42	Pale roseate

Contd.

Table 3 (Contd.)

10	101.4	84	28	Pale roseate	10	100.8	84	42	Pale roseate
11	101.6	86	34	Pale roseate	11	101.0	90	38	Pale roseate
12	102.2	86	36	Pale roseate	12	102.4	88	44	Pale
					13	100.6	86	38	Pale roseate
					14	101.2	98	48	Pale roseate
					15	101.6	78	36	Pale roseate
					16	102.0	76	42	Pale roseate
					17	101.2	72	38	Pale
					18	101.8	94	44	Pale roseate
					19	101.6	78	46	Pale roseate
					20	102.2	70	40	Pale roseate

Healthy cows		Ketotic cows	
Average temperature	= 101.6°F	Average temperature	= 101.4°F
Average pulse rate	= 79/min	Average pulse rate	= 80/min
Average respiration rate	= 30/min	Average respiration rate	= 41/min

Table 4. Haematological and biochemical values of healthy control cows

Sl.No.	Hb (g%)	PCV (%)	Blood glucose (mg%)	Blood ketone bodies (mg%)	BUN (mg%)	Calcium (mg%)	Phosphorus (mg%)	Magnesium (mg%)	Sodium (mmol/ lit)	Potassium (mmol/ lit)	Total protein (g%)	Albumin (g%)	Globulin (g%)
1.	7.8	22	62.90	5.21	23.33	7.00	3.79	2.40	156.31	4.25	6.21	2.90	3.31
2.	7.0	26	47.08	7.63	19.91	11.00	6.32	2.70	133.37	4.56	7.60	3.53	4.07
3.	9.0	28	52.47	3.52	20.90	11.50	6.13	3.00	138.91	4.22	8.22	3.61	4.61
4.	9.2	26	56.83	2.15	18.34	10.50	5.94	2.30	167.29	4.94	7.83	3.41	4.42
5.	7.4	22	69.80	6.71	20.19	8.78	4.28	3.70	145.98	5.40	5.70	4.70	1.00
6.	9.0	38	60.35	3.18	24.28	11.08	6.80	2.35	153.59	5.06	8.93	4.22	4.71
7.	8.0	22	42.50	2.18	22.22	10.51	5.08	2.50	130.11	3.82	6.65	3.84	2.81
8.	7.0	32	58.75	3.07	17.54	7.96	5.25	2.70	150.33	3.96	8.62	3.88	4.74
9.	12.0	44	55.20	2.76	23.17	10.56	4.81	2.80	155.12	5.36	7.80	3.40	4,40
10.	12.0	45	59.90	5.28	18.75	11.23	6.80	2.15	141.09	4.87	7.61	3.27	4.34
11.	11.0	40	44.35	4.72	19.68	9.78	4.56	2.40	153.37	4.22	7.96	3.15	4.81
12.	13.0	37	43.85	3.61	19.41	10.26	5.03	1.95	139.80	4.73	6.47	2.80	3.67
Mean	9.37	31.83	54.50	4.17	20.64	10.01	5.40	2.58	147.11	4.62	7.47	3.56	3.91
±SE	0.16	2.50	2.48	0.51	0.62	0.40	0.29	0.13	3.12	0.15	0.29	0.16	0.32

Table 5. Haematological and biochemical values in ketotic cows

Sl.No.	Hb (g%)	PCV (%)	Blood glucose (mg%)	Blood ketone bodies	BUN (mg%)	Calcium (mg%)	Phosphorus (mg%)	Magnesium (mg%)	Sodium (mmol/) lit)	Potassium (mmol/lit)	Total protein (g%)	Albumin (g%)	Globulin (g%)
1	9.0	38	30.70	28.00	27.94	9.31	4.21	2.05	150.54	3.95	4.93	2.85	2.80
2.	10.2	32	33.80	27.58	17.80	8.93	3.56	1.75	131.64	4.29	5.62	2.83	2.79
3.	10.8	32	34.00	29.23	39.64	9.54	4.17	1.74	133.37	5.92	4.64	2.61	2.03
4.	9.0	36	38.50	26.43	24.36	8.64	4.04	1.20	167.29	4.17	5.27	2.44	2.83
5.	8.8	32	43.00	21.83	40.74	10.25	3.50	2.80	164.25	4.14	4.77	2.45	2.31
6.	7.6	22	34.90	26.54	37.04	9.24	4.11	2.85	128.59	3.91	7.73	4.63	3.10
7.	8.4	30	31.00	27.92	39.53	9.08	4.43	1.45	154.46	6.53	5.71	2.85	2.86
8.	9.4	34	27.25	32.00	23.39	10.35	2.83	1.60	142.18	4.55	6.22	2.91	3.31
9.	10.0	38	34.55	29.70	20.22	8.64	3.42	2.80	119.35	4.74	5.41	2.43	2.98
10.	9.4	40	32.00	29.00	18.65	8.83	3.74	1.35	136.11	3.74	6.18	2.61	3.57
11.	9.0	36	33.40	24.50	28.15	9.41	4.18	1.30	142.61	4.22	4.15	2.02	2.13
12.	6.2	28	36.10	23.60	33.02	7.39	5.43	1.90	153.37	3.86	5.27	3.00	2.28
13.	8.8	32	39.13	18.70	29.30	8.98	3.77	1.95	130.22	6.24	7.71	4.65	3.06
14.	9.2	33	38.20	21.20	24.16	9.31	4.44	2.10	139.79	3.69	5.52	2.75	2.77
15.	7.6	26	28.80	31.20	28.04	9.85	4.13	2.35	143.59	3.62	4.61	2.65	1.96
16.	8.0	34	34.20	18.80	37.66	10.28	3.75	2.05	150.55	4.12	7.23	3.93	3.30
17.	7.2	26	25.20	32.30	39.33	9.79	4.27	1.20	128.70	4.38	4.10	1.76	2.34
18.	8.4	36	32.20	25.20	16.19	8.78	4.47	1.35	144.46	5.92	4.99	2.00	2.99
19	8.0	28	39.20	16.10	23.70	5.59	2.95	1.45	165.98	3.92	6.16	3.45	3.15
20.	8.2	34	29.80	33.20	18.58	10.37	3.85	2.80	132.73	3.99	6.16	3.69	2.47
Mean ±	8.66	32.35	33.80	26.13	28.37	9.13	3.96	1.90	142.99	4.50	5.62	2.93	2.71
SE	0.24	1.03	0.99	1.09	1.89	0.26	0.13	1.13	3.01	0.20	0.24	0.18	0.11

Table 6. Analysis of blood - Comparison between healthy and ketotic cows

	Healthy cows	Ketotic cows
Hb (g%)	9.37 ± 0.16	8.66 ± 0.24
PCV (%)	31.83 ± 2.50	32.35 ± 1.03
Blood glucose (mg%)	54.51 ± 2.48	33.80 ± 0.99*
BUN (mg%)	20.64 ± 0.62	28.37 ± 1.89*
Ca (mg%)	10.01 ± 0.40	9.13 ± 0.26
P (mg%)	5.40 ± 0.29	3.96 ± 0.13*
Mg (mg%)	2.58 ± 0.13	1.90 ± 0.13*
Total protein (g%)	7.47 ± 0.29	5.62 ± 0.24*
Albumin (g%)	3.56 ± 0.16	2.93 ± 0.18*
Globulin (g%)	3.91 ± 0.32	2.71 ± 0.11*
Na (mmol/lit)	147.11 ± 3.12	142.99 ± 3.01
K (mmol/lit)	4.62 ± 0.15	4.50 ± 0.20
Ketone bodies (mg%)	4.17 ± 0.51	26.13 ± 1.09*

* Significant ($P<0.05$)

Table 7. Blood glucose and ketone body levels in blood, urine and milk in ketotic cows

Case No.	Blood glucose (mg%)	Ketone body levels		
		Blood (mg%)	Urine	Milk
1	30.70	28.00	++++	+
2	33.80	27.58	++++	+
3	34.00	29.23	++++	+
4	38.50	26.43	++++	+
5	43.00	21.83	++++	+
6	34.90	26.54	++++	+
7	31.00	27.92	++++	+
8	27.25	32.00	++++	+
9	34.55	29.70	++++	+
10	32.00	29.00	++++	+
11	33.40	24.50	++++	+
12	36.10	23.60	++++	+
13	39.13	18.70	+++	+
14	38.20	21.20	++++	+
15	28.80	31.20	++++	+
16	34.20	18.80	+++	+
17	25.20	32.30	++++	+
18	32.20	25.20	++++	+
19	39.20	16.10	+++	+
20	29.80	33.20	++++	+

Table 8. Assessment of economic loss

Case No.	Milk production				Duration of disease (days)	Actual* loss due to reduction of milk yield (Rs.)	Cost of** treatment (Rs)
	Pre-disease (l)	During disease (l)	Loss of milk production (l)	Post recovery (l)			
1	6	4	2	5	4	88	250
2	9.5	6.5	3	9.5	7	231	500
3	7	5	2	7	7	154	250
4	7.5	6	1.5	7.5	5	82.5	250
5	4.5	1	3.5	4.5	5	192.5	250
6	7.5	5	2.5	6	6	165	500
7	15	12	3	15	6	198	250
8	8	6.5	1.5	8	5	82.5	250
9	5	2.5	2.5	5	6	165	250
10	13	11	2	12	9	198	500
11	11	9	2	11	4	88	250
12	11	8.5	2.5	10.5	5	137.5	250
13	7	6	1	7	3	33	250
14	16	13.5	2.5	16	9	247.5	500
15	6	2.5	3.5	6	7	269.5	250
16	6.5	4	2.5	6.5	5	137.5	250
17	10	7	3	10	6	198	250
18	8	5.5	2.5	7.5	5	137.5	250
19	7	5	2	7	5	110	250
20	5	3.5	1.5	5	6	99	250

Average duration of disease = 5.75 days
Average milk production before the onset of disease = 8.5 l/day
Average milk production loss = 2.3 l/day
Average economic loss due to actual loss of milk =

Duration of illness x milk loss during that period x 11 (Std. milk price)
= Rs.146/-

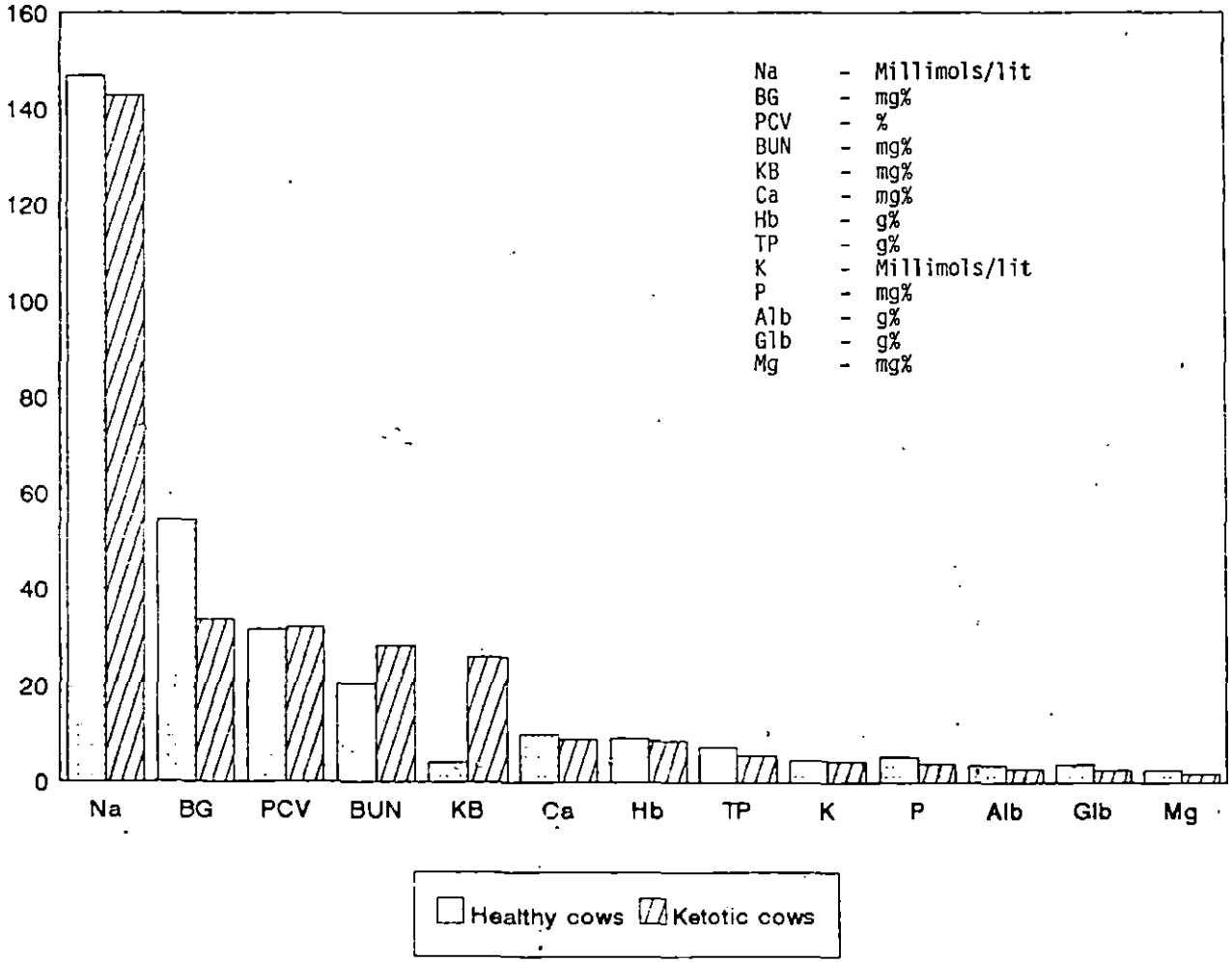
Average cost of treatment = Rs.300/-

Total average economic loss
= Average economic loss due to actual loss of milk
+ Average cost of treatment
= 146 + 300
= Rs.446/-

* Actual loss of milk production = Duration of illness x
milk loss during that period x 11 (standard milk price)

** Cost of treatment = Cost of medicine + Service charges + Conveyance charges

FIG.1 ANALYSIS OF BLOOD - COMPARISON BETWEEN HEALTHY AND KETOTIC COWS



Discussion

DISCUSSION

Epidemiology

Data collected from the Veterinary hospital, Mannuthy showed that the maximum number of ketosis cases occurred during the period from March to May. This may be due to the fact that during this period the environment is hot and humid and the high yielding animals are in heat stress. When a high yielding cow starts producing milk, there is a huge drain of nutrients through milk resulting in a negative energy balance and all are subclinically ketotic . It takes only a small additional nutritional or metabolic insult for them to develop into clinical ketosis (Radostits *et al.*, 1994).

In the present study occurrence of ketosis was more in cows aged three to seven years. This is because the cows are at its peak production between three to six years of age. This is in agreement with the earlier reports of Fox (1971), Ziauddin *et al.* (1992) and Venkateshwarulu and Rao (1993).

Ketosis occurred mostly within the first five weeks of calving. The higher incidence during early part of lactation might be due to excessive energy demands following parturition and stress of lactation. This finding is in accordance with Jazbec (1967), Baird (1982), Reddy *et al.* (1986), Singh and Kasaralikar (1988), Willadsen *et al.* (1993) and Radostits

et al. (1994). The highest incidence of ketosis in the first month of lactation is attributable to the post parturient stress, higher demands of energy and stress of lactation during this stage of lactation.

The study indicated that most of the ketotic cases were observed at the second and the third lactation. This finding is in agreement with Willadsen *et al.* (1993). However, Kauppinen (1983) and Rautmare *et al.* (1989) noted that the most of the cases were at the third or fourth lactation.

Crossbred Jersy cows were mostly affected, although crossbred Brown Swiss and crossbred Holstein Friesians were also affected. The finding is in accordance with Bhuin *et al.* (1993).

The increased incidence in crossbred Jersy cows may be due to the fact that there are more such cows in Trichur area than the crosses of other breeds. On the contrary there are other reports of Holstein Friesian having more incidence (Krdzalic *et al.*, 1988 and Ziauddin *et al.*, 1992). However, breed wise significance could not be indicated in ketosis (Venkateshwarulu and Rao, 1993).

Clinical signs

The various clinical signs exhibited by ketotic animals agreed with the previous reports.

The body temperature, pulse and respiratory rates and mucous membrane were normal. The finding is similar to the observations of King (1979) and Radostits *et al*. (1994). The reduced frequency of ruminal contractions and rumination agrees with the findings of King (1979). Body wasting reduction in milk yield, anorexia, refusal of concentrates but not to hay etc. are reported by many workers (Baird, 1982; Rautmare *et al*., 1992; Anantwar and Singh, 1993 and Radostits *et al*., 1994). Hard and firm dung with mucus coating was noted by Fox (1971) and King (1979).

Nervous signs like salivation, excitation, incoordination of hind limbs leaning onto walls, licking or sucking of inanimate objects etc. agree with the findings of Schultz (1968), Fox (1971), Rautmare (1992) and Radostits *et al*. (1994).

Other than the symptoms reported by earlier workers, biting the crossbar of manger, chain, rope, etc., and frequent falling down in severe cases were also noticed. The nervous signs in ketosis is thought to be caused by the production of isopropyl alcohol, a breakdown product of aceto

acetic acid in the rumen, although the requirement of nervous tissue for glucose may be a factor (Radostits *et al.*, 1994). This could also be caused by the concurrently occurring hypomagnesaemia.

Haemoglobin (Hb) and Packed cell volume (PCV)

The mean value of haemoglobin obtained in healthy cows was comparable with the values reported by earlier workers (Aleyas and Alikutty, 1973 and Schalm *et al.*, 1975). Similarly the mean PCV values of healthy cows agreed with that reported by Rao *et al.* (1981) and Prasad *et al.* (1987).

A non significant decrease in mean concentrations of haemoglobin and PCV found in diseased cows was in accordance with the report of Steger *et al.* (1972). Kuusksalu (1987) and Fatur and Jazbec (1990) reported significantly low values of Hb and PCV in ketotic cows.

Blood glucose

Mean blood glucose value recorded in healthy cows was in the range reported by Sastry (1968), Payne *et al.* (1970), Wiener and Russell (1980) and Radostits *et al.* (1994).

A significant decrease ($P<0.05$) in the mean blood glucose concentrations (33.80 ± 0.99 mg%) compared to normal observed

in the present study is in agreement with the earlier reports (Shaw, 1956, Panduranga *et al.*, 1975, Horber *et al.*, 1980, Sarode *et al.*, 1981, Anantwar and Singh, 1993, Radostits *et al.*, 1994 and Anantwar *et al.*, 1995). According to Baird (1982), Grohn *et al.* (1983) and Radostits *et al.* (1994) ruminants were more prone for ketosis, compared to other species, because only very little carbohydrates was being absorbed from gut and a direct supply of glucose was needed for tissue metabolism. The formation of lactose and utilization of volatile fatty acids for energy purpose is also dependent on the supply of available glucose. The vulnerability is further exacerbated by the much increased turn over of glucose and relatively poor glycogen stores. From calving till peak milk yield, the demand for glucose is being increased. Although milk production can be reduced by reduction in food intake, this does not automatically reduce the milk in early lactation, since the hormonal stimuli for mammary activity will over come the effects of reduced food intake and this is the factor for spontaneous ketosis in early lactation.

Shaw (1956) also reported that the amount of glucose produced by gluconeogenesis is considerably greater than that derived from propionic acid in ketosis. Faulty gluconeogenesis would result in a severe taxation on the animal owing to the fact that the mammary gland continues to

utilize relatively large quantities of glucose resulting in hypoglycaemia.

Homeostasis of blood glucose in healthy animals is maintained as a result of equilibrium established between glucose supply from intestinal absorption and its removal from circulation by various means such as hepatic uptake and release, removal by peripheral tissues, synthesis of lactose for milk production, the effect of hormones etc. (Kaneko, 1980).

Blood Urea Nitrogen (BUN)

Mean value of BUN obtained for control cows was comparable to the values reported for healthy cows by Kaneko (1980) and Benjamin (1998).

The present study indicated a significant increase ($P<0.05$) in the BUN concentration in ketotic animals. Blowey (1975) reported high BUN values in ketosis and attributed it to a high level of metabolic nitrogen circulating within the body and available for protein synthesis. A concurrent hypoglycemia and elevated BUN level in these cases indicated that urea was not being utilized for protein synthesis. Below normal levels of albumin (reflecting protein storage) probably arises from the energy deficit reducing the level of

nonprotein nitrogen utilization by the ruminal microorganisms which inturn leads to a "leakage" of nitrogen from the rumen.

However, reduced values of BUN, glucose and albumin was detected by Zhabolenko (1976) in ketotic cows.

Calcium

The mean calcium level in the serum of healthy controls in this study was comparable to the values recorded by Moodie (1960), Mylrea and Bayfield (1968) and Kaneko (1980).

A non significant decrease in calcium level was noticed in ketotic cows compared to normal cows. Hypocalcaemia in ketosis was reported by many researchers (Cote *et al.*, 1969 Margolles, 1988, Singh and Kasaralikar, 1990 and Radostitis *et al.*, 1994). Halse and Velle (1958) noticed hypercalcaemia in ketosis whereas Green (1975) concluded that ketosis had no influence on the serum calcium levels.

Hypocalcaemia in normal cows at parturition was caused by drain of calcium into milk (Robertson *et al.*, 1956) or interruption to absorption of calcium from the gut (Marr, 1956). Lean *et al.* (1992) observed elevated fatty acid in serum associated with fatty infiltration of liver impairing the ability to maintain calcium homeostasis due to impaired hydroxylation of vitamin D in the liver.

Inorganic Phosphorus

The mean value of phosphorus in healthy control cows was in the range reported by Mylrea and Bayfield (1968), Payne *et al.* (1970) and Kaneko (1980).

Statistically significant decrease ($P<0.05$) in the mean value of serum inorganic phosphorus was detected in ketotic cows. Low levels of phosphorus was obtained in ketotic cows by Yoshida (1978). However no significant changes in phosphorus level was reported by Singh and Kasaralikar (1990). Contrary to this there were also reports of hyper phosphotaemia among ketotic cows (Caple *et al.*, 1977, Margolles *et al.* 1988 and Fatur and Jazbec, 1990).

Serum phosphorus level appears to be intimately related to carbohydrate metabolism (Simesen, 1970). Since there was hypoglycaemia, it indicated poor carbohydrate reserves or nutritional condition, which was believed to be associated with low levels of phosphorus (Shaw, 1956).

Magnesium

Mean magnesium concentration recorded in the serum of healthy cows was in agreement with the ranges specified by Mylrea and Bayfield (1968), Pyne *et al.* (1970) and Larson *et al.* (1980).

There was a significant reduction (P>0.05) in the magnesium concentration in ketotic cows. This finding was in accordance with that of Breirem *et al.* (1948), Yoshida (1978) and Margolles (1988). On the contrary, Duncan *et al.* (1939); Cote *et al.* (1969) and Goncharova *et al.* (1990) did not observe any change in magnesium concentration in ketotis. Rayssiguier (1977) associated low levels of magnesium with increased fatty acid levels and fatty infiltration of liver in ketosis.

Bovine ketosis also has been described as a result of persistent hypomagnesaemia. The two diseases do occur together in herds subjected to a shortage of feed energy during early pregnancy, so that both may have a common cause. Positive response of ketotic cows to treatment with magnesium was also described (Yoshida, 1978).

Sodium

The mean value of serum sodium in healthy cows was comparable to the values for healthy cows reported by Mylrea and Bayfield (1968), Kaneko (1980) and Salim and Joshi (1992).

In ketotic cows, Rashid (1977) could obtain a positive correlation in serum sodium concentrations with ketone bodies. Whereas, Fatur and Jazbec (1990) reported reduced sodium

concentration in ketotic cows. In the present study there was no significant difference noticed between the normal and ketotic cows.

Potassium

Serum potassium levels obtained for healthy cows was in agreement with the range recorded by Mylrea and Bayfield (1968), Payne *et al.* (1970) Kaneko (1980), Benjamin (1998) and Radostits *et al.* (1994).

In the present study there was no significant difference in the potassium level in the diseased group compared to the healthy ones. This finding agreed with the reports of Saarinen and Shaw (1950). Contrary to the above, low levels of potassium in serum of ketotic cows was reported by Carlstorm (1950).

Total protein

The mean total protein value in healthy control cows in the present study was well within the ranges reported by Mylrea and Healy (1968), Payne *et al.* (1970) and Schalm *et al.* (1975).

Significant decrease ($P<0.05$) in the mean values of total protein of ketotic cows observed in the present study

agreed with the observation of Savoiskii and Fedorov (1976). But an increase in total protein was detected in ketosis by Zhabolenko (1976).

Decreased serum protein could be due to an inadequate hepatic production. In pregnancy and lactation it could also be due to the failure to meet the increased demand (Benjamin, 1998).

Albumin

Mean serum albumin values of healthy control cows, in the present study was comparable with the values reported by Payne *et al.* (1970), Singh and Choudhary (1980) and Radostitis *et al.* (1994).

The significant decrease ($P \leq 0.05$) in the mean albumin concentration in ketotic cows was in agreement with the findings of Blowey (1975), Jovanovic *et al.* (1990) and Asmare *et al.* (1997). Blowey (1975) attributed reduced non protein nitrogen utilization by the rumen microflora to low albumin levels in hypoglycaemic energy deficient animals. Low levels of albumin can also be due to inadequate hepatic production. Similarly, low level of albumin during pregnancy and lactation could be due to failure to meet the increased demand (Benjamin, 1998) as was noticed in the case of total protein.

Globulin

Mean values of serum globulin values in healthy control cows was comparable with the values reported by Swenson and Reece (1993) and Radostits *et al.* (1994) and Rajora and Pachauri (1994).

There was a significant reduction ($P<0.05$) in the values of serum globulin concentration in ketotic cows compared with healthy cows. Increase in the serum globulin resulting in an increased total protein value in post-parturient cows was reported by Rajora and Pachauri (1994). They explained that the increase in globulin was probably due to its increased need for production for excretion in colostrum. Similar observations were made by Singh and Choudhary (1988), McLennan and Willoughby (1973) and Rowlands *et al.* (1975).

In the present study serum globulin levels in ketotic cows were found to be reduced. Most of the animals studied were above two to three weeks after calving. By this time the need for production of globulin for excretion in colostrum might have been over. So no further production of excess globulin occurs. This could be the reason for its reduced level in blood.

The reduced globulin levels can also be attributed to inadequate hepatic production and failure to meet the

increased demand for proteins (Benjamin, 1998) as in the above cases of total protein and albumin. Since ketosis is not an infectious disease, globulin levels in serum is not of much importance.

Ketone bodies in blood

The mean values of ketone bodies in blood of normal healthy cows was within the ranges recorded by Huhold and Thiemann (1979), Margolles *et al.* (1988), Nakajima *et al.* (1993) and Radostits *et al.* (1994).

There was significant increase ($P<0.05$) in the ketone body content (26.13 ± 1.09 mg%) of blood in ketosis. The increase was negatively correlated (-0.776) with blood glucose concentrations. This is in accordance with the findings of Shaw (1943), Rajan and Ganapathy (1973), Huhold and Thiemann (1979), Horber *et al.* (1980), Singh and Kasaralikar (1990), Radostits *et al.* (1994) and Anantwar and Singh (1995). This could be the result of mobilisation of more amount of fatty acids to the liver beyond its capacity to convert to glucose or other forms of energy through the Tricarboxylic acid (TCA) cycle and a relative deficiency of oxalo acetate (Baird, 1982; Rings, 1985).

Urine ketone bodies

All the cases in the present study showed varying degrees of positive results for Rothera's test. This is in agreement with the findings of Sastry (1968), Fox (1971), Kronfeld (1972) and Radostits *et al.* (1994).

The development of the colour in Rothera's test is due to the presence of acetoacetate which was the only reactive ketone body in the urine. The degree of colour change depends on the quantity of acetoacetate present in the urine. The findings are in the line with Sastry (1968) and Fox (1971).

Milk ketone bodies

Several workers viz., Emery *et al.* (1964), Schultz (1974), Piatkowski (1974), Anderson and Emanuelson (1985) and Radostits *et al.* (1994) reported the presence of ketone bodies in milk of cows with ketosis.

Schultz (1974) observed that Rothera's test on milk gave positive reaction when the blood ketone body levels reached 10-15 mg per cent. In the present study average blood ketone body level was 26.13 ± 1.09 mg per cent and all the ketotic cows were positive in Rothera's test in milk. This finding was in agreement with Schultz (1974) and Kronfeld *et al.* (1972).

Conclusion

All high producing cows are in negative energy balance during the early post parturient period. The milk production of cows reaches the peak by four weeks after parturition but the dietary intake on a dry matter basis does not peak until seven to eight weeks. The negative energy balance already existing may further aggrevate in adverse conditions due to anorexia.

In the present study most cases occurred during the period from March to May when there was hot and humid climate in Kerala.

The animals are comfortable at a temperature below 25°C. When the temperature exceeds 25°C, heat stress starts in high producers. This will reduce the milk production, lower the hepatic glycogen stores and increase metabolic rate by 40-50 per cent. As a result anorexia, loss of body weight hypoglycaemia, rise in NPN level etc. occur.

In Kerala there is a practice of feeding the cows with rice gruel alone for some days immediately after calving. If the amount exceeds a limit, in an unadapted it will lead to acid indigestion and varying levels of resultant anorexia will again exacerbate the negative energy balance already existing.

Not only that in case of changes in feeding practises, the rumen microbes must get readopted. As a result the efficiency of utilization of the ration is reduced. This again will increase the negative energy balance.

The cows in the present study were reported to be in good body conditions and many were fat cows. This again is a predisposing factor for ketosis. Good body condition and high dietary protein may lead to excessive lipid metabolism and ruminal fermentation leading to development of ketosis.

Based on the results of the study the following management aspects are suggested for prevention and control of ketosis.

1. Avoid exposure of the animals to incliment weather. In hot and humid climate, they should be kept in shades during day time. Grazing should be allowed in the early and late hours of the day only. Preferably give concentrate feed in small amounts at frequent intervals and avoid feeding at noon times. Spraying water on the body during hot times of the day will reduce the heat stress. Water preferably cool should be available at all times and ad libitum.

2. Feeding practises should not be changed abruptly especially after calving. The quantity of energy ration

should be calculated and supplied according to the milk production. To overcome the heat stress additional feed should be given.

3. The cow should not become over fat during gestation.

4. Avoid feeding poor quality roughages to the high yielding cow, especially during early post partum period.

5. Continued feeding of low digestable carbohydrates is to be avoided. A proportion of the feed is to be in a highly digestable form. After calving the cows should be fed more, along with the increase in milk production and never overfeed the animal at the end of lactation and in advanced pregnancy.

In general, routine screening for ketonuria by Rothara's test is advised for early detection and treatment of ketosis.

Summary

SUMMARY

An investigation was undertaken to study the metabolic profile of ketotic cows under field condition.

Incidence of ketosis among dairy cows which attended the outpatient unit of Veterinary College Hospital, Mannuthy for the last six years was recorded and found that most of the cases were occurred between March and May (Summer season).

Twenty field cases of primary ketosis selected at random which attended the Veterinary College Hospital Mannuthy constituted the diseased group. Similarly, 12 healthy dairy cows maintained below 60 days of postpartum, under similar field condition selected at random, were taken as the control group. Detailed epidemiological and symptomatological data were documented.

Samples of blood, urine and milk collected and analysed. Epidemiological study included the effects factors such as breed, age, season, stage of lactation etc. on the incidence of ketosis.

Haematological study comprised of detecting variation in haemoglobin and PCV among the two groups.

Biochemical parameters such as blood glucose, calcium, phosphorus, blood urea nitrogen, magnesium, sodium, potassium, total protein, albumin and globulin and ketone bodies in the blood were determined by standard methods. Urine and milk were analysed for the presence of ketone bodies.

Analysis of epidemiological data showed that incidence was highest in crossbred Jersey cows during summer season. Cows aged three to six years and in their second and third lactation had more incidence. Most of the clinical cases studied were below five weeks of postpartum.

Other than the usual signs reported by the earlier workers, frequent falling down and biting of the crossbar of manger, chain or neck rope were among the major nervous signs noticed. The clinical data were with physiological limits.

Comparison of mean concentrations of Hb and PCV among the diseased and control groups did not reveal any significant changes. Blood glucose, phosphorus, magnesium and total protein albumin and globulin showed significant decrease in their concentration in diseased group when compared to control cows. Whereas ketone bodies and BUN showed significant increase in the ketotic cows. Urine and milk examination for ketone body concentration revealed varying concentration. However, serum concentration of calcium, sodium and potassium

did not vary significantly. The economic loss occurring in each ketotic case was documented and found to be on an average of Rs.446/- per animal.

Changes in the parameters observed in the ketotic cow in the present study suggested hypoglycaemia (33.80 ± 0.99 mg%), hypophosphataemia (3.96 ± 0.13 mg%), hypomagnesemia (1.90 ± 0.13 mg%), reduced values for total protein (5.62 ± 0.24 g%), albumin (2.93 ± 0.18 g%) and globulin (2.71 ± 0.11 g%) increased blood urea nitrogen values (28.37 ± 1.89 mg%), hyperketonaemia (26.13 ± 1.09 mg%), ketonuria and ketolactia.

References

REFERENCES

Aleyas, N.M. and Alikutty, K.M. (1973). Haematological studies on crossbred cattle with special reference to Australian Jerseys. *Kerala J. Vet. Sci.* 4(1): 63-69.

Anantwar, L.G. and Singh, B. (1992). Treatment of clinical ketosis in buffaloes (*Bubalus bubalis*). *Indian Vet. J.* **69**: 446-448.

Anantwar, L.G. and Singh, B. (1993). Epidemiology, Clinico-pathology and treatment of clinical ketosis in buffaloes (*Bubalus bubalis*). *Indian Vet. J.* **70**: 152-156.

Anantwar, L.G., Singh, B., Awaz, K.B., Ali, M.S. and Rajguru, D.N. (1995). Therapeutic evaluation of glucose and insulin combination in ketotic buffaloes (*Bubalus bubalis*). *Livestock Advisor* **20**(1): 26-28.

Andersson, L. and Emanuelson, U. (1985). An epidemiological study of hyperketonaemia in Swedish dairy cows; determinants and relation to fertility. *Preventive Vet. Med.* **3**: 449-462.

Anonymous (1994). Kerala Agricultural University Annual Research Report. 1994-95.

AOAC. (1990). Official Methods of Analysis. 15th ed. Association of Official Analytical Chemists. Washington D.C.

*Aslan, V. and Nimazlioglu, M. (1985). Blood glucose values in the pregnancy and lactation period of cows and the diagnosis of subclinical ketosis. *Veteriner Fakultesi Dergisi Selcuk Universitesi* 1(1): 57-64.

*Asmare, A.A., Reichel, P. and Bartko, P. (1997). Energy metabolism disorder in black-pied breed (Friesian) dairy cows. *Folia Veterinaria* **41**(1-2): 55-59.

Baird, G.D., Heitzman, R.J., Hibbitt, K.G. and Hunter, G.D. (1974). Bovine ketosis - A review with recommendations for control and treatment. *Bri. Vet. J.* 130: 214-220.

Baird, G.D. (1982). Primary ketosis in high producing cows: Clinical and subclinical disorders; treatment, prevention and outlook. *J. Dairy Sci.* **65**: 1-10.

Barnet, R.N. (1973). *Am. J. Clin. Path.* **59**: 836.

*Bauer, M., Dlesk, D., Jankovic, D. and Harapin, J. (1991). Early diagnosis of ketosis in dairy cows. *Veterinarska Stanica* **22**: 3-7.

Benjamin, M.M. (1998). Outline of Veterinary Clinical Pathology. 3rd edn. Kalyany Publishers, New Delhi, pp. 108-116, 286-292.

Bhuin, S., Chakrabarti, A. and Mukherjee, B.N. (1993). A study on clinical ketosis in cows in Mohanpur Haringhata Complex (W. Bengal). *Indian J. Dairy Sci.* **46**(6): 258-259.

*Bjarnason, B. (1986). Ketosis in dairy cows in Auster Skaftafellssy sla in the winter of 1984-1985. *Dansk Veterinaertidsskrift* **69**: 881-889.

Blowey, R.W. (1975). Practical application of metabolic profiles. *Vet. Rec.* **97**: 324-327.

*Breirem, K., Ender, F., Halse, K. and Slagsvold, L. (1948-1949). Experiments on hypomagnesemia in ketosis in dairy cows. *Acta Agriculture Suecana* 3: 89-120.

Caple, I.W., Pemberton, D.H., Harrison, M.A. and Haplin, C.G. (1977). Starvation ketosis in pregnant beef cows. *Aust. Vet. J.* 53(6): 289-291.

Carlstorm, B. (1950). Deficiency Diseases, Particularly Acetonaemia in Cattle. *Vet. Rec.* **62**: 717.

Choudhuri, P.C. (1990). Health care for high productivity. *Indian Dairy Man* **4**: 186-189.

Cote, J.F., Curtis, R.A., McSherry, B.J., Robertson, J.M. and Kronfeld, D.S. (1969). Bovine ketosis: Frequency of clinical signs, complication and alterations in blood ketones, glucose and free fatty acids. *Can. Vet. J.* 10: 179-187.

Daly, J.A. (1972). *Clin. Chem.* **18**: 263.

Dohoo, I.R. and Martin, S.W. (1984). Subclinical ketosis: Prevalence and associations with production and disease. *Can. J. Comp. Med.* **48**: 1-5.

Dohoo, I.R., Martin, S.W., Meek, A.H. and Sandals, W.C.D. (1983). Disease, Production and culling in Holstein Friesian cows. I. The data. Preventive Veterinary Medicine I: 321-334. (Cited by Dohoo and Martin, 1984).

Doumas, B.J. (1978). *Clin. Chem.* 21: 1159.

Duncan, C.W., Huffman, C.F. and Tobin, H.A. (1939). A chemical study of ketosis in dairy herd. *J. Am. Vet. Med. Assoc.* 95: 690-700.

Emery, R.S., Bell, J.W., Brown, L.D. and Blank, G.N. (1964). Detection, occurrence and prophylactic treatment of borderline ketosis with propylene glycol feeding. *J. Dairy Sci.* **47**: 1074-1079.

Emery, R.S., Bell, J.W. and Thomas, J.W. (1968). Benefits derived from routine testing for milk ketones. *J. Dairy Sci.* **51**: 867-874.

*Fatur, B. (1990). Frequency of clinical and subclinical ketosis in dairy cows in the post partum period and the effect of nutrition on its occurrence. *Zbornik-Veterinarske Fakultete Univerza 'Ljubljana* **27**(2): 167-178.

*Fatur, B. and Jazbec, I. (1990). Metabolic profile of ketotic cows. *Veterinarski-Glasnik* **44**(8-9): 647-653.

*Fatur, B., Jazbec, I., Kopitar, M. and Sketa, J. (1993). Incidence of clinical and subclinical forms of bovine ketosis in Slovenia and some other European countries. *Pri Slovenski VeterinarskiKongres Portoroz* 18th-20th November. Zbornik-1 1993, 165-170.

Fatur, B. and Trenti, F. (1994). Ca, P, Mg and Na levels of dairy cows with subclinical ketosis. Proceedings of the 18th World Buiatrics Congress. 26th congress of the Italian Association of Buiatrics, Bologna, Italy. Vol2. 1257-1260.

Folin, O. and Wu, H. (1920). A system for blood analysis supplement. I. A simplified and improved method for determination of sugar. *J. Biol. Chem.* **41**: 367.

Fox, F.H. (1971). Clinical Diagnosis and treatment of ketosis. *J. Dairy Sci.* 54: 974-985.

*Gitzwiller, A. (1989). Health of the dairy herd at Grangeneuve Research Station (Switzerland). Frequency of the principle disorders between 1979-1988. *Landwirtschaft-Schweiz* 2(9): 518-520.

*Goncharova, Y., Petrova, L., Dryanovski, G. and Simeonov, S. (1990). Some vitamins and minerals in the blood of cows with ketosis and their calves. *VeterinarnaSbirka* 88(3): 11-13.

*Green, U. (1975). Electrolyte equilibrium in cows with clinical disease. *Tierarztliche Hochschule, Hannover* pp. 62.

Grohn, Y., Lindberg, L.A., Bruss, M.L. and Farver, T.B. (1983). Fatty infiltration of liver in spontaneously ketotic dairy cows. *J. Dairy Sci.* 63: 2320-2328.

Halse and Valle (1958). Blood calcium in bovine ketosis. *Am. J. Vet. Res.* 19: 575-579.

Hamakawa, M., Sakai, T., Murata, S., Yamada, K., Ogura, K., Ueno, K., Nagao, S. and Motoi, Y. (1995). Composition of rumen volatile fatty acids and blood components at pre and post parturition in ketonuric cows. *J. Japan Vet. Med. Assoc.*

Hamana, K., Taura, Y., Minamishogaku, K. and Akita, S. (1985). Metabolic profile test and parturition syndrome in dairy cattle. I. Survey of parturition syndrome in Miyazaki. Bulletin of the Faculty of Agriculture, Kagoshima University **35**: 107-111, 113-117.

Hayatgheybi, H. and singh, B. (1993). Blood chemistry of healthy and ketotic buffaloes (*Bubalus bubalis*). *Livestock Advisor* **18**(9): 32-34.

Henry, R.L. (1969). Spectrophotometric method of estimationof acetone and acetoacetic acid from blood. Clinical Laboratory diagnosis. 7th edn. Lea and Febiger, Philadelphia. 465-466.

*Heydrych, E.M. and Wieckowski, W. (1991). Colour test for acetones in milk for the diagnosis of subclinical ketosis. *Medycyana Weterynaryna* **47**: 84-86.

*Horber, H., Mader, F. and Jucker, H. (1980). Ketone bodies in blood, milk and urine of healthy dairy cows and cows with primary ketosis. *Schweizer Archiv fur Tierheilkunde* **122**(10): 553-564.

Hove, K. (1978). Insulin secretion in lactating cows responses to glucose infused intravenously in normal, ketonaemic and starved animals. *j. Dairy Sci.* **61**: 1407-1413.

*Huhold, K. and Thiemann, K.G. (1979). Determination of mean values and limits of ketone bodies in cattle blood serum by using mićrodistillation technique. *Monatshefte fur Veterinarmedizin* **34**(13): 484-487.

*Hupka, E. (1928). Die Azetonamie der Rinder. *Duet. tieraztl wochschr* 36: 98 (Cited by Shaw, 1955).

*Jazbec, I. (1967). Incidence aetiology and treatment of subclinical acetonaemia in a dairy herd. *Duet. tieraztl wochschr* **74**: 599-600.

*Jovanovic, M.J., Stamatovic, S., Damnjanovic, Z., Boros, I., Pivnicki, D. and Ivanov, I. (1990). Blood chemical values of recently calved cows with liver disorders. *Veterinarski-Glasnik* **44**(7): 535-540.

Kaneko, J.J. (1980). Clinical Biochemistry of Domestic Animals. 3rd. edn. Academic Press, New York. pp. 27, 109-116, 382, 583-584.

Kaneko, J.J. and Cornelius, C.E. (1970). Clinical Biochemistry of Domestic Animals. 2nd edn. Academic Press, New York. pp. 46-47.

*Kauppinen, K. (1983). Prevalence of bovine ketosis in relation to number and stage of lactation. *Acta Veterinaria Scandinavica* 24: 349-361.

King, J.O.L. (1979). The effects of ketosis in dairy cows on body weight milk yield and milk composition. *Brit. Vet. J.* 135: 40-43.

*Kondrakhin, I.P. (1976). Milk analysis in the diagnosis of ketosis in cows. *Veterinariya Moscow.* **8**: 88-90.

*Krdzalic, P., Vujovic, M., Jovanovic, M.J., Mihailovic, M., Aleksic, M. and Vukovic, V. (1988). Prevalence of ketosis on farms in the public sector and on small holdings in Serbia. *Veterinarski-Glasnik* **42**(8): 469-475.

Kronfeld, D.S. (1972). Diagnosis of metabolic diseases in cattle. *J. Am. Vet. Med. Assoc.* **161**: 1259-1264.

Kronfeld, D.S. and Emery, R.S. (1970). Bovine Medicine and Surgery (ed. Gibbons *et al.*) 1st edition. *Am. Vet. Pub.* Illinois pp. 350-376.

Kronfeld, D.S., Simesen, M.G. and Dungworth, D,L. (1960). Liver glycogen in normal and ketotic cows. *Res. Vet. Sci.* 1: 242-247.

Kuusksalu, V.G. (1987). Metabolic disorders in lactating cows. Aktual nye voprosy obmena veshchestv. Materialy 3-Konferentsii no-voprosu fiziologicheskogo obmena veshchestv-v-organizme cheloveka-i-zhivotnykh, 9-11 Sept., 1987 134-135.

Larson, L.L., Mabruck, H.S. and Lowry, S.R. (1980). Relationship between early postpartum blood composition and reproductive performances in dairy cattle. *J. Dairy Sci.* 63(2): 283-289.

Lean, I.J., Bruss, M.L., Baldwin, R.L. and Trouff, H.F. (1991). Bovine ketosis: A Review. I. Epidemiology and Pathogenesis. *Vet. Bull.* 61(2): 1209-1218.

Lean, I.J., Bruss, M.L., Baldwin, R.L. and Trouff, H.F. (1992). Bovine ketosis: A Review. II. Biochemistry and Prevention. *Vet. Bull.* 62(1): 1-14.

Lebeda, M., Prikrylova, J., and Bus, A. (1982). Ketonuria in dairy cows Veterinarni - Medicina 27:9, 513-523.

Margolles, E, Colome H and Saez C. (1988) Biochemical characteristics of subclinical ketosis in a herd of high yielding Holstein Cows. I., ketone bodies, Glucose and minerals. *Rivista Cubana de Ciencias Veterinarias*.

Marr, A. (1958). Cited by Moodie (1960). 2. Some aspects of hypocalcaemia in cattle. *Vet. Rec.* 72(50): 1145-1149.

McLennan, M.W. and Willoughby R.A. (1973) observations of serum protein values of cows during oestrus, lactation, parturition and Milk fever *Can. Vet. J.* 14:233-242.

Mironov, N.A. (1975) Treatment of Ketosis in cows. Veterinariya Moscow, USSR, No.3 92-95.

Moodie, E.W. (1960), Some aspects of hypocalcaemia in cattle *Vet. Rec.* 72 (50): 1145-1149.

Mylrea, P.J. and Bayfield, R.F. (1968) COncentration of some blood components in the blood and serum of apparently healthy dairy cattle I Electrolytes and minerals. *Aust: Vet. J.* 44:(12): 565-569.

Mylrea, P.J. and Healey, P.J. (1968) Concentration of some components in the blood and serum of apparently healthy dairy cattle. II. serum protein enzymes, bilirubin and creatinine. *Aust. Vet. J.* **44** (12) 570-573.

*Nakajima, I., Ogawa, M., Hasegawa, K., Nagatsuma, Y., Tokita, T. and Ogura, K. (1993). Effect of metabolic disorders in the early period of lactation on the reproductive performance of dairy cows. *J. Vet. Med. Japan* 46(10): 801-805.

*Nenadovic, M., Radakovic, N., Jovanovic, R., Zivkovic, V. and Plancak, J (1978). The incidence and degree of acetonaemia in cows of different genotypes and ages. *Veterinarski Glasnik* 32(11): 913-921.

Nielen, M., Aarts, M.G.A., Jonkers, A.G.M., Wensing, T. and Schukken, Y.H. (1994). Evaluation of two cow side tests for the detection of sub-clinical ketosis in dairy cows. *Can. Vet. J.* 35(4): 229-232.

Nikalaenka, G.V. and Syamenau, B.Y. (1990). Metabolic disorders in high yielding cows. *Vestsi Akademii Navuk* No.3: 101-108.

Overby, I., Hansen, M., Jonsgard, K., Sognen, E. (1974). Bovine ketosis: Occurrence and incidence in herds affected by ketosis in eastern Norway. *Nordiok. Veterinaermedicin*. 26: 353-361.

Panduranga, G.L., Sundaravandanan, V.K. and Sastry, K.N.V. (1975). Occurrence of ketosis in cows with anaroxic symptoms and its relationship to blood glucose level. *Mysore J. Agric. Sci*. 9: 692-696.

Payne, J.M., Dew, S.R., Manston, R. and Faulks, M. (1970). The use of a metabolic profile test in dairy herds. *Vet. Rec*. 87: 150-158.

Piatkowski, S., Viogt, J., Girschewski, H. and Steger, H. (1974). Diagnosis and treatment of subclinical ketosis in dairy cows. Monat. Vet. Med. 29: 532-534.

Prasad, B., Rathor, S.S. and Sindhu, S.S. (1987). Studies on haematobiochemical profile for forecasting downer syndrome in cows. *Indian J. Vet. Med*. 7(2): 91-95.

Radloff, B.D. and Schultz, L.H. (1967). Blood and rumen changes in cows in early stages of ketosis. *J. Dairy Sci*. 50: 68-72.

Radostits, O.M., Blood, D.C. and Gay, C.C. (1994). Veterinary Medicine. 8th ed. English Language Book Society and Bailliere Tindall, Avion, United Kingdom.

Rajan, T.S.S. and Ganapathy, M.S. (1973). Study on blood ketones in cases of digestive and metabolic disorders in cows. *Indian Vet. J*. 50)8): 759-763.

Rajora, V.S. and Pachauri, S.P. (1994). Blood profiles in pre-parturient and postparturient cows and in milk fever cases. *Indian J. Anim. Sci.* **64**(1): 31-34.

Rao, D.G., Prasad, A.B.A., Krishna, V.J. and Rao, K.S. (1981). Studiesk on some biochemical constituents of blood in Ongole cows. *Indian Vet. J.* **58**(11): 870-873.

*Rashid, R. (1977). The relation between ketose bodies, glucose and mineral contents in bovines under various physiological conditions. *Nahrung* **21**(9): 765-769.

Rautmare, S.S. and Anantwar, L.G. (1993). Prevalence, biochemistry and treatment of clinical ketosis in buffaloes. *Buffaloe-Bulletin* **12**(4): 81-83.

Rautmare, S.S., Anantwar, L.G. and Jadhao, P.T. (1992). Ketosis in buffaloes. *Pashudhan* **7**: 3.

Rautmare, S.S., Anantwar, L.G. and Singh, B. (1989). Prevalence of ketosis in buffaloes (*Bubalus bubalis*). *Indian J. Vet. Med.* **9**: 28.

Rayssiguier (1977). Hypomagnesemia resulting from adrenaline infusion in ewes: its relation to lypolysis. *Hormone Metabolic Res.* **9**: 309-314.

Reece, O.W. (1997). Physiology of Domestic Animals. 2nd edn. Rose tree corporate centre 1400 North Providence Road Building, Pennsylvania, USA.

Reddy, J.S., Syamasunder, N. and Choudhuri, P.C. (1986). Subclinical ketosis in crossbred cattle in an organised dairy herd. *J. Res. Andra Pradesh Agricultural University* **14**(2): 170-172.

Reynaert, R., Top, W. and Peeters, G. (1977). Bovine serum growth hormone levels in clinical ketosis. *Vet. Rec.* **101**(2): 36-37.

*Riemann, H.P., Bjerkelarssen, R. and Simensen, E. (1985). Ketosis in Norwegian dairy herds - Some epidemiologic associations. *Acta Veterinaria Scandinavica* **26**: 482-492.

*Rings, D.M. (1984). Therapeutic consideration in ketosis and hepatic lipidosis in cattle. *Modern Veterinary Practises* 66: 523-526.

Robertson, A., Marr, A. and Moodie, E.W. (1956). Milk fever. *Vet. Rec.* 68(13): 173-180.

Rossow, N., Beier, D., Bethe, W., Dudzus, M., Gerisch, V., Le-minh-Chi Kirchner, K., Kreuzberg, H.J., Launer, P. and Mehnert, E. (1976). Results obtained from metabolic supervision in large scale dairy units. *Monatshefte fur Veterinarmedizin* **31**(13): 486-491.

Rowlands, G.J., Manston, R., Pocock, R.M. and Dew, S.M. (1975). Relationship between stage of lactation and pregnancy and blood composition in a herd of dairy cow and the influences of seasonal changes in management on these relationship. *J. Dairy Res.* **42**: 349-362.

Saarinen, P. and Shaw, J. (1950). Studies on ketosis in dairy cattle. XI. Lipids, minerals and ascorbic acid in the blood of dairy cows with spontaneous ketosis. *J. Dairy Sci.* 33: 496.

Salim, A.N. and Joshi, H.C. (1992). Haematobiochemical changes in cows suffering from arrythmias and myocardial infarctions. *Indian J. Anim. Sci.* 62(10): 920-923.

Sarode, D.B., Nisal, M.B. and Sapre, V.A. (1981). Note on bovine ketosis. *Indian J. Anim. Sci.* 51: 228-229.

Sastry, A.G. (1968). The Textbook of Veterinary Pathology. 3rd edn. CBS Publishers, Delhi. 32: 33-37.

*Savoiskii, A.G. and Fedorov, P.D. (1976). Indices of lipid and protein metabolism in the blood of high yielding dairy cows. *Sbornik-Nauchnykh-Trudov-Moskovskaya Veterinarnaya Akedemiya* 85: 95-96.

*Schafer, M. and Bethe, W. (1976). Occurrence of ketosis in our dairy herds. *Monatshefte fur Veteterinarmedizin* 31(13): 505-508.

Schalm, O.W., Jain, N.C. and Carrol, E.J. (1975). Veterinary Haematology, 3rd edn. Lea and Febiger, Philadelphia. pp. 132-144.

Schultz, L.H. (1968). Ketosis in dairy cattle. *J. Dairy Sci.* 51: 1133-1140.

Schultz, L.H. (1974). Ketosis in: Lactation - A comprehensive Treatise. Volume II (Edrs. Larson and Smith). Academic Press, New York and London. pp. 317-353.

Schwalm, J.W. and Schultz, L.H. (1976). Relationship of insulin concentration to blood metabolites in the dairy cow. *J. Dairy Sci.* 59(2): 255-261.

Shaw, J.C. (1943). Studies on ketosis in dairy cattle V. Development of ketosis. *J. Dairy Sci.* 26: 1079-1090.

Shaw, J.C. (1956). Ketosis in dairy cattle - A review. *J. Dairy Sci.* 39: 402-434.

*Simenov, S. (1978). Ketosis in high yielding cows. *Zemizdat Sofia Bulgaria*. pp. 187.

Simensen, E., Liestol, K. and Svela, E. (1987). Ketosis in dairy cows in Hedmark county epidemiological aspects. *Norsk Veterinaertidsskrift* 99(2): 103-111.

Simesen, M.G. (1970). In Clinical Biochemistry of Domestic Animals. Vol.I. Kaneko, J.J. and Cornelius, C.E. eds. Academic Press, New York. pp. 355.

Singh, A. and Choudhary, R.P. (1988): Biochemical studies in Sahiwal and crossbred cattle. *Indian Vet. J.* 65: 791-796.

Singh, B. and Kasaralikar, V.R. (1988). Prevalence, biochemistry and treatment of clinical ketosis in buffalo (*Bubalus bubalis*). Abstract 367-II World Buffalo Congress, New Delhi, December 12th-17th, 1988.

Singh, B. and Kasaralikar, V.R. (1990). Biochemistry and treatment of clinical ketosis in buffaloes (*Bubalus bubalis*). *Indian Vet. J.* 67: 163-165.

Sjollema, B. and Van Der Zande, J.E. (1923). Metabolism in Acetonemia in milch cows. *J. Metabolic Res.* 4: 525.

Snedecor, G.W. and Cochran, W.G. (1980). Statistial Methods. 7th ed. Iowa State University Press, USA.

Steger, H., Piatowski, B. and Girschewski, H. (1972). Effect of propylene glycol and methionine on the concentration of blood sugar and ketones in high yielding cows in early lactation. *Archiv fur Tierernahrung* 22(4): 217-221.

Swenson, M.J. and Reece, W.O. (1993). Dukes physiology of domestic animals. 11th edn. Panama Publishing Company, New Delhi and Bangalore.

Udall, D.H. (1943). The Practise of Veterinary Medicine. 6th edition. George Badnts Publications, USA. Menasha, Washington. pp. 377-385.

Venkateshwarulu, K. and Rao, D.S.T. (1993). Subclinical ketosis in crossbred cows. *Cheiron* 22(3): 116-119.

Venkateshwarulu, K., Rao, D.S.T, Reddy, K.S., Rao, R. and Gaffar, A.A. (1994). Clinicobiochemical findings in subclinical ketosis in crossbred cows. *Indian J. Vet. Med.* 14(1): 6-8.

Vikharev, V.V. (1975). Treatment and prevention of ketosis in dairy cows. *Veterinariya Moscow* 5: 111-113.

West, H.J. (1990). Effect on liver function of acetonaemia and the fat cow syndrome in cattle. *Res. Vet. Sci.* **48**(2): 221-227.

Wiener, G. and Russell, W.S. (1980). Factors influencing the concentration of minerals and metabolites in the plasma of cattle. *J. Agric. Sci. Comb.* **94**: 369-376.

Willadsen, C.M., Qvesel, J., Willeberg, P. (1993). Ketosis in dairy herds II. epidemiological investigations. *Dansk Veterinaertidsskrift* **76**(7): 275-280.

Wybenga, D.R. (1971). *Clin. Chem.* **17**: 891.

Yao, S.W., Gao, S.N., Li, P., Wulumuhan, Namoruo, Ning, X.P., Yang, X.X., Deng, X.Y., Wang, B.X., Zhang, J.Z., Li, Z.R., Reyimu, Zhang, K.J. and Zhang, G.F. (1988). Pathogenetic characteristics and early diagnosis of subclinical ketonaemia in high-producing dairy cows. *Chinese J. Vet. Sci. Tech.* 5: 7-11.

Yoshida, S. (1978). Studies of bovine ketosis associated with magnesium metabolism. *J. Faculty Fish. Anim. Husb. Hiroshima Univ.* **17**(2): 117-131.

Zhabolenko, A.. (1976). Biochemistry of bovine ketosis. I. Adrenal cortex activity. II. Blood Biochemistry. Sbornik Nauchnykh Trudov Moskovskaya Veterinarnaya Akademiya 85, 72-74.

Zharov, A.V. and Kondrakhin, I.P. (1983). Ketosis of high yielding cows. pp.103.

Ziauddin, K.S., Muralidharan, K., Seshadri, S.J., Hussain, P.M., Sreenivasaiah, P.V., Syed Ziauddin, K., Margoob Hussain, P. (1992). Incidence of ketonuria among local and corssbred cows - a five year observation. *Indian Vet. Med. J.* **16**(4): 310-312.

METABOLIC PROFILE OF KETOTIC CROSSBRED DAIRY COWS

By

MANOJ JOHNSON

ABSTRACT OF THE 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 - 680 651

KERALA, INDIA

1999

ABSTRACT

A study of the metabolic profile in ketotic cows in field conditions was undertaken.

Twenty field cases of clinical ketosis in crossbred cows which attended the outpatient unit of University Veterinary Hospital, Mannuthy selected at random, were utilized for the study. Similarly twelve healthy dairy cows of identical field conditions during the vulnerable period of ketosis., i.e., within two months of calving, selected at random constituted the control group. Samples of blood, urine and milk from both healthy and ketotic animals were collected and analysed for various parameters using standard methods. Analysis of economic loss was also carried out.

Data collected from the diseased animals indicated a high incidence among Jersey crossbreds during summer. Cows aged three to six years and in their second or third lactation showed more incidence. Most of the ketotic cows were within the first five weeks of calving.

Other than the usual signs reported by earlier workers, frequent falling down and biting of chain, rope and crossbars of the manger were also noticed. The clinical data were in physiological limits.

Examination of haemoglobin (Hb) and packed cell volume (PCV) did not reveal any significant difference between ketotic and healthy cows. Biochemically blood glucose, phosphorus,magnesium and total protein, albumin and globulin showed significant decrease and blood urea nitrogen and ketone bodies in blood showed significant increase among diseased cows. However serum calcium, sodium and potassium did not vary significantly among the two groups. Urine and milk from the both the groups were examined and varying concentrations of ketone bodies were detected.

Various managemental aspects for the prevention and control of ketosis were discussed.

Appendices

APPENDIX-A

PROFORMA: CASE RECORDING

Serial No.	:	Date:
Case No.	:	
Owner's name and address	:	
Description of animal	:	
Species	:	Breed:
Colour	:	Age :
Parity	:	
History	:	
Owner's Complaint	:	
Present History	:	
Past History	:	

DESCRIPTION OF THE RATION

Feed given		Quantity
Concentrate Y/N	:	Compounded/Uncompounded
Roughage Y/N	:	
Greens Y/N	:	
Is there is any reduction in yield, if so how much	:	
Time of occurance of the disease	:	Before calving/After calving/ Continuous

GENERAL INSPECTION

General condition :
Behaviour :
Expression :
Bodily condition :
Condition of skin & coat :
Type of respiration :
Abdomen :
Posture :
Gait :
Abnormal acts :

Clinical data

Respiration :Rate /min Pulse: Rate /min

Rhythm: Rhythm

Character Character

Temperature : °F
Mucous membrane :
Conjunctival :
Vaginal :

Rumen motility:

Time interval between two motilities :

SYSTEM WISE EXAMINATION

Digestive system

Appetite : Normal/Anorexia/Inappetence/ Hyperorexia/Pica

Desire for water : Present/Absent/Reduced/Increased

Pattern of loss of appetite

( ) Eating concentrates alone
( ) Eating concentrates and paddy straw
( ) Eating concentrates mixed with water
( ) Eating paddy straw and alone
( ) Eating straw and drinking water
( ) Drinking water alone
( ) No inclination to take anything

Rumination : Liver :

Defecation :

Respiratory System

Palpation : Percussion :
Lung ausculation : Nasal discharge : Cough:

Any other symptom of respiratory distress:

Circulatory System

Palpation : Percussion :
Heart auscultation : Superficial lymph nodes:
Blood vessels :

Nervous & Locomotor System

Reflexes : Muscles :
Bones & joints : Ears :
Eyes :

Urinary System

Micturition :
Volume :
Frequency :
Posture :
Bladder (Palpation) :

Kidneys :
Ureters :
Urethra :

Reproductive System

External genitalia :
Ovary :
Cervix :

Mammary gland & teats :
Fallopian tube : Uterus :
Vagina :

Skin & External surfaces the body

Skin & Coat :
Muzzle : Body temperature:
Any other Symptoms shown :

RESULTS OF LABORATORY INVESTIGATION MADE

Hamatological examination

Hb g%
PCV %

Blood Bio-Chemistry

Blood Glucose :
Blood Urea Nitrogen :
Blood Ketone Bodies :
Total Serum Protein :
Total Serum Albumin :
Total Globulin :

Serum Ca :

P :

Mg :

Na :

K :

Urine

Ketone Bodies : (+) (++) (+++) (++++)

Milk

Ketone bodies : (+) (++) (+++) (++++)

Tentative diagnosis :

Definitive diagnosis :

Treatment given and dose rate :

1st Day :

2nd Day :

3rdDay :

Follow up

Assessement of Economic loss

Due to production loss :

Cost of treatment :

Others :

Total :

APPENDIX - B

ESTIMATION OF BLOOD KETONES BODIES

(HENDRY'S METHOD, 1969)

Principle

Performed acetone and the acetone derived from acetoacetic acid are isothermally distilled into alkaline vanillin when they react to from red vanillal acetone or divanillal acetone.

The following reagents were prepared:

1. **Stock Acetone Solution:** Twenty millilitre of distilled water was transferred to a 50 ml flask. One gram of reagent acetone (Sp. gr. 0.791) was added by means of a pipette directly into the water.

The acetone solution was transferred to a 1 litre volumetric flask, and diluted with distilled water to 1000 ml. The contents were mixed and then transformed to glass stoppered bottles and kept in the refrigerator.

2. **Standard Acetone Solution :** Five millilitre of stock Acetone solution was transferred to 100 ml volumetric flask and diluted to 100 ml with distilled water. The contents were

mixed thoroughly and kept well stoppered. This solution was prepared every day. One ml of the solution contained 0.05 mg of acetone.

3. **Vanillin Reagent :** One gram of reagent vanillin was transferred to a 50 ml volumetric flask and diluted to the mark with 4 N KOH and mixed well. Fresh solution was prepared every day.

Procedure

Two millilitre of vanillin reagent was transferred into the two glass stoppered test tube for the standard and unknowns. 0.2 ml of blood from the Ketotic cow was added to the unknown and stoppered. 0.2 ml of standard acetone solution was added to the other.

The test tubes were immediately closed with stoppers and care were taken not to touch the sides. Rubberbands were used to hold the stoppers tightly. Two millilitre of vanillin reagent was added to another test tube and stoppered tightly, to serve as a reagent blank. The test tubes were placed in a water bath at 50° to 55°c for 60 minutes.

At the end of sixty minutes the test tubes were removed from water bath, cooled to room temperature and to each test tube blank 0.3 ml of distilled water was added. The contents were mixed well and the absorbances of the standard and

unknown were read at 415 μ, setting the reagent blank at '0' absorbance.

Calculations

$$\frac{\text{Absorbance of Unknown}}{\text{Absorbance of standard}} \times 5 = \text{mg of acetone per dl.}$$