METABOLIC PROFILE OF HYPOGALACTIC COWS

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Thesis submitted in partial fulfilment of the requirement for the degree of

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DECLARATION

I hereby declare that this thesis, entitled **"METABOLIC PROFILE OF HYPOGALACTIC COWS"** is a bonafide record of research work done by me during the course of research and that the thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title, of any other University or Society.

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CERTIFICATE

Certified that the thesis entitled **"METABOLIC PROFILE OF HYPOGALACTIC COWS"** is a record of research work done independently by **Dr. Reena George**, under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to her.

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CONTENTS

LIST OF TABLES

LIST OF FIGURES

Introduction

1. INTRODUCTION

India is the largest producer of milk. But productivity per animal is among the lowest in the world. Hence productivity and means of improving it are obvious areas of concern. Optimal milk production is the basic requirement in the management of dairy economics.

A recent trend noticed in the high yielding crossbred cattle of Kerala is a decline in milk production in the subsequent lactations over and above that obtained in the first lactation. Although, lactating animals may develop signs of agalactia, hypogalactia or dysgalactia due to various reasons, a systematic study on the etiology of hypogalactia of non specific origin has not so far been conducted.

A decline in milk production may be caused by specific and non specific diseases. Lactational stress especially during 40-60 days after parturition, effect or sudden change of climatic conditions, unscientific management and feeding practices, both infectious and non-infectious diseases, metabolic diseases, death of the calf, abortions or stillbirth have been attributed to be the possible or frequent causes. This has been cited by Graig and Boddie (1956), Turner *et al.* (1957), Roberts (1986) and Blood *et al.* (2000). Hypogalactous dairy cattle have been defined by Stedmann's Medical Dictionary as those dairy cattle producing or secreting a less than normal amount of milk.

A moderate to severe reduction (about 25per cent) in milk yield over that of the previous lactation causes heavy economic loss to the rural farmers of Kerala. Appropriate clinical management is often found difficult as the exact etiology is not clearly understood. A number of metabolic imbalances are suspected to be

responsible for this kind of hypogalactia of non-specific origin in apparently healthy dairy cattle.

Under these circumstances, the study, "Metabolic Profile of Hypogalactic Dairy Cattle" was undertaken with the following objectives:

(i) To identify the various conditions other than specific diseases causing hypogalactia in cows during early to midlactation, and;

(ii) To evaluate the metabolic profile of dairy cattle with hypogalactia of non-specific origin.

Review of Literature

2. REVIEW OF LITERATURE

High yielding dairy cattle are advantageous both in terms of feed conversion efficiency and financial returns. However, it has been found that crossbred cattle are more prone to metabolic diseases which could be forecasted by monitoring the metabolic profile (Payne *et al.*, 1970).

Modern dairy farming imposes severe strain on the animal's metabolism, whereby every effort is made to achieve high production at minimum cost, which increases the risk of imbalances in the input/ output relationship and precipitates the production disease (Rowlands *et al.*, 1976). Metabolic profiling involves collecting blood samples from small groups of cows at different stages of production and analyzing them for a number of defined parameters in order to establish the nutritional status of the herd as a whole and also helps in the diagnosis of inapparent metabolic disorders.

2.1 FACTORS AFFECTING MILK PRODUCTION

Production of milk is the basic economic trait of dairy cattle. Though it is influenced by various factors, basically it is an inherited character. The phenotypic expression of lactation is affected by heredity, managemental factors, climate as well as the primary health status of the cow.

2.1.1 Genetic Factor

Significant variations in milk yield was seen between Sahiwal Brown Swiss crosses versus Red Sindhi Brown Swiss crosses at Karnal (Chopra *et al.*, 1973).

Breedwise variations in milk yield were reported by Panda and Sadhu (1983). According to them the differences in yield between Hariana and Deshi Bengal crosses as well as Holstein versus Jersey crosses were highly significant.

Singh *et al.* (1986) in their study on factors affecting production and reproduction traits Malvi cattle concluded that differences among sires for milk yield and calving interval were highly significant ($P<0.01$) which suggested the genetic influence on the above parameters.

2.1.2 Effect of Parity

According to Turner *et al.* (1957) the total yield of a dairy cow during a lactation period was depended upon the total number of epithelial cells that were stimulated to grow during the first two-thirds of pregnancy and the initiation and maintenance of the intensity of milk synthesized in each of those cells.

During early lactation, the animals are not only in a productive stage but also in a growing stage and both processes lead to drain of the energy reserve of the animal. So maximum yield was expected when the cows have fully grown at about 3rd to 4th lactation in crossbred cattle (Panda and Sadhu, 1983).

Gradual increase in lactation yield from first to third or fourth lactation has been reported by Pandey *et al.* (1986), Chikkara and Pandey (1988) in Nimari breed and Yadav *et al.* (2001) in Gaolao breed.

2.1.3 Effect of Climate

The climatic environment influences nearly every economic aspect of plant and animal growth, milk production and efficiency of conversion of foodstuffs to economic units.

The critical temperature which led to decline in milk yield is approximately 21 to 25°C for Holstein Fresians and Jerseys,but as high as 30 to 32°C with Brown Swiss and 38°C with Brahman cattle. For crossbreds it is 30°C before production is affected. High producing dairy cattle are more susceptible to heat stress than low producing cows, thus effect of heat and high production are additive (Brody, 1955).

For optimum productivity cattle and buffaloes require a climatic environment having an air temperature of 13.18°C, relative humidity of 60-70per cent and a medium level of solar radiations (Gangwar, 1984). The critical temperature that led to decline in milk yield at higher level was approximately 21-25°C for Friesians and Jerseys and as high as 30-32°C for Brown Swiss.

Housing of buffaloes resulted in increased milk production due to protection from cold stress. Alternatively, buffaloes kept in open loose housing with inadequate shelter showed a decline in milk yield. Higher environmental temperatures were considered as an important factor contributing to lower yield and changes in milk composition (Verma and Hussain, 1991).

Agarwal and Singh (2005) stated that lowered ambient temperatures during cold weather reduced milk yield and increased milk fat and these effects were more marked during early lactation.

2.1.4 Effect of Management

Frequency of milking significantly affected milk yield. Milking twice daily, yields at least 40 per cent more than milking once, milking thrice daily may yield 5-20 per cent and milking four times daily may yield 5-10 per cent more (Bath *et al* .,1978).

Managemental factors like alleviating heat stress also influenced milk yield. During summer, body temperatures of cross-bred cows was brought down by

splashing water on the body surface of milking animals resulted in increased dry matter intake and milk production (Verma and Hussain, 1998).

2.1.5 Nutritional factors

Essential nutrients required for optimum lactation are carbohydrates as the primary source of energy, lipids, protein, minerals, vitamins and water. Of these energy yielding carbohydrates are considered most limiting because it is necessary for the proper metabolism of the remaining nutrients.

2.1.5.1 Protein

Feeding of bypass protein to cows in early lactation resulted in increased production because they were in an energy deficient state (Clark *et al.*, 1973).

Clark and Davis (1980) stated that, the response to increased dietary crude protein levels on milk production was maximum when the crude protein content of the ration was raised from 9-10 per cent to 13-14 per cent. He defined crude protein requirement of dairy cows as the minimum amount of protein that will support maximum production. He also opined that production of milk by cows in early lactation was directly proportional to the quality of protein consumed. Bricano *et al.* (1988) also reported the same finding. However increasing protein content of the diet above the NRI requirements of 15-19 per cent of dietary dry matter had not effect on milk yield and caused only a slight increase in the non protein nitrogen content of milk.

National Research Council (NRC, 1989) recommended 19 per cent crude protein in the diet of high yielding cows during early lactation when the dry matter intake lags behind the milk production.

Tolkamp *et al.* (1998) reported higher dry matter intake by increasing ration crude protein from 13.1 to 18.5 per cent.

Feeds of animal origin such as fish meal, blood meal and meat cum bone meal have also been used successfully as protein supplements to improve production in cows. (Atwal and Erfle, 1992).

Khorasani et al. (1994) observed an interaction between protein and energy sources and reported that while protein source significantly increased milk yield, starch source influenced the milk composition. Inadequate protein supply in the diet will reduce milk yield and milk solids content

2.1.5.2 Carbohydrates

Compromised feed intake and diseases like milk fever, which exacerbate the reduction in energy at and after calving increases the risk of fatty liver and ketosis. Such heavy metabolic demands for energy make liver dysfunctions common soon after parturition (Morrow *et al.*, 1979).

Inadequate energy supply in the diet leads to reduced milk yield and milk solids content (protein and fat) and can have a serious impact on the production performance of the herd (Huxley, 2004).

2.1.5.3 Fats

Fat content of ruminant rations ranged from two to three percent of dry matter content and attempts to increase the fat contents were most beneficial when used in the interval of 5-15 weeks of lactation when body fat reserves are depleted and milk production was at its maximum. Supplying cows with about 15 per cent

fat which is approximately 6-7 per cent fat in dietary dry matter as, long chain fatty acids resulted in maximum efficiency of energy for milk production (Sampath, 2001).

2.1.5.4 Miscellaneous

Supplementation of cobalt, Vitamin B_{12} together with trace minerals viz. zinc, selenium and manganese was found to increase milk production in dairy cows maintained on average feeding conditions (Marston, 1961). et al.,

Italics Feeding of Saccharomyces cerevisiae for the improvement in digestibility and production of the animals was demonstrated by Singh *et al.* (1998).

Yeast supplementation not only increased milk yield but also the fat corrected milk production according to the studies of Kamra and Pathak (2005).

2.2. ETIOLOGY

Hypogalactic dairy cattle have been defined as those cattle producing or secreting less than normal amount of milk.

A decline in milk production may be caused by specific and non specific diseases and lactational stress especially during 40-60 days after parturition. The effect or sudden change of climatic conditions, unscientific management and feeding practices, both infectious and non infectious diseases, metabolic diseases, death of the calf, abortions or still birth on milk yield has been documented by Graig and Boddie (1956), Turner *et al.* (1957), Roberts (1986) and Blood *et al.* (2000).

2.2.1. Non Specific Causes

2.2.1.1 Anaemia

Manstan *et al.* (1975) opined that in lactating animals, minimal protein diet resulted in reduction in the synthesis of haemoglobin. This resulted in decreasing value of haemoglobin concentration is apparent as lactation progressed.

Anaemia is a reduction of the erythrocyte number and or haemoglobin concentration per unit volume of blood Schalm *et al.* (1975).

Ramakrishna *et al.* (1992) concluded that incidence of anaemia in buffalo was mostly nutritional in origin.

Anaemia often resulted in low productivity, depressed reproduction and suppressed resistance thus affected the viability of livestock industry. In anaemic cases of bovine, 80 per cent were due to parasitism and 20 per cent were due to nutritional deficiency. Anaemia which often resulted in low productivity in lactating animals was highly prevalent in crossbred cattle, i.e., 67.5 per cent compared to 48.59 per cent in indigenous cattle (Samanta *et al.*, 1995).

Selukar *et al.* (2001) opined that due to anaemia, the time of circulation of blood through the mammary gland decreases, resulting in reduced blood flow to mammary gland and subsequently reduction in milk yield.

2.2.1.2 Anorexia

Reduced serum calcium level was found to be a cause of inappetance which resulted in reduced rumen motility and decreased milk yield occurring within three days of parturition (Pandey and Parai, 1988). a/b .

Any alteration in the rumen pH affected the microbial population, fermentation and absorption process, tonicity of the rumen wall, metabolism of the rumen, which resulted in altered rumen liquor and blood metabolites. Singh *et al.* (1989) stated that any kind of variation beyond the normal range of metabolites in the rumen leads to digestive disorders which impaired the growth, production and reproductive performance of the animal.

Metabolic diseases were easily precipitated during the first stage of lactation. Hence, good appetite during this period helped in calcium absorption from the gut and restricts the mobilization of fat into the liver while indigestion reversed the process.

Pillai *et al.* (1994) recorded that 35.64 per cent of animals, which suffered from post parturient indigestion had liver dysfunction and this was most frequent in the age group of 3-5 years. Such animals exhibited partial to complete loss of appetite, reduced milk yield, decreased ruminal motility and dark yellow urine.

Singh *et al.* (1996) studied the role of stomachic and galactogogue on changing the rumen environment and restoring suppressed milk production. They opined that around 200 litres of milk could be saved per lactation in animals by maintainence of normal rumen pH and administration of antibiogenic amines.

Anorexia is a common field condition in which efficiency of feed utilization is depressed leading to decreased metabolism, retarded growth rate and lowering of milk production (Dua and Bhatti, 1999).

On a study on clinical cases of primary indigestion, Singh et al. (1989) observed that all cases of indigestion had a history of dullness, depression, off-feed, hypogalactia and agalactia. Any deviation in ruminal pH from its normal values influenced the kind and count of microbes and hence the volatile fatty acid and biogenic amines produced in the rumen environment which altered both the metabolites of rumen liquor and blood. Such changes often resulted in reduced milk production from 25 per cent to nil.

2.2.1.3 Ectoparasities and Endoparasites

Subclinical infection of nematodes in dairy cattle caused considerable economic losses due to decreased milk production (Grishi and Todd, 1978).

Significant adverse effect of gastrointestinal parasitic infection on milk production of adult Murrah buffaloes was studied by Sharma *et al.* (1984). They reported a 12.73 per cent increase in milk yield of infected lactating buffaloes after treatment.

Pediculosis caused economic losses in dairy cattle due to anaemia, unthriftiness, poor feed utilization, which resulted in decreased weight gain and lowered milk production (Joseph *et al.*, 1986).

Treatment with febendazole in buffaloes resulted in an increased milk yield of 1.34 litres per day per animal in cases of *Ostertagia ostertagi* infection in buffaloes (Srivastava, 1990).

Among the haemoprotozoan infections, trypanosome infection of buffaloes was manifested by intermittent fever (upto 105°F), salivation, lacrimation and

congestion of conjunctivae and loss of milk yield (Singh and Joshi, 1991). The low level of milk yield was due to anaemia as a result of haemoprotozoan infection.

Bhongade *et al.* (1993) revealed that clinical symptoms of animals naturally infected with helminth parasites included reduced milk production, low levels of haemoglobin, blood glucose, total protein and calcium.

Drug trials were conducted in 40 infected cattle to compare the efficacy of oxyclosanide, hexachlorophene and nitroxynil against natural infection of amphistomes (Manna et al., 1994). Oxyclosanide proved to be more effective than the other two and it also increased the milk yield upto 25 per cent than that of the untreated control.

2.2.1.4 Infectious Diseases

The physiological functions of importance during the periparturient period are; adaptation of the rumen to lactation diets that are high in energy density, maintenance of normocalcemia and a strong immune system. The incidence of both metabolic and infectious diseases is greatly increased whenever one or more of these physiological functions are impaired (Goff and Horst,1997).

In addition to metabolic diseases, the overwhelming majority of infectious diseases especially mastitis, Johnes disease and Salmonellosis cause a reduction in the lactation yield (Petrie, 1987). Bovine salmonellosis is characterized by haemorrhagic enteritis, inappetance, pyrexia and decrease in milk yield. Dramatic drop in milk yield was also reported in animals suffering from paratuberculosis by the same author.

According to Dutta *et al.* (1995) subclinical mastitis caused reduction in milk yield but without any clinical feature of mastitis except a high somatic cell count. It was responsible for a daily yield loss of 1.24 kg per cow and 2.65 kg per buffalo.

Clinical mastitis especially coliform mastitis was mostly likely to occur during the first month of lactation. During that period the activity of the immune system of the cow was depressed and the serum concentration of immunoglobulin, complement and conglutinin are reduced (Goff and Horst, 1997).

Maiti *et al.* (1997) opined that subclinical mastitis a major constraint to dairy industry due to its very high prevalence, reduction in milk yield and cost of treatment. Moreover, failure to regain milk production increased the economic losses due to this disease entity.

Metritis a post parturient uterine disease that adversely affected the dairy cows milk production during the post parturient period. It was also a risk factor for post parturient diseases like abomasal disorders and ketosis (Overton *et al.*, 2003).

Ghosh *et al.* (2004) states that incontrast to the visible changes in the acute form of mastitis, absence of gross abnormalities in the milk or udder in the case of subclinical mastitis, delays treatment and thus increases the economic loss due to lowered milk production. Apart from this it also was a source of infection to other herd mates and gradually made the secretary tissues of the mammary gland unproductive.

2.2.1.5 Metabolic Diseases

The metabolic diseases that occur during the transition stage in dairy cattle are milk fever, ketosis, retainted placenta and displacement of abomassum. The

metabolic demands imposed on the cow by the formation of colostrum far exeeds the demands of the fetus and results in an energy deficit in early lactation.

Due to the energy deficit occurring during early lactation a certain amount of fatty acid was oxidized completely by the tricarboxylic acid cycle of the liver or transported in the form of very low density lipoprotein, which accumulated in the hepatocyte impairing gluconeogenesis and resulted in hypoglycemia and reduction of milk yield (Reid and Roberts, 1982).

The production of just 10 kg of colostrum requires 11 Mcal of energy, 140 g of protein, 23 g of calcium, 9 g of phosphorous and 1g of magnesium be supplied from the diet or be brought to the mammary gland from the body stores. Thus the onset of lactation imposed tremendous physiological challenges to the homeostatic mechanisms of the cow (Goff and Horst, 1997). The metabolic diseases usually associated with hypogalactia are subclinical ketosis, hepatic lipidosis and left displacement of abomassum..

Andrews (1998) stated that most cases of chronic ketosis were due to lack of access to feed, starvation, reduced appetite, poor quality feed or an excessively high milk production. Most of these animals reduced or stopped milking, or very occasionally they continued lactation and became very thin.

Inappetance to complete anorexia, a marked drop in milk production and varying degrees of ketosis based on ketonuria are the clinical features of LDA (Left-Side displacement of the abomasum) as reported by Blood et al. (2000).

Kumar and Radhika (2001) opined that hypoglyceamia, ketonaemia and fat accumulation in internal organs especially liver was a common feature of hepatic

lipidosis. Clinical manifestations of hepatic lipidosis include anorexia, reduced milk yield in lactating cows, progressive debility, in-cordination and nervous signs.

Anjilappa and Suryanaraya (2002) concluded that hypoglycaemia in sub-clinical ketosis was presumably due to depletion of hepatic glycogen being utilized by mammary gland for lactose synthesis particularly in animals maintained under poor nutritional conditions.

2.2.1.6 Hormonal Imbalances

Hypothyroidism in dairy lactating dairy cattle was characterized by lowered milk production, lowered resistance to infection, increased susceptibility to ketosis, late abortion and retention of placenta (Wilson, 1975).

Ratnakumar *et al.* (1990) from the observations of their study of prevalence of hypothyroidism, recommended iodine supplementation in cases of non-infectious reproductive and production disorders.

Blanter *et al.* (1949) stated that thyroinine favourably influenced feed consumption, the rate and volume of blood flowing to the udder, the composition of blood, especially the precursors of milk and cellular activity of the mammary gland cells. At the same thyroprotein when fed at peak lactation stimulated production by 10 per cent.

Growth hormone increased amino acid uptake and incorporation into protein, stimulated synthesize of new RNA and enhanced glucose utilization (Park and Jacobson, 1988).

Estrogen caused a proliferation of the mammary tissue. It influenced the mineral metabolism causing a retention of phosphorous and sodium which caused water accumulation in tissues and excretion of potassium (Turner *et al.*, 1976).

2.3 THE METABOLIC PROFILE TEST

The Compton Metabolic Profile Test (Payne *et al.*, 1970) was defined to function as an aid in the diagnosis of production disease in dairy herds.

The Metabolic Profile Test is based on the assessment of the levels of certain metabolites in blood, the combination of which is defined as an animal's metabolic profile, used to forecast or diagnose the presence or abscence of metabolic disease. The term "metabolic disease" embraces those diseases also known as production diseases such as hypomagnesemia and ketosis and other less well known conditions which affect an animal's milk production or fertility (Rowlands *et al.*, 1976).

The metabolic profile test was proposed for four general uses. They are, to predict the likelihood of the occurrence of metabolic disease, to diagnose or confirm the diagnosis of metabolic disease, and to assess fertility and nutritional status of the herd as a whole (Ingraham and Kapples, 1988).

Pandey and Parai (1988) opined that the nature of the metabolic disease depended on the feeding, managemental and climatic conditions. This often resulted in complex metabolic problems in lactating cows which were difficult to diagnose and treat.

The constituents commonly monitored under the metabolic profile test are packed cell volume, blood haemoglobin, blood glucose, serum urea nitrogen,

albumin, globulin, inorganic phosphate, calcium, magnesium, copper and thyroxine (T_4) as per Whitaker (2000).

According to Huxley (2004) metabolic profiling involved collection of samples from small groups of cows at different stages of production which were analysed for a number of defined parameters.The animals were grouped on the basis of the stage of lactation as; dry cows within a few weeks of calving, early lactation cows, which calved approximately 10-40 days back and mid lactation which calved approximately 3-4 months back. Animals should not be sampled within a few hours of a large concentrate feed or within a few weeks of a significant change in diet.

2.4 HAEMATOLOGICAL PARAMETERS

2.4.1 Haemoglobin

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

Payne (1974) reported a fall in haemoglobin concentration with increasing milk yield. The value obtained in dry, medium and high yielding cows were 11.7, 11.2 and 10.8 g / 100 ml respectively.

Haemoglobin concentrations showed a definite fall with increasing milk yield irrespective of the season as per the survey conducted by Payne *et al.* (1974).

Amstuz (1981) stated that haemoglobin value of 'downer cow' was 10 g/dl.

Study of the physiological status of fifty apparently healthy lactating Hariana and Sahiwal cattle in the fourth stage of lactation, revealed that the haemoglobin levels were within normal limits and almost identical irrespective of the breed. The

haemoglobin values were 10.15±0.68 and 10.05±0.54 g per cent respectively for Hariana and Sahiwal dairy breeds (Pyne and Maitra, 1981).

The haematobiochemical profile of dry, recently parturited and advanced pregnant dairy cows studied by Prasad *et al.* (1987) revealed that the level of haemoglobin was slightly higher in dry cows compared to the other two groups. The mean haemoglobin levels were 10.11±0.25, 9.22±0.50 and 9.23±0.31 g per cent respectively for the three groups.

The haemogram of 24 healthy lactating cows in second or third lactation with signs of agalactia or hypogalactia revealed the haemoglobin levels was 11.25±0.25g per cent (Galhotra, 1990).

Bhongade *et al.* (1993) in a study on the effect of antehelmintic therapy on haematobiochemical profiles of dairy cows, which calved 3-6 months back and in the second to eight lactation found that the affected animals had reduced milk production and low levels of haemoglobin compared with the clinically healthy cows, kept as control. They recorded the haemoglobin values of cows infested with helminth endoparasites as 7.16 ± 0.32 to 8.0 ± 0.22 g per cent while corresponding values in healthy control cows were 11.34 ± 0.32 to 12.42 ± 0.29 g per cent.

The haemoglobin value of downer cows was found to be 12.20 ± 0.35 g per cent by Khatsu *et al.* (1998).

Pillai and Alikutty (1995) studied the clinico-haematological changes in bovine amphistomosis. They recorded the haemoglobin value as 9.8 g/dl. The loss

of blood due to penetration of the duodenal wall by the parasite exacerbated by the improper protein absorption across the intestinal wall resulted in anaemia.

Baitule *et al.* (1999) studied the efficacy of a herbal glactogogue in treatment of post parturient hypogalactia. They stated that pre-treatment haemoglobin levels of 10.9 g per cent were significantly lower when than post treatment levels of 11.93 g percent. They reasoned that the rise in haemoglobin values was due to the effect 'Aswagandha' which was reported to possess both tonic and haematinic effects.

Kalsi *et al.* (2002) reported significant low haemoglobin values in dairy cows having laminitis. The haemoglobin values in healthy cows were 12.28 ± 0.42 g/dl and in cows having laminitis was 10.49 ± 0.28 g/dl. These changes were indicative of a mild to moderate degree of anaemia probably as a result of reduced feed intake due to decreased rumen mobility and decreased erythropoietic activity.

Soodan *et al.* (2004) found the values for haemoglobin to be 10.58±0.28g per cent for healthy, high yielding crossbred cattle and buffaloes

2.4.2 Volume of packed Red cells

Prasad (1987) reported the PCV values of dry, recently parturited and advance pregnant cows were 32.42±0.65, 29.71±1.60 and 30.71±0.81 per cent respectively for the three groups.

Pandey and Parai, (1988) found that the PCV values of 10 recently parturited normal crossbred cows was 30.20±0.44 per cent while that of cows suffering from production disease was 38.81±0.59 per cent.

Galhotra (1990) on studying the haemogram of 24 healthy lactating cows which were either in their 2nd or 3rd lactation and showing signs of agalactia or hypogalactia, reported that the PCV values was 33.98±0.98per cent.

Khatsu *et al.* (1994) in a study on the metabolic profile of downer cows have recorded that the PCV value of healthy crossbred cows was 29.86±0.62 percent.

Kalsi *et al.* (2002) recorded a low PCV value of 31.67 ± 1.29 percent in lactating dairy cows with overgrown hoofs when compared to 38.00 ± 1.19 percent in healthy control cows. PCV values when correlated with haemoglobin values indicated that such animals were anaemic which inturn resulted in reduced milk yield.

2.5 SERUM BIOCHEMICAL PARAMETERS

2.5.1 Serum Glucose

Payne et al., (1970) reported that the mean blood glucose concentration among dairy animals varied from herd to herd with a range of 36.7-54.1 mg/dl with a mean of 45.4mg/dl.

Kaneko (1980) recorded the blood glucose concentration in healthy adult cow to be 45-75mg per cent with a mean value of 57.4±6.8mg/dl.

Amstutz (1981) reported that the serum blood glucose levels in downer cows were 101 mg/dl as against normal values of 49-82 mg/dl.

Metabolic profile of dairy cows in different stages of lactation revealed that blood glucose levels 31-60 days after parturition was 51.2±1.029 mg per cent which

further declined to 50.5 ± 1.319 mg per cent followed by a gradual rise thereafter (Pradhan and Chakaborti, 1986).

Gupta and Rai (1987) recorded that the blood glucose levels in dairy cattle 3-4 days after calving was 48.74 ± 10.68 and one month after calving was 53.88 ± 11.56 mg percent . They attributed the low blood glucose levels in the early stages to be due to feeding of a low energy ration in advanced pregnancy.

Pandey and Parai (1988a) in a report on the clinico-biochemical profile of complex metabolic disease in cows, recorded the blood glucose values in affected animals as 32.81 ± 0.51 mg per cent as against 36.76± 0.46 mg per cent.

Pandey and Parai, (1988) reported that atypical production disease in crossbred cows was due to marked reduction in blood glucose value (29.47±0.52) as against 47.31±0.99mg/dl in the normal control cows.

Pandey and Parai, (1989) reported that the mean serum glucose value in healthy cows at calving was 41.73±0.71 mg/dl which showed a decreasing trend varying from 36.76±0.46 to 41.73±0.71 mg/dl for almost one month after calving. However blood glucose levels returned to pre-calving glucose level by the 60th day post calving (48.80±2.06 mg/dl).

Anantwar and Singh, (1993) observed significant decrease ($P<0.01$) in blood glucose levels during the post parturient period (51.95±3.83mg per cent) and attributed it to the rapid utilization of glucose by the mammary gland to synthesize lactose coupled with inadequate feed intake.

The blood glucose level of downer cows were 48.85 ± 1.25 mg per cent as reported by Khatsu *et al.* (1994).

Pillai *et al.* (1994) reported that the blood glucose levels of apparently healthy post parturient dairy cows was 49.45±2.36 mg/dl while that of cows suffering from post parturient indigestion was within the range of 40.55-45.68mg/dl.

Gupta *et al.* (1995) reported that the mean serum glucose level in healthy cows one month after calving was 53.88±11.59mg/dl.

Rao and Suryanarayanan (1996) stated that both subclinical ketosis and hypocalcaemia were among the leading causes of post parturient anorexia. They recorded the glucose levels in 1st, 2nd and 3rd month and beyond as 30.25 ± 2.1, 39.4 ± 1.2, 45.5 ± 2.1 mg/dl respectively in cattle and 36.0 ± 1.2, 38.01 ± 1.3 and 47.0 ± 2.2mg/dl respectively in buffaloes.

In a treatment trial on post parturient hypogalactia, the elevation of glucose level post treatment might be due to the fact that milk yield was being increased resulting in high blood glucose levels and more transport of blood glucose to mammary gland for synthesis of milk. This marked effect on glucose levels by a herbal galactogogue was reported by Baitule et al. (1999).

Patel and Jadhav, (2003) while studying the metabolic profile of apparently healthy buffaloes from areas of high and low incidence of post parturient hypocalcaemia reported that the glucose levels of clinically healthy buffaloes was 56.310 mg /dl.

Soodan *et al.* (2004) reported the blood glucose level in dairy cattle during first stage of lactation was 41.75±1.97, during an epidemiological study on metabolic profile status of lactating dairy cattle and buffaloes.

Jagatheesan *et al.* (2005) opined that negative energy balance and post parturient stress might be the reason for reduced blood glucose levels during early lactation compared to the levels at third trimester of gestation (40.03 ± 1.54 mg/dl).

2.5.2 Serum Calcium

Physiological studies on lactating Hariana and Sahiwal cattle over four years of age by Pyne and Maitra, (1981) revealed a calcium level of 9.75±0.12mg/dl and 10.05±0.25mg/dl respectively.

McAdam *et al.* (1982) revealed that there was a tendency for calcium concentrations to decrease shortly after calving particularly in animals that have been through one or more lactations.

Low levels of serum calcium and phosphorus on the day of calving had a significant relationship with cases of retention of placenta as reported by Shukla *et al.* (1983). The serum calcium level was 10.36 ± 0.37 and 9.89 ± 0.07 mg percent in normal and affected cases respectively. There was a gradual increase in serum concentration of calcium 1 to 20 days post partum.

The serum calcium levels of cows yielding six to eight l/day was found to be in the range of 9.17±0.18 to 9.21±0.16mg/dl in a study conducted by Mode *et al.* (1986).

Pradhan and Chakaborti, (1986) reported the serum calcium levels of crossbred cows two to thirty, 31 to 60 and 61 to 90 days after parturition was 10.1±0.197,10.8±0.158 10.7±0.127mg percent respectively.

Gupta and Rai (1987) opined that decreased serum calcium levels one month after parturition may be probably due to the increased milk production during

the period resulting in more excretion of calcium through milk. Serum calcium levels one week and one month after calving were recorded as 11.00 ± 0.96 and 10.26 ± 0.81 mg/dl respectively.

Prasad (1987) reported that the serum calcium levels in recently parturited cows to be 8.79±0.43mg per cent.

Studies on the biochemical aspects of atypical cases of production diseases in crossbred herds revealed a markedly low level of serum calcium, 5.19±0.06 mg percent in clinically ill animals as against normal values of 8.87±0.08mg percent (Pandey and Parai, 1988).

Bhongade *et al.* (1993) while studying the effect of anthelminthic therapy on haemato-biochemical profiles of lactating cows reported the normal serum calcium of non-descript and crossbred cows was 8.99±0.09mg per cent and 9.28±0.24mg per cent respectively.

Rao and Suryanarayana (1996) opined that depletion of serum calcium levels during the post partum period might be due to the sudden drainage of calcium through colostrum, or lactational stress. Insufficiency or inactivity of the parathyroid gland may be another factor. They stated that blood calcium levels in first, second and third month and beyond was 6.12 ± 0.2, 7.0 ± 0.4, 7.0 ± 0.2 mg per cent in cattle and 6.0 ± 0.1, 6.0 ± 0.1 and 7.0 ± 0.1 mg percent in buffaloes.

Goff and Horst (1997) suggested that low calcium and high phosphorus diet should be fed during lactation as it prevented the rapid reduction in serum total and ionized calcium levels associated with parturition. This was possible by inducing

compensatory hypertrophy of parathyroid glands which produced sufficient calcium-mobilizing hormone and averted the drop of blood calcium during early lactation.

Rosol *et al.* (1997) documented that 30 percent of the extra cellular calcium is in the form of ionized calcium which is the biologically active form of Ca^{2+}. Serum Ca^{2+} was lower in post parturient cows with two or more lactations compared to cows in the first lactation.

The serum calcium level of hypogalactous dairy cattle was 10.23 mg/dl. (Jayashree *et al.*, 1999). After treatment with a herbal galactogogue although milk yield increased from 3.83 to 6.70 l/day there was no significant effect on serum calcium level.

The serum calcium values of hypocalcaemic cattle was 5.68 ± 0.18 mg/100 ml compared to normal values of 10.94 ± 0.57 mg/100 ml. (Dasan and Divya, 2001)

Patel and Patel (2001) gave the normal serum calcium level in healthy buffaloes as 8.04±0.49mg per cent.

The mean serum calcium level in early lactation varied significantly from the values of cows in mid and late lactation .The serum calcium level in early lactation stage was low since cows were in a state of negative energy for 8-10 weeks post calving. The level of calcium was 11.03±1.60mg/dl as against the mid lactation and late lactation levels of 12.82±2.03 and 13.2±2.29mg/dl respectively (Bhatt *et al.*, 2002).

Sarkar *et al.* (2004) observed that main cause of metabolic and deficiency diseases in lactating animals were due to the non availability of certain non specific

elements in the soil. Serum calcium level of healthy lactating animals was given as 11.11 ± 0.23 mg/dl.

Soodan *et al.* (2004) reported a calcium deficient status among animals in the first stage of lactation and attributed the cause to be due to excessive feeding of paddy straw, mineral imbalance in soil-plant-animal system and inadequate mineral supplementation. The calcium level in serum of lactating cows was as 8.811±0.08mg/dl.

Jagatheesan *et al.* (2005) stated that difference in levels of serum calcium during the third trimester and early lactation were not statistically significant.Serum calcium levels during third trimester was 5.55 ± 0.33 mg/dl and that during early lactation was 6.12 ± 0.50 mg/dl.

2.5.3 Serum Phosphorous

Payne et al., (1970) reported that the mean serum inorganic phosphorus levels in dairy cattle were 5.42mg/dl and its range varied from 3.2 to 7.2mg/dl.

Kaneko (1980) observed that the serum inorganic phosphorus level in normal adult cattle varied from 4.0 to 7.0mg per cent (1.3-2.3 m mol/l).

Pyne and Maitra (1981) reported the serum phosphorus levels of apparently healthy lactating Hariana and Sahiwal cattle in 4th lactation to be 5.17±0.24 and 5.08±0.18mg/dl.

Peterson *et al.* (1981) in a study on physiological status and blood picture revealed that levels of inorganic phosphorus in lactating non-pregnant, lactating

pregnant and dry cows was 5.01±0.11, 5.55±0.10 and 6.20±0.28 mg per cent respectively.

Kulkarni *et al.* (1983) stated that inorganic phosphorus levels in lactating Gir and crossbred dairy cows was 5.71-6.41 and 5.03 – 5.86 mg per cent respectively.

In a study on the serum biochemistry of cases of retained placenta, Shukla *et al.* (1983) found a relatively low phosphorous levels (4.59 ± 0.10) in affected animals on the day of calving cases compared to the levels in normal cows (5.30 ± 0.10 mg/dl) which calved on the same day.

Kulkarni *et al.* (1984) stated that inorganic phosphorus levels in lactating and dry Indian buffaloes are 4.97 ± 0.15 and 5.41 ± 0.53mg/dl. This difference however was not statistically significant.

Sivaiah *et al.* (1986) reported that recently calved animals (within 7 days of calving) had a phosphorus level of 7.18±0.90mg percent

Higher levels of serum phosphorus levels one month post partum (5.69 ± 0.38) compared to that during one week after calving (4.98 ± 0.39) was reported by Gupta and Rai (1987). The change in calcium to phosphorus ratio and the less levels of parathormone 3-4 days post partum may be the reason for the co-existing hypophosphataemia.

Prasad *et al.* (1987) reported that the mean serum inorganic phosphorus values in normal crossbred was aged 5-8 years during pregnancy and early lactation were 6.50±0.42 and 5.17±0.27mg/dl respectively.

Pandey and Parai (1988b) reported normal inorganic phosphorus in animals with atypical production disease and control animals to be 3.10±0.07 and 4.45±0.11mg/dl respectively.

Pandey and Parai (1989) recorded the serum inorganic phosphorus values of 2.88 ± 0.04 mg per cent in complex metabolic disease of cows.

According to Gupta *et al.* (1995) the mean serum inorganic phosphorus level in healthy cows one month postpartum was 5.69±0.38mg/dl.

Baitule (1999) found no significant increase in serum phosphorus levels in a treatment trial of post parturient hypogalactia with a herbal preparation, although milk yield was improved considerably. The values recorded pre and post treatment are 4.96 and 4.76 mg/dl.

Dasan and Divya (2001) recorded the phosphorus levels of hypocalcaemic dairy cattle as 2.48 ± 0.18 mg/dl while the value in clinically healthy animals were 4.98 ± 0.31 mg/dl.

Jagatheesan *et al.* (2005) reported that the serum phosphorus levels of Murrah buffalo during early lactation was 8.66 ± 0.50 mg/dl as against 8.45 ± 0.40 mg/dl during the last trimester of pregnancy.

2.5.4 Serum Magnesium

Mylrea and Bayfeild (1968) reported that the serum magnesium levels of healthy cows in milk to be 2.4±0.36mg/dl.

Pyne and Maitra (1981) reported the serum magnesium levels in lactating Hariana and Sahiwal cattle to be 2.66±0.06 and 2.70±0.04mg/dl respectively.

McAdam (1982) reported an increase in maternal plasma magnesium concentrations at parturition.

Gupta and Rai (1987) reported that the higher magnesium concentration during the advance stage of gestation phase reduced within 24 hours of parturition but reached normal levels within one month post partum. Serum magnesium values one month after parturition were recorded as 2.57 ± 0.19 mg/dl in cow and 2.67 ± 0.14 mg/dl in buffaloes.

Prasad *et al.* (1987) recorded low levels of serum magnesium levels 3.44±0.18mg/dl as compared with that of dry cows and cows in advanced stage of gestation.

Serum calcium and magnesium levels in dairy cows were examined by Mode *et al.* (1986) to observe their relationship with milk production in different seasons. He reported that levels were higher in summers. Many other factors like plant type fertilizers, soil, climatic factors and the stage of maturing all influence serum magnesium levels. The average serum magnesium concentration of non-lactating cows was lower than those of lactating cows. This was attributed to supplementation of the same in the diets of lactating animals.

Serum magnesium levels in downer cows was reported as 1.8 mg/dl by Amstutz(1981) while normal values are 1.5 to 3.5 mg/dl.

According to Pandey and Parai (1988b) the value of serum magnesium of atypical cases of production diseases was almost similar to normal values. The

values obtained was 2.98±0.08mgper cent as against the values of control cows of 3.09±0.06.

Pandey and Parai (1989) reported that serum magnesium values (3.024±0.136mg/dl) were high on the calving day than that on five days before parturition when the levels were 2.66±0.126mg/dl in healthy crossbred cows with daily milk yield of 15 liters in the previous lactation.

Patel and Patel (2001) gave the normal values of serum magnesium of buffaloes as 2.95±0.54mg per cent.

Bhatt *et al.* (2002) reported that the serum magnesium concentration of early lactation, mid lactation and late lactation to be 48.27±0.829, 29.63±2.49 and 41.05±2.62 respectively. He attributes the cause either due to the inadequate dietary intake by cows in mid and late lactation or it may also be due to high intake from production rations by cows in early lactation.

2.5.5 Total serum albumin and serum protein concentration

Amstutz (1981) recorded the serum protein levels in downer cows as 5.9 g/dl and albumin level as 2.5 g/dl. The corresponding normal range is healthy lactating animals was 5.9-8.1 and 3.0-4.0 g/dl respectively.

Pyne and Maitra (1981) during a study on the blood physiology of lactating Hariana and Sahiwal cattle has given serum protein levels in Hariana and Sahiwal breeds as 6.95±0.42 and 7.02±0.5g per cent respectively.

Kulkarni *et al.* (1984) recorded the total protein and albumin levels of lactating Indian buffaloes as 6.47 ± 0.06 g per cent and 3.17 ± 0.04 g per cent. Corresponding levels in dry buffaloes was 6.00 ± 0.07 g per cent and 3.34 ± 0.07 g per cent.

Pandey and Parai (1988) reported that the total protein value in complex metabolic disease of cattle occurring during first stage of lactation was 7.92 ± 0.10 g per cent.

Pandey *et al.* (1989) reported that the total serum protein value was unaffected by calving.

Bhongade *et al.* (1993) reported a low level of total serum protein in crossbred cows (6.85±0.20) than that in nondescript cows (6.76±0.17). These animals were infected with helminth parasites and had calved three to six months back.

Khatsu (1994) reported a significant decrease in serum protein and highly significant decrease in serum albumin in recently calved cows. This may be due to inadequate production, malnutrition and physiological hypoproteinemia during pregnancy and onset of lactation. Normal values for total protein and albumin in control cows were 7.886±0.32g/dl and 3.375±0.125g/dl respectively.

Baitule (1999) in a treatment trial of a herbal galactogogue in post parturient hypogalactia, reported significant increase in serum total protein and albumin level and that the drug had a stimulatory effect on protein synthesis.

Kumar *et al.* (2001) reported the serum albumin values of clinically healthy lactating buffaloes between two to three years of age to be 2.98±0.72 g/dl.

The total serum protein in hypogalactous dairy cattle with overgrown hoofs was given as 8.99 ± 0.44 g/dl compared to 8.45 ± 0.14g/dl in healthy control cows, by Kalsi *et al.* (2002).

The mean serum total protein levels recorded in hypocalcaemic dairy cattle by Dasan and Divya was 6.77 ± 0.26 g/dl which was higher than those levels in normal animals (6.395 ± 0.19 g/dl). Albumin value was reported as 3.97 ± 0.18 g/dl and globulin level as 2.81 ± 0.26 g/dl in the hypocalcaemic cases.

Soodan *et al.* (2004) in an epidemiological study on metabolic profile status of lactating dairy cattle and buffaloes in first stage of lactation gives the total plasma protein and albumin levels (g/dl) as 6.53±0.16 and 3.53±0.16 respectively.

In a study of blood biochemical constituents in Murrah buffaloes, Jagatheesan *et al.* (2005) opined that serum protein levels were slightly higher during early lactation (9.28 ± 0.30 g/dl) than during the third trimester of pregnancy (8.88 ± 0.24 g/dl).

They found a positive co-relation between serum protein levels and milk yield. Furthermore, synthesised of protein for milk production might be the reason for elevated total protein level during early lactation.

2.5.6 Serum Blood Urea Nitrogen (BUN)

Amstutz (1981) in a case report on downer cow recorded the BUN values as 8 mg/dl while the normal values are given as 4-19 mg/dl.

Peterson *et al.* (1981) in a study on purebred Holstein - Fresians above 2 years of age revealed that the physiological state of the animal (lactating, pregnant, dry cows etc.) and the feeding and management regimen had significant effects on serum blood urea nitrogen levels. Serum BUN was lowest for dry cows, higher in early lactation and increased linearly with the days milked.

Kulkarni *et al.* (1983) while discussing the biochemical indices of lactating Gir and crossbred dairy cows stated that the blood urea nitrogen levels did not vary significantly between breeds. Levels recorded were 13.83-15.50 mg/dl and 13.83-15.83 mg/dl respectively.

The urea nitrogen concentration of serum biochemical constituents of lactating and dry buffaloes was given as 20.29 ± 1.002 mg/dl and 16.15 ± 0.87 mg/dl respectively by Kulkarni *et al.* (1984).

Raina *et al.* (1993) has recorded the non-protein nitrogen values of non-descript Kashmiri cows and Jersey cows to be 27.29±4.64 and 28.56 ±6.89 mg/dl respectively.

Khatsu (1994) has observed that normal blood urea nitrogen concentration of cross bred cattle in Thrissur district to be 21.91±2.24mg/dl and that in downer cows was 17.29 ± 1.74 mg per cent.

In an investigation undertaken to study some parameters of high yielding crossbreds Soodan *et al.* (2004) gives the blood urea nitrogen levels of cows in first stage of lactation as 20.24±0.79mg/dl.

2.5.6 Blood ketone bodies

Henry (1969) reported the spectrophotometric method of estimation of acetone and aceto acetic acid from blood. Preformed acetone and acetoacetate was isothermally distilled into alkaline vanillin, where they react to form red vanillal acetone. The normal ketone body level was 0.5 to 3 mg per cent.

Rajan and Ganapathy (1973) reported that the blood ketone levels in healthy cows was 1.3 to 1.9 mg per cent with a mean of 1.5 mg per cent.

The blood ketone bodies in animals with subclinical ketosis were 5.26 to 6.65 mg / dl. Ketonemia was better correlated with subclinical ketosis but ketonuria with clinical ketosis. Inappetance, hypoglycaemia, ketonaemia, moderate ketonuria and acidosis are significant findings of subclinical ketosis in cross bred cows (Baishya and Hazarika, 1998).

2.6 SERUM ENZYMOLOGY

2.6.1 Estimation of seum aspartate aminotransferase (AST)

Amstutz (1981) in a case report on downer cows recorded the SGOT value as 156 IU/l and the normal value as 36-69 IU/l. Elevated levels of SGOT might be due to muscle damage or concurrent liver damage.

Peterson *et al.* (1981) obsereved that the activity of serum aspartate amino transferase was influenced by the physiological state of the animal and the feeding or management regimen. Dry cows showed a higher activity of serum AST than cows in first stage of lactation.

A study on the physiological values of lactating Hariana and Sahiwal cattle by Pyne and Maitra (1981) revealed that serum AST levels were 51.15±0.85 and 52.35±0.74 Karman Units respectively..

Prasad *et al.* (1987) reported an increased level of serum AST in pregnant animals over that of early parturited animals. The values were 58.43±3.83 and 30.63±2.27IU/ml respectively and this might be due to the gestational stress on the liver.

Pillai *et al.* (1994) reported that 35.64 per cent of the cows suffering from post parturient indigestion (PPI) had liver dysfunction and they were of the age group ranging between 3-5 years. The serum AST levels in healthy lactating animals and that in animals suffering from PPI was 28.67±4.105 U/ml and 78.6±3.04 U/ml respectively.

Significant increase in serum AST levels in subclinical ketosis was reported by Venkateswaralu (1994), which probably might be due to hepatic damage in ketotic cows. Before treatment levels of serum AST ranged between 100.75±6.50, 120.10±5.13 IU/l.

Dua and Bhatti (1999) reported that serum AST levels in healthy lactating buffaloes and cattle vary from 71.0-75.5 and 41.00 to 46.5 U/l respectively.

Kumar *et al.* (2001) recorded that the serum AST value of healthy lactating buffaloes was 62.72 ±3.91 IU/l and those infected by intestinal amphistomes was 68.18±4.24 IU/l respectively.

Kalsi *et al.* (2002) recorded the serum SGOT levels in dairy cattle with overgrown hoofs and with a history of reduction in milk yield as 88.00 ± 10.16 as against 55.71 ± 8.68 IU/l in healthy control cows. They opined that this significant rise in mean activity of SGOT could be ascribed to muscle damage and hepatic insufficiency.

Soodan *et al.* (2004) reported that serum AST levels of multiparous animals in first stage of lactation was 50.94±0.86 IU/l.

Jagatheesan *et al.* (2005) found that the SGOT values remained constant during the third trimester and early lactation in Murrah buffalo. Levels recorded in the third trimester and early lactation was 78.69 ± 3.60 and 88.33 ± 3.90 IU/l. These differences were not statistically significant.

Materials and Methods

3. MATERIALS AND METHODS

The present study was conducted in the Department of Clinical Medicine, College of Veterinary and Animal Sciences, Mannuthy over a period of two semesters.

3.1 DESIGN OF THE STUDY

The study consisted of dairy animals which suffered a 25 per cent reduction in milk yield when compared to the previous lactation and without any signs of clinical disease formed the experimental group (Group I, n=30) and apparently healthy multiparous animals in the first stage of lactation as control (Group II, n=6).

3.2 OUTLINE OF THE STUDY

3.2.1 Clinical Examination

Detailed clinical examination of the animals comprising of both group I and group II were conducted as described by Boddie (1962). The details of age, breed, parity, season of calving, stage of lactation, frequency of milking, milk yield (both present and that in previous calving/ calvings) were recorded.

3.2.2 Sampling and Analysis

About 15ml of whole blood was collected by jugular venipuncture from each animal using a labeled 20ml disposable syringe. Three mililitres of whole blood was transferred to clean, dry, labelled vials containing sodium fluoride at a concentration of 10-20mg/ml of blood collected, as anticoagulant. This was used for estimation of haematological parameters and blood glucose.

The 12ml of whole blood was left undisturbed. After clot retraction, about one-two hours later, the serum was centrifuged at 3000rpm for 15 minutes. The clear serum was then transferred into clean, dry, labelled plastic vials and stored at –20°C, until further analysis was carried out. The urine, milk and dung samples were also taken in labelled vials for performing relevant laboratory examination.

3.2.2 Examination of Clinical Materials

3.2.2.1 Screening of Faecal Samples

Faecal samples were triturated in a mortar and pestle. The suspension was sieved and the filtrate was centrifuged at 3000rpm for five minutes. The sediment was examined under low power of objective of a light microscope for the presence of ova of parasites.

3.2.2.2 Screening of Milk Samples

Milk samples from each animal were subjected to California Mastitis Test (Schalm et al., 1971) to rule out cases of clinical and subclinical mastitis.

3.2.2.3 Screening of Urine Samples

Urine samples from each animal were subjected to Rothera's test (Benjamin, 1975) to rule out the presence of ketosis.

3.2.3 Metabolic Profile

3.2.3.1 Evaluation of Hematological Parameters

Haematological parameters such as haemoglobin content (Hb) and volume of packed red cells (VPRC) were determined on the day of collection as per methods described by Schalm *et al.* (1975).

3.2.3.2 Serum Biochemistry

Total serum glucose, protein, albumin and phosphorous were estimated using the kits supplied by Merck. Blood urea nitrogen was measured by the kit supplied by Agappe. Photometer 5010 (Boehringer Wanheim) under standard conditions of operation was used for biochemical analyses.

Serum calcium was estimated by modified o-cresolphthalein method (Endres and Rude, 2001). Serum magnesium was estimated by calmagnite method (Grindler, 1971).

Serum glucose was estimated by glucose oxidase peroxidase 4 amino antipyrine (GOD-PAP) method (Burtis and Ashwood, 2001).

Serum protein was estimated quantitatively by biuret method (Johnson *et al.*, 2001).

Serum albumin was measured by automated dye binding method using bromcresol green (BCG) dye. This dye has great affinity for albumin, therefore the initial rate of binding is measured and related to the concentration of albumin in the sample (Johnson *et al.*, 2001).

Blood urea nitrogen (BUN) was estimated by glutamate dehydrogenase enzymatic method. (Newman and Price, 2001).

Serum inorganic phosphorus was estimated by phosphomolybdate method as given by Daly *et al.* (1972) using the kit supplied by Labkit, Spain.

Serum blood ketone bodies was estimated by spectrophotometric method using vanillin according to the method of Amlathe and Gupta (1990).

3.2.4 Serum Enzymology

3.2.4.1 Estimation of Serum Aspartate aminotransferase (AST)

Serum aspartate amino transferase (AST) levels was measured by coupling the aminotransferase reaction to specific dehydrogenase reactions (Henderson and Moss, 2001).

3.2.5. Statstical Analysis

The data collected were statistically analysed as per the method of Snedecor and Cochran (1985).

Results

4. RESULTS

Results of the parameters studied viz., haemogram, serum biochemistry and enzymology were statistically analysed and presented.

4.1 HISTORY AND EPIDEMIOLOGY

The animals of the experimental group (Group I, (n=30) were in the third to fifth calving and between 45 to 90 days of lactation. Animals, which showed clinical signs of any systemic disease, either infectious or metabolic, were not considered for the present study. Those animals, which showed a 25 percent reduction in milk yield as reported by the farmers, were selected for the study. All the animals were maintained under identical conditions of feeding and management. Their age ranged between three to eight years and in their third parity. Most of the owners were livestock farmers whose sole source of income was agriculture. The animals were milked twice daily except three animals, which were milked thrice daily. Compounded cattle feed as per the recommendations of package of practice; green grass and paddy straw formed the feed of the animal. Fresh clean water was provided adlibitum.

Animals of control group (Group II, n = 6) consisted of animals, of the same stage of lactation as that of the Group I and yielded upto 9-18 liters of milk per day. They were fed with 25-30 kg green grass and one kilogram of compounded concentrate mixture for every 2.5 to 3 kilogram of the milk produced. They were maintained under similar housing and managemental conditions as Group I.

4.2 CLINICAL PATHOLOGY

4.2.1 Haematology

The comparison of the haemogram values between Group I and II are presented in Table 1.

4.2.1.1 Haemoglobin

The mean values of haemoglobin of animals of Group I and Group II are 9.87±1.24 and 11.37±0.95g per cent respectively. The reduction in haemoglobin level of Group I animals was statistically significant when compared to Group II animals.

4.2.1.2 Volume of packed red cells (VPRC)

Volume of packed red cells for Group I and II ranged between 29 to 34 percent and 28 to 32 per cent respectively. The mean values obtained in Group I and II were 31.68±2.46 per cent and 30.33±3.87 per cent which varied non-significantly. ($P<0.05$).

4.2.2 Serum Biochemistry

Serum biochemical values of animals of Group I and Group II are presented in table 2.

4.2.2.1 Total serum protein

Serum total protein of the animals ranged between 4.1 and 7.8 g/dl and that for Group II ranged between 7.6 and 8.4 g/dl. The mean value of total protein for Group I was 5.64±1.44 g/dl and that for Group II was 8.64±0.30 g/dl. A statistically significant decrease ($P<0.05$) was noticed in Group I animals when compared to control Group.

4.2.2.2 Albumin

Serum albumin levels of the animals of Group I ranged between 2.1 to 3.4 and that for Group II between 3.15 to 3.9g/dl. The mean value for Group I was 2.87±0.56g/dl and that for Group II was 3.59±0.32g/dl. A statistically significant decrease was noticed in Group I animals when compared to Group II ($P<0.05$).

4.2.2.3Globulin

Serum globulin concentrations of Group I ranged from 1.25 to 4.80g/dl with a mean of 2.69±1.08g/dl Group I and 3.50 to 4.8g/dl with a mean of 4.45±0.13g/dl in Group II. The difference in the values were statistically significant ($P<0.05$).

4.2.2.4 Blood glucose

The blood glucose level varied between 26-42mg/dl in animals of Group I with a mean of 36.02±4.27 mg/dl. The corresponding values in Group II varied between 52-62mg/dl with a mean of 57.38±4.31mg/dl. A statistically significant decrease was noticed in Group I animals when compared to Group II ($P<0.05$).

4.2.2.5 Ketone bodies in blood

Blood ketone body concentration of animals in Group I ranged between 3.42 and 6.65mg/dl with a mean value of 5.94±0.91mg/dl and that for Group II was 2.15 to 5.02mg/dl with a mean of 3.69± 1.03mg/dl. A statistically significant increase was noticed in the levels of blood ketone body of animals in Group I when compared to Group II($P<0.05$).

4.2.2.6 Blood Urea Nitrogen

The mean value of blood urea nitrogen of animals Group I was found to be 15.48±2.43mg/dl and that of Group II was 17.8±2.59mg/dl. There was statistically significant reduction in the values of Group I compared to Group II. ($P<0.05$)

4.2.2.7 Serum Calcium

Serum calcium levels of Group I ranged between 6.29 and 10.2mg/dl with a mean of 8.03±1.25mg/dl and that for Group II ranged between 8.5-11.5mg/dl with a mean value of 10.26±1.09mg/dl. The values differed significantly. ($P<0.05$)

4.2.2.7 Serum Phosphorous

The serum phosphorous levels of Group I ranged between 3.28 to 5.9mg/dl and that for Group II between 4.3 to 6.8mg/dl.The mean values for serum phosphorous were 4.79±1.29mg/dl and 5.40±1.08mg/dl respectively for Group I and Group II. The difference when analyzed statistically was found to be non-significant.

4.2.2.8 Serum Magnesium

The mean value of serum magnesium for Group I was 2.26±0.25mg/dl and that for Group II was 2.86±0.28mg/dl. No statistically significant difference was noticed in the serum magnesium values of animals in Group I when compared to Group II.

4.3.3 Serum Enzymology

4.3.3.1 Serum aspartate aminotransferase

The mean value of serum AST for animals of Group I was 72.07±5.65 IU/L and that for Group II animals was 61.00±7.24 IU/L. A statistically significant increase was noticed in the levels of serum AST of animals in Group I when compared to Group II ($P < 0.05$).

Table1. Haemogram of Hypogalactic and Control Animals

Parameters	Group I	Group II
Haemoglobin (g%)	9.87 ± 1.24^{a}	11.37 ± 0.96^{b}
PCV (%)	31.68 ± 2.46^{a}	30.33 ± 3.87^{a}

Table 2. Serum Biochemistry of Hypogalactic and Control Animals

Parameters	Group I	Group II
Total protein (g/dl)	5.64 ± 1.44^{a}	8.64 ± 0.30^{b}
Albumin (g/dl)	2.87 ± 0.56^{a}	3.59 ± 0.32^{b}
Globulin (g/dl)	2.69 ± 1.08^{a}	4.45 ± 0.35^{b}
Glucose (mg/dl)	36.02 ± 4.27^{a}	57.38 ± 4.31^{b}
Blood ketone bodies (mg/dl)	5.94 ± 0.91^{b}	3.69 ± 1.03^{a}
BUN (mg/dl)	15.48 ± 2.43^{a}	17.80 ± 2.59^{b}
Calcium (mg/dl)	8.03 ± 1.25^{a}	10.26 ± 1.09^{b}
Phosphorus (mg/dl)	4.79 ± 1.29^{a}	5.40 ± 1.08^{a}
Magnesium (mg/dl)	2.26 ± 0.25^{a}	2.86 ± 0.28^{a}
AST in IU/l	72.07 ± 5.65^{a}	61.00 ± 7.24^{b}

* Different superscript in a row indicate significant difference ($P<0.05$)

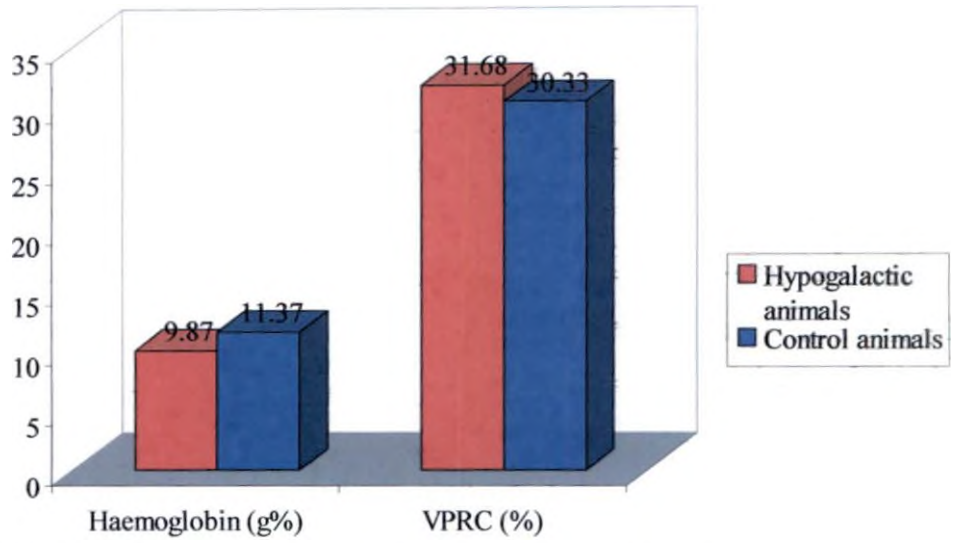

Fig.1 Haemoglobin and VPRC of hypogalactic and control animals

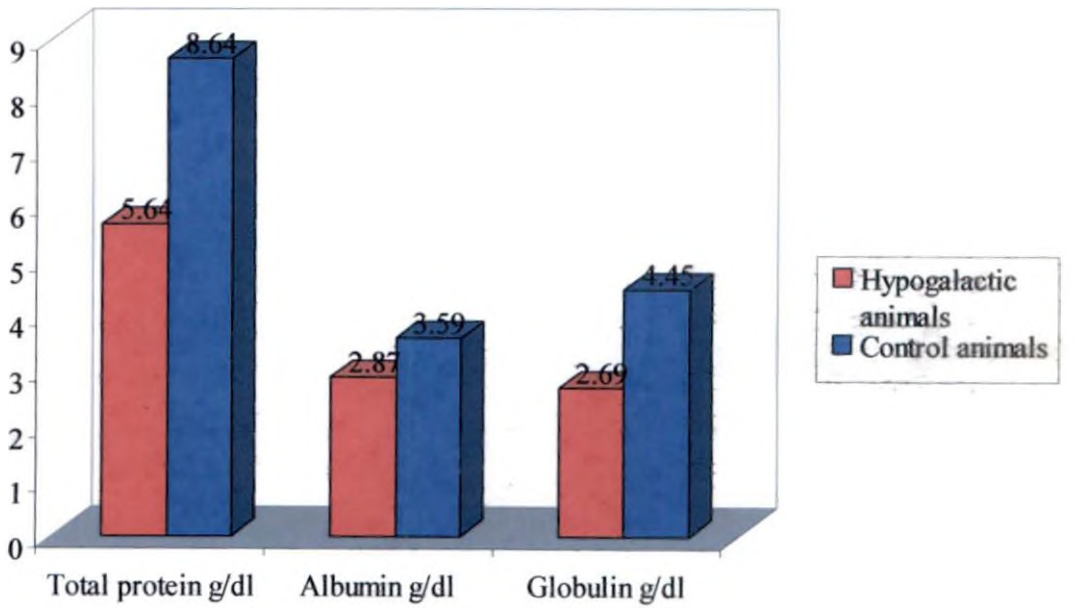

Fig.2 Serum protein status of hypogalactic and control animals

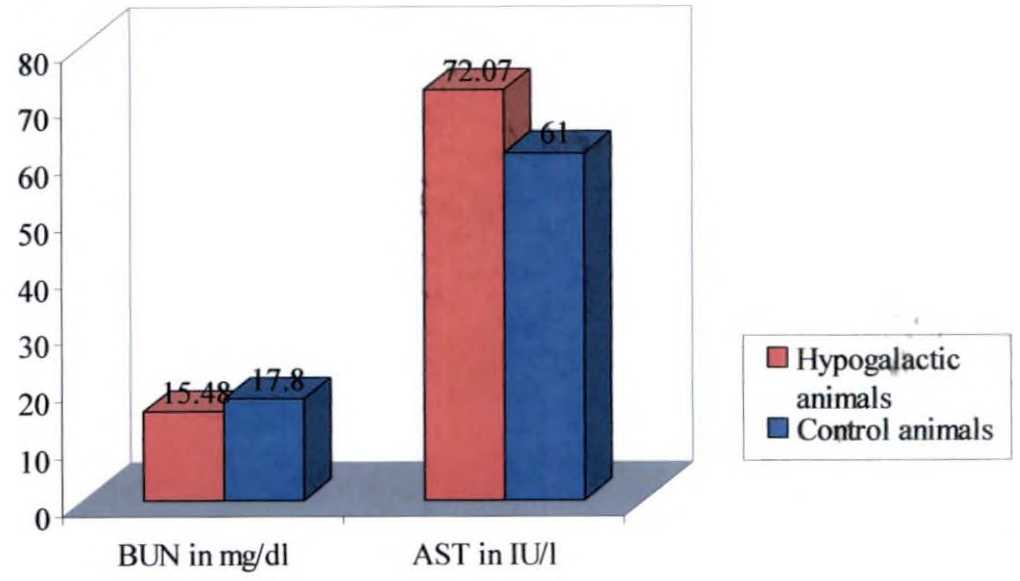

Fig.3 BUN and AST values in hypogalactic and control animals

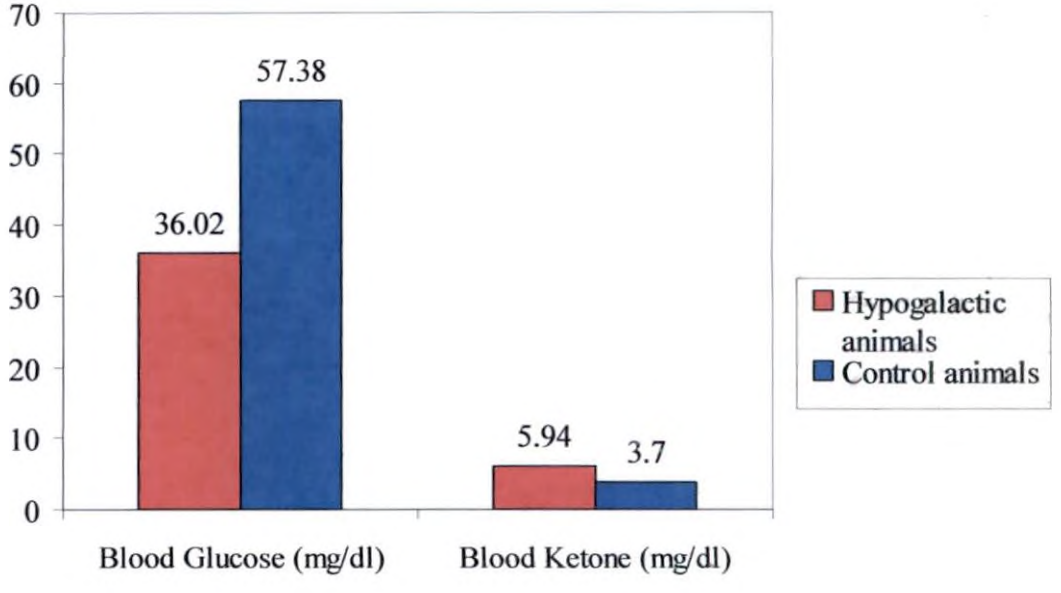

Fig. 4 Blood Glucose and Ketone values in hypogalactic and control animals

Discussion

5. DISCUSSION

Metabolic status of herds can be detected by studying the serum biochemistry. Manston and Allen (1981) stated that metabolic profile tests were intended to aid the diagnosis of metabolic problems. It was therefore sensible to select a profile of constituents which are likely to be pertinent to the problem being investigated. Camy (1985) suggested that a short but adequate metabolic profile obtained more rapidly and cheaply than the standard profile, consisted of glucose, urea, total protein, albumins, globulins, phosphorous, calcium and haematocrit values. The present study envisages an insight into the blood picture of hypogalactic dairy cows and to study the probable etiological factors to formulate a suitable therapeutic regimen for the same.

5.2 METABOLIC PROFILE

5.2.1 Haemogram

5.2.1.1. Haemoglobin

The mean value of haemoglobin obtained in the control group (11.37±0.96) was comparable with the values reported by Schalm (1975), Pyne and Maitra (1981) and Prasad *et al.* (1987).

A statistically significant decrease in the mean concentration of haemoglobin was found in hypogalactic cows (9.87±1.24). The values were similar to the values obtained by Venkateswarulu *et al.* (1994) for sub clinically ketotic animals and Pillai and Alikutty (1995) in cases of bovine amphistomosis. However Galhotra (1990)

gave the haemoglobin values of cows with signs of hypogalactia as 11.25±0.25g percentage.

The mean value obtained in the present study was slightly lower than that reported in hypoglactic dairy cattle by Galhotra (1990). Gopinath (2004) reported a reduction in milk yield due to parasitic anaemia in dairy animals which was manifested clinically by pallor of visible mucous membrane, exaggeration of respiration, anorexia and debility. Nutritional anemia due to copper and cobalt deficiency causing suboptimal productivity was also been reported by Goold and Smith (1975) and Choudhari, (1990).

The low values of haemoglobin obtained in the present study might be attributed to parasitic anemia and concurrent mineral deficiency causing nutritional anaemia.

5.2.1.1. Volume of packed red cells (VPRC)

The mean VPRC values of healthy cows agreed with that reported by Prasad *et al.* (1987) and Pandey *et al.* (1988). A non significant increase in the values of VPRC in cases of hypogalactia was obtained in the present study. The values concur with the findings of Galhotra (1990).

5.2.2. Serum biochemistry

5.2.2.1 Total Protein

The mean total protein value in healthy control cows (8.64±0.30g/dl) in the present study was in concurrence with the values reported by Amstutz(1981).

Khatsu (1994) reported that the normal serum protein levels in crossbred dairy cows was 7.87±0.32g/dl.

Jagatheesan *et al.* (2005) opined that serum protein levels were slightly higher during early lactation than during the third trimester of pregnancy. They found a positive co-relation between serum protein levels and milk yield. Furthermore, increased synthesis of protein for milk production might be the reason for elevated total protein during early lactation.

The mean values of total protein of hypogalactous dairy cattle in the present study was 5.64±1.44g/dl. This might have been due to the low protein diet, decreased hepatic synthesis as well as due to improper absorption across the intestinal wall.

5.2.2.2 Albumin

Mean serum albumin values of healthy control cows, in the present study was comparable with the values reported by Amstutz (1981), Singh and Choudhary (1988), Khatsu (1994) and Soodan *et al.* (2004).

The significant decrease in the mean albumin concentration in the present study (2.87±0.56) may be due to inadequate hepatic production. A lowered serum albumin level in hypogalactic cows was also reported by Baitule *et al.*, (1994).

Larson (1957) opined that the most common causes of hypoprotenenia in ruminants were starvation and protein malnutrition. A less common cause was chronic hepatitis. The author further suggested that hypoalbuminemia often existed despite normal total plasma protein levels due to a concomitant rise in serum globulin levels.

In the present study, starvation, malnutrition and chronic gastro-intestinal disorders that interfered with digestion and absorption resulted in inadequate provision of amino acid substrate for general protein production. Concurrent sub clinical hepatic dysfunction further exacerbated the clinical condition.

5.2.2.3 Globulin

Mean serum globulin values in healthy control cows was comparable with the values of Radostits *et al.*, (2000). There was a significant reduction ($P<0.05$) in the values of serum globulin concentration in experimental cows compared with healthy cows. As most of the animals studied had been 45-90 days in lactation, the need for production of globulin for excretion in colostrums might have been over. So no further production of excess globulin was required. Reduced globulin levels could also be attributed to inadequate hepatic synthesis.

5.2.2.4 Blood Glucose

Mean blood glucose values recorded in healthy control cows was in the range reported by Kaneko (1980) and Amstutz (1981). Pradhan and Chakaborti (1986) found the blood glucose levels during 31-60 days after parturition was 51.2±1.03 mg per cent.

A significant decrease in mean blood glucose concentrations of the experimental animals compared to the control animals was observed. Similar values were reported by Pandey and Parai (1988) in cases of atypical production disease. Rao and Suryanarayana (1996) reported that the blood glucose levels in cases of subclinical ketosis were 39.4±1.2mg/dl.

Hypoglycaemia in post parturient animals was probably due to drainage of large amount of glucose by mammary gland for conversion to lactose or due to

insufficient glucose preccursor in the diet of these animals. Many cows could exist in negative energy balance and ketonuric state but may not show overt clinical signs. (Radostits *et al.*, 2000).

Glucose is obtained by gluconeogenesis in the liver. Any form of hepatic dysfunction will affect the availability of glucose. Glucose is virtually the sole precursor of lactose and about, 60-80 percent of blood glucose is used by the mammary gland for milk production. The two sources of plasma glucose is absorption from gut and gluconeogenesis. In ruminants, only little glucose is absorbed from the gut, so the remaining overwhelming bulk of it has to be synthesized. Most of this synthesis occurs in the liver. Mismatch between mammary drain of glucose for lactose synthesis and gluconeogenesis in liver, will result in hypoglycaemia (Goff and Horst, 1997).

According to Anantwar *et al.*, (1995) in ruminants the formation of lactose and the utilization of volatile fatty acids for energy purpose is dependant on the available supply of glucose. So energy-protein malnutrition will considerably affect the milk yield resulting in sub-optimal productivity.

According to Kronfeild *et al.* (1960) it was depletion of liver glycogen during the development of ketosis that led to hypoglycaemia. The oxaloacetate which is produced by the liver is preferentially utilized for gluconeogenesis. But when the mammary drain for lactose is higher, the liver may not be able to meet the physiological demands, gluconeogenesis becomes impaired, resulting in hypoglycaemia. The cow becomes further depressed, reducing feed intake and reducing milk production

In the present study the moderate degree of hypoglycaemia was attributed to the lactation stress, depletion of hepatic glycogen, greater demands of energy and protein energy malnutrition.

5.2.2.5 Blood ketone bodies

The mean values of blood ketone bodies in the blood of normal healthy cows were 3.69±1.03mg/dl within the ranges recorded by Rajan and Ganapathy (1973) and Radostits *et al.* (2000).

The values of blood ketone bodies obtained in hypogalactic animals (5.94±0.41mg/dl), were in accordance with those values reported by Singh *et al.* (1989) and Venkateswarulu *et al.* (1994). Baishya and Hazarika (1998) recorded that the blood ketone levels of affected animals in cases of subclinical ketosis ranged from 5.26 to 6.65mg/dl.

During early lactation, the amount of energy that is required for maintenance of body tissues and milk production exceeds the amount of energy the cow can obtain from dietary sources. Hence, the cow must utilize body fat as a source of energy. There is a limit to the amount of fatty acids that can be oxidized completely by the tricarboxylic acid cycle of the liver or exported from the liver as very low density lipoprotein. When this limit is exceeded triglycerides accumulate within the hepatocytes, impairing their function, and the acetyl – coenzyme A that is not incorporated into the tricarboxylic acid cycle is converted to acetoacetate and ß hydroxybutyrate (Goff and Horst, 1997). Thus, when liver synthesised maximum amount of glucose which resulted in disturbance of normal functioning of TCA cycle, ketone bodies appeared as byproducts.

Individuals with subclinical ketosis are those animals that have no clinical signs of ketosis but have low glucose levels blood ketones in the range of 10-30mg/dl and milk ketones level of 2mg/dl (Kauppinen,1983). Grohn et al. (1983) opined that dairy cattle could be ketonaemic without significant hypoglycaemia and other signs of ketosis.

Ketonaemia in the present study may not be enough to cause clinical ketosis. Mobilisation of more amounts of fatty acids to liver beyond its capacity to convert it to glucose due to the negative energy balance resulted in formation of ketone bodies,which adversely affected milk production.

5.2.2.5. Blood urea nitrogen

Mean value of blood urea nitrogen obtained for control cows (17.80±2.59 mg/dl) was comparable to the values reported by Amstutz, (1981). Khatsu (1994) also reported similar values (21.91±2.24 mg/dl).

In the present study significant reduction in the blood urea nitrogen levels (15.48±2.43 mg/dl) were observed. Larson (2002) opined that urea nitrogen tends to be low in lactating animals because of their high fluid intake and urine output. Poor nutrition or high fluid intake in the presence of normal renal function will result in a decreased BUN level because relatively little urea will be reabsorbed by the renal tubules. Liver disease can also cause decrease in urea synthesis because of diminished activity of urea cycle in liver.

It was concluded that since urea production occurs almost exclusively in the liver, hepatic failure is frequently associated with a decrease in BUN. A low protein diet may also be another reason for the decreased blood urea nitrogen levels.

5.2.2.6 Serum calcium

The mean calcium levels in the serum of control animals (10.26±1.69) was comparable to the values observed by Pyne and Maitra (1981), Shukla *et al.* (1983), Pradhan and Chakraborti (1986) Gupta and Rai (1987). Deshpande *et al.* (1998) recorded the serum calcium levels on 28th day post partum was 10.49±0.14 mg/dl.

A significant decrease in serum calcium levels was noticed in hypogalactic cows (8.03±1.25 mg/dl) compared to normal cows. In lactating ruminants, the serum calcium homeostasis is maintained by an increased rate of dietary calcium absorption, decreased rate of urinary calcium excretion and mobilization of bone calcium, under the complex physiological actions of parathormone, calcitonim and vitamin D. (Kulkarni *et al.*, 1984).

Clinical signs of milk fever are not precipated until blood calcium levels fall below 4mg/dl.A state of subclinical hypocalcaemia can exisist were plasma concentrations fall between 5 to 7.5 mg/dl. At a plasma concentration of 5mg/dl, abomasal motility is reduced by 70 percent and at 7.5 mg/dl it is reduced by 50 percent (Goff and Horst, 1997).

According to Meyer *et al.* (1966), hypocalcaemia occurs when serum calcium levels falls below 8.4mg/dl. If the parturient cow has a depressed appetite due to undermined causes, hypocalcacemia should be considered even in the absence of characteristic clinical signs.

Animals with serum calcium levels above 5.0mg/dl did not show clinical signs of paresis. Hypocalcaemia can occur without paresis and in some instances without any detectable signs except anorexia and reduced milk yield. Improvement of calcium levels in such animals resulted in a significant increase in milk yield (Mode *et al.*, 1986).

Serum calcium level is lower in postparturient cows with two or more previous lactations compared to cows after their first lactation. The inability of osteoclastic bone activity to increase rapidly enough to compensate for calcium loss in to milk in older animals further exacerbates the imbalance between the inflow and outflow from the extracellular pool calcium pool (Capen and Rosol, 1989). In the present study most of the animals were in their third parity which concurs with the above findings.

Anorexia, hypocalcemia and reduction in milk yield in post parturient animals has also been reported in association with subclinical ketosis state by Baishya and Hazarika (1998) Anjillappa and Suryanarayana (2002).

Hypocalcaemia in the present study may be attributed to parathyroid insufficiency, decreased osteoclastic activity as well as poor absorption from the gut which in turn is caused by inhibition of the oxalate contained in the paddy straw fed to the animals. Borderline hypocalcaemia in the present study might be a contributing factor for the reduction in milk yield.

5.2.2.7 Serum phosphorus

The mean value of phosphorus in the healthy control cows was in concurrence with the values reported by Payne *et al.* (1970), Gupta and Rai (1987), Prasad *et al.* (1987) and Gupta *et al.* (1995).

In the present study, the phosphorous levels were lower compared to group II although the differences were non-significant. Serum inorganic phosphorus levels remained fairly constant throughout pregnancy and lactation. The levels also showed a marked decline with increasing age (McAdam, 1982).

The values were similar to those reported by Baitule et al., (1994) in hypogalactic animals. Serum phosphorus levels appear to be intimately related to carbohydrate metabolism (Simesen, 1970). In the present study hypoglycaemia with concurrent low levels of serum phosphorus indicated poor carbohydrate reserves or poor nutritional status (Shaw, 1956).

5.2.2.8 Serum magnesium

Mean serum magnesium concentrations of the group II were similar to the finding of Mylrea and Bayfeild (1968), Pandey et al., (1998) and Patel and Patel (2001).

The values of the experimental animals were similar to the group II. Serum magnesium concentrations were affected by seasons, i.e., values was slightly higher in summer. Many factors like plant type, fertilizers, soil, climatic factors influence serum magnesium levels. Serum magnesium levels were found to be independent of the physiological state of the animal (Mode *et al.*, 1986).

5.4 SERUM ENZYMOLOGY

5.4.1 Serum Asparate aminotransferase (AST)

The serum AST values obtained for the control group (61.00±7.24 IU/l) were in accordance with findings of Kumar *et al.* (2001) and Soodan *et al.* (2004).

The values obtained for hypogalactous animals (72.07±5.65 IU/l) showed significant increase over the values of the control animals. In ruminants, the effect of anorexia on the activity of liver enzymes is delayed as compared to that of monogastric animals (Dua and Bhatti, 1999). Similarly, Jagatheesan *et al.* (2005) opined that serum AST activity remained constant without any significant change

during the advanced stage of pregnancy and early lactation period. The author concluded that neither advanced pregnancy nor early lactation affected serum AST secretion from liver.

Energy deficit is common in dairy cows in whom milk production outstrips appetite and body reserves. As a result of this energy deficit occurring during peak lactation body reserves are mobilized, this involves loss of muscle and adipose tissue and results in deposition of fat in muscles, kidneys, adrenals as well as liver (Reid, 1980). This will result in mild damage to the hepatic cells. Partial to complete loss of appetite, reduced milk yield and decreased rumen motility were reported by Pillai *et al.* (1994) Mahanta *et al.* (1988) in association with liver dysfunction.

The significant rise in serum AST values in the present case may be attributed to the stress of lactation on the hepatic cells as well as subclinical hepatic injury.

Summary

6. SUMMARY

The most metabolically stressful period in a dairy cow's life occurred during early lactation when milk production abruptly rises and a huge metabolic drain is imposed on the body. During the early stage of lactation most cows could not consume enough feed to satisfy the metabolic demands of milk production and hence they subsequently lost weight and body condition. Dairy cows at this stage were most susceptible to metabolic imbalances. Although most cows weathered this period, without overt signs of clinical disease and they failed to achieve optimum milk production.

Metabolic profile of 30 cows which showed reduction in milk yield during the peak period of lactation was selected for the present study. These animals constituted the experimental group (Group I).

Similarly, six healthy dairy cows which were around the period of peak production and maintained under similar environmental and managemental conditions as the experimental group were selected as control group (Group II).

Detailed anamnesis and signalment of the animals were done. History of previous illness, if any, percentage reduction in milk yield, feeding and managemental conditions were noted. Metabolic and infectious diseases were ruled out by the relevant clinical tests. Metabolic profile of both group I and II were studied and subjected to statistical analysis.

Haemogram consisted of estimation of haemoglobin and volume of packed red cells (VPRC) of the two groups.

Biochemical parameters such as serum glucose, total protein, albumin, globulin, calcium, magnesium, phosphorus, blood urea nitrogen and serum aspartate aminotransferase (AST) were determined by standard methods.

Study of the anamnesis of experimental animals revealed that they were in their third parity and hence were more prone to metabolic stress and imbalances. Their age ranged from three to five years and most of them were in their third lactation.

Mean concentrations of haemoglobin and VPRC of group I were decreased when compared to group II. Serum glucose, total protein, albumin, globulin and calcium of Group I animals when compared to Group II were significantly reduced, with non significant reduction in phosphorus values. Serum magnesium values showed no variation. Serum AST and blood ketone body levels of Group I animals showed significant increase when compared to Group II animals.

Lactation is a complex physiological process, influence by neuroendocrine as well as metabolic factors. From the above study, it was evident that hypogalactia was precipitated as a result of metabolic imbalances due to large turn over for milk production. The negative energy balance that occurred in postpartum dairy cows resulted in hypoglycemia and ketone body formation. Concurrent hypocalcemia further exacerbated the condition precipitating hypogalactia.

References

REFERENCES

Amlathae, S and Gupta, V.K. 1990. Spectrophotometric determination of acetone using vanillin. *Analyst* 115: 1385-1387

Amstutz, H.E. 1981. Downer Cows. *Mod. Vet. Pract.* 62: 299-300

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

Andrews, T. 1998. Ketosis and fatty liver in cattle: *In Pract.* 8:50-57

Anjilappa and Suryanarayana, C. 2002. Clinico-biochemical and therapeutic studies on subclinical ketosis in graded murrah buffalo. *Indian Vet. Med. J.* 26: 328 – 330

Atwal, A.S. and Erfle, J.D. 1992. Effects of feeding fish meal to cows on digestibility, milk production and milk composition. *J. Dairy Sci.* 75:502-507

Baishya, B.C. and Hazarika, G.C. 1998. Clinico-biochemical findings in sub-clinical ketosis in crossbred Jersey cows at Guwahati. *Indian Vet. Med. J.* 22:293-296

Baitule, J., Mode, S.G., Padnaik, B.S., Sadekan, R.D., Kolte, A.Y., Joshi, M.V. and Bijwal, D.L. 1999. Efficacy of herbal preparation in the treatment of post-parturient hypogalactia in crossbred cows. *Indian J. Vet. Med.* 19:66-67

Agrawal & Singh 2005 (19.5).

Bath, D.L., Crickinson, F.N., Tucker, H.A. and Appleman, R.B. 1978. *Dairy Cattle: Principles, Problems, Profits.* Second edition. Lea and Febiger, Philadelphia, p. 574

Benjamin, M.M. 1985. *Outline of Veterinary Clinical Pathology*. Third edition. Kalyani Publishers, New Delhi, p. 351

Bhatt, M.K., Kumar, M., Chandra, R. 2002. Monitoring of mineral profile in dairy animals of different lactation stages maintained in adjoining areas of Pantnagar. *Indian J. Vet. Med.* 22: 75-77

Bhikane, A.G., Anantwar, L.G., Kawithkar, S.B., and Markandaya, W.N. 1963. Effect of phosphorus supplementation on health and production of buffaloes. *Vet. Practit.* 4: 148-149

Bhongade, H.G., Sarode, D.B., Rode, A.M. and Sapra, V.A. 1993. Effect of antehelminthic therapy on haematobiochemical profiles of dairy cows. *Indian J. Vet. Med.* 13: 75-76

Blanter, K.L., Reineke, E.P., Crampton, E.W. and Peterson, W.E. 1999. The role of thyroid materials and synthetic goitrogens in animal production and an appraisal of their practical use. *J. Anim. Sci.* 3: 307-310

Boddie, G.F. 1969. *Diagnostic Methods in Veterinary Medicine.* Sixth Edition. Oliver and Boyd, Edinburg, p. 447

Braak, A.E. and Kloosta, A.T. 1987. Effects of calcium and magnesium intake and feeding level during the dry period on bone resorption in dairy cows at parturition. *Res. Vet. Sci.* 43:7

Blood et al. ... (B)

Blanter et al 1969, ...

Bricano, J.V., Vanttorn, H.H., Haris, B. J. and Wilcox, C.J. 1988. Comparison of different methods of expressing dietary protein for lactating dairy cows. *J. Dairy Sci.* 71:1647-1658

Brody, S. 1955. Climatic Physiology of Cattle. *Br. Vet. J.* 106:156-158

Bronicki, M., Dembinski, Z. 1994. Studies on the activity of selected hepatic enzymes in dairy cows in conjunction with lipid metabolism markers. *Med. Vet.* 50: 268-271

Burtis, C.A. and Ashwood, E.R. 2001. Carbohydrates. *Tietz fundamentals of Clinical Chemistry* (eds. Burtis, C.A. and Ashwood, E.R.) Fifth edition, WB Saunders Company, Philadelphia, pp. 310-318

Camy, G. 1985. Use of short metabolic profiles by the practitioner in mixed practice. *Vet. Pract. France* 69:136-142

Chikkara, B.S. and Pander, B.L. 1988. Expected and actual genetic gains in lactation yield and calving interval in Hariana cattle. *Indian J. Anim. Sci.* 58: 256-262

Choudhari, P.C. 1990. Health care for higher productivity. *Indian Dairy Man.* 4:186-189

Clark, J.H. and Davis, C.L. 1980. Some aspects of feeding high producing cows. *J. Dairy Sci.* 63: 873-882

Clark, J.M., Spires, H.R. and Derrig, R.G. 1973. Post ruminal administration of glucose and sodium caseinate in lactating cows. *J. Anim. Sci.* 37: 441-449

Dasan, P.K. and Divya, P.I. 2001. Serum calcium level estimation in cows. *J. Indian Vet. Assoc.* 6: 45

Chopra et al., 1973 (1973).

Deshpande, S.M., Mantri, A.M., Talvelkar, B.A. and Deshmukh, B.T. 1998. Studies on macroelements during gestation and early postpartum period in Gir and crossbred cows. *Indian J. Dairy Sci.* 51: 275-279

Dua. K. and Bhatti, M.K. 1999. Etiopathogenesis and therapeutic aspects of anorexia due to simple indigestion in dairy animals: A review. *Indian J. Vet. Med.* 19: 1-13

Dutta, G.N., Saxena, R.K and Buragohain, J. 1995. Economic implication of treatment of lactating cows for subclinical mastitis. *Indian Vet. J.* 72:420-422

Endres, D.B. and Rude, R.K. 2001. Mineral and Bone Metabolism. *Tietz fundamentals of Clinical Chemistry* (eds Burtis, C.A. and Ashwood, E.R.) Fifth edition, W.B Saunders Company, Philadelphia, pp. 860-876

Galhotra, A.P. 1990. Effect of Galog on milk yield in buffaloes and crossbred cows with agalactia or hypogalactia. *Indian Vet. Med. J.* 14:245-248

Gangwar, P.C. 1984. Role of climate in production diseases of dairy cattle and buffaloes. *Indian J. Vet. Med.* 4:71-76

Gangwar, P.C. 1984. Role of climate in production diseases of dairy cattle and buffaloes. *Indian J. Vet. Med.* 4:71-76

Gindler, E. 1971. *Clin. Chem.* 17:662. Cited in working manual of Labkit for Magnesium

Goff, J.P. and Horst, R.L. 1997. Physiological changes at parturition and their relationship to metabolic disorders. *J. Dairy Sci.* 80: 1260-1268

Daly et al. 1972 (PJ 40)

Gopinath, R.V. 2004. Clinical investigations on parasitic anaemia in cattle. M.V.Sc. thesis, Kerala Agricultural University, Thrissur, p. 87

Gosh, C.P., Nagpaul, P.K. and Prasad, S. 2004. Subclinical mastitis in cattle and buffaloes and its impact on somatic cell count and milk composition. *Indian J. Anim. Sci.* 8: 329-333

* Graig, S.R. and Boddie, G.F. 1956. *Veterinary Materia Medica and Therapeutics*, Bailliere Tindall Company, London, p. 70.

Grishi, L. and Todd, A.C. 1978. Prevalence of gastrointestinal parasitism among milking cows in Wisconsin, Pennsylvania and North Carolina. *Am. J. Vet. Res.* 39:51-54

Grohn, Y., Lindberg, L.A., Bruss, M.L and Farver, T.B. 1983. Fatty infiltration of liver in ketotic dairy cows. *J. Dairy Sci.* 66: 2320-2328

Gupta, G.C. and Rai, P., 1987. A note on biochemical profile in pre and postpartum states in cattle and buffaloes. *Indian J. Vet. Med.* 7:45-46

Gupta, G.C., Pachauri, S.P. and Rajora, V.S. 1995. Haematological profile in periparturient cattle and buffaloes. *Indian J. Anim. Sci.* 65:1225-1227

Henderson, A.R. and Moss, D.W. 2001. Enzymes. *Tietz fundamentals of Clinical Chemistry* (eds Burtis, C.A. and Ashwood, E.R.) Fifth edition, W.B. Saunders Company, Philadelphia, p. 1091

Henry, R.L. 1969. Spectrophotometric method of estimation of acetone and acetoacetic acid from blood. *Clinical Laboratory Diagnosis.* Seventh edition. Lea and Febiger, Philadelphia, p.1025

Gould & Smith, 1975, pp 50

Huxley, J. 2004. Optimizing health, productivity and welfare of dairy cattle. On-farm nutrition. *In Pract.* 26:466-475

Ingraham, R.H. and Kappel, L.C. 1988. Metabolic profile testing. *Vet. Clin. North Am. Food Anim. Pract.* 4:391-411

Jagatheesan, P.N.R., Selvaraju, M., Kumar, V.R. and Chandrahasan, C. 2005. Effect of advanced pregnancy and early lactation on blood biochemical constituents in buffaloes. *Indian Vet. J.* 82: 401-403

Johnson, A.M., Rohlfs, E.M. and Silverman, L.M. 2001. Proteins. *Tietz fundamentals of Clinical Chemistry* (eds Burtis, C.A. and Ashwood, E.R.) Fifth edition, W.B. Saunders Company, Philadelphia, pp. 582-596

Joseph, S.A., Karunamoorthy, G., Lalitha, C.M. and Chandran, D.J. 1986. A note on the occurrence of *Haematopinus quadripertuses fahrenholz* in dairy cattle in Tamil Nadu. *Cheiron* 15:4

Kalsi, J.S., Randhawa, S.S. and Swaran. R.S. 2002. Haematobiochemical changes in overgrown hooved dairy cows in Punjab state, *Indian J. Vet. Med.* 22 : 91 – 93

Kamra, D.N. and Pathak, N.N. 2005. Improvement in livestock productivity by use of probiotics. *Indian J. Anim. Sci.* 75:128-134

Khatsu, C.M. and Alikutty, K.M. 1999. Metabolic profile of 'downer cow' syndrome in crossbred dairy cattle. *J. Vet. Ani. Sci.* 25:47-49

Khorasani, G.R., De Boer, G., Robenson, B. and Kennelly, J.J. 1994. Influence of dietary in starch on metabolic responses of dairy cows. *J. Dairy Sci.* 77: 813-824

Khatsu et al. 1993 (1918)

Kamra, 1980 (1922)

Jagatheesan, 1991 (1403)

Klusmeyer, J.H. Cameron, M.R., Mcloy, G.C., Clark, J.H. 1990. Effects of feed processing and frequency of feeding on ruminal fermentation, milk production and milk composition. *J. Dairy Sci.* 73:3538-3543

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

Kulkarni, B.A. and Talvelkar, B.A. 1984. Studies on serum biochemical constituents in lactating and dry Indian buffaloes. *Indian Vet. J.* 61: 564 – 568

Kulkarni, B.A., Talvelkar, B.A., Kaushik, R.V., Gokani, S.S., Patankar, D.D and Kulkarni, B.S. 1984. Biochemical studies in Gir and Jersey lactating cows. *Indian Vet. J.* 61: 377 – 381

Kulkarni, B.A., Talvelkar, B.A., Deshmukh, B.T., Kolhatkar, V.P., Patankar, D.D., Gokani, S.S. and Kulkarni, B.S. 1983. Biochemical studies in Gir and crossbred dairy cows. *Indian Vet. J.* 60:17-22

Kulkarni, B.A., Talvelkar, B.A., Kaushik, R.V., Gokani,S.S., Patankar, D.D and Kulkarni, B.S. 1984. Studies on serum biochemical constituents in lactating and dry Indian buffaloes. *Indian Vet. J.* 61:564-568

Kumar, B., Joshi, H.C. and Kumar, M. 1984. Clinico-haematological changes on microfilariae affected buffaloes (*Bubalus bubalis*). *Indian J. Vet. Med.* 4: 45-47

Kumar, A. and Radhika, S. 2001. Post parturient diseases in dairy cows. *J. Indian Vet. Assoc.* 6: 45

Kumar, K.S., Reddy, M.P. and Choudhari, P.C. 2001. Clinico-haemobiochemical and therapeutic aspects of intestinal amphistomiasis in buffaloes. *Indian J. Vet. Med.* 21: 46-47

Larson, B.L. and Kendall, K.A. 1957. Changes in specific blood serum protein levels associated with parturition in the bovine. *J.Dairy Sci.* 40: 659

Mahanta, P.N., Misra, S.K. and Nauriyal, D.C 1988. Biochemical indicators of liver injury and its therapy in post parturient dyspepsia in buffaloes. *Indian J. Vet. Med.* 8: 36 – 37

Maiti, S.K., Sharada, R. and Singh, N. 1997. *Indian Vet. Med. J.* 21: 251-253

Manna, A.K., Pramanik, S. and Mulcheiya, G.S.1994. Comparative efficacy of oxyclosanide, hexachlorophene and nitroxynil against natural infection of amphistomes in cattle. *Indian J. Anim. Sci.* 33:25-27

Manston, R., Russel, A.M., Sally, B., Dev, M. and Payne, J.M. 1975. Influence of dietary protein upon blood composition in dairy cows. *Vet. Rec.* 97:497

Marston, H.R., Allen, S.H. and Smith, R.M. 1961. Primary metabolic defect supervening on vitamin B_{12} deficiency in sheep. *Nature* 190: 1085-1091

Mayer, G.P., Ramberg, C.F. and Kronfeld, D.S. 1966. Hypocalcaemia without paresis in cows. *J.Am.Vet.Med.Assoc.*149:402-405

McAdam, P.A. and O'Dell, G.D. 1982. Mineral profile of blood plasma of lactating dairy cows. *J. Dairy Sci.* 65:1219-1226

Mode, S.G., Sapre, V.A. and Sarode, D.B. 1986. Levels of serum calcium and magnesium in relation with milk production in dairy animals. *Indian J. Vet. Med.* 6: 12 – 16

Morrow, D.A., Hitman, D., Dade, A.W. and Kitchen, H. 1979. Clinical investigation of a dairy herd with the fat cow syndrome. *J. Anim. Vet. Med. Assoc.* 174:161-167

Manston & Allen 1981, pg 49.

Mount, L.E. 1979. Adaptation to thermal environment in domestic animals. *J. Dairy Sci.* 57: 86-95

Mylrea, P.J. and Bayfeild, R.F. 1968. Concentration of some components in the blood and serum of apparently healthy dairy cattle. *Aust. Vet .J.* 44: 565-569

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

Newman, D.J. and Price, P.C. 2001. Non protein nitrogen metabolites. *Tietz fundamentals of Clinical Chemistry* (eds Burtis, C.A. and Ashwood, E.R.) Fifth edition, W.B. Saunders Company, Philadelphia, p. 610-635

NRC. 1989. *Nutrient Requirements of Dairy Cattle.* Sixth Edition, National Academy of Sciences, Washington, D.C. p. 159

Overston, M.W., Sischo, W.M. and Reynolds, J.P. 2003. Evaluation of effect of estradiol cypionate administered prophylactically to postparturient dairy cows at high risk for metritis. *J. Am. Vet. Med. Assoc.* 223: 846-851

Panda, P.B. and Sadhu, D.P. 1983. Some genetic and non-genetic factors affecting milk production in cross-breds of Holstein and Jersey with Hariana and Deshi Bengal cows. *Indian Vet. J.* 60: 99-106

Pandey, A.M., Thatte, V.R. and Desai, R.T. 1986. Studies on the performance of some of the production traits in Gaolao and its crosses with exotic breed. *Indian Vet. J.* 63: 137-142

Pandey, N.N. and Parai, T.P. 1988a. Clinico-biochemical profile of complex metabolic disease in cows and therapy. *Indian Vet. Med. J.* 12: 177 - 178

Pandey, N.N. and Parai, T.P. 1988b. Clinico-biochemical and therapeutic studies of production diseases in cross bred cows. *Indian J. Anim. Sci.* 58: 36-38

Pandey, N.N. and Parai, T.P. 1989. Physiological changes in the metabolic profile at calving. *Indian J. Vet. Med.* 9: 27

Park, C.S. and Jacobson, N.L. 1988. The mammary gland and lactation. *Duke's Physiology of Domestic Animals.* (eds. Swenson, M.J. and Reece, O.W.) Eleventh edition. Panima Publishing Corporation, New Delhi, pp. 719-728

Patel, J.S. and Patel, P.R. 2001. Clinico-biochemical and therapeutic studies on clinical parturient paresis in buffaloes. *Vet. Practit.* 2: 135-136

Patel, M.G. and Jadhav, K.M. 2003. Metabolic profile of apparently healthy buffaloes from areas of high and low incidence of post parturient hypocalcaemia. *Indian J. Vet. Med.* 23: 27-28

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

Payne, J.M., Rowlands, G.J., Manston, R., Dew, S.M. and Parker, W.H. 1974. A statistical appraisal of the results of the metabolic profile test on 191 herds in the B.V.A./A.D.A.S. *Br. Vet. J.* 130: 34-43

Peterson, R.G. and Waldern, D.E. 1981. Repeatabilities of serum constituents in Holstein Friesians affected by feeding, age, lactation and pregnancy. *J. Dairy Sci.* 64:822-831

Petrie, L. 1987. Differential diagnosis of diarrhea in adult cattle. *In Pract.* 8: 50 – 57

Pillai, U. N., Alikutty, K.M. 1995. Clinico-haematological observation in bovine amphistomiosis. *Inidian J. Vet. Med.* 15: 38 – 39

Pillai 1994 49 . 10 .

Park & Jacobson 1976, (1915)

Pillai, K.G., Choudhuri, P.C and Sundar, N.S. 1999. Clinical, biochemical and therapeutic aspects of post parturient indigestion in cows. *J. Vet. Anim. Sci.* 25: 120 – 122

Pradhan, N.R. and Chakaborti, A. 1986. Blood glucose and calcium profile in crossbred cows with relations to lactation. *Indian J. Vet. Med.* 6:60-61

Prasad, B., Rathor, S.S. and Sidhu, S.S. 1987. Studies on haematobiochemical profile for forecasting downer cow syndrome in cows. *Indian J. Vet. Med.* 7:391-395

Pyne, A.K. and Maitra, D.N. 1981. Physiological studies on blood of lactating Hariana and Sahiwal cattle. *Indian Vet. J.* 58:526-528

Radostits, O.M., Gay, C.C., Blood, D.C. and Hinchcliff, K.W. 2000. *Veterinary Medicine.* Ninth Edition, W.B. Saunders Company, London, p. 1877

Raina, A.K., Ramesh, V and Raina, R. 1993. Serum biochemical indices in non-descript Kashmiri and Jersey cows. *Indian J. Vet. Med.* 13: 28-29

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.* 50:759-763

Ramakrishna, K.V., Choudhary, P.C., Satyanarayana, G. and Sivaramakrishnayya, K. 1992. Preliminary studies on anaemia in buffaloes. *Indian Vet. J.* 69:535-538

Rao, D.S. and Suryanarayana, C. 1996. Studies on sub clinical ketosis and hypocalcaemia in lactating cows and buffaloes. *Indian Vet. Med. J.* 20: 275-278

Ratnakumar, J.N., Sreekumaran, T., Vijaykumar, V and Rajan, A. 1990. Prevalence of hypothyroidism in sub fertile or infertile cows. *J. Vet. Anim. Sci.* 21:91-94

Reid, I. and Roberts, J. 1982. Fatty liver in dairy cows. *In Pract.* 4: 164-169

Reid, I.M., 1980. Incidence and severity of fatty liver in cows. *Vet. Rec.* 107: 281-284

Roberts, S.J. 1986. *Veterinary obstetrics and Genital Diseases (Theriogenology)* Third Edition. Edward Brothers. Michigan p. 981

Rosol, T.J. and Capen, C.C. 1997. Calcium regulating hormones and diseases of abnormal mineral (calcium, phosphorous, and magnesium) metabolism. (eds. Kaneko, J.J., Harvey, J.W. and Bruss, M.L.) *Clinical Biochemistry of Domestic Animals*, Academic Press, San Diego, pp. 646-651

Rowlands, G.J. and Manston, R. 1976. The potential uses of metabolic profiles in the management and selection of cattle for milk and beef production. *Livest. Prod. Sci.* 3: 239-256

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

Samanta, A.K., Sarkar, S., Basu, S., Biswas, U. and Biswas, S. 1995. Effect of grazing in production of nutritional anemia in cattle. *Indian Vet. Med. J.* 15: 61-63

Sampath, K.T. Role of bypass fat in dairy animals. 2001. *J. Indian Vet. Assoc.* 6: 45

Sarkar, T.K., Sharma, M.C., Dimri, U. and Joshi, C. 2004. Status of calcium, phosphorous and magnesium in soil, fodder and serum of cattle in a part of U.P. and Uttaranchal, *Indian Vet. J.* 81: 670-673

Schalm, D.W., Jain, N.C. and Carroll, E.J. 1975. *Veterinary Haematology.* Third edition. Lea & Febiger, Philadelphia, p.807

Schmidt, G.H., Warner, R.G., Tyrell, H.F. and Hansal, W. 1971. Effect of thyroprotein feeding on dairy cows. *J. Dairy. Sci.* 54: 481-484

Selukar, P.S. Dakshinkar, W. P., Sarode, D.B., Kothekar, M.D. and Bhojne, G.R. 2001. Association of haemoglobin percentage with milk yield in cows. *Indian Vet. Med. J.* 25: 287-288

Sharma, M.G., Pathak, N.N. and Hung, N.N. 1984. Influence of parasitism on growth and milk production of Murrah buffaloes, *Indian J. Vet. Med.* 4: 97-100

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

Shrikande, G.D. and Sarode, D.B. 1999. Haematobiochemical levels in cows of different age groups. *Indian Vet. J.* 76: 26-28

Shukla, S.P., Kharche, K.G., and Pardeh, H.K.B. 1983. Calcium and phosphorus in relation to retained placenta in cross-bred cows. *Indian Vet. J.* 60:183-188

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

Singh, A., Taylor, C.M. and Singh, B.N. 1986. Factors influencing some reproduction and production traits in Malvi cattle. *Indian Vet. J.* 63:388-392

Singh, A.P., Gahlot, A.K., Sharma, T. and Tanwar, R.K. 2003. Physiological status of serum glutamate oxaloacetate (SGOT), serum glutamate pyruvate transaminase (SGPT) and serum alkaline phosphatase (SAP) enzymes in indigenous and crossbred cattle of arid region. *Vet. Practit.* 4: 80-82

Schalm et al, 1971 Pg 38

Singh, B. and Joshi, S.J. 1991. Epidemiology, clinico pathology and treatment of clinical *Trypanosoma evansi* infection in buffalo. *Indian Vet. J.* 68: 975-979

Singh, N., Kumari Ramesh and Akbhar, M.A. 1989. Concentration of biogenic amines in the rumen liquor and blood of healthy buffaloes suffering from indigestion. *Indian Vet. Med. J.* 13:84-88

Singh, N., Puri, J.P., Nangia, O.P. and Garg, S.L. 1983. Early development of rumen function in buffalo calves: Rumen microbes, metabolites and cellulose digestion invitro as a function of age and diet. *Indian J. Anim. Sci.* 53: 933-937

Singh, R. Choudhary, C.C., Kamra, D.N. and Pathak, N.N. 1998. Effect of dietary supplementation with yeast as suspension on growth in crossbred calves. *Aust. J. Anim. Sci.* 11:267-278

Sivaiah, R., Rao, K.B. and Murthy, A.S. 1986. Serum calcium and inorganic phosphorus levels in Ongole crossbred cows. *Indian Vet. J.* 63: 804-806

Snedecor, G.W. and Cochran, W.G. 1985. *Statistical Methods.* Eighth edition. Oxford and IBH publishing company, Calcutta, p. 534

Soodan, J.S., Sudhan, N.A. and Singh, R. 2004. Epidemiological studies on metabolic profile status of lactating dairy cattle and buffaloes. *Indian J. Vet. Med.* 24: 90-93

Srivastava, P.K. 1990. Effect of fenbendazole treatment on milk yield in buffaloes. *Indian J. Vet. Med.* 10: 54-56

Tolkamp, B.J., Dewhurst, R.J., Friggens, N.C., Kyriazakis, I., Veerkamp, R.F. and Oldham, J.D. 1998. Diet choice by dairy cows. Selection of feed protein content during first half of lactation. *J. Dairy Sci.* 81: 2657-2669

172545

Turner, C.W., Yamamoto, H. and Ruppert, H.L. 1957. Endocrine factors influencing the intensity of milk secretion. A. Estrogen, thyroxine and growth hormone. *J. Dairy Sci.* 40: 37-48

Venkateswarulu, K., Rao, D.S.T., Reddy, K.S., Rao, R. and Gaffer, A.A. 1994. Clinico-biochemical findings in subclinical ketosis in crossbred Jersey cows at Guwahati. *Indian J. Vet. Med.* 14: 6-8

Verma, D.N., Hussain, K.Q. and Bharat, R. 1991. Effect on water splashing on feed consumption and nutrient utilization in buffaloes. *Indian. J. Anim. Nutri.* 7:112-114

Wadhwa, D.R. and Prasad, B. 2002. Haematobiochemical studies on downer cow syndrome. *Indian J. Vet. Med.* 22: 11-14

Whitaker, D.A. 2000. Use and interpretation of metabolic profiles. (ed. Andrews, A.H.) *In the Health of Dairy Cattle.* Oxford Blackwell Science, pp. 88-107.

Wilson, J. 1975. Hypopthyroidism in ruminants with special reference to foetal goiter, *Vet. Rec.* 97:161-64

Yadav, B.P., Tripathi, G.S., Navarati, S. and Joshi, S. 2001. Studies on lactation yield in Gaolao cattle. *Indian Vet. Med. J.* 25:55-56

* Originals not consulted

Turner et al., 1946. (by IC)

METABOLIC PROFILE OF HYPOGALACTIC COWS

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ABSTRACT

A study of the metabolic profile of hypogalactic dairy cows under field conditions was undertaken.

Thirty cases of hypogalactic cows were selected and utilised for the study. Similarly, six healthy dairy cows maintained under identical environmental and managemental conditions constituted the control group. Samples of blood, urine and milk were collected and analysed for various parameters according to standard methods. Data collected from the hypogalactic dairy cows indicated a high incidence in animals of third parity which were around four to five years of age. The clinical data were within normal limits. Haemoglobin was decreased indicating a mild anaemia. Biochemically, serum glucose, total protein, albumin, globulin and calcium showed a significant decrease. Blood ketone body levels and serum aspartate aminotransferase (AST) levels showed significant increase. Serum magnesium and phosphorus levels did not vary significantly.

The metabolic profile of hypogalactic animals revealed that hypoglycaemia, hypocalcaemia, hypoproteinemia and subclinical ketosis may be the possible causes of hypogalactia.