CLINICO-THERAPEUTIC STUDIES ON DOWNER COWS

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DECLARATION

I hereby declare that this thesis, entitled **"CLINICO-THERAPEUTIC STUDIES ON DOWNER 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 **"CLINICO-THERAPEUTIC STUDIES ON DOWNER COWS"** is a record of research work done independently by **Prasanna .K.**, under my guidance and supervision and that it has not previously formed the basis for the award of any degree, diploma, fellowship or associateship to her.

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Introduction

1. INTRODUCTION

Kerala State is well known for high producing cattle after the introduction of cross breeding programme. As the genetic worth has improved, the production has increased a lot. However the input or feed resources are scarce. So at the onset of lactation even if the feed is insufficient, the hormonal surge is too much so that the production is not adjusted with the input. This disparity between the input and output is causing a serious depletion in the blood metabolites like calcium magnesium and glucose (throughput).

Moreover the use of unconventional feeds and lack of scientific knowledge in maintaining the high producing cow during the transitional period i.e., three weeks before and after calving can end up in metabolic disorders during the lactation and seriously reduce the production and productive life of animal. The important metabolic disorder which occur during periparturient period are hypocalcaemia, ketosis, hypomagnesemia and fatty liver syndrome. These diseases when not identified and treated in time can produce various other complications. The milk fever if not treated properly can result in serious professional challenge to veterinarians all over the world. So this is the reason why various metabolic profile test were employed for the timely detection and intervention to curtail the occurrence of these diseases.

As per Radostitis *et al.* (2000), downer cow syndrome is defined as a condition occurring in cows usually following post parturient hypocalcaemia characterised by prolonged recumbency even after two calcium treatments.

However various factors other than metabolic reasons can cause recumbency, around parturition, like toxaemia associated with coliform mastitis, metritis, peritonitis, injuries during or following calving and defective managemental practices like insufficient feeding, slippery floors etc. Malnutrition, parasitism and subsequent anaemia are some of the major factors which can cause recumbency in dairy cows heifers and calves. In true sense the term "downer cow" implicates those cows down with metabolic disparities.

The major metabolic disorders associated with downer cows syndrome are milk fever, ketosis and fatty liver syndrome. Undue delay in the treatment of these conditions especially the delayed or insufficient treatment of parturient hypocalcaemia can result in prolonged recumbency and downer cow syndrome. Prolonged recumbency can cause lack of blood supply to various tissues especially to the muscles causing ischaemic necrosis. Ischaemia can also cause permanent damage to muscles and nerves which will result in downer cow syndrome.

It is believed that the reason for this recumbency and non responsiveness to repeated calcium therapy are due to the deficiency of other elements like magnesium, phosphorus, potassium and glucose along with hypocalcaemia. Moreover correction of calcium homeostasis is considered as one of the important area in the field of management of downer cows. For this purpose vitamin D_3 which was used earlier for prevention of milk fever is considered now for the treatment of this condition also.

Since multivarious etiological factors are attributed to downer cow syndrome, it is important to diagnose this condition keeping in mind the differential diagnosis. In farmers point of view it is at utmost important that veterinarian should forecast prognosis of the condition at the earliest.

Since this condition is economically important for the farmers a study is proposed with following objectives

1. To identify probable etiological factors responsible for recumbency in downer cows
2. To evolve cow side tests for the easy diagnosis of downer cows,
3. To assess the efficacy of the different medical and management practices in downer cows and
4. To streamline the prognostic yardstick for the downers.

Review of Literature

2. REVIEW OF LITERATURE

Downer cow has no universal definition. Cows late in lactation, during periparturient period or even dry cows can go down as creepers (Johnson, 1963).

Fenwick (1969e) described downer cow syndrome in broader sense, as all cows recumbent from unknown causes. Treatment is correction of metabolic deficits and symptomatic therapy. Good nursing and management is at most necessary to have a satisfactory out come. Knowledge of the factors, which predispose to the condition, can help to prevent the occurrence of many cases (Allen and Davies, 1981). Whatever be the cause of downer cow, pressure damage is a common denominator in this syndrome (Cox *et al.*, 1982). In severe cases symptoms like tetany of limbs were noticed and cow would not eat or drink (Gangwar, 1984). Fenwick (1986) opined that virtually all downers occur secondarily to milk fever.

Metabolic disorders, toxaemia, injuries during and following parturition and managemental defects can all result in downer cow syndrome (Andrews, 1986). Also downer cases can occur at any time and were usually associated with parturition. A typical downer would be bright and alert, with normal feed and water in take, urination and defecation will be normal some times would be in lateral recumbency.

Radostitis *et al.* (2000) suggested that any cow recumbent for 24 hours even after two calcium therapies can be called as a downer. Etiology is often obscure. Hypocalcaemia, hypophosphataemia, primary musculoskeletal injuries, pelvic swelling from dystocia, nerve damage associated with recumbency and spinal cord compression may all result in this syndrome (George *et al.*, 2002). Also periparturient recumbency can occur. Diagnosis is based on clinical findings and biochemical analysis of blood and prognosis is based mainly on muscle enzymes (Wadhwa and Prasad, 2002b).

2.1 ETIOLOGY

Fenwick (1969e) opined that virtually all downers occur secondarily to milk fever.

Clinically the non-alert downer cow condition appeared to be one of parturient paresis that had failed to respond to treatment (Fenwick, 1977).

Narayana *et al.* (1977) stated that mechanical fracture; infection and metabolic disorders contributed to the aetiology of downer cows.

Eleven percentage of downer sheep were found to be hypocalcaemic by Shorthose and Shaw (1977).

Allen and Davies (1981) suggested that a syndrome with wide definition would necessarily have many causes like milk fever, fat cow syndrome, hypophosphataemia, vitamin E and selenium deficiencies and trauma.

Amstuz (1981) reported that muscle damage that occurs when the cow is recumbent for an extended period led to persistent recumbency in downer cows.

Erb and Grohn (1988) reported that there are no data regarding age and milk fever in older cows and dystocia in heifer are important risk factors

According to Cox *et al.* (1982), pressure damage was a common denominator involved in the pathogenesis of the downer cow syndrome.

Gangwar (1984) reported that in downers there would be hypophosphataemia, hypomagnesemia, hypokalemia, hyper and or hypo adrenocortical activities and sometimes the syndrome appeared concurrently with milk fever.

Andrews (1986) suggested that even though there were many different causes, muscle damage was more responsible in permanent recumbency even when primary causes like milk fever have been successfully treated. The author

also opined that metabolic disorders, toxaemia, injuries during and following parturition and managemental defects could all result in downer cow syndrome.

The plasma calcium, potassium and magnesium concentrations in non-alert downers were lower than those in milk fever Fenwick *et al.* (1986)

Barzanji and Daniel (1988) opined that deficit in oxygen uptake by peripheral tissues during hypocalcaemia could predispose to the downer cow syndrome.

Prasad *et al.* (1988) suspected that downer cow syndrome had been the outcome of hypocalcaemia and or hypomagnesemia.

Barlet and Davicco (1992) opined that hypophosphataemia played a major role in etiology of downer cow syndrome.

Cox and Marion (1992) concluded that prolonged recumbency might cause trauma to peroneal nerve, which resulted in downers.

Correa *et al.* (1993) opined that cows with retained placenta and dystocia were more likely to develop downer cow syndrome than cows with out either disorders. Authors also observed that clinical hypocalcaemia increased the odds for syndrome six fold and stillbirth by five fold.

Sielman *et al.* (1997) observed hypokalemia in ten dairy cows with weakness and recumbency.

Significant reduction in calcium and phosphorus levels was recorded by Patel and Patel (2001) in laterally recumbent buffaloes.

Probable metabolic disorders causing downer cow syndrome were hypocalcaemia, hypophosphataemia, hypomagnesemia and hypokalemia and the etiology could be due to overlong delay in treatment of cows with milk fever (Radostitis *et al.*, 2000).

Damage of one or more peripheral nerves during parturition or milk fever resulted in the so-called downer cow syndrome. Hypocalcemia, hypophosphataemia, primary musculoskeletal injuries, pelvic swelling from dystocia, nerve damage associated with recumbency and spinal cord compression might result in alert downer. (George *et al.*, 2002).

Septic metritis in twenty percentage of downer cows and hip dislocation in a four year old Jersey cross bred cow was noticed as cause of recumbency (Kavitha *et al.*, 2002).

According to Patel and Jadav (2003) post parturient hypocalcaemia was an important condition causing downer cow syndrome.

Veena *et al.* (2003) suggested that downer cows were likely to be deficient in calcium, phosphorus and magnesium.

2.1.1 Managemental Factors

Long duration in treatment of milk fever, exposure to slightly lower than ambient temperature and higher than average wind velocity can lead to hypothermia and prolonged recumbency (Fenwick, 1969d).

Cows fed 15% crude protein while on corn silage concentrate ration for the entire dry period had a disastrous downer cow rate of eight out of twenty six cows in a feeding trial conducted at Ohio (Julien *et al.*, 1977).

Malnutrion, parasitism and exposure to cold could also result in downer cows (Amstuz, 1981).

Murphy and Power (1995) reported that cows that grazed Italian rye grass which contain average nitrate concentration of 5.5% became recumbent.

2.2 EPIDEMIOLOGY

2.2.1 Incidence and Season

There seems to be a predominance of creeper cow cases in the winter and early spring (Johnson, 1963).

In Canada the incidence of 85 % cases of downer cows were reported from November to April (Curtis *et al.*, 1970).

During summer due to climatic stress of high temperature, there would be deficiency of soluble carbohydrates, minerals and vitamins that increase the incidence and intensity of downer cow syndrome (Gangwar, 1984).

In a mail survey of 723 Minnesota dairy herds of 34,650 cows/year an incidence rate of 21.4/1000 was reported by Cox *et al.* (1986).

Less than two percentage (out of five hundred and eighty four cases) of milk fever developed downer cow syndrome (Fenwick *et al.*, 1986).

The cumulative postpartum incidence rate for downer cow syndrome was one per cent (Correa *et al.*, 1993). The author also reported that no evidence of confounding or effect modification was found for season of calving or parity for the syndrome to occur.

Incidence rate was more during summer (Catherine, 1994).

Radostitis *et al.* (2000) reported higher incidence during coldest month of December, January and February and though the incidence among high producers is high accurate figures were not available due to variation in nomenclature used.

The occurrence of downer cow syndrome was evenly distributed throughout the year (Kavitha *et al.*, 2002).

2.2.2 Breed

Narayana *et al.* (1977) observed that the condition was higher in Jerseycross followed by Holstein Friesian and Red Dane

The mail survey by Cox *et al.* (1986) identified a possible lower risk in Brown Swiss than in Holstein cows, but the rates in Jersey and Guersey were intermediate

Tripathy (1987) reported that Jersey and Holstein Friesian cross showed symptoms of paresis at different times of the year.

Pandey and Parai (1988) opined that half Friesian and half Hariana crosses were most susceptible than half Brown Swiss and half Jersey cross.

Barlet and Davicco (1992) reported seventeen Holstein downer cows.

In thirty four Holstein herds studied for risk factors for downer cow syndrome thirty five developed the syndrome (Correa *et al.*, 1993).

Kavitha *et al.* (2002) reported that out of 20 cases 70% were cattle and Jersey cross, HF cross and non descript cows were uniformly affected.

2.2.3 Age

Johnson (1963) suggested that most cases of downer cows occur in older cows just after calving.

Narayana *et al.* (1977) observed that cows between 4 to 8 years of age were mostly affected.

Gangwar (1984) suggested that high milk yielders in their third or subsequent lactations suffered from this syndrome.

All the cows with paresis reported by Tripathy (1987) were of seven to ten years of age.

Erb and Grohn (1988) opined that there are no data regarding age but heifers because of dystocia and older cows because of milk fever are more prone to this disease condition.

Prasannakumar *et al.* (1992) reported a case of downer cow syndrome in a cross bred cow of eight years.

Horst *et al.* (1997a) opined that first lactation cows almost never develop milk fever.

Patel and Patel (2001) reported majority of the parturient paretic buffaloes were in their third to fourth lactation with history of normal calving.

Kavitha *et al.* (2002) observed forty percentage downer cow cases during first two lactation, forty five percentage between third and fourth lactation and fifteen percentage beyond fifth lactation.

Wadhwa and Prasad (2002b) reported that downer cows were 4-6 yrs of age.

2.2.4 Stage of Lactation

Johnson (1963) suggested that sometimes cows late in lactation or even dry cows could go down as creepers.

Allen and Davies (1981) reported the occurrence of downer cow syndrome immediately after parturition.

Gangwar (1984) recorded downer cow syndrome in high yielding cows within first two or three days after calving with symptoms of paresis.

Most cases of downer cows follow milk fever or occur within few days of lactation (Andrews, 1986 and Fenwick, 1986).

Tripathy (1987) opined that all the cows attended with paresis were within five to seven months of lactation.

Twenty three cows studied for the risk factors for downer cow syndrome by Correa *et al.* (1993) were within thirty days postpartum.

Patel and Patel (2001) reported eighty eight percentage of clinical parturient paresis in buffaloes was within seventy two hours after parturition.

About ninety five percentage of downer cow syndrome occurred in post parturient cows immediately after calving (Kavitha *et al.*, 2002).

According to Wadhwa and Prasad (2002b), 51.5 percentage suffered downer cow syndrome within one month of parturition.

2.3 PATHOGENESIS

Bowen *et al.* (1970) opined that primary cause of hypocalcaemic paresis was depression of neuromuscular transmission.

Prolonged hypoxia of skeletal musculature could lead to impaired muscle function and weakness acted as contributing factor to the lack of complete response to the administration of calcium borogluconate solution in cases of hypocalcaemic parturient paresis in cows (Daniel, 1979).

Cox *et al.* (1982) suggested that pressure damage depend on elasticity of the fascia surrounding the osteofacial compartment and it was observed at necropsy that semitendinous muscle with thicker fascial boundaries was mostly affected. Tissue anoxia leads to cell damage and inflammation which causes an increase in pressure within the tissue which limits further tissue perfusion leading to detrimental cascade of destructive cycle.

Andrews (1986) opined that compression of soft tissues leads to nerve and muscle damage, obstruction of blood supply to distal part of the hind limbs leading to venous congestion, stasis and thrombosis leading to ischaemic necrosis

which eventually leads to permanent recumbency. When the animal struggle further, causes muscle tearing and haemorrhage.

Pathogenesis as explained by Hulland (1993) was that when muscles of limbs were in inflexed or tucked up position pressure within muscle rises to levels higher than both venous and arterial pressure. Intra muscular pressure created within the skin and fascial sheaths of the limbs soon serves to collapse veins, causing congestion and then collapse arteries and thus created ischaemia

2.4 CLINICAL SIGNS

Johnson (1963) suggested that ordinarily the creeper will rest on one hip and leg or flat on its belly and udder, that led to palpable atony of the major muscle mass of hind quarters and flank muscles, suppression of appetite, rumination, micturition and defaecation. Rectal temperature of creeper cows was found to be 100° to 101^0F and constricted pupils were also frequently seen in down cows.

Mayer *et al.* (1966) observed that degree of hypocalcaemia appeared to be more crucial to the development of paresis than duration of hypocalcaemia.

All cows that showed knuckling or complete over flexion of one hind fetlock had a common history of prolonged sternal recumbency and is of the opinion that bilateral knuckling of rear fetlock suggest intra spinal lesions or generalised muscular weakness and if the hock joint could be flexed extremely while the stifle, held in extension, then gastrocnemius muscles was ruptured. Fenwick (1969c).

Fenwick (1969e) reported that fifty six percentage of the cows that showed evidence of severe pain when the spinal column was percussed or pressed remained downers compared with twenty three percentage of cows showing no evidence of pain.

Downer cows often exhibit a frog sitting posture, some with the legs under them but were unable to extend the hocks or fetlock and crawled about (Curtis *et al.*, 1970).

No correlation was found by Mullen (1975) between the severity of clinical signs and the inorganic phosphate concentration.

Marked reduction in cardiac out put, stroke volume and arterial blood pressure was recorded by Daniel and Moodie (1978) in a dairy cattle with induced hypocalcaemia

The downer cows would be usually bright ,.often ruminates and may lie on her side with all four legs outstretched or lie on her brisket with hind legs stretched out behind or flexed under body, fetlock joint often knuckles over and if the cow is recumbent owing to injuries received during calving, from the vulva there would be copious vaginal discharge (Allen and Davies, 1981).

Cox *et al.* (1982) reported that downer cows exhibited rigid and swollen right pelvic limb and dog sitting posture and flexion of fetlock joints were the other signs.

Gangwar (1984) reported that affected cows show no signs of illness until they go down either at calving or fourty eight hours after calving. The cows eat and drink normally and look bright but unable to get up. In some cases, the cows may lie in lateral recumbency with head drawn back. When lifted these cows look normal. In severe cases, there will be hyperaesthesia and tetany of limbs and animal do not usually eat and drink.

Rectal temperature was lower in sheep and goat affected with hypocalcaemia (Vihan and Rai, 1984).

According to Andrews (1986) a typical downer cow would be bright and alert. Feed and water intake, urination and defecation would be normal.

Respiration and rectal temperature will be normal even though pulse rate may be raised.

All six non alert cows attended by Fenwick *et al.* (1986) showed preference for lateral recumbency, mucoid faeces were voided which some times contained blood and some cows produced expiratory moaning.

Symptoms recorded by Singh and Gupta (1987) in parturient paretic buffalo included resting of chin on ground, dullness, depression, suspended rumination and anorexia, relaxation of anus and loss of anal reflex. There was respiratory grunt.

Tripathy (1987) reported that non parturient hypocalcaemic paretic cows were either on sternal or lateral recumbency with dry muzzle, slow respiration, dull heart sound, low rumen movements and pulse varied from 70 to 90 per minute.

Clinical symptoms in induced hypocalcaemia in calves as reported by Pandey and Parai (1988) were extreme weakness of hindquarters, inability to stand, recumbency, typical muscular twitching, restlessness, laboured breathing, increased heart rates and reduced ruminal movements.

Pandey and Dwivedi (1988) reported that in clinically induced hypocalcaemic calves, heart rate was significantly increased and ruminal movements declined from two to zero per two minutes on development of clinical hypocalcaemia.

Serum calcium, blood glucose, erythrocyte potassium and sodium concentrations, calcium:magnesium ratio, rectal temperatures and heart rates all had no significant association with disparities in pupil sizes (Fenwick, 1986).

The clinical signs of downer cows in lateral recumbency were bilateral nystagmus, hypothermia and insensitivity of the hindquarters and resistance to flexion of the hind limbs (Barlet and Davicco, 1992).

Cox and Marrion (1992) reported that the affected downer cow remained in sternal recumbency with left hind limb tucked under body. When they could stand up the fetlock of the right hind limb was seen eventually flexed.

All the cows were exhibiting hindquarter weakness and were struggling to rise. In one animal a characteristic flexed right fetlock was observed and in one cow the right hind leg was extended laterally from body (Catherine, 1994).

Clinical signs of milk fever (down cows) often were not seen until the serum calcium falls below 4 mg/100 ml (Goff and Horst, 1998).

Clinically the animals were normal except for recumbency and most downer cows eat and drink normally, their vital signs were within normal range and their alimentary tract function was normal. However, bizarre movements while lateral recumbency and dorsally extended head and neck moaning frequently, assuming a frog legged posture with their pelvic limbs and 'crawling' or creeping may also be exhibited (Radostitis *et al.*, 2000).

Clinically the thirty one downer cows attended by Wadhwa and Prasad (2002b) appeared bright and alert and had normal defecation and urination, but appetite was slightly reduced. The body temperature was within normal range. Elevated cardiac rate, polypnoea were recorded in seven cows. Twenty eight downer cows were in sternal recumbency while three were in lateral recumbency, non alert, anorectic with markedly accelerated cardiac and respiratory rates.

Clinical signs like tremors, excitement, subnormal temperature and dry muzzle were exhibited by parturient paretic buffaloes that were either sternally or laterally recumbent (Patel and Patel, 2001).

All cows attended by Veena *et al.* (2003) were alert downers exhibiting normal physiological functions. Out of the alert downers some cows did not attempt to rise but others made attempt but were unable to completely extend their hind legs.

2.5 HAEMATOLOGICALS OBSERVATION

2.5.1 Haemoglobin

Payne *et al.* (1974), Catherine (1994) and Kramer (2000) reported a mean value of 11.2-g/ dL ,11.17 g/ dL and 11mg%of haemoglobin in healthy cattle.

2.5.2 Packed Cell Volume

Kramer (2000) reported a mean value of 35 per cent in normal healthy cows.

2.5.3 Total Leukocyte Count

Catherine (1994) reported 4.83 × 10^3/µL as the value of total leucocyte count in normal cows.

Mean total leukocyte value reported by Kramer (2000) was 8x10^3/µl for normal cattle.

2.5.4 Differential Leukocyte Count

Greatorex (1957) reported that the normal values of lymphocytes, neutrophils, monocytes, eosinophils and basophils in adult dairy cattle ranged from 36-72, 15-45, 0-8, 2-30, 0 per cent respectively.

Pyne and Maitra (1981) reported N 27 ± 0.73%, L 64.15 ± 0.14%, M 3.05 ± 0.12%, E 4.74 ± 0.06%, B 0.45 ± 0.11% for Hariana breed and N 26.25 ± 0.92%, L 63.35 ± 0.11, M 3.45 ± 0.18%, E 6.33 ± 0.03%, B 0.62 ± 0.06% in Sahiwal breeds of healthy cattle

Catherine (1994) reported values for differential count as L 62.57 ± 2.43%, N 30 ± 2.16%, E 6.71 ± 0.85%, B 0.21 ± 11%, M 0.5 ± 0.17% for normal cows.

Kramer (2000) recorded values as mean values as L 58%, N 28%, M 4%, E 9%, and B 0.5%.

Wadhwa and Prasad (2002a) reported the values as N 32.75 ± 0.64%, L 63.83 ± 2.66%, M 1.25 ± 0.19%, E 2.05 ± 0.26%, B 0.32 ± 0.1%

2.6 HAEMATOLOGY IN DISEASED ANIMALS

Narayana *et al.* (1977) observed that the values of haemoglobin and total lekocyte in four cases of downer cows in crossbred cows were 8.68 ± 0.24 g/100ml and 11,012–1,051/cmm, respectively. The percentage of neutrophils, lymphocytes, monocytes, eosinophils and bosophils were 44.0 ± 3.10, 48.0 ± 5.48, 4.5 ± 5.48, 4.5 ± 2.29, 3.5 ± 1.66 and 0.00, respectively.

The shift in neutrophils and lymphocytes in a downer was associated with recumbency and ulcers (Amstuz, 1981).

In the study of Vihan and Rai (1984) on hypocalcaemic sheep and goat showed higher leucocyte count and low neutrophils with a lower packed cell volume and increased eosinophils.

Prasad *et al.* (1987) recorded a haemogram in an individual cow as haemoglobin 6.6 g%, packed cell volume 22%, TLC 7.5 × 10^3 µL and lymphocytes 77%, which was later, diagnosed as downer with poor prognosis.

Barzanji and Daniel (1988) reported that hypocalcaemia induced in dairy cows and sheep shown significant increase in blood haemoglobin concentration.

Haemoconcentration and increased haemoglobin was reported by Pandey and Dwivedi (1988) in hypocalcaemic calves.

Pandey and Parai (1988) reported a significantly higher packed cell volume in downer cows.

Catherine (1994) recorded a mean value of PCV 34.93, haemoglobin 12.2 mg/%, WBC 44,200 cells/μL, 56.64% of lymphocytes, 40.21% neutrophils, eosinophils of 2.43% basophils of 0.29% and monocytes of 0.51% .

White blood cells counts were within reference range in eight cows which were recumbent for less than twenty four hours but one cow had neutrophilia with WBC count 18,600 cells/μl (Sielman *et al.*, 1997).

Neutrophilia, lymhopenia and eosinopenia were observed in downer cows (Lopez *et al.*, 2001).

Leucocyte pattern of downer cows showed significant increase in neutrophil 41.42 ± 2.16% and decreased lymphocytes in recumbent cow and a significant decrease in haemoglobin (Wadhwa and Prasad, 2002a).

2.7 BIOCHEMISTRY OF NORMAL CATTLE

2.7.1 Serum Calcium

Sivaiah *et al.* (1986) and Pandey and Parai (1988) reported a range of 7.04 to 8.15 mg% of serum calcium in Ongole crossbred cows and 8.87 ± 0.08 mg/dL in normal cows respectively.

Rosol and Capen (1997) reported that mean value of calcium in normal animals were within the range of 8-11 mg%.

2.7.2 Serum Phosphorus

Mc Adam and O'Dell (1982) Pandey and Parai (1988) and Dasan and Divya (2001)recorded a mean value of 4.6 mg%, 4.45 ± 0.11mg% and 4.98 ± 0.307 mg% respectively in randomly selected healthy cows.

2.7.3 Serum Magnesium

Mylrea and Bayfield (1968),Kelly (1974) Payne *et al.* (1974) and (Sahay *et al*, 2002) reported mean value of magnesium as 2.4 ± 0.36 mg%, 2-3 mg% , 2.4 mg% and 2.26 ± 0.092 mg% respectively in normal cows.

Pyne and Maitra (1981) found a value of 2.66 ± 0.06 in Hariana breeds and 2.70 ± 0.04 mg% in Sahiwal breeds

2.7.4 Serum Sodium

Payne *et al.* (1974) and Payne (1977) reported normal concentration of sodium of 139 mEq/L and138.3 mEq/L in normal cows.

2.7.5 Serum Potassium

Mylrea and Bayfield (1968), Payne *et al.* (1974) and Benjamin (1985) and Catherine (1994) reported a mean value of 4.7 ± 0.48 mEq/L, 4.95 mEq/L 3.9 to 5.8 and 4.72 ± 0.20 mEq/L respectively in healthy cows .

2.7.6 Glucose

Payne *et al.* (1974), Pyne and Maitra (1981) and Pandey and Parai (1988) reported mean value of blood glucose as 45.6 mg%, 47.84 ± 1.60 and 47.81 ± 0.99 m/dL respectively in normal cows.

2.8 BIOCHEMISTRY OF DISEASED ANIMALS

2.8.1 Serum Calcium

Curtis *et al.* (1970) opined that there was consistent hypocalcaemia during initial stage of recumbency in downer cow syndrome and that serum calcium returned to normal levels with in 24 to 48 hours after treatment with calcium and phosphorus.

Mullen (1975) recorded a value of 5.068 ± 1.579 mg% of calcium in cows associated with paresis.

Catherine (1994) recorded a mean value of serum calcium was 8.53 mg% in downer cows.

Fenwick (1986) stated that hypocalcaemia prevailed in most of the cases of non alert downers.

Singh and Gupta (1987) recorded serum Calcium of 4.2 mg% in parturient paretic buffaloes.

Tripathy (1987) reported that serum calcium level ranged between 3 to 6 mg% in non parturient paretic cows

Pandey and Parai (1988) reported serum Ca level of 5.19 ± 0.06 mg% in hypocalcaemic cross bred cows.

Lincoln and Lane (1990) recorded 3.6 mg% of T Ca at pH of 7.5 in hypocalcaemic cows and that the total calcium (TCa) is less affected by blood pH and direct measurement of TCa is less difficult. Conversely ionised calcium (ICa) is appreciably influenced by pH changes and is more accurate using Ca ion selective electrode also is clearly related to the clinical syndrome.

Dasan and Divya (2001) found that mean calcium level obtained in hypocalcaemic cases were 5.68 ± 0.18 mg%.

Lopez *et al.* (2001) reported decreased serum calcium in downer cow syndrome.

Wadhwa and Prasad (2002a) noticed a mean Ca level in downer cows as 7.5 mg%.

2.8.2 Serum Phosphorus

Curtis *et al.* (1970) observed that hypophosphataemia was consistent during the initial recumbency and phosphorus level returned to normal with in 24 to 48 hours of treatment. However few cases were found to have low serum phosphorus level even after the initial treatment and they were given phosphorus parentrally.

Narayana *et al.* (1977) observed normocalaemia in downer cows and serum calcium level was 11.75 ± 0.83 mg/100 ml.

Daniel and Moodie (1979) reported that the mean plasma inorganic phosphorus concentrations were lowered significantly in a group of seven cows during induction of hypocalcaemia by infusing 4.7% disodium EDTA.

Allen and Davies (1981) reported that hypophosphetaemia had been associated with downer cows.

Inorganic phosphorus level varied from 6.11 ± 0.56 to 4.32 ± 0.31 mg% in calves in which hypocalcaemia was induced by Pandey and Dwivedi (1988).

Inorganic phosphorus levels reported by Pandey and Parai (1988) was 3.10 ± 0.07 mg% in hypocalcaemic cross bred cows.

Catherine (1994) recorded a mean value of 4.14 mg% phosphorus in downer cows.

Gerloff and Swenson (1996) recorded serum phosphorus concentration of 1.8 mg% in two recumbent cows that calved approximately three to four weeks earlier.

In hypocalcaemic cases the serum inorganic phosphorus levels reported by Dasan and Divya (2001) was 2.48 ± 0.18 mg%.

Lopez *et al.* (2001) found normal values of sodium in downer cows.

2.8.3 Serum Sodium

Narayana *et al.* (1977) observed normonatraemia in downer cows and the mean plasma sodium value in four cases of downer cows was 146.75 ± 3.42 mEq/L.

There was a significantly lower mean concentration of sodium and significantly lower plasma sodium and serum magnesium ratio in cows with milk fever (Fenwick, I986).

Catherine (1994) reported that there was no significant difference in values for plasma sodium in downer cows than from normal healthy cows.

Lopez *et al.* (2001) found that there was no significant difference in the plasma sodium from normal cows.

2.8.4 Serum Potassium

Littledike *et al.* (1969) found that cows with parturient paresis associated parturition had lower potassium levels in the plasma than dry and mid lactation cows.

Kronfeld (1974) reported that hypokalaemia was present in many cases of downer cow syndrome.

Plasma and erythrocyte potassium levels were low (2.8 mmol/L) in non alert downers (Fenwick, 1977).

Narayana *et al.* (1977) reported that hypokalaemia present in downer cow syndrome and the mean value for the four cases treated was 4.3 ± 0.21 mEq/L.

Allen and Davies (1981) found that hypokalemia is often associated with hypophosphatemia. Also low potassium concentrations in muscle and serum was observed in some downers.

Fenwick (1986) observed no significant change in plasma sodium concentration in non-alert downer cows.

Catherine (1994) reported that there was no significant difference in values of potassium in downer cows.

Profound hypokalemia was detected by Sielman *et al.* (1997) in eight cows that were recumbent for less than twenty four hours with a concentration range of 1.41 to 2.2 mEq/L.

Lopez *et al.* (2001) reported that values of potassium in downer cows were with in normal range.

2.8.5 Glucose

Thirteen percentage of downer sheep showed hypoglycaemia (<50 mg%) and 22 per cent were hyper glycaemic (50-85 mg%) (Shorthose and Shaw, 1977).

Tripathy (1987) reported blood glucose level between 25-50 mg% in downer cows.

Pandey and Dwivedi (1988) reported that there was no significant difference in the mean value of glucose of hypocalcaemia calves.

Pandey and Parai (1988) recorded glucose level to be 29.0.52 mg% in downers.

Catherine (1994) could not record any significant difference in mean value of glucose in downer cows.

Wadhwa and Prasad (2002a) reported that mean blood glucose level of downer cows was significantly higher than control group (105.41 ± 7.57mg%).

2.8.6 Magnesium

Andrews (1986) is of the opinion that use of combined magnesium, urea and cretinine phosphokionase levels , quality of nursing correctly forecast the out come in 92 per cent of cases sampled .

Pandey and Parai (1988) recorded mean value of 2.98 ± 0.08 mg% in downers.

Catherine (1994) recorded mean value of serum magnesium of 2.28 mg% in downer cows.

2.8.7 Enzymes

Highest creatine kinase levels was observed by *Cox et al.* (1982), in downer cows by forty eight hours.

Prasad *et al.* (1988) reported marked enzyme alteration in downer cows. The alkaline phosphatase increased in gluteal muscle, there was marked loss of ATPase in liver and sciatic nerve while it increased in muscles, biceps femoris and sciatic nerve revealed significant depletion of Sorbitol Dehydrogenase (SDH).

According to Wadhwa and Prasad (2002a) the levels of Asparatate amino transferase (AST), Alanine amino transferase (ALT), Creatine phosphokinase (CPK) and Lactate dehydrogenase (LDH) showed increasing trend with recumbency which was attributed to muscle damage in downer cow posture.

2.9 URINE ANALYSIS OF NORMAL COWS

Salt crystals seen in urine deposit are mainly polygonal crystals of calcium carbonate and needle shaped, or elongated crystals of phosphates, cells are normally present in urine in numbers not exceeding 5000 epithelial cells/ml (Grinder, 1979).

2.10 URINE ANALYSIS OF DOWNER COWS

Curtis *et al.* (1970) regularly observed a marked proteinuria in affected downer cows with in 48 hours of recumbency.

Kronfeld (1974) reported albuminuria in downer cows.

Narayana *et al.* (1977) observed albuminuria in all the four cases of downer cow syndrome they studied.

Amstutz (1981) found light yellow colour of urine with traces of protein.

Cox *et al.* (1982) reported that downer cows voided dark yellow to brown urine indicative of myoglobinuria.

Gangwar (1984) suggested there was ketonuria and albuminuria with hyaline and granular casts.

Catherine (1994) reported ketone bodies in urine of one downer cow and traces in another cow. Glycosuria was also detected in urine of one cow.

There may be moderate ketonuria and marked proteinuria would be evident by 18-24 hour after the onset of recumbency and in severe cases the urine would be brown and turbid due to myoglobinuria (Radostitis *et al.*, 2000).

Wadhwa and Prasad (2002a) recorded glycosuria and proteinuria, in 39 and 43 per cent cases respectively in downer cows.

2.11 OBSERVATION ON DUNG OF DOWNER COWS

Fenwick (1986) reported that all the non alert downer cows under observation developed mucoid faeces after one or two days of recumbency, which in many cases contained blood spots.

In microscopic examination of downer cows any evidence of parasitism could not be revealed (Catherine, 1994).

2.12 PROGNOSIS

Andrews (1986) opined that presenting position of the downer cows could be used for arriving in its probable causes and prognosis. Prognosis is good only for 'creeper' or 'crawler cows', mainly good for 'frog legged cows', usually poor for cows whose legs are extended behind the animal and it is grave in cows whose hind limbs are extended rostrally so that they are in contact with elbows of the front. Prognosis is poor for animals that rest on one side and if moved on to other side then returns to original position and had been down for more than 10 days. He also opined that once the animal has been down longer than ten days the prognosis is poor.

Clark *et al.* (1987) opined that test that were most useful in predicting a lack of recovery in periparturient recumbent cows were serum urea and muscle enzymes.

Disparities in pupil sizes had no association with the response of cows following treatment with calcium borogluconate solutions, and therefore this has no prognostic value, however its presence can be taken to indicate that hypocalcaemia has been present during the previous 10 minutes. (Fenwick, 1989)

Prognosis is poor for those which are still recumbent after seven days, although some affected cows have been down for 10 to 14 days and subsequently stood up and recovered (Radostitis *et al.*, 2000).

Wadhwa and Prasad (2002b) suggested that the activities of Creatine phosphokinase (CPK), Asparatate amino transferase (AST) and Alanine amino transferase (ALT) enzymes decreased gradually in responsive cases but in non responsive cows, thus making these enzymes as good prognostic indicators.

2.13 TREATMENT AND MANAGEMENT

According to Johnson (1963) inclusion of 0.5 g to 1 g potassium acetate, along with calcium dextrose in creeper cow was most effective and that even though recoveries were slow there were no relapses.

Fenwick (1969e) treated downer cows with a solution containing three percentage potassium acetate in addition to 25 percentage calcium borogluconate, with out any improvement.

Thornton (1976) opined that 250 ml Methylene blue as 1% solution in conjunction with calcium borogluconate was effective in cows which were down due to excess phosphorus intake.

Mullen (1977a) noticed that single intravenous administration of at least 8 g calcium is the preferred treatment for hypocalceamia than combining intravenous calcium therapy with a subcutaneous depot of calcium at the same time.

Mullen (1977b) is of the opinion that therapy with solution combining magnesium and phosphorus with calcium should be more effective in preventing relapses of milk fever than calcium alone and clinical response with 8 g calcium was superior to 6.2 g.

Schneider *et al.* (1985) found that decline in plasma calcium was attributable to magnesium deficiency and vitamin D_3 helped to restore magnesium level thereby both calcium, phosphorus and magnesium levels within normal limits.

Fenwick (1986) treated several non-alert downer cows with intravenous potassium acetate solution and oral potassium chloride up to 200 g per day but was with out any significant success.

Pandey and Parai (1988) reported that a combined therapy of intravenous calcium borogluconate 5%, dextrose 25% and intramuscular Tonophosphan along with liver and rumen stimulants was effective.

Barlet and Davicco (1992) reported that 1α-hydroxy cholecalciferol induced a significant increase in plasma concentration of calcium and recovery of the entire downer cows within twelve hours after the treatment.

Prasannakumar *et al.* (1992) treated a downer successfully with Mifex one bottle and 10% Dextrose two bottles intravenously for two days, Neurobion 9 ml, Berin 10 ml, Tonophospan 10 ml, Esgipyrin-N 15 ml, Dicrysticin-S one large dose intramuscularly daily for five days. Potassium acetate was administered orally (30 g) on fourth day as 10% solution, 60 ml slow intravenously on seventh day, 9 ml of neurobion mixed with 9 ml of distilled water was administered epidurally wherein 50 percentage was administered in lumbo-sacral region ands another 50 percentage in sacro-coccygeal. It was repeated for five days, cow was lifted with pulley on tenth day and used slings and was given good nursing care, by twentieth day cow was able to get up with ease and could walk and stand on its own for one to two hours.

Sathishkumar and Rao (2000) reported that combination therapy of intravenous calcium borogluconate (250 ml) and intramuscular Tonophosphan (4 ml) with oral Rumenton boli was highly efficacious than other combinations in treating the hypocalcaemia.

Parturient paretic buffaloes treated by Patel and Jadav (2003) with intravenous calcium therapy showed significant elevation in serum levels of calcium and phosphorous but magnesium and glucose levels were with in normal range before and after treatment

Veena *et al.* (2003) treated all affected downer cows with two doses of 3 ml of neurobion injection diluted with 6 ml of distilled water epidurally, one at lumbo-sacral space and another at first coccygeal space, on first day. Polyionic

solution containing calcium, magnesium and phosphorus was given intravenously, Diclofenac sodium 375 mg was given intramuscularly for two days.

Wadhaw and Prasad (2004) reported 37.5 per cent recovery in downer buffaloes by combined therapy comprising of calcium, magnesium, phosphorus and potassium.

Results of the study conducted by Braun *et al.* (2004) did not support the hypothesis that a slow intravenous calcium drip, after an initial rapid infusion of calcium would be better than a rapid single dose infusion of calcium solution for the treatment of parturient paresis.

2.14 MANAGEMENT

Cattle hoist (hip clamp) and tractor jib have been the perfect managemental devices for those cows showing extreme over flexion of all hind limbs joints (Fenwick, 1969c).

Allen and Davies (1981) suggested that nothing is more likely to discourage an animal from rising than slipping on the floor and hence even in very cold weather a downer cow is better housed in the cold with good bedding than in the warm with inadequate bedding and is of the opinion that hoisting is often useless, because the animal when raised makes no effort to use its limbs. When the animal is either hoisted or is able to stand unaided, the blood circulation should be simulated by massaging the limbs with wisps of straw or similar material and encourage the movements.

Cox (1982) suggested that once a cow is found with parturient paresis it is important to change the animal's position so that further ischaemia is prevented also adequate help should be available to assist the cow into a standing position .If the legs are slipping laterally, tie them together with a rope between the hind fetlocks. Cows should be moved to a more favourable site with good footing. Hip lifting devices are useful if used judiciously.

As managemental aspect it is ideal for the animal to be kept outside, milked twice daily, turn from side to side an odd number of times to try and reduce the possibility of ischaemic muscle damage, hypostatic congestion and pneumonia. If there is a tendency for legs to straddle they can be kept together 4 with a soft rope in a figure of eight or hobbles or use of two dog collars and a rope. More animals kept outside tend to rise than those kept indoors and cows in good condition also have a better prognosis (Andrews, 1986). The author also reported the use of inflatable bags have the advantage of being relatively comfortable, support the cow's body and allow restoration of limb circulation. Also only therapeutic agent with a specific indication for use in downer cow is Tripelennamine hydrochloride.

Contact dermatitis occur more with bedding of straw and wood shavings but sand not only easy to keep clean but also provide good footing and slipping was never observed during the brief ataxic movements when the animal was rising (Cox and Marion, 1992).

In eight recumbent cows that were studied by Sielman *et al.* (1997) all cows were housed in deeply bedded box stalls on dirt floor or rubber mats. They were rolled frequently and hoists slings and hip lifters were used to minimize muscle injuries and formation of decubital ulcers. Thick bandages were placed on distal portion of limbs to minimize excoriation to carpal and tarsal joints. Recumbent cows were treated with 60 g of potassium chloride given orally as boluses up to eight times per day and in addition sterile Potassium chloride was added to intravenous infusions at concentration up to 200 mEq/L.

Lifting cows which make no effort to stand on their own would be usually unsuccessful and when lifted they usually do not bear any significant weight (Radostitis *et al.*, 2000).

2.15 RESPONSE TO TREATMENT

Fenwick (1969e) suggested that a dose of 200 ml twenty five percentage of calcium borogluconate appears to be adequate for a maximum response However there is a definite tendency for higher doses to be tolerated by early cases and long standing cases to tolerate smaller doses.

There is a definite tendency for the percentage of downers and deaths to increase with increasing lapse of time (Fenwick, 1969a).

Fenwick (1969b) observed that a rough estimate of sudden deaths when calcium borogluconate is infused to the point of heart failure would be one in thousand cases. The cardiac abnormalities noted during calcium borogluconate administration were decreased, increased or no change in rate, increased intensity, arrhythmias like double beats, odd beats, missed beats or any combination of these abnormalities. There was no set principle for determining the end point that can cause danger to the life except closely observing the subjective changes in heart rate, rhythm and intensity. He also stated that out of 400 cows with milk fever, 57.6 per cent made unassisted recoveries within ten minutes, 19.9 per cent made assisted recoveries within ten minutes, 17.8 per cent were recumbent (downers) and 4.7 per cent were destroyed, when treated with twenty five percentage 200 ml calcium borogluconate.

2.16 PREVENTION

Prevention of milk fever is not always satisfactory but can be attempted by avoiding the excess input of calcium during the dry period and possibly also by massive doses of Vit D_3 (Payne, 1977).

Sachs *et al.* (1977) suggested that 350µg of 1α (OH) cholecalciferol given at least 24 to 30 hours prepartum can be a valuable agent in the prevention of parturient paresis.

Measures suggested by Allen and Davies (1981) to prevent downer cow syndrome include reducing the incidence of milk fever, correct feeding during pregnancy, prompt treatment of metabolic disorders, reduce dystocia by using appropriate bulls for dams, provide deep bedding in calving boxes.

Cox (1982) suggested that any means of preventing the occurrence of parturient paresis would lower the incidence of the downer cow syndrome. Also a well bedded place to calve with good footing will minimize damage from hard concrete floors and slippery surfaces and finally close observation of cows for signs of parturient paresis will shorten the time when they are recumbent before treatment.

Hove and Kristiansen (1982) suggested that a single oral dose of 500 µg 1,25-dihydroxy Vitamin D_3 given one to three days before parturition can prevent hypocalcaemia at calving.

Addition of anions like chlorides of ammonium, magnesium and calcium can increase tissue response to parathyroid hormone and enabling the cow to better adapt to calcium demands of lactation and reduce incidence of milk fever (Goff *et al.*, 1991).

Goff *et al.* (1996) found that calcium propionate given at calving and again at 12 hour after calving was beneficial in reducing the incidence of milk fever.

Vitamin D and its metabolites, calcium gels orally and PTH hormone infusions or injections can be used to prevent milk fever (Horst *et al*, 1997b).

Goff and Horst (1998) reported that inclusion of HCl into the prepartum ration of Jersey cows entering the third or greater lactation significantly reduced the incidence of milk fever and also reduced the degree of hypocalcaemia during periparturient period.

The early detection and treatment of milk fever will reduce the incidence and severity of downer cow syndrome (Radostitis *et al.*, 2000).

Serum analysis of cows, fed 1kg of synthetic zeolite (sodium aluminium silicate) per day 4 week before the expected date of calving and until first signs of calving, by Hansen and Jorgensen (2001) revealed a greater calcium concentration in zeolite treated cows and reported that none of zeolite treated cows contracted milk fever or experienced sub clinical hypocalcaemia.

Materials and Methods

3. MATERIALS AND METHODS

3.1 MATERIALS FOR THE STUDY

The present study was carried out in the Department of Clinical Medicine, College of Veterinary and Animal Sciences, Mannuthy over a period of twelve months from May 2004 to May 2005. Two downer cows presented at Veterinary College Hospital, Mannuthy and other ten field cases reported from Thrissur District were utilised for the present study. Detailed history was collected. Clinical and physical examination was done according to Boddie (1969). Cows which still remained as recumbent, even after two intravenous calcium therapies during a period of two to three days and which lacked clinical signs indicative of any other disorders were subjected to study. Local Veterinarians unsuccessfully treated all these animals for parturient paresis for two to three days. Twelve animals in early lactation, maintained under identical condition were selected at random and utilised as control animals.

3.2 SAMPLING AND ANALYSIS

About 10 ml of blood was collected by jugular venepuncture from each animal, in a 20 ml disposable syringe. Three millilitres of the whole blood was transferred to a clean dry glass vial which contained 3 mg of EDTA for haematological studies and rest of the blood, without adding anticoagulant, was kept undisturbed for collection of serum. After clot retraction serum was separated by centrifugation at 3000 rpm for 15 minutes. Urine was collected in clean dry 30 ml glass vial. Milk from each quarter was collected in separate clean dry vials. Faecal sample from each animal was also collected. All the above three samples were collected before treatment. All the samples were transported to laboratory within three hours of collection.

Haematological parameters were determined as per the method of Schalm *et al.* (1971). Serum calcium was estimated by modified O-cresolphthalein

method (Biggs and Moorehead, 1974) using standard kit of Agappe, inorganic phosphorus by phosphomolybdate method (Tausskey and Schorr, 1953) using standard kit of Labkit, magnesium by Calmagnite method (Gindler, 1971) using Labkit, and glucose by glucose oxidase peroxidase 4-amino antipyrine (GOD-PAP) method (Trinder and Cain, 1969) using Agappe kit. Photometer 5010 (Boehringer Mannhiem) under standard conditions of operation was used for these biochemical analyses. Serum sodium and potassium levels were estimated by emission flame photometry as described by Oser (1976).

3.3 ANALYSIS OF MILK

Milk samples collected from each animal were subjected to California Mastitis Test (Schalm *et al.*, 1971) and Rothera's test (Benjamin, 1985).

3.4 ANALYSIS OF URINE

Urine analyses were carried out to detect ketone bodies, glucose and urinary sediments according to Benjamin (1985).

3.5 FAECAL SAMPLE EXAMINATION

Freshly collected samples were microscopically examined to detect any ova of parasites as per Sastry (1985).

3.6 THERAPEUTIC MANAGEMENT

The general line of treatment adopted for downer cow in the present study is as follows.

(i) Calcium magnesium borogluconate solution[1] 450-900 ml intravenously or subcutaneously.

(ii) Inorganic phosphorus[2] – 40% (10-20 ml) either intravenously or intramuscularly.

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1 Mifex (Calcium borogluconate 25%, Magnesium hypophosphite 5%, Dextrose 20%), Novartis

(iii) Vitamin AD_3E combination[3] having Vitamin D_3 @ 2.5 lakh IU, intramuscularly.

(iv) Multivitamin preparation[4] 10 ml, intramuscularly to all animals.

In addition supportive treatment with non steroidal anti inflammatory analgesic drugs of Meloxicam group[5] (@ 0.5 mg/kg BW) 30 ml intravenously or salicylate phenylbutazone combination[6] (@ 4 mg/kg BW) 15 ml intravenously or intramuscularly (six cases each) were administered. Dextrose 25%[7] was also administered intravenously for clinically weak animals.

Though the treatment of all the downer cows were adopted based on general line of therapy, the dosage of each drug, frequency of medication, and course of treatment were adopted based on clinical picture, biochemical profile and response of animal during medication. Specific therapy with calcium magenesium borogluconate was initiated only after analysis of the biochemical status of animals. One case had mastitis and in four cows ova of Amphistome

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2 Alphos-40 (79.4 mg of inorganic phosphorus/ml), Alved

3 Adcelin (Vitamin A 120000 IU, Vitamin D_3 250000 IU, Vitamin E 150 mg/mL, Glaxo Smithkline

4 XLPLEX (Thiamine hydrochloride 100 mg, Vitamin B2 5 mg, Vitamin B6 7 mg, Vitamin B12 100 mg, Niacinamide 25 mg, D-Pantethenol 25 mg, Biotin 10 mg,Choline Chloride 5 mg, Lysine hydrochloride 50 mg/ml), Alved

5 Melonex (NSAID of Meloxicam class), Intas

6 Artizone-S (Phenyl butozone 200 mg, Sodium salicylate20 mg)/ mL, Alved

7 Wocktrose (Dextrose 25%), Wockardt

could be detected in the dung sample. The one with mastitis was additionally treated with parenteral administration of Amoxicillin and Cloxacillin[8] 3 g @ 10 mg/kg body weight for five days and those with Amphistome infestation was administered Oxyclosanide[9] 3 g bolus @ 10/kg body weight. The details of the treatment given before the commencement of study and the day wise treatment administered are given in the table no: 1

3.7 STATISTICAL ANALYSIS

Data obtained were analysed statistically using standard 't' test, as per Snedecor and Cohran (1980).

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8 Alvicillin (Amoxycillin and Cloxacillin) Alved

9 Hexanide (Oxyclozanide) Sarabhai Zydus

Table 1. Details of previous treatment and day wise treatment administered

Case No.	Previous treatment	Day 1	Day 2	Day 3	Day 4	Day 5
1	Calcium borogluconate 450 ml, calcium magnesium borogluconate 900 ml, Dextrose 25% 500 ml, Artizone S 30 ml (within 3 days of treatment)	Inj. Artizone S 15 ml i/v Inj. XLPLEX 10 ml i/m	Inj. Calcium magnesium borogluconate 900 ml i/v Inj.Vit. AD_3E 10 ml i/m Inj.XLPLEX 10 ml i/m Inj. Alphos 40 10 ml i/v, 10 ml i/m Inj. Woktrose 500 ml i/v Inj. Artizone S 15 ml i/v (morning and evening) Hexanide 3 g orally	Inj. Artizone S 15 ml i/v (morning and evening)	Inj. Calcium magnesium borogluconate 900 ml i/v Inj. Vit. AD3E 10 ml i/m Inj. XLPLEX 10 ml i/m Inj. Alphos 40 20 ml i/m Inj.Woktrose 500 ml i/v (Morning and evening)	
2	Calcium borogluconate 450 ml, Calcium magnesium borogluconate 450 ml Artizone S 30 ml Dextrose 25% 500 ml (during a period of 2 days)	Inj. Artizone S 15 ml i/v Inj. XLPLEX 10 ml i/m	Inj. Mifex 900 ml Inj. Alphos 40 20 ml i/v Inj. Woktrose 500 ml i/v Inj.XLPLEX 10 ml i/m Inj.Vit. AD3E 10 ml i/m Inj. Artizone S 15 ml i/v (morning and evening)	Inj. Artizone S 15 ml i/v (morning and evening)	Inj. Artizone S 15 ml i/v (morning and evening) Inj. XLPLEX 10 ml i/m	Inj. Mifex 900 ml Inj. Alphos 40 20 ml i/v Inj. Woktrose 500 ml i/v Inj. XLPLEX 10 ml i/m Inj. Vit AD_3E 10 ml i/m Inj. Artizone S 15 ml i/v (morning and evening)

3.	Calcium magnesium borogluconate 900 ml Calcium 450 ml Alphos 40 - 30 ml Dextrose 25% 500 ml Rintose 1 bottle Artizone - 30 ml (during a period of 4 days)	Inj. Artizone 15 ml i/v Inj. XLPLEX 10 ml i/m	Inj. Mifex 900 ml Inj. Alphos 40 20 ml i/v Inj. Woktrose 500 ml i/v Inj. XLPLEX 10 ml i/m Inj. Vit AD_3E 10 ml i/m Inj. Artizone 15 ml i/v			
4.	Calcium magnesium borogluconate 900 ml Dextrose 10% 1000 ml Inj. Alphos 40 - 10 ml Inj. Vit AD_3E (5000 IU) Inj. Artizone S 40 ml (during a period of 3 days)	Inj. Artizone S 15 ml i/v Inj. XLPLEX 10 ml i/m	Inj. Mifex 600 ml i/v, 250 ml s/c Inj. Alphos 40, 10 ml i/v Inj. Woktrose 500 ml i/v Inj. XLPLEX 10 ml i/m Inj. Vit. AD_3E 10 ml i/m Inj. Artizone S 15 ml i/v Hexanide 3 g orally		Inj. Mifex 900 ml i/v Inj. Alphos 40, 10 ml i/v Inj. Woktrose 500 ml i/v Inj. XLPLEX 10 ml i/m Inj. Vit. AD_3E 10 ml i/m Inj. Artizone S 15 ml i/v	
5	Calcium magnesium borogluconate 900 ml Dextrose 10% 500 ml Artizone 30 ml (during a period of 2 days)	Inj. Meloxicam 30 ml i/v Inj. XLPLEX 10 ml i/m	Inj. Mifex 900 ml Inj. Alphos 40 20 ml i/v Inj. XLPLEX 10 ml i/m Inj. Vit AD_3E 10 ml i/m Inj. Artizone S 15 ml i/v Inj. Woktrose 500 ml i/v	Inj. Mifex 900 ml Inj. Alphos 40 20 ml i/v Inj. XLPLEX 10 ml i/m Inj. Vit AD_3E 10 ml i/m Inj. Artizone S 15 ml i/v Inj. Woktrose 500 ml i/v		

6	Calcium magnesium borogluconate 900 ml Calcium borogluconate 450 ml Dextrose 25% 3000 ml (during a period of 3 days)	Inj. Meloxicam 30 ml i/v Inj. XLPLEX 10 ml i/m	Inj. Mifex 450 ml Inj. XLPLEX 10 ml i/m Inj. Vit AD_3E 10 ml i/m Inj. Alphos 40 20 ml i/v Inj. Meloxicam 30 ml i/v			
7	Calcium borogluconate 450 ml i/v Calcium magnesium borogluconate 250 ml (during a period of 2 days)	Inj. Meloxicam 30 ml i/v Inj. XLPLEX 10 ml i/m	Inj. Mifex 450 ml i/v Inj. Alphos 40 20 ml i/v Inj. XLPLEX 10 ml i/m Inj. Vit AD_3E 10 ml i/m Inj. Meloxicam 30 ml i/v Hexanide 3 g orally			
8	Calcium magnesium borogluconate 3150 ml (7 bottles) Dextrose 10% 2000 ml (4 bottles) Inj. Alphos 40 40 ml Inj. Vit AD_3E (1.25 lakhs IU) Inj. Artizone S 30 ml (during a period of 4 days at different occasion)	Inj. Artizone S 15 ml i/v Inj. XLPLEX 10 ml i/m	Inj. Mifex 900 ml i/v Inj. Alphos 40 20 ml i/v Inj. XLPLEX 10 ml i/m Inj. Vit AD_3E 10 ml i/m Inj. Woktrose 500 ml i/v Inj. Artizone S 15 ml i/v Hexanide 3 g orally			
9	Calcium borogluconate 450 ml Calcium magnesium borogluconate 450 ml Vit AD_3E 5 ml i/m Dextrose 25% 1000 ml Artizone 30 ml (during a period of 2 days)	Inj. XLPLEX 10 ml i/m Inj. Meloxicam 30 ml i/v	Inj. Mifex 450 ml i/v 200 ml s/c Inj. Alphos 40 20 ml i/v Inj. XLPLEX 10 ml i/m Inj. Vit AD_3E 10 ml i/m Inj. Meloxicam 30 ml i/v			

10	Calcium magnesium borogluconate 450 ml Calcium borogluconate 450 ml Artizone 30 ml Hivit 10 ml Alphos 40 - 10 ml (during a period of 3 days)	Inj. Artisone 15 ml i/v Inj. XLPLEX 10 ml i/m	Inj. Mifex 900 ml i/v Inj. Alphos 40 20 ml i/v Inj. XLPLEX 10 ml i/m Inj. Vit. AD_3E 10 ml i/m Inj. Woktrose 500 ml i/v Inj. Artisone 15 ml i/v			
11	Sodium bicarbonate orally and intravenously Corticosteroids and Dextrose 10% 1000 ml Calcium magnesium borogluconate 600 ml on next day	Inj. Meloxicam 30 ml i/v Inj. XLPLEX 10 ml i/m	Inj. Mifex 900 ml i/v, 250 ml s/c Inj. Alphos 40, 20 ml i/v Inj. XLPLEX 10 ml i/m Inj. Vit AD_3E 10 ml i/m Inj. Woktrose 500 ml i/v Inj. Meloxicam 30 ml i/v			
12	Enrofloxacin 30 ml for 2 days and then Enrofloxacin and Gentamicin for 15 ml each for three days and Calcium magnesium boroluconate 450 ml Dextrose 25% 500 ml i/v Artizone 15 ml (within a period of 2 days)	Inj. Meloxicam 30 ml i/v Inj. XLPLEX 10 ml i/m	Inj. Mifex 450 ml i/v Inj. Alphos 40 10 ml i/v Inj. XLPLEX 10 ml i/m Inj. Vit AD_3E 10 ml i/m Inj. Artizone S 15 ml i/v			

Results

4. RESULTS

The present study was carried out from May 2004 to May 2005. Two downer cows presented at Veterinary College Hospital and other ten field cases reported from Trissur district formed the source of clinical cases. Out of 12 cows, seven were crossbred Jersey, three were crossbred Brown Swiss (BS) and two were crossbred Holstein-Friesian (HF). Age of the cows was within four to eight years. One cow was in first parity, five were in second, four in third and the rest two of them in fifth parity. Nine cows were in early lactation. One cow was in eighth month of gestation or pregnancy and two cows were in mid lactation. Average milk production ranged from 6-10 L/day and in one cow it was 21 L/day.

Table 2. Signalment of downer cows

Case No.	Breed (crossbred)	Age	Parity	Production (L)	Stage of lactation	Days of lactation
1	Jersey	4	P1	10	Early	7
2	Jersey	5	P2	8	Early	4
3	Jersey	5	P2	7	Early	3
4	Jersey	8	P5	9	Early	7
5	Jersey	5	P2	8	Early	5
6	Jersey	5	P2	10	Early	5
7	Jersey	6	P3	8	Early	4
8	HF	8	P3	21	Early	3
9	BS	8	P5	8	Early	3
10	HF	6	P2	Eighth month of pregnancy		
11	BS	8	P3	6	Mid	3 months
12	BS	8	P3	10	Mid	6 months

Fig. 1 Downer cow - Alert

Fig. 2 Downer cow -with characteristic right hind fetlock flexion - before treatment

Fig.3 Recovered cow - after treatment

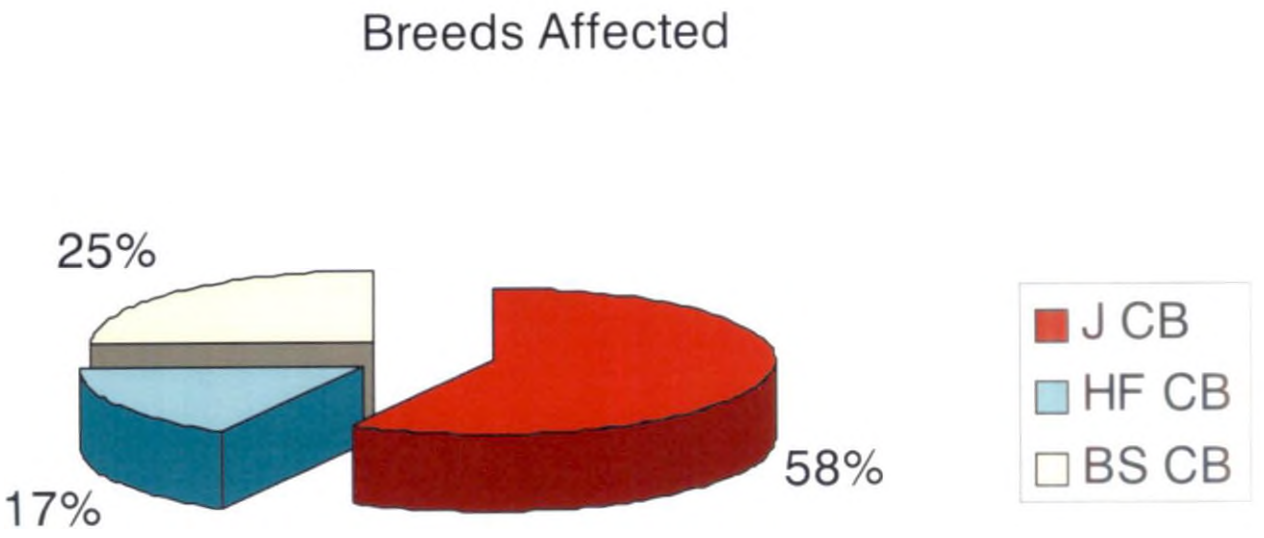

Fig 4: Breeds affected in the present study

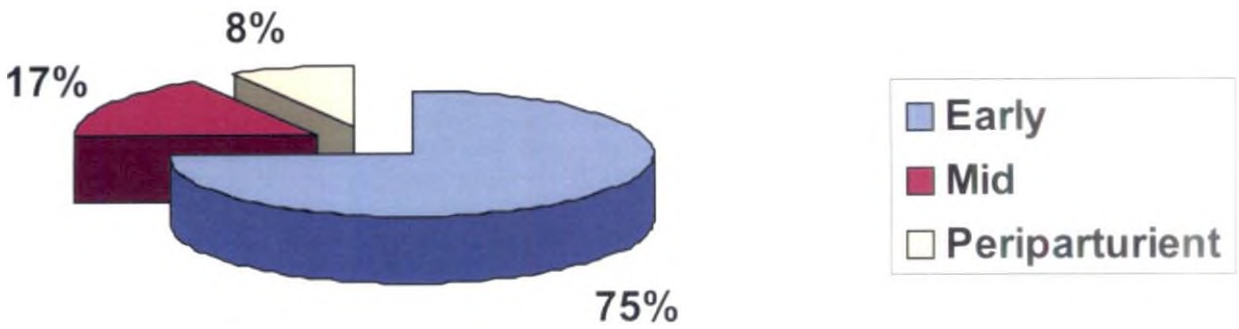

Fig 5: Stage of Lactation

4.1 HISTORY

4.1.1 Feeding Details

History revealed that all the downer cows were fed with a constant quantity (2 to 5 kg in all cows except one cow, which had a yield of 21 L, received 10 kg, irrespective of the stage of lactation) of compounded concentrate ration. Rapid change in feed was reported in one case, where the cow was nearing dry period and to stop milk production the total feed was reduced rapidly and then the disease developed.

4.1.2. Housing Details

All the cows were maintained on concrete floors but none of the cows had the history of a fall or fracture before the development of recumbency. Exposure to extreme climate like heavy rain or summer was also not reported as all the downer cases occurred during the period of May 2004 to May 2005.

4.1.3 History of Previous Milk Fever

Milk fever and ketosis were reported in previous lactation of a cow with high yield (21 L). None of the other cows had the history of either condition during any of the previous lactation.

4.1.4 History of Other Diseases in the Recent Past

One cow was treated by a local veterinarian for acute mastitis, with Enrofloxacin, before the onset of disease condition. One cow was affected with acute ruminal lactic acidosis and subsequently it developed into a downer. One cow had concurrent ketosis before the first treatment.

4.2 CLINICAL SIGNS

All the cows were in sternal recumbency for 24-48 hours at the time of presentation. All the cows were alert. Most of the cows made frequent attempts to rise by using their forelimbs and made their own effort to change the sides, except in one cow, in which attempts were few and animal was reported to be dull and inactive. Three cows could 'crawl' for a short distance. Rectal temperature ranged from 100.8° to 101.2°F. Pulse and respiratory rates were ranged from 60 to 70 per minute and 30 to 45 per minutes respectively. Conjunctival mucous membrane of all the animals were pale roseate. Rumen motility was within a range of 3-5/5 minutes. Feed intake was normal in most cases. Urination and defecation were normal. Mild dehydration was also observed in most of the animals. Normal rumination was also observed. One cow developed mastitis during the course of treatment. Development of bruises over hock, elbow, tuber coxae were the commonly observed complications in downer cows. One cow showed a characteristic right fetlock flexion.

4.3 HAEMATOLOGICAL OBSERVATIONS

Haematological value of normal and downer cow are presented in Table 3.

4.3.1 Volume of Packed Red Cell

The mean value of volume of packed red cell in normal cows was 33 ± 1.20%, whereas the mean value of volume of packed red cell in downer cows was 35.83 ± 1.34 %. No statistically significant difference was observed between the values of normal and diseased group.

4.3.2 Haemolgobin

Mean value of haemoglobin in normal animals was 11 ± 0.35 g/dl while that of downer animals was 12.08 ± 0.34 g/dl. A statistically significant increase

($P< 0.05$) was obtained in mean haemoglobin value of diseased animals when compared to healthy control.

4.3.3 Total Leukocyte Count (TLC)

Mean value of TLC in normal animals was $5.77 \pm 0.27 \times 10^3/\mu l$ and that of downer cows was $7.78 \pm 0.57 \times 10^3/\mu l$. There was statistically significant increase ($P< 0.05$) in total leucocyte count in downer cows when compared with control animals.

4.3.4 Differential Leukocyte Count (DLC)

4.3.4.1 Lymphocyte

Mean value of lymphocyte count in control animals was $64.58 \pm 1.5\%$ while the mean value of lymphocyte in downer cows was $59.67 \pm 1.18\%$. Mild ($P< 0.05$) lymphopenia was observed in downer cows when compared with control animals, which was statistically significant.

4.3.4.2 Neutrophil

The mean value of neutrophil count in normal animals was $31 \pm 1.55\%$ and in downer cows it was $35.17 \pm 1.72\%$ mild. Neutrophilia observed in downer cows was statistically significant ($P< 0.05$), when compared with control cows.

4.3.4.3 Eosinophil

Mean value of eosinophil count in healthy control animals was $3.75 \pm 0.34\%$ and mean value of eosinophil count in downer cows was $4.42 \pm 0.61\%$. There was a slight increase in the eosinophil count in downer cows. But the difference between the values was not statistically significant.

4.3.4.4 Monocyte

Mean value of monocyte count in healthy control animals was 0.5 ± 0.23% and the mean value of monocyte count in downer cows was 0.33 ± 0.14%. Analysis showed that difference between the values were not statistically significant.

4.3.4.5 Basophil

Mean value of basophil count in healthy control animals was 0.17 ± 0.11% while the mean value of basophil count in downer cows was found to be 0.17 ± 0.11%. There was no significant difference in values of basophil between downer cow and normal animals.

Table 3. Haematological values of normal and cows with downer cow syndrome

Parameters	Healthy control cows (Mean ± SE)	Diseased animals (Mean ± SE)	Probability
VPRC (%)	33 ± 1.20	35.83 ± 1.34	NS
Haemoglobin (g/dl)	11 ± 0.35	12.08 ± 0.34	P<0.05
TLC x $10^3/\mu l$	5.77 ± 0.27	7.78 ± 0.57	P<0.05
D L%	64.58 ± 1.51	59.67 ± 1.18	P<0.05
L N%	31 ± 1.55	35.17 ± 1.72	P<0.05
C E%	3.75 ± 0.39	4.42 ± 0.61	NS
M%	0.5 ± 0.23	0.33 ± 0.14	NS
B%	0.17 ± 0.11	0.17 ± 0.11	NS

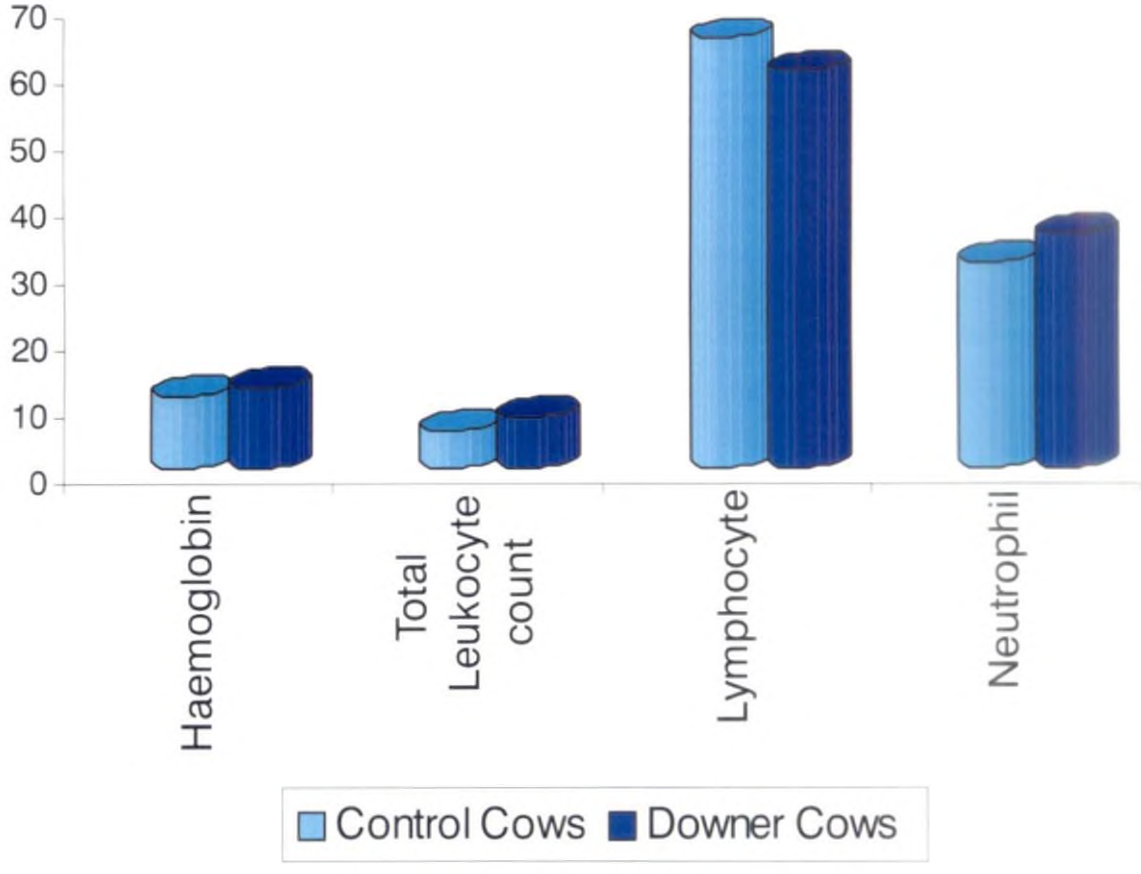

Fig 6: Haemotological changes

4.4 BIOCHEMICAL OBSERVATIONS

Biochemical value of normal and downer cow are presented in Table 4.

4.4.1 Serum Calcium

Mean value of serum calcium in healthy control animals was 8.88 ± 0.20 mg%. The mean value of serum calcium in downer cows was found to be decreased to 6.1 ± 0.33 mg%. Analysis showed that the reduction in the value of serum calcium in downer cow was highly significant ($P< 0.01$) when compared to healthy control.

4.4.2 Serum Phosphorus

Mean value of serum phosphorus in healthy control animals was 4.28 ± 0.12 mg% and downer cows it was decreased to 3.64 ± 0.19 mg%. The difference between the values was statistically significant ($P< 0.05$).

4.4.3 Serum Magnesium

Mean value of serum magnesium in healthy control animals was 2.20 ± 0.07 mg%. Mean value of serum magnesium of downer cows' was found to be in the normal level of 2.27 ± 0.13 mg%. There was no statistically significant difference in the mean values of serum magnesium between the control animals and downer cows.

4.4.4 Serum Sodium

Mean value of serum sodium in healthy control animals was 134.65 ± 1.27 mEq/L and the mean value of serum sodium in downer cows was 134.83 ± 1.14 mEq/L. There was no statistically significant difference in the mean values of serum sodium between downer and control cows.

4.4.5 Serum Potassium

Mean value of serum potassium in healthy control animals was found to be 4.31 ± 0.14 mEq/L. Mean value in downer cows was 4.2 ± 1.70 mEq/L. Difference between the values of serum potassium of downer and control cows was not statistically significant

4.4.6 Serum Glucose

Mean value of serum glucose in healthy control animals was found to be 43.97 ± 1.74 mg% and in downer cows the mean value of serum glucose was 43.96 ± 1.70 mg%. There was no significant statistical difference between the mean values of diseased and control animals.

Table 4. Biochemical values of normal and downer cows

Parameters	Healthy control cows (Mean ± SE)	Diseased animals (Mean ± SE)	Probability
Ca (mg%)	8.88 ± 0.20	6.1 ± 0.33	P<0.01
P (mg%)	4.28 ± 0.12	3.64 ± 0.19	P<0.05
Mg (mg%)	2.2 ± 0.07	2.27 ± 0.13	NS
Na (mEq/L)	134.65 ± 1.27	134.83 ± 1.14	NS
K (mEq/L)	4.3 ± 0.14	4.2 ± 1.70	NS
Glucose (mg%)	43.97 ± 1.74	43.96 ± 1.70	NS

4.5 EVALUATION OF MILK OF DOWNER COWS

4.5.1 California Mastitis Test (CMT)

Result of milk sample examination from all the downer animals was negative for CMT.

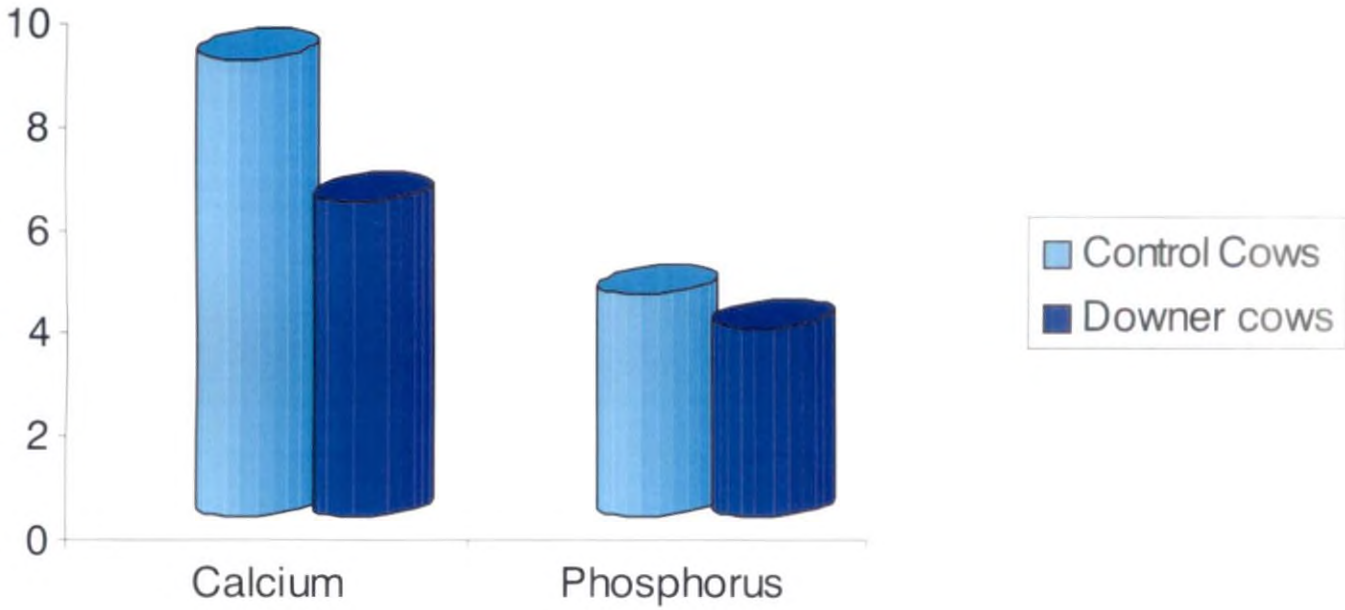

Fig 7: Biochemical changes

4.5.2 Rothera's Test

Rothera's test with milk sample did not reveal any Ketone bodies in the milk of downer cows.

4.6 DATA ON URINANALYSIS

4.6.1 Ketone Bodies

Rothera's test with urine of downer cow showed traces of ketone bodies in case no:3 and no:8 and was moderate in case no : 10.

4.6.2 Glucose

Benedicts test with urine of downer cows revealed that there was no glycosuria in downer cows.

4.6.3 Urinary Sediments

On microscopical examination of urinary sediments of downer, polygonal crystals of calcium carbonate and needle shaped or elongated crystals of calcium phosphate were seen. Large polygonal epithelial cells of bladder mucosa were also noticed. No abnormal crystals or cell could be detected in urine of downer cows.

4.7 FAECAL SAMPLE EXAMINATION

On microscopical examination of dung sample of downer cow Amphistome ova could be detected in case No.1, 4, 7 and 8.

Table 5 Urinanalysis and feacal sample examination in downer cows

Case No.	Breed (Crossbred)	Urine for ketone bodies	Faecal sample
1	Jersey		Amphistome ova (+++)
2	Jersey		
3	Jersey	Traces (+)	
4	Jersey		Amphistome ova (++)
5	Jersey		
6	Jersey		
7	Jersey		Amphistome ova (++)
8	HF	Traces (+)	Amphistome ova (++)
9	BS		
10	HF	Moderate (++)	
11	BS		
12	BS		

4.8 THERAPY

The general line of treatment administered was calcium magnesium borogluconate 450 to 900 ml intravenously or subcutaneously, inorganic phosphorus either intramuscularly or intravenously, vitaminAD_3E combination having D_3 @ 2.5 lakh IU intramuscularly and multi vitamin preparation 10 ml intramuscularly to all animals. Supportive treatment with nonsteroidal anti inflammatory analgesic drugs of Meloxicam group @ 0.5 mg/kg BW 30 ml intravenously or phenylbutazone salicylate combination 15 ml intravenously or intramuscularly for six cases each were administered. Those cows that were clinically weak Dextrose 25% were also administered. Specific therapy was initiated only after analysis of biochemical status of animals. The administration of calcium magnesium borogluconate to each cow was continuously given till the clinical response to calcium therapy like smacking of lips, muscle tremor, belching, eructation etc. were noticed.

Muscle tremors that started from flank and extending to neck were noticed in all animals. The audibility of heart sounds was feeble before the

initiation of therapy. But during the therapy the heart sounds became more audible and rate was increased rapidly.

In four cases hypophosphatemia was recorded and inorganic phosphorus was administered to overcome the deficiency in these cows and in others to prevent further occurrence of hypophosphatemia.

Those animals which did not rise even after 24 hours of last treatment were either repeated with the same drugs on the second day or were given only antiinflammatory drugs and multivitamin with the expectation that as the frequency of unassisted attempts by them were more they would be on feet with in hours.

Vitamin D_3 was administered intramusculary in all animals to maintain calcium homeostatis quickly. Multivitamin preparation was administered as a supportive therapy to facilitate general mobility.

With all these treatments seven cows made assisted recovery with in 5 to 12 hours with 4 days in 2 cows, 5 days in one cow and 2-3 days 4 cows. One cow that required 5 days of treatment was on feet 5 hours of last treatment.

Those cows which were administered phenylbutazone salicylate combination had taken only about 10 hours to rise but those which were given Meloxicam group of NSAIDs had taken 10 to 12 hours to be on feet.

Before the treatment calcium phosphorus ratio was 1.7 : 1 and with sufficient quantity of calcium therapy it was raised to normal ratio of 2.1 : 1

Five cows that did not recover were either slaughtered or test was discontinued because of unwillingness of owners. A total of 58.3 % recovery was obtained.

Table 6. Treatment response in downer cows

Case No.	Duration of treatment	Response	Complications other than bruises	Total days under treatment (days)
1	4	Recovered	Nil	7
2	5	Recovered	Mastitis	7
3	2	Recovered	Nil	6
4	4	Slaughtered	Oedema of hock and fetlock joint	7
5	3	Did not respond, slaughtered	Nil	5
6	2	Slaughtered	Severe traumatic injury	5
7	2	Recovered	Nil	4
8	2	Recovered	Nil	6
9	2	Recovered	Fracture	4
10	3	Slaughtered	Nil	5
11	2	Treatment discontinued	Nil	5
12	2	Recovered	Nil	4

Table 7. Haematological observations after treatment in downer cows

Recovered animals (7 Nos.)	VPRC (%)	Hb (g/dl)	TC ($x10^3/\mu l$)	DLC (%)				
				L	N	E	M	B
Mean ± SE	31.86 ± 1.22	10.37 ± 0.33	5.49 ± 0.24	65.00 ± 1.88	30.57 ± 1.94	3.86 ± 0.51	0.86 ± 0.34	0

Table 8. Biochemical picture of animals recovered from downer cow syndrome

Recovered animals (7 Nos.)	Ca (mg%)	P (mg%)	Mg (mg%)	Na (mEq/L)	K (mEq/L)	Glucose (mg%)
Mean ± SE	9.24 ± 0.20	4.41 ± 0.16	2.34 ± 0.10	135.82 ± 1.22	4.186 ± 0.08	43.75 ± 1.16

Discussion

5. DISCUSSION

Recumbent cow is one of the most challenging problem a field veterinarian can face.

Considering the importance of this problem the present study was carried out to elucidate the probable etiological factors, better therapeutic and managemental regimen in downer cows.

A total number of 12 alert downer cows, which did not respond to field veterinarians' treatment were utilized for the present study.

5.1 SIGNALMENT

5.1.1 Breed Affected

Breeds of downer cows in the present study were Jersey cross (7), Brown Swiss cross (3) and Holstein Friesian (2) cross bred. This observation that highest percentage of cows affected were of Jersey cross was in agreement with the earlier authors (Narayana *et al.* (1977); Cox *et al.* (1986) and Kavitha *et al.* (2002). However this observation was based on phenotypic characters of cows and the association with geno type should be studied.

5.1.2 Age

In the present study all the cows were within 4 to 8 years of age and out of that 41% of cows were of 8 years and there was only one heifer. More occurrence of downer cow in aged animals was in agreement with the earlier workers (Johnson, 1963; Narayana *et al.*, 1977 and Wadhawa and Prasad, 2002b). Though there is no age specificity for occurrence of downer cow syndrome, milk fever in older cows and dystocia in heifers are important risk factors for the development of downer cows (Erb and Grohn, 1988).

5.1.3 Parity

Parity of 12 cows in this study ranged within first and fifth lactation and highest percentage (58.3%) of parity of downer cows lies within third and fifth. Kavitha *et al.* (2002) also found that highest percentage (45%) of downer cows was within third and fourth lactation. One cow was in advanced pregnancy. Catherine (1994), Kavitha *et al.* (2002) and Wadhwa and Prasad (2002a) also have recorded periparturient downer cows.

5.1.4 Production

The cows in this study were high producers and one cow was exceptionally high producer (21 L). Too low production was not recorded in any case. There were no data regarding the risk of downer syndrome for cows in high production rather than low producing ones (Erb and Grohn, 1988). But Cox *et al.* (1986) reported that downer cows were high producers (48%) or average producers (46%) with only 6% being low producers.

5.1.5 Stage of lactation

Seventy five percentage of the downer cows in the present study was in their early lactation and that too within first week of lactation. This agrees with the report of Johnson (1963), Allen and Davies (1981) and Wadhwa and Prasad (2002b), that eventhough downer cows syndrome can occur at any time, during lactation or dry period, highest incidence occur during early lactation or immediately after parturition. There may be many factors that make a cow recumbent but most cases follow milk fever which usually occur within few days after parturition (Andrews, 1986; Fenwick, 1986 and Radostitis *et al.*, 2000).

5.1.6 Season

All the cases were recorded during the period from September to May. Catherine (1994) recorded higher incidence rate of downer cows during summer season in Kerala. However this finding in present study is more corroborative

with the report of Kavitha *et al.* (2002) that occurrence of downer cows syndrome was evenly distributed throughout the year.

5.2 HISTORY

5.2.1 Feeding Details

In the present study all the animals were being fed with compounded concentrate feed and it was revealed from history that the quantity of feed was not proportionate to the production requirements. Neither excessive intake of bran, nor feeds which disturbs calcium metabolism, was reported. Rapid change in quantity of feed was reported in one case. In this case, the cow was eight months of gestation and in order to stop the milk production, concentrate ration and fodder were drastically reduced all of a sudden and subsequently the disease developed. This cow was in severe stress due to both pregnancy and starvation. Stressors like starvation for forty eight hours causes severe depression of serum calcium level, and this may be of importance in the production of hypocalcaemic paresis during periods other than postparturient stage (Radostitis *et al.*, 2000).

5.2.3 Housing Details

Highest rate of downer cow syndrome was for those housed in stanchions and lowest rate for combination stanchions and loose (Cox *et al.*, 1986). The downer cows in the present study were maintained in semi intensive or combination type of housing, so the risk factors of housing did not influence in the present study. Exposure to cold climate (Amstuz, 1981), and higher wind velocity (Fenwick, 1969d) could result in hypothermia and prolonged recumbency. But downer cows in the present study were not exposed to adverse climatic condition. More over none of the owners had reported a history of a fall or fracture. So in this study cows became recumbent probably due to alterations in metabolic profile.

5.2.4 History of Previous Milk Fever

Both milk fever and ketosis were reported in a cow (8.3%) with a high yield of 21 L (case no: 8). During previous lactation none of the other cows had the history of either condition. Radostitis *et al.* (2000) reported that individual cows, and to some extend families of cow are more susceptible than others and the disease tends to reoccur at successive parturitions.

5.2.5 History of Other Disease in the Recent Past

Cows with retained placenta and dystocia are more likely to develop downer cow syndrome than cows with either disorders (Correa *et al.*, 1993). But in this study none of the cows had difficult parturition. One cow (case no: 12) was treated for acute mastitis for past 4 days with antibiotics like Enrofloxacin initially. Another case (case no: 11) occurred in mid lactation and onset of symptoms was immediately after recovery from acute ruminal lactic acidosis. During ruminal lactic acidosis cases there would be a mild hypocalcemia due to temporary malabsorption (Radostitis *et al*, 2000).

5.3 CLINICAL SIGNS

All the cows were in sternal recumbency, alert and without any other abnormalities. Most of the cows (92%) made frequent attempts to rise with their forelimbs but hind legs weakness were noticed. All the physiological parameters were within the normal range. These observations were similar to the reports of Curtis *et al.* (1970), Allen and Davies (1981), Wadhwa and Prasad (2002b) and Radostitis *et al.* (2000) that clinically the animal may be normal except for recumbency, cows eat and drink normally, vital signs within normal range and with normal alimentary tract function. Reduction of appetite in one cow (8%) agrees with the report of Johnson (1963) and Wadhwa and Prasad (2002b). The recumbency and slight reduction in rumen movements might be due to hypocalcaemia (Tripathy, 1987).

Twenty five percentage of cows in present study exhibited creeping or crawling movement simulates the observations reported by Johnson (1963). Allen and Davies (1981) reported similar findings that cows used to crawl around the box or along the ground but were unable to stand. Flexion of hind fetlock in one cow agrees with observation of Curtis *et al.* (1970), Allen and Davies (1981), Cox *et al* (1982), Cox and Marion (1992) and Catherine (1994).

Illthriftiness, frequent urination, staggering gait etc. manifested before going down in two cows (16%) resemble the symptom explained by Radostitis *et al.* (2000) and this could be due to the fact that those animals were calcium cyclers.

Illthriftiness is also a symptom of hypophosphataemia (Clark, 1974). Complications like mastitis and traumatic injury during application of sling were noticed. Bruises were a common complication in all cows. Radostitis *et al.* (2000) reported that complications like coliform mastitis, decubital ulceration, especially over the bony prominences of the hock and elbow joints and traumatic injury around the tuber coxae caused by hip slings are common in downer cows. In this study one cow was slaughtered due to severe trauamatic injury encountered during application of sling. So the slings should be used judiciously in downer cows.

Variations in the clinical signs in individual animals are attributable to factors such as breed, age, feeding, management, environment, milk yield, climate, biochemical and pathological changes. Catherine (1994) also could not draw a conclusion on typical clinical signs of the disease due to the above factors.

5.4 HAEMATOLOGIAL OBSERVATIONS

5.4.1 Volume of Packed Red Cells (VPRC or PCV)

Mean value of volume of packed red cells in normal cows (33 ± 1.20%) was comparable to the values reported by Kramer (2000). There was a slight

increase in the mean volume of packed red cell in downer cows (35.83 ± 1.34%). But the increase was statistically non significant

A higher value of volume of packed red cells was recorded in non alert downers by Fenwick (1986), Prasad *et al.* (1987) and Pandey and Parai (1988). Catherine (1994) attributed the reasons for high volume of packed red cells as release of catecholamines, epinephrine and nor epinephrine during excitement and due to spleenic contraction. In the present study all the cows had mild dehydration as quantum of feed and water intake might not be sufficient during the period of recumbency.

5.4.2 Haemoglobin

The mean value of haemoglobin in normal animals (11 ± 0.35 g/dl) was in accordance with the values reported by Payne *et al.* (1974). Catherine (1994) and Kramer (2000).

A significant increase (12.08 ± 0.34 g/dl) in the value of haemoglobin in downer cows in this study was in accordance with the findings of Catherine (1994). Increased haemoglobin was found in hypocalcaemic cows, calves and sheep by earlier authors (Pandey and Dwivedi, 1988 and Barzanji and Daniel, 1988).

A significant increase in the haemoglobin in the present study could be due dehydration and corresponding increase in the volume of packed red cells in diseased cows.

However Prasad *et al.* (1987) recorded a low haemoglobin level in individual cow and this was later diagnosed as downer with poor prognosis. Wadhwa and Prasad (2002a) also reported a significant lower value in haemoglobin in downer cows. This might be due to concurrent occurrence of severe anaemia in these downer cases.

5.4.3 Total Leukocyte Count (TLC)

Mean values in normal animals (5.77 ± 0.27 x $10^3/\mu l$) were comparable with the values of total leukocyte count reported by Catherine (1994) in normal animals.

The increase in the total leukocyte count to 7.78 ± 0.57 x $10^3/\mu l$ in downer cows was statistically significant when compared to the control cows. However this value was with in the normal range (Kramar, 200). Sielman *et al.* (1997) have reported an increase up to 18,600 cell/μl in an individual cow, which was recumbent. In the present study the increase in TLC could be due to severe traumatic injuries and subsequent inflammation occurred on the elbow joint, hock joint, tubercoxae and stifle. Moreover to one cow the first visit was immediately after treatment for acute mastitis and in another cow mastitis developed during the course of treatment. Leukocytic response observed in the present study cannot be considered due to any systemic pathological condition in downer cows.

5.4.4 Differential Leukocyte Count (DLC)

Mean value of differential leukocyte count obtained in control animals corroborates with the findings of Greatorex (1957), Pyne and Maitra (1981), Catherine (1994) and Wadhwa and Prasad (2002a).

A statistically significant increase in mean value of neutrophils (35.17 ± 1.72%) and decrease in lymphocytes (59.67 ± 1.18%) in the present study was similar to the reports of Amstuz (1981) that there was shift in neutrophils and lymphocytes, which could be due to traumatic injury and subsequent inflammation. Wadhwa and Prasad (2002a) have also reported a similar finding. Catherine (1994) has reported that eventhough there was highly significant increase in the mean value of neutrophils the decrease in mean value of lymphocytes was not statistically significant. There was no significant difference between the mean values of basophil and monocyte. Even though there was a

slight increase in mean value of eosinophil the increase was not statistically significant.

5.5 BIOCHEMICAL OBSERVATIONS

5.5.1 Serum Calcium

The mean value of serum calcium in the control animals (8.88 ± 0.20 mg%) obtained in the present study was well with in the range reported by earlier workers (Sivaiah *et al.,* 1986; Pandey and Parai (1988) and Rosol and Capen (1997).

Mean value of serum calcium in the present study was found to be decreased to 6.1 ± 0.33 mg% which was statistically highly significant ($P<0.01$). This observation agrees with the reports of Curtis *et al.* (1970), Fenwick (1986) and Catherine (1994).

Excessive loss of calcium in the colostrum beyond the capacity of absorption from the intestine at parturition and insufficiently rapid mobilization of calcium from bones to maintain normal serum calcium levels are the factors which affect calcium homeostasis. Calcium lost from the plasma pool must be replaced by increasing intestinal calcium absorption or increasing the bone calcium resorption or both. Parathyroid hormone (PTH) and the active form of vitamin D i.e., 1,25 dihydroxy vitamin D are the two agents that normally respond at the time of hypocalcaemia to increase the plasma calcium by the entry of calcium into the blood from bone stores and from the intestine (Horst *et al.*, 1997b and Radostitis *et al.*, 2000).

Cows develop milk fever as a result of failure of one or both of these factors to maintain adequate blood calcium at the onset of lactation (Goff *et al.*, 1996). At parturition the cow must bring greater than or around 30 g of calcium per day into the calcium pool. As a consequence of this sudden calcium requirement, nearly all cows experience some degree of hypocalcaemia during the

first day after calving as the intestine and bone adapt to the calcium demands within 48 hours of lactation. In some cows, the mammary drain of calcium causes extracellular and plasma calcium concentration to decline to levels that disrupt neuro muscular function, resulting in the clinical syndrome of milk fever (Horst *et al.*, 1997a).

In relapsing cases of milk fever kidneys are temporarily refractory to PTH stimulation i.e., even when there is normal level of PTH, $1,25(OH)_2$ Vit D production from kidney will not be initiated. Aged cows are under greatest risk of developing milk fever because of decreased number of receptors for $1,25\ (OH)_2$ Vit D and decreased response to Vit D_3 at the receptors located in the bone (Goff *et al.*, 1996).

This might be one of the reasons why, even after previous treatments with calcium, the hypocalcaemia persisted especially in aged cows.

Only administration of calcium in the extracellular fluids will restore the responsiveness to PTH, trigger bone resorption and correct the hypocalcaemia (Rosol and Capen, 1997). Considering all these factors it is imperative to give adequate calcium along with Vit D_3 to treat milk fever complicated downer cows.

This is confirmed by the fact that after treatment the mean value elevated from 6.09 to 9.1 mg/dL in recovered cows.

5.5.2 Serum Phosphorus

The mean value of serum phosphorus recorded (4.28 ± 0.12 mg%) in this study for normal animals agrees with the mean value recorded by Mc Adam and O'Dell (1982).

There was statistically significant ($P<0.05$) reduction in the mean value of (3.64 ± 0.19 mg%) phosphorus levels in downer cows than normal healthy cows. This low level of phosphorus obtained in downer cows in this study corroborates with the reports of Curtis *et al.* (1970), Allen and Davies (1981), Catherine (1994),

and Gerloff and Swenson (1996). In hypocalcaemic crossbred cows Pandey and Parai (1988) and Dasan and Divya (2001) have recorded hypophosphataemia.

During hypocalcaemia, in order to maintain calcium phosphorus ratio increased activity of parathormone lowers plasma inorganic phosphorus either by increasing renal phosphate excretion or increasing the salivary secretion of phosphate (Daniel and Moodie (1979).

Also prolonged duration of recumbency and other clinical manifestations and/or excessive fear and excitement response during the onset of hypocalcaemia might result in a marked reduction of energy rich phosphate complexes within muscle cells . (Daniel and Moodie, 1979).

After the treatment the mean value of phosphorus increased from 3.6 ± 0.19 mg% to 4.41 ± 0.16 mg%.

5.5.3 Serum Magnesium

Mean value of magnesium (2.2 ± 0.07 mg%) in healthy control animals recorded agrees with the mean values of normal cows reported by Mylrea and Bayfield (1968), Payne *et al.* (1974), Pyne and Maitra (1981) and Sahay *et al.* (2002).

Mean value of magnesium was found to be with in the normal level as that of control cows. Pandey and Parai (1988) also could not record a change from the normal mean value of magnesium in downer cows. Catherine (1994) also observed a non significant difference in the values of serum magnesium between the healthy and downer cows as there are some form of magnesium reserve in animals which could be greater in cows more than six months pregnant than in non pregnant cows. This is probably the reason for maintenance of normal magnesium level in downer cows even though the appetite and food consumption were reduced.

Hypomagnesemia occur in lactating animals around the period of peak milk production. The occurrence is related to colder climate, high concentration of potassium in soil in which the pasture will be deficient in magnesium (Radostitis *et al.*, 2000). But in the present study cows were not at the peak milk production and soil K/(Ca+Mg) ratio would not be disturbed as most cases have been recorded from coastal areas and during the summer season (Radostitis *et al.*, 2000).

5.5.4 Serum Sodium

Mean value of plasma sodium (134.65 ± 1.27 mEq/L) obtained in healthy cows was comparable to the value for apparently healthy dairy cattle reported by Payne *et al.* (1974).

No significant difference was observed in the plasma sodium levels which agrees with findings of Julien *et al.* (1977) and Catherine (1994).

One reason why hyponatremia is not so valuable diagnostically is that a very high analytical accuracy is needed to measure the small changes in concentration that indicates abnormality (Payne, 1977).

5.5.5 Serum Potassium

Mean value of potassium (4.2 ± 0.08 mEq/L) obtained in healthy control cows was comparable to the values of reported by Mylrea and Bayfield (1968), Payne *et al.* (1974), Benjamin (1985) and Catherine (1994).

Mean value of potassium in downer cows did not differ significantly. This is in accordance with the findings of Catherine (1994) that there was no significant change in the mean value of potassium in downer cows when compared to normal healthy cows.

However hypokalaemia was reported in downer cows by some of the earlier workers (Littledike *et al.* (1969); Fenwick (1977); Allen and Davies (1981)

and Sielman *et al.* (1997). Serum potassium levels will be difficult to interpret because the levels of this electrolyte in serum is not necessarily the true indication of potassium deficiency since primarily it is an intracellular element (Radostitis *et al.*, 2000).

5.5.6 Serum Glucose

Mean value of glucose (43.97 ± 1.74 mg%) obtained in this study is comparable with reports of Payne *et al.* (1974), Pyne and Maitra (1981), and Panday and Parai (1988).

There was no significant difference in the mean value of serum glucose in downer cows from the normal cattle. This normal glucose level simulated the findings of Catherine (1994), Panday and Dwivedi (1988) and Radostitis *et al.* (2000).

Hyperglycaemia is usually seen associated with hypocalcaemia because calcium plays an important role for release of insulin to the circulation from golgi vesicle of cells (Lehninger, 1975). As all the cows in the present study had received calcium therapy at the onset of clinical signs, the effect of calcium to produce hyperglycaemia was not noticed.

5.6 URINE

Urine analysis of downer cows revealed traces to moderate ketonuria which is similar to the findings of Gangwar (1984), Catherine (1994) and Radostitis *et al.* (2000).

Ketonuria noticed in the present study could be due to secondary ketosis as a result of insufficient food intake during early lactation. Glycosuria was not present in any of the downer cows. Catherine (1994) reported that traces of sugar were found in urine of two downers, Wadhwa and Prasad (2002a) recorded glycosuria due to hyper glycaemia. Absence of glycosuria in this study might be due to the lack of hyperglycaemia.

Microscopic examination of urinary sediments revealed polygonal crystals of calcium carbonate and needle shaped or elongated crystals of calcium phoshate. Large polygonal epithelial cells of bladder mucosa were noticed, which is in accordance with report of Grinder (1979). Absence of abnormal crystals or inflammatory cells indicated absence of renal disease. Gangwar (1984) observed granular cast in urine of downer cows without any pathological significance.

5.7 EXAMINATION OF DUNG

On microscopical examination of dung, ova of amphistome could be detected in four cases. In all these cases cows had easy access to forage at water logged area. Intestinal parasitism like Amphistomiasais is reported to be one of the precipitating causes of downer cow syndrome (Amstuz, 1981).

Thus in these cases Amphistome infestation could be a predisposing factor to the occurrence of hypocalcaemia and oxyclozanide was used to prevent further worsening of the condition.

5.8 THERAPY

Number of etiological factors have been reported for the occurrence of downer cows. In this study major biochemical alterations noticed were the decrease in serum calcium and phosphorus levels even after previous treatment with calcium and inorganic phosphorus. Seventy five percentage of cows were in early lactation and were presented mostly after the unsuccessful treatment for the post parturient hypocalcaemia. This corroborates with the opinion of Fenwick (1969e) and Radostitis *et al.* (2000) that most commonly the downer cows is a complication of milk fever.

From the history of previous treatment it was revealed that only minimum quantity of calcium was administered intravenously for 2 to 5 occasions and animals were left alone thinking that they might get up within a reasonable time.

Some of them got up and again became recumbent. Whereas other cows remained recumbent even after repeated calcium therapy.

In the present study calcium, magnesium borogluconate was administered continuously at a stretch till the clinical response of calcium therapy was manifested by each animal. Response to calcium administration during therapy was manifested in the form of belching, smacking muscle tremors, particularly of the flanks and whole body, shivering improvement in the amplitude of pulse, increase of heart sound, urination etc. Inorganic phosphorus preparation up to 10 to 20 ml was also administered intravenously as hypophosphataemia was noticed in 4 animals and for other cows to avoid the occurrence of the same condition. Vitamin D_3 at the rate of 2.5 lakh IU was administered intramuscularly.

Calcium therapy does not materially alter the basic course of hypocalcaemia, but rather corrects some biochemical, neuromuscular and membrane transport defects occurring secondarily to hypocalcaemia (Littledike *et al.*, 1969). Even though it is said that 8 g calcium is superior to 6 g and 12 g superior to 8 g (Mullen, 1977b) it would be better to give sufficient quantity than concentration. Also Mullen (1977b) opined that therapy with solution combining magnesium and phosphorus with calcium should be more effective in preventing the relapses than calcium alone.

In the present study serum calcium and phosphorus levels of all the animals were found to be decreased to 6.1 ± 0.33 mg% and 3.64 ± 0.19 mg% respectively even after two to five intravenous calcium therapies. From this it could be presumed that the intravenous calcium already administered was not sufficient enough to maintain the serum calcium level or the calcium homeostasis mechanism of the downer animals were not triggered to maintain the serum calcium levels in the subsequent days. That could be the reason for those animal that showed transient recovery immediately after calcium therapy by field veterinarians and were subsequently recumbent. In the present study when 900 ml (16 g) of calcium, magnesium borogluconate was administered intravenously at a

stretch until the clinical response was manifested by the animal. Inorganic phosphorus was given intravenously or intramuscularly (10 to 20 ml) followed by intramuscular administration of Vit D_3 (2.5 lakh IU). This would have helped to maintain serum calcium to optimum level as Vit D_3 would have triggered homeostasis resulting in normal serum calcium level. It is a fact that nearly all cows experience some degree of hypocalcaemia during first days after calving. To maintain normal plasma calcium bone mobilisation is stimulated by a concerted effort of PTH and 1,25 $(OH)_2D$ but intestinal calcium absorption is controlled by 1,25 $(OH)_2D$ vitamin alone. The adaptation process begins with dramatic increase in the plasma concentration of PTH and 1,25 $(OH)_2$ D vitamin at the onset of hypocalcaemia. It was evident from the fact that after correction of calcium homoeostasis by administration of Vit D_3 and phosphorus, serum calcium and phosphorus level increased to 9.24 ± 0.20 mg% and 4.41 ± 0.16 mg% respectively after recovery.

The therapy with polypharmacy solutions which contain calcium, phosphorus and magnesium were administered in the present study because earlier workers (Fenwick, 1969; Allen and Davies, 1981; Andrews, 1986; and Barlet and Davicco, 1992) had reported downer cows associated with hypocalcaemia, hypophosphtaemia and hypomagnesaemia .

In concurrent hypocalcaemia the Vit D_3 production will be insufficient. Usually Vit D_3 is used for prevention of milk fever. However in recurrent hypocalcaemia it was used for treatment also. This will help in easy absorption of calcium, magnesium and phosphorus (Payne, 1977; Schneider *et al.*, 1985 and Sturen, 1985).

Multivitamin preparation were used to treat downer cows by the earlier authors (Panday and Parai (1988), Veena *et al.* (2003) and Wadhwa and Prasad (2004). There is a practice of epidiural multivitamin injection to treat downer cows among field veterinarians. This was authenticated by earlier workers also (Veena *et al.*, 2003). However in the present study multivitamin preparation was

administered intramuscularly only. This was done to avoid introduction of infection during epidiural route.

Anti-inflammatory analgesic like phenylbutazone salicylate combination and oxicam like NSAID were used for six cases each. The better responses i.e., unassisted attempts to rise, more frequency of attempts were noticed mainly for cows that were administered with phenyl butazone salicylate combinations. However as number of other drugs were administered it could not ascertain that phenyl butazone salicylate combination was efficacious than oxicam like NSAID. Earlier workers also tried analgesics like Esgipyrine and Diclofenac sodium in downer cows (Prasannakumar *et al.*, 1992 and Veena *et al.*, 2003).

A good managemental care was given to cows that were recumbent for more days. The animal was turned regularly from side to side which helped to prevent decubital sores as advised by Allen and Davies (1981).

Nothing is more likely to discourage an animal from rising than slipping on the floors (Allen and Davies, 1981). In this study all the cows were on non slippery floors and was given good nursing by frequently turning of the sides and hot fomentation which helped to a greater extent for a faster recovery.

Four cows were having sand as bedding, since the owners premises were near to the sea shore. It could be seen that bruises were less when sand bedding was used (Cox and Marion, 1992). Unconventional slings using gunny bags were used in four cases by downer. The recovery were uneventful in these cases.

Unfortunately at present there is no prognostic criteria which would give a reliable answer within two days, eventhough muscle enzymes are prognostic indicators (Cox *et al.*, 1982; Andrews, 1986; Prasad *et al.*, 1988 and Wadhwa and Prasad (2002a).

A better prognosis can presumably made with alert nature, response to therapy like, increased frequency of the unassisted attempts to rise and normal

physiological functions. The prognosis would be bad in non alert downer rather than alert downers. Poor response and death in non alert downer cows were reported by Fenwick *et al.* (1986) and Wadhwa and Prasad (2002).

In the present study there was a recovery rate of 58% and recovered animals were on feet within 5-12 hours after the last treatment. A 58% recovery is comparable with recovery percentage of Wadhwa and Prasad (2002) which was 51%.

To conclude, downer cows which were high producers occurred mostly during the period from September to May. Seventy five percentage of the downer cows were within first week of lactation. Hypocalcaemia and hypophosphotaemia were the biochemical alterations recorded during the analysis. Heavy parasitism and insufficient amount of feed received during pregnancy predisposed the cows to the downer condition.

Sufficient quantity of intravenous calcium 450-900 ml received at a time, intravenous or intramuscular administration of 10-20 ml of inorganic phosphorus and Vit D_3 @ 2.5 lakhs IU would help to retain the normal calcium and phosphorus ratio. Alert nature itself can be considered as a good prognostic sign.

Summary

6. SUMMARY

Downer condition is characterised by recumbency of the cows more than 24 hours without any other clinical signs. It results from metabolic alterations, toxaemia, musculoskeletal or nerve injuries, exposure to adverse climatic condition and or malnutrition. In the present study twelve cows, which were recumbent even after previous calcium therapy, formed the materials for the study. The animals were subjected to detailed clinical examination and all parameters under study such as signalment, history, physical examination, haematology, serum biochemistry, adoption of suitable therapy and treatment response were carried out. Twelve apparently healthy cattle in early lactation maintained under identical conditions were selected as control animals for studying normal haematogical and biochemical profile.

Out of twelve downer cows, highest incidence were in cross bred Jersey cows. Highest percentage of cows (41%) was aged between 4 and 8 and there was only one heifer. Both periparturient downer cows at eight months of pregnancy and post parturient downer cows (75%) with in the first week of lactation was present. Two cows (16%) were in mid lactation. All the cows were average producers with the exception of one (21 L) and parity of twelve cows ranged with in first and fifth, with the highest percentage (50%) with in third and fifth.

All the animals were being fed with compounded concentrate ration. One cow, which was in the eighth month of pregnancy, the feed was drastically reduced all of a sudden, to stop milk production. There was no history of exposure to adverse climatic condition or history of fall before the visit in any of the cows. These cows were maintained on insufficient quantum of feed ie quantity of feed was not proportional to the production. They were reared under semi intensive system and were on concrete floors. None of the cows had any systemic affection.

All the cows were alert downers and were characterised by sternal recumbency. All physiological parameters were within normal range. Rumination, feed in take, defecation and urination were normal. Three cows (25%) exhibited creeping or crawling which was typical to alert downers. One cow developed mastitis as a complication during the recumbency and bruises over elbow, hock and stifle were common to all downer cows.

Clinical pathological studies revealed a statistically significant increase in total leucocyte count, neutrophils and relatively significant decrease in lymphocytes, which could be subsequent to severe traumatic injury and inflammation over, elbow joint, hock joint and tuber coxae.

The reduction in the mean value of serum calcium (6.1± 0.33 mg/dL) of downer cows was statistically highly significant other biochemical changes was hypophosphatemia (3.64 ± 0.19 mg%) and reduction calcium : phosphorus ratio.

The therapeutic agent used for downer cows in the present study were calcium magnesium borogluconate at the dose rate of 450-900 ml intravenously, inorganic phosphorus-40% (10–20 ml) either intravenously or intramuscularly and vitamin AD_3E combination having Vitamin D_3 @ 2.5 lakh IU intramuscularly, multivitamin preparation 10 ml, intramuscularly to all animals. In addition supportive treatment with non steroidal anti inflammatory analgesic drugs of meloxicam group (@ 0.5 mg/kg BW) 30ml intravenously or salicylate phenylbutazone combination (@ 4 mg /kg BW) 15ml intravenously or intramuscularly (six cases each) were administered. Dextrose 25% - 500 ml was also administered intravenously for clinically weak animals.

The dosage of each drug, frequency of medication, and course of treatment were adopted based on clinical picture, biochemical profile and response of animal during medication. Specific therapy with calcium magnesium borogluconate was initiated only after analysis of the biochemical status of animals. The one with mastitis was concurrently treated by parenteral administration of Amoxicillin and Cloxacillin 3g @ 10 mg/kg body weight for

five days and those with Amphistome infestation was administered Oxyclosanide 3 g bolus @ 10 mg/ kg body weight orally.

Two cows were treated for four days and another for 5 days, 2 cows for three days and 7 cows for two days. Of these seven cows recovered and was seen on their feet with in 5-12 hours of last treatment. Five cows were either slaughtered due to severe traumatic injuries or the owners discontinued the treatment.

Sufficient quantity of calcium (450-900 ml) was administered intravenously until the clinical signs of response were noticed. Administration of Vitamin D_3 @ 2.5 lakh IU could be a better agent in helping to maintain the calcium phosphorus homeostasis. After the treatment when the animal responded to calcium therapy, normal serum calcium phosphorus ratio was attained. Cows made unassisted attempts to rise and turned their side more frequently when phenyl butazone salicylate combination was used as anti inflammatory analgesic agent.

The present study can be concluded as

i) Hypocalcaemia and hypophosphataemia have been seen associated with downer cows. Heavy intestinal parasitism like Amphistomiasis and insufficient quantum of feed provided during late gestation and early lactation would have been the predisposing factors.

ii) Recumbency more than 12 hours even after repeated intravenous calcium therapy advocates, therapy with sufficient quantity of calcium, phosphorus, magnesium and Vit D_3 to correct the calcium homoeostasis.

iii) Alert nature can be the best prognostic criteria.

iv) After correcting the calcium homoeostasis the analgesics like phenyl butazone will help in faster recovery.

References

REFERENCES

Allen, W.M. and Davies, D.C. 1981. Milk fever, hypomagnesemia and the downer cow syndrome. *Br. Vet. J.* 137: 435-441

Amstuz, H.E. 1981. Downer cows. *Mod. Vet. Pract.* 62: 299-230

Andrews, T. 1986. The 'downer cow'. *In Pract.* 8: 187-189

Barlet, J.P. and Davicco, M.J. 1992. 1α hydroxy cholecalciferol for the treatment of the downer cows. *J. Dairy Sci.* 75: 1253-1256

Barzanji, A.A.H. and Daniel, R.C.W.1988. The effect of hypocalcaemia on blood gas and acid base parameters in ruminants. *Br. Vet. J.* 144: 93-97

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

*Biggs, H.G., Moorehead, W.R. 1974. *Clin. Chem.* 20: 1458-1460. Cited in working manual of Agappe Calcium kit.

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

Bowen,J.M., Blackmon, D.M. and Heavner, J.E. 1970. Neuromuscular transmission and hypocalcaemic paresis in the cow. *Am. J. Vet. Res.* 31: 831-839

Braun, U., Salis, F., Siegwart, N. and Hassig, M. 2004. Slow intravenous infusion of calcium in cows with parturient paresis. *Vet. Rec.* 154: 336-338

Catherine, M. K.1994. Metabolic profile of 'downer cow' syndrome. MVSc. thesis, Kerala Agricultural University, Thrissur, p.101

Clark, R.G. 1974. Phosphorus deficiency in cattle on two farms in Canterbury. *N.Z. Vet. J.* 22:14-16

Clark, R.G., Henderson, H.V., Hoggard, G.K., Ellison, R.S. and Young, B.J. 1987. The ability of biochemical and haematological tests to predict recovery in periparturient recumbent cows. *N.Z. Vet. J.* 35: 126-133

Correa, M.T., Erb, H.N. and Scarlet, J.M. 1993. Risk factors for downer cow syndrome. *J. Dairy Sci.* 76: 3460-3463

Cox, V.S. 1982. Pathogenesis of downer cow syndrome. *Vet. Rec.* 111: 76-79

Cox, V.S. and Marrion, R.S.1992. Sand as a bedding for a downer cow. *Vet. Rec.* 130: 74-75.

*Cox, V.S., Marsh, W.E., Steuernagel, G.R., Fletcher, T.F. and Onapito, J.S. 1986. Downer cow occurrence in Minnesota dairy herds. *Prev. Vet. Med.* 4: 249-251

Cox, V.S., Mc Grath, C.J. and Jorgenson, S.E. 1982. The role of pressure damage in pathogenesis of downer cow syndrome. *Am. J. Vet. Res.* 43: 26-31

Curtis, R.A, Cote, J.F. and Willoughly, R.A.1970. The downer cow syndrome. A complication, not a disease. *Mod. Vet. Pract.* 51: 25-28

Daniel, R.C.W.1979. Changes in Bromosulphalein fractional clearance and plasma volume in induced hypocalcaemia in cows. *Br. Vet. J.* 135: 25-29

Daniel, R.C.W. and Moodie, E.W.1978. The effect of induced hypocalcaemia on the cardiac out put and blood pressure of dairy cattle. *Res. Vet. Sci.* 24: 380-382

Daniel, R.C.W. and Moodie, E.W. 1979. Induced hypocalcaemia in cows and sheep. Changes in plasma inorganic phosphorus levels. *Br. Vet. J.* 135: 440-451

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

Erb, H.N. and Grohn, Y.T. 1988. Epidemiology of metabolic disorders in the periparturient dairy cow. *J. Dairy Sci.* 71: 2557-2571

Fenwick, D.C.1969a. Parturient paresis (Milk Fever) of cows. The response to treatment and the effect of the duration of symptoms. *Aust. Vet. J.* 45: 111-113

Fenwick, D.C.1969b. Parturient paresis (Milk Fever) of cows. The quantity of calcium solution used in treatment and changes in heart beat characteristics. *Aust. Vet. J.* 45: 114-117

Fenwick, D.C.1969c. Parturient paresis (Milk Fever) of cows. Limb dysfunctions. *Aust. Vet. J.* 45: 118-122

Fenwick, D.C.1969d. Parturient paresis (Milk Fever) of cows: Rectal temperature. *Aust. Vet. J.* 45: 123-125

Fenwick, D.C.1969e. The downer cow syndrome. *Aust. Vet. J.* 45: 184-188

Fenwick, D.C. 1977. Non alert "downer" cows. *Aust. Vet. Pract.* 7: 44-46

Fenwick, D.C.1986. The relationship between certain blood cations in cows with milk fever and both state of consciousness and the position of cows when attended. *Aust.Vet. J.* 65: 374-375

Fenwick, D.C. 1989. The association between pupil sizes and blood constituents in cows with parturient paresis. *Can. Vet. J.* 30: 426 –429

Fenwick, D.C., Kelly, W.R. and Daniel, R.C.W. 1986. Definition of a non alert downer cow syndrome and some case histories. *Vet. Rec.*118: 124-128

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

George, L.W., Smith, B.P. and Lewis, G.W. 2002. Peripheral Nerve Disorders. *Large Animal Internal Medicine* (ed. Smith, B.P.). Third edition. Mosby, Missouri, pp. 1016-1017

Gerloff, B.J. and Swenson, E.P. 1996. Acute recumbency and marginal phosphorus deficiency in dairy cattle. *J. Am. Vet. Med. Assoc.* 208: 716-719

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

Goff, J.P. and Horst, R.L. 1998. Use of hydrochloric acid as a source of anions for prevention of milk fever. *J. Dairy Sci.* 81: 2874-2880

Goff, J.P., Horst, R.L., Jardon, P.W., Borell, C. and Wedam, J. 1996. Field trials of an oral calcium propionate to prevent milk fever in periparturient dairy cows. *J. Dairy Sci.* 79: 378-383

Goff, J.P., Horst, R.L., Mueller, F.J., Miller, J.K., Kiess, G.A. and Dowlen, H.H. 1991. Addition of chloride to a prepartal diet high in cations increases 1,25 dihydroxyvitamin D response to hypocalcaemia preventing milk fever. *J. Dairy Sci.* 74: 3863-3871

Greatorex, J.C. 1957. Observations on the haematology of calves and various breeds of adult dairy cattle. *Br. Vet. J* .113: 29-33

Grinder, H.D. 1979. Microscopic examination of urinary sediment. *Clinical Examination of Cattle.* (eds. Rosenberger, G., Dirksen, G., Grunert, E., Krause, D. and Stober, M.). Second edition. Verlag Paul Parey, Berlin. pp. 78-79

Hansen, T.T. and Jorgensen, R.T. 2001. Prevention of parturient paresis and subclinical hypocalcaemia in dairy cows by zeolite A administration in the dry period. *J. Dairy Sci.* 84: 691-693

Horst, R.L., Goff, J.P., Reinhardt, T.A. and Buxton, D.R. 1997a. Physiological changes at parturition and their relationship to metabolic diseases. *J. Dairy Sci.* 80: 1260 –1268

Horst, R.L., Goff, J.P., Reinhardt, T.A. and Buxton, D.R. 1997b. Strategies for preventing milk fever in dairy cattle. *J. Dairy Sci.* 80: 1269-1280

Hove, K. and Kristeinsen, T. 1982. Prevention of parturient hypocalcaemia: effect of a single oral dose of 1,25 dihydroxyvitamin D_3. *J. Dairy Sci.* 65: 1934-1940

Hulland, T.J. 1993. Muscle and Tendon. *Pathology of Domestic Animals* (eds. Jubb, K.V.F., Kennedy, P.C. and Palmer, N.). Fourth edition. Academic Press Inc, London, pp. 225-227

Johnson, B.L.1963. Observation on the creeping downer cow. *Mod. Vet . Pract.* 68: 217-220

*Julien, W.E., Conrad, H.R. and Redman, D.R. 1977. Influence of dietary protein on susceptibility to alert downer cow syndrome. *J. Dairy Sci.* 60: 210-215

Kavitha, S., Thirunavukarasu, P.S., Vijayakumar, G., Srinivasan, S.R. and Subramanian, M. 2002. A retrospective study on the prevalence of downer cow syndrome in dairy animals of Namakkal district. *Indian J. Vet. Med.* 22: 64-65

Kelly, W.R. 1974. *Veterinary Clinical Diagnosis.* Second edition. Baillire Tindall, London, p.374

Kramer, J.W. 2000. Normal Haematology of Cattle, Sheep and Goats. *Schalm's Veterinary Haematology* (eds. Feldman, B.F., Zinkl, J.G. and Jain, N.C.). Fifth edition. Lippincott Williams and Wilkins, Philadelphia, pp. 1076-1084

*Kronfeld , D.S. 1974. Alert downer cow syndrome. *Mod. Vet. Pract.* 55: 79-81

Lehninger, A.L. 1975. *Biochemistry. The Molecular Basis of Cell Structure and Function.* Second edition. Kalyani Publisher, New Delhi, p.463

Lincoln, S.D. and Lane, V.M. 1990. Serum ionised calcium concentration in clinically normal dairy cattle, and changes associated with calcium abnormalities. *J. Am. Vet. Med. Assoc.*197: 1471-1474

Littledike, E.T., Whipp, S.C. and Schroder, L. 1969. Studies on parturient paresis. *J. Am. Vet. Med. Assoc.*155 : 1955-1963

*Lopez, R.S., Kohayagawa, A., Takahiva, R.K. and Gentile, L.B. 2001. Serum electrolyte, protein and haemogram in clinically healthy cows and those suffering from downer cow syndrome. *Vet. Rec.* 112: 59-63

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

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

Mullen, P.A. 1975. Clinical and biochemical responses to the treatment of milk fever. *Vet. Rec.* 97: 87-92

Mullen, P.A.1977a. Milk fever: influence of treatment before the clinicians visit. *Vet. Rec.*101: 366-367

Mullen, P.A.1977b. Milk fever: A case against polypharmacy solutions. *Vet. Rec.*101: 405-407

*Murphy, S.A. and Power, E.P. 1995. Poisoning of dairy cows by a high nitrate concentration in Italian rye grass. *Irish Vet. J.* 48: 395-397

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

Narayana, K., Setty, D.R.L., Sastry, K.N.V. and Thandaveshwar, M.G. 1977. Downer cow syndrome. *Indian Vet. J.* 54: 148-153

Oser, B.L. 1976. *Hawk's Physiological Chemistry*. Fourteenth edition. Tata Mc Graw-Hill Publishing Company Ltd., New Delhi, p.1141

Pandey, N.N. and Dwivedi, S.K. 1988. Biomedical response to experimental hypocalcaemia in calves. *Indian J .Vet. Med.* 8: 96-99

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

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. 1977. *Metabolic Diseases in Farm Animals*. William Heinemann Medical Books Ltd, London, p.206

Payney, 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 tests on 191 herds in the B.V.A./ADAS. *Br. Vet. J.* 130: 34-43

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

Prasad, B., Rathor, S.S. and Roy, K.S. 1988. Histioenzymopathies in downer cows. *Indian J. Vet. Med.* 8: 79 - 84

Prasannakumar, S., Honnappa, T.G., Pampapathi, G., Setty, D.R.L. and Jayadevappa, S.M. 1992. Successful treatment of downer cow syndrome in a cross bred cow-A case report. *Indian Vet. J.* 69: 940-941

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

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

Rosol, T.J. and Capen, C.C. 1997. Calcium regulating hormones and diseases of abnormal mineral (calcium, phosphorus, 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

Sachs, M., Bar, M., Cohen, R., Mazur, Y. and Hurtwitz, S. 1977. Use of 1α hydroxy cholecalceferol in the prevention of bovine parturient paresis. *Am. J. Vet. Res.* 38: 2039-2041

Sahay, M., Sinha, B.P. and Samantray, S. 2002. Biochemical changes in clinically affected gangrenous syndrome in bovine. *Indian J. Vet. Med.* 22: 107-108

Sastry, G.A. 1985. *Veterinary Clinical Pathology.* Third edition. CBS Publishers and Distributors, Delhi, p.105

Sathishkumar, K. and Rao, D.S.T. 2000. Therapeutic studies on experimental hypocalcaemia in calves. *Indian J. Vet. Med.* 20: 21-22

Schalm, O.W., Carrol, E.J. and Jain, N.C. 1971. *Bovine Mastitis*. Lea and Febiger, Philadelphia, p.360

Scheneider ,K.M., Parkinson, G. B., Houston, J.C. and Leaver, D.D. 1985. 1,25, dihydroxyvitamin D_3 increases plasma magnesium and calcium in sheep fed liquid diets low in calcium and magnesium. *Aust. Vet. J.* 62: *1985-1989*

Shorthose, H.R. and Shaw, F.D. 1977. Plasma constituents of downer sheep slaughtered at an abattoir. *Aust. Vet. J.* 53: 330-333

Sielman, E.S., Sweeny, R.W., Whitlock, R.H. and Reams, R.Y. 1997. Hypokalemic syndrome in dairy cows. *J. Am. Vet. Med. Assoc.* 210: 240-243

Singh, B.P. and Gupta, R.P.1987. Parturient paresis in buffaloes and its treatment. *Indian J. Vet. Med.* 7: 120

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. 1980. *Statistical Methods*. Ninth edition. Oxford IBH Publishing company, Calcutta, p.584

Sturen, M. 1985. The effect of intramuscularly administered Vitamin D_3 on serum vitamin D metabolites and electrolytes in vitamin D_3 deficient dairy cows. B*r. Vet. J.* 126: 179-190

*Tausskey, H.H. and Schorr, E. 1953. *J. Bio. Chem.* 20: 1692-1693. Cited in working manual of Labkit for phosphorus.

Thornton, J.T. 1976. Methylene blue treatment for downer cows. *Mod. Vet. Pract.* 71: 1023

*Trinder, P. and Cain, A. 1969. *Biochem.* 6: 24. Cited in working manual of Agappe Glucose kit.

Tripathy, S.B. 1987. Nonparturient hypocalcaemic paresis in cows. *Indian J. Vet. Med.* 7: 138-139

Veena, P., Ravikumar, A. and Syamasundar, S. 2003. Downer cow syndrome in Jersey cross bred cows. *Indian Vet. J.* 80: 96-99

Vihan, V.S. and Rai, P.1984. Studies on biochemical and haematological changes in metabolic derangements of sheep and goat. *Indian J. Vet. Med.* 4: 9-12

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

Wadhwa, D.R. and Prasad, B. 2002b. Serum enzymes activities as prognostic indicators in downer cow. *Indian J. Vet. Med.* 22: 42-44

Wadhwa, D.R. and Prasad, B. 2002. Clinicotherapeutic observations of downer cow syndrome. *Indian Vet. J.* 79: 484-486

Wadhwa, D.R. and Prasad, B. 2004. Clinicotherapeutic studies on downer syndrome in buffaloes. *Indian Vet. J.* 81: 1143-11456

* Originals not consulted

Appendix

Annexure I

PROFORMA

Name and address of the owner with phone number :

Date :

Details of the animal

Breed Colour Age Parity

History of dam

Reproductive details

- Age at first calving
- Month of calving
- Sex of calving
- Reproductive problems if any
- History of milk fever

Production details

	Morning	Evening
• Milk yield (L)		
• Stage of lactation		

Feeding details

- Roughage
- Concentrates
- Unconventional feeding (Tamarind, bran flour waste etc.
- Changes of feed

Housing details

Floor

Date of onset of symptoms

Treatment (given before) and result

Cost of treatment

Previous history of any disease

OBSERVATIONS

Temp: Pulse: Resp: MM: BP:

Posture Sternal recumbency Lateral recumbency

1. Alert/non alert
   a) with convulsion
   b) without convulsion

2. Frog sitting posture

   Hind limb backwardly placed
   Hind limb forwardly placed

Faeces

Urine

Milk

Haematology

Biochemistry

Rumen liquor

CLINICO-THERAPEUTIC STUDIES ON DOWNER COWS

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Abstract of the thesis submitted in partial fulfilment of the requirement for the degree of

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ABSTRACT

The study entitled "Clinico-therapeutic studies on downer cows" was carried out in twelve cows, which did not respond to the treatment by local Veterinarians. Two of the cows presented at Veterinary Hospital, Mannuthy and other ten field cases from Trissur district formed the subjects for this study.

All the animals were subjected to detailed clinical examination and all parameters under study viz., signalment, history, physical examination haematology, serum biochemistry and treatment response were carried out. The data obtained were analysed statistically. Highest percentage of occurrence was seen in cross bred Jersey cows and in old age group. Seventy five percentage of cows were with in first week of lactation, eight percentage at eighth month of gestation and sixteen percentage in mid lactation. Occurrence was recorded during the period from May 2004 to May 2005.

All the cows were fed on a compounded concentrate ration and quantum of feed were not proportional to the milk production. They were maintained on concrete floors and had no history of a fall or exposure to adverse climatic condition.

All the cows were in sternal recumbency at the time of presentation and were alert without other clinical signs. Frequent attempts to rise and changing the sides by their own was common in most cows. All the physiological parameters were with in the normal range. Feed in take, urination, defecation and rumination were normal. Bruises over hock, elbow and tuber coxae were seen as common complications. One cow that developed mastitis and cows with Amphistome infestation were concurrently treated.

Statistically significant ($P<0.05$) increase in total leukocyte count and neutrophilia were recorded in cases with traumatic injuries and subsequent

inflammation over elbow joint, hock, tuber coxae and stifle. Mild lymphopenia was also seen.

Biochemical alterations included hypocalcaemia and hypophosphatemia and the calcium phosphorus ratio was found to be reduced to 1.69 before the treatment. The general line of treatment adopted for downer cows in the present study were administration of calcium magnesium borogluconate 450-900ml intravenously, inorganic phosphorus-40% (10 –20 ml) either intravenously or intramuscularly, vitamin AD_3E combination having Vitamin D_3 @ 2.5 lakh IU intramuscularly, and multivitamin preparation 10 ml intramuscularly to all animals. In addition supportive treatment with non steroidal anti inflammatory analgesic drugs of meloxicam group (@ 0.5 mg/kg BW) 30ml intravenously or salicylate phenylbutazone combination (@ 4 mg /kg BW) 15ml intravenously or intramuscularly (six cases each) were administered. Dextrose 25% was also administered intravenously for clinically weak animals. Dosage and frequency varied according to biochemical profile, clinical picture and response to therapy.

Out of twelve cows, seven cows recovered from downer state. Three cows were under treatment for 4 to 5 days, two cows for 3 days and seven cows for two days. Serum calcium, phosphorus ratio in the recovered animals returned to normal of 2.1: 1. After treatment five cases were either slaughtered due to severe traumatic injury or owners were unwilling to treat the animal.