Serum Mineral and Haematobiochemical Profile of Microfilariae Infected Cattle in India: Its Effects on Production and Therapy

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ABSTRACT : A survey was under taken of six district of Northern India viz. Bareilly, Pilibhit, Udham Singh Nagar, Nainital, Almora and Rampur. The age, breed, sex and physiological status recorded. A total number of 854 cattle examined out of which lactating (274 cases), non lactating (302 cases) heifers (128 cases), calves (82 cases) and adult male (68 cases) were examined. An incidence of 4.92 percent (42) of microfilarisis was recorded. The highest prevalence was observed in Rudrapur District of Udham Singh Nagar (33.33%, 4/12), followed by Lalkaun in Nanital District (21.74%, 10/46), Rampur (12.50%, 2/16), Bareilly (8.16%, 8/98) and Pilibhit (1.22%, 1/82). No infection was observed in Almora region. Amongst 854 cattle of different group incidence was highest in adult male (12.20%, 10/82), followed by non lactating (3.82%, 12/314) and lactating (2.70%, 2/74), (7.64%, 12/157) was found in Heifers. For haemeto-biochemical, serum minerals estimations and therapeutic study 32 animals suffering from filariasis and 18 healthy animals were taken. 16 animals were treated with ivermectin @200 μg/kg body weight. Effect of this disease on production has also been estimated for which body weight and milk production was observed. The main clinical manifestations observed were anaemia, loss of appetite, debility, oedematous swelling especially in the abdominal region, increased heart rate, and respiration rate. Haematological changes indicated decrease in hemoglobin, total erythrocyte count, packed cell volume, erthrocyte fragility and neutrophil, whereas there was significant increase in erythrocytes sedimentation rate (ESR), total leukocyte count (TLC), lymphocyte and eosinophils. Biochemical changes showed significant reduction in the values of serum albumin, A : G ratio, where as there was significant increase in blood glucose, blood urea nitrogen (BUN), globulin, total lipid, total cholesterol, phospholipids, serum bilirubin. Serum mineral profile also altered markedly, which indicate a significant decrease in Ca, Cu, Fe, Zn, and Mn with increase value of Na and Cl. There was no significant change in P and K values. Enzyme pattern in micro filaria infected animal indicated increased level of AST, ALT, alkaline phosphatase, ornithine carbamyl transferase, sorbitol dehydrogenase, glutamate dehydrogenase, isocitric dehydrogenase and lactate dehydrogenase. In blood gas values and acid/base balance, there was an increase in $PVCo_2$ and PVo_2. It has been observed that microfilaria infected cattle showed decrease in body weight and milk production. Animal treated with ivermectin showed the return of these above values toward normalcy. ***(Asian-Aust. J. Anim. Sci. 2002. Vol 15, No. 3 : 357-365)***

Key Words : Serum Mineral, Microfilariae, Cattle, Haematology, Biochemical, Production, Therapy, Milk Yield, Body Weight

INTRODUCTION

Over a considerable number of years, FAO has emphasized the magnitude of losses in livestock industry due to Filarasis. The disease is present in more than 100 countries of the world including India. In India this disease is confined to geographical areas, which are hot and humid. Tarai areas of India provide a congenial environment for the development of vector and favourable conditions for the development of parasite.

The filarial worms meet their nutrients requirement from the host tissues and fluids and *in situ* transfer of nutrients from the tissues to the body of parasites causes various deficiency symptoms including anaemia, depending on the worm load of the host (Sharma and Pachauri, 1982). Iron and proteins are the main constituents of haemoglobin and copper plays an important role in the catalysis of the synthesis of haemoglobin. Copper is also a component of the enzyme ceruploplasmin (Duncan and Prasse, 1977). The role of zinc and ceruploplasmin in vitamin "A" metabolism is very important. The removal of these constituents from the blood and other body tissues of the host for the development of filarial worm may cause various degree of damage and deficiency of organic and inorganic nutrients (Sharma et al., 1985). The etiological agents causing micro filariasis in animals are infectious larvae of *Setaria, Onchocera, Parafilaria, Dirofilaria, Stephanofilaria, Dipetolonema* (Lec and Wang, 1991; Wahl et al., 1994) the species also have a zoonotic importance (Senthilvel and Pellai, 1999). Microfilariasis in cattle is mostly observed in the sub Himalayan Belt (Tarai region) of Uttar Pradesh. Sharma et al. (2000). Large numbers of animals encounter high level of filarial infections because the environmental condition of the Tarai region favours larval development in vector and their transmission in the host. Usually no apparent clinical symptoms are observed in animals, but dermatitis, sore ear and paralytic symptoms have been observed due to the microfilariae of the various filarial worms in animals (Kumar et al., 1989) and Siddiquie and Sharma (1997). In one study in Andhra Pradesh state in India a low (7.4%) prevalence of micro filariae in buffaloes has been reported (Venu et al., 2000). Estimation of economic loss in terms of production due to parasitism is

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not easy. Losses due to latent and clinical parasitism are considered to be higher than that caused by the microbial agents (Sharma et al., 1980).

The present investigation deals with the detailed alterations on hemato biochemical and serum mineral profiles in cattle naturally suffering from filariasis and its effect on the production capacity of the cattle. Efficacy of the drug ivermectin has been observed and its effect on the body weight and milk yield after successive period of therapy has been studied.

MATERIALS AND METHODS

A survey was under taken of six district of Northern India viz. Bareilly, Pilibhit, UdhamSingh Nagar, Nainital, Almora and Rampur. The age, breed, sex and physiological status viz. lactating (264 cases), non-lactating (302 cases) heifers (128 cases), calves (82 cases) and adult male (68 cases) of 854 cattle was recorded.

Blood samples were collected from jugular vein. Approximately 5 ml of blood was collected in sterile glass vials containing EDTA as anti coagulant. At the same time a set of 5 thin blood smears were prepared and stained with wright stain. For the morphological identification of the microfilaria, modified knott technique (Jackson, 1968) was used for killing and fixing of microfilariae on a glass slide and was examined at 10 and 43 magnifications under a compound microscope.

Observations were made on gait, posture, head carriage and body deformities. Examinations of oral and pharyngeal cavities were also made. Abnormalities, if any, in eyes, ears, lymphnodes, trachea and jugular pulse were evaluated. The lymph glands were palpated and heart and lung was ausculated carefully with stethoscope. Other clinical observations like rectal temperature, heart rate, respiration rate and general conditions were recorded.

For detail haemato-biochemical studies and serum mineral studies group of 18 healthy animals and 32 filaria infected animals were taken.

Examination of blood was carried out for differential leukocytes count (DLC), hemoglobin (Hb), red blood corpuscles (RBC), total leukocytes count (TLC), packed cell volume (PCV), mean corpuscles volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), erythrocyte fragility, total platelets count and clotting time was estimated as per method described by Jain (1986).

Approximately 10 ml blood was collected in a sterilized test tube with out any anticoagulant. The tube containing blood was kept at a room temperature without disturbing. After two hours clot was broken, the serum was separated centrifuged and stored in refrigerator at, -4°C in glass vial. All the biochemical estimations were made within three days of serum collection. Uncoagulated blood of the infected animals was used for the estimations of glucose as described by Folin and Wu (1920) and blood urea nitrogen as per Wootton (1964). Serum total protein and serum albumin were estimated as described by Wootton (1964) and Kaneko (1980), serum globulin were determined by the estimation of total protein and serum albumin, the value of latter was subtracted from the former to give the concentration of the globulin, A:G ratio was determined by dividing the percentage of albumin with percent of globulin in the serum. Estimation of serum total lipids, total cholesterol and total phospholipids was carried out by the method of Wootton (1964) and Kaneko (1980) and Taussky and Shorr (1953). Serum transaminases viz. aspartase amino transferase (AST) and alanine amino transaminases (ALT) were estimated colorimetrically according to the method of Reitman and Frankel (1957). Serum alkaline phosphatase, acid phosphatase, sorbitol dehydrogenase, ornithine carbamyl tranferase, lactic dehydrogenase, isocitric dehydrogenase, glutamic dehydrogenase and creatinine phosphokinase were estimated by the procedures described by Bergmeyer (1983).

The blood gas values viz. pH, P*V*CO_2 (the partial pressure or tension of CO_2 gas in venous blood), P*V*O_2 (the partial pressure or tension of O_2 gas in venous blood) and calculated parameter i.e. HCO_3 (actual bicarbonate concentration of hydrogen carbonate in the plasma). T*V* CO_2 (total carbon dioxide). The concentration of CO_2 (free +bound) in the plasma. BE (Actual base excess in the difference in concentration of strong base in the whole blood and in the same blood treated with strong acid or base). SBE (Standard Blood Excess *in vivo*) or SAT (Oxygen saturation in the amounted oxygen that is combined with Haemoglobin divided by the amount of oxygen that can be combined with haemoglobin. SBC (Standard bicarbonate in the concentration) of hydrogen carbonate in the plasma from blood which equilibrated with a gas with (P*V*O_2) were recorded.

Serum electrolyte viz. calcium (Ca), magnesium (Mg), phosphorus (P), sodium (Na), chlorine (Cl) and potassium (K), trace minerals in serum viz. copper, iron, zinc (Zn), and manganese (Mn) were estimated by atomic absorption spectrophotometer Model AAS 4141 ECIL-India. Atomic Absorption Spectrophotometer (AAS) Model AAS 4141 ECIL (Electronic Corporation of India Limited, Hyderabad -India) was used in present study. It is fully automatic and PC based. It uses a double beam with wavelength of 190-900 nm range, with a four-auto lamp turret. Acetylene flame mixed with air by an air compressor was used as a fuel. The instrument was calibrated using standard of requisite concentration and for every mineral at least, four standards of known concentration were used. Then the readings of unknown samples were recorded. Reading was

noted after five seconds of feeding the sample. The instrument was allowed sufficient water flush after each sample analysis.

Therapy: Five healthy and 9 parasite infected growing male cattle calves of 6-12 months of age, 5 healthy and 9 parasite-infected heifers of 12-24 months of age, were used for therapeutic studies. 8 healthy lactating and 6 parasite infected lactating cows were used for the therapeutic studies 8 infected lactating cattle were left untreated. Infected animals in the treatment group were administrated with Ivermectin @200 micro gm per kg body weight subcutaneously. Two doses at 10 days interval against the infection of the parasite were administered. No treatment was given in the healthy animals of the control group and infected control during experiment. Haematobiochemical and serum electrocyte observation were recorded on 0 day, 7 days, 14 days and 21 days after the first dose.

The observations were recorded for 60 days from the day of treatment and the body weight of the animals was recorded at 15 days intervals for estimating the change in body weight and rate of daily gain in the body weight. Milk yield was observed daily in the morning and evening for sixty days and average milk yield of different group were recorded to observe the effect of therapy on the milk yield.

Data were statistically analysed using *t*-test as per the method described by Snedecor and Cochran (1967).

RESULTS

An incidence of 4.92 percent (42/854) of microfilarisis was recorded. The highest prevalence was observed in Rudrapur District of Udham Singh Nagar (33.33%, 4/12), followed by Lalkaun in Nanital District (21.74%, 10/46), Rampur (12.50%, 2/16), Bareilly (8.16%, 8/98) and Pilibhit (1.22%, 1/82). No infection was observed in Almora region. Amongst 854 cattle of different group incidence was highest in adult male (12.20%, 10/82), followed by non lactating (3.82%, 12/314) and lactating (2.70%, 2/74), (7.64%, 12/157) was found in Heifers.

Through microscopic examination of blood film it was revealed that causative agent of Microfilarisis were larva of *Setaria cervi* measuring 155-290.50 microns in length and 5.50-7.85 microns in width. Nerve ring and the excretory pore were 43.60±2.25 μ and 60.5±3.8 μ away from anterior end respectively. Cattle affected with microfilariae were dull, weak, depressed, debilitated and showed variable appetite. There was an oedematous swelling and cutaneous lesions in three of the affected cattle. There was a slight increase in the body temperature in infected animals as compared healthy ones. There was also a significant increase in the heart rate and respiration rate.

The haematological changes are given in table 1, Hb values were lower in infected cattle as compared to normal healthy cattle this difference was statistically significant at 5% level. There was significant decrease ($p<0.05$) in the total erythrocytes count (TEC) of infected cattle as compared to healthy cattle. Average packed cell volume decreased in affected animals as compare to healthy control animals. This variation was statistically insignificant at 5% level. These values returned towards normalcy with in 14-

Table 1. Showing hematological changes in micro filarial infected cattle and values after treatment

Parameters	Group				
	A	B			
		(0 day)	(7 days)	(14 days)	(21 days)
Clotting time (Mts)	4.99±0.36	6.14±0.42	6.00±0.5	5.45±0.48	5.15±0.32
Hb (g %)	12.76±0.68	8.62±0.96*	9.01±1.01*	10.48±0.66*	11.44±0.58
TEC ($\times10^6$/cm)	6.28±0.33	4.23±0.56*	4.52±0.42*	5.58±0.22	5.92±0.36
PCV (%)	36.14±3.38	28.22±3.68	29.45±3.41	32.18±2.29	34.17±3.20
MCV (cubic-micro)	60.53±4.51	71.10±5.32	68.24±5.11	63.35±4.85	61.85±4.26
MCH (micro-micro gm)	20.57±0.82	18.38±0.73	18.57±0.68	19.03±1.04	19.36±0.99
MCHC (%)	30.36±2.88	26.98±2.86	27.24±2.41	28.15±2.28	29.30±2.63
ESR (m/h)	2.64±0.63	12.36±3.43	10.37±2.81	6.42±1.07	3.58±0.59
Total platetets counts ($\times10^3$/cm)	340.22±23.86	286.27±18.49*	305.24±20.85	321.25±20.8	331.72±21.97
Erythrocyte fragility (% NaCl conc.)	0.469±0.092	0.328±0.069*	0.335±0.675*	0.421±0.85	0.463±0.082
TLC ($\times10^3$ cm)	7.606±0.911	11.386±1.326*	10.482±1.476*	9.211±0.672*	8.916±0.872
Neutrohil (%)	33.65±5.26	18.12±3.22*	22.12±2.15*	26.35±3.35*	29.64±4.14
Lymphocytes (%)	58.11±5.18	72.20±5.36	68.53±3.15	64.33±4.43	62.48±5.87
Esinophils (%)	3.40±0.26	8.40±2.67	6.37±2.25	4.23±1.15	3.65±1.34
Monocytes (%)	4.58±0.59	3.07±0.52	3.15±0.41	3.58±0.55	3.97±0.46

* Significant at 5% level ($p<0.05$).

Group A-Normal healthy cattle (Control group) [Mean of the value on 0, 7, 14 and 21 days after start of therapy].

Group B-Infected cattle (Values on 0, 7, 14 and 21days after start of therapy).

21 days of treatment. Mean corpuscular volume was higher but statistically insignificant in filarial infected cattle. Erythrocyte sedimentation rate was higher in filarial infected cattle as compared to normal healthy cattle. This was statistically significant at 5% level. Total platelets count decreased in filarial positive cattle as compared to normal healthy ones. There was a decrease in fragility of the erythrocyte in infected cattle in comparison to those of healthy control ones and decrease was statistically significant at 5% level. It was observed that values returned towards normal level after treatment. The total leukocyte count increased in infected cattle in comparison to that of control ones. The difference between the infected and control value was statistically significant at 5% level. There was significant decrease ($p<0.05$) in the differential leukocyte count where neutrophil percentage was [18.12± 3.22] in infected cattle, where as in normal healthy animals it was 33.65±5.26. After treatment values showed normalcy. The increase in the number of lymphocytes was statistically insignificant where as the number of eosinophil increased in filarial infected animals as compared healthy non infected animals. The positive influences of therapy in diverting the parameter towards normalcy are given in table 1.

Biochemical changes are given in table 2. Blood glucose, blood urea nitrogen and serum bilirubin level showed a statistically significant increase ($p<0.05$) in the filarial infected cattle, as compared to healthy animals. The values returned towards those of healthy animals after treatment. (table 2). Creatinine value did not show any significant change.

The value of protein fraction in serum of filarial infected cattle showed that total protein did not have any major variation between filarial infected and healthy animals. There was a non-significant decrease in serum albumin value of infected animals in comparison of healthy control. The values of serum globulin showed an increase but it was statistically insignificant. But the albumin and globulin ratio of filarial infected cattle was 0.94 which was lower than that of healthy cattle (1.67). This difference in A:G ratio was significant at 5% level. The total lipid and cholesterol showed a marked increase in its level in infected animals as compared to the healthy animals. This increase was statistically significant ($p<0.05$). These value deviated towards normal level after treatment (table 2). The value of phospholipid showed a rise in filarial infected cattle but the increase was insignificant.

The values of serum minerals are given in table 3. There was notable rise in serum sodium level of infected cattle [158.63±7.23 meq/l] where as normal had value of 123.80± 5.99 meq/l. This rise was statistically significant ($p<0.05$). But there was non-significant decrease in the serum potassium level, of infected cattle. The value of serum chloride increased in the infected cattle as compared to the healthy groups. But there was a decrease in the values of serum calcium in infected animals. This decrease was statistically significant at 5% level. It was observed that values returned towards normalcy after treatment (table 3). The value of serum phosphorus had insignificant variation. There was a significant decrease ($p<0.05$) in the serum copper and iron level of the infected cattle, when compared with the healthy animals of the control group, but the values returned toward normal level after treatment (table 3). Decrease in the level of zinc was also observed in filarial infected cattle. The values of serum manganese was 5.02±1.08 in infected animals as compared to healthy animals (5.77±1.41).

Various changes occurred in the enzymatic activities are shown in table 4. In infected animals value of alkaline phosphatase was 17.36±4.49 units/100 ml, while in control group it was 4.89±1.96 units/100 ml. This increase in the

Table 2. Showing biochemical changes in micro filaria infected cattle and values after treatment

Parameters	Group				
	A	B			
		(0 day)	(7 days)	(14 days)	(21 days)
Blood glucose (mg/100 ml)	48.57±4.32	91.25±6.12*	78.80±5.12*	62.48±4.48	55.39±5.57
Blood urea nitrogen (mg/ 100 ml)	11.09±1.07	18.26±1.25*	15.48±1.18*	12.35±1.68	11.27±1.75
Total protein (g %)	6.34±0.32	6.39±0.42	6.37±0.37	6.32±0.41	6.38±0.46
Albumin (g %)	3.96±0.44	3.11±0.35	3.27±0.48	3.68±0.48	3.80±0.54
Globulin (g %)	2.38±0.25	3.28±0.28*	3.01±0.21	2.76±0.11	2.58±0.17
A:G Ratio	1.67	0.94*	1.01*	1.23*	1.47
Total lipids	401.02±16.05	525.17±19.88*	480.35±20.80*	440.24±17.85*	416.18±18.23
Total cholesterol	160.80±13.80	226.58±15.37*	201.48±12.32*	185.57±16.21*	171.32±14.23
Phospholipids	116.85±9.30	142.63±10.16	138.62±8.27	127.22±7.78	122.91±9.98
Serum bilirubin (mg %)	0.91±0.16	1.68±0.19*	1.37±0.22*	1.18±0.17	1.01±0.14

* Significant at 5% level ($p<0.05$).

Group A-Normal healthy cattle (control group) [Mean of the value on 0, 7, 14 and 21 days after start of therapy].

Group B-Infected cattle (values on 0, 7, 14 and 21days after start of therapy).

Table 3. Showing mineral status in serum of microfilaria infected cattle and values after treatment

Parameters	Group				
	A	B			
		(0 day)	(7 days)	(14 days)	(21 days)
Sodium (meq/l)	123.80±5.99	158.63±7.23*	142.43±5.58*	137.49±6.65*	130.38±6.28
Potassuim (meq/l)	5.17±0.73	4.18±0.47	4.56±0.34	4.85±0.66	5.03±0.57
Chloride (meq/l)	103.06±4.16	137.32±9.67*	122.42±5.57*	111.76±7.79*	105.02±8.92
Calcium (mg/100 μl)	11.20±0.53	8.03±0.61*	9.37±0.54*	10.37±0.62	11.07±0.60
Phosphorus (mg/100 ml)	5.73±0.39	6.04±0.64	5.95±0.60	5.86±0.57	5.78±0.59
Copper (μg/100 ml)	110.56±11.52	78.64±8.82*	88.54±7.75*	96.34±9.23	108.37±7.97
Iron (μg/100 ml)	312.17±28.41	263.43±19.62*	284.54±17.92*	292.46±17.44	301.33±18.72
Zinc (μg/100 ml)	201.66±21.67	161.93±17.52	177.92±14.48	186.88±16.22	196.84±18.20
Manganese (μg/100 ml)	5.77±1.41	5.02±1.08	5.11±1.75	5.21±1.28	5.26±1.18

* Significant at 5% level ($p<0.05$).

Group A-Normal healthy cattle (control group) [Mean of the value on 0, 7, 14 and 21 days after start of therapy].

Group B-Infected cattle (values on 0, 7, 14 and 21days after start of therapy).

level of alkaline phosphatase was significant at 1% level. But there was an insignificant rise in the level of acid phosphatase in infected group as compared in healthy groups. It was observed that the aspartate amino transferase level increased in filarial infected animals, which was statistically significant at 1% level when compared to healthy animals. The activity of alanine amino transferase in filarial infected cattle showed a marked increase at 5% level. The ornithine carbamyl transferase, sorbitol dehydrogenase, glutamate dehydrogenase and lactate dehydrogenase and isocitric dehydrogenase increased markedly in infected animals, in comparison to that of healthy animals, which was statistically significant. After treatment values moved towards normal mark (table 4). But there was insignificant increase in the value of creatinine phosphokinase in the infected animals.

The changes in physical values of the blood are given table 5. No significant difference was observed in the mean value of BE, SBE and SBC. There was insignificant increase in the partial pressure of O_2 gas (PVO_2) in venous blood and HCO_3 concentration in plasma. There was decrease in the value of oxygen that combined with haemoglobin (SAT) in the micro filarial infected animals as compared to healthy animals of control group. The variation in the pH values of infected and healthy animals was insignificant. There was a significant increase (at 5% level) in partial pressure of CO_2 gas in renal blood ($PVCO_2$) in infected animals as compared to healthy animals. Recovery following therapy in these parameters is indicated in table 5.

There was apparent difference in the rate of daily gain in the body weight of healthy and parasite infected animals of both the sexes (table 6). After drugging the animals of Group 3 showed abrupt increase in the body weight during the first fortnight in both the sexes. The animals of untreated infected control group 2 continued to gain at a reduced fluctuating rate. No significant difference was observed in the milk yield of healthy cattle between the average of two month, whereas it was about 19.13% higher in post treatment period after (60 days) in comparison to pretreatment period in infected cattle (table 7).

DISCUSSION

An overall incidence of 4.92% of micro filariasis has been observed in the present study. It is caused by *Setaria cervi* as also reported by Sharma et al. (2000). Higher prevalance in Tarai region of Northern India might be due to favourable agroclimatic conditions for breeding of vectors and subsequent transmission of disease to cattle Sharma et al. (2000) made similar observation. The slight rise in the body temperature may be due to the stress caused by the migration of microfilaria in the body of the host. Increase in heart rate and respiration rate is to compensate anaemic conditions and fulfil body oxygen requirement. Same findings were made by Sharma et al. (1985).

There was a marked decrease in the level of hemoglobin due to rapid disintegration of erythrocytes causing haemolysis. It was associated with significant decrease in the total erythrocytes count, decreased PCV, decreased erythrocytes fragility and platelets count. Increased ESR may be due to increase in the relative density of plasma caused by increase in the ionic content released from the degeneration of erythrocytes by microfilariae. The marked rise in the total leukocyte count (TLC) associated with eosinophilia and lymphocytosis was indicative of chronic parasistaemia. The rise in the eosinophil count was coupled with the micro filariaemia, which indicated body response of defence mechanism through the parasitical action of eosinophil. The degree of lymphocytosis occurred depend upon the intensity of parasitaemia. The results confirmed the earlier studies of Sharma (1985) and Goggin et al. (1997).

Table 4. Showing enzyme activity in microfilaria infected cattle and values after treatment

Parameters	Group				
	A	B			
		(0 day)	(7 days)	(14 days)	(21 days)
Aspartate amino transferase (R.F. units/ml)	42.83±6.40	96.13±8.49**	80.48±7.54**	62.46±7.44**	50.62±9.23
Alanine amino transferase (R.F. units/ml)	16.21±3.72	62.86±5.19*	40.46±6.64*	26.48±4.51*	19.36±6.21
Alkaline phosphatase (Kind and Kings unit/100 ml)	4.89±1.96	17.36±4.49**	11.23±3.11**	8.34±2.57**	5.66±1.17
Acid phosphatase (Gutman and Gutman 100 ml)	2.61±0.96	3.67±0.59	3.21±0.78	2.98±0.91	2.72±0.86
Ornithine carbamyl transferase (vol. activity unit/l)	4.50±1.90	8.632±1.19**	7.75±1.27**	5.21±1.41	4.68±1.87
Sorbitol dehydrogenase (vol. activity unit/l)	13.60±0.96	27.42±5.30	20.35±3.35	16.57±4.41	14.03±0.98
Glutamate dehydrogenase (units/l)	5.38±1.08	11.52±3.92	9.57±2.58	7.42±3.35	6.18±1.18
Isocitric dehydrogenase (units/l)	10.62±2.32	31.82±6.33	27.15±4.48	19.27±3.31	11.37±2.61
Lactate dehydrogenase (units/l)	158.50±9.67	211.35±12.83	190.38 ±10.43	178.41±7.79	167.36±8.76
Creatinine phosphokinase (units/l)	38.66±5.89	46.81±7.96	43.48±6.36	41.28±4.42	39.30±5.39

* Significant at 5% level ($p<0.05$).
** Significant at 1% level ($p<0.01$).
Group A-Normal healthy cattle (control group) [Mean of the value on 0, 7, 14 and 21 days after start of therapy].
Group B-Infected cattle (values on 0, 7, 14 and 21days after start of therapy) .

Table 5. Showing blood gas/acid base changes in microfilaria infected cattle and values after treatment

Parameters	Group				
	A	B			
		(0 day)	(7 days)	(14 days)	(21 days)
pH of blood	7.47±0.17	7.54±0.13	7.52±0.08	7.50±0.04	7.50±0.06
P*v* CO_2 (mm/hg)	38.25±3.40	52.76±3.38*	48.34±3.35*	44.57±3.21*	41.21±3.12
P*v* O_2 (mm/hg)	66.89±3.89	78.65±5.42	74.64±4.52	70.44±4.48	69.42±4.09
HCO_3 (meq/l)	24.38±3.45	27.76±5.53	26.72±3.39	26.11±4.09	25.31±4.07
T*V* CO_2 (mmol/l)	20.20±3.02	28.45±3.72	12.00±0.59	12.02±0.09	24.32±3.52
BE (mmol/l)	010.52±0.80	012.02±0.56	26.21±2.99	25.87±1.97	011.95±0.68
SBE (mmol/l)	011.23±0.68	013.02±0.76	12.98±0.78	12.96±0.67	012.93±0.82
SAT (%)	96.11±4.60	83.89±4.33	85.85±4.21	87.35±3.93	91.24±4.68
SBC (meq/l)	20.18±1.43	16.24±0.43	17.63±0.46	18.11±0.68	18.96±0.95

* Significant at 5% level ($p<0.05$).
** Significant at 1% level ($p<0.01$).
Group A-Normal healthy cattle (control group) [Mean of the value on 0, 7, 14 and 21 days after start of therapy].
Group B-Infected cattle (values on 0, 7, 14 and 21days after start of therapy) .

The significant rise in the blood glucose level indicates lower hepatic function due to which there was marked reduction in the conversion of glucose to glycogen by the liver. This is due to hepatitis caused by migratory microfilariae as also suggested by Duncan and Prasse (1977). These findings also corroborate with the findings of Sharma and Pachauri (1982) and Kumar and Sharma (1994). Increased blood urea nitrogen (BUN) may be due to increased protein catabolism or reduced renal perfusion resulting from inflamatory changes in the kidneys due to circulatory microfilaria. Kaneko (1980) suggested that increase in blood urea nitrogen occur due to the renal change.

Though the change in total serum protein was not significant, there was a significant decrease in the A:G ratio in the infected animals. Total serum proteins does not readily show a change in mild or moderate hepatic dysfunction, but in severe damage the change signify

Table 6. Showing mean daily gain in body weight after treatment

Group	Initial body weight (kg)	Mean daily gains in different days in gms			
		15	30	45	60
Calves of 6-12 months of age					
Healthy control (5)	176.88±12.82	417.64±32.90	423.30±12.35	431.00±33.80	447.64±44.22
Infected control (4)	135.28±11.68	212.39±23.85	199.92±33.52	241.02±16.75	227.66±32.76
Infected treated (5)	134.26±8.92	326.32±35.27	405.37±24.03	415.23±30.43	424.34±22.28
Heifers of 12-24 months of age					
Healthy control (5)	224.80±22.40	365.34±32.97	348.66±43.22	361.92±46.55	355.32±63.73
Infected control (4)	172.00±8.17	178.66±23.95	202.00±22.12	162.14±46.55	242.01±33.82
Infected treated (5)	168.80±6.56	318.66±44.18	312.00±23.85	325.34±34.59	338.63±22.36

Table 7. Showing mean gain in milk yield after treatment

Group	Milk yield in kgs				
	0 day	15 days	30 days	45 days	60 days
Normal healthy animals X=8	6.010	6.025	6.152	6.030	6.218
Infected untreated X=8	4.010 (% decrease)	3.980 (0.74%)	3.512 (12.41%)	3.422 (14.67%)	3.310 (17.46%)
Infected treated X=6	3.727 (% increase)	3.850 (3.30%)	3.950 (5.98%)	4.280 (14.83%)	4.440 (19.13%)

(X=Number of animals taken)

significant decreased albumin and fibrinogen synthesis that occur in liver. Such a variation in A:G ratio was reported in filariasis by Snyder et al. (1967), Ringheim (1974) and Sharma and Pachauri (1982). Increased values of AST and ALT are suggestive of necrotic degeneration of liver and myocardial cell caused by migratory microfilaria.

The decrease in serum albumin and increase in globulin fraction were evident in filaria infected animals since the albumin is synthesized in the liver it is believed that damage to liver cell might cause decrease in albumin production there by causing decrease in plasma albumin content. When plasma albumin decrease it also affects plasma colloids osmotic pressure, which may be a contributing, factor causing ascites in filariasis. Synder et al. (1967) and Ringheim (1974) have also recorded similar findings. In the present study there were no significant difference in alpha and beta fraction of globulin but the gamma globulin percentage was significantly higher in the serum infected animals. The increased level of gamma globulin appears to be a defensive mechanism. The present results agreed with the earlier reports of Dimopeculluos (1970), Barsanti et al. (1970) and Sharma and Pachauri (1982), who also recorded such increase in liver disorder. A significant increase was observed in the content of serum lipids and cholesterol. The fermentation products i.e. volatile fatty acids and higher fatty acid entering the portal circulation were utilized largely in the liver for glycogenesis, damage to liver parenchyma reduced the metabolism of fatty acids which entered the circulation and resulted in the increase of serum lipid. The results corroborated with those of Kronfeld (1965) and Leat et al. (1968) and Sharma and Pachauri (1982) who observed similar changes in the blood of cows and buffaloes. The increase in serum cholesterol level also indicated hepatic damage due to filarial infection which was in agreement with the earlier observation of Kaneko (1980).

There was significant increase in the serum sodium and chloride concentration in filaria infected cattle. The exact cause for such disturbance is not clearly known. It may be due to chronic hepatitis and increased capillary permeability, which favoured the development of diffused oedematous conditions. The loses of circulatory fluid resulted in the increased level of sodium and chloride and also of glucose as was also observed by Coles (1967) and Duncan and Prasse (1977). Little decrease in the content of potassium and calcium was due to the destruction of erythrocytes by microfilaria. This confirmed the earlier observation of Synder et al. (1967) and Sharma and Pachauri (1982). A significant decrease in the serum level of copper, Iron and zinc was observed in the animal infected with the microfilaria. Chronic anaemia caused by filarasis due to disintegration of the erythrocytes resulted in the significant loss of copper and iron. Similarly lower level of calcium effected the concentration of Zinc in the blood as observed by Coles (1967).

There was a significant increase in the activity of aspartase amino transferase, which indicated necrotic degeneration of liver cells caused by microfilariae. Similarly increased serum level of alanine amino transferase indicated the damage of hepatic parenchyma and

myocardial degeneration. Increase in the serum level of these enzyme was used as an indicator of hepatic damage. Similar changes were also reported in cattle, buffaloes, horses and dogs infected with micro filaria, and other infection causing hepatic damage by Rudolph et al. (1957) Dal Santo (1959), Malherbe (1960) and Sharma and Pachauri (1982). The increased level of serum alkaline phosphatase is influenced by several factors. But the present study indicated the hepatic damage and possibly the obstruction of bile ducts were main factors responsible for their increase. The results agreed with those of Drill and Ivy (1944) and Bloom (1957). Acid phosphatase however contributed no significant alterations in microfilaria infection. Since acid phosphatase enzyme is specific to the activity of prostate gland and there appear no involvement of the gland in microfilarial infection the enzyme level thus remain unaffected (Coles, 1967).

The mean pH of blood of filaria infected animals was slightly higher than those of healthy animals. This is probably due to some increase in the concentration of the bicarbonates. Anaemic condition and lower concentration of hemoglobin reduced the oxygen holding capacity of the blood and favoured increased retention of carbondioxide. However these value were highly variable and non-consistent. The mechanism of such imbalance could not be ascertained. Probably necrotic changes in the damaged cells of liver, heart, lung and other tissue in the migratory route of micro filaria produced higher concentration of carbon dioxide. Duncan and Prasse (1977) suggested the use of blood gas and acid base changes in diagnosis of some diseases in large and small animals.

The higher values of BE and SBE in infected animals are in accordance with increased value of carbon dioxide and bicarbonates. The lower SAT value is due to lower oxygen content of the blood due to anaemic condition caused by the infection. The blood gas values can be considered along with other haematological changes for the diagnosis of the filariasis in animals.

Fluctuations in the body weight gain of the parasite infected animals is generally due periodic increase and decrease in the worm load due to hatching of the eggs. The response of treatment on gain in body weight was similar to previous observation of Sharma et al. (1984). The sum of losses due to parasitism has been tentatively viewed to be about 1/4th of the production efficiency in Indian buffaloes (Ranjhan and Pathak, 1979). About 12.73% increase in milk production and 5.57% increase in body weight gain has been observed following treatment of infected buffaloes (Sharma et al., 1984). The positive influence of invermectin in treating microfilaria is well illustrated by the return of all the above discussed haematobiochemical parameters in infected group towards their control group values, of normalcy after 30 days of treatment with the first dose (table 1 to 7).

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