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Haemotobiochemical changes and management of post-partum fatty liver syndrome in Malabari goats#

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Abstract

The fatty liver syndrome was confirmed by haemotobiochemical and hepatic ultrasonographic investigations in 12 Malabari goats within six weeks after kidding. Animals were divided into two groups with six does each which received two different treatments. Ketonex bolus, which contains nicotinic acid, disodium hydrogen phosphate, and live yeast was given to the goats in Group I. The animals in Group II received syrup Bexoliv, which contains tricholine citrate, methionine, inositol, vitamin E, biotin, selenium, silymarin, iron, copper, and B12. In addition to this, animals of both groups received glycerin orally. On the fifteenth day of treatment, the animals were reevaluated. Six apparently healthy adult female goats within six weeks of kidding were selected as control. Before treatment, the neutrophil count showed a significant increase and lymphocyte and monocyte counts showed a decrease in the study groups. Blood beta-hydroxybutyrate (βHB), serum non-esterified free fatty acids (NEFA) and serum urea nitrogen (SUN) levels showed a significant increase before treatment and reduced after treatment among both groups. Hepatic ultrasonography showed hyperechogenicity of parenchyma before treatment when compared

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to after treatment. The animals showed improvement with both treatments.

Keywords: *Fatty liver, goats, haemato-biochemistry, ultrasonography, management*

Goats are predisposed to many gastrointestinal and metabolic disorders. Negative energy balance cause fatty liver during late pregnancy and this condition, "pregnancy toxaemia" is common in sheep and goats. However post-partum negative energy balance leads to the condition "lactational ketosis" with fatty infiltration of the liver frequently remaining undiagnosed and untreated. Poor nutritional levels during late gestation or early lactation result in a negative energy balance, which cause mobilization of body fat depots, particularly from the subcutaneous tissue, leading to increased free fatty acids in the bloodstream and subsequent deposition in the liver. Fatty liver syndrome results in reduced production efficiency and low body condition in goats (Gonenci *et al*., 2003; Yadav *et al*., 2018). This study aims to diagnose and treat fatty liver syndrome in post-partum Malabari goats

Materials and methods

In the present study, 64 multiparous Malabari does, kidded within six weeks and exhibited clinical signs like anorexia and poor body condition were selected. Out of these 12 goats with fatty liver syndrome were identified by evaluating the presence of beta-hydroxybutyrate (βHB) in milk by pen-side strip testing and by evaluating the ultrasonographic changes of the liver. Blood glucose (mg/dL) and blood βHB levels (mmol/L) were estimated by using the FreeStyle Libre reader supplied by Abbott Diabetes Care Ltd, UK. Blood samples were collected in EDTA vials and haematological parameters were estimated using an automatic haematology analyzer. Serum was separated and biochemical parameters were estimated using a semi-automatic biochemical analyzer (MISPAVIVA 2578-10/17) with commercially available biochemical kits. Serum aspartate aminotransferase (AST), total protein, albumin, total cholesterol, triglycerides, high-density lipoprotein (HDL cholesterol), low-density lipoprotein (LDL cholesterol), calcium, phosphorus, total bilirubin, direct bilirubin, serum urea nitrogen (SUN) and creatinine were estimated using standard techniques. Globulin value was calculated by subtracting the value of albumin from total protein. Very low-density lipoprotein (VLDL) was calculated by subtracting the values of HDL and LDL cholesterols from that of total cholesterol. Serum gamma-glutamyl transpeptidase (GGT) was done by using the Szasz methodology. The serum non-esterified free fatty acids (NEFA) was estimated using the colourimetric method described by Soares *et al*. (2018).

The Easi-Scan mobile real-time ultrasound scanner (IMV imaging, Imaging House, Phoenix Crescent Strathclyde Business Park Bellshill, ML4 3NJ Scotland, UK) with BCF Universal Goggles (BUG) and a convex transducer with a frequency range of 4.5 and 8.5 MHz were used in this study. The system-saved scanned images were uploaded to a personal computer for additional analysis.

The liver was imaged using an ultrasound scanning system while the animal was standing. The transducer was moved from dorsal to ventral through the right 6th to 12th intercostal spaces and the image was evaluated for the location of the liver, its parenchymal structure, and the hepatic vessel visibility (El-Khodery *et al*., 2011; Tharwat *et al*., 2012). The selected animals were grouped into two with six goats in each group. In Group I, treatment was done using an oral bolus containing Nicotinic acid, disodium hydrogen phosphate, and live yeast (Ketonex bolus from Zydus AH) one BID along with glycerin 50 mL BID orally for 14 days. In Group II, the animals were treated using the syrup containing tricholine citrate, methionine, inositol, vitamin E, biotin, selenium, silymarin, iron, copper, cobalt, and B12 (Bexoliv syrup by TTK Healthcare Ltd) 10 mL BID along with glycerin 50 mL BID orally for 14 days. The efficacy of the treatments was compared by evaluating the parameters under the study on the fifteenth day after the initiation of treatment. The parameters under the study were compared with that of six apparently healthy goats within six weeks after kidding who served as a control group. The data obtained were statistically analyzed (Snedecor and Cochran, 1994).

Table 1. Results of comparison of haematological parameters of the control group, group I and group II (pre-treatment)

Parameter	Control group (n=6) Mean ± SE	Group I (n=6) Mean ± SE	Group II (n=6) Mean ± SE	p value
WBC (10^3/cmm)	13.583 ± 3.219	16.583 ± 1.595	18.717 ± 1.509	0.298^{ns}
RBC (10^6/cmm)	16.232 ± 1.471	18.428 ± 1.173	14.993 ± 2.143	0.353^{ns}
HGB (g/dL)	9.583 ± 0.554	11.067 ± 0.897	8.633 ± 1.341	0.244^{ns}
VPRC (%)	33.6 ± 2.409	27.383 ± 1.027	26.25 ± 4.035	0.166^{ns}
MCV (fL)	18.267 ± 0.657	17.367 ± 1.093	17.2 ± 0.699	0.636^{ns}
MCH (pg)	5.967 ± 0.226	5.933 ± 0.236	5.567 ± 0.235	0.423^{ns}
MCHC (g/dL)	34.85 ± 1.034	32.767 ± 0.60	32.8 ± 0.593	0.125^{ns}
Platelet count (10^5/cmm)	3.86 ± 0.30	3.627 ± 0.22	3.917 ± 0.176	0.665^{ns}
Neutrophil (%)	41.667 ± 2.390^b	54.667 ± 1.333^a	60.333 ± 1.892^a	0.001**
Lymphocyte (%)	57.667 ± 3.073^a	27.667 ± 2.445^b	27 ± 1.528^b	0.001**
Monocyte (%)	4.333 ± 0.615^a	2.333 ± 0.333^b	2 ± 0^b	0.002**

** Significant at 0.05 level; ** Significant at 0.01 level; ns non-significant*

Table 2. Results of comparison of haematological parameters of group I and group II (pre- and post-treatment)

Parameter	Group I (n=6) Mean ± SE			Group II (n=6) Mean ± SE		
	Before Treatment	After Treatment	p-value	Before Treatment	After Treatment	p-value
WBC (10^3/cmm)	16.58 ±1.595	13.38 ±1.176	0.046*	13.58 ±3.219	11.88 ±0.732	0.549^{ns}
RBC (10^6/cmm)	18.43 ±1.173	17.72 ±0.976	0.226^{ns}	14.99 ±2.143	15.95 ±0.961	0.744^{ns}
HGB (g/dL)	11.07 ±0.897	10.38 ±0.604	0.133^{ns}	8.63 ±1.341	8.98 ±0.538	0.838^{ns}
VPRC (%)	27.383 ±1.027	32.15 ±0.848	0.482^{ns}	26.25 ±4.035	26.17 ±1.731	0.987^{ns}
MCV (fL)	17.367 ±1.093	17.55 ±0.766	0.018*	17.20 ±0.699	16.95 ±0.579	0.515^{ns}
MCH (pg)	5.93 ±0.236	5.58 ±0.206	0.101^{ns}	5.57 ±0.235	5.23 ±0.076	0.195^{ns}
MCHC (g/dL)	32.77 ±0.600	33.27 ±0.749	0.113^{ns}	32.80 ±0.593	32.85 ±0.591	0.927^{ns}
Platelet count (10^5/cmm)	3.63 ±0.220	4.05 ±0.277	0.302^{ns}	3.92 ±0.176	4.66 ±0.175	0.052^{ns}
Neutrophil (%)	54.67 ±1.333	42.67 ±2.108	0.014*	60.33 ±1.892	44.67 ±1.430	0.003**
Lymphocyte (%)	27.67 ±2.445	41.67 ±1.892	0.014*	27.00 ±1.528	48.33 ±1.820	0.001**
Monocyte (%)	2.33 ±0.333	3.00 ±0.447	0.363^{ns}	2.00 ±0.000	3.33 ±0.422	0.025*

** Significant at 0.05 level; ** Significant at 0.01 level; ns non-significant*

Results and discussion

Haematology

In the present investigation, goats with post-partum fatty liver syndrome (both groups) showed a highly significant increase in neutrophil and a decrease in lymphocyte and monocyte counts before treatment when compared to the control (Table 1), whereas, after treatment, there was a substantial decrease in total WBC count, neutrophil count, increased MCV, and lymphocyte count in

Table 3. Results of comparison of blood glucose and blood βHB of the control group, group I and group II (pre-treatment)

Parameter	Control group (n=6) Mean ± SE	Group I (n=6) Mean ± SE	Group II (n=6) Mean ± SE	p-value
Glucose (mg/dL)	45.333 ± 5.116	38.667 ± 2.319	42.833 ± 2.688	0.436 ns
βHB (mmol/L)	0.6 ± 0.058[b]	1.25 ± 0.182[a]	1.2 ± 0.151[a]	0.009**

** Significant at 0.05 level; ** Significant at 0.01 level; ns non-significant*

Table 4. Results of comparison of blood glucose and blood βHB of group I and group II (pre- and post-treatment)

Parameter	Group I (n=6) Mean ± SE			Group II (n=6) Mean ± SE		
	Before Treatment	After Treatment	p-value	Before Treatment	After Treatment	p-value
Glucose (mg/dL)	38.667 ± 2.319	56.50 ± 3.394	0.231 ns	42.83 ± 2.688	51.83 ± 1.302	0.063 ns
βHB (mmol/L)	1.25 ± 0.182	0.60 ± 0.052	0.007 **	1.20 ± 0.151	0.60 ± 0.052	0.017*

** Significant at 0.05 level; ** Significant at 0.01 level; ns non-significant*

Table 5. Results of comparison of serum biochemical parameters of the control group, group I and group II (pre-treatment)

Parameter	Control group (n=6) Mean ± SE	Group I (n=6) Mean ± SE	Group II (n=6) Mean ± SE	p-value
AST (IU/L)	84.28 ± 1.855	141.683 ± 28.947	128.812 ± 17.244	0.126 ns
Total Protein (g/dL)	6.373 ± 0.262	7.467 ± 1.558	8.323 ± 0.943	0.448 ns
Albumin (g/dL)	3.115 ± 0.120	2.97 ± 0.330	3.717 ± 0.520	0.330 ns
Globulin (g/dL)	3.26 ± 0.187	4.713 ± 1.108	4.607 ± 0.471	0.295 ns
Triglycerides (mg/dL)	39.43 ± 2.984	114.423 ± 25.903	95.158 ± 24.862	0.056 ns
Cholesterol (mg/dL)	88.198 ± 7.026	103.913 ± 24.430	109.42 ± 11.359	0.634 ns
HDL (mg/dL)	7.835 ± 2.822	7.732 ± 1.629	9.41 ± 2.741	0.865 ns
LDL (mg/dL)	1.51 ± 0.14[a]	1.408 ± 0.259[a]	2.315 ± 0.312[b]	0.040*
VLDL (mg/dL)	7.887 ± 0.597	22.883 ± 5.181	19.035 ± 4.972	0.056 ns
Serum NEFA (mmol/L)	0.162 ± 0.016[c]	0.373 ± 0.015[a]	0.278 ± 0.024 [b]	0.001**
Calcium (mg/dL)	8.750 ± 0.774	6.545 ± 0.757	7.23 ± 0.990	0.499 ns
Phosphorus (mg/dL)	5.993 ± 0.930	4.636 ± 0.881	4.937 ± 0.485	0.226 ns
Serum urea nitrogen (mg/dL)	13.035 ± 0.610[b]	14.25 ± 1.327[a]	14.98 ± 1.894[a]	0.001**
Creatine (mg/dL)	0.248 ± 0.036	0.337 ± 0.070	0.428 ± 0.079	0.175 ns
GGT (IU/L)	40.345 ± 2.678	44.353 ± 4.787	40.477 ± 3.733	0.708 ns
Bilirubin Total (mg/dL)	0.545 ± 5.220	0.1 ± 0.030	1.025 ± 0.749	0.434 ns
Bilirubin Direct (mg/dL)	0.085 ± 0.046	0.265 ± 0.139	0.148 ± 0.048	0.374 ns

** Significant at 0.05 level; ** Significant at 0.01 level; ns non-significant*

Group I. In Group II, there was a considerable lowering of neutrophil counts and higher lymphocyte and monocyte counts (Table 2) after treatment. According to El-Khodery *et al*. (2011), goats with hepatic lipidosis showed low erythrocyte count, haemoglobin, and PCV levels with eosinophilia and increased numbers of segmented neutrophils. Tharwat *et al*. (2015) noted neutrophilia, monocytopenia and lower MCV in goats after kidding. The present results suggested post-kidding inflammatory response and moderate anaemia associated with post-partum fatty liver syndrome. In both the groups neutrophil counts decreased after treatments, whereas, in Group I MCV increased which indicated that the condition had improved after treatment.

Blood glucose

The blood glucose showed a non-significant decrease in both groups before treatments (Table 3). Even though there was no statistical correlation, there was an increase in blood glucose levels after treatment in both study groups (Table 4). Dutta and Hussain (2020) reported that the minimum level of blood glucose in goats was 40-60 mg/dL. Almost normal blood glucose and the presence of milk ketone bodies obtained before treatment indicated moderate negative energy balance in goats after kidding in the present study.

Blood βHB

Vijayanand *et al*. (2022) noticed that blood βHB levels in apparently healthy goats ranged between 0.2 mmol/L to 0.4 mmol/L. In the present study, the control animals showed blood βHB levels of 0.6 ± 0.058 mmol/L. Before therapy, blood βHB levels increased significantly in both study groups (Table 3). Blood βHB levels varying from 0.8 to 1.6 mmol/L indicated subclinical ketonemia in ewes (Pichler *et al*., 2014). When compared to the pre-treatment values, a statistically significant drop in blood βHB levels was observed after treatment in both groups (Table 4) which indicated clinical improvement in both treatment regimens.

Serum biochemistry

In this present study, there existed a statistically significant increase in serum NEFA and a moderate increase of SUN values between the control group and the study groups during pre-treatment. There was a significant reduction in NEFA and SUN values in both groups after treatment. Taghipour *et al*. (2010) reported that in ewes the levels of NEFA and βHB increased after lambing and showed a positive relationship. Aly *et al*. (2016) attributed the substantial increase in serum NEFA and triglycerides to the significant fat mobilization from adipose tissue in response to the significant reduction in blood glucose during milk production. According to Sadjadian *et al*. (2013), higher NEFA levels during the periparturition phase and an increase in BUN values during the postpartum period in goats could be due to enhanced lipid mobilization and increased protein catabolism. Levels of serum NEFA, BUN, and creatinine were found to be increased in sub-clinical and clinical pregnancy toxaemias of goats also (Vijayanand *et al*., 2022). The serum NEFA and SUN values were reduced after treatments in the present study, which indicated improvement after treatment.

The LDL cholesterol was moderately lowered in Group-I and moderately elevated in Group II before treatment (Table 5). However, no variation in LDL cholesterol after treatment when compared to before-treatment values was observed in both groups (Table 6). Tharwat *et al*. (2015) reported that the reduction in HDL, LDL, and VLDL cholesterols in goats during the transitional phase could be physiological. Nazifi *et al*. (2002) found that during lactation, an increased nor-epinephrine and epinephrine-stimulated free fatty acid synthesis with improved esterification of fat in the liver occurred, which significantly reduced the lipogenesis Triglycerides and VLDL values were reduced in Group-after treatment (Table 6). Reduced triglycerides after treatment in Group-I, indicated less fat mobilization (Sudharsan *et al*., 2020). However, reduced VLDL value after treatment indicated that lipoprotein production from the liver might not have improved using the first treatment regimen (Bobe *et al*., 2004).

Ultrasonography

In the present study the hepatic parenchyma was hyperechoic with engorgement of blood vessels in all affected Does, when compared with that of the control

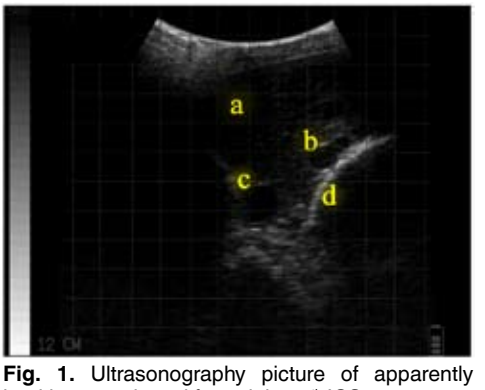

Fig. 1. Ultrasonography picture of apparently healthy goat viewed from right 10th ICS.

a: liver parenchyma; b: portal vein; c: caudal vena cava; d: omasum

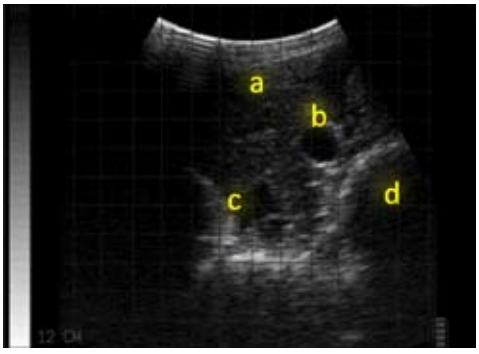

Fig. 2. Ultrasonography picture of hyperechogenicity in fatty liver goat viewed from right 10th ICS before treatment.

a: liver parenchyma; b: portal vein; c: caudal vena cava; d: omasum

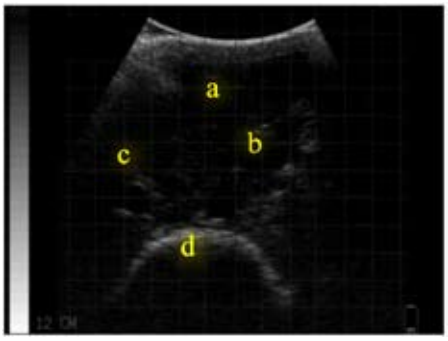

Fig. 3. Ultrasonography of reduced hyper echogenicity of fatty liver in goat viewed from right 10th ICS After treatment.

a: liver parenchyma; b: portal vein; c: caudal vena cava; d: omasum

Table 6. Results of comparison of serum biochemical parameters of group I and group II (pre- and post-treatment)

Parameter	Group I (n=6) Mean ± SE			Group II (n=6) Mean ± SE		
	Before Treatment	After Treatment	p-value	Before Treatment	After Treatment	p-value
AST (IU/L)	141.68 ± 28.947	88.25 ± 8.534	0.160 ns	128.81 ± 17.244	87.33 ± 3.360	0.083 ns
Total Protein (g/dL)	7.47 ± 1.558	5.26 ± 0.741	0.093 ns	8.32 ± 0.943	6.12 ± 0.647	0.149 ns
Albumin (g/dL)	2.97 ± 0.330	2.25 ± 0.448	0.100 ns	3.72 ± 0.520	2.60 ± 0.315	0.113 ns
Globulin (g/dL)	4.71 ± 1.108	3.02 ± 0.665	0.126 ns	4.61 ± 0.471	3.54 ± 0.445	0.244 ns
Triglycerides (mg/dL)	114.42 ± 25.903	39.98 ± 1.612	0.033 *	95.16 ± 24.862	26.42 ± 5.312	0.052 ns
Cholesterol (mg/dL)	103.91 ± 24.430	126.04 ± 15.633	0.523 ns	109.42 ± 11.359	102.50 ± 7.611	0.487 ns
HDL (mg/dL)	7.73 ± 1.629	5.74 ± 0.958	0.188 ns	9.41 ± 2.741	8.15 ± 0.696	0.609 ns
LDL (mg/dL)	1.408 ± 0.259	1.49 ± 0.174	0.848 ns	2.315 ± 0.312	1.76 ± 0.225	0.280 ns
VLDL (mg/dL)	22.88 ± 5.181	7.99 ± 0.322	0.033 *	19.04 ± 4.972	5.28 ± 1.062	0.052 ns
Serum NEFA (mmol/L)	0.37 ± 0.15	0.17 ± 0.008	0.001 **	0.28 ± 0.024	0.16 ± 0.013	0.008 **
Calcium (mg/dL)	6.55 ± 0.757	7.70 ± 0.319	0.121 ns	7.23 ± 0.990	7.31 ± 0.295	0.952 ns
Phosphorus (mg/dL)	4.636 ± 0.881	5.70 ± 0.740	0.486 ns	4.937 ± 0.485	7.54 ± 1.315	0.332 ns
Serum urea nitrogen (mg/dL)	14.25 ± 1.327	13.720 ± 0.860	0.031 *	14.98 ± 1.894	14.550 ± 1.690	0.012 *
Creatine (mg/dL)	0.34 ± 0.070	0.21 ± 0.033	0.126 ns	0.43 ± 0.079	0.21 ± 0.067	0.138 ns
GGT (IU/L)	44.35 ± 4.787	30.41 ± 3.930	0.084 ns	40.48 ± 3.733	37.83 ± 3.290	0.582 ns
Bilirubin Total (mg/dL)	0.10 ± 0.030	0.07 ± 0.037	0.447 ns	1.03 ± 0.749	0.15 ± 0.052	0.311 ns
Bilirubin Direct (mg/dL)	0.27 ± 0.139	0.11 ± 0.037	0.372 ns	0.15 ± 0.048	0.23 ± 0.101	0.582 ns

** Significant at 0.05 level; ** Significant at 0.01 level; ns non-significant*

animals which indicated fatty infiltration of the liver (Fig.1 and 2). There was a reduction in the hyperechogenicity and engorgement of blood vessels in the hepatic parenchyma after both treatments (Fig.3), which indicated the efficacy of both treatments. El-Khodery *et al*. (2011) revealed that the hepatic parenchyma exhibited considerable lipidosis-related generalised hyperechogenicity in goats.

Response to treatment

Both the treatments were found to be equally efficient when considering the rate of changes in key haemato-biochemical parameters and ultrasonography after treatment A significant decrease in plasma glucose levels, free fatty acids and beta-hydroxybutyrate levels were observed with oral administration of nicotinic acid in association with subclinical and clinical ketosis in cows (Fronk and Schultz, 1979). Also, feed supplementation of methionine and choline had the capacity to improve VLDL export from the liver (Grummer, 2008; Neelima *et al*., 2021). Glycerol is the most efficient hyperglycemic agent in ruminants to treat negative energy balance because biochemically it does not require oxaloacetate in the tricarboxylic acid cycle to produce glucose (Kaneko *et al*., 2008).

Conclusion

The fatty liver syndrome in post-partum goats causes low production and body condition in goats and was diagnosed by testing of milk, estimating the levels of blood beta-hydroxybutyric acid and serum NEFA levels and ultrasonography of the liver. Biochemical, inflammatory, and hepatic ultrasonographic changes subsided after giving both treatments under the study.

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Conflict of interest

The authors declare that they have no conflict of interest.

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