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RESEARCH ARTICLE

The Importance of Concentrations of Sorbitol Dehydrogenase and Glutamate Dehydrogenase and B-Mode Ultrasonographic Examination in The Diagnosis of Hepatic Lipidosis in Dairy Cows [1]

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Summary

The aim of this study is to determine the importance of B-mode ultrasonography in the diagnosis of hepatic lipidosis in dairy cows and compare this mode of diagnosis with both the histologic examination of liver biopsy samples and investigation of some biochemical parameters associated with hepatic lipidosis. 15 Holstein cows with moderate hepatic lipidosis and 15 cows with severe hepatic lipidosis and 6 healtyh cows were used as a metarilas. Blood samples were obtained from all cows and analyzed. Liver samples were obtained by biopsy in cattle. The ultrasonographic examination of liver was performed on animal. Serum glutamate dehydrogenase (GDH) and aspartate amino transferase (AST) concentrations were increased in cows with moderate hepatic lipidosis. Serum sorbitol dehydrogenase (SDH), GDH, and AST concentrations were increased in cows with severe hepatic lipidosis. Ultrasonographic examination revealed an increase in diffuse echogenicity of the liver in cows with moderate and severe hepatic lipidosis cows, but the increase was little in moderate hepatic lipidosis. Both serum GDH and SDH levels were found to be increased in severe hepatic lipidosis. However, only the serum GDH level was elevated in moderate hepatic lipidosis. Therefore, both ultrasonographic examination and measurement of specific liver enzymes seem to be beneficial in the diagnosis of hepatic lipidosis.

Keywords: *Hepatic lipidosis, Liver enzymes, Liver ultrasonography, Dairy cow*

Sütçü Sığırların Hepatik Lipidozisinin Teşhisinde B-Mode Ultrasonografik Muayene ve Serum Sorbital Dehidrogenaz ve Glutamete Dehidrogenaz Düzeylerinin Önemi

Özet

Bu çalışmanın amacı; sütçü sığırların yağlı karaciğer sendromunun teşhisinde B-mode ultrasonografinin önemini belirlemek, hastalığın tanısında ultrasonografik muayene ile karaciğer biyopsi örneklerinin histolojik bulguları ve bazı biyokimyasal parametre sonuçlarını karşılaştırmaktır. Bu çalışmada 15 orta hepatik lipidozisli, 15 şiddetli hepatik lipidozisli ve 6 sağlıklı Holştein ırkı sütçü sığır kullanıldı. Bütün sığırlardan kan alındı ve analiz edildi. Sığırların karaciğerinin ultrasonografik muayenesi yapıldı. Hepatik lipidozisli sığırlardan karaciğer biyopsisi alındı. Orta dereceli hepatik lipidozisli sığırlarda serum glutamate dehidrogenaz (GDH) ve aspartat aminotransferaz (AST) seviyeleri artarken, şiddetli hepatik lipidozisli sığırlarda serum sorbital dehidrogenaz (SDH), GDH ve AST seviyeleri arttı. Ultrasonografik maueyenede; orta ve şiddetli hepatik lipidozisli sığırlarda karaciğerde diffuz ekojenite artışı gözlemlenmekle birlikte, orta dereceli hepatik lipidozis olgularında ekojenite artışı daha azdı. Şiddetli hepatik lipidozisde SDH ve GDH enzim düzeylerinde, orta dereceli hepatik lipidozisde ise sadece GDH enzim düzeyinde artış belirlendi. Hem ultrasonografik muayene hem de karaciğerin spesifik enzim ölçümü hepatik lipidozisin teşhisinde faydalı olduğu düşüncesine varıldı.

Anahtar sözcükler: *Hepatik lipidozis, Karaciğer enzimleri, Karaciğer ultrasonu, Sütçü sığır*

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INTRODUCTION

Liver plays a pivotal role in provision of energy for periparturient dairy cows from various precursors [1]. A combination of a deep negative energy balance and fatty liver is a common feature in high producing dairy cows after parturition [2]. Fatty liver syndrome or hepatic lipidosis is characterized by infiltration of triacylglycerol (TAG) in the liver [3]. Fatty liver is a metabolic disorder that is caused by excessive mobilization of body fat, which occurs in high yielding dairy cows in which the demands for energy exceed the supply [4]. Over conditioning is a major risk factor for developing fatty liver [5]. Fatty liver is associated with decreased health status, well-being, milk yield, reproductive performance, and reduced immune response. Severe and moderate fatty liver develops in approximately 15% and 35% of dairy cows, respectively [6]. Fatty liver occurs primarly in the 1st month of lactation in dairy cows [7,8].

Blood profiles and changes in body condition score have been used to monitor metabolic imbalance around parturition and, in early lactation, to investigate problem [9,10] as well as to predict the risk of diseases such as displaced abomasum [11]. Parameters used to monitor imbalance in energy metabolism have included glucose, non-esterified fatty acids (NEFA), betahydroxybutyrate (BHBA), and change in body condition score [12,13]. Fatt liver develops when hepatic availability of lipogenic and glucogenic products is imbalanced. Thereby the oxidation capacity of fatty acids is exceeded and since hepatic secretion of lipids is inherited, low excess hepatic lipids are stored as TAG in the liver tissue [6,14]. In domestic animals, serum enzyme test are grouped into those that indicate hepatocelluler leakage due to hepatocyte damage [15-17]. Hepatocelluler enzymes, particularly AST, GDH and SDH may be useful in monitoring the hepatic lipidosis that commonly occurs at parturition [5,18]. The hepatocelluler leakage enzymes-AST and cholestatic enzymes-GGT activities have been used to evaluate the liver [19]. Blood cholesterol concentration is related to feed intake and low cholesterol concentrations have been associated fatty liver post-partum [20].

Fatty liver can be diagnosed as reliable only by determining TAG content biochemical or histological analysis of a liver puncture biopsy sample. Biopsies are impracticable for on-farm diagnosis because they cause temporary discomfort to cow, pose risk of infection, and can be lethal if a major blood vessel is punctured [21]. Therefore, a non-invasive technique would be very useful. Ultrasonograph has been used routinely for about long time as a diagnostic procudure in hepatic disease of cows [22]. Ultrasound imaging followed by digital analysis of sonograms has potential to non-invasively detect fatty liver and estimate liver TAG content [23]. Haudum et al.[24] reported that ultrasonography has proven useful for the evaluation of hepatic triacylglycerol content in dairy cows. However, detection of fatty liver is more difficult because it results in smaller cages of hepatic echostructure [22].

The aim of this study is to evaluate the importance of B-mode ultrasonography in the diagnosis of hepatic lipidosis in dairy cows and compare this mode of diagnosis with both the histologic examination of liver biopsy samples and investigation of some biochemical parameters (Especially SDH and GDH) associated with hepatic lipidosis.

MATERIAL and METHODS

Animals and Clinical Examination

The institutional ethical committee approved this prospective study. In this study, 15 Holstein cows with moderate hepatic lipidosis and 15 Holstein cows with severe hepatic lipidosis in the first 4 weeks of lactation were used. Cows were aged 3-7 years, and the mean daily milk yield was 25 kg. In the reference group, 6 clinically healthy postparturient Holstein cows from a local dairy farm were used. These cows were aged 3-6 years, were in the first 2-4 weeks of lactation, and were reported by the owner to have a daily milk yield ranging from 23 to 27 kg on milking twice daily. Of 15 cows with moderate and severe hepatic lipidosis, 10 showed left abomasal displacement, and of 10 cows with moderate and severe hepatic lipidosis, 5 had ketosis. Routine physical examination, including simultaneous auscultation and percussion of the abdomen, ballottement of the abdomen for a splashing sound to indicate the presence of an air-fluid interface in a large viscus, palpation per rectum, urinary examination, and ultrasonographic examination, were performed on each cow.

Ultrasonographic Examination of Abomasum and Liver

The ultrasonographic examination of the abomasum is performed on the left side of the standing animal. Ultrasonographic examination of the abomasum was performed at the 10th and 13th intercostal spaces on the left side, and the area was examined ventrally to dorsally using a real-time 3.5-5.0-MHz convex transducer [25,26]. The liver was examined by ultrasonography from the caudal to the cranial region, beginning from the caudal region to the last rib on the right side and ending at the fifth intercostal space, and from the dorsal to ventral region in every intercostal space using a real-time 3.5-5.0-MHz convex transducer [22]. Ultrasonographic examination of the liver was performed in all cows.

Detection of Ketosis

Detection of ketosis; urine was collected with a catheter or free flow urine was collected from all cows and a drop was applied to reagent strips (Multistix® 10 SG; Bayer). Results were scored as positive or negative depending on whether or not there was a change in color from white to purple.

Treatment

A right flank laparotomy was performed under regional analgesia on cows with a presumptive clinical diagnosis of LDA, and the diagnosis was confirmed during surgery using established criteria [27]. Cows with LDA were hospitalized for 1 day after surgery and then discharged. The clinical outcome was investigated at least 2 weeks after surgery by communicating with the owner via telephone. Cows with ketosis were treated. The treatment protocol for cows with ketosis was as follows: intravenous administration of 1.000 mL of 30% serum dextrose (Dekstrose®, Eczacibasi, Baxter) for 3 days, administration of 200 IU insulin (NPH, Humulin®, Lilly Ilac), intramuscular administration of 10 mg of dexamethasone (Devamed®, Topkim Ilac) for 3 days, and oral administration of 150 mL of propylene glycol (Bovical®, Bioteknik) for 4 days. Cows with moderate and severe hepatic lipidosis received ancillary treatment for hepatic lipidosis.

Blood Sample Collection

Blood samples were collected from the jugular vein immediately before surgery in cows with displaced abomasum and before treatment in cows with ketosis and from healthy cattle. An aliquot of blood was placed into glass tubes for serum biochemical analysis. The tubes were centrifuged after clotting, and the serum was harvested and stored at -20°C until analysis.

Biochemical Analyses

Serum SDH concentration was determined by sandwich ELISA (USCN Life Science Inc. Cat No: E91495 Bo, Wuhan, China). The manufacturer of this assay reported the limit of detection in bovine serum as 20 µL and the reference range as 1.56-100 U/L. Serum GDH concentration was determined by sandwich ELISA (USCN Life Science Inc. Cat No: E90293Bo,Wuhan, China). The manufacturer of this assay reported the limit of detection in bovine serum as 100 µL and the reference range as 7.8-500 U/L. Serum AST, ALT, GGT, and ALP activities, as well as serum glucose, cholesterol, triglyceride, blood urea nitrogen (BUN), creatinine, total protein, albumin, Mg^{+}, P^{+}, and Ca^{++} concentrations were measured with an automatic analyzer (BT 3000 plus, Biotecnical Inc, SPA, Via lizenca, 18 00155, Rome, Italy). The serum insulin level was measured by an immunoassay system (Invitrogen immunoassays kit # PL2820085, Advia Center XP, Siemens, REVM). Blood Na^{+}, K^{+}, and Cl^{-} concentrations were measured by using ion-selective electrodes.

Liver Sample Collection and Histological Examination

Liver biopsy samples were obtained preoperatively in cows with LDA and cows with ketosis percutaneously through the right 11^{th} to 12^{th} intercostal space. Liver biopsy samples were not obtained from healthy cattle. Liver biopsy samples were placed in Baker's formal-Ca solution and fixed in paraffin for at least 16 h. From each fixed liver sample, 12-mm sections were cut and stained with oil Red O and Sudan Black B. The sections were examined under light microscopy as described [28], and the percentage volume of visible fat in hepatic parenchymal cells was estimated by a stereological point counting method. The extent of fat infiltration in the liver was categorized as mild (<10%, <10 μm^2/100 μm^2), moderate (10%-20%, 10-20 μm^2/100 μm^2), and severe (>20%, >20 μm^2/100 μm^2) on the basis of the percentage volume of visible fat [29].

Statistical Analyses

Statistical analyses were performed using a package program (SPSS 15.0), as reported by Akgül [30]. One-way analysis of variance (ANOVA) and Tukey's test were used for comparing the data ($P<0.05$).

RESULTS

Twenty cows with LDA did not have evidence of any other clinical disease. Cows with LDA had fair to moderate appetite and decreased rumen contraction frequency, defecation, and milk production. Ten cows with ketosis had depressed appetite, decreased rumen contraction frequency and milk production, dry feces, ketonuria, and ketonemia.

Serum GDH, and AST concentrations were increased in cows with moderate hepatic lipidosis and severe hepatic lipidosis as compared with in the controls *(Table 1)*; however, serum SDH concentration was increased in cows with severe hepatic lipidosis. In addition, serum SDH, GDH, AST, and cholesterol levels were higher in cows with severe hepatic lipidosis than in cows with moderate hepatic lipidosis. In contrast, serum cholesterol, Ca^{++}, Cl^{-}, Na^{+}, and K^{+} levels were decreased in cows with moderate hepatic lipidosis and in cows with severe hepatic lipidosis as compared with in the controls *(Table 1)*. Serum total protein level was increased in cows with severe hepatic lipidosis as compared with in cows with moderate hepatic lipidosis and in the controls.

Increase in diffuse echogenicity of the liver was observed in cows with severe hepatic lipidosis *(Fig. 1)* and cows with moderate hepatic lipidosis, but the increase in the latter was little *(Fig. 2)*. The liver appeared white on ultrasonograms, and it was difficult to differentiate the liver from the surrounding tissue. Echogenicity of the liver was normal in healthy cows *(Fig. 3)*. There was no significant difference between cows with ketosis and cows with LDA in terms of the liver fat percentage. There were 15 cows with moderate (10% - 20%) hepatic lipidosis and 15 with severe (20% - 48%) hepatic lipidosis.

DISCUSSION

The main finding of the present study was that SDH, GDH, and AST enzyme activities were increased in post-

Table 1. *Serum biochemical parameters healthy lactating cows (control) and lactating cows with moderate and severe hepatic lipidosis.*
Tablo 1. *Sağlıklı, orta ve şiddetli hepatik lipidozisli sütçü sığırların serum biyokimyasal parametreleri*

Parameters	Groups			P
	Control N=6	Moderate N=15	Severe N=15	
SDH (UI/L)	18.93±4.32 [a]	42.63±5.28 [a]	75.78±9.75 [b]	0.000
GDH (UI/L)	101.33±8.78 [a]	132.08±9.93 [ab]	149.50±12.18 [b]	0.040
AST (UI/L)	71.33±6.30 [a]	139.33±13.28 [b]	155.07±15.19 [b]	0.007
ALT (U/L)	26.17±2.65	25.31±2.91	28.30±3.17	0.762
GGT (UI/L)	31.50±8.43	46.85±6.29	38.30±5.26	0.297
ALP (UI/L)	47.17±5.15	41.38±3.82	37.30±5.72	0.302
Insulin (µU/L)	0.35±0.07	0.42±0.29	0.50±0.34	0.752
Cholesterol (mg/dL)	176.10±89.03[a]	125.23±68.74[ab]	78.00±11.7[b]	0.36
Tryglyseride (mg/dL)	16.17±2.86	19.92±4.65	22.50±9.30	0.183
Glucose (mg/dL)	101.67±6.64	102.31±13.79	79.90±18.49	0.515
Urea (mg/dL)	29.50±0.99	39.00±4.17	32.70±4.96	0.329
Creatinin (mg/dL)	1.82±0.18	1.30±0.12	1.37±0.12	0.051
Total protein (g/dL)	7.08±0.14 [a]	8.65±0.31 [a]	10.81±0.84 [b]	0.001
Albumin (g/dL)	3.38±0.26	3.86±0.22	4.18±0.15	0.089
Magnesium(mg/dL)	2.00±0.05	1.67±0.17	2.09±0.11	0.101
Calcium (mg/dL)	10.05±0.31 [a]	8.49±0.25 [b]	9.12±0.27 [ab]	0.004
Phosphorus (mg/dL)	6.40±0.66	5.22±0.47	4.68±0.32	0.096
Chlor (mmol/L)	101.50±1.61[a]	94.23±1.76 [b]	96.10±1.66 [ab]	0.045
Sodium (mmol/L)	144.33±0.84 [a]	137.00±0.72 [b]	140.75±1.15 [c]	0.000
Potassium (mmol/L)	3.90±0.18 [a]	3.06±0.22 [b]	3.41±0.15 [ab]	0.043

SDH: *Sorbitol dehydrogenase,* ***GDH:*** *Glutamate dehydrogenase,* ***AST:*** *Aspartate amino transferase,* ***ALT:*** *Alanin amino transferase,* ***GGT:*** *gamma-glutamyl transferase,* ***ALP:*** *Alkaline phosphatase, [a-c] means within row with different supercript differ (P<0.05)*

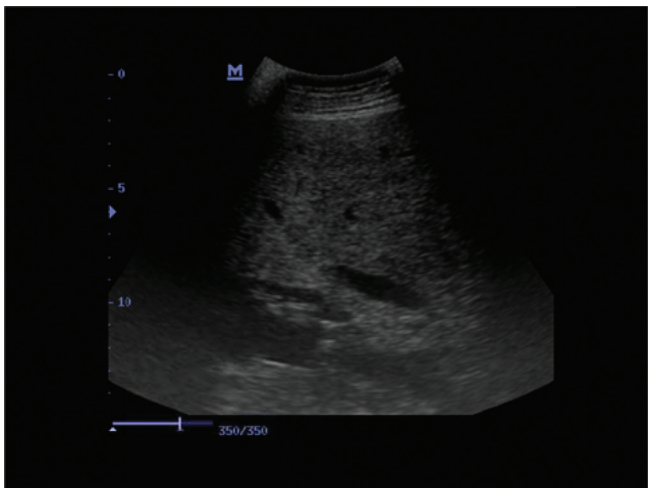

Fig 1. Ultrasonogram of liver of a cow with severe hepatic lipidosis
Şekil 1. Şiddetli hepatik lipidosisli bir inekte karaciğer ultrasonogramı

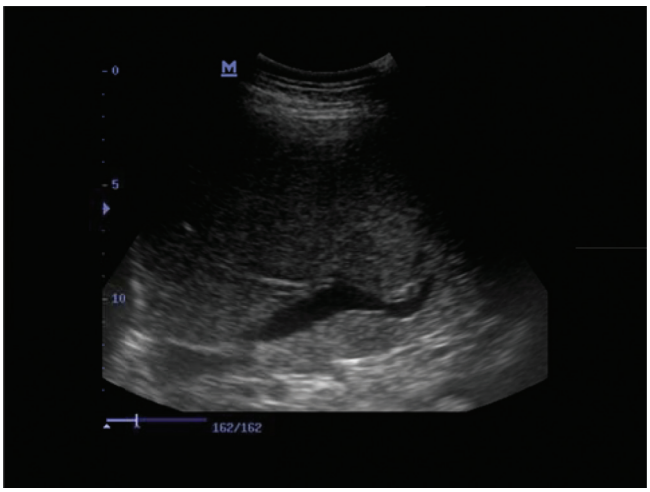

Fig 2. Ultrasonogram of liver of a cow with moderate with hepatic lipidosis
Şekil 2. Hafif şiddetli hepatik lipidosisli bir inekte karaciğer ultrasonogramı

parturient dairy cows with hepatic lipidosis. The second finding was that an increase in diffuse echogenicity of the liver was observed on ultrasonographic examination in cows with severe hepatic lipidosis and cows with moderate hepatic lipidosis, but the increase was little in the latter.

Hepatocelluler enzymes, particularly AST and GDH may be useful in monitoring the hepatic lipidosis that commonly occurs at parturition [5,31]. SDH and GDH are liver-specific enzymes [16,17,32,33]. Increased serum SDH and GDH activity is suggestive of either hepatocyte death or sublethal hepatocyte injury. Sorbital dehydrogenase and GDH have been regarded by many as enzymes of choice for

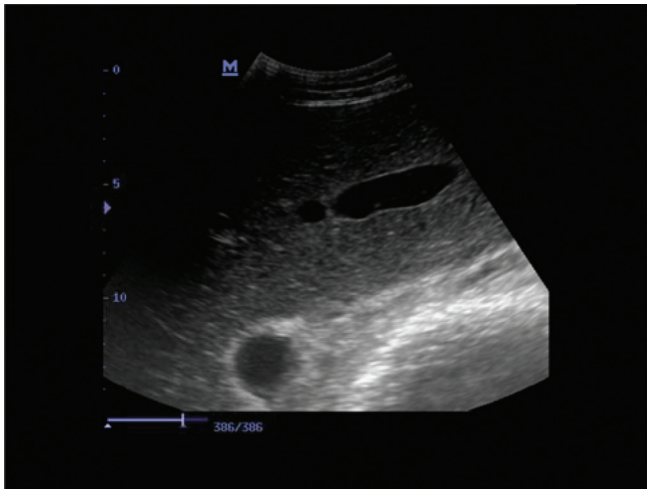

Fig 3. Ultrasonogram of liver of healthy a cow
Şekil 3. Sağlıklı bir inekte karaciğer ultrasonogramı

use as indicators of the degree of hepatic necrosis in sheep, goats, and cattle [16,17,33-35]. Braun et al.[36] reported that SDH, GDH, and GGT concentrations are increased in cows with liver tumors. Kalaitzakis et al.[33] mentioned that AST, SDH, GDH and ornitine carbamobyl transferase, activity was increased in cases of fatty liver syndrome because of the destruction of liver cells. The activities of hepatocellulerr leakage enzymes such as AST and cholestatic enzymes such as GGT have been used to evaluate the liver [19]. Bogin et al.[37] found significantly increased AST levels in cows with severe fatty liver. Sevinc et al.[28] showed that cows with severe fatty liver had high AST and GGT activities. Hepatic lipidosis may contribute to the pathogenesis of abomasal displacement. Komatsu et al.[38] indicated that high levels of AST in cows with LDA were found in fatty liver degeneration. Kalaitzakis et al.[39] reported that OCT and GDH enzymes might be useful in diagnosis fatty liver in downer cows. In this study, serum GDH and AST concentrations were increased in cows with moderate and severe hepatic lipidosis compared with in the controls. However, only the serum SDH concentration was increased in cows with severe hepatic lipidosis. Increased SDH and GDH concentrations in cows with hepatic lipidosis may be related to hepatocyte death or sublethal hepatocyte injury. SDH, GDH, and AST levels were high in cows with moderate and severe hepatic lipidosis compared with in the control group. Although higher levels of SDH and GDH are generally reported in acute liver damage (within 4 to 24 hours of hepatic injury) [19,32]. We found that higher levels of these enzymes were found in cows with hepatic lipidosis. We understand that these enzymes are increased not only in acute liver damage but also in chronic liver damage [15,17,33].

Blood cholesterol concentration is related to feed intake [40] and low cholesterol concentration have been associated fatty liver post-partum [20]. Several authors [1,28,33] reported that triglyceride, cholesterol and HDL- cholesterol concentrations are decreased in the cows with fatty liver. Sevinc et al.[41] found that serum triglyceride and cholesterol concentrations were decreased in the cows with moderate and severe fatty liver. In the present study, serum cholesterol level was decreased in cows with moderate hepatic lipidosis and in cows with severe hepatic lipidosis as compared with in the controls, but triglyceride level in cows with moderate and severe hepatic lipidosis was not different from healthy cows. The low cholesterol level can be thought to be caused by a fat infiltration in the liver and a low output of lipoprotein.

Madison and Trout [42] found that calcium when present at a level of <1.2 mmol/L had a reducing effect on abomasal motility. They also brought attention to the fact that hypocalcemia cannot be a major causative factor for decreased abomasal motility with respect to the development of abomasal displacement. Some authors reported that hypocalcemia is a risk factor for abomasal displacement [43-45]. Metabolic alkalosis is mentioned as a risk factor for abomasal displacement [44]. The fluid accumulating in the distended abomasum indicates continuous secretion of hydrochloric acid. This sequestration of the chloride ion in the abomasum along with some abomasal reflux into the rumen results in metabolic alkalosis [46]. During the dilatation phase, which commonly lasts for several days, there is continuous secretion of hydrochloric acid, sodium chloride, and potassium into the abomasum; thus, the abomasum becomes gradually distended and does not evacuate its contents into the duodenum. This leads to dehydration and metabolic alkalosis with hypochloremia and hypokalemia [47]. In this study, Ca^{++}, Cl^{-}, Na^{+}, and K^{+} levels were decreased in cows with moderate hepatic lipidosis and in cows with severe hepatic lipidosis as compared with in the controls. Decrease in the Ca^{++}, Cl^{-}, Na^{+}, and K^{+} concentrations may be related to metabolic alkalosis resulting from abomasal displacement.

Nutritional stress in dairy cattle can be evaluated by determining the serum NEFA, BHBA, acetoacetate, cholesterol, and glucose and liver fat percentages, with the latter being regarded as the most accurate indicator of nutritional stress [15,16,48,49]. Liver fat percentage is increased in cows with abomasal displacement [50-52] and ketosis [48]. The liver fat percentage in healthy cattle is typically 5%, and is increased to 8% in healthy cows shortly after calving and to 33% in cows with postparturient ketosis [48]. In the present study, cows with abomasal displacement and ketosis had moderate (10% - 20%) and severe (20% - 48%) hepatic lipidosis according to histopathologic evaluation. Increase in serum AST activities is consistently related to fatty liver [41,53].

Ultrasonography has been used routinely for a long time as a diagnostic procedure in hepatic disease of cows [22]. Ultrasound imaging followed by digital analysis of sonograms has the potential to non-invasively detect fatty liver and estimate liver triacylglycerol content [23,24]. Braun [22] reported the ultrasonographic features of fatty livers as an

increase in the size of the liver, round margins, hyperechoic hepatic parenchyma near the abdominal wall, and decrease in the strength of the echo with increase in the distance from the abdominal wall, and poor visualization of hepatic blood vessels. In this study, increase in the diffuse echogenicity of the liver was observed in cows with severe hepatic lipidosis *(Fig. 1)* and cows with moderate hepatic lipidosis *(Fig. 2)*, but the increase in the latter was little. There was a good relationship between the ultrasonographic image findings and 20% and over fatty liver. Large vessels of the liver in cows with moderate or severe hepatic lipidosis were observed, but the small vessels of the liver were poorly imaged or not seen in cows with severe hepatic lipidosis *(Fig. 1)* and only faintly seen or poorly imaged in cows with moderate hepatic lipidosis *(Fig. 2)*. The poor images of the small vessels may be attributable to the deposition of fat on the surface of vessels and the swelling of hepatocytes due to fat deposition in the cells. Otherwise, the cause of increase in diffuse echogenicity of the liver may be the diffuse fat deposition in the cells. Mohamed et al.[54] reported that ultrasonography may provide good images of focal fatty infiltration of the liver. There was also a significant relationship between serum liver-specific enzymes (SDH, GDH, and AST), histopathologic findings, and ultrasonographic imaging findings of the liver.

The results of our study showed that B-mode ultrasonography is a valuable tool in the diagnosis of hepatic lipidosis. Both serum GDH and SDH levels were found to be increased in severe hepatic lipidosis. However, only the serum GDH level was elevated in moderate hepatic lipidosis. Therefore, both ultrasonographic examination and measurement of specific liver enzymes (SDH and GDH) seem to be beneficial in the diagnosis of hepatic lipidosis.

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