*VETERINARSKI ARHIV 84* (4), 355-364, 2014

# Evaluation of serum cardiac troponin I concentration in sheep with acute ruminal lactic acidosis

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**KIRBAS, A., E. BAYDAR, F. M. KANDEMIR, E. DORMAN, O. KIZIL, B. A. YILDIRIM: Evaluation of serum cardiac troponin I concentration in sheep with acute ruminal lactic acidosis. Vet. arhiv 84, 355-364, 2014.**

**ABSTRACT**

Cardiac troponin I (cTnI) is known to be a sensitive cardio biomarker to determine the myocardial damage in diseases affecting the cardiac muscles. However, there has not been sufficient research about cTnI concentration, which is the most sensitive indicator of myocardial damage in sheep with acute ruminal lactic acidosis (ARLA). For this reason this study aimed to evaluate the serum cTnI concentration in sheep with ARLA. Those diagnosed with ARLA (n = 20) from the total of 40 Akkaraman (White karaman) sheep, aged between 1-2 years used in this study comprised the affected group and the healthy ones (n = 20) comprised the control group. Ruminal fluid was obtained from the animals from both groups with the help of a stomach tube, and examined immediately. Blood samples were taken from the jugular vein of the sheep and the serum was separated. Serum cTnI concentration was measured with a commercial immunoassay system, using the one-step sandwich method. Serum enzyme (ALT, AST, CK-MB and LDH) activities were determined via a clinical biochemistry autoanalyzer. The average serum cTnI concentration was at the level of 0.035 ± 0.015 ng/mL (range; 0.02-0.06 ng/mL) in the control group sheep. It was determined that there was a substantial increase in the group with ARLA and the average concentration reached the level of 0.103 ± 0.080 ng/mL (range; 0.03-1.7 ng/mL) ($P<0.0001$). It was observed that another cardio marker, CK-MB, was found in the group with ARLA 454.50 ± 191.88 U/L (range; 214-861 U/L) and increased in comparison with the control group 224.35 ± 83.33 U/L (range; 133-421 U/L) ($P<0.0001$). An increase in LDH ($P<0.001$) and AST ($P<0.001$) from liver enzymes in the group with ARLA and ALT activities compared to the control group was identified ($P<0.01$). In conclusion, this present study determined that the serum cTnI concentration was high in sheep with ARLA and

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ISSN 0372-5480
Printed in Croatia

it was concluded that it could be useful to evaluate cTnI concentration as an important marker to determine the prognosis in sheep with ARLA.

**Key words:** acute ruminal lactic acidosis, cardiac biomarkers, cTnI, myocardial damage, sheep

## Introduction

Acute ruminal lactic acidosis (ARLA), which is a ruminal fermentative disorder, is acute indigestion resulting from the sudden digestion of foods containing easily fermentable carbohydrates, such as barley, wheat, sugar beet and sugar in high quantities in cattle and sheep (BRAUN et al., 1992; GARRY, 2002). The sudden and excessive intake of feeds containing rapidly fermentative carbohydrate results in dominance of Gram-negative and Gram-positive bacteria, producing lactic acid over rumen and increasing lactic acid production. The increase in lactic acid production causes osmotic pressure in the rumen fluid to increase, and results in fluid passage in excessive amounts, mostly from the extracellular compartment into the intra rumen (OWENS et al., 1998; GARRY, 2002). This increase in the volume of rumen fluid causes dilatation of the rumen, severe dehydration and systemic acidosis. In addition, cardiovascular collapse, renal failure, muscular weakness, shock and death are the most important pathophysiological results seen in ARLA (GARRY, 2002; CONSTABLE, 2010).

Cardiac troponins (cTn) are regulatory proteins which have three sub-forms: cTnI, cTnC and cTnT, participating in myocardial contraction by regulating its interaction related to actin and myosin (SARKO and POLLACK, 2002). Both cTnI and cTnT were defined as blood markers which are highly specific and sensitive for noninvasive diagnosis of the increase in cardiomyocyte permeability and the increase up to the level of myocardial damage in humans and animals (O'BRIEN, 2008).

The quantity of cTn in the cytosolic pool is the same as the quantity of creatine kinase myocard band (CK-MB) isoenzyme. However, cTn concentration substantially found in the contractile apparatus is 13-15 times higher than CK-MB quantity per myocard gram. cTn reaches measurable levels because of the oscillation from the cytosolic pool in the early period and the contractile apparatus in the late period, to the peripheric blood during myocyte damage (ADAMS et al., 1994; BABUIN and JAFFE, 2005). The blood levels rise 4-12 hours after acute myocardial damage, peak in 12-48 hours and blood cTn levels continue to be high for 2 weeks (WELLS and SLEEPER, 2008).

Although there are many biomarkers, such as CK-MB, lactate dehydrogenase (LDH), aspartate aminotransferase (AST), to determine myocardial damage, the efficacy of these enzymes is limited because of the unavailability of tissue specificity and sensitivity (JAFFE et al., 1996; O'BRIEN, 2008).

It is emphasized that cTnI is a strongly sensitive biomarker to determine myocardial damage in sheep (TUNCA et al., 2009; KARAPINAR et al., 2012; HAJIMOHAMMADI et al.,

2014), cattle (GUNES et al., 2008), horses (PHILLIPS et al., 2003), dogs (OYAMA and SISSON, 2004) and cats (CONNOLLY et al., 2003).

In this study, the aim was to evaluate cTnI concentration in sheep with ARLA and to investigate interrelations between ARLA and myocardial degeneration.

**Materials and methods**

*Animals.* Twenty Akkaraman sheep aged between 1-2 years with ARLA admitted to the Veterinary Teaching Hospital School of Veterinary Medicine, Firat University, from one herd (ARLA group) and 20 healthy Akkaraman sheep aged between 1-2 years, randomly selected from a different herd (control group) were used. The herds (both the ARLA group and the control group) had been composed of sheep obtained from different farms by the owners.

In routine clinical examinations the pulse and respiratory rates, the number of rumen contractions, rectal temperature and dehydration degrees were determined in all animals.

*Rumen fluid collection and examination.* The samples of rumen fluid were taken from sheep with ARLA and healthy sheep through a stomach tube. The first portion coming from the tube was discharged to minimize the saliva contamination of these samples and the remaining portion of 200-250 mL was examined immediately (GARRY, 2002; KARAPINAR et al., 2008).

These ruminal fluid samples were analysed in terms of odour, colour, pH, methylene blue reduction time and protozoal activity. The odour of the samples was physically examined and classified as aromatic (normal) and acidic. The colour was classified as oily brownish green (normal) or milky grey (abnormal). The pH of the samples was measured with commercial test strips (ColorpHast, Merck KGaA, Darmstadt, Germany) and was classified as normal (6-7), moderately low (5-5.9) and very low (4-4.9). For the methylene blue reduction time of the samples, 1 mL methylene blue of 0.03% was mixed with 20 mL rumen fluid and mixed with rumen fluid in another tube for the colour. While methylene blue reduction time was classified as 3-6 minutes (normal), 6-9 minutes and above >9 minutes, the protozoal activity in the rumen fluid was microscopically evaluated (x40 magnification) and classified as good, reduced and absent (Table 2).

According to their medical history the animals took feed (barley) containing a high amount of carbohydrates. The features of the acidotic rumen fluid (acidotic odour, milky grey colour, reduced pH (≤5), a decrease in protozoal activity or its absence, >6 methylene blue reduction time) were taken into account as findings of ARLA in sheep (KARAPINAR et al., 2008).

*Blood sample collection.* Blood samples were obtained from the jugular vein of all animals. The blood samples were centrifuged at 3000 g for 15 minutes and their serums

were separated. They were preserved at -20°C until their cTnI concentration and enzyme activities (ALT, AST, CK-MB and LDH) were determined.

*cTnI analysis.* cTnI concentration in serums was measured by commercial immunoassay system, using the one-step sandwich method (Unicel Beckman Coulter Access II, USA). The normal range is accepted to be 0.04 ng/mL, (measurrement range, 0.01-100 ng/mL) and their positive and negative status was determined accordingly. The samples below this value were accepted as negative and the samples above this value were accepted as positive.

*Serum biochemistry.* Serum enzyme [CK-MB (CK IFCC method plus Immuno-inhibition), LDH (L-P'a IFCC), ALT (IFCC; with/without pyridoxal Phosphate Activation) and AST (IFCC; with/without pyridoxal Phosphate Activation)] activities were determined using, commercial enzyme kits with a biochemistry autoanalyzer (Beckman Coulter, AU5800, USA).

*Statistical analysis.* The comparison of the data between the two groups was conducted with the SPSS package program using the Mann Whitney U test, which is a nonparametric test (SPSS, Version 11.5 Microsoft, 2002, Chicago, IL). The significance degree between the two groups was determined to be $P<0.05$. The data were presented as an average and standard deviation ($X \pm SD$).

## Results

Table 1. Clinical signs of sheep with acute ruminal lactic acidosis (ARLA) (n = 20) and in control group (n = 20)

| Clinical parameters | ARLA group | Control group |
|---|---|---|
| Rectal temperature (T/°C) | Increased in the whole group | Normal in the whole group |
| Heart rate (P/min.) | Increased in the whole group | Normal in the whole group |
| Respiration rate (R/min.) | Increased in the whole group | Normal in the whole group |
| Ruminal stasis | Available in the whole group | None |
| Ruminal atony | Present in the whole group | None |
| Ruminal contractions (Rh/5 min.) | Decreased in the whole group | Normal |
| Mucous membranes | Dirty hyperemic in the whole group | Normal |
| Scleral congestion (engorged scleral vessels) | Present in the whole group | None |
| Diarrhoea | In ten animals | None |
| Dehydration | From moderate to severe levels present in the whole group | None |
| Teeth grinding | In some animals | None |
| Death | In seven animals | None |

*Clinical signs.* The clinical signs of the sheep with ARLA and sheep in the control group are given in Table 1. In the clinical examination of the sheep with ARLA, in all the animals engorged scleral vessels, dirty hyperemic mucous membranes and dehydration from moderate to severe level, increase in heart and respiratory rates, ruminal atony, ruminal stasis and increase in rectal temperature were determined. Moreover, seven sheep died and diarrhoea was found in ten sheep, and teeth grinding was observed in some sheep. In the clinical examination of the control group, it was detected that their rectal temperature, heart and respiratory rates and rumen contractions were at normal levels.

*Ruminal fluid analysis findings.* The results of the ruminal fluid analyses of the sheep with ARLA and sheep in control group are shown in Table 2.

Table 2. Ruminal fluid findings of sheep with acute ruminal lactic acidosis (ARLA) (n = 20) and in control group (n = 20)

| | | Frequency (%) | |
|---|---|---|---|
| Ruminal fluid parameters | Classification | ARLA group | Control group |
| Odor | Aromatic | 0 | 100 |
| | Acidic | 100 | 0 |
| Color | Olive, brownish-green | 15 | 100 |
| | Milky grey | 85 | 0 |
| pH | Normal (6-7) | 0 | 100 |
| | Moderately low (5-5.9) | 15 | 0 |
| | Very low (4-4.9) | 85 | 0 |
| Protozoal activity | Good | 0 | 100 |
| | Reduced | 20 | 0 |
| | None | 80 | 0 |
| Methylene blue reduction time | 3-6 minutes (normal) | 0 | 100 |
| | 6-9 minutes | 30 | 0 |
| | > 9 minutes | 70 | 0 |

*cTnI and biochemical findings.* cTnI, CK-MB, LDH, AST and ALT concentrations of the sheep with ARLA and of the healthy sheep were presented in Table 3. When the sheep with ARLA (0.103 ± 0.080 ng/mL) were compared to the healthy sheep (0.035 ± 0.015 ng/mL), it was observed that the serum cTnI concentration of the sheep with ARLA was substantially elevated ($P<0.0001$). It was determined that serum CK-MB activity in sheep with ARLA (454.50 ± 191.74 U/L; 224.60 ± 94.88 U/L) had risen substantially in comparison to sheep in the control group ($P<0.0001$). It was determined that LDH and AST activities of the sheep with ARLA (448.25 ± 80.39 U/L; 113.10 ± 26.09 U/L) had

increased substantially in comparison to the sheep in the control group (365.45 ± 58.312 U/L; 86.45 ± 17.84 U/L) (P<0.001). Although it was observed that serum ALT activity increased substantially in sheep with ARLA (18.50 ± 4.89 U/L) in comparison to sheep in the control group (14.25 ± 4.60 U/L), it was determined that this was within the reference range (P<0.01).

Table 3. Serum cTnI concentration and enzyme activities in sheep with acute ruminal lactic acidosis (n = 20) and in control group (n = 20)

| | ARLA group | Control group | |
|---|---|---|---|
| Biochemical parameters | X ± SD | X ± SD | P value |
| cTnI (ng/mL) | 0.103 ± 0.080 | 0.035 ± 0.015 | P<0.0001 |
| CK-MB (U/L) | 454.50 ± 191.74 | 224.35 ± 83.33 | P<0.0001 |
| LDH (U/L) | 448.25 ± 80.39 | 365.45 ± 8.312 | P<0.001 |
| AST (U/L) | 113.10 ± 26.09 | 86.45 ± 17.84 | P<0.001 |
| ALT (U/L) | 18.50 ± 4.89 | 14.25 ± 4.60 | P<0.01 |

**Discussion**

It has been reported that myocardial damage in sheep is caused by some viral (BRELLOU et al., 2007; KARAPINAR et al., 2012) and parasite factors (DUBEY, 1988), some nutritional deficiencies (TUNCA et al., 2009; GUNES et al., 2010), some toxic agents (JONES, 2001; HAJIMOHAMMADI et al., 2014) and ARLA (DSHUROV, 1976).

It has been determined that severe focal myocarditis develops in sheep with ARLA (DSHUROV, 1976). Although clinical signs and ruminal fluid findings are sufficient for the diagnosis of the disease, the dimension of the cardiac damage is not known and prognosis of the disease cannot be fully clarified. For this reason, the present study aimed to evaluate serum cTnI concentration to determine the presence of probable myocardial damage in sheep with ARLA.

Clinical signs and ruminal fluid analyses in the diagnosis of the disease in sheep with ARLA (KARAPINAR et al., 2008; CONSTABLE 2010), and electrocardiography (ECG) for determination of the disorders that develop in the heart due to severe metabolic acidosis are important (JAFFARI-DEKHORDI et al., 2011; ONMAZ et al., 2011). In the same way, in the study, the diagnosis of ARLA was made by evaluating the clinical signs and ruminal fluid analyses (Table 1 and 2).

It has been said that serum and plasma cTnI concentrations increase substantially in cases of diseases in sheep which cause myocardial damage, with various aetiologies, and cTnI is an important and sensitive prognostic and diagnostic cardio marker for the diagnoses of these diseases (TUNCA et al., 2009; KARAPINAR et al., 2012).

It has been determined that plasma cTnI concentrations (146.78 μg/L) rise in lambs with severe focal myocarditis related to foot-and-mouth disease (KARAPINAR et al., 2012). It has been shown that cTnI concentrations (10.49 ng/mL) increased signficiantly in the serum of lambs with nutritional myopathy (TUNCA et al., 2009). It has been found that the increase in plasma cTnI concentration is high on the first day and cTnI concentration moves close to the physiological limit towards the 14$^{th}$ day in a study of the myocardial ischemia process in sheep (LEONARDI et al., 2008). ONMAZ et al. (2011) have reported that in sheep with experimentally induced ARLA, important changes occur in ECG parameters due to metabolic acidosis but positiviy on qualitative cTn kits and cTnI levels indicated myocardial damage is not found and cTns cannot be determined in some cases..

The systemic acidosis and endotoxemia in ARLA causes inflammation and degeneration in the heart, which is a parenchymatous organ (DSHUROV, 1976; NOUR et al., 1998; GARRY, 2002). Moreover, it has been shown that pyogenic liver abscesses may cause severe endocarditis (NAGAJARA and CHENGAPPA, 1998). In the present study, serum cTnI concentrations in sheep with ARLA (0.103 ± 0.080 ng/mL) were found to be at high levels in comparison to the control group (0.035 ± 0.015 ng/mL) ($P<0.0001$). Other cardio markers, CK-MB ($P<0.0001$) and LDH ($P<0.001$) activities, increased substantially in sheep with ARLA in comparison to sheep in the control group (Table 3). The increase in enzyme activities in parallel to an increase in cTnI concentration, may show the presence of myocardial damage.

Depending on damage to the rumen wall, microorganisms scatter in the rumen and endotoxins proceed to the systemic circulation, reach the liver through the vena portae and cause severe damage to liver parenchyma and multifocal abscesses (NAGAJARA and CHENGAPPA, 1998; OWENS et al., 1998). These abscesses develop within a week or month (GARRY, 2002; CONSTABLE, 2010). In some studies in which ARLA was experimentally developed in sheep, it was reported that serum liver enzyme activities did not increase substantially (PATRA et al., 1996).

In conclusion, it was determined that serum cTnI concentrations increased in sheep with ARLA and it was concluded that cTnI could be an important marker to determine the prognosis in sheep with ARLA.

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Received: 26 April 2013
Accepted: 19 December 2013

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**KIRBAS, A., E. BAYDAR, F. M. KANDEMIR, E. DORMAN, O. KIZIL, B. A. YILDIRIM: Procjena serumskoga srčanog troponina I u ovaca s akutnom mliječno-kiselinskom acidozom buraga. Vet. arhiv 84, 355-364, 2014.**

**SAŽETAK**

U bolestima koje zahvaćaju srčani mišić, srčani troponin I (cTnI) poznat je kao osjetljivi biomarker za određivanje oštećenja srčanog mišića. Ipak, nema puno istraživanja o koncentraciji cTnI kao najosjetljivijeg indikatora za oštećenje srčanog mišića kod ovaca s akutnom mliječno-kiselinskom acidozom buraga. Zbog navedenog, u ovom istraživanju namjera je bila procijeniti koncentraciju serumskog cTnI kod ovaca s akutnom mliječno-kiselinskom acidozom. Ovce s dijagosticiranom akutnom mliječno-kiselinskom acidozom (n = 20) u skupini od ukupno 40 akaraman (bijeli karaman) ovaca, u dobi od jedne do dvije godine, činile su pokusnu skupinu bolesnih jedinki. Preostale ovce (n = 20) činile su kontrolnu skupinu zdravih jedinki. Tekućina buraga od svih pretraženih životinja dobivena je sondiranjem i odmah analizirana. Uzorci krvi uzeti su iz jugularne vene te je iz njih izdvojen serum. Koncentracija serumskog cTnI mjerena je komercijalnim imunoenzimnim testom, sendvič postupkom. Aktivnosti serumskih enzima (ALT, AST, CK-MB i LDH) utvrđene su uporabom kliničkog biokemijskog autoanalizatora. Prosječna serumska koncentracija cTnI u kontrolnoj skupini ovaca

bila je na razini 0,035 ± 0,015 ng/mL (raspon: 0,02 - 0,06 ng/mL). U skupini ovaca s acidozom utvrđeno je postojano povećanje koncentracije cTnI koja je dosegla razinu od 0,103 ± 0,080 ng/mL (raspon: 0,03 - 1,7 ng/mL) (P<0,0001). U pokusnoj skupini opaženo je povećanje i drugog srčanog markera CK-MB koji je u ovoj skupini iznosio 454,50 ± 191,88 U/I (raspon: 214 - 861 U/I), a u kontrolnoj 224,35 ± 83,33 U/I (raspon: 133 - 421 U/I) (P<0,0001). Također je ustanovljeno da su ovce s acidozom u odnosu na ovce kontrolne skupine imale povišene jetrene enzime LDH (P<0,001) i AST (P<0,001), odnosno ALT (P<0,01). Zaključeno je da ovce s acidozom imaju povećanu koncentraciju serumskog cTnI koji može poslužiti kao važan biljeg za prognozu bolesti.

**Ključne riječi:** akutna mliječno-kiselinska acidoza, burag, srčani biljeg, cTnI, oštećenje srčanog mišića, ovca