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Acute-Phase Proteins, Oxidative Stress, and Enzyme Activities of Blood Serum and Peritoneal Fluid in Cattle with Abomasal Displacement

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Background: Blood serum and peritoneal fluid acute-phase proteins, oxidative stress indicators, and some enzymes could be used for evaluation of abomasal tissue damage because of displacement in displaced abomasum (DA) cases.

Objectives: The aim of this study was to investigate the concentrations of acute-phase proteins, oxidative stress indicators, and activities of enzymes in blood serum and peritoneal fluid in cattle with right displaced abomasum (RDA) and left displaced abomasum (LDA) and in healthy cows.

Animals: A total of 60 Holstein Friesian cows in early lactation were used, 31 with left and 9 with right displaced abomasum without volvulus diagnosis and no other postpartum disease, and 20 healthy cows as a control.

Materials and Methods: DA diagnosis in dairy cows consisted of physical examination, laboratory, and specific DA tests. Acute-phase proteins, oxidative stress indicators, and enzyme activities were measured in blood serum and peritoneal fluid.

Results: In the RDA group, serum haptoglobin (HPG), serum amyloid A (SAA), malondialdehyde (MDA), adenosine deaminase (ADA), myeleperoxidase (MPO), aspartate aminotransferase (AST), creatine kinase (CK, creatine kinase–MB (CK-MB), and gamma-glutamyl transferase (GGT) activity increased significantly, and serum HPG, MDA, ADA, and AST concentrations increased significantly in the LDA group ($P < .05$). Peritoneal fluid HPG, MDA, ADA, MPO, ALP, GGT, and LDH concentrations increased significantly, whereas NO concentrations reduced significantly in the RDA group, and HPG, MDA, ADA, and TP concentrations increased significantly, whereas concentrations of NO reduced significantly in the LDA group ($P < .05$).

Conclusions and Clinical Importance: There are acute-phase responses, oxidative stress, and abomasal tissue damage because of displacement in DA cases. Especially, HPG, MDA, ADA, and MPO concentrations can provide specific information to help in understanding these changes.

Key words: Acute-phase response; LDA; Malondialdehyde; Myeloperoxidase; RDA.

Displaced abomasum (DA) is a frequent, economically significant and multifactorial disease of high milk-producing cows and 80–90% of cases occur within 3–4 weeks postpartum.[1] Although diseases such as fatty liver, ketosis, and hypocalcaemia are risk factors for displaced abomasum cases,[2,3] the cause of right (RDA) or left displaced abomasum (LDA) cases has not been found by etiology or physiopathology studies.[4] Decreased abomasal contractility, atony, dilatation, decreased ruminal volume, hypocalcemia, endotoxemia, alkalemia, hypergastrinemia, and hyperinsulinemia might be predisposing factors for displacement.[2]

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Abbreviations:

ADA	adenosine deaminase
ALP	alkaline phosphatase
ALT	alanine aminotransferase
AMY	amylase
APP	acute-phase protein
AST	aspartate aminotransferase
BE	base excess
CK	creatine kinase
CK-MB	creatine kinase–MB
Cl	chloride
GGT	gamma-glutamyl transferase
HCO3	bicarbonate
HGB	hemoglobin
HPG	haptoglobin
K	potassium
LDA	left displaced abomasum
LDH	lactate dehydrogenase
MCHC	mean corpuscular hemoglobin concentration
MCH	mean corpuscular hemoglobin
MCV	mean corpuscular volume
MDA	malondialdehyde
MPO	myeleperoxidase
Na	sodium
NO	nitric oxide
O2SAT	oxygen saturation
pCO2	partial carbon dioxide pressure
PCV	packed cell volume
pH	blood pH
PLT	platelet
pO2	partial oxygen pressure
RBC	red blood cell

RDA	right displaced abomasum
RDW	red blood cell distribution width
SAA	serum amyloid A
TP	total protein
WBC	white blood cell

A considerable proportion of DA cases occur on the left side than on the right side.[5] The prognosis of abomasal volvulus cases because of ischemic necrosis of abomasal wall is poor and the survival rate is 61–74%.[3,6,7]

Peritoneal fluid analysis is a useful diagnostic method in abdominal disorders because this fluid generally reflects conditions in the peritoneal cavity. Examination of peritoneal fluid aids in the diagnosis of infectious or chemical peritonitis, intestinal wall infarction, digestive system perforation, bladder rupture, biliary system infiltration, intraperitoneal hemorrhage, and peritoneal neoplasia.[8,9]

Acute-phase proteins (haptoglobin and serum amyloid A), oxidative stress indicators (malondialdehyde and nitric oxide), proinflammatory cytokines, and some enzymes (myeloperoxidase, lactate dehydrogenase, alkaline phosphatase, creatine kinase) can be used to evaluate inflammatory conditions, ischemic effects, and tissue damage caused by obstruction, strangulation, or both in gastrointestinal disorders.[10–13] We tested the hypothesis that changes in oxidative stress, tissue damage, and inflammation in cattle with displaced abomasum can be determined by evaluation of blood serum and peritoneal fluid parameters.

The aim of this study was to investigate the levels of acute-phase proteins, oxidative stress indicators, and some enzymes in blood serum and peritoneal fluid in RDA and LDA cases and in healthy cows.

Materials and Methods

Animals

A total of 60 Holstein Friesian cows at 2–4 weeks postpartum (14–30 DIM) were used in this study; 40 cows (2–6 years old) with displaced abomasum (31 LDA and 9 RDA without volvulus) and no other postpartum disease. As for control group, 20 healthy cows (2–4 years old) were selected from healthy cows in the same farm under the same environmental and management conditions. The control cows were also at 2–4 weeks postpartum (14–29 DIM) and diagnosed to be healthy by analyzing their clinical and hematologic status.

The study was approved by the Ethic Committee of Selcuk University Faculty of Veterinary Medicine. All animals were examined for postpartum diseases (mastitis, metritis, retentio secundinarium, pneumonia, hoof problems, ketosis etc) and positives were excluded.

Blood and Peritoneal Fluid Sampling

Blood was taken by jugular venipuncture. Peritoneal fluid samples from all cows were taken by abdominocentesis. The hair was clipped from umbilical scar region (10 cm caudal to the umbilical scar and 10 cm laterally off the midline toward the right inguinal region). The skin at umbilical scar region was prepared aseptically and was injected subcutaneously with 1 mL of 2% lidocaine. Skin and muscle incision was performed with disposable scalpel blade. Then, a metal tip catheter (10-gauge, 11 cm length) was inserted into the incised site and the catheter was passed through the incision site and a 10-mL sterile syringe was attached to the catheter and gentle aspiration was used to obtain peritoneal fluid. While applying gentle aspiration on the syringe, the catheter was moved gently in its full length and in all directions (dorsally, caudally, or ventrally) within the abdominal cavity to maximize fluid retrieval.

The recovery rate of peritoneal fluid samples was 1–8 mL. After collection, the peritoneal fluid was transferred into a tube and was immediately sent to the laboratory for biochemical analyses.

Diagnosis of Abomasal Displacement

Routine physical examination, laboratory tests (hematological tests and blood gases), and specific DA tests (ping on auscultation and percussion, fluid splashing, liptak test, ultrasonography, and lost percussion area of liver in RDA) were performed for DA diagnosis in dairy cows. Dairy cows with clinical DA diagnosis were sent to the Surgery Clinic of Veterinary Medicine for verification.

Laboratory Analysis

EDTA blood hemogram[a] and gas parameters of heparinized blood[b] were evaluated in DA cases and in control animals. Blood serum and peritoneal fluid (without anticoagulant, 5–10 mL) were analyzed by ELISA tests[c] for haptoglobin,[d] serum amyloid A,[e] malondialdehyde,[f] nitric oxide,[g] adenosine deaminase[h] and myleperoxidase.[i] Alkaline phosphatase (ALP), alanine aminotransferase (ALT), creatinine kinase (CK), creatinine kinase-MB (CK-MB), γ-glutamyl transferase (GGT), lactate dehydrogenase (LDH), amylase (AML), and total protein (TP) levels in blood serum and peritoneal fluid were measured by an autoanalyzer.[j]

Statistical Analysis

Analysis of one-way variance (ANOVA) and the Tukey test were used for data evaluation. The Pearson correlation test was used to evaluate the correlation between the data for the same analyte obtained from blood serum and peritoneal fluid of healthy, RDA, and LDA animals. The level of statistical significance was set at $P < .05$.

Results

Complete Blood Counts and Venous Blood Gas Findings

White blood cell (WBC) counts, red blood cell (RBC) counts, hemoglobin (HGB), red blood cell distribution width (RDW), mean corpuscular hemoglobin concentration (MCHC), partial carbon dioxide pressure (pCO_2), bicarbonate (HCO_3), and base excess (BE) levels were increased significantly ($P < .05$) whereas platelet (PLT) count, partial oxygen pressure (pO_2), oxygen saturation (O_2sat), potassium (K), and chloride (Cl) concentrations were reduced significantly ($P < .05$) in the RDA group compared with the control group. HGB, RDW, mean corpuscular

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Table 1. Hematologic and blood gas parameters of healthy and displaced abomasum animals (mean ± SE).

Parameters	Control (n:20)	RDA (n:9)	LDA (n:31)
WBC $\times 10^3$ mm^3	11.5 ± 0.67^b	13.4 ± 1.58^a	9.27 ± 0.84^{ab}
RBC $\times 10^6$ mm^3	6.73 ± 0.13^b	7.85 ± 0.32^a	7.22 ± 0.17^{ab}
PLT $\times 10^3$ mm^3	419 ± 20.9^a	279 ± 6.51^b	272 ± 14.5^b
PCV%	31.8 ± 0.65	34.8 ± 1.64	33.1 ± 0.96
HGB g/dL	9.39 ± 0.21^b	11.2 ± 0.44^a	11.3 ± 0.28^a
RDW%	18.0 ± 0.26^b	20.8 ± 0.46^a	20.4 ± 0.36^a
MCV fl	47.4 ± 0.75	44.4 ± 1.67	46.1 ± 1.09
MCH pg	13.9 ± 0.27^b	14.4 ± 0.42^b	15.8 ± 0.32^a
MCHC g/dL	29.5 ± 0.24^c	32.5 ± 0.84^b	34.4 ± 0.44^a
pH	7.44 ± 0.01	7.46 ± 0.01	7.43 ± 0.01
pCO_2 mmHg	37.8 ± 0.77^b	42.9 ± 1.95^a	35.8 ± 1.04^b
pO_2 mmHg	39.1 ± 1.95^a	31.2 ± 2.94^b	33.9 ± 1.34^{ab}
O_{2SAT}%	73.4 ± 2.24^a	60.5 ± 6.07^b	65.4 ± 2.02^{ab}
HCO_3 mmol/L	25.1 ± 0.37^b	30.4 ± 2.18^a	23.8 ± 1.09^b
BE mmol/L	0.99 ± 0.42^b	6.66 ± 2.38^a	−0.41 ± 1.24^b
Na mmol/L	141 ± 1.13	139 ± 1.87	143 ± 0.98
K mmol/L	3.49 ± 0.09^a	2.86 ± 0.23^b	3.18 ± 0.08^{ab}
Cl mmol/L	109 ± 1.69^a	98.3 ± 1.74^b	104 ± 1.31^a

Control, healthy animals; RDA, right displaced abomasum; LDA, left displaced abomasum; WBC, white blood cell; RBC, red blood cell; PLT, platelet; PCV, packed cell volume; HGB, hemoglobin; RDW, (red blood cell distribution width); MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; pH, blood pH; pCO_2, partial carbon dioxide pressure; pO_2, partial oxygen pressure; O_{2SAT}, oxygen saturation; HCO_3, bicarbonate; BE, base excess; Na, sodium; K, potassium; Cl, chloride.

[a, b, c]Different letters in the same line are statistically significant (Tukey test, $P < .05$).

hemoglobin (MCH), and MCHC levels were increased significantly ($P < .05$), whereas the PLT count was reduced significantly ($P < .05$) in the LDA group compared with the control group (Table 1).

Serum and Peritoneal Fluid Biochemical Parameters

In blood serum, HPG, SAA, MDA, ADA, MPO, AST, CK, CK-MB, and GGT concentrations were increased significantly ($P < .05$) in the RDA group compared with the control group. HPG, MDA, ADA, and AST concentrations were increased significantly ($P < .05$), whereas ALT and NO concentrations were reduced significantly ($P < .05$) in the LDA group compared with the control group (Table 2). In the peritoneal fluid, HPG, MDA, ADA, MPO, ALP, GGT, and LDH concentrations were increased significantly ($P < .05$) compared with the control group, whereas NO levels were reduced significantly ($P < .05$) in the RDA group compared with the control group. HPG, MDA, ADA, and TP concentrations were increased significantly ($P < .05$), whereas concentrations of NO were reduced significantly ($P < .05$) in the LDA group compared with the control group (Table 3).

The MDA, CK, CK-MB, and GGT enzyme activities in the control group, as well as the MDA levels in the LDA group, in the blood serum and in the peritoneal fluid were positively correlated ($P < .05$) (data not shown).

Table 2. Blood serum biochemical values of healthy and displaced abomasum animals (mean ± SE).

Parameters	Control (n:20)	RDA (n:9)	LDA (n:31)
HPG ng/mL	39.5 ± 14.4^c	1516 ± 105^a	981 ± 90.5^b
SAA ng/mL	23.3 ± 3.73^b	66.7 ± 18.2^a	41.8 ± 6.11^{ab}
MDA mmol/L	4.44 ± 0.16^b	7.51 ± 0.72^a	6.49 ± 0.28^a
NO μmol/L	23.4 ± 2.09^a	18.6 ± 2.73^{ab}	12.9 ± 0.91^b
MPO Eu/mL	1.29 ± 0.48^b	15.8 ± 5.43^a	2.97 ± 1.31^b
ADA U/L	38.8 ± 4.62^b	202 ± 42.8^a	229 ± 27.2^a
ALP IU/L	46.6 ± 2.87^{ab}	60.3 ± 10.9^a	39.1 ± 3.77^b
ALT IU/L	20.0 ± 1.23^a	18.1 ± 1.85^{ab}	14.5 ± 1.15^b
AST IU/L	66.9 ± 1.86^b	136 ± 27.0^a	123 ± 16.5^a
AMY IU/L	52.2 ± 8.21	44.0 ± 4.12	35.1 ± 1.77
CK IU/L	147 ± 16.7^b	575 ± 162^a	358 ± 52.5^{ab}
CK-MB IU/L	244 ± 20.4^b	782 ± 198^a	502 ± 65.3^b
GGT IU/L	21.5 ± 1.51^b	82.1 ± 17.1^a	34.9 ± 3.79^b
LDH IU/L	1621 ± 52.7	1597 ± 157	1533 ± 73.1
TP g/dL	6.79 ± 0.09	6.51 ± 0.24	6.45 ± 0.08

Control, healthy animals; RDA, right displaced abomasum; LDA, left displaced abomasum; HPG, haptoglobin; SAA, serum amyloid A; MDA, malondialdehyde; NO, nitric oxide; MPO, myeleperoxidase; ADA, adenosine deaminase; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; CK-MB, creatine kinase–MB; GGT, gamma-glutamyl transferase; LDH, lactate dehydrogenase; AMY, amylase; TP, total protein.

[a, b, c]Different letters in the same line are statistically significant (Tukey test, $P < .05$).

Discussion

The results of this study indicated that abomasal tissue damage, acute-phase response, and oxidative stress occurred in cattle with left or right displaced abomasum.

In this study, it was concluded that changes in hematological parameters (thrombocytopenia and high RBC, HGB, RDW, and MCHC levels, Table 1) might be as a result of hemostatic dysfunction or disseminated intravascular coagulopathy (DIC). Hemostatic dysfunction and DIC might be as a result of defects in the abomasal mucosa.[14] Lesions in the major abomasal vessels (hemorrhage and thrombosis in gastric and gastroepiploic vessels) because of abomasal necrosis is dependent on the tightness of the volvulus, the duration of ischemia to the abomasum or both rather than on the presence of a thrombosis or rupture of a major vessel;[7] thus, DIC can be an important risk factor for mortality in DA cases.[15] However, for the determination of hemostatic dysfunction and DIC, some specific parameters such as fibrinogen, anti-thrombin III, activated partial thromboplastin time, and prothrombin time in DA cases should be evaluated. High levels of WBC (Table 1) in RDA cases might be the result of an immunological response to endotoxemia, abomasitis, or peritonitis.[16]

It is known that HGP has antioxidant, anti-inflammatory, immune regulation, and antibacterial properties, and thus can be used to detect the presence of disorder/damage, and can aid in preventing a disorder in determining a more precise prognosis and better treatment.[17] The 3 bovine forestomachs and the abomasum can produce APPs, and SAA could be

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Table 3. Peritoneal fluid biochemical values of healthy and displaced abomasum animals (mean ± SE).

Parameters	Control (n:20)	RDA (n:9)	LDA (n:31)
HPG ng/mL	15.2 ± 4.76[c]	1225 ± 147[a]	617 ± 72.8[b]
SAA ng/mL	61.1 ± 10.0	109 ± 19.0	107 ± 17.9
MDA mmol/L	3.85 ± 0.17[c]	6.89 ± 0.63[a]	5.85 ± 0.29[b]
NO μmol/L	60.2 ± 3.97[a]	27.1 ± 14.1[b]	13.9 ± 4.05[b]
MPO Eu/mL	1.48 ± 0.82[b]	15.8 ± 8.63[a]	1.68 ± 0.82[b]
ADA U/L	23.7 ± 3.76[c]	162 ± 24.9[a]	99.6 ± 11.0[b]
ALP IU/L	11.7 ± 2.12[b]	27.2 ± 6.86[a]	17.1 ± 3.59[ab]
ALT IU/L	2.65 ± 0.41	4.11 ± 1.43	3.96 ± 0.65
AST IU/L	33.4 ± 4.19	52.2 ± 11.2	55.4 ± 6.40
AMY IU/L	12.2 ± 1.88	18.6 ± 3.28	14.6 ± 1.38
CK IU/L	127 ± 38.8	132 ± 47.8	90.1 ± 19.5
CK-MB IU/L	146 ± 40.8	182 ± 57.8	118 ± 20.8
GGT IU/L	9.80 ± 1.54[b]	20.0 ± 3.12[a]	16.7 ± 2.22[ab]
LDH IU/L	348 ± 59.2[b]	736 ± 204[a]	482 ± 87.0[ab]
TP g/dL	1.97 ± 0.16[b]	2.66 ± 0.28[ab]	2.87 ± 0.20[a]

Control, healthy animals; RDA, right displaced abomasum; LDA, left displaced abomasum; HPG, haptoglobin; SAA, serum amyloid A; MDA, malondialdehyde; NO, nitric oxide; MPO, myeleperoxidase; ADA, adenosine deaminase; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; CK-MB, creatine kinase–MB; GGT, gamma-glutamyl transferase; LDH, lactate dehydrogenase; AMY, amylase; TP, total protein.

[a, b, c]Different letters in the same line are statistically significant (Tukey test, $P < .05$).

identified only in the abomasum, and expression of HGP was high in the forestomachs and abomasum.[18] It was reported that concentrations of HPG were increased in cattle with DA,[19] HPG and SAA were increased in cattle with DA owing to fatty liver[20] and were increased in cases of fatty liver,[21] mastitis,[22,23] and traumatic reticuloperitonitis.[24] In this study, it was observed that an acute-phase reaction could be generated as a result of tissue damage caused by abomasal tension and increased intraluminal pressure. It is concluded that oxidative stress and the acute-phase reaction, as well as inflammatory response and tissue damage, are severe in RDA cases, but moderate in LDA cases when the increase in hematological parameters, APP, MDA, blood serum, and peritoneal fluid biochemical parameters (Tables 1–3) are taken into consideration.

Ischemia/perfusion stimulates the production of free oxygen radicals, and compounds such as NO and MDA are produced. MDA can be used as a sensor of tissue damage and reperfusion, and NO can be used as a sensor of non-reflow phenomenon.[25] In this study, the increased concentrations of MDA and MPO, as indicators of lipid peroxidation and ischemic damage, were concluded to be significant ($P < .05$) in RDA cases, but not significant ($P > .05$) in LDA cases (Table 2). Levels of NO in both DA groups were lower than those of the control group (Table 2). It was concluded that oxidative stress and apoptosis can be evaluated by the measurement of MDA and NO levels. SAA and HPG concentrations can also be used for evaluation of an inflammatory response.

Concentrations of extracellular adenosine are increased significantly in ischemia, hypoxia, trauma, stress, convulsions, and inflammatory cases.[26] The ADA activity is an important factor in acute and chronic inflammatory responses, and macrophages are its extracellular source.[27,28] The ADA activity in serum or pleural fluid is considered a marker of specific diseases and pathological process.[29–31] It was reported that serum ADA activity is increased in some diseases, including trichophytosis (12.7 U/L),[32] anaplasmosis (8.81 U/L), and theileriosis (16.4 U/L).[33] The ADA activity in pleural fluid with concentrations >40 U/L has a value in diagnosis of tuberculosis in human patients.[31] In this study, there was a significant increase ($P < .05$) in ADA concentration in blood serum and peritoneal fluid in LDA cases (blood serum 229 U/L and pleural fluid 99.6 U/L) and in RDA cases (blood serum 202 U/L and peritoneal fluid 162 U/L) as compared with those of the control group (blood serum 38.8 U/L and peritoneal fluid: 23.7 U/L) (Tables 2 and 3). Increased ADA activity might be related to abomasal tissue inflammation and damage caused by the displaced abomasum. High levels of ADA activity in blood and peritoneal fluid in RDA cases compared with LDA cases may be a useful parameter to evaluate the progression of abomasal damage.

It was reported that the activity of MPO in plasma and peritoneal fluid was increased significantly during pathological displacement of intestines and inflammation in horses.[13] Concentrations of MDA and MPO were increased and concentrations of NO were reduced significantly as a result of tissue damage caused by hypoxia during experimental cases of ischemia-reperfusion damage of the mesenterium of rats.[34] Significantly increased concentrations of MDA and MPO are an indicator of oxidative stress and inflammatory response in relevant tissues during experimental abdominal compartment syndrome in rats.[35] Adenosine release is stimulated by hypoxemic intestinal tissues, and there is a strong correlation between hypoxia and splanchnic ischemia.[36] In this research, severe inflammation, tissue necrosis,[13] and neutrophil response[34,35,37] were observed in abomasal tissue during RDA when the significant increase ($P < .05$) in MPO concentration and WBC in RDA cases was taken into consideration. The nonsignificant MPO increase in LDA cases can be explained because this parameter might be an indicator related to changes in the severity of inflammation, neutrophil response, and tissue necrosis.

An increase in serum CK and CK–MB activity is generally considered related to damage of skeletal muscles or the myocardium[38] and possible changes in activity of this enzyme were reported for colon problems[39] and neoplasia cases.[40] The greater increment in serum CK activity and more severe clinical signs and abomasal damage in RDA cases compared with LDA cases suggest that this increase might be as a result of abomasal damage. Therefore, further research is needed to investigate the relationship

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between CK and CK-MB activity and myocardial ischemia in DA.

Analysis of peritoneal fluid provides useful information for determination of abdominal disorders, propagation, and reasons for inflammation and/or damage.[8,9] LDH activity in the peritoneal fluid might be increased in liver cirrhosis, bacterial peritonitis, and peritoneal tuberculosis. Amylase activity might be increased in pancreatitis, pancreas tumor, and intestinal perforation. ADA activity might be increased in peritoneal tuberculosis and liver cirrhosis. ALP activity and GGT activity might be increased in obstruction, strangulation, intestinal perforation, traumatic hemoperitoneum, hepatitis, and peritoneal carcinoma.[41] In this research, there were significantly increased concentrations of MDA and HPG ($P < .05$) and significantly decreased ($P < .05$) concentrations of NO in LDA and RDA cases. Significantly increased ($P < .05$) ADA activity during LDA and significantly increased ($P < .05$) ADA, MPO, ALP, GGT, and LDH during RDA indicate oxidative/inflammatory changes in the abdomen.[24,35,36,42] The concentration of MDA can be used in the evaluation of oxidative changes in the peritoneal fluid.[43,44] The ADA activity[26,30,36] and the concentration of HPG[24,42] in the peritoneal fluid can be used in the determination of inflammatory response,[34,37,45] whereas ALP, GGT, and LDH enzyme activity can be used in demonstrating tissue damage.[35,41] In consideration of the correlation between blood serum and peritoneal fluid parameters, only LDA cases in the DA group had a positive correlation ($P < .05$), which suggests that, generally, there is no correlation between peritoneal fluid and blood serum parameters in DA cases.

It is concluded that there are acute-phase response, oxidative stress, and abomasal tissue damage because of displacement (obstruction and increased luminal pressure) in DA cases, and evaluation of serum concentrations of HPG, SAA, MDA, NO, ADA, MPO, AST, CK, CK-MB, and GGT, and peritoneal fluid concentrations of HPG, MDA, NO, ADA, MPO, ALP, GGT, and LDH can provide information to help in understanding these changes. Although these results are promising, clinical and prognostic uses of these parameters warrant further research.

Footnotes

[a] Vet MS4-e, Suisse, Switzerland
[b] GEM-Premier 3000, MA
[c] MWGt Lambda Scan 200
[d] HPG, ICL Bovine Haptoglobin Kit, E-10HPT, ICL, Inc, OR
[e] SAA kit, USCN Life Science, E90885Bo, Seattle, WA
[f] MDA, Bioxytech-MDA 586, Oxisresearch, Portland
[g] NO, Colorimetric Assay Kit, 780001 Cayman Chemical Co, MI
[h] ADA, ADA Kit, Biosupply, UK
[i] MPO kit, IMMCO Diagnostics, NY
[j] ILab 300 Plus, Instrumention Laboratory Company, Milan, Italy

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References

1. Geishauser T. Abomasal displacement in the bovine—a review on character, occurrence, aetiology and pathogenesis. J Vet Med A 1995;42:229–251.

2. Van Winden SC, Kuiper R. Left displacement of the abomasum in dairy cattle; recent developments in epidemiological and etiological aspects. Vet Res 2003;34:47–56.

3. Doll K, Sickinger M, Seeger T. New aspects in the pathogenesis of abomasal displacement. Vet J 2009;181:90–96.

4. Geishauser T, Leslie KE, Duffield TF. Metabolic aspects in etiology of displaced abomasum. Vet Clin North Am Food Anim Pract 2000;16:255–265.

5. Constable PD, Miller GY, Hoffsis GF, et al. Risk factors for abomasal volvulus and left abomasal displacement in cattle. Am J Vet Res 1992;53:1184–1192.

6. Constable PD, St-Jean G, Hull BL, et al. Preoperative prognostic indicators on cattle with abomasal volvulus. J Am Vet Med Assoc 1991;198:2077–2085.

7. Sattler N, Fecteau G, Helie P, et al. Etiology, forms, and prognosis of gastrointestinal dysfunction resembling vagal indigestion occurring after surgical correction of right abomasal displacement. Can Vet J 2000;41:777–785.

8. Hirsch VM, Townsend HGG. Peritoneal fluid analysis in the diagnosis of abdominal disorders in cattle: A retrospective study. Can Vet J 1982;23:348–354.

9. Wittek T, Grosche A, Locher LF, Furll M. Diagnostic accuracy of D-Dimer and other peritoneal fluid analysis measurements in dairy cows with peritonitis. J Vet Intern Med 2010;24:1211–1217.

10. Eckersall PD. Measurement of Acute Phase Proteins as Biomarkers of Disease. Arizona: Proceedings of the Annual Meeting of the American College of Veterinary Pathologists And American Society for Veterinary Clinical Pathology - Tucson; 2006.

11. Reeves MJ, Vansteenhouse J, Stashak TS, et al. Failure to demonstrate reperfusion injury following ischaemia of the equine large colon using dimethyl sulphoxide. Equine Vet J 1990;22:126–132.

12. Souza DG, Teixeira MM. The balance between the production of tumor necrosis factor-α and interleukin-10 determines tissue injury and lethality during intestinal ischemia and reperfusion. Mem Inst Oswaldo Cruz 2005;100:59–66.

13. Grulke S, Franck T, Gangl M, et al. Myeloperoxidase assay in plasma and peritoneal fluid of horses with gastrointestinal disease. Can J Vet Res 2008;72:37–42.

14. Wittek T, Furll M, Constable PD. Prevalence of endotoxemia in healthy postparturient dairy cows and cows with abomasal volvulus or left displaced abomasum. J Vet Intern Med 2004;18:574–580.

15. Irmak K, Turgut K. Disseminated intravascular coagulation in cattle with abomasal displacement. Vet Res Commun 2005;29:61–68.

16. Zadnik TA. Comparative study of the hemato-biochemical parameters between clinically healthy cows and cows with displacement of the abomasum. Acta Vet Beograd 2003;53:297–209.

17. Sadrzadeh SMH, Bozorgmehr J. Haptoglobin phenotypes in health and disorders. Am J Clin Pathol 2004;121:97–104.

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18. Dilda F, Pisani LF, Rahman M, et al. Distribution of acute phase proteins in the bovine forestomachs and abomasum. The Veterinary Journal 2011;192:101–105.

19. Stengarde L, Holtenius K, Traven M, et al. Blood profiles in dairy cows with displaced abomasum. Journal of Dairy Science 2010;93:4691–4699.

20. Guzelbektes H, Sen I, Ok M, et al. Serum amyloid A and haptaglobulin concentrations and liver fat percentage in lactating dairy cows with abomasal displacement. J Vet Intern Med 2010;24:213–219.

21. Ametaj BN, Bradford BJ, Bobe G, et al. Strong relationships between mediators of the acute phase response and fatty liver in dairy cows. Can J Anim Sci 2005;85:165–75.

22. Jacopsen S, Niewold TA, Kornalijnslijper E, et al. Kinetics of local and systemic isoforms of serum amyloid A in bovine mastitic milk. Vet Immunol Immunopathol 2005;104:21–31.

23. Suojala L, Orro T, Jarvinen H, et al. Acute phase response in two consecutive experimentally induced *E. coli* intramammary infections in dairy cows. Acta Vet Scand 2008;50:18.

24. Nazifi N, Ansari-Lari M, Asadi-Fardaqi J, Rezaei M. The use of receiver operating characteristic (ROC) analysis to assess the diagnostic value of serum amyloid A, haptoglobin and fibrinogen in traumatic reticuloperitonitis in cattle. Vet J 2009;182: 315–319.

25. Cano CP, Bermudez VP, Atencio HE, et al. Increased serum malondialdehyde and decreased nitric oxide within 24 hours of thrombotic stroke onset. Am J Ther 2003;10:473–476.

26. Yan L, Burbiel JC, Maass A, Müller CE. Basic extracellular adenosine concentrations are in the range of ca. 100 nM, but can dramatically rise up to ca. 100-fold to reach concentrations in the micromolar range under certain, e.g. pathological conditions, such as ischemia, hypoxia, trauma, stress, convulsions and inflammation adenosine receptor agonists: From basic medicinal chemistry to clinical development. Expert Opin Emerg Drugs 2003;8:537–576.

27. Law WR, Valli VE, Conlon BA. Therapeutic potential for transient inhibition of adenosine deaminase in systemic inflammatory response syndrome. Crit Care Med 2003;31: 1475–1481.

28. Conlon BA, Law WR. Macrophages are a source of extracellular adenosine deaminase-2 during inflammatory responses. Clin Exp Immunol 2004;138:14–20.

29. Adanin S, Yalovetskiy IV, Nardulli BA, et al. Inhibiting adenosine deaminae modulates the systemic inflammatory response syndrome in endotoxemia and sepsis. Am J Physiol 2002;282:1324–1332.

30. Tuon FF, Litvoc MN, Lopes MIBF. Adenosine deaminase and tuberculous pericarditis—A systematic review with meta-analysis. Acta Tropica 2006;99:67–74.

31. McGrath EE, Warriner D, Anderson PB. Pleural fluid characteristics of tuberculosis pleural effusions. Heart Lung 2010;39:540–543.

32. Cenesiz S, Nisbet C, Yarim GF, et al. Serum adenosine diaminase activity and nitric oxide level in cows with trichophytosis. Ankara Univ Vet Fak Derg 2007;54:55–58.

33. Guzel M, Askar TK, Kaya G, et al. Serum sialic acids, total antioxidant capacity, and adenosine deaminase activity in cattle with theileriosis and anaplasmosis. Bull Vet Inst Pulawy 2008;52:227–230.

34. Karatepe O, Gulcicek OB, Ugurlucan M, et al. Curcumin nutrition for the prevention of mesenteric ischemia–reperfusion injury: An experimental rodent model. Transplant Proc 2009;41: 3611–3616.

35. Tihan DC, Erbil Y, Seven R, et al. The effect of glutamine on oxidative damage in an experimental abdominal compartment syndrome model in rats. Ulus Travma Acil Cerrahi Derg 2011;17:1–8.

36. Bodnar Z, Keresztes T, Kovacs I, et al. Increased serum adenosine and interleukin 10 levels as new laboratory markers of increased intra-abdominal pressure. Langenbecks Arch Surg 2010;395:969–972.

37. Sahoo G, More T, Singh VK. A comparative study on certain enzymes of the granulocyte from different ruminant species. CIMID 1998;21:319–325.

38. Kerr MG. Veterinary Laboratory Medicine, Clinical Biochemistry and Haematology, 2nd ed. Oxford: Blackwell Science Ltd. 2002.

39. Joseph J, Cardesa A, Carreras J. Creatine kinase activity and isoenzymes in lung, colon and liver carcinomas. BJC 1997;76:600–605.

40. Tsung SH. Circulating CK-MB and CK-BB isoenzymes after gastrointestinal surgery. J Clin Pathol 1982;35:200–203.

41. Burgess LJ. Biochemical analysis of pleural, peritoneal and pericardial effusions. Clinica Chim Acta 2004;343:61–84.

42. Karreman HJ, Wentink GH, Wensing T. Using serum amyloid A to screen dairy cows for sub-clinical inflammation. Vet Q 2000;22:175–178.

43. Jurczuk M, Brzoska MM, Moniuszko-Jakoniuk J. Hepatic and renal concentrations of vitamins E and C in lead- and ethanol-exposed rats. An assessment of their involvement in the mechanisms of peroxidative damage. Food Chem Toxicol 2007;45:1478–1486.

44. Bian GX, Li GG, Yang Y, et al. Madecassoside reduces ischemia-reperfusion injury on regional ischemia induced heart infarction in rat. Biol Pharm Bull 2008;31:458–463.

45. Krawisz JE, Sharon P, Stenson WF. Quantitative assay for acute intestinal inflammation based on myeloperoxidase activity (Abstract). Gastroenterology 1984;87:1334.

19391676, 2012, 6, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/j.1939-1676.2012.01018.x by Turkey National Provision, Wiley Online Library on [22/11/2024]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License