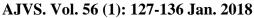


Alexandria Journal of Veterinary Sciences

www.alexjvs.com



DOI: 10.5455/ajvs.285282



New Insights on Left Displaced Abomasum in Dairy Cows

Mahmoud M. Ismael, Ibrahim I. Elshahawy, Ibrahim A. Abdullaziz

Department of Animal Medicine, Faculty of Vet. Medicine, Alexandria University. Egypt

Key words:

Abomasal displacement ultrasound, antioxidants, dairy cows

Correspondence to:

smart_hema2000@yahoo.c om

ABSTRACT

Because left displacements of abomasum cause a great economic losses among dairy cattle production, so this study was aimed to evaluate the main hematological and biochemical alterations, urinalysis, milk ketones, determination acid base balance and ultrasonographic findings of cows with left displaced abomasum (LDA). This study was performed on forty five Holstein dairy cows; fifteen cows were clinically healthy and kept as a control group; the other thirty cows showed signs of abomasal displacement that involve anorexia, sudden drop of milk yield, scanty pasty faeces even some showed diarrhea containing parts of grains, colic and ketotic odour of the breath. By simultaneous percussion and auscultation of the dorsal left flank, particularly on the cranial third of Paralumbar fossa and between 9th and 12th rib, a characteristic tinkling sounds were heard. Ultrasonographically, the displaced abomasum was clearly visible between the left 10th and 12th ICS in all cows. Hypo echoic content of abomasum appeared at left ventral region, gas cap with reverberation artifact appeared at left dorsal region. Laboratory findings of abomasal displaced cows showed a significant hyponatremic hypochloremic hypokalemic metabolic alkalosis with hypocalcaemia, hypophosphatemia, ketonuria and aciduria. Serum values of total protein, albumin, globulins, cholesterol, triglycerides and glucose were significantly decreased. On other hand a significant increase was found in serum concentrations of urea, creatinine, ALT, AST and BHBA in cows suffered from Left Displaced Abomasum (LDA). Oxidantantioxidant study showed a significant increase in the levels of nitric oxide and malondialdehyde while, serum activity of total antioxidant capacity demonstrated a significant decrease in LDA affected cows. LDA increased lipid peroxidation so the using of antioxidants during the treatment of such cases is recommended.

1. INTRODUCTION:

Abomasal displacement is one of the most important metabolic disorders of cattle around the world (Aslan et al., 2003). Feeding of high concentrate diets in early lactation particularly in high producing cows make it more susceptible to LDA. Approximately 90% of cases occur within six weeks following calving. Occasional cases occur few to several weeks before birth (Radostitis et al., 2007). Feeding a large amount of concentrates or corn silage to dairy cows in early postpartum periods, inhibits the abomasal motility resulting gas accumulation followed by dilation and atony; causing displaced abomasum. Once the abomasum is displaced, gas production continues causing distension and further displacement (Cameron et al., 1988 and Radostitis et al., 2007). Other contributing factors decreasing abomasal motility include

metabolic disorders (hypocalcaemia and ketosis), concurrent diseases (mastitis, metritis, retained placenta, or subclinical milk fever), changes of intra-abdominal organs (particularly in late pregnancy), and genetic predisposition Delgado-Lecaroz et al. (2000). In cows with LDA, the abomasum becomes partially or completely displaced between the left abdominal wall and rumen, with caudodorsal distension due to accumulation of gas (Geishauser et al., 2000). Double auscultation of the rumen and percussion and swinging auscultation over the left abdominal ribs have a great importance in diagnosis of such cases (Mueller, 2011). Recently, ultrasonography is described as a useful diagnostic tool of LDA in dairy cattle (Braun, 2009). Although various studies have been performed about the effect of abomasal displacement on some biochemical

particularly those that may reflect the nutritional status of dairy cows during post parturient period, there have been few studies to assess oxidant-antioxidant status in the left displaced abomasum cows. So, the present study was designed to evaluate the oxidative effect with other associated metabolic parameters and blood gas analysis in cows with left displaced abomasum.

2. MATERIAL AND METHODS: 2.1. Animals:

Thirty Friesian dairy cows, 4-7 years old, were used during the present study from February 2015 to March 2017. These cows were admitted with a history of long period of indigestion, scanty Feces, although some cows were diarrheic, and decreased milk yield. All these cases diagnosed as left displaced abomasal (LDA) cases and undergo surgical correction of abomasum with right paralumbar omentopexy technique (Rohn et al., 2004). While another Fifteen healthy Friesian dairy cows were kept as a control group; cows were selected from the same herd and nearly within the same age, breed and calving date.

2.2. Sampling:

2.2.1. Blood Sample:

Three blood samples were collected from jugular vein of each animal. First blood sample collected without anticoagulant (separated serum) were used for measuring serum total proteins, albumin, globulin (by subtracting the albumin from the total protein values), AST, ALT, glucose, Creatinine, Blood urea nitrogen (BUN), Total cholesterol, Beta hydroxyl butyrate (\(\beta \text{HBA} \), triglycerides, Nitric Oxide (No), Malondialdehyde (MDA), total antioxidant capacity (TAC), serum calcium, magnesium, sodium, potassium, chloride and phosphorus. Second blood sample (noncoagulated blood on EDTA) was used for hematological analysis. Third blood sample is venous blood sample was collected into sterilized heparinized vacutainer tubes then gentle inversion of the tubes for several times for total blood gas analysis of left displaced abomasal (LDA) affected cows.

2.2.2. Urine Sample:

Urine was collected from all cows under investigation through spontaneous urination; 5ml was collected in a clean plastic container (Coles, 1986) for determination of pH, protein, glucose, ketone bodies, bilirubin in addition to blood by comber test strips supplied by (Boehringer Mannheim, Germany).

2.2.3. Milk Sample:

Fresh fore quarters strip samples from all cows under investigation were collected after aseptic udder preparation (Denis-Robichaud et al., 2011) for qualitative determination of milk ketone bodies by using Porta BHB milk ketone test strips supplied by (PortaCheck, Inc, Moorestown, USA).

2.3. Clinical examination:-

Full case history and Complete thorough clinical examination of all cows under investigation was performed according to the method described by (Constable et al. 2017) including temperature, pulse, respiration and ruminal movement. simultaneous auscultation and percussion was done which revealed an area of high-pitched tympanic resonance (ping); presented at the line extended from the tuber coxae to the elbow on the left side. On rectal examination a sense of emptiness in the upper right abdomen may be appreciated. The rumen is usually smaller than expected and displaced medially to the right of the midline in cattle with a large LDA. Occasionally, there is chronic ruminal tympany.

2.4. Ultrasonographic examination:-

Sonographic examination was performed by the method described by Braun et al. (1997a) digital Veterinary Ultrasound Scanner machine (Sonoscape Vet-China) linear array transducer (5-10 MHz) was used. After routine skin preparation, ultrasonographic coupling gel (Medico lab- Egypt) applied on the animal's skin during examination, to ensure a good contact between the animal's skin and the probe. Ultrasonographic examination of abomasum was performed in cows at the left side between the 10th and the 12th intercostal spaces. The area was examined ventrally to dorsally with transducer held parallel to the ribs until the abomasum could be imaged.

2.5. Hematological analysis:-

Red blood Cell count (RBCs X10⁶/100ml), hemoglobin concentration (Hb g/l), packed cell (PCV%), mean corpuscular volume volume (MCV fl), mean corpuscular hemoglobin (MCH pg), mean corpuscular hemoglobin concentration (MCHC g/dl), WBCS count $(X10^3/100 \text{ml})$ and differential leucocytic count were determined by using of fully automated veterinary hematology analyzer, Exigo, Boule medical AB., Sweden in the central laboratory, Faculty of veterinary medicine, Alexandria University. Total Blood gas analysis including Blood pH, pCO2 and electrolytes (bicarbonate, sodium, potassium, calcium and chloride) were determined in the heparinized blood sample using blood gas analyzer (ABL90 FLEX, Denmark).

2.6. Biochemical analysis:-

Determination of serum concentrations of calcium, phosphorus, magnesium, sodium, potassium, chloride, AST, ALT, Albumin, Total protien, glucose and creatinine all were carried out by using commercial test kits supplied by (Bio-labo, France), While analysis of βHBA , BUN, Total cholesterol and triglycerides were carried out by using commercial test kits supplied by (Ben-Biochemical Enterprise, Italy) and that of Oxidants/Antioxidant status including TAC, MDA and NO all were analyzed using test kits supplied commercially by (Bio-Diagnostic, Cairo, Egypt) follwing standard methods mentioned in the leaflet of the manufacturer.

2.7. Statistical analysis:-

Data collected were subjected to analysis by T-independent student test to assess significant differences between groups with the aid of **SAS** (2004). All values were expressed as mean \pm standard error (SE). Significance level was set at P \leq 0.05.

3. RESULTS

3.1. Clinical examination:

Clinical examination of diseased cows indicated dullness, poor appetite, sudden drop of milk yield, ketotic breath, scanty pasty faeces and sometimes diarrhea was evident, and left slab-sided abdomen. Diseased cows were afebrile with increased pulse and respiratory rates but some affected cows may show some systemic reaction depending on presence of concurrent disease which was mainly including mastitis, metritis and may retained fetal membranes. Ruminal sounds were reduced (Table 1). Also, high

pitched resonant pings were audible on simultaneous auscultation and percussion of the left dorsal flank in cows with LDA. On rectal palpation, the rumen was displaced medial than normal in LDA affected cows.

3.2. Ultrasonography examination:

Ultrasonography findings revealed that, the abomasal wall appeared as a narrow echogenic line following the abdominal wall and muscles while, the wall of the rumen appeared as a thick echogenic line beside the wall of the abdomen in the ventral region. The rumen was displaced from the abdominal wall by the abomasum on moving the probe dorsally (Fig.1). Dorsally there was gas cap with characteristic reverberation artifacts which appeared as lines run parallel to the surface of the abomasum (Fig.2), while, the ingesta were visualized ventrally and appeared echogenic to hypoechogenic where the abomasal folds may be visible in cows as vague echogenic stripes (Fig.1).

3.3. Hematological analysis

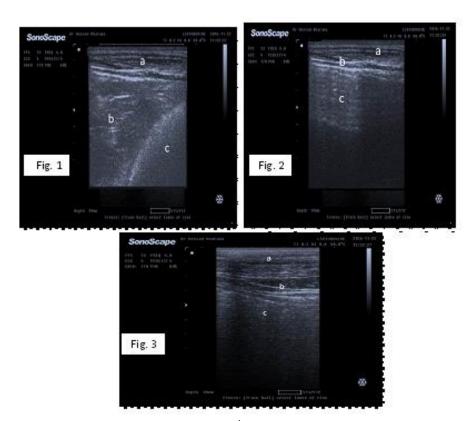
The red blood cells count (RBCS count), MCV, Total lymphocyte count and total number of MID "eosinophils, basophils and monocytes" showed non-significant changes between LDA affected cows and healthy control dairy cows. On other hand the mean values of hemoglobin, packed cell volume (PCV), MCH, MCHC, total leucocytic count (WBCS count) and neutrophils were significantly higher in LDA affected cows than healthy control dairy cows Table (4).

3.4. Biochemical analysis:

The serum calcium, inorganic phosphorus, sodium, potassium, chloride, glucose, total protein, albumin, globulin, cholesterol and triglycerides levels were significantly decreased in LDA group than its level in healthy cows included in this study.

Table (1): Clinical picture of apparently healthy cows and those with LDA.

Groups	Control	LDA
Parameters		
Body temperature °C	38.3 ± 0.4	38.7 ± 0.2
Respiratory rate / min.	21.5 ± 0.5	28.90 ± 0.7
Pulse rate / min.	60.5 ± 0.7	70.3 ± 0.7
Ruminal movement / 2 min.	3	2



Ultrasound of LDA affected cows imaged from the left 12th intercostal space.

- (Fig.1). The abomasum is trapped between the rumen and the left abdominal wall a, left abdominal wall; b, abomasum with hypoechogenic ingesta; c, medially displaced rumen.
- (Fig.2). Dorsal reverberation artifacts at the abomasal surface. a, left abdominal wall; b, abomasal wall; c, reverberation artifacts.
- (Fig.3). Ultrasonography of left abdominal wall after correction of LDA with right paralumbar omentopexy technique. . a, left abdominal wall; b, rumen wall; c, ruminal contents

On contrary the mean values of BHBA, AST, ALT, blood urea and creatinine were significantly increased in abomasal displaced cows. Concerning serum TAC value in LDA affected cows was significantly decreasing, in contrary to NO and MDA levels that significantly increased in affected cows. Results of acid based status revealed presence of metabolic alkalosis in LDA affected cows .This was indicated by the presence of a significant increase in blood pH and bicarbonate with non-significant change in the partial pressure of Co₂ (pCO₂). Tables (3-9).

3.5. Urinalysis:

Urine examination of LDA affected cows revealed ketonuria and aciduria (Table 2).

3.6. Milk ketones:

Fresh Milk samples collected from all examined LDA affected cows revealed positive results for qualitative determination of milk ketone bodies by using PortaBHB milk ketone test strips Table (2).

Table (2): Urinalysis, milk ketone of apparently healthy control and LDA affected cows.

parameter	Control	LDA
pH	7-8	6-6.5
Protein	- ve	- ve
Glucose	- ve	- ve
Ketone bodies	- ve	+++ ve
Blood	- ve	- ve
Bilirubin	- ve	- ve
Milk Ketones	- ve	+ ve

Table (3): Blood Gas Parameters in LDA affected cows.

parameter	LDA Mean ±SE	Reference range*
Blood pH	$7.47 \pm 0.06**$	7.35-7.45
pCO2 mmHg	41 ± 4.01	34-45
Bicarbonate mmol/L	$31.4 \pm 1.04**$	24-30

^{*} Constable et al., 2017

Table (4): Mean values (±SE) of some hematological changes in both healthy control and LDA affected cows.

Group	Control	LDA
RBCS (X10 ⁶ /ml)	5.69±0.10	5.00±0.15
Hb (mg/100ml)	10.94 ± 0.27	12.16±0.57*
PCV (%)	32.60 ± 0.60	39.75±1.16*
MCH(Pg.)	15.08 ± 0.45	17.95±0.26*
MCHC (g/dl)	35.75 ± 0.60	37.25±0.73*
MCV (fl)	48.05 ± 0.52	47.91±0.65
WBCS $(X10^3/ml)$	11.10±0.83	13.32±0.78*
Lymphocytes (X10 ³ /µl)	5.52 ± 0.94	4.19±0.22
$MID(X10^3/\mu l)$	0.98 ± 0.09	1.10 ± 0.10
Neutrophils (X10 ³ /µl)	4.95±1.26	6.04±0.62*

^{*}significant at P<0.05

Table (5): Mean values (±SE) of some Serum minerals and electrolytes in both healthy control and LDA affected cows.

Group	Control	LDA
Ca (mg/dl)	8.92±0.31	6.67±0.55*
Ph. (mg/dl)	5.29 ± 0.30	$3.87 \pm 0.23*$
Mg (mg/dl)	2.73±0.11	2.1±0.11
Na (mmol/l)	133.03±2.34	103.92±0.83*
K (mmol/l)	3.77 ± 0.17	3.15±0.53*
CL (mmol/l)	96.46±1.49	89.49±1.15*

^{*}significant at P<0.05

Table (6): Mean values (±SE) of oxidants /antioxidant profile in both healthy Control and LDA affected cows.

Group	TAC (Mm/l)	NO (µmol/l)	MDA (nmol/ml)
Control	0.8 ± 0.043	13.08±0.40	8.02±0.24
LDA	0.35±0.28*	35.55±0.65*	26.51±1.56*

^{*}significant at P<0.05

Table (7): Mean values (±SE) of some biochemical changes in both healthy control and LDA affected group.

Group	Control	LDA
Cholesterol (mg/dl)	162.05±13.49	85.96±7.62*
Triglycerides(mg/dl)	24.18±1.25	17.88±1.83*
BHBA(mg/dl)	17.44 ± 0.70	33.38±3.70*
Glucose (mg/dl)	52.27±1.49	38.70±8.16*

^{*}significant at P<0.05

Table (8): Mean values (±SE) of some liver function parameters in both healthy Control and LDA affected cows.

Group	Control	LDA
Total Proteins (g/dl)	7.60 ± 0.19	5.34±0.43*
Albumin (g/dl)	4.06 ± 0.20	2.96±0.04*
Globulins (g/dl)	3.54 ± 0.16	2.38±0.33*
ALT (u/l)	26.27±1.91	69.91±2.45*
AST (u/l)	49.53±4.20	182.52±31.39*

^{*}significant at P<0.05

^{**}Detectable changes between examined cows compared to reference range.

Table (9): Mean values (±SE) of some renal function parameters in healthy Control and LDA affected cows.

Group	Urea (mg/dl)	Creatinine (mg/dl)
Control	39.44±1.09	1.00±0.03
LDA	48.64±4.55*	1.37±0.08*

^{*}significant at P<0.05

4. DISCUSSION:

Displaced abomasum can cause economic losses in dairy cattle due to production losses, treatment costs and premature culling. Abomasal displacement frequently occurs in high producing cows during early lactation (Veysi et al., 2003). Cases with displaced abomasum mostly produced within 4 to 6 weeks after calving, these was previously confirmed by Constable et al. (1991), Zadnik (2003), and El-Attar et al. (2007) these may be related to the feeding behavior. In the late pregnancy, animal diet consists mainly of roughage, while after birth the ration is rich in concentrates or corn silage and its feeding with large amount inhibits abomasal motility with subsequent gas accumulation followed by dilatation and atony ended by displaced abomasum (Veysi et al. 2003).

Diseased cows were afebrile with increased heart and respiratory rates and reduced ruminal movements, these findings confirmed by Goetze and Müller (1990) and El-Attar et al. (2007). Moreover, generally ruminal activity declines during moderate hypocalcemia (Jorgensen et al., 1998) that often occurs with abomasum displacement and is considered a reliable risk factor for its occurrence.

Clinical examination of displaced cows showed reduction in appetite, sudden decrease of milk yield, pasty feces and left side abdominal distention. Moreover, on simultaneous percussion and auscultation of the left flank, particularly in the upper third of the abdomen, a high-pitched resonant ping was audible between ribs 9 and 11 in cases with LDA Mokhber Dezfouli et al. (2013). With rectal examination, the rumen was displaced medially than normal in LDA.

Most observed clinical findings were previously recorded by Jubb et al. (1991) and El-Attar et al. (2007). Cows with displacement showed less frequent defecation with scanty feces (Radostits et al., 2007) because the obstructed abomasum cause delaying the emptying its content (Zadnik 2003 and El-Attar et al., 2007).

By ultrasonography, the abomasum is found ventrally and caudal to the sternum at normal healthy cows Braun et al. (1997b) and is generally characterized by homogeneous contents with moderately echogenic stippling. In this study, cows with LDA, the rumen was displaced by the

abomasum more dorsally and the abomasum was seen between the left abdominal wall and rumen El-Attar et al. (2007). The heterogenic appearance of abomasum was imaged in all cows at the area of the left 10th and 12th ICS this result is similar to OK et (2002). Gas cap accumulation produced reverberation which seen dorsally, and the hypoechogenic-to-echogenic ingesta were visualized ventrally El-Deen and Abouelnasr (2014). Reverberation artifact is due to the abomasal reflecting the ultrasound waves reverberation between the transducer and abomasal surface. It appeared as lines of varying echogenicity running parallel to the abomasal surface and became weaker as the distance from the transducer increased Braun et al. (1997a) and Abdelaal (2014). In some LDA affected cows echogenic bands of abomasal folds were observed. this result is in accordance with Braun (2009).

Hematological parameters showed a significant increase in the PCV, MCH, MCHC and hemoglobin concentration which comes in concern with those obtained by El-Gharibe et al. (1996), El-Attar et al. (2007) and El-Deen and Abouelnasr (2014), these changes was attributed to dehydration and hemoconcentration that resulted from fluid trapping in displaced organ and blockage of the transport of fluid into the duodenum Mokhber Dezfouli et al. (2013).

Displaced abomasum can produces abomasitis, peritonitis and endotoxemia, these results in neutrophilia with subsequent leukocytosis Zanik (2003) and El-Deen and Abouelnasr (2014). Increase the total leukocytic and neutrophils counts in LDA cows may be returned to concurrent diseases such as the circulating bacteria, metabolic toxins and inflammation of peritoneum, uterus and udder Geishauser (1995) and Stengarde (2010).

Hypocalcaemia recorded in LDA affected cows may be returned to the development of metabolic alkalosis which is a risk factor for hypocalcemia via a reduced sensibility of the receptors for parathyroid hormone Horst et al. (1997). Inappetence developed with abomasal displacement with reduced absorption from the gut may produce hypocalcemia Rohrbach et al. (1999) and Delgadolecaroz et al. (2000). Puerperal hypocalcaemia considered as an important

predisposing cause for abomasum displacement Geishauser et al. (1998) and Houe. et al. (2001) as hypocalcaemia decreases the abomasal motility which is the predisposing cause for abomasal displacement Radostits et al. (2000) and Delgado-Lecaroz et al. (2000).

The significant reduction in serum phosphorous concentration may be secondary to phosphorus redistribution as a result of metabolic alkalosis that induces intracellular phosphorus entry, phosphorus serum concentration decreasing Amanzadeh and Reilly (2006) and Mahmoud et al. (2016). While, the serum magnesium level in LDA affected cows was non-significantly altered these results agree with De Cardoso et al. (2008), Mokhber Dezfouli et al. (2013) and Mahmoud et al. (2016).

In Generally, The significant reduction of sodium, potassium and chloride electrolytes in cases of LDA can be attributed to acid-base imbalance, anorexia, sequestration in the abomasum, disturbed ingesta transport combined with impaired general condition of cows. Rohn et al. (2004) and El-Attar et al. (2007).

The obtained results revealed a hypochloremia accompanied the cases suffered from LDA; could be attributed to continuous secretion of chloride ions into the abomasum which was refluxed through omasal canal due to pyloric stenosis or obstruction and consequently returned to rumen. The same results were obtained by (Geishauser and Sech, 1996 and Rohn et al., 2004).

Hypokalaemia in LDA cows may be returned to anorexia or shifting of potassium into cells due to alkalosis to allow hydrogen exchange and its continued loss by the kidney, or may explained by shift in potassium ion from the extracellular to the intracellular fluid spaces because of starvation or impaired renal function as mentioned by Hafez &Mottelib (1994), Delgado- Lecaroz et al (2000) and Fouda et al. (2004).

Reduced sodium level in abomasal displaced cows may be attributed to its reduced absorption from the gastrointestinal tract with continuous secretion of isotonic fluid into the abomasum El-Gharibe et al. (1996). Also, the significant reduction of sodium electrolyte in displaced cases may result from the metabolic acid—base imbalance due to the duodeno-abomasal reflux and endotoxemia (Geishauser and Seeh 1996 and Ohtsuka et al. 1997). Sodium is decreased in cows when sodium is shed via the kidney to compensate early-stage alkalosis Mokhber Dezfouli et al. (2013).

The results of metabolic alkalosis in abomasal displaced cows indicated by significant increase in the power of hydrogen (pH) and bicarbonate with non-significant change in the partial pressure of Co (pCo₂). The small intestine does not secrete pancreatic bicarbonate without stimulation by the passage of nutrients so making an elevation in bicarbonate and producing metabolic alkalosis (Cunningham 2002). Metabolic alkalosis is accompanied by an accumulation of bicarbonate in the extracellular fluid owing to sequestration of abomasal juice or potassium depletion so that hydrogen ions move intracellular to replace the lost potassium Rohn et al. (2004). Metabolic alkalosis with hypochloremia and hypokalemia associated with abomasal displacement could be attributed to abomasal atony, continued secretion of hydrochloric acid into the abomasum, and impairment of flow into the duodenum Mokhber Dezfouli et al. (2013).

Unlike Mahmoud et al. (2016) the serum glucose level in LDA affected cows was significantly decreased than control healthy cows, this findings was in accordance with El- Gharieb et al. (1996) and El-Attar et al. (2007) this hypoglycemia may be secondary to LDA due to starvation and negative energy balance that precede LDA and predispose to it (Stengarde, 2010).

The increased ALT and AST values in abomasal displaced cows were agreeable with those of Zadnik (2003), El-Attar et al. (2007) and Mahmoud et al. (2016). Metabolic imbalances in dairy cows have a strong impact on liver functions and vice versa. so, increased Levels of AST activity and ALT could be attributed to hepatic lipidosis, endotoxemia and hepatocyte damage as well.

Unlike El-Gharibe et al. (1996) and El-Attar et al. (2007), the significant decrease of the serum total protein, albumin and globulins in displaced abomasal affected cows also reported by El-Deen and Abouelnasr (2014) and Mahmoud et al. (2016) that because of hypoalbuminemia which resulted from anorexia with decreased food consumption and decreased albumin synthesis in liver. Also endotoxemia associating some concurrent diseases results in an increase in the permeability of capillaries and shifting of albumin and colloids to outside the capillaries resulting in hypoalbuminemia Cullor (1992) and El-Deen and Abouelnasr (2014).

Hypovolemic condition that causes a reduction in renal blood flow with subsequent increased blood urea (Rohn et al. 2004, Breuking and Kuiper 1980 and El-Attar et al., 2007). Furthermore, it is likely that ammonium absorption from rumen increases when microbial protein cannot be transported to the duodenum (Geishauser et al., 1996 and Rohn et al.,

2004). Dehydration and hypovolemia with the subsequent decreased renal blood flow of abomasal displaced cows causing blood urea and creatinine significantly increased El-Attar et al. (2007). Hypovolemia and hemoconcentration appeared to be likely due to blockage of fluid transport from the abomasum into the duodenum Mokhber Dezfouli et al. (2013).

Increased mean values of BHBA confirmed that cows with LDA suffered from obvious and prolonged disturbance in glucose and lipid metabolism due to the combined pathological influences of endotoxemia, energy imbalance (ketosis) and hepatic lipidosis. Similar results were reported by Itoh et al. (1998). The optimal BHBA concentration in dairy cows is below 1.0 mmol/L Whitaker et al. (1983). The concentration begins to increase when the animal is subjected to enhanced energy stress Zadnik (2003).

Serum cholesterol and triglycerides levels were significantly lower in cows with LDA possibly due to disturbances in the lipid metabolism and consequently the existence of fatty liver. At the onset of lactation most dairy cows undergo negative energy balance due to increased nutrient demand than available feed intake Stengarde et al. (2008). Stored body fat reserve will be mobilized to liver in attempt to meet energy demands which will lead to excessive fat accumulation in hepatic cells resulting in fatty liver and reduced lipoprotein synthesis Itoh et al. (1998), LeBlanc et al. (2005) and Mahmoud et al. (2016).

Abomasal displacement is one of the postpartum diseases which induce stress in cattle and has been specified with an oxidative activity. Biochemically, oxidative stress is associated with increased production of oxidizing species or a significant decrease in the effectiveness of antioxidant defenses with the severity depending on the duration and intensity of such stress Devrim et al. (2012). In the present study the significant elevation of serum levels of NO and MDA and the significant reduction in serum total antioxidant capacity in LDA cows confirming the induced oxidative stress in LDA.

Nitric oxide is a free radical which is generated in biological system by nitric oxide synthases (NOS). Nitric oxide functions as a vasodilator by relaxing smooth muscle in the linings of blood vessels Doll et al. (2009). Recent studies proved that cattle with abomasal displacement have an increased activity of neuronal nitric oxide synthase, the primary controller of smooth muscle tone. Based on these facts, the significant increase in nitric oxide could result from increased its generation through increased NOS production which will dilate blood

vessels to increase blood flow to the compromised abomasum due to gas distention Steven and Rogier (2003) and Mahmoud et al. (2016).

Malondialdehyde is the major product of lipid peroxidation that reflect the oxidative stress in cells and tissues Demir et al. (2003) and El-Deen and Abouelnasr (2014). Serum MDA levels were higher in LDA cattle indicating oxidative stress causing an increase in oxidants and decrease in total antioxidants serum parameters Mamak et al. (2013).

On the basis of results of the present study we can conclude that LDA in dairy cows is associated with acid-base imbalance, metabolic disturbances and impairment of liver and kidney functions. The incidence of abomasal displacement appeared to increase during the first 4-6 weeks after parturition; therefore, great attention should be paid to dairy cows during this critical period by maintaining the animals on positive energy and electrolyte balances, adequate roughage intake and avoiding hypocalcaemia. The disease resulted in pronounced oxidative stress response suggesting oxidative stress that may takes part in the aetio-pathogenesis of left abomasal displacement. Thus, adding of antioxidant therapies may play a potential therapeutic role in prevention and treatment of left abomasal displacement in dairy cows. Finally we can conclude that ultrasonography not only considers an easy, fast and accurate diagnostic aid of LDA in dairy cows but also is considered a good decision tool.

4. REFERENCES

Abdelaal, A.M. 2014. Clinical, laboratory and ultrasonographic investigations of left abomasal displacement in Holstein-Friesian cows. Vet. Med. J. Giza 60:1-17.

Amanzadeh, J., Reilly, J.R.F. 2006. Hypophosphatemia: An evidence-based approach to its clinical consequences and management. Nat. Clin. Pract. Nephrol. 2: 136-148.

Aslan, V., Ok, M., Boydak, M., Sen, I., Birdane, F.M., Alkan, F. 2003. The study on the relationship of abomasal displacement and fatty liver syndrome in dairy cows. Acta Vet. Scand. 44:139.

Braun, U., Pusteria, N., Schonmann, M. 1997a. Ultrasonographic findings in cows with left displacement of the abomasum. Vet. Rec. 141 (13): 331-335.

Braun, U., Wild, K., Guscetti, F. 1997b. Ultrasonographic examination of the abomasum of 50 cows. Vet. Rec. 140 (4): 93-98.

Braun, U. 2009. Ultrasonography of the gastrointestinal tract in cattle. Vet. Clin. North. Am. (Food Anim. Pract.) 25: 567-590.

Breukink, H.J., Kuiper, R. 1980. Digestive disorders following obstruction of flow of ingesta through the

- abomasum and small intestine. Bovine Pract. 15: 139-143
- Cameron, R.E.B., Dyk, P.B., Herdt, T.H., Kaneene, J.B., Miller, R., et al. 1998. Dry cow diet, management and energy balance as risk factors for displaced abomasum in high producing dairy herds. J. Dairy Sci. 81: 132-139.
- Coles, E.H. 1986. Veterinary Clinical Path., W.B. Saunders Company. Philadelphia & London.
- Constable, P.D., Jean, G.S., Hull, B.L., Rings, M.R., Hosffsis, G.F. 1991. Prognostic value of surgical post-operative findings in cattle. Am. Vet. Med. Assoc. 198:2077–2085.
- Constable, P.D., Done, H.S., Hinchcliff, K.W., Grünberg, W. 2017. Veterinary Medicine. A textbook of the diseases of cattle, horses, sheep, pigs, and goats. Elsevier Ltd., 11th Ed. 502-510.
- Cullor, J.S. 1992. Shock attributable to bacteremia and endotoxemia in cattle: clinical and experimental findings. J. Am. Vet. Med. Assoc. 200: 1894-1902.
- Cunningham, J.G. 2002. Textbook of vet. Physiol.3rd Ed. Saund. Philad. 255–279.
- DeCardoso, F.C., Esteves, V.S., De Oliveira, S.T., Lasta, C.S., Valle, S.F., et al. 2008. Hematological, biochemical and ruminant parameters for diagnosis of left displacement of the abomasum in dairy cows from South. Braz. Pesq. agropec. bras. Brasília 43 (1): 141-147.
- Delgado-Lecaroz, R., Warnick, L.D., Guard, C.L., Smith, M.C., Barry, D.A. 2000. Cross-sectional study of the association of abomasal displacement or volvulus with serum electrolyte and mineral concentrations in dairy cows. Can. Vet. J. 41(4):301-305.
- Demir, S., Yilmaz, M., Koseoglu, M., Akalin, N., Aslan D., Aydin, A. 2003. Role of free radicals in peptic ulcer and gastritis. Turk. J. Gastroenterol. 14: 39-43.
- Denis-Robichaud, J., DesCôteaux, L., Dubuc, J. 2011. Accuracy of a New Milk Strip Cow-Side Test for Diagnosis of Hyperketonaemia. Bovine Practitioner 45: 297-100.
- Devrim, A.K., Sozmen, M., Yigitarslan, K., Sudagidan, M., Kankavi, O., Atabay, H.I. 2012. Assessment of TNF-α and leptin gene expression by RT-PCR in blood of cows with left abomasal displacement. Rev. Med. Vet. 7: 368-372.
- Doll, K., Sickinger, M., Seeger, T. 2009. New aspects in the pathogenesis of abomasal displacement. Vet. J. 181:90–96.
- El-Attar, H.M., Yassein, M.A., Ghanem, M.M. 2007. Alterations in the clinical, hematological and biochemical pictures in abomasal displacement in cows in Egypt. Vet. Med. J. 102–109.
- El-Deen, A.M.N., Abouelnasr, S.K. 2014. Clinico-pathological and Ultrasonographic Studies on Abomasum Displacement in Cows. Glob. Vet. 13 (6): 1075-1080.
- El-Gharieb, S.A., Hassan, H.Y., Mettwali, A.M. 1996. Occurrence of cows left sided abomasal displacement in Egypt with special references to diagnostic methods

- and certain hemato-biochemical alteration. Benha Vet. Med. J. 7(2): 1-19.
- Fouda, T.A., Zaghloul, A.I., Karrouf, G.I.A. 2004. Differential diagnosis and correction of left abdominal tympanic resonance in cattle and buffalos. SC.V.M.J. VII (2):527-550.
- Geishauser, T., Seeh, C. 1996. Duodeno-abomasal reflux in cows with abomasal displacement. J. Vet. Med. Ser. A. 43: 445-450.
- Geishauser, T., Shoukri, M., Kelton, D., Leslie, K. 1998. Analysis of survivorship after displaced abomasum is diagnosed in dairy cows. J. Dairy Sci.81:2346–2353.
- Geishauser, T., Leslie, K., Duffield, T. 2000. Metabolic aspects in the etiology of displaced abomasum. Vet. Clin.of North Ame. Food Anim. Prac.16: 255-265.
- Goetze, L., Müller, M. 1990. The therapy of hypovolemic shock in cows with right-sided abomasal displacement. Zentralbl Vet. A. 37(4):300–309.
- Hafez, A.M., Mottelib, A.A. 1994. Some biochemical studies on blood serum, ruminal fluid and urine of cattle with abomasal disorders. Zag. Vet. J. 22:88-95.
- Horst, R.L., Goff J.P., Reinhardt T.A., Buxton D.R. 1997. Strategies for preventing milk fever in dairy cattle, J. Dairy Sci. 80: 1269-1280.
- Houe, H., Stergaard, S., Thilsing-Hansen, T., Jorgensen, R.J., Larsen, T., Jorgensen, T., Agger, JF., Blom, J.Y. 2001. Milk fever and subclinical hypocalcaemia. Acta. Vet. Scand. 42:1–29.
- Itoh, N., Koiwa, M., Hatsugaya, A., Yokota, H., Taniyama, H., Okada, H., Kudo, K. 1998. Comparative analysis of blood chemical values in primary ketosis and abomasal displacement in cows. J. Vet. Med. Ass. 45:293-298.
- Jorgensen, R.J., Nyengaard, N.R., Hara, S., Enemark, J.M., Andersen, P.H. 1998. Rumen motility during induced hyper- and hypocalcaemia. Acta. Vet. Scand. 39:331–338.
- Jubb, T.F., Malmo, J., Davis, G.M., Vawser, A.S. 1991. Left sided displacement of the abomasum in dairy cows at pasture. Aust. Vet. J. Vol. 68(4):140-142.
- LeBlanc, S.J., Leslie, K.E., Duffield, T.F. 2005. Metabolic predictors of displaced abomasum in dairy cattle. J. Dairy Sci. 88:159–170.
- Mahmoud, A.A., Nahed, S.S., Tamer, S.A., Hany, G.K. 2016. Evaluation of Clinical, Serum Biochemical and Oxidant-antioxidant Profiles in Dairy Cows with Left Abomasal Displacement. Asian J. of Anim. and Vet. Advan. 11: 242-247.
- Mamak, N., Devrim, A.K., Aksit, H., Aytekin, I., Yildiz, R. 2013. Levels of antioxidant substances, acute phase response and lipid peroxidation in the left and right abomasum displacement in cows. Polish. J. Vet. Sci. 16: 731-733.
- Mokhber-Dezfouli, M., Eftekhari, Z., Sadeghian, S., Bahounar, A., Jeloudari, M. 2013. Evaluation of hematological and biochemical profiles in dairy cows with left displacement of the abomasum. Comp. Clin. Path.22: 175–179.
- Mueller, K. 2011. Diagnosis, treatment and control of left displaced abomasum in cattle. In Pract. 33: 470-481.

- Ohtsuka, H., Ohki, K., Motoshi, T. 1997. Evaluation of blood acid-base balance after experimental administration of endotoxin in adult cow. Jap. Vet. Med. Sci. 59:483–485.
- OK, M., Arican, M., Turgut, K. 2002. Ultrasonographic findings in cows with left and right displacement of abomasum. Revue Méd. Vét., 153 (1):15-18.
- Radastits, O.M., Gay, C.C., Blood, D., Hinch, K.w. 2000. Abomasal diseases, in Veterinary Medicine. A Text Book of The Diseases of Cattle, Sheep, Pigs, Goats and Horses, 9th ed. 259-345.
- Radostits, O.M., Gay, C.C., Hinchcliff, K.W.E., Constable, P.D. 2007. Veterinary Medicine. A textbook of the diseases of cattle, sheep, goats, pigs and horses. W.B. Saund.Co. 259 and1698.
- Rohn, M., Tenhagen, B.A., Hofmann, W. 2004. Survival of dairy cows after surgery to correct abomasal displacement. Association of clinical and laboratory parameter with survival in cows with left abomasal displacement. J. Vet. Med. Physio. Path. Clin. Med. 51(6):300-305.
- Rohrbach, B.W., Cannedy, A.L., Freeman, K., Slenning, B.D. 1999. Risk factors for abomasal displacement in dairy cows. J. Am. Vet. Med. Assoc. 214:1660–1663.
- SAS, 2004. Users guide statistics. As. Institute Cary, North Carolina. USA.
- Stengarde, L., Traven, M., Emanuelson, U., Holtenius, K., Hultgren, J., Niskanen, R. 2008. Metabolic profiles in five High-producing Swedish dairy herds with a history of abomasal displacement and ketosis. Acta. Vet. Scand. 50: 10.1186/1751-0147-50-31.
- Stengärde, L. 2010. Displaced Abomasum and Ketosis in Dairy Cows-Blood Profiles and Risk Factors. Ph.D. Thesis (Clinical Sciences) Fac.of Vet. Med. and Anim. Sci.Swed.Univ. Of Agri. Sci. –Uppsala.
- Steven, V.W., Rogier, Kuiper 2003. Left displacement of the abomasum in dairy cattle: recent developments in epidemiological and etiological aspects. Vet. Res. Bio. Med. Cen. 34 (1): 47-56.
- Veysi, A., Mahmut, O.K., Murat, B., Ismail, S., Fatih, M.B., Fahrettin, A. 2003. The study on the relationship of abomasal displacement and fatty liver syndrome in dairy cows. Abst. Post. Present. at 11th I.C.P.D.
- Whitaker, D.A., Smith, E.J., Kelly, J.M. 1983. Subclinical ketosis and serum beta hydroxybutyrate levels in dairy cattle. Br. Vet. J. 139: 462-463.
- Zadnik, T. 2003. A comparative study of the hematobiochemical parameters between clinically healthy cows and cows with displacement of the abomasum. Acta. Vet.53(5–6):297–309.