

## EVALUATION OF CLINICAL FEATURES, ULTRASONOGRAPHY AND CARDIAC BIOMARKERS IN TRAUMATIC AND NON-TRAUMATIC PERICARDITIS IN COWS

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**Abstract:** A total of 82 female cows with signs of congestive heart failure (CHF) were examined clinically and echocardiography at Veterinary Teaching Hospital of Zagazig University, Egypt. Sixty-seven were diagnosed as traumatic pericarditis (TP) and 15 as non-traumatic pericarditis (non-TP). Inappetence, fever, abnormal heart sounds, brisket edema were the most common clinical findings of both diseases. Recurrent tympany was an additional clinical sign in cows with TP. Respiratory acidosis, anemia, leukocytosis, and hyperfibrinogenemia were the main altered hematological parameters in both diseases. There are significant increases in all cardiac biomarkers concentration (Cardiac troponin I (CTnI), Cardiac troponin T (CTnT), Creatine kinase myocardial band (CK-MB), Lactic dehydrogenase enzyme (LDH), amino-terminal pro B-type natriuretic peptide (NT-proBNP) and endothelin) in TP and non-traumatic pericarditis (non-TP). Whilst CTnI, CTnT, and CK-MB are the cardiac biomarkers that significantly differentiate between TP and non-TP. In conclusion, clinical evaluation couldn't differentiate between TP and non-TP while ultrasonography and CTnI, CTnT and CK-MB were of high diagnostic value for differentiation between both diseases.

**Key words:** cardiac biomarkers; biochemical alterations; cattle; hematology; traumatic pericarditis; non-traumatic pericarditis

### Introduction

Cattle are more prone to pericarditis than horses or small animals (1). There are two main types of pericarditis included non-traumatic pericarditis (fibrinous pericarditis) and traumatic pericarditis (suppurative pericarditis). In pericarditis, inflammatory fluids are accumulated in the pericardial sac, resulting in restriction of ventricular movement and severe systemic infection (2). The most common cause of non-traumatic pericarditis is the spread of organisms hematogenously via bloodstream (*Pasturella species*, *Haemophilus species* and *Streptococcus species*). The causes of traumatic pericarditis in cattle are traumatic reticulo-peritonitis, which is caused by foreign bodies contaminated with pyogenic organisms from the reticulum entering the pericardium (3).

Pericarditis in general, was thought to be a problem due to a sharp and sudden decrease in milk production, costs of treatment and eventually animal, resulting in significant financial costs, which are usually linked to progressive heart failure. In addition, this condition is a common cause of severe abdominal pain in dairy cattle (4).

The occurrence of such condition increased with pregnancy and parturition, where reticulum contractions and fetal pressure allow the foreign object to penetrate the wall, resulting in local or diffuse peritonitis (2, 5). The disease is suspected when green chop, silage, or hay is produced from fields with old, rusted fences or baling wire, or when pastures are located near areas or sites where buildings have recently been built, burned, or torn down. The grain ration could also be a source due to the presence of sharp objects accidentally (6).

Because an early detection of such condition is difficult, additional diagnostic tools such as pain testing, estimation of cardiac biomarkers and ultrasonographic examination are required (1,7,8). Cardiac biomarkers consider an early diagnostic tool of cardiac disorders which include cardiac troponins (cTn-I, cTn-T, and cTn-C), endothelin, amino-terminal proBNP (NT/pro-BNP), creatine kinase myocardial band (CK-MB), and the lactic dehydrogenase enzyme (LDH) (9, 10). Ultrasonography defines as the most non-invasive diagnostic and prognostic imaging modality of pericarditis (11).

Therefore, the aim of the present study was focused on evaluating clinical presentation, the use of the cardiac biomarkers and ultrasonographic examination in the early diagnosis and prognosis of cows with pericarditis either traumatic or non-traumatic.

## Materials and methods

### *Animals and study design*

Eighty-two female cows aged between 2 and 7 years old were admitted at the Animal Medicine Department, Faculty of Veterinary Medicine, Zagazig University, Egypt with signs of CHF. These cows were subjected to thorough clinical examination (12). Ultrasonographic examination was performed to classify the cows into cows with traumatic pericarditis and cows with non-traumatic pericarditis. Additional 10 apparently healthy cows were included as controls.

### *Blood sampling*

Two venous blood samples were collected by puncture of the jugular vein, one placed in vacuum heparinized tube for complete blood count and separation of plasma and the second one in plain tubes without anticoagulant. After centrifugation of the second one, serum samples were collected and then kept frozen at -20 °C until further analysis. blood sample was collected from the middle coccygeal artery for blood gas analysis.

### *Hematological analysis*

A complete blood count (hematocrit (%), hemoglobin (%), erythrocyte count ( $10^6$ /ml) and total and differential leucocyte count) was carried out on whole blood samples using automated cell counter (*HA-Vet Hematology Analyzer*<sup>®</sup>, *Clindia Systems B.V.B.A, Belgium*).

### *Biochemical analysis*

The activities of serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) and concentrations of total protein, albumin and serum creatinine (SC) besides blood urea nitrogen (BUN) were performed spectrophotometrically according to the standard protocols of the test kits (spinreact ,spain). The concentration of cTnI was determined in samples of serum with a commercial kit (Card-I-kit Combo Test; Aboa Tech). The test was carried out according to the manufacturers' instructions. While cTnT was measured quantitatively using electrochemiluminescence technology 3 generation cTnT (Roche Diagnostics, Mannheim, Germany), the concentration of (NT-pro BNP) was measured using sandwich enzyme immunoassay tests (ELISA) and the concentration of CK-MB and LDH was measured spectrophotometrically with commercial kit. Fibrinogen (Fb) estimated in plasma samples using bovine kits according to (13). Blood gas analysis were carried out on arterial blood samples collected in a syringe contains heparin as anticoagulant. Arterial blood gas indices were estimated using blood gas analyzer (*ST-200 CC Blood Gas Analyzer*<sup>®</sup>, *Sensacore, India*).

### *Ultrasonographic examination*

Ultrasonographic examination was performed for both diseased and healthy cows and on both sides of the thorax according to standardized examination techniques (8). Using a 3.5 MHz convex transducer (WED, WELLD<sup>®</sup>, Shenzhen Well D Medical Electronics Co., Ltd., China), the area of the heart and reticulum was examined at the two sides of thorax (from 3<sup>rd</sup> -7<sup>th</sup> intercostal space) to the elbow level. Animals were not sedated and were kept in a standing position. The reticulum and neighboring tissues were examined. The area of examination was clipped, shaved, and cleaned with alcohol to remove excess oil and then apply coupling gel. Firstly, the probe was placed just behind the xiphoid cartilage area, then to assess reticulum passed the probe caudally and laterally to examine the shape, the contour, and the motility of the reticulum per 2 min., the whole area of the heart was scanned ultrasonographically.

*Statistical analysis*

All data were analyzed using the SPSS software (IBM, SPSS Statistics, Version 22, USA). The data were expressed as mean  $\pm$  standard deviation (SD). One way ANOVA followed by Duncan Multiple Range test were used to differentiate between significant means at  $P < 0.05$ .

**Results***Clinical findings*

In present study, a total of 82 cows were diagnosed and classified based on ultrasonographic examination into 67 (82%) cows with TP and 15 (18%) cows with NTP. All cows in this study were Friesian breed and their ages ranged from 2-7 years old. Descriptive data of cows with traumatic pericarditis and those with non-traumatic pericarditis are shown in Table 1. The clinical features of cattle with traumatic and non-traumatic pericarditis were listed in Table 2.

*Hematological findings*

The mean values of hematological indices in cows with pericarditis and non-traumatic pericarditis are summarized in Table 3. The hematological analysis of the diseased groups showed a significant leukocytosis and neutrophilia in traumatic and non-traumatic pericarditis cows in comparing

to cattle in control group. The cattle with TP and non-TP have significant decrease in eosinophils comparing to control groups. While there is a highly significant decrease in eosinophils in cows with TP than cows with non-TP. A significant decrease in Hb % and a significant increase in PCV % in TP and non-TP cows. Arterial blood gas analyses showed a highly significant increase of pCO<sub>2</sub> and HCO<sub>3</sub> and significant decrease in pH in cattle has TP and non-TP.

*Biochemical findings*

The mean values of biochemical indices in cows with traumatic pericarditis and non-traumatic pericarditis are summarized in Table 3. All the diseased cattle showed highly significant hyperfibrinogenemia in cattle with TP and with non-TP. The levels of serum total proteins and albumin were significantly decreased in diseased groups. The hepatic enzymes showed a significant increase in the activities of AST and ALT. All cardiac biomarkers concentration showed a significant increase in diseased cows (cows with TP and non-TP) compared to control ones. While CTnI ( $2.38 \pm 0.05$  and  $2.27 \pm 0.05$ ), CTnT ( $0.59 \pm 0.07$  and  $0.45 \pm 0.32$ ) and CK-MB ( $270.04 \pm 38.82$  and  $212.6 \pm 12.33$ ) are increased in TP more than non-TP.

**Table 1:** Descriptive data of cows with traumatic pericarditis (n = 67) and those with non-traumatic pericarditis (n = 15) with regard to age, sex, season, pregnancy status and diet. The data are presented as absolute number and its percentage (%)

Variables	Cows with traumatic pericarditis (n = 67)		Cows with non-traumatic pericarditis (n = 15)	
Age	2-5 years	22 (33%)	6 (40%)	
	5-7 years	45 (67%)	9 (60%)	
Sex	Male	9 (13%)	1 (7%)	
	Female	58 (87%)	14 (93%)	
Season	Summer	28 (42%)	8 (54%)	
	Spring	20 (30%)	3 (20%)	
	Winter	10 (15%)	2 (13%)	
	Autumn	9 (13%)	2 (13%)	
Pregnancy	Not pregnant	9 (13%)	1 (7%)	
	Early pregnancy	7 (11%)	2 (13%)	
	Mid pregnancy	12 (18%)	2 (13%)	
	Late pregnancy	39 (58%)	10 (67%)	
Diet	Dry	54 (81%)	12 (80%)	
	Green	13 (19%)	3 (20%)	

**Table 2:** Clinical findings in cattle with traumatic pericarditis (TP n=67) and non-traumatic pericarditis (non-TP n=15)

Variables	Clinical findings	Cows with traumatic pericarditis (n = 67)	Cows with non-traumatic pericarditis (n = 15)
Rectal temperature (°C)	Normal	17 (25%) (38.31±0.07)	5 (33%) (38.1±0.05)
	Slight increase	40 (60) (39.6±0.06)	7 (47%) (39.5±0.08)
	Fever	10 (15) (40.22±0.12)	3 (20%) (40.22±0.09)
Appetite	Sudden anorexia	52 (78)	15 (100%)
	Inappetence	15 (22)	0 (0%)
Milk yield	Normal	21 (31)	5 (33%)
	Sudden drop in milk yield	46 (69)	10 (67%)
Bloat	None	0 (0%)	7 (47%)
	Recurrent bloat	67 (100%)	8 (53%)
Ruminal motility	Normal (2-3/2 min)	4 (6%)	0 (0%)
	Hypo-motile	63 (94%) (0.4±0.2)	15 (100%) (0.57±0.2)
Heart rate	Normal (72–80 beat/Min.)	9 (13%) (68.14±3.6)	0 (0%)
	Increased	58 (87%) (92.29±0.9)	15 (100%) (90±0.93)
Intensity of heart sounds	Normal	17 (25%)	0 (0%)
	Muffled	50 (75%)	15 (100%)
Heart rhythm	Normal	9 (13%)	0 (0%)
	Arrhythmic	58 (87%)	15 (100%)
Pericardial sounds	None	37 (55%)	0 (0%)
	Splashing sounds	17 (25%)	8 (53%)
	Tinkling sounds	13 (19%)	7 (47%)
Jugular veins	Normal	34 (51%)	7 (47%)
	Bilateral distension with pulsation	33 (49%)	8 (53%)
Mucous membrane	Normal	16 (24%)	0 (0%)
	Pale	22 (33%)	7(47%)
	Congested	29 (43%)	8(53%)
Edema	Throat region	10 (15%)	3(20%)
	Brisket	13 (19%)	8(53%)
	Umbilicus	10 (15%)	5(33%)
	Cranial to udder	5 (7%)	5(33%)

### Ultrasonographic examination

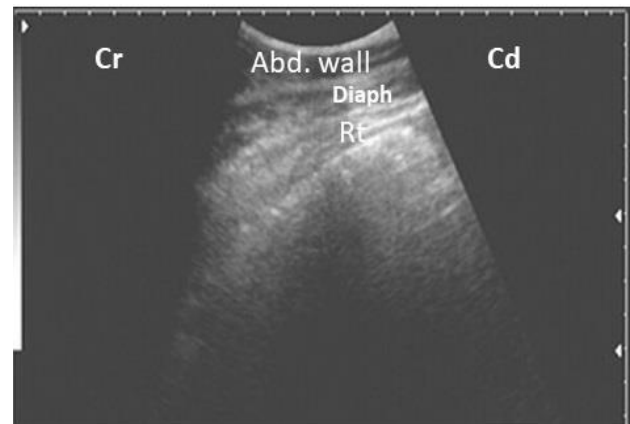
Ultrasonographic examination of reticulum in control cows and in non-traumatic pericarditis cows appeared as a half-moon shaped with smooth contour that contracted with biphasic pattern at a rate of 2 - 3 contraction/2 minutes as in Figure 1. While in cows with traumatic pericarditis, the reticulum appeared corrugated. The motility of the reticulum was monophasic or absent. There was a large amount of fluid with fibrin debris between the reticulum and the dorsal sac of rumen (Figure 2). The cardiac ultrasonography of

the normal heart showed a small distance between the pericardium and epicardium. While in both cows with traumatic pericarditis and those with non-traumatic pericarditis revealed thick pericardium with multiple hyperechoic linear pericardial projections and increased distance between the pericardium and epicardium. Pericardial effusions were also imaged and the fluid in the pericardial sac appeared as either homogeneous anechoic or heterogenous hypoechoic pericardial effusions. Fibrin was imaged as floating shreds within the pericardial effusions or deposited on the epicardium (Figure 3, 4).

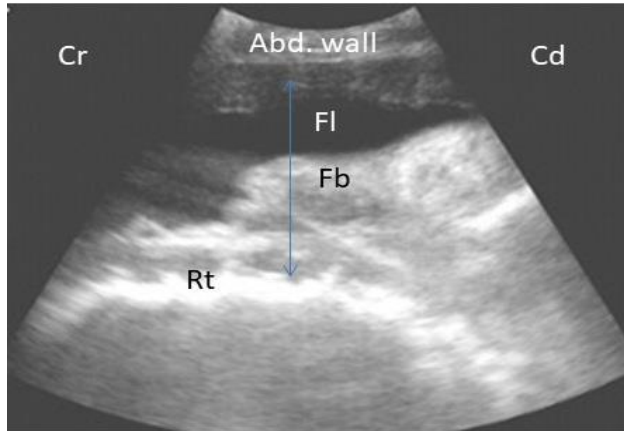
**Table 3:** Hematological and biochemical indices in clinically healthy cows and cows with traumatic pericarditis (n = 67) and non-traumatic pericarditis (n = 15). Results are presented as Mean values  $\pm$  SD.

Parameters	control (n = 10)	Cows with traumatic pericarditis (n = 67)	Cows with non-traumatic pericarditis (n = 15)
Hb (%)	11.5 $\pm$ 0.6 <sup>a</sup>	9.93 $\pm$ 0.23 <sup>b</sup>	9.3 $\pm$ 0.2 <sup>b</sup>
PCV (%)	28.37 $\pm$ 1.03 <sup>b</sup>	32.47 $\pm$ 0.91 <sup>a</sup>	30.87 $\pm$ 1.4 <sup>a</sup>
RBCs ( $\times 10^6$ /ul)	6.85 $\pm$ 0.31 <sup>a</sup>	5.06 $\pm$ 0.05 <sup>b</sup>	5.44 $\pm$ 0.67 <sup>b</sup>
WBCs ( $\times 10^3$ /ul)	6.44 $\pm$ 0.71 <sup>b</sup>	12.71 $\pm$ 0.41 <sup>a</sup>	14.23 $\pm$ 1.83 <sup>a</sup>
Neutrophils (%)	29.06 $\pm$ 1.4 <sup>b</sup>	45.32 $\pm$ 1.1 <sup>a</sup>	47.31 $\pm$ 6.99 <sup>a</sup>
Eosinophil (%)	2.56 $\pm$ 0.39 <sup>a</sup>	0.83 $\pm$ 0.27 <sup>b</sup>	1.99 $\pm$ 0.39 <sup>a</sup>
Lymphocytes (%)	46.32 $\pm$ 2.17 <sup>a</sup>	31.13 $\pm$ 2.25 <sup>b</sup>	25.46 $\pm$ 4.66 <sup>b</sup>
Ph	7.45 $\pm$ 0.03 <sup>a</sup>	7.37 $\pm$ 0.01 <sup>b</sup>	7.38 $\pm$ 0 <sup>b</sup>
pCO <sub>2</sub>	33.87 $\pm$ 1.46 <sup>c</sup>	44.37 $\pm$ 2.1 <sup>a</sup>	39.33 $\pm$ 0.86 <sup>b</sup>
pO <sub>2</sub>	22.17 $\pm$ 1.1 <sup>a</sup>	21.6 $\pm$ 0.72 <sup>a</sup>	21.97 $\pm$ 1.67 <sup>a</sup>
HCO <sub>3</sub>	26.57 $\pm$ 1.56 <sup>b</sup>	32.13 $\pm$ 1.8 <sup>a</sup>	30.53 $\pm$ 0.87 <sup>a</sup>
BE (mmol/L)	2.5 $\pm$ 0.53 <sup>b</sup>	5 $\pm$ 0.7 <sup>a</sup>	4.83 $\pm$ 0.35 <sup>a</sup>
Fibrinogen (mg/dl)	352.67 $\pm$ 58.88 <sup>b</sup>	788 $\pm$ 149.33 <sup>a</sup>	709.67 $\pm$ 96.97 <sup>a</sup>
Total protein (g/dl)	7.08 $\pm$ 0.16 <sup>a</sup>	6.1 $\pm$ 0.12 <sup>b</sup>	6 $\pm$ 0.28 <sup>b</sup>
Albumin (g/dl)	3.43 $\pm$ 0.42 <sup>a</sup>	2.09 $\pm$ 0.17 <sup>b</sup>	1.89 $\pm$ 0.18 <sup>b</sup>
Globulin (g/dl)	3.65 $\pm$ 0.52 <sup>a</sup>	4.01 $\pm$ 0.19 <sup>a</sup>	4.16 $\pm$ 0.2 <sup>a</sup>
AST (u/l)	93 $\pm$ 2.02 <sup>c</sup>	408.8 $\pm$ 9.11 <sup>a</sup>	299.13 $\pm$ 16.49 <sup>b</sup>
ALT (u/l)	31.05 $\pm$ 0.98 <sup>b</sup>	42.31 $\pm$ 2.29 <sup>a</sup>	51.35 $\pm$ 8.87 <sup>a</sup>
Urea (mg/dl)	14.65 $\pm$ 3.07 <sup>b</sup>	30.33 $\pm$ 1.96 <sup>a</sup>	30.53 $\pm$ 1.99 <sup>a</sup>
Creatinine (mg/dl)	0.93 $\pm$ 0.25 <sup>b</sup>	5.97 $\pm$ 1.92 <sup>a</sup>	4.33 $\pm$ 1.04 <sup>a</sup>
CTnI (ng/ml)	0.02 $\pm$ 0 <sup>c</sup>	2.38 $\pm$ 0.05 <sup>a</sup>	2.27 $\pm$ 0.05 <sup>b</sup>
CTnT (ng/ml)	0.07 $\pm$ 0.02 <sup>b</sup>	0.59 $\pm$ 0.07 <sup>a</sup>	0.45 $\pm$ 0.32 <sup>ab</sup>
CK-MB (u/l)	130.59 $\pm$ 1.02 <sup>c</sup>	270.04 $\pm$ 38.82 <sup>a</sup>	212.6 $\pm$ 12.33 <sup>b</sup>
LDH (u/l)	484.32 $\pm$ 33.6 <sup>b</sup>	773.96 $\pm$ 25.2 <sup>a</sup>	740.41 $\pm$ 132.16 <sup>a</sup>
NT/proBNP (pg/ml)	12.13 $\pm$ 0.65 <sup>b</sup>	49.12 $\pm$ 6.8 <sup>a</sup>	49.45 $\pm$ 4.9 <sup>a</sup>
Endothelin (pg/ml)	0.9 $\pm$ 0.06 <sup>b</sup>	9.23 $\pm$ 0.78 <sup>a</sup>	8.99 $\pm$ 0.47 <sup>a</sup>

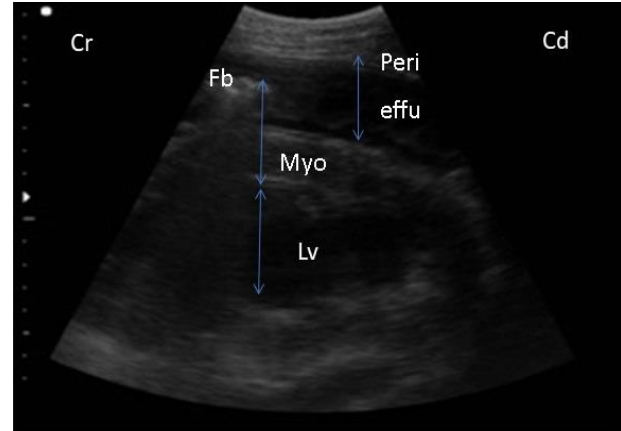
Hb=Hemoglobin, PCV=Packed cell volume, RBCs=Red blood cells, WBCs=White blood cells, AST=Aspartate aminotransferase, ALT=Alanine aminotransferase, CTnI= Cardiac troponin I, CTnT=Cardiac troponin T, CK-MB= Creatine kinase myocardial band, LDH= Lactic dehydrogenase enzyme, NT-proBNP= amino-terminal pro B-type natriuretic peptide



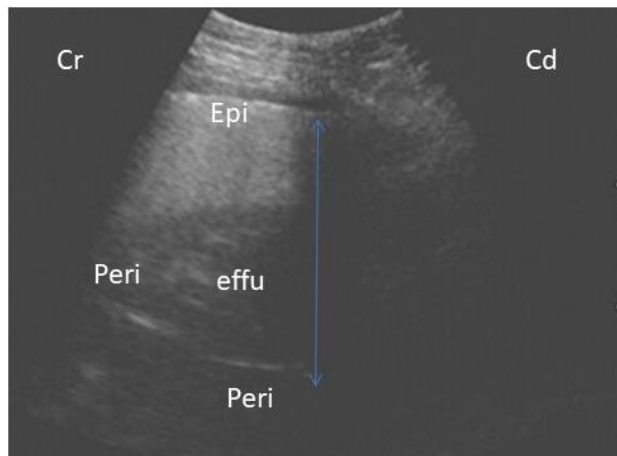
**Figure 1:** Ultrasonographic image of normal reticulum (Rt) in control and non-traumatic cases of pericarditis, notice half-moon shape of the wall, imaged from ventral midline of abdomen, left 7<sup>th</sup> intercostal space (ICS). Abd. wall: Abdominal wall, Diaph: diaphragm, Cr: Cranial, Cd: Caudal



**Figure 2:** Ultrasonographic image of traumatic reticuloperitonitis, thickened and corrugated hyperechogenic reticular wall (Rt). The arrow shows increase distance between reticulum and diaphragm with deposition of the echogenic fibrin (Fb) interspersed with the hypoechogenic fluid (Fl) between the reticulum and cranial sac of the rumen. Imaged from 7<sup>th</sup> ICS at the midline. Abd.wall: Abdominal wall, Cr: Cranial, Cd: Caudal



**Figure 3:** Ultrasonographic image of non-traumatic pericarditis, the arrow show increases the distance between the pericardium (Peri) and epicardium (Epi) by a large amount of heterogeneous hypoechogenic pericardial effusion (Peri effu). Imaged from the left side at 4<sup>th</sup> intercostal space (ICS). Cr: Cranial, Cd: Caudal



**Figure 4:** Ultrasonographic image of traumatic pericarditis, increase the distance between the pericardium and epicardium by a large amount of anechoic pericardial effusion (Peri effu.) and hyperechoic fibrin (Fb) at the pericardial sac. Imaged from the left side at 4<sup>th</sup> ICS. Lv: Left ventricle, Myo: Myocardium, Cr: Cranial, Cd: Caudal

## Discussion

Pericarditis is an inflammation of the pericardium that causes fluid or exudate to accumulate between the visceral and parietal layers of pericardium. It is commonly associated with progressive heart function problems and almost always results in death. In cattle, TP is the most common complication of penetration the gastrointestinal tract by a sharp foreign body then extend to penetrate pericardium (8). The cause of pericarditis is also return to presence of septicaemic microorganism in the blood stream and always the signs of septicemia masking the pathognomonic signs of it (2).

In the present study, the incidence of the disease increased by age this may be attributable to further exposure to foreign bodies (14). Females

were more susceptible to the disease than males and this return to the load of pregnancy and parturition (6).

According to the findings of the present study, 39 (58%) cases were diagnosed in the third trimester of pregnancy. It is possibly due to the combination of weight and height of the gravid uterus, which causes it to behave like a pendulum when a cow gets up and down, creating physical pressure on the rumen and reticulum and leading to perforation by an existing sharp object which allows the foreign body to reach forward and penetrate the pericardium. 54 (81%) cases were found in cows fed a dry feeding diet consisting of chopped roughages and silage that may include old rusted fences or wire (4).

The clinical signs of anorexia, recurrent tympany, tachycardia, dewlap edema and engorgement of jugular vein in pericarditis were in accordance with previous reports (2, 7, 8). The increase in rectal temperature was attributable to toxemia and subsequently the release of inflammatory cytokines causing fever (9). The engorgement of jugular vein and dewlap edema was also evident in animals with pericarditis due to right side cardiac insufficiency and with increase amount of pericardial fluid leading to increase severity of cardiac compression and distension of jugular vein (5). So, case history and clinical signs are not sufficient for the diagnosis of pericarditis.

Due to presence of exudate and fibrin in the pericardium leading to pressure on the heart and according to the amount of the fluid, the tachycardia and muffled heart sound differ in their severity.

Haemato-biochemical analysis indicated leukocytosis and neutrophilia in TP and non-traumatic pericarditis. In case of TP, this return to penetration of the reticulum and the diaphragm by infected foreign body and presence of large amount of exudate causing severe inflammatory reaction (14). While in non-traumatic pericarditis, it might be due to inflammatory changes in pericarditis and endotoxemia due to bacterial infection (15). There were eosinopenia and lymphocytopenia which could be from alteration of lymphocyte kinetics causing decrease in their efflux from lymphoid tissue and redistribution within hematopoietic tissue as a result of increase cortisol level (16). There is significant decrease in eosinophils in TP more than non-traumatic pericarditis which due to stress of penetration leading to reduction in cellular immunity (14). In addition, hypoproteinemia, hypoalbuminemia, hyperglobulinemia and hyperfibrinogenemia were found in diseased cows with increasing the activities of AST and ALT. The results were in agreement with Buczinski et al. (1). The cause was attributed to hepatic dysfunction. Low concentrations of albumin and high concentrations of Fb levels, indicating a severe inflammatory condition. According to (17), the increased activities of AST and ALT may be due to hepatic congestion rather than primary liver disease (3, 18).

The high level of cTnI, cTnT and CK-MB in affected groups due to damage of cardiac myocytes by pressure of pericardial exudate over the

myocytes. Whilst cTnI, cTnT and CK-MB are significantly increased in traumatic pericarditis more than non-traumatic pericarditis due to piercing up of foreign body to the heart (18, 22).

Also, The LDH level elevated due to liver, skeletal muscle, cardiac muscle, and kidney damage. So, due to cardiac myocytes damage and liver dysfunction, there was a significant increase in the LDH level in affected groups.

The significantly high value of endothelin in diseased animals may be returned to the cardiac disease severity which may lead to release of group of vasoconstrictors in blood, such as nor-epinephrine, renin-angiotensin, vasopressin and endothelin which affect kidney function and therefore BUN and creatinine might be increased (9 - 20).

B-type natriuretic peptide (BNP) and amino-terminal proBNP (NT-proBNP) levels in the blood are well-established biomarkers for acutely decompensated heart failure. Both peptides are rapidly released in the plasma of diseased cows with TP at the acute episode as the cardiomyocytes are stretched mechanically, such ventricular overload or increased wall tension, resulting in an increase in the development of the proBNP hormone precursor (pre-proBNP). The significantly high value of NT-proBNP in diseased groups and increased creatinine associated with NT-proBNP concentration, suggesting that this is a useful marker for diagnosing cardiac diseases (11).

Ultrasonography was selected as the gold standard diagnostic tool for identifying heart disease. In the current research, ultrasonography revealed a good tool for detecting the type of heart failure and the size of the lesion. According to the ultrasonographic findings in the present study, the affected cows commonly had a significant amount of hypoechogenic to echogenic pericardial fluid with strands of fibrin on the epicardium. The results were in agreement with the findings reported by Braun (8). In addition, Braun and Goetz (21) had previously identified the ultrasonographic image of the normal bovine reticulum and compared it with the reticular changes that are characteristic of traumatic reticuloperitonitis. Adhesion with the abdominal wall resulted in reticular movement to be reduced or entirely absent. The gap between the epicardium and pericardium increased due existence of hypoechoic

pericardial fluid with fibrin strands. As a result, ultrasonography has been recommended as a reliable tool and process for imaging and assessing the intensity of TP.

## Conclusion

A variety of practical approaches are of great value in early prediction of pericarditis in cattle, the uses of cardiac biomarkers and ultrasonographic examination were ideal diagnostic tools in cows with pericarditis.

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