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Clinical presentation and ultrasonographic findings in buffaloes with congestive heart failure

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Abstract: The objective of this retrospective study was to describe the clinical presentation and ultrasonographic findings in water buffaloes suffering from congestive heart failure. Fifty-three buffaloes with congestive heart failure, which was confirmed postmortem, were included. The final diagnoses were traumatic pericarditis (n = 38) and vegetative endocarditis (n = 15). Ten healthy buffaloes were enrolled in this study as controls. The main clinical presentations were those related to right-sided congestive heart failure including peripheral edema, venous distension and jugular pulsation, and a sharp drop in milk yield. Only 5 buffaloes exhibited signs of left-sided heart failure. Ultrasonographic examinations showed cardiac and extracardiac lesions. Accumulation of massive anechoic fluids in the pericardial sacs, peritoneum, and pleura represented pericardial, peritoneal, and pleural effusions, respectively. In buffaloes with endocarditis, the affected valves appeared as hyperechogenic thick mass structures with nodular or proliferative vegetative lesions. Small (<25 mm) and large (>25 mm) vegetative lesions were imaged. The most affected valve was the tricuspid (n = 8), then the mitral (n = 5) and pulmonary (n = 2). In addition, perireticular abscesses, hepatomegaly, dilated caudal vena cava, and lung consolidations were also imaged. In conclusion, the prognosis seemed poor. Ultrasonography may be helpful in the evaluation of lesions in buffaloes with congestive heart failure.

Key words: Buffaloes, heart, echocardiography, pericarditis, endocarditis

1. Introduction

Congestive heart failure is the terminal stage of various heart diseases that, accompanied with progressive compensatory alterations in the cardiac reserve, lead to negative effects on the myocardium and severe reduction in cardiac output (1). It could be the result of diseases of the endocardium, myocardium, or pericardium that interfere with the flow of blood into or away from the heart, or that impair myocardial function (2). In congestive cardiac failure, reduced myocardial contractility results in the reduction of cardiac output and pulmonary edema when the left ventricle fails, or ascites and/or pitting edema in right-sided congestive failure, when circulatory impairment impedes venous return (3).

Most clinical findings in cattle with heart failure are a consequence of increased hydrostatic pressure (1). These signs are variable and multiple. They include venous congestion, subcutaneous edema, pulmonary edema, general weakness, jugular pulsation, and poor excise tolerance (2). However, in a recent retrospective study of 47 cows with heart diseases, no clinical signs of cardiac failure were noticed (4).

It is of great interest to make a precise diagnosis in the field, as it permits rapid evaluation and decision making regarding treatment options and avoids wasteful supportive treatments in the case of a low-value animal. Ultrasonography is a good choice for imaging and describing the majority of heart diseases (5). Furthermore, cardiac ultrasound examination can be performed easily in field conditions with high sensitivity and specificity (6). Previously, it has been used successfully in the field diagnosis (7,8).

The possible causes of heart diseases were described in a previous study of 59 cows with heart failure (9). Only a few clinical studies of buffaloes with specific heart diseases are available (10). There are no reports listing the possible causes of heart failure in buffaloes. This retrospective study was, therefore, designed to describe the clinical presentations and ultrasonographic findings in water buffaloes suffering from congestive heart failure, determine the possible cause(s), and assign the spread of lesions.

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2. Animals, materials, and methods

2.1. Animals, history, and clinical and postmortem examinations

Fifty-three water buffaloes (*Bubalus bubalis*) were examined at the Veterinary Teaching Hospital, Assiut University, Egypt, between May 2011 and September 2013. The animals ranged in age from 3 to 8 years. Most of the animals were lactating and nonpregnant (n = 44) at different lactation stages of their production cycle; the rest of the animals (n = 9) were pregnant and in the last trimester. Diseased buffaloes were referred to the hospital because of edema, anorexia, sharp drop in milk yield, abdominal distension, and recurrent tympany. The duration of illness varied from 1 to 2 weeks before admission. There was a history of previous medications including antibiotic injections, fluid therapy, and general tonics by the field veterinarians for some diseased buffaloes (n = 15), but without obvious improvement. All animals were examined clinically as described before (11), which included general condition and auscultation of the heart, lungs, rumen, and intestine. In addition, rectal temperature and respiratory and heart rates were also recorded. Testing for foreign bodies in the reticulum or heart included withers pinch technique, strong percussion at the sternum, and mine detector. The general health condition of most of the animals was deteriorated.

Postmortem examination was performed thoroughly on all animals after slaughtering (n = 36) or natural death (n = 17). Postmortem examination was carried out by the first author, except in 14 cases where the authors were unable to conduct a postmortem examination themselves. The postmortem findings of these animals were obtained by telephone conversation with the owners. After complete and thorough physical examinations, 10 buffaloes were included in this study as a control group. These animals did not have a history of previous disease and their physical examination was within normal ranges.

2.2. Hematological and biochemical analyses

Two blood samples were collected from each animal; the first was placed in heparin and the other in plain tubes. The hematocrit, hemoglobin concentration, total erythrocyte count, and total leukocyte count were obtained from the first heparinized sample using a veterinary automated cell counter (Medonic CA620 Vet Hematology Analyzer, Sweden), while differential leukocyte counts were determined manually as described before (12). Venous blood gas indices were estimated using a blood gas analyzer (ABL 5, Radiometer, Denmark) on the first blood sample.

After centrifugation of the second blood sample, serum samples were collected and then frozen at –20 °C for 1 week, after which biochemical parameter analysis was carried out. In the serum samples, commercial test kits were used to determine the concentrations of total proteins, albumin, blood urea nitrogen, creatinine, glucose, total cholesterol, triglycerides, and total bilirubin. The activities of aspartate aminotransferase (AST) and γ-glutamyl transpeptidase (GGT) were also measured in serum samples. The biochemical analyses of the parameters were spectrophotometrically measured according to the standard protocol of the suppliers.

2.3. Ultrasonographic examination

In healthy and diseased buffaloes, ultrasonographic examinations were performed while the animals were standing using 3.5-MHz convex transducers (FF Sonic, UF-4000, Fukuda Denshi Co., LTD, Japan). In preparation for ultrasonography, hair was clipped and shaved on both sides from the 3rd to 11th intercostal spaces and also from the transverse processes of the thoracic vertebrae to the joint of the limb. The area was scrubbed with alcohol to remove excess oil and coupling gel was applied.

Echocardiographic examinations were performed on standing animals according to previous techniques (13). The heart was examined on both sides from the third to the fifth intercostal spaces halfway between the joint of the limb and the shoulder. In the cardiac area, the heart and mediastinal region were thoroughly imaged. Furthermore, the tricuspid, mitral, pulmonary, and aortic valves were also examined.

Ultrasonography of the abdomen was carried out on the ventral part of the thorax on both sides parasternally and along the left and right thoracic wall in the sixth and seventh intercostal spaces, using a 3.5-MHz convex transducer. The reticulum and its contractions, the ruminal atrium, the ventral sac of the rumen, and the spleen, liver, and peritoneum were evaluated as previously (6).

Examination of the pleura and lungs was carried out as described previously (14). Each lung was examined dorsoventrally with the transducer held parallel to the ribs. The lung was considered normal if the characteristic pattern of well-ventilated lung tissue with a smooth visceral surface was observed and if a pleural reflective band and reverberation artifacts were seen (15). A lesion was considered homogeneous when fine-grained, homogeneous, echogenic zones were seen. The lesion was considered nonhomogeneous when anechoic and hyperechoic coarse-grained zones were seen. Fluid was considered to be present when an anechoic zone was seen.

2.4. Diagnosis

Based on the clinical examinations, including ventral edema, venous congestion, tachycardia, and abnormal heart sounds as well as ultrasonographic and postmortem findings, diseased buffaloes were classified into 2 groups: group 1 with traumatic pericarditis (n = 38) and group 2 with vegetative endocarditis (n = 15).

2.5. Statistical analysis

The data were statistically analyzed using SPSS. For hematological and biochemical parameters, data were tested for normality of distribution using the Kolmogorov–Smirnov normality test. Mean and standard deviation for each variable was estimated. Differences between groups were assessed by one-way analysis of variance with post hoc LSD multiple comparison test. Differences were considered significant at $P \leq 0.05$.

3. Results

3.1. Clinical presentation and clinical findings

Clinical presentations and findings in 53 buffaloes with heart failure are listed in Table 1. At admission, the general condition of animals was deteriorated. The most observable clinical presentations were presence of edema at brisket and/or intermandibular space (bottle jaw) (64%) and a sharp drop in milk yield (70%). The appetites of all the animals were abnormal, with either anorexia (57%)

Table 1. Clinical presentation and clinical findings in buffaloes with congestive heart failure.

Variable	Traumatic pericarditis (n = 38) Number of buffaloes (%)	Vegetative endocarditis (n = 15) Number of buffaloes (%)
Clinical presentation		
Anorexia	21 (55)	9 (60)
Edema	29 (76)	5(33)
Inappetence	17 (45)	6 (40)
Recumbency	3 (8)	1 (6)
Cough and respiratory distress	2 (5)	4 (27)
Diarrhea	7 (18)	2 (13)
Weight loss	33 (87)	12 (80)
Sharp drop in milk yield	27 (84)	10 (83)
Abdominal distension	22 (58)	5 (33)
Constipation	5 (13)	3 (20)
Recurrent tympany	15 (39)	0 (0)
Lameness	0 (0)	4 (27)
General weakness	18 (47)	8 (53)
Depression	13 (34)	7 (47)
Clinical findings		
Increased rectal temperature (°C) (>39.5 °C)	23 (61)	8 (53)
Increased respiratory rate (breaths/min) (>33 breaths/min)	2 (5)	4 (27)
Heart rate (beats/min) (>100 beats/min)	28 (74)	10 (67)
Muffled heart sounds	25 (66)	3 (20)
Pericardial frictional sounds	10 (26)	0 (0)
Pericardial splashing sounds	3 (8)	0 (0)
Cardiac murmurs	1 (3)	11 (73)
Cardiac arrhythmia	32 (84)	11 (73)
Impalpable heart beat	27 (71)	2 (13)
Jugular and milk vein congestion and pulsation	33 (87)	13 (87)
Cyanosed mucous membranes	29 (76)	10 (67)
Positive pain tests*	35 (92)	1 (7)
Brisket edema and/or bottle jaw	29 (76)	5 (33)
Arched back posture	27 (71)	3 (20)
Engorged scleral vessels	30 (79)	9 (60)
Pneumonia and respiratory distress	8 (21)	4 (27)
Positive foreign body test (mine detector)	34 (89)	0 (0)
Ruminal atony	33 (87)	11 (73)
Dehydration	17 (45)	4 (27)

*Diseased animal elicits grunting and tearing with protrusion of tongue.

or inappetence (43%). The clinical presentations of some gastric upsets in the form of diarrhea (17%), constipation (15%), or abdominal distension (51%) were observed; furthermore, recurrent tympany (28%) was noticed only in buffaloes with traumatic pericarditis. After thorough physical examinations, the main clinical findings of right-sided heart failure, including jugular and milk vein distensions (87%) and jugular distension and pulsation (79%) (Figure 1), were especially noticeable in buffaloes with traumatic pericarditis. Thirty-five buffaloes (92%) out of 38 with traumatic pericarditis and 1 buffalo (7%) out of 15 with vegetative endocarditis showed signs of pains (grunting, tearing, reluctance to move, and protrusion of tongue) at upward percussion of the reticulum or pinching of the withers. The heart rates were higher than normal in 38 (72%) of the animals. The heart sounds were muffled in 28 (53%) of the animals, and pericardial frictional sounds were audible in 10 buffaloes (19%) and splashing in only 3 cases (6%). Cardiac murmurs (systolic and or diastolic) were heard only in 12 animals (80%) who suffered from vegetative endocarditis. Abnormal respiratory sounds in the form of coughs, wheezes, or crackles and tachypnea were noticed in 6 buffaloes (11%).

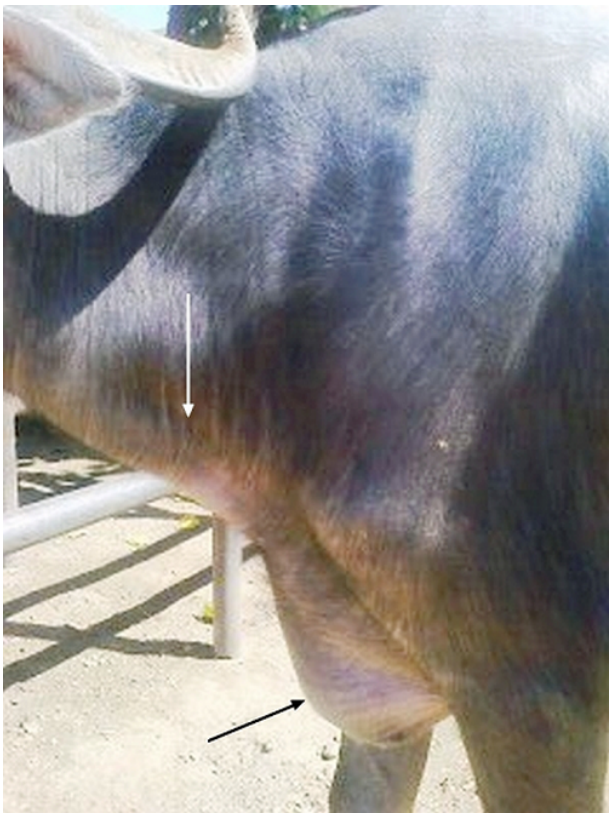

Figure 1. Edema of the brisket region (black arrow) and distended jugular vein (white arrow) are a common clinical presentation in buffaloes with congestive heart failure.

3.2. Hematological and biochemical findings

Table 2 shows the mean hematological and biochemical findings in diseased animals. Hematologically, anemia (erythrocyte count of <5 T/L) was observed in 10 buffaloes with traumatic pericarditis. In comparison with healthy control animals, buffaloes with traumatic pericarditis and vegetative endocarditis showed leukocytosis with regenerative shift to the left ($P < 0.05$). In addition, lymphocyte counts (6.2×10^3 µL) significantly increased in animals with traumatic pericarditis ($P < 0.05$), but it was within the reference range ($3.0–7.5 \times 10^3$ µL). Biochemically, elevated total proteins and hypoalbuminemia were recognized in 30 (79%) and 9 (60%) buffaloes with traumatic pericarditis and vegetative endocarditis, respectively. Hyperglobulinemia was noticed in 34 (89%) and 13 (87%) animals with traumatic pericarditis and endocarditis, respectively. Serum biochemical abnormalities also included elevated activities of AST and GGT in 24 (63%) and 8 (53%) buffaloes with traumatic pericarditis and vegetative endocarditis, respectively. Hyperbilirubinemia was detected in 23 (61%) and 11 (67%) cases with traumatic pericarditis and vegetative endocarditis, respectively. Hypoglycemia was recognized in 17 (45%) and 7 (47%) buffaloes with traumatic pericarditis and vegetative endocarditis, respectively.

3.3. Ultrasonographic findings

Ultrasonographic findings in 53 buffaloes with heart failure are summarized in Table 3. In the cases with traumatic pericarditis, echocardiographic findings revealed thick rugged pericardium (mean [SD] 7 mm [2]) with multiple hyperechoic linear pericardial projections with fibrin strand deposition, indicative of chronic inflammatory changes. Pericardial effusions were imaged in 87% of buffaloes with traumatic pericarditis. These fluids in the pericardial sac appeared as either homogeneous anechogenic (Figure 2A) or heterogeneous hypoechogenic pericardial effusions. Fibrin was imaged as floating shreds within the pericardial effusions or deposits on the epicardium (Figure 2B).

In buffaloes with endocarditis, the affected valves appeared as hyperechogenic thick mass structures with nodular or proliferative vegetative lesions (Figure 3). Small (<25 mm) and large (>25 mm) vegetative lesions were imaged in 5 (33%) and 10 (67%) buffaloes, respectively. The most affected valve was the tricuspid (n = 8), and then the mitral (n = 5) and pulmonary (n = 3).

In buffaloes with traumatic pericarditis, abdominal ultrasonographic findings revealed moderate corrugations of the reticular wall with echogenic constrictive adhesions between the spleen and dorsal ruminal sac in 9 buffaloes (Figure 4A). The amplitudes and frequencies of reticular contractions varied according to the reticular state. In cases where reticular adhesions were noticed, no

Table 2. Hematological and biochemical findings in buffaloes with congestive heart failure.

Variables	Control (n = 10) (mean ± SD)	Traumatic pericarditis (n = 38) (mean ± SD)	Vegetative endocarditis (n = 15) (mean ± SD)
Erythrocyte count (T/L)	6.5 ± 0.5[a]	5.8 ± 0.8[b]	6.0 ± 0.6[ab]
Hematocrit (%)	28 ± 3.0	27 ± 2.8	27 ± 2.6
Hemoglobin (g/L)	103 ± 10	99 ± 7	100 ± 8
Leukocyte count (G/L)	9 ± 1.2[b]	22 ± 3.4[a]	21 ±2.3[a]
Lymphocytes (µL)	5600 ± 700[b]	6200 ± 898[a]	5857 ± 640[b]
Monocytes (µL)	490 ± 74	576 ± 128	487 ± 151
Segmented neutrophils (µL)	2500 ± 527[b]	13,327 ± 3257[a]	13,008 ± 2098[a]
Band cells (µL)	170 ± 72[b]	1474 ± 519[a]	1067 ± 461[a]
Eosinophils (µL)	410 ± 88	423 ± 105	415 ± 139
Platelet count (mm^3) × 10^3	332 ± 75	324 ± 57	326 ± 63
Total proteins (g/L)	71 ± 4.6[b]	79 ± 4.1[a]	76 ± 3.5[a]
Albumin (g/L)	37 ± 3[a]	26 ± 2.6[b]	26 ± 2.9[b]
Globulins (g/L)	34 ± 3[b]	53 ± 4[a]	50 ± 5[a]
Albumin/globulin ratio	1.0 ± 0.1[a]	0.5 ± 0.1[b]	0.5 ± 0.1[b]
γ-Glutamyl transferase (U/L)	27 ± 3[b]	47 ± 7[a]	43 ± 6[a]
Aspartate aminotransferase (U/L)	80 ± 8[b]	131 ± 9[a]	129 ± 7[a]
Total bilirubin (µmol/L)	6 ± 1.9[b]	15 ± 3.4[a]	13 ± 3.5[a]
Blood urea nitrogen (mmol/L)	4.2 ± 0.8	4.0 ±0.7	4.3 ± 0.8
Creatinine (µmol/L)	138 ± 8	140 ± 7	143 ± 9
Glucose (mmol/L)	5.1 ± 0.5[a]	3.2 ± 0.4[b]	3.0 ± 0.4[b]
Total cholesterol (mmol/L)	4.5 ± 1.1	4.2 ± 0.4	4.3 ± 1.2
Triglycerides (µmol/L)	0.49 ± 0.1	0.52 ± 0.1	0.5 ± 0.2
Venous blood gas analyses			
pH	7.35 ± 0.2	7.35 ± 0.4	7.34 ± 0.3
pCO_2 (mmHg)	46 ± 3.4	45 ± 2.7	44 ± 3.9
pO_2 (mmHg)	43 ± 2.8	40 ± 3.2	41 ± 3.8
HCO_3 (mmol/L)	26 ± 2.1	25 ± 2.4	25 ± 3.3
TCO_2 (mmol/L)	27 ± 3.4	26 ± 4.1	26 ± 5.3
BE (mmol/L)	0.8 ± 0.1	0.6 ± 0.3	0.4 ± 0.1

[ab]Values with different superscript letters in the same row differ significantly at $P < 0.05$.

Table 3. Ultrasonographic findings in buffaloes with congestive heart failure.

Organ	lesions	Traumatic pericarditis (n = 38) Number of buffaloes (%)	Vegetative endocarditis (n = 15) Number of buffaloes (%)
Heart	Fibrinous pericarditis	25 (66)	0 (0)
	Suppurative pericarditis	8 (21)	0 (0)
	Pericardial effusions	33 (87)	1 (7)
	Tricuspid valve vegetative lesions	0 (0)	8 (53)
	Mitral valve vegetative lesions	0 (0)	5 (33)
	Pulmonary valve vegetative lesions	0 (0)	2 (13)
Reticulum	Corrugated reticular wall	9 (24)	0 (0)
	Perireticular adhesions	6 (16)	0 (0)
	Echogenic deposits between reticulum, dorsal ruminal sac, and diaphragm	13 (34)	0 (0)
	Perireticular abscesses	5 (13)	1 (7)
Liver	Liver abscesses	3 (8)	1 (7)
	Hepatomegaly	28 (74)	7 (47)
Abdomen	Echogenic deposits between rumen and left abdominal wall	3 (8)	0 (0)
	Abdominal abscesses	2 (5)	0 (0)
	Ascites	32 (84)	8 (53)
Lungs	Pleural effusions	1 (3)	2 (13)
	Lung abscess	0 (0)	1 (7)
	Lung consolidation	10 (26)	2 (13)

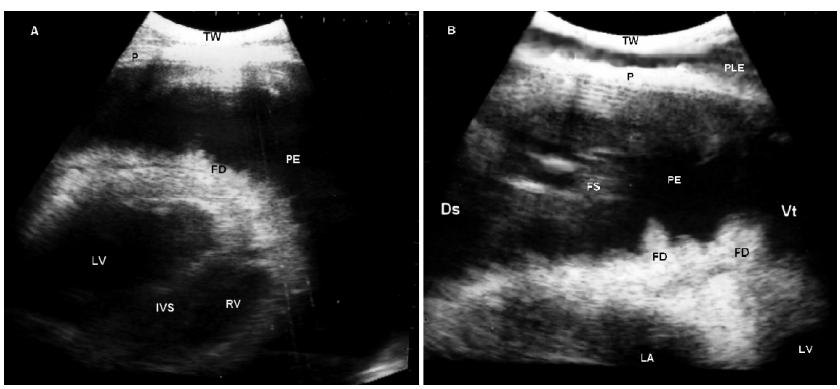

Figure 2. Ultrasonograms in buffaloes with congestive heart failure as a result of traumatic pericarditis, showing **(A)** anechoic pericardial effusion and **(B)** pleural and pericardial effusions with fibrinous pericarditis. TW: Thoracic wall, P: pericardium, PLE: pleural effusion, PE: pericardial effusion, FS: fibrin shreds, FD: fibrin deposits, LV: left ventricle, RV: right ventricle, IVS: interventricular septum, LA: left atrium, Ds: dorsal, Vt: ventral.

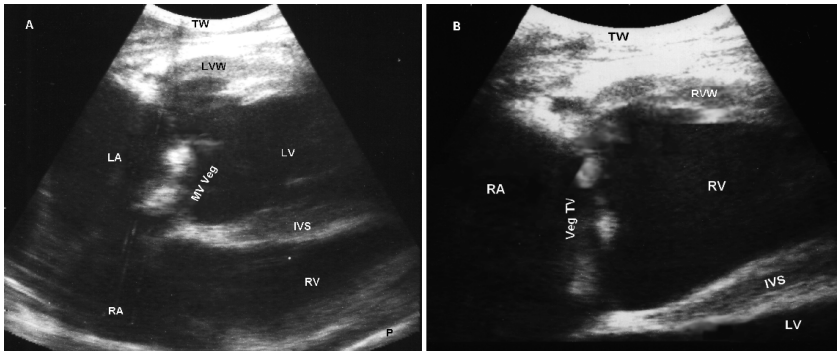

Figure 3. Echocardiography in buffaloes with vegetative endocarditis, showing **(A)** left caudal long-axis view of the heart in a buffalo cow with mitral valve vegetative lesions, **(B)** right short-axis view of the heart in a buffalo cow with tricuspid valve vegetative lesions. LA: Left atrium, LV: left ventricle, RA: right atrium, RV: right ventricle, IVS: interventricular septum, LVW: left ventricular wall, RVW: right ventricular wall, MV Veg: mitral valve vegetative lesions, Veg TV: tricuspid valve vegetative lesions.

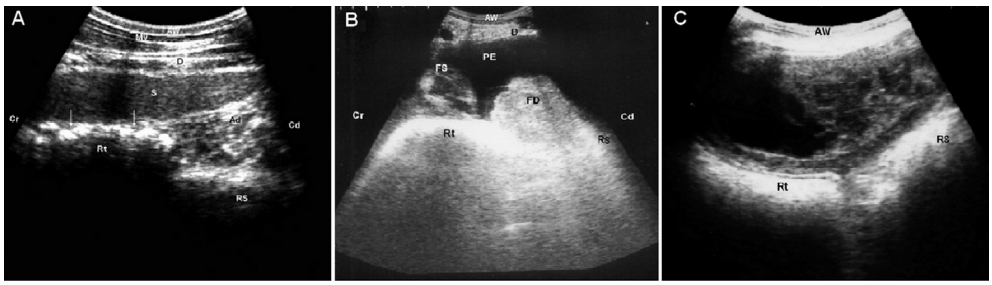

Figure 4. Abdominal ultrasonograms in buffaloes with congestive heart failure as a result of traumatic pericarditis. **(A)** Severe adhesion among reticulum, cranial sac of rumen, and spleen with corrugation of reticular wall (arrows). **(B)** Deposits and shreds of fibrinous tissue with peritoneal effusions. **(C)** Perireticular abscess with heterogeneous contents partitioned with echogenic septae. AW: Abdominal wall, MV: musculophrenic vein, D: diaphragm, S: spleen, Rt: reticulum, RS: cranial dorsal sac of the rumen, Ad: adhesion, FS: fibrin shreds, FD: fibrin deposits, Cr: cranial, Cd: caudal.

reticular contractions were imaged, while in buffaloes with mild reticular lesions, the amplitude of reticular contractions was reduced and the frequency was lowered to 1 contraction per 2-min period. Fibrinous tissue deposits and shreds, which appeared as homogeneously echogenic areas and strands (Figure 4B), were imaged between the reticulum, dorsal ruminal sac, and diaphragm in 13 buffaloes. Perireticular abscesses were seen in 5 buffaloes with traumatic pericarditis and 1 buffalo with vegetative endocarditis. These abscesses appeared as round or oval structures with a well-defined echogenic capsule; furthermore, the content of the abscesses was either homogeneous or heterogeneous (Figure 4C) and it was divided by echogenic septae in 4 cases. No splenic abscessations were observed. Accumulation of nonechogenic fluids in the peritoneal cavity was observed in 36 cases, indicating ascites. Ultrasonographically, the liver was distended with increased echogenicity of its parenchyma in 31 buffaloes. The diameter of caudal vena cava was increased (mean [SD] 4.3 cm [0.8]) with an oval-to-circular shape in cross-section. The hepatic and portal veins were also dilated (Figure 5A).

Ultrasonographic examination of lungs revealed lung consolidation in 12 buffaloes. Depending on the degree of atelectasis, the ventilated lung with consolidation could be identified by the weak, defined, and blurry reverberation artifacts. The extensive hypoechoic zones in the cranioventral lung fields and the cranioventral portions of the main lobes turned out to be consolidated lung tissues, which were confirmed at postmortem examination (Figure 6). Lung abscess was seen only in one buffalo with vegetative endocarditis. It appeared as a relatively well-defined structure that existed in the form of round-to-ovoid anechoic areas. The lung parenchyma had medium echogenicity with a heterogeneous appearance resembling liver parenchyma.

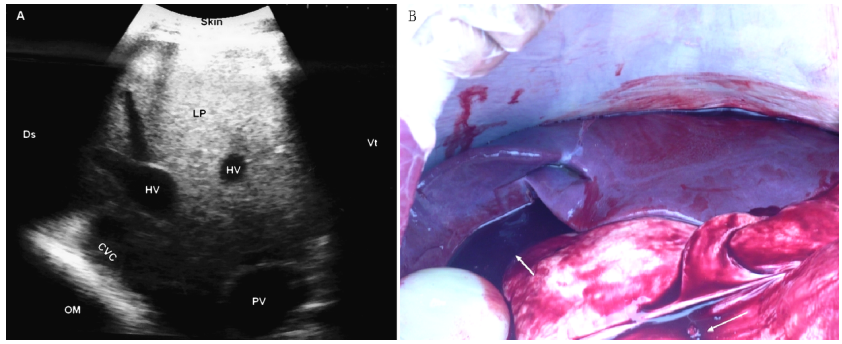

Figure 5. (A) Ultrasonogram of liver in a buffalo cow with congestive heart failure shows dilatation of the caudal vena cava (CVC), hepatic veins (HV), and portal veins (PV). **(B)** Peritoneal cavity of the same cow reveals accumulation of ascetic fluid (arrow) and enlarged liver. LP: Liver parenchyma, OM: omasum, Ds: dorsal, Vt: ventral.

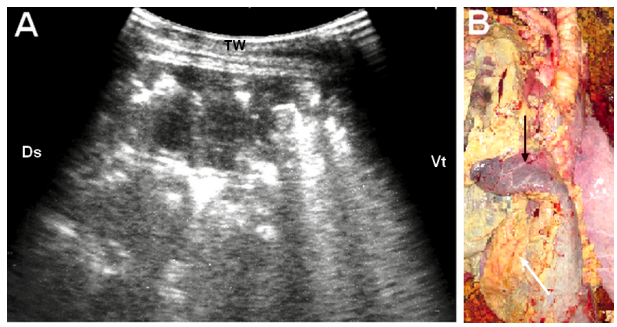

Figure 6. Ultrasonogram and postmortem findings in a buffalo cow with congestive heart failure as a result of traumatic pericarditis. **(A)** Ultrasonogram represents consolidated lung where there are often small pockets of gas remaining, seen as small hyperechoic areas, which cast an acoustic shadow. **(B)** This image represents postmortem findings in the same case showing consolidated right cranial lobe (black arrow) and fibrinous exudates (white arrow). TW: Thoracic wall, Ds: dorsal, Vt: ventral.

3.4. Postmortem findings

Table 4 summarizes the postmortem findings in 53 buffaloes with heart failure. In the cases with traumatic pericarditis, the pericardium was distended, displacing the lung dorsally. Approximately 3 L of turbid yellow malodorous fluid, indicating hydropericardium, were retrieved from the pericardial sac, along with the thick wall of the pericardium; the heart was covered by yellow fibrinous deposits (Figure 7A). Constrictive adhesions between the pericardium and the apex of the heart (Figure 7B) were seen in 7 cases. After opening these adhesions, a tract containing foreign bodies, including nails and wires (Figure 7C), was noticed. In buffaloes with vegetative endocarditis, the tricuspid valve was mostly affected (8 cases), followed by the mitral (5 cases) and pulmonary (3 cases) valves. These lesions appeared primarily as cauliflower-like, verrucose, or wart-like (Figure 7D) with either cardiac dilatation or cardiac hypertrophy.

The examination of other visceral organs revealed extensive fibrinous adhesions between the cranioventral aspects of the reticulum and the ventral abdominal wall and the diaphragm in 6 buffaloes with traumatic pericarditis. In 8 cases, adhesions extended to involve the spleen with splenomegaly. Diaphragmatic hernia of the reticulum was observed in one case. Perireticular abscesses of variable sizes (from 5 to 12 cm in diameter) were observed in 6 buffaloes. Congestion and enlargement of the liver was seen in 34 buffaloes with excessive accumulation of peritoneal effusions (Figure 5B). From 7 to 9 L of peritoneal fluids were retrieved. The lungs were consolidated (Figure 6B) with pleural effusions in 12 cases. After careful postmortem examinations to determine the type of heart failure, it was observed that all buffaloes with traumatic pericarditis exhibited right-sided congestive heart failure. However, animals with mitral vegetative endocarditis showed left-sided heart failure, while buffaloes with tricuspid and pulmonary vegetative endocarditis exhibited right-sided heart failure. All of these lesions indicated the chronicity of illness.

Table 4. Postmortem findings in buffaloes with congestive heart failure.

Organ	Lesions	Traumatic pericarditis (n = 38) Number of buffaloes (%)	Vegetative endocarditis (n = 15) Number of buffaloes (%)	Remarks and descriptions
Heart	Fibrinous pericarditis	26 (68)	0 (0)	Thick pericardium (>5 mm thickness) with accumulation of fibrin shreds
	Suppurative pericarditis	9 (24)	0 (0)	Thick pericardium (>3 mm thickness) with accumulation of purulent exudates
	Pericardial effusions	33 (87)	1 (7)	The pericardial sac filled with much yellowish or greenish fluid, which had very offensive odor
	Fibrinous adhesion between heart and pericardium	7 (18)	0 (0)	A visible tract between heart and pericardium
	Tricuspid valve vegetative lesions	0 (0)	8 (53)	Cardiac hypertrophy or dilatation and the vegetative lesions were clearly obvious on the heart valves
	Mitral valve vegetative lesions	0 (0)	5 (33)	
	Pulmonary valve vegetative lesions	1 (3)	2 (13)	
Reticulum	Perireticular abscesses	5 (13)	1 (7)	Rounded or oval in shape (>10 cm in diameter) containing pus
	Fibrinous adhesion between reticulum and spleen	8 (21)	0 (0)	Splenomegaly and constrictive adhesions either between reticulum and spleen or reticulum and diaphragm
	Fibrinous adhesion between reticulum and diaphragm	6 (16)	0 (0)	
Liver	Hepatomegaly	27 (71)	7 (47)	Enlarged and congested liver
	Liver abscesses	3 (8)	1 (7)	All abscesses were seen near the portal area
Lungs	Lung abscesses	(0)	1 (7)	Located near the diaphragmatic lobes
	Hydrothorax	2 (5)	3 (20)	Pleural effusion with serosanguineous fluids
	Lung consolidation	10 (26)	2 (13)	Mainly diaphragmatic lung lobes were affected
Abdomen	Ascites	32 (84)	8 (53)	Peritoneal cavity filled with ascitic fluid
	Abdominal abscesses	5 (13)	0 (0)	Mostly near rumen and left abdominal wall
Joints	Joint effusions	0 (0)	4 (27)	Mainly hock joint effusions

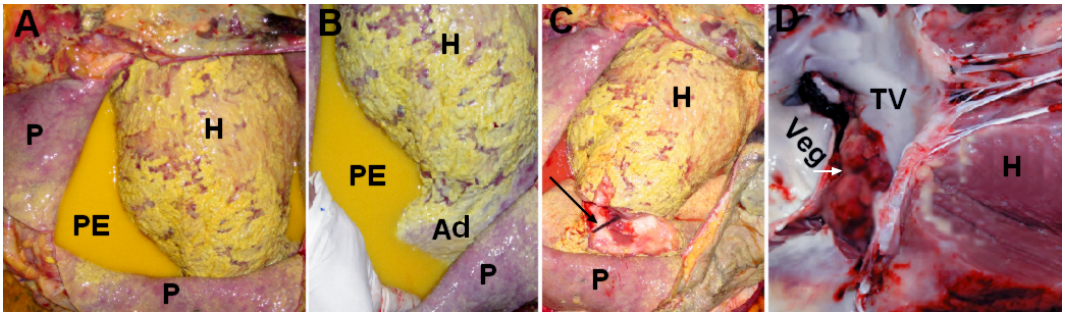

Figure 7. Postmortem findings in buffaloes with congestive heart failure as a result of pericarditis and endocarditis. **(A)** Thick open pericardium (P) contains yellow pericardial effusions (PE) and the heart (H) covered with yellow fibrin. **(B)** Constrictive adhesion (Ad) between heart (H) and pericardium (P). **(C)** Opening this adhesive tract revealed presence of a thin black 10-cm wire. **(D)** Vegetative endocarditis of tricuspid valve.

3.5. Management and outcome

In general, the health condition of all diseased buffaloes was deteriorated. However, from the economic point of view and based on the owner's decision, the pregnant diseased buffaloes (n = 9) were treated and managed until calving. Therapy included procaine penicillin G (6000 IU/kg intramuscularly) as an antimicrobial drug, flunixin meglumine (0.7 mg/kg intravenously) as a nonsteroidal antiinflammatory drug, furosemide (1 ampoule/70 kg intravenously) as a diuretic drug, and vitamin E/selenium (150 mg vitamin E and 1.7 mg sodium selenite/mL; 10 mL subcutaneously) as an antioxidant drug. The duration of the therapeutic trial ranged from 10 to 25 days, according to the date of calving of each buffalo. Management procedures included restricting the movement of diseased buffaloes as much as possible. In addition, diseased pregnant animals were tied up in a sloped posture, where the forelimbs were on a higher level than the hind limbs in order to minimize the pressure load of the growing fetus and abdominal organs on the heart as much as possible. Five animals were found dead before calving. Four buffaloes were able to survive until normal calving, but 1–3 days later their condition progressively deteriorated; therefore, the owners were advised to slaughter these buffaloes for euthanasia. One calf was weak and needed medical intervention, while the remaining 3 calves were in good health.

4. Discussion

Clinical heart failure has been discussed before in cattle (9), but according to the authors' knowledge, a retrospective study of heart failure in buffaloes has not been previously reported. Cardiac failure is defined as ventricular dysfunction leading to the inability to maintain an adequate blood output after the cardiac reserve fails to cope with circulatory demands (16). In the present study, only pericarditis and endocarditis were identified as a cause of heart failure in buffaloes. Congestive cardiac failure in cattle may result from valvular disease, myocardial or pericardial disease, heart tumors, or congenital defects that produce shunts (3,9). In this study, neither heart tumors nor congenital defects were detected. Such differences may be due to species variation and/or the different management systems.

In the current study, the clinical presentations of the affected buffaloes included anorexia, a sharp drop in milk yield, diarrhea, coughing, and recurrent tympany. These findings were inconsistent with previous reports in cattle (2). Compared with cattle, pain reactions, including reluctance to move, arching of the back, grunting, and tearing with protrusion of tongue (10), were common clinical findings in buffaloes with traumatic pericarditis. This is in disagreement with a previous report in buffaloes (8). Such variations could be attributed to the differences in the chronicity and/or severity of illness. Tachycardia, muffled heart sounds, cardiac murmurs, jugular distension and pulsation, and brisket edema were the main clinical findings in affected buffaloes. These findings could be attributed to traumatic pericarditis, valvular endocarditis, cardiomyopathy, or cardiac lymphosarcoma (17). Tachycardia has a low specificity for diagnosing heart disease as multiple noncardiac diseases can lead to tachycardia (7). Jugular venous congestion and pulsation, as well as brisket edema, are cardinal signs of right-sided heart failure in cattle (1). These signs can be of diagnostic value in primary cardiac disease in advanced stages. However, they may lack sensitivity as heart failure is observed in advanced stages of the disease (7,9). In the present study, 53 buffaloes showed signs of heart failure. Right-sided heart failure, manifested by brisket edema, venous distension, ascites, hydropericardium, and hepatomegaly, was the most common finding in buffaloes (38 with pericarditis, 8 with tricuspid endocarditis, and 2 with pulmonary endocarditis). In a retrospective study of cattle, right-sided heart failure was reported in most cases (9). It is well known that tricuspid and pulmonary endocarditis cause right-sided heart failure (2). It may seem

that traumatic pericarditis in buffalo leads to right-sided heart failure. This may be due to the cardiac tamponade, which hinders the normal blood flow in the right heart as the right ventricular wall is thinner than the left ventricular wall (18). Left-sided heart failure, which was manifested by signs of pulmonary congestion and edema, was noticed in 5 buffaloes. Necropsy findings of these animals revealed pulmonary congestion and edema. A low number of animals showing pulmonary edema may indicate that the left heart is rarely involved in heart failure in buffaloes, in contrast to pet animals (19) and horses (20).

In the present study, the results of the clinical presentations and findings may be of diagnostic value for diagnosing congestive heart failure in buffaloes, but they may have little value in differentiating between pericarditis and endocarditis, as in advanced stages of both conditions heart failure could develop. In the present study, hematological findings indicated leukocytosis and neutrophilia in diseased buffaloes; these findings would support the diagnosis of pericarditis (4) and bacterial endocarditis (21) in cattle. However, these values would also support a diagnosis of many other infectious processes (2). Serum biochemistry results showed increased total proteins, globulins, liver enzymes (AST, GGT), and total bilirubin. These findings were compatible with an inflammatory process that was attributable to advanced cardiac disease, impaired liver function, chronic infectious disease or other inflammatory complications (22).

In contrast, cardiac ultrasonography has been described as a reliable tool to detect various types of heart disease (23). The technique has been evaluated in adult cows (13) and calves (24). Cardiac ultrasound examination can be performed easily in field conditions and its sensitivity and specificity are good even when there are no signs of heart failure (4,6,23). In the present study, ultrasonographic results were important for diagnosing traumatic pericarditis and endocarditis in buffaloes with a high degree of assurance. Echocardiography was valuable in detecting the amount and echogenicity of the pericardial, pleural, and peritoneal effusions, and in the verification and differentiation of pericarditis and endocarditis.

Traumatic pericarditis in cattle and buffaloes is most commonly caused by penetrating metallic foreign bodies either migrating from the reticulum or due to impaling (25). In most of the cases in the present study, a large amount of hypoechogenic fluid was seen in the pericardial sac, pleura, or peritoneum, indicating an advanced stage of heart disease, sometimes containing strands of free deposits of fibrin in the cases with pericarditis. Furthermore, ultrasonographic findings appeared to correlate well with those found at postmortem examinations. Previously, cardiac ultrasonography has been suggested as the method of choice for imaging and evaluating the severity of constrictive pericardial effusion (5). Transthoracic echocardiography is a highly sensitive tool for diagnosing endocarditis in cows as also mentioned in previous studies (26). In the current study, the tricuspid valve was the most affected valve (8 buffaloes), followed by the mitral valve (5 buffaloes) and then the pulmonary valve (2 buffaloes). The valve predilections in the present study correlate well with findings reported elsewhere (2,9). In contrast, the pulmonary valve, followed by the tricuspid valve, was the most common location for endocarditis in cattle (27). No aortic endocarditis was found in the present study. The aortic valve seems to be less frequently affected in buffaloes, as found in a recent study of 33 cows (7).

In buffaloes with traumatic pericarditis, abdominal ultrasonography revealed reticular changes, including reduced motility, echogenic deposits, and perireticular abscesses. These alterations could be considered a complication related to traumatic reticuloperitonitis (28). Perireticular abscesses were recognized as round or oval echogenic structures with homogeneous or heterogeneous contents and a distinct hyperechogenic capsule, which was confirmed at necropsy examination. Therefore, reduced amplitude of reticular contractions may presumably be due to the impairments caused by the perireticular adhesions and abscesses.

In buffaloes with traumatic pericarditis and endocarditis, there was frequently moderate to severe ascites, which may be attributable to cardiac insufficiency and advanced stage of heart failure. Other extracardiac ultrasonographic lesions included hepatic enlargement and congestion, and the caudal vena cava dilated so that it appeared round-to-oval instead of triangular in cross section. These abnormalities have been attributed to cardiac insufficiency, thrombosis of the caudal vena cava, and compression of the caudal vena cava in the thorax or in the subphrenic region by space-occupying lesions (29).

Ultrasonographic examinations of lungs revealed pleural effusions (3 buffaloes), lung consolidation (12 buffaloes), and lung abscess (1 buffalo). These findings may be due to disseminated infections from pericarditis or endocarditis (7,28). Results in the present study suggest that the prognosis of congestive cardiac failure in buffaloes as a result of traumatic pericarditis or endocarditis is poor, but from the economic point of view for pregnant ones, it may be helpful to immobilize the diseased animals with therapeutic trials until calving; however, deterioration of their health condition could still develop later on.

It can be concluded that, in the buffaloes described here, heart failure was caused by 2 entities, including traumatic pericarditis and vegetative endocarditis. The main clinical presentations were those indicated in right-sided heart failure, namely peripheral edema, venous distension and jugular pulsation, and a sharp drop in milk

yield. The prognosis of heart disease in buffaloes seems poor; however, in some diseased pregnant buffaloes, restricting movement as much as possible with therapeutic trials may be helpful. Ultrasonographic examinations revealed cardiac and extracardiac lesions that could be easily diagnosed and verified. Ultrasonographic findings were consistent with those observed at postmortem examination; therefore, ultrasonography may be of great help in assessment of the extent and severity of lesions in buffaloes with congestive heart failure.

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