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CLINICOBIOCHEMICAL, ECHOCARDIOGRAPHIC, ELECTROCARDIOGRAPHIC AND POSTMORTEM CHARACTERISTICS IN COWS WITH PULMONIC VALVE VEGETATIONS

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SUMMARY

This paper describes the clinical, hematological, biochemical, echocardiographic, electrocardiographic and pathological findings in 19 cows with pulmonic valve vegetations. Results were compared with those of 10 apparently clinically healthy cows. Clinically, the most striking clinical findings were recurrent fever, dyspnea, tachycardia, weight loss or decreased milk production. Epistaxis and/or hemoptysis were observed in 5 diseased cows. A presumptive diagnosis of pulmonic valve endocarditis was made primarily on the basis of auscultation of the valve at the left 3rd intercostal space with history of fever. Thirteen of the animals had leukocytosis; 11 had neutrophilia and 3 had lymphopenia. Serum chemistry revealed hyperproteinemia, hypoalbuminemia and hypergammaglobulinemia. High serum activities of lactate dehydrogenase (LDH) were also observed. Arterial blood gas analysis showed metabolic acidosis in 14 cows and elevated bicarbonate concentration in 4 cows. A definitive diagnosis was then done by echocardiography and electrocardiography (ECG). Confirmatory diagnosis was finally made after slaughter of 17 cases. Postmortem lesions included also thrombosis of the pulmonary artery inside the lung with secondary pneumonia. Data obtained from this study clearly demonstrate the superior diagnostic effects of echocardiography and ECG in cows with pulmonic vegetative endocarditis.

Key Words: cattle, endocarditis, postmortem findings, pulmonic vegetation, ultrasonography and electrocardiography

INTRODUCTION

Valvular endocarditis is often a serious consequence of bacterial infection in animals as well as in humans [6, 16, 19]. The most commonly affect-

ed valve is the pulmonic valve, followed by the tricuspid, mitral and rarely the aortic valve [5]. The mechanisms behind this endocardial infection and the pathogenesis of the lesions that develop in the valves are, however, incompletely known. There are two possible routes for bacteria in the blood to reach the endocardium of the valves: 1) by contact with the surface endothelium of the endocardium, and 2) from the capillaries in the valves [9]. The generally accepted theory of today in both human and veterinary medicine is that transient or persistent bacteremia, via an endothelial lesion with thrombus formation or through some other immunopathological mechanisms, adhere to the endocardial endothelium and, from there, initiate an inflammation in the valve [2].

In humans, it has been found that endocardial lesions not only induced by hemodynamic disorders as predisposing factors [18] but also that infectious endocarditis may appear in previously normal hearts [6]. In cattle, endocarditis is often a sequel to bacterial infection, with the pulmonic and right atrioventricular valve followed by the mitral valve being the most frequently involved [4]. Fragments of plaque may embolize, resulting in skeletal, pulmonary, splenic, or hepatic involvement [7, 10].

Echocardiography has provided a useful means by which the cardiac chambers and valves in cattle can be imaged [3]. In addition, ECG was approved to be of clinical importance in cardiovascular diseases in cattle [1, 15]. The present paper summarizes the clinicobiochemical, echocardiographic, electrocardiographic and pathologic characteristics in 19 cows with pulmonic vegetative endocarditis compared with controls.

MATERIALS AND METHODS

Six of the animals studied here were seen at the Veterinary Teaching Hospital, Rakuno Gakuen University, Hokkaido, Japan during the period of November 2006 to February 2007 during a fellowship to the author supported by the Egyptian government. The other 13 cows were examined at Zagazig University, Veterinary Teaching Hospital between 2005 and 2006. All animals were examined by the author. A confirmatory diagnosis of valvular endocarditis was made after slaughter of 17 cows. In the remaining 2 cases, the diagnosis was made on the basis of cardiac abnormalities on auscultation, cardiac ultrasonography and ECG. Diseased cows were compared with 10 apparentely clinically healthy cows.

Clinical examination of the animals was carried out as described by Rosenberger (1990). Blood samples on EDTA tubes were collected from each animal for determination of the hematocrit, hemoglobin (Hg), erythrocytes and leukocyte count and differential leukocytic count. In addition, serum samples on plane tubes were taken for the determination of total protein and their fractions (albumin, and α, β and γ- globulins). Albumin globu-

lin ratio was calculated by dividing albumin to total globulin concenration. Serum samples were also used for the determination of the activities of LDH and its isozymes (8). An arterial blood sample was also obtained from the ear artery for blood gas analyses.

Echocardiographic examinations were made while the cows were standing, by the methods described previously [3, 11] using a 3.5MHz linear transducers (Model RT 2600, Yokogawa Medical Systems, Tokyo, Japan and Pie Medical 240 Parus, The Netherlands). An area of hair, 15cm x 15 cm, over the third, fourth and fifth intercostal spaces in the cardiac lesion was clipped on both sides of the thorax. The heart was then examined ultrasonographically on the right and then the left side as illustrated (Figure 1 A, B).

Electrocardiograms were obtained by means of an ECG recorder (Cardiofax GEM, Nihon-Kondem) which allowed recording at a paper speed of 25 mm/ sec. Three leads were used. The right arm ead is placed on the neck, the left arm lead is placed on the chest wall just above the sternum nd the neutral lead is placed on the withers as illustrated in Figure 1 C. The electrocardiogram as only recorded when the heart rate was in the sting range and when the cows was standing. ue to the grave prognosis of the cattle, 17 anials were slaughtered and a complete examinaon was then carried out.

RESULTS

The most common complaint of diseased cases at admission was weight loss and recurrent fever with concurrent anorexia, dyspnea and poor milk production. Five cows had a history of epistaxis and/or hempotysis. The duration of signs prior to admission was 14 to 120 days. The earliest signs included intermittent fever, tachycardia, inappetence, decreased milk production. Since these signs were non-specific, the presence of systolic murmur suggested pulmonic vegetation endocarditis. The murmur was louder over the left 3rd intercostal space in 11 cows and over the right 3rd intercostal space in the other 2 animals. Recognition of a cardiac murmur, usually systolic, was the most specific physical examination finding. However, six of the cows had not detectable murmurs.

Plasma and serum proteins and their fractions and other serum biochemical abnormalities in healthy and diseased cows are summarized in Table 1. Eight cows had high plasma serum proteins and fifteen had hypergammaglobulinemia. Thirteen cases had absolute leukocytosis, 11 had neutrophilia and 3 cows had lymphopenia. Serum biochemical abnormalities also included hyperproteinemia and hypoalbuminemia in 13 cows, hypergammaglobulinemia in 17 and elevated LDH in 10 cases. Arterial blood gas analyses showed metabolic acidosis in 14 cows and elevated bicarbonate concentration in 4 cases.

Compared to the normal pulmonic valves (Fig. 2A, B, C), the vegetative pulmonic abnormalities included hyperechogenicity, thickening, and proliferative or vegetative lesions; vegetations that were oscillating, vibratory, or mobile; lesions with a shaggy, moth-eaten, or fluffy appearance; nodular changes; and focal or discrete lesions (Figs. 2D, E, F). Massive proliferative lesions was also observed in 6 cows (Fig. 3 A, B, C, D, E, F). Compared to control cows (Fig. 4A), ECG recordings in diseased animals demonstrated a deep, irregular QRS complex, and a tall, irregular T wave (Fig. 4B, C, D and E). The QRS complexes were deep in all examined cows and ranged from 1.7 to 2.5 mV (control cows is 1.0 mV). In addition, the T waves were also tall and ranged from 0.8 to 1.5 mV (control cows is 0.3 mV). Both deep QRS complexes and tall T waves were characteristics in all examined cows with pulmonic valve endocarditis.

Seventeen cows were slaughtered where thrombosis of the pulmonic valve was confirmed (Fig. 5A). Other lesions seen at necropsy included thrombosis of the pulmonary artery inside the lung parenchyma (Fig. 5B, C), secondary pneumonia (Fig. 5D, E) and intra pulmonary hemorrhage (Fig. 5F).

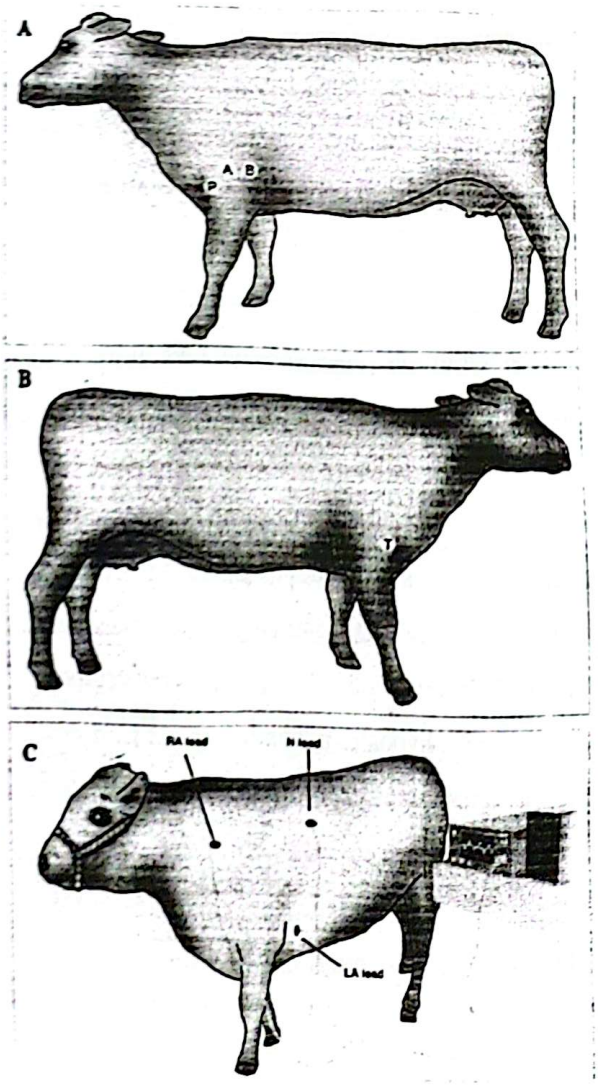

Figure 1: Echocardiographic and electrocardiographic examination. **A:** position of the pulmonary (P), aortic (A) and bicuspid (B) heart valves. The stethoscope is advanced uder the triceps muscle to get as close to the valves as possible. **B:** position of the tricuspid heart valve. **C:** recording a bovine electrocardiograph. The right arm (RA) lead is placed on the neck, the left arm (LA) lead is placed on the chest wall just above the sternum and the neutral lead (NL) is placed on the withers.

Figure 2. Ultrasonograms of the normal pulmonary valve in control cows (A; closed valve during diastole, arrow and B; open valve during systole, arrow). In C, the pulmonary valve is transversely imaged over the aorta in healthy animals. In diseased cows, ultrasonographic imaging of pulmonic valve revealed a highly thickened valve with thrombosis (D, E and F). Image was taken at the left 3rd intercostal space using a 3.5 MHz linear transducer. PA; pulmonary artery, PULA; pulmonary artery, A; aorta, RVOT, right ventricular outflow tract, IVS; interventricular septum, LV; left ventricle, T; thrombus. Arrows indicate vegetations.

Figure 3. Ultrasonograms of [illegible] pulmonary thrombosis in [illegible] (A) [illegible] (B) with [illegible]. Images were taken at the [illegible] using a 7.5 MHz linear transducer. PA, pulmonary artery; [illegible] RVOT, right ventricular outflow tract; VS, interventricular septum; RV, right ventricle; LV, left ventricle; RVOT, right ventricular outflow tract. [illegible] arrows indicate thrombosis.

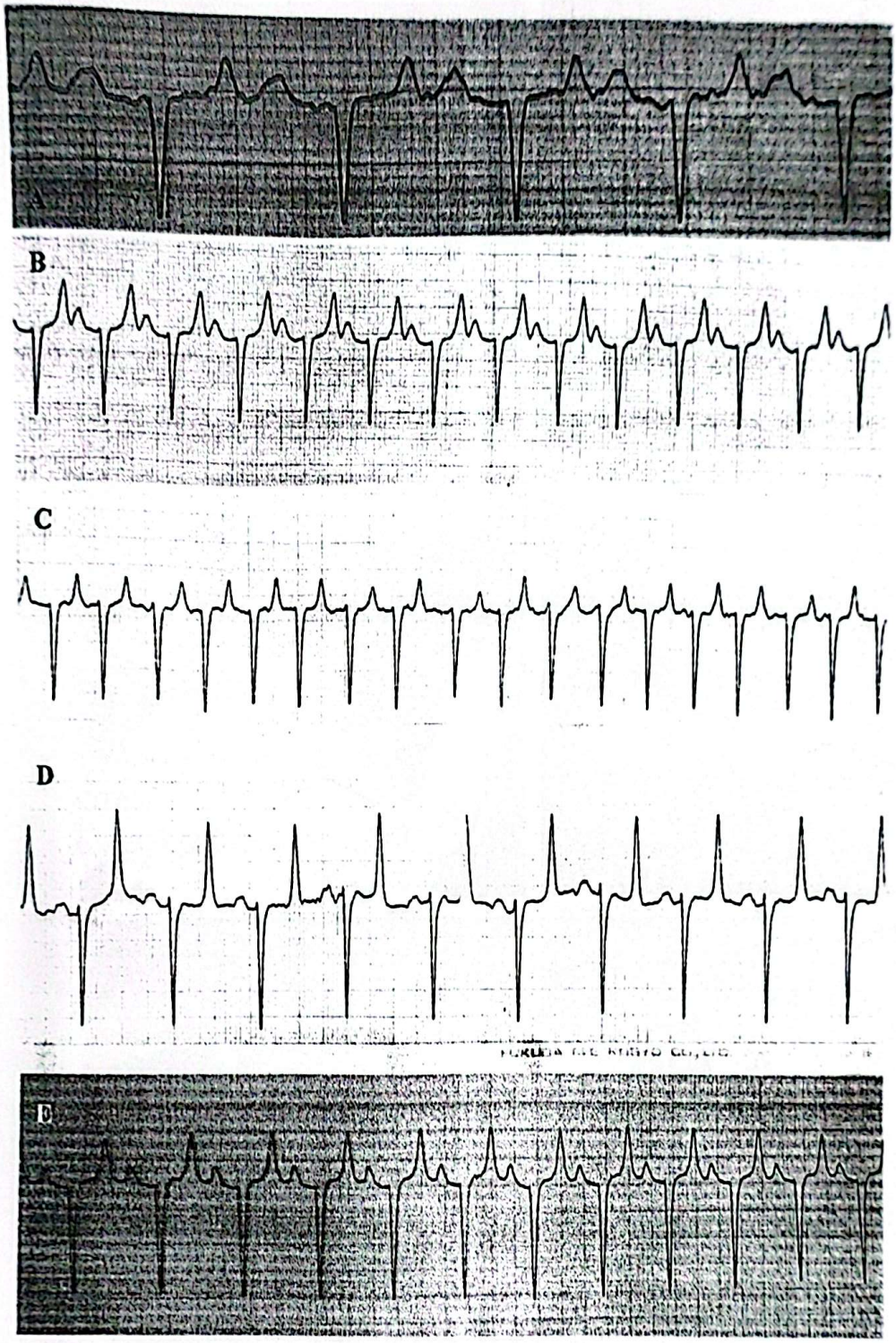

Figure 4. ECG recordings from a control cow (A), and from 4 cows with pulmonic vegetations (B, C, D, E) where deep QS complexes and tall T waves are detected in diseased animals.

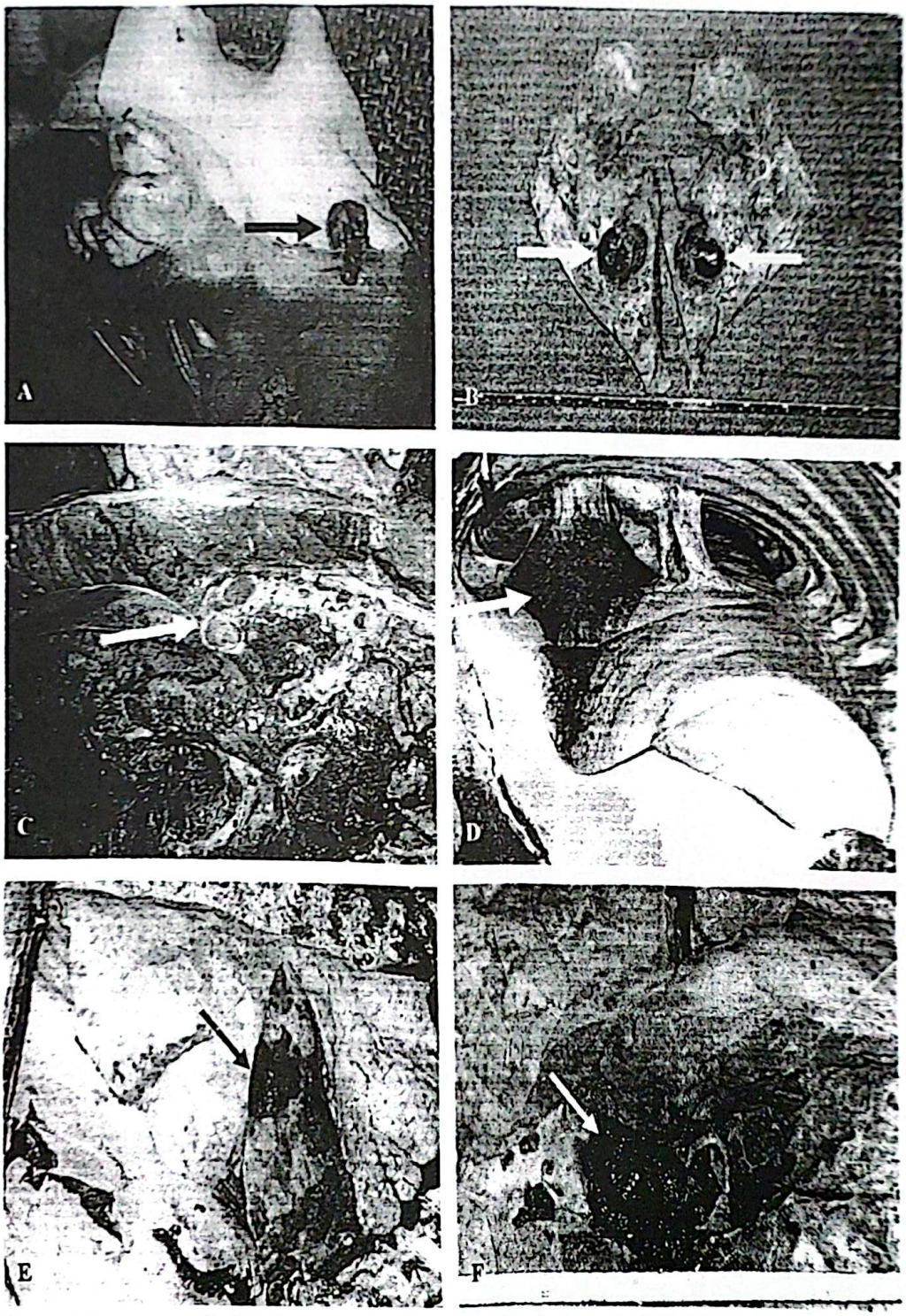

Figure 5. Postmortem findings in cows with pulmonic valve thrombosis. Pulmonic valve vegetations (A, black arrow). Pulmonary thromboembolism (B, C white arrows). Secondary pneumonia (D and E, arrows) and intra pulmonary hemorrhage (F, arrow) are additional findings at slaughter.

Table 1. Hematological and biochemical findings in cows with pulmonic vegetations compared to controls

Parameters		Diseased (n=19)	Healthy (n=10)
Hematocrit (%)		27±4	24-42
Hemoglobin (g/dl)		9.8±3	8.0-12.0
Erythrocyte count ($\times 10^4$/µl)		567±350	500-1000
Leukocyte count (/µl)		18110±5300	5000-1000
Neutrophils (/µl)		11313±1463	700-4500
Bands (/µl)		631±352	0-100
Lymphocytes (/µl)		4300±900	3000-7000
Monocytes (/µl)		650±320	25-500
Eosinophils (/µl)		95±54	0-250
Basophils (/µl)		43±21	0-200
Total protein, plasma (g/dl)		8.6±2.1	7-7.1
Total protein, serum (g/dl)		7.8±0.9	6.6-7.6
Albumin (g/dl)		1.73±0.22	2.0-3.4
α-globulin (g/dl)		1.40±0.18	0.75-0.8.8
β-globulin (g/dl)		0.88±0.14	0.8-0.11
γ–globulin (g/dl)		3.90±0.89	1.69-2.27
A/G raio		0.37±0.09	0.80-0.90
Arterial blood gas analysis	pH	7.489±0.39	7.32-7.45
	PCO^2	36.82±7.31	30-55
	PO^2	90.88±20	85-105
	HCO^{3-}	26.79±4.46	20-30
	BE	4.07±3.85	3.0-5.0
	O_2Sat	96.7±1.37	95-100
Lactate dehydrogenase	(LDH) (U/l)	6733	520-1300
LDH isozymes	LDH-1 %	43±4.6	39.8-63.5
	LDH-2 %	30±2.3	19.7-34.8
	LDH-3 %	18±2.2	11.7-18.1
	LDH-4 %	6±2.3	0.0-8.8
	LDH-5 %	3±2.1	0.0-12.4

Data are expressed as mean±SD

DISCUSSION

The present study represents the largest series of pulmonic valve vegetation in cattle. Before admission, the cases in the present study suffered from weight loss, recurrent fever, decreased milk production and prominent dyspnea. In cows with endocarditis, fever develops early and often is recurrent, but may not be a consistent finding [13]. However, a heart murmur in cattle is not necessarily found in association with bacterial endocarditis [13]. The absence of heart murmur in cattle may be a clinical fact or may reflect our inability to auscultate a slight murmur because of the thick thoracic wall of cattle. Loud heart sounds heard by auscultation are the rule in cattle affected with endocarditis. Dyspnea observed in cows in the present study may be attributed to lung emboli produced by breakdown of pulmonic valve vegetations. In addition, epistaxis and/or hemoptysis that detected in 5 cows may be attributable to intra pulmonary hemorrhage. These observations were confirmed after slaughter and examination of lung parenchyma. Intra pulmonary hemorrhage may be caused by rupture of the pulmonary artery inside the lung parenchyma caused by thromboembolism by fragments from vegetations.

In the absence of a heart murmur, ancillary aids to diagnosis include a complete blood count, total serum protein value and globulin value [13]. Neutrophilia, high serum globulin contents would support the diagnosis of bacterial endocarditis. In the present study, abnormalities detected on complete blood count and serum biochemical analyses included hypergammaglobulinemia, and neutrophilia, which also supported a diagnosis of valvular endocarditis [14]. The prognosis in cows with bacterial endocarditis is guarded to poor. Valvular incompetence and high serum total globulin concentration have been associated with a poor prognosis [12]. In the present study, leukocytosis and hypoalbuminemia were more common laboratory abnormalities in cows.

Complete echocardiographic examination is extremely useful in the evaluation of cows with suspected cardiovascular disease [14]. In the cases of this report, 2-dimensional echocardiography with a 3.5-MHz linear transducer revealed a thick, vegetative pulmonic valve. Findings correlate well with findings reported elsewhere [20]. The source of infection should be determined in all cattle in which bacterial endocarditis is suspected by obtaining a thorough history and performing a complete physical examination. In addition, echocardiographic findings appeared to correlate and run parallel with those found after slaughter. In addition, ECG was sensitive for diagnosis of endocarditis and revealed deep QRS complexes and tall T waves in all examined cows.

As evidenced by the result of the current study, echocardiography is approved to be an excellent

diagnostic technique for cardiac evaluation in cows in which pulmonic vegetative endocarditis is suspected. In the cows of this report, it would have been difficult to reach a confirmatory antemortem diagnosis of pulmonic vegetative endocarditis without the use of echocardiography. Our results also stress the utility of ECG as an effective parallel methodology in the diagnosis of cattle with pulmonic vegetative endocarditis. The results of the present study suggest that in cows with manifestations such as dyspnea, epistaxis and/or hemoptysis, pulmonary thromboembolism and/or pulmonic valve vegetations may be suspected and a differential diagnosis should be carried out in order to reach a final diagnosis.

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