



Clinical, haemato-biochemical, radiographic and ultrasonographic findings in bovines suffering from pericarditis and pleural effusions

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Pericarditis, an inflammation of the pericardium, results in the accumulation of fluid or exudates between visceral and parietal pericardium (Reef and McGuirk 2002). Causes of pleuritis and pleural effusions include traumatic reticulopericarditis, tuberculosis, liver abscesses, tumours (lymphosarcoma), right ventricular failure, hypoproteinemia, ruptured thoracic duct and haemothorax from trauma or haemangiosarcoma (Smith 2002). Animals with advanced pleural effusions or congestive heart failure from nonpericardial causes may appear similar clinically to pericarditis, so efficient differentiation is required (Streeter and Step 2007). Keeping these points in mind, the present study was aimed to record ultrasonographic features of pericarditis and pleural effusions and to correlate it with clinical, haematobiochemical, radiographic findings.

Buffaloes (12) and cows (4) suffering from pericarditis and pleural effusions, presented at Teaching Veterinary Hospital were used. The signalment, duration of illness, feed intake, rumination, defecation, milk yield and reproductive status were recorded. Clinical examination of animals was carried out as per Rosenberger *et al.* (1979). Rectal temperature, heart rate and respiration rate were recorded. The animals were divided into 2 groups, which were based on the clinical, haematological, biochemical, radiographic and ultrasonographic findings and thoracocentesis/pericardiocentesis. Group 1, included 4 cows and 6 buffaloes diagnosed for pericarditis while group 2 included 6 buffaloes having pleural effusions.

Blood was estimated for Hb, PCV, TLC and DLC by standard methods. The plasma was subjected to the estimation of total protein, albumin, sodium, potassium and chloride using auto analyser. Plasma fibrinogen was measured by heat

precipitation test, using hand held refractometer. Thoracic radiographs in right lateral recumbency were taken in 6 animals of group 1 and in 4 animals of group 2. In other animals the owners did not give consent to cast the animal for radiography. Ultrasonography was performed on the standing animals restrained in a cattle crate without any sedation in all the animals. The concept/MCV veterinary ultrasound scanner, with a 3.5 MHz micro convex transducer was used.

Thoracocentesis was performed under ultrasound guidance by using a 16 gauge, 4 inch spinal needle in 5 animals of group 2 in standing position under local anaesthesia through a puncture in the left lateral thoracic wall in the 6th/7th intercostal space. TLC, DLC and total protein of pleural fluid was estimated. Pericardiocentesis was also performed under ultrasound guidance in 5 animals of group 1 using 16 gauge, 4 inch spinal needle through puncture at the level of left 4th/5th intercostal space just dorsal to the elbow. Representative samples of pericardial fluid were collected in sterile syringes and subjected to culture and antibiotic sensitivity test.

All the animals suffering from pericarditis and pleural effusions were female. The mean age of animals suffering from pericarditis was 5.4 ± 0.42 years, which was higher in comparison with Braun *et al.* (2007) who reported lower mean age of 3.8 years in 28 cattle suffering with traumatic pericarditis. Animals of group 2 had a mean age of 7.17 ± 1.19 years. Majority of the animals had reduced appetite which may be attributed to the depression of gastric centre activity and subsequent forestomach hypomotility due to pain (Leek 1983). The mean duration of illness was 9.70 ± 1.61 and 9.17 ± 2.64 days respectively, in animals of group 1 and 2 respectively. Majority ($n=10$) of the animals were non-pregnant in both the groups. Decreased amount of faeces was seen in 6 animals of group 1 and 4 animals of group 2. Off smelling loose faeces was seen in 2 animals of group 1. Rest of the animals passed normal faeces. Passage of diarrhoeic faeces in some animals was similar to the findings of Subramaniam *et al.* (2003). History of reduction in milk

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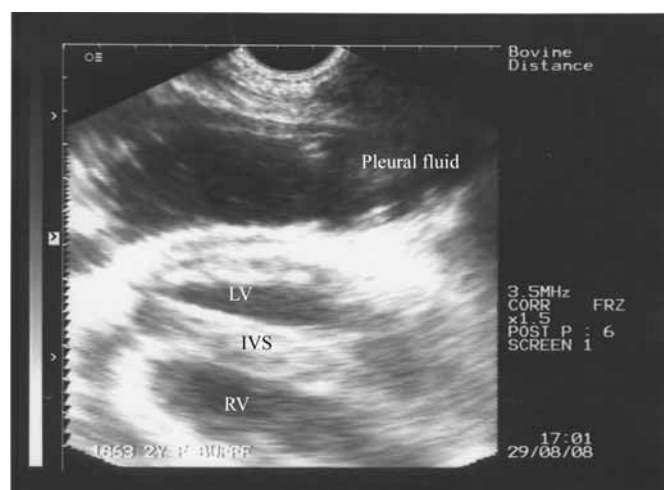
yield and fever was recorded in animals of both the groups.

Dyspnoea was seen in 3 and 2 animals of group 1 and 2, respectively. Jugular pulse and brisket oedema was seen in 7 and 3 animals of groups 1 and 2 respectively. Most of the animals with pericarditis appeared reluctant to move and had abducted elbows. The clinical signs of brisket oedema and jugular engorgement were less prominent in buffaloes as compared with cattle which may be attributed to species differentiation. Distension of jugular veins may be attributable to right-sided cardiac insufficiency, the most important cause of which is traumatic pericarditis, valvular endocarditis, cardiomyopathy and cardiac lymphosarcoma (Grunder 2002). Oedema has been attributed to the increased hydrostatic pressure. However, in some animals, absence of these signs could be due to drainage of pericardial fluid into the reticulum via a patent foreign body tract (Grunder 2002).

Heart rate, respiratory rate and rectal temperature were elevated in both the groups. Increased volume of fluid in the

pericardial sac increases the severity of cardiac compression, resulting in tachycardia and distension of jugular veins (Braun 2008). The elevated rectal temperature may be ascribed to the presence of infection. Auscultation of heart revealed muffled heart sound in majority of the animals. Pleural effusions without cardiac disease must be considered in cattle with muffled heart sounds (Braun 2008). It has been reported that auscultation of muffled heart sounds have high specificity and likelihood ratio for detecting cases of pericarditis (Bexiga *et al.* 2008).

Increase in total leukocyte count with neutrophilic leucocytosis was observed in both the groups, which might be in response to stress and infection. Abnormal biochemical changes included lowered A/G ratio due to decreased albumin and increased globulin levels. In general, plasma sodium, potassium and chloride levels were slightly below the normal range. The hypoalbuminemia could be due to sequestration of plasma proteins into extravascular spaces such as body



Figs 1-4. 1. Fibrinous deposits on the epicardium of the heart in animals suffering from pericarditis seen on B mode ultrasonogram. 2. Ultrasonogram in B mode showing pericardial sac filled with contents of mixed echogenicity indicating abscess. 3. Ultrasonogram in B mode showing anechoic fluid present outside the heart indicating pleural effusions: LV (left ventricle), IVS (interventricular septum), RV (right ventricle). 4. B mode ultrasonogram showing fibrinous strands in animals suffering from pleural effusions.

Table 1. Clinical and haemato-biochemical parameters in animals suffering with pericarditis and pleural effusions

Parameter	Pericarditis	Pleural effusions
Rectal temperature (°F)	103.80±0.43	103.20±0.25
Heart rate (per min)	95.00±6.87	76.00±2.19
Respiration rate (per min)	30.00±3.84	39.5±5.06
Hb (g/dL)	9.30±0.32	9.23±0.86
PCV%	28.32±3.28	33.53±2.50
TLC/µL	15970.00 ±1901.80	11283.33 ±1259.42
Neutrophil %	70.00±5.29	67.00±6.56
Lymphocyte %	28.00±4.24	31.67±5.65
Monocyte %	1.20±0.99	1.33±1.33
Eosinophil %	1.40±0.75	0.00±0.00
Total protein (g/dL)	7.27±0.24	7.98±0.11
Plasma albumin (g/dL)	2.42±0.09	2.85±0.09
A/G ratio	0.50±0.01	0.56±0.02
Plasma fibrinogen (g/dL)	0.85±0.25	0.73±0.10
Sodium (mEq/L)	128.70±1.80	128.50±2.55
Potassium (mEq/L)	3.50±0.20	5.08±0.04
Chloride (mEq/L)	93.20±2.01	93.00±3.10

cavity effusions and vasculopathy or decreased production, viz. malnutrition, hepatic failure, malabsorption, maldigestion (Jain 1986). Hyper-globulinaemia in the animals may be due to increase in α , β and α -globulin (Fisher and Pirie 1965). Elevated plasma fibrinogen concentration in the present study was suggestive of inflammatory process (Jain 1986).

The values of DLC, TLC and total protein (Table 1) of pleural fluid were within the normal reported reference range (Step *et al.* 2001), suggesting that the fluid was a transudate and could have resulted secondary to congestive heart failure. The exact cause was however not established in these cases. The low plasma levels of sodium, potassium and chloride may be due to the inappetence/anorexia in these animals.

Pericardiocentesis in 6 animals suffering from pericarditis yielded straw coloured to blood tinged fluid with offensive smell. Antibiotic sensitivity test (AST) was negative for 4 out of 6 samples. In 2 cases, microorganism isolated were *Corynebacterium* sp. and *Streptococcus* sp. *Corynebacterium* sp. was found susceptible to enrofloxacin, erythromycin and ofloxacin and resistant for cefotaxime, streptomycin, gentamicin, doxycycline, amikacin whereas *Streptococcus* sp. was susceptible to ceftriaxone, gentamicin, erythromycin and enrofloxacin and resistant for ampicillin, streptomycin, amoxicillin. Radostits *et al.* (2000) stated that the smell of the pericardial fluid which was reminiscent of retained placenta and toxic metritis was sufficiently diagnostic in cattle with traumatic pericarditis. Negative isolation and culture in 4 cases may be due to anaerobic nature of the organisms.

Radiographic examination in both pericarditis and pleural

effusions revealed that contour of diaphragm was lost and the cardiac silhouette was not visible and most parts of the thorax appeared as a homogenous area of opacity.

On ultrasonographic examination there was separation of parietal and visceral pericardium with hypoechoic to echogenic pericardial fluid and in some cases echogenic deposits and strands of fibrin were seen on the epicardium (Fig. 1). Strands of fibrin were seen floating in the fluid between the epicardium and pericardium. In 4 cases abscess was visible within the pericardial sac (Fig. 2).

Ultrasonographic examination in all the animals suffering from pleural effusions revealed a large amount of hypoechogenic fluid in the thorax (Fig. 3). In some cases, interlacing pattern of fibrin and anechoic fluid with fibrin shreds (Fig 4) was also present in the thoracic cavity. Similarly, Flock (2004) reported that pleural effusions appear as an anechoic area between the lung, thoracic wall, diaphragm and heart with acoustic enhancement deep to the lesion and often with septa floating within it.

The ultrasonographic findings of present study are similar to earlier findings (Abedaal *et al.* 2009 and Braun 2008). Anechoic fluid represents transudate, increased echogenicity points toward an increased cell count or total protein concentration (Reef *et al.* 1991). Pericardial effusions are differentiated from pleural effusions by finding the echo-free space surrounding the right ventricular wall and left ventricular free wall, but pericardial effusion is rarely imaged behind the left atrium (Salcedo 1985). Echocardiography has been described as the most valuable diagnostic tool for the evaluation of cardiovascular system and in differentiation of effusive and fibrinous pericarditis (Scott 2010 and Streeter and Step 2007).

Results of the present study suggested that clinical, haematobiochemical and radiographic findings could not differentiate pericarditis from pleural effusions. Ultrasonography was found to be a reliable tool for the diagnosis and differentiation of pericarditis and pleural effusions.

SUMMARY

The present study was conducted on 16 bovines suffering from pericarditis (6 buffaloes and 4 cows) and pleural effusions (6 buffaloes). Most of the animals were reluctant to move, had muffled heart sounds and brisket oedema. Haematobiochemical changes included increased total leukocyte count, neutrophilia, increased plasma globulins concentration, decreased albumin/globulin ratio and increased fibrinogen concentration. Indistinct line of diaphragm, loss of thoracic detail, ground glass appearance of thoracic cavity and loss of cardiac silhouette were the most common radiographic findings in both the conditions. Ultrasonographic findings in cases of pericarditis were separation of parietal and visceral pericardial layers filled with hypoechogenic to echogenic contents. Anechoic fluid

was present outside the heart and caudally in cases of pleural effusions.

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