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The Pharma Innovation

ISSN (E): 2277- 7695
ISSN (P): 2349-8242
NAAS Rating 2017: 5.03
TPI 2017; 6(6): 49-53

www.thepharmajournal.com
Received: 10-04-2017
Accepted: 11-05-2017

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Clinico-diagnostic aspects of right sided heart failure in dogs

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Abstract
A study on clinico- diagnostic aspects of right side heart failure in dogs was conducted among 10 dogs that were presented to Veterinary Hospital, Bhoiguda, Hyderabad, Telangana state. Dogs suspected for Right side heart failure based on history and clinical signs were subjected to detailed clinical, hemato-biochemical, radiographic, electrocardiographic and ultrasonographic exami nation. Ascites, dyspnoea, lethargy, exercise intolerance, weakness, pedal edema, fluid thrill, tachycardia were clinical manifestations. Decreased levels of hemoglobin, packed cell volume, total erythrocyte count, total protein and albumin with increased levels of ALT, AST, Creatine Kinase-Mb and LDH were the significant hemato-biochemical findings recorded. Thoracic and abdominal radiography revealed cardiomegaly and ground glass appearance respectively. Electrocardiography revealed low voltage QRS complexes suggestive of abdominal effusion. Ultrasonography of the ascitic abdomen revealed anechoic ascitic fluid with floating organs and congested hepatic and portal veins in the hepatic parenchyma. B and M-mode echocardiography and Doppler study revealed dilated right ventricular lumen with turbulent jet flow pattern and mosaic pattern suggestive of Right side heart failure.

Keywords: Right side heart failure, echocardiography, dogs

1. Introduction
Right heart failure results from increased ventricular after load i.e secondary to chronic pulmonary arterial disease and thromboemboli [2]. Failure of right side of the heart to pump blood at a sufficient rate to meet the needs of the body or to prevent blood from pooling within the veins of the body. Right sided heart failure could be one of the causes of cardiomegaly, pulmonary congestion and ascites manifested by pleural, pericardial and abdominal effusions that requires special attention [11]. Echocardiography is an important noninvasive tool for imaging the heart and surrounding structures and also to evaluate cardiac chamber size, wall thickness, wall motion, valve configuration and proximal great vessels [7].

2. Materials and Methods
The study was conducted in 10 dogs of various breeds, age and sex presented with the history and signs of Right sided heart failure such as ascites, exercise intolerance, respiratory distress, inappetance were selected for the present study. Six apparently healthy dogs with no clinical condition presented for routine clinical examination in the age group of 3-5 years irrespective of age, breed and sex were chosen as healthy control for the present study. Whole blood and serum was collected on day of presentation for complete blood picture and biochemical parameters estimation. Ascitic fluid was collected aseptically with a sterile disposable syringe and subjected for analysis. Electrocardiography was recorded from the affected dogs in right lateral recumbency by using standard bipolar and augmented unipolar limb leads at 25 mm/ sec speed. Lateral and ventro- dorsal aspects of radiographs were taken to evaluate the size and shape of the heart. Abdominal ultrasound and echocardiography was performed with *Ixos vet* Doppler ultrasound machine by using micro-convex 3.5 -5 MHz transducer.

3. Results
The clinical signs observed in the present study were ascites, dyspnoea, lethargy, exercise intolerance, weakness, pedal edema and inappetance (Fig.1 and 2). Tactile percussion of the abdomen revealed fluid thrill suggestive of ascites. Physical examination findings revealed normal rectal temperature and upon auscultation, tachycardia with arrhythmias were evident. The mean values of hemoglobin, packed cell volume, total erythrocyte count were 10.85±0.94 g/dl, 36.35± 0.83%, 5.35±1.25 x 10^6 /µl respectively, which were significantly lower as compared with healthy control group. While, total leucocyte count (10.43±0.35 x 10^3 /µl) and

Differential leucocyte count (Neutrophils: 63.13±1.23, lymphocytes: 12.26±0.88, monocytes: 1.10±0.33 and eosinophils: 1.58±1.14) were within normal ranges as compared to healthy control group. Serum biochemical profile revealed a significant increase in the mean values of Creatine Kinase -Mb (68.74± 2.87 U/L) and LDH (287±10.65U/L) values with a non-significant increase in the mean values of ALT (82.48±7.22 U/L) and AST (95.37±9.47 U/L). There was a significant decrease in the mean values of total protein (4.68± 2.77 g/dl) and albumin (1.72±1.63 g/dl). The results were presented in table 1. Ascitic fluid analysis in affected dogs revealed total protein (3.24±0.18 g/dl), nucleated cell count (1846±12.83/cmm), specific gravity (1.008±0.64) indicating modified transudate. Electrocardiography of the affected dogs revealed low voltage QRS complexes (Fig. 3) indicating that the fluid was evident. Lateral radiographs of abdomen revealed ground glass appearance (Fig.4) indicating ascitic fluid. While, lateral and ventro-dorsal views of chest radiographs revealed cardiomegaly with sternal contact (Fig.5). Ultrasonography of the ascitic abdomen revealed anechoic abdominal fluid with floating viscera (Fig.6) and dilated hepatic and portal vessels in the hepatic parenchyma (Fig.7). Two dimensional B and M-mode echo cardiographic examination of the affected dogs revealed dilatation of the right ventricle (Fig.8) along with turbulent jet flow pattern upon pulse wave Doppler (Fig.9) and mosaic pattern upon color flow Doppler (Fig.10) at tricuspid valves confirming presence of Right side heart failure.

Table 1: Hemato-biochemical parameters in Right side heart failure affected dogs

S. No	Parameter	Healthy control	RHF affected dogs
1.	Hb (g/dl)	12.92±0.44	10.85±0.94*
2.	PCV (%)	41.73±1.34	36.35± 0.83**
3.	TEC (x 10^6 /µl)	7.19±0.23	5.35±1.25*
4.	TLC (x 10^3 /µl)	9.86±1.65	10.43±0.35
5.	Neutrophils (%)	65.98±2.70	63.13±1.23
6.	Lymphocytes (%)	12.77±1.23	12.26±0.88
7.	Monocytes (%)	1.18±0.27	1.10±0.33
8.	Eosinophils (%)	1.68±1.22	1.58±1.14
9.	CK-Mb (U/L)	21.29±0.61	68.74± 2.87**
10.	LDH (U/L)	48.48±1.39	287±10.65**
11.	ALT (U/L)	32.80±1.60	82.48±7.22
12.	AST (U/L)	43.05±1.22	95.37±9.47
13.	TP (g/dl)	6.22±0.03	4.68± 2.77*
14.	Albumin (g/dl)	2.81±0.07	1.72±1.63*

* Significant at (p<0.05), ** Significant at (p<0.01)

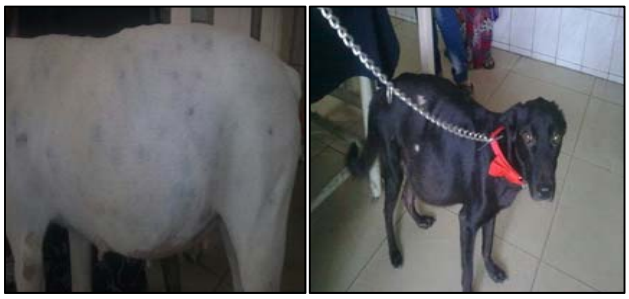

Fig 1: Abdominal distension suggestive of ascites in RHF affected dogs.

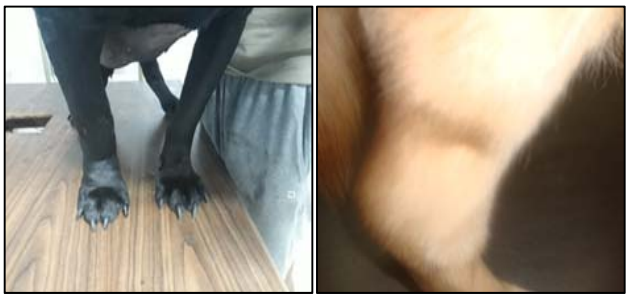

Fig: 2. Pedal edema with pitting nature in RHF affected dog.

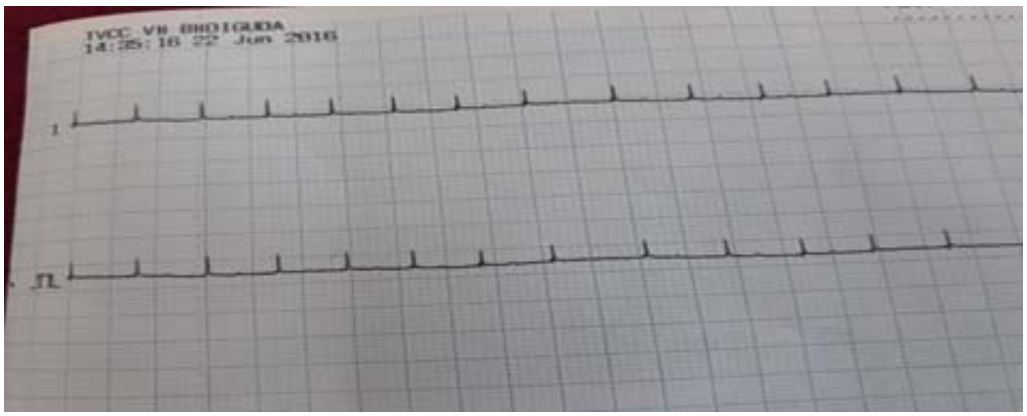

Fig 3: Electrocardiograph- Low voltage QRS complex in suggesting abdominal fluid in RHF affected dog.

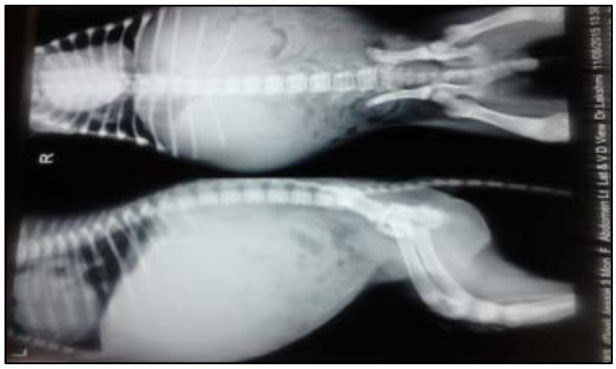

Fig 4: Lateral & ventro dorsal view of Radiograph- Ground glass appearance in RHF affected dog.

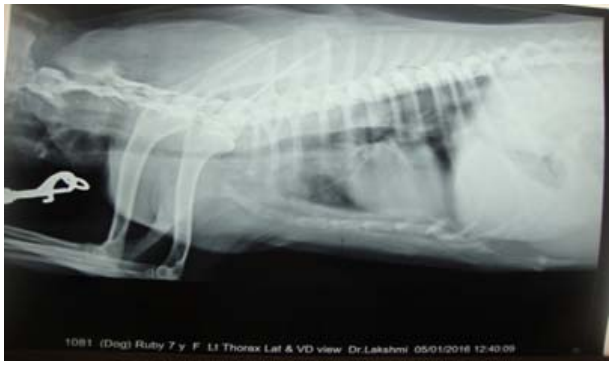

Fig 5: Lateral view of Radiograph- Cardiomegaly with sternal contact in RHF affected dog.

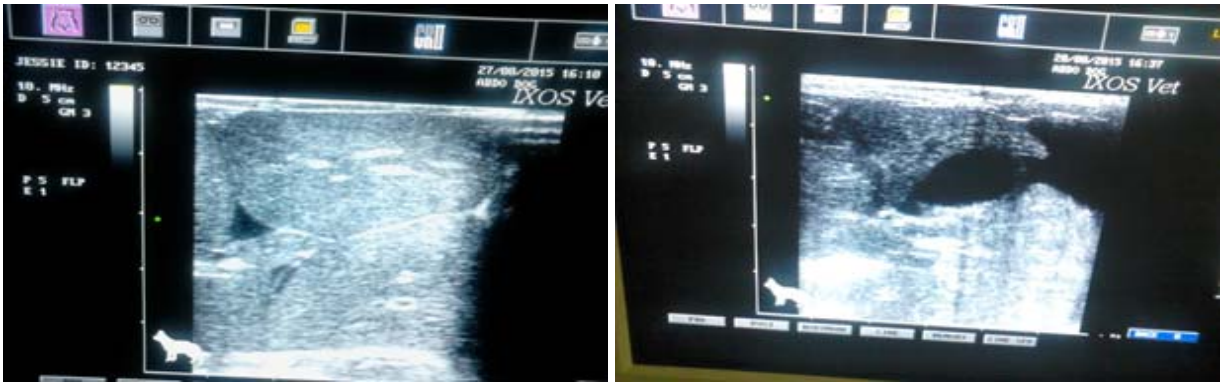

Fig 6: Ultrasonography of the abdomen- Anechoic ascitic fluid with floating viscera in RHF affected dog.

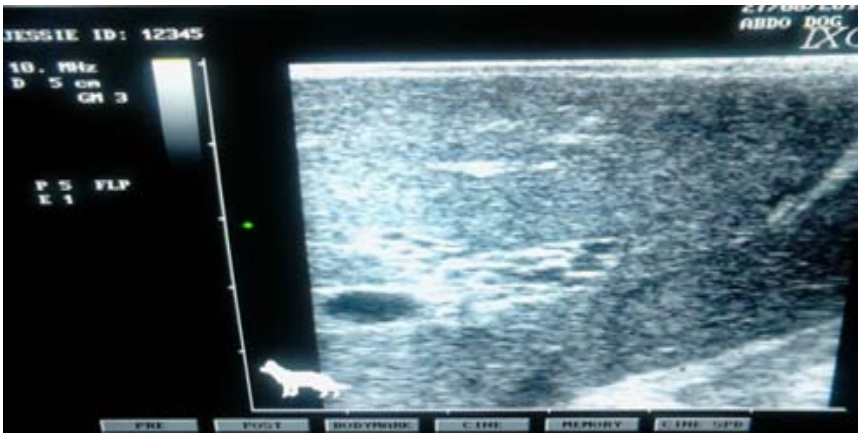

Fig 7: Ultrasonography of the abdomen- Dilated hepatic and portal vessels suggesting hepatic congestion in RHF affected dog.

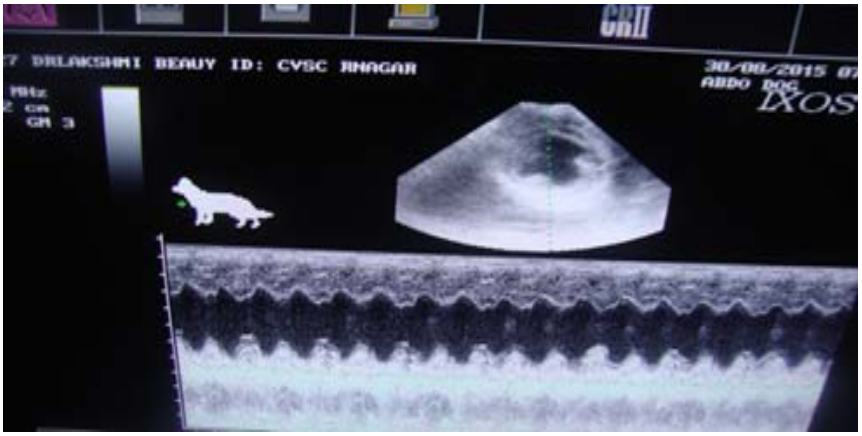

Fig 8: Echocardiography of the heart- Dilated right ventricular lumen suggesting RHF in a dog.

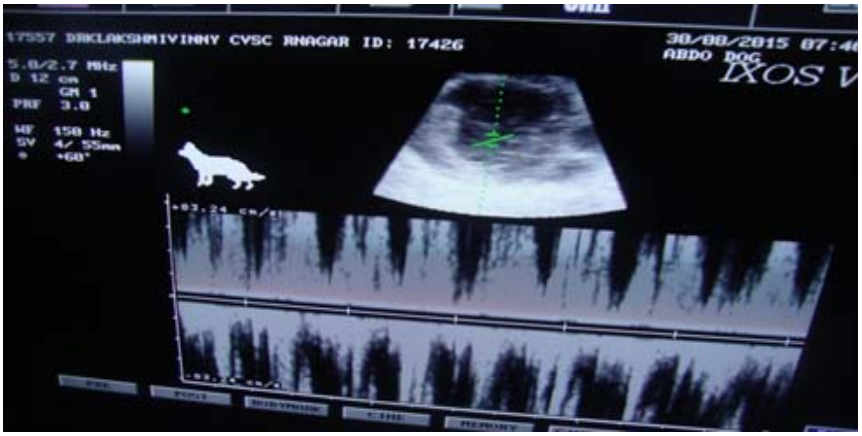

Fig 9: Echocardiography of the heart- Turbulent jet flow pattern upon pulse wave Doppler in RHF affected dog.

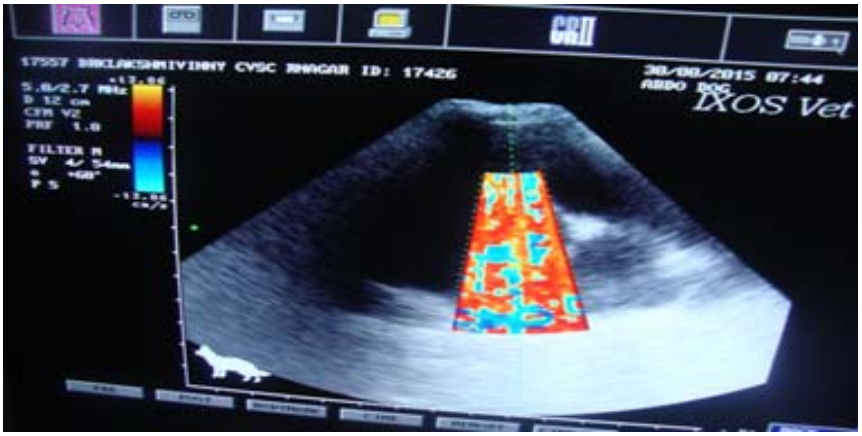

Fig 10: Echocardiography of the heart- Mosaic pattern upon continuous wave Doppler in RHF affected dog.

4. Discussion

The clinical findings in the present study were in agreement with the reports of [2, 3] who observed exercise intolerance, prolonged capillary refill time, ascites, dyspnoea, jugular venous distension, pulsation, arrthymia with pulse defecits and adventitial lung sounds (crackles and wheezes) as clinical manifestations and dyspnoea in the present study might be due to pulmonary infiltrates, abdominal effusion or pleural effusion. Exercise intolerance may be an early sign of decompen sation with heart disease. Peripheral vasoconstriction caused by poor cardiac output may lead to pale mucous membranes [5]. Right sided heart failure caused poor venous return to the heart, accumulation of fluid in the body cavities leading to ascites [11]. Decreased levels of total protein and albumin in the present study might be due to inappetance and hepatic enlargement. Elevated levels of ALT and AST were in accordance with [8, 11] who reported that increase in ALT and AST was a consequence of hepatic congestion. Pre renal azotemia, high ALT levels and electrolyte abnormalities may be evident in severe heart diseases. Increase in values of cardiac biomarkers CK-Mb and LDH were in agreement with [12] who opined that these are more specific to cardiac muscle and helps to differentiate myocardial ischemia from skeletal muscle damage. CKMb is functionally unbound, dissolved in cytosol and predominantly found in the myocardium of the heart at 1-42% [10]. Modified transudate ascites with pleural effusion and distended jugular veins were suggestive of congestive right sided heart failure [3]. Radiographically, increased cardiac sternal contact with a cranial cardiac wall bulge in combination with a greater increase in cranio-caudal cardiac diameter as compared to apicobasilar diameter and electrocardiographically, small R wave amplitude with increased P wave duration and low voltage QRS Complexes were suggestive of right ventricular enlargement [1, 9]. With the impairment of right side of the heart, systemic venous pressure rises resulting in hepatomegaly and ascites [4]. Right sided heart failure results in pleural effusion, enlarged caudal venacava, ascites and hepatomegaly. Echocardiography revealed severe dilatation of right ventricle and atrium with right ventricular hypocontractility. Right ventricular dilatation classically develops as a sequelae to either myocardial failure or ventricular volume overload conditions. Reduced contractility of the right ventricle leads to right heart systolic ejection failure, with a resultant increase in right ventricle volume overload, widening of tricuspid valvular annulus resulting in tricuspid regurgitatation and secondary right atrial dilatation [6].

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