

LEFT SIDED CONGESTIVE HEART FAILURE IN CANINES: AN OVERVIEW

^{1*}Arpita Pattnaik,²Rewa Rajeswari,³Vivek Kirti,¹Prakhar Gupta

¹M.V.Sc Scholar, Division of Medicine, ICAR-Indian Veterinary Research Institute, Izatnagar, Bareilly, 243122, ²M.V.Sc Scholar, Division of Pathology, College of Veterinary Science and Animal Husbandry, Orissa University of Agriculture and Technology, Bhubaneswar, 751003, ³College of Veterinary Science and Animal Husbandry, OUAT, Bhubaneswar, 751003

Corresponding author e-mail: @pattnaikarpita9@gmail.com

DOI: <https://doi.org/10.5281/zenodo.14997704>

ABSTRACT

Left sided heart failure is a quite common in Indian and Exotic dog breeds. Prognosis of the condition is quite poor unless proper management and care is not taken. The affected dogs are mainly seen with the signs like coughing, fatigue, lethargy, weakness, dyspnoea which can have numerous causes like valvular disease, parasitic or bacterial endocarditis, arrhythmias or dilated cardiomyopathy. There are various treatment and medications available for the condition but once the critical period of the pathogenesis has surpassed, the condition will lead to death of the animal. This article is an overview of how left side heart failure condition in canines occur, diagnosis, treatment, and management.

Keywords: Left sided heart failure, Exotic dog breeds, CHF, pulmonary edema

I. INTRODUCTION

Left-sided congestive heart failure is the most common form of CHF. CHF is the failure of the cardiac output to meet the requirements of body with retention of excess fluids and sodium. It is the inability of the heart (specifically the left atrium) to accommodate the blood volume returning from pulmonary circulation. Increased Left atrial pressure causes the pulmonary veins to become congested and unable to transmit blood volume into the chamber. Blood leaks through the mitral valve back into the left atrium and then backs up into the lungs-fluid then seeps into the lungs tissue causing pulmonary edema. The compensatory mechanisms of the heart are overwhelmed and result in congestive failure with excess blood volume in the pulmonary veins.

II. ETIOLOGY

- ✓ Valvular diseases (endocarditis, congenital valvular defects)
- ✓ Myocardial diseases (Myocarditis, Cardiomyopathy)
- ✓ Congenital anatomical defects producing shunts
- ✓ Pulmonary hypertension
- ✓ Toxins affecting cardiac conduction
- ✓ Pressure load (aortic or pulmonary valve stenosis)
- ✓ Volume load (aortic and mitral valve insufficiency)
- ✓ Pumping defects (systolic failure)
- ✓ Filling defects (diastolic failure)

III. PATHOGENESIS

- Increased pulmonary venous pressure causes pulmonary venous congestion
- decreased compliance of the lungs,
- increase in respiratory rate
- increase in the work of breathing

- exercise intolerance
- Bronchial capillary congestion and edema results in encroachment on airways and a decrease in ventilatory efficiency (Tanai et al, 2015)
- When the venous hydrostatic pressure is exceptionally high the net force for filtration of fluid across the pulmonary capillary bed is greatly increased
- Resulting in pulmonary edema with presence of fluid around septal vessels and in alveolar spaces accompanied by marked impairment of gas exchange

IV. CASE REPORT

A Female Labrador Dog of 7yrs age, 53kg weight at TVCC, OUAT, History- Reduced appetite to anorexia, Reduced activity, Progressive abdominal distension, Exercise intolerance, listlessness during night hours, Dyspnoea temp-101degF

Palpation revealed-Fluid accumulation in abdominal region

Lungs auscultation-Stridor sound

ECG-Tall and wide QRS complex suggesting cardiac chambers enlargement, tachycardia,

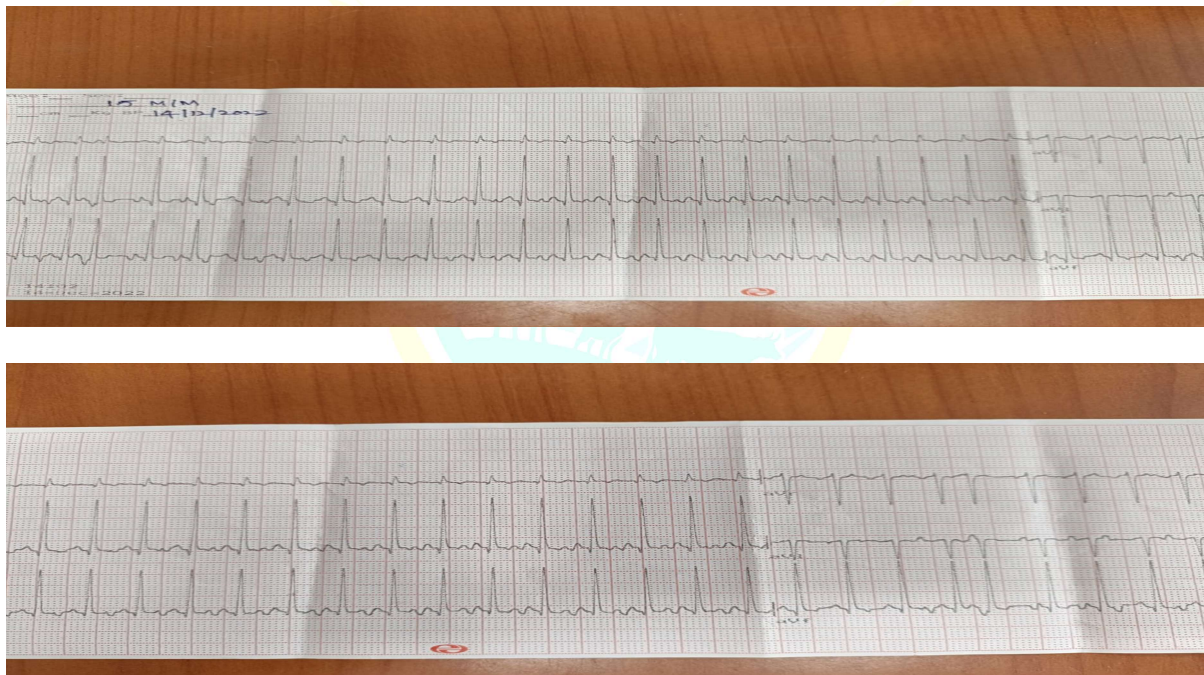


Fig 1 (a) & (b): ECG-Tall and wide QRS complex suggesting cardiac chambers enlargement, tachycardia, Echo-Left Ventricular dilatation, Stroke Volume increased

V. HAEMATOLOGICAL AND BIOCHEMICAL RESULTS OF CASE

- ✓ Blood Examination-Hb-14.3gm%
- ✓ TCL-9000/Cumm, TEC- 5.79×10^6 /microL
- ✓ PCV-41%, PLT- 1.28×10^5 /microL
- ✓ DC-N-54%, E-1%, B-1%, L-40%, M-4%

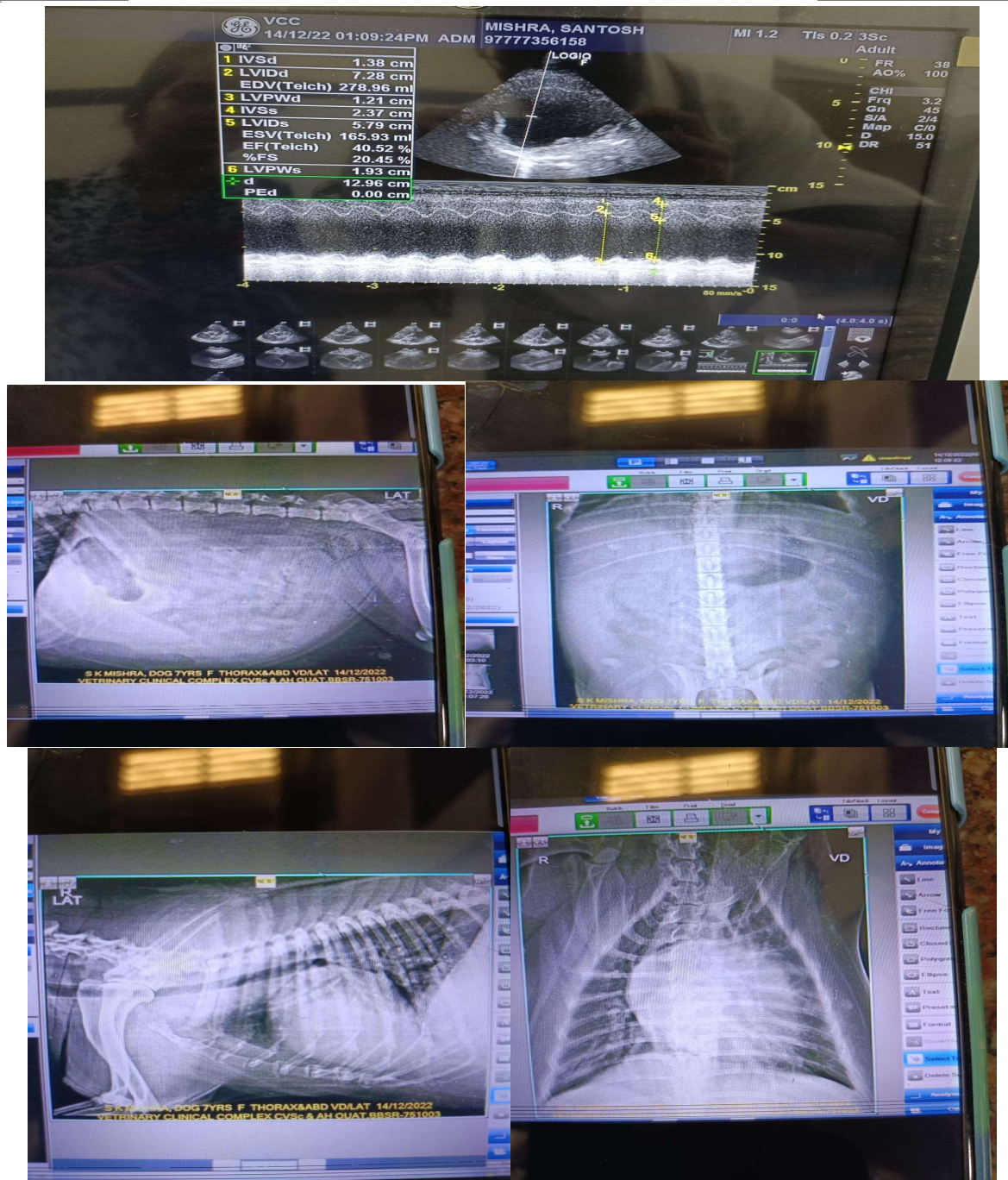


Fig 2: (a), (b), (c) & (d): Xray-Cardiomegaly, Congestion of Lung, Hepatomegaly, Stomach empty filled with gas, Fecaliths presence

Biochemical tests

- ✓ ALP-41.4IU/L
- ✓ ALT-51.2 IU/L
- ✓ AST-55.5 IU/L
- ✓ Urea-39.2IU/L
- ✓ Creatinine-1.54IU/L

- ✓ TP-3.51 IU/L

VI. CLINICAL SIGNS

- ✓ Heart rate increased
- ✓ Increase in rate and depth of respiration at rest with cough

- ✓ Presence of crackles at the base of the lungs
- ✓ Increased dullness on percussion of the ventral borders of the lungs
- ✓ Severe dyspnoea, cyanosis, exercise intolerance, excessive panting, persistent loss of appetite, swollen belly, pale/bluish M.M, generalized weight loss & muscle wasting

VII. CLINICAL PATHOLOGY

- ✓ Proteinuria
- ✓ Increase S.G.P.T level
- ✓ Urea and alkaline phosphatase level may be high
- ✓ Serum or plasma concentration of cardiac troponin provides an excellent cardiac biomarker in large animals

VII. DIAGNOSIS

Diagnosis of the typically involves evaluating the signalment, history and physical examination findings as well and physical examination findings, as well as results of diagnostic tests such as radiography, electrocardiography, and tests such as catheterization, CT, MRI, or nuclear studies. Physical examination like auscultation by a pediatric stethoscope, knowing the left apex beat by palpation, detection of a continuous murmur, palpation of ventral thorax, detection of mucous membrane colour, palpation of arterial pulse, a weak pulse indicates decreased stroke volume, hypovolemic shock, a bounding pulse is usually caused by reduced diastolic pressure can be useful in diagnosing the disease (Atkins et al,2009). Radiography of thorax on the lateral view, dogs can be normal, shallow chested and on dorsoventral view, they can be normal, narrow chested or barrel chested. In ECG the condition shows prominent R waves and deep S waves along with potential ST-T wave changes.

VIII. TREATMENT

Pimobendan, an inotropic drug and vasodilator approved by FDA in 2007, is used in dogs with left side heart failure @0.2-0.3mg/kg. Loop diuretics are effective to decrease the circulating blood volume and reduce signs referral to edema and effusion like Furosemide @4-8mg/kg body weight (Beaumier et al,2018). Dobutamine administered i/v @2.5-20 mcg/kg/min to increase the conduction of the AV node. Cardiac glycosides like digitoxin that increase the vagal tone of the heart can sometimes be used along with the ACE (angiotensin converting enzymes) inhibitors which blunts the increase in systemic vascular resistance, hypertrophy and aldosterone release. Vasodilators exert a positive effect in left side heart failure like sodium nitroprusside, isosorbide dinitrate, hydralazine. Phosphodiesterase inhibitors like sildenafil, tadalafil are used to relax the smooth muscle in pulmonary arterioles. Apart from this Oxygen therapy, thoracocentesis, nutritional considerations etc should be implemented,

IX. MANAGEMENT AND CONTROL

Its important to provide a balanced diet including sodium restriction, supplementation of Omega 3 fatty acid, recommendation for exercise or walking along with weight management. Stress control, regular monitoring and follow up appointments should be done. Routine health checkup, monitoring weight, blood pressure (Davies et al,2015) and electrolyte levels should be strategically carried out.

X. CONCLUSION

The dogs affected with left side heart failure have a very poor prognosis. So it's necessary to take proper care, and management and overall health for your dog to lead a happy and comfortable life. Early detection by noticing the signs and symptoms and effective treatment have proved to be very effective in certain cases. There is a hope for reduction in the cases of left side heart failure in dogs in

future if there is a collaborative effort between the owners and veterinarian and agree to the preventive guidelines.

XI. REFERENCES

- Atkins C, Bonagura J, Ettinger S, Fox P, Gordon S, Haggstrom J, Hamlin R, Keene B, Luis-Fuentes V, Stepien R. Guidelines for the diagnosis and treatment of canine chronic valvular heart disease. *J Vet Intern Med.* 2009 Nov-Dec;23(6):1142-50. doi: 10.1111/j.1939-1676.2009.0392.x. Epub 2009 Sep 22. PMID: 19780929.
- Beaumier A, Rush JE, Yang VK, Freeman LM. Clinical findings and survival time in dogs with advanced heart failure. *J Vet Intern Med.* 2018 May;32(3):944-950. doi: 10.1111/jvim.15126. Epub 2018 Apr 10. PMID: 29635788; PMCID: PMC5980388.
- Davies T, Everitt S, Cobb M. Variation in the management of congestive cardiac failure in dogs. *Vet Rec.* 2015 Apr 25;176(17):435. doi: 10.1136/vr.102818. Epub 2014 Dec 31. PMID: 25552475; PMCID: PMC4431339.
- Tanai E, Frantz S. Pathophysiology of Heart Failure. *Compr Physiol.* 2015 Dec 15;6(1):187-214. doi: 10.1002/cphy.c140055. PMID: 26756631.

