CASE REPORT

Therapeutic Management of Congestive Heart Failure Secondary to Dilated Cardiomyopathy in a Male Labrador Dog: A Case Report

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Dilated cardiomyopathy (DCM) is characterized by significant systolic dysfunction and expansion of the heart chambers (Oyama, 2015). In dogs, DCM is one of the most prevalent acquired cardiovascular conditions. Since idiopathic DCM accounts for the majority of cases in dogs, most treatments are categorized as ''Band-Aid therapies,'' which temporarily relieve symptoms but don't address the underlying source of the illness (Sanderson, 2006). Without any indication of heart symptoms, the concealed stage of this illness is typified by structural or electrical alterations. The main morphologic signs of systolic dysfunction are left and rarely right ventricular dilatation in systole and later diastole. Ventricular premature complexes are the most common type of electrical abnormalities (Wess *et al.,* 2017). Clinical indicators of congestive heart failure (CHF), such as elevated respiratory rate, dyspnoea, coughing, or exercise intolerance, are what define the overt stage of the condition (Friederich *et al*., 2020). The present report describes a case of congestive heart failure secondary to dilated cardiomyopathy in a Labrador dog and its successful therapeutic management.

Case History and Observations

An 11-year-old male Labrador dog weighing 28 kg was reported to the Veterinary Clinical Complex (VCC) of the College in Guwahati (Assam, India) with the chief complaints of persistent coughing for 1 month, blood in stool, swollen abdomen, inability to pass urine, decreased appetite and reduced activity. Physical examination of the dog revealed that the general appearance of the animal was depressed with a normal body temperature of 100.6°F and pale mucous membrane. Auscultation of the lungs and heart revealed congestion and the heart rate was 62 beats/min. The animal had a swollen abdomen but ribs were prominent, fluid thrill on percussion of the abdomen and pitting pedal oedema was also observed (Fig. 1).

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Fig. 1: Animal with swollen abdomen with prominent ribs and pitting pedal oedema

An electrocardiogram (ECG) of the heart showed widening of the QRS complex and ST depression (Fig. 2) with left ventricular enlargement and systolic dysfunction (Fig. 3). Radiographic image of the lateral thoracic region showed increase in width of the heart with vertebral heart score (VHS) of 11 (Fig. 4a) and the radiographic image of the ventro-dorsal aspect revealed biventricular enlargement

of the heart (Fig. 4b). The abdominal ultrasound showed floating liver lobes indicating ascites (Fig. 5a), enlargement of portal vein and marked hepatic congestion (Fig. 5b), cystic sludge with cystitis (Fig. 5c) and degenerative changes of the kidney (Fig. 5d).

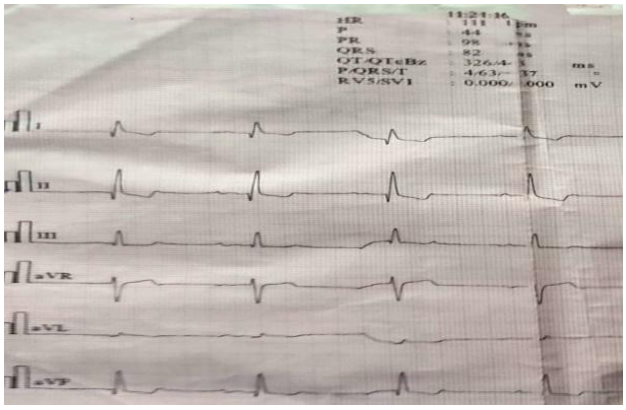

Fig. 2: ST Depression and QRS widening (82 ms)

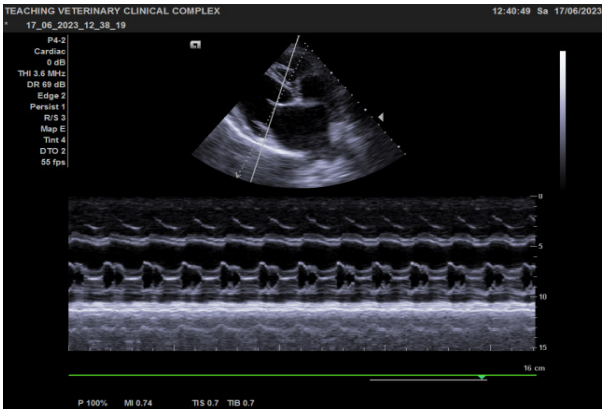

Fig. 3: Left ventricular enlargement and systolic dysfunction

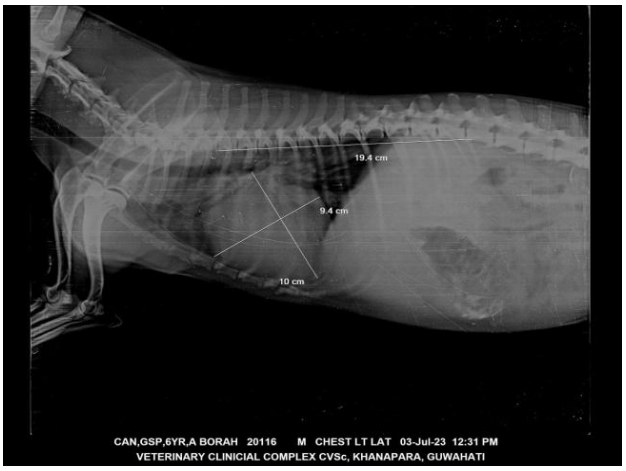

Fig. 4a: Vertebral heart score (VHS) of 11(lateral thoracic)

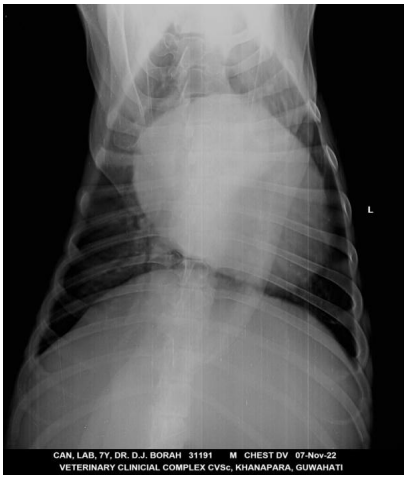

Fig. 4b: Biventricular enlargement of the heart (ventro-dorsal)

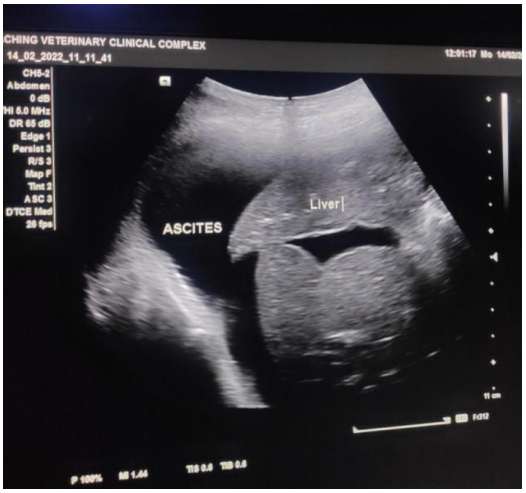

Fig. 5a: Floating liver lobes indicating ascites

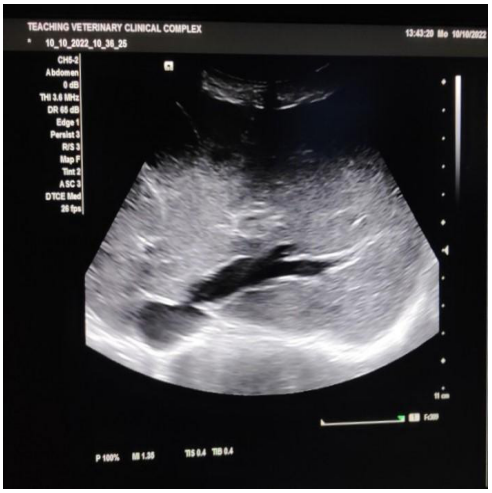

Fig. 5b: Portal vein enlargement and hepatic congestion

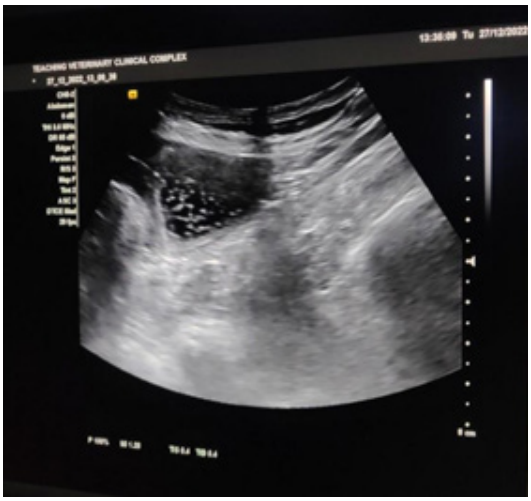

Fig. 5c: Cystic sludge with Cystitis

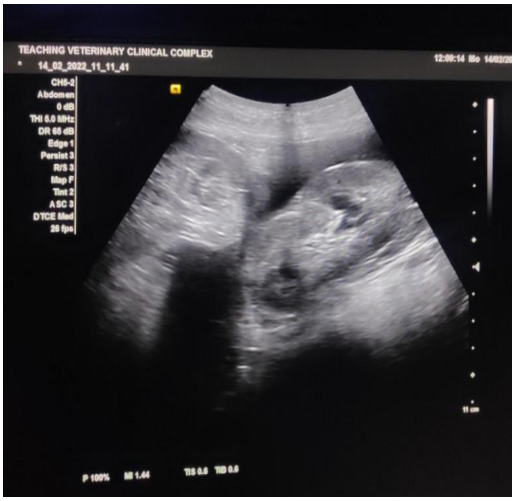

Fig. 5d: Degenerative changes in kidney

The haemato-biochemical analysis revealed anaemia, thrombocytopenia, neutrophilia elevation of the ALT, AST, BUN and creatinine (Table 1). The blood smear examination was negative for *Babesia* spp., *Ehrlichia canis* and *Anaplasma* spp. Based on the history, clinical signs, laboratory findings, electrocardiogram, echocardiography, radiographic imaging and abdominal ultrasound, it was confirmed that the dog was affected with congestive heart failure secondary to dilated cardiomyopathy.

Table. 1: Haemato-biochemical parameters of the CHF-DCM affected dog before and 15 days after treatment

Parameters	Before Treatment	After Treatment	Reference Range*
RBC (million/mm^3)	5.35	6.00	5.50-7.87
PCV/HCT (%)	29.0	33.0	35-57
Haemoglobin (g/dL)	9.0	10.4	11.9-18.9
Thrombocytes (m/mm^3)	104.0	180	120-600
MCH (pg)	16.8	19.0	19.5-24.5
MCHC (cells/mm^3)	31.0	31.9	32-36.3
MCV (fL)	63.0	66.9	66-77
Neutrophils (%)	87.5	80.0	58-85
Lymphocytes (%)	11.1	11.0	10.0-30.0
Monocytes (%)	2.8	2.3	2.0-10.0
ALT (U/L)	110.9	76	10-109
AST (U/L)	64.3	23	13-15
Direct bilirubin (mg/dL)	0.3	0.1	0-0.1
Indirect bilirubin (mg/dL)	0.1	0.1	0-0.3
Total bilirubin (mg/dL)	0.4	0.1	0-0.4
BUN (mg/dL)	70.6	33.4	8-28
Creatinine (mg/dL)	2.9	1.2	0.5-1.7

*The Merck Veterinary Manual (2016), 11th edn.

Treatment and Discussion

The dog was initially stabilized by emergency abdominocentesis to relieve the distension of the abdomen. Thereafter, Tab Pimobendan (Safeheart) @ 0.5 mg/kg b.wt. orally BID, Inj. Furosemide (Lasix) @ 4 mg/kg, i/v, Inj. 20% Mannitol @ 0.5 g/kg i/v, and Taurine and carnitine supplement (Vencard) @ 1 tab orally BID were administered. Supportive medications given included an iron supplement for anaemia (Ansurose injection 5 mL) @ 2 mL mixed with 100 mL normal saline solution for 3 days and hepatoprotective syrup (Hepamust) @ 1 tsp/10 kg b.wt., orally BID; multivitamin supplement (Zipvit) @ 1 tsp/10 kg b.wt., PO, BID; and haematinic (Sharkoferrol Pet) @ 1 tsp/10 kg b.wt. orally BID. On the 15th day after initial therapy, the dog showed marked improvement in the haemato-biochemical profile (Table 1) and the medications were advised to be continued lifelong with monthly reviews.

Dilated cardiomyopathy is a myocardial disorder which is characterized by enlargement of the cardiac chambers, thinning of the ventricular wall, and a decrease in the contractile function of the heart (Oyama, 2015). Congestive heart failure is a clinical syndrome brought on by circulatory congestion and oedema as a result of heart disease (De Morais and Schwartz, 2005). The most common cause of congestive heart failure in large breed dogs, such as Labrador retriever, is dilated cardiomyopathy (Kumari *et al.,* 2011; Jeyaraja *et al.,* 2019). In this case, ECG was successfully used for the detection of rhythm disturbances and cardiac chamber enlargement in a dog suffering from DCM. The result of the ECG showed a widening of the QRS complex and ST depression. Dogs with DCM have been found to exhibit a variety of ECG abnormalities, including tall and wide P waves, tall and wide QRS complexes, rhythm disturbances such as supraventricular tachycardia (SVT), atrial premature contraction (APC), atrial fibrillation, ventricular premature complex (VPC) and ventricular tachycardia (VT), which are highly significant in terms of both diagnostic and prognostic factors (Padalkar, 2012; Velhankar, 2013). The electrocardiography, radiography and echocardiographic values followed the reports published earlier (Rajamohan *et al.,* 2018).

Congestive heart failure is a primary cause of progressive chronic kidney disease (CKD), and there is growing evidence that CKD itself plays a key role in severe cardiac damage. Uncontrolled congestive heart failure (CHF) frequently results in a sharp decline in renal function (Silverberg *et al.,* 2004), which was also revealed in this case. The primary cause of the anaemia in CHF is the frequently associated CKD, but cytokines also have an inhibitory effect on the production of erythropoietin and bone marrow activity, as well as on the absorption of iron from the gut and the release of iron from iron stores (Silverberg *et al.,* 2006). Anaemia alone has the potential to exacerbate renal and cardiac dysfunction and increase a patient's resistance to standard CHF treatment. Anaemia in congestive heart failure has been linked to a higher severity of the condition (Silverberg *et al.,* 2006). Heart failure (HF) is characterized by the inability of systemic perfusion to meet the body's metabolic demands and is usually caused by cardiac pump dysfunction and may occasionally be present with symptoms of a non-cardiac disorder such as hepatic dysfunction (Alvarez and Mukherjee, 2011). The primary pathophysiology involved in hepatic dysfunction secondary to heart failure is either due to passive congestion from increased filling pressures or due to low cardiac output and consequences of the impaired perfusion. Passive hepatic congestion due to increased central venous pressure may cause elevation of the liver enzymes as well as both direct and indirect serum bilirubin (Alvarez and Mukherjee, 2011).

Pimobendan, a positive inotropic and vasodilatory medication, is licensed by the US FDA Center for Veterinary Medicine for use in dogs with chronic mitral valve disease and dilated cardiomyopathy-related CHF. It is now a standard of care for the management of the patients with CHF (Reina-Doreste *et al.,* 2014). In this case, the dog was managed with Pimobendan, which, when used as adjunctive therapy, along with diuretics, taurine-carnitine, hematinics, and liver protectants, can prolong the survival period of the animal (Fitton and Brogden, 1994). Furosemide stimulates the kidneys to remove excess fluid from the body and reduces the preload of the heart (Swathi *et al.,* 2020).

In brief, although the condition is irreversible, treatment mainly aims at reducing symptoms of heart failure and improving cardiac function. Once detected, patient needs to be on medications to prolong longevity. A large number of pharmacological options are available for improving cardiac function like ACE inhibitors, cardiac glycosides, beta-blockers, inotropic agents, phosphodiesterase inhibitors etc. In this case, Pimobendan as a positive inotropic drug helped improve cardiac function.

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