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Clinical Article

CLINICO-PATHOLOGICAL AND NECROPSY FINDINGS IN A GREAT DANE DOG WITH DILATED CARDIOMYOPATHY

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SUMMARY

A two-year-old male Great Dane dog was presented with a complaint of exercise intolerance, distension of abdomen and breathlessness since 1 week. Electrocardiography revealed left bundle branch block and electrical alternans. Chest X-ray and echocardiography confirmed pleural effusion. In addition, echocardiography confirmed the diagnosis to be dilated cardiomyopathy. The dog was given standard therapy to which later it became refractory and hence, the animal was euthanized humanely and necropsy was performed. Gross necropsy changes were indicative of cardiomegaly and involvement of other vital organs. Histopathology of vital organs revealed degenerative changes in the cardiac musculature and liver parenchyma; emphysema and consolidation of lungs; renal tubular degeneration and exfoliation and depletion in splenic white pulp.

Keywords: Cardiomegaly, Dog, Pleural effusions, Necropsy

Dilated cardiomyopathy (DCM) is characterized by impaired myocardial contractility with dilation of the left ventricle (LV) or both ventricles and poor cardiac output with or without arrhythmias. DCM as an entity probably represents the end-stage of different pathologic processes or metabolic defects involving myocardial cells or the intercellular matrix (Ware, 2011). DCM is most common in large and giant breeds of dogs such as Doberman Pinschers, Great Danes, Saint Bernards, Irish Wolfhounds, Boxers etc. (Ware, 2011). In case of Great Danes, the condition is genetic/familial in nature with inheritance as an X-linked recessive trait (Meurs *et al.,* 2001). It is most often diagnosed in middle-aged dogs and the prevalence increases with age. Aim of the present communication was to discuss in detail the clinical signs, diagnosis, hemato-biochemical, serum oxidant-antioxidant and necropsy (gross and histopathologic) changes in a Great Dane dog with DCM.

A two-year-old male Great Dane dog weighing 45 kg was presented to Teaching Veterinary Clinical Complex of the college with a history of exercise intolerance, distension of abdomen, breathlessness since 1 week. Animal was being fed homemade diet containing rice and boiled meat for more than a year. Animal was immunized and dewormed as per standard schedule. Clinical examination revealed euthermia, abdominal distension, pale conjuctival mucous membrane, cachexia, positive hepatojugular reflux, normal capillary refill time, muffled heart sound on auscultation, normocardia and eupnoea. ECG revealed left bundle branch block (LBBB) (Fig. 1) and electrical alternans. Chest X- ray (Fig. 2) revealed marked pleural effusion obscuring the cardiac silhouette and interfered in measuring the vertebral heart score. This was further substantiated with the help of 2D echocardiography (Fig. 3). M-mode echocardiography (Fig. 4) revealed increase in left ventricular internal diameter during diastole (LVIDd) (60.6 mm; reference range 44-59 mm), left ventricular internal diameter during systole (LVIDs) (65.8 mm; reference range 34-45 mm) and end point septal separation (EPSS) (12.5 mm; reference range <7 mm) where as decrease in fractional shortening (FS) (7.9%; reference range 33-46%), ejection fraction (EF) (3%; reference range 50-65%) and sphericity index (SI) (1.32; reference range >1.6). LVIDd and LVIDs indicate LV size during diastole and systole, respectively and hence, better predictors of LV dilation. EPSS is a parameter for the evaluation of left ventricular filling and function. Left ventricular FS is the most commonly used echocardiographic index of myocardial function. A reduced FS is normally observed secondary to poor preload, increased afterload, or decreased contractility and seen in cases of DCM and myxomatous mitral valve degeneration. Ejection fraction is a measure of volume leaving the left ventricle regardless of whether it flows through the aorta, a shunt, or the mitral valve and it is inversely related to EPSS. Decreased EF is one of the major criteria for diagnosis of DCM along with decreased FS. SI is an indication of the degree of rounding of the LV.

Human qualitative immunochromatography cardiac troponin-I kit (Instant View Troponin-I Whole blood/Serum Test kit, Alfa Scientific Designs Inc., USA) was used with an analytical sensitivity of 1.5 ng/ mL which

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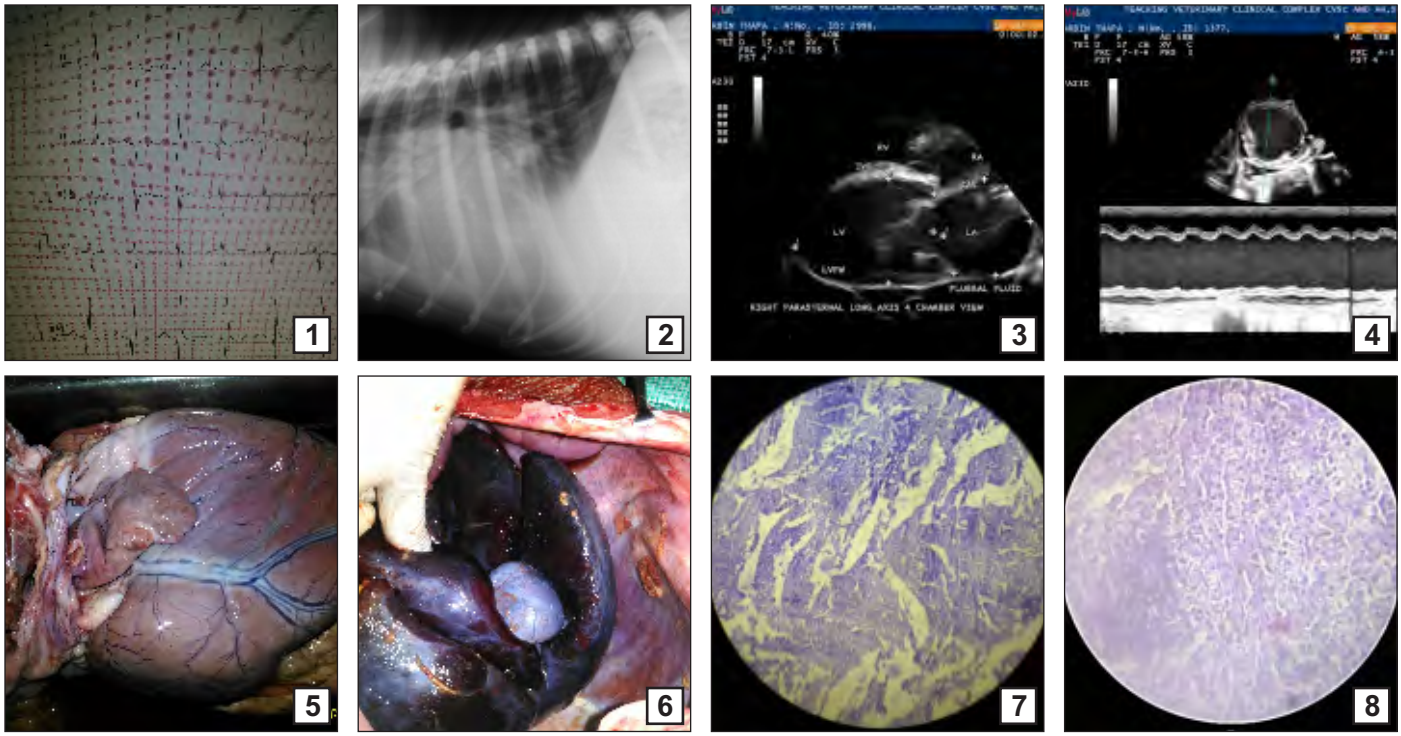

Figs. 1-8. **(1)** ECG showing LBBB with QRS duration 0.07 sec (Lead II_ paper speed 50 mms, 1cm=1mV) (2); **(2)** Chest X–ray showing pleural effusion obscuring the cardiac silhouette (Left lateral); **(3)** Right Parasternal Long Axis 4 Chamber View showing DCM and pleural effusion (2D echocardiography); **(4)** Right Parasternal Short Axis View at the level of Chordae Tendinae showing DCM (M-mode); **(5)** Gross examination of heart showing severe rounding of contour; **(6)** Congested liver and distended gall bladder; **(7)** Heart showing degenerated myofibrils (H and E_ 40x); **(8)** Liver showing hepatocyte degeneration and pyknotic nuclei (H and E×100)

confirmed myocardial injury. So, based on case history, clinical signs, chest X-ray, echocardiography and cTnI assay, the case was confirmed as DCM. Moreover, as per the American/European College of Veterinary Internal Medicine consensus classification system, the case belonged to stage B2 indicating radiographic or echocardiographic evidence of cardiac remodeling (Atkins *et al.,* 2009).

Haematology was found to be within reference range (Table 1). Majority of the plasma biochemical parameters (Table 1) were found to be within reference range except mild hypocalcemia, hypocholesterolemia, hyponatremia and hypochloremia. Serum oxidant-antioxidant status (Table 1) revealed marked decrease in glutathione (GSH) level compared to healthy control.

The dog was administered with standard therapy comprising of tablets Pimobendan @ 0.3 mg/kg twice a day (BID) (before food), Enalapril @ 0.5mg/kg BID, Furosemide-Spironolactone @ 2 mg/kg BID and Cardio-protectants @ 1 tab PO OD for two months. The patient initially responded to therapy (e.g., improved exercize tolerance, resolution of dyspnoea and decrease in abdominal distension) which later on became refractory. The animal was then euthanized humanely upon the owner's request and necropsy was performed.

The major clinical signs of the present case were in agreement with Ware (2011). ECG finding of LBBB was related to underlying disease of the LV (Ware, 2011) which got substantiated by the echocardiographic parameter of sphericity index (SI) of 1.32. The major echocardiographic findings used for diagnosis of DCM were LV dilation, increased EPSS, normal or thin LV wall and interventricular septum and poor FS (Ware, 2011) which were in agreement in this case. Hemato-biochemical parameters in the present case were found to be non specific rather may be used for ruling out concurrent disease (Deepti and Yathiraj, 2015). Hypocholesterolemia could be due to malabsorption, maldigestion and protein losing enteropathy that occurs in dogs with cardiac insufficiency (Smith *et al.*, 2016). Hypocalcemia was in agreement with (Hobai *et al.*, 2001) earlier report hampering contractile function of cardiomyocytes. Hyponatremia and hypochloremia of the present study were in agreement with Boswood and Murphy (2006) and are common complications of congestive heart failure (CHF) due to renal dysfunction, elevation of neurohormonal substances, activation of the RAAS system and diuretic therapy (Smith *et al.,* 2016). Free radicals are highly reactive molecules produced during normal metabolism in body or after exposureto environmental stressors, which are kept in equilibrium by

Table 1

Hemato-biochemical profile of dog with DCM on day 0

S. No.	Parameter	Day 0	Reference range*
1.	Hb (g/dl)	12.12	12-19
2.	Hct (%)	39.0	35-57
3.	TEC (x 10^6/µl)	5.54	5.0-7.9
4.	TLC (x 10^3/µl)	10.50	5.0-14.1
8.	THR (x 10^5/µl)	2.13	2.11-6.21
9.	N (%)	66.0	58-85
10.	L (%)	20.00	8-21
11.	M (%)	8.0	2-10
12.	E (%)	6.0	0-9
Plasma biochemistry			
13.	BUN (mg/dl)	18.87	8-28
14.	Creatinine (mg/dl)	1.13	0.5-1.7
15.	ALT (IU/L)	15.0	10-109
16.	ALP (IU/L)	116.0	1-114
17.	Triglyceride (mg/dl)	85.0	35.0-111.0
18.	Cholesterol (mg/dl)	125.0	135–278
19.	CPK-MB (IU/L)	163.0	78.0-173.0
20.	Ca^{++} (mg/dl)	5.78	9-12
21.	Na^{+} (mmol/L)	114.0	142–152
22.	K^{+} (mmol/L)	4.1	3.9–5.1
23.	Cl^{-}(mmol/L)	88.1	110–124
Serum oxidant-antioxidant status			
24.	LPO (nm)	0.4	0.29-0.48
25.	SOD (U/ml)	0.51	0.44-1.23
26.	GSH (µm)	0.68	1.58-5.1
27.	TA (mM)	1.72	1.23-2.85

*Reference range adapted from The Merck Veterinary Manual, 11th Edition edited by Susan *E. Aiello*, S.E. and Moses, M.A. (2016).

body through endogenous antioxidants. If this balance is disturbed, it results inoxidative stress (Behera *et al.,* 2012). In this case, there was marked decrease in GSH level whereas lipid peroxides (LPO), superoxide dismutase (SOD) and total antioxidants (TA) levels were found to be within normal range. Decrease in GSH level was in agreement with previous report (Viviano *et al.,* 2009) and suggestive of role of oxidant-antioxidant system in the disease pathogenesis of DCM. The unfavorable prognosis of the present case was in agreement with existing report with average survivability not more than three months (Smith *et al.,* 2016). Gross necropsy lesions revealed marked peripheral edema and ascites, cardiomegaly (Fig. 5) along with thin LV wall and a dilated chamber that was consistent with DCM (Saini, 2014), discoloration of kidney cortex, irregular splenic contour, hepatomegaly with a distended gall bladder (Fig. 6) (Ware, 2011). Histopathology revealed degenerative changes in the cardiac musculature (Fig. 7) and liver parenchyma (Fig. 8), renal tubular degeneration and exfoliation which were in agreement with Saini (2014). Degenerated myofibrils of DCM have a high sensitivity and specificity for the disease. Hepatic and renal changes were the after effects of chronic venous congestion in the respective organs due to CHF (Saini, 2014).

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