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A case report on dilated cardiomyopathy-induced pleural effusion in a great Dane

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Abstract
A four-and-a-half-year-old male Great Dane dog was presented at Small Animal Medicine Unit of Veterinary Clinical Complex, Tirunelveli with a history of abdominal distension, severe cough, activity intolerance and panting for past 1 week. A right lateral radiograph was taken which revealed complete border effacement of the cardiac silhouette suggesting pleural effusion. Point-of-care thoracic ultrasound (POCTUS) was performed according to Vet BLUE protocol and it revealed anechoic content at the cranial, middle and Perihilar region. Echocardiographic findings revealed Left Ventricular diameter after diastole - 7.48 cm, Left Ventricular diameter after systole 6.51 cm, Ejection fraction – 27% and fractional shortening – 13%. Needle thoracocentesis was performed as an emergency protocol to alleviate respiratory distress. Patient showed improvement after performing thoracocentesis.

Keywords: Dilated cardiomyopathy, pleural effusion, Point-of-care thoracic ultrasound, vet BLUE, needle thoracocentesis

Introduction
Dilated cardiomyopathy (DCM) is primary disease of the muscles of the heart, characterized by a progressive fall in the contractility of the ventricles, dilation of all the cardiac chambers, leading to congestive heart failure (CHF), arrhythmias, and death [1]. It is the most frequent reason for cardiac disability in dogs that occurs after myxomatous valvular heart disease and it is considered to be a major etiology for heart failure in many breeds of dogs, especially in larger breeds [2]. DCM is characterised by loss of heart muscle function leading to the enlargement of heart and also enlarged ventricles may lead to decreased cardiac output to lungs leading to fluid accumulation in lungs [3]. CHF is generally classified as 'left-sided' (L-CHF), referring to increased pulmonary venous pressures and resulting pulmonary oedema, or 'right-sided' (R-CHF), referring to increased systemic venous pressures and resulting cavitary effusion (ascites, pleural effusion, and/or pericardial effusion) [4].

Case history and observation
A four-and-a-half-year-old male Great Dane dog was presented at Small Animal Medicine Unit of Veterinary Clinical Complex, Tirunelveli with a history of abdominal distension, severe cough, activity intolerance and panting for past 1 week. Animal was in sternal recumbent posture when presented. Clinical examination revealed cardiac cachexia (Fig 1) and dyspnoea. Muffling of heart tones was seen on thoracic auscultation. Animal showed respiratory distress with rapid shallow breathing. A right lateral radiograph was taken which revealed complete border effacement of the cardiac silhouette suggesting pleural effusion. (Fig 2). Point-of-care thoracic ultrasound (POCTUS) was performed according to Vet BLUE protocol and it revealed anechoic content at the cranial (Fig 3), middle (Fig 4) and perihilar region (Fig 5). Ultrasonographic scoring of pleural effusion was done to compare linear measurements from the pleural surface in the midline of the sternebra to the furthest ventrolateral point of both right and left lung edges before (Fig 6) and after thoracocentesis (Fig 7). Hemato-biochemical findings revealed haemoconcentration (Haemoglobin - 13.6 g/dl, Packed Cell Volume – 41%), low serum total protein (TP) (1.90 g/dl), low serum albumin (0.90 g/dl) and elevated serum lactate dehydrogenase (LDH) (261 U/L) levels. Echocardiographic findings revealed Left Ventricular diameter after diastole - 7.48 cm, Left Ventricular diameter after systole 6.51 cm, Ejection fraction – 27% and fractional shortening – 13% (Fig 8). Electrocardiography revealed low voltage QRS complex (Fig 9). Pleural fluid

analysis via diagnostic thoracocentesis was done prior to therapeutic thoracocentesis. Pleural fluid biochemistry revealed TP of 4.9 g/dl and cytological analysis revealed red blood cells, few lymphocytes, reactive mesothelial cells (Fig 10) and total nucleated cell count (TNCC) of 2400 cells/ µl (using Hematology auto-analyzer). Hence, it was diagnosed as dilated cardiomyopathy-induced pleural effusion with modified transudate.

Treatment and Discussion

Needle thoracocentesis was performed as an emergency protocol to alleviate respiratory distress. A 20-gauge 1-inch butterfly needle was used and the site was prepared aseptically for the procedure. 2% lignocaine was locally injected around the prepared site. Needle was inserted at the level of 8th intercostal space close to costo-condral junction and 4 litres of serosanguinous fluid was removed (Fig 11). Animal was treated with crystalloids (Inj. Ringer's lactate @ 10 ml/kg body weight) and diuretics (Inj. Furosemide @ 2 mg/kg body weight intravenously). Owner of the dog was advised to medicate the animal orally *b.i.d.* with Pimobendan (Safeheart®) @ 0.25 mg/kg body weight, Enalapril (Envas®) @ 0.3 mg/kg body weight and L-carnitine (Strongbeat®) @ 100 mg/kg and bring the animal to review every week. The patient revealed no significant clinical improvement after weekly review.

Dilated cardiomyopathy (DCM) has been considered to be primarily an inherited disease with higher prevalence in specific dog breeds. Great Danes typically present with the more classic dilative form of cardiomyopathy [5]. The clinical signs of the dogs with dilated cardiomyopathy produced pleural effusion, such as severe cough, activity intolerance, dyspnoea, and panting which was in accordance with our case study [6]. If bicavitary effusion is present, abdominal distention or an abdominal fluid wave may occur which was seen in clinical examination of the animal [7]. Tachypnoea, tachycardia, and muffled cardiothoracic sounds are the most prevalent physical examination abnormalities some of which was seen in our case study [7]. Most common symptoms of pleural effusion are dyspnoea and exercise intolerance (inactivity) [8] which was present in our case study.

Echocardiography is necessary to definitively differentiate DCM from other cardiovascular diseases [9]. Pleural effusion, identified by thoracic radiography, was the most significant prognostic indicator of survival which was most likely to occur when systemic interstitial pressures are e1evated and in dogs with biventricular CHF [10]. DCM is more common in middle age to old dog with male over dominance in ratio of 2:1 and recognized in several medium-size and giant breed dogs [11] which was seen in our case study. Right sided congestive heart failure leads to fluid accumulation in the abdominal, pleural and/or pericardial cavities [12], which can sometimes be controlled with medication [4].

Measurement of echocardiographic indices by M-mode echocardiogram is a useful technique in recognizing left ventricular dilation and poor contractile function in dogs with DCM [12]. Evaluation of left ventricular diastolic function provides valuable information to determine severity and prognosis of cardiac disease [13] which was evaluated in our case study.

Biochemical analysis is a vital tool in diagnosing the cause of pleural effusion [14]. Pleural fluids are classified as modified transudate when there is a mild increase in TP which is greater than 2.5 g/dL [15] which was reported in our case as pleural fluid TP was 4.9 g/dL.

Tiny amount of tissue damage can cause a substantial increase in LDH levels in the serum, and its extracellular appearance can thus be utilized to identify cell damage or death [16] which was also reported in our case as LDH level in serum was 261 U/L indicating severe tissue damage.

TP and TNCC are used to define effusions as exudate, modified transudate, or pure transudate and can assist to establish an underlying etiology [17].

Pleural effusion can defined as transudate with TP of 3.0g/dl and TNCC >7,000/µl, modified transudate with TP ranging from 2.5 to 7.5 g/dl and TNCC ranging from 1,000–7,000/µl, exudate with TP >3.0g/dl, TNCC >7,000/µl [18]. Our case study showed that pleural fluid TP was 4.9 mg/dL and TNCC was 2400/µL and hence was classified as modified transudate.

Radiographic signs of pleural effusion which are: a) Retraction of the lung lobes away from the thoracic wall by soft tissue opacity b) Presence of single or multiple pleural fissure lines (widest laterally and extend medially as they thin toward the pulmonary hilum) c) Partial or complete border effacement of the cardiac silhouette and diaphragm (different degrees, depending on the severity of the effusion) [19] which is shown in Fig 2. Complete border effacement of the cardiac silhouette will be seen on lateral images in patients with severe effusion which is seen in Fig 2.

POCTUS has been increasingly used in the assessment of critically ill animals and it has been suggested to be beneficial in the assessment of all unstable animals [20] and the POCTUS was performed in our case study [21] as shown in Fig 3, 4, 5 and 6.

Thoracocentesis is an invaluable diagnostic, and often therapeutic, tool and the indications include (1) the presence of any undiagnosed pleural effusion and (2) therapeutic thoracocentesis to relieve respiratory signs caused by large amounts of air or fluid [22] shown in Fig 11.

Thoracocentesis is the prime treatment given to animal since this relieves respiratory distress and also provides time for cyotological examination [23] which in our case showed confirmatory diagnosis of the type of effusion by qualitatively analysing cells shown in Fig 10.

Angiotensin-converting enzyme (ACE) inhibitor such as Enalapril helps in lowering the blood pressure by inhibiting the conversion of peptide hormone angiotensin-I to angiotensin-II, which is one of the prime vasoconstrictor of blood vessels resulting in increase in blood pressure [24] which was advised for our patient twice daily life-long.

Treatment of CHF, for dogs diagnosed with DCM, is similar to other CHF etiologies: decreasing preload and afterload while maintaining systemic blood pressure. In most cases, triple therapy is warranted for treating CHF. Triple therapy includes prescribing pimobendan (0.25 mg/kg by mouth, twice daily), a such as furosemide (typical starting dose is 2 mg/kg by mouth, twice daily), and an angiotensin-converting enzyme (ACE) inhibitor such as enalapril (0.25 to 0.5 mg/kg by mouth, twice daily [25].

L-carnitine supplementation may help improve clinical signs and echocardiographic parameters. The commonly recommended dose of 50 to 100 mg/kg, every 8 hours, may be appropriate for the management of systemic deficiency [26] which was followed in our case study.

In most of the cases the prognosis is poor and the dogs are clinically managed in prolonging the lifetime of the animal [27] which was followed in our case by advising oral medications.

Fig 1: Cardiac cachexia

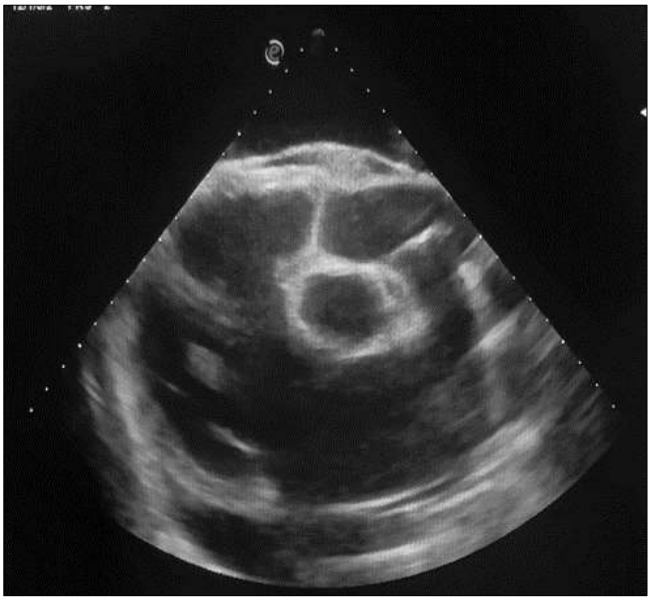

Fig 4: Middle site

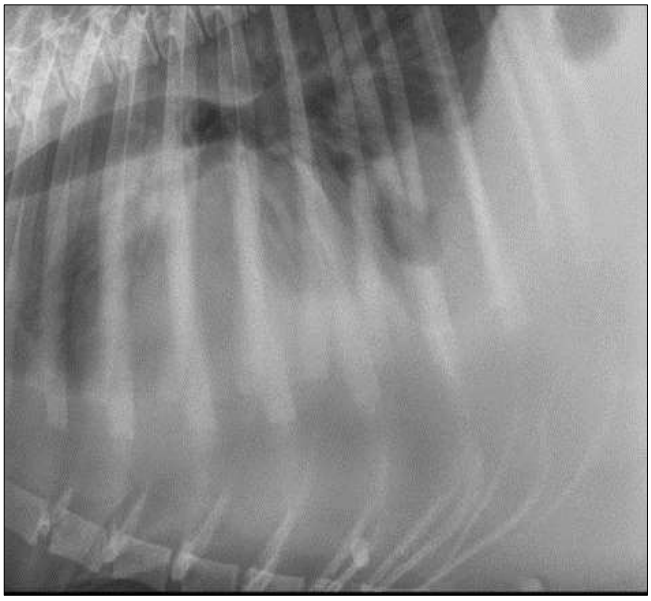

Fig 2: Border effacement of cardiac sillhoutte

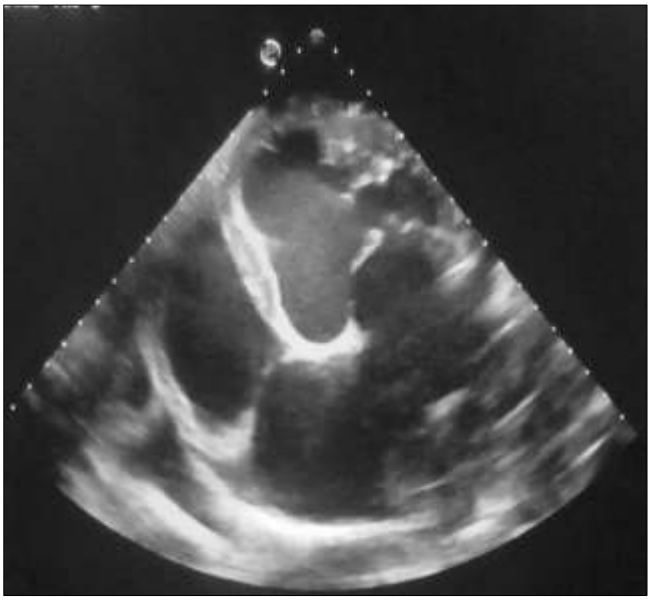

Fig 5: Perihilar site

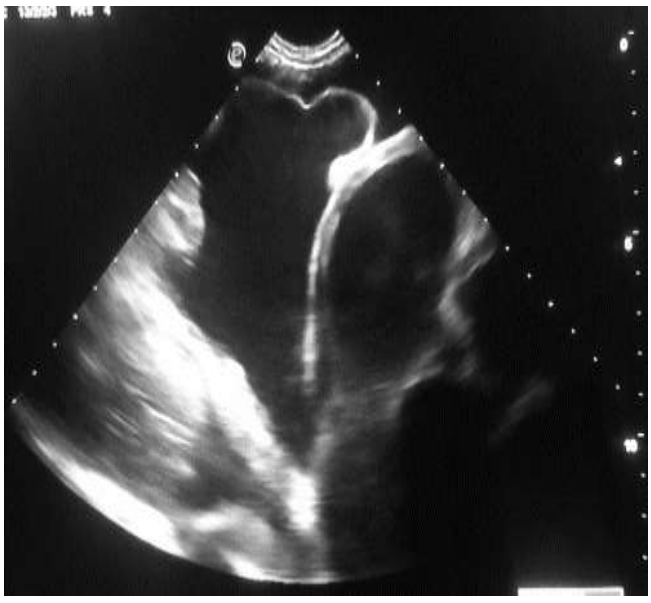

Fig 3: Cranial site

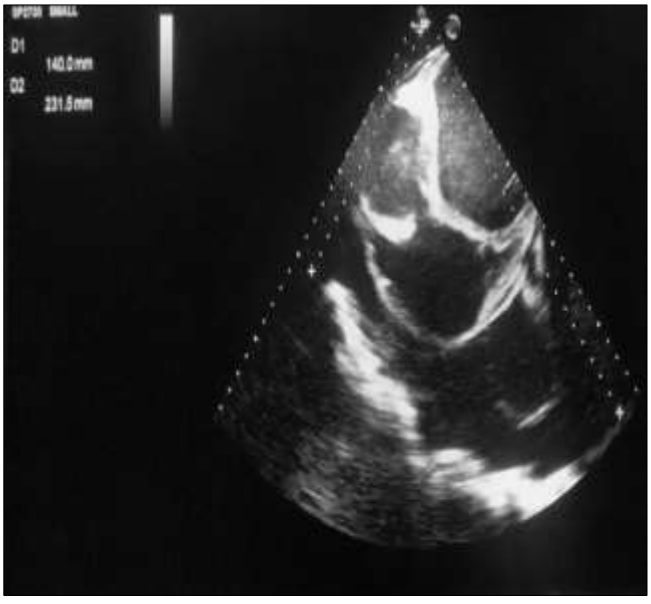

Fig 6: linear measurements before thoracocentesis

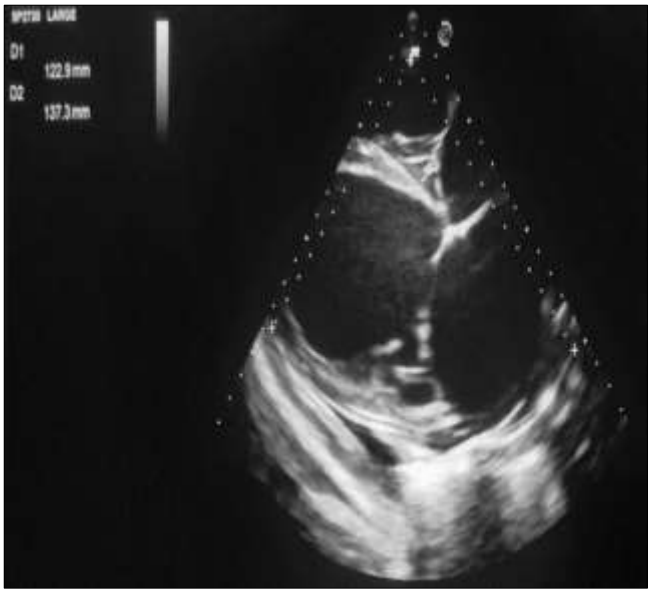

Fig 7: linear measurements after thoracocentesis

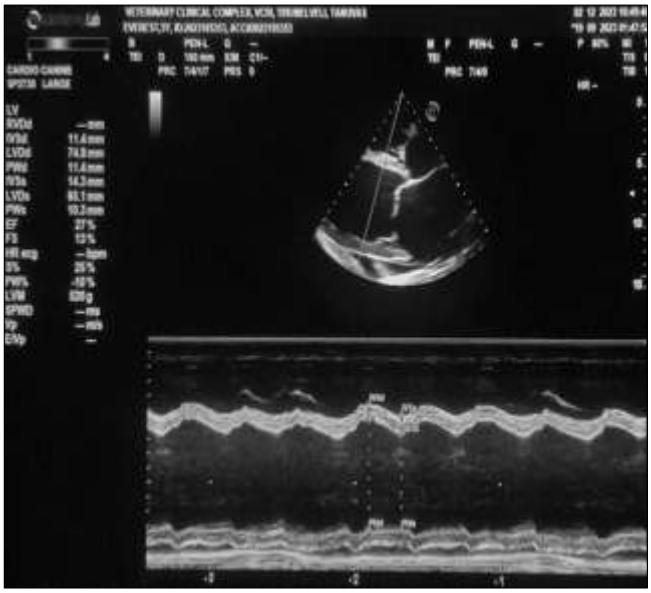

Fig 8: Echocardiography mesearement

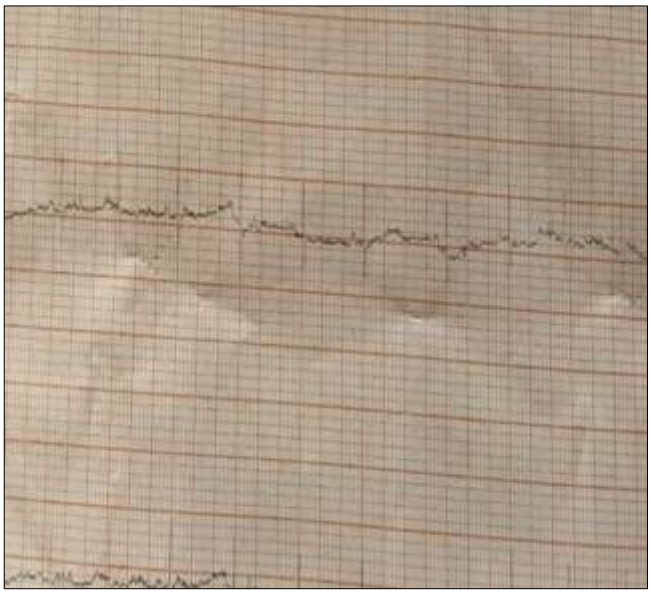

Fig 9: Electrocardiography revealing low QRS amplitude due to low voltage in effusion

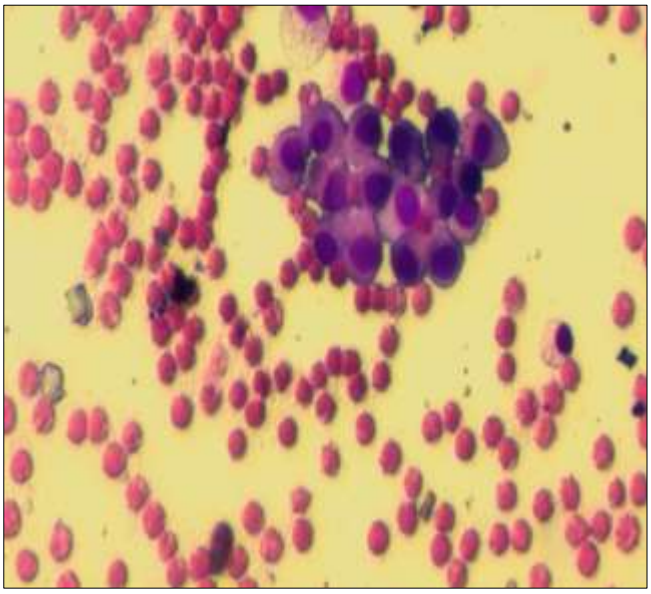

Fig 10: Cytology revealing RBCs, reactive mesothelial cells and lymphocyte

Fig 11: Needle Thoracocentesis

Conclusion

In conclusion, Dilated cardiomyopathy is a very serious disease that must be accurately diagnosed and aggressively treated. DCM induced pleural effusion is easily diagnosed by thoracic radiography and point-of-care Vet BLUE ultrasound technique. Clinical management involves cardiac medication in early stages. Dogs that have developed clinical signs of heart failure such as pleural effusion have a worse prognosis than those that are put on cardiac medication in the early stages of the disease. Long term prognosis for DCM varies considerably with most dogs with signs of congestive heart failure dies within 6 months. Repeated thoracocentesis is the only possible treatment option available to alleviate clinical signs of respiratory system.

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