

A RARE CASE OF TYPE-III BOXER CARDIOMYOPATHY

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ABSTRACT

A three year old female boxer dog was presented with the history of anorexia, coughing, exercise intolerance, severe dyspnoea and retching for the past 10 days. The physical examination revealed pale mucous membranes, normal rectal temperature, respiratory distress and tachycardia. Radiographic examination of chest revealed tracheal elevation and increased opacity in caudal lung lobes. Electrocardiography revealed absence of P waves indicating supraventricular tachycardia. Echocardiographically, enlargement of left side chambers with low ejection fraction (EF%, 10.84%) and fractional shortening (FS%, 4.91%) indicated reduced contraction and systolic failure. Doppler echocardiography revealed severe mitral and tricuspid regurgitation. The present case was diagnosed with type III category of boxer cardiomyopathy which was characterized by myocardial systolic dysfunctions with evidence of congestive heart failure.

Key words: boxer, dog, cardiomyopathy, electrocardiography, echocardiography

Boxer cardiomyopathy is a slow progressive degenerative myocardial process. Its clinical characteristics are unique to Boxer breed as compared with primary myocardial disorders in other large and giant breeds of dogs. There are three clinical categories of the disease i.e. concealed, overt and a myocardial dysfunction form. The concealed form (Type I) is characterized by the absence of clinical signs but presence of ventricular premature complexes (VPCs). In the overt form (Type II), syncope or episodic weakness and tachyarrhythmia's are present; while the myocardial form (Type III) is characterized by myocardial systolic dysfunction (affecting mainly the left ventricle) sometimes with congestive heart failure (CHF) and arrhythmias (Harpster, 1983).

A three year old, female Boxer dog weighing 24 kg was presented to the Teaching Veterinary Hospital of this university with a history of anorexia, coughing, exercise intolerance, lethargy, retching and respiratory distress. Vaccination and deworming status was complete. There was no history of syncope or seizures. The physical, electrocardiographic, radiographic and echocardiographic examination was undertaken. Blood testing for complete blood count (CBC) and biochemical profile was carried out. M-mode, B-mode and Doppler echocardiographic examinations were performed by using a Logiq P5 colour Doppler ultrasound machine equipped with a 5-S transducer. All the M mode measurements were taken by right parasternal short axis view at the level of chordae-tendinae (American Society of Echocardiography). Radiography was performed with

lateral and ventrodorsal (VD) projections of chest. Electrocardiography was performed by using Cardiart 8108 BPL six channel electrocardiographic machine with dog in right lateral recumbency.

On clinical examination the dog was dull with pale mucous membranes with normal rectal temperature (102°F) and tachycardia (heart rate, 180 beats/min). Lateral chest radiograph revealed dorsal elevation of trachea and carina suggestive of right and left atrial enlargement with marked increased opacities in caudal-dorsal lung lobes (Fig. 1). Ventral-dorsal chest radiograph showed enlargement of the heart especially left ventricles and atrium (Fig. 2). Haematological examination revealed neutrophilic leucocytosis (Hb: 13.3 g/dl, hematocrit: 39.9%, TLC: $19.870 \times 10^3/\mu\text{L}$ and neutrophil count: $17.485 \times 10^3/\mu\text{L}$). Electrocardiography (paper speed 50mm/sec) revealed increased heart rate (156 to 300 bpm) with absence of P waves indicating supraventricular tachycardia (Fig. 3).

M mode echocardiography through right parasternal short axis view at the level of chordae-tendinae revealed dilation of left ventricle. Left ventricular internal dimension during diastole (LVIDd) was 6.33cm (reference range 2.90-4.80 cm) and left ventricular internal dimension during systole (LVIDs) was 6.02cm (reference range 1.67-3.30cm) (Boon, 2011; Fig. 4). The EF% (Ejection Fraction) and FS% (Fractional Shortening) were 10.84% (reference range 66.97-79.40%) and 4.91% (reference range 31.25-42.4 %) (Boon, 2011) respectively indicating reduced contractibility and systolic heart failure. M mode echocardiography through right parasternal short axis

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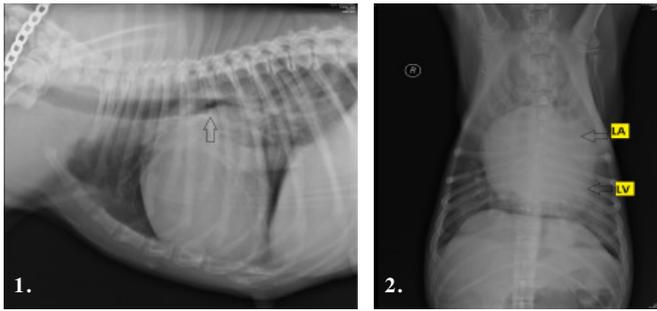


Fig 1. Lateral chest radiograph

Fig 2. Ventral-dorsal chest radiograph showing enlargement of left ventricle and atrium (arrows)

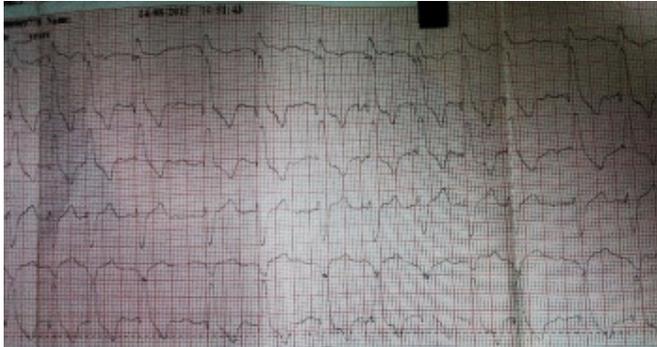


Fig 3. Electrocardiography showing absence of P waves. Paper speed is 50 mm/sec.

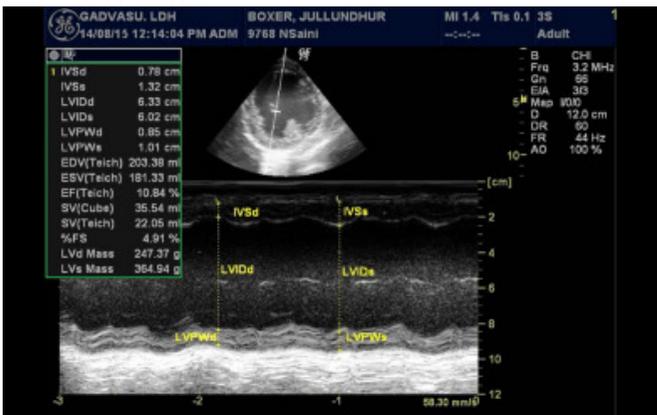


Fig 4. M mode echocardiography through right Para-sternal short axis view at the level of chordae-tendinae revealing dilation of left ventricle



Fig 5. Right Para-sternal short axis view at the level of aorta indicating severe left atrial enlargement

view at the level of aortic valve indicated increased left atrium (LA) dimensions i.e. 5.38 cm (reference range 1.96-3.26 cm) as compared to aortic (Ao) dimensions i.e. 1.23 cm (reference range 1.82-2.69 cm; Boon, 2011) (Fig. 5). As a result, LA/Ao ratio was very high i.e. 4.38 (reference range 1.18-1.92; Boon, 2011) indicating left atrial enlargement.

Pulse wave doppler of mitral valve revealed MV E velocity (Rapid ventricular filling), 1.50 m/s (reference range 0.53-0.96 m/s); MV A velocity (atrial contraction), 0.20 m/sec (reference range 0.34-0.75 m/s); E: A ratio, 7.54 (reference range 0.95-2.22); M V deceleration time, 36.99 m/sec (reference range 60-107 m/s) (Schober and Virginia, 2002) indicating restrictive filling pattern (Fig. 8). Colour doppler echocardiography at left apical 4-chamber view revealed severe mitral and tricuspid regurgitation. Mitral valve and tricuspid valve regurgitant velocity observed was 4.26 cm/sec and 2.76 cm/sec, respectively (Figs. 6, 7). Echocardiographic findings were suggestive of myocardial dysfunctional form of boxer cardiomyopathy with severe congestive heart failure.

The dog was treated with diuretic Furosemide @ 2mg/kg b.wt. orally 12 hourly, and vasodilator, Enalapril @ 0.5mg/kg b.wt. orally 12 hourly for 2 weeks. For myocardial strength, L-Carnitine was given @ 500 mg orally 12 hourly for 2 weeks and antibiotic- Ampicillin and Cloxacillin @ 25mg/kg b.wt. for 12 hourly orally. The animal died on the same day before treatment could be initiated. The owner did not agree for undertaking the post-mortem investigations.

In the present case, the electrocardiographic and echocardiographic parameters were consistent with idiopathic dilated cardiomyopathy characterised by reduced contractibility and ventricular dilation (Kittleson, 1998, Sisson *et al.*, 1999). Hence the present case was categorized as myocardial dysfunction form i.e. Type III boxer cardiomyopathy (Harpster, 1983). Supraventricular arrhythmias recorded in the present study were consistent with the findings of Harpster (1983). Baumwart *et al.* (2005) also reported low FS% (14.4%) and increased LVIDs (4.5cm) in boxer dogs with myocardial dysfunction. The restrictive filling pattern on Doppler echocardiography has been found to be the most common in patients with dilated cardiomyopathy (DCM) and is directly correlated with high filling pressures and poor prognosis in human beings and dogs (Borgarelli *et al.*, 2006). The E wave deceleration time is a powerful independent predictor of poor prognosis in human beings (Giannuzzi *et al.*, 1993). The restrictive trans-mitral flow pattern (E: A>2) and rapid early diastolic deceleration time (<80 m/sec) are

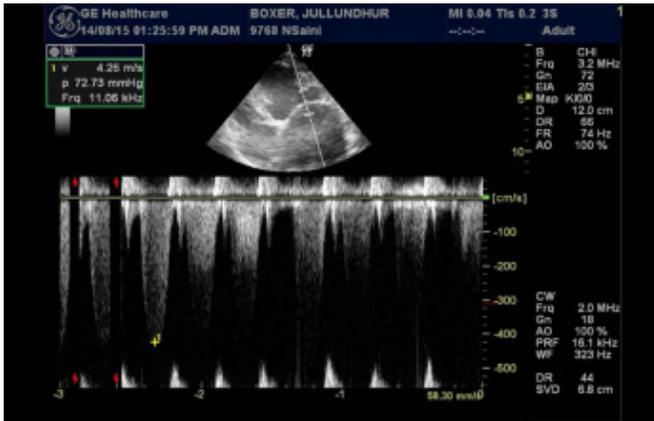


Fig 6. Left apical four-chamber view with continuous-wave doppler at mitral valve measuring the mitral flow regurgitant velocity of 4.26 m/sec.

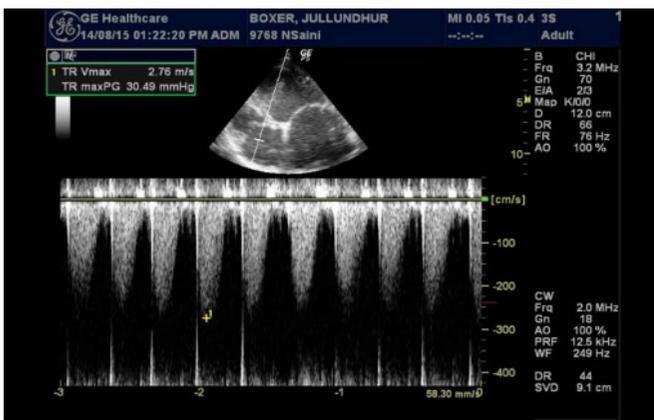


Fig 7. Left apical four-chamber view with continuous-wave doppler at tricuspid valve measuring the tricuspid flow regurgitant velocity of 2.76 m/sec.

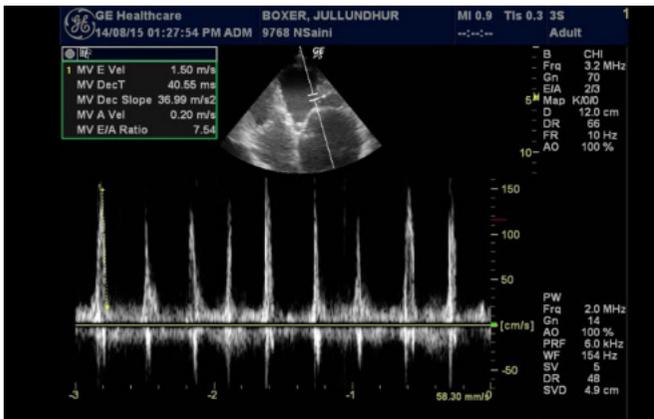


Fig 8. Pulse wave doppler of mitral valve indicating restrictive filling pattern

directly related with a median survival time of 114 days (Borgarelli *et al.*, 2006). Similar findings were observed in present case with low survival time. The echocardiographic diagnosis of DCM was based upon

the criteria of a dilated left ventricle with reduced contractility (Kittleson, 1998; Sisson *et al.*, 1999). Since systolic dysfunctions and left ventricular heart failure were present, so the treatment for canine DCM was provided (Meurs, 2004).

In present study, myocardial dysfunction form of boxer cardiomyopathy characterised by left ventricular dysfunction and congestive heart failure was observed to poor survival time and worse prognosis. Similar observations were also made by Palermo *et al.* (2011).

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