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DIFFERENTIATION OF TWO CANINE CASE STUDIES WITH CONGESTIVE HEART FAILURE: DILATED CARDIOMYOPATHY AND CHRONIC VALVULAR DISEASE

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Chronic valvular disease (CVD) and dilated cardiomyopathy (DCM) are common cardiac diseases in dogs that are important causes of congestive heart failure (CHF) and eventual death. Clinical signs, thoracic radiography (TR) and two-dimensional echocardiography (2DE) can be similar in these two conditions. M-mode echocardiography (MME) of the left ventricle (LV), fractional shortening (FS) and E' point to the septal separation (EPSS) discriminate DCM from CVD but normal FS and EPSS can mask systolic abnormalities. LV diameter in systole (LVDs) and end systolic volume index (ESVI), together with FS and EPSS, however, can give superior differentiation between these two conditions. We compared echocardiography data of two dogs with CHF presented to the Veterinary Teaching Hospital, University of Peradeniya. Both dogs had CVD, but the aim of the study was to find out the exact cause of CHF. Case study 1 (C1) was an 8-year old, cross-bred, male dog, weighing 28 kg and case study 2 (C2) was a 9-year old, cross-bred, male dog weighing 12 kg. They were presented with ascites, dyspnoea, wheezing, weak pulse, tachycardia, tachypnoea and lethargy. Both patients were subjected to clinical examination, TR, 2DE, MME and Doppler. Both dogs were categorised as at the class IV cardiac failure stage with TR. CVD was explained with the findings of 2DE, which showed bilateral chamber enlargement, elevated LA/Ao quantifying LV dilatation, and thickened MV in both dogs. Increased LVDd, reduced IVSd and IVSs with reduced LVWd and LVWs, in C1 and normal LVWd and LVWs in C2, identified eccentric hypertrophy of the LV in both cases. FS was severely reduced in C1, indicating reduced myocardial contractility. Normal FS led to the surmise that C2 had normal myocardial contractility. Elevated EPSS in C1 indicated reduced systolic function. Further evidence for preserved myocardial function in C2 was the normal EPSS. Thus, we concluded that C1 had DCM while C2 did not. However, increased LVDs and ESVI indicated impaired systolic function in both C2 and C1. Normal FS in the presence of volume overload reflected impaired contractility in C2. As chronic volume overload in CVD can be causative for impaired myocardial function, we conclude that C2 had CVD with myocardial systolic dysfunction due to chronic volume overload. Despite the presence of CVD and volume overload in both patients, the MME parameters and ESVI differentiated between the causes of CHF in these dogs, with DCM being implicated in C1 and CVD with impaired myocardial contractility being the cause of CHF in C2.

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