ASCITES OF HEPATO-CARDIAC ORIGIN IN A LABRADOR RETRIEVER DOG

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The present study describes the diagnosis of an ascites of hepato-cardiac origin in a two year old Labrador Retriever bitch. The animal was suffering from chronic cough and recurrent ascites for more than six months. The physical examination revealed ascites which was confirmed through ultrasonography along with hepatic cyst. All the hemato-biochemical reports were found normal except TLC. Upon electrocardiography, it was revealed biphasic QRS complex (rS), widening of S wave and right axis deviation suggestive of right bundle branch block.

Key word: Ascites, Bundle branch block, ECG, Ultrasonography

A two years old, female, Labrador Retriever dog was presented to the clinic with the history of chronic cough (tachypnea, respiratory rate 45 breaths per minute),

exercise intolerance along with recurrent ascites since last six months. Upon physical examination it was for ascites. Radiograph showed hepatomegaly.

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The animal exhibited normal hemogram viz. haemoglobin 13.6 gm%, packed cell volume 41.8%, total erythrocyte count $6.16 \times 10^6/\text{mm}^3$, total leukocyte count $15.2 \times 10^3/\text{mm}^3$, neutrophil 63%, lymphocyte 31%, eosinophil 02%, and monocyte 04% and platelet counts $25 \times 10^5/\text{mm}^3$. All the erythrocyte indices were also within normal range.

Serum biochemistry showed blood glucose 80 mg/dL (random), total protein 7.32 gm/dL with albumin: globulin ratio of 0.73. Liver function test appeared normal with total bilirubin 0.19 mg/dL, bilirubin-direct 0.13 mg/dL, bilirubin-indirect 0.06 mg/dL, SGPT 50.2 IU/L, SGOT 49.3 IU/L and ALP 89.2 IU/L. The kidney function test also showed normal (creatinine 0.82 mg/dL, uric acid 0.93 mg/dL) with higher blood urea 37.0 mg/dL.

Blood electrolyte profile showed sodium 142.0 mEq/L, potassium 5.0 mEq/L and chloride 109.0 mEq/L.

In ultrasonography, anechoic free fluid in the abdomen with floating viscera was appreciated (Fig. 1). The kidneys appeared normal with normal size and shape (left kidney, 4.9cm x 3.2cm and right kidney, 5.6cm x 3.4cm) with clear cortico-medullary demarcation. Urinary bladder showed normal without any rupture, uroliths or neoplasia. The liver showed normal

echogenicity with hepatomegaly indicated as round edged hepatic lobes. A hepatic cyst (2.7cm x 2.4cm) was also appreciated (Fig. 2).

The ECG (Lead-II) showed bi phasic QRS complex (rS), widening of S wave (Fig. 3). The equilateral triangle measurement system (Spasojevic-Kosic, 2011) using QRS complex of Lead I and Lead III was employed to determine the MEA and it was found that there was right axis deviation suggestive of right bundle branch block.

As the animal was diagnosed with ascites of hepato-cardiac origin, the animal was treated with enalapril @ 0.5 mg/kg body weight, orally bid, furosemide @ 4 mg/kg body weight, orally, bid and liver tonic. However after 3 months of treatment the animal was still suffering and prognosis is grave.

The aetiology of ascites in dogs may be of hepatic (Pradhan *et al.*, 2008), renal (Peden and Zenoble, 1982) or cardiac origin (Kumar and Srikala, 2014; Mukherjee *et al.*, 2017). The higher TLC in ascites in this study was in accordance with earlier reports of (Pradhan *et al.*, 2008). In this case, the animal had normal kidney function profiles. So, the renal involvement in ascites can be ruled out. Cardiac origin ascites were associated with cardiomyopathy (Peden and Zenoble, 1982), right heart failure (Kumar and Srikala, 2014)

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Fig. 1. Sonograph showing anechoic free fluid with floating viscera

Fig. 2. Sonograph showing hepatic cyst

Fig. 3. ECG showed biphasic QRS complex (lead-II)

or myocardial ischaemia (Mukherjee *et al.*, 2017). The appearance of bi-phasic QRS complex (rS) with low amplitude R wave along with widening of S wave in ECG was suggestive of right bundle branch block (Leininger, 1984; Tilley and Burtnick, 1999). Leininger (1984) also reported right axis deviation as in our findings. The pathogenesis of ascites due to right heart failure was reviewed by Peden and Zenoble (1982). They stated that right heart failure causes portal hypertension and escape of plasma proteins into the interstitial space of

Disse from the sinusoidal capillaries. Leakage of plasma proteins increases the hydrostatic pressure and the fluids accumulate in the abdominal cavity.

Conflict of Interests: The authors declare there is no conflict of interests amongst them.

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