

# Haematology, Biochemical And Haemodynamic Observations In Clinical Cases Of Idiopathic Cardiomyopathy In Dogs

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During the years, cardiopulmonary disorders have become recognised as an important condition in dogs. Among the cardiopulmonary disorders idiopathic cardiomyopathy (ICM) is a significant risk factor for cardio vascular morbidity and mortality. Idiopathic cardiomyopathy (ICM) was recognised as a distinct disease in dog in 1970 (Ettinger and Suter, 1970). Since then only few reports have described the haematological, biochemical and certain haemodynamic observations in clinical cases of ICM in dogs and hence this study.

## Materials and Methods

By correlating the clinical signs, X-rays, ECG and Echocardiographic findings, sixty cases diagnosed as idiopathic cardiomyopathy, which attended the Madras Veterinary College Hospital formed the basis for the study. The blood samples for haematology were collected from these cases. Haematology including haemoglobin (Hb), red blood cell count (RBC), packed cell volume (PCV), total and differential white cell count were done as per the standard methods. The erythrocytic indices (MCV, MCH and MCHC) were calculated. Blood samples were also processed for the estimation of Aspartate Amino Transferase (AST), serum total creatine phosphokinase (CPK) and creatine Kinase-MB (CK-MB). Haemodynamic recordings included heart rate and blood pressure. Both systolic and diastolic blood pressure was recorded using a sphygmomanometer callibrated in mm of Mercury. Twenty apparently healthy animals were selected as control groups to compare the various parameters included in the study.

## Results

The haemogram values viz., Hb, PCV, RBC, MCV, MCH and MCHC recorded in dogs affected with ICM do not vary significantly from the control animals. The total leucocyte and neutrophil of control animals were  $9348 \pm 352$  and  $6779 \pm 299$ . There was a significant ( $P < 0.01$ ) elevation of total leucocytes ( $14157 \pm 500$ ) and neutrophils ( $11441 \pm 471$ ) in dogs affected with ICM. There was no

significant variations in lymphocytes, monocytes and eosinophils during the disease.

The serum aspartate amino transferase (AST), creatine phosphokinase (CPK) and creatine kinase-MB (CK-MB) of control animal were  $23.31 \pm 0.81$  IU/L,  $43.53 \pm 2.74$  IU/L and  $7.16 \pm 0.46$  IU/L respectively. These values were significantly ( $P < 0.01$ ) elevated to  $67.99 \pm 3.68$  IU/L,  $70.89 \pm 2.53$  IU/L and  $14.35 \pm 0.68$  IU/L in ICM.

The mean  $\pm$  SE values of heart rate in control and ICM group were  $114.50 \pm 3.94$  and  $168.50 \pm 2.78$  beats per minute (bpm). There was a significant ( $P < 0.01$ ) difference between control and sick animals. The mean systolic and diastolic blood pressure of control animals were  $158.50 \pm 2.59$  mm Hg and  $88.75 \pm 1.77$  mm Hg. There was significant ( $P < 0.01$ ) elevation of diastolic blood pressure ( $99 \pm 2.51$  mm Hg) in dogs with ICM.

## Discussion

O'Brien *et al.* (1993) found that most haematological values of ICM did not significantly differ from healthy animals. The present findings with regard to haemogram agreed with O'Brien *et al.* (1993).

Ettinger and Suter (1970) observed neutrophilic leucocytosis in most conditions associated with myocarditis. The present study also revealed neutrophilic leucocytosis in ICM. Stress due to impaired cardiac function was reported to be the cause. Radiographic findings in 58.34 percent of these cases were suggestive of cardiomegaly with bronchitis/pneumonia. Myocardial changes together with pulmonary changes might have caused neutrophilic leucocytosis.

There was an increase of AST upto  $67.99 \pm 3.68$  IU/L in ICM group as against  $23.31 \pm 0.81$  IU/L in the control group. O'Brien *et al.* (1993) also found high AST values in clinical cases of ICM. This probably might indicate cardiac muscle degeneration though confirmatory evidences were lacking. Earlier literature reported elevated AST values in

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myocardial degeneration (Ettinger and Suter, 1970). Increased AST activity in ICM might have also resulted from hepato cellular injury developed probably because of passive congestion and anoxia (O'Brien *et al.* 1993). Inadequate tissue perfusion due to cardiac performance might develop muscle injury and thus could be another cause for elevated AST. Coles (1980) reported that pathological conditions involving either skeletal or cardiac or hepatic injury or all allowed the escape of large quantities of AST in the blood. The present observations on AST values in ICM group were consistent with earlier literature.

Atkas *et al.* (1993) gave the reference value for CK activity in dogs as 46 IU/L. In the present study, the CPK of the apparently healthy animals were found to be  $43.53 \pm 2.74$  IU/L and were comparable to that of Atkas *et al.* (1993).

O'Brien *et al.* (1993) observed increased CPK ( $128 \pm 62$  IU/L) in chronic cardiomyopathy. In the present study, there was a significant increase in CPK value ( $76.89 \pm 2.53$  IU/L) in dogs with ICM compared to healthy animals and it concurred with the findings of O'Brien *et al.* (1993).

While going through the raw data of the present investigation pertaining to the CPK values in clinical cases of ICM, it was observed that 10 per cent of the animals had CPK values within the control range. The observation concurred with that of Sottiaux (1986), who reported that CPK activity was found unchanged in dilated cardiomyopathy.

CPK is a dimeric enzyme having three different isoenzymes : MM, BB and MB. Distribution of CK-MM and MB isoenzymes in the myocardium of dog as percentage of total CK activity was 97 and 3 respectively and BB isoenzyme was not present in the myocardium. In dogs CK - MM predominated in myocardium and skeletal muscles (Atkas *et al.*, 1993). In concurrence with the above reports, CK-MB though relatively low in the myocardium was found significantly elevated in the idiopathic cardiomyopathy group compared to healthy animals and it

contributed to the elevation of total CPK also.

The systolic blood pressure recorded in the idiopathic cardiomyopathy group remained on par with control group; whereas the diastolic blood pressure in idiopathic cardiomyopathy ( $P < 0.01$ ) increased compared to control group. The diastolic blood pressure recorded was elevated and nearer to the diagnostic criteria for hypertension prescribed by Dukes (1992). Bovee (1991) found that increased heart rate resulted in elevation of diastolic blood pressure. Dukes (1992) also opined that blood pressure was related intimately to heart rate and cardiac output. So sinus tachycardia recorded in clinical cases of idiopathic cardiomyopathy resulted in elevated diastolic blood pressure.

The present study clearly indicated that clinical biochemical analysis of serum might be useful in assessing the progression of idiopathic cardiomyopathy. The elevated serum enzymes (AST, CPK and CK-MB) along with persistent leucocytosis with neutrophilia, sinus tachycardia and elevated diastolic blood pressure give clues for the clinical diagnosis ICM, in sick animals with clinical signs suggestive of ICM.

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