Hemato-Biochemical Evaluation of Heart Failure in Canines

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Abstract

The present study was conducted in eighty two (82) dogs with cardiac insufficiency to evaluate hemato-biochemical profiles in heart failure. Thirty four dogs (28 male and 6 female) were found to be having heart failure on the basis of hematobiochemical studies. The results of the study indicated ischemia with acute infection and increased red cell distribution width (RDW), which is associated with increased morbidity and mortality in dogs with heart failure.

Keywords: Heart failure; hematology; RDW; serum biochemistry

Introduction

Chronic heart failure (CHF) is accompanied by high co-morbidity and mortality and is clinically manifested as dilated cardiomyopathy (DCM). This syndrome is common in large and giant breeds of dogs and causes enlarged myocardium, impaired systolic function. It is characterized by vitiated myocardial contractility (Bodh *et al.,* 2016) and diagnosed by clinical signs, physical examination, radiography, electrocardiography and echocardiography with color flow doppler.

The hemato-biochemical indices may not be helpful in mild cases but elevated liver enzymes and azotemia are useful in serious cases (Olsen *et al.,* 2010). The present study was carried out to evaluate the hematological and biochemical alterations in dogs with DCM as a tool for the prediction of prognosis and guidance of treatment of CHF. In this study we aimed to evaluate predictive value of RDW for severity of heart failure and mortality.

Materials and Methods

A total of 34 dogs exhibiting signs like cough, ascites, exercise intolerance, tachypnoea, syncope, dyspnoea suggestive of CHF and a total of twelve clinically healthy dogs were taken for the study. Blood samples were collected for hematology and serum biochemical analysis. The hematological studies were undertaken by auto-hematology analyser (Rayto RT-7600, China). The serum biochemical profiles were estimated by fully automated biochemistry autoanalyser (Biosystems-A15, Spain).

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Statistical Analysis

All data were analyzed statistically using student's t-test (Snedecor and Cochran, 1994).

Results and Discussion

Out of 82 cases that were diagnosed, thirty four dogs (28 male and 6 female) were confirmed as CHF supported by electrocardiography (atrial fibrillation), radiography (cardiomegaly, pulmonary edema), and echocardiography (decreased systolic function/ DCM, mitral regurgitation, valve thickening) findings. Hematological observations revealed a significant ($p \leq 0.05$) alterations suggestive of moderate anemia and leucocytosis with neutrophilia probably due to neurohormonal changes like cortisol production (Ristic, 2004). Increased RDW (red blood cell distribution width) represents the variability in sizes of circulating erythrocytes but mechanism of association between CHF and hematological parameters is not well understood.

Chronic inflammation, iron deficiencies worsen the CHF condition (Inuzuka and Abe, 2015). Further, anisocytosis, anaemia and osmolality changes markedly decrease the ability of RBC to deform, thus reduces micro vascular perfusion and tissue hypoxia aggravating heart failure (Reinhart *et al.,* 2015). This might be the reason for elevated RDW in case of CHF. In the current study, observed reports positively correlated with neutrophils and negatively correlated with RDW and also with leukocytosis which concurs with earlier studies of CHF with leukocytosis and elevated inflammation markers (Kaneko *et al.,* 2008).

Table - Hemato-biochemical values of control and dogs with heart failure (Mean± SE)

Parameter	Healthy dogs (n= 12)		Dogs with heart failure (n=34)	
	Male (n= 6)	Female (n= 6)	Male (n= 28)	Female (n= 6)
PCV (%)	42.56^{b}±0.78	40.12^{b}±0.52	34.79^{a}±1.46	32.79^{a}±1.86
Hemoglobin (g/dL)	12.44^{b}±1.19	10.89^{b}±1.94	9.68^{a}±1.05	8.68^{a}±1.05
TEC (×10^{6}/µl)	6.11^{b}±1.68	5.35^{b}±1.8	4.9^{a}±0.93	4.29^{a}±0.93
RDW (%)	12.2^{a}±0.78	11.81^{a}±0.78	14.5^{b}±0.78	13.8^{b}±0.78
PLT (×10^{3}/µl)	203^{b}±1.24	195^{b}±1.24	173 ^{a}±1.24	163^{a}±1.24
TLC (×10^{3}/µl)	9.48^{a}±1.09	8.17^{a}±0.94	17.82^{b}±2.44	14.82^{b}±2.08
Neutrophils (N) (%)	74.10^{a}±2.34	70.10^{a}±1.94	78.58^{b}±2.13	76.98^{b}±1.88
Lymphocytes (L) (%)	19.87^{b}±2.06	16.87^{b}±1.8	14.72^{a}±1.86	12.74^{a}±0.53
N/L ratio	3.21^{a}±1.24	3.81^{a}±1.24	5.57^{b}±1.24	6.33^{b}±1.24
BUN (mg/dL)	28.65^{a}±1.93	25.78^{a}±2.93	97.64^{b}±3.58	95.64^{b}±3.58
Creatinine (mg/dL)	0.92^{a}±0.59	0.86^{a}±0.44	5.13^{b}±0.21	5.03^{b}±0.21
Total Protein (g/dL)	6.17^{b}±0.77	5.72^{b}±0.85	4.48^{a}±0.25	4.28^{a}±0.25
Albumin (g/dL)	3.23^{b}±0.26	2.83^{b}±0.66	2.21^{a}±0.54	2.05^{a}±0.89
AST (U/L)	32.96^{a}±0.42	29.42 ^{a}±0.23	81.46^{b}±0.58	78.24^{b}±0.18
ALT (U/L)	68.12^{a}±0.41	64.37^{a}±0.71	96.88^{b}±1.24	85.98^{b}±1.24
ALP (U/L)	42.37^{a}±0.71	36.12^{a}±0.41	76.88^{b}±1.24	66.88^{b}±1.24
LDH (U/L)	146.88^{a}±1.24	132.88^{a}±1.24	206.88^{b}±1.24	196.88^{b}±1.24
CKMB (U/L)	59.42^{a}±0.23	52.96^{a}±0.42	71.46^{b}±0.58	62.24^{b}±0.18

PCV: Packed Cell Volume; TEC: Total Erythrocyte Count; RDW: Red Cell Distribution Width; PLT: Platelet; TLC: Total Leucocyte Count; BUN: Blood Urea Nitrogen; AST: Aspartate Amino Transferase; ALT: Alanine Amino Transferase; ALP: Alkaline Phosphatase; LDH: Lactate Dehydrogenase; CKMB: Creatine Kinase MB.
Values bearing different superscripts in a row differ significantly ($p \leq 0.05$)

Kojima *et al.* (2015) found that RDW is positively correlated with central venous pressure and negatively with venous oxygen saturation. Thus, RDW (an index of anisocytosis) may be regarded as an inflammatory indicator which reflects MCV and the results suggested that RDW has a strong negative correlation with hemoglobin in severe CHF.

The association of RDW and CHF involves several factors but decreased RDW during or after treatment may be used in clinical practice as an indicator of reduced risk of CHF. RDW is inexpensive and widely available as a tool for prediction of prognosis and guidance for the treatment of CHF (Liu *et al.,* 2016) regardless of disease progression.

The serum biochemical values of dogs revealed a significant ($p \leq 0.05$) increase in activity of liver enzymes (AST, ALT, ALP) with CKMB, LDH indicated impaired liver function due to congestion in the small circulation associated with changes in heart muscle. Increased concentration of blood urea nitrogen and creatinine explained the background congestion, impaired the kidneys too (cardio renal syndrome in heart failure). This is also confirmed by hypoproteinemia and hypoalbuminemia (Jeyaraja *et al.,* 2015). CHF is regarded as a systemic disease with the involvement of other organ systems based on the chronic inflammation status (Bomassi *et al.,* 2017).

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