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SHORT COMMUNICATION

Evaluation of Myocardial Damage in Dogs with Heat Stroke

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ABSTRACT

Twenty cases of heat stroke in dogs, observed during hot/hot humid months, were investigated for myocardial damage, if any, employing electrocardiography and estimation of cardiac troponin-I. Heat stroke in dogs was clinically characterized by sudden onset of hyperthermia (temperature > 107.0 °F) during hot/hot humid months, impaired consciousness, severe panting and associated symptoms. Their haemogram reflected thrombocytopenia and increased levels of haemoglobin, packed cell volume, total erythrocytes and total leucocytes. Electrocardiographic abnormalities such as sinus tachycardia (heart rate 240 to 320 beats per minutes), ventricular premature complexes (8-10 per minute), ventricular tachycardia (ventricular rate 180-220), ventricular fibrillations, and 3rd degree heart block associated with increased level of cardiac troponin- I (mean 3.34±1.11 ng/ml) suggested myocardial damage in the dogs with heat stroke.

Keywords: Cardiac troponin-I, dogs, electrocardiography, heat stroke

Heat stroke in dogs, a medical emergency, is quite common in tropical countries. It is characterized by sudden hyperthermia, impaired consciousness, altered acid base balance and multiple organ damage. In the initial phase, cardiac output increases and with progression of the disease there is decrease in circulating blood volume resulting into decrease in cardiac output and fall in blood pressure. Thermal injury to the endothelium causes widespread vascular damage, disseminated intravascular coagulopathy and thrombocytopenia. Cellular damage in organs is the result of destruction of cell membrane lipids and chemical bonds, denaturation and inactivation of enzymes, tissue hypoxia, acidosis, hyperkalaemia (Spurr, 1970) and hypophosphataemia. Cardiac damage in such circumstances is well speculated (Kew and Tucker, 1969) that may have influence on the survival rate of dogs with heat stroke. In humans heat stroke has been shown to be associated with an increased risk of sudden cardiac death (Empana *et al*., 2009). Increased level of cardiac troponin-I (Hausfater *et al*., 2010) and electrocardiographic abnormalities (Mimish, 2012) has confirmed cardiac damage in humans with heat stroke. Cardiac arrhythmias and markedly elevated level of cTn-I has also been reported in a dog with heat stroke (Mellor *et al*., 2006). Such studies involving considerable number of cases are lacking in India wherein heat stroke is rather very common in dogs and humans. Therefore, the present investigation was carried out to study myocardial damage, if any, in cases of heat stroke in dogs.

Twenty clinical cases of heat stroke, observed during a period of six years (2012-2016) in hot/hot humid months at the Nandini Veterinary Hospital Surat, formed the material for the present investigation. Dogs having sudden rise in temperature (rectal temperature > 107.0 °F) and panting during dry/hot humid months without any coexisting disease were included in the study. The dogs were subjected to detailed clinical examination, blood smear cytology, haemogram (Celltac MEK64- Nihon Kohden), electrocardiography (Magic R - Maestros) and quantitative estimation of Troponin-I (chemiluminescent enzyme immunoassay method (CLEIA) PATHFAST, Mitsubishi Kagaku Iatron Inc, Tokyo, Japan) on the day of referral. Routine statistical analysis was carried out as per standard procedures.

During the period of last six years, 20 cases of heat stroke in dogs of different breeds (Labrador-7, German Shepherd-10, Great Dane-1, Golden Retriver-1, Pug-1), were investigated for myocardial damage, if any. The onset of clinical illness was sudden with rapid development of different clinical signs. The dogs were refereed at the hospital within 2-6 hours of illness. The disease was clinically characterized by high rectal temperature (107.0 °F to 109.8 °F), severe panting, open mouth breathing, tachycardia, injected mucus membranes, weak rapid pulse, dry extremities, bloody diarrhea (3 cases), semiconciousness to unconsciousness, nervous signs (7 cases), crying (1 case), petechiae (2 cases), melena (3 cases), oligouria (8 cases), turbid and brown urine (1 case) agreeing with the symptoms described for heat stroke in dogs (Bruchim *et al*., 2006). Sudden increase in core body temperature (crossing 105.8 °F) with nervous signs in dogs (Flournoy *et al*., 2003) during hot/hot humid environment (Drobatz and Macintire, 1996) has been classed as heat stroke. The disease is quite common in tropical countries like India. Prominent haematological changes were characterized by cytological negative status for blood protozoan and ehrlichial infections, thrombocytopenia (mean 1.04±0.036, Range 0.84 to 1.5, Median 1.0 lac), increased haemoglobin (mean. 17.77±1.92, Range 13.4-21.6, Median 17.9 g/dl), increased packed cell volume (mean 53.63±1.50 Range 40.0-64.0, Median 54.0 %), increased total erythrocyte count (mean 7.35± 0.259, Range 5.6-10.0, Median 7.11 million per cubic milimeter); and leukocytosis (mean 24168.42± 843.47, Range 18000-31800, Median 24000 per cubic millimeter) agreeing with the observations of other workers (Bruchim *et al*., 2006). Increased values of packed cell volume, total erythrocytes and haemoglobin suggested haemoconcentration due to dehydration. Thrombocytopenia was most likely caused by secondary platelet consumption due to vasculitis, gastrointestinal bleeding, and hyperthermia-induced platelet aggregation (Segev, *et al.,* 2004).

The diagnosis of heat stroke in these cases was based on sudden onset of high temperature in hot/hot humid months and negative cytological status for blood protozoan and rickettsial infections. To ascertain the type of arrhythmias, observed during chest auscultation, dogs were subjected to electrocardiography. Sinus tachycardia (heart rate varying from 240 to 320 bpm,), ventricular premature complexes (8-10 per minute), ventricular tachycardia (ventricular rate 180-220), ventricular fibrillations, and 3rd degree heart block were observed in 06 (30%), 03 (15%), 08 (40%), 1 (5.0%) and 02 (10.0%) cases respectively. Such electrocardiographic changes have frequently been observed in humans with heat stroke and exhaustion (Akhtar *et al*., 1993; Mimish, 2012) and have been ascribed to direct thermal injury to myocardium (Fluorney *et al*., 2003), hypoperfusion, lactic acidosis, electrolyte imbalance and micro thrombosis (Knight and Allen, 1997). Though cardiac arrhythmias or conduction disturbances were observed in dogs with heat stroke in the present study, these changes lacked specificity to predict myocardial insult as diagnostic sensitivity of electrocardiography (ECG) to diagnose minor myocardial injury is poor. An early diagnosis of myocardial injury in these cases is of immense value from a therapeutic and prognostic perspective. Recently cardiac troponins (I and T) have been reported to exhibit myocardial tissue specificity and high sensitivity. The level of cTn-I remains elevated for a much longer period of time (6-10 days), thus providing for a longer window of detection of cardiac injury (Oyama and Sisson, 2004). Therefore serum cTn-I, a more sensitive indicator of myocardial cell injury, was estimated in the dogs suffering from heat stroke having electrocardiographic changes.

The study revealed that cardiac troponin- I levels varied from 1.4 to 6.0 ng/ml with an average of 3.34 ± 1.11 ng/ml and median of 3.1 ng/ml in these dogs at the time of referral. The variability in the concentration of cTn-I in these cases may be due to different degree of cardiac damage owing to varying duration of the disease at the time of presentation of the cases at the hospital. Elevation in the level of cardiac biomarkers has frequently been reported in humans with non-exertional heat related illness during heat wave (Hausfater *et al*., 2010; Chen *et al*., 2012). Extremely high level (180 ng/ml) of cardiac troponin-I has been observed in a dog with heat stroke (Mellor *et al.*, 2006). A rather low initial elevation is followed by a more pronounced release of structurally bound cardiac troponins depending on severity of myocardial damage. cTn-I concentration is correlated with survival i.e. higher cTn-I concentration reduces the chance of survival (Oyama, 2006). Acute or chronic cardiac injury induces release of cTN-I into circulation. In normal dog without cardiac insult, cTn-I levels are very low (sometimes remain undetected) and normal cTn-I ranges for dogs have been reported at 0.03

to 0.07 ng/ml with a median of 0.02 ng/ml (Sleeper *et al.,* 2001).

CONCLUSION

The present clinical study was undertaken to study myocardial damage, if any, in dogs presented with heat stroke at the hospital. Electrocardiographic changes such as sinus tachycardia, ventricular arrhythmias and 3rd degree heart block; and increased cardiac troponin-I level in dogs with severe heat stroke confirmed varying degree of myocardial damage in them.

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