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SHORT COMMUNICATION

Coagulation parameters in dogs with heat stroke – A short study

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ABSTRACT

The present study was performed to detect the usefulness of coagulation parameters that helps in the diagnosing the severity of changes associated with heat stroke. Twenty five previously healthy dogs with the history of epistaxis, hyperthermia and dyspnea after exposure to high environmental temperature were selected and subjected to detailed clinical examination, physical examination, laboratory tests and coagulation analysis. Bilateral epistaxis, hyperthermia and respiratory distress were the common clinical signs recorded. Prothrombin time and activated partial thromboplastin time were significantly increased in affected dogs than normal. Dogs with normal coagulation parameters responded to the treatment.

Keywords: hyperthermia, prothrombin time, activated partial thromboplastin time, DIC, prognosis.

Heatstroke is an acute, rapidly progressive life-threatening condition. Heat stroke is characterized by a nonpyrogenic rise in core body temperature above 41°C causing direct hyperthermal effect to tissues, especially central nervous system, possibly leading to multiple organ dysfunctions (Bruchim, 2012). It results from exposure to a hot and humid environment (classical HS) or due to strenuous physical exercise (exertional HS) (Reiniker and Mann, 2002).

In dogs, more than 70% of the total body heat is dissipated through radiation and convection from body surfaces. Evaporation, through panting, becomes more important in maintaining normothermia when the environmental temperature increases. Non-pyrogenic hyperthermia occurs when heat dissipating mechanisms cannot adequately compensate for heat production, or when these are impaired (Flournoy *et al.,* 2003). These lead to complications such as DIC, acute renal failure, CNS abnormalities and cardiac arrhythmias which have a delayed onset, and present serious risk factors for mortality (Johnson *et al.,* 2013). The diagnosis of heat stroke is not just based on hyperthermia upon admission but should be based on the combination of the history, clinical signs and laboratory results. Thus heatstroke requires prompt, aggressive therapeutic intervention and continuous critical-care monitoring to avoid serious secondary complications and death. The coagulation parameters like Prothrombin time (PT) and activated partial thromboplastin time (aPTT) aid in the determining the severity of changes associated with heat stroke and thus helps in therapeutic management of affected dogs (Diehl *et al.,* 2000 and Hemmelgarn and Gannon, 2013).

This study reports the usefulness of Prothrombin time and activated partial thromboplastin time that helps in determining the severity of changes associated with heat stroke.

The dogs selected for this study consisted of 25 previously healthy animals admitted to Critical Care Unit – Referral Unit, Madras Veterinary college Teaching hospital during the the summer of 2014. The selection criteria for all cases included presence of systemic clinical signs (epistaxis, hyperthermia and dyspnea) of an acute onset, along with a history of exposure to a warm environment in otherwise healthy dogs, with no other concurrent disease. The

animals were subjected to detailed clinical examination, physical examination, laboratory tests and coagulation analysis. Control dogs were selected from those which were brought for routine checkup and vaccination.

For coagulation analysis 0.9 ml of blood was collected in a syringe with 0.1ml of 3.2 % Trisodium citrate solution (0.109M) and centrifuged immediately for 15 min at 3000 RPM. The undiluted plasma samples were analysed using a semiautomatic coagulometer - MISPA CLOG from M/s. Agappe Diagnostics, India. It is an opto-mechanical coagulation analyser, which applies turbodensitometric measuring principles for estimation.

During the study period of five months, from February to June, 25 cases of dogs with heat stroke were recorded among the dogs presented to Critical Care Unit – Referral Unit, Madras Veterinary college Teaching hospital. However, maximum cases had occurred during the hot summer months of April (6 cases) and May (10 cases).

The males were affected more in number than the females in the study (17 of 25 versus 8 of 25). The age of dogs ranged from 1-14 years of age. Among breeds Spitz (7) and Labrador (6) had maximum cases followed by German shepherd (4) and Rottweilers (3). Most of the dogs in this study were of large breed, suggesting that the body weight/body surface ratio is an important factor in the heat dissipation mechanism under heat stress. This is in concurrent with Flournoy *et al.* (2003) who reported that several factors are associated with the risk of developing HS which include obesity, breed (brachycephalic, Golden and Labrador retrievers) and body weight (>15kg). However, the possibility that large dogs are more active compared with small and toy breed dogs and thus are overrepresented. Owners of large breed, obese, and brachycephalic dogs should probably restrict the activity and sudden exposure of such dogs to hot and humid weather leads stress.

Table 1. Coagulation parameters (Mean ± S.E) in normal and heat stroke dogs

Coagulation parameters	Normal dogs	Dogs with Heat stroke
Prothrombin time (PT)	11.75±1.23s	25.93 ±51.16s
Activated partial thromboplastin time (aPTT)	19.05±0.78s	62.52 ± 81.56s

Coagulation parameters were found to be prolonged than control dogs (Table 1). This may be due to widespread endothelial damage in dogs with heat stroke result in tissue thromboplastin and factor XII release, with consequent activation of the coagulation and complement cascades. This may culminate in systemic inflammatory response syndrome (SIRS) and widespread microthrombosis and hemorrhagic diathesis as a result of disseminated intravascular coagulation (DIC) (Leon and Helwig, 2010b). Along with this, hepatic damage further exacerbates hemostatic abnormalities. It has been suggested that prothrombin time and activated partial thromboplastin time help in determining the severity of changes associated with heat stroke (Johnson *et al.,* 2006).

The pathophysiology of DIC involves the release of tissue thromboplastin and factor XII by injured endothelium, which activates the coagulation and complement cascades, inducing SIRS and widespread coagulation (Diehl *et al.,* 2000 and Leon and Helwig, 2010a). As DIC may appear hours to days after the initial hyperthermic insult, dogs with heat stroke should be monitored for coagulation abnormalities and clinical signs of DIC at least during the first 24 hours post insult (Drobatz and Macintire, 1996 and Bruchim *et al.*, 2006).

DIC was diagnosed in dogs that also had thrombocytopenia along with at least 2 of the following: prolonged PT, prolonged aPTT, and clinical signs compatible with DIC (petechiae, ecchymoses, hematochezia, hematemesis, hematuria). Dogs having DIC, based on these parameters, were more likely to die (Hemmelgarn and Gannon, 2013). In this study bleeding disorders such as epistaxis, hematochezia and hematemesis were recorded which were in agreement with the above authors. Bilateral epistaxis was recorded more than unilateral epistaxis (Table 2, Fig. 1). Hyperthermia ranged from 41-42 °C.

About 15 cases were recorded with respiratory distress (Table 2). The increased endothelial permeability and the DIC were possibly responsible for the pulmonary oedema, hyperaemia and haemorrhages, resulting in acute respiratory distress syndrome (ARDS), followed by respiratory failure, as has often been observed in heatstroke (Bruchim *et al.,* 2009).

Table 2. Clinical signs in dogs with heatstroke

Signs	Per cent
Bilateral epistaxis	60
Unilateral epistaxis	40
Dyspnea	60
Haematemesis	8
Haematochezia	8

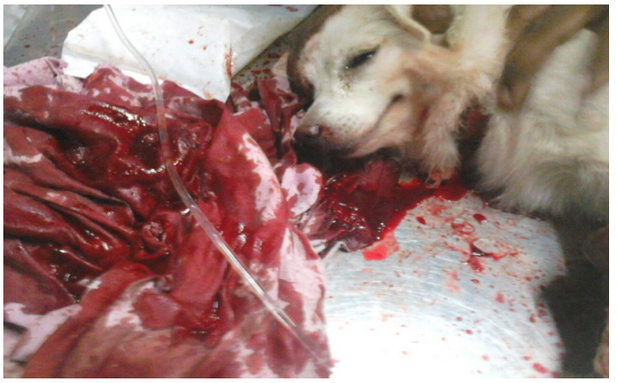

Figure 1. Bilateral epistaxis in a dog with heat stroke

Few dogs were found to have PT and aPTT within normal range. These dogs responded well to the treatment than those which had prolonged coagulation parameters. Treatment given was passive cooling, styptics, fluid therapy and antibiotics. Prolonged coagulation parameters suggested the presence of DIC which showed poor response to treatment. In this study one dog died with the signs of DIC and was subjected to necropsy within 24 h of death. Postmortem revealed widespread haemorrhages of vital organs. The findings were concurrent with Bruchim *et al.,* (2009) and Bruchim (2012) who reported that the most commonly observed lesions in their study of natural cases of canine heatstroke were hyperaemia, oedema, haemorrhages and necrosis in various organs.

From the present study it was concluded that prothrombin time and activated partial thromboplastin time aids in determining the severity of changes associated with heat stroke and thus helps in therapeutic management of affected dogs.

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