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NOTE

Pathology

Canine environmental hyperthermia: a case series

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ABSTRACT. Cases of hyperthermia caused by exposure to high environmental temperature are not uncommonly encountered in dogs. The circumstances surrounding the exposure to high environmental temperature may be accidental or intentional; thus highlighting the importance to perform a forensic autopsy. This report documents three cases of canine environmentally induced hyperthermia and all dogs in this report died less than 6 hr after exposure to the high environmental temperatures. The most commonly observed macroscopic findings included cutaneous petechial and ecchymotic hemorrhage, pulmonary edema and congestion, and epicardial and endocardial hemorrhage. Microscopically, there was systemic vascular congestion and hemorrhage, fibrin thrombi formation, and occasional enterocyte and/or renal tubular necrosis. The findings from all cases were consistent with hyperthermia induced disseminated intravascular coagulation.

KEY WORDS: dog, environmental, forensic pathology, hyperthermia, temperature

Death of a dog due to hyperthermia is not uncommonly reported in the media, especially during the hot summer months. In many of these cases, dogs are left in cars or outside in non-shaded areas for prolonged periods of time, ultimately dying of heatstroke. Accidental canine hyperthermia can occur during aerial transportation and in Japan a number of airlines have suspended certain dogs breeds (including French Bulldogs, Bulldogs, Shih Tzu, Pug) from air travel [6, 8]. Heatstroke is a form of hyperthermia and is characterized by having a core body temperature >106°F (41°C) and the body cannot maintain normal temperature through its regular thermoregulatory system [5]. Heatstroke occurs when heat production exceeds heat dissipation and can result from exposure to environmental heat or from strenuous physical exercise. Clinical findings of heatstroke include hyperthermia, tachypnea, panting, collapse, depression, altered or loss of consciousness, vomiting, diarrhea, and bleeding (petechia(e), ecchymosis, hematochezia) [2, 5].

There is limited information in the scientific literature describing the pathological findings observed at autopsy from dogs that succumbed to heatstroke [2, 5]. In a study reviewing eleven dogs that died due to heatstroke, the most prevalent lesions include hemorrhagic diathesis, microthrombosis, and coagulative necrosis of multiple organ systems. Heatstroke cases present with hyperthermia induced disseminated intravascular coagulation (DIC) and systemic inflammatory response syndrome which ultimately leads to multi-organ dysfunction and death [2].

The dogs selected for this series consisted of three dogs submitted to the diagnostic laboratory with a history of sudden and unexpected death after exposure to environments with high environmental temperatures for less than 6 hr (Table 1). All three dogs were found deceased and no medical interventions were performed. Dog 1 was found within a hot car (measured temperature was 125.4°F/51.3°C) that had failure of the car's cooling system. Dogs 2 and 3 were found within hot rooms (room temperature was not measured for either case). All dogs had a forensic autopsy performed within 36 hr of death. Macroscopic and microscopic examinations of tissues were performed. Tissues were fixed in 10% neutral-buffered formalin and embedded in paraffin wax. Sections were cut at 4 μm and stained with hematoxylin and eosin. Tissues examined microscopically include lung, liver, kidney, spleen, gastrointestinal tract (stomach, small intestine, and colon), adrenal glands, thyroid glands, brain, skeletal muscle, and skin.

All dogs had a minimal to mild degree of autolysis. One dog (dog 1) presented with fixed rigidity of the limbs (Fig. 1). All dogs had cutaneous findings that included petechial and ecchymotic hemorrhage (Fig. 2). Cutaneous and subcutaneous vasculature of all dogs was multifocally congested and there was erythema of the skin. One dog (dog 1) had multiple areas of expansion of the subcutaneous tissues by edema near both shoulders.

All dogs had variable amounts of red and clear fluid within the trachea and/or main stem bronchi and red tinged fluid oozed from the lungs upon sectioning. Lungs from all dogs were heavy on palpation and were mottled pink to red. There was a mild amount of serosanguinous pericardial effusion in one dog (dog 1). The epicardium from two dogs (dogs 2 and 3) had mild to

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Table 1. Signalment of dogs examined in this study

Dog	Age	Sex	Breed	Weight (kg)	Location of heat exposure	Month of incident	Maximum daily temperature	Interval between heat insult and death (hr)
1	2 years	Castrated male	Belgium Malinois	24	Hot automobile	August	85°F (29.4°C)	<6
2	Juvenile	Intact male	Pitbull	9	Hot room	September	97°F (36.1°C)	<4
3	Juvenile	Intact male	Pitbull	9	Hot room	September	97°F (36.1°C)	<4

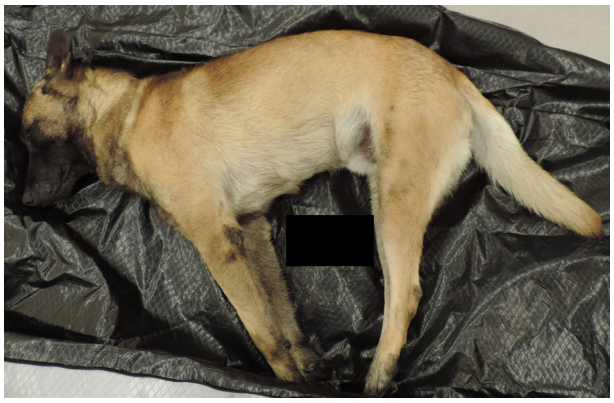

Fig. 1. Dog 1. This dog has fixed rigidity of all four limbs.

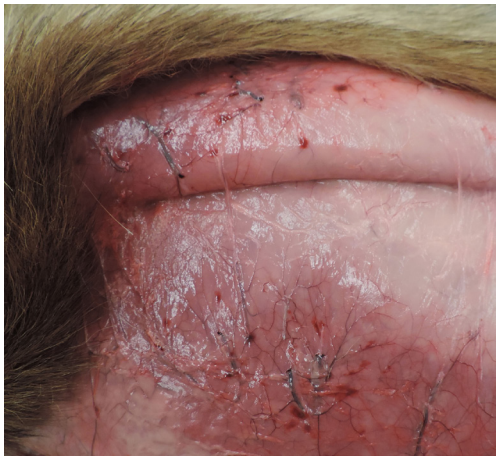

Fig. 2. Dog 1. Macroscopically identified petechial and ecchymotic hemorrhage of the subcutaneous tissue of the back.

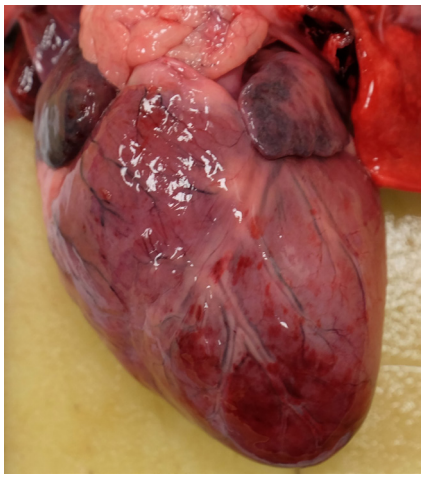

Fig. 3. Dog 2. Macroscopic lesion of the epicardium showing "paint brush" hemorrhage.

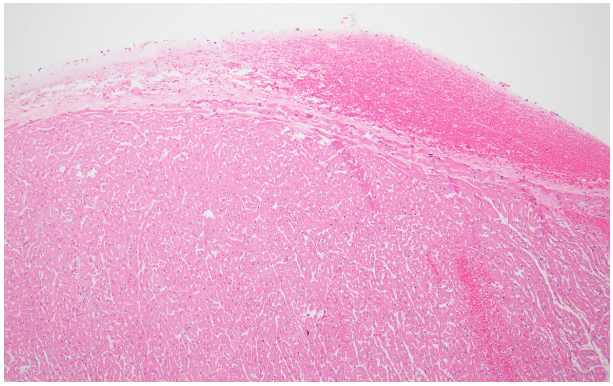

Fig. 4. Dog 2. Microscopic lesion of the epicardium showing acute hemorrhage.

moderate hemorrhage with a paintbrush-like pattern (Fig. 3) and a marked amount of hemorrhage of the endocardium within the left and right ventricles. Thymic hemorrhage was observed in 2 dogs (dogs 2 and 3).

There are pinpoint foci of hemorrhage on the surface of the liver from one dog (dog 1). There was petechial hemorrhage of the mucosal surface within the urinary bladder of one dog (dog 2). One dog has marked congestion of mesenteric blood vessels. All dogs have marked congestion of meningeal blood vessels.

The dermis and/or panniculus of all dogs had multiple areas of hemorrhage. There was lack of erythrophagia and there was no identification of erythrocyte break down products (hemosiderin, hematoidin). Vasculature within the dermis and panniculus were congested.

All dogs had pulmonary edema within sections of lung examined. There was multifocal hemorrhage within alveoli and vasculature within the interstitium was congested. Expanding the epicardium and endocardium from 2 dogs (dogs 2 and 3) were multiple regions of hemorrhage (Fig. 4). One dog had several small regions of hemorrhage extend into the myocardium (dog 2). There was lack of erythrophagia and there was no identification of erythrocyte break down products (hemosiderin, hematoidin).

There was massive congestion of hepatic sinusoids within all dogs. Within 2 dogs (dogs 2 and 3), there was degeneration of centrilobular hepatocytes and fibrin thrombi were identified within hepatic sinusoids of these dogs. Within the kidneys, there are fibrin thrombi within glomerular capillaries of all dogs. There is congestion of renal vasculature (glomerular and interstitial) within all dogs. Two dogs (dogs 2 and 3) had occasional renal tubular necrosis. Incidentally, a single dog (dog 1) had mild thickening of the basement membrane of Bowman's capsule and rare glomerular sclerosis. All dogs had congestion of meningeal and intraparenchymal blood vessels. No significant histologic abnormities were identified within the remaining organs examined.

There are a number of risk factors associated with the development of heat stroke. Besides exposure to high environmental temperature, risk factors include obesity, dog breed (Golden Retriever, Labrador Retriever, brachiocephalic breeds), lack of access to potable water, and confinement to poorly ventilated spaces [1, 5]. Potential scenarios where an animal is exposed to high environmental temperature include being left in a car, being left outside in full sun, and being left in a poorly ventilated room or building. In humans, it has been shown that core body temperature rises between 0.15–0.2°C for every 1% of bodyweight lost due to dehydration from exercise [3].

The most common pathological findings in this case series of canine heatstroke included congestion, hemorrhage, and thrombosis of multiple organs. Vasculature congestion was most prominent within the skin, brain, and lungs. Microscopically, centrilobular congestion and hepatocellular degeneration in the liver was prominent within all dogs. Hemorrhage was most commonly encountered within the skin, heart, and lungs. Fibrin thrombi were identified within the liver of two dogs and the kidneys from all three dogs.

One dog in this series presented in a state of permanent rigidity. This finding needs to be differentiated from rigor mortis which is a transient process that will terminate by approximately 36 hr after death. The fixed rigidity results from the coagulation of muscle protein and ultimately results in muscle shortening and permanent rigidity [4].

The pathologic findings in this series is similar to many of the findings in the 2008 retrospective study by Bruchim *et al*. looking at eleven dogs that died due to heatstroke. One major difference was the lack of "paint brush" hemorrhage of the gastrointestinal tract in the current study; whereas, all dogs in the previous study had gastrointestinal hemorrhage. There also appeared to be less severe necrosis observed in the current study with only slight necrosis within the kidney and small intestine. Renal tubular degeneration and necrosis are common findings in cases of heat stroke and result in severe hemoconcentration and azotemia [5]. Renal tubular necrosis and renal infarcts was reported in 5 of 6 dogs that died of heatstroke [1]. In addition to renal tubular damage, damage to renal glomeruli is also suspected in cases of heatstroke [7].

In contrast to the previous studies, the dogs in this report all died prior to receiving any veterinary medical intervention for hyperthermia and the time from exposure to the elevated environmental temperature to death for all cases was less than 6 hr. The limited timeframe from exposure to death is suspected to have played a role in the difference between the severity of the lesions observed in this case series and previous studies. We suspected that histopathological changes might need additional time to develop; whereas, derangement of cell function would develop more rapidly in these instances. In a previous study, the median time to presentation to a veterinary hospital was 4 hr; whereas, the median time to death was 13 hr with some of the deaths occurring over 20 hr from exposure [2]. In that study, one dog with a short time to death (4 hr) had minor postmortem lesions compared to other dogs in the study. Death in cases of heatstroke are thought to be due to coagulation abnormalities, DIC, and pulmonary abnormities [5].

In this series, all dogs had macroscopic and microscopic evidence of DIC. Given the short duration from the initial exposure to high environmental temperatures to death of these dogs, the pathological changes observed were mild compared to most of the reported cases in the literature. It is suspected that not enough time elapsed to allow for observation of more fulminant cellular changes at the light microscope level. Although the lesions observed were mild, overall the pathological findings were consistent with DIC. A review of the clinical history provided valuable information about the circumstances around the death of these dogs to allow for determination of the cause of death as heatstroke.

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