Heat stroke in a Great Pyrenees dog

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Abstract — A 2-year-old, male Great Pyrenees presented with a history and clinical signs suggesting heat exhaustion. Treatment with intravenous fluids, antibiotics, and surface cooling was unsuccessful, and euthanasia was elected. Histological evaluation of the dog’s tissues revealed lesions consistent with severe hyperthermia and shock.

Résumé — Coup de chaleur chez un chien des Pyrénées. Un chien mâle des Pyrénées âgé de deux ans se présente avec une anamnèse et des signes cliniques qui suggèrent un coup de chaleur. Un traitement à l’aide de solutions intraveineuses, d’antibiotiques et d’un refroidissement de surface échoue et l’euthanasie est choisie. L’évaluation histologique des tissus du chien a révélé des lésions correspondant à une hyperthermie et à un choc grave.

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A 2-year-old, intact male, Great Pyrenees was presented as an emergency because of collapse from suspected heat exhaustion. The dog, reported to be normal that morning, had been found unconscious and in lateral recumbency. The ambient temperature that day was about 30°C, not taking into account the relative humidity. Although the owner had attempted to cool the dog by spraying him with water from a garden hose, the dog remained unresponsive. Diarrhea was noted, but vomit had not been observed. There was no history of trauma, seizures, or potential exposure to toxicants. The vaccination status of the dog was unknown, and previous medical history was unremarkable, except for a minor laceration associated with being struck by a car 14 mo earlier. The dog was presented to the clinic approximately 45 min after being discovered by the owner.

The dog was in thin body condition (body weight [BW] 43 kg) and unable to stand, but he was responsive to external stimuli. Rectal temperature was elevated (40.6°C), breathing was labored (44 breaths/min), and heart rate was increased (120 beats/min). Oral mucous membranes were pink, and the capillary refill time was approximately 2.5 s. Dehydration was estimated at 10%, on the basis of tauty mucous membranes and a moderate skin tent. Malodorous, hemorrhagic diarrhea was noted; it contained 1 adult roundworm. The dog appeared to be in pain, exhibiting prominent abdominal contractions that made deep digital palpation difficult. Auscultation of the thorax was unremarkable. Due to the apparently moribund state of the patient, a full neurological examination could not be performed at that time. The initial differential diagnoses included severe heat stroke, hemorrhagic gastroenteritis (Clostridium perfringens type A or parvovirus), septicemia, exposure to a rodenticide anticoagulant, leptospirosis, and hyperthermia secondary to seizures.

A complete blood cell (CBC) count (QBC VetAutoread Hematology Analyzer; Idexx Laboratories, Westbrook, Maine, USA), serum electrolyte panel (VetLyte Electrolyte Analyzer; Idexx Laboratories), and blood glucose, total serum protein, and blood urea nitrogen (BUN) (VetTest Chemistry Analyzer; Idexx Laboratories, Westbrook, Maine, USA) analyses were performed immediately. The CBC count revealed polycythemia (hematocrit 56.6 L/L; reference range, 37.0 to 55.0 L/L) and leukocytosis (26.8 × 10⁹ cells/L; reference range, 6.0 to 16.9 × 10⁹ cells/L); the lymphocyte: monocyte ratio was 12.1 (reference range, 1.1 to 6.3). All serum electrolyte parameters were elevated: sodium 161.3 mmol/L (reference range, 144.0 to 160.0 mmol/L), potassium 5.88 mmol/L (reference range 3.50 to 5.80 mmol/L), and chloride 126.5 mmol/L (reference range, 109.0 to 122.0 mmol/L). The blood glucose was 3.8 mmol/L (reference range, 3.4 to 6.0 mmol/L), and total protein values were increased (85 g/L; reference range, 55 to 75 g/L). The BUN was 14.49 mmol/L (reference range, 2.50 to 9.64 mmol/L). Lateral and ventrodorsal plain survey radiographs of the abdomen appeared to be normal. On the basis of the hematological and radiographic findings, none of the initial differential diagnoses could be eliminated. Because the dog had bitten a staff member and its vaccination status was unknown, rabies was added to the list of differential diagnoses.

The dog was initially treated with lactated Ringer’s solution USP (Abbott Pharmaceuticals, Toronto, Ontario),
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4 L, IV, via catheters placed in both cephalic veins at a rate of 90 mL/kg BW/h; dexamethasone (Vétoquinol, Lavaltrie, Quebec), 5 mg/kg BW, IM, q24h; and penicillin (Penicillin G Procaine; Rhone Merieux Canada, Victoriaville, Quebec), 20 000 IU/kg BW, IM, q12h. Cold wet towels were draped over the animal’s body, and isopropyl alcohol was poured on the distal limbs in an attempt to decrease core body temperature.

During the next several hours, the dog’s condition continued to deteriorate despite aggressive fluid therapy. He remained depressed and did not attempt to sit or stand up. Abdominal contractions and labored breathing continued. Petechiation of the oral mucosa and ecchymotic hemorrhages on the ventral abdomen appeared, while bradycardia developed (60 bpm) and the peripheral pulses became weak and thready. Potential seizure activity, which consisted of whole body tremors and rigidity of the limbs, was noted once by the owner during this time. The dog became comatose and, due to the poor response to treatment, was euthanized.

A postmortem examination was performed immediately. Grossly, the dog was thin, with minimal SC body fat stores. Petechiation of the myocardium and colonic serosa was evident. There were areas of diffuse hemorrhage in the colonic mucosa, which appeared very thickened, restricting the intestinal lumen. The small intestine contained approximately 1.0 L of blood-tinted fluid, and its mucosal surface appeared hyperemic. Both kidneys appeared normal in size and texture, but they were brick red. All other organs and tissues appeared grossly normal. Samples of lung, liver, kidney, intestine, spleen, heart, stomach, lymph node, urinary bladder, and skeletal muscle were placed in 10% buffered formalin and submitted to the Animal Health Laboratory (University of Guelph, Guelph, Ontario) for histopathologic examination. Sections of frozen lung, liver, kidney, and intestine were also submitted for bacteriological culture. The dog’s brain was submitted to the Canadian Food Inspection Agency to be tested for rabies virus infection. A tentative diagnosis of heat stroke was made on the basis of the necropsy findings and the circumstances surrounding the dog’s death. The rabies test was negative.

Histopathologic examination revealed extensive intestinal mucosal hemorrhage, consistent with vascular shunting associated with shock. Similar ischemic lesions were noted in other tissues. Observation of swollen myofibers with hypereosinophilic sarcoplasm in skeletal muscle was consistent with a diagnosis of hyperthermia. Bacterial culture revealed heavy growth of Enterococcus spp. in lung, liver, kidney, and intestine. Although this organism is normally found in a healthy intestinal tract, the excessive growth in other tissues represented bacteremia that had existed antemortem, rather than due to postmortem sample contamination.

Heat stroke (hyperthermia) in dogs occurs when heat production within the body overwhelms mechanisms responsible for heat loss. It is important to distinguish heat stroke from pyrexia, since heat stroke does not affect the hypothalamic set point. Hyperthermia has been reported in a variety of animals, including dogs and horses (1–4). As in this case, it is most commonly diagnosed during the summer months, when ambient temperatures and humidity are high. Restricted access to water aggravates the condition (2,5). Heat stroke is associated with a marked elevation in core body temperature, often resulting in cellular damage as temperatures approach 42°C (4,6). Cells subjected to sublethal injury (as in hyperthermia) produce heat shock proteins (HSPs), which, in their role of reestablishing normal cellular activities, target and degrade severely damaged proteins (6). Cellular integrity and function may be lost due to denaturation of intracellular proteins and lipids, and if this is severe, multiorgan failure results (1,2,4).

Heat stroke, the most serious of the heat-induced illnesses (5), is most commonly diagnosed when sudden acute signs occur in an animal previously considered normal. Risk factors for developing heat stroke include age extremes, thick haircoat, and prior history of heat-related illness, with a predisposition in giant and brachycephalic breeds (3,7). This dog, a young Great Pyrenees without access to water, fell into a high-risk category. The clinical signs associated with heat stroke may vary, depending on the degree and duration of exposure to elevated environmental temperatures. Animals are usually presented on an emergency basis, with some combination of vomition, diarrhea, tachypnea, inability to rise after collapse, seizures, and other central nervous system abnormalities, and petechial or ecchymotic hemorrhages, or both, on the visible mucous membranes (2–5).

As core body temperature approaches 42° to 43°C, hemostatic mechanisms within the body deregulate. Heat damage to endothelial cells results in the release of thromboplastin from necrotic cells and subsequent disseminated intravascular coagulation (DIC). Consequently, platelets and clotting factors are activated and consumed within the bloodstream (2,4–6,8). However, these effects are variable, as core body temperature varies between individuals with heat stroke (6). As platelets are sequestered, partial thromboplastin time, prothrombin time, and fibrin degradation products in plasma may increase, and thrombocytopenia may be evident. The severe dehydration experienced by patients suffering from hyperthermia may cause hepatic injury as a result of hypoperfusion, which may further exacerbate hemostatic abnormalities (6,8). A coagulation profile is recommended for all patients suffering from heat stroke (5). Since a full biochemical profile and coagulation profile were not performed in this patient, it is difficult to ascertain if the hemorrhages noted on the oral mucosa and ventral abdomen, and internally at necropsy, were the result of DIC, hepatic injury, or some other cause.

Gastrointestinal signs associated with heat stroke may be directly related to heat damage to the intestinal mucosa, or they may be secondary to inadequate perfusion during hypovolemic shock. Hemorrhagic diarrhea may occur as a result of generalized coagulopathy or direct mucosal damage (4,5). The intestinal lumen contains numerous gram-negative bacteria and their associated lipopolysaccharide (LPS). The intestinal wall is normally impermeable to LPS, and LPS is rapidly detoxified by the reticuloendothelial system if small amounts happen to be absorbed. During heat stroke, ischemia damages the mucosal surface, allowing LPS to leak into the portal and systemic circulations, thereby overwhelming hepatic and splenic detoxification...
mechanisms. The end result is vascular collapse, shock, and death (9). Gathrium et al (9) assessed the benefits of treating heat stroke patients with antibiotics and reported that decreasing the intestinal bacterial load increases survival rates in heat-stressed dogs. The bacterial culture results suggest that this dog may have suffered from endotoxic shock, but antibiotic therapy was initiated too late in the course of the illness to be effective.

Successful treatment of patients suffering from heat stroke requires early attempts to reduce core body temperature, while providing cardiovascular support and treating secondary complications (4). Ideally, owners should initiate the process at home by covering the dog with cool wet towels. Cold-hosing these animals is generally not recommended, as it causes peripheral vasodilation that impedes heat loss (1–5). Maximum heat loss occurs when the skin temperature is 22° to 28°C. Other cooling measures that can be employed include gastric and peritoneal lavage with a cold solution and evaporative cooling (4,5). It is important to note that cooling continues after cessation of treatment, and cooling measures should stop before body temperature reaches the normal range (1,4). Large doses of a balanced electrolyte solution should be administered at a shock rate of 90 mL/kg BW/h to correct dehydration and associated hypovolemia (1–5). Corticosteroids may be administered to treat shock and potential cerebral edema; however, treatment with corticosteroids is controversial, as they may induce immune suppression and gastrointestinal ulceration. Heparin and freshly thawed frozen plasma may be useful when the patient suffers from a coagulopathy (2–5).

The prognosis for heat stroke is guarded to good, depending on the extent of organ system failure, the duration of exposure to high ambient temperatures, and the aggressiveness of treatment. Client education is essential in preventing this condition.

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References