Heatstroke

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Heat-related illnesses are most common in dogs during the summer months. Severity can range from heat cramps to life-threatening heatstroke. As the name implies, heatstroke leads to CNS dysfunction. Heatstroke involves a systemic inflammatory response (SIRS) leading to multiorgan dysfunction (MODS) with CNS dysfunction predominating. Rapid recognition and treatment are essential for successful outcomes. Owners should be trained to recognize signs of heat-related illness. Heat cramps may be difficult to recognize, but signs of heat prostration or exhaustion include fatigue, muscle tremors, vomiting, and diarrhea. Heat damage can be due to direct cellular damage such as denaturing proteins or inducing apoptosis. Certain temperatures thresholds have been shown experimentally to induce damage, but the rectal temperature on presentation is not useful for determining the severity of illness. The rectal temperature may not reflect core body temperature. In fact, peripheral vasodilation may lead to overestimation of core body temperature. Furthermore, a normal temperature does not rule out heatstroke.

Patients typically present with a history of increased heat production or decreased heat loss. Examples of increased heat production include exercise, seizures, and toxins (metaldehyde, strychnine, amphetamines, etc.). Examples of decreased heat loss include increased humidity, confinement/inadequate ventilation, brachycephalic airways, laryngeal paralysis, obesity, thick hair coat, and dehydration. The major mechanisms of heat loss include convection, conduction, radiation, and evaporation. These mechanisms are important to understand not only for preventing heatstroke but also for treating it. Convection is the loss of heat into the surrounding air (e.g. fan). Conduction is the transfer of heat to a solid object in contact with the body (e.g. cool metal table). Radiation is the loss of heat into the atmosphere. Evaporation is a loss of heat through changing water to vapor. This occurs as sweating in people and mainly panting in dogs. In normal conditions, dogs rely on radiation and convection. When the ambient temperature and body temperature become similar, evaporation (panting) becomes the primary heat loss mechanism. A dog trapped inside a vehicle illustrates a disastrous combination of risk factors for heatstroke.

Patient Assessment

An elevated temperature is common, but prior cooling or severity of the disease may lead to normal or decreased body temperatures. Active cooling is recommended for temperatures above 103.5F and will be discussed in the treatment section. Like all emergency patients, triage and an initial focus on the ABCs is recommended. Patients commonly have distributive shock secondary to massive peripheral vasodilation. Mucous membranes are usually hyperemic with a fast capillary refill time. Hypovolemic shock is also common due to evaporative fluid loss, vomiting, and diarrhea.

Respiratory abnormalities may have predisposed the patient to heat-related illness. Loud/noisy breathing may indicate upper airway issues such as laryngeal paralysis or edema, obstruction, or tracheal collapse. Aspiration pneumonia is also possible due to vomiting and decreased mentation. Pulmonary hemorrhage is also possible due to heat damage and impaired coagulation (e.g. DIC).

Mentation may range from obtunded to comatose. Seizures are also possible. Causes of CNS dysfunction includes poor perfusion, direct heat damage, cerebral edema, intracranial hemorrhage, and hypoglycemia.

A minimum database is recommended. Hematocrit typically shows hemoconcentration. Blood glucose measured by glucometers may be falsely decreased due to hemoconcentration but may also be decreased due to utilization or sepsis. Urinalysis may show evidence of acute kidney injury (glucosuria, red blood cells, casts, etc.). A blood smear should be performed to verify platelet count and assess for nucleated red blood cells (nRBCs). One study found that having 18 or more nRBCs per 100 WBCs was 91% sensitive and 88% specific for mortality. This may be a useful prognostic tool or useful diagnostic tool for patients presenting hours after the initial insult or unknown history. nRBCs decrease rapidly after the first 24 hours. Direct heat damage can activate coagulation and result in the consumption of platelets and clotting factors. This may lead to decreased platelets, increased PT and aPTT, and ultimately DIC. It is important to keep in mind that petechiae may be caused by direct heat damage to capillary blood vessels and do not always indicate thrombocytopenia and DIC.

Treatment

Active cooling until 103.5F is recommended. Methods include spraying the entire dog with cold water and using fans. IV fluids, placing the patient on a cool metal table, and adding ice cubes inside the facemask during oxygen supplementation may also help increase heat loss. Contrary to most veterinary textbooks, ice baths are recommended in humans with exertional heatstroke. Theoretical concerns of ice water/baths causing peripheral vasoconstriction, shivering, and heat-trapping have been disproven. Several human meta-analyses have concluded that cold-water immersion is the most effective cooling strategy for human exertional heatstroke. In fact, studies show excessive cooling and hypothermia are the biggest concerns when using ice-water or cold-water immersion.

Oxygen supplementation with flow-by is quick and easy to apply. This may decrease the work of breathing and heat production. Any underlying airway/breathing issues should be treated such as providing sedation for laryngeal paralysis. Intubation and positive pressure ventilation may also be necessary.

IV fluids aid in cooling as well as achieving adequate perfusion. Many patients present in hypovolemic shock. Isotonic crystalloids are typically given first. Synthetic colloids are controversial and may increase the risk of coagulopathy and acute kidney injury. Hypertonic saline may be considered and still effective in the face of dehydration. Following hypertonic saline with isotonic crystalloids is recommended. Severely affected patients may require vasopressors (norepinephrine) and inotropic (dobutamine) support.

Although CNS dysfunction is the hallmark finding in human heatstroke, dog brains appear to be more tolerant of heat compared to humans. Dog brains contain higher levels of heat shock proteins that serve to protect cellular function from heat. Mentation/CNS function should be assessed after resuscitation. Poor perfusion and hypoglycemia must be addressed rapidly. If CNS dysfunction persists, cerebral edema may be present. Mannitol (0.5-1 g/kg) or hypertonic saline (4 ml/kg of 7.5% NaCl) administration should be considered.

Acute kidney injury is possible from direct heat damage, decreased perfusion, rhabdomyolysis, and vascular insults secondary to DIC. Serial monitoring of urine output, kidney values, and UA may be indicated. Maintaining adequate hydration and perfusion and addressing the underlying disease is the mainstay of treatment. Unproven therapies include mannitol, furosemide, and diltiazem administration. For severely affected patients, dialysis or continuous renal replacement therapy (CRRT) may be necessary.

Gastrointestinal compromise is common due to direct heat damage and poor perfusion leading to mucosal sloughing and ulceration. GI protectants such as sucralfate and proton pump inhibitors are commonly used. Bacterial translocation is also possible and a potentially higher risk in patients with GI sloughing/bleeding. Antimicrobials are not indicated in all patients and should only be considered in severely affected patients. Adequate nutrition is important and options to consider include nasal feeding tubes, e-tubes, and parenteral nutrition. Nasogastric tubes may be useful to decompress the stomach if ileus is present.

Other treatments include liver support (N-acetylcysteine, SAMe, milk thistle) and blood products. FFP is recommended in coagulopathic patients with clinical bleeding. Red blood cell transfusion may be necessary for patients with clinically significant blood loss.

Prognosis

Heat-related illnesses can quickly become life-threatening if heatstroke develops. This leads to SIRS followed by MODS and ultimately death if not rapidly recognized and treated. Most deaths occur during the first 24 hours. Dogs still alive at 72 hours all survived to discharge. The prognosis for heatstroke is typically guarded, and owners must be warned about the possibility of MODS. Client education about prevention, rapid recognition, and cooling measures en route to the veterinarian all play a role in tackling heat-related illnesses.

References available from the author.