

A perianesthetic approach of heat stroke in a dog with laryngeal paralysis

De perianesthetische benadering van hittedslag door larynxparalyse bij de hond

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ABSTRACT

In this case report, a seven-year-old, male, castrated Landseer presented with chronic respiratory distress, occasional coughing, dysphonia and exercise intolerance is described. The stress caused by transportation and the physical examination rendered the dog severely dyspneic and cyanotic. At that moment, the core body temperature was increased up to 42.5 °C. Based on the clinical signs, laryngeal paralysis causing heat stroke was the most likely diagnosis. The dog was anesthetized and intensive temperature control methods, like active cooling and fluid therapy, were applied. As soon as the dog was cardiovascularly stable, emergency treatment for laryngeal paralysis was performed. As a result of timely intervention, the dog recovered completely without any persisting complications.

SAMENVATTING

Een zeven jaar oude, mannelijke, gecastreerde landseer werd aangeboden met chronische ademhalingsklachten, occasioneel hoesten, dysfonie en inspanningsintolerantie. Door stress veroorzaakt tijdens het transport en het klinisch onderzoek vertoonde deze hond al snel zeer ernstige dyspneu met cyanose. Op dat moment werd een rectale lichaamstemperatuur van 42,5°C gemeten. Gebaseerd op de klinische symptomen was een hittedslag veroorzaakt door larynxparalyse de meest waarschijnlijke diagnose. De hond werd met spoed onder anesthesie gebracht en intensieve verkoelende behandelingen, zoals actief koelen en vloeistoftherapie, werden uitgevoerd. Vanaf het moment dat de hond cardiovasculair stabiel was, werd een spoedbehandeling voor larynxparalyse uitgevoerd. Dankzij een tijdige interventie herstelde deze hond volledig zonder blijvende complicaties.

INTRODUCTION

Heat stroke is a life-threatening condition that veterinarians regularly face in practice during summer. It is frequently observed in dogs but rarely in felines (Johnson et al., 2006). This complex form of hyperthermia is characterized by body temperatures above 41°C and dysfunction of the central nervous system (Bruchim et al., 2009; Hemmelgarn and Gannon, 2013a). Typical for this condition is the direct lethal damage to organ systems as a result of a non-pyrogenic increase in body temperature (Flournoy et al., 2003). Heat stroke is associated with a systemic in-

flammatory response, leading to progressive multiple organ failure and possibly resulting in death (Bruchim et al., 2009; Hemmelgarn and Gannon, 2013b).

Multiple organ systems are involved in this condition, with the central nervous system playing a crucial role. Also the gastrointestinal tract, the renal, the pulmonary and the coagulation systems are disturbed. Common side effects of heat stroke are disseminated intravascular coagulation, acute kidney injury, and acute respiratory distress syndrome (Bruchim et al., 2009; Hemmelgarn and Gannon, 2013a). This condition has high mortality rates of 50% to 56%. To achieve an increased chance of survival, early detec-

tion, elimination of the cause, aggressive therapy and critical monitoring are necessary (Flournoy et al., 2003; Hemmelgarn and Gannon, 2013a).

CASE HISTORY

A seven-year-old, male, castrated Landseer was presented at the Small Animal Department, Faculty of Veterinary Medicine (Ghent University) with chronic respiratory distress, occasional coughing, dysphonia and exercise intolerance. The symptoms had gradually started six months before presentation. Since a couple of days before presentation, the dog had become very dull and had developed an inspiratory stridor at rest. At the local veterinarian, the dog was tested for laryngeal paralysis, but findings were inconclusive.

On physical examination, the dog was alert. He showed mixed inspiratory and expiratory dyspnea and stridor. Body weight was 84.5 kg and a body condition score (BCS) of 6/9 was given. He was tachypneic (40/min) and his heart rate was 120 beats per minute (bpm). Rectal temperature at that point was moderately increased (40.5°C). The red mucous membranes showed a capillary refill time (CRT) of less than two seconds. Pulmonary and cardiac auscultations, as well as pulse quality were normal. During consultation, the dog became progressively more dyspneic and cyanotic. Based on the severity of the respiratory symptoms, an extensive preanesthetic examination was not possible. An ASA-score 'V E' (5/5) was given because the patient was not expected to live 24 hours without surgery and was presented as an emergency. An intravenous (IV) catheter was urgently placed into the vena cephalica, followed by induction of anesthesia with propofol (2.1 mg/kg IV, PropoVet® Multidose, Zoetis Belgium S.A., Louvain-la-Neuve, Belgium). By this time, the core body temperature was increased up to 42.5 °C and the heart rate was increased to 229 bpm. Drastic temperature control methods were performed and consisted of applying cold water to the skin with presoaked towels and enhancing evaporation with a fan. At the same time, fluid therapy was initialized. A cooled shock bolus of a Ringer-lactate solution (Vetivex® 500 ml, Dechra Veterinary Products, Belgium) was infused IV at a rate of 90 ml/kg/h over 40 minutes and afterwards continued at a maintenance rate of 5 ml/kg/h throughout the rest of the anesthesia. Laryngeal paralysis was confirmed by laryngeal inspection, and an endotracheal tube, size 11, was placed to secure the airway.

When stabilized, 65 minutes after initiating maintenance anesthesia, the dog was taken to medical imaging, where left-right lateral and ventrodorsal radiographs of the thorax were taken. The caudal esophagus was mildly distended by gas due to stress aerophagia, general anesthesia or polyneuropathy. A diffusely increased lung opacity, likely caused by expiratory phase and obese body condition, as well as an alveolar

lung pattern with loss of volume in the right middle and caudal lung lobes, compatible with atelectasis, was visible. Hematology, biochemistry and coagulations tests were performed. Except for a hematocrit of 51%, no abnormalities were detected. All the findings supported the diagnosis of heat stroke due to laryngeal paralysis, probably as a clinical manifestation of a generalized peripheral polyneuropathy, also called geriatric onset laryngeal paralysis polyneuropathy.

With the owner's consent, a unilateral cricoarytenoid lateralization (CAL) was performed. Anesthesia was maintained with isoflurane (IsoFlo® 100%, Zoetis Belgium S.A., Zaventem, Belgium) vaporized in oxygen with an initial setting of 1.8% on the vaporizer. The end tidal isoflurane concentration (F_E Iso) gradually rose and ranged from 0.61-1.5% over the anesthetic period. An electrocardiogram was connected, to measure the electric activity of the heart muscle and heart rate (HR). The peripheral oxygen saturation (SpO_2) and the end tidal pressure of carbon dioxide (P_E CO₂) were continuously monitored using a pulse oximeter and by side stream capnography. By non-invasive Doppler blood pressure measurement, a systolic blood pressure of 70 mmHg at the time of induction was estimated. During the rest of the procedure, anesthesia was maintained with isoflurane vaporized in oxygen using a Bain breathing system in the preparation room and a circle rebreathing circuit in the operating room with appropriate fresh gas flows. Manual ventilation was performed in the preparation room and later switched to mechanical ventilation in the operating room. The peak inspiratory pressure (PIP) varied between 12-16 cm H₂O. Respiratory rate (F_R) ranged between 14-19/min. P_E CO₂ ranged between 19-61 mmHg, with an average of 39 mmHg. The fraction of inspired oxygen (FiO_2) ranged between 88% and 96%, while the peripheral oxygen saturation (SpO_2) varied between 93-99%. Invasive intra-arterial blood pressure measurement via a catheter (20G) in the dorsal pedal artery showed a mean arterial blood pressure (MAP) of 56-77 mmHg. The femoral pulse pressure was checked regularly and quickly changed from a powerful pulse into a weak pulse ten minutes after initiating inhalation anesthesia, but normalized 45 minutes later. During the first 90 minutes, the patient showed hyperemic mucous membranes, followed by normal pink appearance till the end. The CRT stayed less than one during the whole procedure and only normalized, to less than two seconds, at the very end. The palpebral reflexes were monitored regularly and remained negative during the whole procedure. The heart rate started to decrease gradually when active cooling methods were applied and normalized during surgery (ranging between 120-84 bpm). At the end of anesthesia, a rate of 75 bpm was registered. A body temperature of 40.4°C was measured 45 minutes after inducing anesthesia, and further decreased to a constant temperature of 37.6°C 105 minutes later until the end of anesthesia.

The procedure consisted of placing two monofilament nylon 0 (Ethilon, Ethicon™, Johnson & Johnson Medical N.V./S.A., Diegem, Belgium) sutures from the caudodorsal aspect of the cricoid cartilage to the muscular process of the left arytenoid cartilage, thereby resulting in an abducted position of this arytenoid. Closure was performed routinely.

One hour after initiating anesthesia, amoxicillin/clavulanic acid (20 mg/kg IV, Augmentin P500®, GlaxoSmithKline, Waver, Belgium) was injected and repeated 2.5 hour later. Fentanyl (Fentadon®, Eurovet Animal Health, Belgium) was given as an intraoperative analgesic by constant rate infusion, ranging from 5 to 7 µg/kg/hour, after a loading dose of 1 µg/kg. Around the same time, 5.9 mL/kg (500 ml) of a plasma expander solution (Voluven®, 6% hydroxyethyl starch 130/0.4, Fresenius Kabi BV, Schelle, Belgium) was given over 30 minutes to address hypotension. Propofol was injected (0.41 mg/kg IV) halfway the procedure and ten minutes before the end of surgery, dexamethason (0.5 mg/kg IV, Rapidexon®, 2mg/mL, Dechra, Belgium) was administered. At the end of the anesthesia, the dog was injected with dexmedetomidine (1 µg/kg IV, Dexdomitor®, Orion Corporation Orion Pharma, Finland) to ensure a smooth recovery.

Postoperatively, the dog was hospitalized for 23 hours in the intensive care unit for monitoring. The dog received Ringer-lactate (2.84 mL/kg/h IV, maintenance + 100% correction) fluid therapy. To keep the dog sedated, acepromazine (20 µg/kg IV, Placivet®, Kela N.V., Hoogstraten, Belgium) was given three times postoperatively. Amoxicillin/clavulanic acid (0.2 mg/kg IV, q8h) was administered and omeprazole (2.5 mg/kg IV, q12h, Losec® AstraZeneca SA/NV, Brussels, Belgium) was initiated. Analgesia consisted of methadone (0.2 mg/kg IV, q4h, Comfortan, Dechra Veterinary Products, Belgium) for the first 19 hours. Afterwards, the dose was reduced to 0.1 mg/kg, q4h, until discharge. Dexamethasone (0.25 mg/kg IV, q8h) was repeated once. The dog was regularly turned from side to side. An inspiratory stridor was observed once at 3:00 during the night. The patient was fasted for the first 12 hours, followed by small portions of food and water. The dog was discharged one day postsurgery, as soon as he was able to eat and drink without coughing and showed a constant body temperature within normal ranges. He was discharged from the clinic on oral antibiotics (11.8 mg/kg, sid, amoxicillin/clavulanic acid, Synulox 500 mg, Zoetis BV, Louvain-la-Neuve, Belgium) and omeprazole (0.95 mg/kg, sid, Omeprazole Sedacid®, 20 mg, Laboratoires SMB N.V., Brussels, Belgium) for ten days. A prescription was given for tramadol hydrochloride (1.8 mg/kg, tid, Tramadol EG®, 50 mg, Eurogenerics NV, Brussels, Belgium) for five days and prednisolone (0.25 mg/kg, bid, Prednisolon 20 mg, Kela, Hoogstraten, Belgium) for five days, followed by 0.25 mg/kg, sid, for another five days. The owners were informed about the basics of wound care and the possible complications. They were advised to schedule a control appointment after one month.

DISCUSSION

In the present case, a heat stroke caused by the inability to remove heat from the body, due to a laryngeal paralysis as a clinical manifestation of generalized peripheral polyneuropathy is described. Large- or giant-breed dogs, like the Landseer, are predisposed to develop geriatric onset laryngeal paralysis polyneuropathy (Rudorf et al., 2001; Kitshoff et al., 2013). The disease is more common in middle-aged to older male dogs (Dixon and Pratschke, 2004; Kitshoff et al., 2013). Airway subobstruction results in a reduced capacity to release heat by panting (Gough, 2008). Tachypnea, dyspnea, an abundant haircoat, body weight (84.5 kg) and BCS (6/9) are additional endogenous predisposing factors affecting heat dissipation (Johnson et al., 2006; Hemmelgarn and Gannon, 2013b). Predisposing factors should be eliminated to prevent subsequent episodes of heat stroke (Flournoy et al., 2003; Mazzaferro, 2009).

The hyperthermia in this patient was most likely caused by the inability to dissipate heat, but other causes of increased body temperatures, such as fever, pain or stress, should also be taken into consideration (Gough, 2008). In general, respiratory problems can originate from the respiratory system or have non-respiratory causes, such as central nerve system diseases, heat stroke and anemia. In the differential diagnosis of inspiratory stridor, all causes of laryngeal and pharyngeal obstruction should be taken into consideration. The simultaneous occurrence of an inspiratory and expiratory stridor with dyspnea is very suggestive for laryngeal disease, while inspiratory dyspnea with stridor along with exercise intolerance, dysphonia and coughing point in the direction of laryngeal paralysis (Koufman and Block, 2008; Kitshoff et al., 2013). Because the primary complaints of this dog were mainly of a chronic nature, a mass, polyp or neoplastic process are also possible causes for airway obstruction (White, 2002; Gough, 2008; Kitshoff et al., 2013). Expiratory stridor is mainly caused by an intrathoracic obstructive process of the lower airways. However, asthma, chronic obstructive pulmonary disease and thoracic neoplasms were less likely in the present case due to the absence of abnormal lung sounds (Gough, 2008).

On presentation, extended clinical, neurological, blood and urine examinations should be performed for further diagnosis and detection of complications due to heat stroke (Hemmelgarn and Gannon, 2013a). Based on the preanesthetic examination, a decision was made towards the anesthesia protocol and further patient work-up. The patient of the present case was attributed an ASA score V E related to aggravating dyspnea, cyanosis and hyperthermia. Since the blood work was normal, the dog was most likely in an early state of heat stroke without multiple organ failure, which however should be treated with immediate attention (Mazzaferro, 2009). As an urgent surgical intervention was desirable, immediate induction of an-

esthesia with low-dose propofol IV (2.1 mg/kg) was performed followed by endotracheal intubation for securing airways, supplying oxygen and maintaining anesthesia by inhalation (Mazzaferro, 2009). Average propofol dosages for non-premedicated dogs range between 4-8 mg/kg. The deteriorating general condition of the dog emphasizes the importance of dose-titration of the induction agent to the desired clinical effect (Hofmeister et al., 2009). In the meantime, a quick intervention by applying cooling methods, such as using fans, humidifying the patient with cold water and the use of presoaked towels, was started. The primary goal of treatment was to control hyperthermia, to decrease the body temperature within a normal range (38 – 39.2°C) and to prevent further damage to vital organs.

When initiating anesthesia, the patient showed hyperemic mucosae, a strong pulse, a very pronounced tachycardia (229 bpm) and a CRT of less than one second, which suggested an early stage of compensatory shock (Mittleman Boller and Otto, 2009). Immediate cardiovascular support was provided by a shock bolus of Ringer-lactate (Flournoy et al., 2003; Mazzaferro, 2009).

During maintenance of anesthesia, the dog showed a MAP ranging from 56 to 77 mmHg. A sudden drop in systemic arterial blood pressure induces a baroreceptor-mediated reflex, which results in vasoconstriction and an increased heart rate and cardiac contractility, a short-term solution to normalize the systemic blood pressure (de Laforcade, 2017). As such, the cardiovascular system plays a crucial role in early states of heat stress by increasing the cardiac output, along with central vasoconstriction and peripheral vasodilatation. Failure of these compensatory mechanism lead to a decrease of systemic vascular resistance resulting in distributive shock (Hemmelgarn and Gannon, 2013b), further stressing the importance of cardiovascular support by fluid therapy in case of heat stroke (Flournoy et al., 2003; Mazzaferro, 2009). During the procedure, HR decreased gradually, ranging between 75-230 bpm. The pulse quality evolved from a strong to moderate beaten pulse and normalized 55 minutes after shock infusion therapy. One and a half hour after initiating inhalation anesthesia, MAP dropped again to 56 mmHg. A plasma-expander infusion was given, resulting in clinically acceptable MAP values (60-80 mmHg) throughout the rest of the procedure. Halfway the procedure, propofol was administered because the dog tended to wake up.

Managing heat-stroke patients can be really challenging. The type of ventilation during inhalation anesthesia has an influence on the body temperature. While preparing the patient for surgery, the patient was temporarily connected to a non-rebreathing Bain system and ventilated manually. The Bain circuit is an economic and ecologic unfavorable anesthetic breathing system, specifically for giant breeds due to the high fresh gas flow (200 ml/kg/min). However, the high gas flow helps to decrease the body tempera-

ture of the patient rendering a non-rebreathing system related inconvenience into an advantage in case of hyperthermia (Johnson, 2009). From the moment the patient reached a normal body temperature, it was switched to a rebreathing circle system (2 L/min). A disadvantage of using a rebreathing system in patients with hyperthermia is the risk of heat production by the CO₂ absorbent granules, which could lead to an increase in body temperature. In addition, body heat of exhaled air is also recycled with this system and for this reason undesirable (Johnson, 2009).

Because heat stroke is a life-threatening condition, it is crucial that these patients are strictly monitored for the next 24-48 hours after admission (Flourney et al., 2003). To reduce the postoperative risk of developing recurrent upper airway obstruction, dexamethason was given at the end of anesthesia for its anti-inflammatory properties. Immediate postoperative sedation was achieved with a microdose of dexmedetomidine (1 µg/kg IV). During the surgical procedure, a broad-spectrum antibiotic (amoxicillin/clavulanic acid) was administered IV and was continued in the postoperative period. Antibiotic therapy is strongly advised in patients with heat stroke, because these patients are more likely to develop septicemia due to bacterial translocation, which may lead to multiple organ failure (Mazzaferro, 2009).

During hospitalization, the patient received Ringer-lactate infusion (240 ml/h IV) to cover for expected losses due to hyperventilation. Methadone was subscribed for pain control and if needed, the patient could be sedated with acepromazine. Buprenorphine might have been a good alternative for postoperative analgesia, since it does not cause panting due to re-setting of the thermoregulatory center as methadone does. However, buprenorphine is far more expensive and due to the dog's body weight, methadone was used to reduce the costs. The dog went home with a subscription to continue oral antibiotic treatment (amoxicilline/clavulanic acid) and omeprazole, to prevent damage to the stomach. Pain medication and prednisolone (gradually reduced in dosage) were also subscribed to decrease the swelling and to make to dog more comfortable. Finally, the owners were informed about the risk of relapse of heat stroke; ideally, management- and weight loss measures should be taken to decrease these risks. Unfortunately, the patient was lost for follow-up since the owners did not come back for a control visit.

CONCLUSION

Heat stroke is a life-threatening disease that should be treated immediately when recognized. Active cooling and fluid therapy should be an essential part of the (pre)anesthetic protocol and should be performed as soon as possible to prevent further damage and multiple organ failure.

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