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CRITICAL CARE

Treating Environmental Lung Injuries: Drowning and Smoke Inhalation

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Environmental lung injury can result from several causes, including drowning or smoke exposure from enclosed-space fires. Management of patients with environmental lung injuries differs from management of patients with the many other types of respiratory compromise. This article describes treatment of lung injury caused by drowning and smoke inhalation.

DROWNING

We will first discuss the definition of drowning, which has undergone modifications over time. Drowning is currently defined as respiratory impairment resulting from submersion or immersion in liquid. In the current literature, the term "nonfatal drowning" has replaced "near-drowning."[1] Also, the terms "wet" and "dry" drowning have been abandoned. Forensic science has shown that most victims previously thought to have "dry" drowned were probably apneic before entering the water, and "dry" lungs are now believed to occur in only <2% of drowning victims.[2]

Dogs and cats drown for several reasons: falls into bodies of water from which they cannot escape,[3,4] accidents while swimming,[3] seizures or other physical conditions that impair swimming,[3] or intentional submersion by a person.[3,5] Drowning accounts for a small percentage of dogs and cats presented for respiratory distress, and the literature with regard to its treatment in small animals is scarce. Thus, most recommendations for management of dogs and cats that have drowned are extrapolated from the literature about humans. However, some of the considerations for management of animals with respiratory compromise resulting from drowning are unique to animals.

Pathophysiology

When an animal experiences drowning, respiratory impairment results from aspiration of water, which causes surfactant dilution and dysfunction, bronchoconstriction, increased permeability of the alveolar–capillary membrane, local inflammation and edema, and lung unit

RESPIRATORY DISTRESS
Management of a dog with an environmental lung injury—e.g., due to drowning—differs from the care given for other types of respiratory compromise.

BOX 1 Management of Drowning Patients

- Provide oxygen therapy
- Dry animal thoroughly
- Treat hypovolemia with appropriate fluid resuscitation
- Treat hypothermia
- Take baseline thoracic radiographs
- If the stomach contains fluid, consider placing nasogastric tube to remove gastric contents
- Provide supportive care
  - Fluid therapy
  - Warmth
  - Possibly bronchodilators
- Complete systems review to investigate for neurologic or cardiac abnormalities that may have predisposed the patient to drowning, if appropriate
- Look for evidence of pneumonia
  - Monitor vital signs
  - Evaluate complete blood counts
  - Perform repeat thoracic radiographs if pneumonia is suspected (fever, leukocytosis)

collapse.[6] Alveolar collapse decreases lung compliance and increases the work required for breathing while the repetitive open-to-collapse-to-open cycling of the delicate respiratory epithelial layer incites further pulmonary damage and inflammation. Clinical severity is associated with the volume of water inhaled, regardless of water type (salt, fresh, or chlorinated).

Presentation and Physical Examination

Most cases of small animal drowning occur during warm weather.[3] However, drowning should be considered for any animal that has been rescued from water. Examination may reveal abnormalities in body temperature, heart rate, respiratory rate or effort, and neurologic status. Patients in obvious respiratory distress should be provided oxygen as soon as possible.

Diagnostic Testing

Determining the severity of respiratory compromise involves physical examination, pulse oximetry, and arterial blood gas analysis. Contrary to findings in experimental canine drowning models, after spontaneous drowning most people and dogs do not seem to have clinically important electrolyte abnormalities.[3,7,8] This discrepancy is probably related to the relatively small volume of liquid aspirated in drowning victims brought to the hospital alive. For animals that have drowned, results of complete blood count, serum biochemistry panel, and urinalysis are usually nonspecific. However, because animals with unrelated disease processes seem to be at increased risk of drowning,[3] a complete systems review is appropriate, particularly when the cause of drowning is not obvious.

The appearance of initial chest radiographs of a drowned patient is highly variable. Serial chest radiographs can be used to monitor for later-developing infiltrates, which may indicate bacterial pneumonia (**FIGURE 1**) or development of the pulmonary form of acute respiratory distress syndrome. If fever or leukocyte changes develop, new chest radiographs are indicated. If pneumonia is suspected, airway sampling by tracheal wash or bronchoalveolar lavage should ideally be performed before empirically initiating antimicrobial therapy while awaiting culture results. In people, aspiration of swimming pool water does not usually cause bacterial pneumonia, although polluted or contaminated water seems more likely to lead to infection.[6]

Treatment

Treatment for drowning is similar to treatment for respiratory distress from other causes of pulmonary

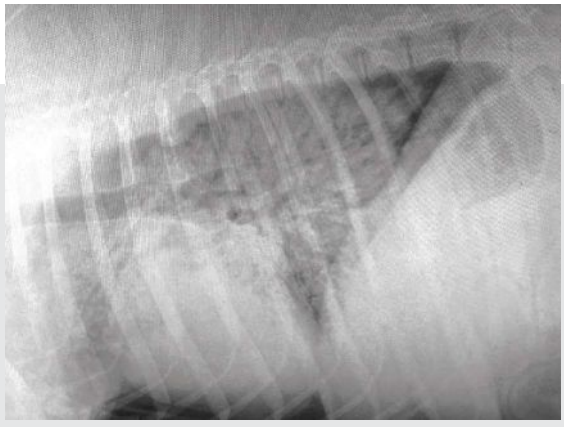

FIGURE 1. Right lateral thoracic radiograph of a mature Labrador retriever that developed severe bacterial pneumonia after non-fatal drowning in a contaminated lake. The dog responded well to aggressive oxygen therapy and broad-spectrum antimicrobials and was discharged alive from the hospital.

disease (**BOX 1**). Oxygen therapy is required for any animal with respiratory difficulty and should be provided as soon as possible. Supportive measures should also be instituted to treat hypothermia, shock, and other concomitant problems. Drowning victims may have swallowed large volumes of water, which can cause electrolyte abnormalities and predispose them to emesis with aspiration.[9] Therefore, if the patient's respiratory status allows, consider passing a nasogastric tube to remove water from the stomach. Prophylactic administration of antimicrobials is not recommended because drowning is not usually associated with bacterial pneumonia and because if pneumonia should occur, prophylactically administered antimicrobials can select for resistant organisms.[6] Glucocorticoids are not routinely recommended for drowning victims.[6] Bronchodilators may be considered. Patients with severe hypoxemia or severely increased respiratory effort require mechanical ventilation. For human drowning victims, a lung-protective strategy with low tidal volumes is generally recommended.[6]

Prognosis

The prognosis for drowning victims who survive the initial event and associated hospitalization is good. For human patients who have experienced prolonged hypoxemia associated with a drowning event, long-term neurologic consequences are possible; however, for veterinary patients, such outcomes have not yet been reported.

SMOKE EXPOSURE

Companion animals are most commonly exposed to clinically meaningful amounts of smoke during enclosed-space fires. With regard to their exposure to wildfires, information about injury patterns is scant; however, available information suggests that many dogs and cats rescued from wildfire or outdoor fire conditions have dermal burns without meaningful respiratory compromise.[10,11] Those with respiratory compromise caused by an outdoor fire usually have burns,[11] whereas many animals rescued alive from enclosed-space fires may have respiratory and neurologic compromise without extensive dermal burns.[12-16] For individuals with respiratory compromise caused by smoke inhalation, the clinical course is complicated and the prognosis is worsened by the presence of dermal burns.[17]

The combination of toxic gas, inhaled particulate matter, and thermal airway injury leads to a unique pathophysiologic circumstance for animals after smoke inhalation.

Pathophysiology

The combination of toxic gas, inhaled particulate matter, and thermal airway injury leads to a unique pathophysiologic circumstance for animals after smoke inhalation. Smoke contains the metabolically toxic gas carbon monoxide (CO) and may also contain hydrogen cyanide and other toxic gases, depending on the materials being burned. Smoke also contains particulate matter that may be inhaled and can be difficult to clear, particularly when upper and lower airways are thermally burned.

Carbon Monoxide

CO is ubiquitously present in fire smoke. Although CO is not irritating to the airways, it causes problems by competing with oxygen for hemoglobin binding sites. The affinity of CO for the hemoglobin molecule is approximately 250 times that of oxygen; thus, even short-term inhalation of only 0.05% to 0.1% of CO gas can be lethal.[18] CO also shifts the oxyhemoglobin equilibrium curve to the left (i.e., increases hemoglobin's affinity for oxygen), thus impeding oxygen release from hemoglobin to the tissues. This combination of toxicologic effects leads to tissue hypoxia by decreasing oxygen availability to tissues.

Some of the clinical signs associated with CO toxicosis cannot be readily explained by simple tissue hypoxia. For instance, many of the neurologic sequelae of CO toxicosis differ from those seen in animals recovering from cardiopulmonary arrest, which also causes cerebral hypoxia. CO affects cellular metabolism and function in many ways, which has led to "cellular" theories regarding these nonhypoxic mechanisms of CO neurotoxicosis.[19] CO molecules may affect mitochondria, cellular enzymes, leukocyte and platelet function, and neuronal signaling.[19] These effects help

explain some of the complexities of the clinical signs of smoke inhalation.

Hydrogen Cyanide

The presence of cyanide gas in enclosed-space fire smoke varies according to what is burned. Cyanide is produced by the burning of certain fabrics, synthetic materials, and paper products. Cyanide is another element that is not irritating to the airways. It is, however, a metabolic toxin that inhibits mitochondrial function and thus leads to poor cellular energy production even when delivery of oxygen to cells is adequate.

Particulate Matter

Smoke contains many particulates. Smaller particles cause lower airway inflammation and edema, and clearance can be impaired because of lower airway thermal injury and secondary mucociliary escalator dysfunction. Larger particulates can become lodged in bronchi and lead to lung lobe collapse.[11] Inhalation of these particles is not generally associated with bacterial pneumonia; however, secondary pneumonia can develop in patients that are managed with an artificial airway (e.g., an endotracheal or tracheostomy tube) because of the presence of the device and circumvention of upper airway defenses against infection.[20]

Thermal Injury

Most airway burns occur in the upper respiratory tract because the upper airway efficiently dissipates the heat, which limits thermal injury to the lower airways. Inflammation and edema of the upper airways can lead to upper airway obstruction. However, superheated particulates can be inhaled deeply and cause severe lower airway burns. Thermal injury to the lower airways causes erosions, inflammation, bronchoconstriction, edema, and hyaline membrane formation. These changes impede mucociliary escalator function, decrease pulmonary compliance, and in severe cases can cause lower airway and alveolar collapse. These problems increase the work required for breathing and cause hypoxemia.

Presentation and Physical Examination

The history for patients exposed to smoke is generally straightforward in that clients are usually aware that the animal has been exposed to smoke or fire; however, clinical signs may or may not have been present at the fire scene. Because early oxygen therapy is vital and not all clinical signs may be immediately obvious, all animals exposed to smoke, even those that seemed unaffected at the fire scene, should receive supplemental oxygen as soon as possible and be evaluated by a veterinarian. When clinical signs are expressed at the fire scene, they can include coughing, gagging, ptyalism, and overt respiratory distress; neurologic signs (e.g., agitation, ataxia, obtundation, recumbency, seizure, or coma); ocular and upper respiratory signs (e.g., apparent pain and discharge); and dermal burns.[12,13]

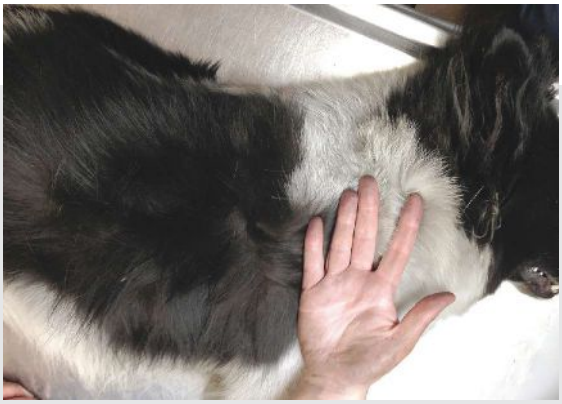

FIGURE 2. A dog presented after being trapped in a house fire. Note the soot on the dog's coat and the rescuer's hand.

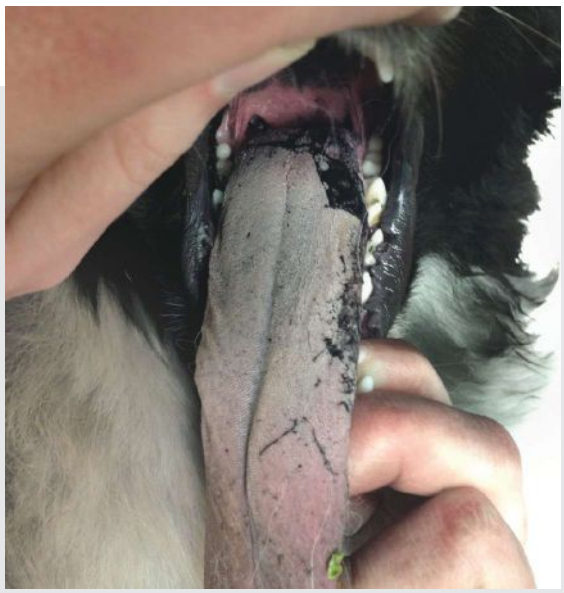

FIGURE 3. Soot in the saliva and on the oral tissues after being trapped in a house fire. When an animal is exposed to a fire but inhalation of smoke is uncertain, examination of the oral cavity or respiratory secretions can help confirm exposure.

Most animals that have been exposed to smoke smell of smoke and/or have soot on their coats (**FIGURE 2**). However, for some animals, smoke inhalation can be difficult to determine, and clues such as soot on the tongue or in saliva can help confirm smoke inhalation (**FIGURE 3**). Initial physical examination findings can vary widely according to the severity and length of exposure. Animals exposed to smoke are often dehydrated. The main systems involved are usually neurologic, respiratory, ophthalmic, and dermal. The most common acute neurologic signs are mentation changes, ataxia, extensor rigidity, and seizures.[12-16,21] Mucous membrane color is rarely the cherry red that is supposedly classic for CO or cyanide toxicosis.[12-14,16] Not all animals are tachypneic,[12,13] and increased respiratory effort may be more clinically relevant than increased respiratory rate.[15] Smoke exposure often leads to ophthalmic injuries, such as corneal ulceration, blepharospasm, and poor tear production.[10,12]

Neurologic signs resulting from smoke inhalation include blindness, deafness, paresis, ataxia, mentation changes, and seizures.[14,15,19] However, CO toxicosis can cause delayed neurologic signs, which can appear days after exposure and in animals that did not have obvious neurologic dysfunction at the time of presentation. These delayed neurologic signs are believed to be secondary to the cellular toxicologic effects of CO. Magnetic resonance images of 1 dog with delayed neurologic signs after smoke inhalation did not show evidence of cerebral anoxic insult, which may suggest that nonhypoxic mechanisms were the more likely cause of the neurologic signs.[15] For 2 chihuahuas caught in a house fire and in which seizures occurred 2 to 3 days later, necropsy results revealed myelin and neuronal changes, which are consistent with changes found in people with acute CO toxicosis.[21]

Diagnostic Testing

For smoke-inhalation patients, the results of complete blood count, serum biochemistry panel, and urinalysis are generally nonspecific. Unfortunately, standard pulse oximetry may not be useful because it does not distinguish dysfunctional carboxyhemoglobin from healthy oxyhemoglobin. In other words, a pulse oximeter will read artificially high in animals with CO toxicosis or falsely normal in hypoxemic animals. Arterial blood gas measurement will reveal hypoxemia (low partial pressure of oxygen [PaO_2]) for animals with concurrent lower airway or pulmonary insult secondary to smoke inhalation but is not helpful in the evaluation of CO toxicosis because CO does not affect lung function or PaO_2. Co-oximetry, which measures the percentages of various forms of hemoglobin in relation to total hemoglobin, can be used to confirm smoke inhalation and to monitor response to treatment. However, although co-oximetry can quantify carboxyhemoglobin, the level of carboxyhemoglobin does not predict severity of clinical signs or prognosis.[15] In addition, co-oximetry is rarely performed in clinical veterinary practice. Cyanide toxicosis leads to increased venous oxygen tension (PvO_2) because of decreased oxygen diffusion into cells at the tissue level and is indicated by a decreased gradient between PaO_2 and PvO_2.

Thoracic radiographic findings after smoke inhalation are variable. In one study of dogs exposed to smoke, radiographs showed patchy, asymmetrical alveolar infiltrates in various lung regions,[12] whereas in another study of cats exposed to smoke, radiographs tended to show diffuse bronchointerstitial patterns with various alveolar patterns.[13] Thoracic radiographs of smoke-exposure patients may be nonremarkable, but that finding does not rule out inhalation of CO or cyanide because these gases are not irritating to airways.

BOX 2 Management of Smoke-Inhalation Patients

- Provide aggressive oxygen therapy as soon as possible. Consider intubation and delivery of 100% oxygen
- Provide fluid resuscitation, if indicated
- Closely monitor respiratory rate and effort
- Consider arterial blood gas measurement (Note that pulse oximetry is unreliable because of CO exposure)
- Take thoracic radiographs
- Provide supportive care
  - Provide saline nebulization and coupage
  - Rehydrate with fluid therapy
  - Consider bronchodilators
- Manage burns
- Provide ophthalmic care
  - Stain corneas with fluorescein
  - Perform Schirmer tear test
  - Treat accordingly with ophthalmic medications
- Monitor for onset of delayed neurologic signs up to 6 days or more following the event

Neurologic signs resulting from smoke inhalation include blindness, deafness, paresis, ataxia, mentation changes, and seizures.[14,15,19]

Patients in which respiratory signs worsen after initial improvement or fever or leukocytosis develop after initial evaluation may have bacterial pneumonia. Diagnosis of pneumonia is based on clinical suspicion, thoracic radiographs, and cytology and culture results of airway samples obtained by endotracheal wash or bronchoalveolar lavage.

Treatment

The cornerstone of management of all smoke-inhalation patients is oxygen therapy, which should be instituted as soon as possible (**BOX 2**). A high fraction of inspired oxygen helps displace CO molecules from hemoglobin so they can be eliminated and replaced by oxygen, thereby restoring oxygen delivery to the tissues. Because oxygen displaces CO from hemoglobin, it is the best treatment for the hypoxic injury caused by CO toxicosis and is the only treatment readily available to combat the toxic effects of CO on cells. A study of human smoke-inhalation patients showed that the mean half-life of carboxyhemoglobin was decreased by almost half (from approximately 130 minutes to 74 minutes) for those who received 100% oxygen inspired at atmospheric pressure compared with those who received less intensive oxygen therapy.[22] Our veterinary patients should thus receive the *highest percentage of oxygen that is reasonable for the patient*, and the best approach may be to induce general anesthesia, intubate, and deliver 100% oxygen. Although hyperbaric oxygen therapy should theoretically be superior for treatment of CO toxicosis, its use in people has shown mixed results and, as such, it is not widely used to treat CO toxicosis.[23] When available, co-oximetry can be used to monitor effectiveness of oxygen therapy for elimination of CO.

To circumvent upper airway obstruction resulting from laryngeal burns and subsequent swelling, you may need to perform a temporary tracheostomy. To treat severe hypoxemia or excessive work of breathing resulting from severely inflamed, noncompliant lungs, you may need to administer intermittent positive pressure ventilation.

Patients should receive appropriate supportive care, and burns should be managed. Routine use of glucocorticoids or prophylactic antimicrobials is not recommended. Saline nebulization is appropriate, and gentle coupage may be considered for animals that have pulmonary infiltrates but are not actively coughing. Patients should be monitored closely for development of neurologic signs and supported appropriately if they occur.

Prognosis

The amount of information about prognosis for smoke-inhalation patients is limited. However, the literature that is available suggests that the prognosis for dogs and cats that improve by the day after presentation without development of delayed neurologic signs is good[12,13] and that those that survive the acute onset of delayed neurologic signs usually recover.[14-16] The prognosis for smoke-inhalation patients with moderate to severe dermal burns is poorer than that for those without burns; the difference may be attributed to the high fluid requirements of burn victims and the likelihood of secondary infection. Regardless of burn status, some survivors may experience poor tear production[10,12] and/or have long-lasting neurologic signs.[12] TVP

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