

Fire and Smoke Inhalation Injury in Horses

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Exposure to fire and smoke can cause devastating traumatic injury. In general, providing medical management of this type of injury is not common for the equine veterinarian. Much of the information about such types of injury has been generated by research related to human injury. Although the incidence of burn injuries in people is on the decline in the United State, there are still 1 to 2 million people treated for burns annually [1–3]. Most of the injuries are minor; however, 20,000 to 50,000 people require hospital care. The most severely affected patients are treated in specialized burn units, wherein the fatality rate is approximately 4% [1,3]. Although trauma from fires, with or without concurrent smoke inhalation injury, in horses is comparatively quite low (with no information reported on the incidence), when it does occur, the results are typically tragic. Sporadic reports of barn fires most commonly note high mortality, with few, if any, horses removed alive from burning buildings. Although large wildfires make national news, there is limited information on the number of horses affected and the types of injuries that occur.

The injuries that horses incur after being exposed to fire or smoke can range from mild to severe, with multiple body systems involved. Thermal injury to the surface produces one of the most prominent types of lesions affecting these victims, and in human medicine, estimation of the extent and depth of burn injury is an important aspect in the initial management [3]. Classification systems based on the depth of injury are routinely used in human medicine and have been extrapolated to horses [4,5].

Respiratory tract lesions are also common in these types of patients and can require emergency therapy. In addition to these more familiar types of lesions, fire and smoke trauma can produce numerous physiologic responses,

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and depending on the severity, the systemic response can be life threatening. Significant systemic involvement is termed *burn shock*, and one of the most striking features is the development of severe edema. The wide variety of possible body systems that can be involved can make these cases challenging to manage medically.

The role of an equine emergency veterinarian in these situations can be varied, ranging from being part of the first-response team, to providing care to critically ill individuals, to helping clients be as prepared as possible for such types of disasters. The goal of this article is to provide a basic review on the pathophysiology of fire and smoke injury as well as to discuss diagnostic and therapeutic goals to aid the practitioner when he or she is faced with such a potentially overwhelming and emotionally charged situation.

First response and disaster preparedness

The two most common ways for horses to receive fire and smoke injury are by being trapped within a burning barn or being in the path of a wildfire. Expected types of injuries vary in each of these situations. In general, barn fires occur with no warning; depending on the material of the barn, the fire can consume the building rapidly, with only several minutes to move animals out. If one is part of the first-response team, it is imperative to follow all instructions of fire control personnel. Entering a burning building is highly dangerous, and it is difficult to predict how an animal is going to react in such situations. Disaster planning is becoming more widespread, and a search of the Internet yields several horse protection groups conducting training sessions specific to removing horses from a burning barn. Practices seen in movies, such as covering the horse's eyes, may not work and may cause more panic in some situations.

As part of the first-response team, a veterinarian may be most productive by focusing attention on the animals removed from the fire. At first, these animals may not outwardly show the extent of their injuries, dermally or systemically. Initially, the goal should be to stop the burning processes [6]. Remove any blankets or wraps that can continue to hold heat next to the animal. Cooling of the patient to limit thermal injury could be considered within the first few minutes with the use of lukewarm water [6]. Causing vasoconstriction of poorly perfused hypoxic tissue by using ice or extremely cold water is contraindicated, however [7]. Primary treatment should be limited to that needed to stabilize the patient and, most commonly, would include sedation to minimize excitement and anti-inflammatory drugs. Also, if feasible, securing intravenous access might be considered during this initial evaluation. Substantial edema can occur within hours, making venous access difficult. After a complete physical examination, movement of an affected horse to a location where further diagnostic evaluation and

intensive treatment can occur is recommended. Because exposure to hypoxic conditions has most likely occurred, availability of oxygen therapy is ideal.

There is little to no information specific to the types of injuries that horses incur when they are in the path of a wildfire. In general, smoke inhalation injury would be expected to be less common, and in addition to thermal injury to the body surface, cuts and contusions might be seen in animals left to fend for themselves. Moving across hot surfaces may also cause thermal damage to the solar surface of hooves. Although wildfires can move rapidly under the right conditions, there is often time for evacuation. Helping clients who live in high-risk areas to create a rational plan, including helping them to develop a transport plan with information on safe holding locations as well as on providing permanent identification, would be useful. Veterinarians who practice in high-risk areas often play a vital role in community disaster planning activities.

Pathophysiology of fire injury

The type and extent of injury depend on factors related to the fire as well as on the duration of exposure and location of the victim relative to the point of origin of the fire. Thermal injury causes a local response that includes microvascular insult and direct tissue coagulation, leading to local inflammation, edema, and, finally, necrosis. Extensive local injuries drive a systemic response. The initial systemic response is attributable, in part, to loss of the skin barrier as well as to release of multiple mediators that can initiate the inflammatory and coagulation cascades [2]. The effects of hypoxia and subsequent reperfusion injury on various organs and tissue beds can complicate recovery from severe thermal trauma [8].

Initially, there is a decrease in cardiac output; however, in focally damaged areas, there is an increase in capillary pressures. These characteristics, which are typical in early shock, represent the initial phase of burn shock. Directly after these changes, there is formation of generalized edema. The pathophysiology of burn edema is complex and involves endothelial damage, protein shifts, and alteration of the interstitial architecture. All these factors lead to a net accumulation of fluid in the interstitial spaces [9]. In addition to the changes incited by direct thermal injury, release of various mediators plays an important role in edema formation. Some of the most prominent mediators include neutrophils, which are a known source of oxidants, as well as prostaglandins, bradykinins, and histamines. Oxidants are produced as a result of focal tissue hypoxia, followed by reperfusion. The results of reperfusion injury cause endothelial cell damage as well as denaturation and fragmentation of interstitial matrix components [9]. Prostacyclin and thromboxane are reported to be detected in burn edema [9]. Although they have opposite effects (prostacyclin is a vasodilator, and thromboxane has vasoconstrictive activities), when expressed in locally

damaged tissues, both contribute to cardiovascular instability and edema formation. Bradykinins and histamines are known to increase vascular permeability. Edema develops not only in tissue directly affected by thermal injury but is seen at distant sites when the burn size exceeds approximately 20% of the body surface [2].

In the long run, high cardiac output, insulin resistance, and increased oxygen consumption with protein and fat wasting, all of which may create a hypermetabolic state, characterize major thermal injury [2,10,11]. Endogenous catecholamines have been implicated as the initiating mediators of this hypermetabolic response [12]. Loss of skin as a barrier, release of inflammatory mediators, and hypermetabolism play a role in the development of immunosuppression. Systemic changes may lead to gastrointestinal tract dysfunction, and disruption of this system can lead to bacterial translocation, increasing the risk of significant infections [13]. Other features noted with burn injury in the horse include anemia secondary to acute hemolysis, renal failure, laminitis, and myositis [14].

Pathophysiology of inhalation injury

The combination of thermal injury with smoke inhalation is known to result in increased morbidity and mortality in human patients [9,15,16]. In closed burning buildings, victims close to the point of origin are more likely to be overcome by direct thermal injury, whereas others at some distance may incur injuries as a result of smoke inhalation [17]. There are infrequent reports of horse with primary smoke inhalation injury [18,19]. The pathophysiology of smoke inhalation injury is complex and multifactorial. Insult to the respiratory system by smoke inhalation depends on the fuels that burned, completeness of combustion, and generated heat intensity.

There are three primary mechanisms of injury: direct thermal effects, toxic gas effects, and hypoxia. Similar to thermal injury of the skin, edema formation is a key component of the initial respiratory injury. Direct thermal injury to the respiratory tract can be limited to the upper respiratory tract by laryngeal reflexes and efficient heat exchange within the nasal passages. Toxic chemicals in the smoke can cause damage, directly and indirectly, through inflammatory mediators. Carbon monoxide intoxication is commonly associated with human injuries from smoke and is a product of incomplete combustion [20]. Newer synthetic building materials contain other potential toxins, and forensic fire investigations have revealed that hydrogen cyanide is likely to be present in appreciable amounts in the blood of fire victims [17]. Finally, with combustion, there is consumption of oxygen, and the resulting low P_{aO_2} can lead to pulmonary vasoconstriction as well as generalized hypoxia.

Three phases of pulmonary dysfunction have been described in the horse [4,18]. The first stage is acute pulmonary insufficiency caused by several

mechanisms. Carbon monoxide may be present in a sufficiently high concentration to cause toxicity within a short time after exposure. Carbon monoxide combines with hemoglobin to form carboxyhemoglobin, which reduces the circulating oxygen-carrying capacity. Hemoglobin has a 200 to 250 times greater affinity for carbon monoxide as compared with its affinity for oxygen [20]. High levels of circulating carboxyhemoglobin result in a shift of the oxyhemoglobin dissociation curve to the left, thereby decreasing oxygen release at the tissue level and leading to tissue hypoxia. Carbon monoxide can also combine with myoglobin, leading to impaired diffusion of oxygen to muscles [17]. Other processes occurring during this acute phase include progressive edema and necrosis in the upper respiratory tract leading to airway obstruction, bronchoconstriction in the lower respiratory tract from the irritating effects of noxious products, and altered pulmonary blood flow [15,16].

These insults produce the second stage, which includes the formation of pulmonary edema, lower airway obstruction, and pulmonary parenchymal lesions. Within 24 to 72 hours after exposure, driven by pulmonary macrophages, neutrophils are called into the area of insult. They release cytokines, proteolytic enzymes, and oxygen-derived free radicals. Expression of the inflammatory cascade in excess of balance causes microvascular damage, leading to increased extravascular lung water. Local insult also results in the release of tissue factor initiating the coagulation cascade to produce fibrin. Debris from the inflammatory cascade, along with fibrin and material directly deposited from smoke inhalation, creates pseudomembranous casts, which may obstruct the small airways. Widespread plugging of the airways may significantly increase airway pressure, causing barotrauma and alveolar damage [15]. Bronchopneumonia is the last stage and occurs as a result of the impaired host immune system, locally and systemically. This phase, if it occurs, may take up to 1 to 2 weeks after the initial injury to become clinically apparent.

Clinical signs

Horses exposed to fire with smoke have a variety of clinical signs depending on the duration and type of exposure and the length of time from the insult. Initially, the extent of damage to the skin may be difficult to ascertain. Horses rescued from burning barns most commonly incur thermal injury of the head and dorsum. At first, the damage may not be clinically visible or may show as singed areas with a leather-type appearance (Fig. 1). Over the first 24 to 48 hours, surfaces affected by direct thermal injury often become edematous. The degree of local edema depends, in part, on the elasticity of the skin and surrounding tissues. Typically, this is limited along the dorsum of the horse, and the extent of injury may not be fully realized for days to weeks (Figs. 2 and 3). Blepharospasm and epiphora are commonly present and often indicate corneal damage (Fig. 4).



Fig. 1. Initial thermal injury along the back of a horse rescued from a barn fire. Note the smaller focal singed areas as well as the generalized leather-like appearance with hair loss across the back.

Depending on the depth of surface injury, sensation may be altered to these areas. In general, more superficial burn injuries cause more pain than deeper injuries, in which permanent damage to cutaneous sensation can occur. Altered sensation or pruritus associated with healing lesions can cause some horse to rub and self-traumatize damaged areas. A complication of successful long-term outcome in human injury includes the formation of restrictive scars. There are limited data on long-term outcomes in horses; however, extensive scar formation along the dorsum of an equine patient would certainly affect future use.

Acutely, within the first 6 hours, clinical signs of shock may be noted and include tachycardia, tachypnea, and altered mucous membrane color. It is during this period that the effect, if any, of carbon monoxide toxicosis may become clinically apparent. The patient may show signs of severe hypoxemia, which is evident by depression, disorientation, irritability, ataxia, or even a moribund to comatose status. As edema and necrosis progress in the upper respiratory tract, dyspnea and stridor may develop. Auscultation of the thorax may reveal decreased air movement, crackles, or wheezes, but these changes may not become apparent for 12 to 24 hours. If edema of the airways is sufficiently severe, air flow may be severely restricted. Edema fluid may be visible at the nostrils and, later, may be replaced by inflammatory exudate. During this same period, concurrent generalized edema may be forming.

Severely affected patients that are successfully resuscitated may start to show clinical improvement with stabilization of vital signs; however, secondary hypoxia in multiple tissue beds and generalized edema may lead to dysfunction of distant organs, such as the kidneys and muscles. It may take several days for organ dysfunction to become clinically apparent. Signs of infection may be difficult to differentiate from clinical signs of other

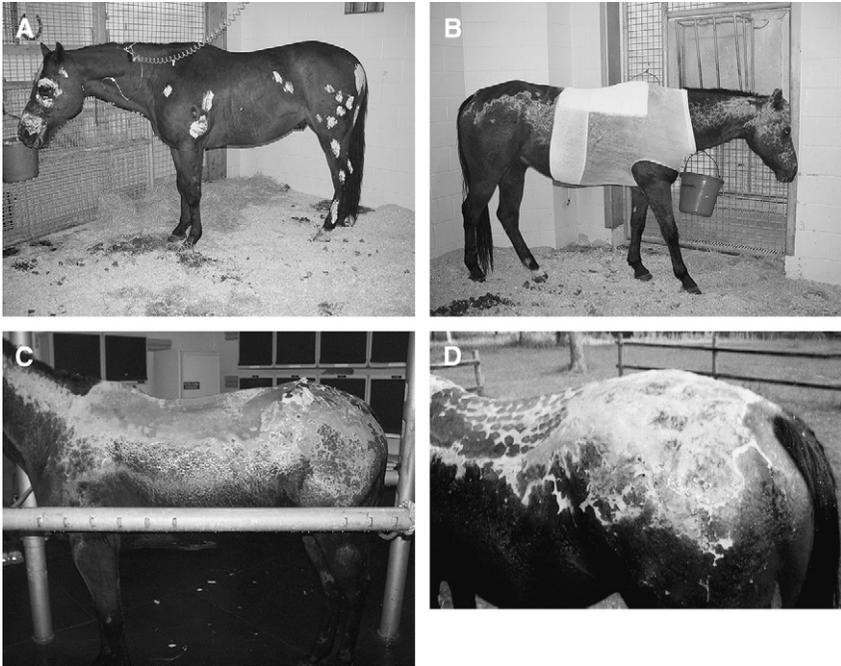


Fig. 2. This series of photographs documents the progression of thermal injury. This horse was rescued from a barn fire. Initially, it is difficult to ascertain the extent of the lesions. (A) At 24 hours after the trauma, edema of the muzzle and singed areas are noted as the most striking features. (B, C) Same patient at 6 and 20 weeks after injury. (D) Approximately 1 year after injury, the patient continues to require wound care. Note the black circles over the dorsal thorax, which are coalescing areas of healing skin grafts. Further resolution for this patient was obtained by the use of multiple punch skin grafts, with the exception of an area over the left gluteal muscles. Hot and humid conditions predisposed this individual to self-trauma of this region.

problems, and severely burned patients are at an increased risk of significant infection for a prolonged period. It is imperative to be vigilant for worsening of clinical signs after initial improvement as well as to monitor the horse for fever as an indication of possible infection.

Long term, the effects of a hypermetabolic response may become clinically evident as loss of body mass. Human patients with 40% body surface burn area can lose a quarter of their weight, even in the face of vigorous enteral alimentation [10]. In horses, this may be exhibited as those individuals that seem to waste away rapidly despite maintaining a good appetite. Healing of burn wound depends on depth and surface area affected. Even with aggressive surgical and medical management, large deep wounds can take prolonged time to heal and there is often scarring. Because many of the wounds involve the horse's back, it is important to remember that scarring can prevent future use of the equine patient for riding.

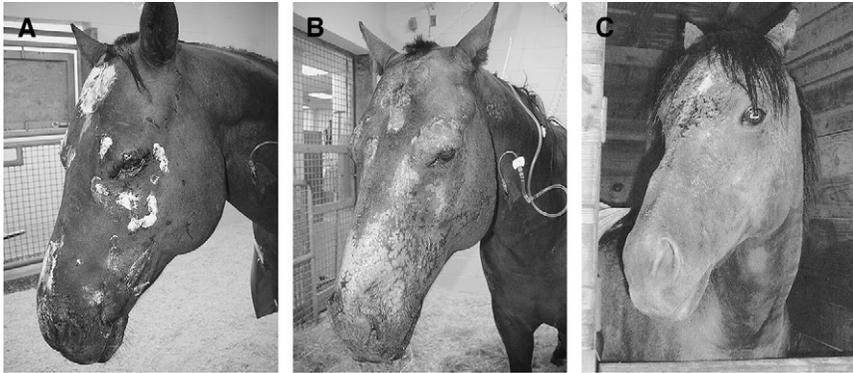


Fig. 3. Burn injury of the head is a common location for lesions. This series reveals clinical changes in lesions of the head in the same patient as in Fig. 2. There is a significant increase in the amount of edema present from the time of the photograph in *A*, which was taken within 6 hours of injury, to that in *B*, which was obtained 48 hours after the trauma. (*C*) Resolution of the lesions around the head occurred approximately 1 year later.

Diagnosis

Diagnosis is typically based on history and physical examination. A normal initial examination does not rule out exposure, because the onset of clinical signs may be delayed for several days. In human burn trauma, determining the extent of the surface area affected and the depth of the injury is an important diagnostic tool. Such information is useful to help determine the type of care required and for prognostic purposes. There are several protocols used to estimate the surface area, such as the “Rules of Nines” or the Lund and Browder charts [3]. There are also diagnostic techniques for determining the depth of injury, including biopsy and laser Doppler perfusion imaging. Even though a variety of theoretic methods



Fig. 4. Evidence of corneal trauma noted immediately in a horse rescued from a barn fire.

exist, this area is still an inexact science with human trauma. Again, there is limited information specific for the horse.

Diagnostic tools useful to judge the severity of cardiovascular compromise include blood pressure monitoring, measuring central venous pressure, serial lactate measurements, and monitoring urine production. Protein loss is thought to be a key component in edema formation; therefore, monitoring albumin levels as well as colloid oncotic pressure would be useful. Serial hematologic and serum chemistry analyses may be helpful to evaluate for dysfunction of various organ systems, such as renal failure or hemolytic anemia.

Various diagnostic tests are useful in determining the extent of respiratory injury. These include endoscopy of the upper respiratory tract and tracheobronchial tree, thoracic radiographs, blood gas analysis, hematology, and cytologic evaluation of tracheal aspirates. Any or all of these tests can be performed on a serial basis as prognostic aids. Within a short time after exposure, carboxyhemoglobin concentration in venous blood can be measured directly with a spectrophotometer. Serum levels fall rapidly; thus, evaluation for carboxyhemoglobin should occur as close as possible to the time of exposure. A level greater than 10% is consistent with carbon monoxide toxicity in the horse [4]. Carboxyhemoglobin and oxyhemoglobin reflect at the same wavelength; thus, pulse oximetry cannot be used to diagnosis carbon monoxide toxicity [21].

Treatment goals and prognosis

Treatment depends on the stage of injury. The initial therapeutic goals, as described previously, are to stop the burn process and gain intravenous access. Therapy should then be directed at improving or maintaining cardiovascular homeostasis. Intravenous fluid administration is the cornerstone of initial resuscitation of burn shock victims. Volumes required vary and should be tailored to the individual's needs based on clinical response. Monitoring urine output is a reasonable indicator of organ perfusion. A balanced polyionic electrolyte fluid is most commonly used. The use of a colloid during the initial phase of shock is controversial in human burn trauma. Some burn trauma centers tend to use colloid administration 12 to 24 hours after injury when the endothelial integrity may be returning to normal. Colloids like hydroxyethyl starch and fresh-frozen plasma have been described in treating equine patients [5]. The use of hypertonic saline to mobilize water from cells that may be overaccumulating fluid holds intuitive appeal; however, careful monitoring of sodium levels is required, and a recent study using hypertonic saline in burn shock revealed an increased incidence of renal failure and death [22].

Other therapeutic goals during the acute phase of burn shock include attenuation of inflammatory mediators and the use of free radical scavengers to treat reperfusion injury. Drugs like flunixin meglumine and pentoxifylline

have been proposed for the inhibition of prostaglandins and tumor necrosis factor, respectively. Dimethyl sulfoxide (DMSO) and acetylcysteine are examples of medications that have been used to treat oxidative stress. Use of corticosteroids for the treatment of burn shock is not recommended for human injury and is controversial in equine patients because of the potential for immunosuppression and laminitis [4].

Attention to the respiratory system is also important in the acute phase. Oxygen support is of benefit. It is a treatment for carbon dioxide toxicity and helps to reduce hypoxemia [19]. Humidified oxygen can be supplied by nasal insufflation or by means of a transtracheal catheter. Upper respiratory tract obstruction may require a tracheostomy. Attention should be paid to keeping the airways clear, and nebulization may be useful, especially when pseudomembranous casts are suspected. Bronchodilators may be useful in counteracting reflex bronchoconstriction. Decreasing inflammation and pulmonary edema may require the use of diuretics and nonsteroidal anti-inflammatory drugs.

Burn wound management is complex, and the details are beyond the scope of this article. A recent report discusses the details of wound care management [5]. Topical antimicrobial therapy is used to help protect damaged areas from infection during the healing phase. Silver sulfadiazine is the most widely used topical treatment in burn patients, and this medication has been used in horses. A possible effect of this topical treatment, especially if used over large areas, is the development of leukopenia. When large areas of skin are damaged, there is loss of effective thermoregulation, and environmental temperatures should be controlled if possible. To help prevent infection, strict hygiene, meticulous nursing care, and optimal nutritional support should be provided [1,5]. Prophylactic systemic antimicrobial use is not recommended in human patients. Documented infection should be treated with appropriate antimicrobial agents based on the results of culture and sensitivity patterns [5].

Although it can be difficult to differentiate signs of pain from the clinical signs of shock, developing a comprehensive pain management plan is recommended. In addition to the use of nonsteroidal anti-inflammatory drugs, other options include opioid medications, such as morphine and butorphanol [23]. Lidocaine and ketamine have also been described for use in pain management in the horse [24,25]. All these medications can be administered as continuous rate infusions as needed. Providing a high-caloric and well-balanced diet is vital, and to help maintain healthy gastrointestinal function, the enteral route is preferred. Human patients with a hypermetabolic response require 1.4 to 2 times the resting energy requirements [10]. In the equine patient, supplementing high-quality hay and grain with energy-rich fat, such as vegetable oil, can meet these needs.

Novel therapies may include inhalation treatment with medications to inhibit inflammatory mediators, coagulation factors, or oxidative stress. This is useful in patients with significant respiratory dysfunction associated

with plugging of airways. Treatments used have included inhalation of heparin, pentoxifylline, or acetylcysteine [26–28]. Another area of active research is the development of strategies to reduce catabolic effects of the hypermetabolic response. The use of medications, such as long-term propranolol, to block the β -adrenergic effects stimulated by high catecholamine levels has been helpful [29,30].

In the care of severely affected equine patients, it can be medically challenging to manage the multiple issues that can occur with fire- and smoke-associated trauma. Also, treatment of burn wounds can be prolonged and may require a lifetime of care with some individuals. During the initial emotional trauma in clients faced with such events, it is important to discuss the possible duration and cost of extended therapy as well as the chances of scar formation. There is no information on the long-term outcome of treatment of horses, especially in regard to the amount of surface area or depth of thermal injury. Although it may be possible to resuscitate the severely affected patient, it should be noted that euthanasia may be a reasonable option for patients with significant burn injury.

Summary

Fire and smoke can produce horrific traumatic injuries, and when such events involve horses, there is often high mortality. In addition to personal loss, there is often an emotional public response. Equine practitioners may be called on to perform several different types of services, ranging from being at the scene, to providing medical management, to helping with disaster plan development. Although injury may be mild, severe trauma can lead to complex critical cases with multiple organ system involvement that are a challenge to manage. Published information on these types of injuries specific to horses is limited. The extent of direct thermal injury may not be clinically evident initially. Severely affected equine patients may be successfully resuscitated but still face a poor prognosis because of the extent of dermal injuries.

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