

Management of Burn Injuries in the Horse

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Burns are uncommon in horses, with most occurring as a result of barn fires. Thermal injuries may also result from contact with hot solutions, electrocution or lightning strike, friction (eg, rope burns), abrasions, radiation therapy, and chemicals (eg, improperly used topical drugs, maliciously applied caustic agents) [1,2].

Most burns are superficial, easily managed, and inexpensive to treat and heal in a short time. Serious burns, however, can result in rapid severe burn shock or hypovolemia with associated cardiovascular changes. Smoke inhalation and corneal ulceration also are of great concern [1,2]. Management of severe and extensive burns is difficult, expensive, and time-consuming. The large surface area of the burn dramatically increases the potential for loss of fluids, electrolytes, and calories. Burns covering up to 50% or more of the body are usually fatal, although the depth of the burn also influences mortality. Massive wound infection is almost impossible to prevent because of the difficulty of maintaining a sterile wound environment. Long-term care is required to prevent continued trauma, because burn wounds are often pruritic and self-mutilation is common. Burned horses are frequently disfigured, preventing them from returning to full function. Therefore, before treatment, the patient must be carefully examined, with particular attention paid to cardiovascular function, pulmonary status, ocular lesions, and the extent and severity of the burns. The cost of treatment as well as the prognosis should be thoroughly discussed with the owner [1–4].

Classification of burns

Burns are classified by the depth of the injury [1–3]. First-degree burns involve only the most superficial layers of the epidermis. These burns are

painful and characterized by erythema, edema, and desquamation of the superficial layers of the skin. The germinal layer of the epidermis is spared, and the burns heal without complication (Fig. 1) [4]. Second-degree burns involve the epidermis and can be superficial or deep. Superficial second-degree burns involve the stratum corneum, stratum granulosum, and a few cells of the basal layer. Tactile and pain receptors remain intact. Because the basal layers remain relatively uninjured, superficial second-degree burns heal rapidly with minimal scarring within 14 to 17 days [5]. Deep second-degree burns involve all layers of the epidermis, including the basal layers. These burns are characterized by erythema and edema at the epidermal-dermal junction, necrosis of the epidermis, accumulation of white blood cells at the basal layer of the burn, eschar (slough produced by a thermal burn) formation, and minimal pain (Fig. 2) [1,3]. The only germinal cells spared are those within the ducts of the sweat glands and hair follicles. Deep second-degree wounds may heal spontaneously in 3 to 4 weeks if care is taken to prevent further dermal ischemia, which may lead to full-thickness

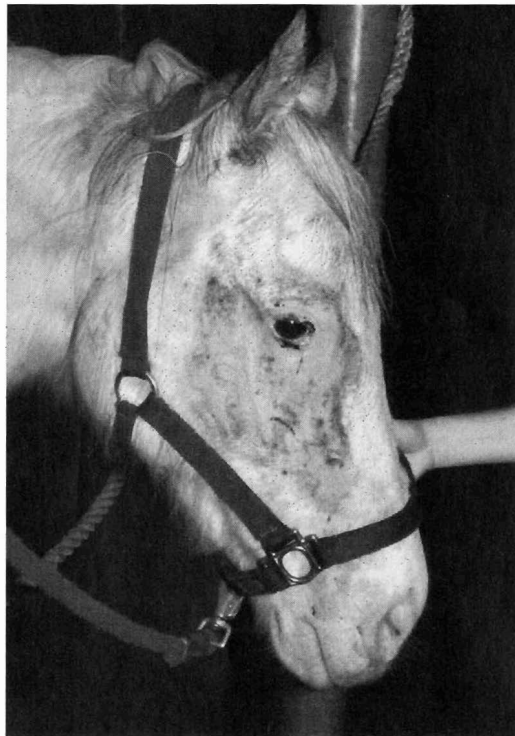


Fig. 1. First-degree burn of the right facial and periocular area. This type of burn involves only the most superficial layers of the epidermis. These burns are painful and characterized by erythema, edema, and desquamation of the superficial layers of the skin. The germinal layer of the epidermis is spared, and the burns heal without complication.



Fig. 2. Deep second-degree burn of the right dorsum and right hind limb. Deep second-degree wounds may heal spontaneously in 3 to 4 weeks if care is taken to prevent further dermal ischemia, which may lead to full-thickness necrosis.

necrosis. In general, deep second-degree wounds, unless grafted, heal with extensive scarring [6]. Third-degree burns are characterized by loss of the epidermal and dermal components, including the adnexa. The wounds range in color from white to black (Fig. 3). There is fluid loss and a marked cellular response at the margins and deeper tissue, eschar formation, lack of pain, shock, wound infection, and possible bacteremia and septicemia. Healing is by means of contraction and epithelialization from the wound margins or acceptance of an autograft. These burns are frequently complicated by infection. Fourth-degree burns involve all the skin and underlying muscle, bone, ligaments, fat, and fascia (Fig. 4) [7].

Mechanism of burn injury

The extent of tissue destruction is dependent on the temperature of the heat source, duration of exposure, blood supply, and local environment of the wound [7]. At the initial injury, there are three levels of injury: the



Fig. 3. Third-degree burn of the dorsal gluteal region incurred during a barn fire as a result of hot asphalt roof shingles falling on the horse. The central burn area is surrounded by deep and superficial second-degree burns.



Fig. 4. Fourth-degree burn of the right cervical neck region and pectoral area. Fourth-degree burns involve all layers of the skin as well as underlying muscle, bone, ligaments, fat, and fascia.

central zone of coagulation, the intermediate zone of vascular stasis, and the outer zone of hyperemia. The central zone of coagulation corresponds to the area that was closest to the heat source. At temperatures higher than 45°C, protein denaturation exceeds the capacity for cellular repair and cell death ensues [6]. The severity of injury decreases radially from this center as heat is dissipated (Fig. 5). Adjacent to the coagulation core is an intermediate zone of vascular stasis caused as dermal vessels thrombose during the initial 24 hours after injury. The damaged cells in this zone survive only under ideal circumstances. The use of heparin and thromboxane inhibitors may moderate the amount of tissue damage in this zone [8–10]. The outer region is the zone of hyperemia; the epidermis is lost, but the dermis remains intact. Cellular recovery in this area is rapid. In human beings, the total body surface area affected and the depth of the injury correlate highly with the degree of morbidity and mortality associated with thermal injury [6].

Pathophysiology of burn injury

After severe burns, there is a dramatic cardiovascular effect termed *burn shock*, which resembles hypovolemic shock. A dramatic increase in local and systemic capillary permeability occurs as a result of heat and the release of cytokines, prostaglandins, nitric oxide, vasoactive leukotrienes, serotonin, histamine, and oxygen radicals [11]. Local tissue damage results from massive protein coagulation and cellular death. In the immediate area of the burn, arteries and venules constrict and capillary beds dilate. Capillary wall permeability is increased in response to vasoactive amines released as a result of tissue damage and inflammation. These vascular responses result in fluid, protein, and inflammatory cells accumulating in the wound. There is vascular sludging, thrombosis, and dermal ischemia, resulting in further



Fig. 5. Deep second-degree burn of the right hind limb. The central burn area is surrounded by less severe skin burns, illustrating the dissipating radiating effects of the heat and damage to the skin.

tissue damage. Tissue ischemia continues for 24 to 48 hours after injury and is caused by the local release of thromboxane A_2 . Before any change in blood or plasma volume, there is a dramatic drop in cardiac output attributable to circulating levels of myocardial depressant factors [12]. Fluid loss into the extravascular space leads to an acute reduction in blood volume. With reductions in blood volume and cardiac output, peripheral and pulmonary vascular resistance increases, peripheral tissue perfusion decreases, and organ failure ensues [11,12].

The extent of fluid loss parallels the severity of the burn. Fluid losses result in increased heat loss from evaporation and an increased metabolic rate. The heat loss is in part responsible for the increased oxygen consumption and metabolic rate as the horse tries to generate heat. Depletion of fat stores and some endogenous protein supplies are two means by which metabolic compensation is achieved. In turn, this hypermetabolic rate leads to weight loss, a negative nitrogen balance, and delayed wound healing. Thus, the nutritional condition of the patient before injury is a prime prognostic consideration.

In burn injury, the vascular compartment remains permeable to proteins up to 15 nm in size, including albumin [13,14]. With moderate thermal injury, up to two times the total plasma albumin pool can be lost from the vascular compartment [15]. Loss occurs through the open wound and into the vascular space. Protein concentration can reach 3 g/dL in the extracellular fluid, which is sufficient to cause large fluid shifts because of osmotic pressure differences [16,17]. The resultant burn edema is clinically recognized within 60 minutes of injury [18].

Accompanying the fluid and protein shifts are electrolyte disturbances. Immediately after a burn, hyperkalemia may occur because of cellular disruption and potassium leakage [19]. When counterbalanced by increased mineralocorticoid secretion, the urinary sodium/potassium ratio is reversed and a subsequent potassium deficit may develop 3 to 5 days after injury [20]. Simultaneously, hypernatremia may result as sodium is reabsorbed after restoration of vascular membranes [21]. During replacement therapy, frequent determinations of protein concentrations, circulatory volume, and electrolyte concentrations are indicated [19-21].

Anemia is not usually a significant concern immediately after a burn. Anemia can, however, become a progressive problem in patients with burns exceeding 30% of the total body surface area [22]. An early anemia resulting from red cell hemolysis and splenic sequestration may be present but is often masked by hemoconcentration. Initial anemia is caused by the immediate destruction of red blood cells by heat and wound hemorrhage [23]. Subsequently, erythrocyte loss occurs from intravascular and extravascular removal of damaged cells as well as during eschar removal [21]. Thrombocytopenia may result from platelet aggregation on damaged capillary endothelium. If damage is extensive, a hemorrhagic diathesis may result from exhaustion of clotting factors.

Immunoglobulin levels in the serum drop, with the lowest values at 2 days after a burn. Serious defects in neutrophil function, such as an inefficient chemotaxis, and impaired phagocytic rate and bactericidal capacity have also been observed in severely burned horses. In addition to destruction of the mechanical barrier of the skin, decreased neutrophil chemotaxis and bactericidal properties, defective fibronectin, and complement opsonization, a decrease in IgG concentration often results. Their combined effect results in a compromised host that is prone to infection [24,25].

Metabolic rate increases in a curvilinear fashion proportional to the size of the thermal injury exceeding 10% of the total body surface area. This causes an increase in body core temperature from 1°C to 2°C and increases in oxygen consumption, fat degradation, and protein and glucose use [26–28]. Caloric expenditure and protein catabolism are greater in burn injury than in any other physiologic stress state. In patients with burns exceeding 30% of the total body surface area, energy expenditure doubles and fuel substrates are metabolized at two to three times the normal rate [29]. To avoid the rapid depletion of skeletal muscle, delayed wound healing, and impaired cellular defense mechanisms, caloric and protein intakes must be adjusted to maintain body weight [26,29,30]. Environmental temperatures should be kept between 28°C and 33°C to minimize the metabolic expenditure required to maintain the elevated core temperatures [26,30,31].

Pathophysiology of inhalation injury

Inhalation injury is a common sequela of closed-space fires and develops through three mechanisms: direct thermal injury, carbon monoxide poisoning, and chemical insult. Direct thermal injury causes edema and obstruction of the upper airway, but because of the efficient heat exchange capacity of the nasopharynx and oropharynx, superheated air is cooled before entering the lower respiratory tract [32].

Carbon monoxide interferes with oxygen delivery in several ways [33]. It has a 230 to 270 times greater affinity for oxygen, thus shifting the oxygen-hemoglobin curve to the left. The resultant carboxyhemoglobin is incapable of oxygen transport. Carbon monoxide also binds to myoglobin, thereby impairing oxygen transport to muscles [34,35]. Carbon monoxide is excreted by the lungs at a rate related to ambient oxygen tensions. In room air, carbon monoxide has a half-life of 3 to 4 hours. An increase in oxygen tension promotes the dissociation of carbon monoxide and hemoglobin; thus, 100% oxygen therapy reduces the half-life to 30 to 40 minutes. Hyperbaric oxygen therapy at 2.5 atm further decreases the half-life to 22 minutes [36].

Chemical insult depends on the material that was burned [32,37]. Combustion products, such as hydrogen cyanide, hydrochloric acid, phosgene, sulfuric acid, and aldehydes, may induce severe tracheobronchitis when combined with the moisture in the airways. Initially, only erythema

may be present, but chemical injury continues as long as chemical-covered carbon particles remain attached to the airway mucosa, with particle size determining where damage occurs within the respiratory tree (Fig. 6). Combustion products cause increased pulmonary artery pressure, peribronchial edema, mucosal sloughing, bronchoconstriction, decreased mucociliary transport and bacterial clearance, and altered surfactant action [35,38]. Subsequently, significant pulmonary ventilation/perfusion mismatches may develop [34,39].

Pulmonary infection is a potential complication in every smoke inhalation patient. Alveolar macrophages, as the primary cellular defense in the lung, are increased in number after the injury but have decreased phagocytic and bactericidal functions [40–42]. Susceptibility to pulmonary infection, pulmonary edema, and lung dysfunction increases greatly in patients that also have cutaneous thermal injury. The interrelation between inhalation and surface burns is unclear but seems to be additive [40–42]. Major cutaneous burns alone have been reported to cause pulmonary dysfunction in as many as 25% of patients, whereas inhalation injury increases the morbidity and mortality rates for a given cutaneous thermal injury [34,38,43,44].

Physical examination findings

Because heat is slow to dissipate from burn wounds, it is often difficult to evaluate the amount of tissue damage accurately in the early phase of injury. The extent of the burn depends on the size of the area exposed, whereas the

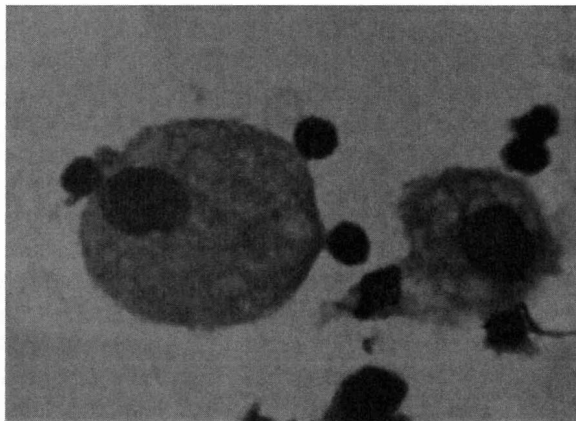


Fig. 6. Carbon particles associated with alveolar macrophages in the bronchoalveolar lavage as a result of inhalation smoke injury. Chemical injury continues as long as chemical-covered carbon particles remain attached to the airway mucosa, with the size of the particles determining where damage occurs within the respiratory tree.

severity relates to the maximum temperature the tissue attains and the duration of overheating. This explains why skin injury often extends beyond the original burn [45].

A complete physical examination should be performed on any burned animal before the wound is evaluated. Only after the patient's condition is stable should the burn wound be assessed. Physical criteria used to evaluate burns include erythema, edema and pain; blister formation; eschar formation; presence of infection; body temperature; and cardiovascular status [45]. In general, erythema, edema, and pain are favorable signs because they indicate that some tissue is viable, although pain is not a reliable indicator for determining wound depth [45]. Often, time must elapse to allow further tissue changes so that an accurate evaluation of burn severity can be made (Fig. 7).

Burns are most commonly seen on the back and face. Erythema, pain, vesicles, and singed hair are present depending on the extent of the injury. Increases in heart and respiratory rates are present in association with abnormal discoloration of mucous membranes. The burned horse may have blepharospasm, epiphora, or both, which signify corneal damage. Coughing may indicate smoke inhalation, whereas a fever signals or confirms a systemic response.

The percentage of total body surface area involved usually correlates with mortality, whereas the depth of the burn determines morbidity [16]. The rule of nine is used commonly in human beings to evaluate the total body surface area involved. Using this method, an approximate extent of the burn can be used to estimate the prognosis. Each forelimb represents 9%, each hind limb represents 18%, the head with the neck represents 9%, and the thorax and abdomen each represent 18% of body surface area [4]. Special attention should be taken to identify injury to major vessels of the lower limbs and the presence of eye, perineal, tendon sheath, and joint involvement. Initial laboratory data, including complete blood cell count, clotting profile, serum chemistry, urinalysis, arterial blood gas, carbon monoxide concentration, chest radiograph, and bronchoalveolar lavage, are helpful in the initial evaluation [7].

Laboratory findings may reveal a low total protein with anemia that may be severe and steadily progressive. Hemoglobinuria may be present. Hyperkalemia may be present initially, but hypokalemia is more likely later in the course of the condition and may often be associated with fluid therapy.

Although specific guidelines do not exist for burned large animals, euthanasia should be recommended for animals with deep partial-thickness to full-thickness burns involving 30% to 50% of the total body surface area [20,46]. The availability of adequate treatment facilities, cost of treatment, and pain experienced by the horse during long-term care should be considered when deciding whether or not to treat. Because convalescence may take up to 2 years, euthanasia is often an acceptable alternative [47].

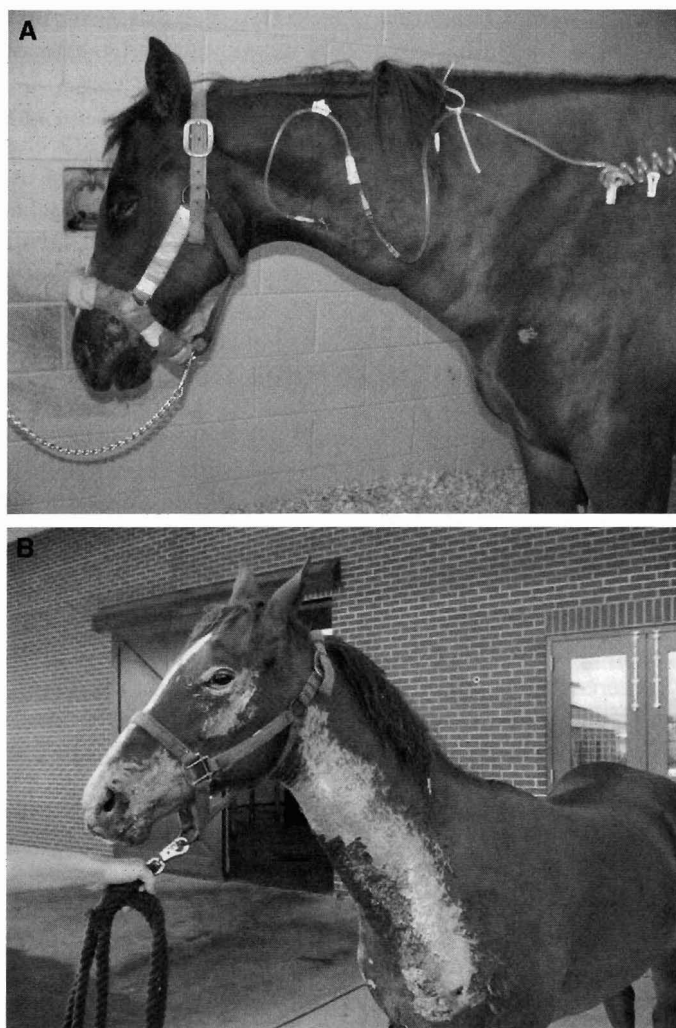


Fig. 7. (A) Severe burn edema along the ventral neck region in a horse 24 hours after burn injury caused by a barn fire. (B) The extent of the burn is more evident after the skin has sloughed because of the latent thermal injury to the skin.

Treatment of pulmonary injury

Maintenance of airway patency, adequate oxygenation and ventilation, and stabilization of hemodynamic status are the cornerstones of therapy for smoke inhalation injury. Early intervention and respiratory support are essential even before the diagnosis of respiratory injury is confirmed. Nasal or tracheal insufflation with humidified 100% oxygen counteracts the effects of carbon monoxide and facilitates clearance by decreasing the half-life of carbon monoxide in the blood. Oxygen insufflation rates of 15 to 20 L/min

can be achieved through a tracheostomy and should be continued until the patient is able to maintain normal oxygenation. Humidification can relieve excessive airway drying or mucous plugging. Nebulizing with N-acetylcysteine and heparin and the use of humidified air can reduce the formation of pseudomembranous casts and aid in the clearance of airway secretions [35,39,44,48]. Nebulized dimethylsulfoxide (DMSO) helps to decrease lung fluid formation [39,44]. The β -adrenergic agonist albuterol can be aerosolized to reduce bronchospasm. DMSO and heparin may protect against airway damage caused by smoke [49–52]. Maintenance of optimal fluid status is essential; patients with concurrent surface burns and inhalation injury require 2 mL per percentage of burns per kilogram more fluid than those with cutaneous burns alone to support adequate cardiac and urine output [53].

Antibiotics and corticosteroids do not influence survival rates and should not be routinely administered to smoke inhalation patients. Systemic antimicrobials are indicated only for proven infections, the incidence of which increases 2 to 3 days after smoke inhalation. Intramuscular penicillin is effective against oral contaminants colonizing the airway. If signs of respiratory disease worsen, a transtracheal aspiration sample should be submitted for culture and sensitivity testing and the antibiotic regimen adapted accordingly [49,53].

Patients with suspected significant smoke inhalation should be observed closely for several hours and hospitalized in the presence of extensive burns. Therapy should be adjusted based on the clinical response and the results of serial blood gas analyses, complete blood cell counts, chest radiographs, airway endoscopy, and cultures. Successful treatment depends on continuous patient reassessment and early aggressive patient care.

Treatment of burn shock

With burn shock, large volumes of balanced electrolyte solution are generally the fluid of choice unless serum electrolyte analysis dictates otherwise. In patients with burns exceeding 15% of the total body surface area, intravenous fluid therapy is required to avoid circulatory collapse [54]. Inadequate fluid resuscitation results in decreased renal and gastrointestinal perfusion that could lead to gastrointestinal bacterial translocation and sepsis [55]. Administration of isotonic fluids at a rate of 2 to 4 mL/kg for each percentage of surface area burned is recommended, but fluid resuscitation is best titrated to maintain a stable and adequate blood pressure [26]. An alternative is to use hypertonic saline solution, 4 mL/kg, with plasma, hetastarch, or both, followed by additional isotonic fluids. If there has been smoke or heat inhalation injury, crystalloids should be limited to the amount that normalizes circulatory volume and blood pressure. The same continued rate of administration of electrolyte solutions after the

resolution of burn shock results in edema in excess of any improvement in cardiovascular dynamics [56]. Plasma at a rate of 2 to 10 L is an effective albumin source as well as an exogenous source of antithrombin III for coagulopathies. One should carefully monitor the hydration, lung sounds, and cardiovascular status during fluid administration.

Flunixin meglumine (0.25–1.0 mg/kg administered intravenously every 12–24 hours) and pentoxifylline (8.0 mg/kg administered intravenously every 12 hours) are effective analgesics and improve blood flow in the small capillary networks. DMSO (1 g/kg administered intravenously) for the first 24 hours may decrease inflammation and pulmonary edema. If pulmonary edema is present and is unresponsive to DMSO and furosemide treatment, dexamethasone can be administered intravenously once at a dose of 0.5 mg/kg.

Nutritional needs

Assessment of adequate nutritional intake is performed with a reliable weight record. Weight loss of 10% to 15% during the course of illness is indicative of inadequate nutritional intake. Nutritional support can include parenteral and enteral routes, with the latter being superior [44]. Early enteral feeding not only decreases weight loss but maintains intestinal barrier function by minimizing mucosal atrophy. This reduces bacterial and toxin translocation and subsequent sepsis [44].

Gradually increasing the grain, adding fat in the form of vegetable oil (4–8 oz), and offering free-choice alfalfa hay increase caloric intake. An anabolic steroid may be used to help restore a positive nitrogen balance. If smoke inhalation is a concern or there is evidence of burns around the face, the hay should be soaked in water and fed on the ground with good ventilation provided [45].

Wound care

First-degree burns are generally not life threatening and are simply managed. Topical therapy in the form of cool compresses, cold-water baths, and wound coverings may provide relief. Pain control can be accomplished with nonsteroidal anti-inflammatory drugs or narcotics.

Second-degree burns are associated with vesicles and blisters. These vesicles should be left intact for the first 24 to 36 hours after formation, because blister fluid provides protection from infection and the presence of a blister is less painful than the denuded exposed surface. After this interval, the blister is partially excised and an antibacterial dressing is applied to the wound or an eschar is allowed to form [1,2,16].

Third-degree burns can be difficult to manage. The patient's condition should be stabilized as rapidly as possible before undertaking wound management. Destruction of the dermis leaves a primary collagenous

structure called an eschar. Dry exposure is a treatment method that operates according to the principle that bacteria do not thrive on a dry surface. The goals of therapy are to keep the wound dry and protected from mechanical trauma. Heat and water loss from the uncovered wound, however, is a disadvantage.

There are several methods to treat burn wounds in the horse, and the choice depends on the extent and location of the injury. Full-thickness burns can be managed by occlusive dressings (closed technique), continuous wet dressings (semiopen technique), eschar formation (exposed technique), or excision and grafting [45].

The closed method uses occlusive artificial dressings. Wound cleansing and debridement are performed at each of the frequent dressing changes. Temporary dressings can, by adhering to the underlying wound bed, decrease the bacterial population, decrease heat and water loss, protect the bed of granulation tissue, and hasten wound healing. With large burns, however, frequent bandage changes and debridement can be painful and extensive bandaging may not be feasible or affordable in some animals [45].

With the semiopen method, the eschar is left in place but is kept covered with an antimicrobial-soaked dressing. The dressing provides protection against trauma, bacterial contamination, and evaporative losses. The wet dressings enhance eschar removal [45].

With the open technique the wound is left exposed to the air to form its own biologic barrier composed of exudate, collagen, and layers of dead skin known as the burn eschar. The eschar does not prevent bacterial contamination or heat or water evaporation, and the depth of tissue destruction may be marginally increased during the drying process. The eschar is covered with an antibacterial agent twice daily. Wound contraction does not occur while the eschar is intact [6]. The eschar is sloughed by bacterial collagenase activity within 4 weeks [57]. The exposed bed can then be grafted or allowed to contract.

Eschar excision and grafting are useful for smaller burns but cannot be used for large burns because of lack of donor skin. Commercially available xenografts (porcine skin) can be used to cover large defects after excision; however, the cost can be prohibitive [1,16]. Eschar excision and open treatment are not practical for extensive burns in horses because of the likelihood of environmental contamination and massive losses of fluid and heat [45]. Therefore, the most effective and practical therapy for large burns in horses is the open method, leaving the eschar intact, with continuous application of antibacterial agents [1,2,45,58].

Initially, the surrounding hair should be clipped and the wound debrided of all devitalized tissue [16]. Attempts should be made to cool the affected skin using an ice or cold-water bath. Copious lavage with a sterile 0.05% chlorhexidine solution should be performed [16]. A water-based antibiotic ointment should be applied liberally to the affected areas to prevent heat and moisture loss, protect the eschar, prevent bacterial invasion, and loosen

necrotic tissue and debris. This slow method of debridement allows removal of necrotic tissue as it is identified, thereby preventing possible removal of healthy germinal layers by mistake. The eschar is allowed to remain intact with gradual removal, permitting it to act as a natural bandage until it is ready to slough.

Dry flakes or sheets of a sterile starch copolymer are available and form a moldable gel when mixed with water. This material absorbs 30 times its weight in exudate, prevents further eschar formation by keeping tissues moist, and does not interfere with topical antibiotics, which can be applied before the gel or mixed with the gel.

Although bacterial colonization of large burns in horses is not preventable, the wound should be cleansed two or three times daily and a topical antibiotic reapplied to reduce the bacterial load to the wound. Occlusive dressings should be avoided because of their tendency to produce a closed-wound environment that may encourage bacterial proliferation and delay healing.

Systemic antibiotics do not favorably influence wound healing, fever, or mortality and can encourage the emergence of resistant microorganisms. Additionally, circulation to the burned areas is often compromised, making it highly unlikely that parenteral administration of antibiotics can achieve therapeutic levels at the wound. Short-term prophylactic intravenous antibiotic therapy may be indicated in the immediate postburn period if quantitative biopsy cultures or a more rapid slide dilution method yields more than 100,000 cells per gram of tissue [59].

The most commonly used topical antibacterial for the treatment of burns is silver sulfadiazine in a 1% water miscible cream (Par Pharmaceutical, Spring Valley, NY). It is a broad-spectrum antibacterial agent able to penetrate the eschar. Silver sulfadiazine is active against gram-negative bacteria, especially *Pseudomonas*, with additional effectiveness against *Staphylococcus aureus*, *Escherichia coli*, *Proteus*, Enterobacteriaceae, and *Candida albicans* [16,45,56]. It causes minimal pain on application but must be used twice a day because it is inactivated by tissue secretions. Although pseudoeschar formation that may preclude wound evaluation, transient leukopenia, skin hypersensitivity, and the development of bacterial resistance have all been reported in human beings, silver sulfadiazine has few systemic effects and provides good results in the horse [45,56,60].

Aloe vera is a gel derived from a yucca-like plant and has antithromboxane and antiprostaglandin properties [61]. It is reported to relieve pain, decrease inflammation, stimulate cell growth, and kill bacteria and fungi. Although used successfully in the acute treatment of burns, it may actually delay healing once the initial inflammatory response has resolved [61]. Aloe vera and silver sulfadiazine are good first choices in antibiotic therapy for burns and are used extensively in human medicine.

Other effective topical antimicrobials include nitrofurazone (Furacin; Phoenix Pharmaceuticals, St. Joseph, MO), mafenide acetate, chlorhexidine

(Nolvasan; Fort Dodge Animal Health, Fort Dodge, IA), povidone-iodine (Betadine; Purdue Frederick Co., Norwalk, CT), and gentamicin sulfate ointment (Gentamicin sulfate ointment USP; Clay-Parks Labs, Bronx, NY). Nitrofurazone has a fairly narrow range of antibacterial activity, resistance can develop, and it does not penetrate the eschar well. Chlorhexidine is active in vitro against a number of gram-positive vegetative bacteria, yeasts, and dermatophyte fungi but has questionable effectiveness against gram-negative organisms. Because of its cationic nature, chlorhexidine binds strongly to skin, mucosa, and other tissues and is thus poorly absorbed. Chlorhexidine can be applied as a cream or solution. Povidone-iodine causes some patient discomfort but is effective against bacteria, yeast, and fungi. Its hyperosmolality causes severe hypernatremia and acidosis because of water loss [56], such that it should not be used on extensive burns where systematic absorption is likely. Immune system depression has also been reported in human patients [62]. Gentamicin is excellent for serious gram-negative infections but should be used only in selected cases, because resistance can develop and it may be nephrotoxic in patients with renal problems. Topical aqueous antibacterial preparations have also been used to treat burns. The solution (mixture of nitrofurazone, glycerin, and distilled water) can be applied to the wound as a mist from a spray bottle several times a day [1]. The nitrofurazone kills bacteria, whereas the moisture loosens the eschar and promotes debridement. Other agents that are occasionally used include neomycin, bacitracin, and polymyxin B. Their use is generally associated with the rapid development of bacterial resistance and systemic toxicity. They are not recommended for routine use in long-term wound care [60].

It is appropriate to change antibacterial creams according to clinical results. In large burns, quantitative wound biopsy analysis is advantageous. Wound flora densities of more than 10^5 organisms per gram of tissue predispose the patient to bacterial invasion of healthy tissue [57]. Preventing conversion of superficial wound sepsis to full-thickness infection with the risk of systemic sepsis is accomplished by administering local antibiotics. The use of systemic antibiotics is not recommended because they are ineffective in penetrating the avascular eschar, where the risk of contamination is greatest [60].

Many burned equine patients are pruritic, and measures must be taken to prevent self-mutilation of the wound. Reserpine can be effective in decreasing the urge to scratch by successfully breaking the itch-scratch cycle.

Skin grafts

Burns heal slowly, and many weeks may be required for the wound to close by means of granulation, contraction, and epithelialization. Closure of the burn wound by primary suturing or skin grafting after eschar removal allows for more rapid healing and superior pain relief and prevents loss of

heat, water, and protein-rich exudate from the wound surface. Burns involving only the superficial dermis heal well within 3 weeks and do not need grafting. Conversely, deep partial-thickness wounds require several months to heal, during which time bacterial contamination of the wound develops. Second-intention healing results in a thin and hairless epithelium that is vulnerable to trauma. Excision, followed by grafting, of the wound is recommended in these cases. Full-thickness grafts from a cadaver donor can be used early in the clinical course of the burn to encourage healing, whereas split-thickness autogenous mesh grafts can be applied once healthy granulation tissue has formed. Early excision and grafting may also benefit horses that do not tolerate daily wound debridement and cleansing.

Complications

Healing burn wounds are pruritic [2,45]. Significant self-mutilation through rubbing, biting, and pawing can occur if the horse is not adequately restrained or medicated. Usually, the most intense pruritic episodes occur in the first weeks during the inflammatory phase of repair and during eschar sloughing. To prevent extreme self-mutilation, the animal must be cross-tied and/or sedated at this time. Other complications include habronemiasis, keloid-like fibroblastic proliferations, sarcoids, and other burn-induced neoplasia [2,47]. Hypertrophic scars, which commonly develop after deep second-degree burns, generally remodel in a cosmetic manner without surgery within 1 to 2 years. Because scarred skin is hairless and often depigmented, solar exposure should be limited. Chronic nonhealing areas should be excised and autografted to prevent neoplastic transformation. Delayed healing and poor epithelialization, complications of second-intention healing, may limit the return of the animal to its previous use (Fig. 8).

Summary

Extensive thermal injuries in horses can be difficult to manage. The large surface of the burn dramatically increases the potential for loss of fluids, electrolytes, and calories. Burns are classified by the depth of injury: first-degree burns involve only the most superficial layers of the epidermis; second-degree burns involve the entire epidermis and can be superficial or deep; third-degree burns are characterized by loss of the epidermal and dermal components; and fourth-degree burns involve all the skin and underlying muscle, bone, and ligaments. Burns cause local and systemic effects. Routine use of systemic antibiotics is not recommended in burn patients. Topical medications should be water based, be easily applied and removed, not interfere with wound healing, and be readily excreted or metabolized. Weight loss of 10% to 15% during the course of illness is

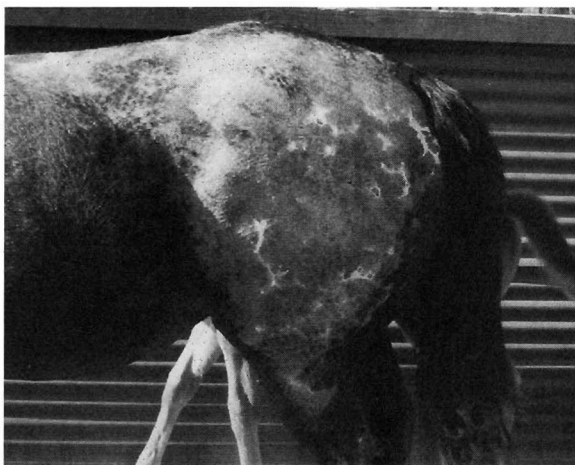


Fig. 8. Horse that sustained deep second- and third-degree burns of the dorsum and left hind limb 7 months previously. The entire wound has epithelialized. The skin is thin and brittle because of lack of sufficient subcutaneous tissue.

indicative of inadequate nutritional intake. Gradually increasing the grain, adding fat in the form of vegetable oil, and offering free-choice alfalfa hay increase caloric intake.

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