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Review Article

Open Wound in Equine and Its Management-Review

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Abstract

Open wounds are the most frequently recorded health and welfare problem in working animals in developing countries that need proper wound dressing, as they are prone to bacterial infection. Wound dressing is one of the main external effects during the healing process of wounds. Understanding the process of wound healing and knowing the phases of wound healing is vital to intervene in the management of open wounds. It is based on repeated bandaging and debridement as needed sugar dressings, honey dressings, wound lavage, wound debridement, and using antiseptics. Besides, antibiotic treatment is necessary to avoid secondary bacterial complications and Natural polymers such as polysaccharides and derivatives (e.g., carboxymethyl cellulose, alginates, chitosan, and heparin), proteoglycans, and proteins (e.g., collagen, gelatin, fibrin, and keratin) facilitate the healing process. The development of wound dressing materials is the main objective of this paper, which will provide general information on future wound dressing materials.

Introduction

Wounds come in all types and varieties. Horses can be injured with anything from a simple scratch to deeper cuts and lacerations that involve tendons, joints, major vessels, and nerves as well as most of the other important structures. Due to this wide variance, it is of utmost importance that a horse receives a thorough physical examination to determine the extent of the injury as well as to determine the overall health and status. If a major blood vessel has been severed, it is easy for the horse to lose a substantial amount of blood. Also, it is very important to realize the areas on your horse where lacerations could result in a career or life-ending injury [1,2].

Horses commonly sustain wounds owing to their instinct to flight, and in the way, we confine and use them close to traumatic objects. Management of traumatic wounds is therefore one of the most common challenges facing veterinarians working with horses. Treatment must be planned on an individual animal level, influenced not only by clinical but also by financial and logistical factors. Rapid healing with the optimal functional and cosmetic outcome is the aim of treatment [3].

There are many options for wound cleaning and it is very important not to cause further trauma when treating a wound. Everything veterinarians have to work with has the potential of causing increased trauma to the wound area, and all therapies must be evaluated to determine if the benefit will outweigh the trauma that occurs. In general, the use of antiseptics in wounds as they can kill the healthy cells we need for healing, and will not kill all of the bacteria in a wound. Saline is the least disruptive of all washing solutions and should be used frequently. Surfactant-based wound cleansers work by reducing the surface tension around the wound, allowing for the removal of fluid, cells, and other substances that naturally seep from the wound area without the need to scrub the wound. This leaves healthy tissues to continue their good work of healing the wound [4,5]. This review aims to assess the principles of initial wound management, treatment, and aftercare.

Review of Open Wound in Equine and Its Management

Types of an Open Wounds

An open wound is a break in the skin surface resulting in external bleeding. It may allow bacteria to enter the body, causing an infection [2,6].

Puncture Wounds

Penetrating wounds that generally look minor by making small skin tears or holes, but can cause significant trauma beneath. Most puncture wounds are complicated by infection because contamination is introduced deep into the wound. Often, the skin heals before the underlying tissue. These wounds should be cleaned, lavage, and encouraged to drain and remain unsutured [7].

Incised Wounds

Generally slicing type wounds that have smooth and clean edges, caused by sharp objects. After a thorough cleaning, these wounds are usually best suited for primary closure (suturing, stapling, or gluing) [8].

Lacerations

Generally, traumatic injuries that leave rough, jagged edges of the skin and possibly underlying soft tissue damage. These wounds are at greater risk for infection due to contamination and generally require some debridement. Depending on the severity and location of the wound, second intention healing or open wound management may be the treatment of choice [6,8].

Abrasions

This is non-penetrating wounds of the skin. These wounds are generally minor, and other than cleaning, require minimal treatment [7].

Wound Healing

Wound healing is divided into three phases: inflammatory, proliferative, and remodeling. Understanding the process of wound healing is essential for the effective management of wounds. Topical medications should provide a specific desired effect during the appropriate stage of healing. 'Wounds are dynamic and treatment may need to be modified during progression through the different stages of healing. Manufacturer's instructions and veterinary literature are important sources of information for the appropriate concentration, frequency of administration, and mode of application of topical agents [6].

Phases of Wound Healing

Coagulation and Haemostasis Phase

Immediately after injury, coagulation and hemostasis take place in the wound. The principal aim of these mechanisms is to prevent exsanguination. It is a way to protect the vascular system, keeping it intact so that the function of the vital organs remains unharmed despite the injury. A second aim is a long-term one, which is to provide a matrix for invading cells that are needed in the later phases of healing. A dynamic balance between endothelial cells, thrombocytes coagulation, and fibrinolysis regulates hemostasis and determines the amount of fibrin deposited at the wound site, thereby influencing the progress of the reparative processes [6].

Inflammatory Phase

The humoral and cellular inflammatory phase follows next, to establish an immune barrier against invading micro-organisms. It is divided into two separate phases, an early inflammatory phase and a late inflammatory phase [9].

Early Inflammatory Phase

Starting during the late phase of coagulation and shortly thereafter, the early inflammatory response has many functions. It activates the complement cascade and initiates molecular events, leading to the infiltration of the wound site by neutrophils, whose main function is to prevent infection. The neutrophils start with

the critical task of phagocytosis to destroy and remove bacteria, foreign particles, and damaged tissue. Phagocytotic activity is crucial for the subsequent processes because acute wounds that have a bacterial imbalance will not heal [10].

Late Inflammatory Phase

As part of the late inflammatory phase, 48-72hr after injury, macrophages appear in the wound and continue the process of phagocytosis. These cells are originally blood monocytes that undergo phenotypic changes on arrival into the wound to become tissue macrophages. Attracted to the wound site by a myriad of chemoattractive agents, including clotting factors, complement components, cytokines such as PDGF, TGF-β, leukotriene B4, and platelet factor IV, as well as elastin and collagen breakdown products, macrophages have a longer lifespan than neutrophils and continue to work at a lower pH [9].

Proliferative Phase

When an ongoing injury has ceased, hemostasis has been achieved and an immune response successfully set in place, the acute wound shifts toward tissue repair. 16-19 the proliferative phase starts on the third day after wounding and lasts for about 2 weeks thereafter. It is characterized by fibroblast migration and deposition of the newly synthesized extracellular matrix, acting as a replacement for the provisional network composed of fibrin and fibronectin. At the macroscopic level, this phase of wound healing can be seen as an abundant formation of granulation tissue [4,11].

Remodeling Phase

As the final phase of wound healing, the remodeling phase is responsible for the development of new epithelium and final scar tissue formation. Synthesis of the extracellular matrix in the proliferative and remodeling phases is initiated contemporarily with granulation tissue development. This phase may last up to 1 or 2 years, or sometimes for an even more prolonged period. The remodeling of an acute wound is tightly controlled by regulatory mechanisms to maintain a delicate balance between degradation and synthesis, leading to normal healing. Along with intracellular matrix maturation, collagen bundles increase in diameter, and hyaluronic acid and fibronectin are degraded. The tensile strength of the wound increases progressively in parallel with collagen collection [12].

Factors Affecting Wound Healing

Oxygenation

Oxygen is important for cell metabolism and is critical for most wound healing processes. Vascular disruption and high oxygen consumption by active cells lead to depletion of oxygen in an early wound. Systemic conditions such as advancing age and diabetes can create further impaired vascular flow. When oxygenation is not restored, healing is impaired. Prolonged hypoxia will lead to a chronic wound status. One therapeutic option for tissue hypoxia is hyperbaric oxygen therapy [11,13].

Infections

Contamination is the presence of non-replicating organisms in a wound. Colonization is defined as the presence of replicating microorganisms. Wounds may have either. Bacteria in infected wounds occur in the form of biofilms. Mature biofilms develop protected environments and are resistant to conventional antibiotics Many chronic wounds do not heal because of the presence of biofilms [4].

Age

Delayed wound healing in the aged is associated with an altered inflammatory response. A review of the age-related changes in healing demonstrates that every phase of healing undergoes characteristic changes, including enhanced platelet aggregation, increased secretion of inflammatory mediators, delayed infiltration of macrophages and lymphocytes, impaired macrophage function, decrease secretion of growth factors, delayed re-epithelialization, delayed angiogenesis, reduced collagen turnover and remodeling, and decreased wound strength [14].

Sex Hormones in Aged Individuals

Compared with aged females, aged males have been shown to have delayed healing of acute wounds. Estrogen affects wound healing by regulating a variety of genes associated with regeneration, matrix production, protease inhibition, epidermal function, and inflammation [15,16].

Stress

Studies in both humans and animals have shown that psychological stress causes a substantial delay in wound healing. This stress impairs normal cell-mediated immunity at the wound site [6].

Nutrition

Nutrition has been recognized as a very important factor that affects wound healing. Malnutrition or specific nutrient deficiencies can have a profound impact on wound healing after trauma or surgery. Carbohydrates, proteins, fat, vitamins, and mineral metabolism all can affect the healing process [14].

Initial Wound Management

The six basic steps of wound management are prevention of further wound contamination, debridement of dead and dying tissue, removal of debris and contaminants, provision of adequate wound drainage, promotion of a viable vascular bed, and selection of an appropriate method of closure [17]. Therapy aims to facilitate wound healing mechanisms by providing a warm, clean environment and an adequate blood supply [18,19].

Open Wound Management

When a wound cannot or should not be closed, open wound management (i.e., second-intention healing) may be appropriate. Such wounds include those in which there has been a loss of skin that makes closure

impossible or those that are too grossly infected to close. Longitudinal degloving injuries of the extremities are especially amenable to open wound management. Open wound management enables progressive debridement procedures and does not require specialized equipment (such as may be needed with skin grafting). However, it increases cost, prolongs the time for healing, and may create complications from wound contracture [20].

Open wound management is based on repeated bandaging and debridement as needed until the wound heals. Traditional therapy calls for wet-to-dry dressings initially. The initial wide meshed gauze dressings help with mechanical debridement at every bandage change. Until a granulation bed-forms, the bandage should be changed at least once daily. In the early stages of healing, the bandage may need to be changed as often as twice daily. After granulation tissue develops, the bandage should be changed to a dry, non-stick dressing so the granulation bed is not disrupted. Both the granulation bed and the early epithelium are easily damaged, and disruption of the granulation bed delays wound healing [17].

With the concept of moist wound healing, bandaging is combined with autolytic debridement to promote wound healing. The use of moist wound dressings keeps white cells healthier, allowing them to aid in the debridement process. A variety of dressings is available. Alginate dressings are commonly used in the exudative wound to stimulate granulation tissue. Hydrogels are used to maintain moisture levels in drier wounds. Foam dressings may be used to absorb excessive exudate or protect granulation beds. These newer dressings are changed only every 2-5 days [21].

Sugar Dressings

Sugar has been used as an inexpensive wound dressing for more than three centuries to control odor and infection. The use of sugar is based on its high osmolality drawing fluid out of the wound and inhibiting the growth of bacteria. The use of sugar also aids in the debridement of necrotic tissue while preserving viable tissue. Granulated sugar is placed into the wound cavity in a layer 1-cm thick and covered with a thick dressing to absorb fluid drawn from the wound. The sugar dressing should be changed once daily or more frequently whenever "strike-through" is seen on the bandage. During the bandage change, the wound should be liberally lavage with warm saline or tap water. Sugar dressings may be used until granulation tissue is seen. Once all infection is resolved, the wound may be closed or allowed to epithelialize. Because a large volume of fluid can be removed from the wound, the animals' hemodynamic and hydration status must be monitored and treated accordingly. Hypovolemia and low colloid osmotic pressure are complications that may be associated with this therapy [22].

Honey Dressings

Honey has also been used for wound dressings over the centuries. Honey's beneficial effects are thought to be a result of hydrogen peroxide production from the activity of the glucose oxidase enzyme. The low pH of honey also may accelerate healing. Honey used for wound healing must be unpasteurized, and the source of the honey appears to be a factor in its effectiveness. Manuka honey may be the best option for wound care. The contact layer wound dressings should be soaked in honey before application. The bandage should be changed daily or more frequently as needed [11].

Wound Lavage

Irrigation of the wound washes away both visible and microscopic debris. This reduces the bacterial load in the tissue, which helps decrease wound complications. Assuming the solution is nontoxic, the most important factor in wound lavage is the use of large volumes to facilitate the removal of debris. The recommended lavage is a moderate pressure system using a 35-mL syringe and a 19-gauge needle that delivers lavage fluid at 8 lb/sq. in. The use of antibiotics in the lavage fluid is controversial [23].

The ideal lavage fluid would be antiseptic and nontoxic to the healing tissues. Although isotonic saline is not antiseptic, it is the least toxic to healing tissue. Surgical scrub agents should not be used because the detergent component is damaging to tissue. Dilute antiseptics can be used safely. Chlorhexidine diacetate 0.05% have sustained residual activity against a broad spectrum of bacteria while causing minimal tissue inflammation. However, gram-negative bacteria may become resistant to chlorhexidine. Stronger solutions of chlorhexidine are toxic to healing tissue. Povidone-iodine 1% is an effective antiseptic, but it has minimal residual activity and may be inactivated by purulent debris [24].

Wound Debridement

Debridement is the removal of devitalized tissue from a wound to encourage the rapid onset of the proliferative phase of wound healing. Wound debridement can be surgical, enzymatic, mechanical, or hydrodynamic [25]. Enzymatic debridement agents may be indicated for wounds where adequate surgical debridement is not possible or in locations such as distal limbs where excessive debridement of healthy tissues should be avoided. Properly used enzymatic agents dissolve wound exudate, coagulum, and necrotic debris without directly harming living tissue. Bacteria lose their protective proteinaceous and nuclear material and are exposed to the effects of cellular and humoral immunity and antimicrobial agents [17].

Advantages include the ability to apply enzyme solutions without anesthesia and to use them in areas with important structures such as nerves and tendons [2]. Applying wet saline bandages over the wound will enhance the enzymatic action. Dis advantages include expense, the time required for adequate debridement, frequency of dressing changes, and potentially insufficient debridement of burned skin [1].

Antiseptics

The ideal wound antiseptic is effective against likely contaminants and pathogens, fast-acting with prolonged residual activity after a single dose, nontoxic. noncarcinogenic and nonteratogenic to host cells, nonallergenic, inexpensive, widely available, incapable of promoting bacterial resistance, and has minimal systemic absorption (Waldrom and Trevor). Chlorhexidine (CHD) and Povidone-iodine (PI) are the most common and effective antiseptics used in veterinary medicine [1,2].

Chlorhexidine

Chlorhexidine has a wide spectrum of antibacterial activity, good residual activity, and low systemic absorption and toxicity. CHD is available as acetate, gluconate, or hydrochloride salts CHD diacetate as a

0.05% aqueous solution, significantly reduces bacterial populations in a contaminated wound without increasing tissue inflammation [17]. CHD has a long residual activity, even in the presence of organic matter, as it binds to proteins in the stratum corneum leaving a persistent residue for at least 48hr. Higher concentrations result in compromised wound epithelialization, granulation tissue formation, and wound contraction, and decreased tensile strength. CHD diluted in electrolyte solutions results in the formation of a precipitate within 4 hr but the precipitate does not delay wound healing or affect the efficacy of CHD as an antiseptic. Gram-negative organisms, such as Proteus and Pseudomonas, have developed a resistance or have an inherent resistance to CHD. *In vitro* studies have shown that 0.05% CHD is 100% lethal to *Staphylococcus intermedius,* epidermal cells and fibroblasts.-*in vitro* comparisons are not reliable indicators of *in vivo* efficacy as bacteria have developed better mechanisms for survival in an abnormal environment and host cells in cell cultures are more susceptible to toxic insults. CHD can cause acute contact dermatitis, synovitis, and synovial ulceration if used as joint lavage and ototoxicity if used to lavage the middle ear [1,17].

Povidone-Iodine (PI)

PI is an iodine solution containing free iodine and Polyvinylpyrrolidone (PVP). The bactericidal activity of PI is proportional to the concentration of free iodine. PVP has no antibacterial activity but its affinity for cell membranes enhances the efficacy of free iodine and reduces the staining, instability, and tissue irritation associated with Gram-negative and positive bacteria, Candida, and fungi. Bacterial resistance to iodine has not been identified. PI has a residual activity of only 4 to 6 hr and hence requires a frequent application. It is inactivated by organic matter and hence adequate debridement and irrigation are required for effective antisepsis [2].

In vitro, PI results in fibroblast and leukocyte cytotoxicity, inhibited neutrophil migration, reduced lymphocyte blastogenesis, and limited granulocyte and monocyte viability. PI can cause acute contact dermatitis, metabolic acidosis, thyroid dysfunction, and ototoxicity [17].

Other Antiseptics

Other skin antiseptics include alcohol, sodium hypochlorite (or Dakin's solution). Quaternary ammonium compounds, acetic acid, hydrogen peroxide, and silver nitrate. These antiseptics do not have the broad-spectrum efficacy or wide margin of safety of CHD or PI [22].

Topical Antibiotics and Sulphonamides

The use of topical antimicrobials is controversial. The Potential Advantages of these agents over antiseptics include selective bacterial toxicity efficacy is not reduced in the presence of organic matter and combined efficacy with systemic antimicrobial therapy [25]. They are proposed to promote normal healing by protecting the wound from superficial infection. Potential disadvantages include expense, reduced antimicrobial spectrum, the potential for bacterial resistance, the creation of superinfections, and increased nosocomial infections. Important considerations in the selection of topical drugs include the antimicrobial spectrum, dose, pharmacokinetics, tissue and systemic toxicity timing, route of administration, and type of preparation

(lavage, ointment, cream, or powder). Topical and systemic antibiotics have less benefit once the infection has become established as the presence of wound coagulum prevents antibiotics from reaching effective levels in deep tissues and systemic antibiotics from reaching superficial bacteria [22].

Wound Dressings

The purpose of bandaging is to minimize hematoma and edema formation, reduce dead space, protect against additional contamination or trauma, absorb drainage, establish adequate oxygen tension, maintain a moist environment, and minimize motion. A moist environment encourages angiogenesis which is essential for the delivery of cellular components for wound healing [18].

Adherent Wound Dressings

Wounds in the inflammatory phase will require adherent dressings to remove necrotic debris, foreign matter, and viscous exudate. Bandage material adheres to wounds when granulation tissue penetrates the interstices of the dressing [26]. Fibrous and capillary invasion entrap the primary layer and proteinaceous exudate and necrotic debris penetrate the bandage [2].

The degree of adherence depends on the size of the interstices in the dressing material. Wide mesh gauze results in better adherence and debridement. Adherent dressings may be applied wet or dry depending on the nature of the exudate and degree of debridement required [22]. The wet-to-dry adherent dressing is most commonly used. Sterile iodine is used as a wetting agent, and soluble medications, antibiotics, enzymes, and/or antiseptics may be added. The dry-to-dry bandage is indicated for low viscosity exudates. These dressings will disrupt granulation tissue and hence should only be used during the debridement phase [18].

Non-Adherent Dressings and Semi Occlusive Dressings

A nonadherent dressing should be used when the wound is in the reparative stage of healing with the formation of granulation tissue and the production of a more sanguineous exudate. Non-adherent dressings are either semi-occlusive or occlusive. Nonadherent dressings have either an absorptive secondary layer or are natural or synthetic fibers impregnated with petroleum or polyethylene glycol (Swaim and Wilhall, 1984).

Petroleum-based dressings (Jelonet and Bactigras, Smith, and Nephew) are inert, non-toxic, non-sensitizing, no irritating, and water-insoluble, thereby maintaining permanent lubricity and permitting no painful removal. Petroleum-based dressings increase wound contraction and result in the absorption of bacteria and exudate from full-thickness skin wounds on dogs. However, they may delay epithelisation [2].

Occlusive Dressings

Fully occlusive dressings are used for Healthy wounds in the repair phase where exudation is minimal. They are broadly classified as biological or synthetic [25]. Occlusive dressings require less frequent Changing and will accelerate epithelialization by up to 50% and protect the new epithelium from abrasion. They act as a physical barrier to contamination by bacterial pathogens, stimulate collagen synthesis, and reduce fluid loss

from wounded tissues. Occlusive dressings are thin, transparent, and biodegradable, and adhere to the surrounding skin but not the wound. Careful clipping around the wound is required for effective adhesiveness (Swaim and Wilhall, 1984). Occlusive dressings can be changed every 5 to 14 days. However, retained moisture may lead to bacterial contamination, tissue maceration, and bandage separation [25,27,28].

Conclusion and Recommendations

The effective management of wounds will reduce the number of complications and allow rapid return to normal function. The wound should be handled with an aseptic technique, thoroughly irrigated under adequate pressure, and judiciously debrided. Debridement should be delayed if tissue viability is questionable. The use of antibiotics or antiseptics in lavage solutions is debatable but should be avoided unless the infection is likely, such as in bite wounds or burns. Aqueous chlorhexidine is the preferred antiseptic solution. The wound should be protected with dressings that are chosen according to the stage of healing. Adherent dressings, usually wet-to-dry, should be used during the inflammatory stage. Alginate dressings may be used during the transition from the inflammatory stage to the proliferative phase when wound exudation is decreasing. Petroleum and then polyethylene glycol-based semi-occlusive, non-adherent dressings are effective during the early proliferative phase. Thus, the occlusive hydrogels are particularly useful and recommended during this phase. Wounds in difficult areas or with inherent poor healing qualities may require specialized topical applications or dressings. Honey is effective, especially on extensive shearing injuries. The use of growth factors to enhance wound healing is a rapidly expanding field and may play a significant role in wound management in the future.

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