An overview of the topical management of wounds

JM LIPTAK
Department of Companion Animal Medicine and Surgery, The University of Queensland, Queensland 4072

Wounds in animals are a common and frequent reason for seeking veterinary attention. The way in which wounds are managed affect the rate of healing, the time to return to normal function, the final cosmetic appearance, and hence the satisfaction of customers. The management of wounds depends on the stage of wound healing and can include irrigation, mechanical and chemical debridement, the use of antiseptics and antimicrobials, adherent and nonadherent dressings, and miscellaneous topical applications such as aloe vera, honey and live yeast cell derivative. The advantages, disadvantages and indications for initial wound management, topical applicants and dressings are discussed.
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PI	Povidone iodine
CHD	Chlorhexidine
PVP	Polyvinylpyrrolidone

Wound healing is divided into three phases: inflammatory, proliferative, and remodelling.[1] An understanding of the process of wound healing is essential for 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.[1] Manufacturer's instructions and veterinary literature are important sources of information for the appropriate use, concentration, frequency of administration, and mode of application of topical agents.[1]

Classification of wounds

Wounds can be broadly classified as open or closed. Open wounds include abrasions, avulsions, ballistic and penetrating wounds, hernias, lacerations, and excised or surgical wounds.[2] Further, woulds can be classified as clean, clean-contaminated, contaminated or infected (see Box).[1,2]

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.[3] The aim of any therapy is to facilitate wound healing mechanisms by providing a warm, clean environment and an adequate blood supply.[4]

Wound lavage

The principal actions of wound lavage are mechanical and dilutional.[1,5] Wound irrigation reduces bacterial numbers and removes necrotic tissue, foreign debris, exudate and wound gels.[1,2] There are many possible lavage solutions including tap water, isotonic saline, and lactated Ringer's solution. These have been combined with PI or CHD to reduce bacterial numbers, but the addition of antiseptics is considered unnecessary by many authors.[1] Wound irrigation is effective with large volumes of fluid delivered at a pressure equivalent to that obtained by spraying with a large syringe and an 18 gauge needle.[2,3] Higher pressure can result in further tissue trauma, deeper seeding of bacteria within the wound, and decreased resistance to infection.[1,2]

Wound debridement

Debridement is the removal of devitalised tissue from a wound to encourage rapid onset of the proliferative phase of wound healing.[2,5] Wound debridement can be surgical, enzymatic, mechanical or hydrodynamic.[2]

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.[5] 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.[1] Advantages include the ability to apply enzyme solutions without anaesthesia and to use them in areas with important structures such as nerves and tendons.[2,5] Wet saline bandages over the wound will enhance the enzymatic action (PR Watt personal communication). Disadvantages include expense, time required for adequate debridement, frequency of dressing changes, and potentially insufficient debridement of burned skin, necrotic bone and connective tissue.[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 avail-

Classification of wounds and basic treatment options

Clean	Nontraumatic, aseptic surgical wound	Primary closure
Clean-contaminated	Surgical wound entering a viscus	Primary closure
Contaminate	Open traumatic wound or incision in area of nonpurulent inflammation	Delayed primary closure
Dirty	Open traumatic wound with infection	Secondary intention healing

able, incapable of promoting bacterial resistance and has minimal systemic absorption.[1,2] CHD and PI are the most common and effective antiseptics used in veterinary medicine (see Box).

Types of antiseptics, recommended concentration, and spectrum of activity

Chlorhexidine	0.05%	Gram-positive and some Gram-negative bacteria
Povidone Iodine	1%	Gram-positive and -negative bacteria, and fungi
Sodium hypochlorite	0.125 to 0.25%	Gram-positive and -negative bacteria, fungi and viruses
Quaternary ammoniums	0.002 to 0.007%	Gram-positive bacteria, fungi, protozoa and viruses
Acetic acid	0.25 to 0.5%	Gram-positive and negative bacteria
Hydrogen peroxide	1 to 3%	Bacterial spores
Silver nitrate	0.5%	Gram-positive and some Gram-negative bacteria

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.[1] CHD diacetate, as a 0.05% aqueous solution, significantly reduces bacterial populations in a contaminated wound without increasing tissue inflammation.[2] 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 48 h.[6] Higher concentrations result in compromised wound epithelialisation, granulation tissue formation and wound contraction, and decreased tensile strength.[1] CHD diluted in electrolyte solutions results in formation of a precipitate within 4 h but the precipitate does not delay wound healing or affect the efficacy of CHD as an antiseptic.[7] Gram-negative organisms, such as *Proteus*, *Serratia* and *Pseudomonas*, have developed a resistance or have an inherent resistance to CHD.[1,2] In vitro studies have shown that 0.05% CHD is 100% lethal to *Staphylococcus intermedius*, epidermal cells and fibroblasts.[7] 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.[1] CHD can cause acute contact dermatitis,[1] synovitis and synovial ulceration if used as a joint lavage,[6] and ototoxicity if used to lavage the middle ear.[1]

Povidone iodine

PI is an iodine solution containing free iodine and PVP. The bacteriocidal activity of PI is proportional to the concentration of free iodine.[1,6] 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 free iodine.[1] PI has good antimicrobial activity against Gram-positive and Gram-negative bacteria, *Candida* and fungi.[6] Bacterial resistance to iodine has not been identified.[1] PI has a residual activity of only 4 to 6 h and hence requires frequent application.[1] It is inactivated by organic matter and hence adequate debridement and irrigation is required for effective antisepsis.[6] In vitro, PI results in fibroblast and leukocyte cytotoxicity, inhibited neutrophil migration, reduced lymphocyte blastogenesis and limited granulocyte and monocyte viability.[1,6] PI can cause acute contact dermatitis, metabolic acidosis, thyroid dysfunction, and ototoxicity.[1] Detergents formed by combining surfactants with PI are deleterious to wound tissue and potentiate infection.[6]

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.[1]

Topical antibiotics and sulphonamides

The use of topical antimicrobials (see Box) is controversial.[1] 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.[1] They are proposed to promote normal healing by protecting the wound from superficial infection. Potential disadvantages include expense, reduced antimicrobial spectrum, potential for bacterial resistance, creation of superinfections and increased nosocomial infections.[1] Important considerations in the selection of a 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).[1] Topical and systemic antibiotics have less benefit once 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.[1]

Topical antimicrobials are indicated prior to the development of the granulation tissue bed as they may prevent or control infection while devitalised tissue and foreign material are still present in the wound. Epithelialisation may be delayed by topical antimicrobials with a petroleum base.[1,2] In vitro studies have shown that topical antimicrobials administered at bacteriocidal concentrations are either cytotoxic or impair local cell function. *Staphylococcus* and *Streptococcus* spp are the most common pathogens found in traumatic and postoperative wounds but Gram-negative and mixed infections are possible.[1]

Beta lactams

Cephazolin (Kefzol, Eli Lilly), locally applied at 20mg/kg provides high concentrations of antibiotic in wound fluid above the mininum inhibitory concentration for a longer time than systemic cephazolin at the same dose rate.[1] Powdered cephazolin has been used topically to provide higher and more prolonged tissue concentrations than solutions.[1] Other antibiotics are poorly absorbed from topical sites and are less effective in treating established infection.[1]

Bacitracin-polymyxin and B-neomycin

The triple combination of bacitracin, polymyxin B and neomycin (Neosporin Topical, Glaxo Wellcome) has a wide range of antimicrobial activity, especially against Gram-positive organisms, but is ineffective against *Pseudomonas aeruginosa*.[8] The zinc component of bacitracin stimulates re-epithelialisation of partial thickness wounds in pigs but can retard wound contraction.[8] These antimicrobials

are poorly absorbed and hence systemic toxicity is rare.[6] Toxicities include ototoxicity, neurotoxicity and nephrotoxicity.[8]

Silver sulphadiazine

Silver sulfadiazine (Silvazine, Smith and Nephew) has a broad spectrum of activity against many bacteria, especially *Pseudomonas* spp and fungi.[1] It enhances epithelialisation in pigs and mice, and is a carrier agent for topical growth factor administration.[1] This agent has in vitro toxicity to human keratinocytes and fibroblasts, and inhibits the function of polymorphonuclear cells and lymphocytes.[9]

Other antimicrobials

Other antimicrobials used in open wound management include gentamicin and nitrofurazone.[6]

Antimicrobials available for topical wound management and their spectrum of activity

Cephazolin	Gram-positive and some Gram-negative bacteria
Bacitracin-polymyxin B-neomycin	Gram-positive and -negative bacteria, not *Pseudomonas* spp
Silver sulphadiazinine	Gram-negative and some Gram-positive bacteria and fungi
Gentamicin	Gram-negative bacteria
Nitrofurazone	Gram-positive and -negative bacteria, not *Pseudomonas* spp

Types of wound dressings and their indications

	Adherent dressings
Wet to dry	Inflammatory stage, high viscosity exudate
Dry to dry	Inflammatory stage, low viscosity exudate
	Nonadherent dressings
	Semiocclusive dressings
Calcium/calcium-sodium alginate	Transition from inflammatory to reparative stages
Petroleum impregnated	Early reparative stage, viscous to sanguineous exudate
Polyethylene glycol	Early to mid-reparative stage, sanguineous exudate
	Occlusive dressings
Polyurethane film	Partial thickness injuries or hydrogel covering
Hydrocolloid	Healthy granulation tissue bed with low exudate
Hydrogel	Reparative stage
Hydrophilic	Deep granulating wounds or high exudate or transudate
Foam	Deep wounds with low exudate
Biologic	Reparative stage

Wound dressings

The purpose of bandaging is to minimise haematoma and oedema formation, reduce dead space, protect against additional contamination or trauma, absorb drainage, establish adequate oxygen tension, maintain a moist environment and minimise motion.[10] A moist environment encourages angiogenesis which is essential for the delivery of cellular components for wound healing.[10] Various types of would dressings are available (see Box).

Adherent dressings

Wounds in the inflammatory phase will require adherent dressings to remove necrotic debris, foreign matter and viscous exudate.[1,11] Bandage material adheres to wounds when granulation tissue penetrates the interstices of the dressing. Fibrinous and capillary invasion entrap the primary layer and proteinaceous exudate and necrotic debris penetrate the bandage.[1,5] The degree of adherence depends on the size of the interstices in the dressing material. A wide mesh gauze results in better adherence and debridement.[11] Adherent dressings may be applied wet or dry depending on the nature of the exudate and degree of debridement required.[11]

The wet-to-dry adherent dressing is most commonly used. Sterile saline is used as a wetting agent, and soluble medications, antibiotics, enzymes and/or antiseptics may be added.[4] The dry-to-dry bandage is indicated for low viscosity exudates.[5,11] These dressings will disrupt granulation tissue and hence should only be used during the debridement phase.

Nonadherent dressings

Semiocclusive dressings

A nonadherent dressing should be used when the wound is in the reparative stage of healing with formation of granulation tissue and production of a more sanguineous exudate.[1] Nonadherent dressings are either semiocclusive or occlusive. Nonadherent dressings have either an absorptive secondary layer or are natural or synthetic fibres impregnated with petroleum or polyethylene glycol.[11] Petroleum-based dressings (Jelonet and Bactigras, Smith and Nephew) are inert, nontoxic, nonsensitising, nonirritating and water insoluble, thereby maintaining permanent lubricity and permitting nonpainful removal.[11,12] Petroleum-based dressings increase wound contraction and result in absorption of bacteria and exudate from full thickness skin wounds on dogs. However, they may delay epithelisation.[11,12]

Calcium or calcium-sodium alginate dressings

Calcium alginate dressings (Kaltostat, BritCair; Sorbsan, Steriseal) are flat, nonwoven pads of either calcium-sodium alginate fibre or pure calcium alginate fibre. They are salts of alginic acid extracted from certain species of brown seaweeds. These dressings are used during the transition from debriding dressings to hydrocolloids.[5] They form a gel via ion exchange when they absorb wound exudate, and encourage epithelialisation and granulation. Calcium contributes to the clotting mechanism. They are not occlusive[5,10] and useful in deep, soft tissue cavities and fistulas.[10]

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.[12] Occlusive dressings require less frequent changing and will accelerate epithelialisation by up to 50% and protect the new epithelium from abrasion.[5,9,10] They act as

a physical barrier to contamination by bacterial pathogens, stimulate collagen synthesis and reduce fluid loss from wounded tissues.[13] Occlusive dressings are thin, transparent and biodegradable, and adhere to the surrounding skin but not the wound.[12] Careful clipping around the wound is required for effective adhesiveness.[11] Occlusive dressings can be changed every 5 to 14 days. However, retained moisture may lead to bacterial contamination, tissue maceration and bandage separation.[12]

Polyurethane films

Polyurethane films (Allevyn, Smith and Nephew; Tegaderm, 3M) are used on partial thickness dermal injuries or as coverings for hydrogels, hydrophilic pastes or powders.[1] They are waterproof, semipermeable to vapour, transparent, adhesive and analgesic.[10] Polyurethane films are indicated for wounds where granulation tissue is established and wound exudate is declining.[1] They are contraindicated in infected wounds or wounds with copious drainage.[1] Advantages include high conformability, effectiveness for superficial wounds and transparency.[10] Disadvantages include potential for channelling of bacteria to the wound site, periwound maceration and damage to new epithelial tissue when removed.[10] Polyurethane films are changed every 2 to 3 days or when exudate has accumulated.[10]

Hydrocolloid dressings

Hydrocolloid dressings (Granuflex, Convatec; Tegasorb, 3M) are suspensions of starch polymers in an adhesive matrix. They are indicated for the protection of healthy granulating wounds.[1,5] Hydrocolloid dressings become nonadherent gels when they contact a moist wound surface and form a barrier between the wound and the dressing.[4,10] Although the polyurethane backing is water resistant, it allows evaporation as it is permeable to water vapour, oxygen and carbon dioxide. It is not permeable to bacteria and water.[10] Wounds dressed with hydrocolloid material epithelialise more rapidly but wound contraction is inhibited due to greater production of wound exudate.[5,10] Hydrocolloids increase epidermal healing by 30 to 36%.[10] These dressings are indicated for pressure sores, minor burns, granulating wounds, cavity wounds, wounds with slough or necrotic tissue, and wounds with moderate exudate.[1]

Hydrogel dressings

Hydrogels (Intrasite Gel, Smith and Nephew) are thin composites of hydrogel adhered to a fine mesh, thin synthetic fibre, or as a paste in which water is the major constituent of the dispersal phase.[11] They are composed of insoluble hydrophilic polymers that absorb variable amounts of wound fluid, permit autolytic debridement at the wound surface, are easy to apply, encourage a moist wound environment, and are compatible with topical agents.[10] Hydrogels increase the collagenase activity in second degree burns. This activity is further enhanced by pulsed electrical stimulation.[14] These dressings are indicated for flat granulating surfaces such as shallow abrasions, blisters and superficial wounds,[10] whereas gel pastes are better for deep defects in the repair phase.[1]

Hydrophilic dressings

Hydrophilic beads, flakes, powders and pastes are polymers which absorb large amounts of wound exudate or transudate and are indicated for deep granulating defects.[1] Hydrophilic agents pull body fluids through the wound tissues to bathe them from the inside.[6] Particulate matter, micro-organisms and plasma proteins are absorbed from the wound surface as capillary flow draws fluid from the wound between the beads.[6] The beads absorb electrolytes and prostaglandins.[6] They may also activate chemotactic factors which attract polymorphonuclear and mononuclear cells.[6] Examples are Debrisan (Johnson and Johnson), Avalon Copolymer Flakes (Summit Hill Laboratories), Intracell (Technivet) and Intrasite Cavity Filler (Smith and Nephew).

Foam dressings

Foam dressings (LYOfoam, Ultra; Allevyn cavity, Smith and Nephew) are cavity wound fillers which have good conformability and provide physical protection and comfort.[4] They are made from polyurethane and are often backed by a semipermeable film with a nonadherent contact surface.[10] The dressings are thin and only absorb a small amount of wound exudate.[10] They are indicated for moderate exudative wounds and wounds in difficult locations such as the inguinal and axillar areas.[1]

Biological dressings

Biologic dressings are pliable, reduce pain, stimulate epithelialisation and collagen synthesis, and increase wound contraction.[1] A porous bovine-derived collagen membrane has been found to be ineffective on the epithelialisation and contraction of open wounds on horse extremities.[15] Equine amnion prevents fluid, protein and electrolyte losses from wounded tissue, decreases pain at the wound site, promotes an earlier return to normal function, decreases bacterial numbers in wound tissue and stimulates re-epithelialisation.[13] Amniotic fluid contains allantoin and lysosome, facilitates bacterial clearance and may also contain an intrinsic bacteriocidal compound.[13] Angiogenic or growth factors in amnion are postulated to accelerate healing.[13] Amnion dressings are not commercially available but can be prepared with 0.05% CHD diacetate in sterile water and stored in a 0.025% CHD solution.[13]

Other topical agents

Live yeast cell derivative

Live yeast cell derivatives, a water soluble extract of brewer's yeast, increases wound oxygen consumption, angiogenesis, epithelialisation and collagen synthesis in human wounds.[1,6] They are used in wounds with healthy granulation tissue and in the proliferative stage of wound healing.[16] In horses, they have prolonged healing time by delaying epithelialisation and inhibiting contraction. However, in dogs granulation tissue epithelialises more quickly.[6]

Honey

Honey or sugar paste has been used by veterinary and medical practitioners to treat chronic, nonhealing and infected wounds. Proposed advantages include wound debridement, reduction of oedema, antibacterial activity, promotion of granulation tissue and epithelialisation, improved wound nutrition, and wound deodourisation.[1] Honey-treated wounds show little neutrophilic infiltration and marked proliferation of angioblasts and fibroblasts.[17] The high content of glycine, methionine and proline in honey results in a higher collagen and hydroxyproline concentration in healing tissues.[17] Honey has antimicrobial properties due to production of hydrogen peroxide by enzymatic oxidation of glucose, the presence of inhibin, hypertonicity, low pH and

unidentified floral sources.[17,18] Manuka honey, floral honey and lime honey have all been associated with better healing than commercial honey and honey from sugar fed bees.[18] Manuka honey does not have hydrogen peroxide activity.[18] Honey is an excellent energy source which may enhance the healing process.[17]

Aloe vera

Aloe vera has antibacterial activity against *Pseudomonas aeruginosa*.[19] It stimulates fibroblast replication and has antiprostaglandin activity against thromboxane A_2 which is produced in burned dermal tissue and pressure sores.[6,8] Allantoin and acemannan are components of the topical aloe vera extract gel. Allantoin has been reported to enhance epithelialisation in suppurating wounds and resistant ulcers.[8] Acemannan stimulates macrophages to produce the cytokines interleukin 1 and tumour necrosis factor which, in turn, stimulate angiogenesis, epithelialisation and wound healing.[19] Aloe vera has anti-inflammatory and analgesic activity due to the presence of a salicylate-like substance which has precluded its use in full thickness wounds, especially during the inflammatory phase.[6]

Growth factors

Growth factors are cytokines that are released normally during the inflammatory process of healing and are produced by many cells including platelets, macrophages, lymphocytes, neutrophils, fibroblasts, and epithelial cells.[20,21] A variety of growth factors have been studied (see Box).[1,20] Growth factors enhance repair of poorly healing wounds such as radiation injuries, pressure sores and wounds affected by endogenous or exogenous corticosteroids.[21]

Miscellaneous agents

Tripeptide and tetrapeptide copper complexes are chemotactic for mast cells, have a stimulatory effect on fibroblasts resulting in increased collagen synthesis and stimulate angiogenesis.[22] They enhance the rate of healing, epithelialisation and wound closure.[22] Zinc-deficient animals have a delayed closure and reduced tensile strength in the first 7 days of healing.[8] Administration of zinc to deficient animals results in enhanced wound epithelialisation, increased tensile strength and synthesis of collagen and other proteins.[8]

Chitosan, a preparation isolated from Atlantic krill shells, stimulates granulation and epithelialisation of open wounds in animals and has antipruritic properties.[23] The application of glucan to open wounds on rabbits promoted macrophage migration into the wound site, granulation tissue formation and epithelialisation.[24] Topical nitroglycerin has been used for benign anal disease in humans for the reduction of pain associated with internal anal sphincter hypertonia.[25] Vitamin A soaked gelfoam sponges increase wound breaking and tensile strength in steroid treated rats.[26] Phenytoin has a biphasic action where it only stimulates cell proliferation and collagenase activity if fibroblasts are in a nonretracting wound under tension.[27]

One study investigated the healing of porcine skin wounds of partial thickness in a liquid environment and reported less inflammation and scar formation without tissue maceration in comparison to air exposed wounds.[28] Hyperbaric oxygen stimulates angiogenesis and increases bursting strength of wounds.[29] Promoting early angiogenesis may provide the metabolic environment necessary at the cellular level to boost proliferation and synthetic activity of wound fibroblasts.[29]

Some of the growth factors involved in the wound healing process

Epidermal growth factor	Epidermal growth factor-like peptide
Platelet derived growth factor	Nerve growth factor
Transforming growth factor α	Transforming growth factor β
Insulin-like growth factor	Basic fibroblast growth factor

Summary of recommendations for the management of topical wounds

- Aseptic handling of wound
- Copious wound lavage, antiseptics added if bite wound or thermal injury
- Judicious debridement
- Adherent dressing during inflammatory stage of wound healing, especially wet-to-dry dressing
- Nonadherent dressings during reparative stage of wound healing, especially petroleum semiocclusive
- Dressings and occlusive hydrogels

Conclusions

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 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 semiocclusive, nonadherent dressings are effective during the early proliferative phase. The occlusive hydrogels are particularly useful and recommended during this phase.

Wounds in difficult areas or with inherent poor healing qualities may require specialised topical applications or dressings. Honey has been found to be 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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Bilateral ureteral tears in a foal

TJ CUTLER, RJ MACKAY, CM JOHNSON and R PAPENDICK
College of Veterinary Medicine, PO Box 100136, University of Florida, Gainesville, Florida 32610, USA.

Ureteral tear is an uncommon cause of uroperitoneum in foals but should be considered when the urinary bladder and urachus are intact. Three cases of ureteral rupture have been previously reported,[1-3] but only one with histopathologic findings, and no mechanism has been described. In this report we describe the presentation of a colt with bilateral tears, report histopathological findings and review relevant literature. Recommendations for diagnosis and treatment of the condition are made.

Case report

A 12-hour-old male Appaloosa foal was referred because of multiple bite wounds inflicted by a pack of dogs. During attempts to strike one of the dogs, the dam had inadvertently kicked the foal and fractured its jaw. Before this, the foal had appeared normal and stood and suckled its dam within 2 h of birth.

At presentation, the foal was slightly depressed but standing and able to walk. Body temperature, pulse and respiratory rates were within normal limits. There was an open, comminuted fracture of the mandible involving the caudal aspect of the right body. In addition, there were multiple full-thickness skin lacerations involving the right thoracic limb and both pelvic limbs. These were 1 to 8 cm long, up to 3 cm deep and associated with subcutaneous emphysema. Haematological tests at admission showed mild haemoconcentration (PCV 0.41 L/L, total plasma protein concentration 70 g/L) and there was slight azotaemia (serum creatinine concentration 212 µmol/L); these were interpreted as prerenal azotaemia due to dehydration. Serum electrolyte concentrations were normal. Surgical repair of the mandibular fracture was not attempted, but the wounds were debrided and flushed with 0.9% saline and dressed with povidone iodine soaked gauze sponges. The dressings were changed twice daily. Antibiotic therapy was begun with amikacin and potassium penicillin G, with flunixin meglumine for analgesia and ranitidine to reduce the risk of gastric ulceration. An indwelling nasogastric tube was placed and dam's milk was fed at 200 mL/kg/day.

By day 3, the foal was bright and more active and sucking the mare frequently. On day 4 some wounds were becoming necrotic and malodourous and leukopaenia (2.3 x 10^9 cells/L) was found. Metronidazole was added to the antimicrobial regimen. Apart from hyponatraemia (125 mmol/L), serum biochemical values were normal. Treatment with flunixin was discontinued. Nine days after admission the foal began to show signs of abdominal pain, strained frequently to urinate in small volumes and was noted to have developed abdominal distention. Transcutaneous ultrasonography revealed a large amount of anechoic peritoneal