

Compendium

Dermatologic Emergencies: Identification and Treatment

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Abstract: Skin disease is one of the most common reasons dogs and cats are taken to the veterinarian. While many dermatologic conditions cause mild, localized signs, some, such as erythema multiforme, toxic epidermal necrolysis, cutaneous vasculitis, cutaneous drug eruptions, and thermal burns, can cause severe cutaneous signs and may have serious systemic consequences. These patients may present on an emergency basis and require intensive monitoring, diagnostics, and care. Lack of familiarity with these conditions may delay diagnosis and appropriate treatment. Pyotraumatic dermatitis may also prompt owners to seek emergency veterinary care due to the severe appearance of associated lesions.

Skin disease is one of the primary reasons owners take a pet to the veterinarian.¹ Signs often consist of pruritus and primary and secondary lesions (e.g., pustules, papules, crusting, alopecia). However, pets affected by severe diseases such as erythema multiforme (EM), toxic epidermal necrolysis (TEN), vasculitis, and cutaneous drug eruptions (CDE) may also develop systemic signs. These patients may present to the veterinarian on an emergency basis. There is little information in the veterinary literature about these dermatopathies. In addition, many veterinarians are unfamiliar with these diseases, making identification difficult and delaying treatment.

Erythema Multiforme

EM is an uncommon, acute, inflammatory condition of the skin and/or mucous membranes that can cause significant morbidity.² The pathogenesis has not been fully elucidated but is proposed to be a host-specific, cell-mediated hypersensitivity reaction to various antigens.³ It has been reported in dogs secondary to administration of specific drugs (TABLE 1), infection (staphylococcal dermatitis, parvovirus), neoplasia, dietary components (beef and/or soy, chicken and/or egg), and idiopathic causes.²⁻⁸ In one review of 44 cases of EM in dogs, 10 (22.8%) were idiopathic.⁷ Reported causes in cats include administration of specific drugs

(TABLE 1), herpesvirus infection, and application of d-limonene-based shampoo.^{3,9}

EM can range from mild to severe. The term *erythema multiforme minor* is used when ≤ 1 mucosal surface and $< 50\%$ of total body surface area (TBSA) are affected. *Erythema multiforme major* is similar, but > 1 mucosal surface is affected. In both forms of EM, epidermal detachment, in which the epidermis separates from the dermis, may be present and comprises $< 10\%$ of TBSA.¹⁰ Lesions vary in appearance and consist of erythematous macules or papules that spread peripherally and clear centrally, producing serpiginous or annular lesions ("target lesions"). However, such lesions are not classic target lesions as described in humans, and the use of this term in veterinary EM cases is controversial. Vesicles or bullae, urticarial plaques, or a combination of lesions may also be present.³ The center of the lesion may become necrotic and slough, leaving behind erythematous erosions and ulcers⁷ (FIGURE 1). The most commonly involved sites in dogs include the ventrum, mucocutaneous junctions, oral cavity, pinnae, and footpads. Lesions in cats are typically ulcerative or vesiculobullous and are found on the trunk and mucocutaneous junctions.³ Some animals present with these lesions; others are evaluated for systemic signs (e.g., pyrexia, inappetence, lethargy) without obvious initial skin involvement.³ Diagnostic differentials are listed in BOX 1.^{3,11} Definitive diagnosis

Table 1. Drugs Implicated in Select Dermatologic Conditions in Dogs and Cats^a

Condition	Drugs	
	Dogs	Cats
Erythema multiforme	Penicillins Cephalosporins Trimethoprim-sulfamethoxazole Diethylcarbamazine Levamisole Levothyroxine d-Limonene-based flea dip	Penicillin Amoxicillin Cephalexin Trimethoprim-sulfamethoxazole Griseofulvin Aurothioglucose Propylthiouracil
Toxic epidermal necrolysis	Levamisole Cephalexin 5-Fluorocytosine Ampicillin Flea dips	Levamisole Cephalexin 5-Fluorocytosine Ampicillin Flea dips
Cutaneous vasculitis	Itraconazole Metronidazole Trimethoprim-sulfamethoxazole Vincristine Phenobarbital	Itraconazole Metronidazole Trimethoprim-sulfamethoxazole Vincristine Phenobarbital
Cutaneous drug eruptions	Sulfonamides (especially if potentiated with trimethoprim) Penicillins Cephalosporins Levamisole Diethylcarbamazine Topical agents	Sulfonamides Penicillins Cephalosporins Topical agents

^aThis is not a comprehensive list.

necessitates a thorough history, physical examination, and skin biopsy. Single-cell keratinocyte apoptosis with lymphocyte satellitosis in all layers of the epidermis is characteristic of EM.³

Treatment is directed at removal of the underlying cause, if present (i.e., discontinue the offending drug, treat the underlying infection, and/or avoid suspect dietary antigens).⁷ A complete blood count and serum chemistry panel should be evaluated and may help determine the underlying cause. In severe cases and in patients with systemic signs, venous blood gases and blood lactate level should be measured. Hospitalization and intravenous (IV) fluid therapy consisting of crystalloids, colloids, and/or plasma to replace fluid, electrolytes, and proteins lost via large cutaneous defects may be required. Additional supportive care in the form



Figure 1. Erythematous erosions and ulcerations in a 3-year-old mixed-breed dog with erythema multiforme of unknown etiology. (A) Lesions along mucocutaneous junctions. (B) Lesions on the ventral abdomen. *Courtesy of UC Davis School of Veterinary Medicine*

of antibiotics, analgesia, nutritional support, and wound management should also be initiated in severe cases^{9,11} (TABLE 2). The use of immunosuppressive agents (e.g., glucocorticoids, cyclosporine, azathioprine) is controversial but may be beneficial in idiopathic cases in which they are not contraindicated (e.g., infection).^{3,7} A food trial with a novel or hydrolyzed protein diet should be considered in suspected idiopathic cases not responding to conventional therapy.³ Human immunoglobulin (TABLE 2) has been successful in treating refractory CDE (severe EM, TEN, Stevens-Johnson syndrome) in one cat and four dogs.¹²⁻¹⁴

Mild cases of EM may spontaneously regress over a few weeks. Chronic, idiopathic, or relapsing cases may require lifelong therapy. Patients with widespread skin lesions or extensive oral and mucosal involvement have a poor to guarded prognosis and may die of septicemia, disseminated intravascular coagulation, or other causes.^{2,4,9}

Box 1. Diagnostic Differentials for Select Dermatologic Conditions in Dogs and Cats

Erythema multiforme

- Bacterial folliculitis
- Demodicosis
- Dermatophytosis
- Urticaria
- Autoimmune diseases (pemphigus vulgaris, bullous pemphigoid, systemic lupus erythematosus)
- Vasculitis
- Thermal or chemical burn
- Epitheliotropic lymphoma
- Cutaneous drug eruptions
- Other pustular and vesicular disorders

Toxic epidermal necrolysis

- Erythema multiforme
- Cutaneous drug eruptions
- Vasculitis
- Thermal or chemical burn

- Autoimmune diseases (pemphigus vulgaris, bullous pemphigoid, lupus erythematosus)

- Epitheliotropic lymphoma

Cutaneous vasculitis

- Autoimmune diseases (pemphigus vulgaris, bullous pemphigoid, lupus erythematosus)
- Erythema multiforme
- Toxic epidermal necrolysis
- Cutaneous drug eruptions
- Cold agglutinin disease
- Frostbite

Cutaneous drug eruptions

Variable based on lesions present

Pyotraumatic dermatitis

- Superficial pyoderma
- Dermatophytosis
- Demodicosis

Toxic Epidermal Necrolysis

TEN is a rare mucocutaneous disorder affecting dogs, cats, and humans.¹² Reported causes in dogs and cats include administration of specific drugs (TABLE 1), bacterial infections, hepatopathy, neoplasia, and idiopathy. Although the exact pathophysiology is unknown, an immunologic role is suspected.^{4,15}

Typically, patients initially develop systemic signs (pyrexia, lethargy, inappetence) with little dermal involvement. Early erythema is followed by vesicles, bullae, necrosis, and ulceration. Lesions may affect skin diffusely over the body, although the face and extremities are more commonly involved. Mucocutaneous junctions, mucosae (e.g., oral, gastrointestinal, rectal, conjunctival, tracheal), and footpads may also be affected. A positive Nikolsky sign (separation of the superficial layer of the epidermis from the basal layer with gentle digital pressure) and significant pain are usually present.^{2,4,15,16} BOX 1 lists diagnostic differentials.^{4,11,15} Skin biopsy is required for definitive diagnosis; histopathology reveals full-thickness coagulation necrosis of the epidermis with minimal dermal inflammation.¹⁵

Treatment is similar to that of EM (TABLE 2) and is aimed at the underlying cause. Extensive dermal involvement results in massive fluid, electrolyte, and plasma protein losses. Supportive care akin to that for burn patients (aggressive fluid replacement, antimicrobial therapy, wound care, analgesia, nutritional support) is required.^{15,17}

Decreased effective circulating volume is possible secondary to fluid losses, and perfusion parameters (venous blood gas, lactate, blood pressure, urine output, heart rate) should be monitored. Use of glucocorticoids is controversial. Human immunoglobulin has been used successfully.^{12,13} Resolution of signs may take 2 to 3 weeks if the underlying cause is corrected.¹⁵ However, the prognosis is often guarded to poor and depends on the severity of the lesions. Mortality rates in humans can be as high as 30% to 50%, with sepsis being the primary cause of death.^{3,4,17}

Cutaneous Vasculitis

Vasculitis is an uncommon disorder that causes inflammation within blood vessel walls, resulting in disruption of the vessel architecture, altered blood flow, and ischemic injury to end tissues.^{18,19} In most cases, the proposed pathophysiology involves a type III hypersensitivity reaction with immune complex deposition along the endothelium. A type I reaction may be important during the initial stages of the disease. Activation of the complement cascade results



Figure 2. Crusts and erosions in a dog with cutaneous vasculitis of unknown etiology. Courtesy of Dr. Randy Thomas, Southeast Veterinary Dermatology and Ear Clinic



Figure 3. Erythema, crusts, erosions, and ulcerations along the ear pinna margins in a dog with cutaneous vasculitis. Courtesy of Dr. Thomas



Table 2. Treatment and Supportive Care of Selected Dermatologic Emergencies

Therapy	Drug	Dosage	Indication for Use	Disease/Condition
Immunosuppressants	Prednisone	2–4 mg/kg PO q24h	Immunosuppression; reserved for idiopathic cases of EM/TEN	EM, TEN, CV
	Cyclosporine ^a	5–10 mg/kg PO q12–24h		
	Azathioprine ^b	2.2 mg/kg PO q24h (dogs)		
	Dapsone ^b	1 mg/kg PO q8h ²⁰ (dogs)		
	Sulfasalazine ^b	20–40 mg/kg PO q8h ²⁰ (dogs)		
Antibiotics ^c	Amoxicillin-clavulanic acid	13.75 mg/kg PO q12h	Prevent secondary infection/septicemia in patients with compromised epidermal barrier	EM, TEN, CDE, burns
	Ampicillin-sulbactam	22–30 mg/kg IV q8h		
	Cephalexin	22 mg/kg PO q12h ¹³		
	Cefazolin	20 mg/kg IV q8h		
	Enrofloxacin	5 mg/kg PO, IV q24h		
Crystalloids	<ul style="list-style-type: none"> Plasma-Lyte (Baxter Healthcare) 0.9% NaCl Lactated Ringer solution Normosol (Hospira) 	<ul style="list-style-type: none"> 90 mL/kg/h: shock (dogs) 60 mL/kg/h: shock (cats) Weight (kg) × % estimated dehydration: replacement 60–66 mL/kg/d: maintenance 	Hypovolemic/maldistributive shock; dehydration; maintain effective circulating volume	EM, TEN, CDE, burns
Colloids	<ul style="list-style-type: none"> Hetastarch Pentastarch Dextran-70 	<ul style="list-style-type: none"> 5–15 mL/kg: shock (dogs) 5–10 mL/kg: shock (cats) 10–20 mL/kg/d: maintenance 	Hypovolemic/maldistributive shock; hypoproteinemia, low colloidal oncotic pressure	EM, TEN, CDE, burns
Opioids	Fentanyl	2–5 µg/kg/h IV or transdermal patch	Analgesia	EM, TEN, CDE, burns
	Hydromorphone	0.05–0.2 mg/kg IV q4–6h		
	Buprenorphine	0.005–0.02 mg/kg IV, buccal (cats) q6–12h		
Nutrition	<ul style="list-style-type: none"> Enteral (nasoesophageal, nasogastric, esophageal) Parenteral (PPN, TPN) 		Supportive care; promote wound healing	EM, TEN, CDE, burns
Other	Pentoxifylline	10–15 mg/kg PO q8h ¹³	Immunomodulation; antiinflammatory; antithrombotic	CV
	Human IVIG	1 g/kg IV over 4h ^{12,13}	Immunomodulation	EM, TEN

CDE = cutaneous drug eruption, CV = cutaneous vasculitis, EM = erythema multiforme, IVIG = intravenous immunoglobulin, PPN = partial parenteral nutrition, TEN = toxic epidermal necrolysis, TPN = total parenteral nutrition.

^aBioequivalent to Neoral (Novartis; modified cyclosporine).

^bNot recommended for use in cats due to the possibility of serious adverse effects.

^cIdeally, choice of antibiotics should be based on cytology at a minimum, but also on results of culture and sensitivity testing, if possible.

in endothelial necrosis, thrombosis, vascular occlusion, hemorrhage, and ischemia of recipient tissues.¹⁸ The skin and visceral organs may be affected. Causes of cutaneous vasculitis include administration of specific drugs (TABLE 1), vaccines (most commonly rabies), infections (viral [feline immunodeficiency virus, feline leukemia virus, feline infectious peritonitis], rickettsial [Rocky Mountain spotted fever, ehrlichiosis, borreliosis], bacterial, parasitic [diro-

filariasis]), food hypersensitivity, insect bites, and neoplasia.^{18–20} At least 50% of veterinary cases are idiopathic.^{18,20}

Cutaneous signs are variable: erosions, ulceration, crusting, palpable purpura, edema, plaques, alopecia, and necrosis.^{18–20} The extremities, ear tips, tail, footpads, oral mucosa, and nasal planum are commonly affected^{19,20} (FIGURE 2 and FIGURE 3). Systemic manifestations such as pyrexia, lethargy, anemia, gastroenteritis,

Key Points

- Serpiginous or annular lesions are characteristic of erythema multiforme.
- Successful treatment of erythema multiforme, toxic epidermal necrolysis, cutaneous vasculitis, cutaneous drug eruptions, and pyotraumatic dermatitis requires removal of the underlying cause or drug.
- Supportive care for severe erythema multiforme, toxic epidermal necrolysis, cutaneous drug eruptions, and thermal burns consists of aggressive fluid therapy (crystalloids, colloids), antibiotics, wound management, analgesia, and nutritional support.
- Fleas and flea-bite hypersensitivity are the most common causes of pyotraumatic dermatitis.

glomerulonephritis, myopathy, and arthropathy may develop.^{18,20} Diagnostic differentials include autoimmune diseases and other dermatologic emergencies^{11,20} (**BOX 1**). **Definitive diagnosis is based on skin biopsy and strong clinical suspicion.**¹⁸ Early lesions (within 8 to 24 hours of onset) are the most diagnostic, as chronic lesions may only show remnant vessels with little active inflammation.^{19,20} A thorough history is important for assessing drug or vaccine administration and exposure to infectious agents. To evaluate for specific etiologies, a minimum database (complete blood count, serum chemistry panel, urinalysis) should be performed. Spe-

cial testing for autoimmune diseases, coagulopathy, and infectious diseases (serology and DNA antigen testing) may be required.

Therapy centers on removal or treatment of the underlying cause and immunomodulation (**TABLE 2**).^{18–20} Systemic glucocorticoids may be required for resistant or idiopathic cases. Pentoxifylline has been successful, presumably due to its hemorrheologic, immunomodulatory, antiinflammatory, and antithrombotic properties.^{18,21} Dapsone, sulfasalazine, tetracycline/niacinamide, cyclophosphamide, chlorambucil, and azathioprine have also been used.^{19,20} The prognosis depends on the extent of visceral involvement and the underlying etiology.²⁰

Rabies vaccine-induced vasculitis has been reported.^{18,20} It is characterized by a focal cutaneous vasculitis at the site of vaccination and occurs primarily in toy breed dogs with long hair growth cycles. Clinical signs include roughly annular areas of variable alopecia, hyperpigmentation, and, less commonly, scaling or erythema overlying a variably indurated dermis and subcutis. The caudal or lateral thigh, cervical region, or shoulder is typically affected. Lesions generally appear 2 to 6 months after subcutaneous administration of the vaccine and persist for months to years. Lesions may also develop at other classic vasculitis sites yet still be associated with the typical localized process.^{22–24} Lesions may respond to pentoxifylline therapy with up to 75% reduction in lesion size; however, they may also spontaneously resolve.^{20,24} Other specific vasculopathies include juvenile dermatomyositis, familial cutaneous vasculopathy of German shepherds, cutaneous and renal glomerular vasculopathy of greyhounds, proliferative arteritis of the nasal philtrum, proliferative thrombovascular necrosis of the pinnae, and solar vasculopathy.

Cutaneous Drug Eruptions

CDE are adverse reactions to any oral, parenteral, or topical drug.^{4,11}

The incidence of CDE in dogs and cats out of all canine and feline dermatology cases examined at one university hospital was 2% and 1.6%, respectively.^{25,26} The pathogenesis may be immunologic or nonimmunologic. Immunologic reactions involve type I (immediate), II (cytotoxic), III (antigen-antibody complex), or IV (delayed) hypersensitivity. Nonimmunologic mechanisms include stimulation of complement pathways, the release of mast cell and basophil mediators, and interference with arachidonic acid metabolism.⁴ The most commonly implicated drugs in dogs and cats can be found in **TABLE 1**, although adverse reactions to NSAIDs, fenbendazole, and carboplatin have also been reported in dogs.^{25–30}

CDE are manifested by variable cutaneous or mucocutaneous lesions that can mimic many other dermatopathies. Lesions may be focal, multifocal, or generalized.¹¹ Typical eruption patterns include urticaria-angioedema, maculopapular eruptions, fixed drug reactions, exfoliative erythroderma, purpuric reactions, vesiculobullous reactions, lichenoid reactions, superficial suppurative necrolytic dermatitis, EM, and TEN. A complete description of these classifications is outside the scope of this article and may be found elsewhere.^{4,31} A pemphigus foliaceus-like drug reaction has also been reported.^{32,33} Diagnostic differentials for CDE vary based on the specific lesions present.

Diagnosis of CDE is often difficult. Clinical signs typically develop within 1 to 3 weeks of starting the offending drug.^{4,31} However, CDE may develop after months or years of therapy with a particular drug or up to 3 weeks after discontinuation of a drug.⁴ Clinicopathologic changes reflect protein and electrolyte loss secondary to cutaneous lesions and the overall health of the patient rather than the cause of the lesions.¹¹ Dermatohistopathology is often nonspecific (except in cases of EM and TEN) and reflects only the lesions present, but it may help rule out some diagnostic differentials. Definitive diagnosis requires rechallenge with the suspect drug to determine if clinical signs recur, but this is considered unethical by most clinicians and may have fatal consequences.^{4,11,31}

Primary treatment consists of discontinuation of the drug in question; cutaneous lesions typically resolve within 2 to 4 weeks after discontinuation. **Lesions should be treated with topical agents (e.g., benzoyl peroxide, chlorhexidine) to help prevent secondary infection and promote healing.** Supportive care (IV fluids, systemic antibiotics, nutritional support) may be warranted in more severe cases (**TABLE 2**). Glucocorticoid therapy is controversial but may be required in some cases to control pruritus and achieve complete resolution of cutaneous signs. The offending drug and any related drugs should be avoided in the future. The overall prognosis is good unless extensive epidermal necrosis or multiple organ involvement is present.^{4,11,31}

Thermal Burns

Thermal burns are relatively uncommon in veterinary patients and occur after accidental or deliberate exposure to a heat source.^{34,35} Reported causes include building fires, electric heating blankets, improperly grounded electrocautery units, hot-air dryers,



Figure 4. Thermal burns in a 4-month-old mixed-breed dog. (A) 10 days after the dog was in a house fire. (B) The same dog 14 days after suffering burns. Penrose drains have been placed in the sterile abscesses seen over the lumbar area in A. An eschar is present along the caudal half of the dorsum. *Courtesy of Veterinary Specialists of South Florida Surgery Department*

scalding water, automobile exhaust pipes, radiators, and malicious torture.³⁴⁻³⁶

Burns are classified according to the depth of the burn and TBSA involved. Superficial (first-degree) burns are restricted to the epidermis and are erythematous, dry, and painful to the touch. Partial-thickness (second-degree) burns involve the epidermis and outer dermis. Depending on the depth of the burn, the skin may be black to yellow-white; hair follicles may or may not be intact; and the area may be painful to the touch or may exhibit decreased pain sensation. In full-thickness (third-degree) burns, the entire epidermis, dermis, and, potentially, underlying connective tissue, muscle, and bone are affected. Prognosis depends on the depth and extent of the burn, which may take 3 to 14 days to fully “declare” itself^{34,35} (FIGURE 4). TBSA affected can be estimated by using the “rule of 9s” established for human burn patients: the head and neck are approximately 9% of TBSA, each forelimb is 9%, each hindlimb is 18%, and the thorax and abdomen are each 18%.³⁷ Severe burns covering $\geq 50\%$ TBSA require intense, long-term nursing care, incur large expenses, and generally have a poor prognosis; euthanasia may be warranted.³⁷

Patients with severe thermal burns may present with hemodynamic compromise from hypovolemic shock. Burn wound edema can result in massive fluid and plasma protein losses during the

first 12 to 24 hours due to increased vascular permeability, thus necessitating aggressive crystalloid fluid resuscitation (TABLE 2). However, patients suffering thermal burns from house fires will likely also have lung damage from smoke inhalation, and fluid therapy should be used judiciously. Specific fluid replacement formulas have been adapted from those used for human burn patients.³⁴ After 12 to 24 hours, vascular permeability stabilizes and use of synthetic colloids may counteract the loss of plasma proteins. Oxygen supplementation should be provided as needed. If $>20\%$ TBSA is involved, significant hematologic, electrolyte, and metabolic abnormalities may develop, including anemia, hyper- or hyponatremia, hyper- or hypokalemia, and metabolic or respiratory acidosis.^{34,35} Clinicians should anticipate these changes and monitor and treat appropriately. Analgesia is of utmost importance, and single-agent (e.g., opioids) or combination therapy (e.g., a morphine, lidocaine, and ketamine constant-rate infusion) may be required. Burn patients are in a hypermetabolic state; early and aggressive nutritional support is required to offset protein catabolism and promote wound healing.^{36,37} If the patient is unable or unwilling to eat on its own, enteral (via nasogastric, esophagostomy, or gastrostomy tube) nutrition should be initiated.

Proper wound management is imperative for preventing infection, protecting the wound, and facilitating healing. If the patient presents within 2 hours of injury, the site should be cooled with cold water or saline (37°F to 63°F) for 30 minutes to reduce heat retention and the depth of the burn. With the patient stable and sedated or anesthetized, the hair over the area should be carefully clipped, a broad-spectrum topical ointment (e.g., silver sulfadiazine, nitrofurazone) applied to the area, and a sterile occlusive dressing placed. Daily wound care consists of conservative debridement, lavage, topical therapy, and bandage replacement, typically under general anesthesia, and continues until wound closure.^{34,37} Healing is rapid for superficial burns and occurs through reepithelialization over approximately 7 days. In superficial partial-thickness burns, the wound may reepithelialize in 10 to 21 days with minimal scar formation. In deep partial-thickness burns, healing may occur through contraction and reepithelialization; however, because severe scarring is typical, surgical closure is recommended. Full-thickness burns often require surgical intervention in the form of skin flaps, advancement techniques, skin grafts, or vacuum-assisted closure.³⁵

Pyotraumatic Dermatitis

Pyotraumatic dermatitis (acute moist dermatitis, “hot spot”) is a rapidly developing superficial to deep skin infection that occurs secondary to self-inflicted trauma from licking, chewing, or scratching.^{38,39} Although the condition is not life-threatening, the sudden onset, pruritic nature, and hemorrhagic appearance often provoke owners to seek veterinary care on an emergency basis. Fleas and flea-bite hypersensitivity are the most common inciting cause when patients present with caudal truncal distribution, but other ectoparasites (e.g., scabies, lice, ticks), hypersensitivity (e.g., adverse food reactions, atopic dermatitis), otitis externa, folliculitis, contact dermatitis, trauma, anal sac disease, swimming (in densely



Figure 5. Acute moist dermatitis in an 8-month-old soft-coated wheaten terrier before (A) and after (B) clipping. Papules along the perimeter of the lesion suggest an expanding superficial pyoderma.

haired dogs), and a dirty, unkempt coat are additional causes.^{38,39} Predisposing factors for development of pyotraumatic dermatitis in dogs are a dense coat, male sex, age <4 years, fleas, and hot, humid weather.³⁸

Clinical signs include erythema, alopecia, and well-circumscribed, moist skin that is typically painful (FIGURE 5). Mild hemorrhage may be present secondary to self-induced trauma. Papules and pustules along the perimeter of the lesion suggest an expanding superficial pyoderma. Lesions most often appear on the trunk, tail base, lateral thigh, neck, or face. Diagnostic differentials include superficial pyoderma, dermatophytosis, and demodicosis (BOX 1), although the latter two typically do not lead to acute onset of severe pruritus at the time of lesion development. Diagnosis is based on the history, clinical signs, and cytology revealing suppurative inflammation with mixed bacteria present.³⁹

Treatment consists of identifying and treating the underlying cause and ensuring aggressive flea control. The lesion should be clipped and cleaned, which may require sedation depending on the location or severity of the lesion. A nonirritating, topical drying

agent or astringent (e.g., 5% aluminum acetate) should be applied every 8 to 12 hours for 2 to 7 days. To control pruritus, a topical or systemic corticosteroid should be used for 5 to 10 days. Antibiotics should be initiated and continued for 1 to 4 weeks. Many studies have shown resolution of clinical signs with topical antibiotic therapy (e.g., fusidic acid, neomycin).^{40,41} However, systemic antibiotics (e.g., cephalosporins, potentiated penicillins, potentiated sulfonamides) should be used for lesions with cytologic evidence of infection or positive culture results that do not show improvement with topical therapy alone.^{38,39} An Elizabethan collar, T-shirt, or socks should be used to protect the affected area and prevent further self-induced injury.³⁹ The prognosis is good if the underlying cause can be corrected or controlled.³⁸

Conclusion

Dermatologic emergencies are uncommon, but they may become life-threatening. In some cases, patients may present with vague systemic signs, with skin lesions developing later. Large cutaneous defects may lead to sepsis or significant plasma protein losses. Certain dermatopathies (e.g., severe EM, TEN, vasculitis, CDE, burn injuries) can cause metabolic, electrolyte, and hematologic abnormalities. Veterinarians should be aware of these conditions so that a proper diagnosis can be made and appropriate therapy instituted.

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1. What is the characteristic histopathology finding used to diagnose EM?

- a. full-thickness coagulation necrosis of the dermis with minimal dermal inflammation
- b. single-cell keratinocyte apoptosis with lymphocyte satellitosis
- c. leukocyte adherence to blood vessel walls
- d. suppurative inflammation with mixed bacteria present

2. Which of the following has/have been used to treat EM?

- a. immunosuppressive agents (e.g., glucocorticoids, cyclosporine)
- b. dietary trial
- c. human immunoglobulin
- d. all of the above

3. Which of the following has/have been reported to cause TEN?

- a. adverse drug reaction
- b. bacterial infections
- c. neoplasia
- d. all of the above

4. Which statement is false with regard to TEN?

- a. Patients often develop systemic signs (e.g., pyrexia, anorexia) before cutaneous lesions.
- b. Massive fluid, electrolyte, and protein losses occur with large dermal involvement.
- c. Mortality rates in humans can be as high as 30% to 50%.
- d. Resolution of signs typically takes 3 to 4 months.

5. The most common proposed pathophysiology of cutaneous vasculitis is a type ____ hypersensitivity reaction.

- a. I
- b. II
- c. III
- d. IV

6. Which statement is false with regard to cutaneous vasculitis?

- a. The extremities, ear tips, tail, footpads, oral mucosa, and nasal planum are the most commonly affected sites.
- b. Rabies vaccine-induced vasculitis has been reported.
- c. Diagnosis is based on skin biopsy and strong clinical suspicion.
- d. Chronic lesions (>24 hours after onset) are the most diagnostic.

7. Which of the following therapies is/are important in managing burn patients?

- a. aggressive fluid resuscitation
- b. nutritional support
- c. wound care
- d. all of the above

8. Which statement is false with regard to burn wound management?

- a. If the patient presents within 12 hours of injury, the site should be cooled with cold water or saline to reduce heat retention.
- b. A topical ointment, such as silver sulfadiazine or nitrofurazone, should be applied to help prevent infection.
- c. Debridement, lavage, and bandage changes are performed on a daily basis initially.
- d. Wound care must continue until wound closure.

9. The most common inciting cause of pyotraumatic dermatitis is

- a. atopy.
- b. fleas and flea-bite hypersensitivity.
- c. otitis externa.
- d. contact dermatitis.

10. Which of the following may be involved in the pathogenesis of CDE?

- a. release of mast cell and basophil mediators
- b. type III hypersensitivity reaction
- c. interference with arachidonic acid metabolism
- d. all of the above