

Dermatological emergencies

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FILIPPO DE BELLIS DVM, CertVD, DipECVD, MRCVS describes the pathogenesis, diagnosis and suggested treatment for a variety of skin diseases in dogs and cats that can be presented by worried owners

DERMATOLOGICAL emergencies are not common in general practice as, in most cases, dermatological conditions are chronic in nature. However, when presented as acute problems, although non-life threatening, skin issues may alarm the owners, who will subsequently seek veterinary attention.

On the other side, there are some conditions, often rare, that may appear initially mild, but may prove serious or fatal at a later stage. Serious conditions can be difficult to differentiate from each other, so the process of history taking is very important, including specific attention to previous treatment, because adverse drug reactions (ADRs) can be some of the most serious dermatological presentations. It is also very important to take skin biopsies of early lesions and submit the specimens to a pathologist with a special interest and expertise in dermatohistopathology.

Urticaria and angioedema

Urticaria and angioedema are uncommon in small animals. They can be triggered by both immunological and non-immunological processes and the pathological changes are restricted to the dermis in urticaria and can involve the connective tissue in angioedema.

Pathogenesis

The causative agents may be non-identified in up to 80 per cent of human cases, and similar numbers are anecdotally reported in veterinary patients. The pathomechanism can be of an immunological or non-immunological nature and clinical signs are classically due to the result of local mast cells and/or basophils degranulation leading to vascular dilation and recruitment of inflammatory cells. Immunological reactions are classic type one hypersensitivity disorder triggered by vaccinations, allergens in drugs, topical agents or arthropod bites. Non-immunological reactions can be triggered by arthropod venom, food items, drugs, dermatographism or by environmental factors (cold-induced urticaria, actinic urticaria).

Clinical signs are acute in onset and classically consist of pruritic wheals or large areas of oedematous swellings ([Figure 1](#)). They can be localised or generalised – with angioedema tending to be localised to the head ([Figure 2](#)). Respiratory distress can occur if the angioedema involves the nares, larynx, or pharynx.

Diagnosis is based on clinical appearance, disappearance of the signs on diascopy and, when necessary, supportive histopathology; however, in most cases, history and clinical signs play a major role. Differential diagnoses for urticaria include bacterial folliculitis (especially for short-coated breeds), early cases of erythema multiforme, mast cell tumours, cutaneous mastocytosis and focal mucinosis. Differential diagnoses of angioedema include early stages of juvenile cellulitis, early stages of bacterial cellulitis and neutrophilic immunologic vasculitis.

Treatment

Prompt identification and correction of the offending factors is crucial for successful management; however, most often it is important to proceed with symptomatic treatment, including systemic glucocorticoids (dose and route administration depend on the severity of the clinical signs), antihistamines (described as ineffective in treating acute cases, but may be helpful in chronic cases or to prevent future reactions), in the case of vasculitis, pentoxifylline (15mg/kg three times daily) and, in the case of angioedema, given the risk of laryngeal oedema, intravenous glucocorticoids (methylprednisolone succinate 1mg/kg or dexamethasone sodium phosphate 0.25mg/kg).

Canine juvenile cellulitis

Canine juvenile cellulitis (CJC) is an uncommon pustular and granulomatous disease affecting mainly the face, pinnae and lymph nodes and is seen usually in puppies. However, there are rare reports of dermatoses resembling CJC in adult dogs. This condition has been described with increased occurrence in some breeds (golden retriever, dachshund, Gordon setter).

The aetiology and pathogenesis of CJC are unknown; an infectious aetiology has been considered, but not demonstrated, and the prompt response to high doses of oral prednisolone supports a non-

infectious aetiology. A causative link with a vaccine against distemper virus, adenovirus and parainfluenza virus has also been suggested. The fulminant onset is a typical characteristic of CJC.

Commonly, the initial feature is acute swelling of the face, especially on the eyelids, lips and muzzle ([Figure 3](#)), with submandibular lymphadenopathy. Within 24 to 48 hours, papules, pustules and crusting develop rapidly. At presentation typical cutaneous lesions usually appear on the head (muzzle, pinnae, and periocular areas). The inner aspect of the pinnae is commonly oedematous and shows the presence of crusting and purulent exudate. Additionally, involvement of the feet, abdomen and thorax, preputial and perianal areas have been reported. Secondary bacterial infections are possible. Pyrexia and depression, along with joint pain, are inconsistent clinical signs.

The clinical differential diagnoses of CJC include mainly deep pyoderma, demodicosis and ADR. In early stages, when papules and pustules are absent, it has also to be differentiated from angioedema. For adult dogs, sterile granuloma pyogranuloma syndrome or eosinophilic furunculosis of the face should also be considered. Diagnosis is based on history, clinical signs and is confirmed by histopathology.

Therapy of CJC requires immune-suppressive doses of glucocorticoids (prednisone or prednisolone at 2mg/kg), to be administered until remission of the clinical signs is achieved. Combination of prednisolone and ciclosporin has also been effective. If there is cytological evidence of secondary bacterial infection, systemic antibiotics should be given.

The prognosis is generally good, although relapses after discontinuation of treatment have been reported.

Pyotraumatic dermatitis

As previously mentioned, sometimes common and non-life-threatening diseases, due to their acute onset, can alarm the owners and, therefore, be presented as emergencies. Pyotraumatic dermatitis “hot spots” is an example. This condition is well known in general practice, affecting mainly young dogs with a higher prevalence in breeds such as St Bernard, golden retriever and Rottweiler. A dense coat seems to be a predisposing factor. Pyotraumatic dermatitis is considered a complication of underlying problems – most commonly allergies. Clinically it is characterised by acute onset with well-circumscribed areas of alopecia with moist, matted hair on the cheek, neck, dorsolumbar region and flanks.

Diagnosis is based on history and clinical signs, but histopathology may be needed to differentiate cases with surface pyoderma from cases with folliculitis and furunculosis consistent with deep pyoderma.

Clinical management consists of clipping and the use of topical antiseptic agents. However, lesions

can be often painful and, therefore, caution should be used when handling, and sedation should be considered at least initially. The use of systemic antibiotics is needed when deep pyoderma is present or suspected and, markedly, pruritic cases, in the absence of deep pyoderma, should be treated with a short course of prednisolone at anti-inflammatory doses.

Primary irritant contact dermatitis

Another condition that can be alarming for owners is primary irritant contact dermatitis. This is a heterogeneous disease with various clinical manifestations and history is usually important to allow identification of the causative factors, which often include soaps, insecticides, topical antimicrobials and caustic substances.

Typical clinical manifestations are erythema and papules seen as primary lesions, with chronic cases showing lichenification, excoriations, crusts and ulcerations. Typically, lesions occur on glabrous areas where the skin is easily exposed to the offending substances.

Differential diagnoses include other hypersensitivity disorders, ectoparasitic diseases and infections, such as *Malassezia* dermatitis.

The treatment of choice includes removing the offending substance and washing the affected area and, with pruritus, anti-inflammatory doses of systemic prednisolone may be indicated.

Eosinophilic furunculosis of the face

Eosinophilic furunculosis of the face (EFF) is an uncommon skin disease in dogs. It is characterised by peracute onset and predominantly nasal and muzzle distribution of lesions. EFF is believed to represent some type of hypersensitivity reaction due to possible contact with arthropods (including bees, hornets, wasps, ants and spiders), but the cause-effect relationship has not been proved.

Affected dogs present with papules, nodules and plaques with various degrees of erosions, ulcerations, crusting, serum leakage and haemorrhage present on the muzzle ([Figure 4](#)) – often periocularly; however, similar lesions may be seen also on the pinnae, ventral abdomen, lips and interdigital areas. Pruritus can be variable. Severely affected dogs may present with pyrexia and depression.

The differential diagnoses of EFF include mainly bacterial nasal folliculitis and furunculosis, dermatophytosis and demodicosis when the lesions persist, and autoimmune diseases with predominant facial lesions; however, EFF presents a fulminant onset, whereas autoimmune diseases commonly develop gradually. Although histopathology is required for definitive diagnosis, cytology, with large numbers of eosinophils, is of value in supporting a clinical diagnosis of EFF and, histopathology results pending, can provide justification for the use of glucocorticoids when

other symptomatic treatments have failed to prevent progression of the disease.

Rapid response within 10 to 14 days to moderately high doses of prednisolone is typical of this disease. In some cases scarring alopecia may be seen. Prognosis is excellent because of the prompt response to the therapeutic agents.

Erythema multiforme

Erythema multiforme (EM) is an uncommon disease characterised by eruption of the skin and/or mucous membranes and has been reported in humans, dogs, cats and horses. EM is considered to be a cutaneous reaction pattern associated with a T cell-mediated hypersensitivity reaction directed against altered keratinocyte antigens. It has been recognised in associations with infections, drug therapy (especially trimethoprim-potentiated sulphonamides, penicillins and cephalosporins), neoplasia, connective tissue diseases and adverse food reactions.

EM is frequently characterised by pleomorphic eruptions. Typically, there is acute onset of maculopapular lesions that spread peripherally and clear centrally, producing annular or arciform patterns ([Figure 5](#)). Other clinical presentations include urticarial plaques, vesicles, bullae and ulcers. Feline EM is reported to exhibit predominantly vesiculobullous and ulcerative lesions, but generalised exfoliative dermatitis with alopecia has also been seen. Severely affected animals may be depressed and anorectic.

The clinical differential diagnoses of EM in this case include mainly paraneoplastic diseases, early lesions of bullous autoimmune diseases and epitheliotropic lymphoma. Due to the pleomorphic nature of the clinical signs, histopathology is essential for making a definitive diagnosis. Interface dermatitis and apoptosis are typical features of EM where apoptosis is prominent at all levels of the epidermis, contrary to thymoma-associated exfoliative dermatitis, which shows milder transepidermal apoptosis. There are cases of EM featuring large histiocytic round cells in clusters in the epidermis mimicking lesions of epitheliotropic lymphoma with apoptosis.

Treatment of EM is based on identifying and removing the offending factor and on use of supportive care measures. In idiopathic cases, glucocorticoids used with or without azathioprine have been successfully used; anecdotal reports suggest the use of ciclosporin. Intravenous human immunoglobulin administration (1g/kg during a four-hour period for two consecutive days) has been reported successful in a feline case. The prognosis is variable, depending on the nature of the lesions and on the identification and correction of the triggering factor.

Toxic epidermal necrolysis

Toxic epidermal necrolysis (TEN) is a rare immune-mediated disease characterised by extensive, painful vesiculobullous and ulcerative lesions affecting both the skin and the mucosae. It has been reported in humans, dogs, cats and cattle and is most commonly associated with drug eruptions

although, in humans, cases are associated with vaccine reactions, neoplasia, infections and pregnancy. These appear to also be possible in dogs and cats.

The pathomechanism of TEN is not exactly known and there are cellular immunologic factors involved, primarily in the death of keratinocytes. The pathways leading to the pathological changes are different and have been extensively studied in humans – the pathogenesis is still unknown in dogs. Stephen Johnson syndrome (SJS), and SJS-TEN overlap, are considered the same entity, albeit characterised by a less severe clinical manifestation.

There is no apparent age, breed or sex predilection, and typical presentations include acute onset, pyrexia, anorexia, lethargy and depression. Dermatological clinical signs consist of erythematous macular dermatitis, focal to widespread mucocutaneous vesicles with epithelial detachment and consequent ulcerations ([Figure 6](#) and [Figure 7](#)). A positive Nikolsky sign is often present. Other clinical signs may include loss of claws, corneal ulcerations and ulcerative otitis. Internal involvement may manifest as renal failure, hepatopathy and blood dyscrasias. Fluid and electrolyte losses and secondary infections can be often seen.

Differential diagnoses include urticaria, thermal burns, deep infections, other immune-mediated blistering skin diseases, vasculitis, epitheliotropic lymphoma and cutaneous drug reactions. Histopathological lesions typically consist of hydropic basal cell degeneration, full thickness necrosis of the epidermis with dermoepidermal separation, vesicles and ulcerations. Cell-poor interface dermatitis may occasionally be present.

Treatment is similar to that for second degree burns and symptomatic and supportive measures to counteract the fluids, colloids and electrolytes losses are vital. Antibiotic cover is important to prevent secondary infections. In humans, the use of systemic glucocorticoids has been associated with increased morbidity and mortality. In veterinary cases intravenous immunoglobulins have been reported to be effective in dogs and cats.

The prognosis of TEN is guarded to poor, depending on the underlying cause.

Cutaneous drug eruptions

An ADR is any undesired consequence derived from the administration of a drug. It can be divided into two categories:

- undesired pharmacological effects, dose dependant, foreseeable and relatively frequent; and
- toxic effects, unforeseeable and of idiosyncratic nature.

A drug eruption is a drug reaction that involves the skin.

Pathogenesis

Generally, any drug can cause undesired effects, although certain drugs are more frequently associated with dermatological manifestations. The pathomechanism can be immunological or non-immunological. Immunological drug reactions include hypersensitivity mechanisms of type one to type four. Non-immunological drug reactions may clinically mimic those of their immunological counterpart, but are non-elicited by their interaction between the drug and the immune system. Some examples include drugs that can elicit the release of mast cell mediators, drugs that can activate an alternate pathway to complement activation, drugs able to alter the metabolism of arachidonic acid, and drugs able to suppress regulatory lymphocytes.

Diagnosis is based on the history of drug administration, clinical signs supported when needed by histopathology, and resolution after drug withdrawal. Reproducibility of the clinical signs after drug re-challenge is important, but is usually not advised for ethical reasons.

The list of differential diagnoses can be extensive as the clinical signs are various, including urticaria and angioedema, erythroderma and exfoliative reactions, maculo-papular eruptions, vasculitis, auto-immune diseases (pemphigus foliaceus, systemic lupus, diseases of the dermo-epidermal junction), Wells'-like syndrome (canine eosinophilic dermatitis with oedema), EM-SJS-toxic epidermal necrolysis, pruritus, injection site reactions, fixed reactions and contact reactions ([Figure 8](#)).

Given the extreme variability of clinical signs and the various criteria needed to address a drug reaction, an ADR probability scale was developed by Naranjo et al in human medicine ([Table 1](#)). This is a method for estimating the probability of an adverse reaction and, to do so, a series of questions should be addressed, giving a pertinent score to the answers.

Treatment is based on withdrawal of the suspected drug. Fluid therapy is needed until an improvement of the general condition is obtained and antibiotic cover (not related to the possible responsible drug) may be needed in cases with ulcerations and pustular lesions. The use of systemic glucocorticoids, apart from cases of urticaria and autoimmune disease, is controversial.

Prognosis is usually good, but can be guarded in cases of anaphylactic shock, EM or toxic epidermal necrolysis.

- Some drugs mentioned are not licensed for veterinary use.

Further reading

- Birne K P and Giger U (2002). Use of human immunoglobulin for treatment of severe erythema multiforme in a cat, *Journal of the American Veterinary Medical Association* **220**(2): 197-201.

- Curtis C F, Bond R, Blunden A S, Thompson D G, Mcneil P E and Whitbread T W (1995). Canine eosinophilic folliculitis and furunculosis in three cases, *Journal of Small Animal Practice* **36**(3): 119-123.
- Holm B R, Rest J R and Seewald W (2004). A prospective study of the clinical findings, treatment and histopathology of 44 cases of pyotraumatic dermatitis, *Veterinary Dermatology* **15**(6): 369-376.
- Miller W H, Griffin C E and Campbell K L (2013). Hypersensitivity disorders. In *Muller and Kirk's Small Animal Dermatology (7th edn)*, Elsevier, St Louis: 363-431.
- Miller W H, Griffin C E and Campbell K L (2013). Autoimmune and immune-mediated dermatoses. In *Muller and Kirk's Small Animal Dermatology (7th edn)*, Elsevier, St Louis: 432-500.
- Naranjo C A, Busto U, Sellers E M, Sandor P, Ruiz I, Roberts E A, Janecek E, Domecq C and Greenblatt D J (1981). A method for estimating the probability of adverse drug reactions, *Clinical Pharmacology and Therapeutics* **30**(2): 239-244.
- Neuber A E, Van den Broek A H, Brownstein D, Thoday K L and Hill P B (2004). Dermatitis and lymphadenitis resembling juvenile cellulitis in a four-year-old dog, *Journal of Small Animal Practice* **45**(5): 254-258.
- Reimann K A, Evans M G, Chalifoux I V, Turner S, Deboer D J, King N W and Letvin N L (1989). Clinicopathologic characterization of canine juvenile cellulitis, *Veterinary Pathology* **26**(6): 499-504.
- Scott D W and Miller W H (1999). Erythema multiforme in dogs and cats: literature review and case material from the Cornell University College of Veterinary Medicine (1988-96), *Veterinary Dermatology* **10**(4): 297-309.
- White S D, Rodney A W, Stewart L J, Cape L and Hughes B J (1989). Juvenile cellulitis in dogs: 15 cases (1979-1988), *Journal of the American Veterinary Medical Association* **195**(11): 1,609-1,611.



Figure 1. Multiple circular areas of tufted hair in a boxer dog with urticaria and angioedema. Note oedematous limbs and erythema on the abdomen and ventral neck.



Figure 2. Oedematous swelling of the face in a Shar Pei puppy. Angioedema secondary to a vaccination reaction.



Figure 3. A Staffordshire bull terrier puppy showing alopecia, papular dermatitis and crusting involving the periocular areas and the muzzle. Oedema of both pinnae.



Figure 4. Alopecia, coalesced papules, oedematous nodules, plaques and mild erosions involving the dorsum of the muzzle. The planum nasale is not affected.



Figure 5. Multiple coalescing erythematous macules with annular to serpiginous pattern on the ventral abdomen.

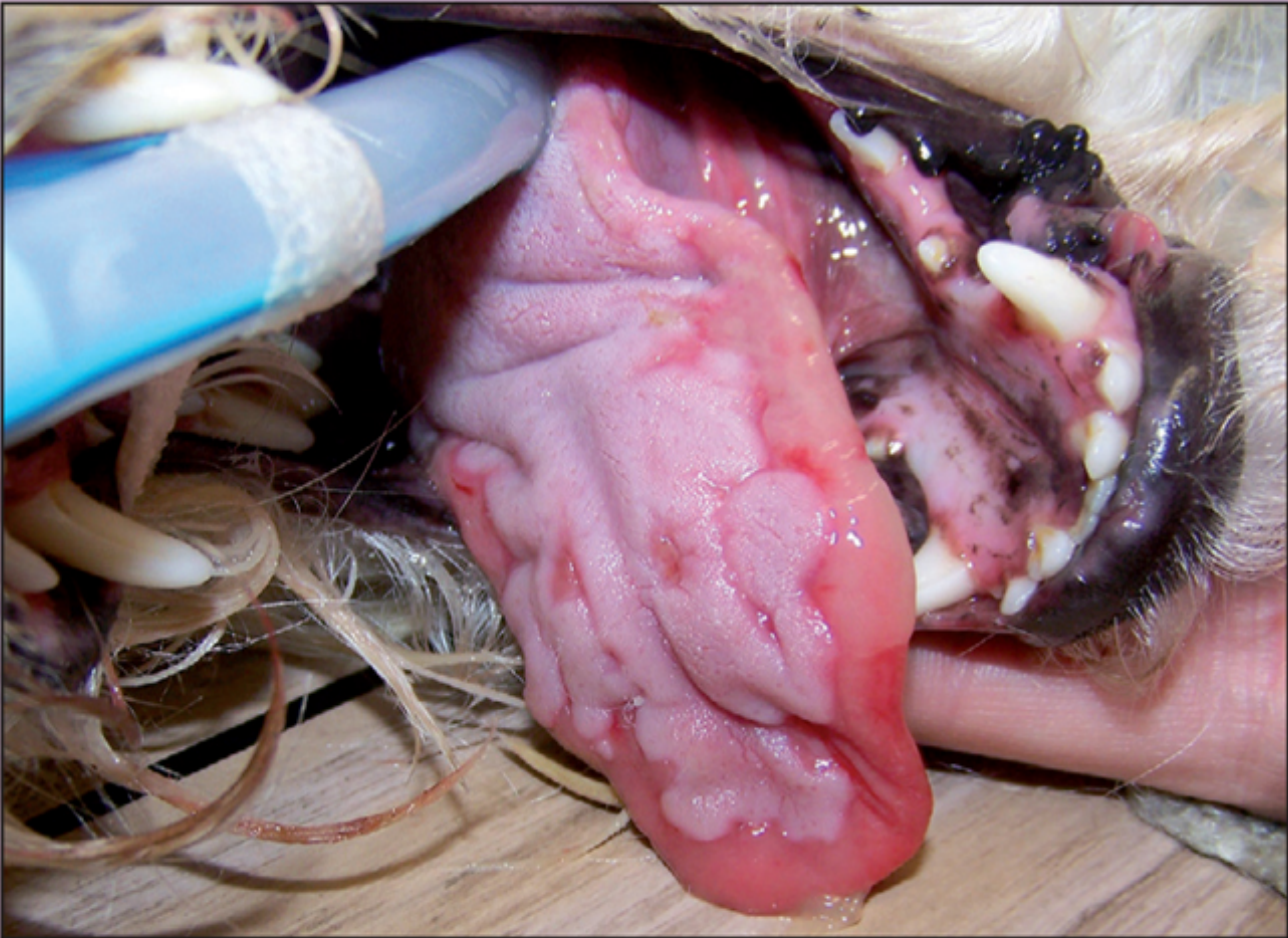


Figure 6. Punctate to widespread ulcerations affecting the tongue mucosa.



Figure 7. Same dog as in [Figure 6](#). Lesions extended to the chest, abdomen and inner aspects of the hindlimbs with widespread epithelial detachment.



Figure 8. Alopecia, erythema, exudation and mild crusting in a domestic shorthair cat on application site of a spot-on product.

	Yes	No	Don't know	Score
1. Are there previous conclusive reports on this reaction?	+1	0	0	
2. Did the adverse event appear after the suspected drug was given?	+2	-1	0	
3. Did the adverse reaction improve when the drug was discontinued or a specific antagonist was given?	+1	0	0	
4. Did the adverse reaction appear when the drug was re-administered?	+2	-2	0	
5. Are there alternative causes that could have caused the reaction?	-1	+2	0	
6. Did the reaction reappear when a placebo was given?	-1	+1	0	
7. Was the drug detected in any body fluid in toxic concentrations?	+1	0	0	
8. Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	+1	0	0	
9. Did the patient have a similar reaction to the same or similar drugs in any previous exposure?	+1	0	0	
10. Was the adverse event confirmed by any objective evidence?	+1	0	0	
Adverse drug reaction (ADR) score: >9 = definite ADR; 5-8 = probable ADR; 1-4 = possible ADR; 0 = doubtful ADR.				

Table 1. Adverse drug reaction probability scale (from Naranjo et al, 1981)