Ischemic dermatoses fall into three categories: dermatomyositis, vaccine- and drug- caused cases, and idiopathic. Dermatomyositis has been reported primarily in collies and Shetland sheepdogs and their crosses, although other breeds have a sporadic incidence of this disease. As its name implies, the disease affects both the muscles and the skin. Cutaneous changes include crusts, ulcerations, vesicles, and/or alopecia around the mucocutaneous junctions, front legs, ear tips, and tail, though other body areas may be affected. Claws may be misshapen. Muscular atrophy may be generalized or may be selective, often affecting the temporal and masseter muscles. Clinical manifestations vary, with some dogs showing only skin or muscular signs, while in others both systems are affected.

Serum enzymes such as creatinine phosphokinase (CPK) are usually normal, and muscle involvement often may be proved only by biopsy or electromyography. Skin biopsies generally reveal perifollicular mononuclear inflammation, and occasionally show intracellular edema of the basal cell layer of the epidermis, with subepidermal clefts; advanced cases show a loss of the normal follicular structures. Dermal blood vessels may be decreased in number, be over-distended, smudged, hyalinized and/or sclerotic in appearance. The onset of clinical signs usually occurs before the age of 6 months. The severity of the disease varies greatly, with some dogs improving with age. Females should be spayed as estrus may exacerbate clinical signs. Diagnosis is based on clinical signs and skin biopsy. Recently, the disease in Shetland Sheepdogs has been linked to a change in chromosome 35.

Drug- or vaccine induced ischemic dermatosis is most commonly associated with rabies vaccination, manifesting itself as alopecia, scale and crusts, and occasionally ulceration, in the area of the vaccine administration, and rarely, in other places as well (especially the pinnal margins and tail tip. Diagnosis is by clinical signs and biopsy. Histology shows involuting, small hair follicles with prominent connective tissue associated with the external root sheaths – the follicles will sometimes disappear permanently. Deep dermal vessels will show a plasmacytic/lymphocytic vasculitis; more superficial vessels will appear as noted above for dermatomyositis.

Idiopathic ischemic dermatosis can occur at any age and any canine breed. Typically, the tail tip, ear margin, face (nasal planum) and sometimes trunk are affected. Claws may be misshapen. Histopathology is as for the vaccine caused type. Recently, there have been anecdotal reports of dogs with this disease having positive Ehrlicia titers and responding completely to doxycyclines – this is probably worth pursuing.

Treatment of ischemic dermatoses
Pentoxifylline (PTX) 10-30 mg/kg q8h, is often effective in controlling ischemic dermatoses. PTX is derived from theobromine. Pentoxifylline, and other methylxanthines produce anti-inflammatory effects. PTX also improves blood flow through narrowed arteries because of the rheological property which allows red blood cells to change shape. It is not known if the improvement in patients with ischemic dermatoses are caused by improved blood flow or via the anti-inflammatory mechanisms. Vomiting is occasionally seen as a side-effect. Eventually some dogs will be able to be tapered down to a q48 hour regimen, or occasionally weaned off the drug.

When PTX is not effective, Atopica® may be used. For small lesions, topical tacrolimus (Protopic®) may be effective. Corticosteroids have had variable efficacy in the author’s experience.

Erythema multiforme (EM) is an acute eruption of the skin and mucous membranes. It is characterized in human beings clinically by annular ("target") lesions. While these have been observed in animals, more common signs are mucocutaneous vesicles, ulcers, maculae, and/or urticarial plaques may also be seen. In widespread lesions, the ventrum and peri-ocular areas are often involved. EM may be self-limiting, although by the time the animals arrive at the specialist’s office, this is not often the case. EM histologically shows apoptotic (programmed-cell death mechanism-activated) keratinocytes, with satellitosis (lymphocytes surrounding the keratinocytes, presumably triggering the programmed cell death mechanism). The incidence/recognition of this disease seems to be increasing. While erythema multiforme has been reported to have an association with drug eruptions, recent work points to TEN (see below) and ‘cross-over syndromes’ between the two diseases as more likely to be due to drug involvement. Viruses have also been hypothesized to cause EM in small animals, and there is one report of EM caused by parvo virus in a puppy. The author has seen a few cases that seemed to have an ischemic component, or occurred concurrently with an ischemic dermatitis. While theoretically pentoxifylline should be helpful, the author has seen 2 dogs which had EM induced by pentoxifylline! Intravenous human immunoglobulin has been reported as successful in two dogs when infused on 2 consecutive days (1 g/kg per day). This is a relatively expensive treatment.

Rare disaster syndromes
These include 4 rare conditions which are often typified by neutrophils in the skin, and 1 by eosinophils in the skin, but with different underlying causes, and therefore treatments.
Necrotizing fasciitis (‘flesh-eating’ bacteria) caused by Streptococcus canis (biotype 3). Dogs present with fever, swelling, erythema, disproportionate pain on palpation, draining tracts and ulcers. There may be pockets of fluid, usually malodorous. It has been hypothesized that Great Danes and Sharpeis may be predisposed, but too few cases have been reported in the literature to confirm this. There is rapid progression of this disease, as there is of all three diseases in this group. Diagnosis is by clinical signs, skin biopsy and bacterial culture and susceptibility; imaging such as ultrasound, CT scan and/or MRI may be helpful (if available) to find exudates advancing along fascial planes that may not be evident on physical examination. Treatment is widespread surgical debridement and antibiotics – eventually based on susceptibility results, but initially with clindamycin and/or amoxicillin-clavulanate, plus an aminoglycoside. Do not use fluoroquinolones, as these have been associated with possibly engendering or enhancing the extreme toxicity of these Streptococcus strains. NSAIDS should also be avoided, as there is some evidence they may suppress neutrophil activity and mask clinical signs.

Sweet’s (sterile neutrophilic dermatosis) syndrome is a non-infectious, presumed immune mediated condition typified by erythema, fever, malaise, neutrophilia, lameness and neutrophilic effusions into the joints. This is also a rapidly progressive disease, and may be caused by certain medications, as well as arising spontaneously. There is one report of a dog with internal organ involvement. Diagnosis is based on skin biopsy, the lack of bacteria on culture of intact skin lesions or joint aspirates, and response to corticosteroids (prednisolone 1 mg/kg bid initially).

Sterile putular erythroderma of miniature schnauzers

A rare, severe, often fatal disease, seemingly limited to miniature Schnauzers, often preceded by bathing. This condition presents with severe depression and malaise, often with fever. Skin lesions are (often dramatic) erythema, pustules or epidermal collarettes, and/or wheals. These dogs are very sick – treatment, when successful, consists of high dose corticosteroids. In a recent abstract, a contact reaction to one of the components in an aloe-based shampoo was implicated.

Staphylococcal Toxic Shock is caused by Staphylococcus sp, presumably Spseud-intermedius. Erythema, fever, malaise, and neutrophilia are seen initially; the malaise may be severe and edema of the legs may develop as the disease progresses. This may be the most rapidly progressing of these three diseases. Diagnosis is based on clinical signs – a skin biopsy and bacterial susceptibility should be performed, but any suspicion on the veterinarian’s part for this disease should initiate the immediate use of staphylocidal antibiotics – cephalosporins have been recommended. Pugs may be over-represented.

Well’s like syndrome is a eosinophilic, generalized dermatitis to cellulitis. It has been associated with either GI signs and/or drugs in some, but not all cases. Peripheral eosinophilia is rare. Skin lesions are papules, macules, and erythema. Pruritus is variable. Histopathology shows an eosinophilic dermatitis, often with eosinophilic ‘flame figures’ (collagen surrounded by eosinophils or their granules). Treatment consists of prednisolone/prednisone at 1-2 mg/kg, then tapered. Treatment duration is variable, but should be continued for at least one month.

References


