Cutaneous Vasculitis in Small Animals

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KEYWORDS

• Cutaneous vasculitis • Necrotizing vasculitis • Neutrophilic vasculitis
• Immune mediated • Cutaneous adverse drug reactions • Canine • Dog • Cat

KEY POINTS

- Cutaneous vasculitis is a reaction pattern and not a disease in itself.
- A thorough workup to identify underlying triggers should always be made.
- The diagnosis must be confirmed histologically, and one should obtain deep tissue samples to make sure vascular damage is not missed.
- If a drug is suspected or confirmed as the cause of vasculitis, withdrawal and future avoidance is the most important treatment.
- Very high doses of steroids may not be the best treatment for patients presenting with large areas of ulcerated skin, because this increases the risk of secondary wound infections and delays wound healing.

Vasculitis is a reaction pattern, characterized by an aberrant immune response directed toward blood vessels.[1] The pathophysiology is not fully understood, and is most likely complex, involving a variety of mechanisms acting in concert to induce necrotizing inflammatory changes in the blood vessel wall.[2]

Clinical presentation of patients affected by this multifactorial reaction pattern depends mainly on the extent of vascular destruction, and both cutaneous and systemic forms have been reported.[1] Impaired vascular function may lead to edema formation, hemorrhage, and purpura. Full-thickness skin necrosis and crateriform ulcers may follow. Patients affected with systemic or cutaneous vasculitis are most often sick, presenting with constitutional signs. A varying degree of pain is common and may range from mild to severe. Extensive ulcerations of large areas of skin predispose these patients to secondary bacterial wound infections and sepsis, much like a patient presenting with extensive burns, so proper wound management is essential.

Numerous triggering factors have been identified as a cause of vasculitides in dogs and cats; therefore, a thorough drug history and early workup for underlying ongoing disease processes are important steps when presented with these patients.[1,3] Diagnosis should be based on history and clinical findings along with compatible histopathology reports.

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Treatment must be tailored to the individual patient, and should be based on underlying triggering factors as well as the extent and severity of cutaneous lesions.

Vasculitic diseases currently recognized in dogs and cats, including cutaneous necrotizing vasculitis or neutrophilic immunologic vasculitis, are covered herein. Specific familial vasculitides will also be reviewed briefly. For information about ischemic dermatopathies please refer to the article by Morris elsewhere in this issue.

IMMUNOPATHOGENESIS

The primary immunopathogenic events that initiate the process of vascular inflammation and blood vessel damage are poorly understood in both people and animals; however, immunologic mechanisms appear to play an active role.[2]

Applying the traditional Gell-Coombs classification of hypersensitivity reactions, it is thought that immediate hypersensitivity reactions as well as type II and type III reactions may all be involved in the immunopathogenesis of vasculitic diseases.[4] This immunologic heterogeneity also illustrates the importance of not viewing vasculitic diseases as a diagnosis in itself, but a reaction pattern that warrants further investigations to achieve a proper diagnosis and prognosis for the individual patient.

Type I Hypersensitivity Reactions

Immediate hypersensitivity reactions, characterized by the formation of immunoglobulin E antibodies have been stated to be involved especially in the early stages of cutaneous vasculitides in animals[5]; however, type I reactions are unlikely to be the major player in most cases of canine and feline vasculitides.[1]

Type II Hypersensitivity Reactions

In a classical type II hypersensitivity reactions, antigen–antibody interactions result in the local production of anaphylotoxin (C5a), the recruitment of polymorphonuclear leukocytes, and subsequent tissue injury owing to the release of hydrolytic neutrophil enzymes after their autolysis. In human medicine, a subset of vasculitides are characterized by the formation of autoantibodies; so called antineutrophil cytoplasmic antibodies (ANCA) and the diseases associated with the production of these antibodies are referred to as ANCA vasculitides. In ANCA vasculitides (Wegener's granulomatosis [WG], Churg–Strauss syndrome, and polyarteritis nodosa), ANCA bind directly to neutrophil granules and the release of toxic mediators leads to a direct damage of vessel walls.[1,2]

Human antibody-associated vasculitides, where antibodies bind directly to the vessel walls, are thus classical examples of type II reactions.[2]

Type III Hypersensitivity

Immune complex reactions occur when antibodies present in the blood result in the formation and deposition of antigen–antibody complexes. The very presence of these complexes lodged in blood vessels, in addition to the polymorphonuclear leukocytes attracted by complement activation, results in tissue injury and compromised function. Vasculitis associated with connective tissue diseases such as systemic lupus erythematosus are examples of a type III hypersensitivity reaction.[1,6] This is currently the most widely accepted pathomechanism of cutaneous vasculitis in animals.

HISTOLOGIC FEATURES

Histologic examination is needed to confirm a diagnosis of cutaneous vasculitis, and samples from affected patients show pathologic changes in the vascular structures of