Treatment of canine adult-onset demodicosis

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Demodicosis can present in a variety of clinical patterns including adult-onset generalised demodicosis with bacterial pyoderma. The latter is the most common pattern seen in dogs presented to our dermatology service.

Assessing the case for therapy

The onset of demodicosis in dogs over three years of age has been associated with a number of underlying diseases that apparently deregulate the commensal relationship between parasite and host. The treatment protocol and overall prognosis for demodicosis in these cases is dependent to a large extent on the prognosis for the underlying disease. Concurrent underlying trigger factors reported for adult-onset demodicosis include neoplasia, spontaneous or iatrogenic hyperadrenocorticism, hypothyroidism and immunosuppressive therapy.[1,2] Unlike juvenile-onset demodicosis, a familial or breed disposition is not reported.

In a study of 25 cases of adult-onset demodicosis (unpublished) all were pure-bred dogs and 15 were small terriers, predominantly West Highland White Terriers (six) and Shih Tzus (four). The terrier breeds were overrepresented when compared to the British pure-bred dog population and the hospital dog population. Most (84%) of the affected dogs had more than one risk factor for generalised demodicosis: 21 cases had received a systemic glucocorticoid for four weeks or longer for existing skin disease, including allergy and pemphigus foliaceus, and of these, six were classified as having iatrogenic hyperadrenocorticism. Five dogs had neoplasia, four dogs had hypothyroidism and one had protein losing glomerulopathy. In only one case was no risk factor identified. Follow-up periods from the time of diagnosis of demodicosis were two to seven years during which time only three dogs died or were euthanased. Clinical, but not necessarily parasitological, remission was achieved in the remaining dogs with appropriate miticidal therapy and management of the underlying cause. However, all cases experienced intermittent periods of clinical relapse and some dogs required continuous long-term miticidal therapy.

Any therapeutic protocol in adult-onset demodicosis must include management of the underlying disease. However, although long-term clinical response compatible with good quality of life is achieved in more than 80% of our cases, repeated or continual therapy is required, as is the practical and financial commitment from the owners.

Therapeutic options

The majority of dogs with adult-onset demodicosis treated at our institution receive a combination of miticidal and antimicrobial therapy. Our most frequently used systemic antimicrobial agent is cephalexin at a dose rate of 20 to 30 mg/kg twice daily for a minimum of three to four weeks and for six to eight weeks in cases of deep pyoderma and pododemodicosis. This is used in combination with topical 2% chlorhexidine or 10% ethyl lactate-based shampoos. Benzoyl peroxide shampoos are not used in our cases as they have been associated with potentiation of pruritus.

Amitraz

In the UK, the only licensed product for the treatment of canine demodicosis is amitraz applied topically at 0.05% solution once every five to seven days. We recommend total-body clipping or, at least, clipping of the affected areas. The antimicrobial shampoo of choice is applied first and the dog towel-dried prior to amitraz application.

Side effects associated with amitraz acting as an α2-adrenergic agonist include hypotension, bradycardia, hypothermia and mydriasis. Its use may also be associated with hyperglycaemia, sedation for 24 to 72 hours, convulsions, ataxia, personality change, diarrhoea, anorexia, vomiting and transient pruritus. Care should be taken to ensure that amitraz therapy does not exacerbate any underlying disease. Small dogs with generalised skin disease are overrepresented in our cases and increased systemic absorption of amitraz with the risk of side-effects is an issue in treatment choice. In small dogs, we may use 0.025% solutions or occasionally only 50% of the body surface is treated at any one time. The use of a residual dip such as amitraz does limit the use of topical antibacterial shampoos that may be useful in controlling pyoderma.

Orally-administered miticides

For many of the above reasons amitraz may not be the treatment of choice in an individual dog. If the risk of side-effects or exacerbation of underlying disease in unacceptable or if the response to amitraz is poor or already causing adverse effects, owners are commonly offered milbemycin or ivermectin alternatives.

Ivermectin – In the UK, this product is not licensed for use in the dog. The injectable ivermectin formulation (10mg/mL) used in cattle, sheep and pigs is the most commonly used product. We use it in non-herding dog breeds orally at a dose rate of 600 μg/kg daily. Prolonged treatment may be required in adult-onset cases: 10 to 18 weeks being the average for parasiticidal remission. Ivermectin is never given to Rough-Coated Collies, Shelties, Old English Sheepdogs or any individual with a merle- coloured coat or blue iris. If in doubt about the toxicity in an individual dog, a test dose of 100 μg/kg subcutaneously is given and the dog monitored for ataxia and mydriasis for 24 hours. The margin of safety for ivermectin in dogs is much narrower than in other species and unexpected toxicity can occur in any individual. One of our dogs, which had previously tolerated a 10-week course of oral ivermectin, was inadvertently given a single dose of 900 μg/kg for one day by her owner. The dog developed typical but fortunately reversible signs of toxicity within hours. Clinical signs of toxicity include ataxia, hypermetria, disorientation, depression, mydriasis, tremors, ptyalism, hyperaesthesia, blindness and coma.

Milbemycin oxime – A number of studies have reported the use of milbemycin oxime in dogs with generalised demodicosis in the USA, Australia and the UK.[3,4] A study performed here investigated the response of dogs with adult-onset demodicosis to treatment with milbemycin oxime.[5] Fifteen dogs, from 2 to 14 years old, were treated including three West Highland White Terriers and four Shih Tzus. Pododemodicosis was a major or a partial feature of the disease in seven dogs. None of the dogs had unmanageable underlying disease. Milbemycin was administered for 120 days at a dose rate of 0.5 mg/kg daily and thereafter at 2 mg/kg for up to a further 120 days if required. Adjunctive antimicrobial therapy was used as the case demanded. Three dogs were parasitologically in remission after treatment for 120 days but four dogs deteriorated during the trial. The remaining eight (53%) dogs were clinically controlled but were not cured.

In our experience, milbemycin is a useful drug for adult–onset demodicosis because of its minimal side effects, good efficacy (including cases of pododemodicosis) and its excellent owner compliance.

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Suggested reading

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Generalised demodicosis in the dog: the unresponsive or recurrent case

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Many cases of seemingly unresponsive or recurrent generalised demodicosis in the dog are not truly resistant at all. Instead, the apparent failure of therapy results either because a treatment program has not been devised or adhered to correctly or the animal has a concurrent underlying disease. The following discussion outlines five common reasons for apparent failure of therapy in generalised demodicosis in the dog.

1. Failure to identify and control concurrent superficial and/or deep bacterial pyoderma

Concurrent bacterial pyoderma is present in most cases of canine generalised demodicosis and will contribute to immunosuppression. The bacterial pyoderma may be superficial, with papules and pustules, or deep, with furuncles, oedema, cellulitis, severe pain and draining tracts that coalesce to form large exudative erosions and ulcers with adherent crusts. Many dogs with deep bacterial pyoderma are lethargic and febrile with a generalised lymphadenopathy and septicaemia.

Staphylococcus intermedius is the organism most often cultured from dogs with pustular demodicosis. Gram-negative organisms such as *Pseudomonas aeruginosa* and *Proteus mirabilis* are less frequently isolated but are commonly resistant to routinely prescribed antimicrobial drugs.

Routine cytologic evaluation of the contents of an intact pustule, bulla or exudate from a draining tract should be performed on every case of generalised demodicosis. A direct impression smear of a pricked lesion should be assessed for the presence of degenerate neutrophils, cocci and rod-shaped bacteria. Samples containing rods should be submitted for culture and susceptibility testing.

An antimicrobial drug with bactericidal activity that achieves good concentration in the skin may be empirically selected for the management of superficial bacterial pyoderma but the treatment of deep bacterial pyoderma should be based on culture and susceptibility testing of the contents of an intact bulla or tissue biopsy. Antimicrobial therapy must be administered at the appropriate dose rate for a minimum of four weeks for superficial bacterial pyoderma and six to eight weeks for deep bacterial pyoderma.

Topical antibacterial therapy with chlorhexidine or benzoyl peroxide shampoo is an important adjunct to antimicrobial therapy.

2. Failure to identify and manage a concurrent medical problem.

Several recent studies have highlighted the causal relationship between adult-onset demodicosis and the presence of concurrent systemic disease. Adult-onset demodicosis is defined as onset of disease after 12 months of age for small, medium or large breed dogs and after 18 months of age for giant breed dogs. Diseases causally associated with adult-onset demodicosis