Sebaceous adenitis management

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Sebaceous adenitis (SA) is an uncommon condition – thought to be an immune-mediated reaction – that leads to eventual destruction of the sebaceous glands. It is more frequently seen in dogs, but has been reported in other mammals, including cats and rabbits (Jazic et al, 2006; White et al, 2000).

Figure 1. Follicular casts present in association with sebaceous adenitis from an akita.

The disease is characterised by scale, follicular casts and eventual alopecia. SA can be localised or generalised, with both mild to severe forms recognised. The condition can result in significant distress for owners, and quick identification and diagnosis is imperative for a good outcome.

Two forms of SA exist – idiopathic or secondary to underlying disease. The latter can occur secondary to diseases such as demodicosis, feline acne, juvenile cellulitis (puppy strangles) and leishmaniosis (Miller et al, 2012; Bardagí et al, 2010).

Pathogenesis

Idiopathic SA is suspected to be an immune-mediated condition, but the exact pathogenesis is

unknown.

An autosomal, recessive mode of inheritance has been identified in akitas and poodles, suggesting genetic inheritance may also play a role in pathogenesis (Reichler et al, 2001; Scarff, 1994). SA is likely triggered by a cell-mediated response against an unidentified component of the sebaceous gland (Rybnicek et al, 1998). Other possible causes include abnormalities in lipid metabolism or storage, as well as keratinisation defects.

Topical oil and triglycerides have been used in mild cases, providing further suspicion lipid management could be involved in pathogenesis.

Keratinisation defects resulting in sebaceous gland obstruction, with resultant inflammation and destruction, have been suggested as a mechanism of SA (Miller et al, 2012). Vitamin A, which is essential to normal keratinisation of the skin and hair follicles, and synthetic retinoids have been used to treat SA (Lam et al, 2011).

Concurrent disease

It has been hypothesised SA is triggered after stress, such as systemic disease, general anaesthesia and illness (Reichler et al, 2001).

A study of Swedish dogs found 43% had concurrent disease, with hypothyroidism accounting for 36% and atopy for 17% (Tevell et al, 2008). However, some of these dogs may have been euthyroid, as only total T4 without thyroid-stimulating hormone was measured before instigation of thyroxine supplementation.

History and clinical examination

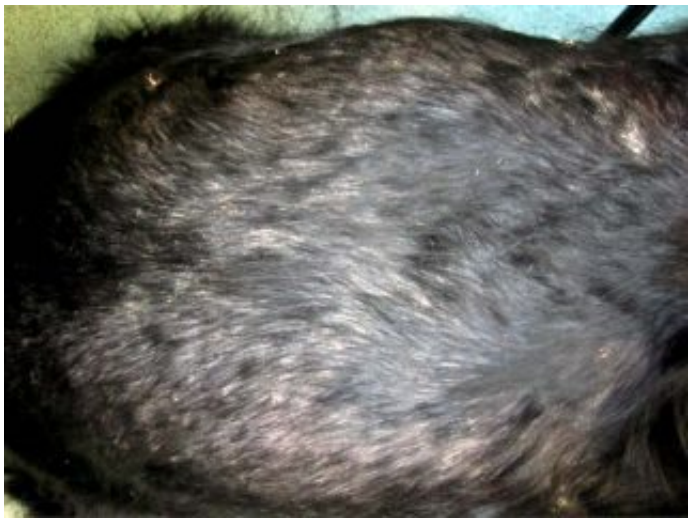

Figure 2. A two-year-old Bernese mountain dog with sebaceous adenitis showing diffuse scale, follicular cast and alopecia.

Both a detailed general and dermatological history are required. It has been identified in many breeds, with Lhasa apsos, springer spaniels, standard poodles, akitas and Samoyeds most frequently encountered.

Signalment is important in this disease, with males over-represented in most studies (Tevell et al, 2008; Frazer et al, 2010).

Pertinent questions include age of onset of disease, initial appearance and location, and coat changes. The clinical presentation is slightly different in long and short-coated breeds.

Short-coated breeds, such as the Vizsla, present with fine scale, with alopecia forming in circular patterns, resulting in a moth-eaten appearance. This is often around the head and cervical region. This can become multifocal and generalised over the dorsal part of the body (Miller et al, 2012; Lortz et al, 2010). Secondary pyoderma is rare in short-coated breeds.

In long-coated breeds, such as akitas, changes can be seen in the hair colour or type – from wavy to straight. The hairs become dull and brittle. Follicular casts may develop, formed by the accumulation of keratin and follicular material attaching around the hair shaft (**Figure 1**). Alopecia occurs due to a combination of the aforementioned factors, which often creates multifocal symmetrical alopecia (Miller et al, 2012; **Figure 2**).

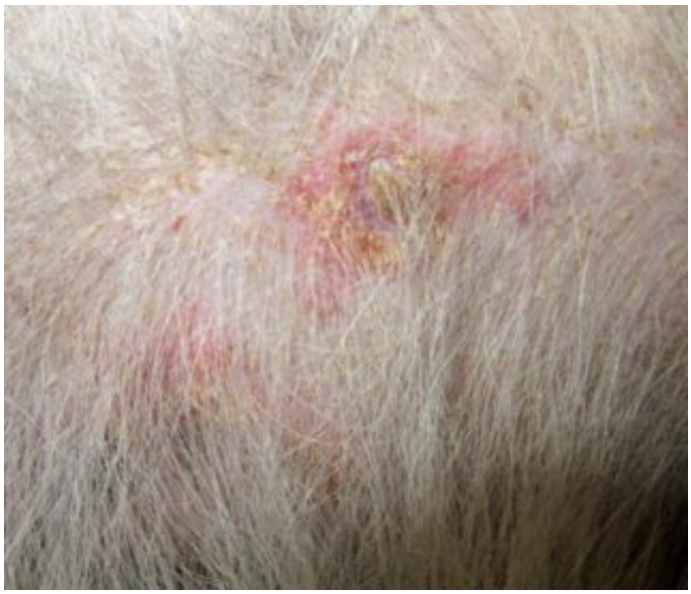

Figure 3. Pyoderma associated with sebaceous adenitis in an akita. Erythema, crusting and follicular casting are present.

Otitis externa may present in some patients. Pruritus may also present in some cases and appears to be more prominent in patients with pyoderma. Secondary bacterial folliculitis or furunculosis can also develop and be present in up to 40% of cases (Frazer et al, 2010; **Figure 3**), with up to 80% of akita owners reporting repeated episodes of pyoderma (Tevell et al, 2008).

Diagnostics

As these clinical signs can mimic several other diseases, routine dermatology diagnostics should be performed before considering more invasive tests.

Scale should be collected using acetate tape strips. Modified Romanowsky staining of the tape can then be evaluated for presence of secondary bacterial or yeast infection, or overgrowth. Unstained samples can then be assessed for ectoparasites, such as Cheyletiella, fleas, lice and Sarcoptes. Coat brushing and skin scrapes may also be performed to rule out Demodex and Sarcoptes parasitism.

Hair plucks should be taken from the areas of scale and alopecia, and examined for dermatophytosis, demodicosis and abnormalities of the hair shaft. Fungal culture should be carried out if any concern arises regarding dermatophytosis.

Definitive diagnosis of SA requires biopsy, which should be sent to a laboratory with a dermatohistopathologist. Biopsy can be performed in patients using sedation and local anaesthetic, and requires careful selection of lesions – early lesions with erythema and scale should be selected.

Skin should not be aseptically prepared as this can remove a wealth of information.

Biopsies should be performed using a 6mm to 8mm biopsy punch and the skin closed routinely. Biopsies taken of later lesions, such as those with mild alopecia, may be necessary in certain circumstances.

Before performing biopsy, a fine, permanent pen should be used on the skin to draw the direction of normal hair growth. Transverse sectioning of the hair follicles is required for proper examination. By marking the normal direction of hair growth, the sample can be processed correctly by a histopathology laboratory (Bond and Brooks, 2013).

Finally, a small section of biopsy should be processed for bacterial culture and susceptibility, and fungal culture, by a microbiology laboratory.

Histopathological findings can vary not only between breeds, but samples from the same patient, with different levels of inflammation present. Early lesions will often show severe orthokeratotic hyperkeratosis (Whitbread, 2014). Pyogranulomatous to granulomatous inflammation of the

sebaceous glands can be identified.

Secondary bacterial infection can also be present as intra-epidermal pustular dermatitis, folliculitis and furunculosis (Miller et al, 2012). Inflammatory changes in the glands are often not present in chronic cases, while no sebaceous glands are present in occasional cases.

Treatment

Due to the lack of full understanding of pathogenesis, many treatment options are described in the literature. Response to therapy can be unpredictable and many clinicians treat on the basis of personal preference.

Mild cases can be controlled with keratolytic shampoos, emollients or humectants. Keratolytic shampoos, such as those containing sulphur, salicylic acid or zinc, can be used. Oil soaks can be a useful adjunct therapy after removal of crust and scale by bathing.

A two-hour soak in baby oil, followed by thorough rinsing and application of 70% propylene glycol, was performed in one study. When used one to three times weekly and then tapered, it was found to be as effective as ciclosporin treatment alone (Lortz et al, 2010).

Figure 4. The Bernese mountain dog in **Figure 2** after four weeks of systemic treatment with ciclosporin, essential fatty acids and keratolytic shampoos.

Coconut oil can also be used as an alternative; however, this can be more difficult to apply and remove due to its consistency. Again, this requires bathing before and after to thoroughly remove any residual oil.

Essential fatty acids (EFAs), given both systemically or applied topically, seem to work in some patients (Tevell et al, 2008). Given the relatively benign side effects from this treatment, EFAs and topical treatment are often considered before immunosuppressive treatment is instigated.

Vitamin A, which is responsible for normal keratinisation and follicular maturation, has been used to

treat many keratinisation disorders. It has been used on its own and in conjunction with topical and/or systemic therapy. Varying degrees of success with this treatment have seen some owners report more than 25% improvement in the appearance of their dog, but others report no improvement. Not enough data exists to support or dismiss its use in treating SA (Lam et al, 2011).

Ciclosporin at 5mg/kg/24h alone, or with topical treatment, has been found to be an effective treatment of SA (Lortz et al, 2010; Linek et al, 2005; **Figure 4**). When both topical treatment of baby oil soaks, as aforementioned, and oral ciclosporin was used, a possible synergistic effect was seen (Lortz et al, 2010).

Prednisolone seems to be less effective as a treatment modality. The disease still progresses and loss of the sebaceous glands occurs (Miller et al, 2012). However, it can be used to control pruritus associated with secondary infection as required.

Rabbits and cats

Treatment of this condition in rabbits has been described using both topical treatments, vitamin A and synthetic retinoids, and systemic ciclosporin, with varying degrees of success (White et al, 2000; Kovalik et al, 2012).

SA is rare in cats. It has been reported in 10 shorthair kittens; however, no treatment regime has been published (Yager et al, 2012). One study found a five-year-old neutered male Norwegian forest cat was treated successfully with topical essential oils and vitamin E after oral supplementation with EFAs found no improvement (Glos et al, 2016).

Prognosis

Prognosis varies between individual cases and their response to treatment. Many cases do not fully resolve and long-term management is required. This can be time consuming and frustrating for owners. These cases often benefit from referral to a specialist dermatologist, if possible.

Euthanasia of patients with SA due to recurrent pyoderma does occur; therefore, prompt diagnosis and treatment of this disease should not be neglected (Tevell, et al, 2008).

- Some medications mentioned in this article are prescribed under the cascade.

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