Lick granuloma (LG) (also known as acral lick dermatitis or acral pruritic nodule; acral meaning ‘extremity’) is a common canine dermatitis wherein repetitive licking results in the development of a well circumscribed, raised, firm, alopecic, eroded to ulcerated plaque or nodule. Lesions are most commonly found on the dorsal aspect of the front limb, anywhere from the metacarpal area to the elbow. LGs are less commonly noted over the metatarsus or tarsal or tibial regions. Breed predispositions include the Doberman Pinscher, Labrador Retriever, Golden retriever, Great Dane, boxer, Weimaraner and Irish Setter.

The etiology of LG is multi-factorial. Primary factors are defined as those that initiate the licking. The most common is allergy, either atopy or food sensitivity. Although some Dermatologists find food sensitivity to be most commonly incriminated, in our experience, the overwhelming majority of patients are atopic. Other, more generalized signs of allergy may vary from severe to very subtle in a given individual. Other primary factors are uncommon to rare and include trauma, underlying bone or joint pain (fracture / arthritis), foreign body, peripheral neuropathy (e.g. secondary to cervical vertebral instability), parasthesia, fungal infection (dermatophytosis or deep fungal infection) and behavior problems. We would agree with others who suggest that having a lick granuloma as the only manifestation of a behavior abnormality is rare. Individuals with behavior problems that result in lick granulomas often also have other behavior problems, including separation anxiety, phobias, or other stereotypic behaviors including tail chasing, circling, fly biting or rhythmic barking. A ‘behavioral’ tendency to lick appears to be much more a perpetuating factor for an already existent LG.

Perpetuating factors are those that amplify the tendency to lick affected areas. They may keep the ‘lick cycle’ going, even if the primary factor is transient (e.g. transient ‘flare’ of allergy). They include:

1. Deep bacterial infection (deep bacterial pyoderma). This is the most common and important of the perpetuating factors. They may contribute very significantly to pruritus. In one study, 29/31 (94%) of dogs with lick granulomas had a deep bacterial component to their LG 1. The most common bacteria isolated were Staphylococcus (60%), Pseudomonas (8%) and Enterobacter (8%). 50% of cases were multi-drug resistant and 25% were methicillin resistant. Cultures from the surfaces of the lesions did not correlate well with those taken by tissue biopsy 1. This appears to be a strong argument for
culturing such lesions by biopsy or by swabbing the lesions after ‘squeezing’ them to bring exudates up from deep within the tissue.

2. Ruptured hair follicles (exposing free keratin to the dermis which is very irritating to these tissues) and hairs that are driven down into the lesion by self trauma are capable of causing significant inflammation which in turn serves as a significant perpetuator. In addition, apocrine glands also become very hyperptrophied, inspissated, dilated and may rupture. These secretions may illicit a significant inflammatory response.

3. Compulsive, behavioral component. Some of this compulsive behavior may be related to a transient release of ‘feel good’ endorphins associated with licking which, along with transient analgesia associated with licking are strong re-inforcements for the tendency to self traumatize. The release of proteases from damaged epidermal cells may also have a pruritogenic effect on naked nerve endings at the dermo-epidermal junctions. These factors all contribute to the development of a relentless itch-lick cycle. Environmental influences such as boredom or stress, confinement or interactive conflict may also be contributing factors.

The work-up of a patient with one or more lick granulomas should always include a thorough history and physical examination, especially looking for other evidence of allergic disease (e.g. seasonality of signs, presence of otitis externa etc.). Questions should also include those directed at assessing environmental influences (e.g. boredom etc.). If the history contains evidence of other behavior abnormalities (e.g. other steryotypic behaviors), then greater emphasis can be placed upon the LG also having a strong behavior component. The data base should include cytology of exudates that have been ‘squeezed’ from the depths of the lesion, hair plucks or scrapes for demodex and a fungal culture. The histologic appearance of LG is relatively characteristic. Skin biopsy would be indicated if the lesion appears at all atypical (to rule out some other pathologic process, e.g neoplasia or deep fungal infection). Strong consideration should be given to the early performance of a bacterial culture. This is especially true if the patient has been on significant systemic antibiotic therapy in the past, or the lesion is persisting in the face of current antibiotic therapy or if the lesion is severe. Cultures are best taken by deep tissue biopsy, or by swab after ‘squeezing’ the lesion to bring up exudates from deep within. Radiographs may be of value to look for underlying bone or joint problems, neoplasia or deep mycotic infections. It is common to see secondary peristeal reaction beneath more severe, active lesions. This is a product of chronic irritation of the overlying skin. In itself it is not a significant finding, other than attesting to the severity of the lesion and possible prognosis for effective medical management.

The therapy of lick granuloma should always involve removal or control (if possible) of the primary factor, treatment for secondary bacterial infection and breaking the itch-lick cycle.

1. Because the primary factor is usually allergy (atopy and/or food sensitivity), symptomatic allergy medication can be very beneficial in reducing the tendency to lick (e.g. course of oral glucocorticoid such as prednisone / prednisolone, starting at 0.5 - 1.0 mg/kg/day for 1-2 weeks, then gradually tapering).

2. Secondary bacterial infections are treated empirically with a good anti-staphylococcal antibiotic (e.g. cephalosporin, amoxicillin-clavulonate or clindamycin) or based on results of culture and sensitivity testing. Antibiotic therapy, if effective, should be maintained until at least a couple of weeks after resolution of the lesion (i.e. good hair regrowth noted on lesion).

3. Sock, or leggings or bandage or E-collar or a topical ‘lick’ deterrent to break the ‘itch-lick’ cycle.
For topical therapy we currently use a mix of HEET (Pain killing linament; a capsaicin containing product available from Medtech Laboratories) and bitter apple at a 1:2 ratio. This is applied directly on or around the granuloma three times daily.

4. +/- topical steroid to help resolve the inflammatory component of the problem more rapidly (e.g. Synotic, Pfizer; fluocinolone and DMSO; apply BID); use only if the patient is not able to lick at the lesion.

5. +/- Behavior Modification - if there is any suggestion that this could be a contributing factor, manipulating these behaviors would be in order (e.g. less confinement, more interaction with owner etc.).

If the problem is responsive to the above medications, but is recurrent (with or without concurrent allergic signs) or if the problem persists in the face of oral steroids and an appropriate antibiotic regimen and ‘less than ideal’ attempts to keep the patient from licking, then a restrictive diet trial should be performed to rule out a food sensitivity component to the problem. The diet trial should be of at least 8 - 12 weeks duration. Secondary bacterial infections must be cleared up early in the course of the diet trial.

For atopic individuals, control of the underlying atopy will be required to keep the LGs from being recurrent (e.g. antihistamines, glucocorticoids, cyclosporine, testing and hyposensitization).

Where a behavioral component is strongly suggestive or for the management of otherwise referactory cases, consideration can be given to behavior modifying drugs. Those that are used most frequently by the author are:

1. Amitriptyline - tricyclic anti-depressant; 2.2 mg/kg PO BID; side effects rare (lethargy, hyperactivity).

2. Clomipramine - tricyclic anti-depressant; started at 1 mg/kg/day; dose can be increased by 1 mg/kg/day every two to three weeks to maximum of 3 mg/kg/day; side effects include lethargy, anxiety, inappetance, dry mouth, vomiting and diarrhea.

3. Fluoxetine - serotonin reuptake inhibitor; 1mg/kg/day; side effects - lethargy, wheals and polyuria / polydipsia.

4. Hydrocodone - 0.25 mg/kg BID or TID.

Of the group, amitriptyline is least expensive and best tolerated, but also less effective. Chlomipramine has been the author’s ‘go to’ choice as a more dependably effective therapy. Trial period on any of these drug therapies is usually 6 - 8 weeks. Therapy is usually maintained until at least a couple of weeks beyond resolution of the LG.

Other therapies, including surgery, cryotherpay, laser ablation, radiation therapy and acupuncture have been reported to have variable success and have only been rarely used by the author.

References