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Hereditary alopecias - overview
Congenital alopecia occurs either at birth or within 2-4 weeks of life. Hereditary alopecia can occur at an early age or may have a tardive onset. Alopecia with a late onset may be more difficult to differentiate from endocrinopathies and other acquired alopecias.

Classic examples of hereditary alopecia are the hairless breeds. These dogs and cats have been purpose bred for a lack of a haircoat, but this does represent a hereditary disorder. Recognized breeds are mexican hairless, american hairless terrier, chinese crested dog, peruvian inca orchid and the sphynx cat. In the mexican hairless an autosomal dominant mode of inheritance has been demonstrated and in american hairless terriers it has been shown to be an autosomal recessive trait. Inherited alopecias have no known specific therapies. Dietary manipulations, bathing and nutritional supplements have been proposed but have not been shown to be effective. Melatonin has been used with some success in some of the acquired, endocrine and hereditary alopecias.

Diagnosis is usually based on clinical signs, signalment and biopsy. Biopsy samples should be taken from affected areas and “normal” areas for comparison.

Since there are no known cures the goal is to obtain an accurate diagnosis and educate the owner on care of the hairless skin. Generally speaking the hairs and sebaceous glands will be miniaturized and therefore the skin will be dry. Comedones may also form and may lead to secondary infections. Good gentle, cleansing, moisturizing and protection from the elements (cold, sun etc) should be recommended.

Specific disorders
Hair shaft disorders
Trichoptilosis- a longitudinal splitting of the hairs (split ends) may be an acquired disorder from over grooming, but has been reported as a hereditary disorder in three golden retrievers.

Medullary trichomalacia- is an unusual disorder where the medulla vacuolates. This may be an acquired disorder but was reported in six german shepherd dogs. Hairs on these dogs felt thicker and stiffer and broke easily. This will usually resolve with the next shed cycle but may recur.

Pili torti- has been recognized in cats and bull terriers. In cats all hairs are affected in the dogs only some hairs were affected.

Shaft disorder of abyssinian cats- this is a rare disorder that affects the primary hairs and whiskers. Hairs have a bulb like swelling usually at the tip of the hair. This is visible to the naked eye.

Congenital hypotrichosis
Congenital hypotrichosis- is a term used for animals that are born without their normal haircoat or who have non-color linked alopecia with in the first month of life. This has been described in american cocker spaniels, belgian shepherds, german shepherds, toy and miniature poodles, whippets, beagles, french bulldogs, rottweilers, yorkshire terriers, labrador retrievers, bichon frise, lhasa apsos and basset hounds. These dogs may develop hyperpigmentation and seborrhea and may also have abnormal dentition.

In cats congenital hypotrichosis has been described in birman, burmese, devon rex and siamese cats. An autosomal recessive mode of inheritance has been described in siamese and birman cats. The birmans were found to lack a thymus on necropsy and the burmese cats lacked claws, whiskers or lingual papillas.

Color-linked alopecias
Color-linked alopecias are inherited alopecias that occur at a young age in specific coat colors.

Black hair follicle dysplasia- occurs in bicolor or tricolored coats in the black haired regions only. The disorder appears to be an autosomal recessive disorder of pigment transfer and hair cuticles which leads to hair breakage. This disorder has been seen in the bearded collie, border collie, basset hound, papillion, saluki, beagle, jack russell terrier, american cocker spaniel, schipperke, cavalier king charles spaniel, dachshund, gordon setter, large munsterlander and pointer.
Clinical signs are usually first seen at 4 weeks of age. The first change may be a lack of luster in the black hairs. As the alopecia progresses an excessive scaliness may occur in affected areas.

Color dilute alopecia (color mutant alopecia)- occurs in
diluted coats (blue, fawn), but not all dogs with a blue or fawn coat will be affected. This disorder appears to be a polymorphic disorder of melanin transfer and storage leading to large clumps of melanin that break the hair cuticle. Recognized breeds affected are doberman pinchers, dachshunds, great danes, whippets, italian greyhounds, chow chows, standard poodles, miniature doberman pinchers, yorkshire terriers, silky terriers, chihuahuas, boston terriers, salukis, newfoundland, german shepherds, shetland sheepdogs, schnauzers, and bernese mountain dogs. A report of five weimaraners describes an alopecia that resembles color dilute alopecia. The first clinical signs are seen between 6 months and 3 years of age and often begin with a dry, dull coat, comedone formation and recurrent folliculitis. The pyoderma needs to be treated with appropriate antibiotics and antimicrobial shampoos.

Non-color-linked alopecias
Pattern baldness- occurs in specific locations regardless of hair color in certain breeds. There are three defined syndromes.

1 Pinnal alopecia is usually seen in dachshunds, but has been seen in other breeds and cats.
2 American water spaniels, irish water dogs, curly-coated retrievers and portuguese water dogs have a pattern alopecia that usually starts at about 6 months of age and affects the ventral cervical region, caudomedical thighs and tail. Concerted efforts by breed clubs have reduced the incidence of this disorder. 3) The most common form of pattern alopecia is seen in dachshunds, boston terriers, chihuahuas, whippets, manchester terriers, greyhounds and italian greyhounds. This is seen almost exclusively in female dogs and begins to be apparent at about 6 month of age. Alopecia affects the ventrum, ventral cervical region and post-auricular area.

Seasonal flank alopecia (cyclic flank alopecia)
Alopecia with irregular margins and hyperpigmentation occurs in the flank regions in the fall or spring. This can be recurrent or self-resolve and not recur again. This is seen in airedale terriers, english bulldogs, boxers, mastiffs, doberman pinchers, miniature poodles, bouvier des flandres, scottish terriers, staffordshire terriers, french bulldogs, affenpinschers and schnauzers. This disorder often responds to melatonin therapy 3-6 mg po q 8-12 hours.

Postclipping alopecia
Failure of the hair coat to regrow for as long as 24 months after clipping is relatively common in plush coated breeds, such as siberian huskies and chow chows. Most of these dogs will however regrow their hair given time.

Follicular lipidosis
Affects the red colored points of young rottweilers of either sex. Histopathology reveals hair matrix cells that are swollen and vacuolated. Special stains reveal the vacuoles to be lipid filled. Most of these dogs have recovered but there was one dog that had chronic renal failure and another reported with hypothyroidism.

References
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Hereditary/congenital diseases
Demodicosis is caused by a proliferation of demodex mites. This mite is a normal inhabitant of mammals skin, but the mite numbers can increase when there is immunosuppression or a hereditary defect of the immune system.

The akita breed of dog is affected by a number of distinct immune-mediated diseases, including thyroiditis, sebaceous adenitis, pemphigus foliaceus, uveitis, polyarthritis, myasthenia gravis, and uveodermatologic (uv) syndrome. This study confirmed loss of dla genetic diversity in the american akita dog in common with other pure breeds of dog and suggested a role for certain dla class ii gene alleles in the pathogenesis of uv. A mutation in the canine bhd gene is associated with hereditary multifocal renal cystadenocarcinoma and nodular dermatofibrosis in the german shepherd dog. Hereditary multifocal renal cystadenocarcinoma and nodular dermatofibrosis (rcnd) is a naturally occurring canine kidney cancer syndrome that was originally described in german shepherd dogs. From hum mol genet - dec 2003 - frode lingaas, kenine e comstock, ewen f kirkness, et. Al.