CASE I: N1400608 (JPC 4066457).

Signalment: Mature gravid female white-tailed deer (*Odocoileus virginianus*).

History: This doe was found dead, but was observed alive in the same yard the previous evening.

Gross Pathology: Approximately 95% of the skin was alopecic, hypopigmented, and covered by small coalescing crusts. A small amount of hair remained over the inguinal, axillary, and perianal regions and the distal extremities. There was consolidation and dark red to black discoloration of the right cranial lung lobe. The remainder of the thoracic and abdominal viscera was grossly normal.

Laboratory Results: None

Histopathologic Description: Diffusely, there is a defect in hair development. Hair shafts are frequently absent from follicles; remaining hair shafts are angular or kinked, attenuated, and fragmented, and rarely extend to follicular ostia at the skin surface. The hair cortex is composed of hyaline, relatively thinned keratin. Follicular infundibula...
Haired skin, white-tailed deer. Hair follicles are empty or contain fragmented, poorly formed hair shafts and keratin debris. Diffusely sebaceous glands are hyperplastic, and apocrine glands are dilated by excessive secretory product. HE, 55X).

are multifocally dilated and misshapen, and some are filled with excessive orthokeratin. Occasional hair bulbs and inferior portions of hair follicles are present at the level of the infundibular-isthmic junction. Sebaceous glands are multifocally hyperplastic, with ectatic ducts that contain fragments of keratin debris. There is mild dermal inflammation with few scattered lymphocytes and histiocytes. The epidermis is variably mildly acanthotic and hyperpigmented.

Contributor’s Morphologic Diagnosis: Haired skin: Follicular dysplasia, diffuse

Name of disease: Toothpaste hair disease of white-tailed deer

Contributor’s Comment: Toothpaste hair disease of the white-tailed deer is a rare condition sporadically affecting individuals with features of widespread alopecia and multifocal crusts. Previous cases have reportedly arisen in the Great Lakes region and the Mississippi Valley. Despite there being some knowledge of this entity for decades, little has been determined as to a cause, including an underlying genetic defect. Moreover, to date there are no cases described in the primary literature, and there are no similar reports in other cervid species. The likely cause of death in this case was attributed to the regional bronchopneumonia.

This condition calls into question the distinction between follicular dystrophy and dysplasia. A true follicular dystrophy suggests a degenerative process and possible association with ‘malnutrition’ of hair follicle cells. The result is defective and impaired development of hair in spite of a structurally normal hair follicle. In contrast, follicular dysplasias not only feature abnormal hairs but are also accompanied by abnormal hair follicles. While this entity has been classified as a form of follicular dystrophy in the past, we consider a
The diagnosis of follicular dysplasia appropriate in this case given the occasional abnormal morphology of hair bulbs, and their often aberrant location within the superficial dermis. In prior cases of toothpaste hair disease, a direct link with malnutrition, though suspected, has not been confirmed. A nutrient/mineral assay was not performed on tissues from the present case.

Follicular dysplasias are not uncommon in veterinary medicine. Color dilution alopecia and black hair follicular dysplasia are well-described conditions in various breeds of dog and cattle. The former has also been reported in the horse. Non-color dependent follicular dysplasias have also been reported for various dog breeds including Siberian huskies, Irish water spaniels, and Portuguese water dogs amongst others. The hairlessness trait of the Sphynx cat and Chinese crested and Mexican hairless dogs is another widely recognized form of follicular dysplasia (congenital hypotrichosis) brought about by the intentional propagation of spontaneous genetic mutations. Hair cycle disorders (including cyclic flank alopecia and follicular arrest) are also classified by some to be follicular dysplasias.

Follicular dystrophies are comparatively quite rare. There are several well-characterized follicular dystrophies in mice. Recently, a spontaneous autosomal recessive mutation was discovered on mouse chromosome 2, termed follicular dystrophy (fold), affecting the P/J mouse strain. A primary follicular dystrophy has also been described in a substrain of B6 mice. The phenotype is one of focal alopecia progressing to ulcerative dermatitis and scarring, and is attributed to polymorphism in alcohol dehydrogenase (Adh4) and differential expression of epithelial retinol dehydrogenase (DHRS9), leading to the impaired removal of excess retinol. Yet another follicular dystrophy phenotype has been defined in B6.C mice, resulting from the Angora mouse mutation brought about by a deletion in fibroblast growth factor 5 (Fgf5) gene.

Humans are also subject to follicular dystrophies. An entity known as acquired progressive kinking of hair is an androgen-dependent disorder that causes affected hairs of the scalp to resemble pubic hair in morphology. A subset of follicular dystrophies are known to stem from deficiencies in one of various nutrients, including copper (Menke’s kinky hair syndrome), sulfur (trichothiodystrophy), and amino acids (Netherton’s syndrome).

**JPC Diagnosis:** Haired skin and subcutis: Follicular dysplasia with sebaceous gland hyperplasia, duct dilation and hyperkeratosis.

**Conference Comment:** The conference description focused on the numerous empty and/or keratin filled, malformed, ectatic hair follicles as well as disorganization and hyperplasia of the sebaceous glands and ectatic sebaceous gland ducts. The moderator was careful to point out that dilated sebaceous gland ducts should not be
confused with dilated hair follicles as both are present in this case. Participants also noted the presence of dilated apocrine glands, malformed hair bulbs and shrunken, fragmented, malformed and hypereosinophilic hair shafts, with absence of normal hair shaft architecture, which led to interpretation as a form of congenital hypotrichosis.

Although uncommon, a similar condition has been reported previously in white tailed deer. In the other reported case, hair follicle density was normal, follicles were ectatic and either empty or contained keratin debris and hair shaft fragments, and apocrine ducts were dilated and hair bulbs were abnormal, similar to what was seen in this case. In that case there was normal hair present on the ventral thorax and sebaceous gland hypertrophy and hyperplasia was variably present. There was also mild epidermal hyperplasia and hyperpigmentation, similar to what is seen in this case. The authors of that manuscript went on to discuss the types of congenital hypotrichosis described in cattle including forms which are lethal, forms associated with dental abnormalities and viable hypo-trichosis, which shares many similarities with this case. Viable hypotrichosis is reported to affect Guernsey, Jersey, Holstein and Hereford cattle with an autosomal recessive mode of inheritance resulting in dysplastic hair follicles that don’t produce hair shafts, generalized alopecia and cystic apocrine glands. Ectodermal defects resulting in congenital alopecia can be restricted to the hair follicle, or also be associated with other ectoderm derived tissues (i.e. teeth, nails).

Formation of a hair follicle and hair shaft involves complex molecular signaling pathways that begin with formation placode, a condensed mesodermal structure which lies just below an epidermal invagination. The placode grows down into the mesenchyme which is followed by differentiation of the follicular mesenchyme, and formation of the dermal papilla and connective tissue sheath, which leads to formation of the hair bulb. The hair bulb is responsible for formation of the hair shaft. When the mesenchymal cells of the dermal papilla become enclosed by keratinocytes, formation of the hair shaft begins. Hair shaft formation is accomplished by the matrix keratinocytes of the hair bulb. Hair shafts are composed of a cortex, which is covered by a cuticle protecting the hair from damage, and many hair shafts have a pigmented medulla. Surrounding the hair shaft is the inner root sheath, which disappears at the level of the follicular infundibulum. The outer root sheath forms at the same time as the inner root sheath and hair follicle, but is not derived from matrix keratinocytes. Downgrowth of the outer root sheath pushes the hair bulb toward the subcutis while matrix keratinocytes are producing the hair shaft and inner root sheath, which grow toward the skin surface. As mentioned above by the contributor, hair follicle dysplasias, which involve defects in the hair follicle and shaft, are often differentiated from the alopecic conditions where the hair follicle appears normal, but the shaft itself is abnormal. Additionally, it is important to differentiate between alopecic conditions with a decreased number of relatively normal hair follicles, vs. alopecic conditions with a normal number of abnormal follicles. Congenital forms of alopecia may involve the epithelial and mesenchymal cells of the hair follicle, but may also involve follicular melanocytes, derived from neuroectoderm, and include color dilution alopecia and black hair follicular dysplasia in dogs.

Contributing Institution:
References:


CASE II: 3140429021 (JPC 4066087).

Signalment: 8-year-old, male, European shorthair domestic cat (Felis catus).

History: Firm, plaque-like thickening of the pinna which is growing.

Gross Pathology: The cut surface of the pinnal thickening is beige and firm.

Laboratory Results: None

Histopathologic Description: Pinna: The deep dermis of the pinna is multifocally thickened by large numbers of neutrophils and fewer lymphocytes and plasma cells in the dermis surrounding the abnormal auricular cartilage. The inflammatory in-
Pinna, cat. Multifocally, the pinna cartilage is surrounded and infiltrated by a cellular infiltrate which extends into the surrounding dermis (arrows). (HE, 6X)

Filtrate sometimes extends into the cartilage, which in some sections is occasionally split. The chondrocytes in these areas show degeneration and necrosis characterized by cytoplasmic hypereosinophilia and pyknotic or karyorrhectic nuclei. The necrotic cells are occasionally surrounded by small numbers of lymphocytes. Multifocally the cartilage is expanded by nodular areas that lack the normal architecture and are occasionally surrounded by dense connective tissue (fibrocartilagenous noduli). The deep dermis shows mild to moderate oedema, with dilation of dermal lymphatics, and there is multifocal proliferation of fibroblasts. The superficial dermis shows a moderate perivascular and periadnexal lymphoplasmacytic infiltrate with scattered neutrophils and mast cells. There is hyperemia of dermal capillaries.

**Contributor’s Morphologic Diagnosis:**

**Pinna:** Marked multifocal, chronic lymphoplasmacytic and neutrophilic chondritis and dermatitis with degeneration splitting and necrosis of auricular cartilage.

**Contributor’s Comment:**

Auricular chondritis has been reported in rats, mice, cats, dogs, in a horse and very rarely in cattle. It has been classified among the immune-mediated diseases due to similarities to rheumatoid arthritis and lupus erythematosus as well as its favorable response to immunomodulatory therapy. In humans, it manifests as part of relapsing polychondritis complex, a rare systemic autoimmune disease characterized by episodic destructive inflammation of cartilaginous tissues throughout the body especially those of the ear, nose, joints and respiratory tract. It has been reported rarely in cats and dogs and both ears are typically affected. Clinical signs include pain, swelling, erythema and deformation of the pinnae. Other organs such as joints, eyes and
heart may be present as well. Histologically, lesions consist of lymphoplasmacytic infiltrates and loss or necrosis of cartilage.\textsuperscript{3,6}

In cattle, one case report characterizes the lesions by lymphoplasmacytic infiltrates and presence of few macrophages within the cartilaginous plate of the pinna, which can be expanded by multiple basophilic cartilaginous nodules, vascularization and perivascular fibrosis. Chondrocytes in the center of the cartilaginous nodules may be swollen and found in clusters (proliferation). Rarely, low numbers of spindle cells surrounded by lacuna were present within these dense collagenous bundles (interpreted as early osseous metaplasia).\textsuperscript{6} In humans, histologically similar lesions may involve the pinnae, nose, trachea, joints, eyes and heart.\textsuperscript{3,6}

\textbf{JPC Diagnosis:} Ear pinna: Auricular chondritis, chronic-active, multifocal, moderate with cartilage degeneration.

\textbf{Conference Comment:} The conference histologic description of this well characterized lesion was very similar to the contributor’s description and emphasized the targeting of auricular cartilage. The cartilage was described as discontinuous, pale, degenerate and deformed with infiltration by a mixed inflammatory cell population, dominated by neutrophils. Multifocal thickening of the dermis and perichondral fibrous tissue coupled with granulation tissue associated with and surrounding foci of the most affected cartilage (a histologic feature that varies between slides) resulted in a brief discussion amongst conference participants regarding the chronicity of the lesion. In this case, the term “chronic-active” used in the JPC diagnosis denotes the chronic degenerative changes associated with the auricular cartilage as well as the active, on-going acute inflammatory component consisting heavily of neutrophils and lesser numbers of mononuclear cells.

Feline relapsing polychondritis, more commonly known as auricular chondritis, is an uncommon condition in cats with no sex predilection affecting predominantly young to middle aged cats, although the lesion has been documented in older cats as well. The current published veterinary literature is unclear regarding whether additional cartilage tissues may be involved; although there has been speculation regarding the presence of joint, ocular lesions and cardiac involvement which is documented in the corresponding human condition as discussed above.\textsuperscript{4} There is a case reported by Baba et al. which involved systemic joint and cartilage inflammation. In that case, the
Histologic ear lesions were similar to other cases reported in the literature. Additionally, costal cartilages were swollen, laryngeal cartilages thickened, and the articular cartilage of most peripheral joints eroded. There was also destruction of the subchondral bone, lymphocytic inflammation in the trachea and larynx and chondrolysis of the thyroid cartilage with associated inflammation. The costal cartilages were described as “irregularly hypertrophic” with bone formation and mixed inflammatory infiltration. Involvement of the respiratory tract and costal cartilage was not previously reported in cats. Uveitis was also reported in that case. Overall, the lesions described in Baba et al.'s case report were more similar to human relapsing polychondritis than other cases of feline auricular chondritis previously reported. However, there were also discrepancies with the human condition, including the nature of the joint lesions, which were more consistent with, and diagnosed as chronic progressive arthritis. Additionally, the cat also had lymphoma, which was considered as a possible cause of the inflammatory lesions in the joints / cartilage.\(^2\)

Histologic lesions in this case are similar to cases described in the veterinary literature and include inflammation and loss of basophilic staining of the ear cartilage with degeneration and necrosis. Fibroblast and capillary endothelial cell proliferation surrounding inflamed cartilage is also described, and is consistent with the presence of granulation tissue as previously discussed. The condition is most often bilateral, fever is reported in some cases and there has been no documented association with FeLV or FIV; unlike the condition in humans, it does not seem to have a relapsing nature. In some cases, cats have been documented to improve without treatment. Due to the rarity of the condition and the lack of thorough follow up in documented cases, the prognosis is unclear.\(^4\)

**Contributing Institution:**
Utrecht University
Department of Pathobiology
www.uu.nl/faculty/veterinarymedicine/EN/abs_services/vpdc

**References:**


**CASE III:** N9619660 (JPC 4033565).

**Signalment:** 2-year-old male ferret (*Mustela putorius furo*).

**History:** Soft subcutaneous mass on head caudal to the right ear growing over 2 months. Otherwise healthy.

**Gross Pathology:** 2 cm in diameter firm white-tan mass with overlying ulcer.

**Laboratory Results:** Fine needle aspirate of mass consisted only of blood.

**Histopathologic Description:** Expanding the dermis and subcutis, there is a well-demarcated, expansile proliferation of neoplastic spindle cells surrounded by a pseudocapsule of compressed adjacent connective tissue. Neoplastic cells are arranged in long interwoven bundles and streams supported by a fine collagenous stroma. Cells are spindle-shaped to strap-shaped with moderate to abundant eosinophilic fibrillar cytoplasm and an oval to irregular nucleus with coarsely clumped chromatin, occasional nuclear vacuoles, and one to multiple prominent nucleoli. There is moderate anisocytosis and severe anisokaryosis with occasional binucleation or multinucleation with lining up of nuclei, and the mitotic index varies depending on the section from 2-5 per 10 high power fields. There are few coalescing large areas of necrosis within the tumor (<50% of the neoplasm) and in some sections, there are small aggregates of lymphocytes within the peripheral aspect of the neoplastic proliferation. There is an overlying ulcer covered by a serocellular crust with fibrin and within the subjacent dermis, there are numerous necrotic and viable neutrophils. The surgical margins are clean (present in the majority of tissue sections).

**Immunohistochemistry** (Figure 2A and 2B): Nearly all of the neoplastic cells have diffuse strong cytoplasmic immunoreactivity for smooth muscle actin and approximately 60% of the neoplastic cells have weak to moderate cytoplasmic immunoreactivity for desmin.
Contributor’s Morphologic Diagnosis: Skin and subcutis (head): Piloleiomyosarcoma.

Contributor’s Comment: The histopathology is consistent with a mesenchymal neoplasm and the cellularity, pleomorphism and mitotic activity are consistent with a sarcoma. Based on the histomorphology and growth pattern of the cells, the immunohistochemistry findings, and the location within the dermis and subcutis, this tumor is consistent with a piloleiomyosarcoma. Piloleiomyosarcomas are neoplasms arising from the arrector pili muscles, which are the smooth muscle bundles supporting the hair follicle and allowing for piloerection. Tumors arising from the arrector pili muscles have been reported in ferrets,1,5,7 as well as in dogs and cats.1-3 The majority of cases in ferrets have been piloleiomyosarcomas, with fewer reports of benign piloleiomyomas.4

In ferrets, piloleiomyosarcomas most commonly arise from the head and trunk, with fewer cases from the limbs, and may have surface ulceration.5,7 Histologically, despite being well-demarcated, the neoplastic cells have significant nuclear pleomorphism, and reports vary regarding mitotic activity, which may be uncommon (1-2 per 10 high power fields)1 or more frequent (≥ 2 per 10 high power fields).5 The nuclear pleomorphism7 or increased mitotic activity (mitotic index of ≥ 2)5 are the foundation for the diagnosis of malignancy in these cases. Foci of lymphocytes may be seen at the periphery of the neoplasm7 and areas of necrosis within the tumor may be identified,5 as in the current case. Direct connection of the tumor with the adjacent arrector pili muscles may or may not be found,5,7 and is not clearly present in this case. The majority of piloleiomyosarcomas in ferrets are positive for desmin and smooth muscle actin with immunohistochemistry5,7 (Figure 2A and 2B). The prognosis for piloleiomyosarcomas in ferrets is good following complete surgical excision with no reports of metastasis and good long-term survival in those cases with follow-up.5,7 Follow-up is not available for the present case.

Ferret, head. Neoplastic smooth muscle cells exhibit marked anisocytosis and anisokaryosis as well as multinucleation. (HE, 320X)
Neoplastic cells show strong diffuse cytoplasmic positivity for desmin (A), and multifocal moderate cytoplasmic positivity for smooth muscle actin. (Photo courtesy of: Animal Medical Center, 510 East 62nd Street, New York, NY 10065 www.amcny.org

In dogs and cats, general criteria to support a diagnosis of cutaneous leiomyosarcoma, as compared to a benign leiomyoma, include mitotic index of $\geq 1$, evidence of invasion, and/or necrosis within the tumor.\(^1\) In dogs and cats, piloleiomyomas and piloleiomyosarcomas are considered likely to be cured by complete excision, and local recurrence or metastasis of cutaneous leiomyosarcomas in general is uncommon.\(^1\) - 3

In humans, leiomyosarcomas arising from the arrector pili muscles have a moderate local recurrence rate of 30% and metastasis is not reported.\(^7\)

In conclusion, this case is a classic example of a piloleiomyosarcoma in a ferret and has many of the characteristic gross and histologic features reported with this entity.

**JPC Diagnosis:** Haired skin and subcutis: Piloleiomyosarcoma.

**Conference Comment:** The conference description was very similar to the contributor’s histologic description above. The neoplastic cell nuclei were described as cigar shaped with blunted ends and the cells themselves were described as strap-shaped, which is often a feature used to describe rhabdomyosarcomas. Eosinophilic nuclear vacuoles were also discussed and described as cytoplasmic invaginations, although their actual nature/origin is unclear. There was spirited discussion on the designation of the neoplasm as a piloleiomyosarcoma as opposed to piloleiomyoma. While the cellular characteristics of malignancy, such as bim- and multinucleation, anisocytosis/anisokaryosis, and a high mitotic rate is evident in this neoplasm and well-documented in the literature, metastatic potential is extremely low, and has not been described in the literature.

Cutaneous smooth muscle tumors can also arise from vascular smooth muscle and deep dermal smooth muscle of the genital area. Vascular origin smooth muscle tumors are termed angioleiomyoma/angioleiomyosarcoma. Regardless of origin, the tumors have similar histologic characteristics. Anatomic location can help differentiate these tumors, such as location in the genital area, or other features such as entrapment of hair follicles within the neoplasm, contiguity with erector pili muscle, or the presence/absence of a prominent vascular component may also be helpful.\(^5\) Other neoplasms involving the skin of ferrets include mast cell tumors, basal cell tumors and neoplasms of apocrine sweat
glands. Mast cell tumors are most common on the head, neck, shoulders and trunk but can occur at any location. Tumors of basal cell origin (most commonly sebaceous epithelioma of the skin can also occur at any location and are benign. The most common malignancy of the skin of the ferret are those of apocrine glands, which are most often seen around the prepuce and vulva, but may also be seen on the head and neck. Other less common tumors in the skin of ferrets include squamous cell carcinoma, cutaneous lym- phoma, hemangioma/hemangiosarcoma, and fibroma/fibrosarcoma.6

**Contributing Institution:**
Animal Medical Center
510 East 62nd Street
New York, NY 10065
www.amcny.org

**References:**


**CASE IV**: B14-15532 (4073336).

**Signalment:** 1-year-old spayed female golden retriever (*Canis familiaris*).

**History:** The dog has an acute history of skin lesions that were noticed 12 to 14 hours prior to examination by a veterinary dermatologist. One week prior to presentation, the local veterinarian gave the dog a rabies vaccine, and no abnormalities were noted on physical exam. Three days prior to presentation, the dog was routinely bathed at a commercial grooming facility. On physical examination, the dog was febrile (104°F), lethargic, and painful and had a mild neutrophilia with left shift. Two 6-mm punch biopsies were taken from the dorsolumbar skin via local anesthesia.

**Gross Pathology:** Coalescing erythematous to hemorrhagic papules and pustules on the dorsal midline.

**Laboratory Results:** None
Haired skin, dog. The skin of the dorsal midline was covered with hemorrhagic papules and pustules. (Photo courtesy of: University of Pennsylvania, School of Veterinary Medicine, Department of Pathobiology http://www.vet.upenn.edu/diagnosticlabs)

Histopathologic Description: The main histologic change is multifocal outer root sheath necrosis with suppurative inflammation and acute hemorrhage in the superficial and mid-dermis. Dense localized aggregates of neutrophils and fewer macrophages surround adnexal units and are admixed with free keratin and erythrocytes. The suppurative aggregates form large subepidermal pustules. The epidermis is expanded by spongiosis with mild acanthosis. There is mild superficial dermal edema.

Contributor’s Morphologic Diagnosis: Furunculosis, necrosuppurative and hemorrhagic, acute, multifocal, severe.

Etiologic diagnosis: Bacterial furunculosis

Name of disease: Post-grooming furunculosis

Contributor’s Comment:
The clinical and histologic features are typical of “post-grooming furunculosis” (PGF). This acute and severe inflammatory reaction is associated with the use of contaminated grooming products. While contaminated shampoos or conditioners are the most common causes, vigorous brushing (e.g. coat stripping), whirlpools, and contaminated surgical scrub have also been documented. Pseudomonas spp. is the most common isolate from the skin. In some cases, the bacteria can be cultured from both the skin and the affected product.

The clinical lesions typically arise on the dorsal midline as hemorrhagic pustules and crusts. The dogs may be febrile and very painful on thoracolumbar palpation. In dogs with a dense or thick haircoat (e.g. Golden retriever), the lesions may be missed on initial examination. In fact, some patients have been worked up for discospondylitis, back pain, tick borne diseases or pancreatitis.

In contrast to other causes of furunculosis (e.g. demodicosis, dermatophytosis, staphylococcal infection), PGF is acute. The outer root sheath is necrotic rather than hyperplastic and the rupture occurs in the superficial dermis. Furthermore, the inflammation is hemorrhagic and fibrinosuppurative rather than pyogranulomatous. Bacteria may be difficult to discern on H&E and Gram stain as they are often small gram negative bacilli.

JPC Diagnosis: Haired skin: Furunculosis, superficial, necrosuppurative and hemorrhagic, acute, multifocal to coalescing, marked.
Conference Comment: The presence of erector pili muscles allows localization of the section to skin of the back/dorsum. At subgross magnification, a distinctive histologic pattern is identified characterized by inflammation targeting the hair follicle in the superficial dermis. A coagulum of predom-inantly degenerate neutrophils, fibrin, hemor-rhage and debris overlies the epidermis; folliculosebaceous units are infiltrated by neutrophils with fewer macrophages and plasma cells admixed with hemorrhage and edema within the superficial dermis and extending into the mid dermis. The infiltrate results in a mural folliculitis with progression to furunculosis. The inflammatory milieu associated with the corresponding furun-culosis contains basophilic fragmented ma-terial, interpreted as keratin, along with free necrotic keratinocytes which can be differentiated from macrophages by the distinctive rounded cellular margins in the former. The moderator noted that in many inflammatory conditions targeting the hair follicle, the follicular epithelium is typically hyperplastic which is not a microscopic feature observed in cases of PGF.

The superficial nature of the lesions, acute onset, hemorrhage and lesions isolated to the dorsal trunk can help differentiate this condition from other causes of folliculitis and furunculosis in dogs. The predilection for lesions over the dorsum in PGF is postulated to be associated with the nature of grooming or bathing activities that may concentrate brushing and shampooing efforts on this area. The dorsum also has increased hair density and hair shaft size. Cytologic features of im-pression smears may include neutrophils and macrophages with or without eosinophils, red blood cells, and intracellular or extracellular bacteria. Clinicopathologic abnormalities associated with this condition may include neutrophilia, with or without a left shift, monocytosis, lymphopenia and mild thrombocytopenia. These abnormalities, when
considered with the presence of fever, may suggest a systemic inflammatory response.\textsuperscript{1}

\textit{Pseudomonas aeruginosa}, a gram negative bacillus, is the most common bacterial agent associated with post-grooming furunculosis. \textit{Pseudomonas aeruginosa} is a common bacterial contaminant associated with water and can survive in the presence of some disinfectants. Other bacteria, such as \textit{Serratia marcescens} and \textit{Burkholderia cepacia}, are associated with this condition. In the veterinary setting, \textit{S. marcescens} has been implicated as a contaminant of intravenous catheters, and \textit{B. cepacia} has been documented as a contaminant of ear cleaning solutions.\textsuperscript{1}

\textbf{Contributing Institution:}
University of Pennsylvania, School of Veterinary Medicine, Department of Pathobiology
http://www.vet.upenn.edu/diagnosticlabs

\textbf{References:}

